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Lumbar disc herniation. From epidemiology and neuroimaging to operatory management and postoperative complications

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ABSTRACT

Lumbar disc hernia represents one of the most frequent neurosurgical pathologies, consisting of a posterior migration of the nucleus pulposus which protrudes through the surrounding protective connective tissue. The patient usually exhibits leg pain, paraesthesia or a variable degree of discomfort at the lower extremities, those clinical characteristics are correlated to the extent of nucleos pulposus protrusion. Regarding neuroimaging diagnosis, the gold standard of evaluation is Magnetic resonance imaging in different sequences which highlights a precise topography of the herniated disc. Currently, in a well-digitalized era, machine learning and deep learning algorithms are used to assess the sensibility of detecting a lumbar disc hernia, with promising results. In those cases requiring surgical intervention, besides the open lumbar microdiscectomy, a less invasive surgical approach regularly used in the medical practice is endoscopic lumbar discectomy, being limited to cases where a wide intraoperative perspective is not necessary. In the actual neurosurgical management, postoperative complications following lumbar disc hernia surgery are rare and accessible to manage.

GENERAL DATA

Lumbar intervertebral disc herniation is characterized by the disruption of the annulus fibrosus, attributed to a multitude of factors including lumbar degeneration and sustained strain. This pathological condition leads to the lateral or posterior displacement of the nucleus pulposus, which extends significantly laterally or directly posteriorly through the compromised annulus.

Lumbar Disc Herniation (LDH) emerges as a predominant etiology for leg pain, with clinical presentations encompassing a spectrum from mild to severe discomfort in the lumbar region and lower extremities, muscular spasms, sciatica, paresthesia, and reduced muscular strength in the lower limbs. Exceptionally, it may precipitate acute cauda equina syndrome. Activities such as sneezing, coughing, or bending from the waist have been documented to intensify discomfort, adversely affecting the patient's clinical status. Therapeutic approaches to LDH predominantly include conservative management and surgical intervention. Surgical procedures aim to alleviate the pressure exerted by the protruding lumbar discs; nevertheless, they are not devoid of significant risks, including the potential for substantial trauma and the emergence of subsequent symptomatic manifestations (1,2). In contrast, conservative treatments may not furnish substantial pain alleviation in certain instances, with prolonged conditions potentially culminating in complications such as uroschesis or foot-drop.

Contemporary classification paradigms for LDH are predicated upon radiographic findings and pathomorphological characteristics. LDH can be categorized into three distinct types—central, paramedian, and foraminal—based on the specific location of disc protrusion. Furthermore, the degree of protrusion allows for subclassification into bulging, protrusion, or extrusion categories. Additionally, distinctions are made between nonruptured, ruptured, and sequestered discs, guided by the surgical pathomorphological findings (3,4).

INCIDENCE AND PATHOPHYSIOLOGY

Low back pain (LBP) represents a significant public health issue, exerting a profound impact on a vast demographic, particularly among individuals engaged in extended periods of sitting. Individuals afflicted with LBP often modify their movement patterns as a compensatory mechanism for the diminished functional mobility, employing various strategies (5). This modification potentially leads to either localized or widespread musculoskeletal strain, which is postulated to contribute causatively to the intensification of back-related maladies or discomfort (6).

Contemporary estimations concerning the incidence, prevalence, and disability-adjusted life years (DALYs) associated with LBP denote 245.9 million incidences annually (accounting for 3.2%; ranked as the 15th leading global cause), 577.0 million cases (7.6%; also the 15th leading global cause), and 64.9 million DALYs (representing 2.6% of all DALYs; the 6th leading global cause), respectively. These metrics have exhibited an approximate 50% escalation over the preceding two decades. The overall burden of LBP cases is slightly more prevalent in women than in men, demonstrates a gradual escalation commencing from birth, peaks during the 40–50-year age range, and subsequently exhibits a gradual decrease (7).

Lumbar disc herniation (LDH) constitutes a significant source of morbidity and substantially influences worker compensation claims. The incidence rate of LDH ranges from 1–3%, predominantly impacting male subjects (with males experiencing double the frequency of females) and individuals within the age bracket of 30–59 years (8,9). Notably, LDH prevalence is higher in the lower lumbar spine compared to the upper or middle sections; however, the incidence of herniation at upper levels tends to increase with advancing age (10).

In elderly individuals who have experienced falls, the displacement of the center of gravity from the vertical axis directly above their feet—particularly when elevated on their toes—differs significantly from that of healthy counterparts. With age, the restriction of trunk movement becomes more challenging, leading to an enhanced risk of imbalance. Moreover, medial-lateral (ML) motion is particularly vulnerable to external disturbances and necessitates the activation of dynamic feedback mechanisms, potentially involving visual, vestibular, and other sophisticated central nervous system functions, to ensure stability. The functional decline in older adults complicates the maintenance of balance in the ML direction, especially when faced with unexpected disturbances (11).

Mechanical stress is a pivotal factor in the genesis of disc herniation and the degeneration of facet joints. The persistent application of mechanical stress on one side may lead to unilateral symptoms and asymmetrical deterioration of the intervertebral joints and discs. Additionally, the onset of intervertebral joint arthritis can be prompted by routine activities. For instance, right-handed men are more likely to rotate their spine to the left while engaging in walking or seated activities, suggesting a correlation between the preferred direction of spinal rotation in daily activities and dominant hand usage (8). Previous investigations have highlighted that the left and right sides of the body exhibit varying degrees of lateral flexion and lumbar spine rotation, which implies potential distinctions in the characteristics of left and right LDH, although no disparities between left and right-sided LDH have been conclusively identified.

NEUROIMAGING: CURRENT AND EMERGING TECHNIQUES

The determination of therapeutic strategies for individuals with intervertebral disc herniation (IDH) is predicated upon clinical assessments and diagnostic imaging results. An initial approach of conservative management is advocated for a duration of 6–8 weeks for patients diagnosed with IDH. Should there be an inadequate response to conservative measures, surgical intervention may be contemplated, with magnetic resonance imaging (MRI) being systematically employed to evaluate the existence of nerve root compression (12).

Research indicates that approximately 10–40% of patients do not experience a marked improvement in symptoms post-lumbar disc surgery, despite advancements in diagnostic and surgical methodologies (13). The suboptimal results observed postoperatively are attributed predominantly to diagnostic inaccuracies, rather than the surgical procedure itself or its associated complications (14). The presence of discrepancies in the interpretation of spinal MRI scans can detrimentally impact therapeutic decision-making, leading to suboptimal clinical management in cases of erroneous positive or negative diagnoses of nerve root compression. The inconsistency in interpreting MRI findings may further complicate the establishment of a correlation between specific imaging features and patient prognoses.

Consequently, it is imperative to comprehend the variability in MRI interpretation among individuals considered for lumbar disc surgery, to enhance diagnostic accuracy and inform optimal treatment pathways. MRI and computed tomography (CT) scans now possess the capability to delineate disc herniations in both intraforaminal and extraforaminal locations with high fidelity. While CT imaging is less efficacious than MRI in identifying radicular compression and exhibits inferior resolution for spinal and paraspinal soft tissues, it demonstrates proficiency in the detection of osteophytes and calcifications (15).

Conventional MRI protocols often do not prioritize the imaging of extraforaminal regions, and visualizing this area, especially at the L5-S1 level, poses challenges due to the overlapping bony structures of the sacral alae and iliac bones. Additionally, degenerative alterations at the L5-S1 disc, which frequently result in a reduction of disc height, complicate the imaging process. Inaccuracies in MRI techniques frequently lead to misdiagnoses. For accurate detection of subtle disc margin aberrations and differentiation of genuine root dislocations from benign asymmetries in root positioning between the two sides, axial slices should be aligned parallel to the intervertebral disc on the sagittal plane. Furthermore, to identify far-lateral herniation, imaging protocols should include paracoronal sections (angled between 15 to 30 degrees) and sagittal sections that extend significantly laterally, covering the entire foramina length. The use of contrast agents is generally not obligatory, although contrast-enhanced imaging may be necessary to differentiate between a sequestered disc fragment and other conditions such as schwannomas. In such instances, fat-saturation pulse T1-weighted spin-echo sequences alongside axial and sagittal T1-weighted spin-echo sequences are recommended. Typically, the sequestered fragment exhibits peripheral enhancement, likely indicative of an inflammatory response in the surrounding tissue (16).

Following images highlights different levels of lumbar disc hernia using MRI in sagittal and axial sections (Fig. 1, Fig. 2, Fig. 3, Fig. 4).

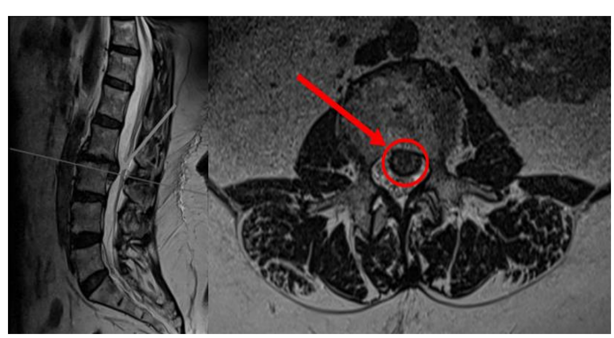

Figure 1. Sagittal section (left) and axial section (right) of a LDH at the level of L2-L3 (Personal case of Prof. Dr. A. V. Ciurea).

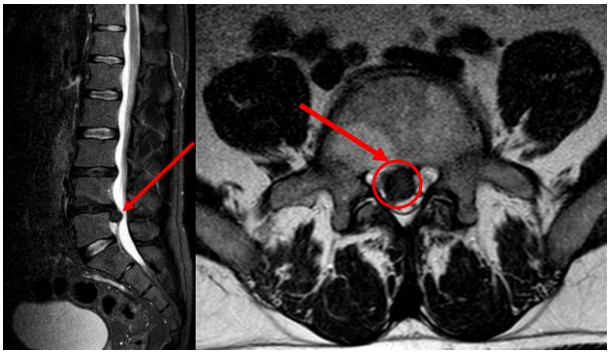

Figure 2. Sagittal section (left) and axial section (right) of a LDH at the level of L4-L5 (Personal case of Prof. Dr. A. V. Ciurea).

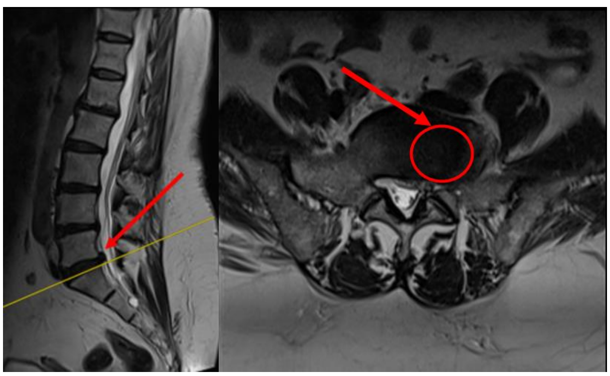

Figure 3. Sagittal section (left) and axial section (right) of a LDH at the level of L5-S1 and spinal stenosis (Personal case of Prof. Dr. A. V. Ciurea).

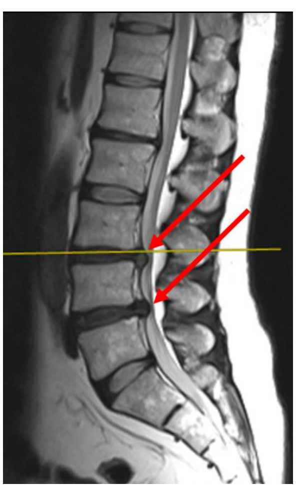

Figure 4. Sagittal section of a multilevel LDH, L3-L4 and L4-L5 (Personal case of Prof. Dr. A. V. Ciurea).

Deep learning architectures are engineered for the rapid and precise identification of images, showcasing their efficacy in enhancing the quality of medical diagnostics within clinical environments. These models significantly augment diagnostic processes and assist healthcare professionals in detecting lesions that may otherwise go unnoticed. Deep learning is particularly beneficial in the analysis of large-scale, repetitive tasks involving the recognition of biomedical imagery. Utilizing sophisticated graphical processing units (GPUs), these methodologies facilitate the computation of intricate features and the automatic classification or recognition of objects within images. Currently, the availability of validated diagnostic MRI images for lumbar vertebrae datasets is limited, underscoring the necessity for the expansion of deep learning models and the enlargement of lumbar MRI data collections (17).

Beyond mere image identification, deep learning is employed to efficiently and iteratively discern critical elements within biomedical images, such as the detection of anomalous and potentially malign lesions throughout the diagnostic evaluation process. Region-based Convolutional Neural Networks (RCNNs) introduce innovative techniques for object detection within images, utilizing a designated object region proposal strategy for CNN training followed by object classification. Subsequent iterations aimed at enhancing processing efficiency, namely Fast-RCNN and Faster-RCNN, strive to refine these complex object detection methodologies, yet challenges persist in achieving optimal deep learning model efficiency. The performance of deep learning is influenced by several factors, including the paucity of images within datasets, which may precipitate overfitting or underfitting scenarios. Previous research has incorporated data augmentation strategies to mitigate data imbalances and minimize overfitting risks in deep learning applications (18). Common data augmentation techniques employed in deep learning encompass Flips, Gaussian Noise, Jittering, Scaling, Powers, Gaussian Blur, Rotations, and Shears (19).

The delineation and pinpointing of distinct discs constitute critical operations in the computer-aided diagnosis of disc herniation. Over the recent half-decade, methodologies rooted in deep learning have redefined benchmarks across numerous domains of computer vision and pattern recognition studies. The aim of this research is to forge an automated framework leveraging a deep convolutional neural network (CNN). This network is designed to analyze input from MRI across various contextual scales, thereafter integrating the extracted high-level features. Such a process amplifies the network's proficiency in identifying discs within the lumbar spine region (20).

OPERATORY MANAGEMENT

Since the advent of the first open discectomy over half a century ago, there has been a significant evolution in surgical methodologies and technological advancements, culminating in procedures that are increasingly minimally invasive. In 1967, Yasargil introduced the utilization of a microscope for discectomy, leading to the development of microdiscectomy (MD) (21). A further reduction in invasiveness was achieved through the introduction of the tubular retractor (TD), which employs a transmuscular route to access the lamina (22). Another innovative approach is the percutaneous discectomy (AUTD), which adopts a posterolateral route. Initially, this procedure was conducted with fluoroscopic guidance alone, but the subsequent integration of endoscopic techniques facilitated the execution of percutaneous endoscopic discectomy (PED) under direct visualization (23).

The primary objective of these surgical interventions remains the excision of disc herniation, typically involving an incision of the disc to extract the herniated material along with the nucleus pulposus contained within the annulus fibrosus (24). Such interventions usually result in the prompt alleviation of radiating leg pain for the majority of patients. However, the degenerated disc and the annular defect are not addressed, which may contribute to a relatively elevated rate of reoperation due to recurrent disc herniation, as additional nucleus material may extrude through the annular breach (25). Previous studies have established a correlation between the size of the annular defect and an increased risk of reherniation, with reported incidences of reherniation varying significantly across studies, ranging from 3-18% (26). A radical approach to mitigate reherniation involves the extensive removal of the nucleus pulposus. However, aggressive discectomy has been associated with a drawback of heightened back pain, likely attributable to expedited disc degeneration (27).

In the 1970s, Caspar and Yasargil pioneered the technique of open lumbar microdiscectomy (OLMD) as a therapeutic intervention for LDH. Characterized by its minimally invasive nature, involving smaller incisions, OLMD demonstrated efficacious outcomes and diminished surgical trauma for patients (28), (29). Consequently, OLMD has been established as the benchmark surgical procedure for the treatment of symptomatic LDH. Despite its successes, OLMD is associated with certain drawbacks, including muscle damage, the necessity for partial laminectomy, and nerve retraction, which may elevate the risk of postoperative lumbar instability and symptomatic epidural scar formation (30,31).

In the early 1990s, in pursuit of enhancing surgical outcomes and minimizing complications associated with LDH interventions, the percutaneous endoscopic lumbar discectomy (PELD), employing either a transforaminal or interlaminar approach, was introduced (32,33). PELD has gained popularity over time, with a substantial body of research indicating its comparability to OLMD in terms of patient outcomes, alongside benefits such as reduced soft tissue damage and improved conservation of bone structure (34,35,36).

Although the incidences of complications common to OLMD, like postoperative spinal instability and epidural scarring, are infrequent with PELD, the technique is not without its challenges (37,38). A significant limitation of PELD is its steep learning curve and the potential for serious complications. Moreover, cases of incomplete decompression during PELD procedures are not rare, necessitating a meticulous morphological assessment of each LDH case prior to opting for PELD as the treatment of choice.

Full-endoscopic spine surgery (FESS) has seen significant advancements over the last thirty years, driven by the evolution of both the instruments employed and the procedural techniques. Initially, full-endoscopic lumbar discectomy (FELD) was designed for implementation via a posterolateral or transforaminal entry. However, the posterolateral route encounters specific challenges at the L5-S1 level, such as a pronounced iliac crest, the least expansive intertransverse space, and a relatively constricted foramen in comparison to superior vertebral levels (39,40). Consequently, interlaminar endoscopic lumbar discectomy (IELD) emerged as a viable solution to navigate the complexities associated with disc herniation at the L5-S1 juncture (41,42). A distinctive aspect of IELD is the likelihood of encountering neural structures prior to reaching the herniated disc, as opposed to the transforaminal approach where the nerve is typically accessed post-herniation removal. Therefore, IELD necessitates precise surgical techniques to mitigate the risks of neural damage and herniation recurrence.

The administration of anesthesia in IELD procedures can be tailored to local, regional (epidural), or general modalities, contingent upon the surgical complexity, the degree of nerve manipulation required, and the surgeon's proficiency (43). General anesthesia is particularly advantageous for securing the airway in patients positioned prone. Procedures characterized by extensive bone removal or spanning multiple vertebral levels may prolong surgery duration, particularly for surgeons with limited experience in these complex scenarios.

A myriad of etiological factors contribute to the persistence of low back pain, encompassing conditions such as spinal stenosis (SS), disc herniation, facet joint pathology, sacroiliac joint dysfunction, adjacent segment degeneration, ligamentous pathology, and failed back surgery syndrome (FBSS) (44,45). Addressing back and leg pain subsequent to spinal surgical procedures presents a formidable challenge. The International Association for the Study of Pain characterizes FBSS as the persistence or recurrence of low back pain, with an undetermined origin, in the same anatomical region, despite the surgical intervention. The genesis of FBSS is multifaceted, influenced by a spectrum of preoperative, intraoperative, and postoperative variables (46). While the precise cause of FBSS remains elusive, a multitude of factors are implicated in its development (47).

Post-surgical epidural adhesions are believed to significantly contribute to the development of epidural fibrosis, a condition associated with FBSS (48,49). Numerous studies have validated the efficacy of percutaneous epidural neuroplasty (PEN) in patients afflicted with FBSS and SS (50,51,52,53). PEN has been recognized as an effective intervention for managing intractable back and leg pain that remains unresponsive to conventional therapeutic approaches, including epidural steroid injections (54).

A treatment modality that occupies the therapeutic niche between conservative management and the administration of epidural steroid injections has been examined in various studies. These studies suggest that between 50% and 87% of patients experience short-term relief (approximately 3 weeks) from symptoms following epidural steroid injection therapy (55,56,57,58). The application of epidural steroid injections is recommended for patients experiencing acute radiating pain and neurogenic claudication that significantly impede daily activities, despite the utilization of analgesics and rest, which are anticipated to mitigate symptoms (59). Moreover, recent research has explored the efficacy of using ropivacaine and dexmedetomidine, in addition to epidural neuroplasty, as adjunctive treatments in thoracolumbar surgical interventions (60,61). Epidural neuroplasty is utilized to address back pain and/or radiating pain arising from mechanical compression on nerve structures within the vertebral column or neuroinflammation. The technique of epidural neuroplasty, including the separation of epidural adhesions with epidural glue, has gained traction and demonstrated promising results in recent times. Nonetheless, there have been reports of serious complications associated with this procedure, including epidural abscesses, irreversible nerve damage, and cardiovascular incidents (62).

Postoperative complications

In the realm of endoscopic lumbar spine surgery, when performed by adept practitioners, complications are infrequent. Nonetheless, endoscopic approaches are not devoid of potential iatrogenic risks, one of which includes the rare but significant risk of operating at an incorrect spinal level. Thus, meticulous examination of preoperative imaging on both sagittal and axial planes is crucial. Accurate radiographic determination of the target level is essential to mitigate the risk of wrong-site surgery. Additionally, precise care is required during the insertion and removal of endoscopic tools to minimize risk.

Comparative analyses have demonstrated that the rates of individual and cumulative complications associated with endoscopic lumbar surgery are significantly lower than those reported for open or minimally invasive translaminar approaches. An aggregate complication rate of 1.42% has been documented by some researchers, with the rate of surgical complications, excluding medical complications, reported at 0.32%—significantly lower than the rates observed in conventional open lumbar spine surgeries. It is noted that approximately 75% of patients undergoing lumbar endoscopy via the transforaminal approach experience a complication-free postoperative recovery.

Considering the full spectrum of complications, which includes durotomy, foot drop, infection (each reported at 0.11%); exacerbation of preexisting medical conditions (0.6%); recurrent herniation of extruded disc fragments (0.6%); postoperative issues such as dysesthesia (12.45%), spinal headaches (0.44%), or surgical site swelling due to irrigation fluid infiltration (3.75%); ecchymosis (0.76%); failure to achieve symptomatic relief (4.35%); and instances of acute care readmission (0.49%), a total adverse event rate of 24.04% has been reported during the postoperative period (63).

A particularly concerning complication of full endoscopic lumbar surgery involves damage to the vascular structures positioned anteriorly and laterally to the spine. Injuries to segmental arteries and major vascular conduits are especially worrisome. Segmental artery injuries are predominantly associated with transforaminal procedures, particularly during the decompression of the exiting nerve root, due to the anatomical path of the segmental artery beneath the exiting nerve root.

Conclusions

Lumbar disc herniation remains one of the most common causes for LBP worldwide. The treatments for LDH have improved in both scope and quality, from interventional pain therapies to various surgical approaches to post-surgical management of recurrence.

Microdiscectomies remain the gold standard surgical approach, and alternative approaches such as tubular discectomies and transforaminal foraminotomy may yield similar symptomatic relief with better clinical outcomes such as lower blood loss and shorter hospital stays.

The exact event leading to disc herniation remains unclear. Non-operative treatments should be the first-line treatment for most patients with lumbar disc herniation. Operative treatment remains the current gold standard, with minimally invasive endoscopic microdiscectomy techniques showing best results with respect to postoperative pain and function. Regenerative medicine is promising.

Endoscopic spine surgery is a popular option in minimally invasive spine surgery with good clinical results in the current scientific literature. With advancement in endoscopic optics, instrumentation and techniques, we can potentially unfold a new chapter of understanding of spine pathologies, and how the pathological processes interact with patients with time as more revision spine surgeries are performed under endoscopic view.

Conflict of interest

The authors declare no conflict of interest, and received no specific funding regarding this scientific research.

Abbreviations

Lumbar Disc Herniation (LDH)
Low back pain (LBP)
Disability-adjusted life years (DALYs)
Medial-lateral (ML)
Intervertebral disc herniation (IDH)
Magnetic resonance imaging (MRI)
Computed tomography (CT)
Graphical processing units (GPUs)
Region-based Convolutional Neural Networks (RCNNs)
Convolutional neural network (CNN)
Microdiscectomy (MD)
Tubular retractor (TD)
Percutaneous discectomy (AUTD)
Percutaneous endoscopic discectomy (PED)
Open lumbar microdiscectomy (OLMD)
Percutaneous endoscopic lumbar discectomy (PELD)
Full-endoscopic spine surgery (FESS)
Full-endoscopic lumbar discectomy (FELD)
Interlaminar endoscopic lumbar discectomy (IELD)
Spinal stenosis (SS)
Failed back surgery syndrome (FBSS)
Percutaneous epidural neuroplasty (PEN)

References

1. Son ES, Lee SH, Park SY, Kim KT, Kang CH, Cho SW. Surgical treatment of t1-2 disc herniation with t1 radiculopathy: a case report with review of the literature. Asian Spine J. 2012 Sep;6(3):199–202.
2. Ducati LG, Silva MV, Brandão MM, Romero FR, Zanini MA. Intradural lumbar disc herniation: report of five cases with literature review. Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc. 2013 May;22 Suppl 3(Suppl 3):S404-408.
3. Payer M. “Minimally invasive” lumbar spine surgery: a critical review. Acta Neurochir (Wien). 2011 Jul;153(7):1455–9.
4. Imaad-ur-Rehman null, Hamid RS, Akhtar W, Shamim MS, Naqi R, Siddiq HI. Observer variation in MRI evaluation of patients with suspected lumbar disc herniation and nerve root compression: comparison of neuroradiologist

and neurosurgeon's interpretations. JPMA J Pak Med Assoc. 2012 Aug;62(8):826–9.

5. Koes BW, van Tulder MW, Thomas S. Diagnosis and treatment of low back pain. BMJ. 2006 Jun 17;332(7555):1430–4.
6. Maher C, Underwood M, Buchbinder R. Non-specific low back pain. Lancet Lond Engl. 2017 Feb 18;389(10070):736–47.
7. Mattiuzzi C, Lippi G, Bovo C. Current epidemiology of low back pain. J Hosp Manag Health Policy. 2020 Jun 1;4:15–15.
8. Jordan J, Konstantinou K, O'Dowd J. Herniated lumbar disc. BMJ Clin Evid. 2009 Mar 26;2009:1118.
9. Fjeld OR, Grøvle L, Helgeland J, Småstuen MC, Solberg TK, Zwart JA, et al. Complications, reoperations, readmissions, and length of hospital stay in 34 639 surgical cases of lumbar disc herniation. Bone Jt J. 2019 Apr;101-B(4):470–7.
10. Takahashi Y, Yasuhara T, Kumamoto S, Yoneda K, Tanoue T, Nakahara M, et al. Laterality of cervical disc herniation. Eur Spine J. 2013 Jan;22(1):178–82.
11. Gao X, Shen F, Wang L, Ma Y, Niu H, Fan Y. Two-dimensional dynamic walking stability of elderly females with a history of falls. Med Biol Eng Comput. 2021 Aug;59(7–8):1575–83.
12. Barzouhi A el, Vleggeert-Lankamp CLAM, Nijeholt GJL à, Kallen BFV der, Hout WB van den, Verwoerd AJH, et al. Magnetic Resonance Imaging Interpretation in Patients with Sciatica Who Are Potential Candidates for Lumbar Disc Surgery. PLOS ONE. 2013 iul;8(7):e68411.
13. Peul WC, Hout WB van den, Brand R, Thomeer RTWM, Koes BW, Group for the LTHSIPS. Prolonged conservative care versus early surgery in patients with sciatica caused by lumbar disc herniation: two year results of a randomised controlled trial. BMJ. 2008 Jun 12;336(7657):1355–8.
14. Cihangiroglu M, Yıldırım H, Bozgeyik Z, Senol U, Ozdemir H, Topsakal C, et al. Observer variability based on the strength of MR scanners in the assessment of lumbar degenerative disc disease. Eur J Radiol. 2004 Sep 1;51(3):202–8.
15. van Rijn JC, Klemetso N, Reitsma JB, Bossuyt PM, Hulsmans FJ, Peul WC, et al. Observer variation in the evaluation of lumbar herniated discs and root compression: spiral CT compared with MRI. Br J Radiol. 2006 May;79(941):372–7.
16. Chen CY, Chuang YL, Yao MS, Chiu WT, Chen CL, Chan WP. Posterior epidural migration of a sequestrated lumbar disk fragment: MR imaging findings. AJNR Am J Neuroradiol. 2006 Aug;27(7):1592–4.
17. Zhou Y, Liu Y, Chen Q, Gu G, Sui X. Automatic Lumbar MRI Detection and Identification Based on Deep Learning. J Digit Imaging. 2019 Jun;32(3):513–20.
18. Abdelhafiz D, Yang C, Ammar R, Nabavi S. Deep convolutional neural networks for mammography: advances, challenges and applications. BMC Bioinformatics. 2019 Jun 6;20(Suppl 11):281.
19. Tsai JY, Hung IYJ, Guo YL, Jan YK, Lin CY, Shih TTF, et al. Lumbar Disc Herniation Automatic Detection in Magnetic Resonance Imaging Based on Deep Learning. Front Bioeng Biotechnol. 2021 Aug 19;9:708137.
20. Mbarki W, Bouchouicha M, Frizzi S, Tshibasu F, Farhat LB, Sayadi M. Lumbar spine discs classification based on deep convolutional neural networks using axial view MRI. Interdiscip Neurosurg. 2020 Dec 1;22:100837.
21. Yasargil MG, Vise WM, Bader DC. Technical adjuncts in neurosurgery. Surg Neurol. 1977 Nov;8(5):331–6.
22. Foley KT, Smith MM, Rampersaud YR. Microendoscopic approach to far-lateral lumbar disc herniation. Neurosurg Focus. 1999 Nov 15;7(5):e5.
23. Kambin P, Sampson S. Posterolateral percutaneous suction-excision of herniated lumbar intervertebral discs. Report of interim results. Clin Orthop. 1986 Jun;(207):37–43.
24. Weinstein JN, Lurie JD, Tosteson TD, Skinner JS, Hanscom B, Tosteson ANA, et al. Surgical vs nonoperative treatment for lumbar disk herniation: the Spine Patient Outcomes Research Trial (SPORT) observational cohort. JAMA. 2006 Nov 22;296(20):2451–9.
25. Clark R, Weber RP, Kahwati L. Surgical Management of Lumbar Radiculopathy: a Systematic Review. J Gen Intern Med. 2020 Mar;35(3):855–64.
26. Ambrossi GLG, McGirt MJ, Sciubba DM, Witham TF, Wolinsky JP, Gokaslan ZL, et al. Recurrent lumbar disc herniation after single-level lumbar discectomy: incidence and health care cost analysis. Neurosurgery. 2009 Sep;65(3):574–8; discussion 578.
27. McGirt MJ, Ambrossi GLG, Datoo G, Sciubba DM, Witham TF, Wolinsky JP, et al. Recurrent disc herniation and long-term back pain after primary lumbar discectomy: review of outcomes reported for limited versus aggressive disc removal. Neurosurgery. 2009 Feb;64(2):338–44; discussion 344-345.
28. Apostolides PJ, Jacobowitz R, Sonntag VK. Lumbar discectomy microdiscectomy: "the gold standard." Clin Neurosurg. 1996;43:228–38.
29. Casal-Moro R, Castro-Menéndez M, Hernández-Blanco M, Bravo-Ricoy JA, Jorge-Barreiro FJ. Long-term outcome after microendoscopic diskectomy for lumbar disk herniation: a prospective clinical study with a 5-year follow-up. Neurosurgery. 2011 Jun;68(6):1568–75; discussion 1575.
30. Schoeggl A, Maier H, Saringer W, Reddy M, Matula C. Outcome after chronic sciatica as the only reason for lumbar microdiscectomy. J Spinal Disord Tech. 2002 Oct;15(5):415–9.
31. Schick U, Döhnert J, Richter A, König A, Vitzthum HE. Microendoscopic lumbar discectomy versus open surgery: an intraoperative EMG study. Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc. 2002 Feb;11(1):20–6.
32. Brayda-Bruno M, Cinnella P. Posterior endoscopic discectomy (and other procedures). Eur Spine J Off Publ

Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc. 2000 Feb;9 Suppl 1(Suppl 1):S24-29.
33. Destandau J. A special device for endoscopic surgery of lumbar disc herniation. Neurol Res. 1999 Jan;21(1):39–42.
34. Chen Z, Zhang L, Dong J, Xie P, Liu B, Wang Q, et al. Percutaneous transforaminal endoscopic discectomy compared with microendoscopic discectomy for lumbar disc herniation: 1-year results of an ongoing randomized controlled trial. J Neurosurg Spine. 2018 Mar;28(3):300–10.
35. Meyer G, Rocha IDD, Cristante AF, Marcon RM, Coutinho TP, Torelli AG, et al. Percutaneous Endoscopic Lumbar Discectomy Versus Microdiscectomy for the Treatment of Lumbar Disc Herniation: Pain, Disability, and Complication Rate—A Randomized Clinical Trial. Int J Spine Surg. 2020 Feb 1;14(1):72–8.
36. Sinkemani A, Hong X, Gao ZX, Zhuang SY, Jiang ZL, Zhang SD, et al. Outcomes of Microendoscopic Discectomy and Percutaneous Transforaminal Endoscopic Discectomy for the Treatment of Lumbar Disc Herniation: A Comparative Retrospective Study. Asian Spine J. 2015 Dec 8;9(6):833–40.
37. Gempt J, Jonek M, Ringel F, Preuß A, Wolf P, Ryang Y. Long-term follow-up of standard microdiscectomy versus minimal access surgery for lumbar disc herniations. Acta Neurochir (Wien). 2013 Dec 1;155(12):2333–8.
38. Rasouli MR, Rahimi-Movaghar V, Shokraneh F, Moradi-Lakeh M, Chou R. Minimally invasive discectomy versus microdiscectomy/open discectomy for symptomatic lumbar disc herniation. Cochrane Database Syst Rev. 2014 Sep 4;(9):CD010328.
39. Reulen HJ, Müller A, Ebeling U. Microsurgical anatomy of the lateral approach to extraforaminal lumbar disc herniations. Neurosurgery. 1996 Aug;39(2):345–50; discussion 350-351.
40. Ebraheim NA, Xu R, Huntoon M, Yeasting RA. Location of the extraforaminal lumbar nerve roots. An anatomic study. Clin Orthop. 1997 Jul;(340):230–5.
41. Choi G, Lee SH, Raiturker PP, Lee S, Chae YS. Percutaneous endoscopic interlaminar discectomy for intracanalicular disc herniations at L5-S1 using a rigid working channel endoscope. Neurosurgery. 2006 Feb;58(1 Suppl):ONS59-68; discussion ONS59-68.
42. Ruetten S, Komp M, Godolias G. A New full-endoscopic technique for the interlaminar operation of lumbar disc herniations using 6-mm endoscopes: prospective 2-year results of 331 patients. Minim Invasive Neurosurg MIN. 2006 Apr;49(2):80–7.
43. Chen HT, Tsai CH, Chao SC, Kao TH, Chen YJ, Hsu HC, et al. Endoscopic discectomy of L5-S1 disc herniation via an interlaminar approach: Prospective controlled study under local and general anesthesia. Surg Neurol Int. 2011;2:93.
44. Katz JN, Harris MB. Clinical practice. Lumbar spinal stenosis. N Engl J Med. 2008 Feb 21;358(8):818–25.
45. Golob AL, Wipf JE. Low back pain. Med Clin North Am. 2014 May;98(3):405–28.
46. Baber Z, Erdek MA. Failed back surgery syndrome: current perspectives. J Pain Res. 2016;9:979–87.
47. Chan C wern, Peng P. Failed back surgery syndrome. Pain Med Malden Mass. 2011 Apr;12(4):577–606.
48. Brzezicki G, Jankowski R, Blok T, Klimczak A, Szymas J, Huber J, et al. Postlaminectomy osteopontin expression and associated neurophysiological findings in rat peridural scar model. Spine. 2011 Mar 1;36(5):378–85.
49. Rönnberg K, Lind B, Zoega B, Gadeholt-Göthlin G, Halldin K, Gellerstedt M, et al. Peridural scar and its relation to clinical outcome: a randomised study on surgically treated lumbar disc herniation patients. Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc. 2008 Dec;17(12):1714–20.
50. Helm S, Knezevic NN. A review of the role of epidural percutaneous neuroplasty. Pain Manag. 2019 Jan 1;9(1):53–62.
51. Manchikanti L, Cash KA, McManus CD, Pampati V. Assessment of effectiveness of percutaneous adhesiolysis in managing chronic low back pain secondary to lumbar central spinal canal stenosis. Int J Med Sci. 2013;10(1):50–9.
52. Manchikanti L, Knezevic NN, Sanapati MR, Boswell MV, Kaye AD, Hirsch JA. Effectiveness of Percutaneous Adhesiolysis in Managing Chronic Central Lumbar Spinal Stenosis: A Systematic Review and Meta-Analysis. Pain Physician. 2019 Nov;22(6):E523–50.
53. Helm S, Racz GB, Gerdesmeyer L, Justiz R, Hayek SM, Kaplan ED, et al. Percutaneous and Endoscopic Adhesiolysis in Managing Low Back and Lower Extremity Pain: A Systematic Review and Meta-analysis. Pain Physician. 2016 Feb;19(2):E245-282.
54. Taheri A, Khajenasiri AR, Nazemian Yazdi NA, Safari S, Sadeghi J, Hatami M. Clinical Evaluation of Percutaneous Caudal Epidural Adhesiolysis With the Racz Technique for Low Back Pain Due to Contained Disc Herniation. Anesthesiol Pain Med. 2016 Apr 27;6(3):e26749.
55. Katz JN, Zimmerman ZE, Mass H, Makhni MC. Diagnosis and Management of Lumbar Spinal Stenosis: A Review. JAMA. 2022 May 3;327(17):1688–99.
56. Thiengwittayaporn S, Koompong P, Khamrailert S, Wetpiriyakul P. Comparison of Clinical Outcomes of Different Rates of Infusion in Caudal Epidural Steroid Injection: A Randomized Controlled Trial. Asian Spine J. 2021 Apr;15(2):244–51.
57. Friedly JL, Comstock BA, Turner JA, Heagerty PJ, Deyo RA, Bauer Z, et al. Long-Term Effects of Repeated Injections of Local Anesthetic With or Without Corticosteroid for Lumbar Spinal Stenosis: A Randomized Trial. Arch Phys Med Rehabil. 2017 Aug;98(8):1499-1507.e2.
58. Lee JW, Myung JS, Park KW, Yeom JS, Kim KJ, Kim HJ, et al. Fluoroscopically guided caudal epidural steroid injection for management of degenerative lumbar spinal stenosis: short-term and long-term results. Skeletal Radiol. 2010 Jul;39(7):691–9.
59. Patel HA, Cheppalli NS, Bhandarkar AW, Patel V, Singla A. Lumbar Spinal Steroid Injections and Infection Risk after

Spinal Surgery: A Systematic Review and Meta-Analysis. Asian Spine J. 2022 Dec;16(6):947–57.
60. Oh Y, Shin DA, Kim DJ, Cho W, Na T, Leem JG, et al. Effectiveness of and Factors Associated with Balloon Adhesiolysis in Patients with Lumbar Post-Laminectomy Syndrome: A Retrospective Study. J Clin Med. 2020 Apr 16;9(4):1144.
61. Qureshi F, Meena SC, Kumar V, Jain K, Chauhan R, Luthra A. Influence of Epidural Ropivacaine with or without Dexmedetomidine on Postoperative Analgesia and Patient Satisfaction after Thoraco-Lumbar Spine Instrumentation: A Randomized, Comparative, and Double-Blind Study. Asian Spine J. 2021 Jun;15(3):324–32.
62. Kim SE, Choi SS. Epidural neuroplasty/epidural adhesiolysis. Anesth Pain Med. 2016 Jan 31;11(1):14–22.
63. Lewandrowski KU. Incidence, Management, and Cost of Complications After Transforaminal Endoscopic Decompression Surgery for Lumbar Foraminal and Lateral Recess Stenosis: A Value Proposition for Outpatient Ambulatory Surgery. Int J Spine Surg. 2019 Jan;13(1):53–67.

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Pre-rupture syndrome. Oculomotor nerve palsy in the case of an aneurysm of the posterior communicating artery

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Keywords
oculomotor nerve palsy, posterior communicating artery aneurysm, subarachnoid haemorrhage, endovascular coiling, aneurysm morphology

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ABSTRACT

Oculomotor nerve palsy can be a strong indicator of an internal carotid artery aneurysm, specifically of the posterior communicating artery, due to the anatomical positioning within the basal cisterns of the subarachnoid space, which allows for nerve compression. This condition can predict an acute subarachnoid haemorrhage, associated with a high mortality rate, often presenting with sudden, severe headaches, with or without deficits. The pathophysiology theories include direct mechanical compression by the aneurysmal sac, arterial pulsations, and nerve tissue oedema from venous obstruction. In cases of unruptured aneurysms, nerve irritation is less common. This palsy occurs in about one-third of cases.

Aneurysmal orientation can vary (lateral, superior, inferior, medial, posterior), and due to the nerve's anatomical position, aneurysms in the postero-lateral-inferior position are more likely to cause nerve damage. Treatment opinions vary from observation to intervention, with options including traditional neurosurgery and endovascular embolization. The latter is increasingly preferred due to its efficacy and lower risk profile.

A clinical case involves a 56-year-old female presenting with ptosis, diplopia, and ipsilateral mydriasis. Imaging confirmed a posterior communicating artery aneurysm with a maximum diameter of 5.3 mm and a 3 mm bleb. Endovascular coiling was performed successfully, excluding the aneurysm from circulation without complications. Post-treatment, the patient was discharged in good condition with recommendations for periodic monitoring.

In conclusion, while no single best treatment exists for posterior communicating artery aneurysms with oculomotor nerve palsy, early intervention (surgical or endovascular) and patient monitoring are crucial for preventing complications and ensuring quality of life.

1. INTRODUCTION

The pre-rupture syndrome associated with posterior communicating artery aneurysms represents a complex clinical entity characterized by neurological signs such as oculomotor nerve palsy. This syndrome holds significant importance in the diagnosis and management of cerebral aneurysms, bearing critical consequences for patient prognosis. In this context, identifying and understanding the pathophysiological mechanisms underlying this syndrome are essential for adopting an appropriate therapeutic approach and improving clinical outcomes.

Beginning with anatomical and pathophysiological aspects, this paper explores the complexity of the pre-rupture syndrome in the context of posterior communicating artery aneurysms. Aneurysms in this anatomical region pose an increased risk of oculomotor nerve palsy due to their proximity to this crucial neural structure.

Characterizing the orientation and dimensions of the aneurysm is of major importance in assessing the risk of nerve compression and determining the optimal treatment strategy.

With the advancement of medical technologies, multiple therapeutic options have been developed for managing cerebral aneurysms. From classical neurosurgical approaches to modern endovascular techniques, there is a diversity of methods available for treating these vascular lesions. However, the decision regarding the optimal treatment of aneurysms, especially in cases of pre-rupture syndrome associated with oculomotor nerve palsy, remains a subject of debate and controversy in the medical community.

Presenting a specific clinical case illustrates the importance of early diagnosis and appropriate management of posterior communicating artery aneurysms. In this case, a 56-year-old patient presented with characteristic symptoms of pre-rupture syndrome, such as eyelid ptosis and diplopia, with imaging and angiographic evidence confirming the presence of an aneurysm. Endovascular management of the aneurysm was associated with favorable clinical outcomes, highlighting the effectiveness and importance of this approach in preventing complications and improving patient prognosis.

The pre-rupture syndrome associated with posterior communicating artery aneurysms represents a complex clinical entity that requires a multidisciplinary and personalized approach. Early diagnosis, careful risk assessment, and selection of the most appropriate therapeutic strategy are crucial for the efficient management of these vascular lesions and for optimizing clinical outcomes.

2. Materials and methods

The study presents the case of a patient diagnosed with posterior communicating artery aneurysms and pre-rupture syndrome. The case manifested with oculomotor nerve palsy, confirmed through radiological imaging showing the presence of an aneurysm, and neurological examination findings consistent with pre-rupture syndrome. Diagnostic evaluation comprised MRI and cerebral angiography for anatomical visualization and aneurysm characterization. Endovascular treatment was administered under general anesthesia, involving femoral arterial access, catheterization to the internal carotid artery, and embolization using detachable coils. Follow-up angiography was conducted to assess treatment efficacy. Post-procedure, clinical outcomes were evaluated, with a focus on symptom resolution and complications. Descriptive statistics summarized patient demographics, procedural details, and outcomes. Ethical considerations were addressed with institutional review board approval and informed consent. Acknowledgment of limitations, such as the retrospective design, was made, with implications for future research highlighted.

3. Case Report

3.1. Oculomotor Nerve Palsy as a pre-rupture sign

A 56-year-old female patient presented to our neurosurgery department with complaints of eyelid ptosis, diplopia, and ipsilateral mydriasis, which began several months ago and were completely resolved at the time of consultation. Upon neurological clinical examination at admission, the patient was conscious, cooperative, oriented, with a GCS score of 15, without signs of intracranial hypertension or meningeal irritation, with normal speech, cranial nerves within normal limits, and without sphincter disturbances. The brain MRI examination revealed the presence of a right posterior communicating artery aneurysm. The DSA confirmed the presence of the aneurysm, measuring 5.3 mm in maximum diameter, with a bleb in its postero-inferior portion measuring 3 mm. Considering the aneurysm's location, morphology (presence of a bleb), and the occurrence of right third nerve palsy episode, it can be inferred that the aneurysm exhibited a pre-rupture syndrome.

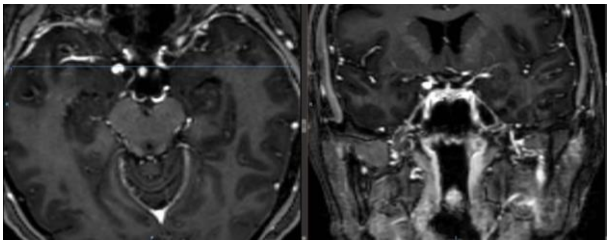


Figure 1. Native brain MRI TOF seg (a-b) right pCom aneurysm.

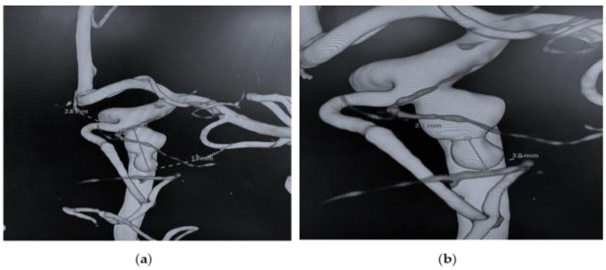


Figure 2. Digital subtraction angiogram (DSA) of the right internal carotid artery: (**a-b**) Giant posterior communicating saccular aneurysm with Bleb sac.

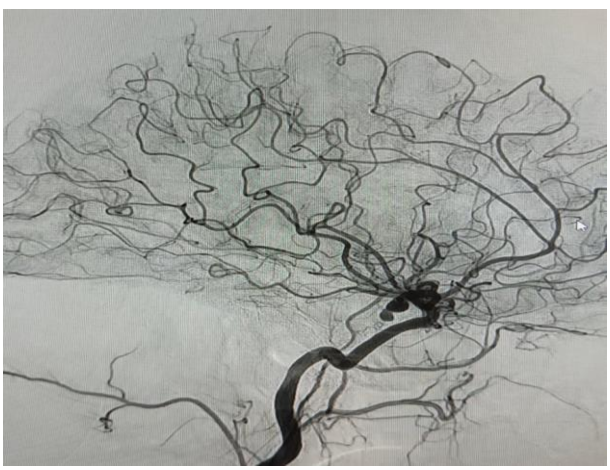


Figure 3. Digital subtraction angiogram (DSA) in lateral view of the right internal carotid artery: giant posterior communicating saccular aneurysm with Bleb sac.

3.2. Endovascular treatment

The patient was placed in the dorsal decubitus position and a right femoral arterial puncture was performed under general anesthesia. A 7F femoral introducer was inserted and connected for invasive blood pressure measurement. A BENCHMARK 6F, 071 catheter was advanced over a 5F SELECT vertebral catheter to the level of the right internal carotid artery for the acquisition of diagnostic angiographic series in anteroposterior, lateral, and 3D projections. Using coaxial technique, a Scepter 4/11 balloon was advanced to the cavernous segment level of the right internal carotid artery, followed by an Echelon 14 microcatheter, pre-curved at 90 degrees, utilizing Transend14 guidewires. The distal tip of the microcatheter was positioned at the neck of the aneurysm. Embolization of the aneurysm was performed using coils sized 5/15, 4/8, 3/6, 2/4, 2/3, and 2/1. Control angiographic series were obtained in working projections, anterior-posterior, and lateral, with contrast injection through the catheter. At the end of the procedure, after angiographic control at the inguinal level, the femoral introducer was removed, and hemostasis was achieved at the puncture site by manual compression and compressive dressing. During the endovascular procedure, the following prophylactic antithrombotic medication was administered: Heparin bolus of 2500 IU and infusion of 1000 IU/h, and intravenous Aspirin 250 mg.

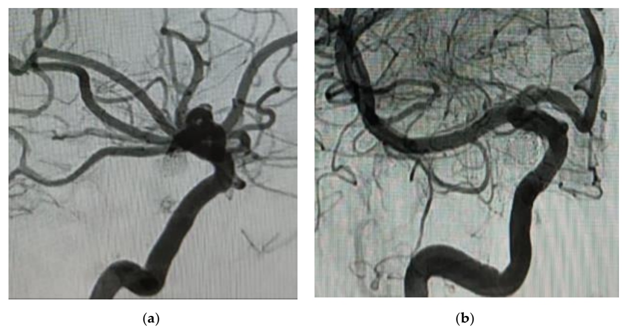
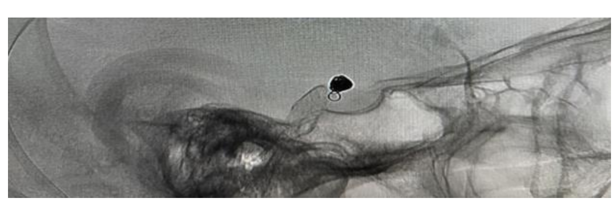


Figure 4. DSA of the right internal carotid artery – coiling session

3.3. Post-procedural result

Diagnostic series highlighted the posterior communicating artery aneurysm with a neck diameter of 2.95mm, maximum diameter of 5.34mm, and a small Bleb measuring 3.41mm. During the operation, aneurysm embolization was performed using simple coiling technique. Follow-up series demonstrated complete exclusion of the aneurysm without any embolic or hemorrhagic complications. The patient was discharged conscious, cooperative, with a GCS score of 15, afebrile, and advised for periodic monitoring and clinical and endovascular/neurosurgical imaging control.



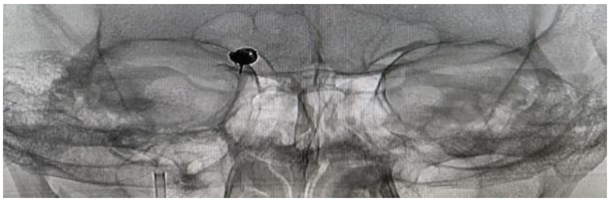


Figure 5. Control radiography highlighting the slipping of a coil loop into the aneurysm's bleb, ensuring better hemostasis at that level.

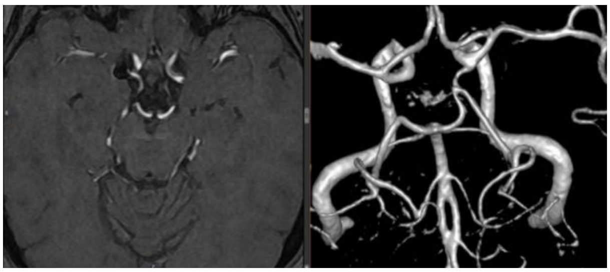


Figure 6. Postprocedural - brain MRI +TOF: exclusion from circulation of the posterior communicating artery as well as of the aneurysmal component.

4. Discussion

The management of posterior communicating artery (PCOM) aneurysms, particularly those associated with oculomotor nerve palsy, requires a nuanced approach that balances immediate intervention with long-term care. A comprehensive comparative analysis of treatment options, including endovascular embolization and traditional neurosurgical approaches, is essential. This involves evaluating success rates, potential complications, recovery times, and long-term outcomes to determine the most beneficial approach for specific patient profiles.

Advancements in imaging techniques, such as 3D angiography and MRI, play a crucial role in the early detection and characterization of PCOM aneurysms. These technologies enhance diagnostic accuracy, aid in treatment planning, and enable precise monitoring of aneurysm progression or resolution. Understanding the various risk factors and predictive indicators of aneurysm rupture, such as patient demographics, genetic predispositions, and lifestyle factors, alongside clinical signs like sudden severe headaches or oculomotor nerve palsy, is vital for preventative care and timely intervention.

Delving into the pathophysiology of oculomotor nerve palsy reveals the complexities of the condition. Theories such as direct mechanical compression by the aneurysmal sac, arterial pulsations, and nerve tissue edema from venous obstruction provide insights into the mechanisms at play. Analyzing specific patient case studies can highlight the variability in presentations, treatments, and outcomes, offering valuable lessons for clinical practice.

Long-term outcomes and the importance of regular follow-up care in patients treated for PCOM aneurysms cannot be overstated. Recommended follow-up schedules, imaging modalities for monitoring, and strategies for managing residual or recurrent aneurysms are crucial for ensuring patient safety and preventing future complications. An interdisciplinary approach to management, involving neurologists, neurosurgeons, and interventional radiologists, is essential for optimizing patient outcomes. Collaboration among specialists enhances treatment processes and ensures comprehensive care.

Innovations in endovascular techniques, including new coil materials and stent-assisted coiling, are revolutionizing the treatment of PCOM aneurysms. These advancements enhance the safety and efficacy of endovascular procedures, offering promising alternatives to traditional surgical methods. The impact of different treatment modalities on the quality of life and neurological function of patients post-treatment is another critical area of discussion. Understanding recovery experiences, long-term functional outcomes, and the psychological impact of living with or recovering from a PCOM aneurysm is vital for the patient care.

Finally, the emergency management of ruptured PCOM aneurysms requires well-defined protocols and strategies to minimize mortality and morbidity. Effective pre-hospital care, in-hospital emergency interventions, and rapid imaging and surgical responses are crucial for improving patient outcomes. Through these multifaceted discussions, the complexities of managing PCOM aneurysms can be better understood, ultimately leading to improved patient care and outcomes.

5. Conclusions

In conclusion, the management of posterior communicating artery (PCOM) aneurysms with associated oculomotor nerve palsy is complex and multifaceted. Early diagnosis is crucial due to the high risk of acute subarachnoid hemorrhage, which significantly increases mortality. Imaging techniques such as cerebral angiography are vital for identifying aneurysm characteristics, including size and bleb formation, which guide treatment decisions. While traditional neurosurgical approaches remain effective, endovascular embolization is increasingly favored for its lower risk profile and comparable efficacy. The case study of a 56-year-old patient successfully treated with endovascular coiling highlights the potential for positive outcomes with this method. Nonetheless, regular monitoring and follow-up are essential to ensure ongoing patient safety and to promptly address any complications. Ultimately, individualized treatment plans that consider the aneurysm's morphology and patient-specific factors are key to optimizing outcomes and maintaining quality of life.

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Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Conflicts of Interest: The authors declare no conflict of interest.

REFERENCES

1. Wang, B., Liu, S., Na, SJ. et al. Effects of endovascular treatment and prognostic factors for recovery of oculomotor nerve palsy caused by posterior communicating artery aneurysms: a multi-center retrospective analysis. BMC Neurol 22, 380 (2022). https://doi.org/10.1186/s12883-022-02911-y
2. Tan H, Huang G, Zhang T, Liu J, Li Z, Wang Z. A retrospective comparison of the influence of surgical clipping and endovascular embolization on recovery of oculomotor nerve palsy in patients with posterior communicating artery aneurysms. Neurosurgery. 2015;76(6):687–94.
3. Tan H, Huang G, Zhang T, Liu J, Li Z, Wang Z. A retrospective comparison of the influence of surgical clipping and endovascular embolization on recovery of oculomotor nerve palsy in patients with posterior communicating artery aneurysms. Neurosurgery. 2015;76(6):687–94.
4. Samuel Hall, Ahmed-Ramadan Sadek, Alexander Dando, Adam Grose, Borislav D. Dimitrov, John Millar, Jason H.M. Macdonald, Adam Ditchfield, Owen Sparrow, DiederikBulters,The Resolution of Oculomotor Nerve Palsy Caused by Unruptured Posterior Communicating Artery Aneurysms: A Cohort Study and Narrative Review,WorldNeurosurgery,Volume 107,2017,Pages 581-587,ISSN 1878-8750, https://doi.org/10.1016/j.wneu.2017.07.123
5. Matsukawa, H., Fujii, M., Akaike, G., Uemura, A., Takahashi, O., Niimi, Y., & Shinoda, M. (2014). Morphological and clinical risk factors for posterior communicating artery aneurysm rupture: Clinical article. Journal of Neurosurgery, 120(1), 104-110. https://doi.org/10.3171/2013.9.JNS13921
6. Zhang, .W., Zhang, .S. & Wu, .B. A study on the sectional anatomy of the oculomotor nerve and its related blood vessels with plastination and MRI. Surg Radiol Anat 24, 277–284 (2002). https://doi.org/10.1007/s00276-002-0052-3
7. Sebouh Z. Kassis, Emmanuel Jouanneau, Florence B. Tahon, Fadi Salkine, Gilles Perrin, Francis Turjman, Recovery of third nerve palsy after endovascular treatment ofposterior communicating artery aneurysms, World Neurosurgery, Volume 73, Issue 1, 2010, Pages 11-16, ISSN 1878-8750. https://doi.org/10.1016/j.surneu.2009.03.042
8. C.G. Chen, J.W. Wang, J.F. Li, C.H. Li, B.L. Gao, Factor saffecting resolution of oculomotor nervepalsy following endovascular embolization of posterior communicating artery aneurysms, Neurología (English Edition), Volume 39, Issue 4, 2024, Pages 315-320, ISSN 2173-5808. https://doi.org/10.1016/j.nrleng.2021.07.006

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Outcomes of dynamic lumbar fixation in young patients with minimal lumbar instability

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Keywords
pedicle screw-rod–based hybrid stabilization,
interspinous device–based hybrid stabilization,
dynesys stabilization,
rod contour,
visual analogue score

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ABSTRACT

Background data: Lumbar instability is considered an essential aetiology of low back pain (LBP) which is always accompanied by considerable disability. The term 'instability' remains poorly defined. But it is most broadly explained by any change of the normal mechanism of spinal motion which leads to pain and/or neurological impairment. It was reported that, 'Segmental instability can be defined as a pathological reaction to the applied loads, characterized by motion in motion segments more than normal constraints. The dynamic system can stabilize an unstable segment adequately but permits more mobility in the fixed levels than the conventional fixation systems.

Study design: prospective, consecutive study

Patients and methods: This study was conducted in Mansoura University hospitals over one year (starting from September 2019 till August 2020) Twenty patients who presented with back pain fulfilled our criteria and were included in this study and underwent surgery for lumbar fixation using a hybrid performance system with dynamic coupler. The history of all patients was taken and they were subjected to clinical examination and preoperative full radiological examination. Visual Analogue Scale (VAS) was used to evaluate POP. Regular follow-up visits were arranged for our cases 6 weeks, three months and 6 months postoperative.

Results*:* This study included 20 patients (7 males and 13 females). The ages of the studied patients ranged from 20 to 33 years. 9 patients presented with L4- 5 affection, 8 patients with L3- 4 affection and three patients with L2-3 affection. The mean basal VAS score was 5.45 (range=4-6) while the mean six months VAS score was 0.40 (range=0-1) with a statistically significant difference from the basal VAS score ($p < 0.001$). Also, the results show that post-operative QoL scale scores was significantly higher ($p < 0.001$) compared with pre-operative QoL scale scores (Physical function (54.30 vs. 67.10), limitations due to physical health (55.55 vs. 67.90)).

Conclusion*:* The current study suggested that Dynamic lumbar fixation provides favourable and stable clinical mid-term results in patients suffering from a degenerative lumbar spinal disease (DLSD) and can be a considerable alternative to fusion surgery in these patients.

