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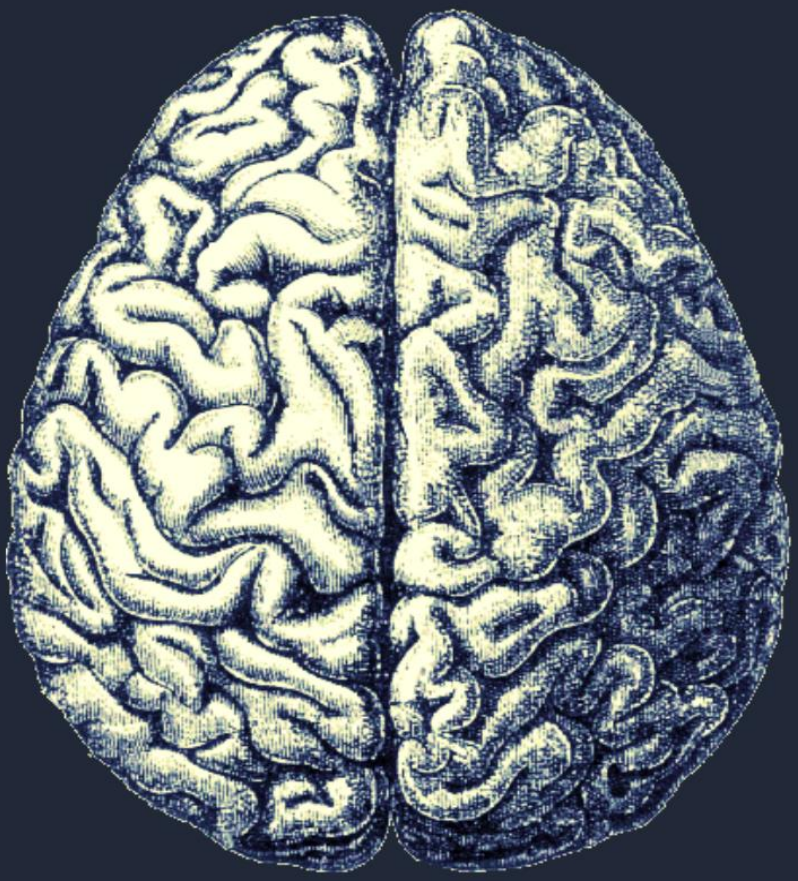

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Post-traumatic epileptic seizures revealing hydrocephalus

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Keywords
cerebrospinal fluid, epilepsy, head trauma, hydrocephalus, ventriculo-peritoneal shunt

ABSTRACT

Background: Hydrocephalus is one of the late complications of head injury. More often, post-traumatic epileptic seizures are due to intracranial hematomas and brain contusion. Hydrocephalus is rarely reported in this clinical registry. The literature is not very rich in cases of epileptic seizures linked to posttraumatic hydrocephalus.

Objective: We report the results of our study, the aims of which were to establish the epidemiology of post-traumatic hydrocephalus in our practice, to determine the mechanisms of trauma onset, to describe the initial traumatic lesions and to highlight the timing and pathophysiological mechanism of epileptic seizures in post-traumatic hydrocephalus.

Materials and methods: We conducted a descriptive, retrospective study over a period of 4 years at the Neurosurgery Department of the Gabriel Touré University Hospital in Bamako. We included patients of both genders and all ages who had at least one seizure after head injury and in whom the CT scan performed during the investigation of the seizure showed hydrocephalus.

Results: Between 1 January 2020 and 31 December 2023, out of 1024 cases of traumatic brain injury (TBI), hydrocephalus was diagnosed in 37 patients (3.6%) during the investigation of epileptic seizures. All patients were male with a mean age of 27.4 years. The trauma was caused by road traffic accidents in 48.6%. The initial brain injuries were dominated by hemorrhagic contusions (29.7%), osteo-meningeal injuries caused by fracture of the frontal bone (19%) and traumatic subarachnoid haemorrhage (16.2%). The mean time to onset of the first attack was 28.3 days. Seizures were generalised in 86.4%. Sudden onset without prodrome occurred in 64.8% of patients. Tetraventricular hydrocephalus was diagnosed in 89.1% of cases. Ventriculoperitoneal shunting was performed in all patients. After 5 months of follow-up, seizures had completely resolved in 87.5% of patients despite complete cessation of antiepileptic treatment. No deaths were reported.

Conclusion: Epilepsy may be a clinical manifestation of post-traumatic hydrocephalus. Morphological imaging is essential for diagnosis. Management is based on cerebrospinal fluid drainage.

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INTRODUCTION

Head injury is a major public health problem in our developing countries. It is responsible for late complications including hydrocephalus. Most often in the torus, all post-traumatic hydrocephalus is attributed to subarachnoid hemorrhage. Hydrocephalus secondary to head trauma can occur outside of any subarachnoid hemorrhage. The etiology can be multiple with varied mechanisms. Post-traumatic hydrocephalus (PTH) occurs in a significant number of patients after head trauma and may affect the outcome of head injury (32). It is a complication of head injury and refers to excessive accumulation of cerebrospinal fluid (CSF) in the brain ventricles due to altered CSF dynamics (32). Post-traumatic hydrocephalus occurs in 0.7% to 29% of patients with head injury (8, 10, 21, 30, 38). In children, the incidence ranges from 0.07% to 1% (6, 16, 33). Posttraumatic epilepsy accounts for approximately 20% of the causes of epilepsy in the general population and 5% of all patients seen in specialized epilepsy centers (2, 26).

Objectives

The aims of this study were to establish the epidemiology of this condition in our practice, to determine the mechanisms of trauma onset, to describe the initial traumatic lesions and to highlight the delay and the physio-pathological mechanism of epileptic seizures in posttraumatic hydrocephalus.

MATERIALS AND METHODS

We conducted a retrospective descriptive study over 4 years, from 1 January 2020 to 31 December 2023. It took place in the Neurosurgery Department of the Gabriel Touré University Hospital Centre in Bamako. It focused on patients with head injuries. Patients of both sexes and all ages who had at least one epileptic seizure after a head injury and whose CT-scan confirmed the diagnosis of hydrocephalus were included in the study. Patients with a history of previous head injury, cranial surgery, stroke or epilepsy were excluded from the study. Data were collected from medical records and the operative report registry. SPSS version 2.0 software was used for data processing. Sociodemographic parameters, causes and mechanisms of head injury, time to onset and treatment modalities were examined.

RESULTS

From 1 January 2020 to 31 December 2023, 1024 patients with head injury were treated in the department. Of these, 37 patients had epileptic seizures, the investigation of which led to the diagnosis of hydrocephalus. This corresponds to a prevalence of 3.6% and an average of 9.3 patients per year. The mean age of the patients was 27.4 years (extremes 17 and 49 years). All the patients were men. Motorcycle mechanics were most affected (21.6%), followed by students (19%) and workers (13.5%). Table I shows the distribution of patients according to their occupation. Traffic accidents were responsible for 48.6% of head injuries, followed by falls from height (29.7%), assaults (18.9%) and accidents at work (2.7%). Traffic accidents were caused by loss of control of the motorcycle in 19% of cases (7 cases), a collision between a motorcyclist and a car in 10.8% of cases (4 patients), a pedestrian hit by a motorcyclist in 10.8% of cases (4 cases), and a car overturning with passengers in 8.1% of cases (3 patients).

Table 1: Distribution of patients according their occupations

Occupations	Number	Frequency (%)
Motorcycle Mechanic	8	21,6
Student	7	19
worker	5	13,5
Farmer	4	10,8
School pupil	4	10,8
Military	4	10,8
Male nurse	2	5,4
Trader	3	8,1
Total	37	100

Table 2. Distribution of patients by mechanism of trauma brain injury

Head injury mechanisms		Effective	Frequency (%)
Public road accidents	Motorcycle fall	7	19
	Motorcyclist-car collision	4	10,8
	Pedestrian hit by motorcyclist	4	10,8
	Overturned car with passengers	3	8,1

Fall from a great height	Fall from scaffolding	5	13,5
	Falling tree	3	8,1
	Fall down stairs	2	5,4
	Fall from ladder	1	2,7
Assaults and battery	with a stick	3	8,1
	with stones	1	2,7
	with an iron bar	1	2,7
	with a gas cylinder	1	2,7
	with a stepladder	1	2,7
Work accident	receiving a bag of coal on the head	1	2,7
Total		37	100

Falls from heights were due to falls from scaffolding in 5 patients (13.5%), from trees 8.1% (3 cases), stairs 5.4% (2 cases) and ladders in 2.7% (1 case). Assaults were perpetrated using blunt objects such as sticks in 3 cases (8.1%), stones, iron bars, gas canisters and stepladders in one case each (2.7%). The only accident at work was due to a head impact with a bag of coal. The mechanisms of head trauma are detailed in Table II. Clinically, 29 patients (78.4%) presented with an initial loss of consciousness of short duration. On admission to the emergency department, 19 patients had a Glasgow score of 15, six patients (16.2%) had a Glasgow score of 13, and two other patients had a Glasgow score of 12. There were two cases of non-febrile meningeal syndrome. None of the patients had pupillary abnormalities. None of the patients had focal motor deficits. Initial brain computed tomography (CT) showed edemato-hemorrhagic contusion in 11 patients (29.7%) (Figure 1) and osteomeningeal breach due to fracture of the anterior skull base associated with pneumocephalus in 7 cases (19%) (Figure 2).

Traumatic subarachnoid hemorrhage was found in 6 patients (16.2%) (Figure 3). CT scans were normal in 35.1% of cases (13 patients). Within ten days of the trauma, 3 patients (8.1%) had developed infectious meningitis, with hyperthermia, headache associated with stiff neck and vomiting in one patient. Analysis of the cerebrospinal fluid (CSF) after lumbar puncture failed to isolate any germs (bacteria). The mean time to onset of the first seizure was 28.3 days, with extremes of 17 and 41 days after head injury. The number of seizures prior to diagnosis was known in 15 patients. There was one seizure in 4 patients (10.8%), 3 seizures in 2 patients (5.4%) and 6 to 8 seizures in 9 patients (24.3%).

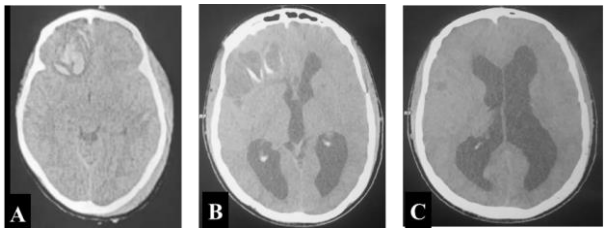

Figure 1. Evolution of brain contusion towards hydrocephalus on brain CT-Scan:
A. Post-traumatic right frontal edemato-hemorrhagic contusion.
B. Dilatation of the lateral ventricles and the 3rd ventricle and the stigmata of the right frontal contusion.
C. Periventricular hypodensity in the frontal horns (trans-ependymal suffusion).

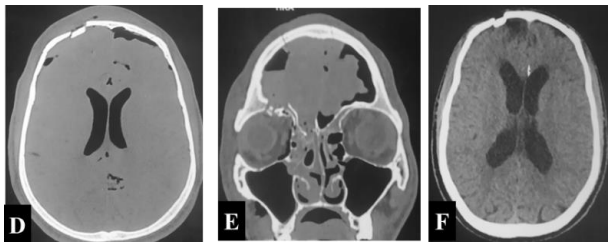

Figure 2. Occurrence of hydrocephalus on an osteomeningeal gap on brain CT-Scan:
D. Right frontal fracture with diffuse pneumocephalus extending to the lateral ventricles.
E. Coronal reconstruction, bony window, diffuse pneumocephalus reaching the base of the skull.
F. Ventricular dilatation associated with peri-ventricular hypodensity of the frontal horns (trans-ependymal suffusion).

Seizures were generalized tonic-clonic in 86.4% of cases (32 patients). They had a sudden onset without prodrome in 24 patients (64.8%). They were preceded by headaches in 8 patients (21.6%). Five patients (13.6%) had secondary partial generalized seizures. Clinical signs associated with seizures were progressive. They included intermittent headaches of moderate intensity in 13 cases (35.1%), vomiting in 8 patients (21.6%) and slowed thinking in 3 cases (8.1%). One patient (2.7%)

presented gait disorders, with small steps and widening of the weight-bearing polygon.

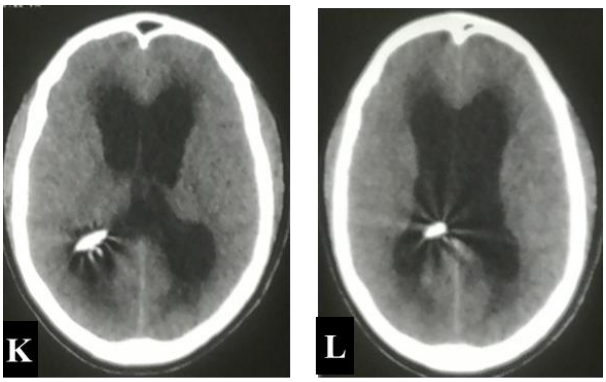

Figure 3. Early postoperative monitoring of hydrocephalus due to traumatic meningeal hemorrhage on cerebral CT-scan:
K. Presence of the catheter in the occipital horn of the right lateral ventricle.
L. Visualisation of the tip of the catheter at the level of the ventricular carrefour not far from the septum pellicidum.

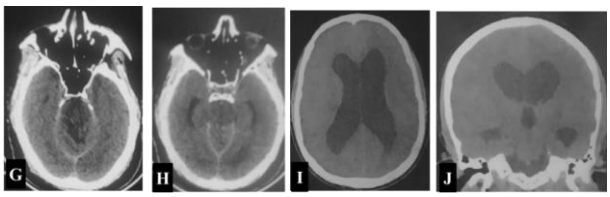

Figure 4.

Fundus examinations were performed in 7 patients (19%). Papilledema was observed in 6 patients (16.2%). An electroencephalogram (EEG) was performed in 11 patients (29.7%). It revealed generalized epileptiform abnormalities in 8 patients (21.6%) and temporal seizures in 2 patients (5.4%). It revealed active hydrocephalus in the form of significant ventricular dilatation associated with diffuse periventricular hypodensity reflecting transependymal suffusion in all patients (figures 1-C; 2-F). Hydrocephalus was tri-ventricular in 4 patients (10.8%) and tetra-ventricular in the remaining 33 (89.1%). Prior to neurosurgical treatment, 29 patients (78.4%) had received oral antiepileptic therapy. These included sodium valproate in 14 patients, phenobarbital in 9 and Levetiracetam in 6. Treatment was initiated on the advice of neurologists. Six patients (16.2%) were treated by traditional medicine.

This was a non-medical treatment consisting of drinking herbal teas and decoctions of medicinal plant leaves or barks. Two patients (5.4%) received no regular treatment. The mean time from diagnosis of hydrocephalus to surgical treatment was 7.3 days, with extremes of 2 and 16 days. Surgery involved ventriculo-peritoneal diversion of cerebrospinal fluid using a medium-pressure valve in all patients (Figure 4-K; 4-L). Concurrently with surgery, patients received a short course of corticosteroids for 3 to 7 days immediately after surgery. Anti-epileptic treatment was gradually reduced over 10 days in 11 patients (37.9%). After 3 weeks, antiepileptic therapy was discontinued in 22 patients (75.8% of cases). Two months later, three patients (8.1%) reported a recurrence of seizures. In one of them, the seizures were partial, and in the other two, they were generalized. They were put back on sodium valproate. After 3 months' follow-up, one patient showed significant ideomotor slowing (2.7%). Radiological studies showed a recurrence of hydrocephalus due to valve dysfunction. Revision surgery was performed. After 5 months' follow-up, 13 patients were lost to follow-up (35.1%), one patient still had ideomotor slowing and 2 patients were still on antiepileptic monotherapy. Progression was favourable in 21 patients (87.5%), characterized by a complete absence of seizures despite complete cessation of antiepileptic therapy. No deaths were reported.

Discussion

General and epidemiological data

Post-traumatic hydrocephalus has been known since Dandy's report in 1914 (21). It is a sequela of head trauma (32). It occurs in a significant number of patients following head trauma and may influence the outcome of head trauma (32). Its incidence ranges from 0.7 to 29% (8, 10, 21, 30, 38). This complication is characterised by a progressive accumulation of cerebrospinal fluid (CSF) in the ventricular cavities of the brain, which compresses the neurological structures within the skull (21, 38).

Pathophysiology

Head trauma may be responsible for the disruption of the normal dynamic balance between CSF production and absorption (29, 34). Clot formation following intraventricular or subarachnoid hemorrhage leads to obstruction of the CSF pathway, causing acute hydrocephalus (9, 29). This subarachnoid hemorrhage may also be responsible for the formation of adhesions in the basal cisterns

and inflammation of the arachnoid villi, leading to impaired CSF absorption and the development of chronic hydrocephalus (34). Sixteen point two percent of patients in our series presented with traumatic subarachnoid hemorrhage. As with subarachnoid hemorrhage, meningitis causes inflammation of the arachnoid villi, which reduces CSF absorption and leads to fibrosis (1). Post-traumatic meningitis affected 8.1% of patients in our cohort. The pathophysiological mechanisms of epileptic seizures associated with hydrocephalus are complex and multifactorial. Intraventricular hyperpressure due to CSF accumulation leads to dilatation of the cerebral ventricles. This causes compression and stretching of the surrounding brain tissue.

This disturbance can lead to desynchronization of the brain's electrical activity, favouring the triggering of epileptic seizures. Increased intracranial pressure can also reduce cerebral blood flow, leading to local ischemia. Lack of oxygen and nutrients can damage neurons and disrupt ionic balance, increasing the risk of seizures. Transependymal suffusion, the diffusion of cerebrospinal fluid through the ependyma of the ventricles, is responsible for inflammation of the brain tissue and the formation of gliosis. These changes can alter neural networks and create an environment conducive to epileptic seizures. CSF imbalances can also affect the concentration and dynamics of neurotransmitters such as glutamate and GABA, which play a key role in neuronal excitability. Too much excitation or too little inhibition can trigger seizures (7, 24, 28, 40,). Neuro-inflammatory pathways and neurochemical imbalances play a crucial role in the development and progression of this pathology.

Following head trauma, activation of glial cells (microglia and astrocytes) leads to the release of pro-inflammatory cytokines such as TNF-α, IL-1β and IL-6. This inflammatory response can lead to disruption of normal brain function and contribute to CSF accumulation. The resulting inflammatory response could alter the permeability of the blood-brain barrier. This allows pro-inflammatory molecules and immune cells to enter brain tissue, exacerbating brain damage and influencing CSF production and circulation. Activation of inflammatory signalling pathways such as NF-κB (nuclear factor kappa B) and MAPK (mitogen-activated protein kinase) can influence cytokine production and the cellular response to trauma. After head trauma, neurotransmitter levels can be disrupted. Excess glutamate can lead to excitotoxicity. The presence of inflammatory proteins or changes in ependymal cells can disrupt CSF metabolism (5, 19, 25, 32).

Clinical presentation

Whether or not associated with head trauma, hydrocephalus in its acute form may manifest as intracranial hypertension (ICH). This results in the onset of headaches of increasing intensity, most commonly in the morning, accompanied by light and jerky vomiting which may (temporarily) relieve the patient, and visual disturbances ranging from a decrease in visual acuity to blindness due to optic atrophy (4, 21, 22 ,41). In the chronic form, the clinical manifestations are those of the Adams-Hakim triad, characterized by progressive cognitive decline, gait disturbances consisting of short steps, widening of the supporting polygon and urinary syndrome (3, 12, 18, 21). The occurrence of epileptic seizures in hydrocephalus is not common. The few articles reported on the subject in a traumatic context are clinical cases (3). Our series is one of the rarest with such a large cohort. In addition to seizures, 56.7% of patients in our study presented with signs of ICH. These were headache (35.1%) associated with vomiting (21.6%). The Adams-Hakim triad was observed in one patient. According to the authors, this clinical manifestation is most often secondary to subarachnoid hemorrhage (9, 29). On the other hand, this does not explain the presence of the same signs in patients presenting with post-meningitic hydrocephalus due to traumatic osteomeningeal rupture.

Treatment options

CSF diversion is the treatment of choice for hydrocephalus regardless of the cause. In our study, ventriculo-peritoneal shunt (VPS) was performed in all patients. It consisted of connecting the cerebral ventricular system to the peritoneal cavity by means of a medium pressure valve. Some authors had proposed external ventricular drainage of CSF for the treatment of acute hydrocephalus (36). This approach is indicated for acute hydrocephalus associated with ventricular hemorrhage or active infectious meningitis.

Authors agree on the need for CSF diversion in post-traumatic hydrocephalus (13, 14, 27, 31). Ventriculo-peritoneal shunt diversion remains the surgical technique of choice because of the reduced compliance due to the condition and the lack of CSF resorption (6, 20, 35). In our series, only fixed and medium pressure valves were used. The use of other valve types, namely programmable valves or the flow-regulated valve system, has been reported (17, 41). The success of surgical treatment is assessed by regression or complete resolution of signs.

A gradual normalization of intracranial pressure would eliminate the compression of neurological structures inside the skull. This improvement led to the total and definitive cessation of antiepileptic treatment in 75.8% of patients in our study after 3 weeks. The placement of a shunt brought a clear clinical improvement in patients with hydrocephalus who had a low Glasgow score after the trauma or post-traumatic meningitis (35, 39). The inflammation of the periventricular cerebral cortex by the transependymal suffusion of the CSF also involved in the genesis of the seizures had justified the short-term corticosteroid therapy administered to all patients in the immediate postoperative period thanks to its anti-inflammatory effect. Some complications may occur during treatment. These include corticosteroid-induced diabetes, of which we have not recorded any cases, and valve dysfunction. Valve disconnection is a mechanical failure of the bypass system. One case occurred in our cohort and required reoperation for valve revision.

The valve revision rate in the literature ranges from 17.9% to 24% (6, 35). Some authors had proposed endoscopic surgery for internal drainage of CSF in post-traumatic hydrocephalus (11, 15). The rationale for performing this endoscopic ventriculocisternostomy (ETV) was that it could restore intracranial compliance by reducing CSF stagnation in the ventricular system through an "internal shunt". A review of the literature suggested that ETV should be reserved for cases of obstruction of the midbrain aqueduct by hemorrhage of traumatic origin, or in cases of shunt dysfunction or repeated infection of the ventriculo-peritoneal drainage (15, 35, 36). This ETV may fail. A study from India reported a failure rate of 60% in the treatment of post-traumatic hydrocephalus (35). In the same series, only 37% of patients treated with VCS showed improvement compared to 73% after ventriculo-peritoneal shunt placement (35). In our cohort, all patients had undergone VPS placement with a favourable outcome. Medium-pressure valves had been the type of shunt used in all the patients in our series. According to Svedung Wettervik and al, patients with medium pressure hydrocephalus (chronic adult hydrocephalus) have a better response to ventriculo-peritoneal shunt (37).

Conclusion

Epilepsy may be the mode of expression of intracranial hypertension in patients after head trauma. Hydrocephalus may be the cause. In any case of post-traumatic seizures, morphological imaging becomes essential even if the first imaging was normal so as not to miss hydrocephalus. Effective and early treatment of this hydrocephalus can save patients from having to take antiepileptic drugs for a very long time.

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A narrative review in managing ICA aneurysms

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ABSTRACT

Introduction: Internal carotid aneurysms are among the most common intracranial aneurysms, with nearly 20-25% of the ruptured aneurysms patients succumbing to death before reaching hospital. Most aneurysms are discovered in the context of an SAH, unruptured aneurysms can also be detected in patients presenting with other clinical symptoms or incidentally through neuroimaging**.**

Materials and methods: We searched Google and indexed articles with the keywords "ICA aneurysm management". We took 45 articles to identify epidemiology, diagnosis and management of these aneurysms. Both microscopic and endovascular treatments were taken into account.

Results: Clipped aneurysms had a relatively lower percentage of recurrences as compared to endovascular modality, while the incidence of peri-procedural complications was higher with clipping patients.

Conclusion: The volume of cases an institution handles influences the procedure's success. Endovascular management of ICA aneurysms has recently gained in numbers compared to open procedures.

Keywords
ICA aneurysm, clipping, coiling

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INTRODUCTION

Cerebral aneurysms are localized dilatations occurring at weakened areas within the brain's arterial circulation. They often occur at the branching points of smaller vessels and are usually saccular in shape, but they can also have fusiform or blister-type shapes. These aneurysms can vary in size, with small aneurysms being less than 0.5 mm, medium ranging from 6 to 25 mm, and large ones exceeding 25 mm. The majority are saccular (berry-shaped), characterised by a thin or absent tunica media and a severely fragmented or absent internal elastic lamina. Less commonly, aneurysms may be fusiform (circumferential) or mycotic (infectious). Most cerebral aneurysms are asymptomatic and are often discovered incidentally during neuroimaging or autopsy (1,2). Around 85% of these aneurysms are found in the anterior circulation, primarily at junctions or bifurcations along the circle of Willis. When rupture occurs, it often leads to subarachnoid haemorrhage (SAH), which is associated with high morbidity and mortality (2).

Wiebers. et al. (2003), in the International Study of Unruptured Intracranial Aneurysms (ISUIA), reported that the most common location for a cerebral aneurysm was the Internal Carotid Artery (29.9%), followed by the Middle cerebral artery (29%) (3). Bouthillier's classification (1996) described seven parts of the Internal Carotid artery based on anatomy which was based on the original Fischer's classification.C1, cervical; C2, petrous; C3, lacerum; C4 cavernous; C5, clinoid; C6, ophthalmic; and C7, communicating (4).

Paraclinoid aneurysms are complex intracranial aneurysms arising from the internal carotid artery proximal to the posterior communicating artery and distal to the distal dural ring (DDR). They have complicated anatomy and project surgical difficulty (5). Paraclinoid aneurysms have a variable relationship with the distal dural ring that cannot be determined radiographically.

Paraclinoid aneurysms spanning the distal dural ring are partially in the subarachnoid space and at risk of subarachnoid haemorrhage. The paraclinoid aneurysms account for 5-10% of all intracranial aneurysms (6). Paraclinoid aneurysms are divided into four types based on the lateral view of ICA angiogram: Carotico-ophthalmic aneurysms (COA), Dorsal (D) or anterior wall aneurysms, Ventral (V) or posterior wall aneurysms and Transitional (T) aneurysms.

Epidemiology

Internal carotid artery aneurysms are believed to represent 30% to 50% of all intracranial aneurysms (3). Research suggests that patients with ICA aneurysms are more likely to have multiple aneurysms (7). They are more frequently seen in women and are typically diagnosed in individuals in their fifties or sixties (8).

Clinical manifestation

Approximately half of these aneurysms present with subarachnoid haemorrhage or symptoms caused by mass effect, while the other half are found incidentally (8). Subarachnoid haemorrhage (SAH) is often a catastrophic event. Around 20 to 25 per cent of patients succumb before arriving at the hospital, and among those who do receive timely medical care, only about one-third experience a favourable outcome post-treatment (9,10). Most SAHs result from ruptured intracranial saccular (berry) aneurysms (11,12).

Diagnosis

The majority of cerebral aneurysms are unruptured and are asymptomatic clinically. However, a ruptured cerebral aneurysm presents with a sudden onset headache, which is severe in intensity (thunderclap headache). In about 30% of cases, the pain tends to be localised on the ipsilateral side. Sudden death can happen in 10 to 15% of patients. Physical examination may reveal signs of raised ICT, i.e. elevated blood pressure, dilated pupils, visual field or cranial nerve deficits, etc. The Hunt and Hess grading system, commonly used to predict patient outcomes based on their initial neurological status, consists of 5 grades, reflecting symptoms' severity and correlating with mortality rates (2). While most aneurysms are discovered in the context of an SAH, unruptured aneurysms can also be detected in patients presenting with other clinical symptoms or incidentally through neuroimaging. Unruptured cerebral aneurysms are often detected on MRI, CTA, or conventional angiography; however, ruptured aneurysm resulting in subarachnoid haemorrhage (SAH) can be detected on a non-contrast CT(NCCT) brain. Once SAH is diagnosed, the bleeding source must be identified through CTA, MRA, or digital subtraction angiography (DSA) (2).

Treatment

The management of the ventral ICA aneurysm can be broadly divided into two approaches: Open surgical and endovascular approach. Anatomical factors—including size and location—and other shape-related characteristics often play a crucial role in determining the most suitable treatment for a patient.

Microsurgical approach

The surgical management of cerebral aneurysms, involving the placement of a clip across the aneurysm's neck, can be used in both unruptured or ruptured aneurysms.

It involves ipsilateral Pterional craniotomy followed by removal of the anterior clinoid process, Extradural drilling of the optic strut and then linear dural opening along the Sylvain fissure, which is followed by distal dural ring dissection, then

Proximal dural ring dissection and lastly application of clip under the ventral ICA.

For aneurysms projecting inferiorly or medially, extradural clinoidectomy is safe and may require less time than intradural clinoidectomy

Inferiorly projecting aneurysms can be clipped with an up-curved clip rather than the usual fenestrated clips.

Clipping requires complete visualisation of the aneurysm, so the rate of intraoperative difficulties and complications like inadequate exposure, injury to brain tissue, vessel injury leading to haemorrhage, and vessel occlusion causing ischemia is relatively high. Taha et al., in a centre retrospective analysis of cases undergoing surgical clipping, found a notable 19.35% incidence of periprocedural technical complications (13).

