ISSN 1220-8841 (Print)
ISSN 2344-4959 (Online)

ROMANIAN NEUROSURGERY

Vol. XXXVI | No. 1 March 2022

Official Journal
"The Romanian Society of Neurosurgery"
- since 1982 -

LONDON ACADEMIC PUBLISHING

Editorial and Advisory Board

ROMANIAN

NEUROSURGERY

Vol. XXXVI | No. 1 March 2022

London
Academic Publishing

ISSN 1220-8841 (Print)
ISSN 2344-4959 (Online)

First Printing: March 2022
London Academic Publishing Ltd.
27, Old Gloucester Street
WC1N 3AX
London, United Kingdom
Email: contact@lapub.co.uk

london-ap.uk
lapub.co.uk
journals.lapub.co.uk
journals.lapub.co.uk/index.php/roneurosurgery

Company Reg. No. 10941794
Registered in England and Wales

Contents

Romanian Neurosurgery (2022) XXXVI (1): pp. 9-13
DOI: 10.33962/roneuro-2022-001
www.journals.lapub.co.uk/index.php/roneurosurgery

Multimodal treatment of glomus jugular tumours. Case series and literature review

F. Stoica[1], G. Popescu[1], Francesca Paslaru[1], Anamaria Gheorghiu[1], A.C. Paslaru[2,3], M. Apostol[1], M.C. Zaharia[1], R.M. Gorgan[1,2]

[1] 4th Neurosurgical Department, "Bagdasar Arseni" Clinical Emergency Hospital, Bucharest, ROMANIA
[2] "Carol Davila" University of Medicine and Pharmacy, Bucharest, ROMANIA
[3] Department of Genetics, "Dr. Victor Gomoiu" Children's Clinical Hospital, Bucharest, ROMANIA

ABSTRACT

Glomus jugulare tumours are extremely rare, slow-growing, hypervascular tumours that arise within the jugular foramen of the temporal bone and frequently involve the lower cranial nerves. We performed a retrospective study for patients treated between January 2005 and December 2019, reviewing clinical and radiological data for 91 cases of glomus jugulare tumours. Data were available for 91 patients presenting with 96 tumours. Surgery was 1st intention of treatment for 13 cases, the endovascular approach was 1st intention for 6 cases and GKRS was primarily performed in 72 cases. Combined treatment options were used in 19 cases. The median age at the time of treatment was of 57 years. The tumour volume varied between 0.5 and 73.4 cm3 with a median value of 8.3 cm3. For the cases treated with GKRS, the peripheral dose ranged between 8 and 35 Gy on the 35% to 65% isodose, with a median of 14 Gy on the 45% isodose. The average follow-up was 38 months with a maximum of 94 and consisted of contrast-enhanced MRI every six months in the first year after the procedure and every 1 to 2 years afterwards. Overall tumour control rate was of 95.6% using multimodal treatment options for glomus jugulare tumours. Multimodal treatment for glomus jugulare tumours offers the patient the chance for the best possible outcome and long-term survivability. Individual treatment approach for this kind of very rare head and neck tumour (0,6% of all head and neck tumours) is recommended to choose the best risk-versus-benefit treatment option.

Keywords
multimodal,
jugular tumours,
glomus

Corresponding author:
G. Popescu

"Carol Davila" University of Medicine and Pharmacy,
Bucharest, Romania

george_popescu39@yahoo.com"

Scan to access the online version

BACKGROUND

Glomus jugulare tumor is a benign neuroendocrine tumor that arises from the jugular foramen. This tumor is characterized by a slow-growing pattern. Paragangliomas, also known as chemodectomas represent benign tumors with the origin from neural crest derivates also known as the parangalia [1],[2],[3]. These tumors are highly vascularised. They can receive blood supply from both from the external carotid artery and internal carotid artery. The localization of

these tumors can widely vary, from carotid bifurcation to the auricular branch of the vagus nerve. The most frequent localization is the carotid body, accounting for almost half of the tumors [5],[6] whereas glomus jugulare tumors represent 24% of them [6]. Even though they are benign tumors, the symptomatology can be the cause of the mass effect [2].

Usually, these tumors are diagnosed in the fourth to sixth decade of life, with a moderate female predilection. Most of the glomus jugulare tumors are isolated lesions but around 20% of them present hereditary components [4]. The inherited tumors are usually bilateral and the onset of the symptoms is reported to be earlier than the onset symptomatology of the sporadic tumors. The reported malignancy of these tumors is less than 5% [2].

Around 25% of the paragangliomas remain silent and are incidentally discovered. The symptoms caused by these tumors depend on their location. Lower cranial nerves impairment is reported in more than 10% of patients [8]. The most common neurological deficits reported are tongue deviation, hoarseness, facial palsy, dysphagia, and shoulder weakness [2],[7],[8]. Additionally, patterns of cranial nerve palsies were described and these include [12]:

- Vernet syndrome that represents motor paralysis of cranial nerves IX, X and XI; [9]
- Collet- Sicard syndrome described as the palsy of cranial nerves IX,X,XI and XII; [10]
- Horner syndrome – oculosympathetic palsy. [11]

Due to its slow-growing pattern and the complex anatomy of the skull base and neck, observation of the patient is considered a good treatment alternative. In more than 60% of the cases, tumor volume remains stable or decrease in size [14]. However, if the tumor tend to be symptomatic, surgical excision or stereotactic radiosurgery will be take into account.

CASE SERIES

We performed a retrospective study for patients treated between January 2005 and December 2019, reviewing clinical and radiological data for 91 cases of glomus jugulare tumors. Data were available for 91 patients presenting with 96 tumors.

Surgery was the treatment of choice for 13 cases, endovascular embolization was performed as first intention treatment for 6 cases and GKRS was primarily performed in 72 cases.

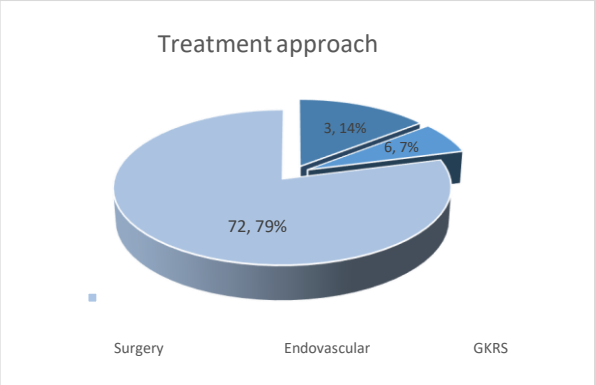


Figure 1. Distribution of the treatment option

Combined treatment options were used in 19 cases (1 surgery with GKRS, 18 endovascular with GKRS). 44 glomus jugulare tumors were identified on the right side, and 47 on the left side. In the study group, we had 23 male patients and 63 female patients. 11 patients were in the 15-39 age group while 80 patients were older than 40 yrs. The median age at the time of treatment was of 57 years. The tumor volume varied between 0.5 and 73.4 cm3 with a median value of 8.3 cm3.

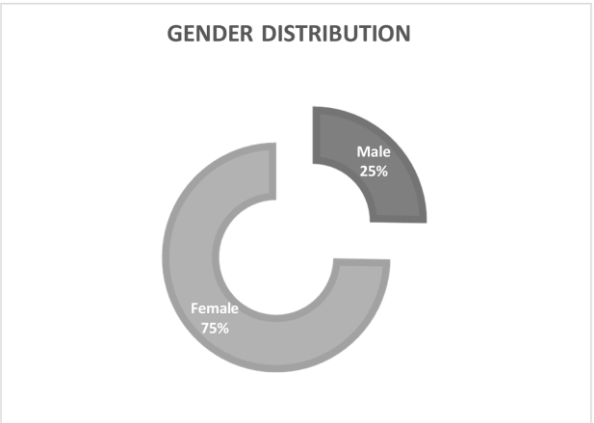


Figure 2. Gender distribution of the patients

For the cases treated with GKRS, the peripheral dose ranged between 8 and 20 Gy on the 35% to 65% isodose, with a median of 14 Gy on the 45% isodose. The average follow-up was 38 months with a maximum of 94 and consisted of contrast-enhanced MRI every six months in the first year after the procedure and every 1 to 2 years afterward. Overall

tumor control rate was 95.6% using multimodal treatment options for glomus jugulare tumors.

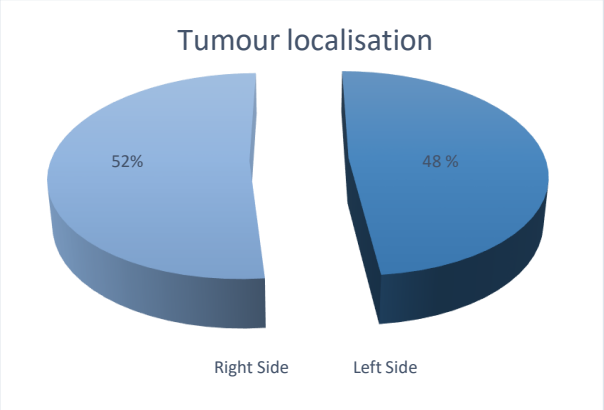


Figure 3. Tumour localisation

COMORBIDITY
Lower cranial nerves deficits
Dizziness
Tinnitus
Hearing impairment
Hemorrhages
Hydrocephalus
Hemiparesis

Table 1. Most common comorbidities

Comorbidities were noted in 37 patients (40.6%) and consisted of lower cranial nerves deficits (26.4%), dizziness, tinnitus, partial or complete hearing loss in 21.9% of cases, 2 hemorrhages, 2 secondary hydrocephalus, and 1 hemiparesis. 23 patients (24.3%) presented recurrences: 13 after surgery, 6 after embolization, and 4 after GKRS. However, the mortality rate was 0.

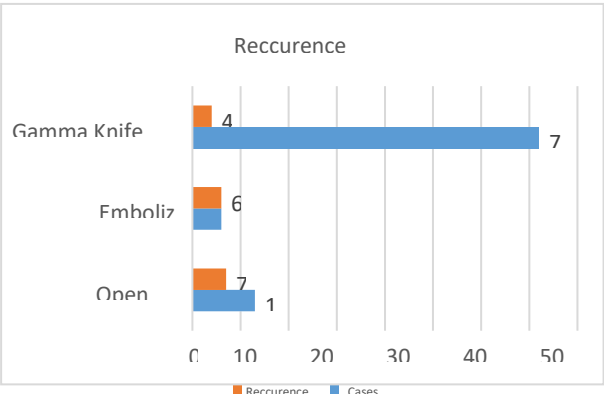


Figure 4. Recurrence cases based on the therapeutical approach

CASE 1

The first case is represented by a female patient of 53 years old, whose symptoms are vertigo, hearing loss, and pulsating tinnitus. Soon it is discovered the typical aspect of “salt and pepper” for a glomus tumor with temporal localization, millimetric extracranial extension into jugular vein lumen and damage to the structures of the inner ear. 9 years after GKRS, a cerebral MRI was performed, which showed that the irradiated tumor volume has been reduced in circumferential dimensions, with a homogeneous intake of contrast substance ("densified" appearance due to sclerosis and obliteration of intratumoral blood vessels) without adverse reactions due to irradiation. No new neurological deficits were recorded after GKRS.

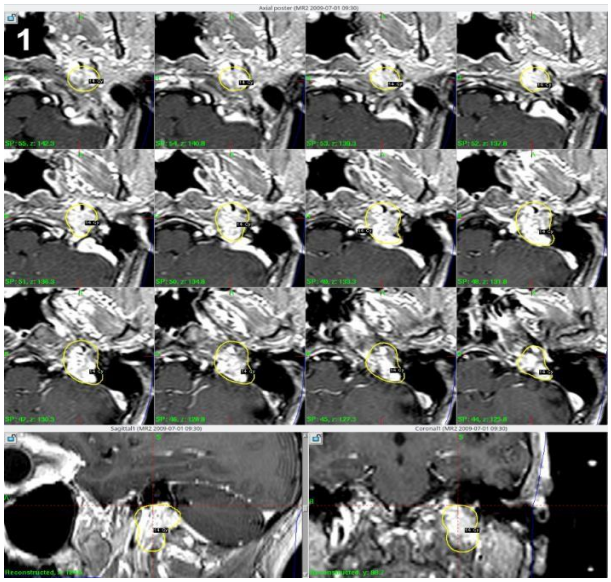


Figure 5. Preoperative aspect of the tumour

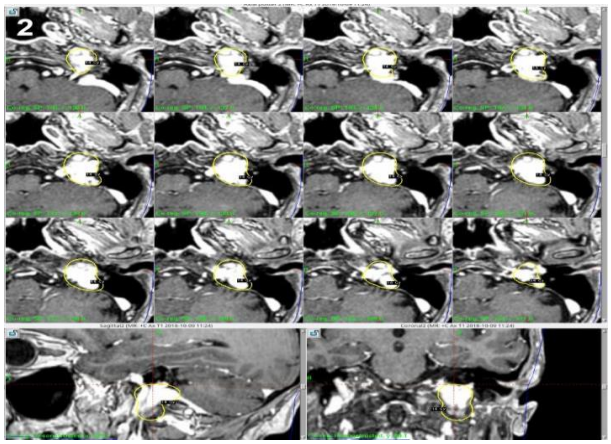


Figure 6. Postoperative aspect after Gamma Knife Radiosurgery

Case 2

The second case is represented by a 62 years old female patient, whose symptoms onset with injury to multiple cranial nerves (VII, VIII, IX, XI, XII). The diagnosis was represented by a large glomic tumor with an important intracranial component with a mass effect on the brainstem.

A cerebral MRI was performed 4 years post GKRS, showing a marked reduction in the size of the irradiated tumor, especially in the intracranial component, with a significant decrease in the mass effect, without perilesional reactive edema.

No new neurological deficits were recorded. 18 months after irradiation, a complete remission of facial paresis has been noticed and other cranial nerves neurological status remained stationary.

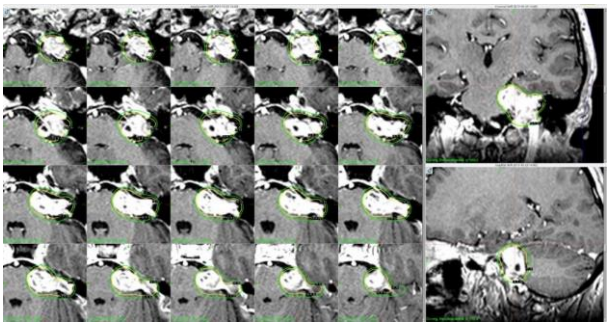


Figure 7. Preoperative aspect of the tumour

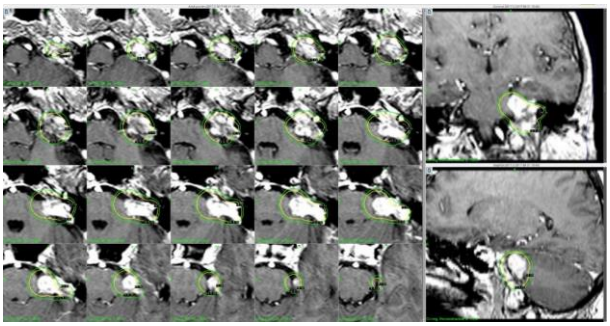


Figure 8. Postoperative aspect after Gamma Knife Radiosurgery

Case 3

For the third case, we have a 35 years old male patient, who presented with glomic tumor located in the temporal bone, onset with hearing disorders (hearing loss and pulsating tinnitus).

6 years after GKRS, a periodic follow-up MRI has been performed. the inferior recurrence in the lumen of the jugular vein was identified outside the irradiation field with a volume of 4.6 cm3, asymptomatic. The decision is made in order to irradiate the recurrence with 14 Gy on 47% isodose.

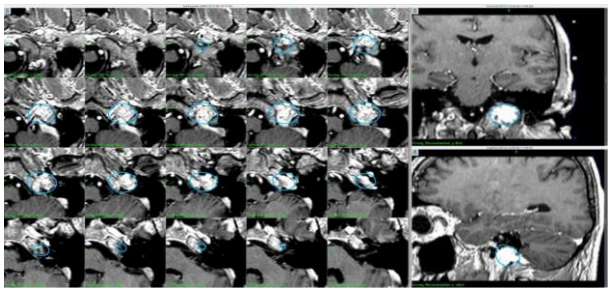


Figure 9. Preoperative aspect before Gamma Knife Radiosurgery

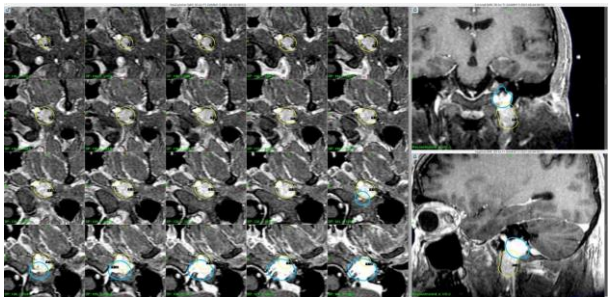


Figure 10. The reccurence aspect, 6 years afte GKRS

Discussions

The best treatment option for glomus jugulare tumors is yet to be debated. Due to their localization, surgical treatment does not represent the gold standard because of the complex anatomy of the region, high rates of morbidity, subtotal resection, and the alternative behavior of the tumors that could be very aggressive in some cases. In 2003, Roberto Pareschi et al [15] described their experience in the surgical treatment of glomus jugulare tumors. 42 patients with glomus jugulare tumors were identified, 3 of them previously undergone surgery for this pathology, and 3 patients presented bilateral temporal lesions. The otoscopic evaluation revealed in 80% of the patients the typical red middle ear mass. 70% of the cases had no preoperative cranial nerve deficit. 37 seven patients were elected for surgical intervention. In 33 cases, infratemporal fossa approaches were used and in 4 cases, conservative jugulopetrosectomy was performed, in order to preserve the facial nerve. In 20% of the cases, cranial nerves IX and X were injured. No recurrence after total resection was reported. An extensive dissection of the posterolateral skull base is required for surgery of glomus jugulare tumors [15]. Even though cranial nerve preservation is an

extremely important goal in the surgical approach, in 22% of the cases facial nerve is sacrificed [15]. The authors concluded that the focus should drift away from total resection to increasing the quality of life of the patient, a philosophy that our clinic shares. Only 14% percent of our cases were surgically treated, in order to avoid the decrease in the quality of patient`s life.

Endovascular treatment is an alternative treatment approach for patients with glomus jugulare tumors. In 2017, Kocur et al [17] presented their experience and the outcome of embolization in 3 cases of glomus jugulare tumors. They described the technical difficulty of achieving complete obliteration of the glomus jugulare tumors and concluded that increased risk of revascularization is not beneficial compared to the diminished clinical symptoms. In our clinic, only 6 endovascular treatments were performed.

A promising approach for this pathology is represented by radiosurgery. Due to the high degree of accuracy, rapid radiation dose falloff at the periphery of the target tumor, and their high precision, radiosurgery became a popular treatment choice. In their meta-analysis, Guss et al [13] included 19 studies, compounding 335 glomus jugulare patients. They reported a reduced or unchanged tumoral volume after radiosurgery, sustained by imagistic findings. Clinical control was reported as improved or unchanged after radiosurgery in 95% of the cases. The authors emphasized the effectiveness of this treatment option. In our clinic, 72 patients benefited from radiosurgery.

Conclusions

Multimodal treatment for glomus jugulare tumors offers the patient the chance for the best possible outcome and long-term survivability. Individual treatment approach for this kind of very rare head and neck tumor (0,6% of all head and neck tumors) is recommended to choose the best risk-versus-benefit treatment option. GKRS in these kind of tumors seems to be the option of choice, considering that in our experience, has the lowest comorbidity, reccurence rate and mortality.

References

1. Fussey JM, Kemeny AA, Sankar S, Rejali D. Successful management of a catecholamine- secreting glomus jugulare tumor with radiosurgery alone. J Neurol Surg B Skull Base. 2013 Dec;74(6):399-402.
2. Ramina R, Maniglia JJ, Fernandes YB, Paschoal JR, Pfeilsticker LN, Neto MC, Borges G. Jugular foramen tumors: diagnosis and treatment. Neurosurg Focus. 2004 Aug 15;17(2):E5.
3. Petropoulos AE, Luetje CM, Camarata PJ, Whittaker CK, Lee G, Baysal BE. Genetic analysis in the diagnosis of familial paragangliomas. Laryngoscope. 2000 Jul;110(7):1225-9.
4. Semaan MT, Megerian CA. Current assessment and management of glomus tumors. Curr Opin Otolaryngol Head Neck Surg. 2008 Oct;16(5):420-6.
5. Tokgöz SA, Saylam G, Bayır Ö, Keseroğlu K, Toptaş G, Çadallı Tatar E, Akın İ, Korkmaz MH. Glomus tumors of the head and neck: thirteen years' institutional experience and management. Acta Otolaryngol. 2019 Oct;139(10):930-933.
6. Singh S, Madan R, Singh MK, Thakar A, Sharma SC. Head-and-neck paragangliomas: An overview of 54 cases operated at a tertiary care center. South Asian J Cancer. 2019 Oct- Dec;8(4):237-240.
7. Wanna GB, Sweeney AD, Haynes DS, Carlson ML. Contemporary management of jugular paragangliomas. Otolaryngol Clin North Am. 2015 Apr;48(2):331-41.
8. Jackson CG, Harris PF, Glasscock ME, Fritsch M, Dimitrov E, Johnson GD, Poe DS. Diagnosis and management of paragangliomas of the skull base. Am J Surg. 1990 Apr;159(4):389-93.
9. Monteiro, F., Oliveira, P., Peneda, J. and Condé, A., 2019. Vernet syndrome: intracranial extension of a slow-growing mass. BMJ Case Reports CP, 12(5), p.e228039.
10. Neo S, Lee KE. Collet-Sicard syndrome: a rare but important presentation of internal jugular vein thrombosis. Practical Neurology. 2017 Feb 1;17(1):63-5.
11. Spector GJ, Druck NS, Gado M. Neurologic manifestations of glomus tumors in the head and neck. Archives of Neurology. 1976 Apr 1;33(4):270-4.
12. Spector GJ, Gado M, Ciralsky R, Ogura JH, Maisel RH. Neurologic implications of glomus tumors in the head and neck. The Laryngoscope. 1975 Aug;85(8):1387-95.
13. Guss ZD, Batra S, Limb CJ, Li G, Sughrue ME, Redmond K, Rigamonti D, Parsa AT, Chang S, Kleinberg L, Lim M. Radiosurgery of glomus jugulare tumors: a meta-analysis. International Journal of Radiation Oncology* Biology* Physics. 2011 Nov 15;81(4):e497-502.
14. Prasad SC, Mimoune HA, D'Orazio F, Medina M, Bacciu A, Mariani-Costantini R, Piazza P, Sanna M. The role of wait-and-scan and the efficacy of radiotherapy in the treatment of temporal bone paragangliomas. Otol Neurotol. 2014 Jun;35(5):922-31.
15. Pareschi R, Righini S, Destito D, Raucci AF, Colombo S. Surgery of glomus jugulare tumors. Skull Base. 2003 Aug;13(03):149-58.
16. Kocur D, Ślusarczyk W, Przybyłko N, Hofman M, Jamróz T, Suszyński K, Baron J, Kwiek S. Endovascular approach to glomus jugulare tumors. Polish journal of radiology. 2017;82:322

Romanian Neurosurgery (2022) XXXVI (1): pp. 14-17
DOI: 10.33962/roneuro-2022-002
www.journals.lapub.co.uk/index.php/roneurosurgery

Single stent transverse-configuration in assisted coiling treatment of a double anterior communicating aneurysm

A. Chiriac, N. Dobrin, Georgiana Ion, Z. Faiyad, Dana Turliuc, I. Poeata

"Gr. T. Popa" University of Medicine and Pharmacy, Iasi, ROMANIA
"Prof. Dr. N. Oblu" Clinic Emergency Hospital, Iasi, ROMANIA

ABSTRACT

The stent-assisted coiling technique is more and more used for the anterior communicating aneurysms (AcomA) treatment due to the increasing incidence of complex and wide-necked aneurysms at this level. Different arrangements of a single stent for assisted coil embolization have been described. The transverse-configuration from A1-Acom-Contralateral A1 stent-assisted coiling procedure was previously reported as a feasible, effective, and relatively safe endovascular technique used to treat wide-necked complex AcomA aneurysms. In this article, we presented a technique involving transverse stenting through the AcomA via the dominant A1 and discuss some particular technical difficulties.

Keywords
transverse-configuration,
stent assisted coiling technique,
intracranial aneurysm

Corresponding author:
Dana Mihaela Turliuc

"Prof. Dr. N. Oblu"
Clinic Emergency Hospital,
Iasi, Romania

chiriac_a@hotmail.com

Scan to access the online version

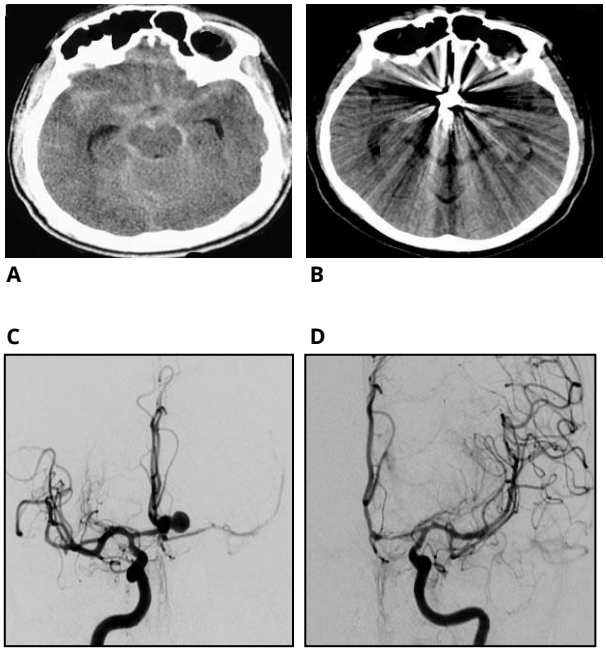
INTRODUCTION

The anterior communicating artery (AcomA) was reported to be one of the most common location of ruptured aneurysms over the last decades. These lesions are of significant importance due to their higher incidence but also to their complex anatomical features, often associated with a wide neck, difficult vascular angles and a variety of abnormalities. The use of self-expanding stents under various arrangements in coil assisted embolization technique can help to optimally treat aneurysms with this location and to achieve more long-term results. Also, due to the numerous disadvantages and technical difficulties specific to double stenting, the single stenting technique seems to be the first option considered in the case of anterior communicating artery aneurysms. In our study we describe the use of a single stent–assisted coiling technique in a transverse configuration for the treatment of double communicating artery aneurysm. The efficacy and limits of this technique were also discussed referring to the few cases already presented in the literature [1,2].

CASE PRESENTATION

A 51-year-old male patient was transferred to our neurosurgical department from a local hospital for a subarachnoid haemorrhage revealed on cranio-cerebral CT scan. At admission he was conscious, confused, agitated, accusing severe headache. The family declare the onset of symptoms four days ago with progressive evolution and a history of medication-treated hypertension. The brain CT angiography demonstrated a double sac complex AcomA aneurysm as source of hemorrhage. The patient was proposed for an immediately endovascular aneurysm occlusion. A written informed consent was signed by patient family after discussions with the operating team.

The procedure was performed on a biplane angiography system (INFINIX, Toshiba, Canon Medical System) with the patient under general anesthesia. Both carotid arteries were evaluated and a large complex double AcomA aneurysm was angiographicaly documented. A slight right side A1 dominance with a better contrast enhancement of the two aneurysmal sac was noticed. The 3D DSA reconstruction image showed wide-neck involving the entire anterior communicating artery segment.
A single stent support technique in a transversal configuration was deemed necessary for a safe endosaccular coiling with all anterior communicating artery complex branches preservation (Fig 1).



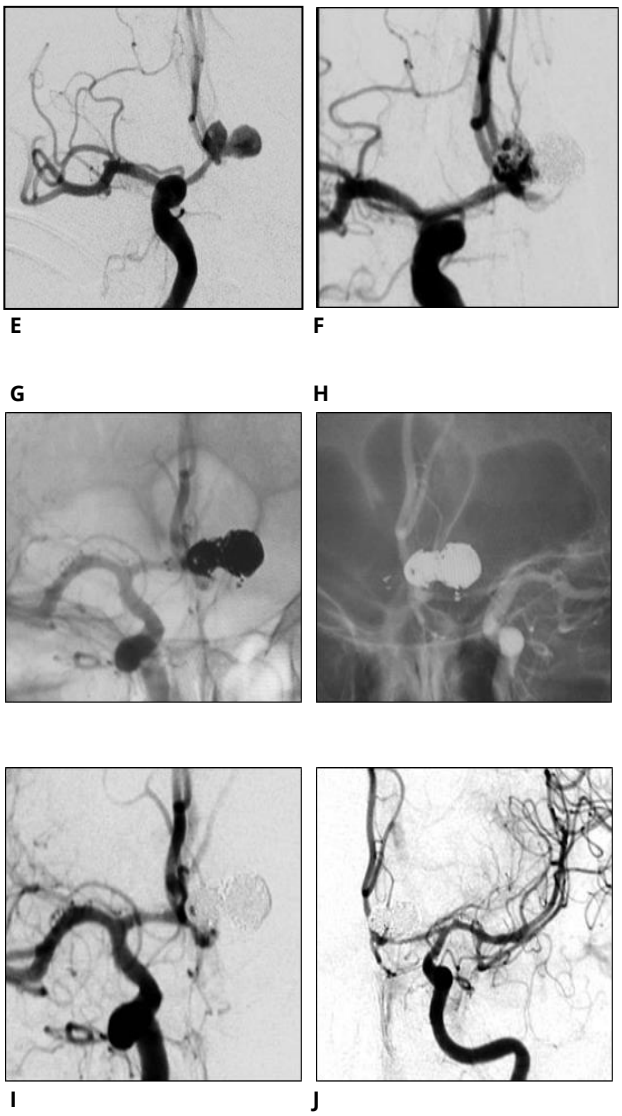


Figure 1. A,B - Brain CT diagnosis highlighting SAH and post interventional brain CT control; C,D,E - ; D,E –DSA from the right and left ICA showing the double AcomA aneurysm; F,G,H,I,J– Intraprocedural DSA images and the final DSA control from both ICA injection.

The patient received 150 mg aspirin before procedure and 5000 IU bolus dose of intravenous heparin was administered after 6F femoral sheath (Merit Medical) placement. A 6-Fr Chaperon guiding catheter was placed at the proximal cervical internal carotid artery carefully advanced over 0.035 glidewire. 3D angiographic imaging working projection was used for the guidance on roadmap images. We use a transvers-configuration stent–assisted coiling technique with step by step trans-stent catheterizing of both aneurysmal sac to avoid

multiple catheter maneuvering through the right A1 arterial segment.

A Prowler Select Plus microcatheter (Codman J&J) was then very carefully advanced over a 0.014 Transed microwire (Boston Scientific) via the right A1 through the AcomA into the middle part of contralateral A1. A 4/30 mm Enterprise 2 stent is then deployed from the middle part of the left A1 over the aneurismal neck through the AcomA until the middle part of right A1. After the full deployment of the stents, a Prowler 10 microcatheter is placed through the stent into the large sac aneurysm. Its complete angiographic coil occlusion was followed by the microcatheter repositioning in to the smaller aneurysm and continued coiling until no further coils could be safely deployed within the aneurysm sac. The control DSA images demonstrated a "Raymond class I" complete aneurysm occlusion with no neck remnant, coil herniation, clot formation or branch occlusion, and with no perioperative or postopective complications. A bolus dose of 5 ml of nimodipine (Nimotop, Bayer Health Care AG) diluted in 15 ml of 0.9% NaCl solution was infused through the microcatheter in to the proximal part of right A1 for 10 minutes to combat procedural vasospasm. The patient evolution was favourable, and he was discarded home after 17 days of hospitalization.

Discussion

The large clinical trial studies showed that AcomA is the most common location of ruptured intracranial aneurysm. Treatment of wide-neck AcomA aneurysms with complex anatomy such as double sac aneurysm usually requires a stent assisted coiling technique for a safety and optimal occlusion. The difficulties and complications described for double stenting techniques as insufficient wall apposition and endothelialisation, and increased risk of acute or chronic thromboembolism have made the single stenting technique to be considered as the first option. However, the most used single stenting configurations through the dominant A1 to the ipsilateral or contralateral A2 may not provide sufficient neck coverage for a double wide-necked AcomA aneurysm, resulting in a coil protrusion and normal branches occlusion. For such particular anatomical cases an A1-AcomA-contralateral A1 stenting could be performed. This particular transverse stenting configuration may successfully prevent the aforementioned inconveniences with optimal results on long-term aneurysmal occlusion [2,3,4].

The few reports in the literature on the use of single transverse-configuration stent assisted coiling for AcomA aneurysms have mentioned beside the success of using this technique, the difficulties encountered in its implementation. The technical challenges with possible related complication such as suboptimal stent deployment, dissection and thrombosis may be increased by difficult navigation through even curved and smaller calibre arteries [3,4,5].

Rhoton's publications have shown a higher probability of AcoAnt aneurysm developing as bigger the diameter difference between the bilateral A1 segments. Thus, due to hemodynamic influence, most AcomA aneurysms appear to arise from the dominant A1 segment of the anterior cerebral artery and form as junction aneurysms between the dominant A1, AcomA and A2 ipsilateral. Yasargil reports that 81.3% of ACoAnt aneurysms usually occurred at the junction of the dominant A1 and AcomA and only 18.7% occurred in the middle of the AcomA. This correlate with the fact that only 22% of cases have comparable diameters of A1 segments of the bilateral anterior cerebral arteries, suggesting a relatively low percentage of patients with AcomA aneurysms who may benefit from the application of single stent assisted coiling technique with stent in a transverse A1 to A1 configuration [2,3,6].

Another aspect of the application of the single stenting technique in the transversal configuration is related to the need for a dual femoral approach with double guidance for a retrograde approach through AcomA. Even if there are many reports of this type of approach, we believe that the use of a single guide catheter and a single femoral puncture is sufficient in many of these cases for both stent and coils delivery and a clear view of the lesion and surrounding vascular shaft [2,3].

Conclusions

Any of the strategies for placing a stent for the treatment of an AcomA aneurysm are dependent on the configuration of the aneurysm, the feasibility of incorporating the neck of the aneurysm into the implant, the difficulty of microcatheter crossing of the AcomA channels, and the degree of hypoplasia of one of the A1 segments. The placement of a single transverse-configuration stent via the dominant A1

across the AcomA into the contralateral A1 can be a feasible, efficient and relatively safe endovascular technique for the treatment of a wide-necked double AcomA aneurysm. This could offer a good long-term occlusion rates with reasonable complication rate.

References

1. Ahmed, M. E., Lum, C., Lesiuk, H., Iancu, D., & dos Santos, M. (2014). Navigation of stents across communicating arteries for aneurysm embolization. Canadian Journal of Neurological Sciences, 41(2), 193-199.
2. Adeeb, N., Griessenauer, C. J., Patel, A. S., Foreman, P. M., Baccin, C. E., Moore, J. M., ... & Thomas, A. J. (2018). The use of single stent-assisted coiling in treatment of bifurcation aneurysms: a multicenter cohort study with proposal of a scoring system to predict complete occlusion. Neurosurgery, 82(5), 710-718.
3. Ban, S. P., Kwon, O. K., & Kim, Y. D. (2022). Long-Term Outcomes of Placement of a Single Transverse Stent through the Anterior Communicating Artery via the Nondominant A1 in Coil Embolization of Wide-Necked Anterior Communicating Artery Aneurysms. Journal of Korean Neurosurgical Society, 65(1), 40.
4. Choi, H. H., Cho, Y. D., Yoo, D. H., Ahn, S. J., Cho, W. S., Kang, H. S., ... & Han, M. H. (2019). Stent-assisted coil embolization of anterior communicating artery aneurysms: safety, effectiveness, and risk factors for procedural complications or recanalization. Journal of NeuroInterventional Surgery, 11(1), 49-56.
5. Kocur, D., Zbroszczyk, M., Przybyłko, N., Hofman, M., Jamróz, T., Baron, J., ... & Kwiek, S. (2016). Stent-assisted embolization of wide-neck anterior communicating artery aneurysms: Review of consecutive 34 cases. Neurologia i Neurochirurgia Polska, 50(6), 425-431.
6. Raslan, A. M., Oztaskin, M., Thompson, E. M., Dogan, A., Petersen, B., Nesbit, G., ... & Barnwell, S. L. (2011). Neuroform stent-assisted embolization of incidental anterior communicating artery aneurysms: long-term clinical and angiographic follow-up. Neurosurgery, 69(1), 27-37.

Romanian Neurosurgery (2022) XXXVI (1): pp. 18-22
DOI: 10.33962/roneuro-2022-003
www.journals.lapub.co.uk/index.php/roneurosurgery

Postero-lateral arterio-venous fistula with particular histological aspects as the main cause of severe spinal thoracal stenosis. Case report

D. Balasa[1], Al. Tunas[1], L. Mocanu[2], M. Enescu[3]

[1] Department of Neurosurgery, Clinical County Hospital Constanta, ROMANIA
[2] Department of Anatomopathology, Clinical County Hospital Constanta, ROMANIA
[3] Department of Orthopaedics, Private Clinic Medstar, Constanta, ROMANIA

ABSTRACT
Spinal dural arteriovenous fistulas (AVFs) are rare spine vascular malformations. We report a case of AVF in the epidural space of T10 and degenerative medial osteophytes leading to clinical symptoms of severe spinal stenosis operated with good evolution.

Keywords
epidural fistula,
thoracal spine stenosis

Corresponding author:
Mocanu Liliana

Department of Anatomopathology, Clinical County Hospital Constanta, Romania

lilianamcn@gmail.com

Object: Our report's objective is to present a particular, extremely rare case of spinal stenosis at T10-T11 level, mostly on the right side, result of the cumulative effect of a vertebral postero-lateral AVF, medial degenerative osteophites and a synovial cyst at the same level. The AVF is the main cause of the spine stenosis.
Case Report: A 69 year-old man presented for 5 years' pain in the thoracal spine, intermitent paresthesias on the right leg. From one week he complains of painful paresthesias on both legs (predominant on the right side), gait with progressive difficulty.
On examination we observed incomplete paraplegia (Frankel C+/E), sensory examination revealed hypoesthesia with sensitive level T10, knee and ankles reflexes increased bilaterally with predominance on the right side, Babinsky sign present bilateral.
On MRI imaging: Thoracal spine stenosis T10-T11, by degenerative osteophites T10-T11 predominant on the right side, synovial cyst in the lateral recess who severelly compresses the dural sac and the spinal cord.

The patient was operated (Laminectomy T10 and T11 on the right side, microsurgical complete resection of a heterogenous extradural

Scan to access the online version

lesion (bony and vascular, intensively bleeding), severely compressive over the spinal cord.

The postoperative evolution was favorable: the incomplete paraplegia has improved (ASIA C+ on the right side, D on the left side), hipoesthesia diminished.

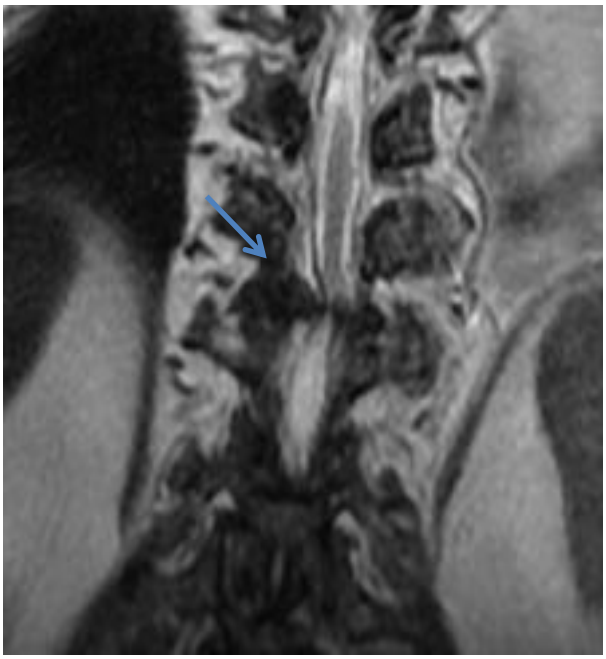


Figure 1. Coronal T2.: Dorsal spine stenosis T10-T11, by degenerative osteophytes T10-T11 predominant on the right side (Blue arrow) who severely compress the spinal cord.

Figure 2. Sagittal T2: Severe spine stenosis produced by degenerative osteophytes T10-T11 and synovial cyst (green arrow).

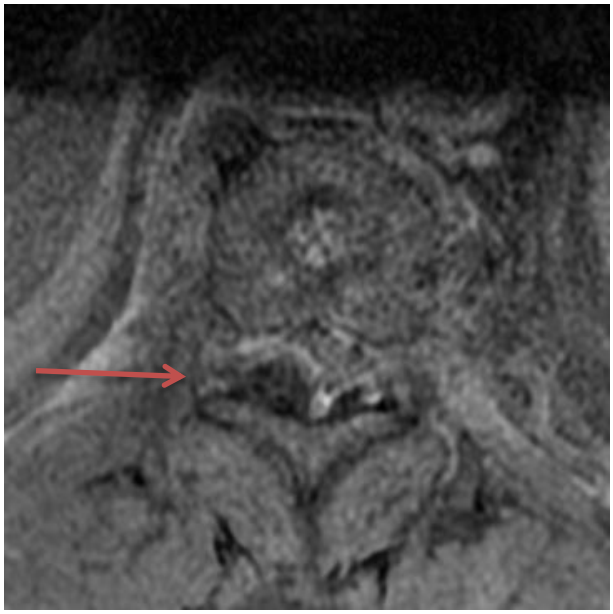


Figure 3. Axial contrast T1: Dorsal spine stenosis T10-T11, by medial degenerative osteophytes T10-T11 predominant on the right side (red arrow).

THE HISTOLOGICAL EXAM

Material and method: The probe was sent fresh with a cold ischemic time of 20 minutes, then it was paraffined embedded.

The conventional slides (Haematoxylin – eosine stained) revealed a conjunctive tissue with coiled vessels, some dilated, some collapsed, attached on a spinal node

Immunohystochemical method was done on Ventana platforme GX, with antibodies CD34 (CLONA QBEnd10); WT1 (CLONA 6FH2)

COMMENTS

Wilms' tumor-1 (WT-1) was originally described as a tumor suppressor gene based on its mutational inactivation in a subset of Wilms' tumor. It plays an essential role in haematopoiesis and angiogenesis by regulating vascular endothelial growth factor, angioproteins, nestin, and proliferation of vascular smooth muscle cells1 Human skin vasculature shows cytoplasmic WT1 protein expression, detected by an antibody recognizing the C-terminal of the protein (6F-H2). Reports have demonstrated that WT-1 protein is expressed in a variety of vascular anomalies. Defects in WT1 signalling might underlie the inability of endothelial cells in vascular malformations to undergo physiological apoptosis and remodelling

CD34 - Commonly used marker of hematopoietic progenitor cells and endothelial cells; Also called hematopoietic progenitor cell antigen CD34; CD34+ stromal cells are called dendritic interstitial cells;

CD34: Membranous stain
WT1: nuclear stain
Endothelium of normal vessels acts as a positive internal control

In our case, the positive reaction was noted in a greater number of vessels to CD34 than to WT1, so the interpretation, correlated with clinical and imagistic data, was of a vascular spinal fistula with loss of WT1 expression (as in AVMs). We couldn't find other reports about WT1 expression in spinal AVFs.

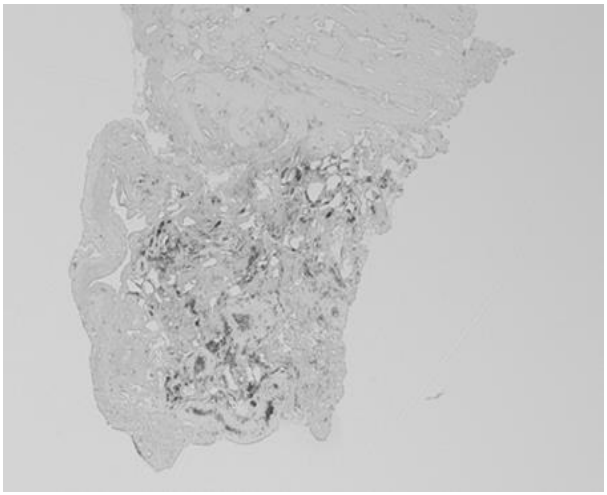


Figure 4. Cd34, ob4x

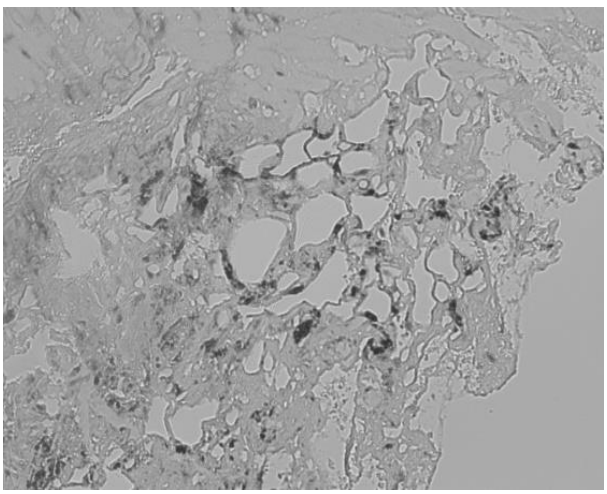


Figure 5. Cd34, ob 10x; positive in endothelial cells and in some interstitial dendritic cells

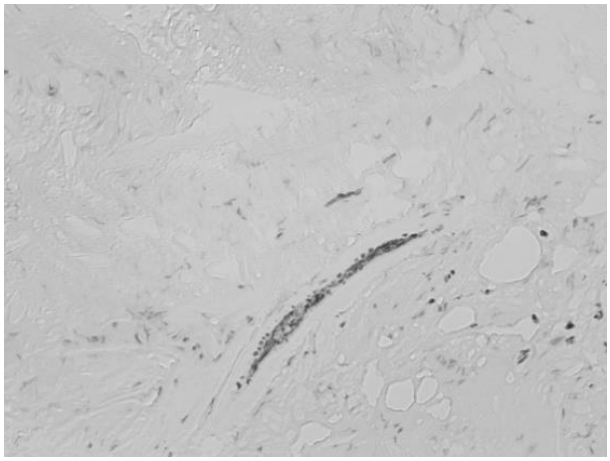


Figure 6. Wt1 positive in a normal vessel, negative in other vessels

Postoperative control was favourable

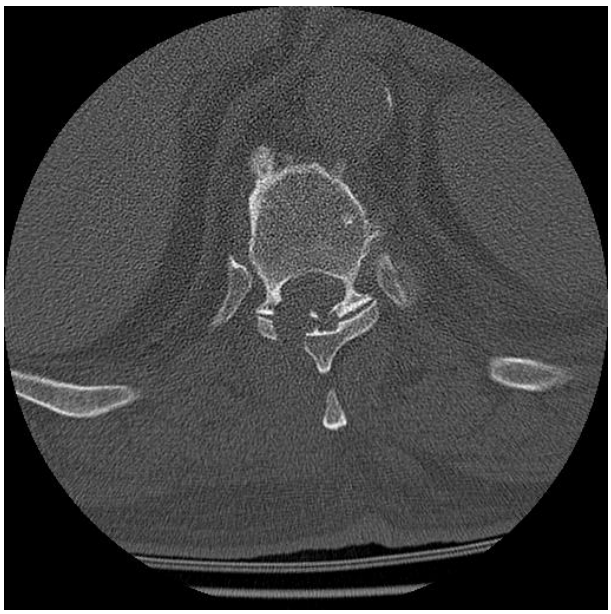


Figure 7. Postoperative CT scan

FOLLOW UP AT 6 MONTHS

Discussion: Extradural (or epidural) AVFs are relatively uncommon malformations characterized by anomalous communication between an extradural branch, usually of a radicular artery, and the epidural venous plexus[2].

Spinal arteriovenous malformations (AVMs) (constituting 3-4% of all spinal cord space occupying lesions)[3].

After Lim the symptoms of epidural AVFs are related to the pattern of venous drainage[4]. If they are

exclusively epidural, they may present with local pain or radicular pain or with progressive myelopathy[4].

Spetzler et all[5] proposed a modified classification of the spine/spinal cord arteriovenous lesions to arteriovenous malformations (AVMs) and arteriovenous fistulas (AVFs). AVFs areclassified according to their location as intradural and extradural. According to Geibprastert et all[6], dural AVFs are classified on anatomical bases in ventral group (most of them) and dorsal epidural group

The dorsal group of dural AV fistulas is related to veins that normally drain the spinous process and lamina at the spinal level[7]. Patients with dural AV fistulas within this space typically present with spontaneous epidural hematomas. These symptomatic lesions are extremely rare[7]. Spinal dural AV fistulas are fed by the radicular arteries and/or the surrounding vertebral branches[7]

After classification of Spetzler et al[5] there are extradural AV fistulas ventral or dorsal, with single or multiple feeders. There is no gender predilection (male 52.9%) of AVFs.[8]

Common to unruptured AVFs are symptoms of myelopathy and pain, such as lower or upper extremity weakness, abnormal sensory, disturbance of gait, back pain, and bladder and/or bowel incontinence[8].Most cases of ruptured AVFs are manifested as spontaneous epidural hematoma (64.7%)[8]. Typically, initial symptoms of spinal epidural hematoma are rapid development of excruciating back pain, with or without neurological deficit, followed by rapidly progressive severity of myelopathy, such as abnormal sensory, disturbance of gait, bladder and/or bowel incontinence, lower or upper extremity weakness/paralysis due to the location of hematoma[9]

After Steinmetz[10] there are two options in the treatment of spinal dural AV fistulas; surgical occlusion of the intradural reflux vein, and endovascular therapy employing embolic material into the fistula. Surgery is a relatively simple and safe intervention, resulting in long-term shunt occlusion in 98% of cases[10]. Any AVFs with progressive myelopathy have to be treated as soon as diagnoses are made. After Lim[4], surgical disconnection of the intradural radicular vein is the curative method, so is endovascular occlusion of the fistula and proximal part of its venous drainage with liquid embolic material. Prompt diagnosis and emergency surgical treatment are crucia[11].

The best management for AVFs is still surgical operation[11].

Our case illustrates a severe compression of the thoracal spinal cord produced by a combination of degenerative lesion (medial degenerative osteophites T10-T11) and posterior extradural AVF. We have realized a correct treatment of these lesions with good results.

Conclusions

Surgical treatment was the mandatory method of healing of the AVF and decompression of the dorsal spinal cord.

The delay in diagnosis left residual incomplete paraplegia.

References

1. Manisha Choudhary, Ira Sharma, Manveen Kaur, Varsha Dalal,1 and Avninder Singh Immunohistochemical Expression of Wt-1 Helps to Differentiate Cutaneous Vascular Tumors from Vascular Malformations. Indian Dermatol Online J. 2017 Jul-Aug; 8(4): 282–284.
2. Racchiusa S, Cavallaro M, Longo M, et al. Spinal cord arteriovenous malformations: a practical guide. J Neurol Stroke. 2018;8(2):113–115. DOI: 10.15406/jnsk.2018.08.00292
3. Bao YH, Ling F. Classification and therapeutic modalities of spinal vascular malformations in 80 patients. Neurosurgery 1997;40:75 81.
4. Lim S.M., Choi I.S. Spinal Epidural Arteriovenous Fistula:a Unique Pathwayinto the Perimedullary VeinA Case Report. Interventional Neuroradiology 15: 466-469, 2009
5. Spetzler R. , Detwiler P. W. , Riina H., Porter, R. W. Modified classification of spinal cord vascular lesions, J. Neurosurg: Spine / Volume 96 / March, 2002
6. Geibprasert S, Pereira V et Al: Dural arteriovenousshunts: a new classification of craniospinal epidural ve-nous anatomical bases and clinical correlations. Stroke39: 2783-2794, 2008
7. Krings T., Lasjaunias P.L., Geibpraswert S., Hans FJ., Thron A.K. , Terbrugge K.G., Reinges M.H.T. Classification of Spinal Vascular Malformations. The Neuroradiology Journal 22 (Suppl. 1): 97-106, 2009
8. Yamamoto S. , Kim P. Spinal Arteriovenous Fistulasand Arteriovenous Malformations– Complicated Vasculature and Surgical Imaging http://dx.doi.org/10.5772/56367. Intech, Chapter 5, pag 2-21, 2013
9. Strom RG, Derdeyn CP, Moran CJ, et al. Frequency of spinal arteriovenous malformations in patients with unexplained myelopathy. Neurology 66(6), 928-931, (2006).
10. Steinmetz MP, Chow MM, Krishnaney AA, Andrews-Hinders D, Benzel EC, Masar-yk TJ, et al. Outcome after

the treatment of spinal dural arteriovenous fistulae: a con-temporary single-institution series and meta-analysis. Neurosurgery. 2004 Jul;55(1):77-87;

11. Singh, et al.: Spinal arteriovenous malformations: Indications for surgery. Asian Journal of NeurosurgeryVol. 11, Issue 2, April June 2016.