INTRODUCTION

The spinal column, spinal muscles, and brain control unit comprise the three subsystems that make up the spine's stabilizing system. Numerous biomechanical investigations of the spinal column have shed light on the various spinal column components' functions in promoting spinal stability.[1]

Clinical instability is an essential etiology of LBP. As we all know there are many controversies about its exact definition, it is most broadly accepted that any change of the normal pattern of spinal motion leads to pain and/or neurological deficits. It is very important to identify the difference between clinical and mechanical instability. The former defines inability of the spine to withstand spinal loads, whereas the latter involves the clinical consequences of neurologic deficit and/or pain.[1] The lumbar spine checklist uses a lot of elements, as biomechanical forces, neurological insult and anticipated stresses on the spine. A point value system could be utilized to detect clinical stability or instability. The spine is deemed clinically unstable if the total of the points is five or higher.[2]

The Notion of Micro instability: The first phase of the degenerative cascade, described as the phase of unstable dysfunction, involves several pathologic changes affecting the constitutive elements of the motor spinal unit. Spondylarthrosis will inevitably result from those changes over time. These changes are explained by a dynamic stress over of the articular surfaces in the motor spinal unit, which includes the articular processes (which are responsible for the movement) and the intervertebral disc (which are responsible for the characteristics of load and torsion resistance). The alterations are generated by an anomalous hyper movement, an overstress of an articulation that isn't able to bear the applied load. In the phase of unstable dysfunction there are ongoing anatomopathological changes in lack of vertebral slippage.[3]

Adjacent segment disease is a broad term as it can explain most of abnormal changes that happen in the nearest motion segment to a spinal fusion. The most common finding is degenerated disc but also listhesis, instability, facet joints hypertrophy, disc herniation, and canal stenosis were frequently reported. Less commonly reported changes included scoliosis and vertebral compression fractures.[4] Recently, non-fusion stabilization of the lumbar spine has become more popular. These non-fusion systems intend to keep or restore the intersegmental motions to magnitudes of the intact spine and showed no harmful consequences on the adjacent segments.[5]

The dynamic system can stabilize the unstable segment adequately but permits more motion in the segment than the conventional systemms. The neighboring segment doesn't appear to be affected by the stiffness of the fixed segment under the loading situations. [6] Biomechanical changes could impact the nearby disc affection. In vitro experimental researches have shown that increased motion in the disc nearby to a vertebral fusion.[7] Moreover, recent literature have documented that dynamic stabilization can offer better surgical option rather than conventional fusion for managing lumbar degenerative diseases.[8] By prevention of additional progression of in- stability, dynamic stabilization might also avoid the use of a bone graft and its complications (such as wound problems, neurovascular injury, infections, pelvic fracture)[5]

In this study "pedicle screw-based dynamic systems" was used. They consist of titanium alloy pedicle screws and connecting rods that fit between the pedicle screw heads. The rod in the rigid fusion system is rigid, whereas it is movable in the dynamic system. This is the only distinction between the two systems. Thus, in theory, motion in the treated spinal segment can be preserved by the pedicle screw-based dynamic system.[8]

The study was done to evaluate the outcomes of dynamic spine fixation regarding the severity of postoperative pain measured by numerical analogue score (NAS), adjacent segment disease, range of motion (ROM) and quality of life.

PATIENTS AND METHODS

This prospective, randomized, double-blinded, comparative study was conducted at Mansoura university hospitals over one year (starting from September 2019 till August 2020) after approval of ethical committee and getting informed verbal and written consent from all patients. The study included Twenty patients their age ranged from 20 to 35 years. Their Inclusion criteria was Lumbar micro-instability (the concept of micro instability, intended as biomechanical dysfunction of the motor spinal unit, responsible for clinical presentation but not demonstrated by dynamic X-Rays), grade 1 and angular spondylolisthesis, Intact motor power and the Main complaint of the them was low back pain. While the Exclusion criteria was younger than18 years and older than 35 years, patient with more than grade 2 spondylolisthesis, presence of Neurological deficit and if the patient had Previous spine surgery.

Preoperative evaluation was done by History taking from every included case. Assessment of Vital signs, Neurological examination, Investigations as Dynamic X-ray of Lumbosacral spine & MRI of affected segment all were done to the all patients.

Evaluated parameters was done by assessing presurgical and postsurgical pain using Visual analogue score (VAS) score after asking the patient to rate his own pain according to 0 – 10 pain score severity (VAS score) where zero represents no pain while 10 represent worst pain imaginable (Zero = No pain, one = Minimal pain, two = Mild pain, three = Uncomfortable, four = Moderate, five = Distracting, six = Distressing, seven = Unmanageable, eight = Intense, nine = Sever & ten = Unable to move). Pre- and post-operative NSAIDS and needed Hospital stay were also checked in this study.

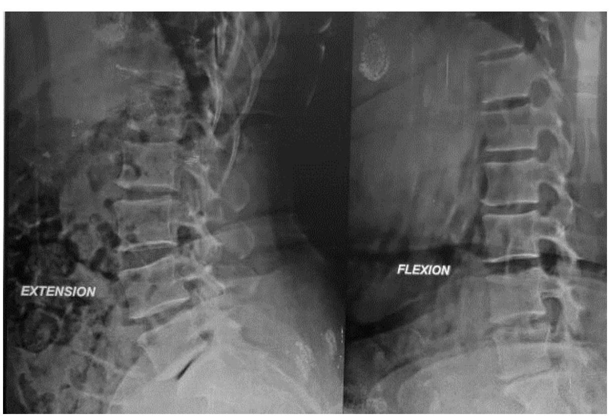

Figure 1. Pre-operative Dynamic Xray LSS (flexion and extension) showing L5-S1 spondylolisthesis.

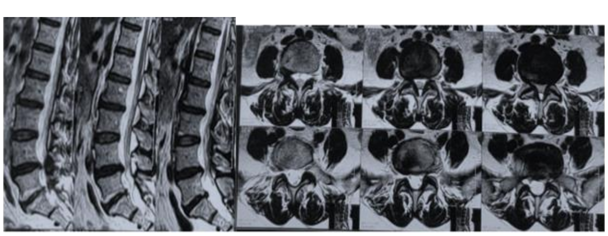

Figure 2. Pre-operative MRI lumbosacral spine showing instability L5 over S1 with modic changes and L4-5 disc prolapse.

Surgical technique

Midline spinal Approach was used. Then the Pedicle was Prepared, the pedicles are exposed then the trocar was used to open the cortical bone and form an initial entry into the pedicle. The pedicle entry point was marked under X-ray. The length and diameter of the Screw was chosen by the depth marks of the awl which commences at 30mm with five mm increments. Screw were placed after usage of K-wires that was placed within the cannulated pedicle, then the pedicle was prepared with a tap prior to screw placement. Determining Rod Contour and Coupler Fit was done through putting the coupler template on the template rod then the template rod was bent as important to accomplish an adequate alignment to the screw heads and to use it for the contouring of the rod. The Rod Length was determined by calculating the following, required rod length = Measured total length minus coupler length (35 mm) and the total rod length has to show overhang of five mm on each side. The screws were Inserted and Tightened by applying the persuader onto the screw head and compress the handle to reduce the rod into place.

Follow up was done by Evaluating the clinical outcome at the time of hospital discharge, 3, 6, 12 weeks then 6 months post procedure and MRI of the spine was done immediate post procedure and 6 months after treatment.

Figure 3. Intraoperative picture using C arm AP view showing fixation by 6screws and 2 rods with couplers.

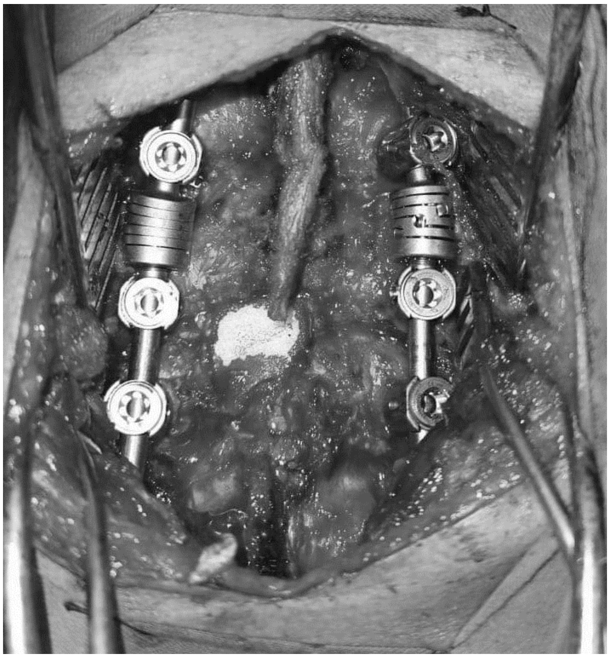

Figure 4. Intraoperative picture showing skin incision with L4-5 decompression with dynamic fixation.

Figure 5. Postoperative MRI LSS sagittal cuts 6 months postoperative, No detected adjacent segment disease.

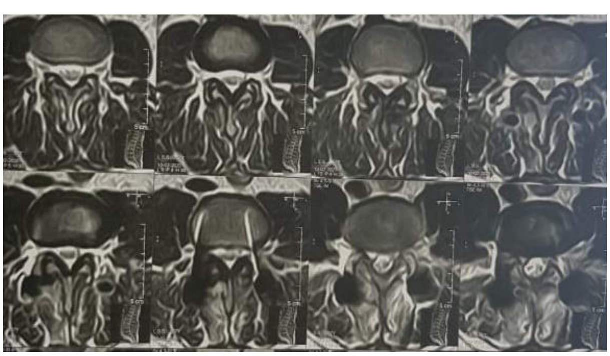

Figure 6. Postoperative MRI LSS axial cuts 6 months postoperative, No detected adjacent segment disease.

Table 1. Demographic characteristics of the studied patients

All patients (n= 20)		Mean & SD	Median	Range	IQR
Age (year)		26.50 ± 4.249	27.50	20, 33	22.25, 29.00
Gender	Male	35.0% (7)			
	Female	65.0% (13)			

Table 2. Affected level and Spondylolisthesis grade of the studied patients

		All patients (n= 20)
Affected level	L2- 3	15.0% (3)
	L3- 4	40.0% (8)
	L4- 5	45.0% (9)

Table 3. Total post-operative hospital stays in the current study:

All patients (n= 20)	Mean & SD	Median	Range	IQR
Hospital stays (days)	3.10 ± 1.683	3.00	1, 5	1.25, 5.00

Results

Ours study comprised 20 patients (7 males and 13 females). The age of the studied patients ranged from 20 to 33 years. The mean age was 26.50 years (table 1). According to level affection, L2- 3 affection was the least commonly reported where it was revealed in 3 patients (15%), L3- 4 affection was revealed in 8 patients (40%) while the majority of patients (9 patients) presented with L4- 5 affection (table 2). The post-operative hospital stay ranged from 1 day to 5 days. The mean post-operative hospital stay was 3.10 days (table 3).

The mean basal VAS score was 5.45 (range=4-6). The mean one-day VAS score was 5.80 (range=4-7) with a statistically significant difference from the basal VAS score (p=0.005). The mean three weeks VAS score was 2.80 (range=1-4) with a statistically significant difference from the basal VAS score ($p < 0.001$). The mean six weeks VAS score was 2.30 (range=4-7) with a statistically significant difference from the basal VAS score ($p < 0.001$). The mean three months VAS score was 1.15 (range=0-3) with a statistically significant difference from the basal VAS score ($p < 0.001$). The mean six months VAS score was 0.40 (range=0-1) with a statistically significant difference from the basal VAS score ($p < 0.001$) (table 4).

The mean basal daily ketolac consumption was 34.50 (range=20-50). The mean six weeks post-operative daily ketolac consumption was 14.50 (range=10-20) with a statistically significant difference from the basal ketolac consumption ($p < 0.001$). The mean three months post-operative daily ketolac consumption was 9.00 (range=0-30) with a statistically significant difference from the basal ketolac consumption ($p < 0.001$). The mean six months post-operative daily ketolac consumption was 4.00 (range=0-10) (IQR: 0.00, 10.00) with a statistically significant difference from the basal ketolac consumption ($p < 0.001$) (table 5).

The final results shows that post-operative quality of life scale scores was significantly higher ($p< 0.001$) compared with pre- operative quality of life scale scores (Physical function (54.30 vs. 67.10), limitations due to physical health (55.55 vs. 67.90), Bodily pain (61.25 vs. 75.65), Social function (55.90 vs. 68.15), limitations due to emotional health (50.90 vs. 62.30), General health (59.65 vs. 73.45), Vitality (54.95 vs. 67.20) and Mental health (58.95 vs. 72.20)) (Table 6).

Discussion

Lumbar instability has been considered as an essential cause of LBP and could be accompanied by considerable disability. The word 'instability' remains poorly defined. But it is most broadly believed that the loss of a normal pattern of spinal motion causes pain and/or neurological impairment. Many authors assumed that, 'Segmental instability is considered to be an abnormal reaction to applied loads, featured by motion in motion segments beyond accepted constraints.[9]

Dynamic stabilization showed an essential role in the management of the degenerative lumbar spine disease (DLSD). Although fusion of one or two motion levels will not cause a big difference in the overall motion range of the lumbar spine, but keeping flexibility of a motion segment could prevent neighboring segment pathology and might allow disc replacement, even when facet joints excision is needed. When a promising environment is kept in the motion segment by preventing overstresses on the disc and providing an acceptable range of motion, the disc can be capable of repairing itself. Although dynamic stabilization can play a role in treating these patients, any new stabilization system should be cautiously approached. Any fusion system only has to work as a temporary stabilization till fusion occurs; in contrast, a dynamic stabilization system should offer stability throughout its life.[10].

Thus, we aimed to evaluate the outcomes of dynamic spine fixation regarding the severity of postoperative pain measured by numerical analogue score (NAS), adjacent segment disease, ROM and QoL. This prospective, randomized, double-blinded, comparative study was conducted at Mansoura university hospitals over one year (starting from September 2019 till September 2020) after approval of ethical committee using Dynamic transpedicular lumbar fixation.

Dynamic stabilization is based on the notion that pain might be relieved and neighboring segment degeneration could be avoided by controlling abnormal motions and increasing physiologic load transmission. It is unlikely that the injured disc will repair itself once normal motion and load transmission are completed, unless the degeneration is evidently very severe [10].As regards, demographic characteristics, the mean age of the studied cases was 26.50 ± 4.249 and the majority of which were female (65%). The current study revealed that, post-operative hospital stay ranged from 1 day to 5 days. The mean post-operative hospital stay was 3.10 days while the median post-operative hospital stays 3.00 days.

Similarly, Baliga et al., 2015 [11] demonstrated that, the average hospital stay of 2 to 4 days in terms of non-fusion lumbar stabilization with mean post-operative hospital stay of 3 days. As regards, VAS score, there were significant differences between basal and post-operative follow-up till 6 months ($P< 0.001$). In the same line, Schnake et al., 2006 [5] demonstrated that, clinical data of their cases improved significantly. Pain on VAS ($P \leq 0.001$) and the use of pain killers ($P = 0.013$) may be diminished and walking distance ($P \leq 0.001$) and neurological manifestations ($P \leq 0.001$) improved. About 87.5% of the studied cases were active at their preceding level without restriction, while only 12.5% of them didn't reach the premorbid level. In general, the majority of the studied cases were satisfied.

The same line, Greiner-Perth et al., 2016 [12] conducted a prospective study on dynamic posterior stabilization for DLSD and demonstrated that, the NRS back and leg pain improved markedly from that reported preoperatively 6.2 and 7.1 (SD 2.4 range 0–

10) points to postsurgical mean 3.5 and 3.5 points, correspondingly (each p=0.0001).

As regards, daily ketolac consumption there was significant difference between basal and postsurgical follow-up till 6 months (P< 0.001). This came in accordance with several researches who demonstrated significant reduction in pain following operation with a subsequent reduction in the analgesic requirements. [12]

As regards comparison of presurgical and postsurgical QoL scale scores in the studied patients, there were significant differences between means of presurgical and postsurgical QoL scale scores as regards Physical function (54.30 vs. 67.10), limitations due to physical health (55.55 vs. 67.90), Bodily pain (61.25 vs. 75.65), Social function (55.90 vs. 68.15), limitations due to emotional health (50.90 vs. 62.30), General health (59.65 vs. 73.45), Vitality (54.95 vs. 67.20) and Mental health (58.95 vs. 72.20). The results shows that post-operative QoL scale scores was significantly higher (p< 0.001) compared with pre- operative QoL scale scores. In the same line, Greiner-Perth et al., 2016[2] [12]conducted a prospective study on 436 cases treated with DSS. In addition, 387 patients (189 male, 198 females; mean age 67.3 years) with DLSD such as degenerative spondylolisthesis (6.1 %) may be assessed. The type of degeneration was primarily spinal stenosis (89.9 %). Following a mean follow-up of 1.94 years, the COMI score and NRS back and leg pain improved significantly. The postsurgical trend analysis couldn't detect a relevant deterioration of such outcomes until four years postoperative. Ten cases were revised (2.6 %) and the implant was excised; in the majority of cases, a fusion was conducted. Another five cases (1.3%) had an extension of the dynamic

Table 4. Basal and post-operative follow-up of VAS score in the studied patients

All patients (n= 20)	Mean & SD	Median	Range	IQR	p
Basal	5.45 ± 0.605	5.50	4, 6	5.00, 6.00	-
One day	5.80 ± 0.834	6.00	4, 7	5.00, 6.00	0.005
Three weeks	2.80 ± 1.005	3.00	1, 4	2.00, 4.00	< 0.001
Six weeks	2.30 ± 0.865	2.00	1, 4	2.00, 3.00	< 0.001
Three months	1.15 ± 0.875	1.00	0, 3	0.25, 2.00	< 0.001
Six months	0.40 ± 0.503	0.00	0, 1	0.00, 1.00	< 0.001
P is significant when < 0.05.					

Table 5. Basal and post-operative daily ketolac consumption (mg) in the studied patients

All patients (n= 20)	Mean & SD	Median	Range	IQR	p
Basal	34.50 ± 10.501	30.00	20, 50	30.00, 40.00	-
Six weeks	14.50 ± 5.104	10.00	10, 20	10.00, 20.00	< 0.001
Three months	9.00 ± 8.522	10.00	0, 30	0.00, 10.00	< 0.001
Six months	4.00 ± 5.026	0.00	0, 10	0.00, 10.00	< 0.001
P is significant when < 0.05.					

Table 6. Basal and post-operative daily ketolac consumption (mg) in the studied patients

All patients (n= 20)	Pre-operative	Post-operative	95% CI	p
Physical function	54.30 ± 16.086	67.10 ± 20.442	- 15.04, - 10.46	< 0.001
limitations due to physical health	55.55 ± 19.508	67.90 ± 24.666	-12.83, -9.07	< 0.001
Bodily pain	61.25 ± 19.298	75.65 ± 24.564	-14.73, -10.57	< 0.001
Social function	55.90 ± 21.957	68.15 ± 26.019	-12.87, -9.53	< 0.001
limitations due to emotional health	50.90 ± 20.576	62.30 ± 24.942	-13.34, -8.86	< 0.001
General health	59.65 ± 18.204	73.45 ± 22.814	-15.43, -10.87	< 0.001
Vitality	54.95 ± 18.597	67.20 ± 22.659	-13.08, -9.82	< 0.001
Mental health	58.95 ± 15.436	72.20 ± 18.831	-14.67, -11.03	< 0.001
P is significant when < 0.05.				

stabilization system to the adjacent level. In addition, 84.2% of cases rated that the surgery had helped a lot or had helped. [5,12,13]

Similarly, Schnake et al., 2006 [5] conducted a study on prospectively assessed 25 cases with lumbar spinal stenosis and degenerative spondylolisthesis demonstrated that, the mean leg pain diminished significantly (P≤0.01), and mean walking distance improved significantly to more than 1000m (P≤0.01) following induction of dynamic stabilization with the Dynesys system. Therefore, they demonstrated that, no significant progression of spondylolisthesis was determined; asymptomatic implant failure was noted in 17% of cases due to screw loosening. Signs of adjacent segment degeneration were demonstrated in seven cases following 24 months.

Furthermore, Delamarter et al., 2006 [14] presented results of a major study conducted on a total of 84 cases managed with Dynesys or instrumented fusion for degenerative lumbar spondylolisthesis and/or stenosis at one or two contiguous levels at 24-month follow-up, ODI scores improved from 56.4 to 31.2, VAS leg pain improved from 82 to 15, and VAS back pain reduced from 52 to 21. Re-operation rate was 4%. Remarkably, the investigators recorded no examples of device failure, though 5% had asymptomatic radiolucency. While, Lee et al., 2017 [15] assumed that, in comparison with normal case, fusion on L4–5 induces 18.8%, 9.3%, 11.7%, and 13.7% increments during motion at L3–4 under flexion, extension, lateral bending, and axial rotation, respectively. Additional stabilization at L3–4 in hybrid models decreased motion range which can occur at that segment. The IH model revealed 8.4%, -33.9%, 6.9%, and 2.0% change in motion at the segment, while the PH model revealed -30.4%, -26.7%, -23.0%, and 12.9%. At L2–3, the PH model revealed 14.3%, 3.4%, 15.0%, and 0.8% of motion increment in comparison to the motion in the IH model. These two hybrid systems expressed law intradiscal pressure at the transition segment compared with that in the conventional system, but the loads at L2–3 (adjacent segment) increased in all loading positions except during extension. Thus, they concluded that, lumbar hybrid stabilization using both Pedicle screw-rod–based hybrid stabilization (PH) and interspinous device–based hybrid stabilization (IH) decreased the fusion-induced excessive motion at the transition segment. At the level next to the transition level, the PH model created more stress than the IH model under the hybrid loading condition. Such a difference could ultimately interfere with the possibility of ASD.

Although this study showed encouraging results and statistically significant percent of improvement, we are still in need for long-term follow up of cases at least 2 years with follow up VAS score and MRI lumbosacral spine to detect adjacent segment disease.

Conclusion

The current study suggested that Dynamic lumbar fixation provides a favorable and stable clinical mid-term results in patients suffering from degenerative lumbar spinal disease (DLSD) and can be a considerable alternative to fusion surgery in these patients.

List of abbreviations:

(ASD): Adjacent segment disease
(COMI): Core outcome measure index
(DLSD): Degenerative lumbar spinal disease
(DSS): Dynamic stabilization system
(IH): Inter-spinous device-based hybrid stabilization
(NAS): Numerical analogue score,
(NRS): Numeric rating scale
(ODI): Oswestny disability index
(PH): Pedicle screw-rod–based hybrid stabilization
(POP): Postoperative pain
(ROM): Range of motion
(QoL): Quality of life
(VAS): Visual Analogue Scale

References

1. Panjabi M. M. Clinical spinal instability and low back pain. J Electromyogr Kinesiol 2003;13(4):371-379.
2. Mulholland R. C., Sengupta D. K. Rationale, principles and experimental evaluation of the concept of soft stabilization. Arthroplasty of the Spine: Springer p. 2004;142-149.
3. Landi A., Gregori F., Mancarella C., Delfini R. Detection of Spinal Microinstability: A Real Clinical and Forensic Problem. J Spine 2015;4:e119,.
4. Park P., Garton H. J., Gala V. C., Hoff J. T., McGillicuddy J. E. Adjacent segment disease after lumbar or lumbosacral fusion: review of the literature. Spine (Phila Pa 1976) 2004;29(17):1938-1944.
5. Schnake K. J., Schaeren S., Jeanneret B. Dynamic stabilization in addition to decompression for lumbar spinal stenosis with degenerative spondylolisthesis. Spine (Phila Pa 1976) 2006; 31(4):442-449.

6. Ross A. Dynamic reconstruction of the spine: Edited by DH Kim, FP Cammisa Jr, RG Fessler Pp. 402. New York: Thieme, 2006. ISBN: 1-58890-484-9. The British Editorial Society of Bone and Joint Surgery; 2008.
7. Marsol-Puig A., Huguet-Comelles R., Escala-Arnau J., Giné-Gomà J. Incidence and risk factors of adjacent disc degeneration after lumbar fusion. Revista Española de Cirugía Ortopédica y Traumatología (English Edition) 2011;55(3):170-174.
8. Qian W., Yin H., Yang H. L., Li C., Hui M. Pedicle screw-based dynamic stabilisation systems versus pedicle screw-based rigid fusion system for lumbar degenerative diseases. Cochrane Database of Systematic Reviews. 2015.
9. Barz T., Melloh M., Lord S. J., Kasch R., Merk H. R., Staub L. P. A conceptual model of compensation/decompensation in lumbar segmental instability. Med Hypotheses 2014;83(3):312-316.
10. Sengupta D. K. Dynamic stabilization devices in the treatment of low back pain. Orthop Clin North Am 2004; 35(1):43-56.
11. Baliga S., Treon K., Craig N. J. Low Back Pain: Current Surgical Approaches. Asian Spine J 2015;9(4):645-657.
12. Greiner-Perth R., Sellhast N., Perler G., Dietrich D., Staub L. P., Röder C. Dynamic posterior stabilization for degenerative lumbar spine disease: a large consecutive case series with long-term follow-up by additional postal survey. European spine journal 2016;25(8):2563-2570.
13. Di Silvestre M., Lolli F., Bakaloudis G., Parisini P. Dynamic stabilization for degenerative lumbar scoliosis in elderly patients. Spine (Phila Pa 1976) 2010; 35(2):227-234.
14. Delamarter R. B., Maxwell J., Davis R., Sherman J., Welch W. 4: 57151. Nonfusion Application of the Dynesys System in the Lumbar Spine: Early Results from the IDE Multicenter Trial. The Spine Journal 2006; 6(5):77S.
15. Lee C.-H., Kim Y. E., Lee H. J., Kim D. G., Kim C. H. Biomechanical effects of hybrid stabilization on the risk of proximal adjacent-segment degeneration following lumbar spinal fusion using an interspinous device or a pedicle screw–based dynamic fixator. Journal of Neurosurgery: Spine 2017; 27(6):643-649.

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Clinical effectiveness of progesterone in acute traumatic spinal cord injury. A randomized single centre placebo-controlled study

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ASIA score

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ABSTRACT
Aim: With only limited treatment options in acute spinal cord injury (SCI) currently available, we aimed to investigate the effect of progesterone on neurological recovery after acute SCI.
Material and Methods: Randomised double-blind placebo-controlled single-centre trial on 128 patients of acute SCI (within 24 hours of injury) was conducted at our institute with the approval of the ethics committee. Eligible patients were allocated to the progesterone or placebo arm. Of 133 eligible patients, one from the placebo arm expired in the acute phase while 4 were lost to follow-up, leaving 128 patients in the study.68 patients remained in the progesterone arm and 60 in placebo. Patients in the progesterone arm received intramuscular progesterone while those in the placebo received intramuscular isotonic saline twice daily for five consecutive days. Neurological assessment was done at baseline, day six, first and sixth months using the American Spinal Injury Association (ASIA) score and motor and sensory actual neural recovery (ANR) scores.
Results: Baseline characteristics were comparable between the groups. At the end of six months, significant improvement occurred in motor and sensory ASIA scores in the progesterone arm (p=<0.01). Compared with the placebo, motor scores were significantly higher in the progesterone arm at 6 months while sensory scores were not (p= <0.01 and p= 0.59 respectively). Additionally, at 6 months, motor ANR was significantly higher in the progesterone arm vs placebo (p=<0.01 vs 0.65). Early progesterone administration (within six hours of injury) was associated with significantly higher motor and sensory ASIA scores at 6 months (p= <0.01vs0.04 respectively).
Conclusion: Administration of intramuscular progesterone within 24 hours in acute SCI was associated with better neurological recovery. Further multicentric studies are required to shed more light on the strength and consistency of this improved outcome.

INTRODUCTION

Approximately, 2,50,000-5,00,000 people per year suffer spinal cord injury (SCI) globally. In the aftermath of trauma, spinal cord injuries carry the most devastating outcomes with severe motor deficits and high socioeconomic burden. [1] Traumatic SCI often results in early necrosis of anterior horn cells leading to a partial or complete loss of motor neurons and consequent severe weakness. [2] Sadly, though the pathophysiology behind traumatic SCI is well understood, the treatment options remain limited with no single drug showing robust effectiveness. Transplant of peripheral nerves, enhancement of axonal growth using fibronectin conduits, usage of olfactory ensheathing cells, stem cells or Schwann cells with an aim to minimize the necrosis associated with SCI have been tried in the past with some success.[3] Pharmacologic approaches, such as neurotrophic factors, antioxidant molecules, antiglutamatergic drugs and steroids have also been previously used for the treatment of SCI. [4,5]

The world neurosurgery society, in contrast to the results of a Cochrane review conducted recently deemed the use of methylprednisolone to be more harmful than beneficial in SCI. The role of corticosteroids in SCI thus stands conflicted. [1,6] The gonadal steroids (17β-estradiol and progesterone) on the other hand in experimental trials have shown consistent optimistic results in traumatic brain and spine injuries.[4]Pertinent to our study, progesterone has been shown to prevent neuronal loss following traumatic brain injury with increased survival of motor neuron, protection against glutamate toxicity and oxidative stress after SCI .[4] Thus, many experimental studies using animal model have abundantly displayed the neuroprotective role of progesterone in SCI. Despite its well proven effectiveness in animal trials, its place in clinical trials is murky. Keeping this in mind, we took up this randomized placebo-controlled study to investigate the effect of progesterone on neurological recovery after acute SCI.

MATERIALS AND METHODS

We performed a prospective, randomized double-blind placebo-controlled trial from December 2019 to December 2020 at our institute. We aimed to assess the neurological recovery after acute SCI in treatment (progesterone administered) and placebo arms by monitoring their American Spinal Injury Association (ASIA) score (motor and sensory) and actual neural recovery (ANR) score {final follow-up ASIA score minus ASIA score at the time of admission} at day six (6), one (1) month and after six (6) months of follow-up}. Institutional ethics committee (IEC) clearance was obtained vide IEC no. 38/MC/EC/2020.

PATIENT SELECTION

All consecutive patients with acute traumatic SCI presenting within 24 hours of the injury between 18 and 65 years of age were included. Patients outside the specified age limit, 24 hours beyond the SCI, those with involvement of only the radicles or nerve roots, with firearm injury, with life-threatening morbidities, history of drug addiction or on steroids, pregnant women and unwilling for a minimum six months regular follow up were excluded from the study.

RANDOMIZATION AND INTERVENTION

Patients underwent simple randomization into the two study groups after fulfilling the inclusion criteria (Figure 1). The treatment group received progesterone (AqSusten, Sun Pharma, Uttarkhand, India) via intramuscular injection with a dosage schedule of 0.5 mg/kg of body weight twice daily for five consecutive days. The placebo group received intramuscular 0.9% sodium chloride injections twice daily for five consecutive days.

STUDY PROTOCOL AND MEASUREMENTS

A thorough clinical history, including demographic information (age, sex) and detailed physical examination assessing the neurological deficit was recorded. All routine investigations were sent as per the standard protocol including complete blood count (CBC) and biochemistry (random sugar, renal and liver function tests). The time of administering progesterone/placebo injection after injury was recorded.

Patients were neurologically assessed at the time of admission, on day 6 and then at first and six months after the injury using the ASIA score. [7] Motor and sensory ASIA scores and the actual neural recovery score were also calculated at the time of admission, day six, one and six months follow up.

STATISTICAL ANALYSIS

A minimum of 60 patients were required to detect any significant differences between the groups as per the ICCP panel for SCI. All relevant data were recorded and GraphPad Prism for Windows, version 9.1.0 (221) was used for data analysis. Per protocol analysis was done. For comparing the serial ASIA scores within the groups, repeated measure one-way ANOVA with Tukey's multiple comparison test was used. Student unpaired t-test was used to compare between the two study groups and $\chi 2$ test was used to compare proportions. We also attempted to compare the ASIA score on the basis of time since injury to administration of progesterone using unpaired t-test. For this, we divided the progesterone group into early (< 6 hours) and late intervention (>6 hours) based on the hours after SCI. Data were expressed as means ± SD or proportions as appropriate. P-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 133 patients met the inclusion criteria, after randomisation 71 and 62 patients were allocated to the case and control group respectively. A team of in-house neurosurgeons blinded to the trial design decided the best treatment plan, surgical or medical management for all patients. Three patients from the treatment arm and one from the placebo arm were lost to follow up and one patient in placebo group expired with sepsis during the acute phase. Thus, at the end of six months, 128 patients (68 cases and 60 controls) were included for the final analysis.

Baseline characteristics of patients in both the study groups were summarized (table 1). Both groups were comparable for age, gender, time between intervention and injury and site of injury. Most common fracture site was the cervical region.76.47% patients in progesterone group and 80% in placebo group underwent surgical management in the form of spinal canal decompression and fixation (anterior or posterior).

Table 1. Baseline characteristics of all patients

	Progesterone (n=68)	Placebo (n=60)	p-value
Age	35.86 ± 13.47	34.97 ± 11.67	**0.99**
Sex			**0.92**
Male	40 (58.8%)	36 (60%)	
Female	28 (41.2%)	24 (40%)	
Time from injury to intervention			**0.74**
< 6 hrs	48	40	
>6 hrs	20	20	
Fracture site			**0.81**
Cervical	**36**	**28**	
C2	2	0	
C3	6	2	
C4	8	8	
C5	10	10	
C6	6	8	
C7	4	0	
Thoracic	26	**22**	
T1-4	6	4	
T5-8	8	12	
T9-12	12	6	
Lumbar	**6**	**10**	
L1	4	6	
L2	2	4	
L3	0	0	
L4	0	0	
L5	0	0	
Treatment			**0.73**
Surgical	52	48	
Conservative	16	12	

ASIA score (motor and sensory for each limb) for both the groups (progesterone and placebo) at the time of admission, 6 days, 1 month and 6 months after injury was recorded (table 2). Mean ASIA score at admission was comparable in both groups (table 2).

At the final (six) months follow up the mean motor ASIA score was significantly higher in the progesterone arm (p=<0.01). Serial improvement in motor ASIA score was noted in both the groups from admission to 6 days, 1 month and 6 months but it reached statistical significance only in the progesterone arm. In this arm, significantly higher motor ASIA score was noted in either of the upper limbs {right and left upper limb (p= <0.01) and in the lower limbs {right and left lower limbs (p=<0.01)}. Further, sub-group analysis showed significant increase in the motor ASIA score of the progesterone group between admission, day 6 and at 1 month without a significant increase between the 1 and 6 month scores. Serial improvement in the motor ASIA score was noted in the placebo group also {right and left upper limb (p=0.07, 0.09 respectively) as well as in right and left lower limb (p=0.07, 0.10 respectively)} but failed to reach statistical significance (table 2).

Table 2. Serial changes in American Spinal Injury Association score in progesterone and placebo group in the individual limbs

		Progesterone group (n=68) American Spinal Injury Association score					Placebo group (n=60) American Spinal Injury Association score				
		At admission	6 days	1 month	6 month	p-value	At admission	6 days	1 month	6 month	p-value
Motor	Right UpperLimb	19.52±6.95	21.32±5.95	22.56±5.09	22.79±5.10	<0.01	20.03±5.90	20.29±5.72	20.40±5.78	20.40±5.78	0.07
	Left Upper Limb	20 ±6.17	21.32±5.69	22.41±5.09	22.64±5.11	<0.01	19.55±6.27	19.92±5.89	20.03±5.96	20.25±5.84	0.09
	Right Lower Limb	13.35±6.17	16.71±6.59	20.06±6.75	20.58±6.82	<0.01	11.66±4.16	11.92±4.08	12.22±3.79	12.22±3.79	0.07
	Left Lower Limb	12.26±6.40	15.82±6.42	19.91±6.69	20.44±6.78	<0.01	11.74±4.14	12.00±4.06	12.11±3.90	12.29±3.72	0.10
Sensory	Right	81.35±23.45	89.23±24.64	92±24.12	92±24.12	<0.01	83.11±20.71	84.81±19.7	86.66±19.8	86.66±19.8	<0.01
	Left	81.82±21.37	88.05±23.41	90.11±23.47	90.11±23.47	<0.01	84.59±19.13	85.48±18.9	87.03±19.9	87.33±20.2	<0.01

Table 3. Comparison of total American Spinal Injury Association total score between the two groups

	Motor			Sensory		
	Progesterone	**Placebo**	**p-value**	**Progesterone**	**Placebo**	**p-value**
Day 0	65.14±21.01	63.03±15.22	0.65	163.18±44.44	168.67±39.44	0.61
6 months	86.47±22.03	64.80±14.00	<0.01	182.11±47.33	176.2±40.54	0.59

Table 4. Serial trend on actual neural recovery in patients who received progesterone and placebo

Actual neural recovery								
Progesterone group					**Placebo Group**			
	6 days	1 month	6 months	p-value	6 days	1 month	6 months	p-value
Motor	10.02±6.42	19.79±11.66	21.32±13.44	<0.01	2.33±4.30	2.7±4.94	2.8±5.18	0.19
Sensory	14.11±16.83	18.94±23.86	18.94±23.86	0.03	2.86±5.27	7.26±8.52	7.53±8.49	0.005

Table 5. Comparison of actual neural recovery

Comparison of Actual neural recovery at Day 6 and at 6 months						
	Motor			**Sensory**		
	Progesterone	**Placebo**	**p-value**	**Progesterone**	**placebo**	**p-value**
Day 6	10.02±6.42	2.33±4.30	0.65	14.11±16.83	2.86±5.27	<0.01
6 Months	21.32±13.44	2.8±5.18	<0.01	18.94±23.86	7.53±8.49	0.02

Table 6. American Spinal Injury Association score between early vs late progesterone administration

American Spinal Injury Association (ASIA) score between Early vs late progesterone administration						
	Motor ASIA score			Sensory ASIA score		
	< 6 hours	> 6 hours	p- value	< 6 hours	> 6 hours	p- value
Day 6	80.54±14.14	62.3±30.05	0.02	186.83±39.87	154.4±58.70	0.06
1 Month	91.58±9.31	69.00±33.14	<0.01	192.66±38.82	156.8±57.89	0.04
6 Month	93.75±8.87	69.00±33.14	<0.01	192.66±38.82	156.8±57.89	0.04

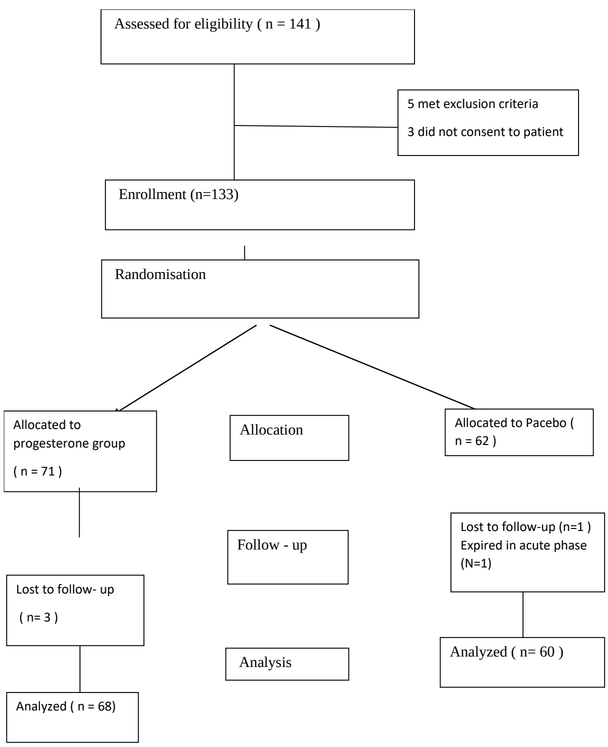

Figure 1. Allocation and analysis

Serial increase in mean sensory ASIA score was also noted from admission to day 6, 1 month and at 6 month follow up in both the arms. Improvement in sensory scores was noted over the time in both groups but a comparison of mean sensory ASIA score on day 0 and at 6 months between the groups did not reach significance (p= 0.59, table 3).

We also compared the serial actual neural recovery score at day 6, 1month and 6 month in both the groups (table 4). Mean motor as well as sensory ANR was found to be higher in the progesterone group. Serial increase in motor ANR at day 6, 1 month and 6 month in both groups was recorded but it was statistically significant only in the progesterone group (p=<0.01). Similarly, sensory ANR also increased in both groups but mean increase in progesterone group was much higher as compared to placebo group. On comparison of motor ANR (table 5) between progesterone and placebo groups at day 6 there was no statistically significant difference (p=0.65) but after 6 months of follow up there was a significant difference (p=<0.01).

On further post hoc analysis, we observed that there was statistically significant increase in mean motor ASIA score at day 6, 1 month and 6 month (80.54±14.14, 91.58±9.31, 93.75±8.87 respectively) if progesterone was administered early (within 6 hours of injury) (table 6). Mean sensory ASIA score also improved at day 6 and 1 month (186.83±39.87, 192.66±38.82) in the early and late progesterone administration groups and there was a significant increase noted with early administration. We also observed, that there was no significant gender difference between men and women in ASIA motor and sensory scores after 6 months of follow up in progesterone group as well as in placebo group. Similarly, neurological recovery was also comparable in surgical vs conservative management groups, in both the progesterone and placebo groups.

Only minor adverse effects such as transient diarrhea in one patient in placebo and nose block with pain at the injection site in the progesterone arm were reported. No major adverse events were recorded.

Discussion

The management of SCI till date remains challenging with no studies showing robust benefit of any drug or surgical intervention. The pathology behind SCI has largely been elucidated in the last decade with primary insult to oligodendrocytes and secondary insult to astrocytes and microglial cells by neuroinflammation.[8] Previous experiments using animal models suggested that SCI leads to increase in oligodendrocytes precursor cell (OPC) numbers but impairs their differentiation and maturation, thus preventing their remyelination.[9] Jure I et al, demonstrated that SCI leads to down regulation of transcription inhibitors in the first few days following injury but during this period even the transcriptional activators remain downregulated and hence the OPC fail to mature and remyelinate.[10] The interest in progesterone in SCI is due to its ability to act as a differentiation factor and enhance expression of transcription activation factors.

Labombarda F et al in animal studies on rats found progesterone to be beneficial and neuroprotective in central and peripheral nerve injuries. [11] They concluded that SCI results in reduced level of choline acetyltransferase and reduced expression of mRNA for α3 catalytic and β1 regulatory subunits of neuronal Na-K-ATPase and stimulates growth-associated protein (GAP-43). Progesterone administration leads to reversal of this effect by enhanced expression of choline acetyltransferase and restoration of mRNA to normal levels which leads to further enhancement of GAP-43 after a three day course of progesterone therapy.

Various transcription factors responsible for OPC differentiation and maturation were also studied previously (Olig2 and Nkx2.2 involved in OPC differentiation and maturation of myelin producing cells, Mash 1 involved in Nkx2.2 expression, Sox10 responsible for stimulation of premyelinating oligodendrocyte cells into myelinating oligodendrocytes via inducing myelin basic protein expression and MRF involved in myelin and internode formation). [12,13,14,15] Progesterone increased Olig 2 and Nkx2.2 expression and also increased the mRNA expression of Sox 10 and Mash 1.[10,16]

Progesterone is known to stimulate OPCs production and stimulate OPC survival and prevent apoptosis via progesterone receptor dependent mechanism.[16,17,18]

Progesterone administration leads to increased TGFβ1 (transforming growth factor beta 1) mRNA levels which in turn promotes OPC maturation [19] In vivo TGFβ pathway up regulates antimitotic genes p15, p21 and p27 as well stimulates pro-oligodendrogenic gene Olig 1 and Olig 2.[19] Progesterone not only increased the oligodendrocytes maturation but also decreased the pro-inflammatory mediators as well as astrocytes and microglial cells after SCI in experimental models.[20] Further, it also attenuated microglial cells toxicity via fractalkine-CX3CR1 signaling.[21,22]

3α, 5α-tetra-hydro-progesterone, a metabolite of progesterone showed promyelinating activity in central and peripheral nervous system in previously conducted studies.[23] Progesterone also increased brain derived neurotropic factor(BDNF), thereby reducing the neuronal loss by neuro inflammation and oxidative stress which are major mechanisms of cell loss in SCI.[5]

Corticosteroids have often been administered in SCI (NASCIS I, II, III trials) but a statement by the world neurosurgery society suggested them to be a double-edged sword recently.[24] The clinical trials on traumatic brain injury (TBI) patients showed no major adverse effects with use of progesterone (such as increased risk of breast cancer, thrombotic risk, feminization effect in male patients).[25] Given its promising role, in experimental studies and safety profile, progesterone seems to be a safe and effective drug in SCI patients.

The anti-inflammatory beneficial effect of progesterone is well established in experimental animal studies but in clinical trials on humans with brain or spinal cord injury the reports have been conflicting. Several randomized controlled trials previously showed progesterone mediated improvement in neurological outcome after traumatic brain injury (TBI).[25,26] However, multicentric larger trials followed soon after and showed no advantage of progesterone in TBI. Interestingly, these negative trials were questioned for their faulty extrapolation of data from pre-clinical trials and use of subjective measures for defining neurological improvement by some researchers.[27] In a very recently published meta-analysis Begemann M et al concluded that progesterone indeed had a beneficial effect in TBI, especially when given early and by the intramuscular route only.[28]

As the pathophysiology of neural injury is similar in TBI and SCI, we hypothesized that progesterone may have a beneficial effect in patients with SCI. A thorough literature review revealed only one study was conducted previously in this regard. In their randomized, double-blind, placebo controlled trial Aminmansour B et al assessed the effect of progesterone and vitamin D on outcome in patients after acute traumatic SCI and concluded that administration of both the drugs in combination were associated with better functional recovery and outcome.[29] In this study, motor and sensory ASIA scores improved significantly in the treatment group after 6 months of follow-up. Similar to the only other previously conducted study, we found a beneficial effect of progesterone in SCI with statistically significant improvement in sensory and motor ASIA scores after 6 months of follow up. On comparing the ASIA scores between the progesterone and placebo arm at 6 months, the motor scores showed significant improvement while the sensory scores between the groups did not. The actual neural recovery scores also showed significant improvement in the progesterone motor and sensory arm at 6 months. Additionally, the early administration of progesterone within six hours of the traumatic insult was more beneficial than the late administration (p= <0.01 vs p= 0.04). This time dependent benefit could be explained with the activation of neuroinflammation and destruction progressing relentlessly after the injury.

Limitations and scope

Firstly, we did not measure the baseline and post administration serum progesterone level, therefore a therapeutic range that would predict outcome could not be ascertained. Secondly, if the surgical intervention contributed towards clinical improvement in the progesterone group cannot be ascertained. However, patients undergoing surgical intervention were equally present in the placebo group also. Therefore, any procedural benefit would be similar in both the groups. Despite these limitations, our results remain valid as both the progesterone and placebo group had no significant baseline differences in demographics, level and clinical severity of injury and surgical management.

The areas that can be researched further include the effect of progesterone on expression of serum or cerebrospinal fluid inflammatory (IL-6, IL-8, TNF-α, MCP-1) and structural (GFAP, NSE, S100b, tau) biomarkers following SCI.[30]

Conclusion

No single agent till date has produced remarkable recovery in SCI. In the past few decades, with improved critical and supportive care the mortality after SCI has substantially reduced but the morbidity and caregiver burden remains high. Our study, shows a small but definitely beneficial effect of progesterone in SCI patients. Larger studies are needed to further understand the depth of this therapy. A deeper understanding into the pathophysiology of SCI should also lead to development of more molecules that are effective in reducing the morbidity associated with this condition.

Abbreviations:
SCI – Spinal cord injury
ASIA score – American Spinal Injury Association score
ANR score – Actual Neural Recovery
TBI- Traumatic Brain Injury

References

1. Walters BC, Hadley MN, Hurlbert RJ, Aarabi B, Dhall SS, Gelb DE, Harrigan MR, Rozelle CJ, Ryken TC, Theodore N; American Association of Neurological Surgeons; Congress of Neurological Surgeons. Guidelines for the management of acute cervical spine and spinal cord injuries: 2013 update. Neurosurgery. 2013 Aug;60(CN_suppl_1):82-91. Doi: 10.1227/01.neu.0000430319.32247.7f. PMID: 23839357.
2. Beattie MS, Hermann GE, Rogers RC, Bresnahan JC. Cell death in models of spinal cord injury. Prog Brain Res 2002;137:37–47.
3. Silva NA, Sousa N, Reis RL, Salgado AJ. From basics to clinical: A comprehensive review on spinal cord injury. Prog Neurobiol. 2014; 114:25–57.
4. Labombarda F, González S, Lima A, Roig P, Guennoun R, Schumacher M, De Nicola AF. Progesterone attenuates astro- and microgliosis and enhances oligodendrocyte differentiation following spinal cord injury. Exp Neurol. 2011 Sep;231(1):135-46.
Doi: 10.1016/j.expneurol.2011.06.001. Epub 2011 Jun 17. PMID: 21704617.
5. González SL, Labombarda F, González Deniselle MC, Guennoun R, Schumacher M, De Nicola AF. Progesterone up-regulates neuronal brain-derived neurotrophic factor expression in the injured spinal cord. Neuroscience. 2004;125(3):605-14. Doi: 10.1016/j.neuroscience.2004.02.024. PMID: 15099674.
6. Bracken MB. Steroids for acute spinal cord injury. Cochrane Database Syst Rev. 2012 Jan 18;1(1):CD001046. Doi: 10.1002/14651858.CD001046.pub2. PMID: 22258943; PMCID: PMC6513405.
7. Ditunno J, Young W, Donovan W, Creasey G. The international standards booklet for neurological and functional classification of spinal cord injury. Spinal Cord 1994;32(2):70–80.
8. Rabchevsky AG, Sullivan PG, Scheff SW. Temporal-spatial dynamics in oligodendrocyte and glial progenitor cell numbers throughout ventrolateral white matter following contusion spinal cord injury. Glia. 2007

Jun;55(8):831-43. Doi: 10.1002/glia.20508. PMID: 17390308.

9. Horky LL, Galimi F, Gage FH, Horner PJ. Fate of endogenous stem/progenitor cells following spinal cord injury. J Comp Neurol. 2006 Oct 1;498(4):525-38. Doi: 10.1002/cne.21065. PMID: 16874803; PMCID: PMC2553041.
10. Jure I, De Nicola AF, Labombarda F. Progesterone effects on oligodendrocyte differentiation in injured spinal cord. Brain Res. 2019 Apr 1;1708:36-46. Doi: 10.1016/j.brainres.2018.12.005. Epub 2018 Dec 5. PMID: 30527678.
11. Labombarda F, Gonzalez SL, Gonzalez DM, Guennoun R, Schumacher M, de Nicola AF. Cellular basis for progesterone neuroprotection in the injured spinal cord. J Neurotrauma. 2002 Mar;19(3):343-55. Doi: 10.1089/089771502753594918. PMID: 11939502.
12. Cai J, Zhu Q, Zheng K, Li H, Qi Y, Cao Q, Qiu M. Co-localization of Nkx6.2 and Nkx2.2 homeodomain proteins in differentiated myelinating oligodendrocytes. Glia. 2010 Mar;58(4):458-68. Doi: 10.1002/glia.20937. PMID: 19780200; PMCID: PMC2807475.
13. Sugimori M, Nagao M, Parras CM, Nakatani H, Lebel M, Guillemot F, Nakafuku M. Ascl1 is required for oligodendrocyte development in the spinal cord. Development. 2008 Apr;135(7):1271-81. Doi: 10.1242/dev.015370. Epub 2008 Feb 20. PMID: 18287202.
14. Emery B, Agalliu D, Cahoy JD, Watkins TA, Dugas JC, Mulinyawe SB, Ibrahim A, Ligon KL, Rowitch DH, Barres BA. Myelin gene regulatory factor is a critical transcriptional regulator required for CNS myelination. Cell. 2009 Jul 10;138(1):172-85. Doi: 10.1016/j.cell.2009.04.031. PMID: 19596243; PMCID: PMC2757090.
15. Hornig J, Fröb F, Vogl MR, Hermans-Borgmeyer I, Tamm ER, Wegner M. The transcription factors Sox10 and Myrf define an essential regulatory network module in differentiating oligodendrocytes. PLoS Genet. 2013 Oct;9(10):e1003907. Doi: 10.1371/journal.pgen.1003907. Epub 2013 Oct 31. PMID: 24204311; PMCID: PMC3814293.
16. Labombarda F, González SL, Lima A, Roig P, Guennoun R, Schumacher M, de Nicola AF. Effects of progesterone on oligodendrocyte progenitors, oligodendrocyte transcription factors, and myelin proteins following spinal cord injury. Glia. 2009 Jun;57(8):884-97. Doi: 10.1002/glia.20814. PMID: 19053058.
17. Garay L, Tüngler V, Deniselle MC, Lima A, Roig P, De Nicola AF. Progesterone attenuates demyelination and microglial reaction in the lysolecithin-injured spinal cord. Neuroscience. 2011 Sep 29;192:588-97. Doi: 10.1016/j.neuroscience.2011.06.065. Epub 2011 Jun 28. PMID: 21736923.
18. Labombarda F, Gonzalez S, Gonzalez Deniselle MC, Garay L, Guennoun R, Schumacher M, De Nicola AF. Progesterone increases the expression of myelin basic protein and the number of cells showing NG2 immunostaining in the lesioned spinal cord. J Neurotrauma. 2006 Feb;23(2):181-92. Doi: 10.1089/neu.2006.23.181. PMID: 16503802.
19. Palazuelos J, Klingener M, Aguirre A. TGFβ signaling regulates the timing of CNS myelination by modulating oligodendrocyte progenitor cell cycle exit through SMAD3/4/FoxO1/Sp1. J Neurosci. 2014 Jun 4;34(23):7917-30. Doi: 10.1523/JNEUROSCI.0363-14.2014. PMID: 24899714; PMCID: PMC4044250.
20. Hussain R, El-Etr M, Gaci O, Rakotomamonjy J, Macklin WB, Kumar N, Sitruk-Ware R, Schumacher M, Ghoumari AM. Progesterone and Nestorone facilitate axon remyelination: a role for progesterone receptors. Endocrinology. 2011 Oct;152(10):3820-31. doi: 10.1210/en.2011-1219. Epub 2011 Aug 9. PMID: 21828184; PMCID: PMC6285137.
21. Aryanpour R, Pasbakhsh P, Zibara K, Namjoo Z, Beigi Boroujeni F, Shahbeigi S, Kashani IR, Beyer C, Zendehdel A. Progesterone therapy induces an M1 to M2 switch in microglia phenotype and suppresses NLRP3 inflammasome in a cuprizone-induced demyelination mouse model. Int Immunopharmacol. 2017 Oct; 51:131-139. Doi: 10.1016/j.intimp.2017.08.007. Epub 2017 Aug 19. PMID: 28830026.
22. Roche SL, Wyse-Jackson AC, Gómez-Vicente V, Lax P, Ruiz-Lopez AM, Byrne AM, Cuenca N, Cotter TG. Progesterone Attenuates Microglial-Driven Retinal Degeneration and Stimulates Protective Fractalkine-CX3CR1 Signaling. PLoS One. 2016 Nov 4;11(11):e0165197.
Doi: 10.1371/journal.pone.0165197. PMID: 27814376; PMCID: PMC5096718.
23. Labombarda F, Pianos A, Liere P, Eychenne B, Gonzalez S, Cambourg A, De Nicola AF, Schumacher M, Guennoun R. Injury elicited increase in spinal cord neurosteroid content analyzed by gas chromatography mass spectrometry. Endocrinology. 2006 Apr;147(4):1847-59. Doi: 10.1210/en.2005-0955. Epub 2006 Jan 5. PMID: 16396987.
24. Bracken MB, Shepard MJ, Holford TR, Leo-Summers L, Aldrich EF, Fazl M, Fehlings M, Herr DL, Hitchon PW, Marshall LF, Nockels RP, Pascale V, Perot PL Jr, Piepmeier J, Sonntag VK, Wagner F, Wilberger JE, Winn HR, Young W. Administration of methylprednisolone for 24 or 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury. Results of the Third National Acute Spinal Cord Injury Randomized Controlled Trial. National Acute Spinal Cord Injury Study. JAMA. 1997 May 28;277(20):1597-604. PMID: 9168289.
25. Shakeri M, Boustani MR, Pak N, Panahi F, Salehpour F, Lotfinia I, Meshkini A, Daghighi S, vahedi P, Khani M, Taghiloo D. Effect of progesterone administration on prognosis of patients with diffuse axonal injury due to severe head trauma. Clin Neurol Neurosurg. 2013 Oct;115(10):2019-22.
Doi: 10.1016/j.clineuro.2013.06.013. Epub 2013 Jul 18. PMID: 23871679.

26. Aminmansour B, Nikbakht H, Ghorbani A, Rezvani M, Rahmani P, Torkashvand M, Nourian M, Moradi M. Comparison of the administration of progesterone versus progesterone and vitamin D in improvement of outcomes in patients with traumatic brain injury: A randomized clinical trial with placebo group. Adv Biomed Res. 2012;1:58. Doi: 10.4103/2277-9175.100176. Epub 2012 Aug 28. PMID: 23326789; PMCID: PMC3544099.
27. Stein DG. Embracing failure: What the Phase III progesterone studies can teach about TBI clinical trials. Brain Inj. 2015;29(11):1259-72. Doi: 10.3109/02699052.2015.1065344. Epub 2015 Aug 14. PMID: 26274493; PMCID: PMC4667711.
28. Begemann M, Leon M, van der Horn HJ, van der Naalt J, Sommer I. Drugs with anti-inflammatory effects to improve outcome of traumatic brain injury: a meta-analysis. Sci Rep. 2020 Sep 30;10(1):16179. Doi: 10.1038/s41598-020-73227-5. PMID: 32999392; PMCID: PMC7528105.
29. Aminmansour B, Asnaashari A, Rezvani M, Ghaffarpasand F, Amin Noorian SM, Saboori M, Abdollahzadeh P. Effects of progesterone and vitamin D on outcome of patients with acute traumatic spinal cord injury; a randomized, double-blind, placebo controlled study. J Spinal Cord Med. 2016 May;39(3):272-80. Doi: 10.1080/10790268.2015.1114224. Epub 2015 Dec 17. PMID: 26832888; PMCID: PMC5073761.
30. Leister I, Haider T, Mattiassich G, Kramer JLK, Linde LD, Pajalic A, Grassner L, Altendorfer B, Resch H, Aschauer-Wallner S, Aigner L. Biomarkers in Traumatic Spinal Cord Injury-Technical and Clinical Considerations: A Systematic Review. Neurorehabil Neural Repair. 2020 Feb;34(2):95-110. Doi: 10.1177/1545968319899920. Epub 2020 Jan 23. PMID: 31971869.

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Multilevel interlaminar fenestration with soft tissue decompression in lumbar canal stenosis. A single centre experience

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ABSTRACT

Introduction: Lumbar Canal Stenosis is the narrowing of the Spinal Canal and/or the intervertebral foramina, which causes compression of the thecal sac and/or caudal roots at a single vertebral level. Stenosis may be local, segmental, or generalised; bone or soft tissue can cause it. Stenosis can involve the bony canal, dural sac or both.[9]

Aim: Assessment of outcomes of multilevel fenestration with soft tissue decompression in lumbar canal stenosis.

Material and method: A prospective study comprised patients with two or more lumbar canal stenosis levels. The Study eliminated patients with infective pathology or recurrent surgery on identical levels. The surgical technique of canal decompression via multilevel fenestrations was used in this study, which enables the decompression of the neural structures while preserving as much of the bony and ligamentous structures as possible. Post-op clinical measures such as motor, sensory, post-op complications, and VAS score improvement assessed study outcomes.

Result: Studies showed improvement in symptoms of pain, motor power and sensory deficit, post-operative improvement in VAS score for pain, and improved quality of life in almost all the patients from day 7 to day 60 after surgery.

Conclusion: The study shows that "Multilevel interlaminar fenestration with Soft Tissue Decompression in Lumbar Canal Stenosis" is a feasible, safe, and effective approach to lumbar canal stenosis and is associated with minimal complications and minimal perioperative morbidity.