Clipping lowers the incidence of residual and recurrent aneurysms. Akyuz et al. (2004) followed 136 patients with 166 aneurysms at an average of 46.6 months after surgery and found that 5.1% of aneurysms had residuals. Still, no recurrences were observed, and Brown et al. (2017), in 431 ruptured and 327 unruptured aneurysms, found that 7.8% had residual aneurysms on early postoperative imaging after one month, with a single recurrence detected at an average of 7.2 years post-discharge (14,15).

Several studies were done to assess survival, morbidity, and mortality rates associated with surgical clipping for unruptured aneurysms. Britz et al. (2004), in a retrospective study, found higher survival rates among patients who underwent clipping, with a 2.3% risk of death due to neurological causes (16). The International Study of Unruptured Intracranial Aneurysms (ISUIA), led by Wiebers et al. (2003), reported an overall morbidity and mortality rate of 11% to 13.7% and 10.1% to 12.6% at 30 days and one-year post-surgery, respectively (3). In a study, Ogilvy et al. (2003) found an overall mortality rate of 0.8% and a morbidity rate of 15.9% (17). Morbidity in these studies encompassed long-term neurological deficits, residual or reformed aneurysms, bleeding, and ischemic stroke due to vessel occlusion.

Mortality and morbidity rates significantly increase when a ruptured aneurysm is treated through surgical clipping. According to the International Subarachnoid Aneurysm Trial, a randomised controlled trial conducted by Molyneux et al. (2005), the combined morbidity and mortality rate was 30.6% in the surgical treatment group. Additionally, the trial demonstrated an absolute risk reduction of 6.9% in dependency or death among those treated surgically (18).

The volume of cases an institution handles also influences the procedure's success. Rinaldo et al. (2017) discovered a negative correlation between case volume and complication rates (19). Barker et al. (2003) conducted a retrospective cohort study. They found that high-volume hospitals, which treated 20 or more cases annually, discharged 84.4% of patients and had a mortality rate of 1.6%. In contrast, low-volume hospitals (handling fewer than four cases per year) discharged 76.2% of patients and had a higher mortality rate (2.2%) (20). With the advent of less invasive procedures like coil embolisation, surgical clipping has become less prominent in managing intracranial aneurysms. Despite ongoing advancements in transcranial approaches and clipping techniques, coiling has emerged as the preferred treatment option in many institutions; nonetheless, clipping remains essential when coiling is not feasible, such as in cases involving very large aneurysms (21).

ENDOVASCULAR APPROACH

There is a majority of endovascular approaches, which include coil embolisation and newer techniques like stent-assisted coiling, balloon-assisted coiling, flow diverters, disruptors, and new embolic materials.

Coil Embolization involves the insertion of platinum coils into the aneurysm's lumen, forming a local thrombus around the coils, effectively obliterating the aneurysmal sac. The procedure begins with gaining vascular access, usually through a peripheral artery like the femoral artery, and then locating the aneurysm, followed by inserting a detachable platinum coil in the aneurysm. Once the coil is placed in the aneurysm, clot formation is triggered. It is followed by dye injection to look for any position of the coil, condition of the parent artery and residual blood flow to the aneurysm. The procedure can be performed under general anaesthesia or sedation (22,23).

Coiling is a relatively new approach to treating intracranial aneurysms. Guglielmi detachable coil was developed and approved in the 1990s for unruptured aneurysms after a study by Eskridge et

al. (1998), who examined 150 cases of both ruptured and unruptured aneurysms that were unsuitable for surgical intervention. They suggested that selected patients(not candidates for clipping) had better outcomes regarding reduced morbidity and mortality than those managed conservatively (24).

The Raymond-Roy Occlusion Classification (RROC) is the standard method for evaluating the success of coiled aneurysms (25). Mascitelli et al. (2015) refined RROC and divided class III into classes IIIa and IIIb. They found that class IIIa aneurysms had a significantly higher likelihood of progressing to class I or II than class IIIb aneurysms (83.3% vs. 14.9%) (25).

Since its initiation, the coiling technique has undergone substantial advancements. Nguyen et al. (2008) assessed aneurysms treated with coiling between 1992 and 2007 and found a rupture rate of 11.7% in very small aneurysms 3 mm or smaller, compared to just 2.3% in larger aneurysms (21,26).

Advances such as softer and smaller coils (around 1 mm in diameter) have made it safer to treat smaller aneurysms. In contrast, larger-diameter coils have been developed to pack larger aneurysms more efficiently (27).

The Analysis of Treatment by Endovascular Approach of Unruptured Aneurysms (ATENA) study, led by Pierot et al. (2008), assessed the effectiveness, morbidity, and mortality rates of coiling in multicenteric prospective study involving 649 patients with 1,100 unruptured aneurysms. They found that procedure-related adverse events occurred in 15.4% of patients, and morbidity and mortality rates after one month were 1.7% and 1.4%, respectively. They concluded that coiling has low morbidity and mortality rates (28).

Numerous studies have explored the safety and effectiveness of coiling, but all found that coiling has a low rate of complete occlusion. Gallas et al. (2008) reported a 70% immediate total occlusion rate in a retrospective study, with a 26.1% subtotal occlusion rate and 14.4% treatment-related morbidity (29). Similarly, Bradac et al. reported a 64% complete occlusion rate, a 34% nearly complete rate, and a 13% complication rate (30). Murayama et al. reported a 55% complete occlusion rate and a 35.4% neck remnant rate, with a 20.9% recanalisation rate primarily linked to the size of the aneurysm's dome and neck and 1.6% delayed aneurysm rupture (31). The International Study of Unruptured Intracranial Aneurysms (ISUIA) found that treatment-related morbidity and mortality were higher in patients with prior subarachnoid haemorrhage than in those without (9.8% vs. 7.1%) (3).

Stent-assisted embolisation as a treatment for intracranial aneurysms was first introduced by Henkes et al. (2002) in a multi-centric prospective study (32).

It was the initially accepted treatment method for unruptured ICA aneurysms with a broad neck and unfavourable neck-to-fundus ratio (dome-to-neck ratio less than 2 or a neck length of 4 mm or more). It was difficult to treat by surgical or traditional coiling. Stent-assisted coil embolisation facilitates proper coil placement while preventing the coils from protruding into the parent vessel. Moreover, intracranial stents may reduce the likelihood of aneurysm recanalisation (33). Stents are positioned in the artery, so antiplatelet regimens are required to prevent arterial thromboembolic complications, making using stents more challenging in cases of recently ruptured aneurysms (34).

Bechan et al. (2016), in a prospective observational study on 45 patients with ruptured and 47 patients with enraptured aneurysms, found that complication rates like visible thrombus, vessel occlusion, rebelled were 10 times more common when Stent assisted coil embolisation was done in ruptured aneurysm as compared to enraptured aneurysm (35).

Recently, its use has expanded to treat all types of aneurysms (34). Coiling is performed mainly in general anaesthesia.

Firstly, stent placement is simulated using computer graphics on the 3D dataset with standard machine software. The software accurately calculated the diameters and length of the targeted vessel segment, allowing for selecting the appropriate stent size and length. A microcatheter is used to navigate past the aneurysm. After placing the stent, the delivery microcatheter is removed, and a lower-profile microcatheter is introduced through the stent struts into the aneurysm. Coils are then deployed to occlude the aneurysm (35).

In a randomised clinical trial, Boisseau et al. (2023) assessed the superiority of Stent-assisted coiling over coiling alone in treating unruptured cerebral

aneurysms in a 10-year study on 205 patients. They found that Stent-assisted coiling did not offer any advantage over traditional coiling in terms of recurrence of the lesions, intracranial bleeding, retreatment, modified Rankin scale of 3-5 or death (34).

Newer endovascular techniques have emerged that may complement or replace coiling. Liquid embolics, particularly OnyxHD500, have gained attention for treating wide-neck aneurysms (27). Dalyai et al. (2011) achieved a 90% complete occlusion rate in patients with wide-neck aneurysms who were unamenable to coiling alone (36).

Additional innovations, including endoluminal flow diversion and aneurysmal neck reconstruction, are being evaluated as treatment options (37,38).

Balloon-assisted coiling, also known as the remodelling technique, was first introduced by Moret et al. (1997) to extend endovascular treatment (EVT) to wide-neck intracranial aneurysms. In this technique, a non-detachable balloon is temporarily inflated across the aneurysm neck during the placement of each coil. The balloon is positioned within the parent vessel adjacent to the aneurysm neck for sidewall aneurysms. Once the coiling is completed, the balloon is deflated and removed unless stenting is required as a follow-up procedure (39,40). A 2006 single-centre retrospective study on both ruptured and unruptured aneurysms suggested that BAC was linked to a higher complication rate. Specifically, the rates of thromboembolic events and intraoperative ruptures in the BAC group were 9.8% and 4.0%, respectively, compared to 2.2% and 0.8% in the coiling-alone group (41). However, more recent data from two large multicenter prospective studies have provided a clearer picture. In the ATENA study (which focused on unruptured aneurysms), the rate of thromboembolic events was similar between the BAC group (5.4%) and the coiling-alone group (6.2%) (28).

Additionally, the rate of intraoperative rupture was 3.2% in the BAC group and 2.2% in the coiling-alone group. Clinical outcomes were also comparable, with a permanent deficit or death in 0.6% of the coiling group and 1.4% of the BAC group. Morbidity rates were 2.2% for coiling alone and 2.3% for BAC, while mortality was 0.9% and 1.4%, respectively (28,42).

In the CLARITY study (which focused on ruptured aneurysms), the two groups' thromboembolic event rates were similar: 12.7% in the coiling group and 11.3% in the BAC group. The rate of intraoperative rupture was 4.4% for both groups, with morbidity and mortality rates being comparable as well (43).

The impact of BAC on anatomical outcomes remains uncertain. One study found that aneurysms treated with BAC had a higher rate of incomplete occlusion (27.7%) compared to those treated with standard coiling (16.9%), as well as a higher rate of retreatment (16.9% versus 9.0%) (41). However, another series reported better initial and follow-up anatomical results with BAC. This study achieved total occlusion in 73% of BAC-treated patients postoperatively compared to 49% with coiling alone, with similar results observed during follow-up (44). Conversely, the ATENA series on unruptured aneurysms did not show better anatomical outcomes with BAC (28,42).

BAC was initially developed for wide-neck aneurysms, but it has also demonstrated utility in cases of intraoperative rupture, where balloon assistance may improve clinical outcomes (45). In this scenario, the balloon is left deflated across the aneurysm neck and is only inflated if rupture occurs, serving as a protective measure. This "sentinel" use of BAC has contributed to its increased adoption in recent years, with one study showing a rise in its use from 23.9% in 2008 to 43.9% in 2010(46). The study also highlighted the versatility of BAC, noting its application in both ruptured and unruptured aneurysms of various locations, particularly those with an unfavourable dome-to-neck ratio (≤1.5) (39,46).

Flow diverters (FDs), developed over the past two decades based on in vivo and in vitro research, were introduced for clinical aneurysm treatment in the late 2000s(39,47,48) . These low-porosity, stent-like implants work through two primary mechanisms:

- Flow Redirection: The FD is placed across the aneurysm neck, reducing blood flow into the aneurysm sac by increasing resistance with its mesh structure while still allowing blood to pass through nearby perforators and side branches. This redirection decreases circulation within the aneurysm, leading to flow stasis and the

formation of a stable thrombus inside the aneurysm.

- Tissue Overgrowth: The FD acts as a scaffold, encouraging neoendothelialization over the aneurysm neck, which helps further seal the aneurysm.

Preclinical studies have shown FDs to be effective and safe in treating aneurysms with good occlusion rates and fewer thromboembolic complications (49,50).

Initially, two FDs were available: the Pipeline Embolization Device (EV3-MTI, Irvine, CA) and Silk (Balt, Montmorency, France). Recently, other devices like Surpass (Stryker, Fremont, CA) and FRED (Microvention, Tustin, CA) have been introduced (51–55). Early clinical experience with FDs, primarily from smaller single-centre or multicenter retrospective studies, demonstrated treatment feasibility, acceptable periprocedural complication rates, and favourable morbidity and mortality outcomes (51–55). Larger retrospective and prospective series have since confirmed these findings (56–58).

Flow diversion is typically used for treating complex aneurysms, such as large, giant, wide-neck, fusiform, or recanalised aneurysms after coiling. A recent international multicenter prospective study focused on treating complex aneurysms in the intracranial internal carotid artery in 108 patients. The treatment was feasible in 99.1% of cases, achieving complete occlusion in 73.6% of aneurysms at 180 days without significant vessel stenosis. The study also showed an acceptable safety profile, with 5.6% of patients experiencing a major ipsilateral stroke or neurological death (59).

Although the precise indications for flow diversion (FD) are still evolving, clinical experience has shown that FDs are primarily used for treating large and giant aneurysms (including fusiform aneurysms), wide-neck aneurysms, aneurysms within diseased arterial segments with multiple aneurysms, and recurrent aneurysms. Due to the need for dual antiplatelet therapy, most aneurysms treated with FD are unruptured. However, small studies have highlighted the effectiveness of FD in managing very small aneurysms, including blister-like aneurysms, which are difficult to treat with standard coiling techniques (60).

As FD use becomes more widespread, more information on potential complications has emerged. Like other endovascular treatments (EVT) for aneurysms, thromboembolic events and intraoperative rupture can occur. The risk of intraoperative rupture is generally lower with FD due to the absence of endovascular manipulation. Still, the risk of thromboembolic events is higher than standard coiling or balloon-assisted coiling (BAC) since FD is placed in the parent artery. To reduce this risk, both preoperative and postoperative antiplatelet therapy (single or dual) is recommended.

More extensive clinical experience with FD has also revealed complications not typically seen with standard coiling or BAC, such as delayed aneurysm rupture and remote parenchymal hematomas. Most of these complications have been reported in large and giant aneurysms, which have a high natural risk of bleeding and were previously untreatable (61–64). The Retrospective Analysis of Delayed Aneurysm Ruptures (RADAR) study showed that delayed aneurysm rupture occurred in about 1% of patients after FD treatment (65). Turowski et al. reported 13 cases of delayed ruptures with the Silk implant, with early ruptures (within 3 months) occurring more frequently than late ones (after 3 months). Early ruptures occurred between 2 and 48 days post-treatment, with patients typically still on dual antiplatelet therapy (aspirin and clopidogrel). Late ruptures, which occurred in patients only receiving aspirin, were seen between 110 and 150 days post-treatment. Delayed ruptures were most common in symptomatic, large, or giant aneurysms, particularly those with high dome-to-neck ratios (61,62,65).

Another severe complication associated with FD use is delayed ipsilateral parenchymal haemorrhage, with its incidence varying across studies. In the Cruz et al. study, delayed haemorrhage occurred in 8.5% of patients, while the RADAR study reported a 1.9% incidence (65,66). This complication was observed between 1 and 6 days post-treatment and was associated with variable clinical outcomes. A separate small series achieved favourable outcomes following surgical hepatoma evacuation after platelet transfusion. The proposed mechanisms behind delayed parenchymal haemorrhage include the haemorrhagic transformation of ischemic lesions,

altered intracranial blood pressure in distal territories, and loss of autoregulation in the distal arteries. Dual antiplatelet therapy may also contribute to the size of the hematoma or trigger spontaneous bleeding, similar to what is occasionally seen after carotid artery stenting (65,66).

Another concern with FD is the patency of perforating arteries and side branches covered by the device.

Finally, long-term follow-up is necessary to monitor for late thrombosis of FDs, which has been reported in certain aneurysms (67).

Flow disruption

Intravascular flow disruption is an endovascular technique akin to intraluminal flow diverter (FD) technology. The primary distinction lies in placing the flow disruptor's mesh directly within the aneurysm sac rather than the parent artery. This strategic placement induces blood flow stasis inside the aneurysm, leading to thrombosis and stabilisation of the aneurysm. Preclinical research has demonstrated that this method is feasible and practical, with a strong safety profile (68). In an initial retrospective multicenter study involving 20 patients treated with the WEB device (Sequent Medical Inc., Aliso Viejo, CA), the treatment achieved a 100% technical success rate, with no mortality and a low % morbidity rate of 4.8%(69). Subsequent prospective single-centre studies have reported comparable outcomes, reinforcing the reliability of the WEB device (70). The preliminary data suggest that the WEB device is particularly suitable for managing wide-neck bifurcation aneurysms in the basilar artery, middle cerebral artery, anterior communicating artery, and internal carotid artery (71). A significant advantage of this intravascular approach is that the flow disruptor remains entirely within the aneurysm, eliminating the need for antiplatelet therapy (71). This feature makes the technique especially promising for treating ruptured aneurysms. Additionally, there have been successful applications of the WEB device in treating recanalised aneurysms, further highlighting its versatility and potential in various clinical scenarios (69).

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Three instruments, two hand technique in biportal endoscopic spine surgery

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ABSTRACT

Background- Unilateral biportal endoscopic spine surgery (UBE) is a popular minimally invasive method for various types of spinal disease. This surgical technique has several advantages over conventional spine surgery, including less tissue damage, less blood loss, shorter hospital stays, and faster recovery. We introduce three instruments, two two-hand technique in UBE surgery.

Methods: Using hook dissector for gently teasing out the disc material and retracting the nerve root, and holding the same in non-dominant hand along with endoscope, we can use the instrument in dominant hand with better efficacy.

Results: A hook dissector can both act as a dissector and retractor while mobilising the nerve root and dural sac, and another instrument can be passed through the same portal, mainly a Kerrison punch or disc forceps for a complete decompression.

Conclusion: Use of 3 instruments in two hands of primary surgeon is a wonderful technique in UBE, particularly while achieving the final stages in decompression.

Keywords
unilateral biportal endoscopy (UBE), hook dissector, endoscope

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INTRODUCTION

Biportal endoscopic spine surgery or Unilateral biportal endoscopy (UBE) is similar to full percutaneous endoscopic spinal surgery in that it uses endoscopic instruments and similar to microscopic spinal surgery in that it uses a floating technique.[3,4] UBE is a useful surgical technique in unilateral or bilateral decompression for the treatment of spinal canal stenosis, foraminal stenosis, ossification of the ligament flavum, low-grade spondylolisthesis, and adjacent segment degeneration.

It involves the usage of two portals- the endoscopic portal, through which the endoscope goes in, and the instrument portal, through which the working instruments goes in. the working instruments are typically held in the dominant hand, and at times during simultaneous retraction and dissection, a hook dissector or a ball probe can also be entered through the working portal along with a Kerrison punch or disc forceps for better retraction and dissection.

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Surgical technique

A 48 years old man presented with low backache with left lower limb radiculopathy since three months. One year back, the patient had right lower limb radiculopathy which had subsided. MRI lumbosacral spine suggested L4/5 ligament flavum hypertrophy with disc protrusion bilaterally.

The patient underwent UBE spine surgery under general anesthesia. Initially drilling of caudal portion of the cranial L4 lamina was performed to separate the insertion margin of ligament flavum cranially, followed by drilling of upper portion of caudal L5 lamina for flavum separation. Excision of ligament flavum was performed and nerve roots freed. Hook dissector was inserted to gently probe the disc area over the shoulder of exiting nerve root, and remove disc fragments. Dissector was placed in position retracting the nerve root, and a disc forceps was inserted simultaneously with the hook through the same instrument portal to remove the disc material. The hook dissector was held in the left hand which is holding the endoscope, and using the thumb, index and middle finger for grip around it.

Complete decompression was performed, nerve roots relieved and closure was done.

Post-operatively, the patient was pain free, mobilized on the same day, and discharged in 24h of surgery.

Discussion

UBE allows visualization of the spinal structures via 2 small incisions on one side of the spine, thus minimizing tissue injury and enhancing postoperative recovery.[2]

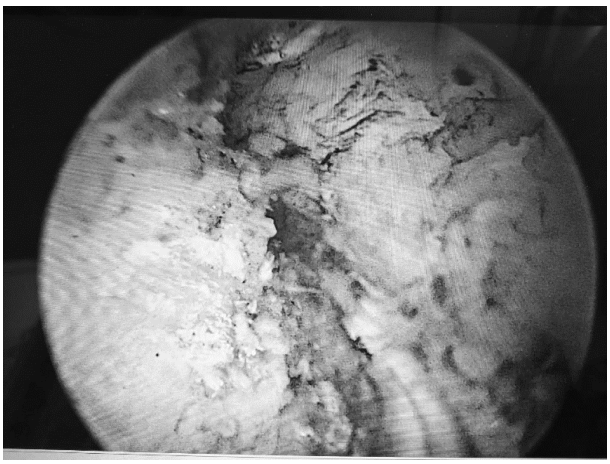

Figure 1. Point of starting drilling at spino laminar junction (12 o`clock- towards spinous process, 9 o`clock- cranial).

The initial docking point of the endoscope and the serial dilator is the location between the pathologic level of the spino-laminar junction and the inferior margin of the caudal lamina. Using the first serial dilator or muscle dissector, the paraspinal muscle should be sufficiently dissected on the lamina around the docking point to ensure sufficient saline patency. (Fig 1)

The laminectomy is started from the lower border of the cranial lamina, using a drill or osteotome until a free margin of LF is obtained. Then, the V-shaped central fissure of the LF is distinguished from the lower border of the cranial lamina, bone work is performed until the cranial, lateral, and caudal sides are freely detached.[1] (Fig 2)

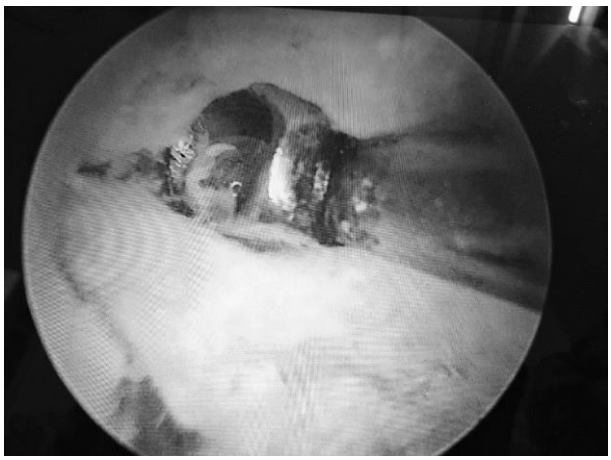

Figure 2. V-shaped central fissure of the LF (12 o`clock- towards spinous process, 9 o`clock- cranial).

Figure 3. Surgeon`s hand showing holding of the three instruments.

In the contralateral sublaminar approach, the LF on the contralateral side and the ventral side of the lamina should be detached using a freer or curette before contralateral decompression.

Usage of a hook dissector can both act as a dissector and retractor while mobilizing the nerve root and dural sac, and another instrument can be

passed through the same portal, mainly a Kerrison punch or disc forceps for a complete decompression. The hook can be held in place using the endoscopic hand, taking the help of thumb, middle and index finger to hold and manipulate the hook, and the other two fingers encircling the endoscope. The dominant hand can then act freely to relieve the decompression. (Fig. 3,4)

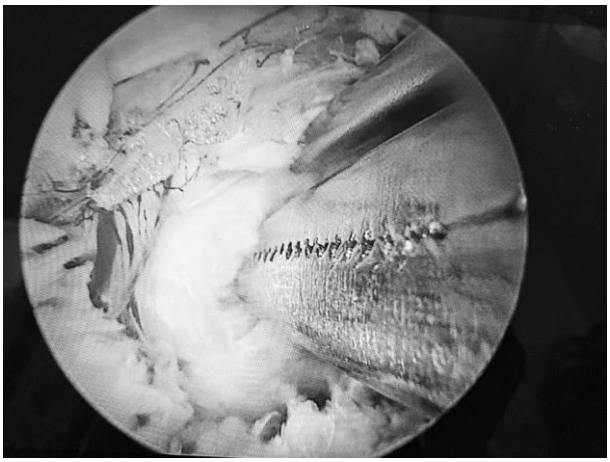

Figure 4. Endoscopic view showing two instruments, hook dissector retracting the nerve root, and disc forceps taking the disc material.

Conclusion

The field of UBE has achieved remarkable advancements in recent years, and endoscopic techniques have become common essential spinal surgery procedures. Use of 3 instruments in two hands of primary surgeon is a wonderful technique in UBE, particularly while achieving the final stages in decompression.

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Intralesional abscess in choroid plexus carcinoma. A case report

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ABSTRACT

Background: Choroid plexus carcinoma (CPC) is a rare, aggressive intraventricular tumour that predominantly affects young children. While CPC typically presents with hydrocephalus and mass effect, intratumoral abscess formation has not been previously documented.

Case Presentation: We report the case of a previously healthy 3-year-old female presenting with central facial hemiparesis and anisocoria. Imaging revealed a solid-cystic, contrast-enhancing lesion in the left ventricular atrium with spinal dissemination. Endoscopic-guided total resection via a parietal approach was performed, revealing a friable, haemorrhagic mass. Histopathology confirmed CPC with high proliferative index (Ki-67: 70%). Postoperative treatment followed the HEAD START III chemotherapy protocol without hematopoietic stem cell transplantation, followed by craniospinal radiotherapy. The patient remains recurrence-free after 12 months of follow-up.

Conclusion: This case highlights the successful management of a CPC complicated by an intratumoral abscess, an unprecedented presentation. Total surgical resection followed by multiagent chemotherapy and radiotherapy resulted in favourable early outcomes. Given CPC's rarity and variable presentation, individualised, multidisciplinary approaches are essential.

Keywords
intralesional abscess, choroid plexus carcinoma

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INTRODUCTION

Choroid plexus carcinoma (CPC) is a rare malignant neoplasm of neuroepithelial and primary of the central nervous system (1,2). The average age of diagnosis of CPC is 3 years and the annual incidence rate is 0.3 per million individuals (3,4). CPC has profound clinical and molecular differences compared to its lower-grade counterpart, choroid plexus papillomas (1). Tomography imaging typically reveals a large, hyperdense, contrast-enhancing intraventricular mass. Associated findings include hydrocephalus, calcifications, and/or hemorrhages. CPC is characterized histologically by friable papillary or cauliflower-like appearance, increased mitotic figures, pleomorphic nuclei, and necrosis. Intraoperatively, CPC may loosely or densely

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adhere to the ventricular wall and minimally or extensively invade adjacent brain parenchyma (2). No abscessed lesions have been described so far, so in this text we report a pediatric case of CPC complicated with intratumoral abscess successfully treated with resection + systemic chemotherapy.

Case presentation

A previously healthy 3-year-old female patient presents with two months of central facial hemiparesis that progressed with anisocoria due to left mydriasis. A computed tomography (CT) scan of the skull showed a space-occupying lesion of a solid and cystic nature in the left ventricular atrium with extraventricular extension and discrete perilesional vasogenic edema. A brain Magnetic Resonance Imaging (MRI) contrast-enhanced with spectroscopy revealed a neoplastic-like lesion located within the left atrium, hinted at the occipital and temporal horn (Figure 1A). Spectroscopy showed a choline/NAA ratio of 1.31.

Extension workup through MRI of the cervical, thoracic and lumbosacral spine showed secondary lesions with contrast enhancement at the C5/C6, T1/T2 and L1 level, on the right side, that compromised the subarachnoid space and the medullary contour (Figure 2).

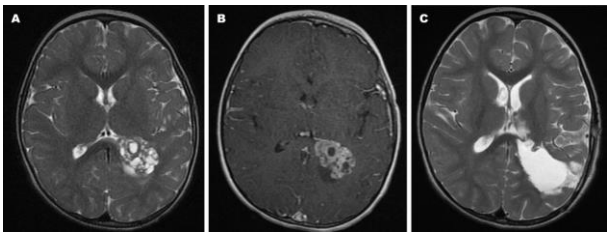

Figure 1. Baseline magnetic resonance of brain in axial T2-weighted (A) and contrast-enhanced (B), and postoperative T2-weighted imaging.

During the hospital stay, she presented vomiting and headaches and received symptomatic management. Endoscopic resection was performed through a parietal approach and guided by neuronavigation. During surgery, the tumor lesion had malignant appearance with a grayish, friable, profusely bleeding and easy to aspirate. A biopsy sample was taken and total macroscopic resection was achieved. There were no complications, with persistence of central facial hemiparesis and left mydriasis in the postoperative period (Figure 1B). Pathology and immunohistochemistry report evidenced a malignant tumor made up of small, ovoid and spindle-shaped cells, marked cytonuclear pleomorphism and mitotic activity. Positive tissue for Cytokeratin, S100, E-Cadherin, Vimentin and P53, with Ki-67 showing 70%, configuring the diagnosis of choroid plexus carcinoma.

Management was started with the HEAD START III protocol without consolidation therapy with autologous transplantation of hematopoietic precursors, there were no major complications during the chemotherapy cycles. Craniospinal radiotherapy and boost in the tumor bed were used as consolidation therapy. MRI of the brain and axis did not reveal neoplastic lesions with the respective post-surgical parenchymal defect. Patient continues in multidisciplinary follow-up 12 months after diagnosis, with no evidence of recurrence.

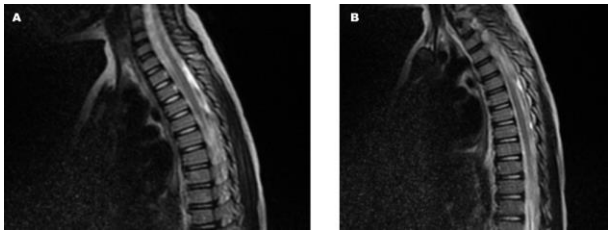

Figure 2. Baseline magnetic resonance of spinal cord showing diffuse lesions in upper (A) and lower (B) thoracic segments.