Romanian Neurosurgery (2022) XXXVI (1): pp. 23-27
DOI: 10.33962/roneuro-2022-004
www.journals.lapub.co.uk/index.php/roneurosurgery

Clinical-pathological features and molecular background of oligodendrogliomas. A single centre retrospective study

G. Popescu[1], Francesca Paslaru[1], A.C. Paslaru[2,3], M. Apostol[1], M.C. Zaharia[1], M. Popescu[4], R.M. Gorgan[1,2]

[1] 4th Neurosurgical Department, "Bagdasar Arseni" Clinical Emergency Hospital, Bucharest, ROMANIA
[2] "Carol Davila" University of Medicine and Pharmacy, Bucharest, ROMANIA
[3] Department of Genetics, "Dr. Victor Gomoiu" Children's Clinical Hospital, Bucharest, ROMANIA
[4] Department of Neurosurgery, Pitesti Emergency Hospital, University of Pitesti, ROMANIA

Keywords
oligodendrogliomas, molecular

Corresponding author:
G. Popescu

"Carol Davila" University of Medicine and Pharmacy,
Bucharest, Romania

george_popescu39@yahoo.com

ABSTRACT

Background: Diffuse gliomas are the most frequent primary central nervous system (CNS) neoplasms, originating from the parenchyma itself, oligodendrogliomas accounting for approximately 10% of cerebral gliomas. For the past 20 years, the study of genetic/molecular mechanisms of gliomagenesis and progression has gradually come into focus. However, the biological and clinical significance of these mutations is still to be completely characterized. The purpose of this article is to describe our clinical experience with oligodendrogliomas and to review the current literature, in order to better describe the characteristics of the molecular/genetic oligodendroglioma subgroups.

Methods: We performed a single-institution retrospective study that included 66 patients with oligodendrogliomas operated in our department between January 2011 and December 2018.

Results: Our study included 26 female patients (39%) and 40 male patients (59%). The mean age at presentation was 39.9year-old (range 26-59year-old). The tumours were located predominantly in the right hemisphere (53%), the majority being situated in the frontal lobe (59%). 64% of the patients had signs of mass effect on the imaging studies, 13% presented with brain herniation syndromes, 16 % of the surgically treated patients had a relapse with regrowth and malignant transformation of the tumour. The most common complaint that the patients had at admission was headaches. Seizures were the second most common symptom that determined the patients to seek medical attention.

Conclusion: The expanding knowledge regarding the genetic alterations of oligodendroglial tumours could lead to significant changes in treatment strategies. However, the utility of each particular marker in planning the treatment has yet to be established. Emerging data will, most likely, improve outcome prediction and adjuvant therapy strategies through identifying the patients most likely to benefit from a particular treatment.

Scan to access the online version

Introduction

Diffuse gliomas are the most frequent primary central nervous system (CNS) neoplasms, originating from the parenchyma [1]. About 75% of gliomas in adults are astrocytic, two-thirds being glioblastomas, the most malignant form. Oligodendroglial tumors account for than 10% of the gliomas [2]. For the largest part of the last century, the diagnosis of oligodendrogliomas has been based on histopathological aspects alone. For the past 20 years, the study of genetic/molecular mechanisms of glioma genesis and progression has gradually come into focus. The 2021 World Health Organization (WHO) Classification of Tumors of the CNS includes molecular features for diagnosis and further classification of oligodendroglioma into IDH-mutant and 1p/19q-codeleted [3]. The biological and clinical significance of these mutations are still to be completely characterized. The purpose of this article is to describe our clinical experience with oligodendrogliomas and to review the current literature, in order to better describe the characteristics of the molecular/genetic oligodendroglioma subgroups.

Materials and methods

We performed a single institution retrospective study that included the patients of the 4th Clinical Department of Neurosurgery of the Bagdasar-Arseni Clinical Emergency Hospital.

We retrospectively reviewed the case files of 66 patients with oligodendrogliomas operated in our department between January 2011 and December 2018. We only included patients operated for oligodendrogliomas with a positive anathomopathological examination for a "classical" oligodendroglioma. Exclusion criteria were diagnosis of primary glioblastoma, oligoastrocitoma with important oligodendroglial compound or patients with a high suspicion for oligodendroglioma, that refused surgery or biopsy for diagnosis. Data were obtained by studying patient files.

Results

Our study included 26 female patients (39%) and 40 male patients (59%). The mean age at presentation was 39.9year-old (range 26-59year-old). The tumors were located predominantely in the right hemisphere (53%), the majority being situated in the frontal lobe (59%). 64% of the patients had signs of mass effect on the imaging studies, 13% presenting with brain herniation syndromes (Figure 1).

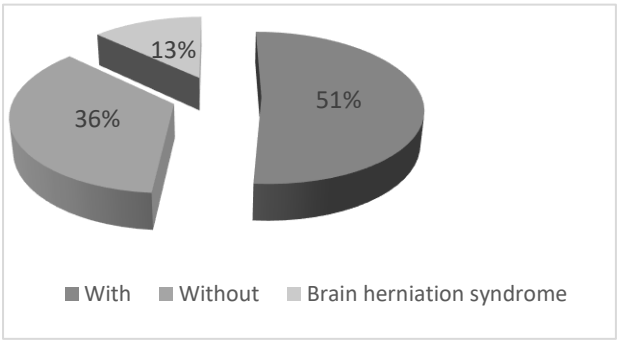


Figure 1. Distribution of patients based on the presence/absence of mass effect on imaging studies

16 % of the surgical treated patients had a relapse with regrowth and malignant transformation of the tumor (Figure 2).

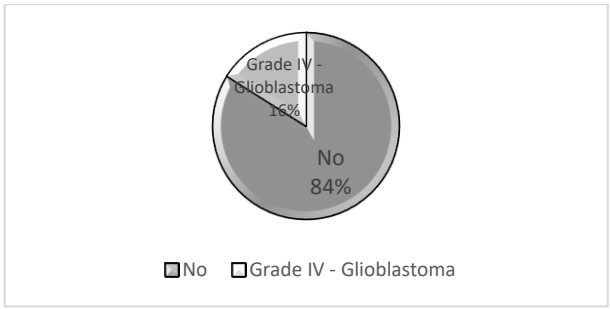


Figure 2. Distribution of the patients based on the presence/absence of relapse with malignant transformation

The most common complaint that the patients had at admission was headache. Seizures were the second most common symptom that determined the patients to seek medical attention (Figure 3).

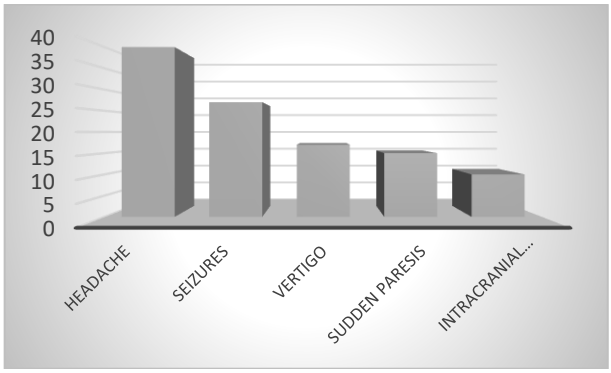


Figure 3. Distribution of the patients based on the clinical symptoms at admission

ILLUSTRATIVE CASE

A 30-year-old male with no significant medical history was referred to our clinic for intense headache, progressively worsened during the month prior to admission. Clinical examination showed no neurological deficit. Gadolinium-enhanced MRI scan revealed a right fronto-temporal tumor, measuring 9.5 cm in diameter, compressing the adjacent structures and producing mass effect on the right lateral ventricle (Figure 4).

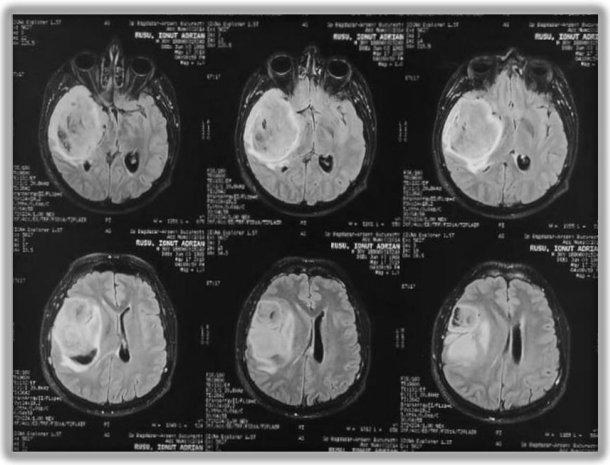


Figure 4. Gadolinium enhanced MRI scan, axial section, showing a right fronto-temporal tumor, measuring 9.5 cm in diameter, compressing the adjacent structures with inhomogenous enhancement

We performed a gross total resection through a pterional approach (Figure 5, Figure 6).

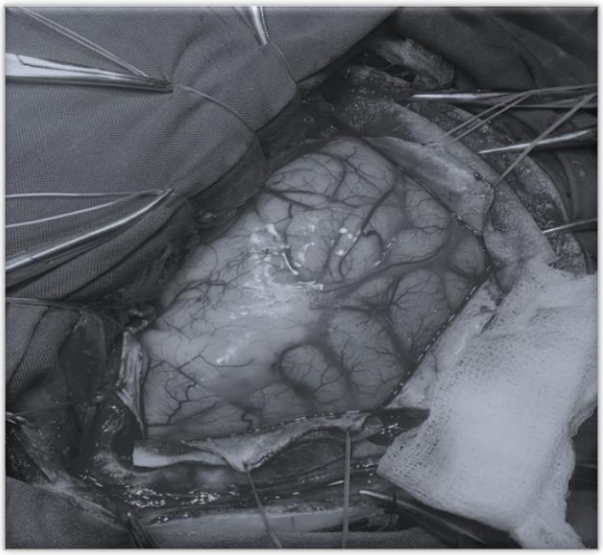


Figure 5. Intraoperative aspect showing the yellow-grey tumour

Figure 6. Intraoperative aspect showing the resection cavity

The patient was discharged 7 days later, with no postoperative neurological deficits. Control CT scan showed a gross total resection of the tumor (Figure 7).

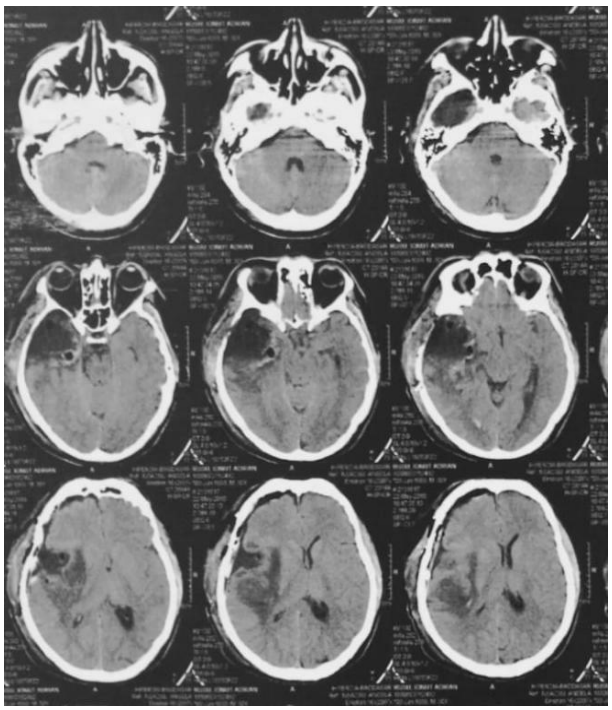


Figure 7. Postoperative CT scan showing the resection cavity

Histopathological examination showed arciform vascularization (figure 8), "boiled egg cells" (figure 9) and high fibrilarity (Figure 10).

Figure 8. Histopathological specimen showing arciform vascularization

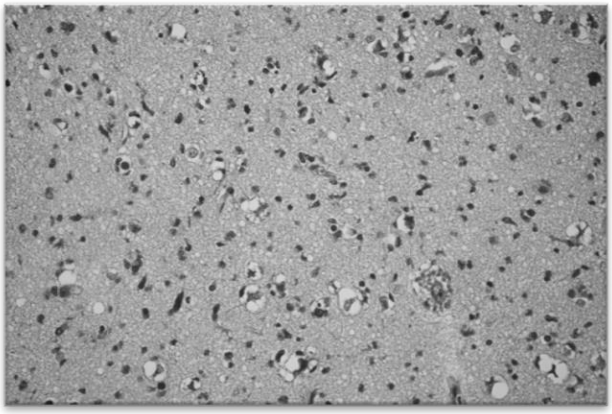


Figure 9. Histopathological specimen showing "boiled egg cells" aspect

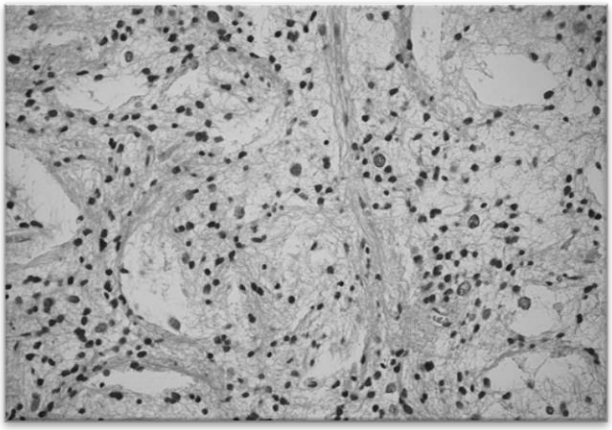


Figure 10. Histopathological specimen showing high fibrillarity

MLPA analysis of the tumor sample identified a codeletion of 1p19q and a c.395G>A (p.R132H) mutation in the exon 4 of IDH1 gene. The analysis was negative for mutations of exons 11 and 15 of BRAF gene, EGFRvIII, MGMT promoter methylation and TERT promoter mutation.

The patient had a favorable evolution, with no neurological deficits and was discharged in the seventh postoperative day.

Discussion

Under the generic term "oligodendroglioma" is a heterogenous group of tumors, with a variable response to adjuvant therapy. This variance highlights the need for markers that can guide the clinical decision-making. Codeletion of 1p19q occurs in 50 to 76% of oligodendrogliomas [4] [5]. EORTC 26951 and RTOG 9402 studies proved that combining radiation therapy with procarbazine, vincristine and lomustine chemotherapy protocol drastically increased overall survival in 1p19q codeleted anaplastic oligodendroglioma patients compared to radiotherapy alone [6] [7]. The EORTC 26951 trial investigated the adding of six cycles of standard procarbazine, vincristine and lomustine to radiation therapy of 59.4 Gy in 33 fractions in anaplastic oligodendroglioma patients and reported a significant difference in overall survival [7] [8]. Patients with 1p19q codeleted oligodendrogliomas benefitted more from the addition of chemotherapy to radiotherapy, the risk reduction in patients with non-codeleted tumors being significantly lower [7]. Regarding both trials, it is also notable the fact that patients with 1p19q codeleted oligodendrogliomas who were treated with adjuvant radiotherapy alone initially, had a lower survival rate at progression, despite being administered a heightened chemotherapy regimen [4] [7]. The CODEL trial has been designed to compare administration of adjuvant therapy consisting of either radiotherapy alone, themozolomide alone or radiotherapy combined with themozolomide. The analysis from the initial study design showed that themozolomide-alone patients experienced a significantly shorter progression free survival, compared to either one of the radiotherapy arms [9]. The study has been subsequently redesigned to compare radiotherapy combined with procarbazine, lomustine and vincristine to radiotherapy and themozolomide regimens and is still ongoing.

Mutations of IDH1 and IDH2 seem to occur in about 70% of oligodendroglioma tumors, mainly

affecting amino acid 132 of IDH1 or IDH2 [10] [11]. IDH1 is known to function as a tumor suppressor, its mutational inactivation leading to tumorigenesis, partially through the induction of the HIF-1 pathway [12] [13]. IDH mutations have been reported in several studies to produce a favorable prognostic impact [7] [10] [14]. However, despite the more favorable prognosis of patients with oligodendrogliomas harboring IDH mutations, it hasn't been proven yet that the treatment strategy should be changed regarding the IDH status.

Conclusions

The expanding knowledge regarding the genetic alterations of oligodendroglial tumours could lead to significant changes in treatment strategies. However, the utility of each particular marker in planning the treatment has yet to be established. Emerging data will, most likely, improve outcome prediction and adjuvant therapy strategies through identifying the patients most likely to benefit from a particular treatment.

Romanian Neurosurgery (2022) XXXVI (1): pp. 28-29
DOI: 10.33962/roneuro-2022-005
www.journals.lapub.co.uk/index.php/roneurosurgery

Management of nontraumatic intracranial haemorrhage (subdural hematoma) in immune thrombocytopenia. Case report

D. Balasa[1], Al. Tunas[1], V. Stan[1], T. Adam[2]

[1] Department of Neurosurgery, Clinical Emergency Hospital Constanta, ROMANIA
[2] Department of Haematology, Clinical Emergency Hospital Constanta, ROMANIA

ABSTRACT
Intracranial haemorrhage is a devastating complication of immune thrombocytopenic purpura [1]. The occurrence of a spontaneous subdural hematoma in immune thrombocytopenia (ITP) is rare [2], affecting 1% or less of patients [3]. In ITP contrary to traumatic SDH the brain parenchyma is well preserved [3]. We present the case of a patient with immune thrombocytopenia, subdural haemorrhage and asymptomatic parietal parasagittal meningioma. Neurological parameters were closely monitored, including the level of consciousness, pupillary size, motor or sensorial deficit. He was managed successfully medically (platelet-rich plasma and steroids) and then surgically (craniotomy, subdural hematoma aspiration).

Keywords
hematoma, subdural, subarachnoid haemorrhage, idiopathic thrombocytopenic purpura

Corresponding author:
Balasa Daniel

Department of Neurosurgery, Clinical Emergency Hospital Constanta, Romania

balasadaniel100@yahoo.com

INTRODUCTION

We present tha case of a patient with imune thrombocytopenia intracerebral hemorhage and parietal parasagital meningioma.

CASE REPORT

We present the case of a patient who suferred a head trauma in uncclеared conditions. He acuse mild left hemiparesis (ASIA 4/5), intense headache VAS 8/10, vomiting and diziness, from 3 days. Few purpuric spots were noted on all the four members.

Medical datas revealed chronical ITP, without continous treatment Hemoglobine: 14,20 g/dl, TLC 4000/cm3. Coagulation tests were normal.

Clinical exam revealed mild hemiparesis (ASIA 4/5), osteotendinous reflexes diminished on the left side, Babinsky on the left side, purpuric lesions on all the four members. Glasgow scale 15.

CT scan of the head revealed hyperdensity in the subdural space in the temporo parieto occipital region on the right side, and in the subdural area in right posterior part of the sagital sinus,left parasagital meningioma.

Scan to access the online version

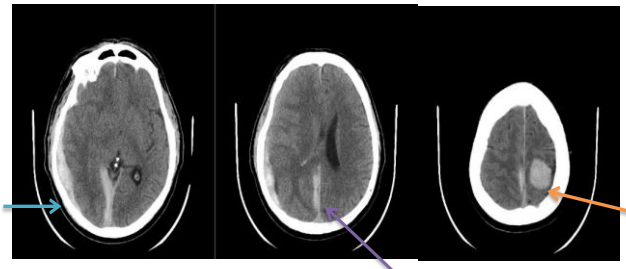


Figure 1. Temporoparietooccipital acute subdural hematoma (blue arrow). Acute subdural interemispheric (falx cerebri) (Pink arrow). Parietal parasagital meningioma (orange arrow).

The patient was treated with Dexametazone 40 mg/day and platelet transfusion.

After 4 days her platelet count rose to 130000/mm3 who allowed surgical intervention. Clinical status was stationary : intense headache (VAS 7/10), left hemiparesis (ASIA 4/5), vomiting 1-2/day, GCS 15.

The patient was operated (frontotemporo-parietoociipital craniotomy, complete evacuation of the subdural hematoma).

Figure 2. Complete evacuation of the subdural hematoma

Clinical postoperative evolution was very good with healing of hemiparesis, of headache, vomiting and diziness. Persisted only slight left pyramidal syndrome.

Discussion

Essential thrombocitopenia is revealed by constant diminution of the platelets without any cause. (Denis, Hayem, Frank.) ITP was first described by Werlhofin 1735[4] as an aquired disorder which leads to immune mediated destruction of platelets characterised by low platelet count and normal coagulation studies[4]. intracranial hemorrhage is a devastating complication of ITP[1,4]. The occurence of a spontaneous subdural hematoma in immune thrombocytopenia (ITP) is rare[2], affecting 1-2% or less of patients[3,4,5]. The clinical features are mainly headache, hemiparesis, signs of raised intracranial tension, altered consciousness[4]. Usually, subdural hematoma occurs, when are associatecd with ITP around the the top and side of the frontal and parietal lobes, in the posterior cranial fossa, near the falx cerebri and tentorium cerebelli[4]

.

Conclusions

- Medical treatment enabled us to achieve an adequate hemostasis wich was esential to be able to perform surgery in proper time.
- Combination between medical treatment of imune thrombocitopenia and surgical treatment of acute subdural hematoma was mandatory for a good clininical and neurological evolution.

References

1. Pavithran K , Thomas M Management of subdural Hematoma in imune thrombocytopenic purpura: Report of seven patients and a literature review. Clinical Neurology and Neurosurgery, Vol 111, Issue 2, February 2009, 189-192
2. Mathews M H, Yu W, Chappell E T. Spontaneous Subdural Hematoma in the setting of Imune Thrombocytopenia Complicated by ischemis infarcts. Neuroradiol J, 2007 Apr 30 (2): 224-227. Epub 2007 Apr 30
3. Sunitha R, Mathew R, Thomas M. Conservative management of subdural hematoma in idiopathic thrombocytopenic purpura: Report of two cases and review of the literature. ANN Indian Acad Neurol 2007; 10: 184-6
4. Chatterjee S, Karmakar PS, Ghosh P, Ghosh A. Subdural Hematoma associated with thrombocytopenic purpura in two different settings.The journal of the association of Phisicians of India, 2010, vol 58, 504-506
5. Meena A K, Murthy J M. Subdural haematoma in a patient with immune thrombocytopenic purpura. Neurol India 1999;47:335.
6. Brill N. E., Rosenthal N. Treatment by splenectomy of essential thrombocytopenia (Purpura hemorrhagica). Arch Intern Med (Chic). 1923; 32 (6): 939-953.

Romanian Neurosurgery (2022) XXXVI (1): pp. 30-38
DOI: 10.33962/roneuro-2022-006
www.journals.lapub.co.uk/index.php/roneurosurgery

Novel technique for intraoperative imaging of the vertebral artery in patients undergoing dorsal osteosynthesis of the cervical spine

O. Pavlov[1], N. Mirchev[2], A. Ruiz[2], J. Obersheimer[3], K. Kallenberg[3], R. Behr[2]

[1] Klinikum Krefeld Helios, Department of Neurosurgery, Krefeld, GERMANY
[2] Klinikum Fulda, Department of Neurosurgery, Fulda city, GERMANY
[3] Klinikum Fulda, Department of Neuroradiology, Fulda city, GERMANY

Keywords
vertebral artery,
cervical osteosynthesis,
hangman fracture,
spinal fractures,
spine,
cervical vertebrae

Corresponding author:
Orlin Pavlov

Klinikum Krefeld Helios,
Department of Neurosurgery,
Krefeld, Germany

orlinpavlov88@gmail.com

ABSTRACT

Background: The incidence of fractures in the upper cervical spine is a common entity among elderly patients. The incidence depends on the health care system and the adopted protocols for a computer tomogram in the emergency unit, in order to assure an easier and earlier detection. Injuries to the vertebral artery could occur with devastating consequences, due to this type of fracture. The aim of the current surgical strategies is to stabilize the fractures and avoid further injuries to the vertebral arteries.

Methods: We adopted a previously hypothesized method for intraoperative imaging of the vertebral artery when performing an O-Arm navigated cervical osteosynthesis. Three patients were included in this group: one patient with dens fracture type II after Anderson and D'Alonzo, one patient with a complex C1/C2 fracture and dissection of the vertebral artery on the left side and one patient with a kyphotic deformity due to Larynx carcinoma Metastasis in C4-C6. After positioning the patients, a 100 ml contrast was applied prior to the primary O-Arm scan, thus obtaining real-time imaging of the vertebral artery in the navigational CT series.

Results: A total of 18 screws were placed. None of the screws bridged the vertebral canal. The average operative time was 174 mins. No new neurological deterioration was observed in the postoperative period.

Conclusions: With the development of this technique, it is possible to reduce the surgery-related injury of the vertebral artery to 0%. Further studies are necessary to assess the feasibility of this technique. This technique could be especially helpful in the cases of distorted anatomical relations.

Scan to access the online version

INTRODUCTION

The incidence of fractures in the upper cervical spine in elderly patients (>65 years) after trauma lies between 1,26% and 3% (Squarza, Uggetti et al. 2019). The precise incidence of those fractures is hard to be determined because it depends on the health care system in every

country and the adopted protocols for emergency computer tomogram (CT) in every emergency department. Robinson et al. reported an increase in the incidence of C2 fractures from 3 to 6 per 100 000 from 1997 to 2014 (Robinson, Olerud et al. 2017), which corresponds to the development of strict protocols for emergency CT of cervical spine in the elderly population. CT has turned to be golden standard for detection of fractures of the upper cervical spine with ability to detect up to 99% of the fractures (Acheson, Livingston et al. 1987). Due to the alternations of the in the upper cervical spine, including reduced bone density and increased rigidity, upper cervical spine is more susceptible to flexion, extension, rotation traumas.

The incidence of the vertebral artery injuries (VAI) after trauma is reported to be between 0, 5% and 88% (Willis, Greiner et al. 1994, Gleizes, Jacquot et al. 2000, Fassett, Dailey et al. 2008, Mueller, Peters et al. 2011) The majority of those VAI remain clinically silent, but this injury could lead to major stroke and neurological deterioration. The mortality rate following VAI reported in the literature varies from 8% to 18% (Fusco and Harrigan 2011). Up to 70% of the traumatic VAI have an associated cervical spine fracture (Miller, Fabian et al. 2001). Three major diagnostic methods are available for detection of VAI, including duplex ultrasonography (sensitivity 38,5%, specificity 100%), magnetic resonance angiography (sensitivity 43%, specificity 97%) and computer tomography angiography (CTA) (sensitivity 53%, specificity 99%) (Eastman, Chason et al. 2006, Harshavardhana and Dabke 2014), whereas the duplex ultrasonography is not reliable for detection of intimal tears. The treatment options of VAI include: 1. Observation, 2. Anticoagulation, 3. Surgery (Nizare, Abdelali et al. 2013, Harshavardhana and Dabke 2014).

The treatment modalities for upper cervical spine fractures (c-spine) vary from halo-fixation to surgery, including fixation with hooks and rods, wiring, transarticular screws fixation, lateral mass screw fixation or ventral fusion (Vergara, Bal et al. 2012). For the treatment of C1/C2 fractures, the lateral screw fixation (Goel/Harms technique) and the transarticular screw fixation (Magerl technique) have shown a superior fusion rate, thus gaining more acceptances among the spine surgeons. Both of these techniques could be performed free-hands, with C-arm fluoroscopy or with navigation for 3D imaging (O-Arm, C-Arm with integrated navigation). The postoperative complications rate in Goel/Harms technique is reported up to 10,6% and in Magerl technique up to 21%(Neo, Sakamoto et al. 2005). The risk for vertebral artery injury, when performing either of both techniques is between 8% and 9,5% (Yeom, Buchowski et al. 2013, Pavlov, Mirchev et al. 2020) when using C-Arm fluoroscopy. To our knowledge, the incidence of vertebral injury during dorsal stabilization with O-Arm navigation or free-hands is unknown.

We already suggested a possible novel technique for the intraoperative visualization of the vertebral artery using hybrid technique from two visualization modalities: O-Arm navigation and CT-Angiography (CTA) (Weller, Rossitch et al. 1999). The core of this technique lies in the administration of intravenous contrast (as for a common CTA) before the intraoperative CT, which is performed by the O-Arm navigation. The aim of this hybrid technique is to bring the intraoperative injuries of the vertebral artery to 0% (Pavlov, Mirchev et al. 2020). To our knowledge, this technique has not been published up to the date of the submission of this paper and we would present the first three cases, utilizing this method.

Material and methods

In our pilot group, three patients were included: Case1, Case 2 and Case 3. A number of possible contraindications have to be considered: known allergic reaction to contrast, uncooperative patients and pregnant patients. In none of the patients, contraindications for this hybrid technique were found. Consent of the patients was obtained prior to surgery. To perform this surgical technique, the following protocol was used(Pavlov, Mirchev et al. 2020):

1. Indications/Contra-indications for this technique, careful anamnesis has to be taken for allergies against contrast.
2. Prone positioning, patient is positioned in the carbon Mayfield clamp, in order to reduce the artifacts from the intraoperative CT
3. O-Arm navigational system positioning over the targeted area, the position is saved and the O-arm needs to be brought to parking position, in order to ensure a maximal working space for the surgeon.

4. Surgical approach to the target area (C1/C2 complex or upper cervical spine)
5. Re-positioning of the O-Arm navigation over the area of interest
6. Bolus manual application of contrast-100ml through central line, 5 seconds before the intraoperative scan for optimal visualization of the vertebral artery and eliminating the venous contrast artefacts.
7. Ventilation pause and carrying out an intraoperative CT scan, then the data is transferred to the surgeon's monitor
8. Screws insertion under 3D guidance with intraoperative visualization of the vertebral artery
8. Ventilation is paused and another intraoperative CT scan is carried out to verify the correct position of the screws.
9. Wound closure

CASE 1

Diagnosis: Dens fracture Type II Anderson and D'Alonzo

Surgical strategy: Dorsal osteosynthesis in Goel/Harms technique C1-C2

Brief summary of the patient's history: A 95-years old patient, in a good clinical condition, presents herself in our emergency room after falling from her sofa backwards. She complained pain in the dorsal region of the c-spine without neurological deficits. The anamnesis showed pre-existing strokes in the left and right medial cerebral artery.

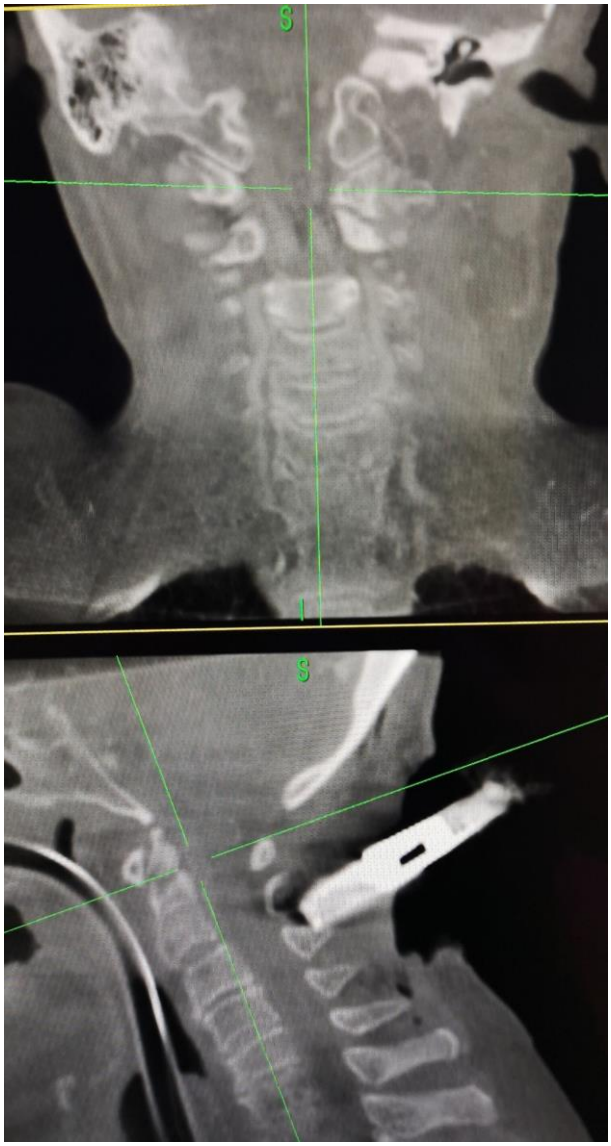


Figure 1 and 2. 3D Data with intraoperative imaging of the vertebral artery

The trauma scan depicted dens fracture type II after Anderson and D'Alonzo classification. The surgery was indicated and after obtaining patient's consent, the surgery was carried out on the 2nd day after the admission of the patient.

The indication for applying contrast, during the surgery, in this patient was based on the pre-existing strokes, suggesting progressive atherosclerosis, and a sufficient kidney function (glomerular filtration rate- 71ml/min and creatinine 1,3 mg/dl).

The application of contrast allowed a good intraoperative imaging of the vertebral artery, optimal length of the screws was chosen without risking a vascular injury (Fig 1-3).

Figure 3. 3D Intraoperative navigation with direct imaging of the vertebral artery

CASE 2

Diagnosis: Complex C1 fracture (Jefferson fracture II)/ C2 fracture (Hangmann fracture) and a. vertebralis dissection links

Surgical strategy: Dorsal fusion in Goel/Harms technique C1/C2 and massa lateralis screws in C3

Brief summary of the patient's history: A 79-year old patient presented at our emergency room after a 6 fall from a roof. The patient complained pain in the dorsal region of the cervical pain without radiculopathy or myelopathy. The neurological examination showed normal neurological status. The anamnesis depicted no pre-existing health issues.

The trauma scan depicted a complex C1/C2 fracture, where the consecutive CTA showed a dissection of the vertebral artery on the left side. Because of the dissection of the vertebral artery, the patient was put on antiplatelet therapy with aspirin. Surgery was indicated and after obtaining the patient's consent, it was carried out on the 7th day.

The indication for applying a contrast in this patient was based on the previous traumatic injury of the vertebral injury on the left side and preserved kidney function (glomerular filtration rate- 69ml/min and creatinine 1,5 mg/dl.

During surgery, after the application of contrast and after carrying out the intraoperative CT, the occlusion of the left vertebral artery was still visible (Fig. 4).

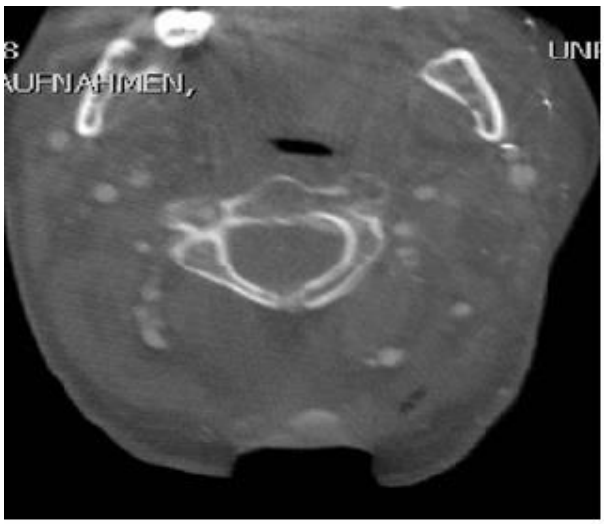


Figure 4. Intraoperative CT Navigation with contrast, performed with O-Arm, where dissection of the left vertebral artery is still visible

Polyaxial screws on both sides in massa lateralis and in the pedicles were placed. The postoperative condition of the patient deteriorated due to postoperative delirium, which was treated with atypical antipsychotic (Risperidone) and Benzodiazepines (Diazepam).

The postoperative c-spine CT showed an optimal placement of all screws.

CASE 3

Diagnosis: Larynx carcinoma Metastasis C4-C6, cervical instability and kyphotic deformity C4-C6

Surgical strategy: Dorsal osteosynthesis (massa lateralis and pedicle screws) C3-D1

Brief summary of the patient's history: The case of the 57-years old patient with larynx carcinoma was discussed at Neuro-Oncological Board due to a new metastasis in the c-spine with progressive deformity. The patient complained pain in the middle cervical spine with irradiation of the pain in shoulders. The neurological examination showed intact neurological status. The patient had tracheostoma because of the larynx carcinoma, making a ventral approach to the C-Spine not possible.

An MRI and CT of the c-spine showed destructive process in C4 to C6 with kyphosis, without tumour bridging in the spinal canal. After interdisciplinary discussion of this case, regarding life-expectancy, neurological outcome and patient's will, surgery was indicated.

After obtaining patient's consent, the surgery was carried out on the 3rd day after admission.

The indication for applying intraoperative contrast was based suspicion of tumour infiltration on left vertebral canal C4-C6.

The intraoperative CT-navigation with contrast depicted a tumour mass from C4 to C6, infiltrating the left vertebral canal, without pressuring the artery.

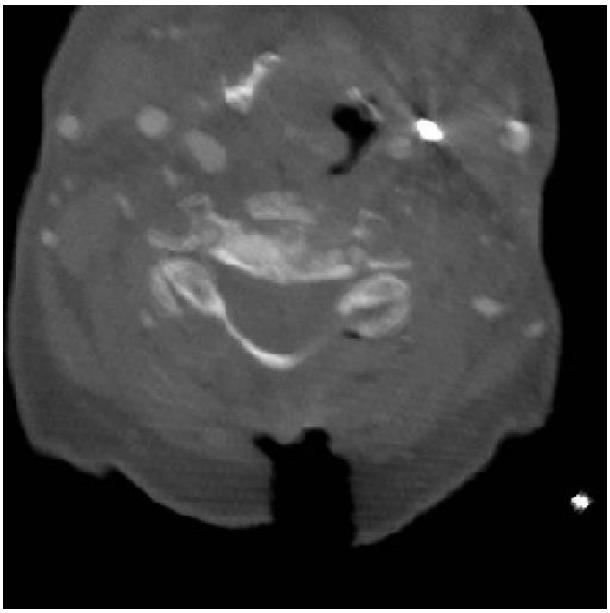


Figure 4. Tumour infiltration in the left vertebral canal C5

After the screw insertion, a second scan was carried out. The second scan depicted a perfect positioning of the screws, unfortunately the vertebral artery was not visible any longer (94 minutes after the initial scan) Fig.8.

Postoperatively the patient recovered well and was discharged on the 7th day.

Results

A total of 18 screws were placed (massa lateralis or pedicle screws). None of them breached the walls of vertebral canal. The average operative time was 174 minutes in the three surgeries.

Using this imaging technique, the vertebral artery was depicted from segment V1 to segment V3, without discontinuity in any of the vertebrae levels. On the second scan, in none of the cases, the vertebral artery was visible any longer.

Discussion

The interest of fusion techniques in intraoperative neuroimaging exists for a long time, especially when vascular structures are involved. Leng et al. were the first to describe the fusion between intraoperative three-dimensional rotational angiography and flat panel detector computed tomography for cerebrovascular navigation.(Leng, Rubin et al. 2013) In this case report, the treatment of a patient harbouring two aneurysms was described, using this fusion/hybrid technique. Although, it is not obligatory to use this technique, this method adds to the arsenal of surgical strategies, which could be used for intraoperative support. Since both the c-spine trauma and the stabilisation technique independently add up to the risk of VAI, it is mandatory to utilize all the possible techniques to preserve the functional vertebral artery, in order to avoid further neurological deterioration. In all patients, where VAI is suspected, aggressive actions need to be taken, in order to confirm the diagnosis because of its therapeutic significance.

The preoperatively obtained CT/CT-Angiography is a valid method for intraoperative navigation in trauma cases. Fiorenza et al. conducted a retrospective study in a series of 21 patients, who underwent a posterior upper cervical spine fixation using a navigational system with intraoperative single level vertebral registration on preoperative CT/CT-Angiography (Fiorenza and Ascanio 2019). No neurovascular damage occurred in any of the patients. In the treatment of upper cervical trauma cases, however, it is not mandatory to conduct a preoperative CT-Angiography (Lockwood, Smith et al. 2016). The development of strict protocols for CT-Angiography in trauma patients is still in development. Lockwood et al. presented one of the most significant studies for CT-Angiography in patients with upper cervical spine, up to date (Lockwood, Smith et al. 2016). Using the Denver Criteria (Biffl, Moore et al. 1999, Kerwin, Bynoe et al. 2001, Cothren, Moore et al. 2003, Parks and Croce 2012) for high risk fracture pattern, the authors analysed 1435 patients, where only combined C1-C2

fractures, subluxations and transverse foramen involvement were predictive for VAI. The odds for developing of VAI were 3,8, 4,8 and 6,3 respectively. Of the 1435 patients with cervical spine fractures, only 10 (0,7%) suffered from posterior circulation stroke(Lockwood, Smith et al. 2016).

The preoperative CT-Angiography gives the surgeons valuable information about the position of the vertebral artery and its position the osseous structures. However, it is interesting to point out, that many studies have shown, that VAI shown in preoperative CT-Angiography and applying treatment (anticoagulation or antiplatelet therapy) does not prevent stroke (Eastman, Chason et al. 2006, Scott, Sharp et al. 2014, Lockwood, Smith et al. 2016). This data would suggest that the obtained CT-Angiography would not have any effect on the neurological outcome of the patients after trauma. The use of our suggested technique shows a real-time intraoperative visualization of the vertebral artery. One must consider that the patient's head is fixated in a rigid collar for the CT-Angiography and prior to the operation, the collar is taken off and patient's head needs to reclined and fixated in Mayfield for the posterior fixation. In this manoeuvre, the position of the vertebral artery does not change in respect to the osseous anatomy, however its blood flow could be changed because of kinking due to fractures (George and Carpentier 2001, Berti, Zafar et al. 2018). Our suggested technique could provide the surgeon with certainty, that prior to the screw insertion, that both of the vertebral arteries are intact. O-Arm navigation has also shown to reduce the operative time, in comparison with the preoperative scans with image guidance (Costa, Cardia et al. 2011) and to reduce the radiation exposure compared to fluoroscopic guidance(Costa, Cardia et al. 2011, Nottmeier, Pirris et al. 2013). Performing intraoperative CT at the end of the procedure, reduces the need to return the patients to the operating room.(Van de Kelft, Costa et al. 2012)

Associated atlas and axis fractures account for 3% of acute cervical spine lesions and 12% of the upper cervical spine lesions (Jung, Jung et al. 2010), where the normal anatomy of the C1/C2 complex is altered, with possible displacement of the vertebral artery. In those cases, even with conventional O-Arm navigation, an injury of the vertebral artery is possible, even with 99,9% accuracy of the screw positioning(Bydon, Martin Ma et al. 2014).

Tumours of the craniocervical junction (CCJ) represent a challenging entity for the neurosurgeon(Bydon, Martin Ma et al. 2014). Bydon et al. showed that the rate of vertebral artery involvement significantly effects the rate of radical resections of the tumours, located in the CCJ. The patients without involvement of the artery had a total resection, statistically significant to those with involvement of the vertebral artery. Pirotte et al. and Talacchi et al. showed that aiming for a total resection of the tumors in CCJ, which involve the vertebral artery, is related to many postoperative complications, including lateromedullary infarction, respiratory failure, cranial nerve palsy, etc.(Pirotte, Brotchi et al. 2010, Talacchi, Biroli et al. 2012). With this technique for intraoperative imaging of the vertebral artery, it could be possible to increase the percentage of tumor resection, extending the life-expectancy, tumor-free period. Kern et al. were the first to use O-Arm navigational system in 2014 for resection of a chordoma in CCJ, however no angiography was performed at the time(Kern, Indro et al. 2014). Because of the distortion of the normal bony structures due to the tumor, it is difficult to appreciate intraoperatively many important landmarks. The use of O-Arm navigation has proven to be a valuable tool in the tumor resection process.

The developed in John Hopkins University algorithm by Sciubba et al.(Sciubba, Mavinkurve et al. 2006), proved successful in treatment of hemangioblastomas in the cervical spine. 3D digital subtraction angiography provides high-resolution images of the vascular structures, however often it does not provide reliable anatomical information about the nearby osseous structures, or in the cases, when it does, resolution of the vascular anatomy in the immediate vicinity of bone sacrificed. (Sciubba, Mavinkurve et al. 2006) The authors used a novel angiographic reconstruction by combining two separate sequences of images of bone and blood vessels in a single 3D Fusion Imaging series. Although, only two case reports were presented by the authors, the need for a better osseous intraoperative imaging is noted. Su et al. already used this fusion technique between O-Arm and on-table angiography for the resection of the occipitocervical tumor in 12/2020, independently from our published hypothetical usage of this

method in 02/2020(Pavlov, Mirchev et al. 2020, Su, Prezerakos et al. 2020). This facilitates safe resection by mapping the surgical field occult to direct vision.

There are two major factors for injury of the vertebral artery, when performing a cervical osteosynthesis: High riding vertebral artery (HRVA), as an anatomical variant and narrow pedicle of the C2 vertebrae(Wang, Xia et al. 2013, Wajanavisit, Lertudomphonwanit et al. 2016). Both of these factors could be present in the setting of spinal trauma or tumor in the CCJ.

Morin et al. demonstrated the close relationship between traumatic brain injury (TBI) and the cervical spine involvement (Morin, Langevin et al. 2016) and thus the neurological outcome. Outcomes from TBI can be temporary or permanent dysfunction of cognition, motor function, physiology, and psychology (Blennow, Hardy et al. 2012) and post-TBI neurological disorders such as posttraumatic epilepsy, chronic traumatic encephalopathy and dementia (Papa, Mendes et al. 2012, Cotter, Kelso et al. 2017). The resulting costs for the treatment of these patients are increased exponentially. The patients with extracranial involvement (c-spine fractures, lung contusion, etc.) have worse outcome, compared to the patient without additional injuries(Konar, Pavlov et al. 2020).

The learning curve for a posterior fixation in the upper cervical spine represents a steep due to the complex anatomy of the CCJ, subaxial spine and its neurovascular relations (Heo, Lee et al. 2019). The proposed technique could improve the learning curve, making the posterior fixation easier for the young neurosurgeons. With the better understanding of the anatomic relations of the bones to the neurovascular structures, one would improve the operating time and minimize the blood loss.

Conclusions

With the development of this technique, we do not aim to set it as a "golden standard" for the treatment of pathologies in the c-spine. We would suggest that this technique would be "good to know" in the cases, where it is needed. Since we present only a technical note, no statistical analysis is carried out.

In the setting of polytrauma or multitrauma, where a cervical dorsal osteosynthesis is required, all measures for reducing the risk for additional injuries need to be considered. The procedure-associated risk for additional neurological deficit, due to injury of the vertebral artery, must be kept as low as possible. This technique offers the possibility for a real-time intraoperative imaging of the vertebral artery and thus protecting it.

This is a pilot cohort of patients with very promising results. Further studies will be carried out to confirm the diminished rate of VAI, to compare the average blood loss, operation time, perioperative mortality and morbidity between the patients undergoing c-spine dorsal osteosynthesis with and without this technique.

Disclosures

No disclosures

References

1. Acheson, M. B., et al. (1987). "High-resolution CT scanning in the evaluation of cervical spine fractures: comparison with plain film examinations." American Journal of Roentgenology 148(6): 1179-1185.
2. Berti, A. F., et al. (2018). "Recurrent posterior circulation infarcts secondary to vertebral artery external compression treated with endovascular deconstruction." Interventional neuroradiology : journal of peritherapeutic neuroradiology, surgical procedures and related neurosciences 24(2): 178-182.
3. Biffl, W. L., et al. (1999). "Optimizing screening for blunt cerebrovascular injuries." Am J Surg 178(6): 517-522.
4. Blennow, K., et al. (2012). "The neuropathology and neurobiology of traumatic brain injury." Neuron 76(5): 886-899.
5. Bydon, M., et al. (2014). "Surgical outcomes of craniocervial junction meningiomas: a series of 22 consecutive patients." Clinical neurology and neurosurgery 117: 71-79.
6. Costa, F., et al. (2011). "Spinal navigation: standard preoperative versus intraoperative computed tomography data set acquisition for computer-guidance system: radiological and clinical study in 100 consecutive patients." Spine (Phila Pa 1976) 36: 2094-2098.
7. Cothren, C. C., et al. (2003). "Cervical spine fracture patterns predictive of blunt vertebral artery injury." J Trauma 55(5): 811-813.
8. Cotter, D., et al. (2017). "Genetic biomarkers of posttraumatic epilepsy: A systematic review." Seizure - European Journal of Epilepsy 46: 53-58.
9. Eastman, A. L., et al. (2006). "Computed tomographic angiography for the diagnosis of blunt cervical vascular injury: is it ready for primetime?" J Trauma 60(5): 925-929; discussion 929.

10. Fassett, D. R., et al. (2008). "Vertebral artery injuries associated with cervical spine injuries: a review of the literature." J Spinal Disord Tech 21(4): 252-258.
11. Fiorenza, V. and F. Ascanio (2019). "Safety and Efficacy of Posterior Atlanto-Axial Stabilization Using Intraoperative Navigation System with Preoperative Computed Tomographic Scan." World Neurosurg 129: 110-119.
12. Fusco, M. R. and M. R. Harrigan (2011). "Cerebrovascular dissections: a review. Part II: blunt cerebrovascular injury." Neurosurgery 68(2): 517-530; discussion 530.
13. George, B. and A. Carpentier (2001). "Compression of and by the vertebral artery." Operative Techniques in Neurosurgery 4(4): 202-218.
14. Gleizes, V., et al. (2000). "Combined injuries in the upper cervical spine: clinical and epidemiological data over a 14-year period." European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society 9(5): 386-392.
15. Harshavardhana, N. S. and H. V. Dabke (2014). "Risk factors for vertebral artery injuries in cervical spine trauma." Orthopedic reviews 6(3): 5429-5429.
16. Heo, Y., et al. (2019). "The Learning Curve of Subaxial Cervical Pedicle Screw Placement: How Can We Avoid Neurovascular Complications in the Initial Period?" Oper Neurosurg (Hagerstown) 17(6): 603-607.
17. Jung, S. H., et al. (2010). "Tailored surgical approaches for benign craniovertebral junction tumors." J Korean Neurosurg Soc 48(2): 139-144.
18. Kern, H. G., et al. (2014). "The use of intraoperative navigation for complex upper cervical spine surgery." Neurosurgical Focus FOC 36(3): E5.
19. Kerwin, A. J., et al. (2001). "Liberalized screening for blunt carotid and vertebral artery injuries is justified." J Trauma 51(2): 308-314.
20. Konar, S., et al. (2020). "Critical Appraisal of Traumatic Brain Injury and Its Management." Indian Journal of Neurotrauma 17(02): 079-084.
21. Leng, L. Z., et al. (2013). "Fusion of intraoperative three-dimensional rotational angiography and flat-panel detector computed tomography for cerebrovascular neuronavigation." World Neurosurg 79(3-4): 504-509.
22. Lockwood, M. M., et al. (2016). "Screening via CT angiogram after traumatic cervical spine fractures: narrowing imaging to improve cost effectiveness. Experience of a Level I trauma center." Journal of neurosurgery. Spine 24(3): 490-495.
23. Miller, P. R., et al. (2001). "Blunt cerebrovascular injuries: diagnosis and treatment." J Trauma 51(2): 279-285; discussion 285-276.
24. Morin, M., et al. (2016). "Cervical Spine Involvement in Mild Traumatic Brain Injury: A Review." Journal of Sports Medicine 2016: 1590161.
25. Mueller, C.-A., et al. (2011). "Vertebral artery injuries following cervical spine trauma: a prospective observational study." European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society 20(12): 2202-2209.
26. Neo, M., et al. (2005). "The clinical risk of vertebral artery injury from cervical pedicle screws inserted in degenerative vertebrae." Spine (Phila Pa 1976) 30(24): 2800-2805.
27. Nizare, E. F., et al. (2013). "Upper cervical spine injuries: a management of a series of 70 cases." The Pan African medical journal 15: 57-57.
28. Nottmeier, E. W., et al. (2013). "Operating room radiation exposure in cone beam computed tomography–based, image-guided spinal surgery." Journal of Neurosurgery: Spine 19(2): 226-231.
29. Papa, L., et al. (2012). "Mild Traumatic Brain Injury Among the Geriatric Population." Current Translational Geriatrics and Experimental Gerontology Reports 1(3): 135-142.
30. Parks, N. A. and M. A. Croce (2012). "Use of computed tomography in the emergency room to evaluate blunt cerebrovascular injury." Adv Surg 46: 205-217.
31. Pavlov, O., et al. (2020). "A possible novel technique for intraoperative imaging of the vertebral artery during arthrodesis of the upper cervical spine." Medical hypotheses 140: 109641.
32. Pavlov, O., et al. (2020). "A possible novel technique for intraoperative imaging of the vertebral artery during arthrodesis of the upper cervical spine." Medical hypotheses 140: 109641.
33. Pirotte, B. J., et al. (2010). "Management of anterolateral foramen magnum meningiomas: surgical vs conservative decision making." Neurosurgery 67(3 Suppl Operative): ons58-70; discussion ons70.
34. Robinson, A.-L., et al. (2017). "Epidemiology of C2 Fractures in the 21st Century: A National Registry Cohort Study of 6,370 Patients from 1997 to 2014." Advances in Orthopedics 2017: 6516893.
35. Sciubba, D. M., et al. (2006). "Preoperative imaging of cervical spine hemangioblastomas using three-dimensional fusion digital subtraction angiography. Report of two cases." Journal of neurosurgery. Spine 5(1): 96-100.
36. Scott, W. W., et al. (2014). "Clinical and radiological outcomes following traumatic Grade 1 and 2 vertebral artery injuries: a 10-year retrospective analysis from a Level 1 trauma center." J Neurosurg 121(2): 450-456.
37. Squarza, S., et al. (2019). "C1-C2 fractures in asymptomatic elderly patients with minor head trauma: evaluation with a dedicated head CT protocol." Radiol Bras 52(1): 17-23.
38. Su, Z., et al. (2020). "Novel use of intraoperative cone-beam imaging with on-table angiography for excision of an occipitocervical tumour: A technical note and its feasibility." Interdisciplinary Neurosurgery 22: 100784.
39. Talacchi, A., et al. (2012). "Surgical management of ventral and ventrolateral foramen magnum meningiomas: report on a 64-case series and review of the literature." Neurosurg Rev 35(3): 359-367; discussion 367-358.