Keywords
lumbar canal stenosis,
visual analogue scale,
fenestration

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INTRODUCTION

Lumbar Canal Stenosis may be defined as the narrowing of the Spinal Canal and/or the intervertebral foramina, causing compression of the thecal sac and/ or caudal roots at a single vertebral level; narrowing may affect the whole canal or part of it. It may be local, segmental or generalised. The cord's normal dural tube cross-sectional area is about 180mm² (±50). The manifestation of stenosis becomes evident within the range of 100 to 130 mm², while relative stenosis is observed when the cross-sectional area of the dural tube measures 100mm² or below. Absolute stenosis is observed when the cross-sectional area of the dural tube measures 70mm² or below. Spinal stenosis refers to morphology, not the symptoms. Neurogenic claudication, alternatively referred to as pseudo-claudication, is a clinical manifestation that serves as a diagnostic indicator for the presence of spinal canal stenosis.

The condition above is characterized by a constellation of symptoms, including discomfort in the lower extremities, abnormal sensations such as tingling or numbness, and diminished muscular strength. These manifestations are notably exacerbated during ambulation and exertion. The prevailing characteristic observed in this case is the sensation of pain, which can manifest as either unilateral radicular pain or diffuse non-dermatomal symptoms. These symptoms typically originate from the buttock region and can extend to varying distances along the leg.

Wilson [12] classified Neurogenic claudication into postural due to positional accentuation of stenosis and ischemic due to failure to meet the oxygen demand of nerve root due to compressed canal. The primary objective of surgical intervention for lumbar canal stenosis entails alleviating compression exerted on neural tissue within the central and nerve root canal. The prevailing surgical modality for addressing lumbar canal stenosis is the implementation of decompression through a comprehensive laminectomy procedure.

Verbiest [1] suggests that nerve compression is due to encroachment by the articular process on the spinal cord, and the laminectomy alone is not sufficient without the removal of the medial part of the articular process. Amundson et al. [3] stated that radiological changes were more extensive than expected from the clinical picture, and the degree of narrowing did not correspond to the degree of clinical affection. Herno et al. [7] stated that the patient's satisfaction with the surgery result was more important in surgical outcome than the degree of decompression detected on the CT scan. Surgery for lumbar canal stenosis can be very successful in most cases for the leg symptoms. However, depending on the severity of the nerve compression and the length of time the nerve has been compressed, there may be some permanent damage that is not relieved with surgery. Success for back pain relief is less reliable with surgery than the relief of leg symptoms.

Patient selection

All consecutive patients with acute traumatic SCI presenting within 24 hours of the injury between 18 and 65 years of age were included. Patients outside the specified age limit, 24 hours beyond the SCI, those with involvement of only the radicles or nerve roots, with firearm injury, with life-threatening morbidities, history of drug addiction or on steroids, pregnant women and unwilling for a minimum six months regular follow up were excluded from the study.

Aim

This study aimed to evaluate the results of multilevel fenestration with soft tissue decompression in lumber canal stenosis.

Material and method

In this prospective study, 14 patients were included with a clinic-radiological diagnosis of lumbar canal stenosis of two or more two levels. All patients were evaluated clinically and radiologically. Patients having recurrent surgery on the same level, infective pathology, or spinal instability were excluded. Out of 14 patients, four patients had an intraoperative conversion to laminectomy and were excluded. The subjects underwent surgical procedures while positioned in the prone position while under the effects of general anaesthesia. During the procedure of interlaminar fenestration, the bone surrounding the interlaminar space of the affected segment (as indicated by abnormal clinical and myelographic findings) was carefully removed. This removal was performed along with removing the ligamentum flavum and a portion of the facet joint, known as partial undercutting facetectomy. However, it is important to note that this surgical intervention intentionally preserved the neighbouring laminae, spinal process with interspinous ligament, and zygapophyseal joints. The procedure involved a lateral extension of fenestration, specifically a foraminotomy, to alleviate the compression on nerve roots exhibiting signs of swelling and oedema. The operative finding has been duly documented and its correlation with the clinic-radiological impression has been noted. The assessment of decompression adequacy was conducted intraoperatively through the evaluation of root mobility and the probing of the root canal.

The study outcome was evaluated in the post-op period based on clinical parameters like Sensory/Motor examination and improvement in pain assessed by visual analogue scale. The outcome was accessed according to Modified MacNab criteria. [10]

Result

Backache (100%) and radiculopathy (30%) were the most frequent symptoms. The neurogenic claudication was also present in all patients. Only one patient had involvement of the bladder and bowel. The mean duration of symptoms was 44.2±15.92 months. Eighty (80%) patients had a clinical history of more than 24 months. The mean walking capacity was 440±107.5meters, ranging from 300-600 meters, and 60% of patients had a walking capacity of <400 meters. Six (60%) patients also had sensory loss of variable dermatomes. The SLR was positive in 90% of patients on the right side and all patients (100%) on the left side. On motor examination, right and left lower limb weakness was found in 50% and 90%, respectively. In the preop period, 30 % of patients had radicular numbness in the lower limb. 60%population had a sensory deficit, and 90% of the population had a motor deficit of grade 2/5 to grade 4/5. In the post-op period, no patients had a sensory deficit, and 100% had motor power between 4/5 to 5/5. Preoperatively in the right lower limb, SLR tests were positive in 90% of cases, and 10.0% were negative. During the follow-up period at day 14 in the right lower limb, 30% of cases were SLR-positive, and 70% were negative. Preoperatively, in the left lower limb, 100% of cases were SLR test positive. During the follow-up period on day 14 in Lt. Lower limb, 90% of cases were SLR negative, and 10% were positive. On radiological evaluation, it was found that on plain digital X-rays, all patients had features of degenerative changes. The degenerative changes in the ratio of vertebral body diameter vs canal A.P diameter in A.P and lateral view, which was less than 1 cm, showed the presence of osteophytes and bone spurs. The MRI findings are summarised in Table 1 and 2. The disc prolapse was most common at L4-5 levels in 80% of patients. The second most common site was L5-S1 in 40% of patients.

Table 1. Levels of spinal canal stenosis on MRI images

No. of stenosed levels	Up to 2 levels	Up to 3 levels	Up to 4 levels
No. of patients	1	7	2

Table 2. Levels of disc prolapse

PIVD	L1-2	L2-3	L3-4	L4-5	L5-S1
Yes	1	1	2	8	4
No	10	9	8	2	6

The ten patients who underwent multilevel fenestration were analysed. Intraoperative images of representative cases are shown in Figure 1. Interlaminar fenestration was done up to four levels in 2 cases, up to three levels in 7 patients and up to two in 1 case. Based on MRI findings, disc excision was done up to a single level in 5 patients, up to two levels in 3 patients and the rest two patients, up to 3 levels. Facet joint hypertrophy was present in all cases, for which partial medial facetectomy was done in all cases.9 cases out of 10 had lateral root canal stenosis for which foraminotomy was done, and adequacy of canal diameter was checked with probing of the root canal. Postoperatively, patients had significant improvement in radiculopathy and improvement in claudication distance. There was also a significant improvement in SLR in both legs (Table 3) in follow-up. The mean VAS score in follow-up was also significantly lower than preop VAS (Table 4). The average period of ambulation was 48±5.57hrs. No wound infection, CSF leak or residual pain and mobility restriction were noted during the post-op period.

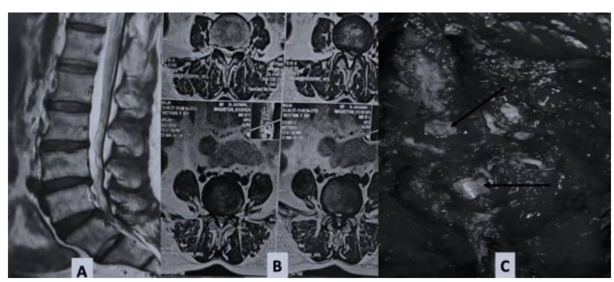

Figure 1. Shows preoperative MRI images of canal stenosis (A & B), intraoperative image (C) with interlaminar fenestration at two levels

Table 3. Pre-op and postop VAS scoring

Patients	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th
Vas score (preop)	7	6	6	6	6	5	6	7	7	7

Vas score Day 7	4	4	5	4	5	5	6	4	4	5
Vas score Day 14	2	2	3	3	2	3	3	2	3	4
Vas score Day 28	2	2	2	3	2	3	3	2	3	3
Vas score Day 60	1	2	2	2	2	2	3	2	3	2

Table 4. VAS score mean value

	Preoperatively	D7	D14	D28	D60
VAS score mean value	6.3	4.6	2.7	2.5	2.1

Discussion

In this study, an effort has been made to "Evaluate the Results Of Multilevel Fenestration With Soft Tissue Decompression In Lumbar Canal Stenosis". All patients presented with a positive history suggestive of lumbar canal stenosis. The average duration of localised back pain was 44.2±15.92 months, shorter than the duration reported in the study conducted by Ahmet Colak et al. [2], where it was 4.4 years. In all of these cases, the patients have had radicular pain before the pain in their lower limb, which is likely due to degenerative changes in the lumbar spine. Only one patient out of 10 has a positive history of bladder-bowel involvement. Neurogenic claudication was present in all cases. Similar findings were recorded by Ahmet Colak et al.

In a study done by Patond KR, Kakodia SC,[11] A total of sixteen patients, consisting of seven males and nine females, who exhibited clinical, neurological, and radiological symptoms of lumbar canal stenosis, underwent interlaminar (fenestration) decompression treatment. During a two-and-a-half-year follow-up, the results were good in 73.3% of cases and fair in 26.7%. The post-operative phase was uniformly uneventful.

According to a study by Puneet Gupta et al., [6] patients with acquired degenerative lumbar canal stenosis multilevel interlaminar fenestration with discectomy, if required, were carried out. Retrospective analysis assessed the outcome by assessing the relief of backache and neurological claudication. The mean age of patients was 50.4 years, and the average duration of neurological claudication was nine months. Diagnosing the degenerative lumbar canal stenosis was made by clinical examination and confirmed by radiological and MRI measurements of the cross-section area of the neural canal. Interlaminar fenestration was done at four levels and three levels in six patients each, while it was done at two levels in the remaining three patients. None of the patients reported immediate or late onset of backache or spinal movement restriction, indicating spinal stability. None of the patients had neurological claudication in the postoperative period.

Our study assessed pre and post-operative pain with VAS score. The VAS score was assessed pre-operatively and post-operatively on day 7, day 14, day 28, and 60 when all the analgesic medications were stopped. VAS score was significantly reduced gradually on follow-up in all the patients. During the preop period, the mean VAS score was 6.3±.67. On day 7, all patients' mean VAS score was 4.60±.69, similarly on day14 was 2.70±.67; on day 28 was 2.50±.52, and on day 60 was 2.1±.56. On comparison from pre-op VAS score vs postoperative VAS score on day 7, day14, day28 and day 60 had a p-value <.05 which shows a statistically significant association in relief of pain during follow up period after surgery done in our institution. All the patients showed a gradual improvement in pain due to lumbar canal stenosis after surgery. In the Ahmet Colak et al. [11] study, the results of VAS scoring during preop. at three months and 12 months after decompression surgery in Lumber canal stenosis was 7.0, 5.5, and 4.0, respectively. According to Devkota UP et al. [4], with minimally invasive open lumber discectomy, 98.33 % of patients had an improvement in the radicular pain and ambulation commenced from the first postoperative day. There were three instances of inadvertent dural tear without fascicle injury and one instance of residual disc requiring reoperation. The outcome was accessed according to Modified MacNab criteria, and results were Excellent in 1 case, Good in 7 cases and Fair in 2 cases. Our results are almost the same compared to the study done by Iwatsuki [8] and Fu YS. [5]

Table 5. Shows the comparison of outcomes among various studies according to Modified MacNab criteria

Pain relief	Iwatsuki K. [8]	Fu YS, Zeng [5]	Patond KR, Kakodia SC [11]	Present study
Excellent	45(95.8%)	68(89%)	0%	1(10%)
Good	1(2.1%)	8 (11%)	11(73.3%)	7 (70%)
Fair	1(2.1%)	0	4(26.7%)	2(20%)
Poor	0	0	0%	0
Total of excellent and good outcome	46(97.9%)	76(100%)	11(73.3%)	8(80.0%)

Conclusion

Degenerative lumbar spinal stenosis can be decompressed adequately with Multilevel Fenestration with Soft Tissue Decompression, including resection of the Interspinous Ligament, except in cases with severe spinal stenosis where Laminectomy is still the procedure of choice. Although resection of the interspinous ligament may be associated with fear of spinal instability, it provides a wider room to work, so fewer chances of intraoperative dural and nerve root injury and complete removal of pressure over the spinal column. The Fenestration operations combined with soft tissue decompression, including resection of the Interspinous Ligament, obtained satisfactory outcomes at low cost, and it can be a standard procedure for the surgical treatment of mild to moderate-grade degenerative lumbar spine stenosis.

Limitation of study

1. Study population is small, so it is hard to generalize the result.
2. Follow-up period is small, especially for long-term recurrence and the need for re-surgery.

References

1. A radicular syndrome from developmental narrowing of the lumbar vertebral canal. The Journal of bone and joint surgery. British volume, 36-B (2), 230–237, 1954.
2. Ahmet Çolak,1 Kıvanç Topuz,1 Murat Kutlay,1 Serdar Kaya,1 Hakan Şimşek, Ahmet Çetinkal,1 and Mehmet N. Demircan1 A less invasive surgical approach in the lumbar lateral recess stenosis: direct approach to the medial wall of the pedicle Eur Spine J 17(12): 1745–1751,2008.
3. Amundson T, Weber H, Lilleas F. Lumbar spinal stenosis; clinical and radiologic features. Spiner 20(10):1178-1186,1995.
4. Devkota UP; Lohani S; Joshi RM Minimally invasive open lumbar discectomy: An alternative to micro-lumbar discectomy. Kathmandu Univ Med J (KUMJ) 7(27):204-8,2009.
5. Fu YS, Zeng BF, Xu JG. Long-term outcomes of two different decompressive techniques for lumbar spinal stenosis. Spine (Phila Pa 1976) 33(5):514-518,2008.
6. Gupta P, Sharma S, Chauhan V, Maheshwari R, Juyal A, Agarwal A. Interlaminar fenestration in lumbar canal stenosis- a retrospective study Indian Journal of Orthopaedics. 01/2005.
7. Herno A, Saari T, Suomalainen O, Airaksinen O. The degree of decompressive relief and its relation to clinical outcome in patients undergoing surgery for lumbar spinal stenosis. Spine 24(10):1010-4,1999.
8. Iwatsuki K, Yoshimine T, Aoki M. Bilateral interlaminar fenestration and unroofing for the decompression of nerve roots by using a unilateral approach in lumbar canal stenosis. Surg Neurol 68(5):487-492,2007.
9. Postacchini F. Lumbar spinal stenosis and pseudostenosis. Definition and classification of pathology. Ital J Orthop Traumatol 9(3):339-350,1983.
10. Macnab I. Negative disc exploration. An analysis of the causes of nerve-root involvement in sixty-eight patients. J Bone Joint Surg Am 53:891–903,1971.
11. Patond KR; Kakodia SC-Interlaminar decompression in lumbar canal stenosis. Neurol India 47(4):286-9,1999.
12. Wilson CB. Significance of the small lumbar spinal canal: cauda equina compression syndromes due to spondylosis. 3: Intermittent claudication. J Neurosurg 31(5):499-506, 1969.

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Anterior cervical surgery for the treatment of cervigonenic headache caused by cervical spondylosis. A case report

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ABSTRACT
Cervicogenic headache (CEH) is a common disorder with cervical spine-specific nociceptive headache symptoms. Cervicogenic headaches have been studied for years, but diagnosis and treatment are continually evolving. Due to non-specific criteria and its relationship with cervical degenerative illness, diagnosis may be underestimated. In many situations, it contributes to myelopathy or radiculopathy. Local anaesthetic blocks are used to confirm the diagnosis; however, non-invasive methods are being explored. Identifying the nociceptive origin improves pain management. Physical therapy and percutaneous interventional procedures are used to treat isolated CEH. However, cervical decompression and/or fusion are often performed in situations of cervical myelopathy and/or radiculopathy. Here we report a 57-year-old female with cervicogenic headache treated with anterior lower cervical discectomy to relieve cervical myelopathy and/or radiculopathy headaches.

Keywords
surgery,
cervigonenic headache,
cervical spondylosis

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INTRODUCTION

Cervicogenic headache (CEH) is common and recurring. The prevailing belief is that the source of discomfort is in the head, although the actual site of the lesion is situated in the neck. Depending on diagnostic criteria, chronic headache (CHE) prevalence varies in the general population. CEH is estimated to affect 1-4.1% of the population.[1] The International Headache Society (IHS) defined cervicogenic headache (CEH) in 2018 as a headache caused by cervical spine or its disc, bone, or soft tissue disruption. It usually causes neck pain, but can manifest as headache.[2]

The precise etiology of CEH is inadequately comprehended. Referred pain, caused by upper cervical nerve degeneration (C1-C3) has long been known. However, cervical spondylosis is most common in the lower cervical spine (vertebrae C4 to C7) and rare in the upper cervical spine (vertebrae 5 and 6).[2]

A recent retrospective study found that anterior lower cervical discectomy may relieve concurrent cervicogenic headache (CEH) according to ICHD-3 beta.[3] Multicenter randomized clinical trial has shown that anterior lower cervical discectomy relieves cervical myelopathy and/or radiculopathy headaches. As shown by follow-up examinations, this intervention's benefits last up to 7 and 10 years.[4] Yang et al reported anterior cervical decompression and fusion (ACDF) can effectively relieve CEH associated with cervical myelopathy and/or radiculopathy.[5]

Case report

The patient was a 57-year-old Asian female. Her chief complaint was pain headache especially at occipital region worsening in the last 1 year. The pain reffered to retro-orbital and left face. She had history of surgery for the left face pain. No abnormality was noted on neurological examination, and only age-related changes were observed on a head magnetic resonance imaging (MRI) scan, showing no abnormality causing headache.

Cervical Xray showed spondylosis cervical with straight cervical and narrowing of intervertebralis disc C4/C5, C5/C6, C6/C7 in flexion and extension position. Patient was diagnosed with cervicogenic headache /cervico cephalic syndrome and spondylosis cervical. Under general anesthesia, ACDF C4-5, C5-6 was performed. After surgery, no new neurological abnormality or other complications developed. Early post operation headache was improved and completely disappear at one month post operation.

Figure 1. Cervical Xray pre-operation.

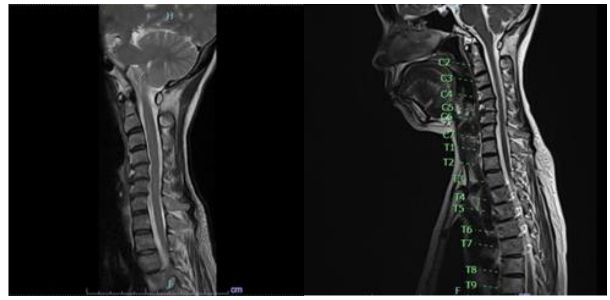

Figure 2. MRI Cervical pre-operation,

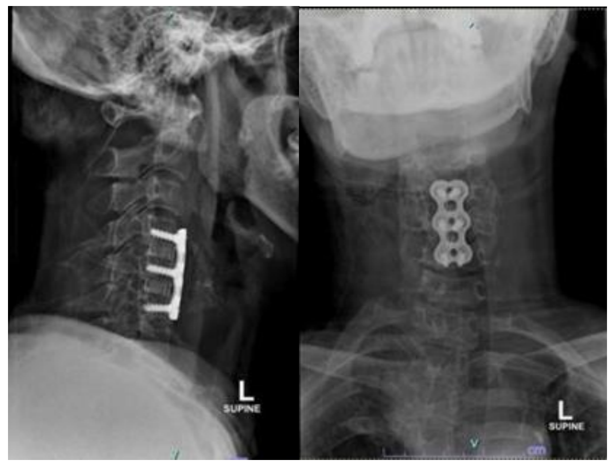

Figure 3. Cervical Xray post operation.

Discussion

Currently, there is a lack of consensus regarding the optimal treatment for CEH due to limited understanding of the underlying mechanism responsible for this form of pain. The therapeutic interventions employed in clinical practice encompass a spectrum of approaches, varying in invasiveness. These include conservative measures such as rest, application of heat, physical therapy involving massage, administration of nonsteroidal anti-inflammatory drugs and muscle relaxants, as well as the utilization of oral anticonvulsant medications. In more severe cases, surgical techniques may be employed as a therapeutic modality. However, none of the aforementioned treatments have demonstrated sufficient results and provide substantial evidence to be considered the optimal therapy for CEH.[6]

Several studies have been conducted to examine the various impacts of anterior cervical surgery on headaches linked to cervical myelopathy and/or radiculopathy in the short, medium, and long term.[7] It is important to note that headaches associated with cervical spondylosis should not be equated with "cervicogenic headache".[8]

The concept of cervicogenic headache (CEH) coming from the lower cervical spine was initially introduced by Diener et al. Their study revealed that in 80% of patients with lower cervical disc herniation (below C4), CEH showed improvement or resolution following surgical intervention.[9]

Nociceptive afferent originating from the lower cervical roots additionally converged upon the trigeminocervical nucleus.[7,8] The distribution of nerves inside the cervical intervertebral region is innervated in a multisegmental manner. The trigeminocervical nucleus has the potential to receive afferent input from both the C1-C3 spinal nerves as well as partially from the C4-C8 nerves. The present study has discovered a correlation between cervical spondylosis in the lower cervical spine and cervicogenic headache (CEH). Furthermore, it has been observed that anterior cervical discectomy and fusion (ACDF) surgery might lead to a substantial improvement in CEH. These findings suggest the possibility of nociceptive afferents originating from lower cervical neurons converging onto the trigeminocervical nucleus.[9,10]

Intervertebral disc degeneration is the prevailing pathological alteration observed in cases of cervical spondylosis. The prevalence of chronic neck discomfort has been widely acknowledged to be associated with degenerative cervical intervertebral disc conditions.[11]

It is postulated that the inflammatory response triggered by the degeneration of cervical discs could potentially activate nociceptors within the intervertebral discs of the neck, thereby leading to the manifestation of neck pain. Simultaneously, the nociceptive excitabilities are sent to the trigeminocervical nucleus located in the upper cervical spinal cord, leading to the manifestation of chronic episodic headache (CEH). Indeed, the presence of cervical radiculopathy or myelopathy can lead to the irritation or compression of cervical nerve roots or the spinal cord, which in turn might impact the nociceptive afferents of the affected cervical intervertebral disc and perhaps exacerbate headaches.[12,13] Hence, it is posited that anterior cervical discectomy and fusion (ACDF) may potentially enhance cervical epidural hematoma (CEH) by eliminating the degenerative cervical disc together with its internal nociceptors, as well as by decompressing the cervical nerve root or spinal cord. Furthermore, it should be noted that posterior laminoplasty has the potential to alleviate cervical spondylotic myelopathy by means of indirect decompression of the spinal cord. Nevertheless, it should be noted that laminoplasty has been found to have a lower level of durability compared to anterior cervical fusion in terms of providing relief from headaches.[14]

Various ideas have been proposed in the existing literature to elucidate the etiology of cervicogenic headache (CEH) in the lower cervical spine. Bogduk and Govind proposed that a direct link between the lower cervical afferents and the trigeminocervical nucleus is not evident. However, they hypothesized the involvement of intermediary mechanisms, such as secondary spinal kinesthesia and muscular tension, which might potentially impact the upper cervical joints. The efficacy of headache alleviation may vary depending on whether anterior arthroplasty or fusion is employed, particularly when considering the specific mechanism of spinal kinesthesia.[15] Lombardi et al. conducted a prospective, multicenter study with a 10-year follow-up to examine the efficacy of arthroplasty and ACDF in alleviating headaches related to cervical radiculopathy and/or myelopathy. Their post-hoc analysis revealed that both interventions were effective in relieving headaches, but the arthroplasty group exhibited lower headache scores compared to the ACDF group. This finding provides evidence for the involvement of spine kinematics in the development of cervicogenic headache.[16]

Additional hypothesized processes include the potential involvement of the sinuvertebral nerve (SVN) or irritation of the sympathetic nerve at the uncovasculoradicular junction, anterior dura mater, or cervical posterior longitudinal ligament (PLL). The cervical dura and posterior longitudinal ligament (PLL) possess distinct sympathetic innervation, which can potentially elicit sympathetic responses.[17] The activity has the potential to traverse the ganglia and the sympathetic trunk, ultimately reaching the trigeminocervical nucleus, hence potentially triggering craniofacial pain.

Furthermore, irritation of the spinal accessory nerve (SVN) at the uncovasculoradicular junction and anterior dura could potentially be the underlying factor contributing to cervicogenic headache (CEH). Given that the inferior branch of the spinal trigeminal nucleus (SVN) has the capacity to extend to three segments below its point of origin, nociceptive signals originating from the lower cervical segment, specifically C6, can transmit to the third cervical nerve and subsequently to the trigeminocervical nucleus. This process ultimately contributes to the development of cervicogenic headache (CEH). Both anterior cervical surgery and posterior decompression have been shown to alleviate headache symptoms.[15]

Three surgical interventions, including ACDF (anterior cervical discectomy and fusion), arthroplasty, and laminoplasty, exhibit substantial efficacy in relieving headache symptoms.[15]

Conclusion

Neck discomfort is a common symptom observed in cases of cervical spondylosis that are aggravated with cervicogenic headache (CEH). The anterior cervical discectomy and fusion (ACDF) procedure has been shown to be a successful intervention for the alleviation CEH symptoms in patients with concurrent cervical myelopathy and/or radiculopathy.

References

1. Bogduk N, Govind J. Cervicogenic headache: an assessment of the evidence on clinical diagnosis, invasive tests, and treatment. Lancet Neurol [Internet]. 2009;8(10):959–68. Available from: http://dx.doi.org/10.1016/S1474-4422(09)70209-1
2. Olesen J. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, 3rd edition. Cephalalgia. 2018;38(1):1–211.
3. Pang X, Liu C, Peng B. Anterior cervical surgery for the treatment of cervicogenic headache caused by cervical spondylosis. J Pain Res. 2020;13:2783–9.
4. Thind H, Ramanathan D, Ebinu J, Copenhaver D, Kim KD. Headache relief is maintained 7 years after anterior cervical spine surgery: Post hoc analysis from a multicenter randomized clinical trial and cervicogenic headache hypothesis. Neurospine. 2020;17(2):365–73.
5. Yang L, Li Y, Dai C, Pang X, Li D, Wu Y, et al. Anterior cervical decompression and fusion surgery for cervicogenic headache: A multicenter prospective cohort study. Front Neurol. 2022;13:1–10.
6. Liu H, Ploumis A, Wang S, Li C, Li H. Treatment of Cervicogenic Headache Concurrent with Cervical Stenosis by Anterior Cervical Decompression and Fusion. Clin Spine Surg. 2017;30(8):E1093–7.
7. Liu JJ, Cadena G, Panchal RR, Schrot RJ, Kim KD. Relief of Cervicogenic Headaches after Single-Level and Multilevel Anterior Cervical Diskectomy: A 5-Year Post Hoc Analysis. Glob Spine J. 2016;6(6):563–70.
8. Huang WC, Chen SP, Wang SJ. Are surgically remediable headaches associated with cervical spondylosis equivalent to "cervicogenic headaches"? Neurospine. 2020;17(2):374–6.
9. Diener HC, Kaminski M, Stappert G, Stolke D, Schoch B. Lower cervical disc prolapse may cause cervicogenic headache: Prospective study in patients undergoing surgery. Cephalalgia. 2007;27(9):1050–4.
10. Persson LCG, Carlsson JY, Anderberg L. Headache in patients with cervical radiculopathy: A prospective study with selective nerve root blocks in 275 patients. Eur Spine J. 2007;16(7):953–9.
11. Peng B, DePalma MJ. Cervical disc degeneration and neck pain. J Pain Res. 2018;11:2853–7.
12. Bir SC, Nanda A, Patra DP, Maiti TK, Liendo C, Alireza M, et al. Atypical presentation and outcome of cervicogenic headache in patients with cervical degenerative disease: A single-center experience. Clin Neurol Neurosurg. 2017;159(May):62–9.
13. Sun Y, Muheremu A, Yan K, Yu J, Zheng S, Tian W. Effect of different surgical methods on headache associated with cervical spondylotic myelopathy and/or radiculopathy. BMC Surg [Internet]. 2015;15(1):3–9. Available from: http://dx.doi.org/10.1186/s12893-015-0092-3
14. Shimohata K, Hasegawa K, Onodera O, Nishizawa M, Shimohata T. The Clinical Features, Risk Factors, and Surgical Treatment of Cervicogenic Headache in Patients With Cervical Spine Disorders Requiring Surgery. Headache. 2017;57(7):1109–17.
15. Schrot RJ, Mathew JS, Li Y, Beckett L, Bae HW, Kim KD. Headache relief after anterior cervical discectomy: post hoc analysis of a randomized investigational device exemption trial: clinical article. J Neurosurg Spine. 2014. 21:217–22. doi.
16. Lombardi JM, Vivas AC, Gornet MF, Lanman TH, McConnell JR, Dryer RF, et al. The effect of ACDF or arthroplasty on cervicogenic headaches: a post hoc analysis of a prospective, multicenter study with 10-year follow-up. Clin Spine Surg. 2020. 33:339–44.
17. Yamada H, Honda T, Yaginuma H, Kikuchi S, Sugiura Y. Comparison of sensory and sympathetic innervation of the dura mater and posterior longitudinal ligament in the cervical spine after removal of the stellate ganglion. J Comp Neurol. 2001. 434:86–100.

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Current insights and surgical interventions in craniovertebral junction instability. A systematic review and meta-analysis

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ABSTRACT
Background: The Craniovertebral Junction (CVJ) is prone to various pathologies, including instability and congenital anomalies. Understanding these conditions and their management strategies is critical for effective treatment.
Materials and Methods: A systematic search was conducted in Science Direct and PubMed databases following PRISMA guidelines. Inclusion criteria encompassed studies addressing craniovertebral instability and associated pathologies. Six systematic investigations were assessed for methodological quality. Data extraction involved 702 patients with CVJ issues, among which 129 had related conditions, while 279 displayed normal CVJ. Surgical interventions encompassed various techniques such as C1-C2 fixation, posterior decompression, and screw placements.
Results: Among 702 patients studied, atlantoaxial subluxation, basilar invagination, and odontoid fractures were observed in 129 cases. Surgical treatments showed favorable outcomes, with fusion achieved within a year post-surgery for both C1-C2 fixation techniques and posterior decompression strategies. Studies highlighted successful outcomes in cases of cervical myelopathy, especially with early occipitocervical fusion.
Conclusion: Managing atlantoaxial instability remains a debated topic, with varying success rates observed in different surgical interventions. Recommendations emphasize the importance of stabilization techniques and imaging modalities for effective preoperative planning and postoperative care. However, limitations in available data underscore the need for further research to refine treatment strategies for better patient outcomes in this complex area of spinal pathology.

Keywords
craniovertebral junction,
atlantoaxial,
instability,
basilar invagination,
fusion

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Introduction

The Craniovertebral Junction (CVJ) constitutes a critical juncture susceptible to various pathological conditions encompassing infections, inflammations, degenerative disorders, neoplasms, and congenital anomalies, such as severe deformities and neurological impairments [1]. Emerging genomic associations, particularly within the fibrillin gene (FBN1), underscore its linkage to Chiari malformation, Basilar Invagination, and atlantoaxial dislocation [2]. Notably, a significant correlation exists between specific gene variants in newborns with Marfan syndrome and morphological irregularities observed in C1-C2 joints among patients experiencing basilar invagination and atlantoaxial dislocation [2].

Remarkably, around 84 syndromes are believed to be intricately connected to CVJ, including both autosomal dominant and recessive forms, often within chromosomal regions like 3p21.1-14.1. Among these syndromes, Larsen syndrome stands out, characterized by deletions in filamin B, a protein crucial for actin binding [3].

The atlantoaxial joint, recognized as the most mobile joint in the neck, embodies a predisposition for instability, allowing extensive circumferential movement owing to its unique articular surfaces, which can vary from rounded to flattened. While instability is common in this joint, its structural variability facilitates motion, albeit with potential complications. Anomalous atlanto-dental alignment captured during neck flexion and extension aids in identifying atlantoaxial instability, which manifests in various forms—vertical, lateral, circumferential, central, or axial—resulting in misalignment of the facet bases [4].

Furthermore, the connection between basilar invagination (BI) and irreducible atlantoaxial dislocation poses a grave concern, as the protrusion of the bulb can compress the cervical spinal cord, potentially leading to irreparable spinal cord injury or stenosis and subsequent limb dysfunction. Advanced imaging technologies like computed tomography (CT) enable precise examination of affected regions, with the primary objective being the correction of atlantoaxial instability to address basilar invagination [5].

This article aims to synthesize the most current insights into craniovertebral junction instability, particularly in patients presenting with concomitant pathologies, emphasizing the critical goal of stabilization.

Materials and methods

Search Strategy and Selection Criteria

A systematic search strategy was executed in the Science Direct and PubMed databases utilizing Mesh terms encompassing Craniovertebral Junction Diseases, Atlanto-Axial Joint, Fusion of C2-C3 Vertebrae, Basilar Invagination, and related pathologies. The search encompassed articles from database inception until August 2023, aligning with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. Adhering to the PRISMA guidelines, the search process involved meticulous screening of articles (Figure 1).

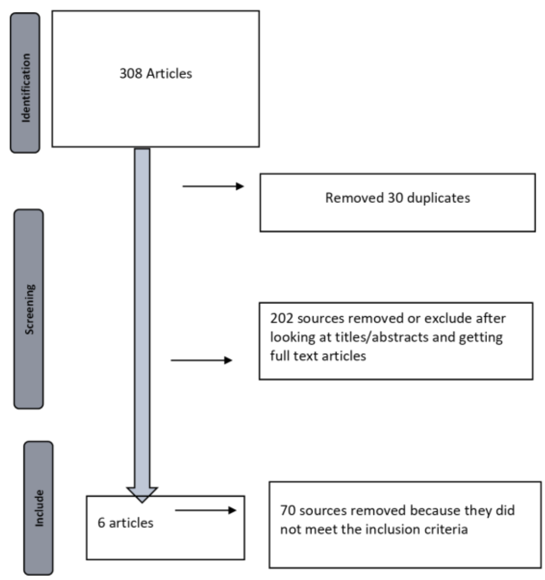

Figure 1. PRISMA protocol outlining the systematic review and meta-analysis of CVJ Atlantoaxial instability.

Inclusion/Exclusion Criteria

The search strategy was structured around comprehensive Mesh terms and keywords linked to diseases associated with basilar invagination, C2-3 fusion, atlantoaxial instability, and craniovertebral junction instability.

Inclusion criteria comprised:

- Analysis addressing craniovertebral instability associated with pertinent pathologies.
- Investigations focusing on atlantoaxial dislocation and the progression, alterations, and stabilization of craniovertebral instability.

Criteria for Exclusion

- Exclusion of patients under 18 years without known medical conditions related to the craniovertebral junction or atlantoaxial instability.
- Elimination of cases that did not meet the specified inclusion criteria.

Data Extraction

Data extraction encompassed comprehensive retrieval of relevant information, including atlantoaxial instability, C2-3 Fusion, Basilar invagination, demographic details, interventions, controls, and pertinent methodologies, following standardized protocols in alignment with the study parameters.

Risk of Bias Assessment

The methodological quality of six systematic investigations was evaluated using the risk of bias assessment technique.

Analytical Statistics

Summary statistics such as mean difference and odds ratio (OR) were utilized for each relevant occurrence. Outcomes of interest and specific data extracted from included studies were defined using weighted mean difference (OR) and 95% confidence interval (CI). Statistical significance was set at $P < 0.05$ for main and subgroup analyses. Analysis software employed included Review Manager, Rayyan version 5.3, Jasp, and GraphPad 8.0.

RESULTS

The review encompasses various studies focusing on cervical spine and craniovertebral junction (CVJ) conditions and surgical interventions. In this systematic review, 308 articles were found using the various databases previously mentioned. We then went ahead and eliminated 30 duplicates and 202 articles that did not fit the criteria for our study, regardless of whether they were full texts, titles, abstracts, or any combination of these. Six publications were included for the standard base of our study after 70 articles that did not fulfill the inclusion criteria were removed, in accordance with the PRISMA systematic review process. fig. As stated by the writers, particularly Chang et al. [6]. conducted a retrospective analysis of 129 instances, with the control group accessing 297 cases for 44% of CVJ patients' C1–C2 fixation and decompression.

For instance, the authors state that previous research support their paper by measuring the various techniques. See the table for methods that bolster the study, the most common decompressions at the C1–C2 level, the various scales that have been demonstrated, the development of craniovertebral approaches in the treatment of their instability, and the favorable reaction to these kinds of methods. 1. Figure 2 shows an illustration of research on craniovertebral instability, and Figure 3 shows the placement of the transperdicular screw insertions in C2 and the lateral mass of C1. Figure 4 of a research in Excel compares the many unstable craniovertebral diseases. The cerebellar amygdalae and brainstem of a 3-year-old child are observed to be shifted downward, which may be an indication of Chiari type II associated with instability of the craniovertebral junction. B.

A remarkably lengthy syringomyelia exhibiting scoliosis stretched from cervical level 6 to the lower thoracic levels. C. A young child undergoing craniocervical decompression has both Chiari type 2 and cervical syringomyelia. D. At the level of C1–C2, a severe herniation of the cerebellar amygdala and related brainstem, which is symptomless, is caused by an expansion of the spinal canal and foramen magnum. [Twenty]. as seen in figure 5. Figure 6. Error standard against effect size radial diagram Our meta-analysis indicates instability of the craniovertebral junction.The Smart Iris imaging system in Taiwan conducted a retrospective comparative study involving 702 consecutive patients who underwent MRI examinations of their cervical spine or CVJ junctions. Among these, 129 had CVJ issues, 279 showed normal CVJ, and there were 294 controls ($p=0.009$). The normal CVJ group had significantly fewer male patients (15%) compared to the diseased CVJ (34%) and control (61%) groups. Conditions observed included atlantoaxial subluxation, basilar invagination, and odontoid fractures. [6].

One study evaluated 140 patients with posterior arches of the C1 vertebra measuring >4 mm. Treatment options involved fixing the lateral mass or screw-fixing the C1 pedicle. Both groups achieved fusion within a year post-surgery, with group A procedures taking less time and using less blood than group B ($p<0.05$). [13]. Another investigation focused on 81 cases of atlantoaxial instability treated with C1 and C2 screws. Despite some screw placement issues, the fixation technique was deemed effective for patients of all ages. [19]. In a Korean study of 32 CVJ lesions, various causes were identified, including rheumatoid arthritis, trauma-induced instability, tumors, and basilar invagination. Different surgical approaches were taken, such as posterior decompression with fusion, transarticular screw fixation, and anterior decompression with fusion. Most cases of cervical myelopathy showed clinical improvement, especially when early occipitocervical fusion was recommended. [21]. A study involving young Down syndrome patients (38 out of 1056) with CVJ instability reported various symptoms such as myelopathy, paralysis, gait abnormalities, discomfort, and torticollis. Surgical interventions included internal fixation using different graft structures, resulting in reduced external orthotic needs. However, there was a 3% mortality rate and a 36% morbidity rate. [22]. Additionally, a study on occipitocervical fusion and biomechanical stabilization in cases of craniocervical instability reported successful fusion in 16 cases over an average of 35 months, with few complications and improvements in Nurick scores. [23].

State the following. Relative studies are shown in Table 1 below.

Table 1.

Author	Year	Kind of study	Cases	Control	Pathology aso. CVJ	Localization	Approaches	Means measures pre-	Post operation reduction	Follow up	P-value
Chang et al. [6].	2021	Retrospective comparison study	129/702	294	Rheumatoid arthritis	C1–C2	Screw fixation	1.67 ± 0.51 cm^2	1.03 ± 0.39 cm^2.	1-2 Years	p=0.001
. Singh et al. [3].	2020	Retrospective, observational study (Pediatric population).	10	N/A	Larsen syndrome	C1–C2 instability	Screw fixation /Descm	4	2	86 months /2	$P < 0.05$
Goel et al. [8].	2005	Review article 20 years of experience	160	N/A	CVJ atlantoaxial instability, Syringomyelia Basilar invagination	C1-C2 Atypical vertebrae	Screw fixation /Descm	3.5	2	3 months	N/A
Dastagirzada et al. [7].	2023	Review	60	206	CVJ instability Chiari Malformation I	C1-C2	Screw fixation /Descm	0.72-0.76	2.7	6-12 months	N/A
Joaquim et al. [12].	2023	Systematic review	23 Studies	8/1233	Basilar invagination	C1-C2	Screw fixation /Descm	0.63	1.18	-	N/A
Yan et al. [13].	2016	RCT	140	N/A	Atlantoaxial instability C1	C1-C2	Lateral screw fixation	1.8	1.4	13 months	$p < 0.01$

Salunke et al. [15].	2021	Retrospectively analyzed	268	17	Chiari and vertebral artery injury	C1-C2	C1-C2 fixation	7.3	0.48	6-12	(p < 0.01
Lohkamp et al. [16].	2022	Systematic review	78	N/A	Craniocervical Instability in Ehlers-Danlos Syndrome	C1-C2	the clivo-axial angle (CXA	5	4	6 Months	N/A
Ottenhausen et al. [17].	2023	Narrative review	212	N/A	Chondroma	C0-C2	Fixation	2	4	-	-
Ramírez-Paesano et al. [18].	2023	Review	2 studies	N/A	Ehlers-Danlos Syndrome	C1-C2	occipitocervical fixation	4.3	2.6	N/A	N/A
Guvin et al. [19].	2018	Retrospective cohort analysis	43	N/A	Traumatic-Rheumatoid arthritis	C1-C2	C1-C2 fixation	1	0.3	N/A	N/A
Encarnación-S D. et al. [20].	2023	Retrospective study	100	N/A	Chiari type II	C1-C2	CVJ descomprensión	1.5	1.7	6-12 months	(p 0.01)

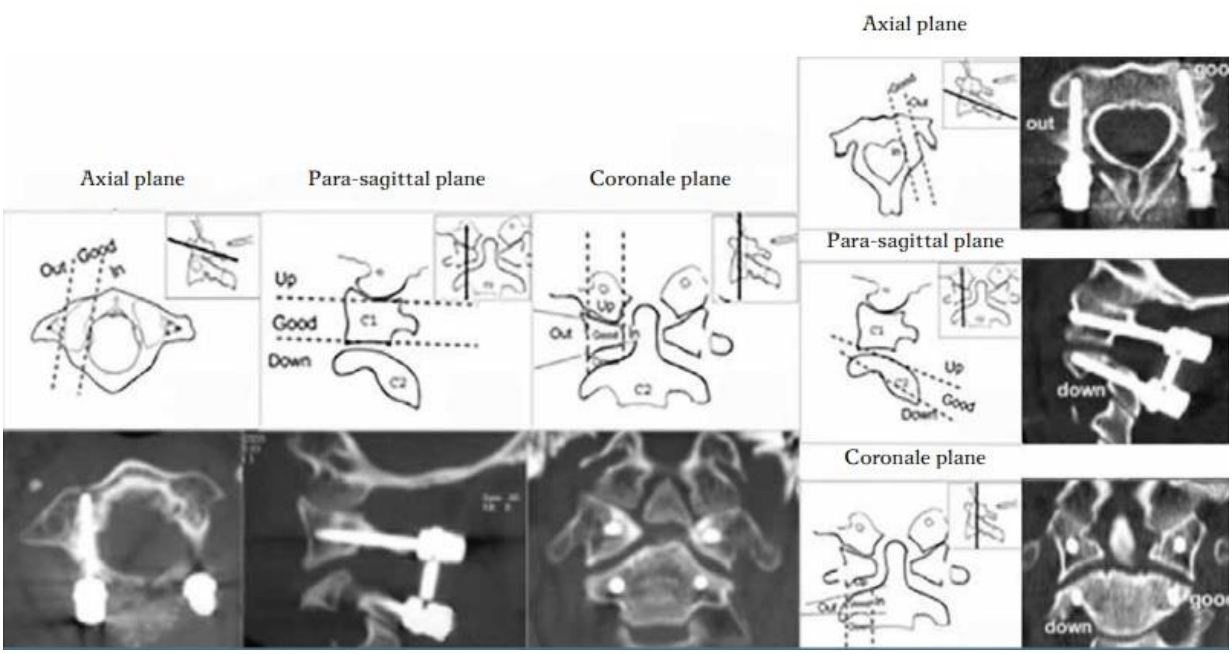

Figure 3. The location of the screw that was transpedicularly inserted into C2 and placed into the lateral masses of C1 [9].

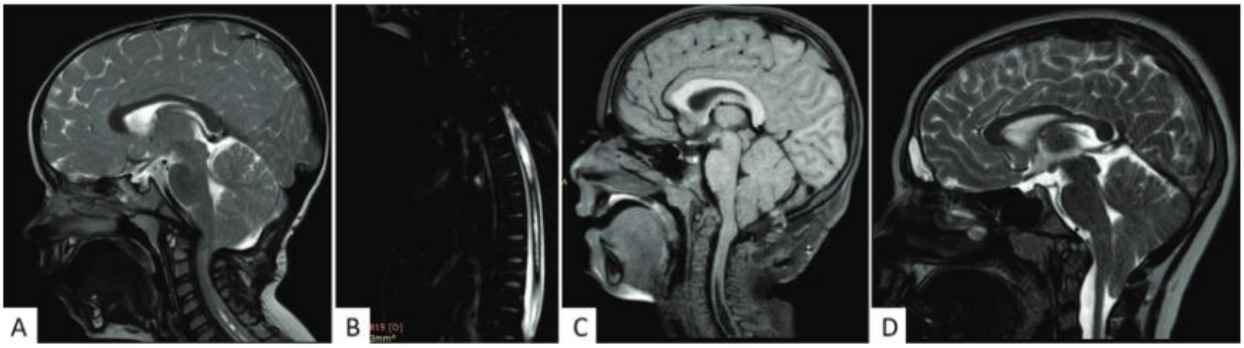

Figure 5. A 3-year-old child's brainstem and cerebellar amygdalae are seen to be displaced downhill, representing a potential example of Chiari type II connected to instability of the craniovertebral junction. B. An extraordinarily long syringomyelia with scoliosis extended from C6 to the lower thoracic levels. C. Cervical syringomyelia and Chiari type 2 are present in a young patient having craniocervical decompression. D. At the level of C1-C2, an extension of the spinal canal and foramen magnum results in a severe herniation of the cerebellar amygdala and associated brainstem, which lacks symptoms. [20].

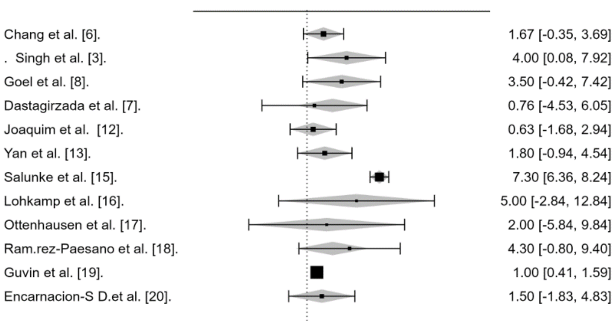

Figure 2. Shows the group effect size of consequence studies for craniocervical junction instability.

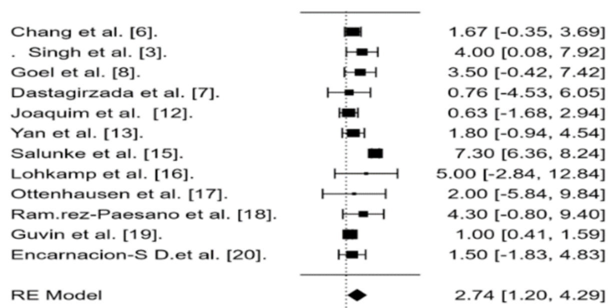

Figure 6. Standard error vs Effect size Radial plot Craniovertebral junction instability

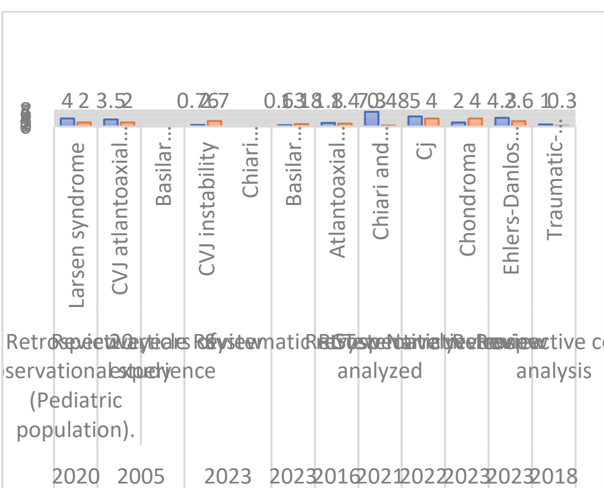

Figure 4. Craniovertebral junction studies and, approaches with pathologies associates

Discussion

These studies emphasize the importance of understanding the surgical anatomy and structures involved in CVJ instability. They highlight the necessity of preventing damage to the vertebrae, especially C1 and C2, and the significance of the vertebral artery's course and groove in surgical planning. Atlantoaxial alignment was evaluated based on facet alignment during neutral head positioning. [9]. Different types of atlantoaxial facet instabilities were identified, emphasizing the need for careful assessment and physical validation during surgery. Imaging examples showed potential instances of Chiari type II and craniovertebral instability-related complications such as syringomyelia and herniation of the cerebellar amygdala. Specific surgical techniques like atlantoaxial facetal fixation and fixation methods for basilar invagination were discussed in various studies, highlighting the challenges and strategies for stabilization in these conditions. [10]. According to Atul Goel, the preferred treatment for basilar invagination is typically atlantoaxial joint fixation or distraction. [11].

When evaluating occipital-atlantoaxial movements, crucial measurements include the basion-atlas interval, the horizontal section of the clivus, the anterior arch of the atlas, and the dens interval in the atlas, including its angle. Additionally, the relationship between the clivus angle and the atlas, or the clivus-atlas angle, should be considered. [16]. CT scans of the atlantoaxial region allow measurements in both sagittal and coronal slices. Lateral radiographs indicate flexion and extension, aiding in the identification and evaluation of craniovertebral junction (CVJ) stability using magnetic resonance imaging (MRI) during these movements. Instability may manifest in various symptoms such as atlantoaxial and atlanto-occipital ligament loss, neck pain, restricted neck muscles, and sensory or motor abnormalities. [17]. Patients with connective tissue diseases may experience spasticity, involuntary contractures of cervical and thoracic muscles, and craniocervical instability. Postoperative discomfort can be managed with painkillers, and drugs like tizanidine or baclofen may assist in pain management. It's important to note that individuals diagnosed with postural orthostatic tachycardia syndrome (POTS) should avoid haloperidol. [18].

Trauma is associated with connective tissue disorders and congenital abnormalities of the craniocervical junction, leading to craniocervical instability. [24]. Symptoms can be effectively managed through postural cues, mid-range stabilizing exercises, and manipulative axial traction techniques. [25]. Halo immobilization, followed by regular lateral cervical radiographs, may be employed initially. If the craniocervical alignment is unsatisfactory after a week, readjustment of the halo device under fluoroscopic supervision might be necessary. [26]. Basic radiography serves as a baseline for occipitoatlanto or atlantoaxial joint instability assessment. [27]. Craniometric studies indicate basilar invagination in healthy individuals and those with Chiari malformation, highlighting changes in the clivus canal angle, craniocervical kyphosis, and thickening of the lordotic cervical column. [28]. The CVJ contributes significantly to cervical spine function, enabling 50% axial rotation and 25% flexion and extension of the neck. [29]. Maintaining sagittal balance and realigning the cervical spine may impact postoperative clinical outcomes positively. [30]. Clinical arrest testing aids in identifying precise craniocervical ligament instability and hypermobility. [31]. Computed tomography angiography is crucial for surgical planning to detect vertebral artery injuries, minimize risks, and identify vascular anomalies early. [32]. Endoscopic endonasal techniques offer a highly adjustable, ventrally situated, and steep learning curve approach for performing safe decompression in the cervicomedullary region. [33].

Variability exists in basilar processes concerning shapes and sizes in relation to the atlas and its axis. [34]. Anterior techniques employing anterior odontoid screws contrast with posterior procedures using anchor rods, atlantoaxial fixation, or occipitocervical fixation with screws. [35].

A comprehensive understanding of anatomy and biomechanics is pivotal in evaluating diseased processes in the affected region. Patients without pre-existing instability or dislocation and in good health pre-surgery might qualify for transoral surgery. [36].

Limitations

Study Selection Limitations: Despite the use of specific inclusion and exclusion criteria, the selection of studies might be subject to some degree of subjectivity, and some relevant studies might have been unintentionally excluded.

Risk of Bias in Included Research: Taking in a certain number of studies for the review might carry a risk of bias, especially if these studies have methodological limitations such as selection, reporting, or confounding biases.

Limitations in Outcome Assessment: The review might not provide a comprehensive overview of all possible outcomes or long-term complications related to various craniovertebral junction stabilization techniques.

Limitations in Formulating Recommendations: Final recommendations or conclusions might be influenced by the limited availability of data or lack of general consensus within the medical and surgical field.

Conclusion

The management of atlantoaxial instability remains a subject of significant debate, especially concerning its implications in genetic connective tissue disorders. Craniovertebral junction instability presents a complex challenge that demands careful consideration. Key recommendations arising from these discussions include:

1. Prioritize key stabilization techniques, especially at the C1-C2 level, in cases of craniovertebral junction instability.
2. Consider the use of benzodiazepines for muscular relaxation and other opioid medications for effective postoperative management.
3. While a majority lean towards transpedicular pedicle screws, individual preferences vary considerably.
4. Techniques advocated by Harms and Atul Goel are favored due to their requirement of only 22 degrees.
5. Consider utilizing odontoid screws for fixation and exploring posterior procedures involving anchor rods, atlantoaxial fixation, or occipitocervical fixation using screws. Additionally, computed tomography angiography and Halo immobilization can offer valuable insights and aid in postoperative care.

In the management landscape for atlantoaxial instability is multifaceted, demanding a nuanced approach tailored to individual patient needs. Further research and consensus-building efforts are essential to refine treatment strategies and enhance patient outcomes in this challenging area of spinal pathology.

Declarations
The authors have no conflict of interest to declare.
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All authors have read and approved the final manuscript.
Ethical approval No applicable
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References

1. Dahdaleh N, El-Tecle N, Cloney M, Shlobin N. et al. An Approach to Managing Disorders. July 2023. https://doi.org/10.1016/j.wneu.2023.03.099
2. Ropper A. et al. From Anatomic to Genetic Understanding of Developmental Craniovertebral Junction. 2020 Dec 31. doi: 10.14245/ns.2040548.274
3. Singh S, Sardhara J, Raiyani V. et al. Craniovertebral junction instability in Larsen syndrome. 2020 Nov 26. doi: 10.4103/jcvjs.JCVJS_164_20
4. Goel A. et al. Cervical Fusion as a Protective Response to Craniovertebral Junction. December 2018. DOI: https://doi.org/10.14245/ns.1836236.118
5. Shi L, Xue D, Wang Y, Chou D. et al. Efficacy of a Lateral Mass Fusion Device. March 2022. https://doi.org/10.1016/j.wneu.2021.12.012
6. Chang CC, Wu C-L, Tu TH, Wu J-C. et al. Cranio-Vertebral Junction Triangular Area. 2021 Jan 6. doi: 10.3390/brainsci11010064
7. Dastagirzada Y, Kurland D, Hankinson T. et al. Craniovertebral Junction Instability in the S. Neurosurg Clin. 2023. https://doi.org/10.1016/j.nec.2022.09.006
8. Goel A, Sharma P, Dange N, Kulkarni AG. et al. Techniques in the treatment of craniovertebral instability. December 2005. DOI: 10.4103/0028-3886.22625
9. Goel A. et al. A Review of a New Clinical Entity of 'Central Atlantoaxial Instability. 2019 Jun 30. doi: 10.14245/ns.1938138.069
10. Goel A. et al Craniovertebral Junction Instability. 2015 July. doi : https://doi.org/10.4184/asj.2015.9.4.636
11. Goel A. et al. Instability and basilar invagination. Jun 2012. DOI: 10.4103/0974-8237.110115
12. Joaquim A, Evangelista A, Walter J, Botelho R. et al. Chamberlain's Line Violation in Basilar Invagination Patients. May 2023. https://doi.org/10.1016/j.wneu.2023.02.057
13. Yan L, He B, Liu T, Yang L. et al. A prospective, double-blind, randomized controlled trial of treatment of atlantoaxial instability with C1. 2016 Apr 14. doi: 10.1186/s12891-016-1017-8
14. Klepinowski T, Limanówka B, Sagan L.et al. Management of post-traumatic craniovertebral junction dislocation. August 2020. https://doi.org/10.1007/s10143-020-01366-4
15. Salunke P, Karthigeyan M, Singh A. et al. The enigma of acute worsening after a latent interval. August 2021. https://doi.org/10.1016/j.clineuro.2021.106741
16. Lohkamp L, Marathe N, Fehlings M. et al. Craniocervical Instability in Ehlers-Danlos Syndrome. 2022 Feb 23. doi: 10.1177/21925682211068520
17. Ottenhausen M, Greco E, 2, Bertolini G, Gerosa A. et al. Craniovertebral Junction Instability after Oncological Resection. April 2023. https://doi.org/10.3390/diagnostics13081502
18. Ramírez-Paesano C, Clarens C, Segovia A. et al. Perioperative opioid-minimization approach as a useful protocol. July 2023. https://doi.org/10.1186/s13023-023-02829-9
19. Gubin V, Burtsev V, Ryabykh O, Klimov S, Evsyukov V, Ivliev S. et al. Analysis of C1, C2 screw fixation for atlantoaxial instability 2018. DOI: http://dx.doi.org/10.14531/ss2018.3.6-12
20. Encarnacion-S D, Chmutin G, Chaurasia B, Bozkurt I. et al. Hundred Pediatric C. Treated for C. Type II. 2023 Jun 6. doi: 10.1055/s-0043-1768572
21. Song G, Cho K, Yoo D, Huh P, Lee S. et al. Surgical Treatment of Craniovertebral Junction Instability. 2010. doi: https://doi.org/10.3340/jkns.2010.48.1.37

22. Isaacs A, Narapareddy A, Nam A. et al. Surgical treatment of craniovertebral junction instability. 28 Apr. 2023. https://doi.org/10.3171/2023.3.PEDS22353
23. Choi S, Lee S, Park C, Kim W. et al. Surgical Outcomes and Complications after Occipito-Cervical Fusion. April 30, 2013. DOI: https://doi.org/10.3340/jkns.2013.53.4.223
24. Mao G, Kopparapu S, Jin Y, Davidar D. et al. Craniocervical instability in patients with Ehlers-Danlos síndrome. December 2022. https://doi.org/10.1016/j.spinee.2022.08.008
25. Mathers S, Schneider M, Timko M. et al. Occult Hypermobility of the Craniocervical Junction. June 2011. https://www.jospt.org/doi/10.2519/jospt.2011.3305
26. Ghatan S, Newell D, Grady S. et al. Severe posttraumatic craniocervical instability. August 2004. https://seattleneurosciences.com/wp-content/uploads/2017/10/Severe-posttraumatic-craniocervical-instability-in-the-very-young.pdf
27. Hendam H, Taha A, Youssef M. et al. Rod and Screw Fixation for Cranio-Cervical Instability. January 2020. DOI: 10.4236/ojmn.2020.101003
28. Botelho R, Diniz J. et al. Basilar Invagination: cranio-cervical kyphosis. March 2017. https://www.jneurology.com/articles/basilar-Invagination-cranio-cervical-kyphosis-rather-than-prolapse-from-the-upper-cervical-spine-neuromed-1-1110.php
29. Clark J, Abdullah K, Mroz T, Steinmetz M. et al. Biomechanics of the Craniovertebral. September 2011. DOI: 10.5772/21253
30. Huang H, Sheng M, Zeng G, Sun C, Li R. et al. Establish a new parameter "horizontal view-axial angle. January 2023. https://doi.org/10.3389/fsurg.2022.947462
31. Hutting N, Gwendolijne G. Scholten-Peeters, Vijverman V. et al. Diagnostic Accuracy of Upper Cervical Spine. December 2013. https://doi.org/10.2522/ptj.20130186
32. Tian Y, Xu N, Yan M, Passias P. et al. Atlantoaxial dislocation with congenital "sandwich fusión. December 2020. https://doi.org/10.1186/s12891-020-03852-8
33. Halderman A, Barnett S. et al. Endoscopic endonasal approach to the craniovertebral j. 2022 Mar. doi: 10.1002/wjo2.8
34. Saccheri P, Travan L. et al. The craniovertebral junction, between osseous variants and abnormalities. 2022 Mar. doi: 10.1007/s12565-021-00642-7.
35. Takayasu M, Aoyama M, Joko M, Takeuchi M. et al. Surgical Intervention for Instability of the Craniovertebral J. 2016 Aug. doi: 10.2176/nmc.ra.2015-0342.
36. Lopez A, Scheer J, Leibl K, Smith Z. et al. Anatomy and biomechanics of the craniovertebral. 2015 Apr. doi: 10.3171/2015.1.FOCUS14807.