Discusion

CPC is a rare cause of a hemispheric brain tumor arising from the lateral ventricles in children. Possible differential diagnoses for a hemispheric brain tumor of this type include choroid plexus papilloma, ependymoma, atypical teratoid rhabdoid tumor, glioma, astrocytoma, and primitive neuroectodermal tumor (PNET). Radiopathological correlation with tissue immunohistochemistry is essential to differentiate and establish a confirmatory diagnosis. (5).

Due to the extremely low incidence of CPC, it is not possible to standardize medical conduct, so individualization based on case studies and clinical expertise determine the therapeutic vector in these patients. However, higher success rates have been reported in patients undergoing total resection with subsequent multiagent chemotherapy. (6). Radical surgery is the most important prognostic factor, although survival remains relatively low. (7). Despite the merits of surgical resection, due to the

macroscopic characteristics of the tumor (large size, high vascularity, diffuse infiltrative nature, and excessive friability), it is difficult to achieve complete resection, resulting in the necessity of a surgical resection. neoadjuvant therapy later. In the pediatric population, the risk of bleeding from surgical resection is considerable, as all circulating blood volume may be lost during resection of these highly vascularized tumors.

The planned surgical approach should allow good visual access to tumor blood flow and maximum exposure of the mass. An effective intraoperative surgical strategy is to identify and ligate the choroidal feeding vessel, which facilitates block-removal of the tumor mass. (5). McEvoy et al. reported a mortality rate attributable to intraoperative bleeding between 5 and 12%. In this report, four of nine surgeries for CPC in their series were suspended due to excessive bleeding, achieving only 33% of CPCs grossly resected completely. (8).

Due to this, it is recommended that if for some reason complete resection is not achieved, reoperation is a reliable option, which can be preceded by the administration of chemotherapy to reduce intraoperative bleeding and tumor size. (1), which will allow for a complete posterior resection rather than an incomplete resection (5). In order to reduce bleeding, for certain anatomical dispositions of the tumor it is feasible to submit the patient to preoperative embolization and thus achieve total gross resection. (9). Embolization also reduces the production of cerebrospinal fluid, the reduction of which is a goal of comprehensive CPC treatment. (10).

Like surgical resection, there is a lack of global consensus on chemotherapy and neoadjuvant regimens and it has not yet been standardized. The following drugs are used in treatment: carboplatin, etoposide, cyclophosphamide, high-dose methotrexate, and vinca alkaloids (1). The best chemotherapy regimen has yet to be determined, but a combination using platinum and etoposide as a base is preferred (11). Its combination with radiotherapy is also controversial due to evidence that it presents a greater benefit in chemotherapy alone (12). Radiotherapy is an important aspect of management, but its implementation is limited in young children (<2 years). Among a database of 524 patients, 5-year survival was better in irradiated CPC patients (13).

The effects of surgery and radiation therapy were evaluated in patients with or without TP53 germline mutations to provide vital information to help define neuro-oncology response assessment specific to pediatric patients with CPC. Li et al. showed an improvement in patients who received total tumor resection and avoided radiation therapy. CPC have been linked to germline TP53 mutations. In particular, patients with the TP53 germline mutation showed a significant survival advantage, demonstrating that radiation therapy should not be considered for patients who have the TP53 germline mutation (4). Despite these findings in the literature, it was decided to use radiotherapy in this patient due to the high risk of consolidating high doses of myeloablative chemotherapy and rescue with autologous transplantation of hematopoietic precursors.

Conclusion

CPC is a rare malignant neoplasm of neuroepithelial origin and primary central nervous system, there is still no consensus on the neoadjuvant alternative for the management of these neoplasms, on the other hand, robust evidence suggests that total resection achieves greater survival, although it continues being relatively low.

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Primary spinal tumours. A single centre study: clinical assessment, treatment and outcome

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ABSTRACT

Primary spinal cord tumours are rare conditions that comprise 3% of all primary CNS tumours in adults. The present study analyses the clinical presentation, prognostic factors, radiological assessment, treatment, histological examination and their correlation with the outcome of these spinal tumours, in an urban setup of India. The study presents our single institution's surgical experience and clinical outcomes on patients who have undergone surgical excision for spinal tumours. *Methodology:* This retrospective and prospective observational study was conducted among 36 patients admitted to the Department of Neurosurgery at Rabindranath Tagore Medical College and Associated Hospital, Udaipur, between May 2022 and September 2024. All patients were clinically evaluated along with preoperative and post-operative modified Nurick grading with 3 months of follow-up. Statistical analysis was performed using SPSS. *Results:* Our study included 36 patients; mean age was 38.4 years. Of the participants, 13 (36.1%) were men and the remaining 23 (63.9%) were women. Motor Weakness was the most common symptom noted in all patients. The tumours were commonly seen in the thoracic region, 14 cases (38.9%). Schwannoma was the commonest lesion, and there was a male preponderance. *Conclusion:* Our study clearly demonstrates that surgical treatment of spinal tumours offers very good functional outcomes irrespective of the age of the patient or the neurological status.

Keywords
primary,
spinal,
tumours

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INTRODUCTION

The prevalence of spinal tumors is rare in comparison to brain tumors which encompass most central nervous system tumors.[1,2] Tumors of the spine can be divided into primary and metastatic tumors with the latter being the most common presentation. Primary tumors are subdivided based on their location on the spinal column and in the spinal cord into intramedullary, intradural extramedullary, and primary bone tumors.[3,4] Back pain is a common presentation in spine cancer patients; however, other radicular pain may be present.[5] Magnetic resonance imaging (MRI) is the imaging modality of choice for intradural extramedullary and intramedullary tumors.[6] Plain radiographs are used in the initial diagnosis of primary bone tumors while Computed tomography (CT) and MRI may often be necessary for further characterization. Complete surgical resection is the treatment

of choice for spinal tumours and may be curative for well circumscribed lesions. However, intralesional resection along with adjuvant radiation and chemotherapy can be indicated for patients that would experience increased morbidity from damage to nearby neurological structures caused by resection with wide margins. Even with the current treatment options, the prognosis for aggressive spinal cancer remains poor. Advances in novel treatments including molecular targeting, immunotherapy and stem cell therapy provide the potential for greater control of malignant and metastatic tumors of the spine.

Materials and methodology

2.1 Study design: This is a retrospective and prospective observational study.

2.2. Study population, place, duration: Data for this study was obtained from 36 patients admitted in the Department of Neurosurgery at Rabindranath Tagore Medical College and Associated Hospital, Udaipur between May 2022 and September 2024.

2.3. Inclusion and exclusion criteria: All patients with a diagnosis of Intra-medullary, IDEM and extradural spinal tumor were included in the study. Patients with recurrent tumors also included, secondaries, infective collection and vascular abnormalities were excluded from the study.

2.4. Study procedure: Pre-operative MRI was done in all cases satisfying the inclusion criteria. All patients were clinically evaluated along with preoperative and post-operative modified Nurick grading with 3 months of follow up. Patient centered questions were present for assessment and follow-up. The number of points in Joa score and European assessment score was more. The people educational status was the main factor in which they could be assessed with the questions of Nurick grade.

2.5. Statistical analysis: Statistical analysis was performed using Statistical Package for Social Science (SPSS). Multiple Logistic regression method was used to predict the independent variables. A two-sided p value less than 0.05 was considered statistically significant.

Result

Our study included 36 patients, age ranging from 06 to 64 yrs. The majority of the patients (72.2%) were between 20 and 60 years of age. Mean age of our cohort was 38.4 years. (Table 1). Of the participants, 13 (36.1%) were men and the remaining 23 (63.9%) were women. There was a correlation between age and outcomes, older age portends a worse outcome, with a p value of 0.04. There was no correlation between gender and outcome. (Table 1). Motor Weakness was the most common symptom noted in all patients (100 %) followed by spasticity noted in 34 patients. Sensory Impairment was seen in 30 patients. There was a considerable overlap of the symptoms as well. Bladder or bowel symptoms were noted in 50 % of our study population. (Table 2). Motor Symptoms were the predominant complaints in patients with spinal tumors while the proportion of patients with bladder and bowel involvement had advanced disease. Twenty four patients had a worse Nurick Grade of 4 or worse. All 30 patients with bladder dysfunction had poor Nurick Grade. (Table 2). The tumors were commonly seen in thoracic region 14 cases (38.9%) followed by cervical 8 cases (22.2 %) and lumbar 5 cases (13.9 %). We also had four cases with cervico-dorsal and five cases with dorso-lumbar involvement (Table 3).

Table 1. Socio-demographic details of the participants.

S.No.	Variable	Frequency	Percentage
1. Gender			
Male		13	36.1 %
Female		23	63.9 %

Table 2. Symptoms of the participants.

S.No.	Variable	Frequency	Percentage
1. Type of symptoms			
Motor weakness		36	100 %
Spasticity		34	94.4 %
Sensory impairment		30	83.3 %
	Bladder or bowel Involvement	18	50 %
2. Complications			
CSF leak		0	0 %
Worsening of power		3	8.3 %

Table 3. Tumor characteristics.

S.No.	Variable	Frequency	Percentage
1. Region of tumor			
Thoracic	14	38.9 %	
Lumber	5	13.9 %	
Cervical	8	22.2 %	
Cervico-dorsal	4	11.1 %	
Thoracolumbar	5	13.9 %	
2. Tumor Diagnosis			
NST- Schwnomma	11	30.6 %	
Neurofibroma	2	5.6 %	
Meningioma	7	19.4 %	
Ependymoma	3	8.3 %	
Astrocytoma	2	5.6 %	
Dermoid/epidermoid (recurrent)	4	11.1 %	
Lymphoma/ leukemic infiltration	2	5.6 %	
lipoma	1	2.8 %	
hydatid cyst	2	5.6 %	
arachnoid cyst	2	5.6 %	

All the patients included in the study underwent excision of the lesions identified by imaging. Standard posterior midline approach employed. Laminectomies and hemi-laminectomies were employed. Microscope was used to achieve maximum safe excision of the lesion. Histopathological examination was done for all the cases. Dorsal approach was used in all patients. Gross Total Excision of the lesion was possible in extramedullary tumors with tumor remnant in case of intramedullary tumors. The epidural space was obliterated with fat after water-tight Dural closure.

Three pathologies, namely, Schwannoma, Neurofibroma and Meningioma accounted for 55.6 % of all primary spinal tumors (n = 20). Other lesions with multiple occurrences include three cases of ependymomas, two cases of arachnoid cyst, two cases of Lymphomas, one case of lipoma. Schwannoma was the commonest lesion and there was a male preponderance. The mean age of presentation was 40.8 years and the mean duration of symptoms was 7 months. Nerve sacrifice was necessary in 6 patients. Meningiomas were common in women and had a mean age of 40.3 yrs and average duration of symptoms was 8 months. Involved dura was cauterized in the 3 cases where the tumor had adherence to the dura. Rest of the tumors were easily separated from the dura.

Nurick Grade was assessed at the time of presentation and discharge as well as during the periodic follow up. 28 patients were available for follow up for 3 months. The average duration of follow up was 7 months. The outcome at the time of discharge is discussed here. Seven patients had a pre operative Nurick grade of 3 and worse while 29 patients had Grades 0, 1 and 2. The average pre operative Nurick's was 1.87. Post operatively, 27 patients belonged to Grade 2 or better. Four patients had Grade 3, while one patient had Grade 4. One patient had Grade 5. The average Post Operative Nurick's Grade was 1.1. thirty two of the 36 patients had a good outcome with a Nurick Grade of 2 or less. 2 patients had a fair outcome while 2 patients had a poor outcome.

At 3 months follow up, none of the two patients with a fair outcome and none of the two patients with poor outcome improved. Six of the 30 patients with bladder dysfunction had some degree of improvement at 3 months follow up. Both of the patients with a poor outcome had a thoracic lesion. None of the lesions were predominantly ventral in location. We had 2 patients that developed wound dehiscence and none had CSF leak from the wound site. Secondary suturing was done in these cases and resolution was achieved. One patient had meningitis which was medically managed. There was one mortality in our study due to respiratory complication.

Discussion

Spinal tumors are rare, comprising of 5%-12% of total tumor of central nervous system, with an overall incidence of 0.74/ 100,000 person years.[7-8] Primary spine tumors are generally uncommon. However, this remains an important topic because they can cause considerable morbidity for patients by causing pain and affecting motor and sensory function. Spine tumors can arise within the spinal cord itself, or from the adjacent structures. These tumors can broadly be divided into primary and secondary (metastatic) tumors. Metastatic spine tumors spread to the vertebral column via hematogenous route (eg, via the Batson plexus). Contrary to primary spine tumors, metastatic spine disease (MSD) is extremely prevalent. In a postmortem study involving patients with breast or prostate cancer, the prevalence of MSD was between 70% and 90%.[9]

In general, spine tumors can be classified according to their anatomic location into intradural-

intramedullary, intradural-extramedullary, and extradural. Intradural-intramedullary tumors are neoplasms arising within the spinal cord. They account for 20% of all intraspinal tumors in adults and 35% of all intraspinal tumors in children.[10] Intradural-intramedullary tumors may arise from intrinsic cells within the spinal cord (intra-axial lesions), or via seeding/ systemic spread. Most primary intra-axial tumors are either ependymomas or astrocytomas. Tumors are labeled as intradural-intramedullary if the epicenter arises at the level of C1 to the level of the conus (L1/L2). Lesions above C1 and involving the medulla are labeled as cervicomedullary. Intradural-extramedullary tumors are located within the dura but outside the spinal cord.

Meningiomas and nerve sheath tumors (NSTs) are the 2 most common types of tumors.[11] Extradural tumors are the most common (60% of all spine tumors). These tumors arise outside the dural sac, typically from the vertebral bodies. These are usually metastatic in nature. These tumors are particularly important because of the risk of epidural spinal cord compression. In this review article, we will provide a broad overview of the various types of tumors that can occur in the spine. We will discuss primary tumors of the spinal cord (benign and malignant), primary vertebral column tumors (benign and malignant), and metastatic spinal tumors separately. For each we will provide brief epidemiology, radiological and histopathological pearls, and touch on the natural history of the entity.

Primary Spinal Cord Tumors

Primary spinal cord tumors are rare conditions that comprise 3% of all primary CNS tumors in adults. Age adjusted incidence rates are slightly higher for men than women, 0.67 compared with 0.59 per 100 000 population, respectively.[12]

Benign Spinal Cord Tumors

Spinal Meningioma. Meningiomas can arise from arachnoidal cap cells anywhere along the neural axis. Most of them occur intracranially, whereas approximately 10% can be found adherent to the spinal dura. The age adjusted incidence is 0.33 per 100 000 with women in the seventh to eighth decades having the highest incidence.[13] Approximately 80% of spinal meningiomas arise in the thoracic region, with the cervical region being the second most common (15%).[14] Prior exposure to ionizing radiation and neurofibromatosis type 2 (NF2) syndrome are known risk factors.[15] Radiologically, the majority are intradural extramedullary in location, having a well-circumscribed appearance on MRI. They often have a broad-based dural attachment and most exhibit a dural tail sign. They can be isointense on T1 and T2 imaging and exhibit homogeneous contrast enhancement. Histologically, they show a lobulated architecture with whorls and psammoma bodies, and stain positive for vimentin. The majority are World Health Organization (WHO) grade 1, with the meningothelial subtype being the most common in the spine. WHO grade II clear cell subtypes have a predilection for the spine and are thought to arise from the denticulate ligaments.[16] Surgical removal is the treatment of choice, with a consideration for adjuvant radiotherapy in higher grade, or recurrent, tumors.

Nerve Sheath Tumors. NSTs are the most common intradural-extramedullary lesions. Most NSTs arise from the dorsal sensory roots. Spinal schwannomas are most common, followed by neurofibromas and ganglioneuromas. The peak incidence of schwannomas occurs in the fifth to seventh decade.[10] About 35% to 45% of patients with NST have neurofibromatosis. Neurofibromas are associated with NF1, and schwannomas are associated with NF2. These are well circumscribed and indistinguishable radiologically. The majority are isointense on T1, hyperintense on T2, and all exhibit contrast enhancement. CT imaging may exhibit chronic changes such as widening of the neural foramina and scalloping of the adjacent bone. Histologically, key features are that of a biphasic tumor with a highly ordered cellular component (Antoni A) that palisades (Verocay bodies) plus myxoid hypocellular component (Antoni B). These stain strongly for S100. Management is via surgical excision, and because of their well-circumscribed nature, can be peeled away from the parent nerve easily. Most do not recur and hence do not need adjuvant radiation. A minority of cases can undergo malignant transformation into malignant peripheral nerve sheath tumor, angiosarcoma, or epithelioid malignant change, for which the prognosis is poor.

Malignant Spinal Cord Tumors

Spinal gliomas. The majority of intramedullary spinal cord tumors are gliomas. The word glioma refers to a tumor with histological similarity to normal glial cells. The major types of spinal glioma tumors are ependymomas and astrocytomas.

Ependymomas. Spinal ependymomas are the most common intramedullary tumors.[17] Ependymomas are glial tumors arising from ependymal cells and most commonly occur adjacent to the ventricular surface, along the spinal canal, or in the film terminale.[18] Though spinal cord gliomas are rare compared with cerebral lesions, ependymomas comprise approximately 60% to 80% of spinal gliomas compared with 3% of intracranial gliomas.[19] Among all spinal tumors in ages 0 to 19, ependymomas are the most common histology, comprising about 20%.[20] NF2 is a dominant hereditary condition and is manifested by multiple tumors of the nervous system.

Imaging evidence of ependymomas is seen in one-third of these patients. In contrast to sporadic spinal cord ependymomas, those associated with NF2 tend to display an indolent growth pattern, and potentially can be observed if found incidentally. In the WHO classification of brain tumors, ependymal tumors are divided into 4 major groups: subependymoma (grade I), myxopapillary ependymoma (grade I), ependymoma (grade II), and anaplastic ependymoma (grade III). [21] Radiologically, ependymomas present a well-circumscribed lesion with variable enhancement. These may be associated with cystic change, hemorrhage, or calcification. Key histological features include perivascular pseudorosettes and ependymal rosettes. Gross total removal should be attempted for all patients. Compared with lower-grade ependymomas, anaplastic tumors appear to have a higher recurrence rate and poorer survival.

Astrocytic tumors of the spinal cord. Spinal astrocytomas account for approximately 6% to 8% of all spinal cord tumors.[17] In children, they are the most common intramedullary tumor, and the second most common in adults. These tumors may occur throughout the spinal cord. The peak incidence is in the third decade, with males being affected more commonly. There is an increased incidence in patients with NF1. They arise from astrocytic glial cells. Compared to their intracranial counterparts, they generally have a lower histological grade (with ~ 75% being low grade). However, high-grade spinal astrocytomas have an increased risk of leptomeningeal dissemination.

Radiologically, astrocytomas arise from the cord parenchyma (as opposed to ependymomas, which arise from the central canal). They often span several segments and appear exophytic in nature, sometimes being mistaken for an extramedullary tumor. Margins are poorly defined because of their infiltrative nature. Peritumoral edema and cysts are seen in less than half of patients. They appear hyperintense on T2 imaging, and most lesions show contrast enchantment on T1 imaging. Astrocytic tumors are composed of infiltrative cells with irregular, hyperchromatic nuclei and eosinophilic, GFAP-positive cytoplasm. These tumors are graded histologically according to their most anaplastic-appearing areas.[22] Molecular parameters, used in the WHO classification, are based growth pattern, behavior, and isocitrate dehydrogenase–mutation status.[8-21]

The mainstay of treatment for primary spinal cord astrocytoma is surgical resection, with the goal of preservation of neurologic function, guided by intraoperative neuro-monitoring. Adjuvant radiotherapy and chemotherapy may be used depending on the extent of resection and tumor grade.

Conclusion

Our study clearly demonstrates that surgical treatment of primary spinal tumors offers very good functional outcomes irrespective of the age of the patient or the neurological status. Hence surgery should be offered to all patients presenting with primary spinal tumors.

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Cranioplasty timing and its effect on the functional neurological outcome in traumatic brain injury

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ABSTRACT

The best time to do cranioplasty (CP) and how it will affect the neurological outcome is still in debate. Moreover, being one of the most commonly performed interventions in neurosurgery, its complication rate is among the highest in literature. The aim of this study is to assess the effect of timing of cranioplasty (CP) on the functional neurological outcome and post-operative complications in patients of traumatic brain injury. A retrospective cohort study was conducted in patients who underwent CP between 2018 and 2022. Early and late cranioplasties were defined as surgeries performed within and more than 90 days of decompressive craniectomy, respectively. The Glasgow Outcome Score- Extended (GOSE) and Functional Independence Measure (FIM) were used to assess the neurological outcome. Late CP cohort patients presented with subdural hygroma (SDG), which had a significant correlation. In post-CP complications, SSI, post-CP hydrocephalus and new onset seizures had a significant correlation with late CP ($p < 0.001$). In early CP GOS E score of 6 was seen, whereas a better score of 7 was seen late cohort. In FIM rating, both had maximum number of cases in Minimal Assistance group. The neurological outcome in patients who underwent early versus late CP is almost identical. We drew the conclusion that early CP often resulted in less post-operative morbidity. A lower number of early CP cohort subjects experienced post-CP HCP, seizures, and SSI. In order to reduce postoperative problems in TBI cases receiving DC, we advise an early CP based on the study's findings.

Keywords
bone flap,
Glasgow outcome scale,
FIM scale,
cranial reconstruction,
surgical site infection

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INTRODUCTION

Cranioplasty (CP) after decompressive craniectomy (DC) is necessary not only for cosmetic reason but cranial bone also offers crucial support and maintains normal cerebrospinal fluid (CSF) flow dynamics, and safeguarding crucial structures. Although a common neurosurgical operation, cranioplasty can have serious side effects; rates of total severe problems range from 10.9% to 40.4%. [1, 2] A study done by P Schuss et al stated that complications of CP exceed those of other elective cranial procedures. The overall complication rate after cranioplasty was 16.4% in their study. [3] Chang and associates also published a series with an overall complication rate of 16% following CP. [4]

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There are multiple papers which suggest that delaying cranioplasty may lead to more complication and morbidity but literature is scarce regarding the timing of cranioplasty on the overall outcome and functional neurological outcome of the patient. In a study that used transcranial colour doppler to evaluate the effect of CP timing on cerebral blood flow (CBF) velocity in patients who underwent DC, showed better improvements in CBF in the early CP group compared to the late CP group and no difference in complication rate between the two groups. They concluded that optimizing patient selection, treatment course along with early CP has potential benefits for cerebral perfusion.[5]

In numerous articles, there has been contentious discussion about the timing of cranioplasty. Numerous studies suggested that early CP might be advantageous for patients.[6,7] Cranioplasty following DC at an early stage (5-8 weeks after DC) is safe and may result in neurologic improvement, according to Liang and colleagues.[8] Their analysis lacked specific information regarding the frequency of complications following cranioplasty. It is also unknown if the timing of a cranioplasty affects the overall complication rate. Given the high rates of complications connected with the treatment, it is crucial to analyze the neurological outcome in relation to the timing of cranioplasty. The purpose of the present study is to assess the neurological outcome and postoperative complication rates among patients who underwent early versus late cranioplasty with autologous bone flap at a major Level I trauma center.

Materials and methods

Patients' eligibility and study setting

This is a retrospective cohort study, reviewing the neurological outcome and postoperative complications, in cranioplasty patients. Data were collected for all eligible cranioplasties performed between January 2018 and December 2022 at King George's Medical University, Lucknow, Uttar Pradesh, a Level I trauma center serving north western and central state region and receives referrals from all over the state.

All patients who underwent cranioplasty and had preserved bone in the abdominal pocket, during the specified period were included in the study. The exclusion criteria were; patients with congenital cranial defects repaired by cranioplasty, patients who had nonautologous cranioplasty, and patients who had more than 50% of the defect replaced with nonautologous bone.

Data collection

Data were retrieved from the archives of the neurosurgery department using two distinct methods; case sheets and the hospital's electronic system from 2018 to 2022. Data included patients' demographics and postoperative complications. The primary indications for performing DC were TBI. Early and late cranioplasties were defined as surgeries performed within and more than 90 days of DC, respectively.

The Glasgow Outcome Score Extended (GOSE) and functional independence measure scores (FIM) were calculated to assess the neurological outcome. The GOSE 8 point scale is scored as follows; (1 = Death), (2 = Persistent vegetative state), (3 = Lower Severe disability), (4 = Upper Severe disability), (5 = Lower Moderate disability), (6 = Upper Moderate disability), (7= lower good recovery) and (8= upper good recovery).[9] Each item on the FIM is scored on a 7-point, Likert scale and the score indicates the amount of assistance required to perform each item (1 = total assistance in all areas, 7 = total independence in all areas).[10] The ratings are based on performance rather than capacity and was acquired by observation, patient interview or telephone interview. A final summed score is created and ranges from 18 – 126, where 18 represents complete dependence/total assistance and 126 represents complete independence.

Statistical analysis

Data were coded before being entered into IBM SPSS (version 23; Armonk, New York, USA: IBM Corporation). Categorical data were presented as frequencies and percentages using descriptive statistics. Age, GOS, FIM, and length of stay are examples of numerical variables for which mean and standard deviation have been determined. To determine the average differences in numerical data between early and late cranioplasty, the independent sample T-test was used. To examine the relationship between categorical data and the date of cranioplasty, the Chi-square test was used.

The P-value's statistical significance level was set at 0.05.

Results

A total of 104 cases of autologous cranioplasty were performed in the time period of which 93 were included in the study. 57 (61.3%) of them underwent late cranioplasty and 36 (38.7%) early cranioplasty. 78.50% were male and 21.50% were female. Maximum patient were in the age group of 23-32 years. 27.28% patients in early carnioplasty group were in the age group of 13-22 years, whereas 36.84% of late cranioplasty group were in the age range of 23-32 years (Fig 1).

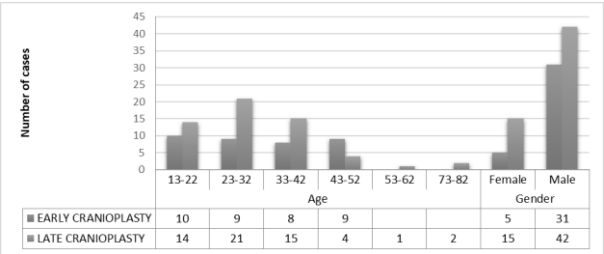

Figure 1. Showing distribution of patients in age groups and gender

The mode of injury in 78.50% was road traffic accident, with 12.90% had history of fall from height and an 8.60% cases had alleged history of physical assault. In the type of injury sustained, 45.20% had mixed type of injury which includes components of both acute SDH and multiple hemorrhagic contusions, 34.40% had acute SDH (Table 1).

62.40% cases had mild head injury, 25.80% were moderate and 11.80% were of severe head injury. This disparity in the presentation GCS might be attributed to the fact that maximum of the severe head injury cases succumb to their injuries in the follow-up period, thus not reporting for cranioplasty. 42 cases underwent right sided decompressive craniectomy, followed closely by left sided decompressive craniectomy (41) and 10.80% cases had bilateral frontal decompressive craniectomy (Table 2).

13 cases underwent ventriculo-peritoneal shunt before cranioplasty in the early cranioplasty group, whereas 7 cases in late cranioplasty group underwent CSF diversion before surgery. 3 cases presented with subdural hygroma warranting the need of subduro-peritoneal shunt before surgery in the late cranioplasty cohort. 30.10% cases (n: 28) in total had post operative complication. 36.85% (n: 21) in late cranioplasty cases and 22.20% (n: 8) in early cranioplasty cohort had complications. Hydrocephalus was identified in a total of 9 patients (early, *n* = 3, 8.30%; late, *n* = 6, 10.52%. p: <0.001). A total of 11 patients presented with surgical site infection (SSI) postoperatively (early, *n* = 3; late, *n* = 8, p: <0.001). Seizures were noted in four patients, and all them underwent late cranioplasty (early, *n* = 0; late, *n* = 4, p<0.001) (Table: 3).

Table 1. Timing of cranioplasty and its relation with mode of injury and type of injury.

		Timing of cranioplasty						
		Early cranioplasty		Late cranioplasty		Total		
		Count	Column n %	Count	Column n %	Count	Column n %	P-value
Mode of injury	Ffh	4	11.10%	8	14.00%	12	12.90%	0.051
	Physical assault	0	0.00%	8	14.00%	8	8.60%	
	Rta	32	88.90%	41	71.90%	73	78.50%	
Type of head injury	Acute sdh	12	33.30%	20	35.10%	32	34.40%	0.964
	Contusion	6	16.70%	13	22.80%	19	20.40%	
	Mixed	18	50.00%	24	42.10%	42	45.20%	

Table 2. Correlation between side of decompressive craniectomy and severity of injury.

	Timing of cranioplasty			
	Early cranioplasty	Late cranioplasty	Total	

		N	Column n %	N	Column n %	N	Column n %	P-value
Side of decompressive craniectomy	Bilateral frontal	5	13.90%	5	8.80%	10	10.80%	0.733
	Left	15	41.70%	26	45.60%	41	44.10%	
	Right	16	44.40%	26	45.60%	42	45.20%	
Gcs at presentation	Mild	18	50.00%	40	70.20%	58	62.40%	0.143
	Moderate	12	33.30%	12	21.10%	24	25.80%	
	Severe	6	16.70%	5	8.80%	11	11.80%	

Table 3. Timing of cranioplasty and its correlation with post operative complications.