40. Van de Kelft, E., et al. (2012). "A prospective multicenter registry on the accuracy of pedicle screw placement in the thoracic, lumbar, and sacral levels with the use of the O-arm imaging system and StealthStation Navigation." Spine (Phila Pa 1976) 37(25): E1580-E1587.
41. Vergara, P., et al. (2012). "C1-C2 posterior fixation: are 4 screws better than 2?" Neurosurgery 71(1 Suppl Operative): 86-95.
42. Wajanavisit, W., et al. (2016). "Prevalence of High-Riding Vertebral Artery and Morphometry of C2 Pedicles Using a Novel Computed Tomography Reconstruction Technique." Asian spine journal 10(6): 1141-1148.
43. Wang, J., et al. (2013). "An anatomic consideration of C2 vertebrae artery groove variation for individual screw implantation in axis." European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society 22(7): 1547-1552.
44. Weller, S. J., et al. (1999). "Detection of vertebral artery injury after cervical spine trauma using magnetic resonance angiography." J Trauma 46(4): 660-666.
45. Willis, B. K., et al. (1994). "The incidence of vertebral artery injury after midcervical spine fracture or subluxation." Neurosurgery 34(3): 435-441; discussion 441-432.
46. Yeom, J. S., et al. (2013). "Risk of vertebral artery injury: comparison between C1-C2 transarticular and C2 pedicle screws." Spine J 13(7): 775-785.

Romanian Neurosurgery (2022) XXXVI (1): pp. 39-42
DOI: 10.33962/roneuro-2022-007
www.journals.lapub.co.uk/index.php/roneurosurgery

Spontaneous acute spinal subdural hematoma in patient on oral anticoagulant therapy. A rare case report

Svetlana Kocić[1], Vuk Aleksić[2], Milenko Stanić[2], Aleksandra Živković[3], Radiša Vojinović[4], Igor Popović[2]

[1] Department of radiology, Clinical hospital center Zemun, Belgrade, SERBIA
[2] Department of Neurosurgery, Clinical hospital center Zemun, Belgrade, SERBIA
[3] Department of Anaesthesiology, Clinical hospital center Zemun, Belgrade, SERBIA
[4] Department of Radiology, Clinical center of Kragujevac, Kragujevac, SERBIA

Keywords
spinal subdural hematoma, spontaneous spinal subdural hematoma, magnetic resonance imaging

Corresponding author:
Vuk Aleksić

Department of Neurosurgery, Clinical Hospital Center Zemun, Serbia

aleksicvuk@hotmail.com

Scan to access the online version

ABSTRACT

Introduction: Spontaneous spinal subdural hematoma as a result of coagulation deficit is a very rare and serious condition, with an overall incidence of less than 1%. Symptoms can vary according to the level of the bleed. Prompt recognition of the presence of spinal subdural hematoma is crucial for treatment. MRI is the investigation of choice for diagnosis as well as for planning the surgery. The differential diagnosis includes abscess, lipomatosis, significant discal hernia and tumours. There are no definite guidelines for the management of spinal subdural hematomas. In cases with serious neurological deficits, prompt surgical evacuation may lead to the resolution of symptoms and complete neurological recovery.

Case report: We present a case of a 75-years-old man with bilateral lower limb radiculopathy, paresthesias, and spinal claudications for four days, progressing to bilateral paraplegia with urine incontinency. The patient was diagnosed with cauda equina syndrome due to spontaneous spinal subdural hematoma in the region from Th12 to L2. He was immediately operated and the subdural hematoma was evacuated. Minimal recovery was achieved and the patient was referred for further rehabilitation with severe paraparesis and urine incontinence.

Conclusion: Early diagnosis of spinal subdural hematoma is essential for treatment. Any delay of correct diagnosis can lead to a devastating neurological deficit. Spontaneous spinal subdural hematoma is one of the rare conditions where an emergency MRI is indicated and crucial for diagnosis.

INTRODUCTION

Spinal subdural hematoma (SSDH) is exceedingly uncommon condition with incidence lower than 1%. Even rarer finding is spontaneous SSDH. Spinal subdural hematomas are usually associated with trauma,

lumbar puncture, anticoagulant therapy (cause of spontaneous SSDH) or spinal surgery. The prevalence of subdural hematoma in the thoracic and lumbar region as a result of coagulation deficit is a rare cause of spinal cord compression. Symptoms can vary according to the level of the hematoma including pain, lower limb weakness, radiculopathy, paresthesias, paraplegia, and urine incontinency. Prompt recognition of the presence of SSDH is essential for successful treatment (1, 2). MRI is the diagnostic modality of choice for diagnosis as well as for planning the surgical procedure. The differential diagnosis includes severe discus hernia, lipomatosis, abscess, and tumors (1).

We present the case of 75-years-old man with bilateral lower limb radiculopathy and paresthesias lasting for four days and progressing to cauda equina syndrome due to spontaneous subdural hematoma at thoracolumbar junction level.

Case report

A 75-years-old man presented with progressive bilateral lower limb radiculopathy, paresthesias and lower back pain progressing to paraplegia and urine incontinency in 4 days, after which he was admitted to our institution with complete cauda equina syndrome. There was no history of trauma. However, the patient had aortic valve prosthesis inserted 5 years prior to admission to our institution and was on anticoagulant therapy (Figure 1). His initial international normalized ratio (INR) was 6.020. An initial thoraco-lumbar computed tomography (CT) was performed in another institution two days after the onset of symptoms, and except for L1 vertebral body compression fracture without signs of dislocation, no other pathological finding was described (Figure 2). After neurological deterioration to cauda equina syndrome, patient was referred to our institution. Upon admission MRI of thoraco-lumbar junction as well as lumbosacral spine was performed and revealed a subdural lesion at the thoraco-lumbar junction level which was hyperintense on T1W imaging and hypointense to spinal cord on T2W image. The STIR sequence showed hyperintensity to spinal cord, so presumptive diagnosis of subdural hematoma was made (Figure 3). Patient was immediately operated and Th12, L1 and L2 laminectomies were performed. The underlining dura was blue, tense, and without pulsations. Upon opening the dura with linear incision semi-liquid, dark blood clot came out under high pressure (Figure 4). The subdural space was irrigated after which flow of liquor was obtained. The dura was sutured watertight and the operation was finished in usual fashion. Postoperatively the patient was painless with minimal improvement of lower extremities strength. No improvement of urine incontinence was obtained. Patient was referred for further rehabilitation 7 days after the operation. Unfortunately, no significant improvement was achieved 2 months after the operation.

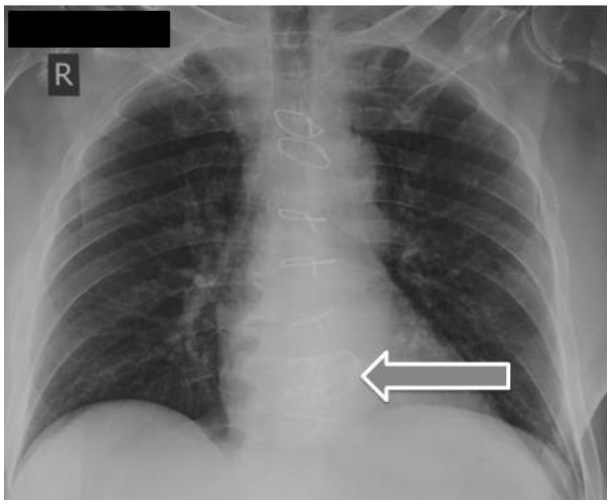


Figure 1. Chest X ray showing condition after heart valve replacement as indirect evidence that the patient is on anticoagulant therapy (arrow is pointing at artificial heart valve).

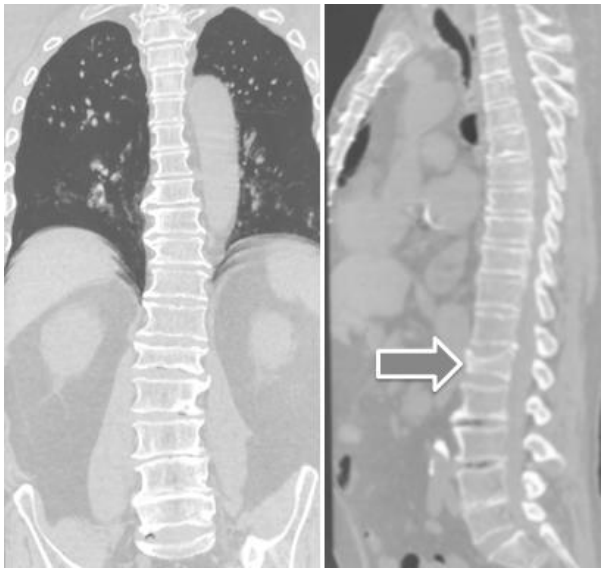


Figure 2. Spine CT scan. Arrow is pointing at L1 vertebral body compression fracture without signs of dislocation. No other pathological finding was found.

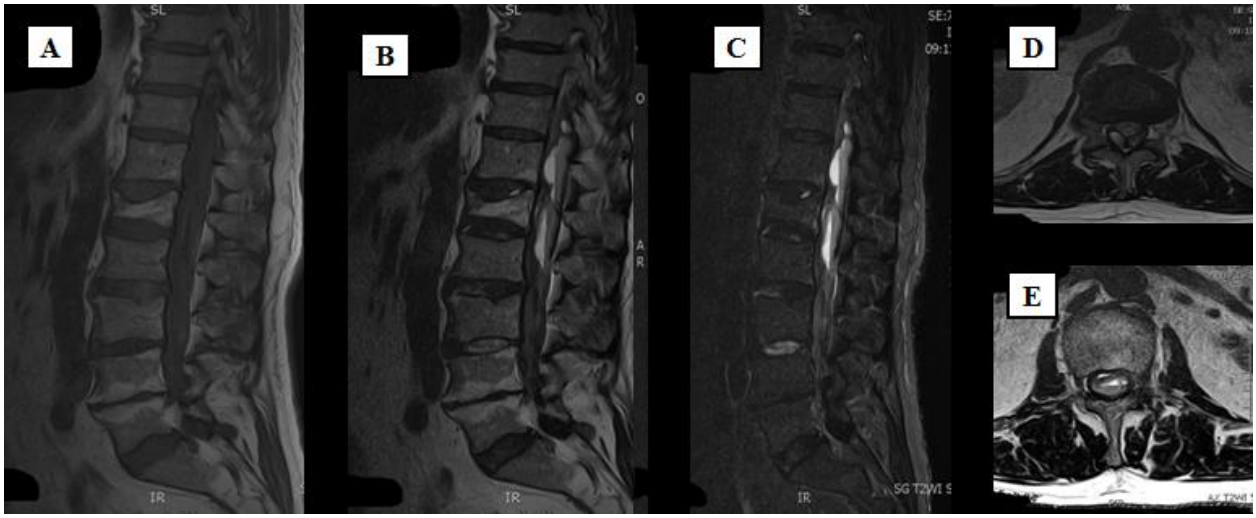


Figure 3. Spine MRI perofrmed 4 days after the onset of symptoms. (A) Sagittal T1W image showing Th12-L2 isointense subdural hematoma; (B) Sagittal T2W image showing Th12-L2 hyperintense subdural hematoma; (C) Sagittal STIR sequence showing hyperintense signal of SSDH; (D) Axial T2W showing the "Y" shaped sign of the SSDH at the level of L1; (E) Axial T2W showing SSDH at the level Th12 causing absolute spinal stenosis.

Figure 4. Intraoperative finding of SSDH

Discussion

SSDH is a rare condition, and most often these hematomas are associated with trauma, lumbar puncture or spine surgery (1). Spontaneous SSDH is exceedingly uncommon finding. De Beer et al. give classification of SSDH into: *(1)* posttraumatic, *(2)* iatrogenic (following spinal surgery or lumbar puncture), and *(3)* spontaneous (associated with underlying vascular malformations or coagulation deficits) (3). We presented a case of spontaneous SSDH due to coagulation deficit with INR 6.020, since the patient was on anticoagulant therapy because of heart valve replacement.

Clinical presentation of spinal subdural hematoma is not specific (1). Severe back pain with a radicular component is often the first complaint. The pain followed by the development of weakness and numbness progressing to a complete sensorimotor paraplegia over a few hours to days is typical clinical finding since most of SSDH are located in thoracic or lumbar region (3). According to study of Dampeer, spontaneous SSDH are most often located in the thoracic region and presenting with paraparesis or paraplegia, sensory level and pain, and more than 40% are due to coagulation deficit (3, 4). In our case patient had typical clinical picture for lesion located in thoraco-lumbar junction level (Th12-L2) with complete cauda equina syndrome.

MRI is the imaging modality of choice for diagnosis SSDH. The most important factor to distinguish SSDH from other spinal lesions is identification of blood products on MRI. Also, MRI gives better visualization of the longitudinal extent and size of the hematoma. Subdural hematoma can be divided into hyperacute, acute and chronic. The hyperacute bleed is iso/hypointense on T1W sequence and hyperintense on T2W sequence. The acute hematoma is hypo-/isointense on T1W images and hypointense on T2W images. In early subacute hematoma the T1W image is hyperintense and hypointense on T2W images. The late subacute hematoma is hyperintense on Tl- and T2- weighted images. The chronic hematoma is usually hypointense on T1W and T2W images (5, 6). Our patient had acute SSDH from Th12 to L2 spinal segment. CT is the workhorse for emergency cases and is usually done before. Unfortunately, SSDH can easily be missed in the acute setting. After having identified a subdural hematoma on MRI, it is good practice to revise the CT scan in an attempt to identify the SSDH (7). In our case initial CT scan was performed in another institution, and except for L1

vertebral body compression fracture, no other pathological finding was described. We revised CT scan after obtaining MRI finding that was suggestive of SSDH, however, no consensus has been reached among 9 neurosurgeons in our department. SSDH is one of the rare conditions where emergency MRI is indicated and crucial for diagnosis. It is important to distinguish between SSDH and other spinal subdural space occupying lesions such as empyema, hygroma, lipomatosis, tumors, and arachnoiditis. The "Y" shaped sign we noted, is similar to the "Inverted Mercedes Benz" sign described by Kasliwal et al. This sign is a result of the encasement of blood around arachnoid lined neural structure. This helps to differentiate between an epidural and subdural location of the hematoma (8).

There are no definite guidelines for management SSDH. The location and symptoms are the most important factors for treatment decision. Treatment involves conservative management in cases with preserved neurology or laminectomy and drainage in cases with serious neurological deficit. Cervical and thoracic SSDH mostly require surgical treatment, while SSDH below the conus medullaris can be treated conservatively. In general SSDH at the cervical or thoracic level are associated with poor outcome. Apart from the location, the duration of symptoms is one of important prognostic factors (9). In cases of spontaneous SSDH due to coagulation deficit, anticoagulant therapy should be stopped immediately (10). Our patient had complete cauda equina syndrome, so laminectomy and evacuation of SSDH was performed immediately after obtaining the diagnosis with MRI. However, due to long duration of symptoms, functional recovery was minimal. In our case we can modify famous neurological maxima "the time is brain" into "the time is spine".

Conclusion

Early diagnosis of SSDH is essential for treatment. So, in patients with cauda equina syndrome and severe back pain, and especially if there is information and evidence of anticoagulant therapy, spine MRI should be performed. If SSDH is confirmed in patient with any neurological deficit we suggest prompt surgical decompression and hematoma evacuation, although there are no definite guidelines for management SSDH and personalized medical approach is always reasonable. Patient's clinical symptoms, the location of the SSDH, the amount of spinal canal narrowing, and duration of symptoms are the most important factors which should be taken into account when choosing the most appropriate treatment.

References

1. Braun P, Kazmi K, Nogués-Meléndez P, Mas-Estellés F, Aparici-Robles F. MRI findings in spinal subdural and epidural hematomas. Eur J Radiol. 2007;64(1):119–25.
2. Schwartz FT, Sarrawi MA, Fox JL. Unusual hematomas outside the spinal cord. Report of two cases.J Neurosurg. 1973;39:249–245.
3. De Beer MH, Eysink Smeets MM, Koppen H. Spontaneous Spinal Subdural Hematoma. Neurologist. 2017;22(1):34-39.
4. Dampeer RA. Spontaneous spinal subdural hematoma: Case study. Am J Crit Care. 2010;19(2):191-193.
5. Mattle H, Sieb JP, Rohner M, Mumenthaler M. Nontraumatic spinal epidural and subdural hematomas. Neurology. 1987;37(8):1351-1356.
6. Konitsiotis S, Glantzouni A, Argyropoulou MI, Tsapoga T, Elisaf M, Efremidis SC. Acute spontaneous spinal subdural haematomas in a patient with essential thrombocythaemia. J Neurol. 2003;250(9):1109-1111.
7. Pierce JL, Donahue JH, Nacey NC, Quirk CR, Perry MT, Faulconer N, Falkowski GA, Maldonado MD, Shaeffer CA, Shen FH. Spinal Hematomas: What a Radiologist Needs to Know. Radiographics. 2018;38(5):1516-1535.
8. Kasliwal MK, Shannon LR, O'Toole JE, Byrne RW. Inverted Mercedes Benz sign in lumbar spinal subdural hematoma. The Journal of emergency medicine. 2014;47(6):692-693.
9. Thiex R, Thron A, Gilsbach JM, Rohde V. Functional outcome after surgical treatment of spontaneous and nonspontaneous spinal subdural hematomas. J Neurosurg Spine. 2005;3(1):12–16.
10. Toledo E, Shalit MN, Segal R. Spinal subdural hematoma associated with anticoagulant therapy in a patient with spinal meningioma. Neurosurgery. 1981;8(5):600-603.

Romanian Neurosurgery (2022) XXXVI (1): pp. 43-48
DOI: 10.33962/roneuro-2022-008
www.journals.lapub.co.uk/index.php/roneurosurgery

Bilateral chronic subdural hematoma: clinical presentation, outcome and review literature. A single centre experience from India

Yadav Kaushal, Jaiswal Gaurav, Lodha Krishna Govind, Gupta Tarun Kumar, Patel Pratik Bipinbhai, Kumawat Suresh

Department of Neurosurgery, Maharana Bhupal Government Hospital & Ravindra Nath Tagore (RNT) Medical College, Udaipur, Rajasthan University of Health Sciences, Jaipur (Rajasthan), INDIA

Keywords
bilateral CSDH, Glasgow coma scale, head injury, outcome

Corresponding author:
Gaurav Jaiswal

Professor & Head,
Department of Neurosurgery,
RNT Medical College, Udaipur,
India

drgauravjaiswal@gmail.com

Scan to access the online version

ABSTRACT

Study design: Prospective hospital-based study.

Background: Annual incidence of bilateral chronic subdural hematoma (CSDH) is increasing due to an increase in the ageing population, associated medical comorbidities such as haemodialysis, anticoagulant and/or antiplatelet therapy.

Objectives: 1. To determine the socio-demographic and clinical profile of bilateral CSDH patients. 2. To determine treatment outcome and its association with the socio-demographic profile.

Method: A prospective hospital-based study was carried out on 100 confirmed patients of bilateral CSDH. Information of patients such as socio-demographic profile, clinical presentation and laboratory investigation, along with treatment and outcome were recorded and analysed.

Results: Among 100 patients, male and female were 74% and 26%. The mean age of patients was 63.03±13.57 years. A history of head injury was reported by 49% of patients. The mean Glasgow coma scale (GCS) was 12.14±2.38. Common presenting symptoms were hemiparesis (69%), headache (58%), aphasia (18%) and complete loss of consciousness (16%). Clinical improvement was observed in 81% while 09% had no change, 06% shows clinical deterioration and 04% of patients die during treatment.

Conclusion: Bilateral CSDH is common in the elderly and prognosis is poor with increasing age however gender has no association with poor outcomes. More than 80% of patients recover with timely interventions.

Key message: Surgery leads to achieving good outcomes in cases of bilateral chronic SDH, but not all such patients will recover completely. Similarly, good functional outcomes can also be achieved in those presented early after symptom onset when managed promptly.

INTRODUCTION

A subdural hematoma (SDH) is collection of blood between the dura and the arachnoid membranes. SDH has been classified into three

verities i.e. acute, sub-acute and chronic types. Acute SDH is usually caused by head injuries and is symptomatic within 24 hours of injury.[13] The Chronic subdural haematoma (CSDH) are usually characterized by history of trivial head trauma.[7] CSDH is an encapsulated collection of old blood, which can be liquefied and located between the dura mater and arachnoid. CSDH patient become symptomatic more than 2 weeks after the initial injury.[14] It was first described by Virchow in 1857 as "pachymeningitis haemorrhagica interna".

Incidence of bilateral CSDH is about 1-5.3 cases per one lac population. The incidence is increasing due to increase in aging population, associated medical comorbidities such as hemodialysis, anticoagulant and/or antiplatelet therapy.[10,8]

The presentation of CSDH could vary from no symptoms to focal neurological signs such as hemiplegia, seizures, confusion, decreased memory, and signs of raised intracranial pressure such as headache, vomiting, and papilledema. Patients may present with difficulty in speech, swallowing, and walking. There may be weakness or numbness of arms, legs, and face.[16,19] CSDH is usually diagnosed by CT scan. Hematomas usually present as hypodense lesions, but sometimes isodense or mixed density lesions can also be observed. They are concavo-convex, but rarely may mimic acute epidural hematomas.

CSDH can be surgically treated, which has the potential for significant improvement or even resolution of symptoms postoperatively although multiple surgeries may required which leads to multiple hospitalization and deterioration of activities of daily living.[6,18] There is lack of uniformity about the treatment strategies, such as the role of burr hole, twist drill, craniotomy, etc., in CSDH amongst various surgeons. There is also disparity regarding use of drain, irrigation, and steroid. In addition, bilateral CSDHs are operated unilaterally or bilaterally depending on symptoms or hematomavolume.[17] Bilateral sites of CSDH are identified as a risk factor for recurrence by some researchers.[11]

Materials and methods

Study design: A prospective hospital-based study.

Study setting: Neurosurgery department of RNT Medical college, Udaipur, Rajasthan, India

Study period: From July 2013 to June 2021.

Study Population: All patients of bilateral CSDH admitted at neurosurgery department during study period.

Inclusion criteria: Patient of bilateral CSDH showed isodense to hypodense hematomas with respect to the adjacent brain at Computerized tomography (CT) scan.

Exclusion criteria: 1. Patient had concomitant occurrences of other types of traumatic brain injury; 2) CSDH had resulted from complications or history of prior neurosurgical procedures, such as craniotomy or cerebrospinal fluid shunting.

Sample size: 100

Study tools: 1) Individual case sheet. 2) CT scan report.

Method

A prospective hospital-based study was conducted at neurosurgery department on patients of bilateral CSDH admitted during July 2013 to June 2021. Based on inclusion and exclusion criteria, records of 100 bilateral CSDH patients were assessed and analysed. Information of socio-demographic profile, clinical presentation at the time of admission, associated medical comorbidities, routine investigation along with neuro-image findings, treatment and outcome were assessed. All informations were entered in individual case sheet. Ethical permission was taken from Institutional Ethic Committee before starting of study.

Statistical analysis

The collected data were compiled and tabulated using MS Excel 2010 and analyzed using statistical software SPSS trial version 20. Appropriated tables and figures were generated. The results were expressed in percentages. Chi-square test was applied to determine association. P value of < 0.05 was considered statistically significant.

Results

Records of 100 patients with radiologically confirm cases of bilateral CSDH were reviewed. Among 100 patients, 74 (74%) were male and 26 (26%) were female. Mean age of patient was 63.03±13.57 years. History of head injury was reported by 49% of patients. Underlying medical condition such as hypertension, diabetes mellitus, stroke and End Stage Renal Disease (ESRD) with hemodialysis was found among 58%, 38%, 19% and 04% of patients

respectively. Total 19% of patients were on antiplatelet therapy and 8% were on anticoagulant therapy at the time of admission. Glasgow coma scale (GCS) was assessed for each patient at admission and mean GCS was 12.14±2.38. (Table 1)

Table 1. Socio-demographic and clinical characteristic of study participants

Variables	Male (n=74)	Female (n=26)	Total
Mean age	63.81±12.33	60.80±16.69	63.03±13.57
Age (years)			
< 40	04 (5.4%)	02 (7.7%)	06
41 – 60	25 (33.8%)	10 (38.5%)	35
61 – 80	42 (56.8%)	10 (38.5%)	52
>80	03 (4.1%)	04 (15.4%)	07
History of head injury			
Yes	37 (50%)	12 (46.15%)	49
No	37 (50%)	14 (53.85%)	51
Underlying medical condition			
Hypertension	35 (47.29%)	23 (88.46%)	58
Diabetes mellitus	21 (28.38%)	17 (65.38%)	38
Stroke	09 (12.16%)	10 (38.46%)	19
Antiplatelet therapy	09 (12.16%)	10 (38.46%)	19
Anticoagulant therapy	05 (6.76%)	03 (11.53%)	08
ESRD with hemodialysis	02 (2.70%)	02 (7.69%)	04
Alcoholism	11 (14.86%)	02 (7.69%)	13
Mean GCS at admission	11.72±2.44	12.82±2.32	12.14±2.38

Table 2. Neuro-image findings of study participants at admission

Neuro-image findings	Numbers (%) / Mean±SD
Midline shift	12 (12%)
Mass effect	89 (89%)
Presence of layering	22 (22%)
Multiplicity of hematoma cavities	21 (21%)
Thickness of hematoma (mm)	32.09± 11.72
Density of hematoma (HU)	34.82 ± 10.38
Mean total hematoma volume in cm^3	178.8 ± 71.4

Neuro-image findings shows midline shift (12%), mass effect (89%), presence of layering (22%) and multiplicity of hematoma cavities (21%) among patients of bilateral CSDH. Mean thickness of hematoma was 32.09± 11.72 mm, mean density of hematoma was 34.82 ± 10.38 HU and mean total hematoma volume was 178.8 ± 71.4 cm3 among patients. (Table 2).

All patients of bilateral CSDH were treated by Biparietal burrhole with subgaleal drain placement. Mean duration of hospital stay was 18.12±8.34 days. At the time of discharge, 81% patient shows clinical improvement, 09% had no change in condition while 06% shows clinical deterioration and 04% patients die during treatment. During follow up, 4% of patients shows recurrence of SDH.

Table 3. Treatment and outcome of study participants

Variables	Numbers (%)
Treatment	
Surgical	100 (100%)
Conservative	00 (0%)
Outcome (at discharge)	
Improved	81 (81%)
No change	09 (9%)
Worsened	06 (06%)
Death	04 (4%)
Mean hospital stay (day)	18.12±8.34
Recurrence during follow up	04 (4%)

Table 4. Association of treatment outcome with Socio-demographic variables

Variables	Improved (n=81)	Others (no change, worsen and death) (n=19)	Total (n=100)	P value *
Age (years)				**0.013**
< 40	06 (100%)	00 (0%)	06	
41 – 60	31 (88.57%)	04 (11.43%)	35	
61 – 80	42 (80.76%)	10 (19.24%)	52	

>80	02 (28.57%)	05 (71.42%)	07	
Gender				**0.53**
Male	61 (82.43%)	13 (17.57%)	74	
Female	20 (76.92%)	06 (23.08%)	26	
Antiplatelet therapy				
Yes	12 (63.16%)	07 (36.84%)	19	**0.06**
No	69 (85.18%)	12 (14.52%)	81	
Anticoagulant therapy				
Yes	05 (62.5%)	03 (37.5%)	08	**0.35**
No	76 (82.6%)	16 (17.4%)	92	
Presence of layering				
Yes	16 (72.73%)	06 (27.27%)	22	**0.26**
No	65 (83.33%)	13 (16.67%)	78	
Multiplicity of hematoma cavities				
Yes	14 (66.67%)	07 (33.33%)	21	**0.059**
No	67 (84.81%)	12 (15.19%)	79	
Thickness of hematoma (mm)	31.6±10.56	34.16±11.88	32.09±11.72	**0.35**
Density of hematoma	34.61±10.20	35.66±10.76	34.82 ± 10.38	**0.69**
Mean total hematoma volume in cm^3	174.28±68.2	196.6±72.22	178.8 ± 71.4	**0.20**

*Chi square and student t test were used as test of significance.

For analysis purpose, outcome of patients was categorized into improved and others (no change, worsen and death). Statistically significant (p=0.013) association was observed with age as outcome is favourable with younger age. No association was found between gender(p=0.53), antiplatelet therapy(p=0.06), anticoagulant therapy (p=0.35), presence of layering (p=0.26) and treatment outcome of bilateral CSDH. Association of multiplicity of hematoma cavities (p=0.059), thickness of hematoma (p=0.35), density of hematoma (p=0.69) and mean total hematoma volume (p=0.20) was insignificant with outcome of CSDH.

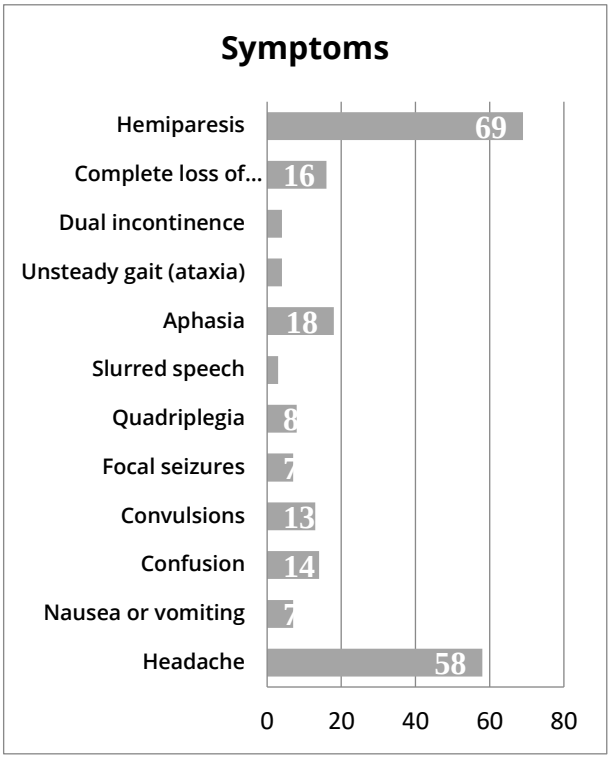


Figure 1. Symptoms of study participants at admission

Patients of bilateral CSDH were present with multiple symptoms at admission. Among them most common symptom was hemiparesis (69%) followed by headache (58%), aphasia (18%) and complete loss of consciousness (16%) excreta. (Figure 1).

Discussion

The increasing global incidence of bilateral CSDH because of an aging population has a great disease burden.[8,4] Clinically, patients often present with a history of gradually increasing altered level of consciousness and/or focal signs. A relatively simple neurosurgical treatment can improve functional outcome; however, high mortality is still observed in patients.[5]

In present study, a prospective study was planned on 100 radiologically confirm cases of bilateral CSDH. Among 100 patients, 74% were male and 26% were female. Age of patients varies from 35 years to 95 years with mean age of 63.03±13.57 years. Head injury was precede CSDH among 49% of patients. Patients were present with multiple symptoms at admission such as hemiparesis (69%), headache (58%), aphasia (18%), complete loss of consciousness (16%) excreta. Total 19% of patients were on antiplatelet therapy and 8% were on anticoagulant therapy at the time of admission. Yuji

Agawa et al[2] study 368 cases of bilateral CSDH. Average age at onset of disease was 74.2±12.8 years and 246 (66.8%) patients were male and 122 (33.2%) were female. Motor weakness (74.45%) was the most common clinical presentation followed by headache (19.56%). Forty-one patients (11.1%) had warfarin use, 67 (18.2%) patients had a history of malignancy, 46 (12.5%) had dementia and 16 (4.3%) had history of depression. Nina Christine et al[15] observed 291 patients of bilateral CSDH and find that out of 291 patients, 71.1% were male and 28.9% were female. Age is between 40 to 98 years, with a mean age of 73.0 years for males and 76.7 years for females. A history of head trauma was obtained in 53.3% patients. Approximately half of the study population (47.8%) received anticoagulant or antiplatelet therapy upon admission.

In this study, midline shift (12%), mass effect (89%), presence of layering (22%) and multiplicity of hematoma cavities (21%) was found in neuro-imaging. Mean thickness, density and mean total hematoma volume was 32.09± 11.72 mm, 34.82 ± 10.38 HU and 178.8 ± 71.4 cm^3 among patients. In neuro imaging of 25 patients of bilateral CSDH, Yu-Hua Huang et al[20] found midline shift (12%), mass effect (96%), presence of layering (20%) and multiplicity of hematoma cavities (20%) among patients of bilateral CSDH. Mean thickness of hematoma was 32.16 ±10.81 mm, mean density of hematoma was 35.80±11.30 HU.

In present study, management of all patients of bilateral CSDH were remain surgical. Mean duration of hospital stay was 18.12±8.34 days. At the time of discharge, clinical improvement was shown in 81% patient while 04% patients die during treatment. Treatment outcome was significantly associated with age but not with gender of patient. Nina Christine treat 264 (90.7%) patients surgically while 27 (9.3%) patients conservatively. Sakina Mehboob Rashid found improvement in 53.3%, worsening of condition among 6.6%, no change in 16.7% and deceased in 23.3% of patients. Similar to our study, Yuji Agawa observed poor clinical outcome among 03 (5.7%) patients and Yu-Hua Huang observed death among 01 (04%) of patients with bilateral CSDH. Olufemi Babatola et al[14] studied 73 patients with age range was 24 to 82 years. Among 73 patients of bilateral CSDH, in majority outcome was favourable (91.3%) in patients with a recurrence rate of 12.5% and mortality rate of around 6.3%. David Kitya et al[9] assess 205 patients of bilateral CSDH and found that 202 patients underwent surgical intervention with burr holes and drainage and 22.8% (46) were admitted to the ICU. Two patients had a recurrence, 5 suffered postoperative wound infection, and 18 died. GCS score on admission was a significant predictor of the discharge GCS score ($p = 0.004$), ICU admission ($p < 0.001$), and death ($p < 0.001$). Presenting symptoms differed by age. Hundred patients of bilateral CSDH was managed by Malaya Patel et al,[12] among them Burr hole drainage was carried out in 94 patients (94%). Primary craniotomy along with membrane excision was carried out in about 5 patients. Secondary craniotomy was performed in 1 patient. With all efforts, total five deaths were reported.

Conclusion

Bilateral CSDH are one of the most rewarding among neurosurgical procedures. Bilateral CSDH is common in elderly and prognosis is poor with increasing age however gender has no association with poor outcome. Head injury was preceding in bilateral CSDH among 49% of patients. Patients were present with multiple symptoms at admission. With timely diagnosis and management, improvement was observed in more than 80% of patients.

Conflicts of interest

The authors declare no conflict of interest.

Informed consent

Informed consent was obtained from all individual participants included in the study.

References

1. Adhiyaman V, Asghar M et al. Chronic subdural haematoma in the elderly. Postgrad Med J 2002;78:71–75.
2. Agawa Y, Mineharu Y et al. Bilateral Chronic Subdural Hematoma is Associated with Rapid Progression and Poor Clinical Outcome. Neurol Med Chir (Tokyo) 56, 198–203, 2016.
3. Babatola O, Salman A et al. Chronic subdural haematoma: clinical presentation, surgical treatment and outcome at the lagos university teaching hospital. African Journal of Neurological Sciences 2011 - Vol. 30, No 1.
4. Balser D, Farooq S, Mehmood T, Reyes M, Samadani U. Actual and projected incidence rates for chronic subdural hematomas in United States Veterans Administration and civilian populations. J Neurosurg 2015;123:1209–15.

5. Bankole OB, Yusuf AS, Kanu OO, Ukponwan E, Nnnadi MN, Arigbabu SO. Chronic subdural haematoma: clinical presentation, surgical treatment and outcome at the Lagos university teaching hospital. Afr J Neurol Sci 2011;30:10–7.
6. Dekker MCJ, Urasa SJ, Howlett WP. Neurological letter from Kilimanjaro. PractNeurrol. 2017;5:412–6.
7. Jobse IC, Feitsma MT. Presentation of chronic subdural hematoma in the elderly. TijdschrGerontolGeriatr. 2011;42:139–43.
8. Karibe H, Kameyama M, Kawase M, Hirano T, Kawaguchi T, Tominaga T. Epidemiology of chronic subdural hematoma. No ShinkeiGeka. 2011;39:1149–53.
9. Kitya D, Med M et al. Causes, clinical presentation, management, and outcomes of chronic subdural hematoma at Mbarara Regional Referral Hospital. Neurosurg Focus Volume 45:October 2018.
10. Krupa M. Chronic subdural hematoma: A review of the literature. Part 1. Ann Acad Med Stetin. 2009;55:47–52.
11. Kurokawa Y, Ishizaki E, Inaba K. Bilateral chronic subdural hematoma cases showing rapid and progressive aggravation. SurgNeurol 2005;64(5):444e9.
12. Malaya Patel et al., Clinical Presentation and Surgical Outcome in Chronic Subdural Haemorrhage Patients. Journal of Clinical and Diagnostic Research, 2021 Mar, Vol-15(3): PC08-PC12.
13. Mayer S, Rowland L. Head injury. In: Rowland L, editor. Merritt's neurology. Philadelphia: Lippincott Williams & Wilkins; 2000. p. 401.
14. Miller JD, Nader R. Acute subdural hematoma from bridging vein rupture: a potential mechanism for growth. J Neurosurg. 2014;120(6):1378–84.
15. Nina Christine Andersen-Ranberg et al. Bilateral chronic subdural hematoma: unilateral or bilateral drainage. J Neurosurg 126:1905–1911, 2017.
16. Rashid S M , Deliran S S et al. Chronic subdural hematomas: a case series from the medical ward of a north Tanzanian referral hospital Egyptian Journal of Neurosurgery (2019) 34:29.
17. Santarius T, Lawton R, Kirkpatrick PJ, Hutchinson PJ. The management of primary chronic subdural haematoma: A questionnaire survey of practice in the United Kingdom and the Republic of Ireland. Br J Neurosurg. 2008;22:529–34.
18. Takahashi S, Yamauchi T et al. Proposal of Treatment Strategies for Bilateral Chronic Subdural Hematoma Based on Laterality of Treated Hematoma. Asian Journal of Neurosurgery | Volume 13 | Issue 4 | October-December 2018.
19. Yadav Y R, Parihar V et al. Chronic subdural hematoma. Asian J Neurosurg. 2016 Oct-Dec; 11(4): 330–342.
20. Y.-H. Huang et al. Bilateral chronic subdural hematoma: What is the clinical significance? International Journal of Surgery 11 (2013) 544e548.

Romanian Neurosurgery (2022) XXXVI (1): pp. 49-51
DOI: 10.33962/roneuro-2022-009
www.journals.lapub.co.uk/index.php/roneurosurgery

Spontaneous height restoration of cervical traumatic non-pathological vertebral compression fracture. Short report

Khelifa Adel, Aichaoui Fayçal, Berchiche Lakhdar, Morsli Abdelhalim

Algiers University/Faculty of Medicine; and Department of Neurosurgery, Mohamed Lamine Debaghine University Hospital (BEO), Algiers - ALGERIA

ABSTRACT

Spontaneous regaining of radiological normal shape with vertebral compression fracture is very uncommon in the cervical spine; conducting confusion regarding the presence of a potentially surgical lesion. We report the case of 48 years old man without past medical history who presented post traumatic compression fracture that resolved spontaneously after transcranial traction. The patient was operated on with a good outcome. This report aims to confirm the presence of such phenomena already reported in the dorso-lumbar spine.

Keywords
cervical spine,
neuro-trauma,
compression fracture,
height restoration

✉

Corresponding author:
Khelifa Adel

Department of Neurosurgery,
Mohamed Lamine Debaghine
University Hospital (BEO);
Algiers - Algeria

drkhelifaadel@gmail.com

INTRODUCTION

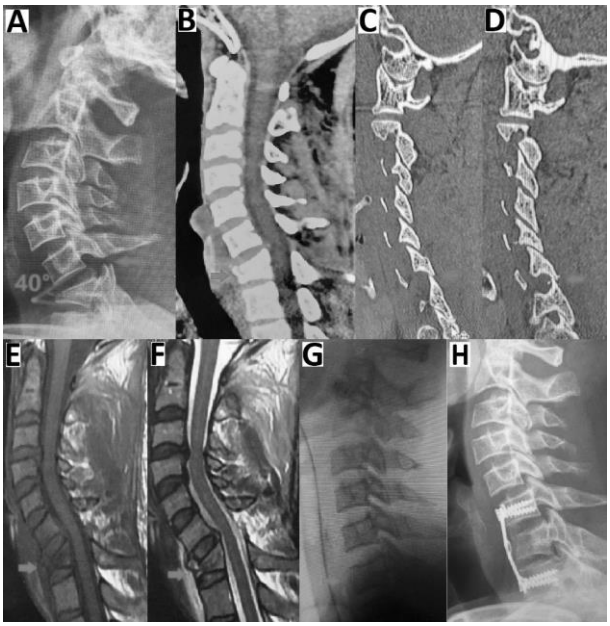
Vertebral compression fracture is a common lesion in the cervical spine trauma; characterized by a loss of the vertebral height. Spontaneous regaining of the initial radiological normal shape is very uncommon in the cervical spine; conducting to confusion regarding the presence of potentially surgical lesion.

CASE REPORT

The patient is a 48 years old man without past medical history victim of road traffic accident. The clinical exam at the admission found a conscious patient without neurologic deficiency presenting neck pain. The patient is scored E on the ASIA Impairment scale. Initial X-rays performed in standing position objectified C6-C7 bilateral facet joint dislocation and distraction; with anterior compression fracture of the C7 body causing an angulation of 40°; without body listhesis; resulting in a significant regional kyphosis (Figure A). With slight careful distraction-extension movement while putting the cervical collar the spine CT performed in supine position objectified partial facet joint reduction (Figures B, C and D). Cervical spine MRI objectified no significant intervertebral disk hernia and no spinal compression (Figure E and F). The patient was put under transcranial traction using Gardner-

Scan to access the online version

Wellstongs, gradually to 6 Kg (1/10 the boy weight). Impressionably per-operativefluoroscopy at the operating room objectified not only a total reduction offacet joint dislocation, but also C7 spontaneously regained totally a normal height. Through a Smith Robinson approach; a C6-C7 and C7-D1 discectomy were performed with partial corpectomy of C7, autografting and C6-D1 fixation. Postoperatively, the patient preserved his neurologic integrity.



A: initial lateral X-rays; showing the degrees of angulation caused by C7 compression lesion and the arrow is showing the amount of facet joint initial dislocation. **B**: Sagittal CT, the arrow is showing the slight improvement in vertebral height in supine position. **C&D:** Parasagittal CT passing through left and right facet joints, the arrow is showing the partial reduction of facet joint dislocation. **E&F**: Spinal MRI in sagittal T1 and T2 WIs, the arrow is showing the pre-vertebral hematoma. **G**: per-operative lateral fluoroscopy, the arrow is showing a total reduction of C7 body height. **H**: Post-operative lateral X-rays.

Discussion

McKiernan*et al.*described on dorso-lumbar levels; the phenomena of "Dynamic Mobility of Vertebral Compression Fractures" in osteoporotic patients where a postural height reduction was noted in many patients candidates for vertebroplasty[1]. In our case -although a slight normalization in vertebral height was noted in CT performed in supine position compared to X-rays performed in standing position- we explain height restoration by the transcranial traction force applied on the superior end plate of the inferior vertebra transmitted by an undamaged disk. In fact, based on the relatively healthy disk aspect on MRI, the absence of body listhesis and the per-operative harmonious aspect of the disk; we think that the relatively integrity of the disk contributed to this phenomena. In the presented case the compression fracture was associated with bilateral facet joint dislocation and thoughthe mechanism is hyperflexion, posterior distraction and anterior compression[2]. In such cases closed reduction with transcranial traction is highly indicated; and giving the absence of significant disk hernia it is without risks. Although the patient is neurologically intact and kyphosis has been corrected; the lesion is still highly instable, giving the facet joint dislocation[2], sign of sever ligamentous damage [3].With reduced bilateral facet joint dislocation, anterior fixation is our choice; it offers the possibility of rigid fixation with both arthrodesis and osteosynthesis, allows to perform a discectomy and especially in that case to perform a corpectomy. In fact although preoperative X-rays body aspect seemed to be normal we insisted on corpectomy and body reconstruction using graft, because we think that histological architecture of the body has been severely damaged and though will not be able to support the force transmitted by head and neck weight. We find this rational in dorso-lumbar compression fractures where vertebroplasty is performed with best outcome in patients with spontaneous height reduction after hyperextension positions [1, 4]

Conclusion

In this report we confirm the presence of spontaneous height restoration of compression fractures in cervical spine. This radiological normalization should not hold back body replacement in case of surgical management.

Funding

None.

References

1. McKiernan F, Jensen R, Faciszewski T. The dynamic mobility of vertebral compression fractures. J Bone Miner Res. 2003 Jan;18(1):24-9. doi: 10.1359/jbmr.2003.18.1.24. PMID: 12510802.
2. A Sayadipour. D G Anderson.S Mlyavykh. O Perlmutter. AR Vaccaro.Subaxial Cervical Spine Injuries. In: Benzel, Edward C. Spine Surgery 2-Vol Set E-Book: Techniques,

Complication Avoidance, and Management (Expert Consult-Online). Elsevier Health Sciences, 2012.

3. McLain RF, Aretakis A, Moseley TA, Ser P, Benson DR. Sub-axial cervical dissociation. Anatomic and biomechanical principles of stabilization.Spine (Phila Pa 1976). 1994 Mar 15;19(6):653-9. doi: 10.1097/00007632-199403001-00003. PMID: 8009329.

4. Yang JJ, Koo KH, Kim K, Park S. Efficacy of Postural Reduction of Vertebral Compression Fracture with Extension Lateral Radiograph Before Vertebroplasty.World Neurosurg. 2020 Nov;143:e430-e441. doi: 10.1016/j.wneu.2020.07.188. Epub 2020 Aug 1. PMID: 32750516.

Romanian Neurosurgery (2022) XXXVI (1): pp. 52-61
DOI: 10.33962/roneuro-2022-010
www.journals.lapub.co.uk/index.php/roneurosurgery

Planning neurosurgical interventions in patients with anticoagulant therapy

Quiñones-Ossa G.A.[1-3], Angulo-Mariño S.L.[1], Garcia-Ballestas E.[2,3], Moscote-Salazar L.R.[2-4], Agrawal Amit[5], Amrita Ghosh[6], Ranabir Pal[7]

[1] Faculty of Medicine, Universidad El Bosque.Bogotá, COLOMBIA
[2] Faculty of Medicine, Universidad de Cartagena. Cartagena, COLOMBIA
[3] Consejo Latinoamericano de Neurointensivismo- ClaNi, COLOMBIA
[4] Neurosurgeon-Critical Care. Center for Biomedical Research (CIB). Director of Research Line Cartagena Neurotrauma Research Group. Faculty of Medicine - University of Cartagena. Cartagena de Indias, Bolivar.
[5] Department of Neurosurgery, All India Institute of Medical Sciences, Saket Nagar, Bhopal 462020, Madhya Pradesh, INDIA
[6] Department of Biochemistry, Medical College, 88, College Street, Kolkata, INDIA
[7] Department of Community Medicine, MGM Medical College & LSK Hospital, Kishanganj, Bihar, INDIA

Keywords
traumatic brain injury, neurosurgery, neurosurgical procedure, neurological surgery, anticoagulant, anticoagulation agents

Corresponding author:
Amit Agrawal

MM Institute of Medical Sciences & Research,
Mullana (Ambala), India

dramitagrawal@gmail.com

ABSTRACT

Clinical practice guideline on anticoagulation is intended to manage patients undergoing neurosurgical procedures for the best possible short and long-term outcomes. In the clinical office practice, anticoagulation is offered to prevent thromboembolism with Warfarin, Heparin, Novel Oral Anticoagulants. The management approach starts with the mitigation plans from a reversal of pre-procedural anticoagulants for impending neurosurgical procedures by estimating procedural bleeding risk on the patients. The haemorrhage criteria and the timing of procedures are best assessed by the proceduralist during and after the intervention, standing on ground situations. Yet, intra- and post-procedure anticoagulant therapy should induct a multidisciplinary consultation paradigm for the best outcome in any emergent scenario. Further, each anticoagulation event should be monitored closely with competence in the optimum reversal process. Different neurosurgical procedures also should be weighed for their inherent hazards along with the probabilities of the bleeding and thromboembolism. The treating team should also concur to suggest a resumption of the pre-procedure anticoagulant therapy which may have been in place for altogether different morbidities. Regarding the anticoagulant agent, there are special conditions and recommendations to bear in mind in the daily medical practice for patient management. In the clinical practice guidelines for neurosurgical procedures, decisions about initiation and continuation of anticoagulants require experience and thorough internalization of the planned procedure, to avoid the risks of inherent risks of bleeding and thromboembolism.

Scan to access the online version

Introduction

Human brain has poor tolerance to constant bleeding and major hemorrhage of brain occurs from non-compressible locations. In ER, the physicians are confronted with the challenging scenario of patients, requiring surgical treatments, under anticoagulant, antiplatelet or thrombolytic medications; these therapies interfere with operative hemostasis (pre-, intra-, or post-operative hemostasis). The anticoagulant therapy is the cornerstone of the standard clinical care practice to avoid of thromboembolic episodes caused by diseases viz. atrial fibrillation (AF), pulmonary embolism (PE), heart disease or deep venous thrombosis (DVT) [1-7]. Intracranial hemorrhages and the higher bleeding risks, are higher incident to the Emergency Room (ER) irrespective of the trauma characteristics (minor or high impact trauma) in those patients on anticoagulant (or over anticoagulated) [7]. Thus, anticoagulation in neurosurgical patients represents two major implications, firstly, healthcare cost, and secondly, safety as well as prognosis [6]. Rapid identification and optimum interventions of anticoagulated neurosurgical patients are related to less healthcare and system cost with improved outcomes and good prognosis [8]. The purpose of this study was to review the current literature about anticoagulation therapy before, during and after the neurosurgical procedure, while considering the co-morbidities and patient current status.

Overview

Anticoagulant medications increased due to higher atherosclerosis prevalence among the elderly[2, 9 8, 9] who are also at higher risk of Traumatic Brain Injuries (TBI) and Intracranial Hemorrhage (ICH) from falls or violence [10] causing huge burden of mortality, morbidity, and disability. Devastating consequences and fatal sequel have been reported after these traumas, especially when they are under anticoagulant therapy [8, 11]. So, before neurosurgical interventions, it is important to reverse or counteract the effect depending on the type of anticoagulant[1], if the patient is under anticoagulation therapy. Those who are under oral anticoagulants therapy, have worse outcomes as reversal agents for these drugs are largely not available [2].

Type of anticoagulant

Warfarin

Warfarin, inhibits Vitamin K dependent coagulation factors viz. II, VII, IX, and X [1, 3]; takes 3 days to achieve complete inhibition of the factors in the order of VII, IX, X, and II; effect reversion also takes 3days after stoppage of doses, and is not an option when the patient needs an urgent neurosurgical procedure [1] and is linked to hematoma expansion in the ICH patient with consequent poor prognosis[12]. Warfarin advice require strict monitoring of Internationally standardized Ratio (INR) and, has known drug interactions [8, 9]. INR value reflects anticoagulant effect: <1.0= non-anticoagulant effect; 2-3 indicates active effect, >3= hemorrhage risk [1, 6, 7, 13-15], INR increase is exponential depending on the dose and individual patient response [16]. Clinico-social factors also affect these levels viz. female gender, advanced age, black race, heart diseases, substance use, psychiatric disorders, and frequent hospitalizations [16, 17]. Further, in presence of or with risk of intracranial bleeding, Prothrombin Time (PT) is strictly kept below 1[1], INR >1.2 provides poor outcomes as in ICH or a TBI [1, 6]; in neurosurgical patients a target minor of 1 to 1.5 (or 1.3) of the INR is recommended [6].