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www.journals.lapub.co.uk/index.php/roneurosurgery

Clinical outcomes of tubular microdiscectomy with 18mm diameter tubular retractor for lumbar disc herniation. A prospective study over a 2-year period

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ABSTRACT

Introduction: Tubular microdiscectomy is one of the minimally invasive techniques for spine surgery. This prospective study aims to evaluate the clinical outcomes of using a tubular microdiscectomy with an 18 mm dilator for the treatment of lumbar disc herniation over a 2-year period.

Methods: A prospective observational study of 57 patients who had undergone a first-time, single-level lumbar discectomy presented with single-sided radiculopathy with or without backache was done. Perioperative and postoperative results were assessed by documenting operative time, estimated blood loss, length of stay, rate of wound infection, neurological deficits in post-op period, rate of cerebrospinal fluid leak and resumption of work. Pain assessment was done with VAS score at admission, at discharge and in follow up.

Results: A total of 57 patients were included in the study. The average duration of surgery was 64 minutes. The average duration from surgery to discharge was 35.5 hours. The average time for complete resolution of radicular symptoms was 8.5 days. The median time for return to work was 20 days. Two patients experienced cerebrospinal fluid (CSF) leak due to inadvertent durotomy. The mean Visual Analog Scale (VAS) value at admission was 9.5087, which significantly reduced to 1.49 at discharge.

Follow-up assessments at 15 days, 1 month, 3 months and 1 year revealed sustained improvements in clinical outcomes.

Conclusion: Tubular microdiscectomy with an 18 mm dilator demonstrates favourable clinical outcomes for patients having single-level lumbar disc prolapse intervertebral disc causing radiculopathy and low backache, less blood loss intraoperatively, small scar, lesser hospital stay and early return to work.

Keywords
lumbar disc herniation (LDH),
sciatica,
microdiscectomy,
tubular

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INTRODUCTION

Sciatica is a prevalent condition affecting millions of individuals globally, commonly attributed to lumbar disc herniation (LDH) [1]. LDH, characterized by degeneration and swelling of the lumbar intervertebral disc's nucleus pulposus, ranks among the most prevalent musculoskeletal disorders. The surgical treatment of LDH was first documented by Mixter and Barr in 1934 [2]. Later advancements in the field, notably by Caspar [3] and Yaşargil [4] in 1977, introduced microsurgical techniques to lumbar disc surgery, pioneering the concept of microdiscectomy. This procedure, heralded as the gold standard for open discectomy, boasts superior visibility, reduced invasiveness, and decreased perioperative morbidity [7]. Despite its advantages, traditional microdiscectomy necessitates midline ligament incision and the separation of paraspinous muscle tendons from the spinous process, potentially resulting in postoperative back pain and spinal instability.

In pursuit of minimizing incision size and sparing paraspinal structures, micro endoscopic discectomy emerged as a refinement of microdiscectomy [5]. Employing a small-diameter tubular retractor (18 mm) and sequential dilators, this technique creates a surgical pathway to the lumbar spine between fascicles of the lumbar paraspinous muscles, circumventing the conventional detachment of multifidus muscles from the spine observed in open discectomy and microdiscectomy [6]. An articulated, repositionable arm secures the tubular retractor to the operating table, freeing the surgeon's hands. The tube's diameter accommodates the simultaneous use of 2 or 3 microsurgical instruments, such as a high-speed drill, suction device, and nerve root retractor. Initially coupled with an endoscope for visualization, the tubular retractor later integrated the operating microscope into its setup.

Tubular microdiscectomy (TD) adopts a muscle-space approach, diverging from the traditional subperiosteal muscle dissection, thereby minimizing tissue damage and expediting recovery.

MATERIALS AND METHODS

Study Design: This prospective observational study was conducted at a tertiary care center and teaching institute in Bareilly city, India, spanning from June 1, 2021, to May 31, 2023.

Participants: A total of 57 consecutive patients, aged 18–70 years, with symptomatic lumbar intervertebral disc prolapse unresponsive to conservative management, neurological deficits, or refusal of conservative treatment, were included. The procedures were performed by a single experienced surgeon proficient in tubular microdiscectomy techniques.

SURGICAL TECHNIQUE

Positioning: prone on bolsters with pelvis and thighs flexed

Steps:

- The midline is identified and marked on the patient's back at site of expected pathology (fig. A)
- A 18 G needle is inserted 1.5 cm ipsilateral to the side of the disc herniation (Fig. A).
- The needle is adjusted until lateral fluoroscopy confirms a trajectory that is coaxial (dashed line) with the disc of interest (Fig. B).
- An 18-20 mm vertical incision is made, with the needle mark serving as the midpoint. This incision is limited to the subcutaneous tissue only. ((Fig. C)
- Following the needle trajectory the first tubular dilator is inserted (Fig. D).
- Placement of Tubular Retractor: A small-diameter tubular retractor (18 mm) is positioned over sequential dilators, establishing a surgical pathway to the lumbar spine between the fascicles of the lumbar paraspinous muscles. This approach avoids the conventional detachment of multifidus muscles from the spine.
- The tubular retractor is secured in place by an articulated, repositionable arm affixed to the operating table.
- The diameter of the tube allows for the simultaneous use of 2 or 3 microsurgical instruments, such as a high-speed drill, a suction device, and a nerve root retractor. The operating microscope is then positioned. (Fig. E)
- Residual soft tissue is excised, and haemostasis is achieved using bipolar and/or monopolar cautery.
- The inferior edge of the lamina is identified, and a hemilaminotomy is performed using the high-speed drill. The lamina is excised until the superior insertion of the ligamentum flavum.
- A nerve root retractor is introduced to safeguard the traversing nerve root, retracting it medially.
- Epidural veins are coagulated using bipolar cautery to minimize bleeding.
- Extruded/compressing/offending disc fragments are extracted using a disc forceps.
- Nerve and foramen is assessed using nerve hook, haemostasis achieved and closure in layers.

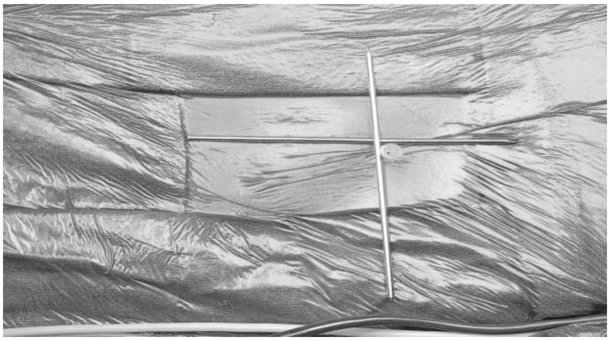


Figure A.

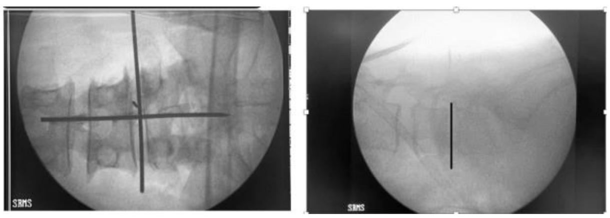


Figure B.

Figure C.

Figure D.

Figure E.

Inclusion Criteria:

- Single-level lumbar disc prolapse /extrusion/ sequestration with unilateral radiculopathy.
- Age between 18 and 70 years.
- Lack of symptom relief after a minimum of 6 weeks of conservative management.

Exclusion Criteria:

- Cauda equina syndrome.
- Spinal instability/spondylolisthesis.
- Failed back surgery syndrome.
- Degenerative lumbar canal stenosis.
- Multi-level disc prolapse.
- Pregnancy.

Data collection

Patient data were recorded in a case record sheet. Preoperative assessments, including comprehensive clinical examinations and radiographic investigations, were conducted, documenting baseline Visual Analogue Scale (VAS) scores for leg and back pain. Intraoperative data, such as incision length, surgical duration, blood loss, specific intraoperative findings, complications, and other relevant details, were also recorded.

Outcome measures

Postoperative complications were categorized into wound complications, surgical site infections, neurological deficits, spinal instability, symptom exacerbation, cerebrospinal fluid (CSF) leakage, discitis, and others. Postoperative pain was evaluated using the VAS scale on the first postoperative day after mobilizing the patient for 50 meters. Diclofenac sodium 75 mg intravenous was administered during surgery and postoperatively at 10:00 p.m., with oral administration advised upon discharge if needed.

Follow-up

Patients were followed up for a minimum of 6 months and a maximum of two years postoperatively, with scheduled visits at 15 days, 1 month, 3 months, 6 months, and 12 months. During follow-up, VAS scores for radicular symptoms were recorded, and any signs of infection or changes in symptoms were documented.

Results

A total of 57 patients were included in study from 1st June 2021 to 31st may 2023 with symptomatic single level lumbar intervertebral disc prolapse.

Of these 57 patients 41(71.9%) were male, 16 (28.07%) were female. Out of 57, 34 cases (59.64%) had involvement at L4-L5 and 23 cases (40.35%) at L5-S1 level. In the study 31(54.38%) patients presented with left-sided radiculopathy and 26 (45.61%) patients with right-sided radiculopathy.

Duration of symptoms ranged from 15 days to 48 months. The average duration of LBA symptoms was 8.8 months and lower limb radiculopathy was 4 months.

The operative time ranged from 32 minutes to 120 minutes and the median duration of surgery was 64 minutes.

The intraoperative blood loss was always less than 10 ml except in two patients in which the blood loss was 50 ml with mean blood loss of 9.92 ml.

Most patients 35 out of 57(61.40%) were discharged within 24 hours of surgery, 2 (3%) after 36 hours, 14(25%) after 48 hours. One patient (2%) was discharged on 5th postoperative day and 5(9%) patients took 72 hours to be discharged. The average duration from surgery to discharge was 35.5 hours, with a median of 24 hours.

Post operative pain was assessed on VAS SCALE on 1st postoperative day by mobilising the patient for 50 metres.

Three patients reported with almost complete resolution of pain after 24 hours of surgery. Eight patients in 2 days after surgery, 3 patients in 3 days, 2 in 4 days, 3 in 5 days, 1 in 10 days, 1 in 12 days, 21 Patients (36.84%) responded with almost complete resolution of radicular pain in 7 days, 12 patients (21.05%) in 15 days, 1 in 28 days and 2 patients in 30 days.

The median hours of complete resolution of radicular symptoms were 7 days, with an average of 8.5 days.

All the patients returned to work within 30 days (Fig-3). Most of the patients 27(52%) of 57 returned to work in 16-30 days. 22(42%) patients were able to resume jobs in 15 days. Six percent viz. 3 patients took merely 7 days to return to work. The median time to return to work was 20 days.

Two patients (3.5%) experienced cerebrospinal fluid (CSF) leak due to inadvertent durotomy (Table-1). One patient among this required conversion to open for dural tear repair. Other patient was managed with fat patch only.

We did not observe any discitis, post op wound infection, recurrent herniation of disc at same level in one year follow up

The mean visual analogue scale (VAS) score at admission was 9.5087, which significantly decreased to 1.49 at discharge (Fig.1 and Fig.2).

Patients were followed up at 15 days, 1 month, and 3,6 and 12 months postoperatively and assessed for pain, wound infection and other complications. None of the patients developed any complication in follow up. Sixty five percent patients had VAS SCORE of 1 at 12 months as depicted in Fig.4

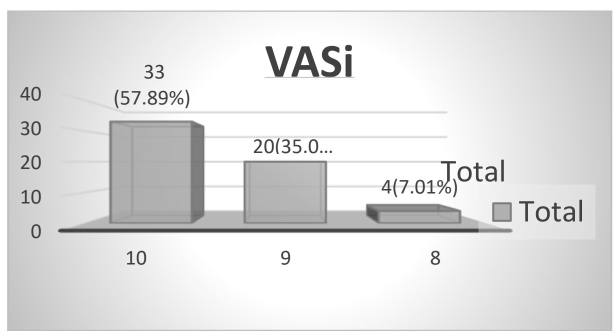
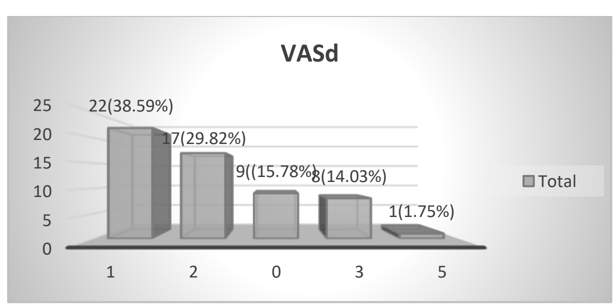


Figure 1. VAS score at admission.

Figure 2. VAS score at discharge.



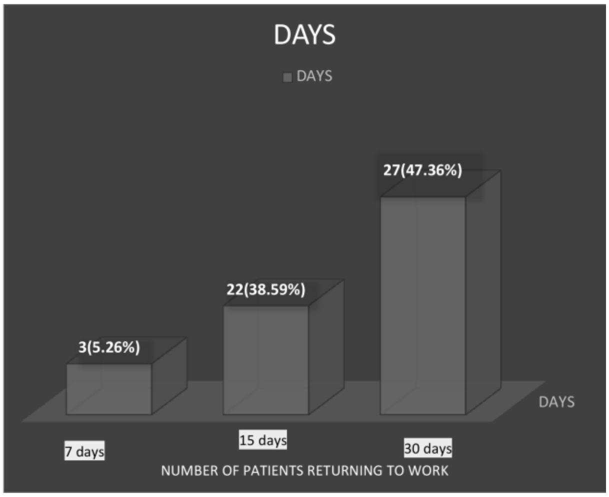


Figure 3. Patients returning to work (Median time- 20 days).

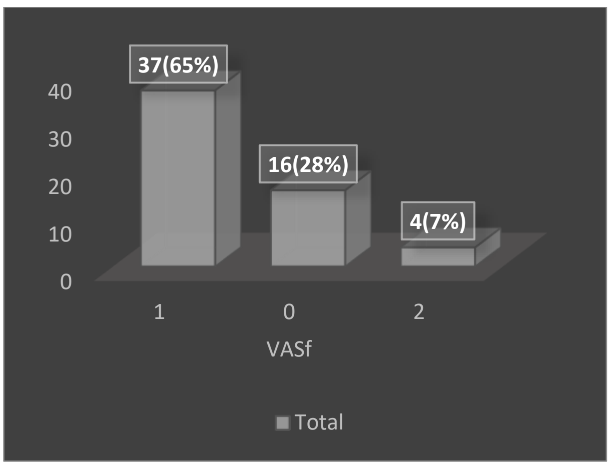


Figure 4. VAS score at 12 month follow up.

Table 1. VAS

Parameters	Number of patients
Total patients in study Male Female	57 41(71.9%) 16 (28.07%)
Site of PIVD: L4L5 L5S1	 34(59.64%) 23(40.35%)
Average duration of symptoms: LBA Radiculopathy	 8.18 months 4 months
OPERATIVE PARAMETERS Intraoperative time(skin incision to skin closure) Average blood loss Inadvertent durotomy	 64 minutes 9.9 ml 2(3.5%)
Average stay (surgery to discharge - hours)	35.5
Average duration of complete resolution of symptoms(days)	8.5
Average VAS SCORE At admission At discharge	 9.5 1.49
Patients returning to work in 7 days 15 days 30 days	 3(5.26%) 22(38.59%) 27(47.36%)
Discitis	0
Wound infection	0
Recurrent disc herniation at same level in 1 year	0
Segmental instability	0
Converted to open	1
Any other	none

Statistical analysis

Study data were entered in MS Excel and then analysed using SPSS Version 27.0. Data are presented as means and percentages depending on the type of variable.

Discussion

Lumbar disc prolapse causing low back ache and lower limb radiculopathy is the most common spinal pathology affecting all age groups. Patients not responding to conservative management for a minimum of 4-6 weeks, or routine activities of daily living affected are treated surgically [8]. Discectomy performed either through an open approach or by minimally invasive techniques remains the gold standard management. Open microdiscectomy surgery requires muscles dissection and retraction which might induce iatrogenic morbidity of the soft tissues in spite of providing greater direct visualization of anatomic structures and obtaining the optimal angle for disc removal; however, discectomy with tubular retractors which minimizes the tissue injury and ensures that deeper tissues are less exposed to potential pathologic organisms due to restricted surgical field. Minimally invasive tubular lumbar microdiscectomy is a refinement of the standard open microscopic lumbar discectomy technique. Advantages of this minimal invasive technique includes less perioperative pain, early ambulation, shorter hospital stay and early return to work with smaller incision [9].

In study of Art et.al [9]mean day of mobilisation of patients was done on 2nd day post operatively, while in this study most of the patients were mobilised with in 36 hours.

In study of Nayak et.al [10] The mean surgical incision length in the tubular microdiscectomy group was 2.45±0.41 cm and 22 mm diameter tubular retractor, while in my study we used 1.8cm-2 cm incision only and 18 mm diameter tubular retractor. Ryang et.al[11] reported average blood loss in minimal invasive group 26.2 ml and 63.8 ml in open microdiscectomy group, while in our study average blood loss was 9.92 ml only. Average operative time was 47 mins in study of Art et.al [9]and 82 minutes in study of Ryang et.al[11] , but it was 64 minutes in our study which is comparable to former. In study of Art et.al [9]mean duration of hospital stay was 3.3 days while in our study mean hospital stay was 35.5 hours. In the trial by Brock et al.[12] postoperative analgesic usage was significantly lower in the tubular group, an observation that is supported by our study too.

We had only two patients with intraoperative dural tear causing CSF leak mean of 3.5% which lesser than reported by Sonawane et.al[14] in which mean was 6.5%. and 19.5% in study of Nayak et.al[10]

We did not observe any discitis, post op wound infection, recurrent herniation of disc at same level in one year follow up which were observed in other studies [9,10,13,14].

The study of Clark et.al[13] for lumbar discectomy says that Level I evidence supports equivalently good outcomes for tubular microdiscectomy compared with standard microdiscectomy, my study strengthens the same.

Conclusions

Tubular microdiscectomy gives good outcome in single level lumbar prolapse intervertebral disc causing radiculopathy and low backache, less blood loss intraoperatively, small scar, lesser hospital stay and early return to work. Moreover the other complications like discitis, post-operative wound infection and recurrent herniation are nil as per my study.

Owing to lesser hospital stay and shorter operative time it may be cost effective too.

References

1. Konstantinou K, Dunn KM. Sciatica: review of epidemiological studies and prevalence estimates. Spine. 2008;33(22):2464-2472.
2. Mixter WJ, Barr JS. Rupture of intervertebral disc with involvement of the spinal canal. N Engl J Med. 1934;211:210–5.
3. Caspar W: A new surgical procedure for lumbar disc herniation causing less tissue damage through a microsurgical approach. Adv Neurosurg 4:74–80, 1977
4. Yasargil MG. Microsurgical operations of herniated lumbar disc. In: Wüllenweber R, Brock M, Hamer J, Klinger M, Spoerri O, editors. Adv Neurosurg. Advances in Neurosurgery. Berlin: Springer; 1977.
5. Foley KT, Smith MM: Microendoscopic discectomy. Tech Neurosurg 3:301–307, 1997
6. Perez-Cruet M.J., Foley K.T., Isaacs R.E., et al. Microendoscopic lumbar discectomy: technical note. Neurosurgery. 2002;51(5 Suppl):S129–S136.
7. Riesenburger RI, David CA. Lumbar microdiscectomy and microendoscopic discectomy. Minim Invasive Ther Allied Technol. 2006;15:267–70.
8. Jensen MP, Karoly P, O'Riordan EF, Bland F Jr, Burns RS. The subjective experience of acute pain. An assessment of the utility of 10 indices. Clin J Pain. 1989;5(2):153–9.
9. Arts MP, Brand R, van den Akker ME, Koes BW, Bartels RHMA, Peul WC, et al. Tubular diskectomy vs conventional microdiskectomy for sciatica: a randomized controlled trial. JAMA. 2009;302(2):149–58.
10. U KRN, Bhat SN, Ampar N, Kundangar RS. Microdiscectomy and Minimally Invasive Discectomy Using a Tubular Retractor System for Lumbar Disc Herniation: A Comparative Study. GMJ 2024;35:30-37.
11. Ryang YM, Oertel MF, Mayfrank L, Gilsbach JM, Rohde V: Standard open microdiscectomy versus minimal access trocar microdiscectomy: results of a prospective randomized study. Neurosurgery 62:174–182, 2008
12. Brock M, Kunkel P, Papavero L: Lumbar microdiscectomy: subperiosteal versus transmuscular approach and influence on the early postoperative analgesic consumption. Eur Spine J 17:518–522, 2008
13. Clark AJ, Safaee MM, Khan NR, Brown MT, Foley KT. Tubular microdiscectomy: techniques, complication avoidance, and review of the literature. Neurosurg Focus. 2017 Aug;43(2):E7. doi: 10.3171/2017.5.FOCUS17202. PMID: 28760036.
14. Sonawane, Dhiraj V; Chobing, Habung; Kolur, Shivaprasad S; Chandanwale, Ajay; Jawale, Sagar A; Ansari, Naved A F A; Pawar, Eknath. Conventional versus tubular microdiscectomy for lumbar disc herniation: A prospective randomized study. Indian Spine Journal 7(1):p 59-65, January-June 2024. | DOI: 10.4103/isj.isj_30_23

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Variation in optic recess angle with optic chiasm position. Imaging characteristics of 140 cases

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ABSTRACT

Background: The position of the optic chiasm relative to surrounding structures is critical in planning surgical interventions for suprasellar lesions. This study explored the relationship between the angle of the optic chiasm and lamina terminalis (OC-LT) and the position of the optic chiasm.

Methods: The study comprised 140 individuals who underwent midsagittal and axial MRI-T2 scans. The position of the optic chiasm was classified into three categories: sellar, prefixed, and postfixed. The OC-LT angle was measured in the midsagittal section.

Results: The angle between OC-LT varied from 30 to 66 degrees with a mean of 46.6 degrees. The sellar position of the optic chiasm was predominant (85.2%), with smaller angles (30-39 degrees) significantly associated with a sellar chiasm location. Most cases with postfixed optic chiasm fell within a 40-49 degree angle range.

Conclusions: The OC-LT angle variability can be linked to the position of the optic chiasm. Predominantly, smaller angles correlated with a sellar position of the optic chiasm, while larger angles were associated with a postfixed optic chiasm. This information is crucial for surgical planning in the suprasellar region.

INTRODUCTION

The chiasmatic recess (CR) is a distinctive structure in the anterior region of the third ventricle. It is formed by the lamina terminalis (LT) as its anterior wall and the upper surface of the optic chiasm as its floor. Together, these adjacent surfaces create a margin that resembles an inverted U shape (11). The anatomical relationship between the lamina terminalis, hypothalamus, chiasm, sella turcica, pituitary gland, diaphragma sellae, basal cistern, and other adjacent structures is of great significance. The lamina terminalis forms the anterior wall of the third ventricle, enclosing the hypothalamus above the chiasm. Below the chiasm, the sella turcica, pituitary gland, and diaphragma sellae are situated (8). This intricate network of structures, including the lamina terminalis, hypothalamus, chiasm, sella turcica, pituitary gland, diaphragma sellae, basal cistern, and other adjacent elements, highlights the profound anatomical interplay within this region.

The optic chiasm, formed by the convergence of the optic nerves, plays a crucial role in visual perception by transmitting visual information from one side of both eyes to the occipital cortex (8). The location of the optic chiasm classified into three positions. The terms "prefixed" and "postfixed" were introduced in 1924 to describe these positions, and a subsequent study by Bergland et al. in 1968 confirmed the existence of these chiasm types. Prefixed chiasms were found above the tuberculum sellae, while postfixed chiasms were observed above the dorsum sellae (1). This classification provides valuable insights into the anatomical variations of the optic chiasm.

Suprasellar lesions, including craniopharyngiomas, gliomas, pituitary adenomas, germinomas, hamartomas, and meningiomas, have a profound impact on the optic chiasm. In addition, vascular disorders like aneurysms and arteriovenous malformations, as well as granulomatous diseases affecting the infundibulum and hypothalamus, can further complicate the situation (9). Understanding the intricate anatomy of the optic chiasm and its surrounding region is essential for effective surgical planning in these cases, enabling the development of optimal strategies and reducing the risk of complications.

The aim of this article is to investigate the anatomical variations of the angle between the optic chiasm and lamina terminalis (OC-LT), with a focus on its relationship with different positions of the optic chiasm.

Methods

The study was conducted at the Department of Neurosurgery in Baghdad, Iraq, spanning from 2021 to 2023. A diverse group of participants underwent midsagittal magnetic resonance imaging (MRI)-T2 scans for various medical reasons. Participants without abnormalities in the ventricular system were included, while scans lacking essential parameters were excluded. Measurements of the optic chiasm position and the angle between the optic chiasm and the lamina terminalis were taken using Perfect Screen Ruler 3.0 software and the accompanying drawing scale. The measurements were conducted by an experienced researcher to ensure accuracy and minimize bias. The focus was on examining the chiasmatic recess, which is a small extension located below the anterior portion of the third ventricle. The CR is characterized by the lamina terminalis serving as its anterior wall, while the upper surface of the optic chiasm forms its floor as showen in (Figure 1). Within this anatomical region, the focus was on measuring the angle between the optic chiasm and the lamina terminalis. This angle was measured and recorded according to the methodology illustrated in (Figure 2). Statistical analysis was performed using SPSS version 25 to determine frequencies and percentages of categorical variables.

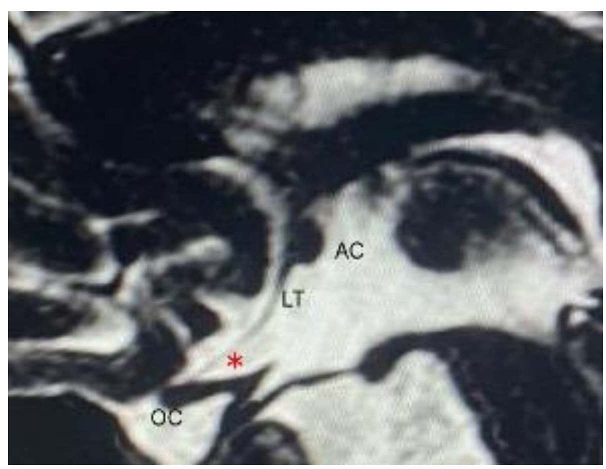

Figure 1. Mid sagittal MRI T2 shows chiasmatic recess (red star), the optic chiasm (OC), lamina terminalis (LT) and anterior commissure (AC).

Results

A group of 146 individuals participated in the study and underwent midsagittal and axial MRI-T2 scans for different reasons. The average age of the participants was 55 years, ranging from 10 to 87 years. Among the participants, 54.4% were female and 43.6% were male. Following a careful evaluation based on predetermined criteria for inclusion and exclusion, 140 subjects were selected for comprehensive analysis out of the initial sample. Out of the 140 patients included in the study, one patient (2.9%) had a prefixed chiasm, while ten patients (7.1%) had a postfixed chiasm (Table 1).

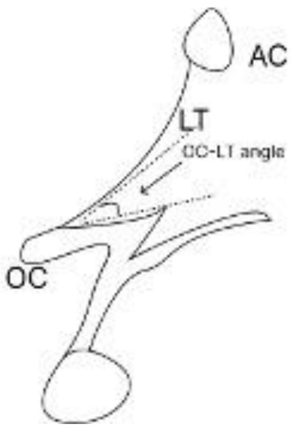

Figure 2. Schematic illustration of the OC-LT angle, the optic chiasm (OC), lamina terminalis (LT), and anterior commissure (AC).

Table 1. Optic chiasm positions

Optic chiasm position		No. (%)
	Sellar	129 (90.0%)
	Postfixed	10 (7.1%)
	Prefixed	1 (2.9%)
	Total	**140 (100.0%)**

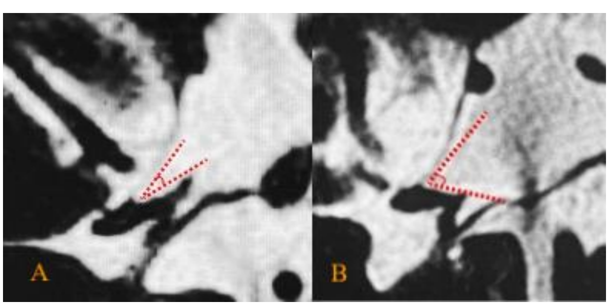

Figure 3. Mid sagittal MRI T2 shows variation in OC-LT angle ranging from 30 degrees in A to 60 degrees in B.

Table 2. OC-LT Angle ranges

	Angle range	No. (%)
OC-LT angle	30-39	*37 (26.6%)*
	40-49	*54 (38.5%)*
	50-59	*29 (20.7%)*
	≥60	*20 (14.2%)*
	Total	**140 (100%)**
Abbreviations: OC; optic chiasm, LT; lamina terminalis.		

Table 3. OC-LT angle ranges in different optic chiasm positions

Variables		**OC-LT Angle range**			
		30-39	**40-49**	**50-59**	**≥60**
		No. (%)	No. (%)	No. (%)	No. (%)
OC positions	**Sellar**	35 (94.5%)	46 (85.2%)	28 (96.5%)	20 (100.0%)
	Postfixed	2 (5.5%)	8 (14.8%)	0 (0.0%)	0 (0.0%)
	Prefixed	0 (0.0%)	0 (0.0%)	1 (3.5%)	0 (0.0%)
	Total	***37* (100.0%)**	***54* (100.0%)**	***29* (100.0%)**	**20 (100.0%)**
Abbreviations: OC; optic chiasm, LT; lamina terminalis.					

The optic chiasm and LT angle, was measured on the midsagittal sections ranged from 30 to 66 degrees (Figure 3), with a mean angle of (46.6) degree and standard deviation (SD) equal to 9.9. The largest proportion of cases (38.5%) exhibited angles between 40 and 49 degrees, followed by cases with angles between 30 and 39 degrees, accounting for 26.6% of the total (Table 2).

When considering optic chiasm positions, the analysis revealed that the sellar position was predominant, observed in 85.2% of cases. Conversely, a smaller percentage (14.8%) exhibited a postfixed optic chiasm. In cases with angles between 30 and 39 degrees, the sellar location was highly prevalent, accounting for 94.5% of cases, while a minority (5.5%) had a postfixed optic chiasm. Only one case was identified with a prefixed optic chiasm and an angle falling within the 50-59-degree range, as shown in Table 3.

Discussion

The chiasmatic recess is a distinctive structure located in the front part of the third ventricle. It is created by the anterior wall, known as the lamina terminalis (LT), and the upper surface of the optic chiasm, which forms its floor. These adjacent surfaces come together to form a border that resembles an inverted U shape (11). The anatomical relationship between the lamina terminalis, hypothalamus, chiasm, sella turcica, pituitary gland, diaphragma sellae, basal cistern, and other nearby structures holds significant importance. The lamina terminalis acts as the front wall of the third ventricle, surrounding the hypothalamus above the chiasm. Below the chiasm, you can find the sella turcica, pituitary gland, and diaphragma sellae situated (8).

Lesions located in the suprasellar and anterior ventricular regions can have an impact on the optic chiasm. Among these lesions, craniopharyngiomas are the most commonly encountered, characterized by their benign nature and slow growth originating from remnants of squamous epithelium in Rathke's pouch. Although primarily found in children and young adults, craniopharyngiomas can also occur in adults and manifest with a range of symptoms, from growth failure to visual disturbances, depending on their size and precise location (10). Another type of lesion in this region includes chiasmatic and hypothalamic gliomas. Chiasmatic gliomas, associated with neurofibromatosis type I, are gradual-growing tumors that infiltrate the visual pathways. On the other hand, hypothalamic gliomas, predominantly observed in adults, tend to exhibit more aggressive behavior (5). Pituitary adenomas can cause hormone hypersecretion microadenomas or visual impairment and headaches in non-secreting adenomas (12). Other rare lesions include choristoma, epidermoid and dermoid tumors, Rathke's cleft cyst, suprasellar arachnoid cysts, and metastatic tumors (2,3,7).

Since the 18th century, surgeons have been exploring different approaches to the base of the skull for the management of suprasellar lesions. The evolution of trans-sphenoidal approaches, including endoscopic techniques, has revolutionized contemporary skull base surgery (6). These techniques offer advantages such as minimally invasive access, excellent visualization of the operative field, and the ability to manage various types of lesions. However, there are potential complications, including cerebrospinal fluid (CSF) leaks, bleeding, damage to critical structures like the internal carotid artery and optic nerves, meningitis, and ophthalmic complications. Preoperative radiological studies, careful planning, and a multidisciplinary approach are crucial for successful outcomes and minimizing complications in these complex surgeries (4). OC-LT angle can be utilized as assessment parameter to determine the feasibility of employing different approaches for the surgical resection of suprasellar lesions.

In our study OC-LT angle ranged from 30 to 66 degrees, with a mean angle of (46.6) degree and SD of 9.9. This mean angle is slightly higher than the mean reported by Tsutsumi S et al. (ranging from 16.5 to 62 degrees, with a mean of 34 degrees) (11). Among the cases analyzed, the largest proportion (38.5%) exhibited angles between 40 and 49 degrees, indicating that this range is the most prevalent configuration. Additionally, 26.6% of the cases fell within the range of 30 to 39 degrees, while angles ranging from 50 to 59 degrees accounted for 20.7% of the sample. Furthermore, cases with angles exceeding 60 degrees represented 14.2% of the total cases. These observations highlight the variability in the angulation of the OC-LT angle in the chiasmatic recess.

The distribution of optic chiasm positions within specific angle ranges is also noteworthy. In cases with angles between 40 and 49 degrees, the majority (85.2%) displayed an optic chiasm located at the sellar position, while a smaller proportion (14.8%) exhibited a postfixed optic chiasm. This suggests association between the angle of the optic chiasm and its anatomical position within this angle range. Similarly, in the subset of cases with angles between 30 and 39 degrees, the vast majority (94.5%) exhibited an optic chiasm positioned at the sellar location, with only a minority (5.5%) displaying a postfixed optic chiasm. These findings indicate a predominant sellar position of the optic chiasm in cases with smaller angles.

The cases with a postfixed optic chiasm were distributed across two angle categories: 30-39 degrees and 40-49 degrees. As expected, the distribution of angles in the postfixed position encompassed a wider range. However, it is important to note that the absence of cases in the wider-angle range may be attributed to the relatively small sample size utilized in this study. a single case featuring a prefixed optic chiasm with an angle ranging from 50 to 59 degrees. Surprisingly, this particular case exhibited a wider-angle range compared to the postfixed cases, which is unexpected. Several factors could contribute to this unusual observation, such as the small sample size or the presence of unrecognized associated conditions that may have influenced the optic chiasm's positioning and angulation. Further investigation is warranted to explore the underlying reasons for this atypical occurrence. These results contribute to our understanding of the anatomical variations in optic chiasm orientation and position relative to the measured angle.

The chiasmatic recess is a distinct structure in the front part of the third ventricle, formed by the lamina terminalis and the upper surface of the optic chiasm. It has an inverted U shape and is important for the anatomical relationship with nearby structures. Lesions in the suprasellar and anterior ventricular regions, such as craniopharyngiomas, chiasmatic and hypothalamic gliomas, and pituitary adenomas, can affect the optic chiasm, resulting in various symptoms including visual disturbances, hormone hypersecretion, and headaches. Other rare lesions in this area include choristoma, epidermoid and dermoid tumors, Rathke's cleft cyst, suprasellar arachnoid cysts, and metastatic tumors. In our study, the angle between the optic chiasm and lamina terminalis ranged from 30 to 66 degrees, with a mean angle of 46.6 degrees.

This mean angle is slightly higher than the average angle reported by Tsutsumi S et al., which ranged from 16.5 to 62 degrees, with a mean of 34 degrees. Among cases with angles between 40 and 49 degrees, the majority (85.2%) had an optic chiasm located at the sellar position, while a smaller proportion (14.8%) exhibited a postfixed optic chiasm. The cases with a postfixed optic chiasm were distributed across two angle categories: 30-39 degrees and 40-49 degrees. Additionally, there was a single case with a prefixed optic chiasm and an angle ranging from 50 to 59 degrees. These findings enhance our understanding of the variations in the angle between the optic chiasm and lamina terminalis and positions of optic chiasm.

Conclusion

The angle in the chiasmatic recess is variable and the variability can be related to the position of optic chiasm. Cases with smaller angles (30-39 degrees) showed a predominant sellar position of the optic chiasm. Most postfixed optic chiasm cases displayed in a range of angles (40-49 degrees).

References

1. Bergland RM, Ray BS, Torack RM. Anatomical variations in the pituitary gland and adjacent structures in 225 human autopsy cases. J Neurosurg. 1968 Feb and 28(2):93-9.
2. Boyko OB, Curnes JT, Oakes WJ, Burger PC. Hamartomas of the tuber cinereum: CT, MR, and pathologic findings. American Journal of Neuroradiology. 1991 Mar 1 and 12(2):309-14.
3. Cockerham KP, Kennerdell JS, Maroon JC, Bejjani GK. Tumors of the meninges and related tissues: meningiomas and sarcomas. Walsh and Hoyt's Clinical Neuro-Ophthalmology. Baltimore, Williams & Wilkins. 1998:2017-82.
4. Emanuelli E, Zanotti C, Munari S, Baldovin M, Schiavo G, Denaro L. Sellar and parasellar lesions: multidisciplinary management. Acta Otorhinolaryngologica Italica. 2021 Apr;41(2 Suppl 1):S30.
5. Gailloud P, Ruíz DS, Muster M, Murphy KJ, Fasel JH, Rüfenacht DA. Angiographic anatomy of the laterocavernous sinus. American journal of neuroradiology. 2000 Nov 1 and 21(10):1923-9.
6. HIRSCH O. ENdonasal method of removal of hypophyseal tumorswith report of two successful cases. Journal of the American Medical Association. 1910 Aug 27;55(9):772-4.
7. Iglesias A, Arias M, Brasa J, Paramo C, Conde C, Fernandez R. MR imaging findings in granular cell tumor of the neurohypophysis: a difficult preoperative diagnosis. European Radiology. 2000 Nov and 10(12):1871-3.
8. Kidd D. The optic chiasm. Handbook of Clinical Neurology. 2011 Jan and 102:185-203.
9. Mar, Ruscalleda J. Parasellar lesions. European Radiology. 2005 and 15:549-59.
10. Nov, Hayward R. The present and future management of childhood craniopharyngioma. Child's Nervous System. 1999 and 15:764-9.
11. Tsutsumi S, Ono H, Yasumoto Y. The chiasmatic recess of the third ventricle: delineation with magnetic resonance imaging. Surgical and Radiologic Anatomy. 2016 Oct and 38:881-6.
12. Wilson CB. Endocrine-inactive pituitary adenomas. Clinical neurosurgery. 1992 Jan and 38:10-31..

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Configurational changes of ruptured intracranial aneurysms at the window between pre-operative CTA and the definite clipping surgery

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ABSTRACT

Background: Ruptured intracranial aneurysms may face configurational changes in size and shape resulting from extreme weakness in their wall. These configurational changes are associated with an amplified risk of rupture and surgical challenges as these aneurysms have proven to be unstable. However, to our knowledge, no previous studies have addressed the issue of configurational aneurysmal changes between the patient's presentation (radiological images) and intraoperative findings. This paper aims to compare aneurysmal size and shape between pre-operative Computed tomography angiography and intraoperative lesion characteristics in a cohort of patients presented to our centre.

Methods: A retrospective analysis was performed on cases admitted to the Neurosurgery Teaching Hospital in Baghdad, Iraq, and underwent microsurgical clipping of ruptured aneurysms. Their records were checked for aneurysmal configurational changes by comparing pre-operative radiological images and intra-operative findings.

Results: Of the 275 patients, 5 cases were enrolled with aneurysmal configurational changes. Three of them were females, and two were males. The pre-operative aneurysmal shapes based on Computed tomography angiography were (3 unicyst and two conical shapes) and the range of aneurysmal size was (7-11 mm) with a mean of 9 mm. Compared to intra-operative findings, there were total aneurysmal shape changes (connected cyst in 4 patients and spherical aneurysm in one patient). The range of aneurysmal size was (7-11 mm) with a mean of 9 mm.

Keywords
intracranial aneurysm,
pre-operative CTA,
configurational changes

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Conclusion: Intracranial aneurysms may undergo configurational changes through the time between pre-op imaging and surgery; early detection of these changes may ensure optimal management of such cases.

INTRODUCTION

Intracranial aneurysm (ICA) is a common cerebrovascular disease with high morbidity and mortality in patients around 40–60 years old, characterized by abnormal focal dilation of cerebral arteries due to arterial wall weakness (13). Aneurysmal rupture is the most admissible cause of non-traumatic spontaneous subarachnoid hemorrhage (14). Despite advanced surgical techniques and peri-operative management, the morbidity and mortality associated with aneurysmal rupture are still very high, leading to an acute disability in 30% of cases and death in 30–50% of them (9,10). Ruptured intracranial aneurysms may face configurational changes in size and shape resulting from extreme weakness of the aneurysmal wall (6). It has been reported that approximately 10% of the aneurysms presented growth and changing size through follow-up imaging (5). These configurational changes are associated with an amplified risk of rupture and surgical challenges as these aneurysms have proven to be unstable (12).

Furthermore, this explains that the angiographic pathological finding is not correlated with the characteristics and nature of the aneurysm. Direct aneurysmal observation should be done accurately to detect if there are any abnormal configurational changes in the aneurysm. So, repeated regular follow-up computed tomography angiography (CTA) of an aneurysm is beneficial throughout the time between the first pre-operative (pre-op) CTA and the time of surgery. However, to our knowledge, no previous studies have addressed the issue of configurational aneurysmal changes between the patient's presentation (radiological images) and intraoperative findings. This paper aims to compare aneurysmal size and shape between pre-op CTA and intraoperative lesion characteristics in a cohort of patients presented to our center.

METHOD

A retrospective chart analysis was performed on cases admitted to the Neurosurgery Teaching Hospital in Baghdad, Iraq, and underwent microsurgical clipping of ruptured aneurysms for the period of (September 2018- March 2022). Their records were checked for the presence or absence of aneurysmal configurational changes by comparing pre-op CTA and intra-op findings. Patients with the presence of aneurysmal size and shape changes were analyzed according to the following parameters:

1. Pre-operative data: (A) patient demographics (age, sex). (B) Aneurysmal characteristics: location/side, shape based on CTA, size based on CTA.
2. Intra-operative data: (C) intra-op findings through changes in (size or shape or together), the time between the pre-op CTA and surgery.
3. Post-operative data: final outcome and last follow-up.

RESULT

Of the total 275 patients, 5 cases were enrolled with aneurysmal configurational changes. Three of them were females, and two of them were males. The range of patients' age was 41-62 years with a mean of 50 years. Regarding aneurysmal location and side, 3 out of 5 patients had posterior communicating artery (Pcom) aneurysm (two of them on the left side and one on the right side), while one patient had anterior communicating artery aneurysm and the last one had a right middle cerebral artery (MCA) aneurysm. The pre-op aneurysmal shapes based on CTA were (3 unicyst and two conical shapes) and the range of aneurysmal size was (7-11 mm) with a mean 9 mm. Compared to intra-op findings, there were total aneurysmal shape changes (connected cyst in 4 patients and spherical aneurysm in the last one). The range of aneurysmal size was (7-11 mm) with a mean of 9 mm. The time range between the pre-op CTA and surgery that the configurational aneurysmal changes occurred was (10-21 days) with a mean of 16 days.

Regarding the outcome, two patients were good with no deficit, one patient had intra-op rupture (IOR) and right-sided weakness (grade 3-4) for two months then was good with left side grade 4+ when followed up, one patient had a left-sided weakness (grade 2) then was good with left side grade 4+ when followed up. The last patients had incomplete clipping, left-sided weakness (grade 3), redo surgery, and complete clipping that was good with no deficit when followed up. The range of the last follow-up of the patients was (6-30 months) with a mean of 20 months. The included patient data is described in Table 1. In addition, examples of the included cases are depicted in figure1 and 2.

Table 1. Ruptured intracranial aneurysm cases with pre-operative configurational changes.

ID	Age	Sex	Aneurysmal location/Side	Pre-op Aneurysmal shape based on CTA	Pre-op Aneurysmal size based on CTA	Intra-op findings: change in size, and shape	Time between the pre-op CTA and surgery	Final Outcome	Last Follow up
1	45	F	L Pcom	unicyst	4 mm	Size 9 mm, shape 3 connected cysts	17 days	IOR, right side weakness (grade 3-4) for 2 months final outcome is Good with no deficit	24 months
2	58	F	AcomA	Conical shape dome	3 mm	Size 8 mm, shape 2 connected cysts	12 days	left side weakness (grade 2), final outcome is Good with left side grade 4+	6 months
3	47	M	R MCA	conical	4 mm	size 7 mm, spherical	21 days	incomplete clipping, left side weakness (grade 3), redo surgery and complete clipping, the final outcome is Good with normal	18 months
4	62	F	L Pcom	unicyst	5 mm	Size 10 mm, shape 2 connected cysts	10 days	Good with no deficit	24 months
5	41	M	R Pcom	unicyst	6 mm	Size 11 mm, shape 3 connected cysts	18 days	Good with no deficit	30 months

AcomA; Anterior communicating artery CTA; Computed tomography angiography, Pre-op; Pre-operative, Intra-op; Intra-operative Pcom; posterior communicating artery, A2; post-communicating anterior cerebral artery, MCA; middle cerebral artery.

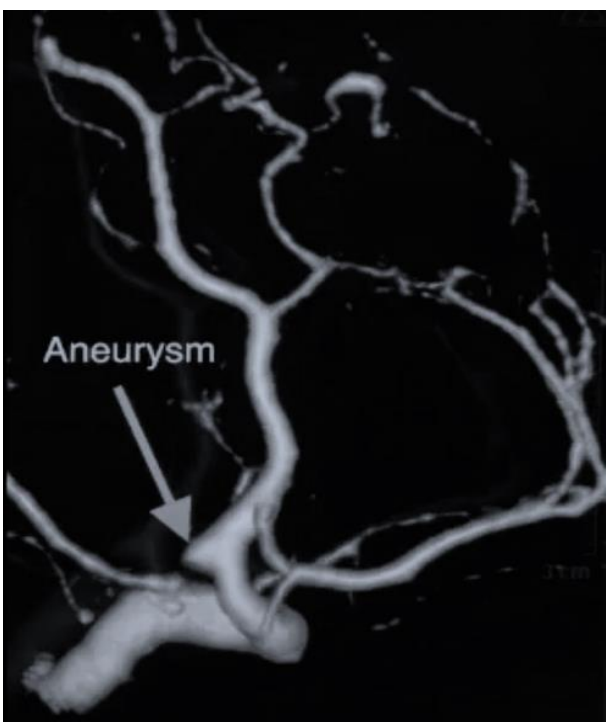

Figure 1. A. 45 yrs. female, presented with sudden severe headache and vomiting, CT showed SAH in basal cisterns (ID 1 in Table 1). Pre Op CTA showed a wide neck Left Pcom aneurysm measuring 4 mm.

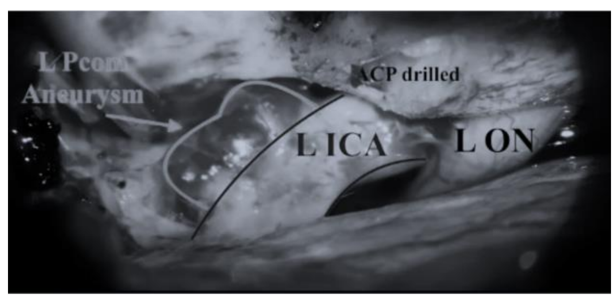

Figure 1. B. Intra-op view through Left pterional approach with the Pcom aneurysm and surrounding structures were dissected. 17 days after the rupture (CTA time), showed significant changes in the size and shape of the Pcom aneurysm. The intraoperative size was 9 mm, and the shape was 3 connected cysts.

Discussion

ICA, described as a pathological dilation of cerebral arteries found in the subarachnoid spaces, is considered the most challenging vascular lesion in the brain for clinicians (3). Aneurysmal formation is a multi-factorial disease that interferes with genetic and environmental factors and cerebral vessels. It is reported that ICA affects approximately 1-6% of the world's population and, specifically, 3–5% of the adult population (4). ICA is classified according to its size (small < 5mm, medium 5-10mm, large 10-15mm, giant > 25mm) and shape as saccular (berry), fusiform and mycotic aneurysms (1). aSAH is a devastating medical event with high morbidity and mortality and can be found in approximately 0.7-1.9% of cases (2). Follow-up assessment of ruptured aneurysms is recommended because the aneurysm can change its size and shape over time, which may affect the surgical planning and patient's outcome (11). These configurational changes are slightly different as some grow wider, whereas others' growth ends with forming a bleb. The difference in change mechanisms might have different risks to the patient's surgical outcome as different pathological processes underlie the aneurysmal growth and size (8).

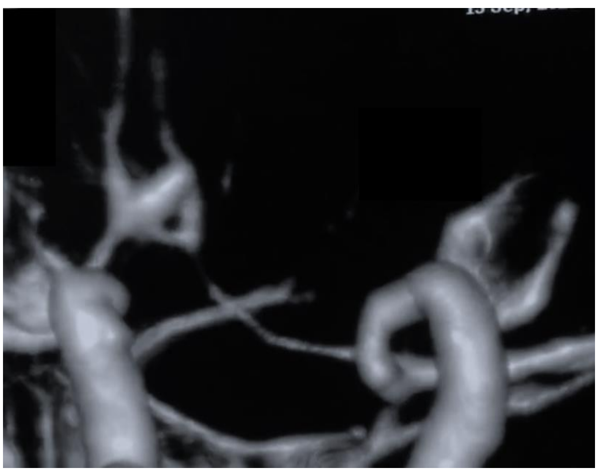

Figure 2. A. 58 yrs female, presented with sudden onset of severe headache, CT showed SAH in basal cisterns (ID 2 in Table 1). Pre-op CTA showed a conical shape aneurysm dome of an anterior communicating artery aneurysm measuring 3 mm.

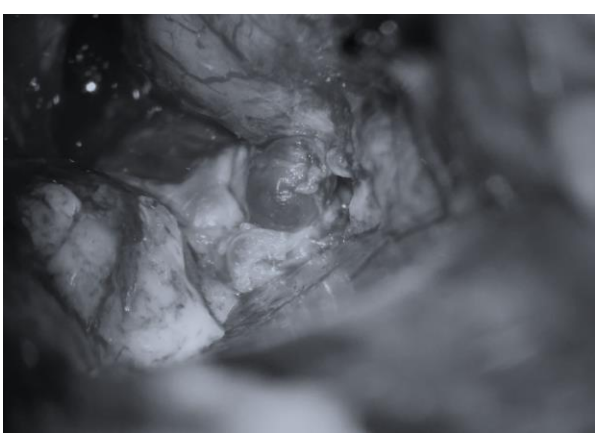

Figure 2. B. Intra-op view through Right pterional approach with the Acom aneurysm and surrounding structures dissected. of the aneurysm 12 days after the rupture, showed significant changes in the size of the Acom aneurysm with two connected cysts forming the dome and size 8 mm.

Moreover, the risk of aneurysmal configurational changes was found to depend on factors such as (age, sex, smoking, hypertension, history of a SAH, irregular shapes, multiple aneurysms, and anatomical site). However, the influence of individual risk factors on ICA growth is poorly understood. Ogwa et al. reported in their paper that the first aneurysmal angiogram of three patients revealed a tiny blister aneurysm. In contrast, in the second angiogram, there was an increase in aneurysmal sizes and shape change into the saccular aneurysm. During operation, these aneurysms faced extremely fragile walls with no firm aneurysms (7). This was reported in the literature; however, the description and impact of aneurysmal configurational changes in the period between the patient's presentation and surgical management are not addressed well. Our study will show the differences in aneurysmal growth and size between pre-op CTA and intra-op findings concerning possible factors and patient outcome assessment. Out of 5 patients who enrolled in our study, we noticed huge differences between the pre-op CTA aneurysmal characteristics and intra-op findings represented by configurational changes through size and shape, as shown in Table 1. The most commonly found aneurysm in those patients was PCOM aneurysm, whether it was right or left. The estimated aneurysmal size difference was 5 mm between pre- and post-aneurysmal changes.

Further, the time between CTA and surgery was 2-3 weeks which is a long period in which the configurational changes silently take place and lead to operative challenges regarding these changes. Fortunately, the patients were suitable after the operation, some with some complications resolved with time. So, the essential factor that can detect the aneurysmal change through its size and shape is the time between the last pre-op CTA and the time of surgery which should not be more than one week. As part of the Iraqi population, we faced many situations that obligate the surgeons to delay the operation, so this delay if it was more than one week; new CTA should be done to ensure that any configurational changes affect the patients and operation.

The small sample size of cases in our study is one of the limiting factors that may not render the statistics definitely accurate. However, it has a significant impact on operative challenges and patients' outcome.

The potentials and advantages of our study are primarily better understanding the intra-op aneurysmal changes and how to deal with these circumstances. Also, possible future studies may make this study a severe issue in decreasing intra-op challenges and improving patient outcomes.

In summary, the presence of aneurysmal differences between pre-op CTA and intra-op findings may be attributed to configurational size and shape changes due to the enormous fragility of the aneurysmal wall.

Conclusion

Intracranial aneurysms can face configurational changes through the time between pre-op imaging and surgery; early detection of these changes may ensure optimal management of such cases.

Abbreviations:
ICA = Intracranial aneurysm
CTA = Computed tomography angiography
aSAH = Aneurysmal subarachnoid haemorrhage Pre-op = Pre-operative
Intra-op = Intra-operative
Pcom = posterior communicating artery
A2 = post-communicating anterior cerebral artery MCA = middle cerebral artery

References

1. Alg VS, Sofat R, Houlden H, Werring DJ. Genetic risk factors for intracranial aneurysms: a meta-analysis in more than 116,000 individuals. Neurology. 2013 Jun 4;80(23):2154-65
2. Alwalid O, Long X, Xie M, Yang J, Cen C, Liu H, Han P. CT angiography-based radiomics for classification of intracranial aneurysm rupture. Frontiers in neurology. 2021 Feb 22;12:619864
3. Bo L, Wei B, Wang Z, Li C, Gao Z, Miao Z. Bioinformatic analysis of gene expression profiling of intracranial aneurysm. Molecular medicine reports. 2018 Mar 1;17(3):3473-80.
4. Etminan N, Dreier R, Buchholz BA, Beseoglu K, Bruckner P, Matzenauer C, Torner JC, Brown Jr RD, Steiger HJ, Hänggi D, Macdonald RL. Age of collagen in intracranial saccular aneurysms. Stroke. 2014 Jun;45(6):1757-63
5. Etminan N, Rinkel GJ. Unruptured intracranial aneurysms: development, rupture and preventive management. Nature Reviews Neurology. 2016 Dec;12(12):699-713.
6. Kim JH, Kwon TH, Kim JH, Park YK, Chung HS. Internal carotid artery dorsal wall aneurysm with configurational change: are they all false aneurysms?. Surgical neurology. 2006 Oct 1;66(4):441-3.
7. Ogawa A, Suzuki M, Ogasawara K. Aneurysms at nonbranching sites in the supraclinoid portion of the internal carotid artery: internal carotid artery trunk aneurysms. Neurosurgery. 2000 Sep 1;47(3):578-86.
8. Raghavan ML, Ma B, Harbaugh RE. Quantified aneurysm shape and rupture risk. Journal of neurosurgery. 2005 Feb 1;102(2):355-62
9. Rikhtegar R, Mosimann PJ, Rothaupt J, Mirza-Aghazadeh-Attari M, Hallaj S, Yousefi M, Amiri A, Farashi E, Kheyrollahiyan A, Dolati S. Non-coding RNAs role in intracranial aneurysm: General principles with focus on inflammation. Life Sciences. 2021 Aug 1;278:119617.
10. Rincon F, Rossenwasser RH, Dumont A. The epidemiology of admissions of nontraumatic subarachnoid hemorrhage in the United States. Neurosurgery. 2013 Aug 1;73(2):217-23.
11. Thompson BG, Brown Jr RD, Amin-Hanjani S, Broderick JP, Cockroft KM, Connolly Jr ES, Duckwiler GR, Harris CC, Howard VJ, Johnston SC, Meyers PM. Guidelines for the management of patients with unruptured intracranial aneurysms: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2015 Aug;46(8):2368-400
12. Villablanca JP, Duckwiler GR, Jahan R, Tateshima S, Martin NA, Frazee J, Gonzalez NR, Sayre J, Vinuela FV. Natural history of asymptomatic unruptured cerebral aneurysms evaluated at CT angiography: growth and rupture incidence and correlation with epidemiologic risk factors. Radiology. 2013 Oct 1;269(1):258-65
13. Wei L, Wang Q, Zhang Y, Yang C, Guan H, Chen Y, Sun Z. Identification of key genes, transcription factors and microRNAs involved in intracranial aneurysm. Molecular Medicine Reports. 2018 Jan 1;17(1):891-7.
14. Zhang B, Fugleholm K, Day LB, Ye S, Weller RO, Day IN. Molecular pathogenesis of subarachnoid haemorrhage. The international journal of biochemistry & cell biology. 2003 Sep 1;35(9):1341-60.

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Neurosurgical management of posterior communicating artery aneurysmal perforators. Technical note through two case examples

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Keywords
posterior communicating artery aneurysms, true perforators, microsurgical clipping

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ABSTRACT

Background: Posterior communicating artery (PCom) aneurysms are prevalent intracranial aneurysms, frequently leading to subarachnoid haemorrhage and third nerve palsy. "True" perforators originating directly from the PCom aneurysmal sac are rare and pose unique surgical challenges. We present two illustrative cases where "true" PCom perforators were identified during surgery, necessitating tailored microsurgical approaches.

Case Reports: In case one, a 35-year-old female presented with a large PCom aneurysm. During surgery, a modified clipping technique successfully preserved a perforator originating from the aneurysmal sac. In case two, a 36-year-old male with a PCom aneurysm was found to have a perforator supplying the mesial temporal cortex. Sacrificing this perforator was necessary for aneurysm clipping.

Conclusion: This paper underscores the rarity of "true" PCom perforators and their impact on microsurgical approaches. Preoperative imaging techniques often fail to detect these small-calibre vessels, highlighting the importance of intraoperative identification. Tailored approaches based on individual anatomy and clinical context are essential. While endovascular procedures provide alternatives, adaptability in microsurgical techniques remains crucial for cases requiring intraoperative perforator management. Overall, understanding the complex vascular intricacies of PCom aneurysms, including "true" perforators, is vital for neurosurgeons. The delicate balance between preservation and necessity, along with ongoing research for improved preoperative identification, stands as the cornerstone for enhancing surgical outcomes in these complex cases.