		Timing of cranioplasty						
		Early cranioplasty		Late cranioplasty		Total		
		Count	Column n %	Count	Column n %	Count	Column n %	P-value
Post op complication	Absent	28	77.80%	36	63.15%	65	69.90%	0.138
	Present	8	22.20%	21	36.85%	28	30.10%	
	Hydrocephalus	3	8.30%	6	10.52%	9	9.67%	P<0.001
	Cardiac complication	1	2.80%	2	3.50%	3	3.20%	
	Pulmonary complication	1	2.80%	1	1.75%	2	2.20%	
	Seizure	0	0.00%	4	7.01%	4	4.30%	P<0.001
	Ssi	3	8.30%	8	14.03%	11	11.82%	P<0.001

Table 4. Timing of cranioplasty with GOS-E and FIM score.

		Timing of cranioplasty						
		Early cranioplasty		Late cranioplasty		Total		
		Count	Column n %	Count	Column n %	Count	Column n %	P-value
Gos e	1	5	13.90%	4	7.00%	9	9.70%	0.288
	2	2	5.60%	0	0.00%	2	2.20%	
	3	4	11.10%	4	7.00%	8	8.60%	
	5	3	8.30%	4	7.00%	7	7.50%	
	6	12	33.30%	17	29.80%	29	31.20%	
	7	9	25.00%	24	42.10%	33	35.50%	
	8	1	2.80%	4	7.00%	5	5.40%	
Fim rating	Complete independence	1	3.20%	3	5.76%	4	4.30%	0.637
	Maximal assistance	5	16.12%	4	7.69%	9	9.70%	
	Minimal assistance	10	27.80%	20	38.46%	30	32.30%	
	Moderate assistance	6	32.25%	11	21.15%	17	18.30%	
	Modified assistance	0	0.00%	1	1.92%	1	1.10%	
	Supervision	8	25.80%	14	26.92%	22	23.70%	
	Total assistance	1	3.20%	0	0.00%	1	1.10%	

In early cranioplasty cohort GOS E score of 6 (upper moderate severity) was seen in 32.25% cases, whereas a better score of 7 (lower good recovery) was seen in 42.10% cases in late cranioplasty cohort. Overall statistics of both cohort shows a lower good recovery in 35.50% cases. In FIM rating early cranioplasty and late cranioplasty both had maximum number of cases in Minimal Assistance group (early, *n* = 10, 27.80%; late, *n* = 20, 35.10%). This was closely followed by supervision in both groups. (Table 4) The length of stay in early cranioplasty cohort is 9.97 ± 4.61 SD and in late cranioplasty cohort it is 10.67 ± 5.63 SD days. A slight better outcome in FIM score (79.32 ± 21.33) has been noted in late cranioplasty cohort in comparison to the early group (74.94 ± 22.22).

Discussion

The current study looked into how the scheduling of a CP at a Level l trauma center affected the neurological outcome and postoperative complications. There was no statistically significant difference in the overall postoperative complication rates between patients who had early versus late CP (p=0.138). Early versus late subgroup P-values were statistically significant in relation to post operative complications i.e., seizures, post CP hydrocephalus and surgical site infections. Along with that statistical significance has been found in late CP cohort cases who presented with subdural hygroma. The timing of the CP had no effect on the neurological outcome as measured by the FIM and GOSE

Although early CP is defined differently in the literature, it is typically defined as occurring 90 days after DC. As a result, the 90-day cut off criterion was used in the current investigation.[11] Being a trauma center, we also saw a lot of patients with severe TBI, which resulted in malignant cerebral edema. Therefore, it is noteworthy to mention that the FIM and GOSE are expected to slowly improve overtime after decompressive craniectomy (DC). As a result, the FIM and GOS-E were recorded after cranioplasty and not after DC.

30.10% cases (n: 28) in total had post operative complication in our study. 36.85% (n: 21) in late cranioplasty cases and 22.20% cases (n: 9) in early cranioplasty cohort had complications. Cranioplasty, although a routine neurosurgical procedure, can cause significant morbidity; rates of total major complications between 10.9% and 40.4% have been reported in the literature.[1, 12]

In our study 13 cases presented with post DC hydrocephalus and 8 had post cranioplasty ventriculomegaly (early, *n* = 3, 8.30%; late, *n* = 5, 10.52%; *P* <0.001). Hydrocephalus may start to develop secondarily to the disturbance in the dynamics of cerebrospinal fluid. Several studies reported the incidence of hydrocephalus after cranioplasty.[13, 14] Some studies defined hydrocephalus as the need of ventriculoperitoneal shunt insertion. On the contrary, other studies defined hydrocephalus as the finding of dilated ventricles on CT images with or without neurological deficit or poor improvement. The incidence of hydrocephalus reported by these studies were in range 1.4–12.2% which was similar to our findings.[13, 14, 15]

In the late CP cohort 3 patients presented with subdural hygroma (SDG), which had a significant correlation with presenting late following DC for cranioplasty. The generally preferred pathophysiological theory postulates a one-way flap valve mechanism as a result of the traumatic arachnoid membrane rupture that causes cerebrospinal fluid (CSF) entrance in the subdural space.[16, 17] The dura-arachnoid interface, a layer made up of tiny collagen and cellular components kept together loosely by an amorphous substance susceptible to breakdown by physical stress, is thought to be the primary cause of this occurrence.[18] There have, however, been numerous hypotheses put out as to how the SDG came to be. According to the blood brain barrier failure theory, a major contributing cause to the buildup of water in the subdural space and the SDG expansion is the increased permeability of blood capillaries, which raises the osmotic pressure in the subdural space.[18, 19, 20] We postulate that one way flap valve mechanism secondary to arachnoid membrane rupture following traumatic brain injury can result in accumulation of fluid over time. The more duration post DECRA, the more would be the fluid being collected. Along with that post DECRA external cerebral herniation can result in furthering the process of fluid collection and as such we found significant cases of SDG in the late cranioplasty cohort.

A total of 11 patients presented with surgical site infection (SSI) postoperatively (Early, *n* = 3, 8.30%; Late, *n* = 8, 14.03%, p<0.001). The SSI rate in our study is 8.60% which is significantly low than that in literature.[21] The rate of infection in early versus late cranioplasty was statistically insignificant, according to a comprehensive review by Yadla et al.[22] Many published research also support the correlation of DC for TBI and greater post-CP infection rates.[6, 23] The initial contamination of the skull by traumatic discontinuities in the skin, as well as the presence of numerous scars from trauma and surgery impair the vascularization of the surgical flap and delay the healing of the wound after CP. The bigger skull abnormalities that developed after DC after TBI and their connection to a delayed integration of the reimplanted bone flap may also be at play in this situation. Lastly, the possible involvement of the air

cavities of the skull, involved by fractures or by the decompressive flap, may also increase the infection rate of the reimplanted bone after CP. [24, 25, 26] But in our study, we hypothesize that the longer we wait after DC, the greater the likelihood of increased adhesion between the various layers of the scalp and the underlying duramater and its augmented portion. As a result, it must undergo substantial dissection during the process, requiring significant tissue handling over a prolonged period of time. Due to the flap's constant contact against the borders of the craniectomy site, there is a risk that there may be microischemic alterations in addition to the increase in intraoperative duration. Higher intraoperative duration, higher adhesions between the layers and the potential for ischemia alterations in the flap around the borders of the craniectomy site may all contribute to an increase in the post-CP infection rate in the late cranioplasty.

Diffuse cerebral edema and ischemia cause toxic mediators and excitatory cellular components to be released, which cause the onset of early seizures. As an extradural procedure, cranioplasty requires handling of brain tissue during the extradural plane dissection, which could result in cerebral edema. The dissection will lessen localized cerebral blood flow due to the severance between the scalp and dura mater, which may cause relative ischemia in the underlying cortex. In order to enable the improved shape of the bone flap after cranioplasty, brain tissue is additionally altered. These modifications may cause seizure activity in already seizure suceptible brain tissue and is the principal hypothesis behind post-cranioplasty seizures.[27, 28 29] Seizures are a significant, well-documented side effect of cranioplasty. In our study, there were 4.30% occurrences of post-cranioplasty new-onset seizures.

All the cases were in the late cranioplasty cohort, thus providing a significant correlation between new onset seizures with late cranioplasty. According to a systemic analysis by Malcom JG et al, the incidence of new-onset seizures after cranioplasty is 5-6%.[30] In their study 37% of early seizures following cranioplasty occurred within the first 24 hours, 16% occurred during the first week, and 47% occurred after that. In a study published in 2018, Yeap et al. looked into the prevalence of post-cranioplasty seizures among patients who received the procedure at their facility. 89 patients (26.5%) out of 336 patients with no prior history of seizures developed new-onset seizures. Similar studies have stated development of post CP seizures but the incidence of seizures in both groups (early vs. late) were statistically insignificant.[30, 31, 32]

Bitemporal and convexity cranioplasties were linked to seizures in a research by Zanaty et al., however the link was not statistically significant.[33] Increased seizure risk has been linked to surgery after cranioplasty for hematoma evacuation, but it has also been reported to be minor.[33, 34] In our investigation, all four patients of newly developing seizures underwent convexity cranioplasties, with hematoma evacuation performed in all. Timing of cranioplasty has been shown to be significant predictor for developing new onset seizure. As with our study, wherein we found significant correlation between late cranioplasty and occurrence of post CP, other studies also showed increased risk of seizure in patients who underwent late cranioplasty. [35, 36] Some studies also showed the benefits of early cranioplasty in minimizing the risk of seizure. [35, 36, 37]

Myocardial infarction was seen in 3.2% cases in our study, which were managed in consultation with cardiology. In a study done by Lester Lee et al 0.82% cases developed a non-ST elevated myocardial infarction (NSTEMI), immediately post-operatively.[38] A systematic review by De Cola *et al*. showed that motor improvement occurs in the early cranioplasty group, coupled with improved other parameters, such as cognition.[39] In another study by Archavlis *et al*., showed improved neurological function, including motor power if cranioplasty is performed within 7 weeks, compared to later than 7 weeks.[40] The speculate mechanism of why motor improvement occurs is mainly due to post cranioplasty physical therapy, precluding the timing difference of cranioplasty on motor improvement.[39, 40]

In our study we had a slight difference in the overall GOSE and functional independence measure in both the cohort. In early cranioplasty cohort GOS E score of 6 (upper moderate severity) was seen in 33.30% cases; whereas a better score of 7 (lower good recovery) was seen in 42.10% cases in late cranioplasty cohort. This may be attributed to the fact the patients in late cranioplasty cohort had longer duration of time to recover from the primary insult. In FIM rating early cranioplasty and

late cranioplasty both had maximum number of cases in Minimal Assistance group (early, *n* = 10, 27.80%; late, *n* = 20, 35.10%). A slight better outcome in FIM score (79.32 ± 21.33) has been noted in late cranioplasty cohort in comparison to the early group (74.94 ± 22.22). Di Stefano et al. demonstrated a greater degree of recovery after CP at 6 months, however Honeybul et al. (2016) found that CP performed between 3 and 6 months results in a greater degree of cognitive recovery.[41, 42] This improved outcome in cases of late CP is a result of the physiological cerebrospinal fluid circulation being restored, which in turn allows for an effective restoration of blood circulation and, as a result, of the large-scale neural networks important for cognition (Corallo et al., 2017). [43] The follow-up assessment was completed in Honeybul et al. (2016) after 3 days of CP, but Corallo et al. (2017) and Di Stefano et al. (2016) completed it after 1 month, indicating the significant discrepancy that could influence the results. This observation from the studies are crucial in our study as well, which might explain the better FIM and GOS-E score in the late CP cases. Similiarly, in many studies the greatest improvements were evident many months after cranioplasty and most of the clinical improvement due to cranioplasty is secondary to prolonged effects on brain physiology, rather than immediate changes. [41, 42, 43, 44]

In the early cranioplasty cohort, the length of stay was 9.97 ± 4.61 SD days, whereas in the late cranioplasty cohort, it was 10.67 ± 5.63 SD days. A Aloraidi et al, in their study also stated an increase length of stay in the group of patients who had late cranioplasty.[44] The longer length of stay in the late CP group can be due to the fact that they required more time to recover from postoperative complications, particularly SSI and seizures, which both required close monitoring and rapid therapy to stop the patient's further deterioration.

Conclusion

There are a few limitations that need to be considered before interpreting the results of the present experiment. The study's retrospective methodology makes it vulnerable to selection and information bias, which is its first flaw. Second, fewer people took part in the study because none of the patients who received non autologous cranioplasty were included. Despite these drawbacks, the current investigation demonstrated the significance of comparing the neurological outcome and post surgical complications. The neurological outcome in patients who underwent early versus late cranioplasty is almost identical with a slight better outcome in late CP cohort which can be attributed to longer recovery period since DC.

Early and late cranioplasty had statistically significant variations in the rates of all postoperative complications. We draw the conclusion that early cranioplasty often resulted in less post-operative morbidity. A lower number of early CP cohort subjects experienced post-CP HCP, seizures, and SSI. Early CP cases also typically have shorter hospital stays, which puts less strain on the hospital's resources. In order to reduce postoperative problems in TBI cases receiving DC, we advise an early treatment for CP based on the study's findings. Overall, the neurological result was not significantly affected by the time of the cranioplasty. Owing to the mentioned limitations in the study we recommend a multicenter, prospective studies investigating the neuropsychological outcome pre/postoperatively in relation to cranioplasty timing.

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Abbreviations

cranioplasty (CP)
decompressive craniectomy (DC)
subdural hygroma (SDG)
traumatic brain injury (TBI)
standard deviation (SD

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Lumbar neurofibroma: aetiology, clinical presentation, surgical indications, and surgical technique. A focused view about surgical experience in Viseu, Portugal

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ABSTRACT
Lumbar neurofibroma is a benign neoplasm that originates from peripheral nerves, specifically from Schwann cells. While it can occur at various anatomical sites, its manifestation in the lumbar region has specific clinical and neurosurgical aspects, which are of great relevance for accurate diagnosis and effective treatment. This article addresses the causes, clinical presentation, indications for surgical treatment, and the surgical technique applied to lumbar neurofibromas, based on key studies and articles published in the last ten years.

Keywords
neurofibroma, lumbar, root, plexus

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ETIOLOGY AND CAUSES

Neurofibromas can be classified as either sporadic or hereditary, with the latter often associated with Neurofibromatosis Type 1 (NF1), an autosomal dominant condition that results in multiple neurofibromas across various parts of the body. NF1 is caused by mutations in the NF1 gene located on chromosome 17, which codes for neurofibromin, a tumor suppressor protein. Patients with NF1 are predisposed to developing neurofibromas in various areas, including the spinal column, particularly in the lumbar region [1,2].

On the other hand, sporadic neurofibromas have no identifiable genetic cause and can occur at any age, with higher prevalence in young adults [3]. Pathogenesis involves abnormal proliferation of Schwann cells, fibroblasts, and other peripheral nervous system cells.

CLINICAL PRESENTATION

The clinical presentation of lumbar neurofibromas can vary significantly depending on the size, location, and compression of adjacent structures, such as nerve roots and the spinal cord. Common symptoms include low back pain, radiculopathy, sensory loss, or

muscle weakness, with patients often affected by compression of spinal nerves exiting the lumbar spine [4].

In more severe cases, spinal cord compression can lead to more significant neurological symptoms, such as paralysis, urinary and fecal dysfunction, and loss of reflexes. Pain is a predominant symptom, which may be continuous or intermittent, often exacerbated by movement of the spine [5].

Additionally, patients with NF1 may present characteristics associated with the syndrome, such as café-au-lait spots and Lisch nodules, which can assist in the differential diagnosis [6]. Magnetic resonance imaging (MRI) is the imaging modality of choice for evaluating lumbar neurofibromas, enabling the identification of lesion extent, compression of surrounding structures, and the type of tissue involved.

Surgical indication

The main indication for surgery in cases of lumbar neurofibroma is the presence of progressive neurological symptoms or failure of conservative treatment. Surgery is indicated when there is evidence of nerve root or spinal cord compression causing debilitating pain, neurological deficit, or functional impairment [1].

Furthermore, surgical removal is recommended in symptomatic neurofibromas that interfere with the patient's daily activities or when malignancy is suspected, a rare but possible condition that can occur in long-standing neurofibromas. The evaluation of malignancy is based on clinical and histological characteristics, with malignant transformation to neurofibrosarcoma being a severe complication that requires urgent intervention [7].

Surgical technique

The surgical approach to lumbar neurofibromas must be carefully planned to minimize the risk of neurological injury and maximize tumor removal. The standard procedure involves lumbar laminectomy or extended laminectomy to ensure access to the neurofibroma, which is often located in extradurally or paraspinal regions. The choice of laminectomy type depends on the tumor's location and size [8].

The initial step in surgery involves careful exposure of the lumbar spinal structures and removal of the vertebral lamina, followed by identification and dissection of the neurofibroma. In cases of intradural neurofibromas, a more invasive approach may be necessary, with manipulation of the dura mater and nerve roots. The neurofibroma should be excised as completely as possible without compromising the integrity of the nerve roots, with attention to prevent damage to the spinal cord.

In some cases, intraoperative neurophysiological monitoring (IONM) may be helpful to protect neurological structures during excision, especially in tumors located in difficult-to-access areas [2]. After tumor resection, the lumbar spine may be stabilized, if necessary, with instrumentation, depending on the extent of bone removal. The goal is to ensure functional recovery without additional neurological deficits.

In our series in two cases mini retroperitoneal approach was uses for tumor removal and in one case the tumor was approached through extended laminectomy.

Postoperative considerations

Postoperative management of patients undergoing lumbar neurofibroma resection includes pain control, neurological monitoring, and early physiotherapy to promote mobility recovery. Full recovery may vary depending on the severity of preoperative symptoms and the extent of surgery. In NF1 cases, continuous monitoring is important to detect the development of new neurofibromas over time [4].

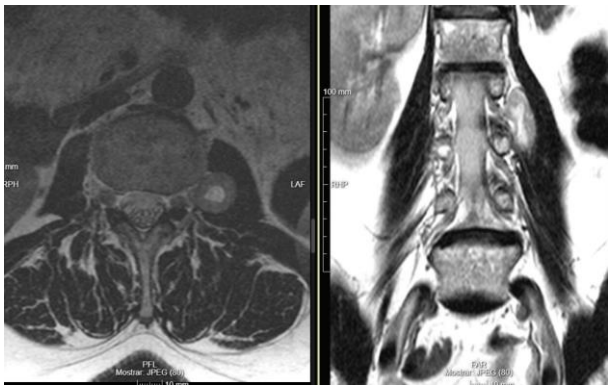


Figure 1. MRI extra canalar, intrapssoas muscle tumor, arising from left side L2 root.

Case 1.

59 years old lady with history of renal TB treated, later left side nephrectomy due to lithiasis, presenting left side radicular pain L3 treated conservatively with no effect. Lumbar MRI showed extracanalar, intrapssoas muscle tumor, arising from left side L2 root (Fig. 1). She was operated through left side mini retroiperitoneal approach with total removal of the tumor with resolution of complaints. Two years after surgery she is doing well, MRI revealed no tumor recurrence (Fig. 2).

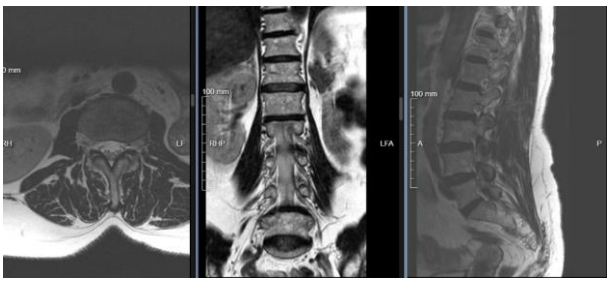
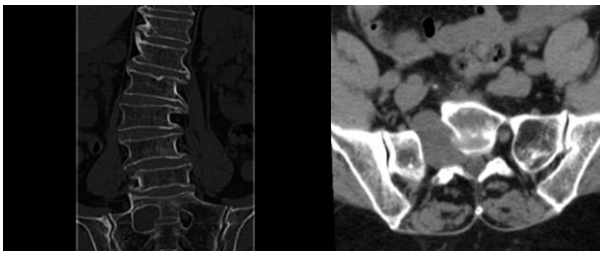


Figure 2. Postoperative MRI 2 years after surgery showing no recurrence of tumor.

Case 2.

73 years old lady with a long lasting hystory of right side ciatic pain, worthened by association of progressive neurologic claudication. CT scan showd severe L5-S1 stenosis and sacral errosion with enlagement of right side S1 foramina by a tumor arizing from extracanalar para of S1 root with evolution to presacral, retroperitoneal space (Fig. 3). She was submitted to psterior decompression of low lumar spine with fixation with transpedicular screws and S1 tumor total removal. 4 years after surgery she is doing well, complaining of numbness of righr leg, independent, using sporadically pain killers. CT scan 4 years after surgery shows no recurrence and the tumor bad is filled with bone (Fig. 4).



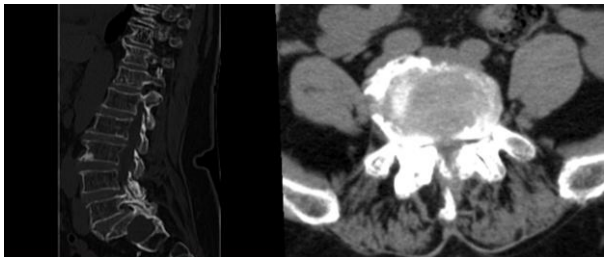


Figure 3. CT scan revealing enlargement of right-side S1 foramina by huge tumor, associated by L5-S1 severe central canal stenosis.

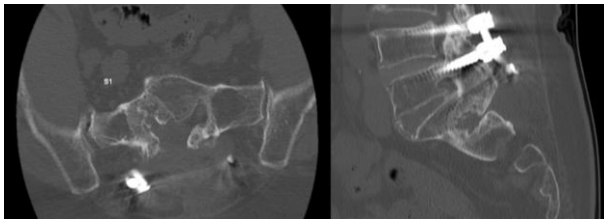


Figure 4. SC scan 4 years after surgery, partial bone filling of the tumor bed.

Case 3.

45 years old lady with complants of right side L2 pain with few effect with pain killers. MRI found left-side intrapssoas tumor adjacent to L3 root (Fig. 5). She was operated through left-side mini retroperitoneal approach with total removal of tumor and after two year MRI revealed no recurrence (Fig. 6).

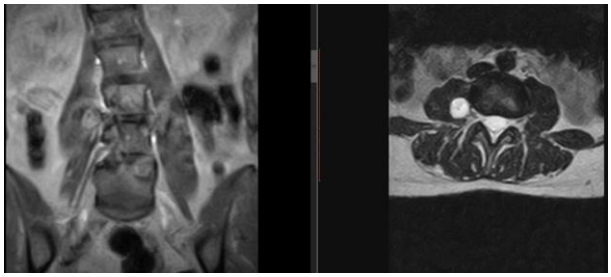


Figure 5. MRI right-side extra canalar, intrapssos tumor razing from L3 root.

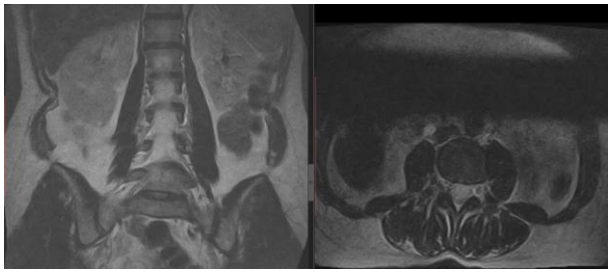


Figure 6. Postoperative MRI, 2 years after surgery, showing no recurrence of tumor.

Conclusion

Lumbar neurofibromas represent a significant clinical and neurosurgical challenge, requiring early diagnosis and appropriate management to prevent neurological complications. Surgical intervention is crucial in symptomatic cases, with tumor resection being the most effective strategy to alleviate symptoms and improve the quality of life of patients. Modern intraoperative monitoring techniques and spinal stabilization options contribute to a high surgical success rate.

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Eagle Syndrome. A contemporary review and clinical challenges

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ABSTRACT
Eagle Syndrome (ES) is a rare but clinically significant condition characterised by the elongation or calcification of the styloid process or stylohyoid ligament, leading to a spectrum of symptoms such as cervical, pharyngeal, and facial pain. The pathophysiological mechanisms remain inadequately understood, with potential etiological factors ranging from developmental anomalies to post-traumatic alterations. This comprehensive review explores the clinical presentation, diagnostic advancements, epidemiological trends, and management strategies for Eagle Syndrome. By enhancing awareness and understanding of this often-misdiagnosed condition, this review aims to facilitate improved patient outcomes through timely recognition and tailored management.

Keywords
styloid process, Eagle

✉
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INTRODUCTION

Eagle Syndrome, initially characterized by Dr. William Eagle in 1656, presents a unique constellation of symptoms arising from elongation or abnormal calcification of the styloid process and stylohyoid ligament. Patients often exhibit oropharyngeal and cervical discomfort, frequently mistaken for common disorders such as temporomandibular joint dysfunction or pharyngitis. The clinical significance of this syndrome lies in the potential morbidity associated with its late diagnosis, underscoring the necessity for a high index of suspicion in clinical practice. (1)

The styloid process typically measures between 2.5 cm and 3 cm. Elongation exceeding this threshold may result in irritation or compression of adjacent anatomical structures, including cranial nerves, which leads to characteristic pain syndromes. Despite its infrequency, a heightened awareness of Eagle Syndrome as a differential diagnosis in cases of unexplained orofacial pain is increasingly recognized within the medical community. (1, 3, 4)

Epidemiology

Eagle Syndrome affects an estimated 4% to 10% of the population, though true prevalence might be obscured due to clinical underreporting. The condition is predominantly diagnosed in adults aged 30 to 60 years, with a notable female predominance (female-to-male ratio of 2:1 to 3:1). Geographic prevalence varies significantly, with increased rates observed in regions with higher incidences of head trauma. (5)

Populational risk groups

Though Eagle Syndrome can affect anyone, certain populations show heightened susceptibility. Those with histories of blunt trauma, particularly whiplash injuries, are at increased risk of developing symptomatic presentations. Chronic throat irritation, frequent episodes of tonsillitis, and anatomical variations such as congenital elongation of the styloid process also contribute to the syndrome's onset. (5)

Clinical diagnosis

Diagnosing Eagle Syndrome is a complex endeavor that necessitates both a thorough clinical evaluation and a high degree of clinical suspicion. Patients commonly report recurrent or persistent oropharyngeal pain, a sensation of a foreign body in the throat, and dysphagia-all of which can overlap with a myriad of other medical conditions. (2, 3)

Advanced imaging techniques, including panoramic radiography and computed tomography (CT), are imperative for diagnostic confirmation. These modalities enable the detailed visualization of elongation, supporting the accurate diagnosis of Eagle Syndrome. (2, 3)

Management and treatment

The management of Eagle Syndrome is tailored to the severity of symptoms. First-line approaches typically involve conservative treatments, including nonsteroidal anti-inflammatory medications, corticosteroid injections, and targeted physiotherapy to alleviate musculoskeletal tension. (4)

Surgical intervention becomes critical for those with refractory symptoms. Styloidectomy, the surgical resection of the elongated styloid process, remains the treatment of choice for patients with persistent or severe manifestations. Contemporary advancements in minimally invasive techniques have revolutionized surgical outcomes, allowing for quicker recoveries and reducing complication rates. (4, 5, 6)

Comparing treatment modalities: Historical vs. Current practices

The management of Eagle Syndrome has evolved significantly over the years. While early treatments were primarily symptomatic (pain management, physical therapy, and corticosteroid injections), modern approaches are more focused on surgical interventions, with endoscopic and minimally invasive procedures becoming more common.

1. **Conservative Management:** Conservative management is still considered for mild cases, particularly for patients who may not be suitable candidates for surgery due to comorbidities or personal preferences. Nonsteroidal anti-inflammatory drugs (NSAIDs) and corticosteroid injections have been used with varying degrees of success. For patients who experience less severe symptoms, conservative approaches may provide satisfactory relief without the need for invasive procedures. Additionally, physical therapy targeting the neck and jaw can sometimes help reduce symptoms related to muscle tension or spasm.
2. **Surgical Management:** The gold standard for treatment, especially in moderate to severe cases, has become surgical intervention, specifically styloidectomy (removal of the elongated styloid process). Traditional open surgical approaches, while effective, can result in extended recovery times and higher complication rates. However, with advancements in minimally invasive surgery, such as endoscopic styloidectomy, recovery time has been reduced, and complications have become less common. Endoscopic surgery offers greater precision, reduced scarring, and faster recovery, making it a preferred option for many surgeons today.
3. **Outcome and Efficacy:** Studies suggest that surgical intervention, particularly styloidectomy, offers the most effective and durable relief for patients with Eagle Syndrome. One of the primary reasons for the success of surgical

treatments is that they address the underlying cause of the condition – the elongated styloid process. In contrast, conservative treatments only provide symptomatic relief without addressing the anatomical abnormality. Post-operative outcomes are generally favorable, with many patients reporting complete or significant resolution of symptoms.