Heparin

This parenteral anticoagulant act as prophylaxis of DVT, by binding with plasma proteins and affect molecular configuration [1]. Heparin antithrombin complex rapidly interacts with circulating thrombin to inhibit the coagulation enzymes and reduce platelet aggregation by the inhibition of the Von Willebrand Factor [1]. The efficacy of heparin is measured by the partial thromboplastin time (PTT) to be 1.5-2.0 times of the patients baseline value [1]; reversal effect after stoppage of dosage administration takes up to 1 hour which is also huge time gap to start an emergency neurosurgical intervention [1]; protamine is used reversal at the dose of 1 mg per 100 units of heparin when an urgent surgery is contemplated.

Novel Oral Anticoagulants (NOACs) versus Direct Oral Anticoagulants (DOACs)

These are of two types: A) direct factor Xa inhibitors (Endoxaban, Apixaban, Rivaroxaban) and B) direct thrombin inhibitors (Dabigatran) [8]; used as first-line therapy in atrial fibrillation. There is need to review

the clinical history in absence of clear information on anticoagulant intake[16] as these increase the bleeding risk or the progression of the ICH [8]. Research groups reported reduction of venous thromboembolic episodes of NOACs used as chemoprophylactic anticoagulation therapy in TBI patients within 24 hours without Computed Tomography (CT)-Scan changes [4]. NOACs are safer and simpler alternative compared with Warfarin with shorter half/life with predictable therapeutic rapid onset effects and do not require continuing monitoring[8, 9], change the dietary pattern, less drug interactions; issues with lack of specific antidotes [3] and reversal antidotes are still evolving [8]. Few studies reported worse prognosis than Warfarin with higher progression rate of ICH and mortality after TBI[8], while others, noted lower risks against vitamin K antagonist [6,12]. NOAC used with a low aspirin dosage was reported safer and more effective than Warfarin by other researchers in preventing strokes and intracranial hemorrhage [9, 18]. During use of Dabigatran (direct thrombin inhibitor), the normal ranges of Thrombin Time (TT) or the dilute TT (dTT) is rider for associated anticoagulation effect [6]; "safe-zone" for TT before surgical interventions is <30 ng/ml; in case of a higher values (> 30 ng/ml) or with heavy bleeding, the antidote must be administered [6].

Activated Partial Thromboplastin Time (APTT) help approximate time since the last dose[19] as prolongation result due to the anticoagulant effect[3] and suggest risk of bleeding if the value is twice the normal ratio[19]; not done in lupus syndrome or clotting factors deficiency disorders due to the intrinsic prolonged effect of APTT that may mask the true effects[3]. Reversal with Idarucizumab depends on the available tests; in absent of testing facilities and with active bleeding doses have to be repeated [6]. Studies reported rapid hematoma expansion and bad prognosis in ICH patients with NOACs intake even with minor intracranial bleeding [8, 20, 21]. For the X-factor inhibitor, anticoagulants activity evaluation involves Anti-activated factor X (Anti-Xa) calibrated to LMWH or the corresponding "xaban" available on limited scale[6]. NOACs usage has increased in a colossal way in the last years replacing conventional anticoagulants especially in patients with trauma in ER[8]. Patients with ICH, regardless of the origin, under Dabigatran require urgent reversal, should be treated with idarucizumab[3]; on factor Xa inhibitors intake to be treated with PCC [3]. NOACs use may need observation [6]in circumstances viz. normal CCT-Scan and GCS, an open head injury with injured scalp reflecting normal coagulation status and with unilateral chronic subdural hematoma without neurological deterioration or red flags or minimal neurological symptoms[6].

Management approach in anticoagulated patients

ER personnel should assess clinical status, triage and neurosurgical intervention with review of clinical records for co-morbidities, medications and anticoagulant use in TBI cases or with ICH suspects followed by Cranial Computed Tomography (CCT) as the anamnesis or neurological status in anticoagulated patients is usually attributed to vascular origin[3]. The latter has high sensitivity for extent of intracranial damage with acute onset of the hemorrhage [6]; contrast enhanced CT helps to identify the risk of bleeding expansion within the hematoma (spot signs)[3]. The prognosis of ICH varies on age, clinical status, volume of hematoma and degree of anticoagulant activity[7]. Regardless of the type of bleeding and anticoagulant agent, every life-threatening hemorrhage should be managed initially with basic ABC resuscitation protocol[3]; no uniform recommendation in the primary ICH exists for NOACs,[3]; ICH is 11times worst compared to extracranial with VKA therapy [3]. Even with normal CCT on anticoagulant medication, patients should be observed in-hospital for 24 hours to exclude delayed intracranial hemorrhage; repeat CCT scan needed in neurological deterioration[6]; with intubation, sedation, neurological concomitant disease, follow-up to be made by CCT-Scan [6].

There is need to optimize neurosurgical procedure, reversal agents, risk of thromboembolism versus anticoagulation and re-induction of anticoagulation after procedure [3]; correct management of blood pressure is related to a less neurological damage, hematoma expansion and unfavorable outcomes including improvement in functional recovery[3]. Total correction of VKA is achieved with a PCC infusion (20 UI/kg) or a bolus of 25 IU/kg and a single dose of 5 mg of Vitamin K in order to get a value from 1.2-1.5 with an approximate 6hours effect [16]; neurosurgical procedures require INR <1.3 [16]. [Table 1] The surgical procedures can be unscheduled invasive surgery or emergency surgery, semi-urgent, relative delayable surgery, urgent diagnosis procedures (e.g. lumbar

puncture) or scheduled invasive procedures than can present high or moderate risk of hemorrhage, each needs special recommendations (Table 2.)[16]

Table 1. Major hemorrhage criteria[16]

- Excessive/fatal bleeding
- Symptomatic hemorrhage with a critical organ compromise (intracranial, spinal, intraocular, pericardial, intramuscular hemorrhage with a compartment syndrome, retroperitoneal hemorrhage).
- A drop in hemoglobin levels that requires transfusion of more than two units of packed red blood cells or whole blood compounds.
- Damaged organ or special location (intracranial, spinal, digestive, thorax, pericardium, abdominal cavity).
- Massive hemorrhage that cannot be managed with conventional procedures.
- Requirement of invasive treatment (surgery, interventional radiology or endoscopy).

Table 2. Recommendations according the timing of the procedure (8)

Type of procedure	Recommendation
Emergency surgery	Administration of PCC is effective in the first 30 minutes after administration and could last for 5 hours. In this case, is important to measure the INR after 5 hours after the initial dose. In neurosurgical procedures - recommended to achieve an INR <1.3.
Semi-urgent surgery	If it is performed within 24 hours the recommended values are still under 1.3 of the INR, but because of the allowed delayed time a single dose of Vitamin K (5 to 10 mg) might be effective to achieve the hemostatic safety threshold.
Invasive unscheduled procedure with a high risk of hemorrhage (e.g. lumbar puncture)	Thrombotic and hemorrhage risk should be considered, in these types of procedures, if the bleeding can be controlled with local pressure there is no need to revert the anticoagulation effect.
Scheduled invasive procedures with moderate/ high bleeding risk	In these cases, is recommended to stop VKA treatment 5 days before the procedure and monitorization the INR levels.

MINIMAL AND HIGH-RISK PROCEDURES

Neurosurgical patients under anticoagulant therapy have inherently higher risk of hemorrhages (ICH) [10] though the thromboembolic event also carry of 3-43% risk [22, 23] which should be kept in mind as 50-50 chance in order to assess the risk versus benefit [22, 23]. Neurosurgical procedures can be invasive and non-invasive, the emergency procedures with other types and sub-types [22].

A. Lumbar puncture

Lumbar Puncture (LP) is useful for therapeutic and diagnostic use in daily medical practice to help analyze Cerebrospinal Fluid (CSF) especially for suspected neuroinfection[24, 25], biomarkers for TBI prognosis [11, 26-28], to diagnose elevated Intracranial Pressure (ICP) (syn. Intracranial Hypertension) [24, 29]. After LP multiple complications can occur viz. epidurals, subarachnoid or subdural hematomas (trivial or massive) as Traumatic Lumbar Puncture (TLP) due to a direct puncture in the radicular vessels and the sliding of the arachnoid on the dura[30-32]. The diameter of needle and catheter add higher risk of bleeding, when the patients are under anticoagulant therapy [30, 33]. The spinal hemorrhages can lead to irreversible complications like paraplegia or paraparesis of lower limb[34], medullar or compressive radicular syndrome (due to exacerbated fibrinolytic property of the CSF related to a higher red blood cells count after the TLP) [33]. It is recommended to avoid anticoagulation therapy with Enoxaparin 24 hours before LP and 48 hours if under NOACs therapy [34].

B. Decompressive craniectomy

Decompressive Craniectomy (DC) is commonest treatments in treatment of high ICP since last century to maintain intracranial equilibrium[35] in high ICP from cerebral tumors, neuroinfections, TBI, ICH (whether spontaneous or traumatic)[36-39] to avoid neurological complications, secondary insults, brain herniation and unfavorable outcomes including death [40, 41]. There is a high risk of DVT after DC that need antithrombotic measurements and imaging studies [22]; patients under anticoagulation should have coagulation profile; if abnormal, suspend the therapy and/or restore the coagulation time within 48 hours[1, 22] as damaged tissues and platelets produce excessive thromboplastin and vasoconstrictors that might produce acidosis status and ischemia [22]. The preventive management are recommended to avoid hypercoagulation and thrombosis episodes, initiate mechanical

compressive; reinitiating of anticoagulant therapy done after 15 days if there is pulmonary embolism in the postoperative period [22, 42]; anticoagulation therapy are also used by others within first 24 hours; yet there is no clear consensus of the timing of the anticoagulation therapy after the surgical procedure[42, 43]. In impending risk of Cerebral Venous Thrombosis (CVT), neurological monitoring and imaging studies considered; risk of intracranial bleeding and hematoma expansion should be thought in patients under anticoagulant therapy[8, 12, 43-45].

C. Craniotomy in Tumor Resection

Cerebral tumors have frequent post-operative complications due to high risk of DVT (27-45%) or prothrombotic status related to the tumor itself [22]. Tumors predispose to a venous stasis and atherosclerosis by the intimal dysfunction, disturbance of vessels and major procoagulant factors[22, 44] add higher risk of thromboembolic events. The enoxaparin or NOACs treatment lead to major ICH; it is better to use mechanical measurements to prevent thrombosis to decrease post-surgical bleeding[22, 46]. Chronic anticoagulant treatment has not been associated with a post-surgical hemorrhage recurrence within first 72 hours compared to non-anticoagulated patients[46, 47]. LMWH usage in the first 48 hours after the procedure as a prophylactic therapy is recommended to avoid the thrombotic complications[46, 47].

D. Ventriculoperitoneal Shunt and Ventriculostomies

Ventriculoperitoneal shunt (VPS) is used treat high ICP especially in hydrocephalies[48]. External Ventricular Drainage system drains CSF and reduces ICP; the hemorrhage risk is 7% but a significant hemorrhage reported in minority (0.8%); Heparin is recommended in VPS [9, 49, 50]

Dose adjustment

No clinical practice guideline can replace clinical acumen and judgment on ground situations though many standard office procedures are based on 2012 ACCP guidelines for antithrombotic therapy and discussions are needed regarding different qualitative and quantitative approaches [51].

Warfarin

In Warfarin over-anticoagulated patients with high INR, 5-10mg Vitamin K (oral or intravenous) is administered; takes up to 24 hours to full reversal [1, 6]. Thus in ER, Vitamin K as antidote or reversal agent is not recommended in hemorrhagic TBI or urgent surgery; useful as an adjunct therapy[6]. Prothrombin Complex Concentrate (PCC) dosage depends on initial INR value, has the advantage of immediate reversal effect, Vitamin K can be used to maintain effect [6]. Plasma transfusion therapy require high volumes and can lead to circulatory overload, pulmonary edema, congestive heart failure and immune-suppression; also takes longer time compared to PCC to reverse and normalize INR[6]. Warfarin use causes higher postoperative bleeding than NOACs[3].

Pre-operative and peri-operative thromboembolism vs. bleeding risk prediction

At first, bleeding versus embolism risk stratification needed using CHA2DS2-VASc and HAS-BLED scores are user-friendly for rapid assessment of thrombotic and hemorrhagic risk respectively [3, 52, 53]. Additionally, we have to consider risk factors of ICH viz. older age, hypo- or hypertension, micro-bleeds on echo-magnetic resonance imaging gradient, and ICH in lobar location[3]. To reach at correct treatment strategy, every patient under anticoagulation treatment requires an evaluation and categorization on urgency of the invasive procedure with a special consideration of thrombotic and hemorrhagic risk [16]; otherwise carry risk of thrombosis or pulmonary embolism (PE) in 25-60% [52]. Other researchers prefer initiation of thromboembolism prophylaxis after first 24 hours only in radiographically and neurological stable TBI [6]; restart of antithrombotic prophylaxis within first 72 hours has lower incidence of DVT and PE [6].

Post-operative management – when to restart anticoagulation therapy?

Intracranial bleeding represents a special condition for resumption of anticoagulation as in hemispheric location of hemorrhage the VKA therapy should be permanently discontinued[16] Resumption of anticoagulation regimen is a clinical dilemma in ICH or any neurosurgical procedure[3, 9]; after hemostasis achieved and ICH has stopped, the resumption of

anticoagulants can add risk of bleeding or a future re-bleeding in TBI [6]. ICH management guidelines indicates that therapeutic anticoagulation should be reinitiated after 2 weeks post-trauma with stable injury and high cerebral ischemia risk secondary to mechanical valve prosthesis or atrial fibrillation with a high a CHA2DS2VASc score[6, 54, 55]; with low risks of thromboembolism, anticoagulation therapy are reinitiated after 8 weeks [6]. Literature reports that VKA's therapy might be initiated within 7 days and with heparin after 3 days in ICH without re-bleeding complications[56] but others recommend anticoagulation therapy after the first 2 weeks to avoid hemorrhagic complications[57]. Restarting the anticoagulation with Warfarin within 14 days is associated with an increased hemorrhagic complications, thus anticoagulation after 2weeks is recommended[16]; some studies reported as ideal time to reinitiate Warfarin after a week of the procedure[9, 58].

Strategies to reduce the thromboembolism risk and non-pharmacological treatment

Thrombosis and embolism episodes are global public health problem with increasing mortality with co-morbidities the risk and incidence is doubled[59]. Risks of impending thromboembolism are well assessed by the treating physician though the multidisciplinary and integral paradigm of prevention and medical approach. Adoption of healthy lifestyle with good dietary habit, reduction of alcohol and tobacco consumption, oral anticonceptives, hormone replacement therapy is recommended [60] with non-pharmacological therapy for the patients management viz. pneumatic intermittent compression, compression stockings and drugs[52]. The prevention may start even before the surgical intervention regardless the concomitant pathologies that predispose to DVT or pulmonary embolism [22, 59]. Graduated Compression Stocking compress the lower extremities, graduated from the bottom (more intense) to the top (less compressive) to increase blood-flow; advised to use them from early deambulation till 2 years post- procedure[61, 62], the Intermittent Mechanical Compression increases blood flow in the veins of the lower limbs and is superior to graduated compression stocking to significantly reduce DVT by 7.3% and pulmonary embolism 1.2-2.8%; recommended to use it jointly with pharmacological prophylaxis. [61, 62]

Current issues with the anticoagulant therapy in anticoagulated neurosurgical patients

Anticoagulants inhibits the coagulation factors metabolism to avoid thrombotic complications including that of post-neurosurgical interventions [63, 64]. The bone of contention here is decision of precise moments of interruption or resumption of anticoagulantion after neurosurgery [62, 65, 66] where the risk-benefit analysis of the associated factors, choice of drugs, type of procedure and neurosurgeons criteria must be taken into account[63, 67]. Regarding INR measurement to analyze the effect, Thromboelastometry (viscoelastic analysis method that qualitatively assesses coagulation and fibrinolysis through rates of clot formation, resistance and degradation, and interaction of coagulation factors) is useful, yet, not established for neurosurgical use [23, 68-70].

Further, human inherent anticoagulation genetic factors with two allelic variants (2C9*2 and 2C9*3) of the CYP2C9 enzyme [71, 72] usually require a minor anticoagulant dosage. On the other hand, the variant of Vitamin K Epoxide Reductase Isoenzyme 1 (VKERI-1) generates resistance to the VKA agents requiring higher dosages o achieve the therapeutic effect [73, 74]. Non-genetic factors are associated to personal habits, adherence to therapy, preference for alternative treatments, dosage mistakes and co-morbidities the confounding variables [17].

Among the complications previously mentioned, one of the most important is the intracranial bleeding [75], followed by thrombosis that increase 2.5 times daily in anticoagulated patients whose therapy is interrupted for neurosurgical intervention; in addition the risk of developing hypercoagulability increases due to the limitation in postoperative ambulation [76-78].

Several studies recommend to avoid resumption of anticoagulation before 24 hours due to the risk of reactivation of bleeding in the intervened area, yet early restart between 4-7 days is the ideal time, with lesser complications compared to 14-day late restart, which increases the risk of cardioembolic infarction and ischemic stroke [58, 76]. Heparin use may be recommended after intracranial surgery within 24 hours with the intention to reduce DVT and PE [79, 80]. The considerations to restart anticoagulant therapy, it is important to recap type of medication, possibility of control or not with INR, cognitive deterioration or diseases associated with memory disorders, labile

INR, high risk of stroke[79, 80], and risk of bleeding or thromboembolic disease associated with non-surgical scores such as CHA2DS2-VASc and HAS-BLED [53, 81]. In addition, certain precautions should be taken in patients suffering from CKD (Chronic Kidney Disease), patients over the age of 65, on treatment with macrolides and the use of antifungals such as ketoconazole and itraconazole [80-82].

Conclusions

There is a widespread concern on the outcome of neurosurgical procedures while the patient is on anticoagulant therapy. A multipronged approach is needed involving specialities and sub-specialities ranging from Haematology, Biochemistry, Pathology, Pharmacy services, Internal Medicine, Emergency Medicine, Family Medicine, Anaesthesiology, Nephrology, and Pre-hospital consult services roped in for specific patients who need more complex continuum of care with dosing and monitoring of anticoagulant medications. There is urgent need to develop consensus guidelines for the health care professionals regarding management of anticoagulation which may be playing as double aged sword in both risk factor and outcome. Basic rules should be, while a more specific reversal agent of anticoagulant is available and approved to be used in the medical practice, antiplatelet agents can be continued throughout the perioperative period.

References

1. Powner DJ, Hartwell EA, Hoots WK. Counteracting the effects of anticoagulants and antiplatelet agents during neurosurgical emergencies. Neurosurgery 2005;57:823-831; discussion 823-831.
2. Batey M, Hecht J, Callahan C, Wahl W. Direct oral anticoagulants do not worsen traumatic brain injury after low-level falls in the elderly. Surgery 2018;164:814-819.
3. Raval AN, Cigarroa JE, Chung MK, et al. Management of Patients on Non-Vitamin K Antagonist Oral Anticoagulants in the Acute Care and Periprocedural Setting: A Scientific Statement From the American Heart Association. Circulation 2017;135:e604-e633.
4. Raychaudhuri R, Litofsky NS. Which traumatic brain injury patients should be treated with anticoagulants and when? Expert review of neurotherapeutics 2014;14:237-239.
5. Roark CD, Haines S. Pharmacological anticoagulation and mechanical compression versus mechanical compression alone for venous thromboembolism prophylaxis for post-operative neurosurgical patients. Cochrane Database of Systematic Reviews 2009.
6. Wiegele M, Schöchl H, Haushofer A, et al. Diagnostic and therapeutic approach in adult patients with traumatic brain injury receiving oral anticoagulant therapy: an Austrian interdisciplinary consensus statement. Critical care (London, England) 2019;23:62.
7. Auer C, Wurm G. Outcome after acute head trauma needing neurosurgical intervention in patients with oral anticoagulants or anti-thrombotic agents. J Trauma Treat 2012;1:2167-1222.1000132.
8. Zeeshan M, Jehan F, O'Keeffe T, et al. The novel oral anticoagulants (NOACs) have worse outcomes compared with warfarin in patients with intracranial hemorrhage after TBI. The journal of trauma and acute care surgery 2018;85:915-920.
9. Passer JZ, Loftus CM. Postoperative Anticoagulation After Neurologic Surgery. Neurosurgery Clinics 2018;29:575-583.
10. Tator CH. Concussions and their consequences: current diagnosis, management and prevention. CMAJ 2013;185:975-979.
11. Quiñones-Ossa G, Padilla-Zambrano H, Pal R, et al. Biomarkers in acute brain trauma: A narrative review. Journal of Acute Disease 2019;8:1.
12. Adachi T, Hoshino H, Takagi M, Fujioka S. Volume and Characteristics of Intracerebral Hemorrhage with Direct Oral Anticoagulants in Comparison with Warfarin. Cerebrovascular diseases extra 2017;7:62-71.
13. Levine AR, Laliberte B, Lin H, et al. Weight based heparin dosing for thromboembolic disease is associated with earlier anticoagulation in surgical patients. International journal of surgery (London, England) 2014;12:1416-1419.
14. Lillemäe K, Järviö JA, Silvasti-Lundell MK, Antinheimo JJ, Hernesniemi JA, Niemi TT. Incidence of Postoperative Hematomas Requiring Surgical Treatment in Neurosurgery: A Retrospective Observational Study. World neurosurgery 2017;108:491-497.
15. Vakharia VN, Tai D, Marcus H, Vakharia NN, Nandi D. New oral anti-coagulants: Implications for neurosurgery. British journal of neurosurgery 2015;29:182-188.
16. Lapostolle F, Siguret V, Martin A-C, et al. Vitamin K antagonists and emergencies. European Journal of Emergency Medicine 2018;25:378-386.
17. Anguita Sánchez M, Bertomeu Martínez V, Cequier Fillat Á. Quality of Vitamin K Antagonist Anticoagulation in Spain: Prevalence of Poor Control and Associated Factors. Revista espanola de cardiologia (English ed) 2015;68:761-768.
18. Bennaghmouch N, de Veer A, Bode K, et al. Efficacy and Safety of the Use of Non-Vitamin K Antagonist Oral Anticoagulants in Patients With Nonvalvular Atrial Fibrillation and Concomitant Aspirin Therapy: A Meta-Analysis of Randomized Trials. Circulation 2018;137:1117-1129.
19. Kimpton G, Dabbous B, Leach P. New oral anticoagulant and antiplatelet agents for neurosurgeons. British journal of neurosurgery 2015;29:614-621.

20. Beynon C, Potzy A, Sakowitz OW, Unterberg AW. Rivaroxaban and intracranial haemorrhage after mild traumatic brain injury: A dangerous combination? Clinical neurology and neurosurgery 2015;136:73-78.
21. Pakraftar S, Atencio D, English J, Corcos A, Altschuler EM, Stahlfeld K. Dabigatran etixilate and traumatic brain injury: Evolving anticoagulants require evolving care plans. World journal of clinical cases 2014;2:362-366.
22. Schoihet S. Prevención de enfermedad tromboembólica en pacientes neuroquirúrgicos y neurológicos. Revista chilena de cirugía 2007;59:311-316.
23. Maegele M, Schöchl H, Menovsky T, et al. Coagulopathy and haemorrhagic progression in traumatic brain injury: advances in mechanisms, diagnosis, and management. The Lancet Neurology 2017;16:630-647.
24. Jovel CAE, Ramos ML, Moreno CM, et al. Utilidad y rendimiento diagnóstico de la punción lumbar en el servicio de urgencias. Acta Neurol Colomb 2015;31:39-48.
25. Tan IL, Smith BR, von Geldern G, Mateen FJ, McArthur JC. HIV-associated opportunistic infections of the CNS. The Lancet Neurology 2012;11:605-617.
26. Mondello S, Robicsek SA, Gabrielli A, et al. αII-spectrin breakdown products (SBDPs): diagnosis and outcome in severe traumatic brain injury patients. Journal of neurotrauma 2010;27:1203-1213.
27. Weiss ES, Wang KK, Allen JG, et al. Alpha II-spectrin breakdown products serve as novel markers of brain injury severity in a canine model of hypothermic circulatory arrest. The Annals of thoracic surgery 2009;88:543-550.
28. Zetterberg H, Smith DH, Blennow K. Biomarkers of mild traumatic brain injury in cerebrospinal fluid and blood. Nature reviews Neurology 2013;9:201-210.
29. Gu J, Huang H, Huang Y, Sun H, Xu H. Hypertonic saline or mannitol for treating elevated intracranial pressure in traumatic brain injury: a meta-analysis of randomized controlled trials. Neurosurgical review 2019;42:499-509.
30. Sánchez-Menoyo J, Ruiz-Ojeda J, Martínez-Arroyo A, García-Moncó J, Paz A-D, Vicente-Olabarría I. Complicación espinal hemorrágica secundaria a una punción lumbar diagnóstica. Revista de Neurología 2009;48:418-420.
31. Brown MW, Yilmaz TS, Kasper EM. Iatrogenic spinal hematoma as a complication of lumbar puncture: What is the risk and best management plan? Surgical neurology international 2016;7:S581-589.
32. Park JH, Kim JY. Iatrogenic Spinal Subarachnoid Hematoma after Diagnostic Lumbar Puncture. Korean Journal of Spine 2017;14:158-161.
33. Ruff RL, Dougherty JH, Jr. Complications of lumbar puncture followed by anticoagulation. Stroke 1981;12:879-881.
34. Avila A, Rivarola S, Oliveros K, et al. Hematoma espinal secundario a punción lumbar en paciente anticoagulado con rivaroxabán. Revista Hematología 2019;23:93-96.
35. Lang SS, Kofke WA, Stiefel MF. Monitoring and intraoperative management of elevated intracranial pressure and decompressive craniectomy. Anesthesiology clinics 2012;30:289-310.
36. Durango-Espinosa Y, Moscote-Salazar L, Keni R, Deora H, Agrawal A. The puzzle of spontaneous versus traumatic subarachnoid hemorrhage. Apollo Medicine 2019;16:141-141.
37. Heit JJ, Iv M, Wintermark M. Imaging of Intracranial Hemorrhage. J Stroke 2017;19:11-27.
38. Kim JY, Bae HJ. Spontaneous Intracerebral Hemorrhage: Management. J Stroke 2017;19:28-39.
39. Parker D, Jr., Rhoney DH, Liu-DeRyke X. Management of spontaneous nontraumatic intracranial hemorrhage. Journal of pharmacy practice 2010;23:398-407.
40. Lacerda Gallardo ÁJ. Craniectomía descompresiva en el tratamiento del traumatismo craneoencefálico grave. Revista Cubana de Neurología y Neurocirugía 2013;3.
41. Lubillo S, Blanco J, López P, et al. Papel de la craniectomía descompresiva en el enfermo neurocrítico. Medicina intensiva 2009;33:74-83.
42. Pizzi MA, Alejos DA, Siegel JL, Kim BY, Miller DA, Freeman WD. Cerebral Venous Thrombosis Associated with Intracranial Hemorrhage and Timing of Anticoagulation after Hemicraniectomy. Journal of stroke and cerebrovascular diseases : the official journal of National Stroke Association 2016;25:2312-2316.
43. Andrade-López A, Lara-Ortega R, Narvaez-Rojas A, Padilla-Zambrano HS, Moscote-Salazar LR. Anticoagulación y trombosis venosa cerebral: el dilema de la craniectomía descompresiva. Acta neurol colomb 2018:223-224.
44. Gessler F, Bruder M, Duetzmann S, et al. Risk factors governing the development of cerebral vein and dural sinus thrombosis after craniotomy in patients with intracranial tumors. Journal of neurosurgery 2018;128:373-379.
45. Wassef SN, Abel TJ, Grossbach A, et al. Traumatic intracranial hemorrhage in patients taking dabigatran: report of 3 cases and review of the literature. Neurosurgery 2013;73:E368-373; discussion E373-364.
46. Greuter L, Ullmann M, Mariani L, Guzman R, Soleman J. Effect of preoperative antiplatelet or anticoagulation therapy on hemorrhagic complications in patients with traumatic brain injury undergoing craniotomy or craniectomy. Neurosurgical focus 2019;47:E3.
47. Lan M, Dambrino RJt, Youssef A, et al. Repeat Surgery After Decompressive Craniectomy for Traumatic Intracranial Hemorrhage: Outcomes and Predictors. World neurosurgery 2020;133:e757-e766.
48. Huertas González N. Características clínicas, manejo y pronóstico de las hemorragias cerebrales asociadas al tratamiento con anticoagulantes orales. 2014.
49. Bruder M, Schuss P, Konczalla J, et al. Ventriculostomy-Related Hemorrhage After Treatment of Acutely Ruptured Aneurysms: The Influence of Anticoagulation

and Antiplatelet Treatment. World neurosurgery 2015;84:1653-1659.
50. Scheller C, Strauss C, Prell J, Simmermacher S, Brandt S. Increased rate of ventriculostomy-related hemorrhage following endovascular treatment of ruptured aneurysms compared to clipping. Acta neurochirurgica 2018;160:545-550.
51. Blostein M, Kerzner R. Practice guidelines for anticoagulation management [online]. Available at: https://cdn.ciussscentreouest.ca/documents/hgj/Hematology/JGH_ACO_Guidelines_Final_06-29-2012.pdf?1541434879. Accessed 26 Apr.
52. Borab ZM, Lanni MA, Tecce MG, Pannucci CJ, Fischer JP. Use of Computerized Clinical Decision Support Systems to Prevent Venous Thromboembolism in Surgical Patients: A Systematic Review and Meta-analysis. JAMA Surg 2017;152:638-645.
53. Stanton R, Woo D, Moomaw C, Haverbusch M, Flaherty M, Kleindorfer D. Abstract WP327: CHADSVASC and HASBED Scores in Patients With Intracerebral Hemorrhage and Atrial Fibrillation. Stroke 2018;49:AWP327-AWP327.
54. Kernan WN, Ovbiagele B, Black HR, et al. Guidelines for the prevention of stroke in patients with stroke and transient ischemic attack: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2014;45:2160-2236.
55. Sahai T, Tavares MF, Sweeney JD. Rapid response to intravenous vitamin K may obviate the need to transfuse prothrombin complex concentrates. Transfusion 2017;57:1885-1890.
56. Halvorsen S, Storey RF, Rocca B, et al. Management of antithrombotic therapy after bleeding in patients with coronary artery disease and/or atrial fibrillation: expert consensus paper of the European Society of Cardiology Working Group on Thrombosis. European heart journal 2017;38:1455-1462.
57. Kuramatsu JB, Sembill JA, Gerner ST, et al. Management of therapeutic anticoagulation in patients with intracerebral haemorrhage and mechanical heart valves. European heart journal 2018;39:1709-1723.
58. AlKherayf F, Xu Y, Gandara E, Westwick H, Moldovan ID, Wells PS. Timing of vitamin K antagonist re-initiation following intracranial hemorrhage in mechanical heart valves: Systematic review and meta-analysis. Thrombosis research 2016;144:152-157.
59. Huisman MV, Barco S, Cannegieter SC, et al. Pulmonary embolism. Nature reviews Disease primers 2018;4:18028.
60. Villar AB, Iturriaga LAR. Tromboembolismo pulmonar. Archivos de Bronconeumología 2010;46:31-37.
61. Figueroa G, Labarca E, Cornejo R, et al. Recomendaciones de la Sociedad Chilena de Medicina Intensiva para la Prevención del Tromboembolismo Venoso en Pacientes Críticos Médico-Quirúrgicos Adultos. Revista Chilena de Medicina Intensiva [internet] 2016;31:162-174.
62. Ubaldini J. Consenso de enfermedad tromboembólica. Consenso Argentino SAC. Revista Argentina de Cardiología 2009;77:411-428.
63. Lara ML, Pérez ML, Lucio AV. Eficacia del tratamiento anticoagulante oral e incidencia de complicaciones en clínica de anticoagulantes. Revista de Especialidades Médico-Quirúrgicas 2013;18:292-298.
64. Mateo J. Nuevos anticoagulantes orales y su papel en la práctica clínica. Revista Española de Cardiología Suplementos 2013;13:33-41.
65. DeWald TA, Washam JB, Becker RC. Anticoagulants: Pharmacokinetics, Mechanisms of Action, and Indications. Neurosurgery clinics of North America 2018;29:503-515.
66. Dornbos D, 3rd, Nimjee SM. Reversal of Systemic Anticoagulants and Antiplatelet Therapeutics. Neurosurgery clinics of North America 2018;29:537-545.
67. Dudley RR, Aziz I, Bonnici A, et al. Early venous thromboembolic event prophylaxis in traumatic brain injury with low-molecular-weight heparin: risks and benefits. Journal of neurotrauma 2010;27:2165-2172.
68. Carrillo-Esper R, Meza-Márquez JM. Monitoreo de la coagulación en el perioperatorio. Revista Mexicana de Anestesiología 2015;38:406-409.
69. Hunt H, Stanworth S, Curry N, et al. Thromboelastography (TEG) and rotational thromboelastometry (ROTEM) for trauma induced coagulopathy in adult trauma patients with bleeding. The Cochrane database of systematic reviews 2015;2015:Cd010438.
70. Schneck MJ. Prophylactic Screening for Venous Thromboembolism in Neurosurgical Patients. Anticoagulation and Hemostasis in Neurosurgery: Springer, 2016: 317-325.
71. Chen X, Jin DY, Stafford DW, Tie JK. Evaluation of oral anticoagulants with vitamin K epoxide reductase in its native milieu. Blood 2018;132:1974-1984.
72. Hodroge A, Matagrin B, Moreau C, et al. VKORC1 mutations detected in patients resistant to vitamin K antagonists are not all associated with a resistant VKOR activity. Journal of thrombosis and haemostasis : JTH 2012;10:2535-2543.
73. Benavides F, Grossman N, Poggi H, et al. Efecto de las variantes de VKORC1 y CYP2C9 sobre la dosis de anticoagulantes orales en individuos chilenos. Revista médica de Chile 2015;143:1369-1376.
74. Kamali F, Wynne H. Pharmacogenetics of warfarin. Annual review of medicine 2010;61:63-75.
75. Chatterjee S, Sardar P, Biondi-Zoccai G, Kumbhani DJ. New oral anticoagulants and the risk of intracranial hemorrhage: traditional and Bayesian meta-analysis and mixed treatment comparison of randomized trials of new oral anticoagulants in atrial fibrillation. JAMA neurology 2013;70:1486-1490.
76. Mehta VA, Wang TY, Sankey EW, et al. Restarting Therapeutic Anticoagulation After Elective Craniotomy

for Patients with Chronic Atrial Fibrillation: A Review of the Literature. World neurosurgery 2020;137:130-136.
77. Divito A, Kerr K, Wilkerson C, Shepard S, Choi A, Kitagawa RS. Use of Anticoagulation Agents After Traumatic Intracranial Hemorrhage. World neurosurgery 2019;123:e25-e30.
78. Sadighi A, Wasko L, DiCristina H, et al. Long-term outcome of resuming anticoagulation after anticoagulation-associated intracerebral hemorrhage. eNeurologicalSci 2020;18:100222.
79. Nassiri F, Hachem LD, Wang JZ, et al. Reinitiation of Anticoagulation After Surgical Evacuation of Subdural Hematomas. World neurosurgery 2020;135:e616-e622.
80. Saraf K, Morris PD, Garg P, Sheridan P, Storey R. Non-vitamin K antagonist oral anticoagulants (NOACs): clinical evidence and therapeutic considerations. Postgraduate medical journal 2014;90:520-528.
81. Baber U, Mastoris I, Mehran R. Balancing ischaemia and bleeding risks with novel oral anticoagulants. Nature reviews Cardiology 2014;11:693-703.
82. Ebright J, Mousa SA. Oral anticoagulants and status of antidotes for the reversal of bleeding risk. Clinical and applied thrombosis/hemostasis : official journal of the International Academy of Clinical and Applied Thrombosis/Hemostasis 2015;21:105-114.

Romanian Neurosurgery (2022) XXXVI (1): pp. 62-69
DOI: 10.33962/roneuro-2022-011
www.journals.lapub.co.uk/index.php/roneurosurgery

Acute phase of traumatic brain injury. Overview of neuroimaging tools and significant findings

Maria Paz Bolaño-Romero[1,2], Gustavo Gaitan-Herrera[2], Daniel Galarza-Garrido[2], Diana Carolina Caicedo-Posso[3], Nathalia Carolina Ordóñez-Veloza[4], Cristian Javier Guerrero-Eraso[5], Jesús David Hidalgo-Pasaje[6], Jesús David Castillo-Martínez[7], Sebastian Pinzon-Vargas[8], Michael Gregorio Ortega-Sierra[2]

[1] Grupo Prometheus y Biomedicina Aplicada a las Ciencias Clínicas, School of Medicine, Universidad de Cartagena, Cartagena, COLOMBIA
[2] Medical and Surgical Research Center, St Mary's Medical Group, Cartagena, COLOMBIA
[3] School of Medicine, Universidad Icesi, Cali, COLOMBIA
[4] School of Medicine, Universidad de Manizales, Manizales, COLOMBIA
[5] School of Medicine, Fundación Universitaria San Martín, Bogotá, COLOMBIA
[6] School of Medicine, Universidad de Antioquia, Medellín, COLOMBIA
[7] School of Medicine, Universidad Nacional, Bogotá, COLOMBIA
[8] School of Medicine, Universidad de Santander, Bucaramanga, COLOMBIA

ABSTRACT

In neurotrauma, diagnostic imaging plays a fundamental role in the early detection of treatable injuries or the mitigation of secondary injuries. Currently, the routine imaging techniques used in the setting of a head trauma patient include non-contrast computed tomography (CT), computed tomography angiography (CTA), conventional magnetic resonance imaging (MRI) with T1, T2 or diffusion imaging. Of the above mentioned, CT is superior to MRI in terms of speed of examination, due to the greater access to portable equipment in the patient's environment, which reduces the risk of secondary complications at the time of transfer to the radiology department. Nevertheless, MRI provides a much higher quality of images than CT. MRI is not indicated for the diagnosis of acute brain injury, but if the results of CT without contrast are normal, and neurological manifestations are present, it is indicated. As a result, CT should be the first study requested to the imaging service by the medical team in charge of the patient during the acute phase of the traumatic brain injury. The main objective of this review is to present some of the advantages and disadvantages offered by the different diagnostic imaging methods when approaching and managing brain-injured patients, with emphasis on the acute phase of trauma.

Keywords
brain injuries,
neurotrauma,
cerebral haemorrhage,
skull fractures,
tomography,
traumatic brain injury

Corresponding author:
Michael Gregorio Ortega-Sierra

Medical and Surgical Research Center, St Mary's Medical Group, Cartagena, Colombia

mortegas2021@gmail.com

Scan to access the online version

Introduction

The improvements in the technology have provoked a modern advent of the image analysis to establish clinical variables at different scales, so that the interpretation of these can create a very powerful diagnostic approach [1]. Diagnostic images play an important role in the detection of treatable lesions and prevention or mitigation of secondary injuries [2]. Currently, imaging techniques routinely used in the context of neurotrauma include computed tomography (CT) without contrast, computed tomography angiography (CTA) and conventional magnetic resonance imaging (MRI) with T1, T2, susceptibility or weighting diffusion [3]. Other studies exist but have minimal impact. Neuroimaging may allow for better characterization of patients for both treatment decisions, and the improved selection for clinical trials [4].

Despite its limitations, the Glasgow Coma Scale (GCS) is a practical, reliable and objective method to assess the level of consciousness. Patients are scored according to their best response in three categories (eye opening, verbal responses and motor score), and classified as mild, moderate and severe [5]. GCS is one of the most frequently used methods to identify patients that benefit from imaging studies [5]. In 2007 the Traumatic Head Injury Guide produced by NICE, replaced the head plain X-ray as the primary imaging modality for traumatic brain injury (TBI) by computerized axial tomography, which at first caused an increase in the cost of attention to the destabilization of the system; therefore, with the intention of balancing clinical benefit with costs, as well as exposure to radiation, strict criteria have been established for its realization [6]. The following are the imaging investigations used for the management of patients with TBI, their indications and relevant findings.

Methods

A bibliographic search was carried out in the databases PubMed and Science Direct and in the Google Scholar search engine using the following terms: Brain injuries, Neurotrauma, Cerebral hemorrhage, Skull Fractures, Tomography, Traumatic brain injury. Articles in English language were included, emphasizing the importance of neuroimaging for a correct approach to the patient with acute brain injury, through different studies where CT stands out as the main imaging method that allows to cover a wide range of pathologies that may go unnoticed or not be identified by other types of imaging during the acute phase. A total of 185 articles were identified, including original articles, subject reviews, systematic reviews, letters to the editor, case reports and case series. 43 articles were selected that matched the aim of the article.

Results

Plane X-rays

Plane X-ray (SR) of the head (skull) has no indication for the initial evaluation of the patient with traumatic brain injury. Even tough skull fractures are associated to brain injuries, its sensitivity is quite low and, in many instances, increase the chances of diagnostic failure. Thus, both the Centers for Disease Control and Prevention (CDC) and the practice guidelines of the American College of Emergency Physicians (ACEP) no longer recommend the use of plain head x-rays for acute traumatic injuries [3].

Hitesh et al. studied the effectiveness of plain x-rays for the identification of skull fractures and compared to CT scan, autopsies and SR and TAC. The SR eluded skull fractures by 19% and CT by 11.9%. Thus, the SR has no advantage and little benefit with respect to the CT, leads to the delay in the diagnosis of the injury and exposes the patient to unnecessary and harmful radiation [7].

Computed tomography

CT is the neuroimaging that is requested in the first instance in cases of TBI [8,9] **(Figures 1-2)**. It is superior to the MRI in terms of the speed of the examination, due to the greater accessibility to portable equipment that can be located around the patient, reducing the risk of complications secondary to transfer to radiology services. In addition, most institutions have tomographic equipment and to a lesser extent have resonators, so the initial examination is the CT scan. However, the resolution quality of the images is lower than the data offered by the MRI [8]. Computed tomography provides important prognostic predictors of severe head injury in children too [10]. At present, the head fixation frame of most CT scanners has certain limitations. the width cannot be effectively adjusted and texture is hard, so the head cannot be effectively fixed. The patient's intentional and unintentional movements cause head movements. Furthermore, the noise during CT operation also causes the test

subject to be agitated to a certain extent. Once the head movement coefficient is too large, it will significantly affect the image data quality. This has forced medical workers to repeat CT scanning, which could expose patients to increased radiation doses and increase the workloads of medical and paramedical staff and machines, leading to increased costs and less efficient emergency service [11]. Current organization-specific practice involves placing the patient for observation and obtaining a repeat CT scan within 24 hours of initial imaging. This requires increased utilization of health care resources with increasing costs and potentially an increase in hospital-related adverse events and may also place a burden on patients and their families. We hypothesize that the vast majority of patients with low-mechanism closed head trauma who receive direct oral anticoagulant without abnormal initial cephalic CT findings will not have a late intracranial rebleed and, in fact, will not benefit from a prolonged hospital stay or repeat imaging [12].

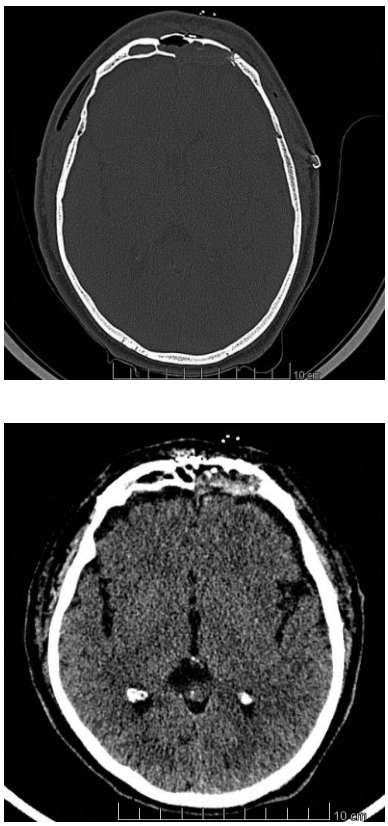


Figure 1. a, b. Computed tomography image showing skull fracture of the frontal bone

Indications: Some guidelines state that simple CT should be performed in patients with moderate or severe trauma (GSC <13) due to the higher incidence of acute intracranial hemorrhage, however different studies have shown that from 16% to 21% of patients with GCS 13 to 15 have acute intracranial hemorrhage so the CT scan should be performed [13].

Figure 2. Computed tomography image showing skull fracture secondary to a firearm

Most relevant findings: Skull fractures, hemorrhages, bruises, or cerebral edema, commonly determine the impact of the lesions on adjacent brain tissue (the mass effect, the compressed ventricle), as well as the extent and location of the injuries [6]. The main objective for the CT scan is to identify lesions can be treated by surgery, but also to monitor the patients to prevent or early identify the appearance of phenomena compatible with injuries secondary to trauma [3,7].

Skull fractures

CT Linear fractures, such as those of the groove of the middle meningeal artery, may be associated with epidural hematoma while temporal bone fractures, due to the bone lesser stiffness and the intracranial venous sinuses.

Depressed skull fractures are defined by the concavity of the affected bone, and is associated with severe complications such as hemorrhages, seizures and neuroinfection [14]. When penetrating fractures are detected, the presence of factors related to a complex prognosis should be ruled out. Basilar fractures may be missed by the CT scan, and should be suspected when facial bones fractures such as

ethmoid, sphenoid, lamina cribrosa, occipital or petrous area of the temporal fractures are identified [15]. The detection of skull fracture on CT can be interpreted not only as an objective indicator of primary injury but also as a factor causing secondary damage, including the release of neuroinflammatory cytokines and coagulopathy [16]. While computed tomography plays an essential role in the management of these particular patients, there is currently no support in the literature to suggest rapid repeat imaging in this population.

There are several risk factors that may be associated with worse outcomes in the setting of depressed skull fractures. These predictors include the GCS score at presentation, fracture site, and fracture type. Fractures involving more than one area and those associated with other intracranial injuries have also been shown to worsen outcome and increase the need for surgical intervention [17].

Intracerebral hemorrhage

CT can also show us the existence or not of bruising. Brain contusions are also detected by CT. The imaging characteristics are related to the time of establishment of the lesion and the presence or absence of bleeding. They are usually located in the frontal and temporal lobes [1,8]. Acute intracranial hemorrhage appears on CT as a region of increased density owing to the linear relationship between attenuation and hematocrit, predominately owing to hemoglobin concentration [18]. With the widely used of the CT, clinicians and researchers are able to qualitatively and quantitatively describe the characteristics of a hemorrhage to guide interventions and treatments. Among these characteristics, the volume of intracranial hemorrhage is an important diagnostic indicator of stroke severity, long-term functional outcome, and mortality [19].

Extra axial hemorrhage

The epidural hematoma classically is characterized by a biconvex morphology, with location outside the axial plane and hyperdensity in the images [1,8]. The majority of patients with acute subdural hematomas have concomitant parenchymal brain injuries. The acute subdural and epidural hematomas are adequately diagnosed with CT and almost never reach the MRI room [20]. While the subdural hematomas have a semilunar appearance. Traumatic subarachnoid hemorrhages are usually located in convex areas such as cisterns [8]. The subarachnoid hemorrhage (SAH) occurs in the space between the arachnoid membrane and the pia. The most common cause of SAH is trauma. In 85% of non-traumatic cases, SAH is caused by ruptured aneurysms, while 10% fit the pattern of so-called non-aneurysmal perimesencephalic hemorrhage. The remaining 5% is due to various rare diseases, such as cocaine abuse [21]. Sources of SAH in trauma include tearing of pial vessels, extra-axial extension of a hemorrhagic contusion, and redistribution of intraventricular hemorrhage caused by damage to subependymal veins. Often the highest concentration of SAH occurs contralateral to the side of direct impact [22].

The quantitative evaluation of the data provided by the CT has not been widely studied, however, the implementation of computer-aided diagnosis technology in the measurement of injuries seems to be very promising. Some studies have shown that the quantification of midline displacement from the day of injury and the volume of bleeding correlate significantly with morbidity and mortality in severe TBI [9].

Other findings

Pathological findings in CT with poor prognosis [9]:

- Herniation
- Injury affecting several lobes
- Intraventricular or subarachnoid hemorrhage
- Injury that compromises the entire brain

There is convincing evidence that TBI increases the incidence of stroke and some epidemiological evidence that stroke outcomes are worse in patients with a history of TBI. Given the very large number of TBI that occur annually it is critically important that we determine why this population is at a greater risk for both more strokes and worse outcomes [23]. Intraventricular hemorrhage on early CT independently predicts poorer short- and long-term outcome in TBI. These findings may help guide intervention, and prognosis when intraventricular hemorrhage (IVH) is present on acute CT imaging. Evidence suggesting that IVH observed on CT may be a surrogate marker for white matter injury warrants further study with MRI imaging [24].

Magnetic Resonance Imaging

Beyond CT and MRI has proven particularly effective in identifying brain regions involved with TBI. In addition, diffusion imaging of white matter (WM) fiber pathways in blast-exposed military veterans who show no visible symptoms of injury sequelae has proven to be a useful diagnostic tool [25]. In complicated mild TBI, magnetic resonance is superior to computed tomography without contrast in diagnosing subarachnoid hemorrhages, contusions and axonal lesions. The sequences used for the TBI are T1, T2, T2-FLAIR, T2-GRE and image by diffusion [26]. Recent contributions to the body of knowledge on TBI favor the view that multimodal neuroimaging using structural and functional magnetic resonance imaging (MRI and fMRI, respectively) as well as diffusion tensor imaging (DTI) has excellent potential to identify new biomarkers and predictors of TBI outcome (27). MRI is the study of choice to further characterize intracranial hemorrhage, offering greater sensitivity in the detection of hemorrhage during all stages of hematoma evolution as well as the ability to more accurately assess the temporal evolution of hemorrhage. MRI also allows more specific investigation of the etiology of intracranial hemorrhage [18].

Indications: MRI is not indicated during the initial management of acute mild TBI. It is indicated when the results of the computed tomography without contrast are normal, and there is persistence of neurological manifestations [28]. MRI can diagnose subcortical lesions that are missed by other imaging techniques [14].

Main findings: Diffuse axonal injury (DAI) is present in a high proportion of patients with severe TBI. MRI has a greater sensitivity/specificity than CT scan for identifying DAI [1,21]. In a study by Cicuendez et al. comparing conventional MR and the diagnosis of DAI, found that the T2, FLAIR, and T2-GRE sequences provide the best visualization in more than 80% of the cases. In addition, with the FLAIR sequence, the hemispheric DAI was better evidenced at the subcortical level while the T2-GRE distinguished hemorrhagic DAI. Therefore, they recommend carrying out MRI in the subacute period of a severe TBI for the accurate diagnosis of DAI, using the sequences T2, FLAIR and gradient echo [29]. Other radiological findings, such as a midline shift, epidural hemorrhage, subarachnoid hemorrhage, and the volume of the hematoma could be additional variables to account for in predicting DAI prognosis [30]. MRI allows the precise quantification, of the size of the lesions as well as, clinically useful prognostic information [28]. The disadvantages of magnetic resonance include prolonged time being a long test and the lower probability of diagnosing fractures [29].

For subacute to chronic TBI, magnetic resonance is the test of choice due to its high sensitivity for the detection of cerebral atrophy [28]. Actually, the modified ultrafast MRI protocol for brain imaging demonstrates clinically acceptable image quality in four out of five sequences and has high accuracy in diagnosing normal and clinically significant abnormalities when compared against the standard MRI protocol for brain imaging. It could potentially benefit a select group of pediatric patients who require neuroimaging [31] routine clinical use of synthetic MRI can be feasible for neuroimaging in daily practice because the overall image quality and conspicuity of anatomical details were acceptable. In addition, the two attending neuroradiologists had no significant challenge during radiologic assessment of all synthetic images.

In previous clinical studies using synthetic MRI, the synthetic images had a similar diagnostic utility with sufficient or better image quality when compared to conventional MRI scans [32]. Also, MRI and CT brain imaging may be equally accurate for detecting acute brain hemorrhages in people with acute focal stroke symptoms. However, MRI may be more accurate than CT imaging for detecting chronic brain hemorrhages [33].