INTRODUCTION

Posterior communicating artery (PCom) aneurysms are common, accounting for 25% of all intracranial aneurysms with a 48% rupture rate (1,2,12). Ruptured Pcom aneurysms have several variable symptoms. Typically, PCom presents with subarachnoid hemorrhage (SAH) and 3rd nerve palsy (1,6). Peri-aneurysmal perforating arteries (perforators) are small millimetre or submillimetre calibre vessels arising from the parent vessel in the vicinity of intracranial aneurysms. Peri-aneurysmal perforators have a variable incidence depending on the aneurysm site (7). Perforators are typically identified intraoperatively; only a small subset is visible in digital subtraction angiography (DSA) or computerized tomographic angiography (CTA) (3,7,9).

Perforating branches derived directly from parts of the aneurysm may be described as "true" perforators, as opposed to "peri-aneurysmal" perforators emerging from the parent vessel (Figure 1: A, B). Reports on “true” perforators are available on the basilar tip, internal carotid artery bifurcation, and anterior communicating artery aneurysms (7). Two cases of ruptured Pcom aneurysms characterized by intraoperatively-identified "true" perforators are reported in this paper, to document this rare finding and highlight its potential intraoperative implications.

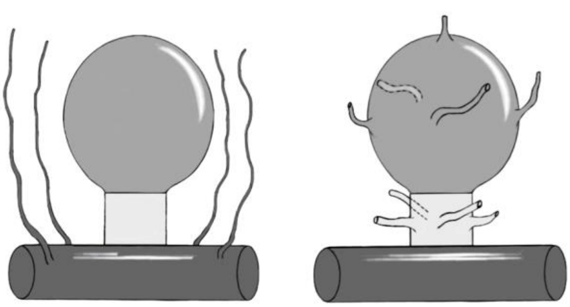


Figure 1. A: Peri-aneurysmal perforators arising from the parent vessel segments near the aneurysm. **B:** Potential locations for “True” Pcom perforators branching directly from the dome or neck of the aneurysm.

ILLUSTRATIVE CASES

Case one

An otherwise healthy, 35-year-old female had a sudden-onset, severe headache associated with meningismus. The patient was awake, alert, and oriented to person, time, and place. Neurological examination revealed a left-sided third nerve palsy. A brain computed tomography (CT) scan showed SAH in the basal cisterns. The CTA showed a large (14mm) Pcom aneurysm (Figure 2A).

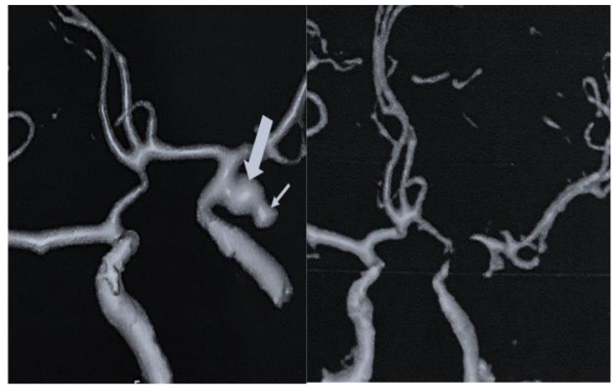


Figure 2. Cerebral CT angiography (3D reconstruction). **A**: Preoperative image showing a large left posterior communicating artery aneurysm (thick arrow) directed postero-laterally. A daughter cyst (thin arrow) is also seen. **B**: Postoperative image showing complete clipping of the aneurysm with no residual neck.

No endovascular facility was available in our country, leaving microsurgical clipping as the only possible mode of intervention. Microsurgical clipping of the aneurysm, through a standard, left-sided pterional, trans-Sylvian approach was planned. The patient was positioned supine with the head tilted 30 degrees to the right side. After the removal of the lateral part of the sphenoid ridge, the carotid cistern and the proximal part of the Sylvian fissure were opened. Dissection of the internal carotid artery was then carried out until the aneurysm dome and neck became visible. Next, the aneurysm neck was dissected. A relatively large perforating artery was found originating from the posterior part of the aneurysmal dome, a few millimetres from the aneurysm neck, (Figure 3A-B). The distal course of the perforator could not be confirmed. Thus, a decision was made to preserve the perforator by reconstructing the aneurysm neck with a modified clipping technique, using two tandem clips to exclude the aneurysm from circulation. The distal clip closed the major part of the aneurysm sac while the proximal one left a small residual neck posteriorly to maintain blood flow to the perforator, (Figure 3C-F). The patency of the perforator was confirmed by intraoperative Doppler ultrasonography.

The postoperative course was uneventful, and the patient's preoperative 3rd nerve palsy recovered completely. The post-operative CTA confirmed that the aneurysm was now clip-secured (Figure 2B). The patient was discharged home seven days post-operatively. At her six-month follow-up, the patient was well and both the CT and CTA studies showed no new significant findings.

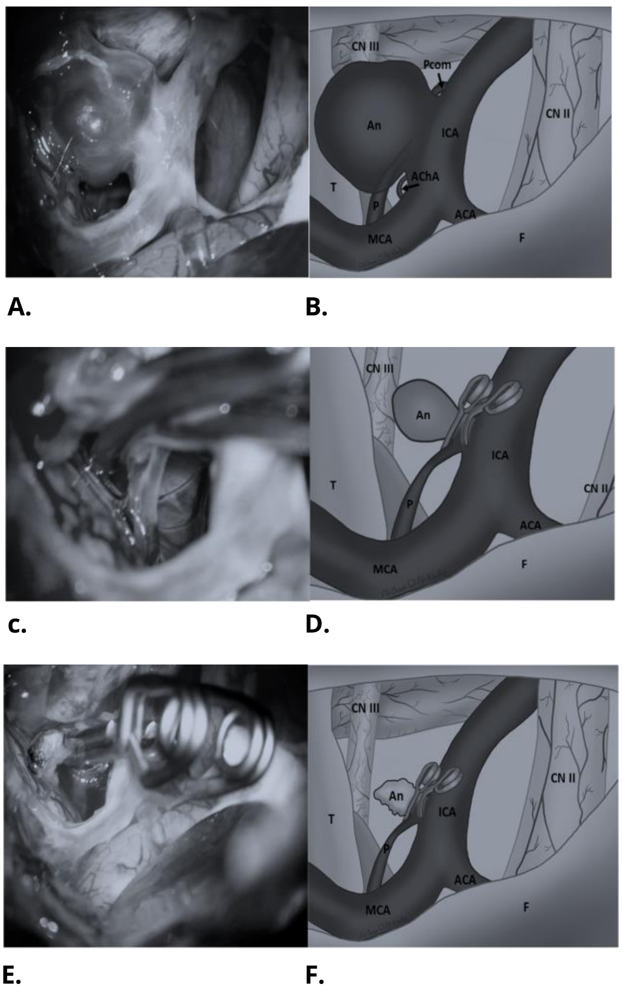


Figure 3. Intraoperative images of microsurgical clipping of posterior communicating artery aneurysm with illustrations. **A-B**: Pre-clipping image showing the perforator (P) arising from the posterior dome of the aneurysm (An), a few millimeters from its neck. The posterior communicating (Pcom) and anterior choroidal (AChA) arteries are seen arising from the proximal and distal neck of the aneurysm, respectively. **C-D**: Zoomed in view showing the application of a clip distal to the origin of the perforating branch, fully occluding the aneurysmal sac distal to it. A second clip is applied at the level of origin of the perforating branch, occluding most of the aneurysmal neck apart from its posterior part, maintaining flow to the perforating artery. **E-F**: Zoomed out view showing dissection and cauterization of the aneurysmal dome from the oculomotor nerve (CN III). ACA: anterior cerebral artery, AChA: anterior choroidal artery, An: aneurysm, CN II: optic nerve, CN III: oculomotor nerve, F: frontal lobe, ICA: internal carotid artery, MCA: middle cerebral artery, P: perforator, T: temporal lobe.

Case two

A previously healthy, 36-year-old male was admitted with the complaint of an acute, severe headache of three-day duration, along with one seizure episode. The patient was awake, alert, and oriented to person, time, and place. Neurological examination was within normal limits. Admission brain CT scan showed SAH in the basal cisterns, particularly in the suprasellar cistern, extending to the left Sylvian fissure. Cerebral CTA revealed a large (10mm) elongated left-sided Pcom aneurysm, with a marked narrowing of the supraclinoid segment of the internal carotid artery (ICA) on the left side, suggesting arterial vasospasm (Figure 4).

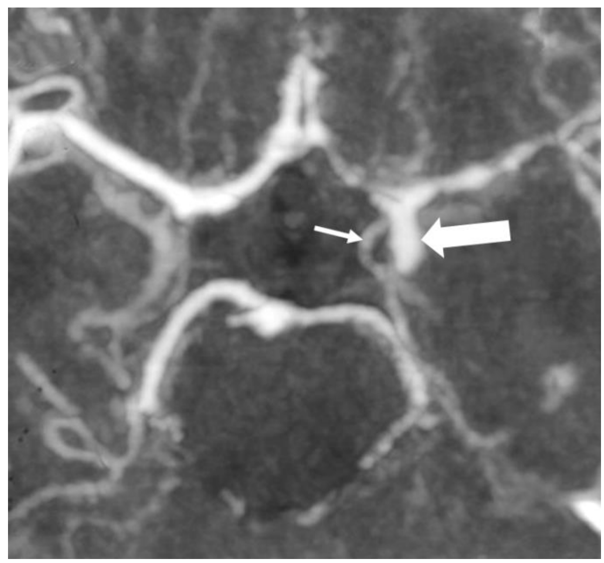
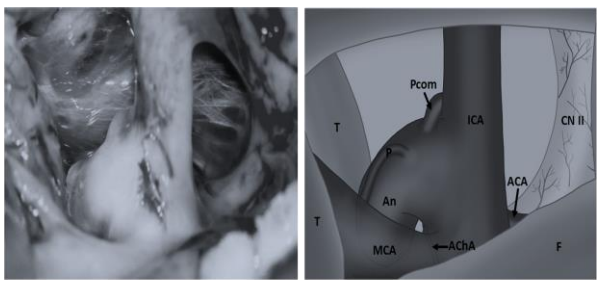


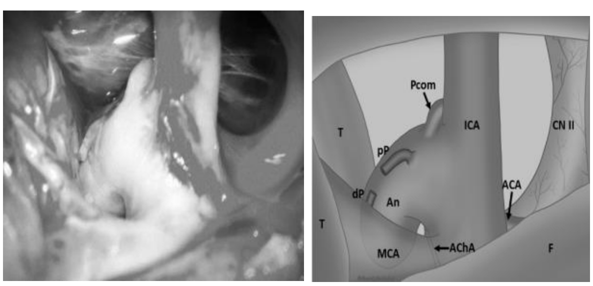
Figure 4. Pre-operative CTA showing a posteriorly projecting saccular aneurysm (thick arrow) originating from the communicating segment of the left ICA. The Pcom artery (think arrow) can be seen originating from the neck of the aneurysm. Marked vasospasm of the left supraclinoid ICA as well as the ACA and MCA can be seen.

Microscopic aneurysm clipping was planned using the left pterional trans-Sylvian approach. Following the dissection of the Sylvian fissure, the ICA and the aneurysmal neck were dissected. A relatively large perforating artery arising from the mid-part of the anterior surface of the aneurysm dome was discovered (Figure 5A-B). Following the course of this perforator showed that it supplied the mesial temporal cortex. Because of this perforator's source site, we were forced to sacrifice it so that the aneurysm could be clipped while holding the Pcom patent (Figure 5C-F).

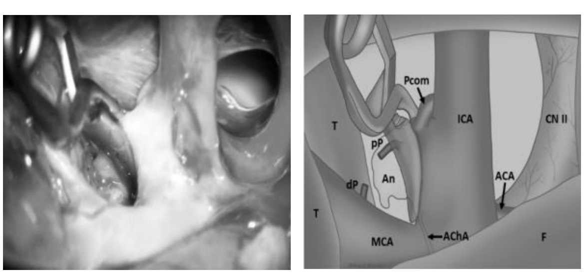
There were no surgical complications and the postoperative recovery was uneventful. The post-operative CTA showed occlusion of the aneurysm with the resolution of the vasospasm (Figure 6). The patient was discharged on day eight post-operatively. At his nine-month follow-up, the patient was well with no neurological deficits and both the CT and CTA studies showed no new significant findings.



A. **B.**



C. **D.**



E. **F.**

Figure 5. Intraoperative images of microsurgical clipping of posterior communicating artery aneurysm with illustrations. **A-B**: Pre-clipping image showing the perforator (P) arising from the anterior mid-sack of the aneurysm (An). The posterior communicating artery (Pcom) is seen arising from the proximal neck of the aneurysm. The anterior choroidal artery (AChA) is coursing behind the internal carotid artery (ICA) with its origin from the distal neck of the aneurysm. **C-D**: The proximal (pP) and distal (dP) ends of the perforator were cauterized and the segment between them cut off. **E-F**: Post-clipping image. The aneurysm was clipped using a curved clip, sparing the origins of both posterior communicating and anterior choroidal arteries. ACA: anterior cerebral artery, AChA: anterior choroidal artery, An: aneurysm, CN II: optic nerve, F: frontal lobe, ICA: internal carotid artery, MCA: middle cerebral artery, P: perforator, pP & dP: proximal & distal segments of the perforator, T: temporal lobe.

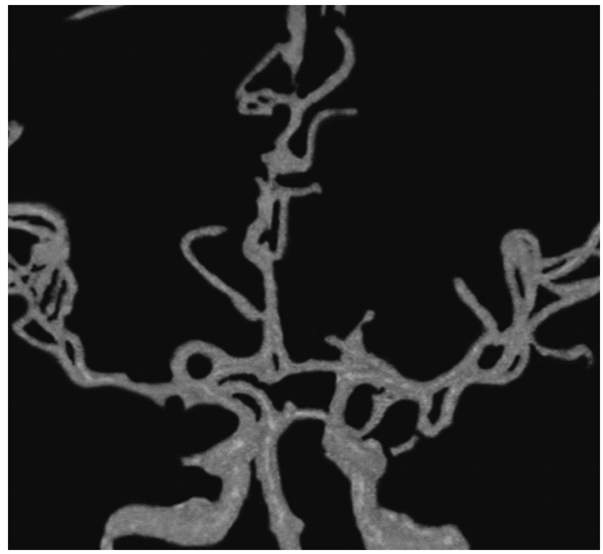


Figure 6. 8-day follow-up 3D-reconstructed CTA showing complete clipping of the aneurysm with no residual neck and resolution of the vasospasm.

Discussion

The current paper presents two case studies of "true" perforators originating from PCom aneurysms, a rarely documented phenomenon in the literature. These "true" perforators, which arise directly from the aneurysmal sac, are distinguishable from the more frequently studied peri-aneurysmal perforators (7). Our study underlines the clinical implications of such anatomic variants and points towards the need for an adapted surgical approach to manage them. Surgically, "true" PCom aneurysm perforators have only been mentioned once in the literature by Reynolds et al, who identified a small perforator originating directly from the fundus of a PCom aneurysm. Dissection of that perforator suggested that it supplied the internal capsule. The aneurysm was deemed unclippable and the perforator was maintained (8).

Aneurysms involving the PCom artery are not uncommon, they stand as one of the most frequent aneurysms encountered by neurosurgeons, ranking second in the overall prevalence of aneurysms, accounting for a quarter (25%) of all aneurysmal cases, and a half (50%) of all internal carotid artery aneurysms (2,12). They not only present with the conventional SAH but also they can manifest as isolated oculomotor nerve palsy or non-traumatic subdural hematoma (6). In terms of surgical treatment, PCom aneurysms can be one of the most straightforward or one of the most difficult aneurysms to treat surgically. This depends on the considerable variations in the anatomy of the PCom complex, which carry substantial surgical implications (1).

Despite the known variability of PCom aneurysms and their associated vascular anatomy, there is a paucity of information about "true" PCom perforators. On average, seven perforating arteries originate from the PCom, with surgical clipping, occlusion of these peri-aneurysmal perforators is considered to cause hemiparesis and prolonged disturbance of consciousness. This presents a heightened challenge in achieving a pure separation of the aneurysm from the surrounding circulation (3,9). In addition, the presence of "true" perforators further complicates these surgical challenges and may potentially impact the choice of surgical approach and technique. In our cases, the identification of these "true" perforators dictated alterations in the conventional clipping process, thus underscoring their clinical significance.

Current imaging modalities like DSA or CTA often fall short in the preoperative identification of these small-caliber vessels. This limitation arises due to the inherently limited resolution of these imaging techniques in visualizing intricate anatomical details at a microscopic level, as the diameter of the perforators within the PCom complex spans from 0.1 to 1 millimeter (4,7,11). Therefore, this makes intraoperative identification crucial in the majority of cases. While the first case in our study emphasizes the need for careful intraoperative investigation, highlighting the pivotal role of surgical expertise, the second patient's case underlines the necessity of making complex decisions in real-time when such perforators cannot be preserved.

In non-complicated cases, most PCom aneurysms are treated with straight or curved clips. A fenestrated clip over the internal carotid artery may be required in a small number of PCom aneurysms that are large or with specific dome projections. The clip is directly positioned around the origin of the aneurysm neck while sparing vital neighboring blood vessels and perforating branches. The primary objective of the technique is to prevent blood flow into the aneurysm sac, thereby reducing the risk of rupture (9). However, our modified clipping technique for PCom aneurysms demonstrates its effectiveness through its application in our complicated cases with “true” perforators. In the first case, a straight clip was positioned distally to the origin of the perforating branch, ensuring complete occlusion of the aneurysmal sac. Subsequently, a second curved clip was positioned at the origin of the perforating branch, partially occluding the aneurysm's neck while maintaining perfusion to the perforating artery (Figure 3). Turning to the second case, prior to clipping, microscopic images revealed the perforator's precise origin from the aneurysm's anterior mid-sac, supplying the mesial temporal cortex. To enable aneurysm clipping while preserving PCom patency, we sacrificed this source. A curved clip was adroitly utilized to manage the aneurysm, without compromising the origins of the PCom and anterior choroidal arteries (Figure 5). These customized approaches were essential to preserving vital blood vessels while achieving the primary objective of optimal aneurysm closure.

The intraoperative implications of "true" PCom perforators, as demonstrated in our case studies, could be profound. They can guide decision-making regarding the preservation or sacrifice of these vessels during surgery. This choice directly relates to the risk of ischemic complications or intra-operative hemorrhage if these vessels are inadvertently injured (3). These decisions become more nuanced when the distal course of these perforators cannot be definitively determined, as seen in the first case. The use of a modified clipping technique to preserve the identified perforator helped avoid potential ischemic consequences, underlining the need for adaptability in neurosurgical interventions.

It is noteworthy that the most recent endovascular procedures are recognized as an effective approach for managing complex PCom variants. A study conducted by Kwon et al. reported a case of a giant PCom aneurysm that was treated with trapping and thrombectomy. The aneurysm had perforator arteries originating from the dome, but these arteries were successfully preserved (5). Another study by Wang et al. introduced a novel endovascular technique called λ stenting for the treatment of PCom aneurysms with fetal-type PCom originating from the aneurysm dome. The technique was found to be safe and effective in preserving the PCom and preventing ischemic complications (10). However, in cases of intraoperative identification, as seen in our study, the versatility and adaptability of the clipping technique, emerge as the method of treatment of such variants, even when faced with unforeseen challenges in a surgical setting.

CONCLUSION

Our findings underline the importance of having a comprehensive understanding of the complex vasculature associated with PCom aneurysms and the potential presence of "true" perforators. While preservation should be the aim where possible, sacrifice may sometimes be unavoidable. Therefore, a tailored approach is crucial based on the individual patient's anatomy and clinical context. It is evident that further reporting and analysis are necessary to fully understand the implications of "true" PCom perforators in the intraoperative management of PCom aneurysms. The development of new imaging modalities that allow for the accurate preoperative identification of these perforators could significantly improve surgical outcomes in the future.

REFERENCES

1. Golshani K, Ferrell A, Zomorodi A, Smith TP, Britz GW. A review of the management of posterior communicating artery aneurysms in the modern era. Surgical neurology international. 2010;1.
2. Hsu SP, Krisht AF, Lin CF, Chen HH, Chen MH, Shih YH, Hsu HS. Immediate results of microsurgical clipping of posterior communicating artery aneurysms using the pretemporal transclinoidal approach. Journal of the Chinese Medical Association. 2012 Sep 1;75(9):454-8.
3. Joo SP, Kim TS. The clinical importance of perforator preservation in intracranial aneurysm surgery: An overview with a review of the literature. Chonnam medical journal. 2017 Jan
4. Kataoka H, Iihara K. Microsurgical anatomy of perforators: The efficacy of ICG videoangiography and neuroendoscopy. Japanese Journal of Neurosurgery. 2015 Jan 1;24(1):12-1;53(1):47-55.
5. Mochizuki Y, Kawashima A, Yamaguch K, Okada Y. Thrombosed giant "true" posterior communicating artery aneurysm treated by trapping and thrombectomy. Asian Journal of Neurosurgery. 2017 Dec;12(04):757-9.
6. Patel K, Guilfoyle MR, Bulters DO, Kirollos RW, Antoun NM, Higgins JN, Kirkpatrick PJ, Trivedi RA. Recovery of oculomotor nerve palsy secondary to posterior communicating artery aneurysms. British journal of neurosurgery. 2014 Aug 1;28(4):483-7.
7. Pritz MB. Perforator and secondary branch origin in relation to the neck of saccular, cerebral bifurcation aneurysms. World neurosurgery. 2014 Nov 1;82(5):726-32.
8. Reynolds MR, Roland JL, Kamath AA, Cross DT, Dacey RG. Microsurgical confirmation of perforating arteries arising from the fundus of a posterior communicating artery aneurysm. Neurosurgical focus. 2015 Jul 1;39(videosuppl1): V16.
9. Tanabe J, Ishikawa T, Moroi J, Sakata Y, Hadeishi H. Impact of right-sided aneurysm, rupture status, and size of aneurysm on perforator infarction following microsurgical clipping of posterior communicating artery aneurysms with a distal transsylvian approach. World Neurosurgery. 2018 Mar 1;111:e905-11.
10. Tanabe J, Nakahara I, Matsumoto S, Suyama Y, Morioka J, Hasebe A, Watanabe S, Suyama K, Kuwahara K, Irie K. λ stenting: a novel technique for posterior communicating artery aneurysms with fetal-type posterior communicating artery originating from the aneurysm dome. Neuroradiology. 2022 Jan;64:151-9.
11. Vogels V, Dammers R, van Bilsen M, Volovici V. Deep cerebral perforators: anatomical distribution and clinical symptoms: an overview. Stroke. 2021 Oct;52(10):e660-74.
12. Zelman S, Goebel MC, Manthey DE, Hawkins S. Large posterior communicating artery aneurysm: initial presentation with reproducible facial pain without cranial nerve deficit. Western Journal of Emergency Medicine. 2016 Nov;17(6):808.

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21 cm long intra-medullary tuberculoma of dorsal spinal cord. A rare lesion and its management

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ABSTRACT
Intramedullary tuberculoma (IMT) is a rare form of spinal tuberculosis. The incidence of this disease is 01-02/100,000 patients (1). Occurs by hematogenous dissemination from focus elsewhere in the body. Magnetic resonance imaging (MRI) is helpful for diagnosing IMT at an early stage and it is also very useful in follow-up. CSF PCR studies are confirmatory. Histopathology is the Gold Standard. AKT drug therapy is the mainstay of treatment for IMT, with surgery indicated in select patients. Here, we describe a female patient with long-segment dorsal intramedullary tuberculoma with compressive myelopathy treated with surgery and put on AKT who improved clinically.

Keywords
intramedullary,
tuberculoma,
21cm

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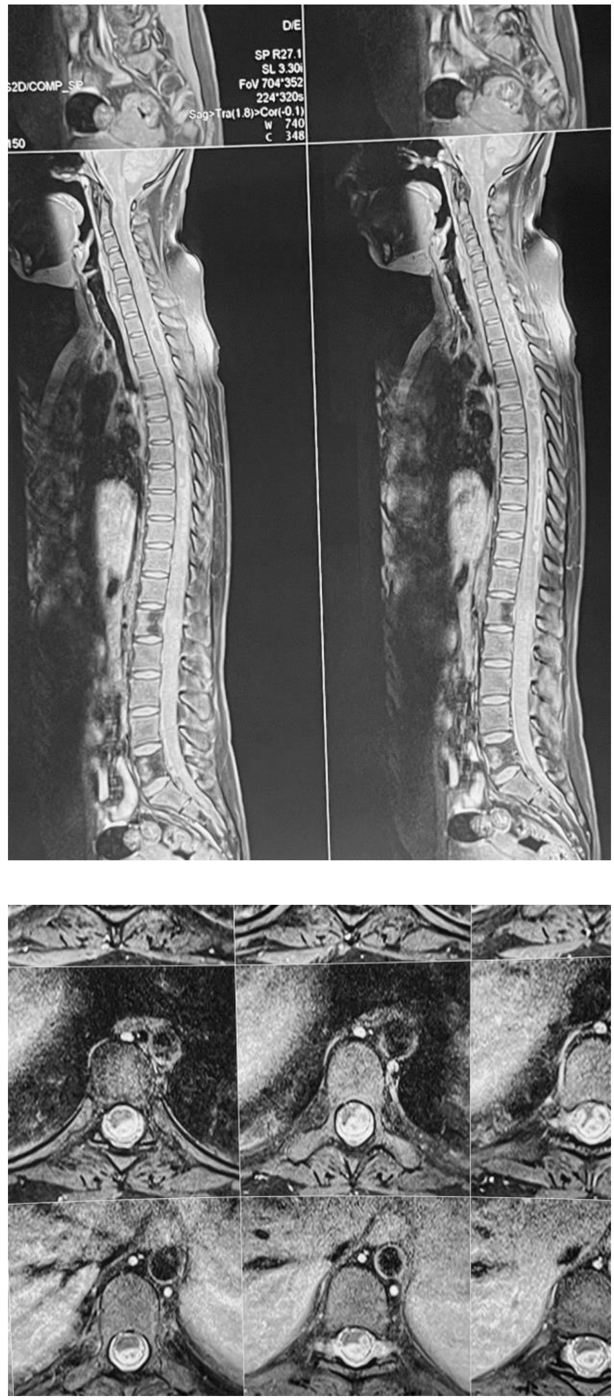
INTRODUCTION

Tuberculosis can theoretically affect any tissue of the human body, but in practicality, there are different predilections and incidences for different tissues. The spectrum of Spinal TB includes – Tubercular Spondylodiscitis – potts disease (most common), TB myelitis (next common) and intra-spinal TB (rare)(2). Intraspinal TB further can be TB arachnoiditis, meningitis and Intra-Medullary Tuberculoma (IMT)(2). IMT is extremely rare entity with incidence. Spinal IMTs are extremely rare, seen in only 1- 2 of 100,000 cases of TB (1) and 2 of 1000 cases of CNS TB (2). The first report of IMT was given by Albercrombie in 1828. (3) In 1960, Lin et al. reviewed literature of IMT and accounted for 104 cases, of which majority were diagnosed post-mortem (3). The first magnetic resonance imaging (MRI) documented description of tuberculoma was given by Rhoton et al. in 1988(4). Clinical presentation is of a compressive myelopathy but with MRI showing Intramedullary lesion, can be misdiagnosed for Intra-Medullary tumour. Presentation of Intra-Medullary cord syndrome is unusual compared to common spinal TB presentations. Here we report such a case with such unusual presentation, where sound knowledge of MRI interpretation, corelation with past history of Koch elsewhere in body, helped to diagnose TB and we discuss the treatments given and clinical outcome.

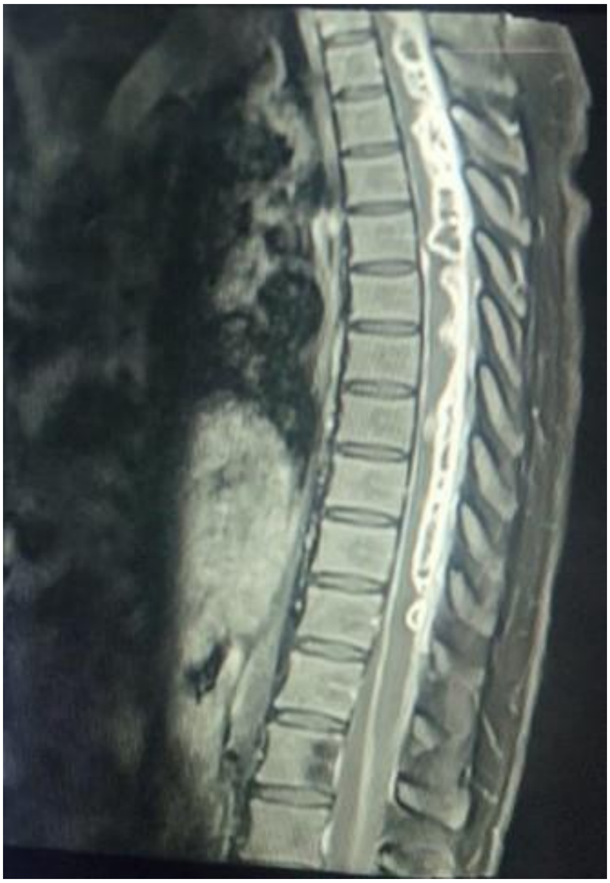
Case report

A 17-year-old female, a student, presented with complaints of back pain which was sudden insidious onset ,was described as persistent dull ache and was radiation to both lower limbs and which was gradually increasing intensity over 1 month until presentation ; this was a/w slow progressive weakness over both lower limbs and pt was rendered bed ridden at presentation ; a/w bladder and bowel incontinence for which she was foley catheterised. She gives a past history of CNS TB with communicating Hydrocephalus for which she was right mpvp shunted at private hospital 06 years back and she had completed AKT - during which she developed Ethambutol induced ocular toxicity. She had no other comorbidities, no other operative history. On examination, her higher mental functions and cranial nerves were intact. Her muscles on lower limbs started to show atrophy.

Tone was increased on both lower limbs. Muscle power was grade 05 in all muscle groups of both upper limbs while it was grade 02 over all muscles of the lower limbs. Reflexes were normal over all 4 limbs. Plantars were mute. She had sensory impairment below D6 corresponding to vertebrae level and altered sensation with hypoesthesia. Clinical provisional diagnosis was made of myelopathy and could be due to TB spondylodiscitis (i/v/o past history) and patient was investigated further. Routine blood investigations were done and analysed. Erythrocyte Sedimentation Rate was done and was found to be elevated. 'Triple H' was found to be normal and Plain chest radiograph was also found to be normal. Contrast enhanced MRI was done – this showed a long segment patchy intradural, intramedullary lesion with T1 iso-intense and T2 hypo-intense extending from c7 to d10 level with maximum thickness at d6 level. It was peripheral rim enhancing on contrast. CSF showed elevated protein, increased cellularity with 100% lymphocytic predominance. There were also small tuberculomas in the basal brain but with no mass effect/ hydrocephalus. These findings lead us to shift diagnosis to that of Intra-Medullary Tuberculoma and hence consulted pulmonologists for starting AKT(i/v/o past history of AKT and ethambutol toxicity) and decision was taken to put her on Bedaquilline based AKT. Simultaneously, we also opted for surgery for spine as she was having frank myelopathy with neurodeficits, aim was to achieve excision of the lesion and decompress where necessary. Intraoperatively we found a intr-dural extrapial long segment tuberculoma from c7 to d10 levels – this was excised totally and cord was free hence laminae placed back. Histopathology was suggestive of chronic granulomatous inflammation suggestive tuberculosis and



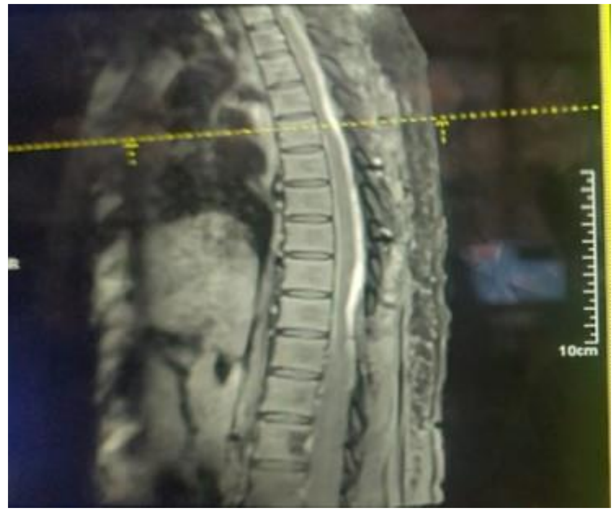
Figures 1, 2, 3. Pre op MRI Cervico dorsal spine with contrast showing intradural intramedullary contrast enhancing lesion.



GeneXpert on the tissue sample was positive for M.Tb and with Rif resistance detected. This confirmed our diagnosis of Spinal Intra-medullary tuberculoma. On POD 02, MRI showed significant radiological resolution and on POD 10, patient was discharged with Power on both lower limbs grade 03- a clinical improvement. Patient kept on follow up with plan to repeat MRI at 03 months.

Discussion

CNS TB accounts for approximately 0.5-2% of all cases of TB, carries a high mortality and neurological morbidity,(1) . Spinal TB is very rare and the common forms of spinal TB are tuberculous spondylitis, TB myelitis, and intraspinal TB. Intraspinal TB could be spinal meningitis, arachnoiditis, IMT, and abscess. IMT is very uncommon and till now roughly 150 cases have been reported(6). The thoracic segment is the most common site of IMTB and hematogenous spread is usually the main etiology followed by CSF seeding – this is a/w longer segmental involvement (7). Most cases of intramedullary tuberculomas are subacute and present with progressive symptoms suggestive of a compressive myelopathy.



Figures 4, 5. Post operative MRI with contrast s/o complete excision of lesion.

Our patient had the picture of such subacute cord compression with past history of TB in brain hence corroborating with CSF seeding theory and also consistent with this is finding of a long segment involvement. Immunoosuppresive states in general are known to favour disseminated Kochs hence was also done and found negative in our patient. Raised ESR is again favourable for diagnosis of Koch. Advent of MRI has made diagnosis of IMT more accurate and earlier. In the early phase, the tuberculoma is characterized by severe inflammatory reaction which causes severe edema. At this stage, the gel capsule is not well formed. During this stage, the enhancement after contrast examination is uniform. T1WI and T2WI both show equal signal intensity(early phase). As the gel content in the tuberculoma increases, the peripheral edema begins to disappear(intermediate phase). As a result, T1WI shows isointense lesions while T2WI shows low or isointense lesions. Contrast MRI shows central hypointensity with rim enhancement. With the development of caseation(late phase), T2WI shows a typical "target sign," which means that it exhibits a range from the low signal target to the high signal rim and also from the center of the low signal rim to the peripheral parts. The caseous substance appears hyperintense at the center, which gives the characteristic target sign. The low signal rim in the external region is composed of collagen fibers produced by fibroblasts.

Figure 6. 21 cm long specimen of tuberculoma.

The target sign is a valuable indicator that helps differentiate spinal tuberculoma from other intramedullary lesions. Rim enhancement and presence of sharp margins also differentiates IMT from intramedullary tumours.(8)(9)(10)(11) . In our patient, there was t2 hypointensity with peripheral rim enhancement and central hypointensity on contrast. This corresponds to the intermediate phase. Further CSF study was done which showed elevated protein and increased cellular count with 100% lymphocytes. This is a well-accepted classical picture for CNS Koch. Hence at this juncture, we decided to start AKT immediately. Now this girl had already received AKT and already got Ethambutol toxicity. So we consulted pulmonologists and ophthalmologists and started her on BDQ based AKT with ophthalmological safe drugs. Indications for surgery in IMT.

1. Gross neurological deficits
2. Worsening of neurological status during Rx
3. Paradoxical enlargement of lesion during Rx

Our patient had neurodeficits and myeopathy which had to be hence operated. Surgical resection was performed through posterior approach from c7 to d10 Level. Midline durotomy was performed and showed a thick mass of 11 cm extrapial tuberculoma. The patient underwent gross total excision of the mass and there was no need as such of bony decompression. The tissue was sent for HPE which revealed a granulomatous lesion with a central area of caseation in keeping with a tuberculoma. Histopathological diagnosis is also confirmatory for TB. We also further confirmed with GeneXpert. Reason for GeneXpert was twofold, we wanted to find out why in a immunocompetent patient there is reccurence and dissemination of TB apart from confirming diagnosis.

GeneXpert showed us Mtb and confirmed resistance to Rif – hence making our calculated assumption correct. This child had initially itself CNS TB in the brain with communicating hydrocephalus which was incompletely evaluated at the time (could be due to treatment done at periphery and was odne in 2016 when GeneXpert may not have been in vogue) and was shunted and received line one AKT but which was already a resistant strain of Mtb. This then over period of time formed small tuberculomas in basal brain and disseminated to long segment of cord by CSF and gave rise IMT. Hence aptly managed with BDQ AKT and optimum surgery.

At POD 2 ther was significant radiological resolution. Clinical improvement was evident at Day 10 with marginal increase in power in both lower limbs. Patient to be kept in follow up and MRI and clinical assessment repeated at 3 months. This case is being reported for its sheer rarity and to highlight diagnostic features and importance of surgery along with medical treatment to achieve good clinical results.

Conclusion

Rare entity of IMT is to be kept in mind in endemic nations like India when patient has intramedullary cord symptoms in picture of history of Kochs disease. Although medical management is to be instituted at earliest, this case report emphasised role of surgery in this disease. Look for drug resistance / immunosuppressive states in such disease and treat those properly which in turns will only help eradicating this entity. This case could provide some evidence-based data, thus contributing to the future research studies and clinical practice.

References

1. RNTCP status report TB India. Central TB division. Ministry of health and family welfare Page. 2010;2:3.
2. A rare case of intramedullary tuberculoma: Complete resolution after medical treatment and role of magnetic resonance imaging in diagnosis and follow-up Suresh Chidambaram Thirunavukarasu and Arunkumar Ramachandrappa-Asian J Neurosurg. 2012 Oct-Dec; 7(4): 223–226
3. Lin TH. Intramedullary tuberculoma of the spinal cord. J Neurosurgery. 1960;17:497–9. [PubMed] [Google Scholar]
4. Parmar H, Shah J, Patkar D, Varma R. Intramedullary tuberculomas. MR findings in seven patients. Acta Radiol. 2000;41:572–7
5. H.L. Yen, R.J. Lee, J.W. Lin, H.J. Chen Multiple tuberculomas in the brain and spinal cord: a case report Spine (Phila Pa 1976), 28 (23) (2003), pp. 499-502
6. Rhoton EL, Ballinger WE, Quisling R, Sypert GW. Intramedullary spinal tuberculoma. Neurosurgery. 1988;22:733
7. Borges MA, Carmo MI, Sambo MR, Borges FC, Araujo CM, Campos MJ, et al. Intramedullary tuberculoma in a patient with human immunodeficiency virus infection and disseminated multidrug-resistant tuberculosis: case report. Int J Infect Dis 1998;2:164-7.
8. Gupta RK, Gupta S, Kumar S, Kohli A, Mishra UK, Gujral RB. MRI in intraspinal tuberculosis. Neuroradiology. 1994;36:39–43.
9. Parmar H, Shah J, Patkar D, Varma R. Intramedullary tuberculomas. MR findings in seven patients. Acta Radiol. 2000;41:572–7.
10. Lu M. Imaging diagnosis of spinal intramedullary tuberculoma: Case reports and literature review. J Spinal Cord Med. 2010;33:159–62.
11. Jinkins JR, Gupta R, Chung KH, Rodriguez-Cebojal J. MR imaging of central nervous system tuberculosis. Radiol Clin North Am. 1995;33:771–86..

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Intestinal perforation due to a ventriculoperitoneal shunting catheter. Case report and review

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ABSTRACT

Introduction: The ventriculoperitoneal (VP) shunt allows the drainage of excess cerebrospinal fluid; among its gastrointestinal complications are intestinal volvulus, pseudocyst, and perforation, the last of which is rare but potentially serious, with a frequency between 0.1% and 0.7% and a mortality rate around 15%. We present the case of a male child who required VP shunt colocation and suffered GI tract perforation with transanal protrusion.

Case report: A three-year-old male with a long clinical history of pilocytic astrocytoma in the posterior fossa that was partially resected in February 2022 and with VP shunt placement since March 2022. He was diagnosed with an intestinal perforation and anal externalization due to a VP shunt. He required surgery and management with combined antimicrobials.

Discussion: Although the aetiology of GI tract perforation due to a VP shunt is not fully established, some risk factors have been described, including the use of abdominal trocars, rigid distal catheters, silicone allergies, and children's thin intestine walls. Treatment must be individualized, with some principles including external drainage of the proximal part until the CSF fluid reaches a biochemically near-normal state and becomes sterile on culture; secondly, the administration of intravenous antibiotics; and finally, the removal of the perforating section of the catheter.

Conclusions: Bowel perforation due to a VP shunt is an extremely rare entity whose aetiology is not fully established, primarily affects children and may be mostly related to chronic-irritative gastrointestinal distress. Individualizing treatment is the cornerstone of the therapeutic approach that includes VP shunt replacement, antimicrobial therapy, and management of life-threatening complications.

Keywords
ventriculoperitoneal shunt,
gastrointestinal tract,
children,
neurosurgery

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INTRODUCTION

The ventriculoperitoneal (VP) shunt allows drainage of excess cerebrospinal fluid (CSF) under circumstances where there is an obstruction in the normal outflow or where there is an absorptive abnormality of CSF (1). Considering the technique of the procedure, some complications may occur, including abdominal complications, which include intestinal volvulus, pseudocyst, and perforation of different anatomical sites like the scrotum, umbilicus, vagina, or gastrointestinal (GI) tract (2,3). Perforation of the GI tract is a rare but potentially serious complication that has a frequency between 0.01% and 0.07% (4-8). Considering the main target population for VP, most VP shunt protrusions manifest in individuals who are younger than 10 years old (9) with up to 70% of the reported patients being children (10).

Since the first case report about GI tract perforation for a VP shunt in 1966 (11) about a hundred cases have been described (10, 12), and half of them were asymptomatic (10). The mortality rate of intestinal perforation by a VP catheter is around 15% (7). The reduction in mortality and morbidity is substantial when early diagnosis and treatment are implemented, particularly in cases of asymptomatic intestinal perforations (10).

Anal extrusion of the VP shunt is a shocking and even more rare condition associated with perforation of the GI tract. Some cases have been described in children (2,4,5,9,12-15) and others in adults (6, 16-18). We present the case of a male child who required VP shunt colocation and suffered GI tract perforation with transanal protrusion.

CASE REPORT

A three-year-old male with a long clinical history of pilocytic astrocytoma in the posterior fossa that was partially resected in February 2022 and with VP shunt placement since March 2022 with the requirement of resection of a cyst at the tip of the VP shunt catheter in January 2023 and with recent Magnetic resonance imaging (MRI) findings of spinal metastasis was brought by his mother to the emergency room after finding a foreign body in the anal region. The mother decided to pull the catheter, suspecting other causes, without achieving extraction.

Initial clinical examination showed a heart rate of 114 beats per minute, a breath rate of 22 breaths per minute, a temperature of 36 °C, oxygen saturation of 97%, and a VP shunt catheter protruding through the anus. Initial labs showed slightly microcytic and hypochromic anemia (hemoglobin (Hb): 11.9 g/dL, hematocrit (Hto): 34.5%, mean corpuscular volume (MCV): 72.2 um3, mean corpuscular hemoglobin (MCH): 24.8 pg.), and positive C-reactive protein (CRP) (48 mg/L). Initial skull, chest, and abdomen radiography showed a VP catheter with a wide travel in the body, making multiple turns to exit through the anus (Figure 1).

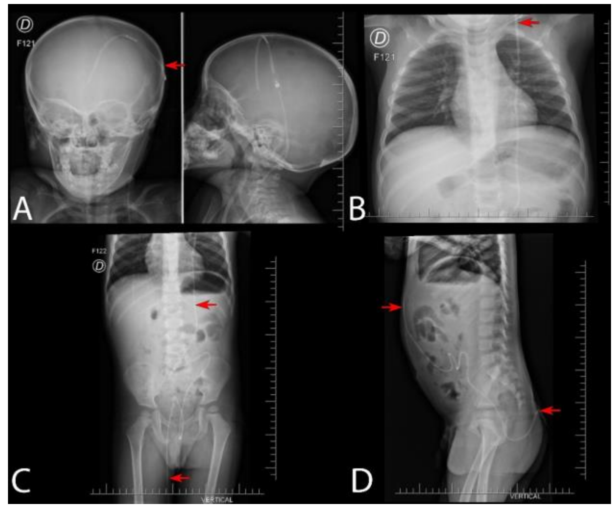

Figure 1.

Abdominal Computerized Tomography (CT) showed colonic perforation migration through the bowel of the distal VP shunt to the anal level (Figure 2) A head CT reported a fossa posterior tumor previously known and a VP shunt in an adequate position (Figure 3).

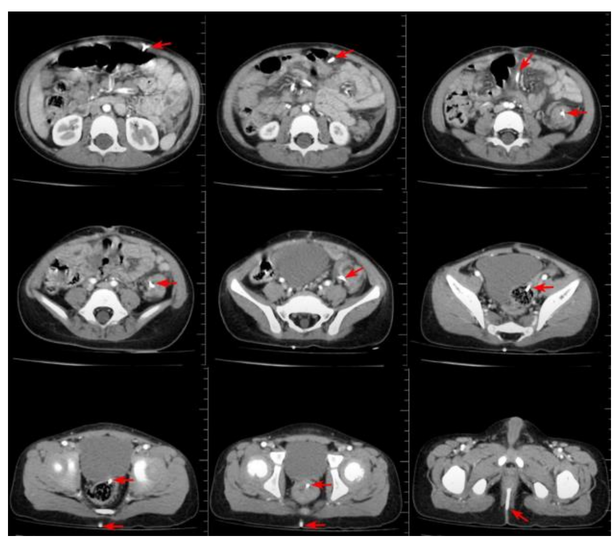

Figure 2.

Initial surgical management was externalization of the VP shunt via clavicular and partial removal of the distal catheter via anal; days later, he underwent surgery for a change of VP He was also given antibiotics through a central venous catheter (CVC) in the left subclavian area before and after surgery.

These included metronidazole (140 mg IV every 8 hours) for ten days, cefepime (700 mg IV every 8 hours) for thirteen days, and vancomycin (210 mg IV every six hours) for sixteen days.

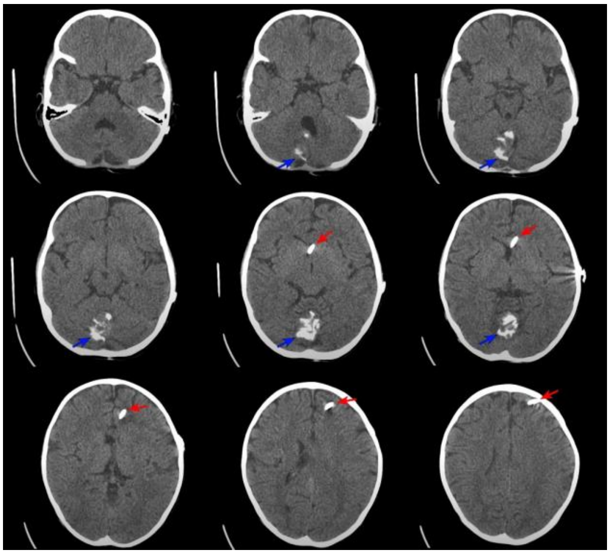

Figure 3.

During postoperative VP shunt replacement, the patient presented with fever (38.4 °C), tachycardia (162 bpm), tachypnea (31 bpm), neck edema, and abdominal pain. Laboratory control reported a deteriorating microcytic and hypochromic anemia (Hb: 8.7 g/dL, Hto: 26.4%, MCV: 74.2 um3, MCH: 24.4 pg.), CRP positive (48 mg/L), SARS-COV-2 antigen negative, neck ultrasonography showed soft tissue edema in the left neck and reactive lymph nodes, and abdominal ultrasonography showed slightly free fluid in the pelvis. Blood cultures and CSF cultures were reported as negative.

The medical staff made an antibiotic switch from cefepime to meropenem (560 mg IV every 8 hours) and a change of CVC to the right internal jugular. Imaging studies reported neck CT with contrast showing edema in soft tissues and reactive adenopathy **(Figure 4)**, and thorax CT with contrast showing both lower lobe consolidation, bilateral lower lobe subsegmental atelectasis, and right pleural effusion.

Despite antibiotic management, the patient continued to have a fever associated with emesis and diarrhea. Laboratory control reported leukocytosis (20.66 * 10^3/mm^3), neutrophilia (69.2%), microcytic and hypochromic mild anemia (Hb: 9.4 g/dL, Hto: 28%, VCM: 72.9 um3, HCM: 24.6 pg.), a rise in CRP (96 mg/L), a respiratory viral panel positive to enterovirus and syncytial virus, and a new blood culture isolated *Staphylococcus epidermidis*. The antimicrobial management was modified by adding clindamycin (140 mg IV every 6 hours) and restarting vancomycin (210 mg IV every 6 hours); however, the pediatric infectologist suspended meropenem and clindamycin while continuing vancomycin. Due to the requirement of a major complexity level considering the need for a new evaluation by oncology due to a relapse evidenced in an MRI, the patient was transferred to the oncology unit.

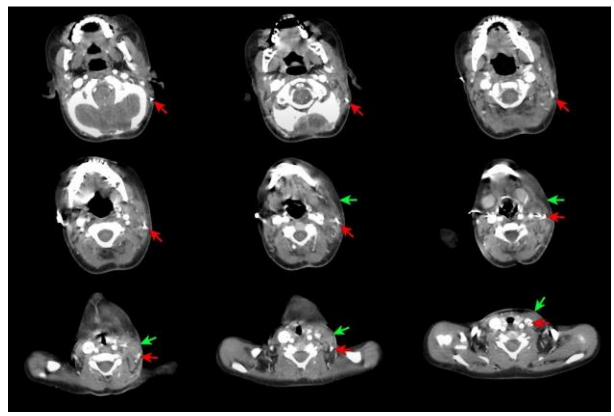

Figure 4.

Discussion

The etiology of GI tract perforation due to a VP shunt is not fully established; however, some risk factors have been described, including the use of abdominal trocars, rigid distal catheters, silicone allergies, and children's thin intestine walls (4, 19). Gmeiner et al. (2020) studied how GI perforation happens and divided it into two types: an acute and traumatic type that happens when the first distal catheter is placed, and a chronic type that is caused by irritative gastrointestinal distress. This latter type is the main cause of late cases of GI tract perforation (19). In addition, patient motion, peristalsis, and CSF pulsation may contribute to bowel wall perforation (19). In this case, our patient presented some of the risk factors described above: first, his age, a thinner intestinal wall, and patient motion. In addition, the relevant background of the resection of a cyst at the tip of the VP shunt catheter in combination with the above-mentioned risk factors may indicate the presence of an event of chronic-irritative gastrointestinal distress as the main cause of GI tract perforation.

The need for surgical treatment must be individualized. According to Alves et al. (2017), three principles are crucial to individualized surgical management: firstly, the implementation of external drainage of the proximal part until the CSF fluid reaches a biochemically near-normal state and becomes sterile on culture; secondly, the administration of intravenous antibiotics; and finally, the removal of the perforating section of the catheter (20). In cases where peritonitis or abdominal abscesses are absent, the removal of the abdominal end of the catheter can be accomplished using percutaneous or endoscopic methods without surgery. Surgical intervention is indicated when intra-abdominal infection occurs or when the fistulous tract fails to shut spontaneously following percutaneous or endoscopic removal (20). Laparotomy, laparoscopic, and transanal repair have been reported as methods for managing the colonic perforation (20). In the presence of severe peritonitis or with a relevant background of severe abdominal problems such as abdominal adhesions (10), laparotomy is mandatory (6). Our patient required surgical procedures. First, our patient was taken for VP shunt externalization and removal of the catheter that was perforating and was externalized via anal, and subsequently, he had a second surgical event, the placement of a new VP shunt.

Abdominal or systemic infections related to GI tract perforation can produce life-threatening conditions. Even though peritonitis was supposed to be the most common infectious outcome, less than 25% of patients present with peritonitis (12). However, other foci of infection can be found; for this reason, the majority of cases explain and address infection prevention and management (4, 9, 10, 13-16, 18, 19, 20, 21-23), some of them found a positive CSF culture and directed a specific antibiotic therapy (15, 16, 21, 22), and notice that in nearly 50% of GI tract perforations due to VP shunt CSF cultures are positive (16) and the most common bacteria isolated in any type of culture is *Escherichia coli* (12, 15, 16). According to Birbilis et al. (2009), mortality can reach up to 22% if a central nervous system (CNS) infection is confirmed due to meningitis, encephalitis, or brain abscesses (16); other authors have also reported these statistics (22, 24). In our case, fortunately, no peritonitis or other abdominal complications from the GI tract perforation were reported; however, despite initial negative blood and CSF cultures, the patient presented clinical symptoms and other laboratory tests suggestive of an infectious event, which made it necessary to switch the antimicrobial management several times, completing long treatment schedules. Finally, the last blood culture isolated *Staphylococcus epidermidis,* allowing the pediatric infectologist to direct specific treatment.

Conclusions

The bowel perforation due to a VP shunt is an extremely rare entity whose etiology is not fully established; however, it is known that it primarily affects children and may be mostly related to chronic-irritative gastrointestinal distress. The treatment required must be individualized, including management of the VP shunt, antimicrobial therapy to prevent or treat concomitant infectious diseases, and control of another wide range of life-threatening complications.

List of Abbreviations

VP: Ventriculoperitoneal
CSF: Cerebrospinal fluid
GI: Gastrointestinal
MRI: Magnetic resonance imaging
Hb: Hemoglobin
Hto: Hematocrit
MCV: Mean corpuscular volume.
MCH: Mean corpuscular hemoglobin.
CRP: C-Reactive protein
CT: Computerized tomography
CVC: Central venous catheter
CNS: Central nervous system

References

1. Fowler JB, Jesus OD, Mesfin FB. StatPearls. 2023. Available from: https://www.ncbi.nlm.nih.gov/books/NBK459351/#:~:text=Introduction,are%20used%20to%20treat%20hydrocephalus. Accessed 15 Aug 2023.
2. Zhou F, Chen G, Zhang J. Bowel Perforation Secondary to Ventriculoperitoneal Shunt: Case Report and Clinical Analysis. J Int Med Res. 2007;35: 926–929.
3. Khizar A, Shah SA, Zahid S, Yadav JK. Total intracranial migration of ventriculoperitoneal shunt: A case report with literature review. Roneuro. 2023; 37(2):224–228

4. Thiong'o GM, Luzzio C, Albright AL. Ventriculoperitoneal shunt perforations of the gastrointestinal tract. J Neurosurg Pediatr. 2015;16:36–41.
5. Ferreira PR, Bizzi JJ, Amantéa SL. Protrusion of ventriculoperitoneal shunt catheter through the anal orifice. A rare abdominal complication. J Pediatr Surg. 2005;40: 1509–1510.
6. Sathyanarayana S, Wylen EL, Baskaya MK, Nanda A. Spontaneous bowel perforation after ventriculoperitoneal shunt surgery: case report and a review of 45 cases. Surg Neurol. 2000;54:388–396.
7. Snow RB, Lavyne MH, Fraser RAR. Colonic perforation by ventriculoperitoneal shunts. Surg Neurol. 1986;25:173–177.
8. Sharma A, Shukla A, Iyengar SN. Rectal Migration of Ventriculo-Peritonial Shunt: A Rare Case Report. Roneuro. 2018;32(2):303–305.
9. Hai A, Rab A, Ghani I, Huda M, Quadir A. Perforation into gut by ventriculoperitoneal shunts: A report of two cases and review of the literature. J Indian Assoc Pediatr Surg. 2011;16:31.
10. Bakal Ü, Poyraz AK, Tartar T, Akdeniz İ, Sürme MB, Çelik S et al. A Rare Complication of Ventriculoperitoneal Shunt: Asymptomatic Small Bowel Perforation. Istanb Med J. 2019;20:571–573.
11. Wilson CB, Bertan V. Perforation of the bowel complicating peritoneal shunt for hydrocephalus. Report of two cases. Am Surg. 1966;32(9):601-3.
12. Liu Y, Li C, Tian Y. Ventriculo-peritoneal shunt trans-anal protrusion causing Escherichia coli ventriculitis in child: Case report and review of the literature. Chin Neurosurg J. 2017; 3.
13. Gedik AH, Uzuner S, Cindemir E, Bayraktar S, Torun E, Seyithanoglu H et al. Trans- anal protrusion of ventriculo-peritoneal shunt related to colon perforation: Two case reports. Turk Arch Pediatr. 2013;48: 255–258.
14. Bakshi S. Spontaneous trans-anal extrusion of caudally migrated ventriculo-peritoneal shunt tip in a child: a case report. Surgl Case Rep. 2020;6(1):50.
15. Jang HD, Kim MS, Lee NH, Kim SH. Anal Extrusion of Distal V-P Shunt Catheter after Double Perforation of Large Intestine.J Korean Neurosurg Soc.2007;42(3):232-234.
16. Birbilis T, Zezos P, Liratzopoulos N, Oikonomou A, Karanikas M, Kontogianidis K et al. Spontaneous bowel perforation complicating ventriculoperitoneal shunt: a case report. Cases J. 2009;2:8251.
17. Yavuz A, Bulduk E, Altiner S, Buyukkasap C, Dikmen K, Koksal H. Colonic Perforation due to Ventriculoperitoneal Shunt Catheter: A Case Report. Gazi Medical Journal 2019; 30:318–319.
18. Li H, Tan TC, Cheung FC.Transanal protrusion of ventriculoperitoneal shunt. Surg Pract. 2008;12:93–96.
19. Gmeiner M, Thomae W, Tolino M, Senker W, Gruber A. Bowel perforation after ventriculoperitoneal-shunt placement: case report and review of the literature. Open Medicine 2020; 15: 71–75.
20. Alves AR, Mendes S, Lopes S, Monteiro A, Perdigoto D, Amaro P et al. Endoscopic Management of Colonic Perforation due to Ventriculoperitoneal Shunt: Case Report and Literature Review. GE Port J Gastroenterol. 2017;24:232–236.
21. Ishizuka N, Komatsu E. Intestinal Perforation Caused by Lumboperitoneal Shunt Insertion Repaired with an Over-the-Scope Clip. Clin Endosc. 2022;55:146–149.
22. Kang HG, Cho KY, Mun JH, Lee LS. Colonic perforation as a complication of ventriculoperitoneal shunt: two case reports with a literature review. J Korean Ster and Func Neurosurg. 2022;18(1):37–41.
23. Scarascia A, Atallah E, Pineda MDA, Rosenwasser R, Judy K. Gastric perforation from a migrating ventriculoperitoneal shunt: A case report and review of literature. Radiol Case Rep. 2022;17:4899–4902.
24. Yousfi MM, Jackson NS, Abbas M, Zimmerman RS, Fleischer DE. Bowel perforation complicating ventriculoperitoneal shunt: Case report and review. Gastrointest Endosc. 2003;58:144–148.