In comparison to the past, the overall prognosis for patients has improved. The advent of modern imaging techniques has made early diagnosis more achievable, and minimally invasive surgical techniques have greatly improved treatment outcomes. Patients who undergo surgical intervention typically experience a reduction in pain and discomfort, with many returning to normal activities within a few weeks. (5, 6)

CLINICAL CASE PRESENTATION

To further enrich the clinical relevance of this review, we propose the inclusion of a dedicated section showcasing clinical case presentations that embody the multifaceted nature of Eagle Syndrome.

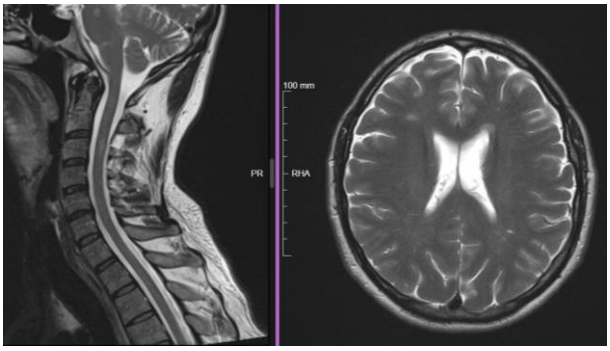

Figure 1. MRI revealing brain and cervical spinal cord lesions.

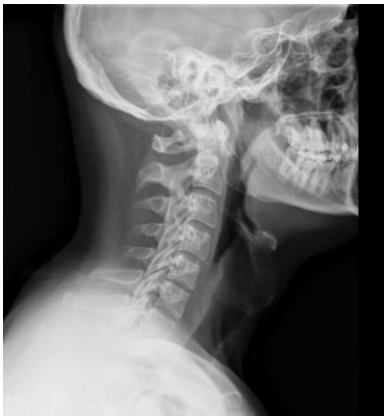

Figure 2. X-ray suspecting Eagle syndrome

Our patient is 46 years old man, diagnosed with multiple sclerosis (Figure 1) for 16 months, treated with immunomodulatory therapy (Natalizumab). He was complaining of neck pain (VAS 6) and other symptoms related to multiple sclerosis and on X-ray was suspected Eagle syndrome (Figure 2), confirmed on CT scan, measuring a little more than 5cm (Figure 3).

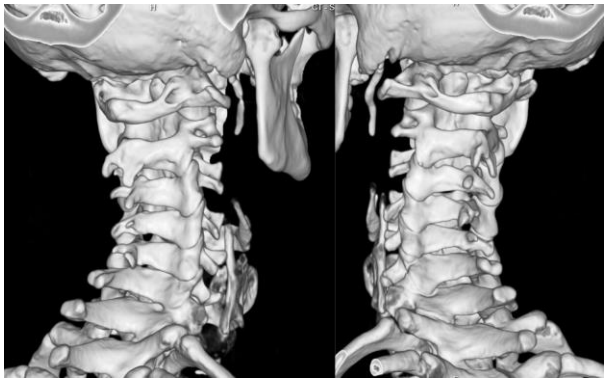

Figure 3. CT scan confirms Eagle syndrome.

The conservative treatment was adopted and, in this case, and he improved with physiotherapy.

DISCUSSION

Eagle Syndrome remains a challenging diagnosis due to its nonspecific symptoms and the fact that it is often misdiagnosed as other more common conditions, such as temporomandibular joint dysfunction, pharyngitis, or even dental problems. In the case of Eagle Syndrome treated conservatively at our hospital, several common diagnostic challenges were observed, overlapping with diagnosis of multiple sclerosis, proving the effectiveness of conservative management strategies.

Diagnostic Challenges

In our case, the patient presented with a constellation of symptoms, including oropharyngeal pain, dysphagia, a foreign body sensation in the throat, and cervical discomfort, which are often attributed to other, more common conditions, in this case multiple sclerosis. This made early recognition of Eagle Syndrome difficult. Common issues included:

1. **Overlapping Symptoms with Other Conditions:** The symptoms of Eagle Syndrome can mimic other conditions such as temporomandibular joint dysfunction (TMJD) or chronic pharyngitis, both of which are far more frequently diagnosed. This overlap led to initial misdiagnoses and delays in proper treatment.
2. **Uncommon Diagnosis:** Eagle Syndrome is not frequently encountered in routine clinical practice, which resulted in a high degree of clinical uncertainty. Despite the syndrome being relatively rare, it is essential for clinicians to maintain a high index of suspicion, especially in patients with unexplained throat pain and a history of trauma (such as whiplash).
3. **Challenges in Imaging:** While panoramic radiographs and CT scans were employed, there was some delay in obtaining the necessary imaging. Patients initially underwent more routine investigations, such dental exams, which failed to reveal the underlying structural abnormality. Only upon obtaining detailed imaging was the elongated styloid process or calcified stylohyoid ligament identified, confirming the diagnosis.
4. **Atypical Presentations:** Some patients presented with atypical symptoms, such as severe headaches or ear pain, which further complicated the diagnosis. These atypical presentations are sometimes overlooked as being unrelated to the primary symptoms, leading to prolonged diagnostic workups.

Efficacy of Conservative Treatment Modalities

Despite the diagnostic challenges, the patient responded well to conservative treatment, underscoring the importance of early intervention and appropriate management, even in cases where surgical intervention was not immediately considered.

1. **Nonsteroidal Anti-Inflammatory Drugs (NSAIDs):** NSAIDs were the first-line treatment used in our cases. These medications helped reduce the inflammation around the elongated styloid process and alleviated the associated pain. In the majority of cases, patients reported significant relief within a few weeks of treatment, which allowed them to better manage their symptoms and return to normal daily activities.
2. **Physiotherapy**: Targeted physiotherapy was employed to address muscle tension in the neck and jaw, which is often a secondary issue due to the discomfort caused by the elongated styloid process. Physiotherapy included gentle stretching exercises and relaxation techniques, this treatment was highly beneficial in reducing associated muscle spasms and improving neck mobility, thus contributing to symptom relief.
3. **Patient Education and Lifestyle Modifications:** Educating patients about their condition and recommending lifestyle modifications were essential in managing symptoms conservatively. The patient was advised to avoid prolonged periods of throat irritation (e.g., speaking loudly or swallowing large food bites) and to engage in relaxation techniques to reduce stress, which could exacerbate muscular tension in the neck.
4. **Observation and Follow-up:** Given the varied response to conservative treatments, close monitoring and regular follow-ups is critical, after several months of conservative treatment, symptoms fully resolved.

Outcomes and Future Considerations

In our case, conservative management was successful in relieving the symptoms of Eagle Syndrome without the need for surgical intervention. This case highlight that, while surgery (such as styloidectomy) is the gold standard for more severe or refractory cases, conservative management can be highly effective, particularly in mild to moderate cases. The outcomes observed in this patient emphasize the importance of early diagnosis and individualized management plans.

However, it should be noted that conservative treatment is not universally successful in all patients. The success of conservative treatment can depend on factors such as the degree of elongation of the styloid process, the presence of calcification, and the overall health and pain tolerance of the patient. For some, the persistent nature of the symptoms may eventually necessitate surgical intervention, especially if the pain is unresponsive to repeated conservative measures.

Conclusion

The case treated conservatively demonstrated that conservative management of Eagle Syndrome can be effective, particularly in mild cases. Early recognition and treatment are crucial in reducing the risk of misdiagnosis and prolonged suffering. The combination of NSAIDs, physiotherapy, and lifestyle modifications provided significant relief for patients, minimizing the need for surgical interventions. However, ongoing research and more case studies are needed to refine the guidelines for conservative treatment, especially for patients with more severe presentations or complex cases.

In summary, this case highlights the importance of a comprehensive, multidisciplinary approach in managing Eagle Syndrome. While surgical options should remain a consideration for refractory cases, conservative management strategies, when applied early and effectively, can significantly improve patient outcomes.

Eagle Syndrome remains an underappreciated yet clinically relevant condition that requires heightened vigilance in its recognition and management. Advances in imaging and surgical techniques have significantly improved patient prognosis, enabling earlier recognition and more effective intervention. While conservative management continues to play an essential role in treating mild cases, surgical options, notably minimally invasive styloidectomy, are becoming the gold standard for more severe manifestations. As our understanding of Eagle Syndrome evolves, further research into its pathophysiological underpinnings and treatment options will be critical in optimizing clinical outcomes for affected individuals.

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Human dorsal pseudotail. A rare congenital anomaly case report with review of literature

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ABSTRACT

There are two different types of human tails: real tails and pseudotails. While pseudotails can be an abnormal extension of the coccygeal vertebra, lipoma, teratoma, chondrodystrophy, or parasitic fetus, true tails are made of adipose tissue, connective tissue, muscles, arteries, nerves, and mechanoreceptors. We are going to present a case of dorsal pseudotail in a 06 months old infant.

Keywords
human tail,
pseudotail

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INTRODUCTION

The human tail is an uncommon anatomical occurrence and a topic of considerate curiosity. Early scientists had a concept of recapitulation including ontogeny and phylogeny, or "reversion to a lower species," because of the human tail (1). The predominant site of the human tail is the lumbosacrococcygeal area, there have been no reports of dorsal pseudotails. The presence of a human tail is an indicator of underlying spinal dysraphism and tethered cord, which may be linked to neurological issues (2, 5). Only about 40 cases have been reported so far, the first case to be reported dates back to 1881 (4). We present an additional case and review the literature.

CASE REPORT

A 06 months old female child was presented to our outpatient department with history of a dorsal tail-like structure since birth. The mother had no significant antenatal history and family history was also not contributory.

On examination, the child had 5.7 cm-long tail shaped soft tissue appendage arising from dorsal region which was fleshy with bony content, non-translucent, completely covered by sensate skin, no obvious meningocele and had no spontaneous movement [Figure 1]. The remainder of neurological examination was uneventful. CT scan [Figure 2] and magnetic resonance imaging (MRI) of the spine [Figure 3] showed a dorsal bone formation of 69 mm at the height of D1, a syringomyelic cavity opposite this formation, opening of the posterior arch from C7 to D5 and L4 posterior arch closure defect.

Our patient underwent excision of the vestigial tail [Figure 4] and made an uneventful recovery. Microscopy of the excised tail showed skin tissue made of a thinned epidermis over a dermis which contains sweat glands, pilosebaceous follicles and small vessels. Deeper we found adipose tissue which surrounds an osteocartilaginous tissue. Absence of glial tissue and meningeal structures.At one month follow-up, the wound had healed well and a neurological examination was normal at subsequent monthly follow-up.

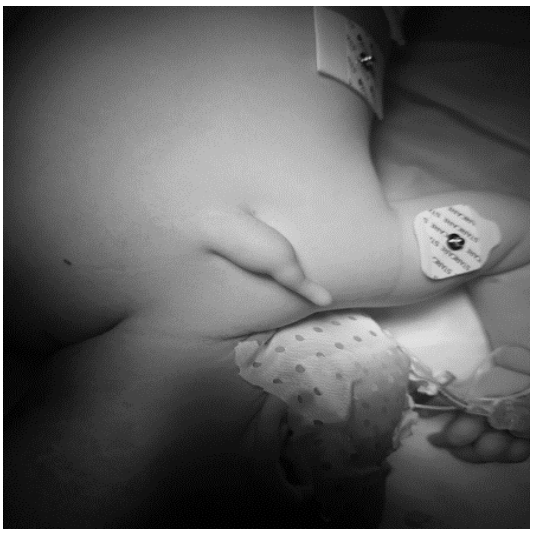

Figure 1. Dorsal congenital pseudotail in our patient

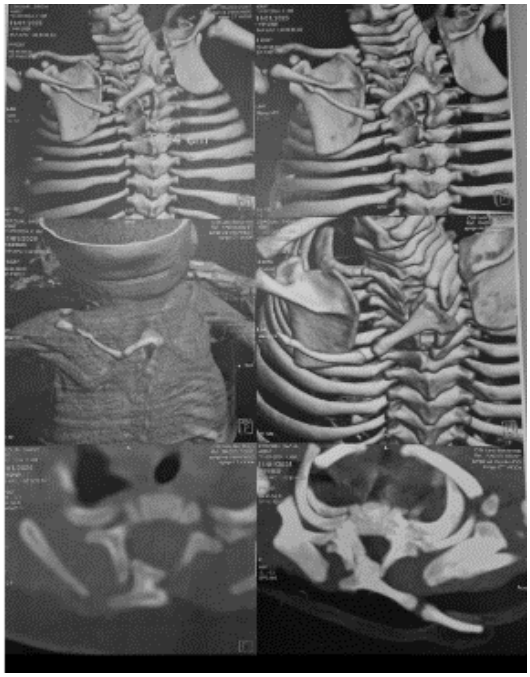

Figure 2. CT Scanning showing the pseudotail.

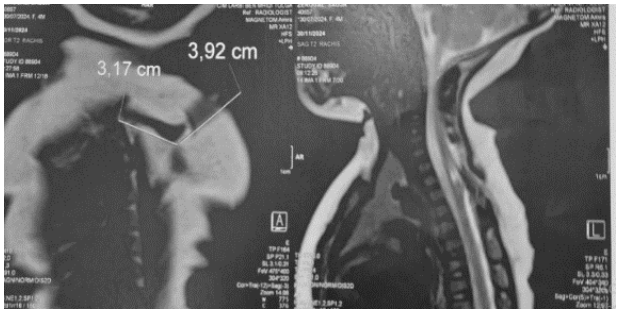

Figure 3. MRI showing the malformation.

Figure 4. Excision of the pseudotail.

Discussion

The human tail is an uncommon and benign congenital condition. As stated by Lu et al., the occurrence of a human tail is an aberration in fetal development, not a regression in the evolutionary process (6).

Human tail is a midline protrusion in the caudal region composed of muscle, adipose tissue, and skin (1). During the fifth and sixth weeks of gestation, the human embryo develops a tail with 10-12 caudal vertebrae (2). The tail typically regresses with fewer vertebrae and fusion, leaving the vestigial coccyx. It normally fades by the eighth week of gestation (2).

Bartelet et al. suggested the first classification of human tails in 1884 (4). He divided human caudal tails into four types based on their morphology and the presence of osseous tissue. Dao and Netsky (2) defined human tails as real or pseudotails based on histology in 1984. According to this classification, true tails contain muscle, adipose tissue, and connective tissue, whereas pseudotails contain

bone, cartilage, and notochord remains. In 2016, a new taxonomy of human tails was proposed, with five categories: soft tissue caudal appendages, bony caudal appendages, bony caudal prominence, true tails, and additional caudal appendages (4).

Lin et al. proposed another classification scheme in which a lesion in the lumbosacral area is classified as a pseudo-tail, regardless of whether it is linked with spinal dysraphism. However, if the same lesion is located in the gluteal or coccygeal region, the presence of other lesions is critical in determining the diagnosis; it is classified as a pseudo-tail if there are any accompanying vertebral or spinal lesions, and as a real tail if not (6). Nonetheless, each of these classifications is more useful for embryology than clinical practice. Clinically, it is difficult to distinguish between the vestigial tail and pseudotails (3).

True human tails are not inherited; however, a case has been recorded in which three female generations from the same family were born with true human tails (6). The incidence of actual human tails is twice as high in males as in females, while the exact incidence is unknown. There have been less than 40 cases recorded in the literature so far (4).
CT scanning and an MRI of the spine are necessary to delineate the underlying disease and assist in surgical planning and prognosis (5).

The management of a true tail involves straightforward removal for aesthetic purposes, whereas the approach for a pseudo tail necessitates excision accompanied by the treatment of the underlying neural tube lesion. True tail 'cosmesis' is assessed at follow-up, whereas pseudo tail is assessed neurologically in the affected spinal region (6).

Conclusion

True human tails are simple skin extensions with an excellent outcome and only require an excision. However, pseudotails are potentially complex lesions with underlying vertebra or spinal anomaly, necessitating additional diagnostic work-up and specialized surgical procedures.

The goal is to distinguish actual tails from pseudotails and to provide appropriate therapy, since the lack of understanding of the condition may raise concerns about their prognosis and the best management options.

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A study of prognostic value of ASIA score in post operative outcome assessment in spine trauma in a tertiary care centre

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Keywords
spinal cord injuries, neurological recovery, American Spinal Injury Association Impairment Scale, prognostic value, traumatic spinal cord injury, rehabilitation

ABSTRACT

Background: Spinal cord injury (SCI) remains a major cause of long-term disability and reduced quality of life, with significant socioeconomic and healthcare implications. The American Spinal Injury Association Impairment Scale (AIS) is a widely accepted tool for assessing the severity of SCI and predicting neurological recovery. While existing studies have examined AIS grade changes in Western populations, limited data are available from resource-limited settings such as India.

Objectives: (A) To determine the percentage of individuals with complete spinal cord injury (AIS A) who convert to incomplete status within the first post-injury year (B) To evaluate the extent of improvement in AIS grade from baseline to one-year follow-up among patients with initial AIS B injuries.

Methodology: This was a prospective observational study conducted at the Trauma Centre, Department of Neurosurgery, SMS Medical College, Jaipur, from December 2022 to December 2023. A total of 400 patients aged ≥16 years with traumatic SCI admitted within 30 days of injury were enrolled. Baseline AIS grades were recorded at admission, and neurological recovery was assessed at a one-year follow-up using the AIS classification. Statistical analysis involved chi-square tests to assess the significance of AIS grade changes.

Results: Of the 146 patients with initial AIS A injury, 38 (26.0%) improved to an incomplete status (AIS B, C, or D), while 108 (74.0%) remained unchanged. Among 66 patients with initial AIS B injuries, 26 (39.4%) showed improvement to a higher AIS grade, while 33 (50.0%) remained unchanged and 7 (10.6%) worsened to AIS A. AIS C and D patients demonstrated higher rates of recovery (56.5% and 54.4%, respectively), while no significant change was observed among AIS E patients. Statistical analysis confirmed a significant association between baseline AIS grade and post-operative outcomes ($p < 0.001$).

Conclusion: The study demonstrates that AIS scores are valuable in predicting post-operative neurological recovery in SCI patients. While AIS A patients exhibit limited potential for improvement, AIS B–D patients show higher recovery rates, reinforcing the importance of early intervention and targeted rehabilitative strategies. The findings underscore the need for tailored clinical management based on initial AIS grades to optimise patient outcomes in resource-limited settings.

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Introduction

Spinal cord injury (SCI) represents a significant global health challenge, often resulting in profound neurological deficits and long-term disability. Traumatic SCI is caused by events such as falls, road traffic accidents, and assaults.[3,5] It affects millions annually - an estimated global incidence of 10.5 cases per 100,000 people - as reported in various studies and systematic reviews.[2,4,10] The impact of SCI is pronounced in developing countries, where resource limitations exacerbate outcomes.[18,19] In India - the epidemiology of SCI is shaped by a high prevalence of falls and vehicular accidents and necessitates focussed research into prognostic tools and recovery patterns.

The American Spinal Injury Association Impairment Scale (AIS), part of the International Standards for Neurological Classification of Spinal Cord Injury, is a widely accepted tool for assessing the severity of SCI. Ranging from AIS A (complete injury with no motor or sensory function below the injury level) to AIS E (normal function), this scale provides a standardized framework for evaluating neurological status and tracking recovery. A study by Kirshblum et al. in 2020 emphasized its prognostic utility, noting that baseline AIS grades strongly correlate with long-term functional outcomes. For patients with AIS A injuries, conversion to an incomplete status (AIS B, C, D, or E) within the first year is a critical indicator of recovery potential, while improvement in AIS B patients reflects the capacity for neurological regain post-intervention.[8]

Research on AIS grade conversion rates has yielded variable findings. A study by Spiess et al. in 2004 reported that approximately 20–30% of AIS A patients in Italy converted to incomplete status within one year, influenced by factors such as injury level and timing of intervention. Conversely, a study by Lee et al. in 2016 found lower conversion rates (10–15%) in cervical SCI patients with AIS A.[9] They emphasized the challenges in predicting recovery for complete injuries. For AIS B patients, improvement rates to higher grades (C, D, or E) range from 30–50%, as noted in a study by van Middendorp et al. in 2020; this indicated greater plasticity in incomplete injuries. These discrepancies highlight the need for region-specific data, particularly in resource-constrained settings like India, where surgical and rehabilitative care may differ from Western contexts.[14]

Epidemiology and Burden of SCI in India

In India, the epidemiology of SCI reflects a unique set of socioeconomic and environmental factors. Road traffic accidents and falls are the leading causes of SCI, with workplace injuries and falls from height accounting for a substantial proportion of cases. One study reported that approximately 60% of traumatic SCI cases in India are caused by road traffic accidents, followed by falls (25%) and violence (5%). The majority of affected individuals are young adults (aged 18–40), which amplifies the long-term socioeconomic impact due to loss of productivity and increased caregiving burden.[13,20]

Despite the high incidence of SCI in India, accurate national data remain limited due to underreporting and inconsistent record-keeping, especially in rural areas. It is estimated that nearly 1.5 million people are living with SCI in India, with an annual incidence of approximately 20,000 new cases.[15] However, fewer than 10% of these individuals have access to specialized spinal care or rehabilitation services. There are gaps in studies and information available on recovery patterns and prognostic factors related to SCI.[1]

Healthcare and Rehabilitation Challenges in India

The healthcare infrastructure for SCI management in India faces several systemic challenges that contribute to poor outcomes. First, access to acute trauma care is limited, particularly in rural and semi-urban areas. A study found that fewer than 30% of SCI patients in India receive surgical intervention within the critical first 24 hours post-injury—a key determinant of neurological recovery.[16,17]

Specialized rehabilitation centres are scarce; India has fewer than 15 dedicated spinal rehabilitation centres for a population of over 1.4 billion.[7] Most rehabilitation services are concentrated in urban centres, making them inaccessible to rural populations. Financial constraints further compound these challenges. The cost of long-term care for SCI patients—including surgical, rehabilitative, and assistive care—places a significant burden on families, particularly in the absence of comprehensive health insurance coverage. A study reported that nearly 70% of SCI patients in developing countries experience catastrophic healthcare expenditures, which can push families into poverty. These healthcare

disparities highlight the need for prognostic models to assist and optimize treatments and outcomes.[11]

Our study sought to address some of these gaps by investigating the prognostic value of AIS scores for assessment of post-operative outcomes in traumatic SCI patients at a tertiary care centre in Jaipur, India. We focussed on heterogeneous sample which included tetraplegia and paraplegia. We aimed to assess the percentage of AIS A patients converting to incomplete status and the extent of AIS grade improvement in AIS B patients over one year.

Methodology

Our study was an observational study conducted at the Trauma Centre, Department of Neurosurgery, SMS Medical College, Jaipur, a tertiary care centre in India. The study duration was one year, from December 1, 2022, to December 31, 2023, with patient data collected for traumatic spinal cord injuries (SCI) and occurred between January 1, 2023 - December 31, 2023.

Participants were patients aged 16 years or older who were treated for traumatic SCI at the study site and provided written consent. Exclusion criteria included patients who did not receive a computed tomography (CT) scan within 24 hours of admission or those who suffered fatal injuries upon arrival. A total of 400 patients were enrolled – which we determined using the below sample size formula.

$$N = \frac{Z^2 \cdot P(1-P)}{L^2}$$

Where Z=1.96 (95% confidence interval), P=0.02 (2% proportion of AIS A conversion from literature), Q=0.98 (1 - P) and L=0.01 (1% precision) and this resulted in our sample size of 400. Data collection involved a comparative analysis of traumatic SCI cases admitted within 30 days of injury. Neurological status was assessed using the American Spinal Injury Association Impairment Scale (AIS) at baseline (pre-operative) and at a one-year follow-up post-operatively. Baseline AIS grades (A through E) were recorded upon admission, and follow-up assessments tracked changes in AIS grade after surgical intervention. Additional data included patient demographics (age, sex), diagnosis (fracture level) and mode of injury (e.g., fall from height, road traffic accident, assault, penetrating injury).

Data analysis was performed using SPSS v28 and reported using descriptive statistics to summarize patient characteristics and AIS grade changes; and distributional analysis. Inferential statistics (chi-square tests) employed to assess significance of AIS grade improvements and test our study hypotheses: the null hypothesis (H_0) stated no significant improvement in AIS score from baseline to follow-up, while the alternative hypothesis (H_1) posited significant improvement. Statistical significance was set at $p < 0.05$. All analyses were performed to address the objectives of determining the percentage of AIS A patients converting to incomplete status and the improvement in AIS grade among AIS B patients over one year.

Results

Demographic and Baseline Characteristics

Our study included 400 patients with traumatic spinal cord injuries treated at SMS Medical College, Jaipur, between January 1, 2023, and December 31, 2023. The mean age was about 37.8 years. Ages ranged from 14 to 73 years. Most patients were male (65.5%) and Female patients accounted for 34.5%. The most common cause of injury was falling from a height (47.0%) which was followed by road traffic accidents (38.5%). At the start of the study we observed that 36.5% of patients had a complete spinal cord injury (AIS A) and 16.5% had an AIS B injury.

Table 1. Demographics

Characteristic	Value
Total Patients	400
Mean Age (years)	37.8
Age Range (years)	14–73
Male, n (%)	262 (65.5%)
Female, n (%)	138 (34.5%)

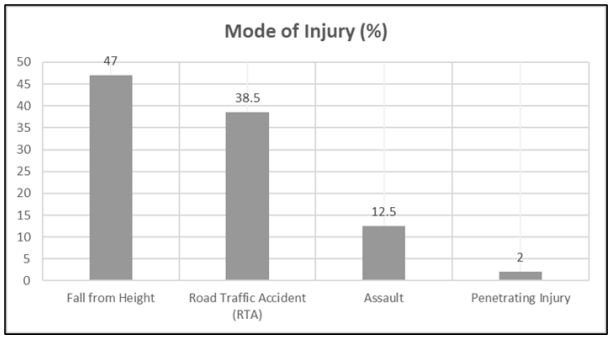

Figure 1. Mode of injury.

Table 2. Mode of Injury

Mode of Injury	Count (n)	Percentage (%)
Fall from Height	188	47
Road Traffic Accident (RTA)	154	38.5
Assault	50	12.5
Penetrating Injury	8	2
p-value (χ^2, df=3)	<0.001	

As illustrated in Table-2 and Figure-1 - Falls from height were the most common (47.0%, 188 patients), followed by road traffic accidents (38.5%, 154 patients). Assault caused 12.5% (50 patients), and penetrating injuries were rare (2.0%, 8 patients). The p-value (<0.001) shows these differences are significant. This indicated that the injury causes were not evenly split—falls and RTAs were the major contributors for mode of injury in our study cohort.

Table 3. Baseline AIS Grades

Baseline AIS Grade	Count (n)	Percentage (%)
A	146	36.5
B	66	16.5
C	62	15.5
D	90	22.5
E	36	9
p-value (χ^2, df=4)	<0.001	

Table-3 and Figure-2 show the starting AIS grades for our 400 patients. AIS A - a complete injury - was the most frequent (36.5%, 146 patients). This was followed by AIS D (22.5%, 90 patients). AIS B had 16.5% (66 patients) and AIS C and E were less common at 15.5% (62 patients) and 9.0% (36 patients). The p-value (<0.001) indicated statistical significance and indicated that more patients started with AIS A and D than expected.

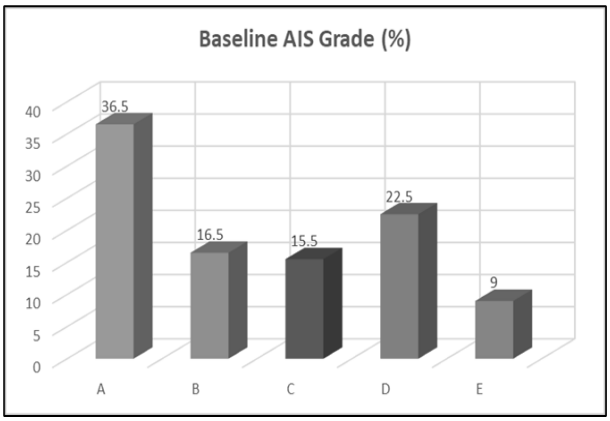

Figure 2. Baseline AIS Grades.

Conversion from AIS A to Incomplete Status

This section examines how many patients with complete spinal cord injuries (AIS A) improved to an incomplete status within one year and the results are illustrated in Table-4 and Figure-3.

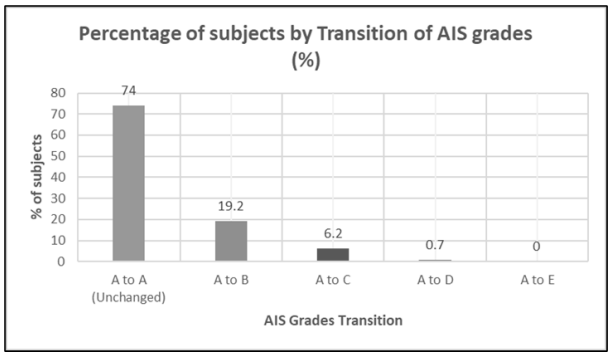

Figure 3. Conversion from AIS A to Incomplete Status.

Table 4. Conversion from AIS A to Incomplete Status

Transition	Count (n)	Percentage (%)
A to A (Unchanged)	108	74
A to B	28	19.2
A to C	9	6.2
A to D	1	0.7
A to E	0	0
p-value (χ^2, df=4)	<0.001	

Table-4 provides details on the 146 patients who started with AIS A (a complete spinal cord injury) out of the total 400 in the study. After one year - 38 of them (26.0%) improved to an incomplete status: 28 (19.2%) moved to AIS B, 9 (6.2%) to AIS C, and 1 (0.7%) to AIS D, with none reaching AIS E. Most, 108 patients (74.0%), stayed at AIS A with no change. The p-value (<0.001) shows these results are significant. This meant that about one in four AIS A patients gained some recovery - though most remained complete.