Functional neuroimaging: A variant deserving mention is proton MRI spectroscopy, which evaluates metabolic and biochemical alterations in patients with TBI. The detection of different metabolites allows the diagnosis of anomalous neurometabolic profiles such as the reduction in the levels of N-acetylaspartate (NAA), NAA/choline and NAA/Creatinine. The metabolite reduction persists for weeks to months and is evident both in apparently healthy white matter tissue and in perilesional areas, even though conventional MRI does not show such structural alterations. Biochemical alterations due to neural structural

deformation caused by TBI imbalances neurotransmitters release that can affect the cellular sodium–potassium (Na^+-K^+) pump and results in distribution of membrane homeostasis [34]. The advantage of this imaging tool is to allow correlation with the functional outcome by 6 months after TBI [13]. Recent meta-analysis showed that significant changes in the ratios NAA / Creatinine and Choline / Creatinine and in the absolute values of NAA are associated with clinical outcome in TBI [35].

Future perspectives

Progress in the advancement of strategies and models to improve sensitivity and specificity in the use of neuroimaging is one of the fields of radiology, neurology and neurosurgery studies with the greatest impact today [36-38]. The investment in robotic neurosurgery and the deepening of neurosurgical education during undergraduate, are objectives to be reinforced in low- and middle-income countries, in order to promote in-hospital care of patients with neurosurgical pathology and, in particular, patients with neurotrauma [39-41]. Translational research in neurosurgery, through the search for biomarkers and gene expression (currently known as neurogenomics and neuroimaging genomics), can substantially improve the diagnosis, management and prognosis of this type of patients, by improving the diagnostic accuracy and management of intervention times [41-43]. It is necessary to continue working from the global neurosurgery, to the approach of strategies that help the technological and academic development of neurosurgery in third world countries [37].

Conclusion

TC scan is the imaging test of choice for the early evaluation of TBI patients. The primary objective is to diagnose injuries amenable to surgery, and have been shown to reduce mortality. Plane head X-rays are no longer indicated. For patients suspected of harboring vascular injuries, computed tomography angiography or magnetic resonance angiography are indicated. MRI is used at a later phase of managing TBI patients and most often for the diagnosis of a diffuse axonal lesion and prognosis determination.

References

1. Su E, Bell M. Diffuse Axonal Injury. In: Laskowitz D, Grant G, editors. Translational Research in Traumatic Brain Injury. Boca Raton (FL): CRC Press/Taylor and Francis Group; 2016. Chapter 3. Available from: https://www.ncbi.nlm.nih.gov/books/NBK326722/
2. Chang WT, Badjatia N. Neurotrauma. Emerg Med Clin North Am. 2014; 32(4):889-905.
3. Shin SS, Bales JW, Edward Dixon C, Hwang M. Structural imaging of mild traumatic brain injury may not be enough: overview of functional and metabolic imaging of mild traumatic brain injury. Brain Imaging Behav. 2017; 11(2):591-610.
4. Newcombe V. Neuroimaging of TBI, moving towards 'big data' and precision management. Journal of the Neurological Sciences. 2019: 405:39-40.
5. Murray GD, Brennan PM, Teasdale GM. Simplifying the use of prognostic information in traumatic brain injury. Part 2: Graphical presentation of probabilities. J Neurosurg. 2018; 128(6):1621-1634.
6. Hodgkinson S, Pollit V, Sharpin C, Lecky F; National Institute for Health and Care Excellence (NICE) Guideline Development Group. Early management of head injury: summary of updated NICE guidance. BMJ. 2014; 348:g104.
7. Chawla H, Malhotra R, Yadav RK, Griwan MS, Paliwal PK, Aggarwal AD. Diagnostic Utility of Conventional Radiography in Head Injury. J Clin Diagn Res. 2015; 9(6):TC13-5.
8. Williamson C, Morgan L, Klein JP, Care N. Imaging in Neurocritical Care Practice. 2017; 1(212):840–51.
9. Pervez M, Kitagawa RS, Chang TR. Definition of Traumatic Brain Injury, Neurosurgery, Trauma Orthopedics, Neuroimaging, Psychology, and Psychiatry in Mild Traumatic Brain Injury. Neuroimaging Clin N Am. 2018; 28(1):1–13.
10. Ortega Rodríguez J, Costa Clar JM, Caritg Bosch J, Martín Rodrigo JM, et al, Severe head injury among children: Computerised tomography evaluation and intracranial pressure monitoring as prognostic factors. Medicina Intensiva. 2001; 25(1):8-13.
11. Wang H, Dong L, Xia B, Li Y, Pei Y, Zhang J. Letter to the Editor Regarding "What Is the Best Timing of Repeated CT Scan in Mild Head Trauma with an Initially Positive CT Scan?". World Neurosurgery. 2020; 141:552.
12. Mourad M, Senay A, Kharbutli B. The utility of a second head CT scan after a negative initial CT scan in head trauma patients on new direct oral anticoagulants (DOACs). Injury. 2021; 52(9):2571-2575.
13. Cook NF. The Glasgow Coma Scale: A European and Global Perspective on Enhancing Practice. Critical Care Nursing Clinics of North America. 2021; 33(1):89-99.
14. Vespa PM. Imaging and decision-making in neurocritical care. Neurol Clin. 2014; 32(1):211–24.
15. Al-Sarraj S. The pathology of traumatic brain injury (TBI): a practical approach. Diagnostic Histopathology. 2016; 22(9):318-326.

16. Fujiwara G, Okada Y, Ishii W, Iizuka R, Murakami M, Sakakibara T, et al. Association of skull fracture with in-hospital mortality in severe traumatic brain injury patients. Am J Emerg Med. 2021; 46:78-83.
17. Berg A, Voigt B, Kaul S. Delayed evolving epidural hematoma in the setting of a depressed skull fracture: A case report and review of the literature. Trauma Case Rep. 2019; 22:100193.
18. Saad AF, Chaudhari R, Fischbein NJ, Wintermark M. Intracranial Hemorrhage Imaging. Semin Ultrasound CT MR. 2018; 39(5):441-456.
19. Kuang Z, Deng X, Yu L, Wang H, Li T, Wang S. Ψ-Net: Focusing on the border areas of intracerebral hemorrhage on CT images. Comput Methods Programs Biomed. 2020; 194:105546.
20. Karantanas AH, Komnos A, Paterakis K, Hadjigeorgiou G. Differences between CT and MR imaging in acute closed head injuries. CMIG Extra: Cases. 2005: 29(1):1-8.
21. Vermeulen L, Goethem AV, van den Hauwe L, Beuls E, Parizel PM, Jacobs W. Pseudo-subarachnoid hemorrhage: A tricky CT finding with serious medicolegal implications. Forensic Imaging. 2021; 24:200427.
22. Hijaz TA, Cento EA, Walker MT. Imaging of Head Trauma. Radiologic Clinics of North America. 2011; 49(1):81-103.
23. Anderst J, Carpenter S, Frazier T, Appavu B, Noetzel M, Beslow LA, et al. Subdural hemorrhage in a cohort with cerebral sinovenous thrombosis: Application to abusive head trauma. Child Abuse Negl. 2021; 117:105119.
24. Atzema C, Mower WR, Hoffman JR, Holmes JF, Killian AJ, Wolfson AB, et al. Prevalence and prognosis of traumatic intraventricular hemorrhage in patients with blunt head trauma. J Trauma. 2006; 60(5):1010-7.
25. Bhattrai A, Irimia A, Van Horn JD. Neuroimaging of traumatic brain injury in military personnel: An overview. J Clin Neurosci. 2019; 70:1-10.
26. Enríquez O. Imaging in trauma. Rev Médica Clínica Las Condes. 2013; 24(1):68–77.
27. Irimia A, Wang B, Aylward SR, Prastawa MW, Pace DF, Gerig G, et al. Neuroimaging of structural pathology and connectomics in traumatic brain injury: Toward personalized outcome prediction. Neuroimage Clin. 2012; 1(1):1-17.
28. Wintermark M, Sanelli PC, Anzai Y, Tsiouris AJ, Whitlow CT. Imaging evidence and recommendations for traumatic brain injury: Conventional neuroimaging techniques. J Am Coll Radiol. 2015; 12(2):e1–14.
29. Lozada Martinez ID, Ortega-Sierra MG, Zambrano-Arenas MDP, Páez-Escallón N, Perea-Martinez DG, Caldera-Arrautt YS, et al. Essentials of cerebral fat embolism syndrome: A hidden enemy in trauma. Roneuro. 2021; 35(3):316-20.
30. Cicuendez M, Castaño-León A, Ramos A, Hilario A, Gómez PA, Lagares A. Magnetic resonance in traumatic brain injury: A comparative study of the different conventional magnetic resonance imaging sequences and their diagnostic value in diffuse axonal injury. Neurocirugia. 2017; 28(6):266–75.
31. Guarnizo A, Chung HS, Chakraborty S. Subcallosal haemorrhage as a sign of diffuse axonal injury in patients with traumatic brain injury. Clin Radiol. 2021 ;76(3):237.e15-237.e21.
32. Ahamed SH, Lee KJ, Tang PH. Role of a modified ultrafast MRI brain protocol in clinical paediatric neuroimaging. Clin Radiol. 2020; 75(12):914-920.
33. Ryu KH, Baek HJ, Moon JI, Choi BH, Park SE, Ha JY, et al. Initial clinical experience of synthetic MRI as a routine neuroimaging protocol in daily practice: A single-center study. J Neuroradiol. 2020; 47(2):151-160.
34. Kidwell CS, Chalela JA, Saver JL, Starkman S, Hill MD, Demchuk AM, et al. Comparison of MRI and CT for detection of acute intracerebral hemorrhage. JAMA. 2004; 292(15):1823-30.
35. Rakib F, Al-Saad K, Ahmed T, Ullah E, Barreto GE, Md Ashraf G, et al. Biomolecular alterations in acute traumatic brain injury (TBI) using Fourier transform infrared (FTIR) imaging spectroscopy. Spectrochim Acta A Mol Biomol Spectrosc. 2021; 248:119189.
36. Brown M, Baradaran H, Christos PJ, Wright D, Gupta A, Tsiouris AJ. Magnetic resonance spectroscopy abnormalities in traumatic brain injury: A meta-analysis. J Neuroradiol. 2018; 45(2):123–9.
37. Acevedo-Aguilar L, Gaitán-Herrera G, Reina-Rivero R, Lozada-Martínez ID, Bohorquez-Caballero A, Paéz-Escallón N, et al. Pulmonary injury as a predictor of cerebral hypoxia in traumatic brain injury: from physiology to physiopathology. J Neurosurg Sci. 2021 Nov 11.
38. Lozada-Martínez I, Maiguel-Lapeira J, Torres-Llinás D, Moscote-Salazar L, Rahman MM, Pacheco-Hernández A. Letter: Need and Impact of the Development of Robotic Neurosurgery in Latin America. Neurosurgery. 2021; 88(6):E580-E581.
39. Ortega-Sierra MG, Durán-Daza RM, Carrera-Patiño SA, Rojas-Nuñez AX, Charry-Caicedo JI, Lozada-Martínez ID. Neuroeducation and neurorehabilitation in the neurosurgical patient: programs to be developed in Latin America and the Caribbean. J Neurosurg Sci. 2021.
40. Miranda-Pacheco JA, De Santis-Tamara SA, Parra-Pinzón SL, González-Monterroza JJ, Lozada-Martínez ID. Medical interest groups and work policies as emerging determinants of a successful career: A student perspective - Correspondence. Int J Surg. 2021; 92:106020.
41. Ortega-Sierra MG, Prado-Grajales V, Martinez-Imbett R, Unás-Perea K, Lozada Martinez ID. Research career in neurosurgery: a challenge for future neurosurgeons. J Neurosurg Sci. 2021 Aug 3.
42. Blanco-Teherán C, Quintana-Pájaro L, Narvaez-Rojas A, Martínez-Pérez R, García-Ballestas E, Moscote Salazar L, et al. Evidence-based medicine in neurosurgery: why and how? J Neurosurg Sci. 2021 Aug 3.
43. Herrera-Martinez MP, García-Ballestas E, Lozada-Martinez I, Torres-Llinás D, Moscote-Salazar L. Letter to the Editor. Creating the conditions for gender equality to

end sexual harassment in neurosurgery. J Neurosurg. 2021 Feb 5:1-2.

44. Lozada-Martínez I, Bolaño-Romero M, Moscote-Salazar L, Torres-Llinas D. Letter to the Editor: 'Medical Education in Times of COVID-19: What's New in Neurosurgery?'. World Neurosurg. 2020; 143:603.

Romanian Neurosurgery (2022) XXXVI (1): pp. 70-72
DOI: 10.33962/roneuro-2022-012
www.journals.lapub.co.uk/index.php/roneurosurgery

Primary intracranial malignant melanoma. A case report and review of literature

Deepak Kumar Singh, Diwakar Shankar, Vipin Chand, Mohammad Kaif

Dr. Ram Manohar Lohia Institute of Medical Sciences, Lucknow, INDIA

ABSTRACT

Primary Intracranial Malignant Melanoma (PIMM) is a very rare neoplasm and accounts for 1% of all melanomas and 0.1% of all intracranial tumours. It carries a poor prognosis with overall poor survival. Diagnosis on mere imaging findings of an intracranial melanoma is a daunting task. Multimodality treatment with surgery followed by chemotherapy and radiotherapy have proved to be effective in improving clinical outcome in these patients.

Keywords
melanoma,
primary intracranial malignant melanoma,
PIMM,
intracranial melanoma case report,
intracranial melanoma literature review

Corresponding author:
Diwakar Shankar

Dr. Ram Manohar Lohia Institute of Medical Sciences, Lucknow, India

dev123diwakar@gmail.com

INTRODUCTION

Primary Intracranial Malignant Melanoma (PIMM) is a very rare neoplasm and accounts for 1% of all melanomas [5]. It accounts for 0.1% of all intracranial tumours [5]. PIMMs are derived from melanocytes that are normally present in the leptomeninges. It is more common in males and has a poor survival rate overall. Pathologically it is of two types, diffuse type which infiltrates pia-mater and the subarachnoid space (SAS); and the solitary type which is present as nodular mass. Treatment of choice is complete resection followed by radiotherapy and chemotherapy. We here present a case of PIMM in a 35-year-old male presenting with signs and symptoms of a left cerebellar hemisphere malignant melanoma with leptomeningeal spread.

CASE DISCUSSION

A 35-year-old male presented to our institute with a complaint of headache, decreased hearing in the left ear, difficulty in walking, and diminished vision bilaterally for the past 2 years. Non-contrast computer tomography (CT) head revealed a hyperdense lesion in the left cerebellar hemisphere with associated perilesional edema (Figure 1, A). Contrast-enhanced MRI brain revealed a 4.5cm x 5cm x 3.5cm homogenous mass lesion in the left cerebellar hemisphere which was hyperintense on T1 and hypointense on T2 with mass effect and perilesional edema (Figure 1, B, C, D). Radiographic features were suggestive of meningioma. No evidence of hemorrhage on the GRE sequence was seen.

Scan to access the online version

The patient was taken for surgery and complete excision was done through the left retro-mastoid suboccipital approach. Meningeal spread of the tumor was evident from the dark-colored dural after removal of the bone flap (Figure 2, A). Intraoperative observation revealed a firm, well-demarcated, highly vascular, dark brown, 5cm x 4cm sized lesion with dural attachment (Figure 2, B, C).

Histopathological examination confirmed melanocytes on H and E stains. The tumor consisted of polygonal to spindle-shaped cells disposed of in sheets with abundant dark melanin pigment deposits in the cytoplasm. These cells stained positively with human melanin black-45 (HMB-45) antibody, S-100, and Melan A. It was reported as malignant melanoma.

The patient was evaluated for the alternative site of melanoma but no such sites were found. The postoperative period was uneventful and the patient was discharged on the tenth postoperative day.

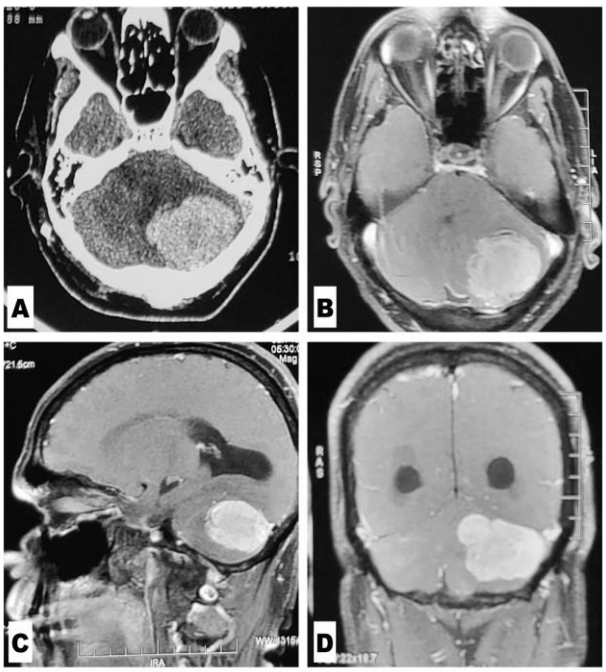


Figure 1. Preoperative CT Non-contrast (CT) and Contrast-Enhanced MRI images. (A) CT images revealed a hyperdense lesion in the left cerebellar hemisphere with associated perilesional edema. (B) Axial view, contrast-enhanced T1WI MRI. (C) Sagittal view, contrast-enhanced T1WI MRI. (D) Coronal view, contrast-enhanced T1WI MRI. Contrast-enhanced MRI brain revealed a 4.5cm x 5cm x 3.5cm intra-axial heterogeneous mass lesion in the left cerebellar hemisphere which was hyperintense on T1 and hypointense on T2 with mass effect and perilesional edema.

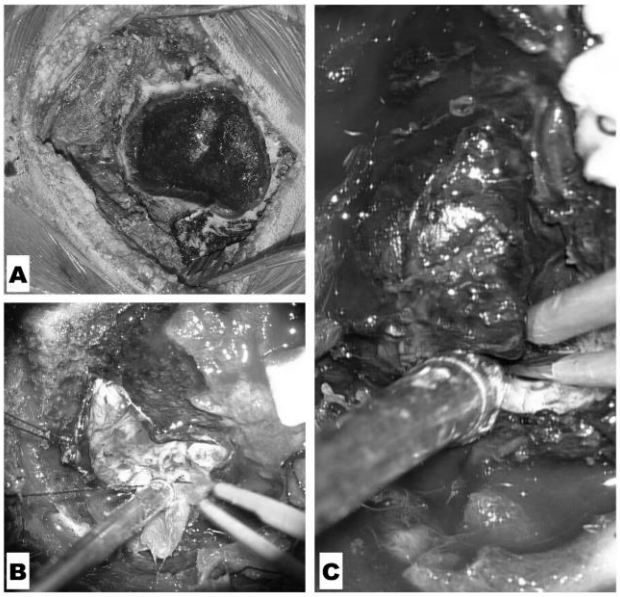


Figure 2. Intraoperative images showing gross total resection of the dark-colored lesion with leptomeningeal involvement and dural attachment.

Discussion

Primary Intracranial Malignant Melanoma (PIMM) is a very rare neoplasm and accounts for 1% of all melanomas [5]. It accounts for 0.1% of all intracranial tumors [5]. PIMMs are derived from melanocytes that are normally present in the leptomeninges. It is more common in males and has a poor survival rate overall [4].

In humans, melanocytes are found in skin, mucous membrane, uvea, etc. There are many theories for the origin of primary intracranial melanomas. The endodermal theory states that some aberrant embryonic ectodermal cells in the central nervous system produce melanin pigment. The neurogenic theory states that the neural crest gives rise to pigment cells. Neural crest later develops into mesodermal and neural elements. The mesodermal theory proposes that the mesodermal pigment cells reach the central nervous system through pial vessels.

Hayward proposed a few features of a primary intracranial melanoma[3]. He suggested that for a PIMM, there should be no malignant melanoma outside the CNS, evidence of leptomeningeal involvement, hydrocephalus, intramedullary spinal lesions, tumor located in the pituitary, or pineal region, and a single intracerebral lesion.

Gibson et al divided intracranial melanomas into

two pathological types [1]. The diffuse type which infiltrates pia-mater and the subarachnoid space (SAS); and the solitary (discrete) type which is present as nodular mass. The diffuse type was more prevalent in younger patients (mean age 26-years). It presented with clinical features of intracranial hypertension, cranial nerve deficit, or meningism. Solitary type is more common in adults (mean age 44-years). Most of the solitary tumors are supratentorial. Leptomeningeal involvement is frequently seen in solitary melanoma. Our patient has a solitary lesion.

Symptoms included headache, vomiting (especially due to raised intracranial pressure), hydrocephalus (43%), focal neurological deficit (due to mass effect, 35%), and convulsion or subarachnoid hemorrhage (16%). Headache and focal neurological deficit were present in our case. Intracranial hypertension and hydrocephalus are more commonly seen in diffuse types. The presence of congenital melanocytic nevus increases the risk of malignant melanoma. 25% of patients with primary intracranial melanomas have congenital nevus. This was not the case with our patient. Extracranial metastasis of PIMM is rare.

It is difficult to differentiate primary central nervous system (CNS) melanomas from metastatic melanomas based on imaging alone. Melanin cells in cerebrospinal fluid (CSF) analysis helps in the diagnosis. 86-97% melanocyte tumors are positive for HMB-45 Antigen. Other commonly used tumor markers are S100 and Melan A. All three were positive in our patient.

Treatment of choice for the intraparenchymal melanocytic tumor is complete resection and postoperative chemoradiotherapy. Survival in adults can be up to 17 years and 8 months in pediatric patients[2]. Mean survival was found to be better (19.6 months) in patients in whom complete resection was done as compared with those where partial resection was done (9.3 months). Also, the prognosis of secondary melanoma was poorer than PIMM. There is 95% mortality in secondary intracranial melanomas.

Initially, radiotherapy (RT) was not offered to patients of PIMM as melanomas were radioresistant tumors but recent studies showed that it was effective in controlling tumor growth. Stereotactic Radiosurgery (SRS) along with whole-brain radiotherapy (WBRT) has been shown to reduce recurrence and improve survival. A high dose of 5000 centigray as SRS in small lesions (size less than 3cm) gave better tumor control in almost 80% of cases. Hydrocephalus caused by the leptomeningeal spread of the disease has been shown to respond well to WBRT.

Dacarbazine is commonly used in the treatment of PIMMs after surgery or radiotherapy. It has 16-20% effectiveness. Other agents are temozolomide and Dimethyl-triazeno-imidazole-carboxamide which is common in use.

Despite optimum treatment, median survival is less than a year. The leptomeningeal spread has a median survival of 10 weeks.

Conclusion

Primary Intracranial Malignant Melanoma (PIMM) is a very rare neoplasm and accounts for 1% of all melanomas and 0.1% of all intracranial tumors. It carries a poor prognosis with overall poor survival. Diagnosis on mere imaging findings of an intracranial melanoma is a daunting task. It is best diagnosed intraoperatively due to its characteristic appearance as well as during preoperative clinical examination. Postoperative chemoradiotherapy plays an important role in preventing recurrence as well as improving overall survival. Collaboration of large-scale data from many such small case series from centers all over the world can confirm findings suggested in this paper and formulate guidelines to improve clinical outcomes.

References

1. Gibson JB, Burrows D, Weir WP: Primary melanoma of the meninges. J Pathol Bacteriol 74:419–438, 1957.
2. Greco Crasto S, Soffietti R, Bradac GB, et al. Primitive cerebral melanoma: case report and review of the literature. Surg Neurol 2001;55:163–8.
3. Hayward RD. Malignant melanoma and the central nervous system. A guide for classification based on the clinical findings. J Neurol Neurosurg Psychiatry 1976;39:526-30.
4. Ma Y, Gui Q, Lang S. Intracranial malignant melanoma: a report of 7 cases. Oncol Lett 2015;10:2171–5.
5. Sloan AE, Nock CJ, Einstein DB. Diagnosis and treatment of melanoma brain metastasis: a literature review. Cancer Control. 2009;16:248e255.

Romanian Neurosurgery (2022) XXXVI (1): pp. 73-83
DOI: 10.33962/roneuro-2022-013
www.journals.lapub.co.uk/index.php/roneurosurgery

Anterior cervical discectomy and fusion in the era of motion preserving surgery. A retrospective study

Arun S., Abhishek V., Prakash Goswami, Sanu V., Jyothish L.S., Sunil Kumar, Suresh Kumar, Anil Kumar, Oommen P. Mathew*

Department of Neurosurgery, Govt Medical College, Thiruvananthapuram, Kerala, INDIA
* Research Investigator, Population Research Centre, University of Kerala, Thiruvananthapuram, Kerala, INDIA

Keywords
adjacent segment disease, anterior cervical discectomy and fusion, cervical spondylotic myelopathy, standalone cage

ABSTRACT

Background. Anterior cervical discectomy and fusion is accepted as the standard surgical treatment of cervical spondylotic myelopathy. Cervical disc arthroplasty has gained widespread acceptance as an alternate choice for ACDF. We intend to present the clinical and radiologic outcomes of patients who underwent ACDF in our department.

Methods. Designed as a retrospective study, the primary objective was to assess the nonunion in patients undergoing ACDF and Anterior cervical Corpectomy and fusion. The article discusses the outcome for the discectomy group. All patients who underwent ACDF for CSM from January 2014 to December 2018 were included. Patients who underwent posterior fusion in addition to anterior approach, revision surgery and congenital anomalies of the spine were excluded. Of the 230 eligible patients,46 subjects were part of the study. They underwent neurologic and radiographic examination and their past records were examined. Neurologic outcome was assessed using Nurick grade and mJOA score. Dysphagia was assessed using the Bazaz score. Neck radiographs were analyzed for fusion, Adjacent segment Disease, subsidence, cervical and segmental lordosis.

Results. The overall response rate was 25.65%. The mean follows up duration was 4 years. The mean age of the population was 47.1 years. The most common operating level was C5/6. The neurologic status of patients improved from the baseline. There was mild transient dysphagia in 5(10.9%) patients. The overall rate of fusion was 91.3%. Subsidence was seen in 10.9%. Degenerative changes were noted in postop x rays of 67.4% of patients. There was no mortality.

Conclusion. ACDF achieves thorough decompression thereby resulting in neurologic improvement. It produces effective and sustained neurologic improvement. Preoperative adjacent segment degenerative changes were significantly associated with the development of ASD during follow up. This is can due to the progression of the disease. Though the procedure improves the lordosis, it tends to decrease with follow up.

Corresponding author:
Abhishek V.

Govt Medical College, Thiruvananthapuram, Kerala, India

vijayan.abhishek@gmail.com

Scan to access the online version

Introduction

Anterior cervical discectomy and fusion(ACDF) was pioneered by Cloward and Smith and Robinson separately in 1950s for the surgical treatment for patients with cervical spondylotic myelopathy(1,2,3) .Thorough and direct decompression of the pathology and subsequent bony fusion of the involved levels , as advocated by the Cloward forms the fundamental principle of the surgery(2). Anterior approach have became the standard treatment for Cervical Spondylotic Myelopathy. Anterior Cervical Corpectomy and Fusion (ACCF) was introduced by Whitecloud and LaRocca to circumvent graft failure following discectomy following multilevel discectomy.(4)

Though anterior cervical discectomy and corpectomy are excellent in achieving the decompression of the spinal cord and restoring the lordosis ; they are not without complications. Variously reported complications include graft subsidence, graft migration , graft collapse , nonunion, loss of lordosis and adjacent segment disease (ASD) (5,6,7,8). Injury to neurovascular structures and oesophagus though rare have been reported (5,7) .The design of the Anterior cervical plates introduced in 1980s to address the problem of graft migration and nonunion has evolved to the presently popular translational plates(9).The translational plates achieve graft loading by permitting controlled subsidence, a prerequisite for fusion (10). Adjacent segment degeneration a described complication of fusion surgeries , was further elaborated by Hilibrand et al as occurring at a rate of 2.5% per yr with cumulated rate of 25.9 % at 10 yrs (11) .

The hypothesis that fusion increases the stress at the adjacent levels and subsequently accelerates degenerative changes at those levels ,brought back motion preserving surgery in to surgeon's armamentorium. Originally introduced by Ulf Fernstorm in 1966, artificial cervical disc was a stainless steel ball bearing device ,which was discontinued due to high failure rate (12,13) . The next era in motion preserving implants happened with introduction of Frenchay (Prestige) and Bryan artificial disc .However both the devices were of different designs(14,15).First decade of 21 st century saw many arthroplasty devices completing trials and getting approval for use in Cervical Spondylotic Myelopathy concurrent with expanding indications for their use(16). Accruing evidence from long term results of RCTs and multiple meta analyses suggested superiority of CDA(Cervical Disc Arthroplasty) over ACDF in overall outcome , adjacent segment degeneration and secondary surgery at the index and adjacent levels(17,18) . Dynamic Cervical Implant(DCI), developed by Dr G Matge et al is a U shaped single piece implant with teeth for fixation into adjacent endplates, with U - limb of the implant facilitating controlled flexion and extension, while preventing axial rotation and lateral bending ,thereby reducing the stress on facet joints. .Matge et al reported excellent short term neurologic outcome and motion preservation in majority of the patients (19).

Though present day neurosurgical literature is replete with high quality evidence from many RCTs which suggest better overall outcome of CDAs over ACDF all these trials however were nonblinded .Recent analysis from a single blind trial for CDA vs ACDF found comparable results for PROM(Patient Reported Outcome Measures) as well as clinical adjacent segment disease(20).Results of two double blinded trials comparing CDA with ACDF and ACD, NECK (NEtherlands Cervical Kinematics Trial)and PROCON trial reported no advantage of CDA over ACDF in either patient outcome variables or in ASD. Data from the same trials show the incidence of Heterotopic ossification(HO) in 68%-85% of patients ,with half of them being motion restricting severe HO (21). Above all CDA is the not the panacea for all patients needing anterior cervical fusion with only 47 % of all patients undergoing ACDF for various indications being candidates for CDA (22,24). Regarding DCI ,the outcomes reported by Matge et al were not replicated in other series(23) .

The present article discusses results of the subgroup analysis of the study conducted in the department to determine the rate of nonunion in patients undergoing anterior cervical fusion for spondylotic myelopathy including anterior cervical discectomy and corpectomy . The radiologic and clinical outcome of the discectomy and fusion subgroup is presented here.

Materials and methods

Patient population

The study was designed as a retrospective design and was approved by Institutional Ethics Committee. The primary objective was to determine the rate of

nonunion in patients undergoing anterior cervical fusion for cervical spondylotic myelopathy and included both discectomy and corpectomy subgroups. The patients operated between January 2014 to December 2018 in our Department and willing to be part of study were included .Those with history of previous cervical spine surgery , with congenital anomalies of spine and those who needed posterior approach in addition to anterior surgery were excluded. The patients underwent a detailed neurologic examination and radiographic examination with X rays of the Cervical Spine. Their hospital records, previous images , and records of the follow up visit were examined. Of the 256 eligible patients 59 patients who were willing to be part of the study and with complete data were included in the study. Of these 46 patients had undergone discectomy while 13 had received corpectomy. The baseline data of the patients are summarised in Table-1.

Table 1 Demographic details of patients

Age in years Mean ± SD	47.1 ± 10.7
Sex (M/F)	32/24
Presence of other comorbidities(%)	16 (34.8)
History of Smoking(%)	14 (30.4)
Single level affected (%)	31 (67.4)
Presence of MRI T2 hypertintensity (%)	19 (41.3)

Surgical procedure

The involved levels were approached using an oblique neck incision after identifying the level preoperatively with C arm. The Caspar retractor system was used to retract the great vessals of the neck and the tracheoesophageal complex. The longus colli was detached from anterior vertebral surface. Subsequent to reconfirming the level annulotomy and discectomy was done with microscopic assistance. The disc space spreader was used to widen the disc space during discectomy. After complete discectomy PLL(Posterior Longitudinal Ligament) was inspected for any defect and disc fragments posterior to the PLL was removed. The osteophytes were thinned using drill and removed using Kerrison punches and was confirmed using C arm. After satisfactory decompression of the cord, the endplates were prepared and appropriately sized cages or standalone cages made of Titanium filled with locally harvested bone pieces were impacted in to the disc space while avoiding overdistraction. The standalone cages have a side flange with a screw hole which allowed placement of a single screw in to the adjacent vertebral bodies. For those with conventional plates, a contoured plate of appropriate length was placed over the adjacent segment and fixed using 4 screws,2 each in to adjacent bodies. For patients undergoing Corpectomy, the upper and lower discs were removed followed by median corpectomy. Osteophytes were drilled thin and removed with punches .Once decompression was confirmed, adequately sized Titanium cages were impacted after filling them with bone harvested from the removed vertebra.

Patients were usually discharged on 5 th postoperative day. Patients were given a cervical collar for 6 weeks. The follow ups were at 6 weeks,3 months,6 months and 1 year and annually thereafter .At 3 months,6 months and at 1 year f/u ,they undergo C Spine x rays .

Clinical and radiologic outcome assessment

Nurick grade and mJOA (modified Japanese Orthopaedic Association) score were used to assess the neurologic outcome . Radiologic assessment was done using plain and dynamic x rays . Bazaz criteria was used for assessing dysphagia. The criteria used for fusion was absence of movement of >2 mm between spinous processes of the fused segment and absence of radiolucency between the implant and the bony surface and absence of bridging bone between the fused vetebrae. Subsidence was interpreted as migration of the cage more than 2mm in to the adjacent bodies(25). The criteria proposed by Chung et al was used to assess ASD (26).The implant complications included - screw pullout, screw breakage, plate loosening and plate breakage. Global Cervical Lordosis was measured using Cobb Angle between inferior endplate of C2 and inferior endplate of C7(27).Segmental angle was defined as the angle between the superior endplate of superior

vertebra and inferior endplate of inferior vertebra (28)

Statistical methods

Categorical and quantitative variables were expressed as frequency (percentage) and mean ± SD respectively. Chi-square test and Fisher's exact test were used to find association between categorical variables. Mann-Whitney U Test was used to compare selected quantitative parameters between type of surgery. For all statistical interpretations, p<0.05 was considered the threshold for statistical significance. Statistical analyses was performed by using a statistical software package SPSS, version 20.0

RESULTS

The discectomy subgroup included 46 patients.The demographic data of the patients is given in Table 1. Mean duration of follow up was 4 years. 31 (67.4%) % of patients had pathology affecting one level while 32.6% had pathologies at multiple levels. The details of the levels is given in Table 2. Cord signal changes ie, T2 hyperintensity were present in 41.3 %. Plate extending in to adjacent disc space was present in 30.4 % patients in postop x ray . Among patients with plate overlap majority of the overlap was at the cranial level (90.5%) and in 9.5% of subjects overlap was at the inferior level. Degenerative changes at adjacent levels were present in preop x rays in 43.5 % of patients. Regarding the height of cage most commonly used was 6 mm cages in 47.8% of patients, 7 mm in 34.8 %, 8 mm in 10.9% and 5 mm in 6.5% patients.

Table 2. Level Affected

Primary level	Count
C3/4	8 (17.4)*
C4/5	11 (23.9)*
C5/6	19 (41.3)*
C6/7	8 (17.4)*

( )* - in Percentages

Neurologic status of patients improved after surgery as reflected by the improvement in Nurick grade and mJOA score (Table 2) and this improvement was sustained till final follow up. Mild transient dysphagia occurred in 5 (10.9%) patients which improved in all during the postop period. Regarding radiologic outcome ,the overall fusion rate was 91.3%(Figure 1).

Degenerative changes were noted in postop x rays of 67.4% patients , 3 patients had implant related complications 2 had screw breakage and one suffered loosening of the screw.There was no mortality in the group.One patient had deteriorated neurologically in immediate postop period due to haematoma and required evacuation , following which patient improved gradually .

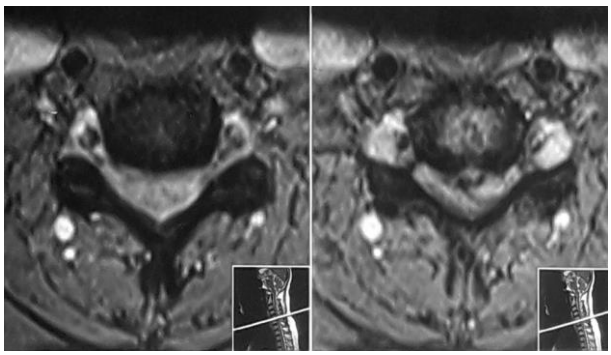


Figure 1. A – Sagital T2 image showing cord compression due to the disc. B -Axial Image .C ,D and E - Lateral radiographs - preop ,Postop and at 2 years f/u respectively . 2 yr radiograph shows solid fusion of the operated level.

The lordosis both cervical and segmental angle improved after surgery , but there was partial loss of this improvement over the follow up period (Table 3). Subsidence occurred in 5 patients .All of them had undergone fusion with standalone cage (p = 0.026)

The degenerative changes on X rays had increased from 43.5% in preop x rays to 67.4% in post op xrays. The affected level, no of operated levels, plate extending to the adjacent disc space , height of cage were not associated with postop degenerative changes .However preop degenerative changes had a significant association with postop X ray changes (p=0.025).

Table 3 Cervical Lordosis and Segmental Angle comparison

		Mean ± SD	Median (IQR)	Pair	p
Cervical lordosi	Pre Op	18.2 ± 6.5	18 (15 - 23)	-	-
	Post Op	22.6 ± 7.5	23 (18 - 26)	Pre Vs Post	p<0.01
	Follow up	19.8 ± 6.5	20 (15 - 23)	Pre Vs Follow up	0.007
Segmental angle	Pre Op	3.5 ± 2.3	3 (2 - 5)	-	-
	Post Op	4.7 ± 2.2	5 (3 - 6)	Pre Vs Post	p<0.01
	Follow up	3.5 ± 2.2	3 (2 - 5)	Pre Vs Follow up	0.351

Table 4.

		Mean ± SD	Median (IQR)	Pair	p
Nurick grade	Pre Op	3.1 ± 1.1	3 (2 - 4)	-	-
	Post Op	2.4 ± 1.4	2 (1 - 4)	Pre Vs Post	p<0.01
	Follow up	1.4 ± 0.9	1 (1 - 1)	Pre Vs Follow up	p<0.01
M JOA	Pre Op	12.2 ± 3.2	13 (11 - 14)	-	-
	Post Op	12.8 ± 3.5	13 (11 - 15)	Pre Vs Post	p<0.01
	Follow up	15.7 ± 3.1	17 (15 - 18)	Pre Vs Follow up	p<0.01

Discussion

The participation rate in the study was (25.65%) .The travel restrictions imparted due to COVID and the patient reluctance to attend the hospital OPD which was a dedicated COVID treatment centre might have contributed to low participation rate.

Anterior Cervical Discectomy and fusion remains as a standard surgical option for patients with Spondylotic myelopathy despite the popularity of cervical disc arthroplasty . Evidence from control arm of CDA -IDE(Cervical Disc Arthroplasty – Investigational Drug Exemption) trials provide high quality evidence about the outcome and complications of ACDF (29).The fusion rate 97 % -98% along with excellent clinical outcome in 94% reported by Cloward in his series of more than 2000 patients, operated for various pathologies underscores safety and efficacy of the procedure as well as the sound scientific basis of this procedure(3). The ability to achieve thorough decompression of the offending pathology and restoration of lordosis are the inherent advantages of the procedure .This is reflected in the neurologic outcome after fusion surgeries ,which shows a sustained improvement in neurologic function on long term follow up (30,31,32). A recent study by Karim et al concluded that the neurologic improvement in all groups ie, mild ,moderate and severe myelopathy , though the improvement was more pronounced in severe group (33).In our study population the mean mJOA score improved from the 12.8 (SD 2.5) to 16.5 (SD 1.9) at final follow up which is similar to the result from the past studies. All these results point to the efficacy of the procedure.

Postop dysphagia is a frequent complication reported after anterior cervical fusion with reported incidence ranging from 12% -35% (34).This subsides in majority of the patients though it can be troublesome for a minor group of patients Various proposed etiological factors for dysphagia include design of the plate, female sex, number of levels operated and use of conventional plate (35,36,37).Mild Dysphagia for solid food present in 10.9% of our patients during immediate postop period resolved in follow up.

Cloward in his article stressed the role of fusion in ACDF as equally important as decompression. He had used variously shaped allografts in his patients with an excellent fusion rates of up to 97% (3). Though studies variously use absence of relative motion between the spinous processes of fused segments , the presence of bridging bone and absence of radiolucency between the endplate and implant as the criteria for fusion there is no uniformity in the definition for fusion. Fraser et al in a metanalysis reported reported an overall fusion rate of 89.2% for anterior fusion surgeries which also included noninstrumented fusion and corpectomies(38). For instrumented ACDF, the fusion rate varied from 82.5% for multilevel to 97.1% for single level(38). However information on fusion rates stratified according to the implant type was not availbale. Noordhoek et al reported comparable fusion rates of >90 % for Titanium and PEEK cages (38,39). Our study population using Titanium cages had a fusion rate of 91.3% . The cohort of patients who didn't achieve radiologic fusion in our group were neurologically stable and were free of symptoms. (Figure 2).

Figure 2. A – Nonunion after C5/6 fusion- Lateral Radiographs (A) preop, (B) Postop,(C) at 2 yrs,(D) Extension and (E) Flexion images .Flexion pronounces defect in the bridging bone and radiolucency around the implant.

Adjacent segment degeneration after anterior cervical fusion was noticed by several authors as the the procedure gained widespread popularity(40,41,42).Hilibrand et al in a series of 409 patients followed up to 21 years reported the annual incidence of adjacent segment degeneration of 2.5% per year . Using a survivorship analysis they estimated cumulative incidence of 25.9 % at 10 years. Authors however had distinguished between adjacent segment disease and symptomatic adjacent segment disease which occured in 14.2 %.They recognized single operated level and C5/6 or C6/7 as risk factors for development of ASD(11) .The etiology of the adjacent segment disease is the subject of a hitherto unsettled discussion. HIlibrand et al after comparing the occurrence of ASD in anterior and posterior surgery groups concluded ASD as outcome of natural history of disease rather

than an outcome precipitated by treatment(43). Other risk factors emerged from further studies includes Plate extending to the adjacent disc space - plate to disc distance < 5 mm (44) , kyphotic postop sagittal alignment in patients with ASD (45) ,preop needle localization at wrong level (46). 0.5 to 1 fold distraction on the other hand has been found to be protective against ASD(47) .In our patient population the number of levels operated, plate overlap in to adjacent disc space and height of the cage were not associated with development of degenerative changes in postop x rays .However degenerative changes in pre op x rays(43%) was significantly associated with development of further changes in post op x rays(67%)(p = .025).This finding supports the argument that the ASD is an outcome of the natural history of the disease rather than a complication of the treatment. The height of the cage , an indirect marker of distraction had no association with the ASD in our study .

Subsidence of the cage in to the adjacent vertebral body is not an infrequent phenomenan with the use of metal cages .There have been contrasting reports of subsidence unfavourably affecting clinical outcome(48,49) and having no impact on clinical outcome(50,51). Various authors have reported patient related, technique related a and implant related factors associated with subsidence.The risk factors include age, sex ,preop cervical alignment ,bone mineral density (52,53,54). Truumees et al reported overdistraction and damage to endplates positively correlated with occurrence of subsidence(55).Cage height and Titanium cages , standalone cages were also reported to be significantly associated with subsidence (56).In our patients five developed subsidence ,all of them had undergone fusion with standalone cage compared to conventional cage and plate ,which was significant (p=.026).However all the patients with subsided cages eventually attained fusion. Both group had similar clinical outcome despite the subsidence. The finding in our patients possible might be due to 2 factors – Titanium cages and greater graft loading with standalone cages compared to conventional cages. Titanium cages have greater modulus of elasticity compared to PEEK and bone , with the resultant modulus mismatch playing a role in subsidence (57).

Restoration of lordosis is one of the advantages of the ACDF over posterior procedures. The improvement in lordosis is reported to be associated with improvement NDI scores and JOA recovery rate .The impact of the correction of lordosis on improvement of JOA score is less clear as to whether this being a result of decompression or directly related to restoration of lordosis (58). Katsuura et al reported the occurrence of local kyphosis in 43% of patients undergoing multilevel discectomy which was a predisposing factor for ASD(59). Reports differ on the long term maintenance of the postop lordosis (60,61).Multilevel procedures tend to lose the lordosis compared to fewer operated segments. In our study the post op cervical lordosis(22.6 degrees) and the segmental angle (4.7 degrees) increased significantly compared to preop levels (18.2 and 3.5) .Though the gain in the lordosis was partially lost over the follow up duration ,the final cervical lordosis(19.8) was higher compared to preop levels and was significant for cervical lordosis ,though not segmental angle at final follow up. We attribute this to the progression of the degenerative disease which was present in 67% of our patients radiologically at final follow up. Thus the occurrence of ASD and loss of lordosis is closely related. However whether this loss of lordosis was due to the progression of the degenerative pathology couldn't be conclusively verified from our study.

Conclusion

Anterior Cervical Discectomy and Fusion is an effective option for the treatment for Cervical Spondylotic Myelopathy . It produces a sustained neurologic improvement .Preoperative adjacent segment degenerative changes was significantly associated with development of ASD during follow up. This is can due to the progression of the disease rather being precipitated by the procedure. Though the procedure improves the lordosis, it tends to decrease with follow up.

References

1. Smith GW, Robinson RA. The treatment of certain cervical-spine disorders by anterior removal of the intervertebral disc and interbody fusion. J Bone Joint Surg Am. 1958 Jun;40-A(3):607-24.
2. Cloward RB. The anterior approach for removal of ruptured cervical disks. J Neurosurg. 1958 Nov;15(6):602-17.
3. Cloward RB. The anterior surgical approach to the cervical spine: the Cloward Procedure: past, present, and future. The presidential guest lecture, Cervical Spine

Research Society. Spine (Phila Pa 1976). 1988 Jul;13(7):823-7.
4. Whitecloud TS, LaRocca H Fibular strut graft in reconstructive surgery of the cervical spine. Spine 1976;1: 33-43
5. Sahjpaul RL. Esophageal perforation from anterior cervical screw migration. Surg Neurol. 2007 Aug;68(2):205-9; discussion 209-10.
6. Jung A, Schramm J, Lehnerdt K et al. Recurrent laryngeal nerve palsy during anterior cervical spine surgery: a prospective study. J Neurosurg Spine. 2005 Feb;2(2):123-7.
7. Orlando ER, Caroli E, Ferrante L. Management of the cervical esophagus and hypofarinx perforations complicating anterior cervical spine surgery. Spine (Phila Pa 1976). 2003 Aug 1;28(15):E290-5.
8. Fountas KN, Kapsalaki EZ, Nikolakakos LG et al Anterior cervical discectomy and fusion associated complications. Spine (Phila Pa 1976). 2007 Oct 1;32(21):2310-7.
9. Böhler J, Gaudernak T. Anterior plate stabilization for fracture-dislocations of the lower cervical spine. J Trauma. 1980 Mar;20(3):203-5
10. Steinmetz MP, Benzel EC, Apfelbaum RI. Axially dynamic implants for stabilization of the cervical spine. Neurosurgery. 2006 Oct;59(4 Suppl 2):ONS378-88; discussion ONS388-9. doi: 10.1227/01.NEU.0000233901.53692.E1.
11. 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 Apr;81(4):519-28.
12. Fernström U. Arthroplasty with intercorporal endoprothesis in herniated disc and in painful disc. Acta Chir Scand Suppl. 1966;357:154-9.
13. Fisahn C, Burgess B, Iwanaga J et al . Ulf Fernström (1915-1985) and his Contributions to the Development of Artificial Disc Replacements. World Neurosurg. 2017 Feb;98:278-280.
14. Cummins BH, Robertson JT, Gill SS. Surgical experience with an implanted artificial cervical joint. J Neurosurg. 1998 Jun;88(6):943-8.
15. Basho R, Hood KA. Cervical total disc arthroplasty. Global Spine J. 2012 Jun;2(2):105-8.
16. Turel MK, Kerolus MG, Adogwa O et al. Cervical arthroplasty: what does the labeling say? Neurosurg Focus. 2017 Feb;42(2):E2.
17. Badhiwala JH, Platt A, Witiw CD et al. Cervical disc arthroplasty versus anterior cervical discectomy and fusion: a meta-analysis of rates of adjacent-level surgery to 7-year follow-up. J Spine Surg. 2020 Mar;6(1):217-232.
18. Nunley PD, Coric D, Frank KA et al . Cervical Disc Arthroplasty: Current Evidence and Real-World Application. Neurosurgery. 2018 Dec 1;83(6):1087-1106.
19. Matgé G, Berthold C, Gunness VR et al . Stabilization with the Dynamic Cervical Implant: a novel treatment approach following cervical discectomy and decompression. J Neurosurg Spine. 2015 Mar;22(3):237-45.
20. Johansen TO, Sundseth J, Fredriksli OA et al .Effect of Arthroplasty vs Fusion for Patients With Cervical Radiculopathy: A Randomized Clinical Trial. JAMA Netw Open. 2021 Aug 2;4(8):e2119606..
21. Yang X, Donk R, Bartels RHMA et al. Comparing Heterotopic Ossification in Two Cervical Disc Prostheses. Spine (Phila Pa 1976). 2020 Oct 1;45(19):1329-1334.
22. Leven D, Meaike J, Radcliff K et al . Cervical disc replacement surgery: indications, technique, and technical pearls. Curr Rev Musculoskelet Med. 2017 Jun;10(2):160-169.
23. Li Z, Wu H, Chu J et al . Motion analysis of dynamic cervical implant stabilization versus anterior discectomy and fusion: a retrospective analysis of 70 cases. Eur Spine J. 2018 Nov;27(11):2772-2780.
24. Auerbach JD, Jones KJ, Fras CI et al . The prevalence of indications and contraindications to cervical total disc replacement. Spine J. 2008 Sep-Oct;8(5):711-6.
25. Yang JJ, Yu CH, Chang BS et al. Subsidence and nonunion after anterior cervical interbody fusion using a stand-alone polyetheretherketone (PEEK) cage. Clin Orthop Surg. 2011 Mar;3(1):16-23.
26. Chung JY, Kim SK, Jung ST et al . Clinical adjacent-segment pathology after anterior cervical discectomy and fusion: results after a minimum of 10-year follow-up. Spine J. 2014 Oct 1;14(10):2290-8.
27. Chen Y, Liu Y, Chen H et al . Comparison of Curvature Between the Zero-P Spacer and Traditional Cage and Plate After 3-Level Anterior Cervical Discectomy and Fusion: Mid-term Results. Clin Spine Surg. 2017 Oct;30(8):E1111-E1116.
28. Chen Y, Lü G, Wang B et al. A comparison of anterior cervical discectomy and fusion (ACDF) using self-locking stand-alone polyetheretherketone (PEEK) cage with ACDF using cage and plate in the treatment of three-level cervical degenerative spondylopathy: a retrospective study with 2-year follow-up. Eur Spine J. 2016 Jul;25(7):2255-62.
29. Zigler JE, Rogers RW, Ohnmeiss DD. Comparison of 1-Level Versus 2-Level Anterior Cervical Discectomy and Fusion: Clinical and Radiographic Follow-Up at 60 Months. Spine (Phila Pa 1976). 2016 Mar;41(6):463-9
30. Fehlings MG, Wilson JR, Kopjar B et al . Efficacy and safety of surgical decompression in patients with cervical spondylotic myelopathy: results of the AOSpine North America prospective multi-center study. J Bone Joint Surg Am. 2013 Sep 18;95(18):1651-8. D
31. Badhiwala JH, Witiw CD, Nassiri Fet al. Efficacy and Safety of Surgery for Mild Degenerative Cervical Myelopathy: Results of the AOSpine North America and International Prospective Multicenter Studies. Neurosurgery. 2019 Apr 1;84(4):890-897.
32. Fehlings MG, Ibrahim A, Tetreault L et al. A global perspective on the outcomes of surgical decompression in patients with cervical spondylotic myelopathy: results

from the prospective multicenter AOSpine international study on 479 patients. Spine (Phila Pa 1976). 2015 Sep 1;40(17):1322-8.