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An exceptional presentation of pituitary apoplexy in thyrotoxicosis. A rare case report

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ABSTRACT

Pituitary apoplexy (PA) is a rare and potentially life-threatening condition characterized by severe headaches, vomiting, visual disturbances, ophthalmoplegia, altered mental status, and possible pan-hypopituitarism. A macroadenoma-induced pituitary haemorrhage is the primary cause. Various factors such as systemic hypertension, altered intracranial pressure, head trauma, anticoagulation, and pregnancy can trigger PA. A 35-year-old non-smoking male presented with headaches and an enlarged thyroid gland. Initial imaging revealed a haemorrhage in the sellar area, prompting a clinical diagnosis of pituitary apoplexy with T3 thyrotoxicosis. Despite the absence of visual impairment, conservative management was chosen, including anti-hypertensive therapy and follow-up MRI after three months. PA diagnosis can be challenging without prior identification of a pituitary tumour. Imaging modalities like MRI are crucial for diagnosis, and radiological follow-up is recommended. The relationship between PA and hyperthyroidism remains unclear, necessitating further investigation in patients presenting with PA symptoms.

Keywords
pituitary apoplexy, pituitary haemorrhage, hyperthyroidism, diagnosis, management

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INTRODUCTION

Pituitary apoplexy (PA) is a rare and potentially life-threatening condition characterized by acute hemorrhage or infarction of the pituitary gland, typically occurring in the setting of a pre-existing pituitary adenoma. It manifests clinically with sudden-onset severe headaches, vomiting, visual disturbances, ophthalmoplegia, altered mental status, and potential pituitary hormone deficiency [1,2]. The condition poses a diagnostic challenge due to its varied presentation and potential overlap with other intracranial pathologies. Various predisposing factors, such as systemic hypertension, altered intracranial pressure, head trauma, anticoagulation therapy, pregnancy, and hemodialysis, have been implicated in triggering PA [3]. Prompt recognition and management are essential to prevent potentially devastating complications such as pituitary hormone deficiency, visual impairment, and neurological sequelae.

CASE REPORT

A 35-year-old man presented to the neurosurgery department with headaches. He was a non-smoker. There was no evidence of ophthalmoplegia, although the thyroid gland was somewhat enlarged as a diffuse goitre on palpation. Hypopituitarism was not present. The patient had a 150/90 mmHg blood pressure, a heart rate of 90 beats per minute, a breathing rate of 18 breaths per minute, and a body temperature of 36.7 oC.Glasgow coma scale (GCS) was 15/15, and there were no additional cranial nerve impairments found. Computer tomography (CT) angiography revealed no evidence of an aneurysm. After ruling out traumatic brain damage and subarachnoid hemorrhage caused by an aneurysm, we were alerted to a suspicious lump in the sellar area of the head on CT. The pituitary gland seemed to be hemorrhaging (Fig 1).

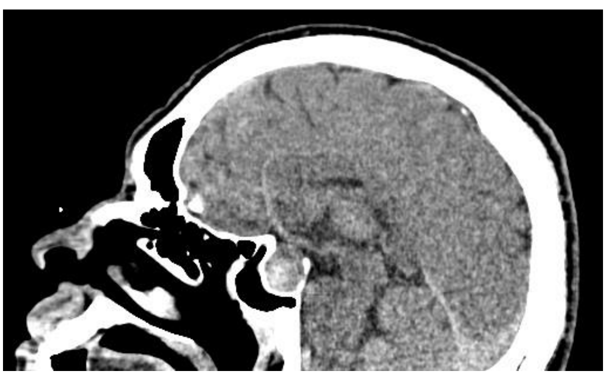

Figure 1. CT Head Saggital shows sign of hemorrhage in the pituitary.

Since the sellar area was of particular interest, we conducted an improved brain magnetic resonance imaging (MRI). Slightly hyperintense T1-weighted and hypointense T2-weighted images were seen in the sellar area of the brain, which indicated a bleeding in the lesion (Fig 2). Laboratory investigations revealed decreased thyroid stimulating hormone (TSH) levels (0.010 IU/mL) but increased free triiodothyronine (T3) (20.80 pg/mL) and thyroxine (T4) levels (> 6.00 ng/dL).

Therefore, a clinical diagnosis of pituitary apoplexy with T3 thyrotoxicosis was made. There were no signs of compression of the optic chiasm and no visual impairment, so surgical intervention was unnecessary. Given the absence of visual impairment or neurological deficits, conservative management was initiated, including anti-hypertensive therapy and close monitoring of hormone levels. Follow-up MRI imaging was scheduled after three months to assess for resolution of hemorrhage and monitor for any changes in pituitary morphology.

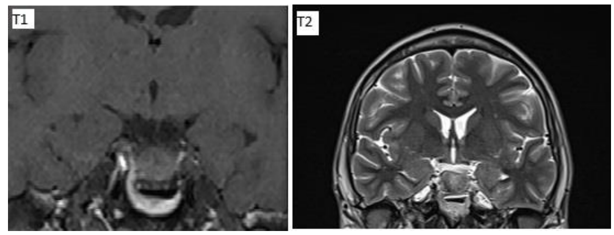

Figure 2. MRI of Brain T1 shows partially hyperintense and T2 shows partially hypointense.

DISCUSSION

Pearce Bailey published the first description of pituitary apoplexy (PA) in 1898 [4]. If a pituitary tumor hemorrhages or infarcates, it may cause pituitary apoplexy, an uncommon but potentially deadly illness [5].

An apoplexy diagnosis might be challenging if a pituitary tumor has not been previously identified. Postpartum hemorrhage and the use of anticoagulants, as well as other bleeding diseases, diabetes, concussions, and other injuries to the brain, are all linked to an increased risk of apoplexy. The most common risk factor was hypertension. Additionally, thyroid dysfunction has been well proven to affect cardiac output, contractility, blood pressure, vascular resistance, and rhythm problems [6,7]. Thus, in individuals with pituitary adenoma, it is theoretically plausible that primary thyroid failure induces pituitary apoplexy. However, the clinical relationship between pituitary apoplexy and thyroid dysfunction has remained ambiguous.

Hyperthyroidism, particularly T3 thyrotoxicosis, might be overlooked in pituitary apoplexy due to normal free T4 and reduced TSH levels. TSH suppression together with increased free T3 but normal free T4 is referred to as T3 thyrotoxicosis [8]. Acute neuro-ophthalmological degeneration with headache is a sign of pituitary apoplexy. The clinical presentation is variable. Fever and headaches are symptoms of infarcted tissue leaking into the subarachnoid space. Ophthalmoplegia may develop if the pituitary enlarges or bleeds into the cavernous sinus [9].

Cortisol deficit is a biological indication of pituitary insufficiency that should be explored. Aside from TSH, free thyroid hormones are required to diagnose secondary hypothyroidism. Imaging of the pituitary fossa, particularly CT, has low diagnostic sensitivity. So 'pituitary' MRI is the gold standard for detecting bleeding. It is critical to do radiological follow-up with an MRI in individuals who have PA. In pituitary apoplexy, fluid and electrolyte levels are monitored to prevent subsequent diabetes insipidus. In cases of reduced state of awareness, visual impairment, or hypothalamic disruption, immediate high dosage corticosteroid replacement and surgical decompression may be necessary [10].

The relationship between pituitary apoplexy and hyperthyroidism, particularly T3 thyrotoxicosis, remains unclear and warrants further investigation. Although hyperthyroidism has been implicated as a potential trigger for pituitary apoplexy, the underlying mechanisms and clinical implications remain poorly understood. Future research should aim to elucidate the pathophysiology of this relationship and identify potential therapeutic targets.

Conclusions

Pituitary apoplexy is a rare but potentially life-threatening condition that requires prompt recognition and management. Clinical presentation can vary widely, and imaging studies such as CT and MRI play a crucial role in confirming the diagnosis. Management is largely supportive and aims to stabilize the patient, control symptoms, and prevent complications. The relationship between pituitary apoplexy and hyperthyroidism remains unclear and warrants further investigation. Optimal management requires a multidisciplinary approach involving neurosurgery, endocrinology, and radiology, with close monitoring of hormone levels and imaging studies to assess for resolution of hemorrhage and monitor for any changes in pituitary morphology.

References

1. Wakai S, Fukushima T, Teramoto A, Sano K. Pituitary apoplexy: its incidence and clinical significance. J Neurosurg. 1981;55(2):187-193. doi:10.3171/jns.1981.55.2.0187
2. BROUGHAM M, HEUSNER AP, ADAMS RD. Acute degenerative changes in adenomas of the pituitary body--with special reference to pituitary apoplexy. J Neurosurg. 1950;7(5):421-439. doi:10.3171/jns.1950.7.5.0421
3. Reid RL, Quigley ME, Yen SS. Pituitary apoplexy. A review. Arch Neurol. 1985;42(7):712-719.
4. doi:10.1001/archneur.1985.04060070106028
5. Wang AR, Gill JR. The Pituitary Gland: An Infrequent but Multifaceted Contributor to Death. Acad Forensic Pathol. 2016;6(2):206-216. doi:10.23907/2016.023
6. Bills DC, Meyer FB, Laws ER Jr, et al. A retrospective analysis of pituitary apoplexy. Neurosurgery. 1993;33(4):602-609.
7. doi:10.1227/00006123-199310000-00007
8. Klein I, Danzi S. Thyroid disease and the heart [published correction appears in Circulation. 2008 Jan 22;117(3):e18]. Circulation. 2007;116(15):1725-1735.
9. doi:10.1161/CIRCULATIONAHA.106.678326
10. Randeva HS, Schoebel J, Byrne J, Esiri M, Adams CB, Wass JA. Classical pituitary apoplexy: clinical features, management and outcome. Clin Endocrinol (Oxf). 1999;51(2):181-188.
11. doi:10.1046/j.1365-2265.1999.00754.x
12. Figge J, Leinung M, Goodman AD, et al. The clinical evaluation of patients with subclinical hyperthyroidism and free triiodothyronine (free T3) toxicosis. Am J Med. 1994;96(3):229-234. doi:10.1016/0002-9343(94)90147-3
13. Semple PL, Webb MK, de Villiers JC, Laws ER Jr. Pituitary apoplexy. Neurosurgery. 2005;56(1):65-73.
14. doi:10.1227/01.neu.0000144840.55247.38
15. Verrees M, Arafah BM, Selman WR. Pituitary tumor apoplexy: characteristics, treatment, and outcomes. Neurosurg Focus. 2004;16(4):E6. Published 2004 Apr 15. doi:10.3171/foc.2004.16.4.7.

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Neuroimaging findings from a case of neurocysticercosis. Case report

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ABSTRACT

Introduction: Human cysticercosis occurs when humans ingest *Taenia solium* eggs from the fecal-oral route. The infection in the central nervous system is called neurocysticercosis and is considered the principal cause of late-onset epilepsy in endemic countries in Latin America, Asia, and Africa.

Case report: 71-year-old female in a state of neglect with a medical history of untreated rheumatoid arthritis. She presented her first seizure episode. Cerebral magnetic resonance imaging with contrast reported parenchymal and subarachnoid neurocysticercosis in different stages.

Discussion: The high incidence of neurocysticercosis in endemic countries is associated with poverty conditions such as less hygienic conditions and permanent contact with domestic animals. The four stages of classification of neurocysticercosis is useful for pathologists and radiologists to identify the life cycle of the parasite within the body and the specific imaging findings of each phase. After diagnosis, treatment includes the use of niclosamide or praziquantel.

Conclusions: neurocysticercosis is a public health problem in endemic countries that requires further attention. Clinical manifestations are variable and neuroimaging findings are essential to making a correct diagnosis.

Keywords
Neurocysticercosis,
Seizures,
magnetic resonance imaging,
treatment

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INTRODUCTION

Human cysticercosis occurs when humans ingest Taenia solium eggs from the fecal-oral route, harbouring the parasite in the intestine. Later, the eggs evolve into oncospheres, which are carried to other tissues, including the central nervous system (CNS) and meninges, where they evolve into larval forms, or cysticerci. (1) The infection in the CNS is called neurocysticercosis (NCC) (1) and is considered the principal cause of late-onset epilepsy in endemic countries in Latin America, Asia, and Africa. (2,3)

Clinical manifestations depend on the number of lesions, size, stage, and location; some of the most common include seizures in up to 70–90% of patients with NCC, hydrocephalus, which can cause headaches, vertigo, loss of consciousness, and an increase in intracranial hypertension due to CSF obstruction. (2) Less often, symptoms include cognitive and psychiatric impairment. (4) Neuroimaging is the cornerstone of diagnosis; in addition, a CSF test would be useful to detect even antibodies/antigen to Taenia solium. (2) Magnetic resonance imaging (MRI) is especially useful to identify four of the four types classified by Escobar of NCC: the vesicular, colloidal vesicular, granular nodular, and calcified nodular stages, with the non-cystic stage detectable only by laboratory tests. (5, 6) We present the case of a 71-year-old female with findings compatible with NCC.

CASE REPORT

A 71-year-old female in a state of neglect with a medical history of untreated rheumatoid arthritis presented to the emergency room with a tonic-clonic seizure with sphincter relaxation of 20 minutes duration. She was treated in a first-level hospital with diazepam and phenytoin with modulation of the crisis and was subsequently transferred to a hospital of greater complexity.

On presentation, she was awake, disoriented in time, with elevated blood pressure (BP: 160/95 mmHg), and all other vital signs were normal (heart rate: 75 bpm, breath rate: 18 bpm, temperature: 36.9 °C, oxygen saturation: 98%). Laboratories reported a complete blood count with anemia (hemoglobin 11.6 g/dL, NR: 13.2 to 16.6 g/dL, hematocrit: 36%, NR: 38.3% to 48.6%), normal leukocytes (7.700 mm3, NR: 4.000–10.000 mm3), platelets in lower limit (156.000 mm3, NR: 150.000–400.000 mm3), antibody to human immunodeficiency virus negative, and syphilis antibodies non-reactive.

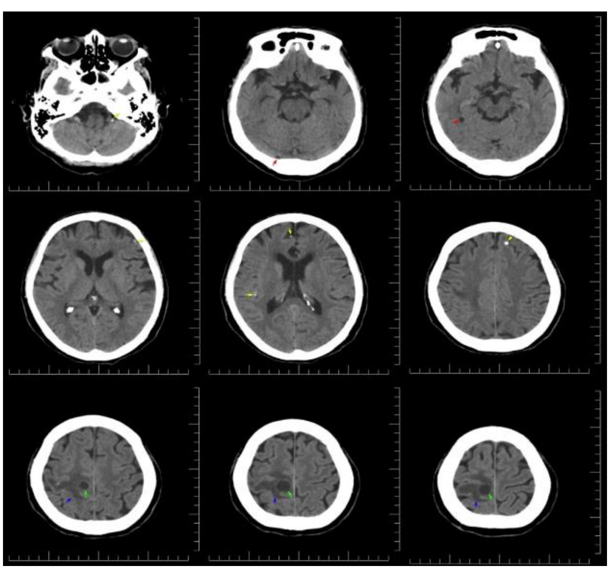

Figure 1. Cranial CT scan showing nonspecific punctate left occipital, bilateral frontal, and right parietal calcifications (yellow arrows), hypodense right temporal intra-axial lesion (red arrows). Right parietal intra-axial lesion rounded, hypodense with liquid density (6.5 HU), hyperdense wall measuring 13x15 mm (green arrows), and vasogenic edema (blue arrows).

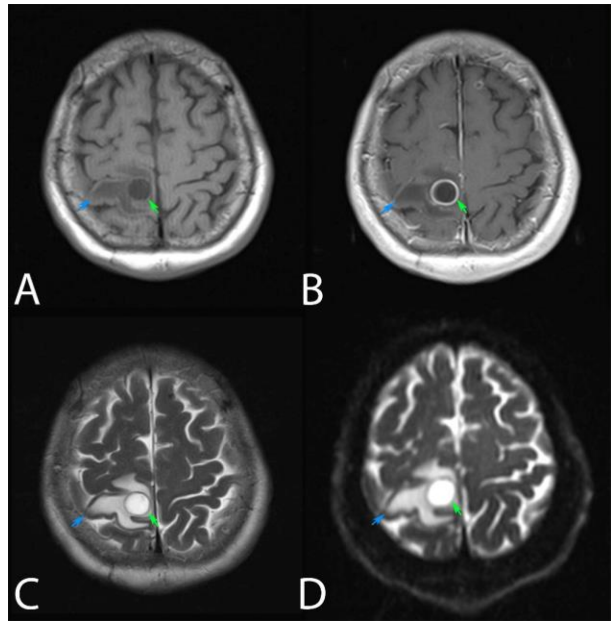

Figure 2. Brain MRI with contrast showing focal cortico-subcortical lesion in the right parietal region (green arrows) (approximately 20x16 mm) with discretely heterogeneous internal signal, predominantly hypointense in T1 (**A**), hyperintense in T2 (**C**), and diffusion-weighted (**D**), identifying iso-intense eccentric focus in all sequences and capsular-pseudocapsular margin less than 2 mm thick. Capsular-septal and eccentric focus enhancement, especially in postcontrast sequences (B), associated perilesional vasogenic edema conditioning discrete local mass effect with effacement of adjacent cortical sulci (blue arrows).

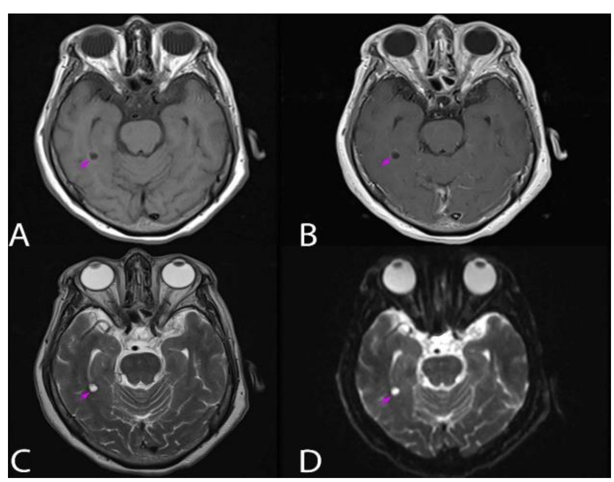

Figure 3. Brain MRI with contrast showing focal cortico-subcortical lesion in the right temporo-basal region (purple

arrow) with hypointense signal in T1 (**A**), hyperintense in T2 (**C**), and diffusion-weighted (**D**) (approximately 7 mm in diameter) with small isointense eccentric focus in all sequences and a thin capsular-pseudocapsular margin less than 2 mm thick. Fine capsular/pseudocapsular and eccentric focus enhancement, especially in postcontrast sequences (B), without evidence of adjacent vasogenic edema.

During a cranial computed tomography (CT) scan, a round and hypodense lesion in the right parietal lobe of 13x15 mm in diameter was reported with vasogenic edema and nonspecific punctate calcifications on the left frontal and right parietal areas (Figure 1). Cerebral magnetic resonance image (MRI) with contrast reported parenchymal and subarachnoid neurocysticercosis in different stadiums: right parietal intra-axial lesion in the vesicular colloid stage and perilesional vasogenic edema (Figure 2); lesions in the vesicular stage in the right temporal intra-axial lobe (Figure 3) and extra-axial frontal bilateral lobe, temporary right and pericallosal; intra-axial lesions in the nodular-calcified stage left frontal, right parietal, and right temporal lobes.

The patient started treatment with albendazole 600 mg PO diary for 10 days, praziquantel 2.000 mg PO every day per 10 days, dexamethasone 4 mg IV every 8 hours per two days, dalteparin 2.500 U SC every day, and antiseizure medications with levetiracetam 500 mg PO every 12 hours. Because of the national shortage of praziquantel, the patient could not continue treatment; furthermore, on day nine, she was evaluated by an infectologist, who recommended continued management with albendazole and dexamethasone for 30 days due to subarachnoid compromise. The patient completed the new recommended therapeutic regimen and was discharged to continue follow-up by a specialist.

Discussion

The high incidence of NCC in endemic countries (predominantly low- to middle-income countries) is associated with poverty conditions such as less hygienic conditions and permanent contact with domestic animals. The rise of cases associated with poor scientific production leads to the abiding of infection. (7,8) Our case is not far from this reality, since the patient lives in conditions of poverty and neglect in an endemic country.

Even before Escobar described the four stages of NCC, Carpio in 1994 proposed a classification of NCC according to the viability and location of the parasite. (8, 9) This classification is also important to identify the parasite activity, being stage active when the parasite is alive, transitional if the parasite is in the degenerative phase, and inactive if the parasite has been eradicated. (9) This classification can be useful for clinicians, just as the Escobar classification is useful for pathologists and radiologists to identify the life cycle of the parasite within the body and the specific imaging findings of each phase. At the vesicular stage, in computed tomography (CT) and MRI, the cyst fluid has similar characteristics to CSF; the vesicular wall measures up to 4 mm and the lesion up to 20 mm. Notice that the scolex is eccentrically within the lesion; it can be iso or hyperintense in both T1 and T2 weighted sequences and hyperdense on CT.

In addition, at this stage, there is no calcification or perilesional oedema. (5, 6) In the next stage, the colloidal vesicular, the cyst fluid becomes turbid due to an inflammatory response at the brain parenchyma after the larval degeneration from the scolex, is hyperdense on CT in comparison with the CSF, and the cyst wall is irregular and thicker. (9) The patient presented multiple small lesions at the vesicular stage in subcortical and deep supratentorial bihemispheric white matter, thalami, lenticular nuclei, internal capsules, midbrain, pons, and in both cerebellar hemispheres, and one colloidal vesicular lesion at the right parietal lobe with surrounding edema.

The cyst gets smaller in the granular stage, turning into a granulomatous nodular lesion. There is often gliosis around the lesion, and there is some edema but not as much as in the previous stage. Finally, the lesion is about a quarter of its original size in the nodular calcified stage, which is a nonactive stage of the disease. There is no oedema around the lesion. (9) In this scenario, CT imaging is better suited to identify this lesion than the MRI because it can clearly depict the calcified nodule; however, in the MRI, it is possible to identify mild contrast enhancement surrounding the calcification, and seizure activity could be present even knowing that this is the non-active stage of disease. (6) In our case, the patient presented lesions compatible with granular and calcified stages at the left parietal and frontal lobes, easily identifiable on CT.

After confirming the diagnosis, treatment includes the use of niclosamide or praziquantel, the latter being more effective. (11) Unfortunately, our patient could not receive Praziquantel treatment due to a supply shortage and was treated with a less effective alternative regimen while waiting to resupply. (11)

Conclusion

NCC is a public health problem in endemic countries that requires further attention. Clinical manifestations are variable; however, the disease may be suspected in cases of late-onset epilepsy. Knowledge of different parasite stages and neuroimaging findings is essential to making a correct diagnosis.

References

1. Del Brutto OH. Human Neurocysticercosis: An Overview. Pathogens. 2022;11:1212.
2. Takayanagui OM, Haes TM. Update on the diagnosis and management of neurocysticercosis. Arq Neuropsiquiatr. 2022;80:296–306.
3. Balderrama J, Arevalo DET, Calderon-Mirada WG, Joaquim AF, Pacheco-Hernandez A, Agrawal A, et al. Intradural spinal neurocysticercosis: case illustration. Romanian Neurosurgery. 2017;31:530–535.
4. Rodrigues CL, de Andrade DC, Livramento JA, Machado LR, Abraham R, Massaroppe L, et al. Spectrum of cognitive impairment in neurocysticercosis: Differences according to disease phase. Neurology. 2012;78:861–866.
5. Venkat B, Aggarwal N, Makhaik S, Sood R. A comprehensive review of imaging findings in human cysticercosis. Jpn J Radiol. 2016;34:241–257.
6. Zhao JL, Lerner A, Shu Z, Gao XJ, Zee CS. Imaging spectrum of neurocysticercosis. Radiol Infect Dis. 2015;1:94–102.
7. Carpio A, Lorenzo N. Neuroimaging in Neurocysticercosis. [accessed on October 17, 2023]. In: Medscape. Available at: https://emedicine.medscape.com/article/1168784-overview?form=fpf&scode=msp&st=fpf&socialSite=google&icd=login_success_gg_match_fpf&isSocialFTC=true
8. Carpio A, Placencia M, Santillán F, Escobar A. A Proposal for Classification of Neurocysticercosis. Can J Neurol Sci. 1994;21:43–47.
9. Gonzalez-Alcaide G, Sosa N, Shevy L, Belinchon-Romero I, Ramos-Rincon J-M. Global research on cysticercosis and neurocysticercosis: A bibliometric analysis. Front Vet Sci. 2023; 10:1156834.
10. Rodríguez-Rivas R, Flisser A, Norcia LF, Hamamoto Filho PT, Bonilla-Aldana DK, Rodriguez-Morales AJ, et al. Neurocysticercosis in Latin America: Current epidemiological situation based on official statistics from four countries. PLoS Negl Trop Dis. 2022;16:e0010652.
11. Zammarchi L, Bonati M, Strohmeyer M, Albonico M, Requena-Méndez A, Bisoffi Z, et al. Screening, diagnosis and management of human cysticercosis and Taenia solium taeniasis: technical recommendations by the COHEMI project study group. Trop Med Int Health. 2017; 22:881–894.

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Spinal arachnoid cysts. A case series and review of literature with subgroup comparison of intradural and extradural arachnoid cysts

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ABSTRACT

Objectives: Spinal arachnoid cysts (SAC) are rare lesions. There are many uncertainties regarding details about Intradural and Extradural SAC. We present a series of 12 cases of SAC along with a comprehensive review of the literature. In this review, we discussed the differences between Intradural and Extradural SAC pertaining to demography, pathogenesis, surgical procedures and outcomes.

Methods: We retrospectively collected the data of 12 patients of SAC treated at our Institute from 2012 to 2023. The age, gender, clinical, radiological, surgical data and outcome were noted. An extensive review of the literature was done to analyse and note the differences between Intradural and extradural SAC and their surgical management.

Results: In our series of 12 patients, 7 had Extradural SAC(58.3%), 3 had Intradural SAC(25%) and 2 had Intramedullary SAC(16.7%). . Of the 12 patients, 4 were males and 8 were females (1:2). Age range was 9 to 64 years and the mean age was 34.42 ± 17.71 years. There were four paediatric patients in the series. The most common symptoms reported at presentation are weakness of limbs(81.9%),back pain(36.4%), sensory symptoms(36.4%), radicular pain (18.2%), and bladder disturbances(9.1%). Out of the 12 patients, surgery was done in 11 patients and one patient was managed conservatively.

Conclusions: From the review of the literature, intradural SAC was twice as common as extradural SAC. Ventrally located SACs are more common in Intradural locations as compared to Extradural. The intradural SACs were more commonly located in the thoracic and cervical regions when compared to extradural SACs which were located commonly in the thoracolumbar, thoracic and lumbar regions. Extradural SACs were mostly Primary and the mainstay of treatment is complete or partial excision of the cyst with identification and ligation of the communicating pedicle. Selective laminectomy, partial excision of the cyst and wide fenestration to sustain CSF flow is the recommended treatment in Intradural SAC.

Keywords
spinal arachnoid, cysts, intradural, extradural

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INTRODUCTION

Spinal Arachnoid cysts (SAC) are rare lesions which can cause myelopathy or radiculopathy.[1,2]Spinal arachnoid cysts were classified into three major types by Nabors et al.: Extradural cysts without spinal nerve root fibers (Type I), Extradural cysts with spinal nerve root fibers (Type II), and Intradural cysts (Type III).[3] Type I was further subdivided into two groups: extradural arachnoid cysts (type Ia) and sacral meningocele (type Ib).[3] We describe here a series of 12 Spinal arachnoid cysts treated at our Institute between 2012 to 2023.

There were many uncertainities regarding details about intradural and extradural SAC. There was a difference in the opinion of authors regarding the incidence of intradural vs extradural SAC. Some authors claimed that intradural SAC[4] were common whereas others claimed extradural SACs were more common.[5] We wanted to clarify this ambiguity along with some other queries regarding SAC. We have done a comprehensive review of literature to find out the demographic, clinical and surgical details and differences between Intradural and Extradural arachnoid cysts.

Methods

We retrospectively collected the data of patients treated for Spinal arachnoid cysts at our Institute between the years 2012 to 2023. All the demographic, clinical, radiological, surgical data and outcome was collected and analysed. The surgical technique and outcome were described in detail.

Comprehensive review of literature and detailed subgroup analysis of Intradural and Extradural SAC's was done.

We have defined Arachnoid cysts as Primary when there is no identifiable cause(Idiopathic). Secondary arachnoid cysts are defined as cysts secondary to inflammatory reactions related to trauma, subarachnoid haemorrhage, meningitis, post intradural surgery, after CT myelography or post lumbar puncture or epidural anaesthesia in few instances.[6]

Outcome analysis

Odom's criteria were used for evaluation of the surgical results and outcome: Excellent (no pain and no neurological deficit); Good (occasional, mild pain and no neurological deficit and no change in work status); Fair (frequent, persistent pain and slightly improved neurological deficit and some change in work status); Poor (disabling pain and unchanged neurological deficit and unable to work).

Statistics

IBM spss25 software was used for statistical analysis.

Our case series

We describe the results of our series of 12 patients of SAC.

Demography and clinical features

Twelve patients of Spinal arachnoid cysts were treated at our centre during the study period. Details of all the patients are described in **Table 1**. Of the 12 patients, 4 were males and 8 were females (1:2). Age range was 9 to 64 years and mean age was 34.42 ± 17.71years. There were four paediatric patients in the series. The most common symptoms reported at presentation are weakness of limbs(81.9%),back pain(36.4%), sensory symptoms(36.4%), radicular pain(18.2%), and bladder disturbances(9.1%). Signs of myelopathy are present in 7 out of 12(58.3%) patients. The duration of symptoms was ranging from 2 days to 6 months.

Imaging features

All the patients underwent MRI spine for diagnosis. Out of the 12 patients, 7 had Extradural SAC(58.3%), 3 had Intradural SAC(25%) and 2 had Intramedullary SAC(16.7%).

Location of the Arachnoid cysts was thoracic in 5(45.5%) patients, cervical in 3 (27.3%) patients, thoracolumbar in 2(18.2%) patients and lumbar in 1(9.1%) patient. All the Extradural SAC were dorsal to the spinal cord/ thecal sac. Both the Intradural SACs were ventral to the cord. In one patient (Case 2) there were associated cervical degenerative disc disease and cord atrophy with myelomalacia changes. CT myelography was done in one patient but we could not delineate the level of communication.

Surgical procedure

Out of the 12 patients, surgery was done in 11 patients and one patient with D12-L2extradural arachnoid cyst (case 10) was managed conservatively.

Laminectomy and excision of the cyst was done in all the six patients with Extradural cysts. Total excision of an extradural SAC(case1) was demonstrated in Figure 1. Intradural communication was identified in 5 patients which was ligated and closed. The site of intradural communication was the junction of thecal sac and dural sleeve of nerve root in 4 patients and posterior midline of the thecal sac in 1 patient.

In two patients with Intradural cysts, Laminectomy, fenestration and partial excision of the cyst wall was done. In the third patient (case 12) with intradural cyst, complete excision of the cyst was done. Preoperative MRI images of an intradural SAC(case 9) were shown in Figure 2.

In one patient with intramedullary cyst, myelotomy and partial excision of cyst wall was done where as in other subpial intramedullary cyst, laminectomy and fenestration of cyst was done (Figure 3).

Table 1. Our case series of 12 cases of SAC

S.no	Age/	Symptoms	Duration	Spinal level (no. of	Horizontal level (Dorsal/ ventral)	Surgery	Complications	Outcome	Follow up
1	14 /M	Spastic paraparesis	45 days	D8-L1 (6)	Extradural (Dorsal)	D8-D12 Laminectomy and complete excision -Communication seen and closed	NIL	Improved in power Excellent	-6years -No recurrence
2	43 /F	-Band like sensation in lower limbs - Weakness of both upper and lower limbs, difficulty in walking	2 mts. 2 mts	C3-C6 (4)	Extradural (Dorsal)	C3-C6 Laminectomy and complete excision -Communication seen and closed	Patient deteriorated and developed Right hemiparesis (Power-2/5)	-Patient improved to 4-/5 power in 3 months Poor	- 3years& 6 months -No recurrence
3	47 /F	-LBA Left L1,L2 radicular pain -Left LL paraesthesia	15 days	D10-L2 (5)	Extradural (Dorsal)	D11-L1 Laminectomy and complete excision -Communication at L1 closed	NIL	-Pain relieved Excellent	-3 years& 6 months -No recurrence
4	44 /F	Right LL weakness, difficulty in walking -Paraesthesia both LL's k/c/o Pulmonary TB & spinal TB arachnoiditis on ATT since 6 months	3 mts	D9-D11 (3)	Intradural extramedullary (Ventral)	D9-D11 Laminectomy, Partial excision of cyst wall	NIL	Improved in power Fair	-3years& 4 months -No recurrence
5	50 /F	LBA Weakness of both LL's	6 mts 2 mts	D9D10 (2)	Intramedullary	D8-D10 Laminectomy, myelotomy, partial excision of cyst wall	NIL	-Improved in power Good	-3 years -No recurrence
6	64 /F	Upper back pain	10 yrs.	D2-D4 (3)	Extradural (Dorsal)	D2- D4 Laminectomy and complete excision	NIL	Improved in power	-8 years& 2 months

		Numbness both LL's Difficulty in walking, Urinary urgency	6 mts			-communication not seen		Good	-No recurrence
7	9/F	Paraplegia	2 days	D3-D6 (4)	Extradural (Dorsal)	D3- D6 Laminectomy and complete excision - Communication seen and closed	NIL	-No improvement of power Poor	-9 years& 6 months -No recurrence
8	16/M	Spastic paraparesis	3 wks.	D4-D6 (3)	Extradural (Dorsal)	D4- D6 Laminectomy and complete excision - Communication seen and closed	NIL	-Improved in power Excellent	-10months No recurrence
9	30/F	Spastic quadriparesis, Right upper limb radiculopathy	10 days	C1-C3 (3)	Intradural extramedullary (ventral)	Laminectomy, subtotal excision of cyst wall& communication to subarachnoid space	NIL	Improved in power Excellent	-9 months -No recurrence
10	53/F	LBA	1 mt.	D12-L2 (3)	Extradural (Dorsal)	Conservative	-	-LBA decreased -Repeat MRI showed no increase in size of lesion	3 years - Noprogression ofsymptoms
11	11/M	quadriparesis	2 mt.	C6C7 (2)	Intramedullary	C6, C7 Laminectomy and fenestration	NIL	-Power improved Good	-10 years& 6 months -No recurrence
12	32/M	Back pain, difficulty in walking	1 year	D6D7 (2)	Intradural extramedullary (dorsal)	D6 -D8 laminectomy andcompleteexcision of cyst	NIL	-Back pain improved Excellent	- 9 months - No recurrence

Complications and outcome

Outcome was excellent in 5 patients, good in 3 patients and fair in 1 patient. Poor outcome was noted in2 patients. One patient (case 2) developed right hemiparesis postoperatively(grade 2/5).This patient gradually improved in power to preoperative status(4-/5) on follow up over 3 months period. The other patient (case 7) with poor outcome presented with paraplegia did not improve after surgery. There was no CSF leak or wound infections in the series.

Follow up

The average follow up of our patients was 52 months. There was no reported recurrence or onset of new symptoms till the last follow up.

Discussion

Spinal arachnoid cysts are rare and may account for only about 1-3 % of Primary spinal lesions.[1,2]Spinal arachnoid cysts were described as Spinal Meningeal cysts by Nabors et al. and were classified into three types.

In our literature review of large case series [5-13] of Spinal arachnoid cysts it was evident that Intradural arachnoid cysts are two times commoner than Extradural arachnoid cysts and Intramedullary arachnoid cysts are very rare.[14]

Ventrally located cysts in relation to the spinal cord were more common in Intradural arachnoid cysts. Ventral location of cysts is seen in about 15-20% of all Intradural arachnoid cysts.[8,7,12,15]

Extradural ventral cysts are very rare.[5,11,12,16,17] Similar findings were seen in our present series.

Intradural SACs were more commonly seen in the Thoracic, cervical and cervicothoracic and thoracic levels[9,6,7,18] whereas extradural SACs were commonly seen in the Thoracolumbar, Thoracic, lumbar and lumbosacral levels. [11,16,17]

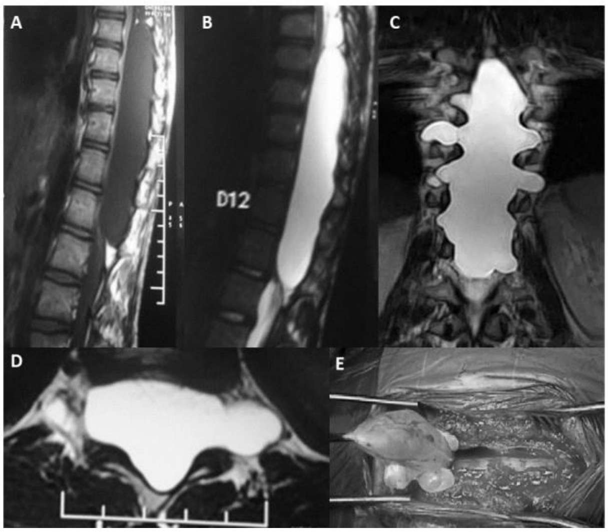

Figure 1. Extradural arachnoid cyst (case 1) extending from D8-L1. Sagittal T1 &T2 weighted (**A, B**) and coronal, axial T2W MRI images (**C, D**) demonstrating extension into neural foramina. Intra operative image(E) showing arachnoid cyst being excised and easily seperable from underlying dura.

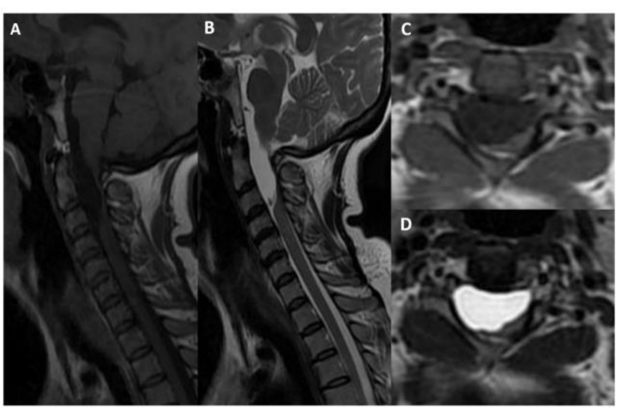

Figure 2. Sagittal T1 and T2 weighted(A,B) and axial T1 and T2W(C,D) MRI images demonstrating an Intradural arachnoid cyst (case 9) at C1 to C3 level with marked thinning of the cord.

Pathogenesis

The origin of Extradural arachnoid cysts in most cases is due to herniation of arachnoid through a congenital dural defect or could be a congenital diverticulum of the dura.[19]The dural defect is located most often at the junction of thecal sac and dural sleeve of the root or in the region of the dural sleeve of the root and less commonly dorsal midline of the thecal sac.[19]Extradural secondary arachnoid cysts are very rare and causes are post traumatic or post surgical dural defects.[20]Various theories were proposed for expansion of Extradural cyst and one suggested theory is ball-valve mechanism in the communicating pedicle associated with pulsatile CSF dynamics and one way CSF flow which leads to cyst expansion.[21]Other proposed mechanisms for cyst expansion are active secretion by cyst wall, Osmosis of water into cyst due to hyperosmolar cyst content.[1]

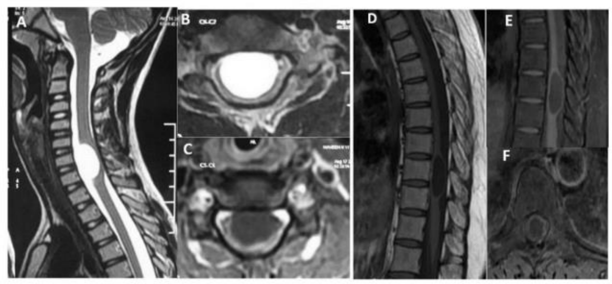

Figure 3. Sagittal and axial T2 weighted(A,B) and Axial T1 weighted (C) images of Case 11 demonstrating a Subpial intramedullary arachnoid cyst.
T1 weighted sagittal(D), Sagittal T1 post contrast(E), Axial T1 post contrast(F) images of Case 5 showing a D9,D10 intramedullary arachnoid cyst with expansion of cord.

The pathogenesis of Primary(congenital) Intradural arachnoid cysts includes various theories and Perret et al.[22] proposed that they arise from or within the septum posticum. Septum posticum is a midline membrane dividing the posterior cervical and thoracic subarachnoid space longitudinally. In our analysis most of the Intradural arachnoid cysts are seen in the thoracic and cervical location. But this theory does not explain the origin of ventral cysts. Another theory proposes that congenital arachnoid cysts originate from deviations in arachnoid trabeculations during the embryonic period.[45]The etiology of SecondaryIntradural arachnoid cysts could be post traumatic, SAH, post surgical, lumbar puncture and infective causes. [6,7,18]

Secondary arachnoid cysts are most commonly Intradural and Extradural secondary arachnoid cysts are very rare. Klekamp et al.[6] described 130 cases of Intradural SAC's out of which 109 were primary and 21 were secondary. They have highlighted certain differences between Primary and Secondary Arachnoid cysts. Primary arachnoid cysts were mostly seen in the thoracic level (94%) whereas

Secondary Arachnoid cysts were seen in the thoracic spine, the cervicothoracic, or the thoracolumbar junction. Secondary Arachnoid cysts were located in a more lateral or even ventral in relation to the spinal cord but only one Primary Arachnoid cyst was located ventrally. They also observed that progression-free survival for 10 years following surgery was 83% for primary compared to 15% for secondary arachnoid cysts. Associated syrinx was more common in Secondary arachnoid cysts. 18 patients with secondary arachnoid cysts (86%) showed a syrinx and 50 patients with primary (46%) demonstrated a syrinx. Baig Mirza et al.[9] also described in their series that a syrinx being present in 5 out of their 11 secondary SAC cases.

Radiology

MRI is the diagnostic imaging of choice as it can delineate exact location of the cyst (extra or intradural), extent and relationship to the cord (dorsal vs ventral)[24]. Arachnoid cysts are similar to that of CSF signal intensity on T1 and T2 weighted sequences.

Extradural cysts show epidural fat capping of the lesion at the superior and inferior poles. The lesions cause spinal cord or thecal sac compression and vertebral body scalloping, expansion of the neural foramina bilaterally, thinning of pedicles which suggest longstanding mass effect from the cyst.[25]

Neo et al.[26] have used Cine-MRI sequence for successful identification of the communication site(dural defect) which shows a pulsating turbulent flow void in an Extradural arachnoid cyst. Funao et al[17] have used Myelography, CT myelography (CTM), and cine MRI to delineate the communication site between the spinal subarachnoid space and the arachnoid cyst cavity. Pooling of the contrast medium at the early phase in Myelography and CT myelography revealed the communication site between the spinal subarachnoid space and the cyst in 7 out of 12 cases in their series. Whereas Cine MRI revealed the communication site in 2 of these 7 patients.

Morizaneetal.[27]have done CT Myelography(CTM) and Cine MRI to detect the communication site in 6 of their 12 Exradural SAC's. They also suggested to do CTM first and to do cine MRI if the communication site could not be detected by CTM.

Nakagawa et al.[28] have used 3D constructive interference in steady state MRI (3D CISS MRI) for visualizing the communication site.

MRI with CISS-3D sequences is also very precise in detection of intracystic septae, trabeculae, and intradural cystoid formations as compared to T2W imaging.[29] The differential diagnosis of Intradural cysts includes neurenteric cysts, neuroepithelial cysts, epidermoid cysts, dermoid cysts, cystic schwannomas, and parasitic cysts.[6]

Surgery

In Extradural arachnoid cysts, the mainstay of management is complete excision of the cyst with closure of dural defect and obliteration of communicating pedicle.[2,3,10] In case of dense adhesions of cyst wall with dura, partial or subtotal removal of the cyst wall, with ligation of the communicating pedicle is recommended.[3,10]Selective laminectomy, incision of the cyst wall and closure of the dural communication has been done by few authors with good neurological outcomes and cyst resolution.

Selective laminectomy or hemilaminectomy, incision of the outer cyst wall and closure of the dural defect microscopically was done by Xu et al.[16] in their series of 10 patients. There was shrinkage or even complete disappearance of residual cysts in this series with an average follow up of 13.2 months.They have not identified any recurrence of cyst with this technique.

Funao et al[17] reported 12 cases of Extradural SAC. Laminectomy and total resection of the cyst was done in 7 patients and closure of the dural defect without cyst resection was done in 5 patients. In the later group, preoperative identification of the communication site was done by CT myelogram and Cine MRI . They performed a selective laminectomy at that level, opening of the dorsal cyst wall, and closure of dural defect with the aid of a flexible neuroendoscope. They observed no difference in functional recovery between the two groups but the mean postoperative kyphotic angle was more (statistically significant) in the patients treated by total resection of the cyst.

Morizane et al.[27]described 12 patients of Extradural SAC out of which 6 patients underwent massive laminectomy along with total resection of the cysts. In the remaining 6 patients in whom dural defects could be detected preoperatively, partial laminectomy and closure of the dural defects without resection of the cysts was done. Selective laminectomy or hemilaminectomy with closure of dural defect is recommended as compared to massive laminectomy and total cyst excision in view of similar results, reduced operative time, less bleeding, less muscle damage, and less incidence of postoperative kyphosis.

If the communication site is not detectable preoperatively, they advocated selective laminectomy and closure of the defect at the mid-level vertebrae affected by the cyst or at the T12-L1 level in patients with a cyst at the thoracolumbar level.

Hatashita et al[30] proposed that closure of the communication site is not important if total removal of the cyst could be done. Hence total Excision of the cyst can be done in cases where communication site cannot be detected either by preoperative imaging or intraoperatively.

The goals of surgery in Intradural SAC are decompression of thecord, establishing sustained flow of CSF and preventing postoperative adhesions[6]. Selective laminectomy, partial excision of cyst and wide fenestration to sustain CSF flow is the recommended treatment in Intradural SAC.[6,8] Klekamp et al.[6] and Schmutzer et al.[8]recommended resection of upper pole or upper half of the cyst with fenestration. Partial excision or fenestration was also recommended in ventrally located cysts or adherent cysts.[3,13]Duraplasty with graft was recommended by few authors[6,7] to prevent post operative adhesions and also achieve the goal of unobstructed CSF flow.

Complete excision of the Intradural SAC was recommended by few authors to prevent recurrence.[3,7]Complete excision of the Intradural SAC can be done wherever possible in case of short segment cysts extending not more than 3 vertebral levels.[6]

Schmutzer et al.[8]observed that there was no significant difference in terms of size reduction of the cyst or clinical outcome between the two groups of total cyst excision vs partial excision and fenestration. Cystoperitoneal shunt was a treatment option in few cases of recurrent intradural cysts.[8,13]

Endo T et al[18] have done Endoscopic assisted surgery in 6 of their 11 cases of Intradural SAC's. In all these six patients, 2 to 3 level hemilaminectomy or laminectomy and microsurgical removal of part of the cyst wall was done. Then a flexible endoscope of outer diameter 2.5 mm was introduced to inspect the cranial and caudal end of the cyst and further fenestration was done. The endoscope was passed forward to exit from the cyst cavity into the subarachnoid space. Endoscope was also used to inspect the area ventral to the cord and to look for adhesions. They concluded that the number of levels of laminectomy were significantly lower and operative times were shorter in the endoscopically treated group.

Limited laminectomy or Laminoplasty whenever possible may decrease the incidence of post operative deformity. Lesions requiring laminectomy of five or more vertebral segments in thecervical and thoracic region may result in kyphosis of the spine.[1,2]

Conclusions

From the review of literature, Intradural SAC was almost twice as common than extradural SAC. Ventrally located SAC are more common in Intradural location and Extradural ventral cysts are very rare. Intradural SAC were more commonly located in the thoracic and cervical region when compared to extradural SAC which were located commonly in the thoracolumbar, thoracic and lumbar regions.

Extradural SACs were mostly Primary and mainstay of treatment is complete or partial excision of the cyst with identification and ligation of the communicating pedicle. After preoperative imaging for detection of communication site, Selective laminectomy or hemilaminectomy with closure of dural defect is recommended in Extradural SACs. Total excision can be reserved to cases where dural communication site could not be detected by preoperative imaging or intraoperatively.

Selective laminectomy, partial excision of cyst and wide fenestration to sustain CSF flow is the recommended treatment in Intradural SAC. Total excision can be done if cyst is not more than 3 vertebral segments and not adherent to cord. Cystoperitoneal shunt can be done in few cases of recurrent intradural cysts.

References

1. Gortvai P. Extradural cysts of the spinal canal. J NeurolNeurosurg Psychiatry.1963;26:223–30.
2. Cloward RB. Congenital spinal extradural cysts: case report with review of literature. Ann Surg.1968;168:851–64.

3. Nabors MW, Pait TG, Byrd EB, et al. Updated assessment and current classification of spinal meningeal cysts. J Neurosurg. 1988;68(3):366-377.
4. Holly LT, Batzdorf U. Syringomyelia associated with intradural arachnoid cysts. J Neurosurg Spine. 2006;5(2):111-6.
5. Garg K, Borkar SA, Kale SS, Sharma BS. Spinal arachnoid cysts - our experience and review of literature. Br J Neurosurg. 2017;31(2):172-178.
6. Klekamp J. A New Classification for Pathologies of Spinal Meninges-Part 2: Primary and Secondary Intradural Arachnoid Cysts. Neurosurgery. 2017;81(2):217-229.
7. Moses ZB, Friedman GN, Penn DL, Solomon IH, Chi JH. Intradural spinal arachnoid cyst resection: implications of duraplasty in a large case series. J Neurosurg Spine. 2018;28(5):548-554.
8. Schmutzer M, Tonn JC, Zausinger S. Spinal intradural extramedullary arachnoid cysts in adults-operative therapy and clinical outcome. Acta Neurochir (Wien). 2020;162(3):691-702.
9. Baig Mirza A, Bartram J, Sinha S, et al. Surgical management and outcomes in spinal intradural arachnoid cysts: the experience from two tertiary neurosurgical centres. Acta Neurochir (Wien). 2022;164(5):1217-1228.
10. Singh S, Bhaisora KS, Sardhara J, et al. Symptomatic extradural spinal arachnoid cyst: More than a simple herniated sac. J Craniovertebr Junction Spine. 2019;10(1):64-71.
11. Shi L, Su Y, Yan T, Wang H, Wang K, Liu L. Early microsurgery on thoracolumbar spinal extradural arachnoid cysts: Analysis of a series of 41 patients. J Clin Neurosci. 2021;94:257-265.
12. Kumar A, Sakia R, Singh K, Sharma V. Spinal arachnoid cyst. J Clin Neurosci. 2011;18(9):1189-1192.
13. Bond AE, Zada G, Bowen I, McComb JG, Krieger MD. Spinal arachnoid cysts in the pediatric population: report of 31 cases and a review of the literature. J NeurosurgPediatr. 2012;9(4):432-441.
14. Thakur VV, Rangnekar RD, Aroor S, Kesavapisharady K, Abraham M. Conus medullaris intramedullary arachnoid cyst- case report and review of the literature. SurgNeurol Int. 2021;12:370.
15. Eroglu U, Bozkurt M, Kahilogullari G, et al. Surgical Management of Spinal Arachnoid Cysts in Adults. World Neurosurg. 2019;122:e1146-e1152.
16. Xu F, Jian F, Li L, Guan J, Chen Z. Surgical Treatment of Ten Adults with Spinal Extradural Meningeal Cysts in the Thoracolumbar Spine. J Korean Neurosurg Soc. 2021;64(2):238-246.
17. Funao H, Nakamura M, Hosogane N, et al. Surgical treatment of spinal extradural arachnoid cysts in the thoracolumbar spine. Neurosurgery. 2012;71(2):278-284.
18. Endo T, Takahashi T, Jokura H, Tominaga T. Surgical treatment of spinal intradural arachnoid cysts using endoscopy. J Neurosurg Spine. 2010;12(6):641-646.
19. Elsberg, C. A., Dyke, C. G., Brewer, E. D., The symptoms and diagnosis of extradural cysts. Bull. Neurol. Inst. N.Y.3 (1934), 395–417.
20. Lesoin F, Rousseau M, Thomas CE 3rd, Jomin M. Post traumatic spinal arachnoid cysts. Acta Neurochir (Wien). 1984;70(3-4):227-234.
21. Rohrer DC, Burchiel KJ, Gruber DP. Intraspinal extradural meningeal cyst demonstrating ball-valve mechanism of formation. Case report. J Neurosurg. 1993;78(1):122-125
22. Perret G, Green D, Keller J. Diagnosis and treatment of intradural arachnoid cysts of the thoracic spine. Radiology. 1962;79:425-429.
23. Teng P, Papatheodorou C. Spinal arachnoid diverticula. Br J Radiol. 1966;39(460):249-254.
24. Krings T, Lukas R, Reul J, Spetzger U, Reinges MH, Gilsbach JM, Thron A. Diagnostic and therapeutic management of spinal arachnoid cysts. Acta Neurochir (Wien). 2001;143(3):227-34
25. Liu JK, Cole CD, Kan P, Schmidt MH. Spinal extradural arachnoid cysts: clinical, radiological, and surgical features. Neurosurg Focus. 2007;22(2):E6.
26. Neo M, Koyama T, Sakamoto T, Fujibayashi S, Nakamura T. Detection of a dural defect by cinematic magnetic resonance imaging and its selective closure as a treatment for a spinal extradural arachnoid cyst. Spine (Phila Pa 1976). 2004;29(19):E426-E430.
27. MorizaneK, Fujibayashi S, Otsuki B, et al. Clinical and radiological features of spinal extradural arachnoid cysts: Valve-like mechanism involving the nerve root fiber as a possible cause of cyst expansion. J Orthop Sci. 2018;23(3):464-469.
28. Nakagawa A, Kusaka Y, Jokura H, Shirane R, Tominaga T. Usefulness of constructive interference in steady state (CISS) imaging for the diagnosis and treatment of a large extradural spinal arachnoid cyst. Minim Invasive Neurosurg. 2004;47(6):369-372.
29. Hingwala D, Chatterjee S, Kesavadas C, Thomas B, Kapilamoorthy TR. Applications of 3D CISS sequence for problem solving in neuroimaging. Indian J Radiol Imaging. 2011;21(2):90-97.
30. Hatashita S, Kondo A, Shimizu T, Kurosu A, Ueno H. Spinal extradural arachnoid cyst--case report. Neurol Med Chir (Tokyo). 2001;41(6):318-321.

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Acute synchronous bilateral extradural hematoma, a scarcely reported, rare entity. Analysis of cases with review of literature

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ABSTRACT
Objective: Extradural hematomas (EDHs) are contact injuries resulting from blunt trauma to the skull. It may be unilateral or bilateral. Bilateral acute extradural hematoma (EDH) was rarely reported before the advent of computed tomography. Mortality rates vary from 10.0%-40.0% and are an index of alertness and efficiency of health care and hospital set-up. EDH is an injury with preventable mortality in most cases. Bilateral acute extradural hematomas no longer confer high mortality of pre-CT era because of early diagnosis on CT, and prompt neurosurgical intervention. We present our experience with Bilateral EDH cases with an aim to investigate clinical, GCS score, and radiological features and also to analyse outcomes in these patients.
Material and methods: We analyzed cases of EDH who presented at our centre from January 2023 to December 2023 and further investigated ten cases of bilateral EDH. All the patients were examined clinically and a plain computerized tomography scan of the head was performed. The clinical details of all patients, GCS at admission and discharge, radiological findings on CT and neurosurgical intervention performed, and GOS (Glasgow outcome score) were noted.
Results: Road traffic accident was the reason for traumatic brain injury in all the cases. The GCS at admission was between 13–15 in 2 patients, 8–12 in 6 and 3–7 in 2 patients. Bilateral EDH was found in all of our patients .9 out of 10 cases were males. On CT head underlying fracture was seen in all cases. The localization of epidural hematomas in CT scans was bifrontal in four of the cases. All cases were treated with surgical management. The GOS Score was 5 in eight of the patients.
Conclusion: Posttraumatic bilateral acute extradural hematoma is a rare entity, but now does not show the high mortality previously seen in the preCT era. This is due to early radiological diagnosis on CT and profuse monitoring combined with expeditious operative procedures.

Keywords
bilateral acute extradural hematoma,
post traumatic,
synchronous,
rare entity,
outcome analysis

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INTRODUCTION

Extradural hematomas (EDHs) are caused due to contact injuries as a complication of blunt trauma to the skull. Extradural hematoma is identified by blood collected between the skull and the duramater. Usually, the bleeding is unilateral and originates from the middle meningeal artery. It may be unilateral or bilateral in location. [14]

Bilateral extradural hematoma (BEDH) is a rare entity accounting for less than 5 % of all acute extradural hematomas in adults but is extremely rare in children [5,13]. It was described by Watson et al in 1884 and was also reported in 1884 by Roy GC [15,17]. BEDH is commonly seen in the supratentorial region although some authors have reported the infratentorial location in few studies. [2,7]. Linear skull fractures are commonly associated with EDH in 61-95% of cases. In over 50% of all patients, the middle meningeal artery gives rise to the hematoma [13].

However, EDH is a major source of preventable mortality. The non-contrast head CT (Computed Tomography) Scan is the gold standard diagnostic investigation done for EDH cases. The size of the extradural hematoma, the patient's low Glasgow coma scale (GCS) score at presentation and deterioration in the neurological status are all indicators to proceed with the evacuation of hematoma. The outcome of BEDH remains quite variable and is dependent on many factors including, the initial neurological state at admission [1,2]. Hematomas present within the cranium may lead to life-threatening complications. Following traumatic head injury, the accumulated blood leads to increased intracranial pressure, which damages the brain and can lead to permanent vegetative state or death.

However, if there is uncertainty about the origin of the bleeding, other imaging modalities such as brain MRI and MRV can be done further to rule out any injury or occlusion of the superior sagittal sinus [9]. CTV can be used in acute sitting instead of time-consuming MRV. Along with finding the location of the hematoma, CT imaging also helps in detecting midline shift, skull bone fracture, basal cistern obliteration, and volume and type of hematomas which decides the further management.

High mortality rates (42-100%) have been reported in previously reported cases of BEDH. However with the widespread use of CT scan, early diagnosis has led to improved surgical results and prognosis. Bilateral acute extradural hematomas no longer show very high mortality in patients which was seen in the pre-CT era because of timely diagnosis, and rapid neurosurgical intervention. However, if the patient presented with a good level of consciousness and remains stable or improves, then it is up to the neurosurgeon to weigh the risks against the benefits of a surgical over conservative treatment [1]. Not much literature is available on traumatic bilateral intracranial hematomas possibly owing to resources like CT imaging being limited in many parts of our developing country. We present our experience with ten cases of bilateral EDH diagnosed on CT Scan along with analysis of clinical features, radiological findings and their outcome.

Materials and methods

We present ten patients of bilateral EDH who were admitted to our tertiary care centre from January 2023 to December 2023. All the cases were examined clinically and GCS (Glasgow Coma Score) at admission was noted. Plain computerized tomography (CT) scan of the head was performed in all the ten cases. We noted the age and gender, mode of injury, site of hematomas, GCS at presentation and discharge, treatment and their outcome in these bilateral traumatic head injury cases. The patients were evaluated by the Glasgow Outcome Score (GOS) after 6 months.