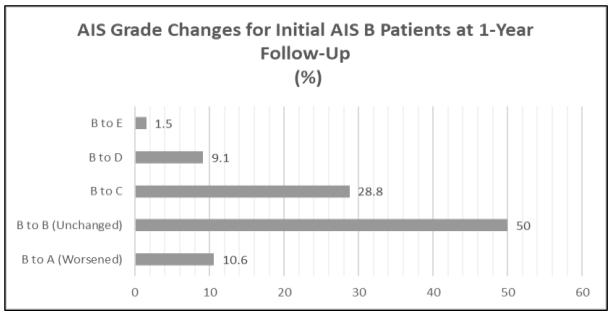

Figure 4. AIS Grade Changes for AIS B subjects.

Table 5. AIS Grade Changes for Initial AIS B Patients at 1-Year Follow-Up

Transition	Count (n)	Percentage (%)
B to A (Worsened)	7	10.6
B to B (Unchanged)	33	50
B to C	19	28.8
B to D	6	9.1
B to E	1	1.5
p-value (χ^2, df=4)	<0.001	

Improvement in AIS Grade for Initial AIS B Injuries

In this section we tracked number of patients with initial AIS B injuries who improved their AIS grade after one year.

Table-5 and Figure-4 focused on the 66 patients who started with AIS B - out of the total 400 in our study. After one year - 26 of them (39.4%) improved: 19 (28.8%) moved to AIS C, 6 (9.1%) to AIS D, and 1 (1.5%) to AIS E. Half, 33 patients (50.0%), stayed at AIS B; 7 (10.6%) got worse and dropped their grade to AIS A. The p-value (<0.001) showed that these changes are significant. This meant that about four in ten AIS B patients improved their grade and showed a good chance of recovery; though some stayed the same or worsened.

Overall AIS Grade Changes

Table-6 summarizes AIS grade changes across all patients from baseline to one-year follow-up. Figure-5 and Table-6 showed the final eventual condition of all 400 patients' AIS grades after one year.

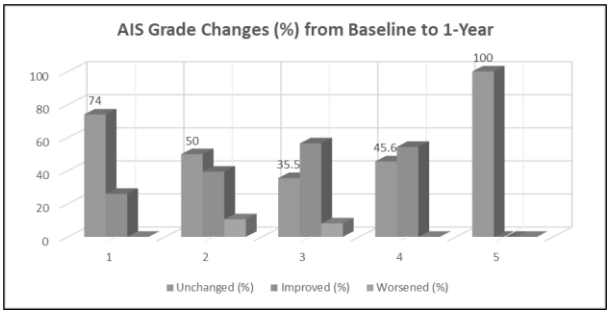

Figure 5. AIS Grade Changes from Baseline to 1-Year Follow-Up.

For AIS A (146 patients), 26.0% (38) improved, but 74.0% (108) stayed the same. For AIS B (66 patients), 39.4% (26) got better, 50.0% (33) didn't fluctuate and 10.6% (7) worsened. AIS C (62 patients) had the highest improvement rate at 56.5% (35), with 35.5% (22) unchanged and 8.1% (5) worse. AIS D (90 patients) saw 54.4% (49) improve to E, with 45.6% (41) staying D. All 36 AIS E patients stayed normal (100%). The p-value (<0.001) shows these patterns are significant. This supports our objectives: 26.0% of AIS A patients improved to incomplete status (Objective 1), and 39.4% of AIS B patients got better (Objective 2), with clear patterns across all grades for charting.

Table 6. AIS Grade Changes from Baseline to 1-Year Follow-Up

Baseline AIS	Count (n)	Unchanged, n (%)	Improved, n (%)	Worsened, n (%)
A	146	108 (74.0)	38 (26.0)	0 (0)
B	66	33 (50.0)	26 (39.4)	7 (10.6)
C	62	22 (35.5)	35 (56.5)	5 (8.1)
D	90	41 (45.6)	49 (54.4)	0 (0)
E	36	36 (100)	0 (0)	0 (0)
p-value (χ^2, df=8)	<0.001			

Statistical Analysis

This section presented statistical tests used to evaluate AIS grade changes and test our study hypotheses. Table-7 showed the statistical tests for our study. For all 400 patients - we observed a strong link between starting AIS grade and their status after one year ($p < 0.001$). This meant outcomes depended on the initial injury. For the 146 AIS A patients - 26.0% improved to incomplete status and the p-value (<0.001) confirmed this isn't random. For the 66 AIS B patients, 39.4% got better with a significant ($p < 0.001$) p-value. These results rejected our null hypothesis (no improvement) and supported our alternative hypothesis (significant improvement) and indicated AIS scores can improve post-surgery in spine trauma patients.

Table 7. Statistical Analysis of AIS Grade Changes

Analysis Focus	Group	Count (n)	Key Finding	Chi-Square (χ^2)	Degrees of Freedom (df)	p-value
Overall AIS Changes	All Patients	400	Significant association between baseline AIS and outcome	114.3	8	<0.001

AIS A Conversion	AIS A Patients	146	26.0% improved to incomplete status	223.45	4	<0.001
AIS B Improvement	AIS B Patients	66	39.4% improved to higher grade	43.39	4	<0.001

Discussion

The findings of our study provide significant insights into the prognostic value of the American Spinal Injury Association (ASIA) Impairment Scale (AIS) in post-operative outcome assessment of spinal cord injury (SCI) patients. Traumatic SCI remains a major cause of morbidity and long-term disability globally, particularly in developing countries where healthcare infrastructure and rehabilitative resources are limited. We conducted Our study at a tertiary care centre in Jaipur, India with the aim of evaluating the degree of recovery in SCI patients post-operatively over a one-year follow-up period. Our results contribute to evidence supporting the utility of AIS scores in predicting neurological recovery.

Age and Gender Distribution

In the present study, the mean age of the patients was 37.8 years (range: 14–73 years), which aligns closely with the findings of Mahmoodkhani et al. (mean age 39.64 years). However, Haque et al. reported a slightly lower mean age of 32.9 years (range: 18–57 years). This suggests that SCI in India and Iran tends to affect individuals in the third to fourth decades of life, reflecting a higher burden on the working-age population. The gender distribution also varied between the studies. In the present study, 65.5% of the patients were male, consistent with the pattern of male predominance observed in Mahmoodkhani et al. (74.7%), but Haque et al. did not report gender-specific findings. The higher proportion of males affected by SCI is consistent with global trends, which have been linked to greater male exposure to occupational and road traffic hazards.[6,12]

Mechanism of Injury

The mode of injury in the present study primarily involved falls from height (47.0%), followed by road traffic accidents (38.5%). In contrast, Mahmoodkhani et al. reported that road traffic accidents were the most common cause of SCI (54.4%); this was followed by falls (36.7%). Haque et al. did not report data on the mechanism of injury. The higher prevalence of falls in the present study may indicate differences in occupational hazards and the rural-urban injury distribution in India. The predominance of road traffic accidents in Mahmoodkhani et al. likely reflects differences in traffic patterns, infrastructure and safety regulations between Iran and India.[6,12]

Baseline AIS Grades

The baseline AIS grade distribution varied across the studies. In the present study, 36.5% of patients presented with complete injuries (AIS A), which was higher than the 15.2% reported by Mahmoodkhani et al. and the 6.45% reported by Haque et al. The higher proportion of AIS A cases in the present study suggests a more severe initial injury profile, which may reflect delays in accessing acute care or differences in injury severity. The proportion of AIS B patients in the present study (16.5%) was similar to Haque et al. (16.1%) but differed significantly from Mahmoodkhani et al., who reported no AIS B cases at baseline. AIS C and D cases were more frequent in Mahmoodkhani et al. (39.2% and 31.6%, respectively) and Haque et al. (41.93% and 35.48%, respectively) compared to the present study (15.5% and 22.5%). This difference may suggest that the population in Mahmoodkhani et al. and Haque et al. included a higher proportion of patients with incomplete injuries, which generally have a better prognosis.[6,12]

One of the key findings of Our study was that 26.0% of patients with complete spinal cord injuries (AIS A) converted to an incomplete status (AIS B, C, or D) within one year. This aligns with previous studies that have reported AIS A conversion rates ranging from 10% to 30% depending on factors such as injury level, early surgical intervention, and the quality of rehabilitative care. Spiess et al. (2004) observed similar rates of conversion in cases where early decompression and aggressive rehabilitative strategies were implemented.[21] The finding that 74% of AIS A patients remained unchanged implicates the challenges associated with neurological recovery in cases of complete injury. Complete SCI - by definition - involves total loss of

motor and sensory function below the level of injury; this limited the potential for spontaneous neurological recovery even with optimal surgical and rehabilitative management. This is also comparable to the 33.3% reported by Mahmoodkhani et al. but higher than rates reported in other Western studies (ranging from 10–15% in cervical injuries). Haque et al. did not report specific conversion rates for AIS A patients but noted that 48.38% of AIS B patients showed improvement by at least one AIS grade at discharge, which is higher than the 39.4% improvement rate observed in the present study.[12]

Our study also demonstrated that AIS B patients exhibited a higher rate of improvement, with 39.4% improving to higher grades (C, D, or E) over the course of one year. This finding is consistent with the work of van Middendorp et al. (2020) – who also reported that AIS B patients show greater neurological plasticity compared to AIS A patients.[14] They attributed this to preserved sensory pathways - which may facilitate motor recovery through neuroplastic mechanisms. Our study also noted that 10.6% of AIS B patients worsened to AIS A, highlighting the dynamic nature of SCI recovery and the potential for secondary complications such as spinal cord ischemia, progressive myelopathy, or mechanical instability contributing to neurological decline. Mahmoodkhani et al. reported that 84% of AIS D and 77% of AIS C patients showed improvement within 24 months post-surgery. This indicated a higher degree of functional recovery in incomplete injuries. Additionally, Mahmoodkhani et al. reported that 38% of all patients achieved full recovery (AIS E), while no AIS A patient in the present study converted to AIS E within one year.[12]

The overall pattern of AIS grade changes across the study cohort further supports the predictive value of the AIS system in SCI prognosis. Notably, 56.5% of AIS C patients and 54.4% of AIS D patients showed improvement, reinforcing the hypothesis that incomplete injuries (AIS B–D) have a higher likelihood of functional recovery due to preserved motor and sensory pathways. The absence of any improvement among AIS E patients reflects the ceiling effect of the AIS system; patients with normal baseline function (AIS E) are unlikely to demonstrate further improvement. The statistically significant association ($p < 0.001$) between baseline AIS grade and outcome highlights the strength of AIS as a prognostic tool.

Mahmoodkhani et al. noted a 5% mortality rate within 24 months, with 75% of the deceased patients being AIS A at the time of admission. The higher mortality among AIS A patients is consistent with the well-established correlation between injury severity and survival outcomes. Haque et al. did not report mortality rates but documented post-operative complications, including dysphagia (35.48%), donor site infection (9.67%), cerebrospinal fluid (CSF) leak (3.22%), and catheter-related urinary tract infections (9.67%). The present study documented similar rates of complications - a higher frequency of urinary tract infections and wound infections[6,12]

Our study's findings have several important clinical implications. (1) The relatively high rate of improvement among AIS B patients suggests that early surgical intervention and comprehensive rehabilitative care should be prioritized for this subgroup to maximize functional recovery. (2) The lower conversion rate among AIS A patients highlights the need for further research into neuroprotective and neuroregenerative strategies. This includes stem cell therapy, pharmacological interventions and rehabilitative techniques - to enhance recovery potential in complete SCI cases. (3) Our study emphasized the importance of patient stratification based on baseline AIS grades to customize their rehabilitative strategies and set realistic recovery goals.

The single-centre design may limit the generalizability of the findings to other settings with different healthcare infrastructure and patient demographics. The short follow-up period of one year may not capture the full trajectory of long-term recovery in cases of incomplete injury where neurological recovery may continue for several years. AIS scores provided a valuable measure of neurological function. However they do not fully capture functional independence or quality of life - which are critical outcomes for SCI patients.

Our study observed prognostic values of AIS scores in assessing post-operative recovery in SCI patients. Our findings highlighted potential for neurological recovery in incomplete injuries (AIS B–D); as well as the challenges associated with complete injuries (AIS A).

Conclusion

Our study delivered insights into how patients with spinal cord injuries (SCI) recover. Especially in a setting with limited healthcare resources. It shows that 26.0% of patients with complete injuries (AIS A) improved to an incomplete status within one year. This means that even patients with severe injuries have a chance to recover, especially if they receive early surgery and proper rehabilitation. The study also found that 39.4% of patients with AIS B injuries improved to a higher grade, showing that incomplete injuries have a better chance of recovery. However, 10.6% of AIS B patients worsened to AIS A, highlighting the unpredictable nature of SCI recovery. This suggests that complications like poor blood flow to the spinal cord or further damage can affect recovery. This makes early rehabilitation and close monitoring very important. Our study also showed that in India, falls from height (47.0%) are the most common cause of SCI, while in other countries like Iran, road traffic accidents are more common. This means that injury prevention strategies in India should focus more on reducing falls - especially at construction sites and in rural areas.

Patients with incomplete injuries (AIS C and D) showed higher recovery rates (56.5% and 54.4%), confirming that patients with some preserved motor and sensory function are more likely to improve with proper care. Our findings highlighted need to improve trauma care facilities and to increase access to rehabilitation centres.

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An inexpective adult diffuse astrocytoma. Case report and literature review

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Keywords
case report, diffuse astrocytoma, gliomas, computer tomography, survival

ABSTRACT

Background: Diffuse gliomas in adults also include astrocytoma and isocitrate dehydrogenase deficiency, oligodendrogliomas with 1p/19q deletion, and also glioblastoma, wild type (IDH). As indicated in the European guidelines on low-grade I gliomas, early extensive surgical resection may be associated with a good prognosis. Molecular markers show that IDH1 R132H has a prognostic role in GBM. Temozolomide chemoradiation has shown beneficial results in the survival of patients with astrocytoma.

Case description: A 76-year-old woman presented to our emergency room; according to her family and emergency medical team, the patient fell at home and received a direct blow to the head with loss of consciousness. The patient had a known history of hypertension for a few years and was taking antihypertensive medications periodically. Unspecified leukaemia. Respiratory rate: 16/min; respiratory rate: regular. On clinical examination, the patient is conscious, oriented, and has right-sided weakness. Glasgow GCS (points): 11 points. after a profound, stunning. Contact is very difficult due to speech disorders. (Dysarthria). He has a right hemiparesis of 2 points, with a Positive Babinski sign on the right side.

Conclusion: The case highlights the importance of multimodal management, including early surgical intervention, molecular diagnostics, and postoperative care, in improving outcomes for patients with astrocytomas. A review of prognostic factors, such as age, GCS scores, and molecular markers like IDH1 mutations, emphasises the need for individualised treatment approaches. Although advances in chemoradiation, particularly with temozolomide, have improved survival rates, astrocytomas remain associated with high morbidity and mortality. This report underscores the critical role of early diagnosis, maximal safe resection, and tailored therapeutic strategies in optimising patient outcomes and quality of life.

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Introduction

The World Health Organization (WHO) has established the fifth classification as the diagnostic criteria for the nomenclature and grading of diffuse gliomas. Diffuse gliomas in adults also include astrocytoma and isocitrate dehydrogenase deficiency, oligodendrogliomas with 1p/19q deletion, and also glioblastoma, wild type (IDH). [1]. Brain tumors are clinically divided into primary and metastatic brain tumors. Primary tumors are due to cells of the central nervous system, accounting for 1% of cancers in the United States and probably 2% of cancer deaths in the United States, mainly with glioma as the primary tumor. [2]. In intrinsic diffuse pontine gliomas, surgical techniques have improved the safe initial biopsy, with a deeper understanding of the biological evolution of the tumor tissue. This has led to the discovery of a recurrent somatic mutation in function leading to the substitution of lysine 27 by methionine [p.Lys27Met, K27M]. Histone 3 is characterized in more than 85% of intrinsic diffuse gliomas, influencing the first epigenomic role of histones in pathogenesis as a specific diagnostic criterion. [3].

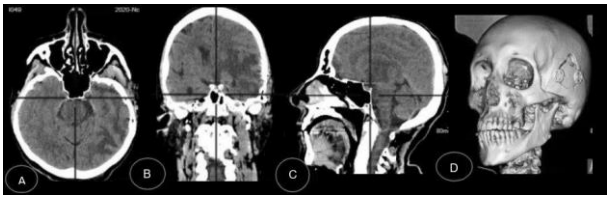

Figure 1. a) Axial negative dynamic CT image of the formation in the left temporal lobe. b) Coronal image of the temporo-parietal-occipital region: significantly increased vasogenic edema on the left; c) Sagittal image, transverse dislocation and edema of the left hemisphere, reversal of pneumocephalus with diffuse atrophic changes in the brain. d) 3D: skull reconstruction after craniectomy in the left temporal region.

Case presentation

A 76-year-old woman presented to our emergency room; according to her family and the emergency medical team, the patient fell at home and received a direct blow to the head. The patient had a known history of hypertension for a few years and was taking antihypertensive medications periodically. Unspecified leukemia. Respiratory rate: 16/min; respiratory rate: regular. On clinical examination, the patient is conscious, oriented, and has right-sided weakness. Glasgow GCS (points): 11 points after profound stunning. Contact is very difficult due to speech disorders. (Dysarthria): He has a right hemiparesis of 2 points, with a positive Babinski sign on the right side. Systolic pressure: 130 mmHg; diastolic pressure: 80 mmHg; pulse: 64/min. Heart rate: not altered. Heart sounds: clear. A CT scan was completed, confirming a space-occupying lesion. The third ventricle. In the parietal-temporo-occipital. The patient was admitted and treated with anticonvulsants and supportive antibiotics. for possible intervention

CT scan of the brain

The midline structures are shifted to the right up to 14 mm (previously up to 7 mm) at the level of the third ventricle. In the parietal-temporo-occipital region on the left, there is a heterogeneous zone, represented mainly by a finger-shaped vasogenic edema zone measuring 68x92x62 mm (previously 61x86x46 mm), against the background of which a group of areas of fluid density of up to 7-10 units of fluid density is formed. Gas and blood inclusions previously identified in the temporal region are not visualized in this study. Periventricular: small areas of leukoaraiosis. Convexital fissures and Sylvian fissures are widened on the right and narrowed on the left; they cannot be traced in the temporal region (they were previously traced). The ventricles of the brain are asymmetrical: the right lateral ventricle is slightly dilated and the left one is compressed. The shunt tank on the right is narrowed. The vendor region is unremarkable. No "fresh" bone traumatic changes were identified.

Procedure

A craniotomy hole is made in the left temporal region, and a 33x38 mm cranioplasty with a bone graft is performed with the presence of metal fixators. Two weeks later, an extended tumor biopsy. See Figure 1-2.

Diagnosis: Tumour (diffuse astrocytoma) of the left temporal lobe.

Postoperative CT control

In the soft tissues of the occipital region, there is a hematoma measuring 4x0.7x5 cm. Condition after craniotomy in the left temporal region. In comparison with a CT scan of the brain, negative dynamics of the formation in the left temporal lobe were noted in the form of an increase in the zone of

vagogenic edema in the temporo-parietal-occipital region. On the left, transverse dislocation and edema of the left hemisphere. Reversal of pneumocephalus. Diffuse atrophic changes in the brain. Shown in Figure 1.

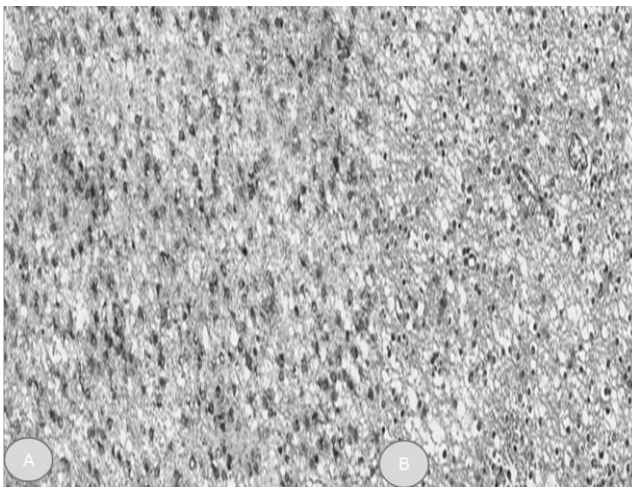

Figure 2. a) Diffuse astrocytoma exhibiting diffuse IDH1 immunoperoxidase staining; original magnification 150x; b) Grade 2 astrocytoma, IDH mutant. Demonstrating a low-cellularity astrocytic neoplasm comprised of inconspicuous, mitotically dormant cells featuring ovoid hematoxylin and eosin-stained nuclei; original magnification 150x.

Follow up, UCI, condition at discharge

The patient's condition is relatively satisfactory; there are no respiratory or hemodynamic disturbances. In neurological status: level of wakefulness—clear consciousness, GCS 15 points. Contact is difficult due to sensorimotor aphasia. A minimal right-sided hemiparesis persists.

Discussion

Our case report is of a patient who was symptomatic for a space-occupying mass after losing consciousness and being brought in via emergency. Imaging and laboratory markers are standard to confirm the diagnosis prior to biopsy. It also shows how this cluster of symptoms presents once the patient has brain dislocation, loss of consciousness, and even hemiparesis, such as a stroke. A retrospective study of 64 patients was conducted to predict the survival of those diagnosed with diffuse astrocytoma as a low-grade glioma. Cox regression models were analyzed, and subsequently a monogram was performed for the prediction of survival by three significant factors. Age ≥ 60 years at hazard ratio (HR) = 5.8 and 95% confidence interval (CI): 2.09-15.91 and Glasgow Coma Scale motor response score < 6 [HR = 75.5, 95% CI: 4.15-1.369.4 and biopsy HR = 0.45 and 95% CI: 0.21-0.92, where mortality will be shown in the monogram at one year from diagnosis. [4]. [9]. [10]. As indicated in the European guidelines on low-grade gliomas, early extensive surgical resection may be associated with a good prognosis. As in the European and American guidelines, they agree that the best therapeutic option is maximum resection, also taking into account observation in already selected patients. [5]. [11]. Molecular markers show that IDH1 R132H has a prognostic role in GBM. Therefore, they tend to have better survival than tumors with PDFGFRA expression with a P=0.066. Therefore, proneural and mesenchymal molecules showed a better benefit in the addition of chemotherapy and radiation therapy. [6]. [12]. Although radiation therapy and chemotherapy have improved survival, mortality remains high in patients with astrocytomas. Remember that each year in the United States, a brain tumor is diagnosed in 51,000 individuals per year, and that more than 75% will die within the first 5 years after diagnosis. Recent studies have shown that, because they are related to suppressive symptoms, the quality of life in patients with astrocytomas decreases. [7]. [8]. [13]. Temozolomide chemoradiation has shown beneficial results in the survival of patients with astrocytoma. IDH is a grade 2 mutation. This chemoradiation can be postponed until the time of progression in younger patients, using extensive or massive resection. Therefore, in high-risk patients, treatment should be earlier. . [14]. [15]. [16].

Conclusion

Our patient had a relapse due to syncope and lack of cerebral oxygenation due to herniation by the space-occupying mass of origin under investigation, which a biopsy determined to be a diffuse astrocytoma. It is worth highlighting the dysarthria caused by this, probably due to further impact. The altered blood pressure, the onset of cerebral infarction, would influence the hemiparesis, affecting his right limbs. The patient after surgery During treatment in the intensive care unit (ICU), positive results were observed, from the recovery of alertness to a clear and partial regression of the

aphasia and hemiparesis. The patient was discharged without complications. He will continue conservative treatment and rehabilitation and will remain under observation for a complete recovery after referral to the neurology clinic.

Author Contributions

Conceptualization. BC.JJA, JFH. , D.E.S.; methodology, D.E.S., .; software, D.E.S., and .; validation, .. I.B., and B.C; formal analysis, G.C., and DRC.; investigation, D.E.S. resources, EC and E.S. Data curation, EC. G.S.; writing—original draft preparation, D.E.S., writing—review and editing, IB, D.E.S., visualization, JJA. JFH. supervision, I.B. MP. ES.

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Cystic meningiomas, literature review and case reports. Nauta classification modification

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ABSTRACT
Meningiomas of central nervous system are common lesion, but among them, the cystic meningiomas are very rare tumours. We report two cases of cystic meningioma successfully treated, and we propose the modification of the widely used Nauta classification of cystic meningiomas. The clinical results depend on meticulous preoperative diagnosis and surgical planning, enhancing the utility of modification of Nauta classification by adding bone invasion.

Keywords
cystic,
meningioma,
dura,
brain

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INTRODUCTION

Cystic meningioma is very rare with an incidence described is 9.6% [1]. According to other studies on cystic meningioma, the reported incidence is 1.6-11.7% [2,3,4]. It is more frequent on children, representing 12%-24% of pediatric meningiomas while it's only of 2%-4% on adult, twice more on female probably due to hormonal factors [5].

Nauta in 1979 classified these tumors according to the location of the cyst relative to the tumor into 4 types. Type 1 cyst is contained wholly within the tumor and, being located centrally, or nearly so, is surrounded by macroscopic tumor throughout, type 2 he cyst is at the periphery of, but still wholly within the margins of the tumor, there being a microscopically visible attenuated rim of tumor cells along the peripheral margins of the cyst, type 3 the cyst again appears to be peripheral, and indeed actually lies within the adjacent brain rather than within the tumor itself, type 4 the cyst appears at the interface between the tumor and brain as a loculation of CSF in the subarachnoid space, and does not appear within either the tumor or brain itself [6]. There is no reference about adjacent bone invasion of the tumor.

There are several hypotheses for how cysts appears in meningiomas. First hypothesis is degenerative phenomenon, the development of the cavity is due to intracellular regressive processes

as vacuolar, myxomatous, mucoid and fatty degeneration [7,18]. Another hypothesis is ischemic: arteriolar hyalinization in the necrotic tissue of tumors that cause the intratumoral cavity [9. The third hypothesis is previous intratumoral hemorrhage in angioblastic meningioma [8,10,11,12,13].

CASE DESCRIPTION

Case 1.

A 76-year-old gentleman came to the emergency room for headaches and left hemiparesis, left homonymous hemianopsia, and mental confusion. An urgent CT scan revealed a large lesion in the right occipital region, and an MRI of the brain showed an extraxial cystic right parieto-occipital neoplasm with extensive parenchymal edema**.** Due to an extraxial lesion in the right parieto-occipital region, the patient underwent craniotomy and Simpson 2 excision.

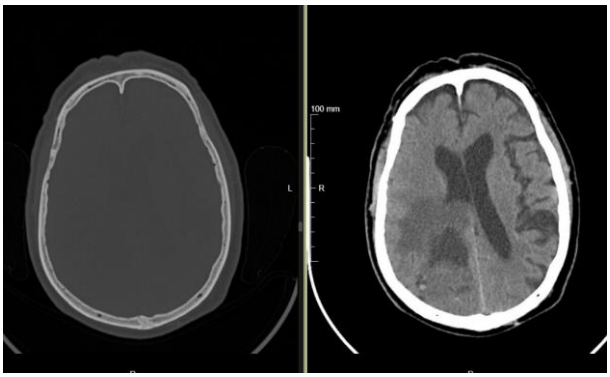

Figure 1. Emergency CT Scan revealing cystic parieto-occipital mass.

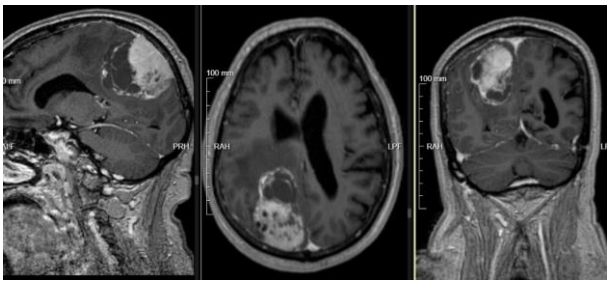

Figure 2. Pre-op MRI cystic meningioma classified by Nauta as type III.

Histologic examination revealed atypical meningioma, grade 2 (of 3) according to the WHO Classification. He followed radiotherapy with total dose: 59.4 Gy 33 fractions/6.5 weeks. The patient remains under oncological and neurosurgical follow-up with a good recovery, postoperative MRI maintains dural thickening adjacent to craniotomy.

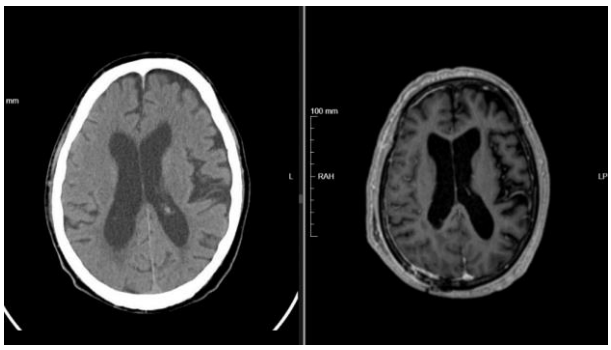

Figure 3. CT scan and MRI 1 year after surgical treatment plus radiotherapy.