33. Karim SM, Cadotte DW, Wilson JR et al . Effectiveness of Surgical Decompression in Patients With Degenerative Cervical Myelopathy: Results of the Canadian Prospective Multicenter Study. Neurosurgery. 2021 Oct 13;89(5):844-851.
34. Rihn JA, Kane J, Albert TJ et al. What is the incidence and severity of dysphagia after anterior cervical surgery? Clin Orthop Relat Res. 2011 Mar;469(3):658-65
35. Lee MJ, Bazaz R, Furey CG et al. Influence of anterior cervical plate design on Dysphagia: a 2-year prospective longitudinal follow-up study. J Spinal Disord Tech. 2005 Oct;18(5):406-9.
36. Riley LH 3rd, Skolasky RL, Albert TJ et al. Dysphagia after anterior cervical decompression and fusion: prevalence and risk factors from a longitudinal cohort study. Spine (Phila Pa 1976). 2005 Nov 15;30(22):2564-9.
37. Zhou J, Li J, Lin H et al . A comparison of a self-locking stand-alone cage and anterior cervical plate for ACDF: Minimum 3-year assessment of radiographic and clinical outcomes. Clin Neurol Neurosurg. 2018 Jul;170:73-78.
38. Fraser JF, Härtl R. Anterior approaches to fusion of the cervical spine: a metaanalysis of fusion rates. J Neurosurg Spine. 2007 Apr;6(4):298-303.
39. Noordhoek I, Koning MT, Vleggeert-Lankamp CLA. Evaluation of bony fusion after anterior cervical discectomy: a systematic literature review. Eur Spine J. 2019 Feb;28(2):386-399.
40. Baba H, Furusawa N, Imura S et al. Late radiographic findings after anterior cervical fusion for spondylotic myeloradiculopathy. Spine 1993;18: 2167–73.
41. Cherubino P, Benazzo F, Borromeo U et al. Degenerative arthritis of the adjacent spinal joints following anterior cervical spinal fusion: clinicoradiologic and statistical correlations. Ital J Orthop Traumatol 1990;16:533–43.
42. Gore DR, Sepic SB. Anterior cervical fusion for degenerated or protruded discs. A review of onehundred forty-six patients. Spine 1984;9:667–71.
43. Hilibrand AS, Robbins M. Adjacent segment degeneration and adjacent segment disease: the consequences of spinal fusion? Spine J. 2004 Nov-Dec;4(6 Suppl):190S-194S.
44. Park JB, Cho YS, Riew KD. Development of adjacent-level ossification in patients with an anterior cervical plate. J Bone Joint Surg Am. 2005 Mar;87(3):558-63.
45. Park MS, Kelly MP, Lee DH et al. Sagittal alignment as a predictor of clinical adjacent segment pathology requiring surgery after anterior cervical arthrodesis. Spine J. 2014 Jul 1;14(7):1228-34.
46. Nassr A, Lee JY, Bashir RS et al . Does incorrect level needle localization during anterior cervical discectomy and fusion lead to accelerated disc degeneration? Spine (Phila Pa 1976). 2009 Jan 15;34(2):189-92.
47. Xiong W, Zhou J, Sun C et al. 0.5- to 1-Fold Intervertebral Distraction Is a Protective Factor for Adjacent Segment Degeneration in Single-level Anterior Cervical Discectomy and Fusion. Spine (Phila Pa 1976). 2020 Jan 15;45(2):96-102.
48. Lee YS, Kim YB, Park SW. Risk factors for postoperative subsidence of single-level anterior cervical discectomy and fusion: the significance of the preoperative cervical alignment. Spine (Phila Pa 1976). 2014 Jul 15;39(16):1280-7.
49. Barsa P, Suchomel P. Factors affecting sagittal malalignment due to cage subsidence in standalone cage assisted anterior cervical fusion. Eur Spine J. 2007 Sep;16(9):1395-400.
50. Bartels RH, Donk RD, Feuth T. Subsidence of stand-alone cervical carbon fiber cages. Neurosurgery 2006;58:502–8 [discussion 502–8].
51. Wu WJ, Jiang LS, Liang Y, et al. Cage subsidence does not, but cervical lordosis improvement does affect the long-term results of anterior cervical fusion with stand-alone cage for degenerative cervical disc disease: a retrospective study. Eur Spine J 2012;21:1374–82
52. Lee YS, Kim YB, Park SW. Risk factors for post-operative subsidence of single level anterior cervical discectomy and fusion: the significance of the preoperative cervical alignment. Spine (Phila Pa 1976) 2014;39:1280–7
53. Lim TH, Kwon H, Jeon CH et al. Effect of endplate conditions and bone mineral density on the compressive strength of the graft-endplate interface in anterior cervical spine fusion. Spine (Phila Pa 1976) 2001;26:951–6)
54. Kao TH, Wu CH, Chou YC, et al. Risk factors for subsidence in anterior cervical fusion with stand-alone polyetheretherketone (PEEK) cages: a review of 82 cases and 182 levels. Arch Orthop Trauma Surg 2014;134:1343–51
55. Truumees E, Demetropoulos CK, Yang KH et al . Effects of disc height and distractive forces on graft compression in an anterior cervical discectomy model. Spine (Phila Pa 1976). 2002 Nov 15;27(22):2441-5.
56. Igarashi H, Hoshino M, Omori K et al . Factors Influencing Interbody Cage Subsidence Following Anterior Cervical Discectomy and Fusion. Clin Spine Surg 2019;32(7):297-302.
57. Heary RF, Parvathreddy N, Sampath S et al. Elastic modulus in the selection of interbody implants. J Spine Surg. 2017 Jun;3(2):163-167.
58. Wu WJ, Jiang LS, Liang Y et al. Cage subsidence does not, but cervical lordosis improvement does affect the long-term results of anterior cervical fusion with stand-alone cage for degenerative cervical disc disease: a retrospective study. Eur Spine J. 2012 Jul;21(7):1374-82.
59. Katsuura A, Hukuda S, Saruhashi Y et al . Kyphotic malalignment after anterior cervical fusion is one of the factors promoting the degenerative process in adjacent intervertebral levels. Eur Spine J. 2001 Aug;10(4):320-4.
60. Liang G, Liang C, Zheng X et al . Sagittal Alignment Outcomes in Lordotic Cervical Spine: Does Three-Level Anterior Cervical Discectomy and Fusion Outperform

Laminoplasty? Spine (Phila Pa 1976). 2019 Aug 1;44(15):E882-E888.

61. Park Y, Maeda T, Cho W et al . Comparison of anterior cervical fusion after two-level discectomy or single-level corpectomy: sagittal alignment, cervical lordosis, graft collapse, and adjacent-level ossification. Spine J. 2010 Mar;10(3):1.

Romanian Neurosurgery (2022) XXXVI (1): pp. 84-87
DOI: 10.33962/roneuro-2022-014
www.journals.lapub.co.uk/index.php/roneurosurgery

Osteochondroma of axis vertebra treated with *en bloc* resection and arthrodesis. A case report and review of literature

Deepak Kumar Singh, Diwakar Shankar, Vipin Chand, Mohammad Kaif

Dr. Ram Manohar Lohia Institute of Medical Sciences, Lucknow, INDIA

ABSTRACT

Osteochondromas are capped benign bony neoplasm that forms on the outer surface of the bone. They are most commonly found in appendicular skeletal but rare in the spine. The cervical spine is commonly involved in spinal osteochondromas. Here we discuss a case of a 24-year-old male with osteochondroma arising from the posterior arch of C2 (axis) vertebra presenting with myelopathy. Most of the cases are asymptomatic needing only observation. Surgery is needed in case of progressive neurologic deficit. It is recommended to do an en bloc resection of the lesion along with cartilage cap to prevent recurrence and spinal stabilization with arthrodesis to avoid postoperative kyphosis.

Keywords
osteochondroma,
C2 osteochondroma,
axis osteochondroma,
spinal tumour,
posterior arch
osteochondroma

Corresponding author:
Diwakar Shankar

Dr. Ram Manohar Lohia Institute of Medical Sciences, Lucknow, India

dev123diwakar@gmail.com

Scan to access the online version

INTRODUCTION

Osteochondromas are the most common benign primary tumor of bone constituting 20-50% of all benign bone tumors and 10-15% of all bone tumors [5]. According to World Health Organization (WHO), osteochondromas are capped benign bony neoplasm that forms on the outer surface of the bone. Tumor originates from the ends of the bone near the growth plate where metaplastic cartilage undergoes endochondral ossification with subsequent proliferation. It is most commonly found in the appendicular skeleton but rare in the spine [6]. More than 50% of spinal osteochondromas are found in the cervical region [4]. Here we discuss a case of a 24-year-old male with osteochondroma arising from the posterior arch of axis vertebra presenting with myelopathy.

CASE REPORT

A 24-year-old male patient presented with complaints of progressively increasing difficulty in walking with weakness in the right upper and lower limbs for the last year. There was no history of trauma. There was no family history of MHE. His neurological examination revealed weakness (Medical Research Council, MRC muscle power grading - 4/5) of the right upper and lower limbs. Deep tendon reflexes across major

joints were brisk. Bilateral plantars reflex were extensor. Bilateral Hoffmann signs were positive. No involvement of bladder and bowel was seen. Sensory modalities were intact. The breath-holding time was 42s. The modified Japanese orthopedic association (mJOAS) score was 16/18.

Plain radiographs and computer tomography (CT) cervical spine revealed lobulated circumscribed bony growth along inner margin of right lamina of the axis (C2) vertebra with extension anteriorly into the spinal canal and producing marked canal stenosis (Figure 1). MRI cervical spine showed a large extradural mass lesion arising from axis vertebral lamina causing major compression at C2 vertebral level with myelopathic changes in the cord which was co-related to the clinical complaints and examination of the patient (Figure 2).

The patient was taken for surgery. Preoperative positioning was as per standard guidelines. Fluoroscopic guidance was used for surgery. Intraoperative en bloc resection of the lesion along with bilateral C2 vertebral lamina was done (Figure 3). Bilateral C1 and C3 vertebra lateral masses were fixed with titanium screws and rods system. Onlay autologous bone grafts were placed between C1 and C3 lamina. The postoperative CT cervical spine showed complete removal of the lesion with an increased effective canal diameter at the level of the C2 vertebral body (Figure 4). Pathologic examination was consistent with osteochondroma. The patient had significant improvement in his symptoms and was discharged with a hard cervical collar for three months. In six months, follow-up patient was improved.

Figure 1. Preoperative computer tomography (CT) images with the 3D reconstruction of the cervical spine showing pedunculated circumscribed bony growth along the inner margin of the right lamina of axis (C2) vertebra with extension anteriorly into the spinal canal and producing marked canal stenosis.

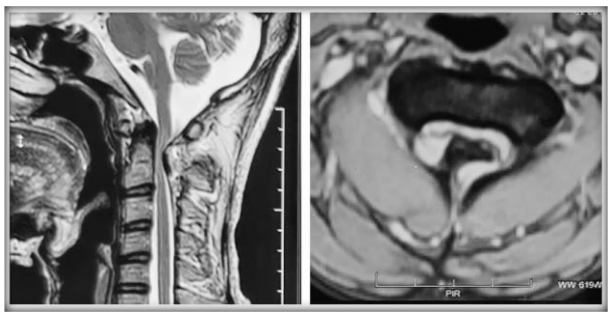


Figure 2. Preoperative magnetic resonance imaging (MRI) of the cervical spine showed a large extradural mass lesion arising from the right C2 vertebral lamina causing major compression at C2 vertebral level with myelopathic changes in the cord.

Figure 3. Intraoperative image showing en bloc resection specimen of the osteochondroma along with C2 (Axis) vertebral lamina.

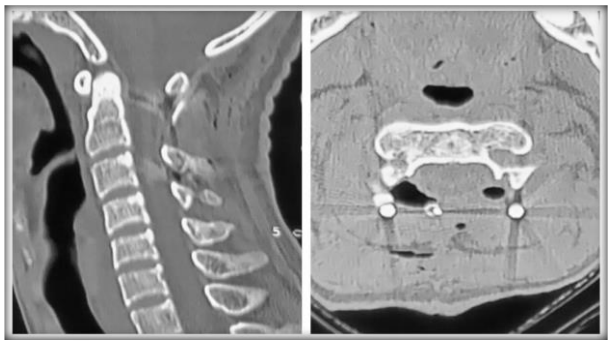


Figure 4. The post-operative computer tomography (CT) of the cervical spine showed complete removal of the lesion with an

increased effective canal diameter at the level of the C2 vertebral body.

Discussion

Osteochondromas are the most common benign primary tumor of bone constituting 20-50% of all benign bone tumors and 10-15% of all bone tumors [5]. It is twice more common in females. The usual age of presentation is 20-30 years. According to World Health Organization (WHO), osteochondromas are capped benign bony neoplasm that forms on the outer surface of the bone. It arises from epiphyseal growth plate where microtrauma can lead to fibroblastic proliferation and the formation of new bone. Osteochondromas can present in two forms, Solitary Exostosis (SE) and Multiple Hereditary Exostosis (MHE). Solitary Exostosis is the sporadic form of the disease and constitutes 85% whereas MHE occurs as Autosomal Dominant syndrome which constitutes 15% of all osteochondromas. Osteochondromas can be sessile or pedunculated. Our patient had a solitary pedunculated lesion over the right lamina of the axis vertebra.

It is most commonly found in the appendicular skeleton including femur, proximal tibia and humerus, and pelvis but rare in the spine[1]. Spinal osteochondromas constitute 1-9% of all cases[4]. More than 50% of spinal osteochondromas are found in the cervical region[4]. This is due to increased mobility leading to the displacement of cartilage and resulting exostosis. Osteochondromas most commonly originate from the posterior element where ossification centers are present. The Axis vertebra is the most common site of osteochondroma in the spine as was in our case followed by the C3 and C6 vertebra [2].

Most of the cervical osteochondromas are asymptomatic as they commonly grow outwards away from the spinal cord. Inward growth causes myelopathy and radiculopathy which is seen in 0.5-1% cases[1]. Other symptoms include pain, weakness, numbness, neck mass, etc. As osteochondroma grows slowly, the patient may develop progressive symptoms. Sometimes it may present early after a trauma leading to compression of the spinal cord.

Plain skiagram and CT scan can be used for establishing the diagnosis. CT is the imaging modality of choice[9]. On CT scan there is a direct continuity of lesion with the cortical and medullary cavity of the underlying bone. Magnetic resonance imaging (MRI) is useful in assessing the degree of spinal cord compression as well as malignant transformation.

The most common treatment modality for asymptomatic cases is observation. Symptomatic patients with pain or progressive neurological deficit require surgery. Also, surgical excision may be done to establish the diagnosis[3]. Previously surgical decompression without fusion was popular but it was associated with postoperative kyphotic deformity, cervical instability, late neurological deficit. Hence, a shift towards en bloc resection with arthrodesis has been seen in recent times. Complete surgical excision of the cartilaginous cap is critical to prevent recurrence[4]. Sclubba et al in their study found that the recurrence rate increased from 11% after en bloc resection to 33% after intralesional resection. There is no role of neoadjuvant or adjuvant radiotherapy or chemotherapy.

Recurrence is seen in 2-11% of postoperative cases[4,8]. Malignant transformation, mostly into chondrosarcoma, is seen in 1% of SE and 3-15% of MHE cases. Features of malignant transformation are rapid growth, growth after skeletal maturity, cartilage cap thickness >3mm, recurrence after complete resection[7]. In our case, complete resection with arthrodesis was done which is found to be more effective than resection alone.

Conclusion

Osteochondromas are the most common benign primary tumor of bone. Most of them are found in the appendicular skeletal but rare in the spine. C2 vertebra is the most commonly affected vertebra. Most of the cases are asymptomatic needing only observation. Surgery is needed in case of progressive neurologic deficit. It is recommended to do an en bloc resection of the lesion along with cartilage cap to prevent recurrence and spinal stabilization with arthrodesis to avoid postoperative kyphosis.

References

1. Brastianos P, Pradilla G, McCarthy E, Gokaslan Z. Solitary thoracic osteochondroma case report and review of the literature. Neurosurgery 2005; 56: E1379.
2. Chatzidakis E, Lypiridis S, Kazdaglis G, Chatzikonstadinou K, Papatheodorou G. A rare case of solitary osteochondroma of the dens of the C2 vertebra. Acta Neurochir (Wien) 2007;149(6):637–8.

3. Gul rkanlar D, Acıduman A, Gul naydın A, Kocak H, Clelik N. Solitary intraspinal lumbar vertebral osteochondroma: a case report. J Clin Neurosci 2004; 11:911–913.
4. Lotfinia I, Vahedi A, Stefanaki K, Tubbs RS, Vahedi P. Cervical osteochondroma with neurological symptoms: literature review and a case report. Spinal Cord Ser Cases. 2017;3:16038.
5. Murphey M, Choi J, Mark J, Kransdorf M, Flemming D, Gannon F. Imaging of osteochondroma: variants and complications with radiologic pathologic correlation. RadioGraphics 2000; 20: 1407–1434.
6. Quirini G, Meyer J, Herman M, Russell E. Osteochondroma of the thoracic spine: an unusual cause of spinal cord compression. AJNR 1996; 17: 961–964.
7. Srikantha U, Bhagavatula ID, Satyanarayana S, Somanna S, Chandramouli BA. Spinal osteochondroma: spectrum of a rare disease. J Neurosurg Spine. 2008;8:561–6.
8. Veeravagu A, Li A, Shuer LM, Desai AM. Cervical Osteochondroma Causing Myelopathy in Adults: Management Considerations and Literature Review. World Neurosurg. 2017;97,752.e755-752.e753.
9. Victory F, Ali K. Solitary, asymptomatic, posterior, vertebral, intracanal, cervical spine osteochondroma. Radiology Case Reports. 2011;6:572.

Romanian Neurosurgery (2022) XXXVI (1): pp. 88-91
DOI: 10.33962/roneuro-2022-015
www.journals.lapub.co.uk/index.php/roneurosurgery

A clinico-radiological study of diagnostic accuracy of special clinical tests in diagnosing compressive myelopathy in patients of early cervical spondylitis symptoms along with its radiological correlation

Anshul Galav

Department Of Neurosurgery. St. John's Medical College Hospital, Bengaluru. Kota Medical College, Kota, INDIA

Keywords
myelopathy, Wartenbergs, spondylitis, Hoffmann, tromner. radiculopathy, interforaminal

Corresponding author:
Anshul Galav

St. John's Medical College Hospital, Bengaluru,
India

dranshul33@gmail.com

ABSTRACT

Background. Cervical spine myelopathy occur due to compression of the spinal cord is present is quite common with a prevalence of around 90. Diagnosis of in the early stages of the condition, the patient may be symptomatic as neck pain, myelopathy or radiculopathy signs clinical examination findings, correlated by MRI findings.

Aims and objectives. To study the clinic-radiological correlation between these clinical signs and radiological imaging in patients with early cervical spondylotic symptoms.

Methods and measures. A minimum of 100 cases shall be considered in this study. All the patients of either sex above the age of 12 years with early cervical spondylotic symptoms of SJMCH during the study period will be considered in this study Thorough clinical history including past medical, treatment and personal history is taken. The following clinical signs are assessed in them: clonus, Hoffman's, Trömner, Wartenberg's sign, Deep tendon reflexes, Plantar reflexes The results of these tests are tabulated. MRI cervical spine is performed and cervical myelopathy is graded. MRI findings are correlated with the clinical signs and thus sensitivity, specificity and accuracy of each of these tests is calculated.

Results. Four clinical diagnostic tests were found to have been quite reliable. None of the tests was self-diagnostic. Among various diagnostic tests Babinski, clonus sign has the highest specificity but low sensitivity.

Conclusion. This study denotes those 6 tests used to diagnose myelopathy are interdependent, none is fully diagnostic.

Scan to access the online version

INTRODUCTION

Degenerative cervical spondolytic myelopathy (CSM) is a progressive disease of the spinal cord. The clinical signs, symptoms and radiology play a key role in diagnosis.1 In the early stages diagnosis is difficult due to subtle signs . various clinical test considered for diagnostic purpose.MRI cervical spine is considered the best method for diagnosis

of cervical stenosis, cord compression, or myelomalacia elements .Spondylosis is common ageing process seen between age of 25 to old age . Most patients are asymptomatic most common symtoms are : neck pain ,cervical radiculopathy, and cervical myelopathy.

Neck pain may be due to degenerative disc or facet changes. Cervical radiculopathy can be acute, subacute, or chronic. Radiculopathy may be due to disc or osteophyte which causes motor or sensory deficits2 Cervical myelopathy due to long tracts involvement due to various static, dynamic factors and ischemic changes. early subtle signs like hyperreflexia, Babinski's sign, Hoffman's reflex,Tromner's sign and/or clonus . other characteristic signs and symptoms include weakness of the hands, hand parathesia,gait disturbances ,bowel and bladder involvement. Signs of LMN at site of lesion and UMN at below level.

Clinical tests

Various clinical tests have been used to diagnose cervical myelopathy – hyperreflexia, Hoffman's, tromners, the Babinski sign, wartenbergs sign, clonus.3 Controversy is there regarding reliability of these tests. Sensitivity and specificity of these tests depends on various factors like inter observer variation ,stage of disease,age of patient,associated comorbidities .Positivity of these tests in normal people not known.Combination of these tests are more reliable then single test.

Hoffmann's sign 4 - stabilise proximal ip joint followed by flicking nail with thumb positive response is flexion of fingers and thumb.

Deep tendon reflex tests - In Biceps tendon testing, the patient is in sitting position ,examiner strikes bicep tendon on his thumb normal response is flexion of elbow.

Babinski sign 5 -patient in sitting position ,strike foot from lateral to medial ,positive response is fanning of fingers and dorsiflexion of thumb.

Tromner sign – on flicking volar apect of middle finger flexion of thumb and index finger.

Wartenberg sign 6–involuntary abduction of little finger when hands r outstretched due to unopposed Action of extensor muscles digiti minimi,digitorum communis.

Clonus7 –on rapid passive dorsiflexion of foot, repititive foot movement due to stretch on Achilles tendon.

Radiological studies

The radiological diagnostic workup often includes static or dynamic plain cervical x-rays, magnetic resonance imaging (MRI) [8].

Magnetic Resonance Imaging (MRI)

MR is useful for evaluating the spinal canal diameter, spinal cord, intervertebral discs,and vertebral ligaments Signal changes on T2-weighted MRI scans at the level of spinal compression are often increased in patients with cervical spondylotic myelopathy. This represents edema, inflammation, ischemia, myelomalacia, or gliosis .9

In DCM, the following are 3 key characteristics that indicate the severity of disc degeneration: loss of T2WI hyperintensity of the nucleus pulposus and resulting loss of delineation between the nucleus pulposus and annulus fibrosus on T2WI, narrowing of the IVD space, and migration of disc material into the spinal canal10.

Methods

This is a descriptive prospective study conducted department of neurosurgery at SJMCH bengaluru and KMC, Kota

Study period: 2018 to 2020

Study Population/Sample Size/
Inclusion/Exclusion Criteria

Minimum of 100 cases shall be considered in this study.All the patients of either sex above the age of 12 years with early cervical spondolytic symptoms of during the study period will be considered in this study. Children less than 12 yrs and other non spondylotic cause of myelopathy.

Procedure

Patients presenting with symptoms of neck pain, radiating pain to upper limbs, paraesthesia, weakness & stiffness in the limbs and fulfilling the inclusion criteria are included in this study.Thorough clinical history including past medical, treatment and personal history is taken.The following clinical signs are assessed in them : clonus ,Hoffman's, Trömner ,Wartenberg's sign, Deep tendon reflexes, Plantar reflexes.

The results of these tests are tabulated.MRI cervical spine is performed and extent of cervical myelopathy changes are graded.MRI findings are

correlated with the clinical signs and thus sensitivity, specificity, accuracy of each of these tests is calculated .The following parameters will be assessed -Patients age and sex, Occupation, Complete Symptomatology ,Co morbidities like HTN,DM, thyroid disorder, RA, AS etc MRI cervical spine and assessing for degree of cord compression Correlating MRI findings with the clinical signs.

Statical Evaluation

As this is a descriptive study, all the mentioned data will be collected and analysed using the parameters described above. Specificity and sensitivity of the clinical tests analysed through the collected data.

Results

100 Patient studied from 2018 to2020, who satisfied inclusion criteria conformed by mri. Mri taken on basis of presence of clinical signs. Among total patients majority 55 females and 45males,with 35 patients associated with various comorbidities . patient included having symptoms of radiculopathy, myelopathy, and cervical strains. 73%admitted with complains of neck pain ,64%admitted with diagnosis hand numbness and gripping weakness and 45 %admitted with gait disturbances.

Four tests are highly diagnostic : Hoffmann's sign , deep tendon reflexes ,tromner and clonus sign. Other 2 wartenbergs and Babinski sign have moderate diagnostic capacity. Most frequent level affected in mri are C5-C6 and C6-C7 levels. Combining clinical examination tests provided significant improvements in the diagnostic accuracy of the clinical tests. Patient associated with various comorbidities has higher positive value of sensitivity and specificity of clinical tests.

Age	Gender	Presence Of Comorbidities {35 Patients}
30 TO 80 YRS	Male =46	Male =15
Mean Age 50	Female =54	Female=20

Neck pain	73
Loss of hand dexterity and medial numbness	64
Clumsiness while walking and gait disturbances	45

Clinical test	Sensitivity	Specificity
Hoffmann Test	43	76
Tromners Sign	45	77
Deep Tendon Reflex	43	70
Babinski Reflex	32	93
Clonus	15	96
Wartenberg Sign	18	78

Discussion

There is high level of agreement for these clinical tests in two examiners .Among these 4 tests highly substantiates diagnosis and other 2 with moderate efficacy. Mri is used to confirm diagnosis,presence of myelomalcia,cord signal changes are highly diagnostic. Tromner sign more sensitive to Hoffman as Hoffman elicited by nipping of nail having no nerve endings while tromner elicited by tapping volar aspect of skin at terminal phalynx of middle finger.[11] As more nerve endings present at volar aspect .most widely accepted explanation for clonus is that it is hyperactive tendon reflexes caused by self excitation. it is caused by interruption of UMN fibres, having low sensitivity but high specificity.

Babinski sign having low sensitivity but high specificity indicates high chances of correctly diagnose cervical myelopathy.[Wartenberg] sign also have low sensitivity value but good specificity.[12]

Conclusion

Our various clinical test shows moderate reliability to diagnose cervical myelopathy. This means these tests may be false negative in presence of myelopathy.There accuracy may be increased by using multiple tests for diagnosis.

Limitations

There are chances of interobserver variability of diagnosing clinical sign ,some interpretations may be wrong .There are chances of variability of signs according to age ,some signs may be more ominous with old age. There is variability in timing of mri in different patients with different symptom profile.

References

1. Batzdorf U, Flannigan BD. Surgical decompressive procedures for cervical spondylotic myelopathy. A study using magnetic resonance imaging. Spine. 1991;16:123-127.
2. Bednarik J, Kadanka Z, Dusek L, et al. Presymptomatic spondylotic cervical cord compression. Spine. 2004;29:2260-2269.
3. Berger JR, Fannin M. The "bedsheet" Babinski. South Med J. 2002;95:1178-1179.
4. Bossuyt PM, Reitsma JB, Bruns DE, et al. Towards complete and accurate reporting of studies of diagnostic accuracy: The STARD Initiative. Ann Intern Med. 2003;138:40-44.
5. Braga-Baiak A, Shah A, Pietrobon R, Braga L, Neto AC, Cook C. Intra- and inter-observer reliability of MRI examination of intervertebral disc abnormalities in patients with cervical myelopathy. Eur J Radiol. 2008;65:91-98. http://dx.doi. org/10.1016/j.ejrad.2007. 04.014.
6. Brennan PF, Hays BJ. The kappa statistic for establishing interrater reliability in the secondary analysis of qualitative clinical data. Res Nurs Health. 1992;15:153-158.
7. Browder DA, Erhard RE, Piva SR. Intermittent cervical traction and thoracic manipulation for management of mild cervical compressive myelopathy attributed to cervical herniated disc: a case series. J Orthop Sports Phys Ther. 2004;34:701-712.
8. Estanol BV, Marin OS. Mechanism of the inverted supinator reflex. A clinical and neurophysiological study. J Neurol Neurosurg Psychiatry. 1976;39:905-908.
9. Fujiwara K, Yonenobu K, Ebara S, Yamashita K, Ono K. The prognosis of surgery for cervical compression myelopathy. An analysis of the factors involved. J Bone Joint Surg Br. 1989;71:393-398.
10. Fukushima T, Ikata T, Taoka Y, Takata S. Magnetic resonance imaging study on spinal cord plasticity in patients with cervical compression myelopathy. Spine. 1991;16:S534-538.
11. Ghosh D, Pradhan S. "Extensor toe sign" by various methods in spastic children with cerebral palsy. J Child Neurol. 1998;13:216-220.
12. Glaser JA, Cure JK, Bailey KL, Morrow DL. Cervical spinal cord compression and the Hoffmann sign. Iowa Orthop J. 2001;21:49-52.

Romanian Neurosurgery (2022) XXXVI (1): pp. 92-97
DOI: 10.33962/roneuro-2022-016
www.journals.lapub.co.uk/index.php/roneurosurgery

Osmotherapy in patients with severe brain trauma: which agents should we take into account?

Randy Reina-Rivero[1], Gustavo Gaitán-Herrera[1], Aixa Eliana García-García[2], Amalia Molina-Trujillo[3], Manuel Alejandro Jaramillo-Acosta[4], Juliana Paniagua-Aguirre[5], Alfonso Barón-Sánchez[6], Antonio Luis Castro-Castro[7], Mohamed Argüello-Manrique[8], Michael Gregorio Ortega-Sierra[9]

[1] School of Medicine, Universidad de Cartagena, Cartagena, COLOMBIA
[2] School of Medicine, Universidad de Santander, Bucaramanga, COLOMBIA
[3] School of Medicine, Universidad de Ciencias Aplicadas y Ambientales, Bogotá, COLOMBIA
[4] School of Medicine, Universidad del Quindío, Armenia, COLOMBIA
[5] School of Medicine, Universidad de Caldas, Manizales, COLOMBIA
[6] School of Medicine, Universidad de Boyacá, Tunja, COLOMBIA
[7] Centro Médico Especializado CEMES, Saravena, COLOMBIA
[8] School of Medicine, Universidad Juan N Corpas, Bogotá, COLOMBIA
[9] Medical and Surgical Research Centre, Future Surgeons Chapter, Bogotá, COLOMBIA

Keywords
osmotherapy,
traumatic brain injury,
intracranial pressure,
mannitol,
sodium lactate,
hypertonic saline solution

Corresponding author:
Michael Gregorio Ortega-Sierra

Medical and Surgical Research Centre, Future Surgeons Chapter, Bogotá, Colombia

mortegas2021@gmail.com

ABSTRACT

Traumatic brain injury is still a major cause of mortality and morbidity in the world and is considered a public health problem that needs to be well attended to. Cerebral oedema due to brain injury compromises the delivery of essential nutrients and alters normal intracranial pressure, whose increase has been shown to be strongly associated with poor neurological outcomes and mortality for patients with head trauma. Intravenous fluids are a fundamental component of trauma care and fluid management influences patient outcomes. Thanks to advances in the research of osmotic agents, mortality has been reduced and there has been greater control in intracranial pressure. The osmotic agents most used for the control of intracranial pressure in patients who have suffered severe brain trauma are mannitol and hypertonic saline. However, in recent years have been studying the benefits that sodium lactate can generate in these cases. It has been found that sodium lactate has generated a greater decrease in intracranial pressure values and lower mortality rates with respect to mannitol and hypertonic saline. This still has been disputed as an application of mannitol, hypertonic saline or sodium lactate for treating patients who have suffered some head trauma. This review aims to show the advantages, disadvantages and recommendations of the different hyperosmolar solutions mentioned previously based on current evidence.

Scan to access the online version

Introduction

Severe traumatic brain injury (TBI) initially causes an injury to the brain by an impact as force propagates towards the cranial cavity, resulting in a stretching and loosening of neural and vascular structures. These direct injuries into the brain parenchyma or blood vessel, prompt inflammation or edema which can be accompanied by bleeding; all these factors ultimately result in an increase in intracranial pressure (ICP) (1,2).

Fluid resuscitation in patients with severe head trauma is vital because of the need to avoid hypotension and secondary neurological injury (3). The increased ICP can generate several consequences. On one hand, as the ICP increases, the cerebral perfusion pressure (CPP) (initially conserved by regulatory mechanisms) when stopping the self-regulating capacity, the CPP will decrease, which implies a risk of secondary hypoxic brain injury. Moreover, the brain can herniate between intracranial compartments and can also lead to secondary brain injury compression and territorial arterial ischemia or by direct compression of the brainstem, becoming in a very mortal issue (1). The main objective of osmotherapy is to preserve or restore the physiology and minimize secondary brain damage, through conservation of CPP and decreased ICP. Moreover, the brain tissue tension of oxygen (PbtO2) has emerged adds as an additional therapeutic goal in the treatment of these patients (3-5).

Osmotherapy has limited efficacy, so prophylactic use should be avoided and must be carefully removed after the maximum period of edema (6). In addition, it is important to pay attention to signs of rebound edema (7). The major hyperosmolar solutions used in patients with traumatic brain injury include hypertonic saline, mannitol and sodium lactate (2). In view of the above, the objective of this review is to synthesize evidence on the use of osmotherapy in the management of patients with traumatic brain injury and related aspects.

Methods

A bibliographic search was carried out in the PubMed and Science Direct databases and in the Google Scholar search engine using the following terms: "Osmotherapy"; "Traumatic Brain Injury"; "Intracranial Pressure"; "Mannitol"; "Sodium Lactate"; "Hypertonic Saline Solution". Articles in Spanish and English were included, emphasizing in the benefits of different osmotic agents in the treatment of severe brain trauma, regardless of the publication date. A total of 325 articles were identified from original articles, topic reviews, systematic reviews, letters to the editor, case reports, and case series. 41 articles were selected (3 in Spanish and 38 in English) that were adjusted to the objective of the article.

RESULTS

Mannitol

Mannitol is an osmotic agent associated with the treatment of intracranial hypertension (IH), used for clinical purposes in 1962 (3). Currently it is highlighted among its indications decreasing refractory ICP, elevated ICP, oliguria and in some cases of acute renal failure. Concentrations ranging from 5% g/100 ml at 25% g/100 ml at an osmolality between 274 and 1372 mOsm/l. As for the dose, it was observed a significant reduction in ICP and more durable responses to treatment when doses are administered between 0.5 and 1.4 g/kg (8,9). The objective is to maintain osmotherapy normal volemia or mild hypervolemia, and maintain serum osmolality between 300 and 320 mOsm/l, so particular vigilance is required to treatment (3,10,11). Mannitol acts on IH after about 20 minutes to be administered and reaches its maximum effect in the brain 30 minutes after; effect duration ranging from 90 minutes to 6 hours depending on the etiology. By decreasing the blood viscosity and the hematocrit and increase the flow of oxygenated blood brain leads to vasoconstriction of cerebral arterioles, and this in turn leads to a reduction in ICP and increasing the CPP. Broadly speaking, also generates a reduction in systemic vascular resistance (and afterload) combined with a transient increased preload and a slight positive inotropic effect, therefore, it improves cardiac output and ventilation (3,12). However, osmotic diuresis with mannitol can cause intravascular dehydration and hypotension (7). In the brain, the mannitol remains in the vascular fluid compartment (13); after prolonged use, it may cross the blood brain barrier and accumulate in the brain tissue, causing an inverse osmotic change, which increases ICP (rebound phenomenon) (7).

Mannitol is filtered at the glomerulus and reabsorbed in the nephron as an osmotic diuretic, it

is excreted unchanged (3). It can induce acute renal failure (ARF) due to renal vasoconstriction, decreased intravascular volume and hyperosmolarity (4). Usually it produces no permanent injury; then removing the drug this is reversed. Several studies report the lowest total dose of mannitol that can cause ARF is 200 g/day (3). Other side effects include electrolyte disorders (hyponatremia, hypochloremia, hyperkalemia), acidosis, heart failure and pulmonary edema (3,7). Restrict the use of mannitol in patients with signs of trans-tentorial hernia or progressive neurological deterioration causes not attributable to extracranial (14).

Hypertonic saline

Hypertonic saline (HS) was used in clinical practice for the first time in 1926 by Silver, who used a 5% HS to treat Burger's disease. Currently, it is positioned as the most popular osmo-agent for hyperosmolar therapy, this due to the multiple complications associated with the use of mannitol (3,15). Continuous HS can be early to reduce brain swelling and IH (16). It is also used in cases of subarachnoid hemorrhage, stroke and liver failure. It is used as adjunctive therapy with mannitol or as an alternative treatment in those patients who failed prior therapy with mannitol (3,7,17).

It is offered in different concentrations (2%, 3%, 7.5% and 23.4%) and is recommended to administer via a central line and should be used at a concentration > 2%, avoiding the risk of thrombophlebitis and peripheral venous thrombosis (3). It is commonly administered as a bolus of 30 ml saline 23.4% (7) or in addition to continuous infusion therapy, decreasing the ICP over a period <72 hours, but this effect extended not maintained for a while (3). Similar to the action of mannitol, HS causes the liquid passage from the parenchyma into the intravascular space, reduces the rate of cerebrospinal fluid (CSF) production and having a lower diuretic effect causes expansion of the intravascular volume, increasing cerebral blood flow and reducing the ICP at the same time. also it has an anti-inflammatory effect reducing leukocyte adhesion (3,18,19). Continuous and frequent use of hypertonic saline lead to the development of hyperchloremic metabolic acidosis, which can be prevented by using hypertonic Sodium/Acetate (7,20). Other problems include ARF, arrhythmias, hemolysis and acute pulmonary edema (3).

Mannitol vs. hypertonic saline

Mannitol and HS are the two most commonly used hyperosmolar solutions (7). No Class I evidence showing the superiority of one over another in the treatment of cerebral edema and intracranial hypertension of different etiologies in critically ill patients (3,21). In 2012, Mortazavi et al. (22) performed a meta-analysis studying the results of HS for the management and control of IH. This review included 36 articles; of these, 9 showed that HS controlled IH better, compared to mannitol. In 6 articles, a reduction in ICP values was present when HS was administered after the use of mannitol.

The potential advantages of hypertonic saline include intravascular volume expansion, to produce less osmotic diuresis, maintains a more stable patients with brain injury systemic and cerebral hemodynamics, and not only decreases the ICP and maintains the CPP, but also increases PbtO2 (7,23,24). As for intracranial hemorrhage have in the acute phase there is an elevated ICP, suggesting a poor prognosis in these patients. Currently it is not known which of these two agents has better performance in this condition (3,25). There are no strong recommendations on which of the two hyperosmolar agents should be used. Mannitol is most often used as first line therapy for IH caused by cerebral trauma, as second line therapy the HS is used when no response to the first. Several authors report that both agents have a similar effect in equimolar dose, and others affirm that the HS is more efficient and safer than mannitol to decrease ICP in severe traumatic brain injury (3,26).

Although the evidence regarding these solutions and their impact on patients is low, there have been studies that have examined various aspects of these fluids with significant results. Pelletier et al. performed a meta-analysis which determined that when comparing the use of HS with other solutions such as mannitol, Ringer's lactate, hypertonic sodium and 0.9% saline, it was found that there is no difference in mortality and maintenance of the ICP (27).

Sodium lactate

To control the elevated ICP as a result of severe brain trauma, it is necessary to use safe and effective osmotic agents. One of these has been mannitol, which has been for many years, solving lesson (27); however, there have been various adverse effects

attributable to this solution. Within these adverse effects are: hypovolemia, hypotension, renal failure, a transient effect, among others (28). Meanwhile, hypertonic saline emerged showing high efficacy in the management of patients neurocritical ill; some of the benefits that this solution is the easy monitoring of its infusion, its high potency and longer duration of effects, his great role as a stabilizer and systemic hemodynamics intracranial, among others. However, HS can cause negative effects that could limit their use; within these it has generated hypernatremia (independent marker of mortality in critically ill patients), osmotic nephropathy, phlebitis, hypokalemia, rhabdomyolysis, and many more (28,29). Therefore, in recent years it has been emerging hypertonic fluid promising for the treatment of severe IH associated with cerebral trauma, sodium lactate. To understand the mechanism of action sodium lactate, it is important to analyze the pathophysiological mechanism by which brain damage occurs in severe brain trauma. This causes trauma injuries dividing two phases: primary and secondary; said phases depend on the time elapsed since the trauma occurred (29,30). The primary stage is given the exact time when the brain injury is generated, and will be characterized by presenting vascular damage, followed by a bleeding in the brain tissue, generating a mass effect in the brain and subsequent IH. As late consequences, at this stage an imbalance of the neurotransmitter regulation, axonal damage and cell death occurs (30). In the secondary phase will be altered cerebral autoregulation mechanisms; generating power failures, which are to be supplied momentarily by increased glycolysis and oxidative phosphorylation; out of ATP stores, anaerobic metabolism begins until this process is unsatisfactory (30,31).

There is a theory that suggests that astrocytes use the process of glycolysis to lactate production; once said lactate enters neuronal cells, this is transformed into pyruvate by the action of lactate dehydrogenase, and thus involved in oxidative metabolism and energy generating (27,32). Under physiological conditions, lactate contributes 8% of the energy required by the brain; however, in cases where there is brain damage, and decreased levels of glucose, lactate can contribute over 60% (31). For this reason, sodium lactate has been a very promising method in controlling the IH in cases of severe brain trauma, requiring more research that yield firm conclusions about the use of this solution with respect to the other, analyzing their effects beneficial and adverse effects. Sodium lactate acts primarily by decreasing the blood viscosity by increasing plasma volume, resulting in an increased blood flow and brain tissue oxygenation (31). Once increased perfusion of the brain tissue, vasoconstriction occurs reflects which contributes to decrease ICP by limiting blood supply to the brain. Due to its hypertonic properties, sodium lactate causes an increase in the osmotic pressure within the blood vessels and a great extent of osmotic gradient between the intra- and extravascular spaces. As a result, decrease the edema fluid introduced in the intravascular space, helping equally to decrease ICP (32,33).

Sodium lactate vs. mannitol

In 2009, Ichai et al. (34) compared the mannitol and sodium lactate in 34 patients who had suffered traumatic brain injury and had IH; in this study it was determined that sodium lactate compared to mannitol, ICP decreased more ($p = 0.016$), longer ($p = 0.009$) and performed with greater success patients ($p = 0.053$). In 2013, Ichai et al. (35) conducted a double-blind, randomized and controlled 60 patients who received a continuous infusion of 0.5 ml/kg/h for 48 hours sodium lactate or normal saline (control group). The results obtained showed that the sodium lactate reduced IH episodes compared to control group ($p <0.05$); this suggests that sodium lactate can be used as an alternative treatment for preventing post IH traumatic brain injury. Moreover, in patients receiving sodium lactate, better urine output and an optimal fluid balance she was found (4,31).

Within the future perspectives on osmotherapy in patients with TBI, it is necessary to mention that the use of one agent or another will vary according to the availability and level of complexity of the hospital where the patient is located (35-38). Therefore, many more studies are needed to evaluate the outcomes of the use of osmotic agents for the management of IH, according to the context of each country, team of professionals and hospital institutions (39,40). This research should be promoted more strongly in low- and middle-income countries, where there is a high incidence of neurotrauma cases, and where high-tech tools that have been shown to further improve neurosurgical outcomes of neurotrauma are not available (35,41).

Conclusions

Traumatic brain injury is a public health problem worldwide. Management and control of intracranial hypertension back to this type of trauma has been being an aspect in which health professionals need to improve as the years pass. Osmotherapy has been a very important mechanism for achieving this objective using fluids such as mannitol and hypertonic saline, which have had great results for patients; however, there have been multiple adverse effects that affect their health. In search of better methods to treat this condition has been introduced as sodium lactate osmotherapy, which has proved very effective in counteracting the secondary damage generated by the traumatic brain injury. Similarly, it has shown better results regarding the cognitive effects after injury. However, conducting research on which these solutions are compared with respect to the benefits and disadvantages that can generate in patients is necessary.

References

1. Wells AJ, Hutchinson PJA. The management of traumatic brain injury. Surgery. 2018; 1–8.
2. Ramos VY, Bedoya OH, Gómez RMC, Corrales SH, Moscote SLR. Osmotherapy in patients with severe brain trauma. Rev Cuba Med Intensiva y Emergencias. 2018; 17:53–6.
3. Llorente G, Claudia M. Mannitol versus hypertonic saline solution in neuroanaesthesia. Rev Colomb Anestesiolgía. 2015; 43(S-1):29–39.
4. Rossi S, Picetti E, Zoerle T, Carbonara M, Zanier ER, Stocchetti N. Fluid Management in Acute Brain Injury. Curr Neurol Neurosci Rep. 2018; 1–9.
5. Jagt M Van Der. Fluid management of the neurological patient: a concise review. Crit Care. 2016; 20(126):1–11.
6. Diringer M. Treatment of fever in the neurologic intensive care unit with a catheter-based heat exchange system. Neurol Crit Care. 2004; 32(2):559–64.
7. Shah S, Kimberly WT. Today's Approach to Treating Brain Swelling in the Neuro Intensive Care Unit. Semin Neurol. 2016; 36(6):502-507.
8. Sorani MD, Manley GT. Dose-response relationship of mannitol and intracranial pressure: a metaanalysis. J Neurosurg. 2008; 108:80–7.
9. Sorani MD, Morabito D, Rosenthal GUY, Giacomini KM, Manley GT. Characterizing the Dose-Response Relationship between Mannitol and Intracranial Pressure in Traumatic Brain Injury Patients Using a High-Frequency Physiological Data Collection System. J Neurotrauma. 2008; 298:291–8.
10. Gondim F de A, Aiyagari V, Shackleford A, Diringer M. Osmolality not predictive of mannitol-induced acute renal insufficiency. J Neurosurg. 2005; 103:444–7.
11. García-Morales EJ, Cariappa R, Parvin CA, Scott MG, Diringer MN. Osmole gap in neurologic-neurosurgical intensive care unit: Its normal value, calculation, and relationship with mannitol serum concentrations. Crit Care Med. 2004; 32(4):986–91.
12. Rickard AC, Smith JE, Newell P, Bailey A, Kehoe A, Mann C. Salt or sugar for your injured brain? A meta-analysis of randomised controlled trials of mannitol versus hypertonic sodium solutions to manage raised intracranial pressure in traumatic brain injury. Emerg Med J. 2014; 31(8):679-83.
13. Hong Y, Yingying S. Hemodynamic effects of mannitol infusion in patients with acute intracerebral hemorrhage. Acta Cirúrgica Bras. 2013; 28(2):106–11.
14. Carney N, Totten AM, Reilly CO, Ullman JS, Bell MJ, Bratton SL, et al. Guidelines for the Management of Severe Traumatic Brain Injury 4th Edition. Brain Trauma Found. 2016.
15. Mangat HS, Chiu YL, Gerber LM, Alimi M, Ghajar J, Härtl R. Hypertonic saline reduces cumulative and daily intracranial pressure burdens after severe traumatic brain injury. J Neurosurg. 2015; 122(1):202-10.
16. Lazaridis C, Neyens R, Bodle J, Desantis SM. High-Osmolarity Saline in Neurocritical Care: Systematic Review and Meta-Analysis. Crit Care Med. 2013; 41(8):1353–61.
17. Kerwin AJ, Schinco MA, Tepas JJ 3rd, Renfro WH, Vitarbo EA, Muehlberger M. The use of 23.4% hypertonic saline for the management of elevated intracranial pressure in patients with severe traumatic brain injury: a pilot study. J Trauma. 2009; 67(2):277-82.
18. Chen H, Song Z, Dennis JA. Hypertonic saline versus other intracranial pressure-lowering agents for people with acute traumatic brain injury. Cochrane Database Syst Rev. 2020; 1(1):CD010904.
19. Rizoli SB, Rhind SG, Shek PN, Inaba K, Filips D, Tien H, et al. The immunomodulatory effects of hypertonic saline resuscitation in patients sustaining traumatic hemorrhagic shock: a randomized, controlled, double-blinded trial. Ann Surg. 2006; 243(1):47-57.
20. Fatima N, Ayyad A, Shuaib A, Saqqur M. Hypertonic Solutions in Traumatic Brain Injury: A Systematic Review and Meta-Analysis. Asian J Neurosurg. 2019; 14(2):382-391.
21. Hooman K, Navi B, Nakagawa Ka, Hemphill C. Hypertonic saline versus mannitol for the treatment of elevated intracranial pressure: A meta-analysis of randomized clinical trials. Crit Care Med. 2011; 39(3):554–9.
22. Mortazavi M, Romeo A, Deep A, Griessenauer C. Hypertonic saline for treating raised intracranial pressure: literature review with meta-analysis. J Neurosurg. 2012; 116:210–21.
23. Anstey JR, Taccone FS, Udy AA, Citerio G, Duranteau J, Ichai C, et al. Early osmotherapy in severe traumatic brain

injury: An international multicenter study. J Neurotrauma. 2020; 37(1):178–84.
24. Jagannatha AT, Sriganesh K, Devi BI, Rao GSU. An equiosmolar study on early intracranial physiology and long term outcome in severe traumatic brain injury comparing mannitol and hypertonic saline. J Clin Neurosci. 2016; 27:68–73.
25. Huang X, Yang L, Ye J, He S, Wang B. Equimolar doses of hypertonic agents (saline or mannitol) in the treatment of intracranial hypertension after severe traumatic brain injury. Medicine (Baltimore). 2020; 99(38):e22004.
26. Gu J, Huang H, Huang Y, Sun H, Xu H. Hypertonic saline or mannitol for treating elevated intracranial pressure in traumatic brain injury: a meta-analysis of randomized controlled trials. Neurosurg Rev. 2019; 42(2):499-509.
27. Gantner D, Moore EM, Cooper DJ. Intravenous fluids in traumatic brain injury: what's the solution? Curr Opin Crit Care. 2014; 20:385–9.
28. Aramendi I, Biestro WMA. Lactato de sodio 0,5 molar: ¿el agente osmótico que buscamos? Med intensiva. 2015; 40(2):113-117.
29. Alnemari AM, Krafcik BM, Mansour TR, Gaudin D. A comparison of pharmacologic therapeutic agents used for the reduction of intracranial pressure after traumatic brain injury. World Neurosurg. 2017; 106:509–28.
30. Reza M, Zuhro A, Ibrahim A, Hafid A. Role of Hypertonic Sodium Lactate in Traumatic Brain Injury Management. Asian J Neurosurg. 2018; 13:971–5.
31. Betancur-Calderón JM, Veronesi-Zuluaga LA, Casta F. Terapia con lactato sódico hipertónico en trauma cráneo-encefálico: ¿se convertirá en la mejor alternativa de manejo? Rev Colomb Anestesiol. 2017; 5(S-2):51–57.
32. Millet A, Cuisinier A, Bouzat P, Batandier C, Lemasson B, Stupar V, et al. Hypertonic sodium lactate reverses brain oxygenation and metabolism dysfunction after traumatic brain injury. Br J Anaesth. 2018; 9:1–9.
33. Berger-Pelleiter E, Émond M, Lauzier F. Hypertonic saline in severe traumatic brain injury: a systematic review and meta-analysis of randomized controlled trials. Can J Emerg Med Care. 2016; 18(2):112–20.
34. Ichai C, Berthier F, Samat-long C. Sodium lactate versus mannitol in the treatment of intracranial hypertensive episodes in severe traumatic brain-injured patients. Intensive Care Med. 2009; 35:471–9.
35. Ichai C, Leverve XM. Half-molar sodium lactate infusion to prevent intracranial hypertensive episodes in severe traumatic brain injured patients: a randomized controlled trial. Intensive Care Med. 2013; 39:1413–22.
36. Pérez-Fontalvo NM, De Arco-Aragón MA, Jimenez-García JDC, Lozada-Martinez ID. Molecular and computational research in low- and middle-income countries: Development is close at hand. J Taibah Univ Med Sci. 2021; 16(6):948-949.
37. Ortega-Sierra MG, Prado-Grajales V, Martinez-Imbett R, Unás-Perea K, Lozada Martinez ID. Research career in neurosurgery: a challenge for future neurosurgeons. J Neurosurg Sci. 2021. Aug 3.
38. Blanco-Teherán C, Quintana-Pájaro L, Narvaez-Rojas A, Martínez-Pérez R, García-Ballestas E, Moscote Salazar L, et al. Evidence-based medicine in neurosurgery: why and how? J Neurosurg Sci. 2021 Aug 3.
39. Silva-Rued ML, Ramírez-Romero A, Guerra-Maestre LR, Forero-Hollmann ÁM, Lozada-Martínez ID. The need to develop specialized surgical centers: the evidence that surgical diseases cannot wait. Int J Surg. 2021; 92:106036.
40. Ortega-Sierra MG, Durán-Daza RM, Carrera-Patiño SA, Rojas-Nuñez AX, Charry-Caicedo JI, Lozada-Martínez ID. Neuroeducation and neurorehabilitation in the neurosurgical patient: programs to be developed in Latin America and the Caribbean. J Neurosurg Sci. 2021 Jun 10.
41. Lozada-Martínez I, Maiguel-Lapeira J, Torres-Llinás D, Moscote-Salazar L, Rahman MM, Pacheco-Hernández A. Letter: Need and Impact of the Development of Robotic Neurosurgery in Latin America. Neurosurgery. 2021; 88(6):E580-E581.

Romanian Neurosurgery (2022) XXXVI (1): pp. 98-106
DOI: 10.33962/roneuro-2022-017
www.journals.lapub.co.uk/index.php/roneurosurgery

Utilization of anticoagulant and antiplatelet medications among geriatric patients with neurosurgical diseases

Abdulrahman Al-Mirza[1], Omar Al-Taei[1], Hatem Al-Saadi[1], Tariq Al-Saadi[2]

[1] Sultan Qaboos University College of Medicine. Sultanate of Oman-Muscat, Al-Khoudh, OMAN
[2] Department of Neurology and Neurosurgery. Montreal Neurological Institute, Faculty of Medicine, McGill University, QC, CANADA and
Department of Neurosurgery, Khoula Hospital, Muscat, Sultanate of Oman, OMAN

Keywords
anticoagulants, geriatric, elderly, bleeding, trauma, neurosurgery, antiplatelet

Corresponding author:
Tariq Al-Saadi

Faculty of Medicine, McGill University, QC, Canada

t.dhiyab@hotmail.com

Scan to access the online version

ABSTRACT

Aim: To study the utilization patterns of anticoagulant and antiplatelet medications among geriatric patients with neurosurgical conditions in the neurosurgical department at Khoula Hospital, Muscat, Sultanate of Oman.