Results

Ten patients were admitted to the emergency at our tertiary care centre who presented with injury due to road traffic accidents in all the patients. Nine out of ten patients were males. Four patients presented with altered sensorium, headache and vomiting. The GCS at admission was between 13–15 in 2 patients, 8–12 in 6 and 3–7 in 2 patients. (Table 1) CT was done in all the patients. Bilateral EDH was diagnosed on CT in all of our patients. On CT head, underlying fracture was also seen in all cases. The localization of epidural hematomas in CT scan were bifrontal in four cases (Figure 2,6,8,9), bitemporal in one case (Figure 4), frontal and parietotemporal in one case(Figure1), frontoparietal and parietal in one case(Figure 5), parietotemporal on both sides in one case (Figure7), parietotemporal and parietal in one case (Figure 3) and bilateral occipital in one case (Figure 10). The size of the hematomas (thickness) ranged between 1.9 to 3.6 cm in all the cases. All cases were treated with surgical management. The GCS at discharge was between 13–15 in nine patients, 8–12 in one patient. GOS Score was 5 in eight of the patients. (Table 1)

Table 1. Clinical and radiological (CT) features, GCS and Outcome of patients

S. no	AGE	GENDER	GCS (admission)	HEMATOMA (RIGHT)	HEMATOMA (LEFT)	TREATMENT	GCS (Discharge)	GOS
1.	13 yr	Male	15	Frontal	Parietotemporal	Surgery	15	5
2.	55 yr	Female	7	Frontal	Frontal	Surgery	14	4
3.	46 yr	Male	5	Parietotemporal	Parietal	Surgery	10	4
4.	35 yr	Male	10	Temporal	Temporal	Surgery	15	5
5.	26 yr	Male	11	Frontoparietal	Parietal	Surgery	15	5
6.	22 yr	Male	15	Frontal	Frontal	Surgery	15	5
7	22 yr	Male	10	Parietotemporal	Parietotemporal	Surgery	14	5
8	21 yr	Male	9	Frontal	Frontal	Surgery	15	5
9	14 yr	Male	10	Frontal	Frontal	Surgery	15	5
10	21yr	Male	9	Occipital	Occipital	Surgery	15	5

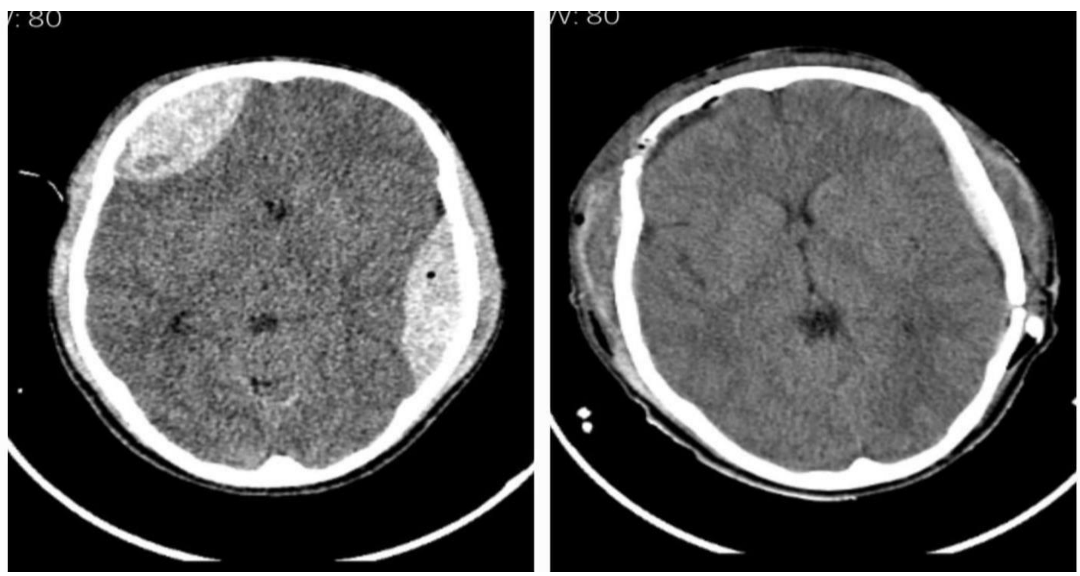

Figure 1. Axial CT Brain shows Frontal and Parietotemporal hematoma.

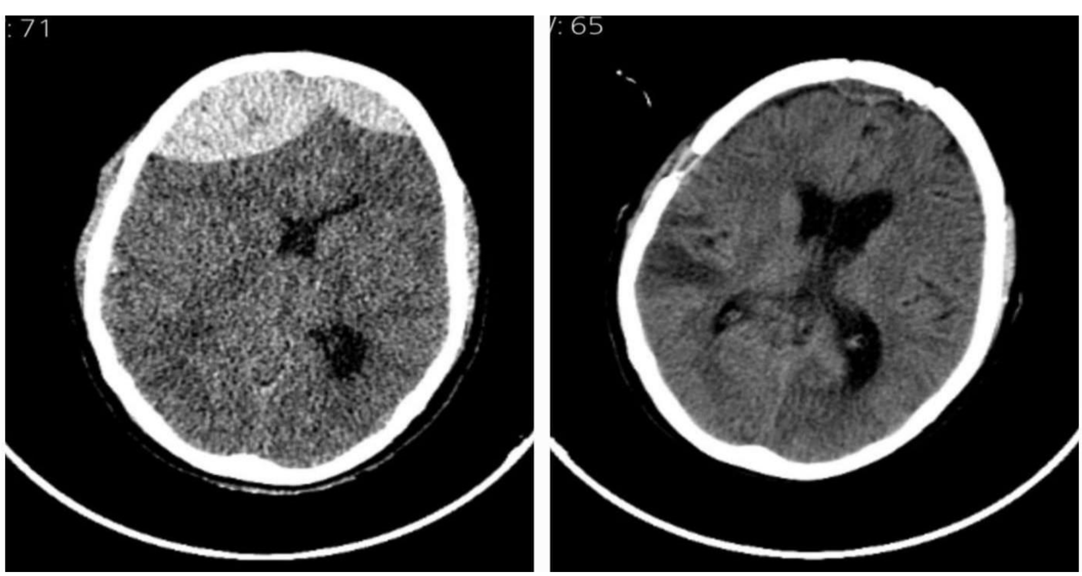

Figure 2. Axial CT Brain shows Bifrontal hematoma.

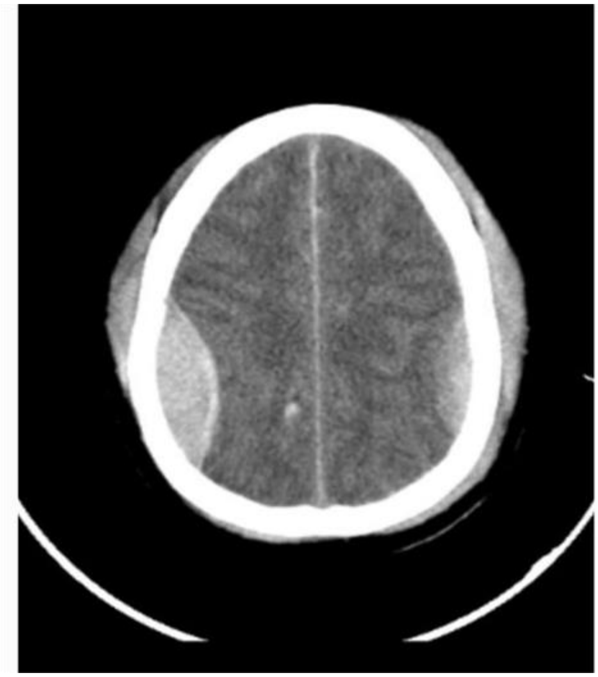

Figure 3. Axial CT Brain shows Parietotemporal and Parietal hematoma.

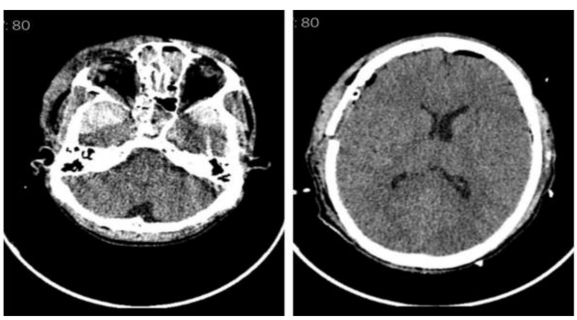

Figure 4. Axial CT Brain shows Bitemporal hematoma.

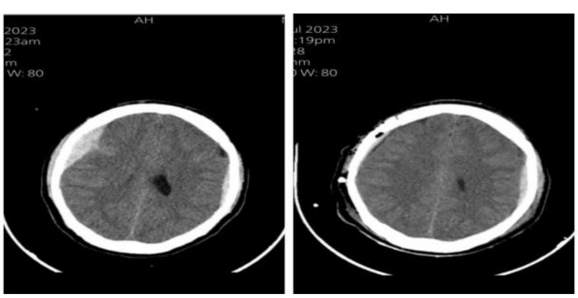

Figure 5. Axial CT Brain shows Frontoparietal and Parietal hematoma.

Discussion

Epidural hematomas are one of the most common complications of closed head injuries. Yet, they rarely show bilateral localization.

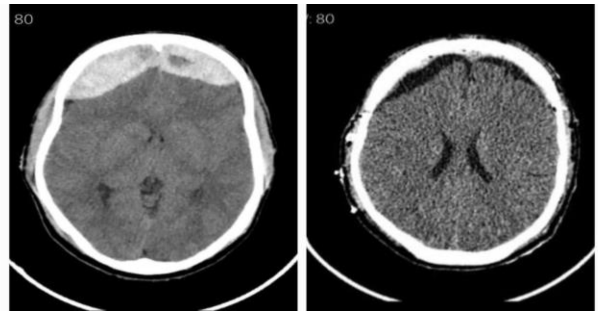

Figure 6. Axial CT Brain shows Bifrontal hematoma.

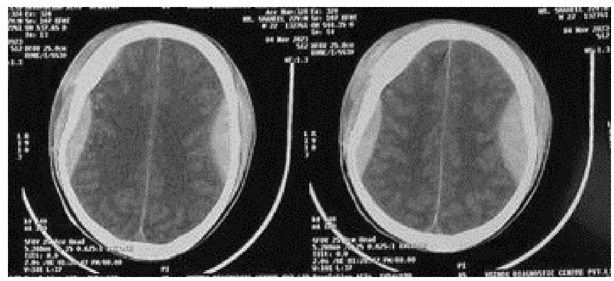

Figure 7. Axial CT Brain shows Bi-Parietotemporal hematoma.

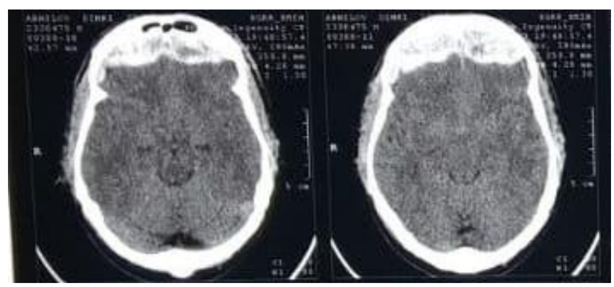

Figure 8. Axial CT Brain shows Bifrontal hematoma.

After an immense review of the literature in the last 20 years, we found fewer studies describing bilateral extradural hematoma. The reported literature has approximately 18 patients with GCS scores of 13–15, 25 patients with GCS between 8 and 12, and the majority 28 with GCS from 3 to 7. More number of male cases have been reported in other similar studies probably due to their work hazards. [1,3,4,6,8,9,10,11,12,16] Similarly our study shows six patients with GCS between 8 and 12 ,two patients with GCS of 13-15 and GCS 3–7 in 2 patients.. Our study also shows predominance of male patients owing to more travelling due to work.

Majority of the studies showed presence of fracture on CT similar to the presence of fracture on CT in all our cases. Bilateral extradural hematoma can be divided into two categories according to the time of bleeding, but most of the cases occur synchronously similar to our cases. The other type is sequential extradural hematoma wherein delayed hematoma is seen by postoperative CT scan on the contralateral side after the evacuation of the prior hematoma – similar case was reported by Fricia M et al. [6] In our study, all the patients developed synchronous bilateral EDH similar to findings in previous studies. [1,3,4,6,8,9,10,11,12,16]

Figure 9. Axial CT Brain shows Bifrontal hematoma.

Figure 10. Axial CT Brain shows Bioccipital hematoma.

All the studies showed that most of the cases of bilateral extradural hematoma were managed surgically with use of various surgical techniques at different centers. However, only two cases were treated conservatively [8,9]. Early surgical evacuation of bilateral extradural hematoma revealed favorable postoperative outcomes and GOS of 5 [1,9,10]. Many of the studies show GOS score of 5 in majority of patients similar to the good outcome in our patients. [1,3,4,6,8,9,10,11,12,16]

In developing country like ours, with limited resources, poor patient economic status, and time delay in reaching to the nearest medical centre, managing such cases of bilateral hematomas is a bigger challenge as prompt neurosurgical intervention is required. Taking care of various obstacles in developing countries and widespread use of CT scan, it is possible to reduce the mortality and therefore we may be able to minimize the postoperative neurological deficit which is hindrance to the patient's whole life.

Conclusions

Bilateral extradural hematomas are extremely rare but may lead to serious complications. The gold standard for diagnosis for these cases is CT Scan. With the widespread avai;ability of CT scan in developing countries, diagnosis before deterioration of the neurological status usually has an impact over the results of surgery, prognosis and even confers the possibility of a conservative treatment. Management of these cases includes careful planning, judicious surgical approach, and timely management for good results. Prompt surgical evacuation leads to good postoperative outcomes despite the surgical risks. In summary, Post traumatic bilateral acute extradural hematoma is a rare entity, but now do not show the high mortality previously seen in the pre CT era in developing countries. This is due to radiological diagnosis on CT and profuse monitoring combined with expeditious operative decompression procedures.

References

1. Abbas M, Khairy S, AlWohaibi M, Aloraidi A, AlQurashi WW. Bilateral Temporal Extradural Hematoma on Top of Bilateral Temporal Arachnoid Cyst: First Case Report and Extensive Literature Review. World Neurosurg. 2018 Jul;115:134-137.
2. Aggarwal A, Salunke P, Futane S, Sodhi HB. Bilateral

posterior fossa and temporo-occipital extra-dural hematomas: A simple novel technique. Asian J Neurosurg. 2017 Apr-Jun;12(2):253-255.

3. Agrawal A. Bilateral symmetrical parietal extradural hematoma. J Surg Tech Case Rep. 2011 Jan;3(1):34-6.
4. Bimpis A, Marcus HJ, Wilson MH. Traumatic bifrontal extradural haematoma resulting from superior sagittal sinus injury: case report. JRSM Open. 2015 May 11;6(4):2054270415579137..
5. Fadalla T, Jalaleldean B, Suliman M, Elsayed M, Elmahdi M, Elsalawi W. Post-traumatic bilateral synchronous acute extradural hematomas: A case report and review of literature. Ann Med Surg (Lond). 2022 Feb 12;75:103377.
6. Fricia M, Umana GE, Scalia G, Raudino G, Passanisi M, Spitaleri A, Cicero S. Posttraumatic Triple Acute Epidural Hematomas: First Report of Bilateral Synchronous Epidural Hematoma and a Third Delayed. World Neurosurg. 2020 Jan;133:212-215.
7. Gelabert M, Prieto A, Allut AG. Acute bilateral extradural haematoma of the posterior cranial fossa. Br J Neurosurg. 1997 Dec;11(6):573-5.
8. Giannakaki V, Triantafyllou T, Drossos D, Papapetrou K. Post-Traumatic Bifrontoparietal Extradural Hematoma with Superior Sagittal Sinus Detachment: A Case Report and Review of the Literature. World Neurosurg. 2016 Sep;93:489.e17-20.
9. Görgülü A, Cobanoglu S, Armagan S, Karabagli H, Tevrüz M. Bilateral epidural hematoma. Neurosurg Rev. 2000 Mar;23(1):30-3.
10. Hou B, Guo X, Wang D, Zhang Y, Zhao Z, Yang W, Wang G, Yan G, Zhou B, Ren H. Traumatic bifrontoparietal extradural hematomas with detachment of superior sagittal sinus: a case report and review of the literature. Br J Neurosurg. 2019 Aug;33(4):425-427.
11. Huda MF, Mohanty S, Sharma V, Tiwari Y, Choudhary A, Singh VP. Double extradural hematoma: an analysis of 46 cases. Neurol India. 2004 Dec;52(4):450-2.
12. Montemurro N, Santoro G, Marani W, Petrella G. Posttraumatic synchronous double acute epidural hematomas: Two craniotomies, single skin incision. Surg Neurol Int. 2020 Dec 11;11:435.
13. Pandey S, Sharma V, Shinde N, Sharma M. Bilateral occipital extradural hematoma in a child. J Pediatr Neurosci. 2015 Jul-Sep;10(3):270-2.
14. Ramzan A, Wani A, Malik AH, Kirmani A, Wani MA. Acute bilateral extradural hematomas. Neurol India. 2002 Jun;50(2):217-9.
15. Roy GC . Fracture of skull; extensive extravasation of blood on dura mater, producing compression of brain; trephining; partial relief of symptoms; death. Lancet 1884;2:319
16. Udoh DO. Bilateral post-traumatic acute extradural hematomas: a report of four cases and review of literature. Niger J Clin Pract. 2012 Jan-Mar;15(1):104-7.
17. Watson A, Orchard J.T .Fracture of the skull; treatment by trephining; recovery1.Lancet 1884 January 123;3149:11.

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A comprehensive review on anterior endoscopic cervical discectomy

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ABSTRACT
Anterior endoscopic cervical discectomy (AECD) represents a groundbreaking surgical technique that has garnered considerable interest in recent times due to its minimally invasive nature for addressing cervical disc herniation. This review endeavours to conduct a thorough examination of the existing literature concerning AECD, encompassing its indications, surgical technique, clinical outcomes, advantages, limitations, and potential complications. Through a meticulous analysis of the available data, this review aims to evaluate the effectiveness, safety, and overall viability of AECD as a substitute for conventional open surgical methods in addressing cervical disc issues. By delving into these aspects, the review seeks to provide insights into the potential role of AECD in enhancing patient outcomes and optimizing the management of cervical disc pathology.

Keywords
cervical spine,
cervical spondylosis,
discectomy,
endoscopy

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INTRODUCTION
Cervical disc herniation is a common cause of neck and upper limb pain, often leading to neurological symptoms and functional impairment. Traditional open surgical techniques, such as anterior cervical discectomy and fusion (ACDF), have been widely used for the treatment of cervical disc herniation. However, these procedures involve extensive tissue dissection, muscle retraction, and fusion, which can result in complications, prolonged recovery times, and potential limitations in spinal motion.[1,2] The development of endoscopic technologies has paved the way for minimally invasive approaches to cervical disc herniation. Anterior endoscopic cervical discectomy (AECD) is a novel technique that aims to minimize tissue trauma while providing effective decompression of the affected disc level. AECD involves accessing the cervical spine through small incisions and using specialized endoscopic instruments to visualize and remove the herniated disc material.[3,4]

The rationale for AECD lies in its potential to achieve similar clinical outcomes as traditional open surgeries while minimizing the associated drawbacks. By avoiding extensive tissue disruption and preserving spinal stability, AECD offers the promise of reduced postoperative pain, shorter hospital stays, faster recovery, and improved patient satisfaction. Additionally, the preservation of spinal motion and adjacent segment integrity are important considerations in the long-term success of cervical disc surgery.[5,6] The growing body of literature on AECD highlights its potential advantages over open surgical techniques. Several studies have reported favorable clinical outcomes, including significant pain relief, improvement in neurological function, and high patient satisfaction rates. Furthermore, AECD has demonstrated advantages such as reduced blood loss, decreased postoperative complications, and improved cosmetic outcomes.

It is also important to acknowledge that AECD is a technically demanding procedure requiring specialized training and expertise. Surgeon experience and patient selection criteria play crucial roles in achieving optimal outcomes. Additionally, long-term follow-up data are still limited, and comparative studies with traditional open surgeries and other minimally invasive techniques are necessary to establish the true efficacy and safety of AECD. In short, AECD represents a promising advancement in the management of cervical disc herniation. Its minimally invasive nature, potential for reduced tissue trauma, and preservation of spinal motion make it an appealing option for both patients and surgeons. However, further research and well-designed studies are needed to establish its long-term outcomes, comparative effectiveness, and potential role as a standard surgical approach for cervical disc pathology.

Discussion

Evolution of surgical techniques for cervical disc herniation:

The surgical management of cervical disc herniation has evolved over the years, with various techniques being developed to improve outcomes and minimize surgical invasiveness. Key milestones in the evolution of surgical techniques for cervical disc herniation are discussed below:

1. **Anterior cervical discectomy and fusion (ACDF):** ACDF has been the traditional surgical approach for cervical disc herniation. It involves the removal of the herniated disc material through an anterior approach, followed by the placement of a bone graft and anterior plate fixation to achieve spinal fusion. ACDF has shown favorable outcomes in terms of pain relief and neurological improvement.[1] However, concerns about adjacent segment degeneration and limited motion preservation have led to the exploration of alternative techniques.

2. **Posterior cervical foraminotomy:** Posterior cervical foraminotomy is a minimally invasive technique that aims to decompress the nerve root by removing the osteophytes and part of the facet joint. This approach allows for direct visualization of the nerve root and preservation of motion segments. Studies have demonstrated comparable outcomes to ACDF in terms of pain relief and functional improvement.[7]

3. **Cervical disc arthroplasty:** Cervical disc arthroplasty, also known as total disc replacement, involves the removal of the diseased disc and the implantation of an artificial disc prosthesis. This technique aims to preserve motion at the treated level and potentially reduce the risk of adjacent segment degeneration. Several studies have shown favorable clinical outcomes with cervical disc arthroplasty, including improved range of motion and similar or better pain relief compared to fusion techniques.[8,9]

4. **Minimally invasive approaches:** Advancements in endoscopic and minimally invasive techniques have revolutionized the field of cervical spine surgery. These approaches, such as AECD, utilize smaller incisions, specialized instruments, and endoscopic visualization to achieve decompression while minimizing tissue disruption. Minimally invasive techniques have been associated with reduced postoperative pain, shorter hospital stays, and faster recovery compared to open procedures.

5. **Full-endoscopic techniques:** Full-endoscopic techniques have gained popularity in recent years for the treatment of cervical disc herniation. These approaches utilize specialized endoscopic instruments to directly visualize and access the herniated disc through a small incision. Full-endoscopic techniques, such as full-endoscopic cervical discectomy, offer the advantages of minimal tissue disruption, reduced postoperative pain, and faster recovery.[3]

6. **Hybrid techniques:** Hybrid techniques combine elements of different surgical approaches to optimize outcomes. For example, hybrid

procedures may involve a combination of anterior and posterior approaches to achieve decompression and stabilization. These techniques aim to address specific pathology while minimizing invasiveness and preserving spinal function.

7. **Navigation-assisted techniques:** Navigation-assisted techniques utilize intraoperative navigation systems to enhance the accuracy and safety of surgical procedures. These systems provide real-time imaging guidance, improving the precision of implant placement and facilitating optimal decompression. Navigation-assisted techniques have shown potential in improving surgical outcomes and reducing complications.

Indications for AECD:
AECD has shown promising outcomes in specific indications. Common indications for AECD are:

1. **Cervical disc herniation:** AECD is commonly performed for the treatment of cervical disc herniation. It is particularly effective in cases where the herniation is causing compression of the spinal cord or nerve roots, resulting in radicular symptoms such as neck pain, arm pain, numbness, or weakness. AECD allows for direct visualization and precise removal of the herniated disc, relieving pressure on the neural structures.

2. **Cervical radiculopathy:** Cervical radiculo-pathy refers to the compression or irritation of a nerve root in the cervical spine, leading to radiating pain, numbness, and weakness in the corresponding upper extremity. AECD can be a suitable treatment option for cervical radiculopathy caused by disc herniation, as it provides targeted decompression of the affected nerve root.[10]

3. **Cervical myelopathy:** Cervical myelopathy is a condition characterized by compression of the spinal cord in the cervical spine, resulting in motor deficits, sensory changes, and loss of coordination. In selected cases of cervical myelopathy caused by disc herniation, AECD may be considered as a treatment option. The removal of the herniated disc material can relieve spinal cord compression and potentially improve neurological function.

4. **Failed conservative treatment:** AECD may be recommended when conservative treatments, such as medication, physical therapy, or spinal injections, have failed to provide adequate relief from symptoms and there is persistent pain or functional impairment. In such cases, AECD offers a minimally invasive surgical option to address the underlying pathology and alleviate symptoms.

Patient selection criteria:
Patient selection plays a crucial role in determining the success and safety of AECD. Proper patient selection ensures that individuals who are most likely to benefit from the procedure are chosen. While specific criteria may vary among surgeons and institutions, some general patient selection considerations for AECD are:

1. **Symptomatic disc herniation:** AECD is typically recommended for patients with symptomatic cervical disc herniation causing radicular symptoms, such as neck pain, arm pain, numbness, or weakness. Imaging studies, such as magnetic resonance imaging (MRI), should confirm the presence of disc herniation correlating with the patient's symptoms.[11]

1. **Failed conservative treatment:** Patients who have failed to respond to conservative treatments, including medication, physical therapy, and spinal injections, may be considered for AECD. A trial of conservative treatment for a reasonable duration should be undertaken before considering surgical intervention.

2. **Neurological deficits:** Patients with neurological deficits, such as muscle weakness, sensory changes, or coordination problems, caused by cervical disc herniation may be candidates for AECD. The presence and severity of neurological deficits should be evaluated through clinical examination and neuroimaging.

3. **Single-level disc herniation:** AECD is commonly performed for single-level disc herniation. Patients with a clear correlation between their

symptoms and a single-level disc herniation are considered suitable candidates. Multilevel herniations or complex pathologies may require alternative surgical approaches.

4. **Absence of severe instability:** Patients with severe cervical spine instability, such as significant spondylolisthesis or degenerative cervical kyphosis, may not be suitable candidates for AECD. Preoperative evaluation of cervical spine stability, including dynamic imaging, can help identify patients who require alternative surgical interventions.

5. **Recurrent disc herniation:** AECD can be considered as a treatment option for patients with recurrent disc herniation in the cervical spine. Recurrent disc herniation refers to the recurrence of herniation at the same level or adjacent levels following previous surgical intervention. AECD allows for targeted removal of the recurrent herniated disc material, providing symptom relief and reducing the need for more extensive revision surgeries.

6. **Favorable anatomy:** Patients with favorable anatomical characteristics, such as adequate access to the target disc space and sufficient disc height, are more likely to be suitable candidates for AECD. Preoperative imaging studies, including lateral radiographs and magnetic resonance imaging (MRI), can help assess the feasibility of the procedure based on the patient's anatomy.[12]

7. **Absence of ossification of the posterior longitudinal ligament (OPLL):** Patients with severe OPLL may not be appropriate candidates for AECD. OPLL can limit the ability to adequately visualize and access the herniated disc during the endoscopic procedure. Preoperative imaging, such as computed tomography (CT) or MRI, should be performed to evaluate the extent and severity of OPLL

8. **Adequate surgical experience:** AECD is a technically demanding procedure that requires specific surgical skills and expertise. Surgeons should have sufficient training and experience in performing endoscopic cervical discectomy to ensure optimal outcomes and minimize complications. Patients should seek surgeons who are well-versed in endoscopic techniques and have a track record of successful AECD procedures.

Cervical disc herniation types amenable to AECD: Some common types of cervical disc herniation that are amenable to AECD are:

1. **Posterolateral disc herniation:** AECD is suitable for treating posterolateral disc herniation, where the herniated disc material protrudes toward the side of the spinal canal. This type of disc herniation can cause compression of the nerve roots, resulting in radicular symptoms. AECD allows for direct visualization and removal of the herniated disc material through an anterior approach.[13]

2. **Central disc herniation:** AECD can also be used for central disc herniation, where the herniation occurs centrally within the spinal canal, causing compression of the spinal cord. In selected cases, AECD can be performed to decompress the spinal cord by removing the herniated disc material and relieving the pressure on the spinal cord.[13]

3. **Subligamentous disc herniation:** Subligamentous disc herniation refers to a herniated disc that is located beneath the posterior longitudinal ligament (PLL). AECD can be utilized to access and remove the subligamentous disc herniation while preserving the PLL. This approach avoids disruption of the PLL, which may help maintain spinal stability.

Surgical technique:

a. Preoperative planning and patient positioning: Preoperative planning and patient positioning are crucial aspects of AECD to ensure a successful procedure. An overview of the preoperative planning and patient positioning is given below:

1. **Preoperative imaging:** Preoperative imaging, such as MRI or CT scans, helps in evaluating the extent and location of the disc herniation, identifying any associated pathologies, and planning the surgical approach.[13]

2. **Neurological evaluation:** A comprehensive neurological evaluation is performed to assess the patient's symptoms, neurological deficits, and functional limitations. It helps determine the

indication for surgery and provides a baseline for postoperative comparison.

3. **Informed consent and patient education:** The surgeon should have a detailed discussion with the patient, explaining the procedure, potential risks, benefits, and possible complications. Informed consent should be obtained, and the patient should be educated about preoperative and postoperative care.

4. **Patient positioning:** Proper patient positioning is crucial for optimal access to the surgical site and to minimize the risk of complications. The patient is usually positioned supine on the operating table with the neck slightly extended. Various positioning aids, such as a headrest or a horseshoe-shaped headrest, may be used to stabilize the head and maintain proper alignment during the procedure.

b. Access routes and portal placement: Access routes and portal placement are important considerations in AECD to ensure safe and effective access to the surgical site.

1. **Access routes:** The access route refers to the path taken to reach the targeted disc level. In AECD, the most common access routes include the anterior approach through the intervertebral disc space or the lateral approach through the uncovertebral joint. The choice of access route depends on the specific anatomical considerations and surgeon preference.

2. **Portal placement:** Portal placement involves creating small incisions for the introduction of endoscopic instruments and visualization. The exact placement of portals may vary depending on the targeted disc level and the specific technique used. Typically, the portals are strategically placed to provide optimal access, visualization, and instrument maneuverability.

c. Step-by-step procedure description: Surgical technique for AECD is given below:

1. **Patient positioning and anesthesia:** The patient is placed in a supine position on the operating table, and general anesthesia is administered to ensure comfort and immobility during the procedure.

2. **Incision and exposure:** A small incision is made in the anterior neck, typically along a natural skin crease. The subplatysmal plane is dissected to expose the anterior cervical spine.

3. **Dilating and portal creation:** Sequential dilators or tubular retractors are used to create a working channel to the targeted disc space. This allows for visualization and instrument access to the disc.

4. **Discectomy and decompression:** Endoscopic instruments, such as a microendoscope or endoscopic forceps, are introduced through the portals. The herniated disc material is visualized and carefully removed, relieving pressure on the spinal cord or nerve roots.

5. **Visualization and confirmation:** Throughout the procedure, a high-definition endoscope or camera is used to provide visualization of the surgical site. This allows the surgeon to ensure complete decompression and confirm the appropriate removal of the herniated disc.

6. **Closure:** After the completion of the discectomy, the instruments are removed, and the incision is closed using sutures or surgical staples. Sterile dressings are applied to the incision site.

d. Intraoperative considerations and precautions: During AECD, several intraoperative considerations and precautions should be taken to ensure patient safety and successful outcomes. Some common intraoperative considerations are given below:

1. **Neural structures identification and protection:** Careful identification and protection of neural structures, such as the spinal cord and nerve roots, are crucial during AECD. Visualization and gentle retraction techniques help minimize the risk of inadvertent injury to these structures.

2. **Hemostasis:** Adequate hemostasis is essential to minimize bleeding during the procedure. Hemostatic agents, cautery techniques, and meticulous dissection can be used to achieve hemostasis and maintain a clear surgical field.

3. **Complete discectomy:** Thorough removal of the herniated disc material is crucial to ensure decompression of neural structures and reduce the risk of recurrence. Visualization through the endoscope and careful inspection of the disc space help ensure complete discectomy.

4. **Spinal stability:** Preservation of spinal stability is important during AECD. Depending on the patient's condition and surgeon's preference, additional procedures such as fusion or artificial disc replacement may be considered to maintain spinal stability.

5. **Neurophysiological monitoring:** Intraoperative neurophysiological monitoring, such as somatosensory evoked potentials (SSEP) and electromyography (EMG), may be employed to assess the integrity of neural structures and guide surgical decision-making.

Clinical outcomes:

a. **Pain relief and functional improvement:** The clinical outcome of AECD is often evaluated in terms of pain relief and functional improvement. Ruetten S et al.[3] compared the clinical outcomes of full-endoscopic anterior decompression (including AECD) with conventional anterior decompression and fusion. The authors found that both approaches provided significant pain relief and functional improvement, but the full-endoscopic approach resulted in a lower complication rate and faster recovery.

b. **Rates of neurological recovery:** Ruetten S et al.[3] compared the neurological recovery rates between full-endoscopic anterior decompression (including AECD) and conventional anterior decompression and fusion. The authors reported significant neurological improvement in both groups, but the full-endoscopic approach resulted in a lower complication rate and faster recovery.

c. **Return to work and quality of life measures:** In this study by Ruetten S et al.[3], the authors compared full-endoscopic anterior decompression (including AECD) with conventional anterior decompression and fusion. They reported a faster return to work and improved quality of life in the full-endoscopic group compared to the conventional group.

d. **Comparison with open surgical approaches:** When comparing AECD with open surgical approaches for the treatment of cervical disc herniation, several factors come into consideration. Some aspects to be considered when comparing AECD with open surgical approaches are:

1. **Surgical approach:** AECD is a minimally invasive procedure that utilizes an endoscope to access and remove the herniated disc material through a small incision. In contrast, open surgical approaches typically involve a larger incision and require more extensive tissue dissection to access the affected disc.

2. **Muscle and tissue trauma:** AECD is associated with reduced muscle and tissue trauma compared to open surgical approaches. The smaller incision and targeted approach of AECD minimize disruption to surrounding structures, leading to potentially faster recovery and reduced postoperative pain.

3. **Blood loss and complications:** AECD is generally associated with less blood loss compared to open surgical approaches. The reduced tissue trauma and smaller incision contribute to lower complication rates, such as infection and wound healing problems, in AECD.

4. **Operative time:** AECD typically requires a shorter operative time compared to open surgical approaches. The minimally invasive nature of AECD allows for a more focused and streamlined procedure, potentially resulting in shorter surgical durations.

5. **Hospital stay and recovery:** AECD is often associated with shorter hospital stays compared to open surgical approaches. Patients undergoing AECD may experience faster recovery and return to their daily activities, including work, compared to those undergoing open surgical procedures.

6. **Long-term outcomes:** Both AECD and open surgical approaches have shown favorable long-term outcomes in terms of pain relief, functional improvement, and patient satisfaction. However, long-term studies directly comparing the two

approaches are limited, and further research is needed to fully assess the comparative effectiveness and long-term outcomes.

Advantages of AECD:

a. Minimally invasive nature: One of the key advantages of AECD is its minimally invasive nature. Some points highlighting the benefits of the minimally invasive approach are:

1. **Reduced tissue trauma:** AECD involves a smaller incision and uses endoscopic techniques to access the cervical disc herniation. This results in reduced tissue trauma compared to traditional open surgical approaches. The smaller incision minimizes disruption to surrounding muscles, ligaments, and other soft tissues, leading to less postoperative pain and faster recovery.

2. **Decreased blood loss:** The minimally invasive nature of AECD results in reduced blood loss during the procedure. This can lead to a lower risk of complications related to blood loss and the need for blood transfusions.

3. **Smaller incision and minimal scarring:** AECD utilizes a smaller incision compared to open surgical approaches. This results in a more favorable cosmetic outcome with minimal scarring. The smaller incision size is particularly advantageous for patients who value aesthetic considerations.

4. **Faster recovery and shorter hospital stay:** The minimally invasive nature of AECD leads to faster recovery and shorter hospital stays compared to open surgical approaches. Patients may experience reduced postoperative pain, require fewer pain medications, and be able to resume their daily activities sooner.

b. Preservation of cervical spine stability: Another advantage of AECD is the preservation of cervical spine stability. Some points highlighting this benefit are:

1. **Preservation of natural spinal anatomy:** AECD allows for targeted removal of the herniated disc material while preserving the integrity of the surrounding spinal structures. This includes preserving the facet joints, posterior ligaments, and adjacent vertebral bodies. By maintaining the natural anatomy, AECD helps to preserve the stability of the cervical spine.

2. **Reduced risk of postoperative instability:** The preservation of spinal structures through AECD can help minimize the risk of postoperative instability. This is particularly important in cases where the disc herniation is associated with minimal or no significant degenerative changes in the adjacent vertebral segments.

3. **Avoidance of adjacent segment degeneration:** By preserving the natural spinal anatomy and minimizing the extent of surgery, AECD can potentially reduce the risk of adjacent segment degeneration. This refers to the accelerated degeneration that may occur in the vertebral segments adjacent to the treated level following spinal fusion procedures.

4. **Potential for motion preservation:** In some cases, AECD may allow for preservation of motion at the treated level, depending on the specific technique employed. This can be advantageous for maintaining normal neck motion and potentially reducing the risk of adjacent segment degeneration associated with spinal fusion procedures.

c. Reduced blood loss and tissue trauma: Reduced blood loss and tissue trauma are significant advantages of AECD. An overview of these benefits is:

1. **Reduced blood loss:** AECD is a minimally invasive procedure that involves a smaller incision and utilizes endoscopic techniques. Compared to traditional open surgical approaches, AECD typically results in reduced blood loss during the procedure. This can lead to several advantages, including a lower risk of complications related to blood loss and potentially less need for blood transfusions.

2. **Minimal tissue trauma:** AECD is designed to minimize disruption to the surrounding tissues, including muscles, ligaments, and other soft tissues. The smaller incision used in AECD reduces the extent of tissue trauma compared to open surgical approaches. As a result, patients may experience less postoperative pain, have a

faster recovery, and require shorter hospital stays.

3. **Preservation of surrounding structures:** AECD focuses on assessing and treating the herniated disc material while preserving the integrity of the surrounding spinal structures. By avoiding excessive tissue disruption, the procedure aims to minimize trauma to the adjacent muscles, ligaments, and bones. This can contribute to reduced postoperative pain and faster recovery.

4. **Improved cosmetic outcome:** The smaller incision used in AECD results in a more favorable cosmetic outcome compared to larger incisions associated with open surgical approaches. The reduced tissue trauma and smaller scar contribute to improved cosmetic appearance, which may be particularly important for patients who value aesthetic considerations.

d. Shorter hospital stay and faster recovery: Shorter hospital stay and faster recovery are significant advantages of AECD. An overview of these benefits is:

1. **Shorter hospital stay:** AECD is a minimally invasive procedure that is associated with shorter hospital stays compared to traditional open surgical approaches. The smaller incision and reduced tissue trauma associated with AECD often result in less postoperative pain and faster recovery. This allows patients to be discharged from the hospital earlier, leading to a shorter overall hospital stay.[14]

2. **Faster recovery:** AECD minimizes disruption to the surrounding tissues, including muscles, ligaments, and other soft tissues. As a result, patients may experience less postoperative pain and have a faster recovery compared to open surgical approaches. The minimally invasive nature of AECD promotes early mobilization, allowing patients to resume their daily activities sooner.

3. **Reduced postoperative complications:** AECD's shorter hospital stay and faster recovery can contribute to a reduced risk of postoperative complications. Prolonged hospital stays are associated with an increased risk of healthcare-associated infections and other complications. By minimizing the duration of hospitalization, AECD helps to lower the overall risk of these complications.

4. **Improved patient satisfaction:** The shorter hospital stay and faster recovery associated with AECD can enhance patient satisfaction. Patients often prefer minimally invasive procedures that allow them to return to their normal activities quickly and with minimal disruption to their daily lives. AECD's ability to offer a faster recovery can positively impact patients' overall experience and satisfaction with the procedure.[14]

e. Cosmetic benefits and patient satisfaction: Cosmetic benefits and patient satisfaction are important considerations when evaluating the advantages of AECD. An overview of these benefits is:

1. **Minimal scarring:** AECD is a minimally invasive procedure that involves smaller incisions compared to open surgical approaches. The use of smaller incisions results in minimal scarring, which can be cosmetically advantageous. The smaller scars are often less noticeable and may be better concealed, leading to improved cosmetic outcomes and patient satisfaction.

2. **Aesthetically pleasing results:** AECD's minimally invasive approach and smaller incisions contribute to aesthetically pleasing results. Patients may feel more confident and satisfied with the appearance of their neck following the procedure, as the minimal scarring and preservation of natural tissue structures enhance the overall aesthetic outcome.

3. **Psychological well-being:** The cosmetic benefits of AECD can have a positive impact on a patient's psychological well-being and self-esteem. Patients may experience improved body image and reduced self-consciousness about visible scars, leading to enhanced satisfaction with their overall appearance and improved quality of life.

4. **Overall patient satisfaction:** The combination of reduced scarring, improved aesthetic outcomes, and enhanced psychological well-being contributes to overall patient satisfaction with AECD. Patients appreciate the cosmetic benefits of the procedure, which can positively

influence their perception of the surgical experience and outcomes.

Limitations and challenges:

a. Learning curve and surgeon expertise: Learning curve and surgeon expertise are important factors to consider in AECD, and they come with certain limitations and challenges. Some key points to consider are:

1. **Learning curve:** AECD is a technically demanding procedure that requires specialized training and experience. Surgeons must develop proficiency in endoscopic techniques and become familiar with the specific equipment used in AECD. The learning curve for AECD can be steep, and it may take time for surgeons to gain the necessary skills and expertise to perform the procedure effectively.[15]

2. **Surgeon expertise:** The success of AECD heavily relies on the expertise and skill of the surgeon performing the procedure. Surgeons with extensive experience in endoscopic spine surgery and a deep understanding of cervical anatomy are better equipped to handle the complexities and potential complications that may arise during AECD. Therefore, it is crucial for patients to seek out surgeons who have a proven track record and specialized training in AECD.

3. **Learning curve-related complications:** During the initial stages of the learning curve, surgeons may encounter higher rates of complications compared to experienced surgeons. This includes the risk of inadvertent damage to vital structures, suboptimal decompression, or incomplete removal of disc material. As surgeons gain experience and refine their skills, the rates of these complications typically decrease.[16]

4. **Patient selection and case complexity:** Surgeon expertise is particularly crucial in appropriately selecting patients for AECD and assessing the complexity of each case. Not all patients with cervical disc herniation may be suitable candidates for AECD, especially those with extensive pathology, instability, or significant spinal cord compression. Surgeons with expertise in AECD can accurately evaluate patient eligibility and identify cases that may require alternative surgical approaches.[16]

b. Patient-specific anatomical variations: Patient-specific anatomical variations can present challenges during AECD. Some key considerations are:

1. **Vertebral anatomy:** The anatomy of the cervical vertebrae can vary among individuals, including the size, shape, and orientation of the vertebral bodies, facet joints, and neural foramina. These anatomical variations can affect the accessibility and visualization of the targeted disc space during AECD. Surgeons need to be mindful of these variations and adapt their surgical approach accordingly.

2. **Disc morphology and pathology:** The morphology and pathology of the cervical discs can also vary among patients. The location, size, and extent of the disc herniation or degeneration can differ, making each case unique. Surgeons need to carefully assess the patient's specific disc pathology and customize the surgical approach and technique accordingly to achieve optimal outcomes.

3. **Neurovascular structures:** Anatomical variations of the neurovascular structures in the cervical region, such as the vertebral artery and spinal nerves, can pose challenges during AECD. Surgeons must have a detailed understanding of the patient's specific neurovascular anatomy to minimize the risk of injury during the procedure.

4. **Adjacent level anatomy:** Patient-specific variations in the adjacent cervical levels, including the disc height, facet joint morphology, and presence of osteophytes, can impact the surgical approach and technique during AECD. Surgeons must carefully assess the anatomy of the adjacent levels to ensure proper planning and execution of the procedure.

c. Potential complications and their management: AECD is generally considered a safe procedure, but like any surgical intervention, it carries potential complications. Some common complications that can occur during or after AECD, along with their management are:

1. **Nerve injury:** Nerve injury, including spinal nerve root or spinal cord injury, can occur during AECD. Surgeons must exercise caution to minimize the risk of nerve damage. In the event of nerve injury, prompt recognition and appropriate management are crucial. Neurological deficits should be evaluated, and if necessary, further imaging studies and consultation with a neurosurgeon or spine specialist should be considered.[17]

2. **Vascular injury:** Vascular injury, such as injury to the vertebral artery or carotid artery, is a potential complication during AECD. Surgeons should be vigilant to minimize the risk of vascular injury. In case of vascular injury, immediate recognition and intervention are crucial. Vascular surgery consultation may be necessary for management, including potential repair or embolization of the injured vessel.

3. **Infection:** Surgical site infection can occur after AECD. Strict adherence to aseptic techniques, proper wound care, and prophylactic antibiotics can help reduce the risk of infection. If an infection occurs, appropriate antibiotic therapy and wound management are essential. In some cases, surgical debridement or drainage may be necessary.

4. **Disc recurrence:** In some cases, there may be a recurrence of disc herniation at the treated level following AECD. Patients should be educated about the possibility of disc recurrence and the need for ongoing monitoring. In case of recurrence, further imaging studies, such as MRI, should be performed to assess the extent of recurrence and guide subsequent treatment decisions.

5. **Dysphagia and hoarseness:** AECD can occasionally lead to postoperative dysphagia (difficulty swallowing) and hoarseness due to irritation or injury to the esophagus or recurrent laryngeal nerve. These symptoms are typically temporary and resolve spontaneously over time. Symptomatic management, such as dietary modifications or voice therapy, may be appropriate in certain cases.[17,18]

6. **Instrumentation-related complications:** Instrumentation-related complications can include implant migration, malposition, or failure. Surgeons must carefully select appropriate implants and ensure their correct placement and fixation. In case of instrumentation-related complications, revision surgery may be required to address the issue, such as repositioning or replacing the implant.

7. **Adjacent segment degeneration:** AECD can lead to increased stress and motion at adjacent cervical levels, potentially contributing to adjacent segment degeneration (ASD) over time. Close follow-up and monitoring of patients are essential to identify and manage the development of ASD. Non-operative measures such as physical therapy or pain management can be considered initially, but in some cases, surgical intervention may be necessary.[17]

Comparative Analysis:

a. AECD versus traditional open discectomy: Comparison between AECD and traditional open discectomy is an important aspect to consider when evaluating the surgical options for cervical disc herniation. Some points of comparison between the two techniques are shown in the following table:

S.no.	Features	ACED	Traditional Open Discectomy
1.	Surgical Approach	Anterior endoscopic cervical discectomy is a minimally invasive procedure performed through a small incision in the front of the neck. It utilizes an endoscope and specialized instruments to access and remove the herniated disc material.	This technique involves a larger incision in the front or back of the neck, allowing direct visualization of the affected disc and removal of the herniated portion.
2.	Tissue Trauma	AECD involves less tissue trauma and	Open discectomy

	and Muscle Dissection	muscle dissection compared to open discectomy. The smaller incision and use of endoscopic instruments help minimize damage to surrounding structures.	involves more extensive tissue dissection, which can lead to increased muscle trauma and potential for postoperative pain and complications.
3.	Visualization and Magnification	The use of an endoscope provides magnified visualization of the surgical site, allowing for better identification and removal of the herniated disc material.	Open discectomy provides direct visualization of the surgical site, but the magnification is limited compared to endoscopic techniques.
4.	Hospital Stay and Recovery	Minimally invasive nature of AECD usually leads to shorter hospital stays and faster recovery times compared to open discectomy.	Open discectomy may require a longer hospital stay and recovery period due to the larger incision and associated tissue trauma.
5.	Complications	AECD is associated with its own set of potential complications, such as nerve injury, vascular injury, infection, and recurrence of disc herniation. However, the overall complication rates are generally reported to be low.	Open discectomy also carries potential complications such as nerve damage, infection, blood loss, and scar formation.

b. Cost-effectiveness considerations: Cost-effectiveness is an important aspect to consider when evaluating medical interventions such as AECD and traditional open discectomy. Some general considerations regarding cost-effectiveness in the context of AECD are:

1. **Direct costs:** Direct costs include hospitalization costs, surgical fees, anesthesia fees, and postoperative care expenses. AECD is often associated with shorter hospital stays and faster recovery times compared to traditional open discectomy. This can potentially result in lower direct costs for AECD due to reduced resource utilization.

2. **Indirect costs:** Indirect costs encompass factors such as lost productivity, time off work, and rehabilitation expenses. Minimally invasive techniques like AECD may offer advantages in terms of shorter recovery times, allowing patients to return to work and daily activities sooner, potentially reducing indirect costs associated with lost productivity.

3. **Complications and reoperation rates:** The occurrence of complications and the need for reoperation can significantly impact the overall cost-effectiveness of a surgical intervention. While AECD is generally associated with lower complication rates compared to open discectomy, it is crucial to consider the long-term outcomes, including the potential need for revision surgery, as these factors can influence the cost-effectiveness of AECD.

4. **Health-related quality of life:** Assessing health-related quality of life is an essential component of cost-effectiveness analysis. While AECD and traditional open discectomy aim to provide pain relief and functional improvement, evaluating the long-term impact on quality of life is important when considering the cost-effectiveness of the procedures.

Future directions and innovations:

a. Advances in endoscopic technology: Advances in endoscopic technology have significantly contributed to the field of minimally invasive spine surgery, including AECD. These advancements have improved visualization, instrumentation, and procedural techniques, enhancing the safety and efficacy of endoscopic procedures. Some notable advances in endoscopic technology include:

1. **High-definition imaging:** Modern endoscopic systems employ high-definition cameras that provide excellent visualization of the surgical field. This allows surgeons to navigate and perform precise interventions with enhanced clarity and detail.[16]

2. **Miniaturized instruments:** Endoscopic instruments have become increasingly compact and specialized, enabling surgeons to access and manipulate anatomical structures through small incisions. These instruments often incorporate advanced features such as adjustable angles, ergonomic handles, and improved durability.

3. **Improved lighting and visualization systems:** Endoscopic systems now incorporate advanced lighting technologies, such as xenon or LED light sources, to provide optimal illumination during surgery. Coupled with high-definition cameras and monitors, these systems enable clear visualization of the surgical field, even in deep or narrow anatomical spaces.[16]

4. **Navigation and robotics:** Endoscopic procedures can benefit from the integration of navigation and robotic technologies. Navigation systems utilize real-time imaging and tracking to assist surgeons in precise instrument guidance and localization within the spine. Robotic systems can enhance surgical accuracy and facilitate complex maneuvers by providing assistance and precision control to the surgeon.

5. **Surgical training simulators:** Virtual reality-based simulators and training modules have been developed to enhance the education and skill development of surgeons performing endoscopic procedures. These simulators allow surgeons to practice surgical techniques in a controlled environment, improving their proficiency and reducing the learning curve.

6. **Tissue management:** Advancements in endoscopic technology have led to the development of specialized tools for tissue management, including bipolar radiofrequency ablation and laser systems. These tools enable efficient and controlled tissue ablation, minimizing the risk of injury to surrounding structures.

b. Training and education for widespread adoption: Training and education are crucial factors for the widespread adoption of AECD and other advanced endoscopic spine surgery techniques. Few key points related to training and education in AECD are:

1. **Specialized training programs:** Surgeons interested in AECD should undergo specialized training programs to gain the necessary knowledge and skills. These programs typically include didactic sessions, hands-on workshops, and observation of experienced surgeons performing AECD procedures. This allows surgeons to understand the surgical technique, become familiar with the instruments and equipment, and develop proficiency in performing the procedure.

2. **Mentorship and proctorship:** Mentoring and proctorship play a crucial role in the learning process of AECD. Experienced surgeons can serve as mentors and provide guidance, supervision, and feedback to novice surgeons. Proctorship involves the presence of an experienced surgeon during the initial AECD procedures performed by a trainee, ensuring patient safety and assisting in challenging cases.

3. **Continuing medical education (CME):** Ongoing education and participation in CME programs are essential for surgeons to stay updated with the latest advancements in AECD and related techniques. CME activities, such as conferences, workshops, and online courses, provide opportunities to learn from experts, exchange knowledge, and enhance technical skills.

4. **Surgical simulation:** The use of surgical simulation platforms and virtual reality technology can aid in the training and education of AECD. Simulators allow surgeons to practice and refine their skills in a controlled environment before performing actual surgeries. These platforms can provide realistic anatomical models, haptic feedback, and interactive scenarios to simulate various surgical scenarios encountered in AECD.

5. **Collaboration and peer learning:** Collaboration among surgeons, multidisciplinary team members, and professional societies promotes peer learning and knowledge sharing. Surgeons can attend conferences, join professional societies, and participate in research and case discussions to exchange experiences, discuss challenging cases, and learn from each other's expertise.

6. **Clinical guidelines and consensus statements:** The development of clinical guidelines and consensus statements by expert panels and professional societies helps standardize the indications, techniques, and postoperative care in AECD. These guidelines serve as valuable resources for surgeons in their training and clinical practice, ensuring safe and effective outcomes.

Recommendations for future research and clinical practice:

Based on the current understanding of AECD in the management of cervical disc herniation, the following recommendations can be made for future research and clinical practice:

1. **Comparative studies:** Conduct more comparative studies that directly compare AECD with traditional open surgical approaches, as well as other minimally invasive techniques such as microdiscectomy or cervical disc arthroplasty. These studies should evaluate outcomes such as pain relief, functional improvement, complication rates, patient satisfaction, and cost-effectiveness. Comparative studies can provide valuable insights into the relative effectiveness and safety of AECD compared to other treatment options.

2. **Long-term follow-up:** Perform long-term follow-up studies to assess the durability of outcomes after AECD. Investigate the long-term success rates, recurrence rates, and complications associated with AECD over an extended period. Long-term follow-up data will provide a better understanding of the sustained benefits and potential complications associated with AECD.

3. **Standardization of outcome measures:** Establish standardized outcome measures to evaluate the effectiveness of AECD consistently across different studies. Consensus on outcome measures will facilitate meaningful comparisons and meta-analyses of the available data, leading to more robust evidence-based conclusions.

4. **Patient selection criteria:** Refine and standardize the patient selection criteria for AECD. Identify specific patient characteristics, anatomical variations, and clinical presentations that are most suitable for AECD. This will help in optimizing patient selection, ensuring better outcomes, and avoiding unnecessary procedures in patients who may not benefit from AECD.

5. **Training and education:** Continue to focus on training and education for surgeons interested in adopting AECD. Develop standardized training programs, including hands-on workshops, surgical simulation, and mentorship opportunities, to enhance surgical skills and ensure safe and effective adoption of AECD. Additionally, promote the exchange of knowledge and experiences among surgeons through conferences, professional societies, and multidisciplinary collaborations.

6. **Cost-effectiveness studies:** Conduct more cost-effectiveness studies to assess the economic impact of AECD compared to other treatment modalities. Evaluate direct costs, indirect costs, and quality of life outcomes to provide a comprehensive understanding of the economic value of AECD. This information can guide decision-making by healthcare providers and policymakers.

7. **Advances in endoscopic technology:** Continue to explore and develop advancements in endoscopic technology to further improve visualization, instrumentation, and surgical techniques in AECD. Investigate new tools, imaging modalities, and navigation systems that can enhance the safety and efficacy of AECD.

8. **Patient-reported outcomes:** Include patient-reported outcome measures in research and clinical practice to assess the impact of AECD on patients' quality of life, functional status, and satisfaction. Incorporating patient perspectives will provide a more comprehensive understanding of the outcomes and benefits of AECD.

Summary of key findings:

- AECD is a minimally invasive surgical technique used for the treatment of cervical disc herniation. It involves accessing the affected disc through a small incision in the front of the neck, using specialized endoscopic instruments and visualization systems.
- The evolution of surgical techniques for cervical disc herniation has led to the development of minimally invasive approaches like AECD. These techniques aim to achieve comparable outcomes to traditional open surgery while minimizing tissue trauma and promoting faster recovery.
- Indications for AECD include cervical disc herniation causing radiculopathy or myelopathy, unresponsive to conservative treatment. Other indications may include foraminal stenosis, discogenic neck pain, and recurrent disc herniation after previous surgery.
- Patient selection criteria for AECD depends on factors such as the location and type of disc herniation, patient's symptoms, and overall health. Factors like the absence of severe instability, presence of intact disc height, and good bone quality are considered when determining the suitability of a patient for AECD.
- The surgical technique of AECD involves various steps, including preoperative planning, patient positioning, access route selection, portal placement, disc space preparation, discectomy, and potential fusion. These steps are performed using specialized endoscopic instruments and visualization systems.
- Clinical outcomes of AECD have shown favorable results in terms of pain relief and functional improvement. Studies have reported significant reductions in neck and arm pain, improvement in neurological function, and high patient satisfaction rates following AECD.
- Rates of neurological recovery after AECD have been promising, with most patients experiencing improvement or resolution of neurological symptoms. The decompression achieved through AECD allows for the relief of neural compression, leading to neurological recovery.
- AECD has demonstrated favorable return-to-work rates and improvements in quality of life measures. Patients who undergo AECD generally experience shorter hospital stays, reduced postoperative pain, and faster recovery compared to traditional open surgery.
- When compared to open surgical approaches, AECD has shown comparable or even superior outcomes in terms of pain relief, functional improvement, complication rates, and patient satisfaction. AECD offers advantages such as reduced blood loss, decreased tissue trauma, shorter recovery time, and improved cosmetic outcomes.
- Cost-effectiveness considerations suggest that AECD can be a cost-effective treatment option due to shorter hospital stays, decreased need for postoperative care, and faster return to work.
- Advances in endoscopic technology have contributed to enhanced visualization and instrumentation in AECD. Techniques such as the use of tubular retractors, microendoscopic approaches, and specialized endoscopic systems have improved surgical precision and outcomes.