Case 2.

A 45-year-old male presented with a clinical onset numbness the face, which progressed to a generalized tonic-clonic seizure. He came to emergency services for these complaints. A CT scan on admission revealed a large right parietal lesion, and an MRI of the brain indicated an extraxial neoplasm in the right occipital region with no edema. CT scan and MRI also detected bone invasion.

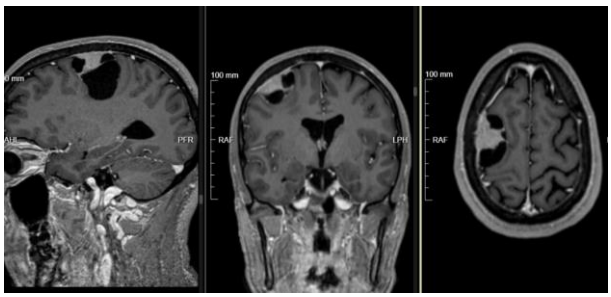

Figure 4. Pre-operative MRI showing cystic meningioma classified by Nauta as type 4.

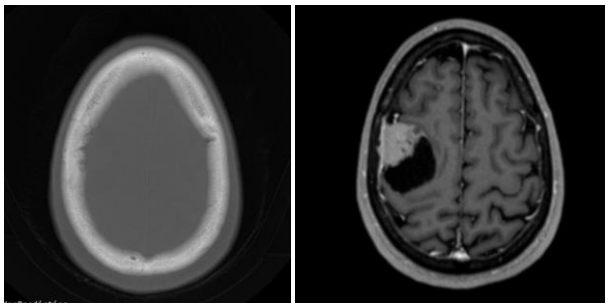

Figure 5. Bone invasion seen on CT scan and MRI slices.

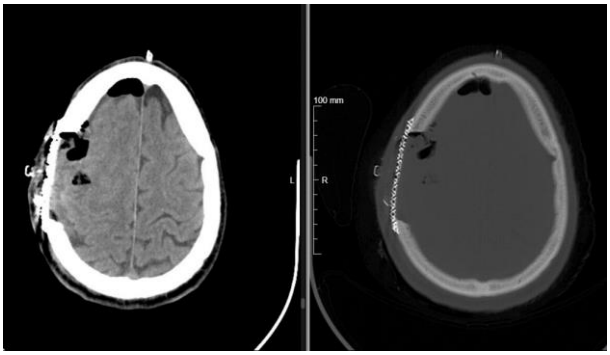

Figure 6. Postoperative CT scan.

Given these findings, the patient underwent the following procedure: right parietal craniotomy guided by navigation, total microscopic resection of the posterior frontal tumor with dural involvement and bone invasion, removal of the dural flap and craniotomy, dural repartion using synthetic dura, and cranioplasty with a titanium plate. The procedure was uneventful. A postoperative CT scan showed complete resection of the lesion.

Discussion

The preoperative diagnosis is a challenging task, MRI with contrast achieves a preoperative diagnostic accuracy of 80%, while CT scan approximately 50% [4,14,15]. The presence of an associated cyst is an uncommon imaging feature that may make it difficult to distinguish the tumor from a primary intra-axial glial neoplasm and the presence of peritumoral edema can also be a misleading finding [16]. The presence of an intratumoral cyst can often create diagnostic challenges, resembling other neoplastic lesions [17,18,19]. The most frequent histological subtype found was the meningothelial subtype [2,20,21]. Atypical meningiomas have the tendency to form a cyst, as opposed to other subtypes of meningioma [21,22].MRI with diffusion-weighted imaging may be efficient in diagnosing the cystic meningioma [4,23], while the value of PET/CT in cystic meningioma requires further investigation and according to some studies it seems that diffusion-weighted imaging may be superior at distinguishing between the various types of meningioma [4,24]. The presence of cystic components within and/or around the mass usually suggests a diagnosis other than meningioma. These tumors can be easily misdiagnosed as metastases, gliomas and hemangioblastomas macroscopically in CT or MRI [25]. Although there are less dilemmas with MRI, cystic meningioma can easily be misdiagnosed, in about 20% of cases, particularly as glial tumor, metastasis, neuroblastoma or haemangioblastoma [26]

Whether total cystic wall excision is necessary remains unclear. Author´s opinions are divided, some recommend that excision of the cyst wall is necessary [3,27], while other authors do not [11,29]. There are opinions that in the case of type II cystic meningiomas, every effort should be made to remove not only the mural nodule but also the cystic wall [2], other authors reported a case of tumor recurrence in type II cystic meningioma in which the cystic wall was not completely removed [8 [29,30,31], there are also reports that cyst wall components contain cells and complete removal of cystic components is essential [32].

We found in the literature only a sporadic description of cystic meningiomas with bony invasion, and curiously it was found in type II and III Nauta (with peripheral tumor adjacent to the bone [33,34,35], like it also was found in our second case. We propose to introduce a modification to Nauta classification, adding bone invasion, like type I,II,III,IV A-without bone invasion and B-with bone invasion.

Conclusion

Accurate diagnosis cystic meningioma is made through CT and MRI, but MRI remains the most effective on preoperative diagnostic, while the anatomopathological study is the only tool to confirm the diagnosis, allowing the correct management which should privilege a total resection as much as possible, total resection of the cyst and its prevents and diminish the risk of postoperative tumor recurrence.

The clinical results depend on meticulous preoperative diagnosis and surgical planning, enhancing the utility of modification of Nauta classification by adding bone invasion.

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Hypochloraemia in patients with severe traumatic brain injury at a tertiary care hospital in India. A possible threat for mortality

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ABSTRACT

Objective: To evaluate the correlation between Individuals with severe traumatic brain injury and death from electrolyte imbalance

Methodology: In a prospective cohort study, patient records suffering from a severe brain injury caused by trauma Glasgow Coma Scale score less than 8, and electrolyte abnormalities were reviewed. To determine a correlation between the patients who passed away and the ones who lived, electrolyte levels were examined. For categorical variables, bivariate analysis was carried out using the $\chi 2$ test, which has a 95% statistical accuracy. To ascertain the correlation between electrolyte fluctuations and mortality, the $\chi 2$ test was employed in conjunction with multiple comparisons. The linkage between fatalities and electrolyte shifts was analysed using logistic regression. 95 per cent of statistical tests were reliable.

Results: In 24.5 % of patients who passed away, Elevated mortality risk was significantly correlated with hypochloremia (P 0.03). It also represents the substantial link between the Examination of Acute Physiology and Chronic Health APACHE II ($P < 0.01$) and age ($P < 0.01$).

Conclusion: Hypochloremia may be a significant prognostic factor for determining death in individuals suffering from severe TBI risk and optimising treatment.

Keywords
hypochloraemia,
severe traumatic brain injury

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INTRODUCTION

All across the world, traumatic brain injury (TBI) is a serious socioeconomic as well as public health issue. Globally, TBI is the leading death cause for young adults. [1-3]

There are 250 traumatic brain injury cases for every 100,000 people worldwide each year.4 In developed nations, falls, auto accidents, violent crimes, and motorcycle accidents are the leading causes of TBI. In Europe, the male-to-female ratio for TBI is 9:1, while in the United States it is 3:1. Traumatic brain injury happens every 7 seconds, and in young people, it happens every 5 minutes.4 TBI accounts for 45% of deaths in patients with polytrauma.1 Additionally, the majority of TBI survivors are thought to have a permanent sequel. In Colombia, there are 200 cases of head trauma for every 100,000 people annually, translating to an 18% TBI death rate. [5].

TBI also affects the blood-brain barrier, primarily through modifications to pressure gradients associated with Starling forces. Significant changes in the levels of other solutes and electrolytes also impact osmolarity. Patients with TBI may experience magnesium deficiency, hypophosphatemia, hypernatremia, hypokalemia, or hypocalcemia soon after the first injury and the recovery process. These changes are likely connected with how the lesion is developing or how medicine is being given. [7, 8]

In clinical studies, the association with in electrolyte levels and TBI fatality is not well established. The majority of trauma studies focus on hyponatremia and its clinical changes, but they don't account for electrolytes like magnesium, potassium, chlorine, or other elements.9, 10 There is only one study in Colombia that looks at patients who have both electrolyte imbalances and renal failure; no other research addresses these issues about TBI.11 Colombia's Ministry of Health and Social Protection wants to create accident prevention programs to lower death rates and secondary lesions among TBI patients. 12–14 This study's primary goal was to determine the association between altered electrolytes and higher mortality in TBI patients by using patient's cohort from Sawai Man Singh Medical College and Hospital, Jaipur.

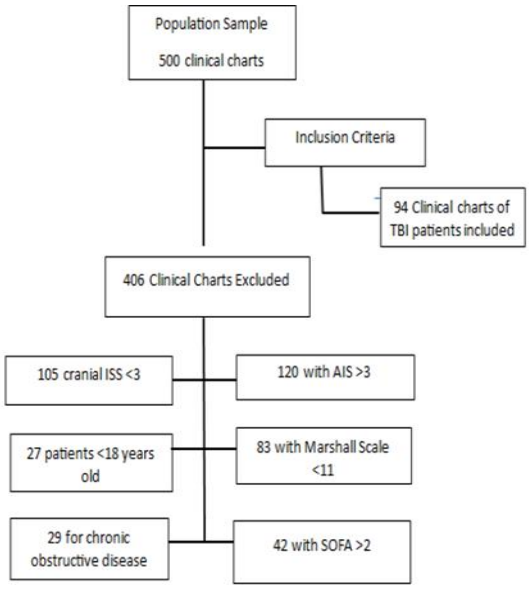

Figure 1: Flowchart of the total patients and record of exclusion. Traumatic brain injury (TBI), Abbreviated Injury Scale (AIS), Cranial Injury Severity Scale (cranial ISS), sequential organ failure score assessment (SOFA).

MATERIALS AND METHODOLOGY

Study Design

The clinical records of patients from Sawai Man Singh Medical College and Hospital, Jaipur who suffered from severe traumatic brain injury have been examined. Patients were divided into two groups based on the presence or absence of electrolyte abnormalities to examine the possible correlation between these abnormalities and mortality. 500 admitted TBI patients' clinical records made up the study's population sample between the November 2023 to April 2024.

The study's inclusion criteria were individuals with TBI who were over 18 and had a Marshall tomography classification of III or above. A total score of 3 was obtained from the Glasgow Coma Scale, 3 from the Trauma Severity Scale of Cranial Trauma, 3 from the Abbreviated Injury Scale data, and 8 from the Trauma Severity Scale of Body Trauma. Individuals undergoing therapy with loop diuretics, mannitol, thiazide, digitalis, or glucocorticoids were not included, nor were patients with chronic illnesses.

Patients with sepsis, septic shock, and multiorgan dysfunction with a Sequential Organ Failure Assessment greater than 2 that was unrelated to a declining Glasgow Coma Scale score were also excluded. Moreover, 406 patients were discharged from the 500 clinical charts that were analyzed; 120 Patients were not treated due to having an Abbreviated Injury Scale score of more than three; A cranial Injury Severity Scale score of less than three excluded 105 patients, and a Marshall classification of less than three excluded 83 patients. With a sequential organ failure score assessment of more than two, 42 patients

suffered from severe infections. 29 patients with chronic illnesses and 27 patients under the age of 18 were assessed.

Consequently, 94 patients fulfilled the study's inclusion requirements (Figure 1). Information was taken from patient records during the initial ten days of hospital stay. Daily electrolyte readings and sociodemographic data were documented.

Analysis of Data

Levels of blood for K, Ca, Na, Mg, and Cl were monitored for 10 days after the admitting date. The Kolmogorov-Smirnov statistical analysis was used to verify the numerical variables, and the various

variables were grouped based on proportions for particular categorical variables. Nonparametric measures were employed to analyze variables that did not exhibit normality patterns. Moreover, the χ2 evaluation was carried out with 95 percent consistency for categorical variables. In the final statistical analysis, the Yates correction was applied when the frequency was less than five.

The correlation between electrolyte disturbances and mortality was ascertained through multiple comparisons by the χ2 test. Additionally, a regressional analysis was done to find out how electrolyte imbalances and mortality are related. A 95% dependability level was maintained throughout the execution of each statistical test.

Results

The medical history of 500 individuals diagnosed with severe head trauma who were admitted to Sawai Man Singh Medical College and Hospital, Jaipur between November 2023 to April 2024 comprised the population sample for this study. Out of 500 clinical charts, 406 were deemed ineligible for inclusion in the study, while 94 clinical charts satisfied the requirements and were added to the database. The clinical charts were based on whether or not there were electrolyte changes.

Table no 1. Sociodemographic and Clinical Characteristics of Patient Population (N= 94)

Characteristic	Value
Sex	
Male	78(92.6%)
Female	16(7.4%)
Age, years, average (SD)	42.1(18.6%)
Acute complications	
Renal Disease	6(4.9%)
Subarachnoid Disease	6(4.9%)
Diabetes insipidus	9(5.9%)
No complications	73(84.3%)
Marshall classification	
III	5(3.8%)
IV	24(25.1%)
V	57(64.9%)
VI	8(6.2%)
Hospital length of stay, days, average (SD) 12(12.6%)	
Patients deceased	34(24.5%)
Patients survived	60(75.5%)

The patients were 42 years old on average, with an 11:1 male-to-female ratio consisting of 78 men (92.6%) and 16 women (7.4%). Side effects, such as acute kidney disease (4.9%), subarachnoid hemorrhage (4.9%), and diabetes insipidus (5.9%), were observed in patients (Table 1).

Table no 2. Electrolyte Alteration in Patients with Severe Traumatic Brain Injury (N=94)

Electrolyte	Value
Potassium	
Hypokalemia, mean 3.1 mEq/L	41 (25.1%)
High plasma osmolarity	(42.2%)
Hyperkalemia, mean 6.0 mEq/L	9 (27.9%)
Normal values	44(46.9%)
Calcium	
Hypocalcemia, mean 6.7 mEq/L and 0.8 mmol/L 19 (16.1%)	
Hypercalcemia, average 13.9 mEq/L and 1.5 mmol/L 11(3.5%)	
Normal values	64 (79.5%)
Magnesium	
Hypomagnesemia, mean 1.3 mEq/L	38 (38.7%)
Hypermagnesemia, mean 3.2 mEq/L	11 (3.5%)
Normal values	45 (57.8%)
Sodium	
Hyponatremia, mean 128 mEq/L	42 (55.6%)
Hypernatremia, average 150 mEq/L	14 (12.7%)
Normal values	44 (31.7%)
Chlorine	
Hypochloremia, mean 94 mEq/L	12 (46.7%)
Hyperchloremia, mean 117.8 mEq/L	9 (3.6%)
Normal values	73 (56.9%)

According to Marshall Tomography classification (any surgically evacuated injuries), 25.1% of lesions were diffuse type IV lesions (shift greater than 6 mm; no high- density or mixed-density injuries greater than 26 mL). Scores 16 and 17 on the Acute Physiology and Chronic Health Evaluation (APACHE II) classification indicated that mortality would be between 156% and 22%. Changes in hydroelectrolyte levels were noted in 78 clinical charts (92.8%); the most common changes were related to sodium and potassium, with hyponatremia accounting for 55.6% and hypokalemia at 46.7 % (Table 2).

The least affected electrolytes were magnesium and calcium, with increases in each occurring in just 3.5 % of the total charts.

In 14% of the charts analyzed, hypochloremia was detected. Just 82.5% of the patients had electrolyte correction of any kind. There was a statistically significant correlation discovered among APACHE II score (P < 0.01), older age, and mortality (P < 0.01). Furthermore, hypochloremia was observed in 25.1% of individuals, which was substantially important (95% confidence interval: 1.0-15.5; P ¼ 0.03).

Despite their prevalence, hyperkalemia, hypomagnesemia, and hypermagnesemia did not significantly correlate with death. Hypochloremia and hyperkalemia, two categorical variables, displayed an odds ratio for a higher chance of dying. Furthermore, 42.2% of patients had high plasma osmolarity (292.14 SD 16.9); this result was statistically significant (P ¼ 0.03). The majority of these patients were deceased.]

Discussion

The study's findings revealed a 24.5% TBI death rate, which is significantly greater than the 10%–20% mentioned in previous research on patients with comparable traits. [17, 18] The association between hypochloremia and an elevated risk of fatality in individuals who passed away, with a statistically accurate difference (P ¼ 0. 03), was the study's most significant finding. It is possible to argue that hypochloremia is a significant threat or a mortality predictor in TBI individuals. It's also crucial to remember that hypochloremia may go undiagnosed, be an isolated finding, or be linked to hyponatremia, which is another condition that has a poor prognostic value.[19] However, hyponatremia was not a statistically important factor for death because it was found in both surviving and deceased patients with high frequency.

The investigation's findings are novel since prior studies have focused on changes in Na and K that are associated with mortality rather than Cl. [9, 20–22] Moreover, some research has demonstrated that variations in chlorine concentrations are linked to adjustments made to the microenvironment of neuronal cells because chlorine is transported out of the cell, acting in opposition to its electrical balance and favor of its chemical balance.[23]Because of this, in most mature neurons, chlorine can act as a signaling agent and a depolarizing current.[24] Potassiumchlorine,KCC1-4, SLC12A4 through SLC12A7, and sodium- dependent cotransporters of chlorine- bicarbonate, like SLC4A8 and the sodium-driven chloride/bicarbonate exchanger NDCBE, are the most significant cotransporters of chlorine. Mature g-aminobutyric acidergic postsynaptic neurons are hyperpolarized by low intracellular chlorine concentrations.[24]

On the other hand, certain immature neurons depend on g-aminobutyric acid for the expression of chlorine currents, which in turn produce postsynaptic potentials that depolarize and are essential for maintaining the stability of recently formed synapses. The partial depolarization of g-aminobutyric acid-dependent neurons, which is made worse by local ischemia that affects the sodium-potassium adenosine triphosphatase pump and reduces the hyperpolarizing effect of chlorine current, may be linked to hypochloremia and mortality.[25, 26]

Patients with TBI may experience hypochloremia due to a reduction in renal perfusion and tubular renal Claudin-2 expression, which obstructs the reabsorption of chlorine. [12, 14, 27]

The analysis of mortality showed a statistically significant correlation with age as one of the variables; the greater the patient's age, the higher their risk. The other significant variable was the APACHE II score (P less than 0.01).

This outcome is consistent with findings from previous research. Age is one risk factor included in the validated APACHE II scale for predicting death in critically ill patients.[28] The standardization of patient characteristics was made possible by the inclusion and exclusion criteria. With an approximate age of 42 and a total hospital stay of 6 months, men made up the majority of the study's patients. These attributes align with a few of the reviewed studies from Latin America. This study's male-to-female ratio of 5:1 was comparable to that reported by Guzmán et al.[5] Clinical studies conducted in the US and Europe produced findings comparable to these (e.g., Brazinova et al.[17]) There could be several reasons for the disparity in frequency of patients by gender, such as lifestyle, environment, cultural background, and occupational risks.

According to the results, water-electrolyte shifts were discovered in 92.8 percent of patients; as in previous clinical trials, potassium (46.9%) and sodium (55.6%) were the most affected electrolytes. Kovesdy et al.[22] discovered abnormal blood and a

correlation that is statistically important between levels of potassium and sodium and mortality in patients suffering from electrolyte imbalances, liver disease, and cardiovascular disease. However, this study did not find a correlation, and the patients who survived had more of these electrolyte abnormalities. The fact that there were methodological variations, the follow-up period was extended, and comorbidities were not utilized as an exclusion criterion must be emphasised.[22, 29]

Despite the patient population or setting, all research has concluded that there is a clear correlation between hyponatremia and a higher risk of death.[30, 31] The association between hyperosmolarity and hyperglycemia was statistically significant with a greater alteration in patients who passed away. Because hyperglycemia causes hyperosmolarity, it also raises insulin levels and causes hypokalemia, and is linked to increased levels of cortisol, glucagon, and catecholamines.[19]

Limitations

Despite the initial analysis of 500 clinical charts, the research was constrained by the ultimate sample size. Another drawback was the follow-up period, although most studies on this subject have a follow-up period of six months. The evaluation of charts from just one hospital represents the last restriction. This research's strengths include its statistical evaluation and the dearth of comparable research containing data on electrolytes more than potassium and sodium.

Conclusion

In TBI patients, hypochloremia may be a prognostic factor or a threatening factor for increased mortality. Osmolarity and electrolytes should be assessed and managed because there is a statistically accurate link between changes in these parameters and fatalities. Patients with TBI need to be treated right away if their hydro-electrolyte levels change. Changes in magnesium or chlorine electrolyte levels should also be taken into account as a potential threat to death in TBI patients.

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Complete embolization of large dural AV fistula having multiple feeders using glue embolizing agent (Squid 12)

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ABSTRACT

Dural arteriovenous fistulas are rare intracranial vascular malformations with a propensity for haemorrhage. The Cognard classification system is the most widespread classification system, wherein type IIB through V must be promptly treated to avoid the risk of haemorrhage. The case presented herein reports a 34-year-old male presenting with vague nonspecific headache found to have a Cognard type I dural arteriovenous fistula with multiple arterial feeders.

Keywords
embolization,
dural AV fistula,
glue embolizing agent,
squid 12

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INTRODUCTION

Cranial dural arteriovenous fistulas (dAVFs) are defined as an abnormal connection between dural feeding arteries and a dural sinus and/or leptomeningeal vein. The current hypothesis conjectures that the pathogenesis of dVAF is based on their association with venous outflow obstruction, i.e. the resultant increase in venous pressure causes enlargement of the physiologic shunts between the dural arteries and sinuses that promote angioneogenesis through regional venous ischemia. They manifest with myriad signs and symptoms that differ based on their anatomical location and may be diagnosed incidentally.[1] These unusual lesions are estimated to account for 15% of all intracranial vascular abnormalities.[1] Borden et al [2] and Cognard et al [3] proposed the two most commonly used classification systems for dAVF. These systems function by stratifying dAVFs depending on the presence or absence of cortical venous reflux and venous drainage patterns. Clinically stable patients can be managed through observation with repeated enhanced cross-sectional imaging; however, prompt endovascular embolization or surgical intervention should be considered in unstable patients or those with debilitating symptoms, as successful treatment can significantly ameliorate symptoms and improve the quality of life. We report the case of a patient who initially ignored right-sided occipital headache and was diagnosed with dAVFs involving the transverse sinus. The patient was successfully managed with a single uneventful session of endovascular embolization.

Case

A 34-year-old man without any past medical history of chronic illness, such as hypertension, diabetes mellitus, or dyslipidemia, came to us in out patient department with complaints of persistent headache only temporarily relieved by medication. NCCT head was done which was unremarkable, later patient underwent CT angio which revealed a large right occipital entangled mass suggestive of right occipital dural fistula unruptured which was later confirmed on DSA. It was cognard type 1. patient was taken up for embolization as the patient had persistent headache only temporarily relieved by medication. Patient DSA and embolization was planned in the same setting. Access was taken through right femoral artery, 9fr femoral sheath introduced in femoral artery using seldinger technique. DSA showed Dural fistula of right transverse sinus with feeders from right occipital artery, right MMA, right SCA, right PICA, left occipital, left post auricular, and left MMA artery with large venous sac. The multidisciplinary treatment plan prescribed endovascular treatment using the transarterial, which was performed under general aneasthesia, to occlude the dAVF.

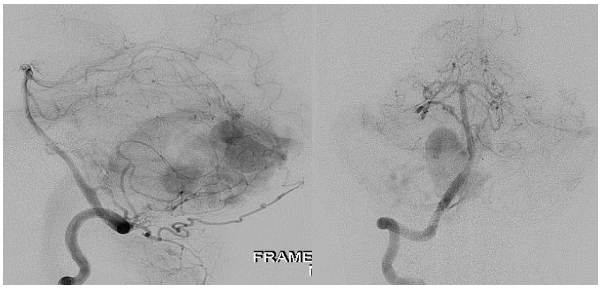

Figure 1, Figure 2. Shows dural arteriovenous fistula in AP and LAT view if injection vertebral artery respectively.

The procedure was initiated by administering an intravenous bolus dose of unfractionated heparin to achieve an activated clotting time of 1.5 to 2 times the baseline value, depending on the patient's weight (as per the hospital's protocol), after positioning the guiding catheter within the right proximal external carotid artery. We decided to embolize the right TS-SS junction dAVF. we passed the right petrous branch of the middle meningeal artery and right occipital artery , followed by injection of Squid 12 under continuous fluoroscopic guidance, to achieve complete occlusion of the fistula which was confirmed post embolization DSA.

No complications were observed during or immediately after the procedure, and the patient's symptoms were resolved. The patient was followed in routine and the patient also confirmed that the symptoms did not reappear during the last follow-up visit and that he was satisfied with the management plan.

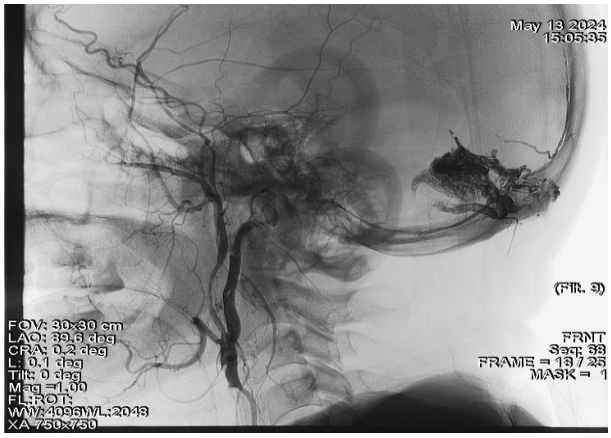

Figure 3. Shows complete embolization of dural avf with residual glue cast/

Discussion

A dAVF is an abnormal arteriovenous communication that links arterial and venous channels within the leaflets of the dura mater. [4] Although the etiology of multiple dAVFs is unknown, the occurrence of cyclical sinus thrombotic occlusion and recanalization is one of the theories postulated to explain their pathogenesis. These fistulas may emerge after angioneogenesis as micro-shunts within the dura; several risk factors are known to increase the incidence of such fistulas, including venous sinus thrombosis, head trauma, transcranial surgery, transsinus procedures, hyper-coagulable states, and oral contraceptive use.[5,6]The etiology of pain in dAVF is attributed to the direct communication between the dural arteries and venous sinuses or meningeal venous lacunae, with antegrade venous drainage that increases flow through the dural sinuses or venous channels; dAVFs commonly produce benign symptoms such as headaches, otalgia due to pulsatile tinnitus, and painful ophthalmoplegia due to accumulated venous drainage toward the cavernous sinus that can sometimes result in painful proptosis [1].

A multidisciplinary approach with several treatment sessions should be considered as part of the treatment plan if a case of complex cranial dAVF cannot be managed with a single session of treatment. Multiple arterial feeders, reflux into multiple cortical veins, sinus entrapment and occlusion, venous aneurysms, segmental stenosis, median or deep location, and associations with the deep venous system are specific angio-architectural considerations that can increase the complexity of the dAVF and its associated management. [7]

Natarajan et al. [8] described a treatment algorithm for complex dAVFs in which endovascular treatment was recommended as the first option. Transarterial ONYX embolization should be the first option for dAVFs located at any site, except cavernous DAVFs. Moreover, ONYX has unique physical properties, which facilitates prolonged injections that can be better controlled, along with a more predictable penetration and higher cure rate compared to other agents.[9,10,11]. Transvenous embolization (TVE) is based on thrombosis of the venous side of the fistula, which involves a part of the dural sinus. This procedure would be well tolerated if the pathological dural sinus is arterialized and does not serve as a site of drainage in normal circulation. Large venous sinuses can often be blocked without complications. It can benefit cavernous or transverse sigmoid sinus fistulas, or cranial dAVFs with multiple arterial feeders with a small or tortuous course, for which TAE would be difficult. Overall, TVE has a success rate of 71–87.5%.[8]. Furthermore, TVE should be considered the first-line treatment approach for condylar dAVFs, because it is safe and effective.[12] Catheterization of the cervical venous plexus is classified as a difficult procedure, owing to the tortuous venous route. The Onyx embolic material is recommended for TAE of dAVFs because it is superior to glue or coils, with respect to safety and facilitates a high rate of complete obliteration. Moreover, it is not associated with neurological morbidity or mortality.[13]

Conclusion

The diagnosis and treatment of multiple cranial dAVFs remain challenging, despite the rapid advancements in surgical technology and techniques. A multidisciplinary strategy should be implemented, after a thorough clinical evaluation, aided by innovative pre- and intra-operative imaging techniques. This will increase the amenability of endovascular management for treating these lesions and allow clinicians and patients to weigh treatment risks against the expected clinical course. In our case, a single endovascular treatment session accomplished favorable long-term outcomes for dAVFs.

Abbreviations

dAVF, dural arteriovenous fistula;
PCC, posterior condylar confluence
TAE, transarterial embolization
TS-SS, transverse sinus-sigmoid sinus
TVE, transvenous embolization
MMA middle meningeal artery
PICA posterior inferior cerebellar artery
SCA superior cerebellar arter

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Surgical outcome of endonasal transsphenoidal approach for pituitary macroadenoma involving cavernous sinus

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ABSTRACT

Background: Pituitary macroadenomas with cavernous sinus (CS) invasion pose significant surgical challenges due to anatomical complexity and high risk of complications. The endonasal transsphenoidal approach (ETSA) is a minimally invasive technique that offers direct access to the sellar and parasellar regions.