Introduction: Anticoagulant and antiplatelet presumption is a worry in neurosurgical patients, that suggests a subtle balance between the risk of thromboembolism against the risk of peri- and postoperative haemorrhage. Patients taking those medications were found to have an increased risk of bleeding from traumatic and traumatic events.

Materials and Methods: A retrospective study of geriatric cases admitted to the Neurosurgery Department in Khoula Hospital (KH) as an example of a neurosurgical center in Sultanate of Oman, from January 2016 to 31st December 2019. Patients demographics, diagnosis, length of hospital stay (LOS), Glasgow Coma Scale (GCS), length of ICU admission, and treatment proposed were recorded.

Results: The most common diagnostic category was trauma (35.4%). 16.0 % of the patients were taking anticoagulant medications. Patients with traumatic brain injury (TBI) were found to have a higher rate of using anticoagulant medications (36.6%). There was a significant difference between the LOS, type of intervention, ICU admission, and the usage of anticoagulant and antiplatelet drugs ($p<0.05$). Enoxaparin was the most commonly used anticoagulant agent. 19.6 % of the patients were taking antiplatelet medications. This study was showed that aspirin is the most commonly used antiplatelet agent among different neurosurgical pathologies.

Conclusion: Patients with TBI were found to have a higher rate of using anticoagulant medications. Decisions regarding prescription and resumption of anticoagulants and antiplatelet medications should be taken on a case-by-case basis involves multidisciplinary and holistic approaches.

INTRODUCTION

Anticoagulant presumption is a worry in neurosurgical patient that

suggests a subtle balance among the risk of thromboembolism against the risk of perioperative and postoperative hemorrhage (1). The ageing populace has resulted in an alteration in the demographics of trauma (2). Numerous of these patients are undergoing anticoagulation therapy and are also at high risk of falls and sustaining injury with possibly serious hemorrhagic complications (2). The main risk of systemic anticoagulation is bleeding, including intracranial hemorrhage, which must be well-adjusted against the recommended chronic anticoagulation for at least the duration of active cancer (3). Clinicians often face a patient with a brain tumor who requires anticoagulation for a deep venous thromboembolism (DVT) or pulmonary embolism (PE) or for cardiac reasons, such as atrial fibrillation or peripheral vascular disease. (3). Head injury `embodies one of the most important and recurrent traumatic pathology in the emergency department (4). Among the diverse risk factors, preinjury use of warfarin has received considerable attention in trauma literature. Several studies were undertaken to elucidate the effect of warfarin anticoagulation on the mortality rate and risk of development of intracranial hemorrhage (ICH) in adult patients with minor head injuries (4). The risk of mortality in a patient anti-coagulated on warfarin with an ICH after head injury ranges in the literature from 16-80% (5). The Food and Drug Administration -USA- projected that 2 millions of people each year start using vitamin K antagonist. The use of anticoagulants is predicted to continue growing in the near future (1). Warfarin is a vitamin K antagonist, and works by inhibiting the enzyme vitamin K epoxide reductase. This is essential for calcium binding, allowing the linking to phospholipids surfaces to promote clotting (5).

Platelets are small, irregularly shaped subcellular fragments that are derived from megakaryocytes and play a major role in the maintenance of haemostasis (6). Antiplatelet agents are frequently being used in the management or prevention of cardiovascular disease (7) such as; deep venous thrombosis, atrial fibrillation, pulmonary embolism and coronary artery disease. They are also given postoperatively for prosthetic heart valves or stent placement (8). Patients taking these medications found to have an increased risk of bleeding from traumatic and traumatic events (7). Therapeutic mechanisms of antiplatelet agents include inhibition of platelet aggregation, so normal haemostasis is impaired, which may lead to an increased incidence of ICH and enlargement of hematomas in TBI, potentially increasing mortality and morbidity (9). For instance, patients taking an antiplatelet agent, with ICH occurring in approximately 3.6%–67.3% of patients on antiplatelet therapy and 1.6%–50.5% of patients not on this therapy (7). Also, posttraumatic ICH is associated with a mortality rate of 21% in patients on antiplatelet therapy. (6). Antiplatelet agents are necessary for patients undergoing neuroendovascular procedures, due to it is preventative effect against thromboembolic consequences (9). Aspirin is the most common used drug while undergoing neuroendovascular procedures. Aspirin is typically initiated at least 3–5 days prior to some vascular procedures such as carotid or intracranial stenting (9). Clopidogrel is the other most common antiplatelet drug used while undergoing neuroendovascular procedures. Variation in individual response to clopidogrel has sparked a great deal of research and controversy in the cardiovascular and neuroendovascular areas. The incidence of clopidogrel hyporesponsiveness has varied from 21% to 53.1% in patients undergoing neuroendovascular procedures (9).

This study is conducted to investigate the prevalence of therapeutic antiplatelet and anticoagulant medications among geriatric patients admitted in neurosurgical department at Khoula Hospital, Muscat, Sultanate of Oman, which is the first of its kind in the region, to the best of our knowledge. The health care services in Oman are considered as having one of the best health care systems in the world according to World Health Organization reports (10,11). The Department of Neurosurgery in our hospital is the main neurosurgical center in the country with an average annual admission of 1600 patients (12,13) In this study, we chose a cut of 65 years and older according to the local definition, taking into account the increase in life span throughout the last decades as well as the improvement in the quality of life.

Methods

Study group

This is a retrospective study conducted at Khoula Hospital located in Muscat, Sultanate of Oman. The study was approved by the Research Ethical

Committee at Khoula Hospital/ Ministry of health (PRO122020072). Medical records of 696 patients who are above the age of 65 and admitted to the neurosurgical ward January 2016 to 31st December 2019 were included. The study includes both Omani and non-Omani patients. Patients with the following features are excluded: non elderly patient (below 65 years), non-neurosurgical conditions, outside the study period (from 1st January 2016 to 31th December 2019), Patients with missing or incomplete data.

Data collection

Data was obtained from the health information system included: patient demographics. diagnosis, length of hospital stay (LOS), Glasgow Coma Scale (GCS), length of ICU admission and treatment proposed. Then the information classified into continues and categorized variables and analysed accordingly.

Data analysis

Research database was analysed and processed using the statistical package for the social sciences (SPSS) software (version23). The categorized variables were cross-tabulated using frequency tables. Chi-square test was used to obtain the significance of the association between categorized variables, using a P value of ≤ 0.05 as the cut-off for significance.

Results

The demographic characteristics of the included cases in the present study is represented in table 1. We have total of 669 patients with mean age of 73.3 admitted in the neurosurgical department at KH in Muscat the capital city of Sultanate of Oman in four years 'period (from 2016 to 2019). 2019 accounted for the highest number of admitted patients (30%).61.9% of the study cohort were more than 75-year-old. Male to female ratio was (1.63:1). Most of the patient were having GCS score of 14-15 (72.3%). The most common diagnostic category was trauma, accounted for 35.4% of the study cohort followed by oncology and vascular equally (16.3%).16.0 % of the patients received anticoagulant medications.19.6% of the patients received antiplatelet medications. Most of the patients underwent surgical intervention (73.1%). Majority of the patients stayed in the hospital less than 15 days (77%).

Table 1. Demographic characteristics of the patients

Category	Number of patients (%)
Number of patients admitted each year	
2019	**202 (30.0%)**
2018	**172 (25.7%)**
2017	**154 (23%)**
2016	**141 (21.3%)**
Total	**669**
Age	
>=75	**414 (61.9%)**
< 75	**255 (38.1%)**
Gender	
Female	**255 (38.1%)**
Male	**414 (61.9%)**
GCS on arrival	
15-14	**484 (72.3%)**
13-12	**49 (7.3%)**
11-9	**36 (5.3%)**
<8	**100 (15%)**
Diagnostic category	
Oncology	**109 (16.3%)**
Trauma	**237 (35.4%)**
Vascular	**109 (16.3%)**
Spine and Peripheral nerve diseases	**176 (26.3%)**
Infection and functional	**13 (2%)**
Hydrocephalus	**25 (3.7%)**
Anticoagulant drugs	
Yes	**107 (16.0)**
No	**562 (84.0)**
Type of interventions	
Surgical	**489 (73.1%)**
Observational	**180 (26.9%)**
Length of stay (LOS)	
<= 15 days	**515 (77%)**
>15 days	**154 (23%)**

Utilisation of anticoagulant medications

Figure 1 illustrates the utilization of anticoagulant medications among different diagnoses (131 patients out of 669 patients). Patients with TBI found to have higher rate of using anticoagulant medications (36.6%) followed by patients with spinal diseases and vascular pathologies, respectively (25.9%, 18.3%). There was no significant relationship between the utilization of anticoagulant medications among different neurological diseases investigated

(p=0.920). The association between usage of anticoagulant medications among patients and other variables (type of injury, LOC, type of intervention, age, and ICU admission) is shown in table 2. It demonstrates that there was no significant difference between the utilization of anticoagulant medications the type of injury, as both traumatic and non-traumatic patients were using anticoagulant medications in a similar way (p=0.674). Also, it is showing that there was a significant difference in the LOS and the usage of anticoagulant drugs, in which patients who were not using anticoagulant medications found to stay shorter in the hospital (p<0.05). Additionally, patients who were undergone surgical intervention were using anticoagulant medications much less than patients who were on conservative management (p=0.001). Added to that the significant relationship that found to be between the ICU admission and the utilization of anticoagulant medications, in which majority of the patients who were not on anticoagulant therapy were not admitted to the ICU (p<0.05). Also, there was no association between the usage of anticoagulant medications and the age of patients (above and below 75 years) (p=0.198). Figure 2 illustrates the utilization of anticoagulant agents among different diagnoses. It is showing that enoxaparin was the most commonly used anticoagulant agent among different neurosurgical pathologies in the present study (n=93), followed by warfarin (n=15). Heparin was only used by 3 patients in this study.

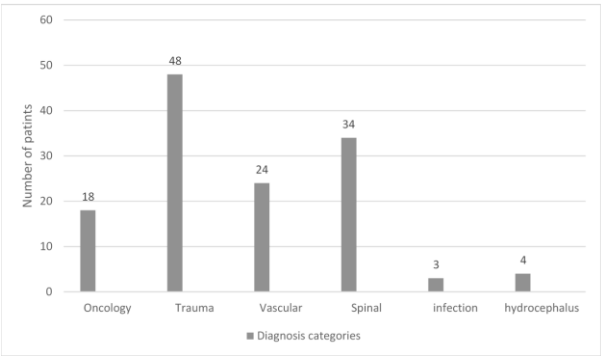


Figure 1. The utilization of anticoagulant medications among different diagnoses.

Utilization of antiplatelet medications

The utilization of antiplatelet medications among different diagnoses (n=107) is illustrated in Figure 3. Patients with TBI found to have higher rate of using antiplatelet medications (33.6%) followed by patients with oncological and vascular pathologies, respectively (26.1%, 21.4%).

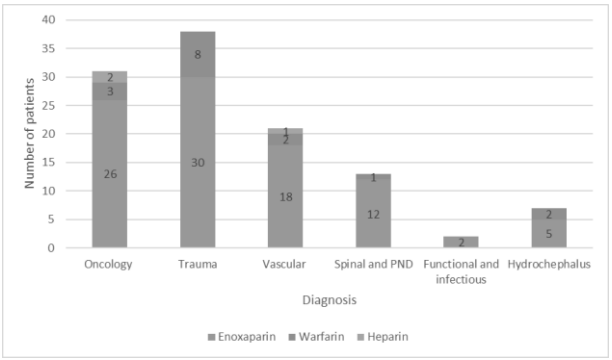


Figure 2. Utilization of anticoagulant agents among different diagnoses.

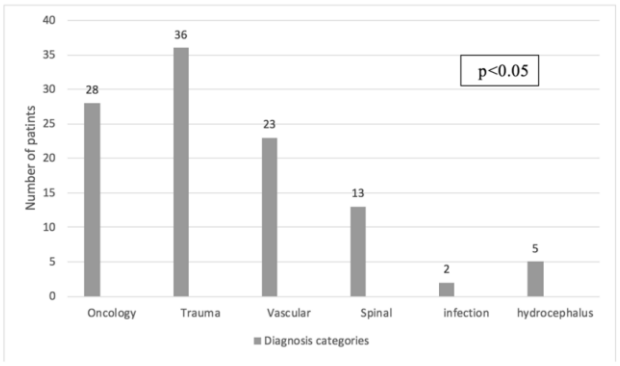


Figure 3. The utilization of antiplatelet medications among different diagnoses.

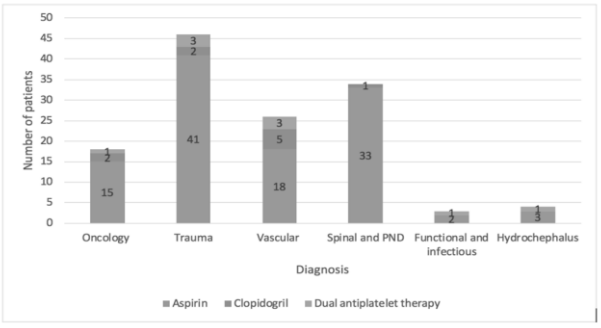


Figure 4. Utilization of antiplatelet agents among different diagnoses

There was a significant difference in the utilization pattern of antiplatelet medications among different neurological diseases investigated (p<0.05). Table 3 is representing the relationship between utilization of antiplatelet agents among patients and other variables. It demonstrates that there was no

significant relationship between the usage of antiplatelet medications and the type of injury (traumatic vs non-traumatic) (p=0.746). Additionally, there was a significant difference in the LOS and the usage of antiplatelet medications (p=0.034). Patients who were undergone surgical intervention were using antiplatelet medications in a much lesser rate compared with patients who were on non-surgical conservative management (p=0.032). Added to that, there was a significant difference between the ICU admission and the utilization of antiplatelet medications (p=0.02). Also, there was no association between the usage of antiplatelet drugs and the age of patients (above and below 75 years) (p=0.906). Figure 4 showed the utilization of antiplatelet agents among different pathologies. Aspirin was the most commonly used agent in the present study (n=112), followed by clopidogrel (n=10). Dual antiplatelet therapy (aspirin with clopidogrel) was only used by 9 patients in this research.

Table 2. The relationship between the usage of anticoagulant medications and type of injury (trauma vs. non trauma), length of stay, type of intervention (surgical vs. observation), age group, and ICU admission.

Category	Type of injury		Length of stay		Intervention		Age		ICU admission	
	Trauma	36	More than 15 days	51	Surgical	92	More than 75 years	56	Admitted to ICU	40
	Non trauma	71	Less than 15 days	56	Observation	15	Less than 75 years	51	Not admitted to ICU	67
P-value	P= 0.674		P<0.005		P=0.001		P= 0.198		P<0.005	

Table 3. The relationship between the usage of antiplatelet medications and type of injury (trauma vs non trauma), length of stay, type of intervention (surgical or observation), age group, and ICU admission.

Category (p-value)	Type of injury		Length of stay		Intervention		Age		ICU admission	
	Trauma	48	More than 15 days	21	Surgical	86	More than 75 years	84	Admitted to ICU	12
	Non trauma	83	Less than 15 days	110	Observation	45	Less than 75 years	47	Not admitted to ICU	119
P-value	P= 0.746		P= 0.034		P= 0.032		P=0.906		P=0.002	

Discussion

Utilization of anticoagulant medications

Anticoagulants are important treatments for numerous medical conditions including DVT, pulmonary embolism, and non-valvular AF. Importantly; The triad of anticoagulation, age above 65 years, and minor head injury was considered lethal by Karni et al. on a retrospective review of 278 patients with minor head injuries and CT-documented ICH (14). Patients with TBI found to have higher rate of using anticoagulant medications followed by patients with spinal diseases and vascular pathologies, respectively. In comparison, Claudia et al reported that 75 patients out of 1410 included in the study was on anticoagulants at the time of TBI (4) An important issue to be raised in elderly patients with higher risk of thromboembolic events, is weighing up the balance between thrombotic risk and falls or bleeding risk which can often be challenging and compounded by the presence of lower number of evidences to guide decisions regarding reinstitution of anticoagulants. (15,16). There was no significant relationship between the utilization of anticoagulant medications among different neurological diseases investigated it the current study (p=0.920). In contrast to our study, Pieracci et al. conducted a study to evaluate the effect of anticoagulants on elderly patients and concluded that patients on warfarin had increased severity of head injury, had more chances of ICH (17). Another study investigated the effect of anticoagulants among patients with glioblastoma stated that anticoagulants was associated with inferior overall survival compared to no use on multivariate analysis (p=0.001) (18). In comparison to this study; another study stated that patients with primary and metastatic brain tumours, can safely use anticoagulants under careful monitoring, except

for patients with untreated tumours with a high rate of intracranial haemorrhage (i.e., metastases from melanoma, choriocarcinoma, thyroid carcinoma, hepatocellular carcinoma, and renal cell carcinoma). In patients with cerebrovascular pathologies such as cerebral aneurysm, it has been showed that oral anticoagulants in safer than parental ones.

Our study ddemonstrates that there was no significant difference between the utilization of anticoagulant medications and the type of injury, as both traumatic and non-traumatic patients were using anticoagulant medications in a similar way. In contrast, another study found that there was an exponential correlation between the intensity of anticoagulation the risk of ICH from traumatic events (19). Additionally, patients who were undergone surgical intervention were using anticoagulant medications much less then patients on conservative management. Kawamata et al addressed post-craniotomy usage of anticoagulants in retrospective review of 27 patients. Seventeen of those patients (63%) underwent craniotomy for evacuation of intracerebral of subdural hematomas. Also, there was no association between the usage of anticoagulant drugs and the age of patients (above and below 75 years) (p=0.198). Another study by Sharma et al reported that Warfarin consumption is independently associated with higher mortality among a cohort of 384 TBI elderly patients (20)

Enoxaparin was the most commonly used anticoagulant agent among different neurosurgical pathologies in the present study followed by warfarin. Another study by Calvin et al found that almost one fifth of elderly patients older than 65 years old admitted to an American level one trauma centre with head injury were on warfarin, reflecting the importance of impact of warfarin on management and outcome of head injuries (21). This can be explained by the co-existence of other comorbidities that requires anticoagulation therapy such as warfarin more than other anticoagulants (21). An observational study on a cohort of 114 patients with severe blunt trauma (severe TBI excluded) showed that use of oral anticoagulants was associated with significantly lower mortality group 8.3% vs. warfarin group 29.5%, $p < 0.015$) (15). Yet; some physicians may be hesitated to prescribe anticoagulants, especially for the very old or frailest patients, as they are perceived to be at higher risk for the consequences of traumatic events (TBI) (20)

Utilization of anticoagulant medications

Patients with TBI found to have higher rate of using antiplatelet medications in our study. In comparison to our study; another study showed that 40.7% who sustained blunt head trauma were taking antiplatelet medications and having consequent acute ICH following the head trauma (2). Posttraumatic ICH was associated with significantly increased mortality rate in patients on antiplatelet therapy when compared to controls in previous studies (9,10). These findings are supported by a study by Major et al who found evidence that posttraumatic ICH is associated with a mortality rate of 21% in patients on antiplatelet therapy (11). The risk for ICH appears to be dose dependent with aspirin, the most studied agent, but exists with other antiplatelet agents as well (12). The utilization of those medications was correlated with increased ICH volume growth at 12 hours, volume of intraventricular haemorrhage increased chance of death at 14 days, and poor outcome at 3 months (13,14,15). Yet, the decision to stop all antiplatelet medication needs to be carefully considered, weighing the size and morbidity of the ICH against the reason the agents were initiated. In another hand, platelet function testing appears advisable prior to neurovascular procedures, particularly in patients with risk factors for variable response to antiplatelet medications (16). Another important issue to be considered is that in all studies the assessment of pre-injury use of antiplatelet agents was based on the patient's history and not on assessment of platelet activity through laboratory examinations, thus, incompliance of patients regarding the intake of prescribed medications as well as ineffectiveness of antiplatelet agents in some will affect the overall effect of antiplatelet medications in the body system (4).

This study also showed that there was a significant relationship between the utilization of antiplatelet medications among different neurological diseases investigated. Correspondingly, a previous study conducted to evaluate the effect of antiplatelet medications on neurosurgical conditions showed that pre-injury antiplatelet agents were associated with three times higher mortality among elderly patients where it was due to a functional rather than quantitative factors (2).

Our study demonstrates that there was no significant difference between the utilization of antiplatelet medications the type of injury. In

comparison to our study, Mina et. al found a statistically significant mortality rate (47%) in those who had TBI and taking aspirin compared with controls (8%). This may be attributed to the severity of TBI or the dose of aspirin consumed by the patients as low aspirin doses showed to has a lower risk of bleeding compared with higher doses (12,17) Also the present study demonstrated that there is a significant difference in the LOS and the usage of antiplatelet drugs, in which patients who were not using antiplatelet medications found to stay shorter in the hospital. In comparable results, Scott et al reported that patients who were using antiplatelet medications were three times more likely to be discharged to long-term inpatient facilities and 14 times higher mortality rate as compared with those not on antiplatelet Additionally, patients who were undergone surgical intervention were using antiplatelet medications much less then patients on conservative management. In contrast to our research; Bachelani et al found that neither history of aspirin intake nor platelet inhibition measured by a specific coagulation tests were associated with an increased risk of incidence or progression of ICH and consequent need to an operative intervention such as craniotomy (18). Added to that the significant relationship that found to be between the ICU admission and the utilization in which majority of the patients who were not on antiplatelet therapy were not admitted to the ICU. In comparison to a study conducted by Sharma et al which stated that antiplatelet therapy with aspirin and clopidogrel did not increase rate of mortality and did not affect length of hospital ICU stay (19). Also, there was no association between the usage of antiplatelet drugs and the age of patients (above and below 75 years). Prior research has shown that the geriatric population suffers from a higher rate of acute ICH. As the brain ages, there is volume loss in the brain parenchyma, making bridging cerebral vein more vulnerable to bleeding along with a significant decline in the elasticity of those veins. Moreover, the increase in aging population due to improvement of health care services in high income developing countries – like sultanate of Oman - has led to increased utilization of antiplatelet medications, likely contributing to the increased incidence of bleeds (2).

Aspirin is the most commonly used antiplatelet agent among different neurosurgical pathologies in the present study followed by clopidogrel. In the same line to our study, a previous study showed that aspirin is the most common utilized antiplatelet drug. This can be attributed to the fact that it is widely available, well tolerated, and has extensive clinical evidence supporting it is use in the cardiovascular protective properties. Yet, currently, the combination of aspirin and clopidogrel is recommended prior to some neuroendovascular surgeries such as stent deployment (4).

Limitations

There were some limiting factors in the present study. It was a retrospective, single-centered study over a period of four-years which limit is our ability to investigate whether there is a prior use of anticoagulants and antiplatelet medications especially in patients with chronic diseases that carry a high thromboembolic risk. Analysis of coagulation profile was not routinely performed, so it is unclear if patients were compliant with their anticoagulant medications. Moreover, the study was conducted in tertiary health care facility so it doesn't include prescription patterns of medications from other health care facilities. Finally, the outcome of using anticoagulant and antiplatelet medications was not illustrated in the present study due to the lack of comprehensive data regarding mortality in the health information system used to collect the data.

Conclusion

Patients with TBI found to have higher rate of using antiplatelet and anticoagulant medications. Aspirin and enoxaparin were the most commonly used antiplatelet and anticoagulant agents among different neurosurgical pathologies in the present study Physiological changes and comorbidities are main contributors to the challenges faced in managing bleeding in geriatric age group which has to be considered essentially. There is a need for further work to explore the balance of thrombotic and antithrombotic factors in elderly patients with neurosurgical diseases and to investigate whether the laboratory measures of thrombotic potential can help identify those at highest risk of thrombosis. Decisions regarding prescription and resumption of anticoagulants and antiplatelet medications should be taken on a case-by-case basis involve a multidisciplinary and holistic approaches. The preventative benefit is of antiplatelet and

anticoagulant therapy from thromboembolic events of stroke or myocardial infarction have to be weighed against the risk of haemorrhage and death in those patients due to decreases ability of their body to with stand high energy traumatic accidents and their increased risk of frequent falls. It's worth mentioning that this study is the first of its kind in the region, to the best of our knowledge.

Abbreviations

DVT: deep venous thromboembolism,
GCS: Glasgow Coma Scale,
ICH: Intracerebral haemorrhage,
ICU: Intensive care unit,
LOS: length of hospital stays,
PE: pulmonary embolism.

Disclosures

Nothing to disclose.

Funding

No funding required

Conflict(s) of Interest

No conflict of interest.

References

1. Prior, Alessandro, Pietro Fiaschi, Corrado Iaccarino, Roberto Stefini, Denise Battaglini, Alberto Balestrino, Pasquale Anania, Enrico Prior, and Gianluigi Zona. 2021. "How Do You Manage ANTICOagulant Therapy in Neurosurgery? The ANTICO Survey of the Italian Society of Neurosurgery (SINCH)." BMC Neurology 21(1):1–14. doi: 10.1186/s12883-021-02126-7.
2. Anon. 2018. "Typhi Strain Ty21a Carrying a Plasmid Encoding Vascular Endothelial Growth Factor Receptor 2 (VEGFR2). The Bacterium Serves as a Vector to Deliver the Plasmid into the Peyer's Patches." (September):2018.
3. Ho, Sandra, Vismay Thakkar, and Jack Jallo. 2013. "Outcomes in Traumatic Brain Injury Patients on Preinjury Anticoagulation and Antiplatelet Agents." JHN Journal 8(1):1–5. doi: 10.29046/jhnj.008.1.002.
4. Alter, Scott M., Benjamin A. Mazer, Joshua J. Solano, Richard D. Shih, Mary J. Hughes, Lisa M. Clayton, Spencer W. Greaves, Nhat Q. Trinh, and Patrick G. Hughes. 2020. "Antiplatelet Therapy Is Associated with a High Rate of Intracranial Hemorrhage in Patients with Head Injuries." Trauma Surgery and Acute Care Open 5(1):1–5. doi: 10.1136/tsaco-2020-000520.
5. Claudia, Casula, Ranalli Claudia, Ognibene Agostino, Magazzini Simone, and Grifoni Stefano. 2011. "Minor Head Injury in Warfarinized Patients: Indicators of Risk for Intracranial Hemorrhage." Journal of Trauma - Injury, Infection and Critical Care 70(4):906–9. doi: 10.1097/TA.0b013e3182031ab7.
6. James, Robert F., Viktoras Palys, Jason R. Lomboy, J. Richard Lamm, and Scott D. Simon. 2013. "The Role of Anticoagulants, Antiplatelet Agents, and Their Reversal Strategies in the Management of Intracerebral Hemorrhage." Neurosurgical Focus 34(5):1–11. doi: 10.3171/2013.2.FOCUS1328.
7. Kim, Keri S., Justin F. Fraser, Stephen Grupke, and Aaron M. Cook. 2018. "Management of Antiplatelet Therapy in Patients Undergoing Neuroendovascular Procedures." Journal of Neurosurgery 129(4):890–905. doi: 10.3171/2017.5.JNS162307.
8. Roth, P., A. Pace, E. Le Rhun, M. Weller, C. Ay, E. Cohen-Jonathan Moyal, M. Coomans, R. Giusti, K. Jordan, R. Nishikawa, F. Winkler, J. T. Hong, R. Ruda, S. Villà, M. J. B. Taphoorn, W. Wick, and M. Preusser. 2021. "Neurological and Vascular Complications of Primary and Secondary Brain Tumours: EANO-ESMO Clinical Practice Guidelines for Prophylaxis, Diagnosis, Treatment and Follow-up †." Annals of Oncology 32(2):171–82. doi: 10.1016/j.annonc.2020.11.003.
9. Beynon, Christopher, Daniel N. Hertle, Andreas W. Unterberg, and Oliver W. Sakowitz. 2012. "Clinical Review: Traumatic Brain Injury in Patients Receiving Antiplatelet Medication." Critical Care 16(4). doi: 10.1186/cc11292.
10. The World Health Report 2000 (Health Systems: Improving Performance). WHO Geneva. Available at: https://www.who.int/whr/2000/en/.
11. Al-Kalbani, H., Al-Saadi, T., Al-Kumzari, A., & Al-Bahrani, H. (2020). Public's perception and satisfaction on the health care system IN Sultanate OF Oman: A cross-sectional study. Annals of the National Academy of Medical Sciences (India), 56(04), 214-219. doi:10.1055/s-0040-1721554
12. G P Mishra, Tariq Al Saadi, Neeraj Salhotra, Samit Biniwale, Mohammad Hadad, Mohammad Hashim, Ali Al Mashani, Qassim Al Hinai and Sharad Samson Rajmani. Brain and Spinal Tumors Incidence Annual Audit 2017 of Dept of Neurosurgery Khoula Hospital Muscat Oman: A Review. American Journal of Medical Case Reports. 2018; 6(7):128-131. doi: 10.12691/AJMCR-6-7-2
13. Al-Saadi T, Al Sharqi A, ALSharqi J, ALBlushi K, ALRasbi A, Al-Farsi M, Al-Ghafri S, Al-Abri S, Al-Saadi H. Leaving Against Medical Advice Among Patients With Brain Tumours in the Middle East: Khoula Hospital Experience. Int Clin Neurosci J [Internet]. 2020Sep.28 [cited 2021Mar.20];7(4):179-84. Available from: https://journals.sbmu.ac.ir/neuroscience/article/view/30700https://www.ncbi.nlm.nih.gov/books/NBK189784/table/appc.t1/
14. Karni A, Holtzman R, Bass T, et al. Traumatic head injury in the anticoagulated elderly patient: a lethal combination. Am Surg. 2001;67: 1098–1100
15. Anon. 2018. "Typhi Strain Ty21a Carrying a Plasmid Encoding Vascular Endothelial Growth Factor Receptor 2

(VEGFR2). The Bacterium Serves as a Vector to Deliver the Plasmid into the Peyer's Patches." (September):2018.
16. View, Point O. F. 2019. "Geriatric Traumatic Brain Injury : An Old Challenge Traumatismo Craneoencefálico En El Anciano : Un Viejo Reto." 43(1):44–46.
17. Chowdhury, Subhankar, and Partha pratim Chakraborty. 2017. "Universal Health Coverage - There Is More to It than Meets the Eye." Journal of Family Medicine and Primary Care 6(2):169–70. doi: 10.4103/jfmpc.jfmpc.
18. Wong, Henna, Nicola Lovett, Nicola Curry, Ku Shah, and Simon J. Stanworth. 2017. "Antithrombotics in Trauma: Management Strategies in the Older Patients." Journal of Blood Medicine 8:165–74. doi: 10.2147/JBM.S125209.
19. Angelozzi, Andrea, Giulia Renda, and Michele Mercuri. 2015. "The Risk of Intracranial Hemorrhage with Anticoagulation in the Elderly - Estimates of Prevalence and Therapeutic Strategies." American College of Cardiology 1–9.
20. Rao LV, Pendurthi UR. Tissue factor-factor VIIa signaling. Arterioscler Thromb Vasc Biol. 2005;25(1):47-56. doi:10.1161/01.ATV.0000151624.45775.13
21. Mak, Calvin H. K., Stephen K. H. Wong, George K. Wong, Stephanie Ng, Kevin K. W. Wang, Ping Kuen Lam, and Wai Sang Poon. 2012. "Traumatic Brain Injury in the Elderly: Is It as Bad as We Think?" Current Translational Geriatrics and Experimental Gerontology Reports 1(3):171–78. doi: 10.1007/s13670-012-0017-2.
22. Mina AA, Knipfer JF, Park DY, Bair HA, Howells GA, Bendick PJ: Intracranial complications of preinjury anticoagulation in trauma patients with head injury. J Trauma 2002, 53:668-672.
23. Ohm C, Mina A, Howells G, Bair H, Bendick P: Effects of antiplatelet agents on outcomes for elderly patients with traumatic intracranial hemorrhage. J Trauma 2005, 58:518-522.
24. Major J, Reed MJ: A retrospective review of patients with head injury with coexistent anticoagulant and antiplatelet use admitted from a UK emergency department. Emerg Med J 2009, 26:871-876.
25. Naidech AM, Bendok BR, Garg RK, Bernstein RA, Alberts MJ, Bleck TP, et al: Reduced platelet activity is associated with more intraventricular hemorrhage. Neurosurgery 65: 684–688, 2009
26. Naidech AM, Bernstein RA, Levasseur K, Bassin SL, Bendok BR, Batjer HH, et al: Platelet activity and outcome after intracerebral hemorrhage. Ann Neurol 65:352–356, 2009
27. Naidech AM, Jovanovic B, Liebling S, Garg RK, Bassin SL, Bendok BR, et al: Reduced platelet activity is associated with early clot growth and worse 3-month outcome after intracerebral hemorrhage. Stroke 40:2398–2401, 2009 hemorrhage. World Neurosurg 76:100–104, 2011
28. Feeney JM, Santone E, DiFiori M, Kis L, Jayaraman V, Montgomery SC.1 Compared to warfarin, direct oral anticoagulants are associated with lower mortality in patients with blunt traumatic intracranial hemorrhage: a TQIP study. J Trauma Acute Care Surg. 2016;81(5):843–848.
29. Alter, Scott M., Benjamin A. Mazer, Joshua J. Solano, Richard D. Shih, Mary J. Hughes, Lisa M. Clayton, Spencer W. Greaves, Nhat Q. Trinh, and Patrick G. Hughes. 2020. "Antiplatelet Therapy Is Associated with a High Rate of Intracranial Hemorrhage in Patients with Head Injuries." Trauma Surgery and Acute Care Open 5(1):1–5. doi: 10.1136/tsaco-2020-000520.
30. Ho, Sandra, Vismay Thakkar, and Jack Jallo. 2013. "Outcomes in Traumatic Brain Injury Patients on Preinjury Anticoagulation and Antiplatelet Agents." JHN Journal 8(1):1–5. doi: 10.29046/jhnj.008.1.002.
31. Bachelani AM, Bautz JT, Sperry JL, Corcos A, Zenati M, Billiar TR, Peitzman AB, Marshall GT: Assessment of platelet transfusion for reversal of aspirin after traumatic brain injury. Surgery 2011, 150:836-843
32. Chowdhury, Subhankar, and Partha pratim Chakraborty. 2017. "Universal Health Coverage - There Is More to It than Meets the Eye." Journal of Family Medicine and Primary Care 6(2):169–70. doi: 10.4103/jfmpc.jfmpc.

Romanian Neurosurgery (2022) XXXVI (1): pp. 107-109
DOI: 10.33962/roneuro-2022-018
www.journals.lapub.co.uk/index.php/roneurosurgery

The use of 3d programs in the treatment of glioblastomas - 3d angio window. Technical note

Ahmet Cemil Ergün, Fatih Demir, Metin Kaplan

Firat University Faculty of Medicine, Department of Neurosurgery, Elazığ, TURKEY

ABSTRACT

The purpose of this study is to define pre- and postoperative planning as a new usage area of 3D imaging programs in the follow-up and treatment of glioblastomas. The thin-section MRI images of the cases were used in this study. These images were analyzed with the 3D angio window of the Radiant Dicom Viewer © Program. In the 3D angio window, it was found that the glioblastoma tumour tissue was revealed in 3D, such as the vascular structures, when calvarium and brain tissue were suppressed. In this examination method, the bleeding in the postoperative surgical area is also suppressed, the contrasting residue tissue is fully visible, and can be monitored in 3D.

Keywords
brain haemorrhage,
glioblastoma,
3d imaging programs

Corresponding author:
Ahmet Cemil Ergün

Firat University Faculty of Medicine, Department of Neurosurgery, Elazığ, Turkey

drahmetcemil@yahoo.com.tr

Scan to access the online version

INTRODUCTION

A good preoperative planning should be made to ensure maximum tumor resection with low surgical complications in glioblastomas. It is very important to make the tumor clear and to define its relationship with environmental structures for a good planning in radiological terms. Also, knowing the feeding of the tumor and its relations with vascular structures will facilitate surgery. Clearer evaluation of patients in terms of postoperative residue is also very important for treatment and follow-up (1). For this reason, efforts are continuing to develop new radiological imaging techniques and expand their use. For this purpose, 3D Imaging Programs are widely used in the medical world in this respect. The purpose of this study is to define pre- and postoperative planning as a new usage area of 3D imaging programs in the follow-up and treatment of glioblastomas.

METHODS

For this purpose, 10 cases who were suspected of glioblastoma and were confirmed with diagnosis after surgery were examined. Also, the images of 10 cases of intracerebral hematoma with contrasting MRI images were included from the archives in the study as the control group to find out if the bleeding was suppressed. The thin-section MRI images of the cases were used in this study. These images were

analyzed with 3D Angio Window of the Radiant Dicom Viewer © (64-bit) Program (Figure 1).

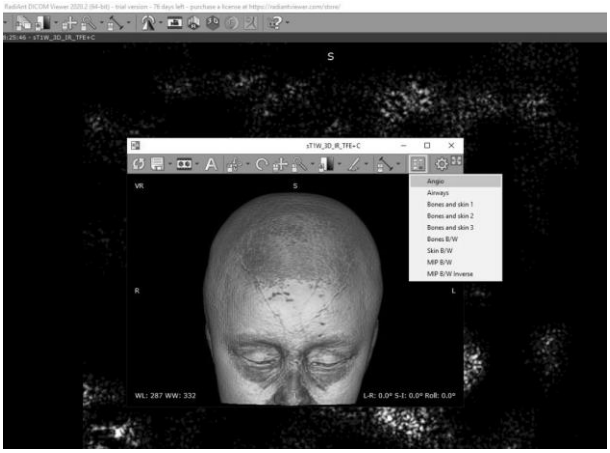


Figure 1. Radiant Dicom Viewer ©- Angio Window Screenshot

Discussion

There are two basic data in the development of our hypothesis. First of all, calvarium and brain tissue are suppressed in this window of the program, and vascular structures are revealed because of its contrast content. This allows to examine the vascular network in 3D. The second one is that the radiologically environmental-ring contrasting is typical for Glioblastoma Multiform. This contrasting area also shows the active tumor area where the feeding occurs, and the main target is the complete resection of this area in surgery. This ring-like contrasting of the tumor external wall was our main reference point. In Radiant Dicom Viewer 3D Angio Window, it was found that the glioblastoma tumor tissue was revealed in 3D, such as the vascular structures, when calvarium and brain tissue were suppressed. Also, this situation constitutes an opportunity to examine the relation and feeding of vascular structures as well as to fully reveal tumor size in 3D (Figures 2-3). It is important to reveal whether there are postoperative residues in the follow-up and treatment of glioblastomas. Sometimes it becomes difficult to evaluate this because of postoperative bleeding areas in control radiological images. It may not be easy to reveal whether it is bleeding or residual tissue, and if it is residual tissue, to determine the exact size of it. In this examination method, the bleeding in the postoperative surgical area is also suppressed, the contrasting residue tissue is fully visible, and can be monitored in 3D (Figure 3). These images will contribute to the creation of a roadmap for the follow-up and treatment of glioblastoma multiforme patients after their surgery. Also, the bleeding will be suppressed with this examination method in atypical bleedings, and it will be easier to understand the underlying pathology, if any (bleeding into the tumor, vascular malformations) (Figure 4-5).

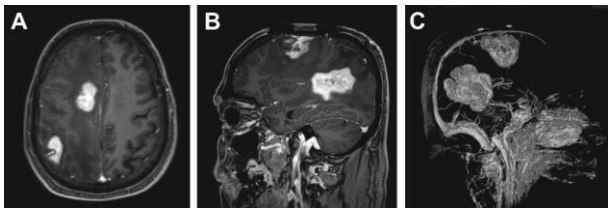


Figure 2. Multiple glioblastoma case; Tumors and vascular structures are observed in a- Axial contrasted MRI, b- Contrasted sagittal MRI, c- 3D Angio Window Image.

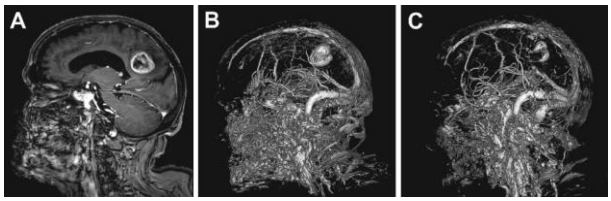


Figure 3. Glioblastoma case; a- Sagittal contrasted MRI, b- Relation with tumors and vascular structures is seen in 3D Angio Window Image, c- The presence of tissue in the postoperative residue in 3D angio window image.

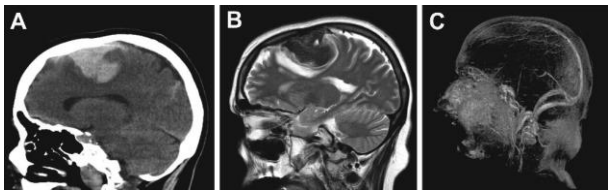


Figure 4. Case of atypical bleeding; a- Sagittal CT, b- Sagittal T2-weighted MRI,
c- Complete suppression of the bleeding is seen as there is no contrasting in 3D angio window.

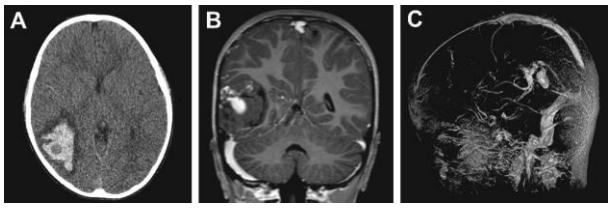


Figure 5. Atypical bleeding case; a- Axial CT, b- Coronal-contrasted MRI, c- It is seen in 3D angio window that bleeding is suppressed, and the presence of contrasted vascular anomaly is preserved.

References

1. Wang LL, Leach JL, Breneman JC, McPherson CM, Gaskill-Shipley MF. Critical role of imaging in the Neurosurgical and radiotherapeutic management of brain tumors. Radio Graphics. 2014; 34:702-721.

Romanian Neurosurgery (2022) XXXVI (1): pp. 110-115
DOI: 10.33962/roneuro-2022-019
www.journals.lapub.co.uk/index.php/roneurosurgery

Full endoscopic lumbar decompression of spinal stenosis through uniportal approach. Technique and preliminary results

Talat Cem Ovalioglu[1], Salih Aydin[2]

[1] Bakirkoy Research and Training Hospital for Neurology, Neurosurgery and Psychiatry, Department of Neurosurgery, Istanbul, TURKEY
[2] Uskudar University Faculty of Medicine, Department of Neurosurgery, Istanbul, TURKEY

ABSTRACT

Introduction: To investigate the certain advantages of full endoscopic lumbar decompression (FELD) surgery over conservative techniques in lumbar spinal stenosis (LSS). Minimal invasive techniques have been introduced recently to treat lumbar spinal stenosis and gained popularity over conservative techniques because of the minimal tissue damage along with the satisfactory postoperative outcome. In our study, the FELD technique using a uniportal approach is described and investigated in accordance with the preliminary clinical results of patients who had degenerative spinal stenosis.

Methods: 55 patients who underwent FELD were retrospectively reviewed. General demographics and parameters including operation time, length of hospital stay, mean time to return to work, complications were recorded. Clinical outcomes were evaluated using the visual analogue scale (VAS) for low back and leg pain, Oswestry Disability Index (ODI) for functional assessment.

Results: The mean follow-up time was 36 months. There was no measurable intraoperative blood loss. The mean operating time was 97,4 minutes. The length of hospital stay after the operation was 27 hours on average. The difference between preoperative and postoperative VAS scores was statistically significant ($p<0.001$). No surgery-related complication was noted.

Conclusions: Amongst other standard techniques, FELD has the technical advantages of less paraspinal muscle dissection, less tissue trauma, less risk of spinal instability, minimal blood loss, quicker postoperative recovery and shorter length of hospital stay.

Keywords
endoscopic,
lumbar decompression,
spinal stenosis,
uniportal approach

Corresponding author:
Talat Cem Ovalioglu

Bakirkoy Research and Training Hospital for Neurology, Turkey

talatovalioglu@gmail.com

Scan to access the online version

INTRODUCTION

Degenerative lumbar stenosis is caused by the hyperthrophy of the surrounding bone structures and disco-ligamentous complex and it is more prevalent in the elderly. The compression may cause clinical symptoms of neurogenic claudication with radicular signs. Back pain is more likely attributable to the degenerative process, such as segmental instabilities or deformities.

As minimal invasive techniques, biportal endoscopic decompression technique and uniportal full endoscopic lumbar decompression technique have been introduced recently and it has been reported to have favorable clinical results and less muscle damage for lumbar spinal stenosis and has begun to attract attention. Since then, several studies were reported satisfactory results with a follow-up of more than two years after unilateral biportal endoscopic lumbar decompression [8, 9, 13]. Thus, FELD has been suggested as an alternative to open laminectomy.

This study evaluates the short-term outcome of unilateral uniportal endoscopic spinal surgery for degenerative spinal stenosis instead of biportal approach.

Material and methods

In this study, 55 patients with symptomatic degenerative lumbar stenosis, who underwent FELD operation between 2014 and 2015 were evaluated retrospectively. The indication for surgery was defined according to present-day standards based on radiographic images, radicular pain symptoms or neurogenic claudication unresponsive to conservative treatment and progressive neurological deficits3. Ethical approval was obtained from the ethical committee and a written informed consent was obtained from participants in the study prior to surgery.

Preoperative and postoperative imaging data, including magnetic resonance imaging (MRI) and computed tomography (CT) images were collected. Follow-up visits were conducted on 3rd, 12th and 36th month as the final follow up. Clinical examination was made during the follow-up visits. In addition to general parameters, pain related information was obtained using these questionnaires: VAS for back and leg pain and ODI for functional assessment.

Clinical inclusion criteria was neurogenic claudication with unilateral or bilateral leg pain with or without paresis. Exclusion criterias were predominant back pain, mono segmental recess stenosis, foraminal stenosis in the lower level, coexisting disc herniation, degenerative spondylolisthesis with maximum Meyerding Grade I, multidirectional rotation slide,, scoliosis (maximum curvature 20°), prior surgery of the same segment.

Full-endoscopic instruments

The working sheath has an outer diameter of 10 mm and an opening with an oblique tip that enables enhanced visualization. Insertion of the working sheath is made bluntly using a dilator. An optic with an outer diameter of 9.5mm is inserted through the working sheath. The optic contains an intraendoscopic, excentric working canal with a diameter of 5.7 mm, a light conductor system, a canal for continuous irrigation and a rod lens system. The angle of vision is 25 degrees. Various instruments including drills up to 5.5 mm in diameter can be used through the working canal. All of the operating instruments and optic products were supplied by the WOLF company (Richard Wolf GmbH, Knittlingen, Germany).

Operative technique

The full-endoscopic interlaminar operation technique was described previously in the literature.[17-19] Under general anesthesia, patients are positioned in prone position under radiographic control (Figure 1). A dilator is inserted bluntly to the lateral edge of the interlaminar window and an operating sheath with an oblique opening is directed toward the ligamentum flavum under constant irrigation. The identification of the medial edge of the ascending facet is made, normal anatomy is recognized (Figure 2). Then ipsilateral decompression is achieved by cranial and caudal laminectomy, partial facetectomy, and ligamantum flavum resection. Then on the contralateral side of the dorsal dura, the ligamentum flavum is initially left intact in order to protect the dura and craniocaudal laminectomy, partial facetectomy is performed (Figure 3). Subsequently, the ligamentum flavum is completely resected. The decompression is concluded when the dura and the spinal nerves are adequately decompressed on both sides (Figure 4). None of the patients had intradiscal nucleotomy along with the fenestration of the annulus.

Statistical Analysis

Numerical variables expressed by mean, standard deviation. Statistical analysis was calculated using the SPSS (Statistical Package for Social Sciences) version 21.0 for Windows (SPSS Inc., Chicago, IL, USA). Paired t-test were applied to compare the

preoperative and postoperative VAS, ODI parameters. p<0.05 was considered significant.

Results

Study group

There were 21 female and 34 male patients aged between 44 and 84 years (mean 58,2 years). The duration of symptoms ranged from 2 to 78 months (mean 19 months). Preoperative average walking distance of the patients was 30 meters.

45 interventions were performed at the L4–L5 level, L3–L4 level was operated on five patients; L5-S1 level was operated on two patients; L1-L2 level was operated on two patients; L2–L3 level was operated on one patient.

Throughout 12 patients who were under the age of 50, 9 patients were operated on the L4-5 level, one patient on the L1-2, one patient on the L3-4 and one patient on the L5-S1 level. No recurrence or complication was noted.

Throughout the rest of the patients who were over 50 years old, 37 patients were operated on the L4-5 level, four patients on the L3-4, one patient on the L1-2, one patient on the L2-3 level. Same as the young age group, no recurrence and complications were noted.

Perioperative and postoperative outcome

The mean operating time was 97,4 minutes (65 to 120min). There was no measurable blood loss. Adequate bleeding control was achieved via continuous irrigation and the use of radiofrequency bipolar coagulation. The patients were mobilized five hours after the operation depending on the effects of the anesthesia. Overall hospital stay was 27 hours on average and mean time to return to work was 14 days.

There were 3 cases of back pain in which the patient recovered well following physical therapy and 5 cases of paresthesia that gradually improved following 2 – 3 weeks of rehabilitation. There were no other complications such as dural tear, hematoma, delayed wound healing, softtissue infection, spondylodiscitis, cauda-equina syndrome or thrombosis. All procedures were successfully completed via endoscopic approach, there was no need to convert to an open approach.

Clinical Outcome

Significant reduction of radicular pain was noted postoperatively, along with the statistically significant results of VAS and ODI questionnaires.

Preoperative VAS leg score on the affected side was 7.9±1.1 and the postoperative VAS leg score improved to 1.1±0.7 early postoperatively. The improvement was statistically significant (p< 0.001). Significant improvement of VAS leg scores was achieved on 3rd month, 12th month and 36th month follow-up visits, 0.3 ± 0.5, 0.2 ± 0.5 and 0.1 ± 0.3 respectively (p< 0.001). Significant improvement of VAS back scores was noted on 3rd month, 12th month and 36th month follow-up visits, 1.6 ± 0.7, 1.01 ± 0.8 and 0.9 ± 0.9, respectively (p< 0.001). ODI scores changed from 48.2 ± 21.9 preoperatively to 26.6 ± 7.9 early postoperatively (p< 0.001). Significant improvement was noted on 3rd month, 12th month and 36th month follow-up visits, 20.4 ± 5.6, 14.5 ± 5.5 and 12.8 ± 5.4 (p< 0.001) respectively. Furthermore, significant development in the walking distance of the patients was observed statistically. Walking distance increased from 30.7 ± 14.4 meters to 124.3 ± 38.8 meters early postoperatively (p< 0.001). Significant improvement in walking distance was noted on 3rd month,12th month and 36th month final follow-up visits; 301.2 ± 81.1 meters, 682 ± 86.2 and 703.6 ± 202.2 meters respectively (p< 0.001).

Discussion

Lumbar spinal stenosis is the result of a dejenerative process, including facet joint hypertrophy, loss of intervertebral disc height, disc bulging, osteophyte formation, and hypertrophy of the ligamentum flavum [1]. The hallmark of spinal stenosis is neurogenic claudication, consisting of lower limb pain and neurological symptoms exacerbated by walking [19]. Nonetheless, people with LSS often avoid walking and have reduced walking capacity because of the pain and discomfort in the lower extremities during walking [10, 21]. The LSS patients also suffer from physical impairments including poor balance, sensory loss (numbness or tingling), and muscle weakness in the lower extremities[10, 11]. Symptoms are generally intermittent and posture-dependent. Mostly they appear with standing for a long time and lumbar extension. The symptoms usually worsen by walking and they are relieved by rest in a flexed or seated position. Radicular pain may be due to a combination of mechanical compression, inflammatory irritation of neural elements, vascular congestion and segmental instability [1].

Depending on the severity of stenosis, various kinds of treatments are available, from surgical procedures to conservative methods. Open microscopic laminectomy procedure has been the common standard surgical treatment method for various types of lumbar spinal stenosis. The standard surgery procedure requires, followed by laminectomy and excision of hypertrophic ligamentum flavum for decompression. The multifidus muscle injury and muscle atrophy occur frequently after posterior lumbar spine surgery, and they are associated with lower back pain and functional disability[7]. Postoperative iatrogenic instability following microscopic laminectomy procedure, is a possible postoperative outcome of this technique. However, it has been reported to occur rarely following these procedures. Silvers et al. reported that lumbar instability rarely occur after open laminectomy procedure [20]. Since several studies have reported favorable long-term results, the technique is currently considered the standard technique [2, 6].