List of Abbreviations:

ACDF: Anterior Cervical Discectomy and Fusion
AECD: Anterior Endoscopic Cervical Discectomy
CME: Continuing Medical Education
CT: Computed Tomography
EMG: Electromyography
MRI: Magnetic Resonance Imaging
OPLL: Ossified Posterior Longitudinal Ligament
PLL: Posterior Longitudinal Ligament
SSEP: Somatosensory Evoked Potentials

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References

1. Fountas KN, Kapsalaki EZ, Nikolakakos LG, et al. Anterior cervical discectomy and fusion associated complications. Spine (Phila Pa 1976). 2007;32(21):2310-2317. doi:10.1097/BRS.0b013e318154c57e
2. Hilibrand AS, Carlson GD, Palumbo MA, Jones PK, Bohlman HH. Radiculopathy and myelopathy at segments adjacent to the site of a previous anterior cervical arthrodesis. J Bone Joint Surg Am. 1999;81(4):519-528. doi:10.2106/00004623-199904000-00009
3. Ruetten S, Komp M, Merk H, Godolias G. Full-endoscopic anterior decompression versus conventional anterior decompression and fusion in cervical disc herniations. Int

Orthop. 2009;33(6):1677-1682. doi:10.1007/s00264-008-0684-y
4. Hu Y, Lv G, Ren S, Johansen D. Mid- to Long-Term Outcomes of Cervical Disc Arthroplasty versus Anterior Cervical Discectomy and Fusion for Treatment of Symptomatic Cervical Disc Disease: A Systematic Review and Meta-Analysis of Eight Prospective Randomized Controlled Trials. PLoS One. 2016;11(2):e0149312. doi:10.1371/journal.pone.0149312
5. Wen Z, Lu T, Wang Y, Liang H, Gao Z, He X. Anterior Cervical Corpectomy and Fusion and Anterior Cervical Discectomy and Fusion Using Titanium Mesh Cages for Treatment of Degenerative Cervical Pathologies: A Literature Review. Med Sci Monit. 2018;24:6398-6404. doi:10.12659/MSM.910269
6. Kulkarni AG, Hee HT, Wong HK. Solis cage (PEEK) for anterior cervical fusion: preliminary radiological results with emphasis on fusion and subsidence. Spine J. 2007;7(2):205-209. doi:10.1016/j.spinee.2006.03.002
7. Bhatia S, Brooks NP. Posterior endoscopic cervical foraminotomy. Neurosurgery Clinics. 2020 Jan 1;31(1):9-16. doi:10.1016/j.nec.2019.08.001
8. Wang T, Wang H, Liu S, An HD, Liu H, Ding WY. Anterior cervical discectomy and fusion versus anterior cervical corpectomy and fusion in multilevel cervical spondylotic myelopathy: a meta-analysis. Medicine. 2016 Dec 1;95(49):e5437. doi:10.1097/MD.0000000000005437
9. Phillips FM, Allen TR, Regan JJ, et al. Cervical disc replacement in patients with and without previous adjacent level fusion surgery: a prospective study. Spine (Phila Pa 1976). 2009;34(6):556-565. doi:10.1097/BRS.0b013e31819b061c
10. Ahn Y. Anterior Endoscopic Cervical Discectomy: Surgical Technique and Literature Review. Neurospine. 2023;20(1):11-18. doi:10.14245/ns.2346118.059
11. Parihar VS, Yadav N, Ratre S, Dubey A, Yadav YR. Endoscopic Anterior Approach for Cervical Disc Disease (Disc Preserving Surgery). World Neurosurg. 2018;115:e599-e609. doi:10.1016/j.wneu.2018.04.107
12. Yeung AT, Yeung CA. Advances in endoscopic disc and spine surgery: foraminal approach. Surg Technol Int. 2003;11:255-263.
13. Ruetten S, Komp M, Merk H, Godolias G. A new full-endoscopic technique for cervical posterior foraminotomy in the treatment of lateral disc herniations using 6.9-mm endoscopes: prospective 2-year results of 87 patients. Minim Invasive Neurosurg. 2007;50(4):219-226. doi:10.1055/s-2007-985860
14. Ahn Y. The Current State of Cervical Endoscopic Spine Surgery: an Updated Literature Review and Technical Considerations. Expert Rev Med Devices. 2020;17(12):1285-1292. doi:10.1080/17434440.2020.1853523
15. Ratre S, Yadav YR, Swamy MN, Parihar V, Bajaj J. Endoscopic Anterior Cervical Discectomy (Disc Preserving). Neurol India. 2020;68(6):1310-1312. doi:10.4103/0028-3886.304078
16. Quillo-Olvera J, Lin GX, Kim JS. Percutaneous endoscopic cervical discectomy: a technical review. Ann Transl Med. 2018;6(6):100. doi:10.21037/atm.2018.02.09
17. Ju CI, Kim P, Seo JH, Kim SW, Lee SM. Complications of Cervical Endoscopic Spinal Surgery: A Systematic Review and Narrative Analysis. World Neurosurg. 2023;178:330-339. doi:10.1016/j.wneu.2023.07.058
18. Tsalimas G, Evangelopoulos DS, Benetos IS, Pneumaticos S. Dysphagia as a Postoperative Complication of Anterior Cervical Discectomy and Fusion. Cureus. 2022;14(7):e26888. Published 2022 Jul 15. doi:10.7759/cureus.26888

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A prospective study to evaluate the role of repetitive trans-cranial magnetic stimulation in treatment of chronic low back pain

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ABSTRACT

Introduction: Chronic Low Back Pain (CLBP) is a disabling musculoskeletal condition with a high prevalence in developing and developed countries. There are many treatment modalities but none of them provides a satisfactory and consistent cure. Recently focus has shifted to neuromodulation to cure chronic pains. The U.S. Food and Drug Administration (FDA) approved the use of Repetitive TMS (rTMS) in the management of Pain in 2013. Since then, centres across the world have been using Transcranial Magnetic Stimulation (TMS) as a noninvasive modality for the treatment of various pain conditions. rTMS is a variation of TMS where stimulation is provided in sessions to create long-term excitation in the brain cortex. Evidence on the effectiveness of rTMS for CLBP is scarce due to limited rigorous clinical trials. This study is the first of its kind undertaken in India to critically analyze the role of rTMS in the treatment of CLBP.

Materials & Methods: In this single institutional prospective, single-blind study, we enrolled 40 patients of CLBP sharing similar clinical profiles. They were divided into a test group and a sham group. In the test group patients were given rTMS in addition to conventional treatment while in the sham group, patients were taken through the procedure of rTMS without actually being administered it. A figure-of-eight-shaped coil was used focusing on the Left M1 area and Dorsolateral prefrontal cortex to administer the rTMS by a trained physician. Visual Analogue Scores (VAS) were noted before and after the procedure. Each patient was continuously monitored during the procedure for any side effects. Subsequently, they were interviewed and followed up for 6 months. At the end of 6 months, data was compiled and conclusions were drawn.

Result: In our study, we found that 90 per cent of patients in the test group reported a reduction in VAS score by 30 per cent reduction while the remaining had a 20 per cent reduction in pain scores. 90 per cent of subjects in the sham group reported a marginal improvement in VAS score which can be attributed to the placebo effect. Most patients in the test group reported an improvement in quality of life at the end of six months. None of the patients suffered any untoward side effects during or after the procedure.

Conclusion: Based on our study we conclude that rTMS is a safe procedure and it can be used as a modality in treating CLBP with satisfactory outcomes. Although the sample size was small, it is the first study of its kind undertaken in India to evaluate the role of rTMS-based neuromodulation in treating this chronic disabling condition. However, to be accepted as a standard of care for CLBP it will require further multi-institutional robust clinical trials with long-term follow-up.

Keywords
repetitive trans cranial magnetic stimulation (RTMS), chronic low back pain (CLBP), neuromodulation

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1. Introduction and background

Low back pain (LBP) refers to pain, muscle tension, or stiffness that occurs below the costal margin and above the inferior gluteal fold, with or without sciatica (pain that spreads from the lower back down to the legs.[1] LBP persisting >3 months is termed as CLBP. Low back pain is one of the most common musculoskeletal diseases among people with chronic pain. Recent research reveals about 45–75% of patients report feeling pain 12 months after the onset of LBP.[2] Lifetime prevalence of low back pain (LBP) is over 70% in industrialized countries with a worldwide lifetime prevalence of 84%.[3] There are many modalities for treating LBP like muscles strengthening exercises, nerve root block and analgesics, despite established benefits of these methods many patient face refractory low back pain. With increasing CLBP prevalence the affected individuals are persistently visiting the health care facilities resulting in loss of working hour and cost to the state and severly diminishing individuals activities of daily living (ADL). Recently focus has shifted on use of Transcranial Magnetic Stimulation (TMS) as a noninvasive modality for treatment of CLBP. The exact mechanism of TMS has not been understood however, it is hypothesized that TMS effects neuromodulation of brain tissue through the production of the high or low-intensity magnetic field to modulate cortical excitability.[4] rTMS refers to applying recurring TMS pulses to a specific brain region(motor cortex). rTMS is a variation of TMS where stimulation is provided in sessions of same condition to create long-term excitation in the brain cortex. The electromagnetic induction generated by rTMS is painless and safely passes through the skin and skull. Then, it produces small electric currents in the region of the brain just under the coil.[5]

rTMS has been approved by the U.S Food and Drug Administration (FDA) for the treatment of major depressive disorder (MDD) by 2008, While approval for treating pain was granted in 2013. Of late, various neurostimulation methods, including rTMS, peripheral nerve stimulation, spinal cord stimulation, deep brain stimulation, and motor cortex stimulation, have been applied to chronic-pain treatments including patient of Chronic low Back ache (CLBP). However, evidence on the effectiveness of rTMS for CLBP is scarce due to limited rigorous clinical trials. The aim of this study is to critically analyze the role of rTMS in treatment of CLBP in Indian population.

2. Material & Methods

2.1 Study Protocol: A Prospective, single center study conducted at Department of Neurosurgery and Department of Psychiatry at Command Hospital SC, Pune from Jan 2023 to June 2023. Total 40 patients of chronic low back pain sharing similar clinical profile were included in this study, with 20 patients in Test group & 20 in Sham group. The Test group was subjected to High frequency rTMS of 15 to 20 Hz, for 20 minutes in each session (6 sessions given per patient within 6 weeks), Sham group underwent the placement of coils without actually giving rTMS.

2.1.1 Inclusion Criteria:

1. Participants aged above 18 years;
2. Willing to participate in the study
3. Having stable pharmacological or nonpharmacological treatments for pain.
4. Low backpain more than 3 months

2.1.2 Exclusion Criteria

1. Specific causes: Autoimmune disease, Inflammatory, infective, malignancy, trauma (e.g., spondylosis, spondyloarthropathy, or vertebral fracture);
2. Pregnancy or nursing female
3. Previous spinal surgery
4. Psychiatric Illness
5. Evidence of Prolapsed disc on MRI imaging
6. Contraindications to use rTMS (e.g., severe head trauma, intracranial hypertension, implanted ferromagnetic devices, history of epilepsy).

2.2 Methodology:

All patient underwent MRI LS Spine before being enrolled for rTMS to exclude inflammatory, infective cause of LBP and prolapsed disc. After obtaining informed written consent, a figure-of-eight-shaped coil was used focusing on Left M1 area and Dorsolateral Pre-Frontal Cortex(Fig-1 & Fig-2). Motor evoked potential was recorded by observing the twitching of right thumb visually, for each patient and frequency of pulse which generates twitching was recorded and frequency which is lesser than Motor Evoked potential for each session was applied. Frequency given to each patient ranges between 20

Hz for 20 mins (total 06 sessions, once per week). Vitals of each patient was monitored pre and post session. Each patient as given a visual analogue scale (VAS) at end of each session and at the end of six weeks. Score of each session was recorded at end of each session and at end of 06 weeks. All patients were followed up for 6 weeks post rTMS for any procedure complications.

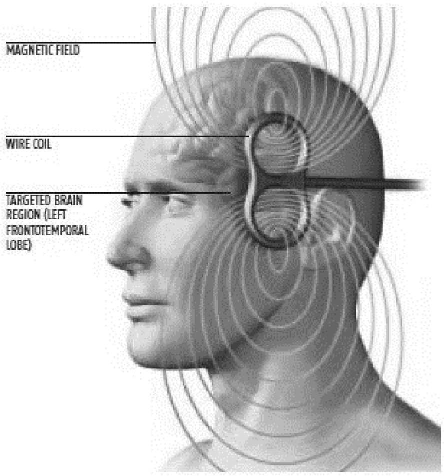

Figure 1. (left) / **Figure 2**. (right)

2.3 Ethical Clearence: (certificate enclosed) was taken by institutional ethical committee and all participants consent was obtained prior to study

Table 1. Pre and Post rTMS VAS

	Group							
	Test group			Sham group				
	Mean	Median	Std. Deviation	Mean	Median	Std. Deviation	p Value	Significance
Pre Procedure VAS	10.00	10.00	0.00	10.00	10.00	0.00	1.000	Not Significant
Post Procedure VAS	7.17	8.00	1.72	8.55	9.00	0.82	<0.001	Significant
Change in VAS	2.83	2.00	1.72	1.45	1.00	0.82	<0.001	Significant
% Change in VAS	28.26	20.00	17.23	14.50	10.00	8.20	<0.001	Significant

The pre procedure VAS was 10 in both the Test and Sham Group however the Post procedure VAS obtained after 04 sessions of rTMS statistically significant reduction in LBP with change in Mean VAS score of 28.26% in Test group compared to no change in Sham group (Table 1.).

Table 2. Change in VAS

		Group			
		Test group	Sham group	p Value	Significance
% Change in VAS	NO CHANGE	0(0)	02(10)	<0.001	Significant
	CHANGE <=30%	12(60%)	18(90)		
	CHANGE >30%	8(40%)	0(0)		
Total		20(100)	20(100)		

In our study we found that all patients in test group reported a reduction in VAS score in which 08 patients (40%) reported ≥30 percent reduction and 12 patients(60%) reported around 20% reduction in pain scores. However, 90 percent subjects in sham group reported a marginal improvement in VAS score which can be attributed to placebo effect. Most patients in test group reported an improvement in quality of life (Table 2.)

Table 3. Post Procedure Complications

		Group		Total		
		Test group	Sham group		p Value	Significance
Complication	Headache	2(10)	1(5.2%)	3(8.1%)	0.577	Not Significant
	NONE	18(90)	19(94.8%)	37(91.9%)		
Total		20(100)	20(100)	40(100)		

Post procedure 02 patients (10%) in test and 01 Patient (5.2%) in Sham group reported with mild episodic headache which relieved with analgesics without requirement of further management (Table-3).

Table 4. Related studies on rTMS effect on CLBP

Author	Year	Subject	Sample Size	rTMS Protocol	Outcome
Ambriz et al [8]	2016	CLBP	n=82,rTMS-44,Sham-12, PT-26	10biphasic pulse/10sec at 20 Hz	Significant reduction in pain score
Lee et al [9]	2019	CLBP	n=21,rTMS-11, Sham-10	10 Mins, 10 Hz	Improvement in pain score in Test group
Olechowski et al [10]	2020	CLBP	n=20	20 mins, 10 hz	Long term improvement in Pian
Masoumbeigi et al [11]	2021	CLBP	n=09	40 Pulse, 28 sec rest period, 20 Hz	Reduction in VAS Score
Present Study	2023	CLBP	n=40, rTMS-20, Sham-20	1 session per week, 20 hz for 20 mins, Total 04 sessions	Statistically significant reduction in Post procedure VAS score

3. Statistical Analysis

The statistical software SPSS version 25 has been used for the analysis. Continuous variables are expressed as Mean, Median and Standard Deviation and compared across the groups using Mann-Whitney U test since the data does not follow normal distribution. Categorical variables are expressed as Number of patients and percentage of patients and compared across the groups using Pearson's Chi Square test for Independence of Attributes/ Fisher's Exact Test as appropriate. An alpha level of 5% has been taken, i.e. if any p value is less than 0.05 it has been considered as significant.

4. Results

The demographic profile was comparable in both the groups with mean age is 39.26 and 39.05 in Test & Sham group respectively. In test group(n=20) 5 patients were female (21.74%) and 15 were male (78.26%) where as in Sham group(n=20) 7 Patients were female (26.67%) and 13 were male (73.33%).

5. Discussion

TMS is based on the Faraday law. As per recent understanding an electromagnetic coil sends a perpendicular magnetic field into the region of interest on the scalp. The mechanism of cortical stimulation for pain relief is believed to be due to the modification of neuronal excitability. rTMS is hypothesized to induce alterations in the activity of cortical and subcortical brain structures that are related to pain modulation and processing, including the orbitofrontal cortices, medial thalamus, anterior cingulate, and periaqueductal gray matter. Also, rTMS is hypothesized to reduce chronic pain by triggering descending inhibitory neural pathways which acts at dorsal-horn level. Other proposed mechanism of action include increase in cerebral blood flow to the affected areas as it is hypothesized that patients with chronic pain have a decreased blood flow(12).. Broadly, rTMS has been classified, High frequency (>1 Hz) and Low frequency (<5Hz).[6] Stimulation frequency is associated with synaptic changes; higher frequencies (> 5 Hz) are excitatory, and lower frequencies (< 1 Hz) are inhibitory.[7] Numerous studies have established that active high-frequency rTMS (HF-rTMS) ranging between 5 Hz to 20 Hz has analgesic effects in management of chronic pain.[12,13] Findings of this study is consistent with results of previous studies which showed a significant reduction in pain score in patients undergoing rTMS Repetitive transcranial magnetic stimulation can be performed as an outpatient procedure for chronic low back pain. At present there are few studies which have evaluated efficacy of rTMS in patients with CLBP.

6. Conclusions

Based on our study we conclude that rTMS is a safe procedure and it can be used as a modality in treating CLBP with satisfactory outcomes. Although the sample size was small, it is the first study of its kind undertaken in India to evaluate the role of rTMS based neuromodulation in treating this chronic disabling condition. How ever to be accepted as a standard of care for CLBP it will require multi-institutional robust clinical trials with long term follow up.

Limitations: Single centre and single blinded study with small sample size.The reproducibility of results post rTMS can be further studied with multicenter study on larger population and long term follow up is required to observe for late onset complication if any.

References

1. Vlaeyen JWS, Maher CG, Wiech K, et al. Low back pain. Nat Rev Dis Primers. 2018;4(1):53
2. Hestbaek L, Leboeuf-Yde C, Manniche C. Low back pain: what is the long-term course? A review of studies of general patient populations. Eur Spine J. 2003;12(2):149–165. doi: 10.1007/s00586-002-0508-5
3. Van Tulder M, Becker A, Bekkering T, et al. Chapter 3. European guidelines for the management of acute nonspecific low back pain in primary care. Eur Spine J. 2006;15(Suppl 2):S169–91
4. Yates E, Balu G. Deep Transcranial Magnetic Stimulation: A Promising Drug-Free Treatment Modality in the Treatment of Chronic Low Back Pain. Delaware Medical Journal. 2016 Mar 1;88(3):90-2.
5. Chen M, Deng H, Schmidt RL, Kimberley TJ. Low-frequency repetitive transcranial magnetic stimulation targeted to premotor cortex followed by primary motor cortex modulates excitability differently than premotor cortex or primary motor cortex stimulation alone. Neuromodulation. 2015;18:678–85.
6. Berlim MT, Van den Eynde F, Jeff Daskalakis Z. Clinically meaningful efficacy and acceptability of low-frequency repetitive transcranial magnetic stimulation (rTMS) for treating primary major depression: A meta-analysis of randomized, double-blind and sham-controlled trials. Neuropsychopharmacology. 2013;38:543–51
7. Ambriz-Tututi M, Alvarado-Reynoso B, Drucker-Colín R. Analgesic effect of repetitive transcranial magnetic stimulation (rTMS) in patients with chronic low back pain. Bioelectromagnetics. 2016 Dec;37(8):527-535. doi: 10.1002/bem.22001. Epub 2016 Aug 22. PMID: 27548757.
8. Lee YS, Bae SH, Kim KY. Effects of repetitive transcranial magnetic stimulation on psychological aspects of patients with chronic lower back pain. Journal of Magnetics. 2019 Sep;24(3):543-8.
9. Olechowski C, Gener M, Aiyer R, Mischel N. Transcranial magnetic stimulation for the treatment of chronic low back pain: a narrative review. Frontiers in Pain Research. 2023 May 5;4:1092158.Latina R, Sansoni J, D'Angelo D, Di Biagio E, De Marinis MG, Tarsitani G. Etiology and prevalence of chronic pain in adults: A narrative review. Prof Inferm. 2013;66:151–8
10. Masoumbeigi M, Alam NR, Kordi R, Rostami M, Afzali M, Yadollahi M, Rahimiforoushani A, Jafari AH, Hashemi H, Kavousi M. rTMS pain reduction effectiveness in non-specific chronic low back pain patients using rs-fMRI functional connectivity. Journal of Medical and Biological Engineering. 2022 Oct;42(5):647-57. Knijnik LM, Dussán-Sarria JA, Rozisky JR, Torres IL. Repetitive transcranial magnetic stimulation for fibromyalgia: Systematic review and meta-analysis. Pain Pract. 2016;16:294–304.
11. Min Cheol Chang, Sang Gyu Kwak, and Donghwi Park The effect of rTMS in the management of pain associated with CRPS. Transl Neurosci. 2020; 11(1): 363–370.
12. Xue Jiang , Wangwang Yan , Ruihan Wan Effects of repetitive transcranial magnetic stimulation on neuropathic pain: A systematic review and meta-analysis Neuroscience & Biobehavioral Reviews Volume 132, January 2022, Pages 130-14.
13. Kliment Gatzinsky , Christina Bergh , Ann Liljegren , Hans Silander Repetitive transcranial magnetic stimulation of the primary motor cortex in management of chronic neuropathic pain: a systematic review Scand J Pain 2020 Sep 7;21(1):8-21.

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Colloid cyst of the third ventricle. Experience of total endoscopic excision in 7 cases

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ABSTRACT

Background: In the past, microsurgical resection was considered the gold standard treatment for colloid cysts. The endoscopic approach is gaining popularity and has been recognized as a safe and effective alternative to open surgery.

Aims**:** To evaluate our results, safety and efficacy of the endoscopic approach

Materials and methods: This retrospective study included seven patients with colloid cysts who underwent endoscopic resection between May 2020 and April 2022. Patient records, radiological images, and surgical notes were assessed. Follow-up data, including clinical and radiological details, were retrieved. Postoperative Computed tomography (CT) was performed, and magnetic resonance imaging (MRI) was performed in all patients.

Result: Seven patients aged range 27-56 years 4 males and 3 females underwent endoscopic resection of the tumor during the study period. All patients presented with headaches. The mean diameter of the cyst was 10.6(range 8 -14mm), and the mean operating time was 126(range 100 -180 min). All patients underwent the single burr hole and single port technique. Six patients underwent transforaminal surgery and one patient underwent trans-septal corridor. GTR was achieved in six patients. One patient underwent near-total resection with coagulation of the capsule. None of the endoscopic procedures was converted to open resection. No patient had a recurrence, and the mean follow-up period was 24.3 (range 16 to 36 months). There were no deaths during the follow-up period. No residual cysts were observed on postoperative MRI in any patient.

Conclusion: Endoscopic excision of colloid cysts is an effective and safe alternative method. Although the follow-up time was short, the residual cyst wall remained asymptomatic without any evidence of growth after near excision and coagulation of the wall.

Keywords
colloid cyst,
endoscopy,
gross total excision,
near-total excision,
transforaminal

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INTRODUCTION

Colloid cysts are rare benign tumors arising from the third ventricular roof attached to the tela choroidea, in proximity to the interventricular foramen of Monro (FOM), and are believed to have a neuroectodermal origin.[1-3] These cysts are surrounded on either side by pillars of the fornix. These lesions are often incidentally found on imaging performed to evaluate various neurological symptoms such as headache, nausea, and/or vomiting; hence, it has been difficult to define management protocols for these lesions. In most cases, these lesions remain silent and the patient remains asymptomatic; however, because of their close proximity to FOM, they can clinically present with the features of obstructive hydrocephalus, rapid clinical deterioration, and in severe cases sudden death [4-5].

On imaging, cysts are well defined, smooth, spherical ovoid masses filled with gelatinous and mucinous fluid. Most cysts are hyperdense on CT. MRI is the preferred modality to investigate colloid cysts and may have variable appearances, with approximately two-thirds of cysts appearing hyperintense on T1. Most cysts appear isointense to hypointense on T2/FLAIR with slight rim enhancement on contrast and do not exhibit diffusion restriction.

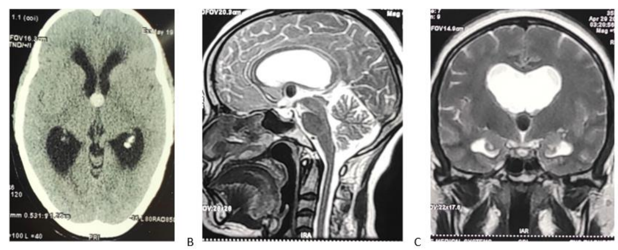
The location of the tumour in the centre and in close proximity to critical neurovascular structures makes surgical management technically difficult. In asymptomatic patients, observation with serial neuroimaging can be performed, and in a few cases, spontaneous resolution of colloid cysts has been reported in the literature [7-9]. Symptomatic cases require surgery as soon as possible after cyst-related symptoms have been identified. Patients with acute symptoms and rapid neurological decline associated with these lesions should undergo CT immediately, and depending on the presence of hydrocephalus, bilateral EVDs should be placed before surgery. Various surgical modalities, such as bilateral shunting in acute cases, stereotactic cyst aspiration in few cases, transcranial approaches, including transcortical and transcallosal approaches, and the recent endoscopic approach to cyst excision [10-12]

Dandy in 1921, using a posterior transcallosal approach, successfully removed colloid cyst [6]

Both microsurgical techniques; "transcortical" and "transcallosal" combined to their different extensions through the choroidal fissure can be used to access this deep part within the diencephalic region without significant morbidity or mortality [13].

2. Material and methods

The current work is a retrospective analysis of patients who underwent endoscopic colloid cystectomy of the third ventricle in a tertiary care hospital during a 2-year period from May 2020 to April 2022. A total of 7 patients were included. Files were reviewed for preoperative data such as patient demographic age, gender, clinical manifestations, and radiological data. Radiological data included pre- and postoperative computed tomography (CT) and magnetic resonance imaging (MRI) of the brain to determine cyst size, site, and density, as well as the status of ventricle dilatation (Fig 1). Intraoperative difficulty and extent of resection were noted. All patients were followed up clinically at 6 weeks, 3 months, and 6 months after discharge and then yearly. Postoperative MRI was performed after 3 months.



A B C

Fig 1 : Preoperative images showing a well circumscribed ovoid hyperdense lesion at the foramen of monro in a computed tomography scan (A) and hypointense lesion located near the foramen of Monro in the anterior third ventricle with mild hydrocephalus in the magnetic resonance imaging (B and C).

Endoscopic approach

The coronal images of the MRI were helpful in deciding the side of approach, and in our study, all patients were approached from the right lateral ventricle. Careful study of the sagittal and coronal MR images was conducted to define the relationship of the cyst with respect to the ventricular structures. After undergoing general anesthesia, the patients were placed in the supine position on a doughnut with head elevation of 10°–30°. Mid-sagittal MRI and neuro-navigation were used to plan the burr hole placement and endoscopic trajectory for optimal visualization of the colloid cyst. It was paramount to target the center of the cyst while avoiding the fornix, septal, and internal cerebral veins. Generally, the burr hole corresponded to 3-4 cm away from midline and 3-4 cms anterior to the coronal suture in the precoronal area.

The surgery was performed using rigid 0-degree endoscopes (Karl Storz, Tuttlingen, Germany with a 6.5 mm outer diameter. A single port was used in all patients. After coagulating the underlying pia with monopolar cautery, free hand cannulation of the lateral ventricle was performed using an endoscopic sheath and obturator. The obturator was removed during lateral ventricle cannulation, and an endoscope was introduced to visualize anatomical landmarks such as the foramen of Monro (fig 2), thalamostriate vein, septal vein, and choroid plexus. In most cases, the colloid cyst bulged through the foramen of Monro into the ipsilateral lateral ventricle. The cyst capsule was coagulated, and using a thin catheter through the neuroendoscope working channel, the cyst contents were aspirated, followed by cyst excision. Meticulous hemostasis was achieved in all cases, and wound closure was performed after insertion of the surgical gel foam. During the postoperative period, all patients received antibiotics according to the institutional protocol. Plain CT was performed on the same day to rule out any hematoma. The ventricular drain, if placed, was first clamped and then removed after 24 h when the CSF became clear.

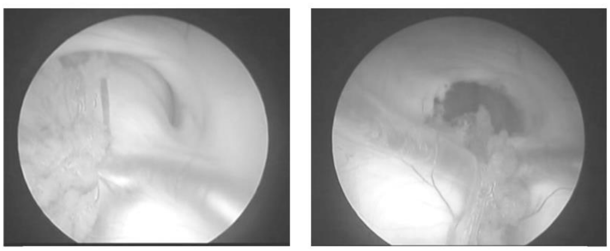


Fig 2: Endoscopic resection of the colloid cyst seen at the foramen of monro (A) before and (B) after total excision

Results

This was a retrospective study of seven patients (4 males and 3 females), with age range 26 -57 years (table 1). Headache was present in all patients. Other symptoms included vomiting in 4, blurred vision in 1, and urinary incontinence in one patient.

The mean cyst diameter was 10.6 mm (range8-14mm). All colloid cysts were associated with hydrocephalus. All patients underwent right-sided burr hole. Six patients underwent transforaminal and one underwent trans septal approach. GTR was achieved in six patients, whereas one patient had near total excision of the cyst with coagulation of the nubbin of the cyst wall.

The mean operating time for the procedure was 126 min (range 100 -160 mins). Septostomy to gain biventricular access was performed in one patient. At the end of the procedure, while separating the cyst from the fornices, intraoperative bleeding was the most common intra operative event and was controlled by prolonged irrigation with Ringer's Lactate solution. Intraventricular drains were used in 3 patients with minor persistent ooze and removed 24 hrs after surgery. All patients underwent post operative CT scan on same day to rule out any hematoma. Following surgery, headache improved in all patients. Vomiting, blurring of vision, and urinary incontinence improved in all. The postoperative hospital stay ranged from 4 to 8 days. The mean follow-up was 24.3 months (range 12- 36). Follow-up imaging was available in all patients, and none of the patient had residual cyst (fig 3). They are under regular follow-up and were asymptomatic till the last visit.

Table 1.

Serial no	Age (yrs)/sex	Size of cyst (mm)	Associated hydrocephalus	Surgical excision	Operative time(min)	Follow up(months)	Outcome
1	34 /m	12	yes	Total excision	140	12	improved
2	27 /f	9	yes	Total excision	120	30	improved
3	50 /m	11	yes	Total excision	110	24	improved
4	43 /f	14	yes	Near total excision	160	28	improved
5	28 /m	10	yes	Total excision	130	22	improved
6	56 /f	8	yes	Total excision	100	36	improved
7	36 /m	10	yes	Total excision	120	18	improved

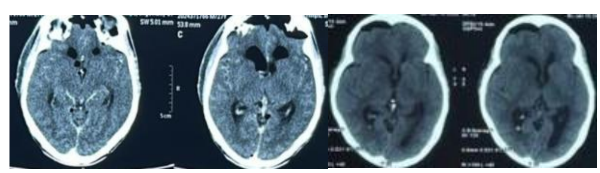


Fig 3 : Post operative images of 2 patients showing gross total excision of the colloid cyst by right side trans foraminal route

Discussion

The best modality to treat this benign disease is debatable, and while deciding the surgical approach, one has to consider the risks associated with radical surgical resection against the chance of recurrence. The endoscopic approach has proven to be a good alternative to microsurgical techniques with good long-term results.[16-17] We were able to manage all the cases with a single port, but some authors have used a bi-port technique.[18] We also used neuro navigation in all of our cases as an intraoperative adjunct because it helped us to better determine the burr hole site and trajectory. It is paramount to study the preoperative images to define the position of the cyst in relation to the foramen of Monro and the roof of the third ventricle.

Hoffman et al. (27) reported that the sensitivity of postoperative MRI is not sufficient and that it is imperative to consider direct intraoperative inspection to accurately predict the extent of resection. The major reason supporting this finding is that cyst wall remnants identified intraoperatively do not necessarily appear on postoperative imaging.

In our study, we also considered the intraoperative completeness of cyst excision and cyst wall with no observable portion of cyst wall to consider GTR. We achieved a GTR in 6 (85.7%) cases, which is similar to other published studies, such as that achieved in 78% patients by Mishra et al [18]; and in 96.9% patients by Engh et al.[24] In our study, no patient required conversion to open craniotomy because of excessive bleeding or poor visualization of the colloid cyst, which proves the safety and applicability of the endoscopic technique. Most cases of bleeding were easily controlled with Ringer's lactate solution. The average hospital stay in our study was 6.2 days; the average operative time was 126 min, which is similar to other similar studies. There were no deaths in our series; similar results were observed in other series[19-23]. There was no recurrence during the follow-up period.

Conclusion

Thought the endoscopic colloid cyst excision requires a steep learning curve and radical resection demands certain degree of technical skill and experience with the endoscope it is a very effective and safe method. Although the limitation of our study is short follow up period the residual cyst wall remained asymptomatic without any evidence of growth after near total excision and coagulation of the wall.

References

1. Barbagallo GM, Raudino G, Visocchi M, Maione M, Certo F. Out-of-third ventricle colloid cysts: review of the literature on pathophysiology, diagnosis and treatment of an uncommon condition, with a focus on headache. Journal of Neurosurgical Sciences. 2016 Sep 7;63(3):330-6.
2. Kabashi A, Dedushi K, Ymeri L, Ametxhekaj I, Shatri M. Colloid cyst of the third ventricle: case report and literature review. Acta Informatica Medica. 2020 Dec;28(4):283.
3. Iacoangeli M, di Somma LG, Di Rienzo A, Alvaro L, Nasi D, Scerrati M. Combined endoscopic transforaminal-transchoroidal approach for the treatment of third ventricle colloid cysts. Journal of neurosurgery. 2014 Jun 1;120(6):1471-6.
4. de Witt Hamer PC, Verstegen MJ, De Haan RJ, Vandertop WP, Thomeer RT, Mooij JJ, van Furth WR. High risk of acute deterioration in patients harboring symptomatic colloid cysts of the third ventricle. Journal of neurosurgery. 2002 Jun 1;96(6):1041-5.
5. Chan RC, Thompson GB. Third ventricular colloid cysts presenting with acute neurological deterioration. Surgical neurology. 1983 Apr 1;19(4):358-62.
6. MES. Benign Tumors of the Third Ventricle of the Brain: Diagnosis and Treatment.
7. Pollock BE, Huston J. Natural history of asymptomatic colloid cysts of the third ventricle. Journal of neurosurgery. 1999 Sep 1;91(3):364-9.
8. Turel M, Kucharczyk W, Gentili F. Spontaneous resolution of colloid cyst of the third ventricle: Implications for management. Asian journal of neurosurgery. 2017 Jun;12(02):2036.
9. Peeters SM, Daou B, Jabbour P, Ladoux A, Abi Lahoud G. Spontaneous regression of a third ventricle colloid cyst. World neurosurgery. 2016 Jun 1;90:704-e19.
10. Deinsberger W, Böker DK, Bothe HW, Samii M. Stereotactic endoscopic treatment of colloid cysts of the third ventricle. Acta neurochirurgica. 1994 Sep;131:260-4.
11. Mathiesen T, Grane P, Lindquist C, von Holst H. High recurrence rate following aspiration of colloid cysts in the third ventricle. Journal of neurosurgery. 1993 May 1;78(5):748-52.
12. Kondziolka D, Lunsford LD. Stereotactic management of colloid cysts: factors predicting success. Journal of neurosurgery. 1991 Jul 1;75(1):45-51.
13. Nair S, Gopalakrishnan C, Menon G, Easwer H, Abraham M. Interhemispheric transcallosal transforaminal approach and its variants to colloid cyst of third ventricle: technical issues based on a single institutional experience of 297 cases. Asian journal of neurosurgery. 2016 Sep;11(03):292-7.
14. Hodges JR, Carpenter K. Anterograde amnesia with fornix damage following removal of IIIrd ventricle colloid cyst. Journal of Neurology, Neurosurgery & Psychiatry. 1991 Jul 1;54(7):633-8.
15. Beaumont TL, Limbrick DD, Rich KM, Wippold FJ, Dacey RG. Natural history of colloid cysts of the third ventricle. Journal of neurosurgery. 2016 Dec 1;125(6):1420-30.
16. Hellwig D, Bauer BL, Schulte M, Gatscher S, Riegel T, Bertalanffy H. Neuroendoscopic treatment for colloid cysts of the third ventricle: the experience of a decade. Neurosurgery. 2008 Jun 1;62(6):SHC1101-9.

17. 17 . Maqsood AA, Devi IB, Mohanty A, Chandramouli BA, Sastry KV. Third ventricular colloid cysts in children. Pediatric neurosurgery. 2006 Apr 21;42(3):147-50.
18. Mishra S, Chandra PS, Suri A, Rajender K, Sharma BS, Mahapatra AK. Endoscopic management of third ventricular colloid cysts: eight years' institutional experience and description of a new technique. Neurology India. 2010 May 1;58(3):412-7.
19. Decq P, Le Guerinel C, Brugières P, Djindjian M, Silva D, Kéravel Y, Melon E, Nguyen JP. Endoscopic management of colloid cysts. Neurosurgery. 1998 Jun 1;42(6):1288-94.
20. 20 . Longatti P, Martinuzzi A, Moro M, Fiorindi A, Carteri A. Endoscopic treatment of colloid cysts of the third ventricle: 9 consecutive cases. min-Minimally Invasive Neurosurgery. 2000 Sep;43(03):118-23.
21. Acerbi F, Rampini P, Egidi M, Locatelli M. Endoscopic treatment of colloid cysts of the third ventricle: long-term results in a series of 6 consecutive cases. Journal of neurosurgical sciences. 2007 Jun 1;51(2):53.
22. Horn EM, Spetzler RF. Treatment options for third ventricular colloid cysts: comparison of open microsurgical versus endoscopic resection. Neurosurgery. 2008 Jun 1;62(6):E1384.
23. Greenlee JD, Teo C, Ghahreman A, Kwok B. Purely endoscopic resection of colloid cysts. Operative Neurosurgery. 2008 Mar 1;62(3):51-6.
24. Engh JA, Lunsford LD, Amin DV, Ochalski PG, Fernandez-Miranda J, Prevedello DM, Kassam AB. Stereotactically guided endoscopic port surgery for intraventricular tumor and colloid cyst resection. Operative Neurosurgery. 2010 Sep 1;67(3):ons198-205.
25. 25 Boogaarts H, El-Kheshin S, Grotenhuis J. Endoscopic colloid cyst resection. min-Minimally Invasive Neurosurgery. 2011 Apr;54(02):95-7.
26. Decq P, Le Guerinel C, Brugières P, Djindjian M, Silva D, Kéravel Y, Melon E, Nguyen JP. Endoscopic management of colloid cysts. Neurosurgery. 1998 Jun 1;42(6):1288-94.
27. Hoffman, Caitlin E. MD*; Savage, Nicole J. BS*; Souweidane, Mark M. MD†‡. The Significance of Cyst Remnants After Endoscopic Colloid Cyst Resection: A Retrospective Clinical Case Series. Neurosurgery 73(2):p 233-239, August 2013. | DOI: 10.1227/01.neu.0000430300.10338.71

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Evaluation of the need for debulking in meningioma surgery by a retrospective analysis of our case series. Is debulking mandatory?

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ABSTRACT

Introduction: The primary aim of meningioma surgery should be total excision. However, subtotal excision is considered first if the main vascular structures and the cranial nerves are at risk. The 'primum non nocere' principle should always be considered. A clinical study where the emphasis is on en bloc resection without debulking in suitable cases, in contrast to the classic approaches, could therefore prove useful.

Method: The meningioma cases operated at the İzmir Bakırçay University Çiğli Training and Research Hospital's Neurosurgery Clinic between 2021 and 2023 were retrospectively evaluated. There were no exclusion criteria. The demographic features of the patients who had undergone debulking or en bloc resection and the anatomical, pathological, surgical and clinical features of the meningiomas were evaluated and the results were presented as percentages.

Results: A total of 21 patients, consisting of 5 males (23.8%) and 16 females (76.2%), were included in the study. The mean age was 58.8 (28-90) years. En bloc resection was performed in 18 (85.7%) and debulking in 3 (14.3%) of the 21 cases. The other results are presented within the article.

Conclusion: En bloc resection could be an important surgical strategy to decrease the surgical duration and bleeding amount in appropriate cases.

Keywords
meningioma,
surgery,
en bloc resection

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INTRODUCTION AND BACKGROUND

Meningiomas constitute 36.6% of all primary central nervous system (CNS) tumors and 53.2% of benign CNS tumors. The general incidence of meningioma is 8.3 per 100,000 people. There is a female dominance of 2.3:1. Besides 81.1% of meningiomas reported as grade 1 (typical), 16.9% as atypical, and 1.7% as anaplastic (8). Ionizing radiation to the skull is considered a risk factor for meningioma development and increases the relative risk six to ten times following a variable latent period without a clear dose-response relationship (9). Epidemiological factors such as a history of head injury, smoking, and cell phone use have not been consistently shown to be associated with an increased risk of meningioma (8). The most common inherited cause is neurofibromatosis type 2 (NF2), which is characterized by autosomal dominant inheritance. Phenotypic NF2 is rare in the general population and occurs in <1% of meningioma cases (10). Other important meningioma-related disorders include the Li-Fraumeni, Gorlin, and von Hippel-Lindau syndromes, Cowden's disease, and multiple endocrine neoplasia (MEN) type 1(11).

Meningioma surgical strategies have played an important role in the development of neurosurgery. Meningioma surgery has always been one of the favorite areas of the discipline. The general principles are complete removal of the tumor, or reducing its volume to a level that eliminates mortality/morbidity if this is not possible; choosing the shortest and safest route for the surgical approach; preserving important vascular structures and neural tissue; and concluding the surgery within a reasonable time. As in every medical intervention, the principle of 'primum non nocere' should always be considered during the surgery. Taking all these into account, we believe it may be useful to present a different perspective that includes en bloc resection for meningioma surgery through our own case series.

Material and Method

Meningioma cases operated in İzmir Bakırçay University Çiğli Training and Research Hospital's Neurosurgery Department between 2021 and 2023 were retrospectively analyzed. The study has ethics committee approval from İzmir Bakırçay University with decision number 1197. We have no conflict of interest about this study. The study inclusion criteria were 'cases that had undergone meningioma surgery', with no exclusion criteria. The cases were evaluated in terms of the patient age and gender, and the meningioma anatomical origin, size (in mm), hemispheric side (right/left), Simpson resection grading, pathology, debulking/en bloc resection technique, Glasgow outcome score, surgical duration, and arterial penetration of the meningioma. The meningiomas were also divided into four groups as Type 1 or small (< 20 mm), Type 2 or moderate (20-39 mm), Type 3 or large (40-59 mm), and Type 4 or giant (>60 mm). Based on these four types, the debulk/en bloc resection technique was then evaluated.

The Simpson resection grade rates of the tumors resected with the debulking/en bloc technique was calculated. Besides, the success of the en bloc resection technique was determined in cases with arterial penetrance. Finally, the en bloc resection rates of moderate and large meningiomas were calculated. All data were evaluated as percentages.

Results

There were 21 patients, consisting of 5 males (23.8%) and 16 females (76.2%), in the study. The mean age was 58.8 (28-90) years. There were 2 spheno-frontal meningiomas (9.5%), 8 convexity meningiomas (38%), 1 tentorial meningioma (4.8%), 2 parasagittal meningiomas (9.5%), 1 falx meningioma (4.8%), 2 lateral sphenoid wing meningiomas (9.5%), 2 medial sphenoid wing meningiomas (9.5%), 1 tuberculum sella meningioma (4.8%), 1 spheno-Sylvian meningioma (4.8%), and 1 thoracic meningioma (4.8%). When evaluated by size, none of the Type 1 'small' size meningiomas were operated on. There were 10 (47.6%) type 2 'moderate' size meningiomas that underwent surgery, 9 (90%) by resection en bloc while debulking was performed in 1 (10%) meningioma of tentorial origin. There were 10 (47.6%) type 3 'large' size meningiomas that underwent surgery with 8 (80%) resected en bloc and 2 (20%) with debulking.

A total of 2 (100%) medial sphenoid wing meningiomas were also resected by debulking. One type 4 'giant' size meningioma was operated on. The Simpson grade was 2 in 18 (100%) patients that underwent en bloc resection, and 2 in 2 (9.5%) patients and 4 in 1 (4.8%) patient that underwent debulking. The pathology result of 4 (19%) of the 21 patients was 'atypical meningioma'. The Glasgow outcome score was 5 in 20 (95.2%) patients and 4 in 1 (4.8%) debulked medial sphenoid wing meningioma. Penetration with important arterial structures was detected in 7 (33.3%) of the 21 cases; en bloc resection was performed in 5 (71.4%) and debulking in 2 (28.6%) of these 7 cases. The number of Type 3 'large' and Type 4 'giant' meningiomas was 11, and en bloc resection was performed in 9 (81%) of them.

Discussion

The classic disciplines in meningioma surgery focus on four basic phases of excision: 1. Devascularization, 2. Detachment from the origin, 3. Debulking and 4. Dissection (1,7). These phases may not always have a sequential relationship during surgery. A partial circumferential dissection can be performed before devascularization, or the debulking phase can precede all these stages. The 360-degree dissection can first be slightly advanced a little deeper by preserving the tumor form. This is related to the relaxation state of the tumor and brain at that moment and the surgeon's perspective on the current clinical picture. For example, the feeding with blood of a falx meningioma may be partially impaired after dural opening. The devascularization and detachment from the falx cerebri, the area to which the tumor is attached, then occur simultaneously. Meanwhile, the dissection proceeds deeper into the falx. Following full devascularization and detachment, the classic discipline recommends moving to the 'debulking' phase.

	Age	**Gender**	**Origin**	**Size (mm)**	**Side**	**Simpson**	**Pathology**	**Debulking**	**En bloc**	**GOS**	**OT**	**AP**
1	76	M	Spheno-frontal	45x45x53	Left	2	Atypical	No	Yes	5	120	-
2	28	F	Parietal Convexity	30x35x24	Right	2	MM	No	Yes	5	90	-
3	44	F	Tentorial	30x30x25	Left	2	Fibrous	Yes	No	5	150	-
4	57	M	Temporal Convexity	25x25x25	Left	2	Transitional	No	Yes	5	100	-
5	54	M	Frontal Convexity	30x51x35	Right	2	Atypical	No	Yes	5	100	-
6	52	F	Parasagittal	40x52x44	Right	2	Transitional	No	Yes	5	140	-
7	39	F	Parietal Convexity	25x24x25	Left	2	Transitional	No	Yes	5	60	-
8	74	F	Frontal Convexity	40x42x32	Right	2	Atypical	No	Yes	5	90	-
9	90	M	Parasagittal	42x41x40	Right	2	Transitional	No	Yes	5	120	-
10	44	F	Spheno-frontal	30x30x30	Left	2	Transitional	No	Yes	5	90	-
11	63	F	Frontal Convexity	56x61x55	Left	2	Transitional	No	Yes	5	120	-
12	59	F	Falx	33x31x33	Left	2	MM	No	Yes	5	120	+
13	50	F	Lateral sphenoid wing	27x25x25	Right	2	MM	No	Yes	5	90	+
14	65	F	Spheno-Sylvian	33x25x34	Left	2	Transitional	No	Yes	5	80	+
15	52	F	Tuberculum sella	30x24x20	-	2	MM	No	Yes	5	150	+
16	65	F	Thoracic	30x20x20	-	2	Psammomatous	No	Yes	5	120	-
17	80	F	Frontal Convexity	33x47x42	Right	2	Atypical	No	Yes	5	90	-
18	67	F	Lateral sphenoid wing	40x40x42	Right	2	Angiomatous	No	Yes	5	120	+
19	56	M	Frontal Convexity	56x55x58	Right	1	MM	No	Yes	5	120	-
20	67	F	Medial sphenoid wing	45x52x46	Left	4	MM	Yes	No	4	180	+
21	52	F	Medial sphenoid wing	46x35x52	Left	2	MM	Yes	No	5	150	+

GOS: Glasgow Outcome Score, OT: Operation Time (minutes), AT: Arterial Penetrance, MM: Meningotheliomatous

The debulking phase means removing the meningioma tissue piece by piece while making sure it remains inside the tumor capsule. The aim here is to facilitate dissection by allowing the capsule to tip towards the center in large meningiomas. By performing a circumferential dissection, the tumor capsule is released and removed. The debulking phase is often performed by using an ultrasonic aspirator or a monopolar cauter with a ring-shaped tip. Our observations have shown that the debulking duration for a meningioma of approximately 4 cm can be up to 60 minutes. When debulking is performed, all tumoral tissue up to the tumor capsule must be removed so that the capsule can become flexible and be bent. This procedure takes quite a long time.

The issue I want to discuss in this article is not whether the debulking phase facilitates tumor removal or not but whether it is absolutely necessary. This stage is emphasized in all written and visual material, resulting in it being perceived as indispensable among trained surgeons. Tumor debulking was actually required in only 14.2% of our cases. Briefly stated, our strategy starts with opening the dura after dural coagulation. Then, we place our cotton pads around the tumor following limited dissection so that the borders are clear. We then proceed according to the source of the meningioma. For example, we proceed to the falx connection in falx meningiomas, and the tubercle connection in tuberculum sella meningiomas.

A detachment is secured both by burning the connection towards the tumor with bipolar cautery as well as with the support of a cotton pad, and the dissection is advanced deeper at the same time. Devascularization and tumor shrinkage are ensured at this stage. We then place cotton pads on the dissection area, paying attention to important vascular structures and preserving the arachnoid plane. The dissection is deepened in the areas where we are sure of the dissection, and the tumor tissue is reduced by burning it towards the center with bipolar cautery. We also make small maneuvers that mobilize the tumor from its place during the burning procedure.

Burning should not be performed without understanding the relationship of the main vascular structures with the tumor, and any forceful maneuvers should be avoided during dissection. At the same time, vascular structures and cranial nerves adherent to the tumor capsule should be patiently and carefully separated from the capsule. If the tension in the tumor capsule during these vital dissections will cause difficulty in separating these structures from the capsule, we always proceed to the debulking procedure. The vascular structures should be separated from distal to proximal by careful dissection that is as sharp as possible following the debulking. Sharp dissection is the key (3).

The arachnoid plane to be cut must be distinguished beforehand, and sharp dissection must be continued with small moves. Otherwise, a vascular injury that will develop, for example in a sphenoid wing meningioma adherent to the MCA branches, may cause serious morbidity and mortality. We continue the circumferential dissection until the tumor tissue is completely released from all its attachments. Finally, and only after full release is achieved, the tumor tissue is removed en bloc. Another important point is that the vascular penetration area is determined exactly if we come across the main vascular structures when turning around the periphery of the tumor or if the main vascular structures providing cerebral perfusion are penetrating into the tumor.

The dissection is continued by skipping this section in this case. The next thing to do after fully turning around the tumor is to follow the vascular structure. This is possible with controlled incisions and partial resections in the tumor tissue in that area. I believe it is not correct to call resection of the capsule together with the pieces debulking. I believe that resection with this strategy is more controlled than with debulking in cases where the main vascular structures are surrounded by the tumor. However, debulking should be performed very carefully if the distal or proximal section of vascular penetration cannot be detected and tumor removal has to be continued.

Radiological results should be thoroughly evaluated before the surgery. Film footage should be available during surgery. The use of neuro-navigation may also be beneficial. The circumferential dissection should be deepened in a controlled manner and should not cause tension in the vascular structure. Gross total resection should be decided on by considering the profit-loss relationship. The principle of 'primum non nocere' should always be considered. For example, it may be more important to decide under the guidance of this perspective when working on medial sphenoid wing meningiomas and paraclinoid meningiomas.

Yasuo Suga et al. describe the birth-like excision of a giant falx meningioma following intraoperative acute brain swelling in their case report. They mention that the mass was pushed out en bloc by the brain after the falx connection was separated. It is emphasized that the relative hyperemia in the tissue surrounding the tumor due to sudden brain swelling induced by the craniotomy and the sudden decrease in intracranial pressure facilitates the en bloc pushing of the tumor from the brain. One of the conclusions that can be drawn here is that brain elasticity may allow circumferential dissection even in giant meningiomas. This may be an indirect indication that internal decompression is not an essential stage even in such meningiomas. (1).

Sivashanmugam Dhandapani et al. have reported that internal decompression may increase the amount of bleeding and the duration of the surgery, especially for meningiomas with high intra-mass vascularity, in their series where debulking was not used. Less bleeding and shorter surgery duration were emphasized as the most important advantages of en bloc removal. En bloc resection was decided on after separation from the dural connection in this article. In our opinion, if a suitable arachnoid plane was located, en bloc removal was performed by deepening the circumferential dissection without internal decompression. We believe there is a clear contraindication for en bloc extraction when the main vascular structures remain inside the tumor. However, the circumferential dissection should be advanced as far as possible to the vascular region while preserving the arachnoid plane even when en bloc extraction is not possible. Entry should be made by cutting the capsule in areas where vascular structures remain on the inside. Otherwise, intra-tumor decompression will increase bleeding from the first moment and the possibility of distinguishing tumor cleavage and proceeding with the case in a controlled manner will be lost. The preservation of intratumoral or peritumoral vascular structures may also become difficult in these cases (2).

Kaarakhan et al. have mentioned en bloc resection to be the most important surgical option in a giant meningioma case with extra- and intracranial spread (4). A long duration of surgery will greatly increase the amount of bleeding in giant meningiomas. The biggest factor in prolonged surgery is the lost time during the debulking phase and continuous bleeding in the form of oozing. The aim should therefore be the rapid decrease of the peroperative tumor burden to the desired levels and if possible total removal, in addition to safe surgery. Surgical maneuvers such as total removal may be the most important strategy for large and giant meningiomas where the main vascular structures are not penetrating. Our recommendation in this regard is the preoperative consideration of en bloc resection, taking our previously mentioned guidelines into account.

Debulking is generally not necessary during surgery for small meningiomas, especially for small meningiomas in the frontal, parietal, or occipital convexity. We believe that the localization of the tumor is more important than its size. Debulking is not required for a 3 cm convexity meningioma but may be primarily required during dissection of the vessels and cranial nerves in the tuberculum sella or clinoid. Debulking was not required during the resection of the 3 cm tuberculum sella meningioma in our series, and the tumor was resected en bloc with no additional neurodeficiency developing in the patient. The decision here will of course be shaped according to the experience and peroperative opinion of the surgeon. Circumferential dissection was not possible in another 3 cm meningioma case of tentorial origin due to adhesions in the pial area, and the tumor was debulked and resected. Tumor pathology of the fibrous type could have contributed to this result.

We performed en bloc resection in 18 of the 21 meningioma cases in this series. No additional neurodeficit occurred in any of these patients. Debulking was performed in 3 of the patients. Two of these were medial sphenoid wing meningiomas while one case was tentorial meningioma. We want to emphasize two cases in particular. In one of the two patients with medial sphenoid wing meningiomas, the M1 segment and the lateral lentriculostriate arteries arising from it were completely surrounded by the meningioma. Subtotal resection was performed in this patient. Although the left upper extremity was 2/5 hemiparetic and the left lower extremity was 4/5 hemiparetic in the postoperative period, a remarkable recovery was observed at the one-year follow-up. In the other medial sphenoid wing meningioma, the MCA segments were adherent to the tumor capsule distally in the supero-posterior section, and the anterior temporal branch course was in the middle posterior. After debulking, all vascular structures were released from distal to proximal by sharp dissection and the tumor was grossly totally removed. The patient was discharged with no postoperative deficit.

En bloc resection is contraindicated in meningiomas where the vascular structures are surrounded by the tumor. Debulking should be performed if the tumor elasticity does not allow dissection in cases where the vascular structures or cranial nerves are adherent to the tumor capsule. However, if the dissection is progressing well, the decision to debulk may be left to the surgeon's opinion and experience. Sharp dissection and vascular dissection from the periphery to the central area should be performed, once a clear view is obtained.

One of the striking points of the current study is that surgery was preferred for some small convexity meningiomas as well. Choosing follow-up or radiosurgery for small meningiomas may cause an increase in the size of the convexity meningiomas. Surgery becomes more complex in the case of growth of small convexity meningiomas when radiosurgery is not successful or the patients do not come for follow-up. The mean postoperative complication rate for these patients is around 10%(5). A large tumor lodge may increase the risk of intracerebral hematoma, CSF fistula, and infection (5).

Surgery is therefore the gold standard for convexity meningiomas up to 2.5 cm in diameter, if the patient's age is also suitable. En bloc resection was performed in all of the convexity meningiomas we operated on. None of them required debulking, enabling a short duration of surgery with a low amount of bleeding.

An interesting technical note regarding convexity meningiomas in the literature is that a vacuum retractor system that is connected to the operating room aspiration system has been reported. The authors emphasized that the pulling effect of this system on the neurovascular structures around the tumor was safer than with classic methods and allowed for fast and safe en bloc resection of convexity meningiomas with little or no pressure on the surrounding cerebral tissue (6). We would like to mention the importance of the current article that highlights en bloc resection with a new technique where a vacuum assistant was used. Although our surgery were more classical, the objective was again safe en bloc resection.

The advantages of the en bloc resection method have been reported as less bleeding, shorter surgical duration, low probability of residual tumor, and low recurrence rate in the meningioma series of Dhandapani. One disadvantage is that the method requires good cleavage, which may not be possible in anaplastic or malignant meningiomas. Fibrous type meningiomas may also not be suitable for en bloc resection, as in our case. In addition, the method is contraindicated if the tumor has encircled major vascular structures. It has been reported that deeply located tumors may not be suitable for this method and we partially agree with this point of view. En bloc resection of deep-seated meningiomas, and especially large-diameter ones, should be avoided if there is a risk of strain or damage to normal cerebral tissue during maneuvering with the bipolar cauter. Debulking is necessary in these cases. We would also like to point out that we performed en bloc resection of a 3 cm tuberculum sella meningioma. Even though this tumor is deeply located, trans-Sylvian intervention may have provided us an advantage as regards retraction of the brain. Therefore, it may be more accurate to decide on the en bloc or debulking method during the surgery

One of the remarkable points in our study is that brain and tumor elasticity allowed the release of the ICA/MCA/ACA and distal branches adhering to the tumor capsule in 5 of the 7 meningioma cases penetrated by ICA/MCA/ACA and branches. This was especially true for the distal MCA/ACA segments. However, release and en bloc resection were also possible in the case of tuberculum sella meningioma, which was also adherent in the ICA, A1-M1 region. The optic nerve was elongated unilaterally due to tumor growth in this case. En bloc resection should not be attempted in any case where the optic nerve is stretched. En bloc resection is possible for spheno-sylvian or lateral sphenoid wing meningiomas. The most appropriate method for medial sphenoid wing or paraclinoid meningiomas is to reduce the tumor volume by debulking. Mobility is also low in the proximal arterial segments and performing gradual resection by debulking can be vital. Debulking was performed for two medial sphenoid wing meningiomas in our series. The main option is total or sub-total resection for tumors with significant arterial structure penetration.

The biggest advantage of en bloc resection may be the significant shortening of the surgery, due to skipping of the debulking phase. The amount of bleeding may also decrease significantly in these cases, as evident in our series.

The Glasgow Outcome Score was 5 in all our cases that had undergone en bloc resection. This may indicate that the en bloc resection technique does not harm the patient in properly chosen cases.

Conclusion

Although debulking is an important step in meningioma surgery, it may not be beneficial for every case. On the contrary, it can prolong the duration of surgery. However, debulking or en bloc resection may be preferred according to the opinion and experience of the surgeon in cases where the main vascular structures and cranial nerves are adherent to the capsule of the meningioma. En bloc resection is contraindicated in meningiomas where the main vascular structures are encircled and subtotal resection can also be considered in these cases.

REFERENCES

1. Yasuo Suga, Satoshi Tsutsumi, Takuma Higo, Akihide Kondo, Yusuke Abe, Yukimasa Yasumoto, Masanori Ito. Huge falx meningioma resected en bloc following acute brain swelling: a case report. No Shinkei Geka. 2008 Sep; 36(9):819-23*
2. Sivashanmugam Dhandapani, Karamchand Sharma. Is “en-bloc” excision, an option for select large vascular meningiomas? Surgical Neurology International 2013; 4:102
3. Michael Salcman, Roberto C. Heros, Edward R. Laws, Jr., Volker K. H. Sonntag. Kempe’s Operative Neurosurgery. Volume 1; Sphenoid wing meningoma; 2004 Springer-Verlag New York, Inc.
4. V. B. Karakhan, V. A. Aleshin,D. M. Below et al. Must oncological principles be in the surgery of meningiomas? En bloc removal of giant right frontal meningioma with extracranial spread. Description of a clinical case. Head and Neck Tumors, 2015 Apr: 50-54.
5. Nader Sanai, Michael E Sughrue, Gopal Shangari, Kenny Chung, Mitchel S Berger, Michael W McDermott. Risk profile associated with convexity meningioma resection in the modern neurosurgical era. Journal of Neurosurgery; 2010 May; 112(5):913-9
6. Benjamin D. Fox, Bartley D. Mitchell, Akash J. Patel, Katherine Relyea, Shankar P. Gophinath, Claudio Tatsui, Bruce L. Ehni. Vacuum-assisted en bloc resection of large convexity meningiomas. Journal of Neurosurgery; 2010, Volume 114, Issue 3, Page: 727-730
7. Dhandapani SS. En-bloc excision of large meningiomas (poster presentation). Post Graduate Institute of Medical Education and Research (PGIMER), Chandigarh.
8. Robin A Buerki, Craig M Horbinski, Timothy Kruser, Peleg M Horowitz, Charles David James, Rimas V Lukas. An overview of meningiomas. Future Oncology 2018 Sep; 14(21): 2161–2177
9. Wiemels J, Wrensch M, Claus EB. Epidemiology and etiology of meningioma. Journal of Neuro-Oncology. 2010;99(3):307–314.
10. Rogers L, Barani I, Chamberlain MC, et al. Meningiomas: knowledge base, treatment outcomes, and uncertainties. a RANO review. Journal of Neurosurgery 2015;122(1):4–23.
11. Chamberlain MC. Meningiomas. In: Norden AD, Reardon DA, Wen PCY, editors. Primary Central Nervous System Tumors: Pathogenesis and Therapy. Humana Press; NJ, USA: 2011. pp. 355–375.

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