Nowadays, the main goal is to preserve normal anatomical spinal alignement without causing postoperative spinal instability. It has been shown that standard microsurgical lumbar decompression may lead to iatrogenic muscle injury and spinal instability, requiring additional surgical intervention for stabilization [5]. Regarding these complications, minimal invasive techniques have been introduced recently. On 2015, Komp et al. reported bilateral full-endoscopic decompression with equivalent clinical results and less complication rate, less operation time when compared to standard microsurgical laminectomy [14].

Minimal invasive techniques include bilateral decompression using biportal unilateral approach and uniportal unilateral approach for patients with lumbar stenosis [9, 13, 16]. The difference between uniportal and biportal technique is the number of the portals in use during the operation. Biportal technique requires two portals whereas uniportal technique requires only one portal for the endoscope and the remaining endoscopic instruments. There isn't adequte proof of one's superiority, since it depends on the surgeon's experience with the technique [9].

Via uniportal endoscopic technique, there may be no need to make a large facet resection intraoperatively, except when there is hypertrophy of the superior facet that compresses the spinal cord through lateral recess. With the novel endoscopic instruments providing better visual control, stability of the facet joint is not violated significantly while resecting bone elements and this prevents lateral wedging motion of the vertebral segment which maintains stability even after decompression. This technique also allows an extended view of the foramen and lateral recess, especially of the contralateral side, adequately. An enhanced visualization may lead to less neural injury, with adequate decompression of lateral recess and neural foramen on both ipsilateral and contralateral sides.

The procedure is performed under general anesthesia with a 1cm vertical incision for each level. This minimal invasive technique with minimal incision site thus leads to less infection rate and it provides an opportunity for even elderly patients who have preexisting comorbidities prior to the procedure.

The benefits of FELD approaches include decreased blood loss, shorter operation time, shorter hospital stay, decreased postoperative narcotic requirement, decreased rate of infection and cerebrospinal fluid leak and a decrease in time to return to work. Lee et al. reported mean time of 2.4 days of hospital stay after uniportal endoscopic laminotomy for spinal stenosis [15]. In our study, despite the lack of comparison with lumbar microsurgery patients, mean time to return to work and total hospital stay is significantly short such as 14 days and 24 hours respectively. This result was consistent with the recent literature [15].

As for complications, dural tear incidence is the most common complication noted during FELD surgery. In 2017, Kim et al. reported 6.25% cerebrospinal fluid leakage rate in 48 patients who had endoscopic lumbar decompression procedure [12]. Since our clinic has fair number of endoscopic cases, there hasn't been any serious complication perioperatively. We have encountered low infection rate and no cerebrospinal fluid leakage was noted. However, it is important to emphasize that the learning curve is steep for endoscopic operations and complications like cerebrospinal fluid leakage may occur at the beginning phase of this process.

As shown in the study of Bresnahan et al., preservation of the posterior elements via endoscopic techniques is associated with better

outcomes regarding lumbar spinal stability. Main conclusions of these studies are less tissue trauma, shortened rehabilitation period and less postoperative complications [4, 14]. Since our study is a case series study, a comparison could not be stated, however, our results were consistent with the literature.

Along with these advantages, there are some certain disadvantages of the procedure, such as; the limited possibility of extending the approach in a case of possible neural injury or cerebrospinal fluid leakage and the steep learning curve of the procedure which may lead to higher complication rate at the beginning of the practice.

Conclusions

The FELD operation through uniportal approach for lumbar degenerative stenosis is a sufficient and safe supplementation and alternative to the conventional microsurgical procedure. In our experience, the advantages of the procedure are; facilitation of the procedure via excellent visualization, good illumination, and expanded field of vision with 25 degrees optics; short operating time, adequate pain relief, reduced tissue trauma and rapid rehabilitation.

References

1. Atlas SJ, Delitto A. Spinal stenosis: surgical versus nonsurgical treatment. Clin Orthop Relat Res. 2006;443: 198-207. https://doi.org/10.1097/01.blo.0000198722.70138.96.
2. Atlas SJ, Keller RB, Robson D, Deyo RA, Singer DE. Surgical and nonsurgical management of lumbar spinal stenosis: four-year outcomes from the maine lumbar spine study. Spine (Phila Pa 1976). 2000;25(5): 556-562. https://doi.org/10.1097/00007632-200003010-00005.
3. Binder DK, Schmidt MH, Weinstein PR. Lumbar spinal stenosis. Semin Neurol. 2002;22(2): 157-166. https://doi.org/10.1055/s-2002-36539.
4. Bresnahan L, Ogden AT, Natarajan RN, Fessler RG. A biomechanical evaluation of graded posterior element removal for treatment of lumbar stenosis: comparison of a minimally invasive approach with two standard laminectomy techniques. Spine (Phila Pa 1976). 2009;34(1): 17-23. https://doi.org/10.1097/BRS.0b013e318191438b.
5. Bresnahan LE, Smith JS, Ogden AT, et al. Assessment of Paraspinal Muscle Cross-sectional Area After Lumbar Decompression: Minimally Invasive Versus Open Approaches. Clin Spine Surg. 2017;30(3): E162-E168. https://doi.org/10.1097/BSD.0000000000000038.
6. Caputy AJ, Luessenhop AJ. Long-term evaluation of decompressive surgery for degenerative lumbar stenosis. J Neurosurg. 1992;77(5): 669-676. https://doi.org/10.3171/jns.1992.77.5.0669.
7. Fan S, Hu Z, Zhao F, Zhao X, Huang Y, Fang X. Multifidus muscle changes and clinical effects of one-level posterior lumbar interbody fusion: minimally invasive procedure versus conventional open approach. Eur Spine J. 2010;19(2): 316-324. https://doi.org/10.1007/s00586-009-1191-6.
8. Huang YH, Lien FC, Chao LY, Lin CH, Chen SH. Full Endoscopic Uniportal Unilateral Laminotomy for Bilateral Decompression in Degenerative Lumbar Spinal Stenosis: Highlight of Ligamentum Flavum Detachment and Survey of Efficacy and Safety in 2 Years of Follow-up. World Neurosurg. 2020;134: e672-e681. https://doi.org/10.1016/j.wneu.2019.10.162.
9. Hwa Eum J, Hwa Heo D, Son SK, Park CK. Percutaneous biportal endoscopic decompression for lumbar spinal stenosis: a technical note and preliminary clinical results. J Neurosurg Spine. 2016;24(4): 602-607. https://doi.org/10.3171/2015.7.SPINE15304.
10. Iversen MD, Katz JN. Examination findings and self-reported walking capacity in patients with lumbar spinal stenosis. Phys Ther. 2001;81(7): 1296-1306.
11. Johnsson KE, Rosen I, Uden A. Neurophysiologic investigation of patients with spinal stenosis. Spine (Phila Pa 1976). 1987;12(5): 483-487. https://doi.org/10.1097/00007632-198706000-00012.
12. Kim HS, Paudel B, Jang JS, et al. Percutaneous Full Endoscopic Bilateral Lumbar Decompression of Spinal Stenosis Through Uniportal-Contralateral Approach: Techniques and Preliminary Results. World Neurosurg. 2017;103: 201-209. https://doi.org/10.1016/j.wneu.2017.03.130.
13. Kim SK, Kang SS, Hong YH, Park SW, Lee SC. Clinical comparison of unilateral biportal endoscopic technique versus open microdiscectomy for single-level lumbar discectomy: a multicenter, retrospective analysis. J Orthop Surg Res. 2018;13(1): 22. https://doi.org/10.1186/s13018-018-0725-1.
14. Komp M, Hahn P, Oezdemir S, et al. Bilateral spinal decompression of lumbar central stenosis with the full-endoscopic interlaminar versus microsurgical laminotomy technique: a prospective, randomized, controlled study. Pain Physician. 2015;18(1): 61-70.
15. Lee CW, Yoon KJ, Jun JH. Percutaneous Endoscopic Laminotomy with Flavectomy by Uniportal, Unilateral Approach for the Lumbar Canal or Lateral Recess Stenosis. World Neurosurg. 2018;113: e129-e137. https://doi.org/10.1016/j.wneu.2018.01.195.

16. Ruetten S, Komp M, Godolias G. An extreme lateral access for the surgery of lumbar disc herniations inside the spinal canal using the full-endoscopic uniportal transforaminal approach-technique and prospective results of 463 patients. Spine (Phila Pa 1976). 2005;30(22): 2570-2578. https://doi.org/10.1097/01.brs.0000186327.21435.cc.
17. 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. 2006;49(2): 80-87. https://doi.org/10.1055/s-2006-932172.
18. Ruetten S, Komp M, Merk H, Godolias G. Full-endoscopic interlaminar and transforaminal lumbar discectomy versus conventional microsurgical technique: a prospective, randomized, controlled study. Spine (Phila Pa 1976). 2008;33(9): 931-939. https://doi.org/10.1097/BR S.0b013e31816c8af7.
19. Ruetten S, Komp M, Merk H, Godolias G. Use of newly developed instruments and endoscopes: full-endoscopic resection of lumbar disc herniations via the interlaminar and lateral transforaminal approach. J Neurosurg Spine. 2007;6(6): 521-530. https://doi.org/10.3171/spi.2007.6.6.2.
20. Silvers HR, Lewis PJ, Asch HL. Decompressive lumbar laminectomy for spinal stenosis. J Neurosurg. 1993;78(5): 695-701. https://doi.org/10.3171/jns.1993.78.5.0695.
21. Tomkins-Lane CC, Holz SC, Yamakawa KS, et al. Predictors of walking performance and walking capacity in people with lumbar spinal stenosis, low back pain, and asymptomatic controls. Arch Phys Med Rehabil. 2012;93(4): 647-653. https://doi.org/10.1016/j.apmr.2011.09.023.

Romanian Neurosurgery (2022) XXXVI (1): pp. 116-121
DOI: 10.33962/roneuro-2022-020
www.journals.lapub.co.uk/index.php/roneurosurgery

Clinical outcomes and satisfaction in patients after lumbar microdiscectomy. A single centre study

Dipak Chaulagain, Volodymyr Smolanka, Andriy Smolanka, Taras Havryliv, Alexandr Shecko

Regional Clinical Centre of Neurosurgery and Neurology, Uzhhord National University, Uzhhorod, UKRAINE

ABSTRACT

Introduction: Lumbar disc herniation is a common cause of low back and radicular pain. Microdiscectomy is the recommended surgical technique for herniated lumbar discs at the moment. It has great success rates and little postoperative morbidity. We aimed to assess the clinical result and patient satisfaction of micro-discectomy in lumbar disc herniation patients.

Methodology: This is prospective observational hospital-based research of 26 patients who had micro-discectomy at the Regional Clinical Center of Neurosurgery and Neurology in Uzhhorod, Ukraine, during August and September 2021. The research excluded patients with recurrent prolapsed intervertebral discs, multiple level herniated discs, and disc surgery requiring stability. During surgery, the kind of prolapsed intervertebral disc, its level, and the duration of the procedure were recorded. Additionally, we recorded the duration of the patient's hospital stay and any complications. The visual analogue scale (VAS), the Oswestry Disability Index (ODI), and the MacNab score questionnaires were used to measure pain, disability, and patient satisfaction, respectively.

Result: All procedures were performed on a single level using micro-discectomy. The mean age of the study population was 45.69 years. Micro-discectomy surgeries were performed in less than an hour in 69.2% of cases. The most often seen lumbar prolapsed intervertebral disc occurred at the L4-L5 level (57.7%). The most often seen kinds of prolapse were disc extrusion (30.8%) and disc sequestration (26.9%). The mean length of stay in the hospital was 3.96 days. After surgery, 57.7% of patients received an excellent rating on the Macnab's scale. There was a statistically significant difference between pre- and postoperative VAS and ODI scores ($p<0.05$).

Conclusion: Overall 65.4% of patients had no postoperative complications. In these instances, a proper surgical technique might help avoid problems. Our findings demand additional investigation with bigger sample sizes and longer follow-up periods.

Keywords
disc herniation, lumbar pain, microdiscectomy

Corresponding author:
Dipak Chaulagain

Regional Clinical Centre of Neurosurgery and Neurology, Uzhhord National University, Uzhhorod, Ukraine

neurodipak@gmail.com

Scan to access the online version

INTRODUCTION

Low Back pain is a prevalent complaint among patients seeking basic care. Lumbar disc herniation (LDH) causes low back pain and leg pain [23]. Nucleus pulposus or annulus fibrosis displacement beyond

normal intervertebral disc space is a typical cause for radiculopathy. 95 percent of lower lumbar disc herniation occur at L4-5 and L5-S1 [12, 17]. A lumbar disc herniation may be treated in many ways, but the diagnosis is made based on the patient's history, physical exam, and radiograph Patients with persistent lower back pain, with or without leg radiating pain, difficult to manage pain, or acute paresis, including cauda equine syndrome, are frequently given surgery [28]. Radiculopathy caused by LDH usually heals without surgery. If nonsurgical treatment fails, micro-discectomy may be explored. Lumbar micro-discectomy has good success rates and little postoperative morbidity. The operation has been shown to be successful in treating lumbar radicular pain and sciatica with success rates ranging from 50% to 98% [26]. Elective lumbar discectomy is one of the most popular neurosurgical treatments for lumbar disc herniation. If sciatica or neurological impairments remain following a period of conservative therapy, a discectomy is regarded the gold standard [4, 13]. More than 70 years, fenestrated discectomy has been the primary treatment for lumbar disc herniation worldwide [15]. A long midline incision and significant muscle retraction with complete laminectomy were first documented in 1934 by Mixter and Barr [3]. LDH surgery entered a new era in 1977 when surgeons started using operating microscopes to remove herniated disc material [6]. As early findings showed, micro-discectomy was equally as effective as regular discectomy and had certain benefits over the latter. This was soon after the launch of the aforementioned invention. An advantage of micro-discectomy over traditional open discectomy is the ability to do surgery with fewer incisions and less harm to the skin and fascia [18]. Oswestry Impairment Index (ODI) measures the degree of disability caused by low back pain and is based on the Oswestry Low Back Pain Questionnaire. VAS was the last scale to be implemented [5, 8]. Following micro-discectomy surgery, we documented the surgical procedures, clinical results, postoperative patient satisfaction (VAS and ODI), length of stay in the hospital and early problems (Macnab score) that we encountered. The study's goal was to evaluate the short-term clinical outcomes after a single-level lumbar micro-discectomy operation.

Material and methods

From August to September 2021 at a Regional Clinical Center of Neurosurgery and Neurology in Uzhhorod, Ukraine, we conducted a cross-sectional observational research to evaluate our patients who had had a microlumbar discectomy. Patients with a single level lumbar disc herniation were eligible to participate in the trial. Cases with repeated disc herniations, several levels of lumbar disc herniation and disc surgery with instrumentation were ruled out.

Types and levels of LDH, and surgical time were all reported during surgery for LDH (central, lateral extrusion, protrusion, lateral disc bulge, and sequestration). Complications that occurred during treatment were also reported. Patient's pain was assessed using VAS and ODI before and after surgery. This was the main outcome. Two questionnaires, the Visual Analogue Scale (VAS, 0-10) and the Oswestry Impairment Index (ODI, 0-100% disability), were used to evaluate patient's levels of pain and disability before and after surgery. When it comes to assessing low back function, the ODI is the gold standard [19, 25]. The Macnab questionnaire was used to measure patient satisfaction and it was rated excellent, good, fair, or bad [16].

Early complications like nausea and vomiting, post-operative cerebrospinal fluid leak, wound infection and discitis were recorded. SPSS version 25 was used for data collection, and the data were then analyzed using the statistical software for social sciences (SPSS). For example, age and gender, type of LDH and complication. ODI and VAS were evaluated by the paired samples t-test method. $P < 0.05$ was assessed as significant.

Results

From August to September 2021, lumbar disc herniation patients who had micro-lumbar discectomy were studied prospectively. The patients were mostly male and average age was 45.69 [Table 4]. All surgeries were single level micro-discectomy including L3-L4 (3.8%), L4-L5 (57.7%), and L5-S1 (38.5%) [Table 1] .69.2% cases were performed surgically by less than one hour whereas 30.8% cases took more than one hour for micro-discectomy [Table 1]. Average day of staying in hospital of patients post-operatively was 3.96 days [Table 4]. Disc extrusion (30.8%) and disc sequestration (26.9%) were the most commonly seen types of prolapse [Table 1]. There was statistically significant difference ($p<0.05$) in pre-operative and post-

operative VAS scores and ODI [Table 5]. After surgery, 57.7% of patients received an excellent rating on the macnab's scale [Table 2] and Overall 65.4% of patients had no postoperative complications [Table 3].

Table 1. Description of patients involved in the study

Total number of patients	26
Average age (range)	30-58
Males	16 (61.5%)
Females	10 (38.5%)
Side of PIVD	
Right side	15 (57.7%)
Left side	11 (42.3%)
Level of prolapse	
L3-L4	1 (3.8%)
L4-L5	15 (57.7%)
L5-S1	10 (38.5%)
Types of PIVD	
Central disc bulge	6 (23.1%)
Lateral disc bulge	8 (30.8%)
Disc protrusion	3 (11.5%)
Disc extrusion	7 (26.9%)
Disc sequestration	2 (7.7%)
Operation duration	
Less than hour	18 (69.2%)
More than hour	8 (30.8%)

Table 2. Description of Macnab's score (post-op) in the study

	Frequency	Percent	Valid Percent	Cumulative Percent
EXCELLENT	15	57.7	57.7	57.7
FAIR	3	11.5	11.5	69.2
GOOD	7	26.9	26.9	96.2
POOR	1	3.8	3.8	100.0
Total	26	100.0	100.0	

Table 3. Description of early post-op complication in the study

	Freq.	%	Valid Percent	Cumulative Percent
CSF LEAK	1	3.8	3.8	3.8
DISCITIS	1	3.8	3.8	7.7
NAUSEA,VOMITING	5	19.2	19.2	26.9
NO COMPLICATION	17	65.4	65.4	92.3
WOUND INFECTION	2	7.7	7.7	100.0
Total	26	100.0	100.0	

Table 4. Descriptive mean statistics of Age and Duration of hospital stay (post-op) in the study

	N	Min.	Max.	Mean	Std. Deviation
AGE	26	30	58	45.69	8.966
POST-OP DURATION OF HOSPITAL STAY(DAY)	26	2	6	3.96	1.311

Table 5. Paired-samples T-test of VAS score and ODI (pre-op and post-op) in the study

							t	df	Sig. (2-tailed)
					95% Confidence Interval of the Difference				
		Mean	Std. Deviation	Std. Error Mean	Lower	Upper			
Pair 1	VAS SCORE (PRE-OP) - VAS SCORE (POST-OP)	6.500	1.105	.217	6.054	6.946	30.007	25	.000
Pair 2	ODI % (PRE-OP) - ODI % (POST-OP)	30.000	14.213	2.787	24.259	35.741	10.763	25	.000

Discussion

If the nucleus pulposus and the annulus fibrosus degenerate, LDH is almost always caused by this. Lifting accidents or other trauma may also be to blame. People who have lower back pain almost always have their intervertebral discs bulging, especially if they push on a nerve root. Numbness and tingling are very common in the lower legs because of the pain caused by this. The L4-L5 and L5-S1 segments of the spinal column are the most often affected by disc disease. There are a variety of therapy options available for lumbar discopathy. 90% of individuals will have a conservative improvement in their pain levels [22].

Patients with lumbar disc herniation had satisfactory long-term treatment outcomes for their sciatica symptoms, regardless of whether they underwent surgery or received conservative therapy. When compared to conservative therapy, surgical surgery alleviated back pain more quickly; nevertheless, after three months, no difference was seen between any of the treatments [21]. Discectomy of the lumbar spine is one of the most frequent surgical procedures performed today. Open surgery and endoscopic surgery are both

acceptable methods of doing the procedure. The discectomy process has gone a long way since Yasargil conducted the first microscopic surgery in 1968 and Schreiber and Suezawa conducted the first endoscopic discectomy in 1986, then Mayer, Brock, and Mathews refined the technique in the 1990s [7, 9,14,24].

In this cross-sectional research, we looked at the surgical result and patient satisfaction rate of micro-lumbar discectomy, which is the most common kind of spine surgery. Discectomy of the lumbar spine is one of the most frequent surgical procedures performed today. Open surgery and endoscopic surgery are both acceptable methods of doing the procedure. The outcomes of this research corroborate the long-held notion that discectomy is a safe and effective therapeutic option for lumbar discopathy associated with sciatic pain in the lower back. Patients have claimed success rates ranging from 88–97% for this treatment; however, more realistic results, as determined by patient-reporting measures, range from 75–80% [1].

Because of rising healthcare expenditures and other costs connected with hospitalization, most neurosurgical centers across the globe perform lumbar microdiscectomy as a day care procedure to save costs. One of the most important considerations during a surgery is the duration of the procedure, especially in light of the probability of blood loss and the existence of intraoperative risk factors for surgical site infections. Micro-discectomy has a number of advantages, one of which is that the quantity of blood lost during the treatment is significantly decreased, according to the findings of various studies [20, 29, 30]. In our study, 69.2% of cases were completed surgically in less than one hour, while 30.8% of patients required more than one hour for micro-discectomy surgery.

According to our study, the median length of hospitalization was 3.96 days, with a range of 2 to 6 days being recorded. Differential results in discectomy-related outcomes are primarily influenced by changes in patient selection, follow-up period, and the tools employed to quantify outcome and their interpretation. Patients who have had lumbar disc herniation surgery are able to return to work in 76% of cases after one year [2].

The kind of disc herniation has also been shown to have a substantial impact on the functional results of patients [27]. Following surgery, 4% of patients report a decrease in their functional condition. Prior to surgery, the following factors are associated with deterioration: lengthy duration of pain and low ODI (greater function) [11].

According to Shriver, Michael F et al., the overall complication rate of micro-lumbar discectomy was 12.5%, with 1.3% reporting new or worsening neurological deficit, 2.6% reporting direct nerve root injury, 0.5% hematoma, 2.1% wound complications (infection, dehiscence, seroma), and 4.1% recurrent disc complications [10]. While in our study, 65.4% of patients had no postoperative issues, we discovered that 3.8%, 3.8%, 19.2%, and 7.7% of cases experienced difficulties such as Cerebrospinal fluid leak, discitis, nausea and vomiting, and wound infection, respectively [Table 3].

While our research had a small number of participants, it was not without its faults. Proposed multi-center study on lumbar micro-discectomy with long-term follow-up should be conducted in order to get more universal and reasonable findings.

Limitations

A single-center research has a smaller sample size. Prospective research with large sample sizes and long follow-up times are also necessary to correctly extrapolate results to the general population.

Conclusions

A lumbar micro-discectomy is a safe and efficient treatment for disc herniation-related sciatic lumbar pain. It will be necessary to conduct additional multi-centric studies with a larger sample size and a longer follow-up period in order to verify our findings.

References

1. Asch, H. L., Lewis, P. J., Moreland, D. B., Egnatchik, J. G., Yu, Y. J., Clabeaux, D. E., & Hyland, A. H. (2002). Prospective multiple outcomes study of outpatient lumbar microdiscectomy: should 75 to 80% success rates be the norm?. Journal of neurosurgery, 96(1 Suppl), 34–44.
2. Bailey, C. S., Rasoulinejad, P., Taylor, D., Sequeira, K., Miller, T., Watson, J., Rosedale, R., Bailey, S. I., Gurr, K. R., Siddiqi, F., Glennie, A., & Urquhart, J. C. (2020). Surgery versus Conservative Care for Persistent Sciatica Lasting 4 to 12 Months. The New England journal of medicine, 382(12), 1093–1102.
3. Brodke, D. S., Goz, V., Lawrence, B. D., Spiker, W. R., Neese, A., & Hung, M. (2017). Oswestry Disability Index: a psychometric analysis with 1,610 patients. The spine

journal : official journal of the North American Spine Society, 17(3), 321–327.

4. Carragee, E. J., Han, M. Y., Suen, P. W., & Kim, D. (2003). Clinical outcomes after lumbar discectomy for sciatica: the effects of fragment type and anular competence. The Journal of bone and joint surgery. American volume, 85(1), 102–108.
5. Caspar, W., Campbell, B., Barbier, D. D., Kretschmmer, R., & Gotfried, Y. (1991). The Caspar microsurgical discectomy and comparison with a conventional standard lumbar disc procedure. Neurosurgery, 28(1), 78–87.
6. Chiarotto, A., Maxwell, L. J., Ostelo, R. W., Boers, M., Tugwell, P., & Terwee, C. B. (2019). Measurement Properties of Visual Analogue Scale, Numeric Rating Scale, and Pain Severity Subscale of the Brief Pain Inventory in Patients With Low Back Pain: A Systematic Review. The journal of pain, 20(3), 245–263.
7. Deyo, R. A., & Mirza, S. K. (2016). CLINICAL PRACTICE. Herniated Lumbar Intervertebral Disk. The New England journal of medicine, 374(18), 1763–1772.
8. Fairbank, J. C., & Pynsent, P. B. (2000). The Oswestry Disability Index. Spine, 25(22), 2940–2952.
9. Fardon, D. F., Milette, P. C., & Combined Task Forces of the North American Spine Society, American Society of Spine Radiology, and American Society of Neuroradiology (2001). Nomenclature and classification of lumbar disc pathology. Recommendations of the Combined task Forces of the North American Spine Society, American Society of Spine Radiology, and American Society of Neuroradiology. Spine, 26(5), E93–E113.
10. Gibson, J. N., & Waddell, G. (2007). Surgical interventions for lumbar disc prolapse: updated Cochrane Review. Spine, 32(16), 1735–1747.
11. Gugliotta, M., da Costa, B. R., Dabis, E., Theiler, R., Jüni, P., Reichenbach, S., Landolt, H., & Hasler, P. (2016). Surgical versus conservative treatment for lumbar disc herniation: a prospective cohort study. BMJ open, 6(12), e012938.
12. Hamawandi, S. A., Sulaiman, I. I., & Al-Humairi, A. K. (2020). Open fenestration discectomy versus microscopic fenestration discectomy for lumbar disc herniation: a randomized controlled trial. BMC musculoskeletal disorders, 21(1), 384.
13. Javedan, S., & Sonntag, V. K. (2003). Lumbar disc herniation: microsurgical approach. Neurosurgery, 52(1), 160–164.
14. Kahanovitz, N., Viola, K., & Muculloch, J. (1989). Limited surgical discectomy and microdiscectomy. A clinical comparison. Spine, 14(1), 79–81.
15. Kulkarni, A. G., Bassi, A., & Dhruv, A. (2014). Microendoscopic lumbar discectomy: Technique and results of 188 cases. Indian journal of orthopaedics, 48(1), 81–87.
16. Macnab I. (1971). Negative disc exploration. An analysis of the causes of nerve-root involvement in sixty-eight patients. The Journal of bone and joint surgery. American volume, 53(5), 891–903.
17. Mathews H. H. (1996). Transforaminal endoscopic microdiscectomy. Neurosurgery clinics of North America, 7(1), 59–63.
18. Mayer, H. M., & Brock, M. (1993). Percutaneous endoscopic discectomy: surgical technique and preliminary results compared to microsurgical discectomy. Journal of neurosurgery, 78(2), 216–225. https://doi.org/10.3171/jns.1993.78.2.0216.
19. Mixter WJ, Barr JS. Rupture of the intervertebral disc with involvement of the spinal canal. N Engl J Med. 1934; 5:210-215.
20. Peul, W. C., van Houwelingen, H. C., van den Hout, W. B., Brand, R., Eekhof, J. A., Tans, J. T., Thomeer, R. T., Koes, B. W., & Leiden-The Hague Spine Intervention Prognostic Study Group (2007). Surgery versus prolonged conservative treatment for sciatica. The New England journal of medicine, 356(22), 2245–2256.
21. Porchet, F., Bartanusz, V., Kleinstueck, F. S., Lattig, F., Jeszenszky, D., Grob, D., & Mannion, A. F. (2009). Microdiscectomy compared with standard discectomy: an old problem revisited with new outcome measures within the framework of a spine surgical registry. European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society, 18 Suppl 3(Suppl 3), 360–366.
22. Reed, M. D., & Van Nostran, W. (2014). Assessing pain intensity with the visual analog scale: a plea for uniformity. Journal of clinical pharmacology, 54(3), 241–244.
23. Ruan, W., Feng, F., Liu, Z., Xie, J., Cai, L., & Ping, A. (2016). Comparison of percutaneous endoscopic lumbar discectomy versus open lumbar microdiscectomy for lumbar disc herniation: A meta-analysis. International journal of surgery (London, England), 31, 86–92.
24. Schreiber, A., & Suezawa, Y. (1986). Transdiscoscopic percutaneous nucleotomy in disk herniation. Orthopaedic review, 15(1), 35–38.
25. Shriver, M. F., Xie, J. J., Tye, E. Y., Rosenbaum, B. P., Kshettry, V. R., Benzel, E. C., & Mroz, T. E. (2015). Lumbar microdiscectomy complication rates: a systematic review and meta-analysis. Neurosurgical focus, 39(4).
26. Solberg, T. K., Nygaard, O. P., Sjaavik, K., Hofoss, D., & Ingebrigtsen, T. (2005). The risk of "getting worse" after lumbar microdiscectomy. European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European

Section of the Cervical Spine Research Society, 14(1), 49–54.

27. Varlotta, G. P., Brown, M. D., Kelsey, J. L., & Golden, A. L. (1991). Familial predisposition for herniation of a lumbar disc in patients who are less than twenty-one years old. The Journal of bone and joint surgery. American volume, 73(1), 124–128.
28. Weinstein, J. N., Lurie, J. D., Tosteson, T. D., Skinner, J. S., Hanscom, B., Tosteson, A. N., Herkowitz, H., Fischgrund, J., Cammisa, F. P., Albert, T., & Deyo, R. A. (2006). Surgical vs nonoperative treatment for lumbar disk herniation: the Spine Patient Outcomes Research Trial (SPORT) observational cohort. JAMA, 296(20), 2451–2459.
29. Williams R. W. (1978). Microlumbar discectomy: a conservative surgical approach to the virgin herniated lumbar disc. Spine, 3(2), 175–182.
30. Yasargil MG. Microsurgical operation for herniated disc. Adv Neurosurg. 1977;4:81.

Romanian Neurosurgery (2022) XXXVI (1): pp. 122-126
DOI: 10.33962/roneuro-2022-021
www.journals.lapub.co.uk/index.php/roneurosurgery

Paediatric traumatic chronic subdural hematoma – a very rare entity. Single institution study of 5 cases

Vivek Kumar Kankane, Avadhesh Shukala, S.N. Iyenger

Department of Neurosurgery, G.R. Medical College; JAH Groups of Hospital Gwalior (M.P.), INDIA

ABSTRACT

Objectives: To study the prevalence of Traumatic chronic subdural hematoma in children.

Material and methods: This is a prospective study conducted at a tertiary care centre at Gwalior, from November 2020 to November 2021. We encountered 5 patients admitted for signs of raised intracranial tension due to Head trauma, showing Chronic subdural Haematoma (CSDH) in plain CT Head or MRI brain. All patients had no history of loss of consciousness, no history of vomiting, no history of seizures, no history of nasal or ear bleed and GCS was 15/15. All blood investigation and coagulation profiles are normal. Frontal and parietal 2.5 cm trephine craniotomy with evacuation of subdural hematoma was done. Patients were followed up at 1 month and 6 months

Result: All children are male. Age ranging from 7 years to 14 years, Mean age of presentation was 10 years. All patients had a history of head trauma and mode of injury road traffic accidents (RTA) and Glasgow coma scale (GCS) at admission was 15 and duration of developed CSDH was 20 days to 42 days'(mean30.40 days). All patients had good outcomes and the average follow-up was 6.5 months. All patients went on Surgical intervention. and outcome assessed by Glasgow Outcome score. 3 out of 5 patients had brownish fluid as a collection in subdural space while the other 2 patients had fluid that has motor oil like consistency in subdural space. All the patients were subjected to a similar procedure. The postoperative course was uneventful. Mean hospital stay was 7 days. A postoperative scan was done on the 7th day.

Conclusion: Chronic Subdural Hematoma is a disease reported in infants and the elderly population. It is uncommon in the age group of 2-14 years. Most of the children present with symptoms of raised intracranial tension due to head injury, with no reported history of repeated head trauma, child abuse & coagulopathy. Traumatic Chronic subdural collection should be considered as one of the diagnoses as the prognosis of this disease is better. However, due to the scarcity of reports in the literature, established guidelines are not available.

Keywords
traumatic,
subdural hematoma,
paediatric

✉
Corresponding author:
Vivek Kumar Kankane

G.R. Medical College,
India

vivekkankane9@gmail.com

Scan to access the online version

INTRODUCTION

Subdural hematoma is the collection of blood on the cortical surface beneath the dura with bleeding from bridging veins or cortical arteries. The term Chronic subdural Haematoma (CSDH) is often applied to

these collections, although the content of the accumulation may vary from thin, watery fluid resembling cerebrospinal fluid to thick "motor oil" often associated with adult CSDH.

The pathophysiology and clinical outcomes of head trauma differ between children and adults.

Blood vessels are absent in the normal dura-arachnoid interface. Neo-vasculature is abundant, but just in the outer CSDH membrane.

Abnormal dilated sinusoids measuring as large as 1000 micrometer, with an incomplete basement membrane and attenuated endothelial cells, share the outer membrane with rapidly growing microcapillaries. Both vessel types are composed of endothelial cells with irregular surface because of numerous pseudopod-like structures extending into vascular lumen. Erythrocyte and platelets in various stages of degeneration are frequently found deposited in the perivascular space. These sinusoids contain gap junction as large as 8 micrometer, sufficient to allow leakage of plasma and even red blood cells into the hematoma cavity.

Inflammatory mediators present in CSDH fluid may potentiate chronic rebleeding of the fragile Neovasculature. Kallikrein, bradykinin, and platelet-activating factor (PAF) have all been identified at significant level in CSDH fluid.

These inflammatory mediators stimulate vasodilation, increase vascular permeability, prolong the clotting time, and release tissue plasminogen activator (t-PA) from endothelial cells. Other work has focused on disturbances of the prostaglandin system as possible components in the pathophysiology [1].

Eosinophil degranulation in the outer membrane may be the source of fibrinolytic factors and inflammatory mediators causing local coagulopathy and cell destruction in the CSDH [2]

The traumatic mass lesions such as subdural and epidural hematomas occur less frequently in children and when present, are associated with lower mortality.

Common manifestations of CSDH are altered mental state and focal neurological deficits [3]. Subdural hematomas (SDH) are associated with an increased morbidity and mortality and generally occur as chronic SDH among older patients [4].

The most frequent signs and symptoms like headache, alteration of consciousness, gait impairment and hemiparesis are also seen among other CNS diseases, so they have to be considered during evaluation. In the case of symptomatic patients with focal neurological deficits surgical intervention should be considered, where as in case of asymptomatic patients or patients with only slight headaches conservative treatment with clinical and radiological follow-up might be a possibility. Also after surgical intervention, the recurrence rate is between 5 and 33% [4].

CSDH are much more common in infants because of associated traumatic deliveries and frequently exist as a single entity. It is rare for chronic subdural fluid accumulations to occur after one year of age and they are more frequent during adolescence [5,6]. Specific traumatic events are usually unrecognized

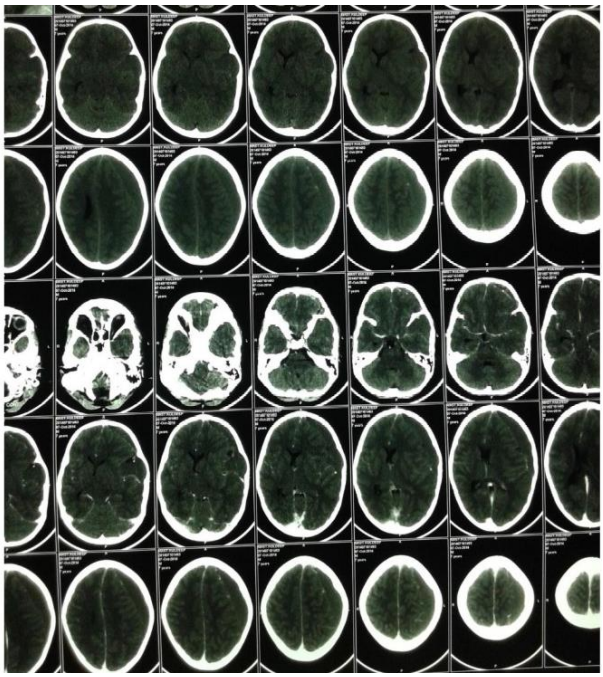


Figure 1. NCCT head revealed chronic subdural haematoma in Left Fronto-temporo-parietal convexity with midline shift toward right side with cisternal effacement.

MATERIALS AND METHODS

This is prospective study conducted at a tertiary care centre at Gwalior, from November 2020 to November 2021. We encountered 5 patients admitted for signs of raised intracranial tension who on radiological examination were found to have hemispheric subdural collection. All patients had No history of loss of consciousness, no history of vomiting, no history of seizures, no history of nasal or ear bleed and GCS was 15/15. All blood

investigation and coagulation profile normal. Frontal and parietal 2.5 cm trephine craniotomy with evacuation of subdural hematoma was done. Patients were followed up at 1 month and 6 months.

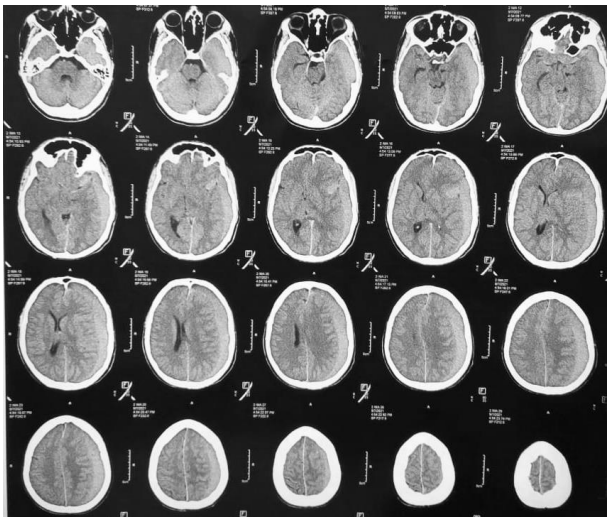


Figure 2. NCCT head revealed chronic subdural haematoma in Left Fronto-temporo-parietal convexity with midline shift toward right side with cisternal effacement.

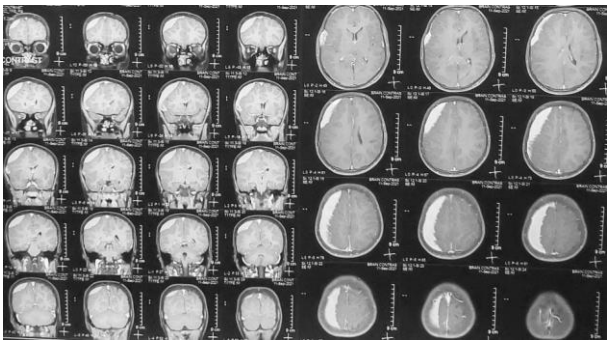


Figure 3. MRI Brain revealed chronic subdural haematoma in Right Fronto-temporo-parietal convexity with midline shift toward right side with cisternal effacement.

Results

All children are male. Age ranging from 7 years to 14 years, Mean age of presentation was 10 years. All patients had history of head trauma and mode of injury road traffic Accidents (RTA) and Glasgow coma scale (GCS) at admission was 15 and duration of developed CSDH was 20 days to 42 days'(mean30.40 days).. All patients had good outcome and average follow-up was 6.5 months. All patients went on Surgical intervention [Table-1] and outcome assessed by Glasgow Outcome score [Table-2].

Table 1. Demographic data, management and outcome of patients

Age (year)	sex	Mode of injury	GCS at the time of Admission	Management	Outcome measure by GOS
14	Male	Road traffic Accident (RTA)	15/15	Surgery	Good
11	Male	RTA	15/15	Surgery	Good
10	Male	RTA	15/15	Surgery	Good
8	Male	RTA	15/15	Surgery	Good
7	Male	RTA	15/15	Surgery	Good

Table 2. Glasgow Outcome Scale

GOS	Functional Status
5	Resumption of normal life; there may be minor neurological and Psychological deficit
4	Able to work in a shattered environment and travel by public transportation
3	Depend on daily support by reason of mental and physical disability or both
2	Unresponsive for weeks or month or until death
1	Death

Table 3. Description of Previous reported case of Traumatic Pediatric Chronic SDH

Author	Year	No. of case	Age/sex of patient	Description of patient
Osaka H et al.	1993	1	10 month/male	Gluteric Aciduria type-1
Narsinghani et.al	2002	1	5 year/Male	strikingly large calcified CSDH in a 5-year-old child with increased intracranial pressure and subfalcine herniation
Mori et al.	2002	12	All patients are paediatric age group	Chronic Subdural Haematoma with Arachnoid cyst
Acakapo satchivi et al.	2007	1	4 month/male	Macrocrania with Chronic Subdural Haematoma

Kumar et al.	2008	20	Range 1 month – 2year	Chronic Subdural Haematoma following minor head injury
Wang et al.	2010	1	1 year/male	Chronic Subdural Haematoma due to repeated minor Dodgeball head injury
Vivek kankane et.al.	2015	1	7 year/male	Chronic Subdural Haematoma due fall from bed 3 month ago

Discussion

Wang, et al. reported a rare case of CSDH in a 9-year-old child due to repeated minor dodgeball head injuries in 2010 [Table-3].

Although such a case has never been reported in sport. No altered mental state or focal neurological deficits were observed; the child presented with intermittent severe headache with nausea and vomiting. There was also no evidence of child abuse; however, the history of repeated minor head injuries during playing was significant [3].

Kumar et al. reported twenty cases of traumatic subdural empyema (SDEs) following minor head injury or unreported. In rare instances, SDH may indicate an underlying bleeding disorder, hematological malignancy or benign expansion of subarachnoid in 2008. The age of these children ranged from 1 month to 2 years. 70% children presented with subtle findings, 30% children presented with overt neurological signs and symptoms. Seizures were the most common mode of presentation.

Bilaterality and ventriculomegaly were more common in the subtle group, each with an incidence of 43%. 35% cases required operative management of traumatic SDEs. Recurrence was seen in 10% cases who had been conservatively managed previously. Only one child showed conversion of traumatic subdural hygroma to CSDH on conservative management [7]

Narsinghani et al. reported a case the dramatic presentation of a strikingly large calcified CSDH in a 5-year-old child with increased intracranial pressure and subfalcine herniation in 2002 [5].

Mori et al. reported that 12 patients with CSDH and arachnoids cyst were significantly younger than the patients with CSDH without arachnoid cyst in 2002. The most frequent symptom was headache followed by vomiting in the patients with arachnoid cyst, while gait disturbance and hemiparesis predominated in patients without arachnoid cyst. CSDH formation may be preceded by subdural hygroma caused by the rupture of arachnoid cyst [8].

In 2007, Acakapo satchivi et al. reported an unusual case of cortical herniation into a CSDH. A 4-month-old boy with a history of Macrocrania and very large bilateral chronic SDHs underwent subduro-peritoneal shunt treatment shortly after presentation.

To the authors' knowledge, this is only the fourth report in the medical literature of cortical herniation through a chronic subdural membrane and the first in which successful treatment with a good outcome is described [9].

CSDH in infant after abusive head trauma is a serious form of child abuse that can lead to severe neuropsychological sequelae or death in infants [10].

Osaka H, et al. reported a 10-month-old male with glutaric aciduria type-1 (GA-1) in 1993. This patient showed frequent partial motor seizures, irritability, and involuntary movements, including oral dyskinesia at the age of 3 months. On admission, magnetic resonance (MR) scanning revealed a chronic subdural hematoma and widening of the bilateral insular cisterns. Urine organic acid analysis showed marked excretion of glutaric acid, 3-hydroxy glutaric acid and glutaconic acid, suggesting GA-1 [11].

New pathophysiologic aspect might have an impact on conservative treatment in future. In particular, detection of the angiogenic cytokines responsible for development of the well-known leaky vessels within the outer membrane of a hematoma might offer new and promising targets to be blocked by pharmacologic agents.

Recently, it was shown that the angiogenic properties of angiotensin converting enzyme inhibitors could reduce the rate of recurrence in CSDH, as well as level of vascular endothelial growth factor within the hematoma [12,13].

Subsequent studies have identified lower levels of all coagulation factors in CSDH fluid than plasma. Factor II, V, VII, VIII, and X are disproportionately depleted. These findings reflected a phase of accelerated fibrinolytic activity after the rapid and defective clot formation. The end result is a milieu of anticoagulant protein (chiefly FDPs) and depleted coagulation factors. Other author have suggested that the PAF derived from lysis of red blood cells may stimulate the synthesis and release of t- PA, as well as induce chemotaxis of inflammatory cells to the CSDH fluid and elevated plasma level of PAF in patient with CSDH versus healthy volunteers. The latter observation may suggest a systemic predilection to the development of CSDH.

Conclusions

Chronic Subdural Hematoma is disease reported in infants and elderly population. It is uncommon in the age group of 2-14 years. Most of the children present with symptoms of raised intracranial tension due to head injury, with no reported history of repeated head trauma, child abuse & coagulopathy. Chronic subdural collection should be considered as one of the diagnoses as prognosis of this disease is better. However due to scarcity of reports in literature, established guidelines are not available.

References

1. Hirashima Y, Endo S, Kato R, Ohmori T, Nagahori T, et al. (1994) Plateletactivating factor and development of chronic subdural haematoma. Acta Neurochir 129: 20-25.
2. Muller W, Firsching R (1990) Significance of eosinophilic granulocyte in chronic subdural hematomas. Neurosurge rev13: 305-308.
3. Wang HK, Chen HJ, Lu K, Liliang PC, Liang CL et al. (2010) A pediatric chronic subdural hematoma after dodgeball head injury. Pediatr Emerg Care. 26: 667-668. 4.
4. Gautschi OP, Gallay MN, Kress TT, Korte WC, Hildebrandt G (2010) Chronic subdural hematoma -assessment and management. Praxis (Bern 1994).
5. Narsinghani U, Burson G, Schmidt MB, Anand KS (2002) Case of the month. Symptomatic chronic calcified subdural hematoma in a 5-year-old child. J Ark Med Soc 98: 300-303.
6. Per H, Gümüş H, Tucer B, Akgün H, Kurtsoy A, Kumandaş S (2006) Calcified chronic subdural hematoma mimicking calvarial mass: a case report. Brain Dev 28: 607.
7. Kumar R, Singhal N, Mahapatra AK (2008) Traumatic subdural effusions in children following minor head injury. Childs Nerv Syst. 24: 1391-1396.
8. Mori K, Yamamoto T, Horinaka N, Maeda M (2002) Arachnoid cyst is a risk factor for chronic subdural hematoma in juveniles: twelve cases of chronic subdural hematoma associated with arachnoid cyst. J Neurotrauma 19: 1017-1027.
9. Acakpo-Satchivi L, Luerssen T (2007) Brain herniation through an internal subdural membrane: a rare complication seen with chronic subdural hematomas in children. Case report. J Neurosurg 107: 485-488.
10. Wuerfel nee Tysiak E, Petersen D, Gottschalk S, Gerling I, Gliemroth J, et al. (2012) Progression of chronic subdural haematomas in an infant boy after abusive head trauma. Eur J Paediatr Neurol 16:736-739.
11. Osaka H, Kimura S, Nezu A, Yamazaki S, Saitoh K, et al. (1993) Chronic subdural hematoma, as an initial manifestation of glutaric aciduria type-1. Brain Dev 15: 125-127.
12. Weighel R, Hohenstein A, Schlickum L, Weiss C, Schilling L (2007) Angiotensin converting enzyme inhibition for arterial hypertension reduces the risk of recurrence in patients with chronic subdural hematoma probably by an angiogenic mechanism. Neurosurgery 61: 788-792
13. Kankane Vivek, Jaiswal Gaurav and Gupta Tarun, A Pediatric Traumatic Chronic Subdural Hematoma: Rare Case Report and Review of Literature. J Spine Neurosurg 2015, 4:3.

Guidelines for authors

1. Ethics

The publication of an article in Romanian Neurosurgery is a direct reflection of the quality of the work of the authors. The prevention of publication malpractice is first the responsibility of every author and also of our editorial board. Authors must submit accurate information and sufficient details, presenting its objective significance; unethical behaviour is unacceptable.

Plagiarism in all its forms constitutes unethical publishing behaviour and is unacceptable.

For Romanian Neurosurgery the publication ethics and publication malpractice statement are consistent with the recommendations and guidelines of the Committee on Publication Ethics, the World Association of Medical Editors, the International Committee of Medical Journal Editors and Consolidated Standards of Reporting Trials.

Links:
Committee on Publication Ethics
(COPE): http://www.publicationethics.org
World Association of Medical Editors
(WAME): http://www.wame.org
International Committee of Medical Journal Editors
(ICMJE): http://www.icmje.org

2. Enclosed Letter

In addition to the manuscript, the Editorial Board should receive an enclosed letter containing the exclusive reservation of copyright guaranteed by all authors whose manuscripts have already been accepted. If the paper was completely or partially published or exposed previously, a copy or a photocopy of it should be also sent. The technical reports should contain a declaration concerning the financial sources that cover the costs necessary for instruments and methodology acquisition.
In order to illustrate different cases, photos of identifiable patients will not be published without their legal consent or that of their legal representative. The letter containing this consent together with the manuscripts should be sent to the editorial office.
If the author wishes his unpublished manuscripts returned, please note this in the enclosed letter.

3. Sending of manuscripts

Authors shall ensure that the article has been "spell and grammar checked" prior to the submission.
The manuscript sent for publishing must be submitted in English. The manuscript will be typed without formatting, with a 1-line space. Please enclose 1 copy of the manuscript, tables, graphics and photos. After publishing, the paper and pictures become the property of Romanian Neurosurgery.

There are no article processing charges (NO APCs) and no article submission charges.

4. Manuscript elaboration

Paper sent for publishing should be in accordance with international standards of manuscript submittance. These standards are mentioned in "British Medical Journal" 1988; 296: 404-405 or in "Annals of Internal Medicine" 1988; 108:258-265. The authors are responsible for the accuracy of the information contained in the essay.

4.1. The title page should contain the whole title of the essay and complete names of authors with their academical degrees. If it is necessary, the department, the hospital or the institution where the search has been undertaken, should be also mentioned.

4.2. Please include an additional page containing the title of the essay and the author responsible for correcting of any and of all mistakes and for maintaining correspondence. The address, phone and fax number should be included (e-mail be available).

4.3. A summary, no longer than 300 words, should be written on a separate page. Key-words, no more than 7, should be listed in alphabetical order on the same page. The use of keywords should be approved by the "Index Medicus".

4.4. Text. The introduction should specify the purpose of the paper. The content and the method should give a minute account of the work methodology so that the experiment conclusion could be reproduced and checked up on in other centres. The experiments and medical studies performed on human beings should respect the principles specified in "The Declaration of Helsinki", whereas the experiments done on animals should be in accordance with "The Principles Charta of Animal's Care and Use". The results should not contain references to previous studies. The discussions should reflect the main features of the research.

5. References

- typed on 1 line;
- quoted in alphabetical order;
- explanatory footnotes are not accepted;
- unpublished data and personal papers should be quoted inside the text and not in the bibliography;
- entries to the bibliography should appear in the following order: the authors, the title of the essay, the title of the periodical (abbreviated according to the list of abbreviation specified in the "Index Medicus"), the volume number, the page number and the data publication.

6. Tables and pictures

There should be 1 copy sent. Each table with its own title should be submitted on separate pages. The photos, radiographies, CT scans should be labelled on the back with the number of each picture, corresponding with the number

included inside the text, and the author's name. The label will be placed on the top of the picture. The drawings may be sent on vellum paper, tracing paper or transparent paper. The use of pictures belonging to other publications is accepted, only if mentioning the original source. Further, the partial use of any previously published text is accepted with the approval of its author and editor. All pages should be consecutively numbered, starting with the title page.

7. Abbreviations

Abbreviations should be consistently used throughout the text and established in a fixed form from the beginning.

8. Submission

Manuscripts should be sent to the Editor:
Dr St.M. Iencean,
Romanian Neurosurgery
"Gr.T. Popa" University of Medicine and Pharmacy
Universitatii Street, No. 16, 700115, Iasi, Romania
or by e-mail to ***mirceasteffan@yahoo.com***.

9. Disclaimer

The editors disclaim any responsibility for opinions expressed in the papers.

10. The review process

After the manuscript submission, the peer review process is broken down into the following steps:
1. The Editor assigns Reviewers to the manuscript.
2. The Reviewers review the manuscript.
3. The Editor drafts a decision to be sent to the author/authors.

The review process takes between three weeks and two months.