Objective: This study evaluates the surgical outcomes of ETSA for pituitary macroadenomas with cavernous sinus invasion based on a cohort of 36 patients.

Methods: A retrospective review of 36 patients who underwent ETSA for pituitary macroadenomas with radiological evidence of CS invasion (Knosp grades 2–4) was conducted. Surgical outcomes were assessed in terms of gross total resection (GTR) rates, hormonal remission, and postoperative complications.

Results: GTR was achieved in 61% of patients, with higher rates in Knosp grade 2 tumours (80%). Hormonal remission occurred in 67% of patients with functioning adenomas. Complications included cerebrospinal fluid (CSF) leaks (6%), transient cranial nerve deficits (8%), and diabetes insipidus (5%). No mortality was reported. Table: Clinical Characteristics of Patients Undergoing ETSA for Pituitary Macroadenomas.

Conclusion: The ETSA is a safe and effective approach for managing pituitary macroadenomas involving the CS, particularly for lower-grade tumours. Advanced intraoperative tools and multidisciplinary care improve outcomes.

Keywords
endonasal transsphenoidal, pituitary macroadenoma, cavernous sinus

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INTRODUCTION

Pituitary macroadenomas account for approximately 10–15% of intracranial tumors, and 6–10% extend into the cavernous sinus (CS). Tumors invading the CS pose significant challenges, including difficulty achieving gross total resection (GTR) and an increased risk of complications such as cranial nerve deficits and cerebrospinal fluid (CSF) leaks [1-3].

The endonasal transsphenoidal approach (ETSA) has become the preferred surgical technique for pituitary tumors due to its minimally invasive nature, allowing direct access to the sellar and parasellar

regions. However, the role of ETSA in cases of CS invasion remains debated [4,5].

This study evaluates the surgical outcomes of ETSA for pituitary macroadenomas involving the CS, with a focus on GTR rates, hormonal remission, and complication profiles in a cohort of 36 patients.

METHODS

Study Design

A retrospective cohort study conducted from 2015 to 2023 at private setup where endoscopic set up is there.

Patient Selection

Inclusion criteria:

- Patients with pituitary macroadenomas (>10 mm in size).
- MRI evidence of CS invasion (Knosp grades 2–4).

Exclusion criteria:

- Microadenomas (<10 mm).
- Recurrent tumors.
- Cases treated with a transcranial approach.

Surgical Technique

All surgeries were performed using a binostril endoscopic ETSA with intraoperative neuronavigation. Cavernous sinus exploration was attempted only in tumors with radiological evidence of accessible tumor portions.

Outcomes

1. Primary Outcome: Gross total resection (GTR) rate, as confirmed by postoperative MRI.
2. Secondary Outcomes: Hormonal remission in functioning adenomas, postoperative complications (e.g., CSF leaks, cranial nerve deficits), and recurrence rates.

RESULTS

Demographics and Tumor Characteristics (Table 1):

- Sample size: 36 patients (20 male, 16 female).
- Mean age: 47.3 ± 12.1 years.
- Tumor type: Functioning adenomas (56%), non-functioning (44%).
- Knosp grades: Grade 2 (30%), Grade 3 (45%), Grade 4 (25%).

Surgical Outcomes:

- GTR Rates (Figure 1): Overall: 61%; Knosp grade 2: 80%; grade 3: 65%; grade 4: 30%.
- Hormonal Remission: Achieved in 67% of functioning adenomas, highest in prolactinomas.

Complications:

- CSF leaks: 6% (resolved with lumbar drainage).
- Transient cranial nerve deficits: 8%.
- Diabetes insipidus: 5%.
- No mortality.

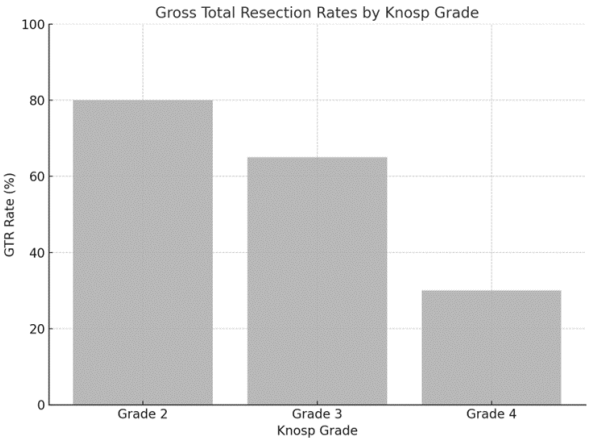

Figure 1. Gross Total Resection Rates by Knosp Grade

Table 1. Patient characteristics with tumor types, grading, resection rate and complications.

Characteristic	Value
Total Patients	36
Gender Distribution	Male: 20 (56%), Female: 16 (44%)
Mean Age (± SD)	47.3 ± 12.1 years
Tumor Type	Functioning: 20 (56%) Non-functioning: 16 (44%)
Knosp Grade Distribution	Grade 2: 11 (30%) Grade 3: 16 (45%) Grade 4: 9 (25%)
Gross Total Resection Rate	Overall: 61% Knosp Grade 2: 80% Knosp Grade 3: 65% Knosp Grade 4: 30%
Hormonal Remission Rate	67% (in functioning adenomas)
Complications	CSF Leaks: 6% Transient Cranial Nerve Deficits: 8% Diabetes Insipidus: 5%
Mortality	0%

DISCUSSION

This study demonstrates that ETSA is a viable surgical option for pituitary macroadenomas with CS invasion, particularly for lower-grade tumors

(Knosp grades 2–3). The GTR rate of 61% aligns with prior studies, while Knosp grade remains a significant predictor of resection success [6-8] .

The hormonal remission rate of 67% in functioning adenomas is consistent with expectations, with the highest success in prolactinomas [9-12] . Complication rates, including CSF leaks (6%) and cranial nerve deficits (8%), were comparable to those in larger series, emphasizing the importance of surgical expertise and intraoperative tools [13-18] .

Advanced techniques such as neuronavigation, Doppler ultrasound, and intraoperative imaging have contributed to improved outcomes and reduced morbidity [19-21] . Despite these advances, tumors with Knosp grade 4 invasion remain difficult to manage due to their extensive involvement of the CS [22-24] .

Future studies should explore adjunct therapies, such as stereotactic radiosurgery, for residual or recurrent tumors [25,26] .

Conclusion

The ETSA provides a safe and effective approach for managing pituitary macroadenomas involving the CS, particularly for Knosp grade 2–3 tumors. Multidisciplinary care and advanced intraoperative techniques are essential to optimizing outcomes.

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Subperiosteal drain versus subdural drain in chronic and subacute subdural hematoma burr-hole evacuation. A comparative study (local experience)

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Keywords
chronic subdural hematoma, subdural drain, subperiosteal drain, burr-hole evacuation

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Abstract

Background: Chronic subdural hematoma (cSDH) is one of the most prevalent neurosurgical conditions, with burr-hole drainage being the standard surgical procedure. While subdural drain (SDD) insertion reduces recurrence rates, subperiosteal drain (SPD) placement has shown comparable recurrence rates with fewer complications.

Objective: To compare the outcomes of SPD and SDD in chronic and subacute subdural hematoma burr-hole evacuation.

Methods: A prospective, randomised study was conducted on 200 patients admitted to the Neurosurgery Department at Mansoura University Hospitals. Patients were allocated into two equal groups: SPD (n=100) and SDD (n=100). Outcomes measured included recurrence rate, infection, seizures, mortality, parenchymal injury, and new neurological deficits.

Results: The recurrence rate was significantly higher in the SDD group (14%) compared to the SPD group (4%). Infection rates were 6% (SDD) and 4% (SPD), seizures occurred in 10% (SDD) and 4% (SPD), mortality was 4% (SDD) and 2% (SPD), and parenchymal injury was observed only in the SDD group (4%). New neurological deficits were noted in 10% (SDD) and 6% (SPD) of cases.

Conclusion: SPD is superior to SDD in terms of recurrence rate, incidence of seizures, parenchymal injury, and new neurological deficits. Although infection and mortality rates were lower with SPD, the difference was not statistically significant.

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Introduction

Chronic subdural hematoma (CSDH) is characterized by liquefied hematoma in the subdural space, often presenting as a hypodense or isodense crescentic collection on CT scans [1]. The elderly are most affected, with incidence rates ranging from 1.7 to 21 per 100,000 people annually [2]. Symptoms vary from headache and confusion to focal neurological deficits [3].

Burr-hole evacuation remains the preferred surgical technique, with postoperative drainage improving outcomes [4]. The choice between subdural (SDD) and subperiosteal drains (SPD) is contentious, with recent studies favoring SPD for its minimally invasive nature [5-9]. This study compares the efficacy and complications of SPD and SDD in a single-center experience.

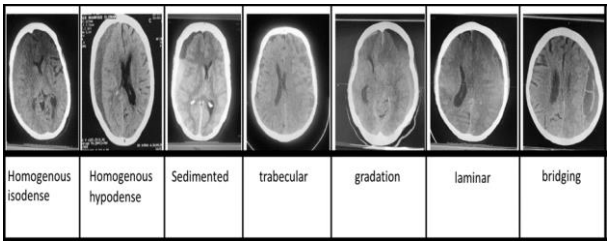


Figure 1. Types of chronic and subacute subdural hematoma in non-contrast CT brain.

PATIENTS AND METHODS

Study Design

A prospective, randomized study was conducted at Mansoura University Hospitals, including 200 patients (100 SPD, 100 SDD) with CSDH or subacute SDH.

Inclusion Criteria

- Adults >18 years.
- Hematoma size ≥10 mm or midline shift ≥5 mm.
- Focal neurological deficit or GCS ≤14.

Exclusion Criteria

- Acute subdural hematoma.
- Previous burr-hole evacuation for CSDH.

Surgical Procedure

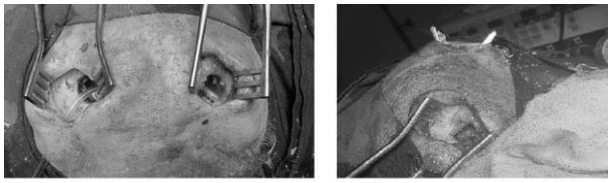
- Patients were positioned supinely under general anesthesia.
- Two burr holes (frontal and parietal) were drilled, followed by hematoma evacuation.
- Drains (SPD or SDD) were placed and connected to a ventriculostomy bag.
- Postoperative care included antibiotics, antiepileptics, and supine positioning for 24 hours.

Outcome Measures

- Recurrence rate.
- Infection, seizures, mortality.
- Parenchymal injury and new neurological deficits.

Statistical Analysis

Data were analyzed using SPSS v26. Qualitative data were compared using Chi-Square and Fisher's exact tests ($p \leq 0.05$).



two subdural drains subperiosteal drain

RESULTS

As regard recurrence rate between studied groups, it was 14(14%) in SD group while it was 4(4%) in SP group. As regard rate of infection between studied groups it was 6(6%) in SD group while it was 4(4%) in SP group.

As regard incidence of seizures between studied groups it was 10(10%) in SD group while it was 4(4%) in SP group. As regard mortality rate between studied groups, it was 4(4%) in SD group while it was 2(2%) in SP group.

As regard incidence of parenchymal injury between studied groups it was 4(4%) in SD group while there was no parenchymal injury in SP group. As regard incidence of new neurological deficits between studied groups there was deterioration in 10(10%) in SD group while there was deterioration in 6(6%) in SP group.

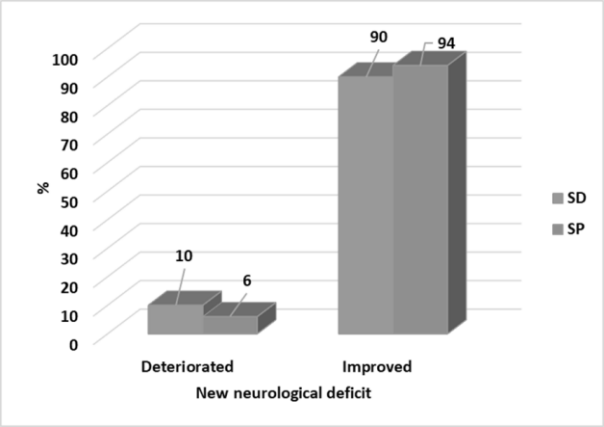
All the outcomes were comparable between the two groups except for the recurrence rate that was significantly higher in the SD group.

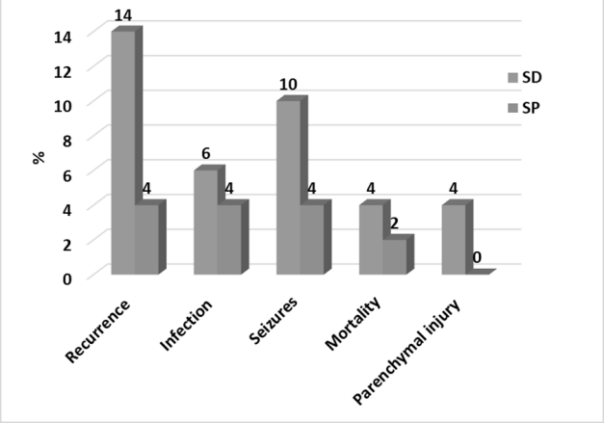
Table 1. Comparison of outcome among studied groups

	SD N=100(%)	SP N=100(%)	Test of significance
Recurrence			
-ve	86(86.0)	96(96.0)	**χ^2=6.11**
+ve	14(14.0)	4(4.0)	**P=0.013***
Infection			
-ve	94(94.0)	96(96.0)	χ^2=0.421
+ve	6(6.0)	4(4.0)	P=0.516
Seizures			
-ve	90(90.0)	96(96.0)	χ^2=2.76
+ve	10(10.0)	4(4.0)	P=0.096
Mortality			
-ve	96(96.0)	98(98.0)	FET =0.687
+ve	4(4.0)	2(2.0)	P=0.683

Parenchymal injury **No** **Yes**	96(96.0) 4(4.0)	100(100.0) 0	FET=4.08 p=0.121
New neurological deficit **Deteriorated** **Improved**	10(10.0) 90(90.0)	6(6.0) 94(94.0)	χ^2=1.08 P=0.297

χ2=Chi-Square test, FET: Fisher exact test *: Statistically significant





Discussion

CSDH is a prevalent disorder in neurosurgical practice. Surgical therapy is necessary for people who exhibit symptoms. Level I research suggests that removing burr holes and installing drains can significantly lower the recurrence rate [10, 11]. According to an international study of surgeons' practices worldwide, the most popular approach for drain insertion was SDD placement (50%), whereas 27% of respondents utilized the SPD and 23% used an SDD predominantly and SPD otherwise [12]. The current study aims to compare the effectiveness of two strategies using our own single-center experience.

Due to its substantial influence on the prognosis of CSDH, the key outcome chosen was the postoperative recurrence rate [13-15].

Our results showed that SPD group has statistically better results than SDD in terms of recurrence. In accordance to our results, Soleman et al. showed that recurrence rate was lower in the SPD group (8.33%) than in the SDD group (12%) [16]

Zhang et al. (11 and 13%) [12], Chih et al. (7 and 10%) [17] and Oral et al. (6 and 8%) [18] did not reveal a noteworthy variation in recurrence rates, whereas Kaliaperumal et al.'s investigation revealed no recurrences at all [19]. On the other hand, Häni et al. (24 and 22%) [20], Glancz e t al. (9 and 8%) [21] and Ishfaq (13 and 10%) [22] revealed that the SDD group experienced fewer recurrences than the SPD group; however, none of these investigations found statistical significance.

A high death rate, poor prognosis, and recurrence are all caused by significant volumes of remaining hematoma. The dura and the subdural membrane were suggested to be involved to initiating an inflammatory response that yields the inflammatory exudates. This subsequently leads to hematoma formation and progression. The postoperative drainage when analyzed was shown to contain increased levels of fibrin degradation products, plasminogen activator, kallikrein, interleukin-6, platelet-activating factor, fibroblast growth factor and vascular endothelial growth. These factors are supposed to be responsible for recurrence of the hematoma [23, 24].

In our study , the risk of seizures and infection was higher in the SDD group, yet it showed no statistically significant difference when compared to SPD group.

This was consistent with the findings of a previous meta-analysis that found no discernible link between the frequency of epilepsy and postoperative infection [6].

The difference was significant in favor of the SPD as shown by Soleman and his colleagues, where the SPD group showed significantly lower rates of surgical infections (P = .0406) [16].

In previous investigations, neither group's participants experienced surgical site infections or post-operative seizures [7, 17].

The brain insult was suggested to be responsible for development of post-traumatic epilepsy. However, it didn't match the findings where the SDD technique is associated with more injury to the parenchyma, but the rate of seizure was comparable between the two techniques [25]. This theory should be furtherly tested in subsequent studies.

It is theoretically possible that the contact from the drainage increases the likelihood of CNS infection in the SDD group. Overall infection rates were modest in all investigations, however, and SPD and SDD did not significantly differ from one another. Most likely, using antibiotics appropriately is enough to lower the risk of infection. However, because SDD insertion is more challenging than SPD, the frequency of superficial infections was shown to be greater in the SDD group [16].

As regard incidence of new neurological deficits between studied groups there was deterioration in 10 (10%) in SD group while there was deterioration in 6 (6%) in SP group.

This was in contrast to Pranata et al.'s findings, which demonstrated that parenchymal damage or new neurological impairments were substantially smaller (almost fourfold) in SPD than in SDD [26]. Compared to SPD, SDD is unquestionably more invasive and more likely to result in parenchymal damage.

In our study, mortality rate showed no statistically significant difference between two studied groups. This was in accordance with many previous studies. The mortality rate reported by Xie et al. was of 3.7 and 3.8% [27] and that reported by Ding et al. was 4.8% and 4.5% [28] while Pranata et al. showed that the mortality rate was 15.7% and 9.4% [26] in SPD and SDD respectively.

Limitations

Short period of follow up and a smaller number of cases limited our detailed assessment of various parameters in our study. surgeon experience may influence outcomes. Larger studies with longer period of follow up are mandatory to make more concise conclusions

Conclusion

SP drain is better than SD drain as regard recurrence rate, incidence of seizures, incidence of parenchymal injury and incidence of new neurological deficits also it was better than SD drain as regard rate of infection and mortality rate with no statistically significant difference between two studied groups.

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Prognostic value of traumatic brainstem injury in early computed tomography in paediatric population

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ABSTRACT

Introduction: In India, children aged <16 years constitute 35% of the total population and contribute to 20–30% of all head injuries. Prognostication of severe TBI in children based on early imaging and biomarkers has been universally challenging. The Marshall, Rotterdam, Stockholm, and Helsinki CT scores were developed to use acute head CT images to predict mortality at 6 months. Notably, none include criteria related to traumatic brainstem injury (BSI). The objective of this study was to compare the long-term outcome of pediatric patients with BSI identified on CT, along with an effort to classify BSI based on lesion volume, lesion location, presence of subarachnoid hemorrhage (SAH) and intraventricular hemorrhage (IVH) and how, the presence of these subset of injuries affect the outcome.

Methods: A retrospective analysis of pediatric patients presenting with TBI was undertaken from 2019 to 2023. CT scans were reviewed for brainstem lesions and, when present, characterised by location, size, and type (traumatic axonal injury (TAI), contusion, and duret haemorrhage). Clinical, demographic, and outcome data were then compared with the type of lesion, position of lesion, lesion volume, presence/absence of SAH and IVH.

Results: We found that lesion volume of more than 1 cm^3 is associated with a poorer GOSE score ($p< 0.001$). Similarly, lesions spanning both anterior and posterior quadrant are associated with poor outcome (GOSE: 3.4 +_ STD 2.9). We also found significant correlation with presence of SAH and IVH related to a poorer outcome ($p < 0.001$).

Conclusion: Early evidence from the current study suggests that certain TBI patients with BSI can have positive outcomes. BSI can further be classified into TAI, duret and brainstem contusions, each with variable outcome. Brainstem lesions with volume of >1 ml have been found to have a poorer outcome. Similarly, lesions spanning both quadrants tend to have a worse prognosis. Although there was no significant difference in outcome when compared with BSI – cases. These findings suggest of patients with brainstem injuries may exist with divergent recovery potential after TBI.

Keywords
traumatic brainstem injury, paediatric population

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INTRODUCTION

Trauma remains one of the most common causes of death in all age groups, but this is especially true in the pediatric population. Traumatic injuries are the leading cause of death and a major cause

of disability among children. [1,2] Greater than 45% of deaths in children aged 1–14 years in the United States are secondary to trauma. [3] However, predicting, in the early phase, the long-term neurologic outcome in head injury children is still a challenge. In the 1990s, Sharples et al. [4] found that with adequate management, prompt and accurate assessment, and early initiation of critical care is of crucial importance and has been shown to decrease mortality by almost 30%. Secondary brain lesions from systemic origin contribute to worsening of primary brain lesion. Hypoxia and hypotension are major sources of secondary brain lesions that worsen outcome. The deleterious influence of uncontrolled secondary brain insults of systemic origin must also be considered when trying to define predictive factors of outcome. [5]

For TBI triage, nonenhanced computed tomography (CT) is still an essential technique when used in conjunction with the neurological assessment. CT scans can be performed quickly and are very sensitive for injuries that need urgent care or neurosurgical intervention. [6] CT provides sufficient sensitivity to identify fractures, contusions, mass effects, and acute, potentially fatal cerebral hemorrhages. For these reasons, nonenhanced head CT is a class I recommendation for patients with moderate to severe TBI. [7] Acute head CT images were used to generate the Marshall, Rotterdam, Stockholm, and Helsinki CT scores, which were designed to predict outcome at six months [8]. Although these classification systems have become increasingly valuable in their contribution to predicting outcome, they remain imperfect and notably, none include criteria related to traumatic brainstem injury (BSI).

The paucity of existing studies utilizing traumatic brain stem injury (BSI) on computed tomography (CT) as an outcome predictor could be attributed to CT's lower sensitivity in identifying these lesions in contrast to magnetic resonance imaging (MRI) [9, 10]. Even after adjusting for other clinical, demographic, and imaging variables, a number of studies show that adding brainstem lesions to models improves prognosis accuracy [11, 12]. Previously thought to herald a catastrophic neurologic prognosis, research has shown that individuals with brainstem TAI have outperformed expectations in terms of their outcomes [13, 14]. Despite ubiquitous use of CT imaging in the acute TBI, ongoing efforts to refine prognostic tools based on head CT and increasing evidence that traumatic BSI on MRI is associated with more variable outcomes than previously assumed, minimal data are available that relate acute traumatic BSI on CT to long-term functional outcome. Moreover, no study has been done yet to analyze the impact of BSI in pediatric population and how the presentation of these cases and the lesion determine the outcome. Hence the need of the hour was to undertake a study solely dedicated to pediatric head injury, to assess the long-term outcome with BSI identified on CT brain and devise a classification to prognosticate such cases. Along with it we wanted to compare the outcome with that of patients with similar TBI injury but without BSI in an effort to identify subsets of patients with divergent probabilities of functional recovery.

Material and methods

A retrospective study was performed on pediatric patients, less than 16 years of age who presented to Trauma Centre, King George's Medical University, Lucknow, India, with intracranial injuries between January 2019 to December 2023. Patients were included if there was any evidence of cranial injury on the basis of imaging findings warranting admission in trauma centre. Pateints with polytrauma was excluded from the study. Data collected and recorded included the patient's age, gender, mechanism of injury, systolic blood pressure, and arterial blood oxygenation levels. In addition, Glasgow Coma Scale (GCS) score, injury description, and in hospital mortality were recorded. The type of neurological injury was determined based on the results of initial CT scans. Hypotension was defined as a blood pressure that for >5 min was below the 5th percentile for age. Hypoxia was defined as PaO2 less than 80 mm Hg at ABG on presentation to emergency department.

874 cases of pediatric head injury were identified, of which 354 were excluded because they underwent operative intervention. 520 cases were managed conservatively. Of the 520 patients, 19 were identified to have brainstem lesions (BSI+). From the remaining 501 patients with TBI, we matched each patient with BSI+ by age, sex, and admission Glasgow Coma Scale (GCS) score to patients with TBI without brainstem lesions (BSI−). Nineteen of the patients with BSI+ were matched

exactly on all three factors, staying within the same severity range (moderate or severe). Matching on admission GCS score was done to ensure comparable levels of brain injury severity in consideration of long-term outcome as the fairest possible way to investigate any unique contribution of brainstem lesions. In total, 38 pediatric patients with TBI, 19 with BSI+ and 19 with BSI−, were used for the analysis in this study. The primary outcome of interest was observed GOSE score. Favorable GOSE scores were defined as 5 to 8, and unfavorable scores were 1 to 3 (lower severe disability).

CT IMAGING

The earliest head CT of interpretable quality from within 48 hours of recorded injury was selected for detailed qualitative and quantitative analysis. CT scans with brainstem lesions were again further characterized. All pathoanatomic TBI lesions were demarcated, measured, and recorded. A suspected lesion was only recorded and used for analysis if it (1) appeared qualitatively to represent hemorrhage over other hyperdense elements, such as bone in the skull base, artifact, etc.; (2) had at least three contiguous pixels with Hounsfield unit measurements equal to or greater than the measured average Hounsfield units parallel to the tuberculum sellae-occipital protuberance line.

BSI+ lesions were analyzed by volume, location and lesion subtype (TAI, contusion, or Duret) (Figure 1).

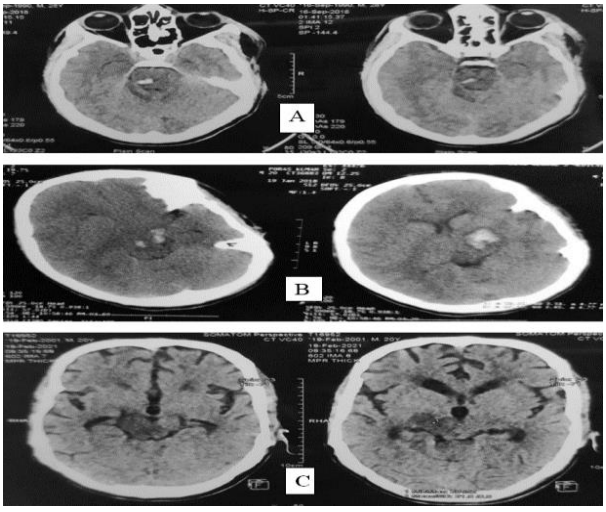

Figure 1. Subsets of BSI+ cohort. **A**. Small, isolated hemorrhagic lesions without associated mass effect or edema most consistent with traumatic axonal injury (TAI). **B**. Larger petechial hemorrhagic lesion with associated edema consistent with brainstem contusion. **C**. Dusky intervening nonhemorrhagic parenchyma consistent with Duret hemorrhage.

Separation of lesions into location by specific brainstem structures was not possible because of bony artifact and low tissue resolution. To improve statistical confidence, location delimitation was limited to anterior and posterior. Lesions were classified as anterior if the majority of the measured volume was situated anterior to a lateral line drawn perpendicular to the anterior–posterior midpoint of involved brainstem structures on relevant axial CT cuts. Patients with BSI+ were then divided into groups based on those with only one or more posterior, those with only one or more anterior, and those with at least one brainstem lesion in both the anterior and posterior halves of the brainstem. (Figure 2)

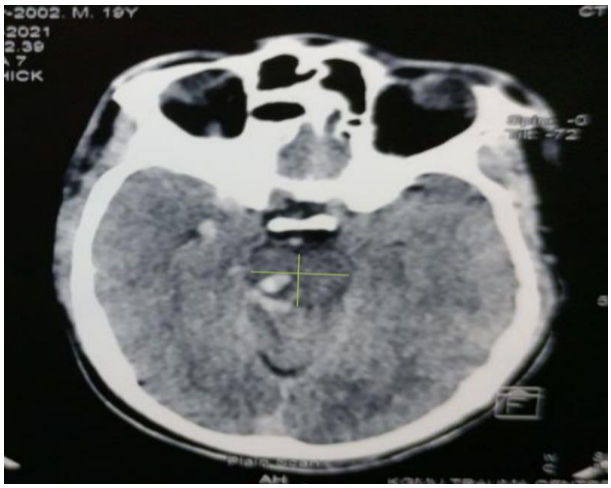

Figure 2. Axial head computed tomography (CT) slice demonstrating right pontine traumatic brainstem injury. The green line drawn perpendicular to each other divide the brainstem into 4 quadrants, where we consider anterior, posterior or lesion spanning both the anterior and posterior.

The association between lesion volume and outcome was analyzed by grouping BSI+ lesions above and below a cutoff point of 1 ml (1 cm3), whereas lesion type was consider as either TAI, duret and contusion. For lesion volume, this cutoff appeared to best segment lesions resulting from microscopic versus macroscopic mechanisms (i.e., isolated TAI versus arterial injury, venous stasis hemorrhage, etc.). Larger, diffuse hemorrhage lesion of at least 1 ml in size were compared with

those with all lesions less than 1 ml with regard to GOSE scores.

Statistical analysis

The distribution of patients with BSI+ and BSI− within subgroups of variables was analyzed using Fisher's exact and Mann–Whitney U-tests, as appropriate. The difference between long-term GOSE scores among groups in variables of interest was analyzed using Mann–Whitney and Spearman correlation, as appropriate. Similarly, bivariate differences in long-term GOSE scores among different subtypes of BSI+ were analyzed using Mann– Whitney and Spearman correlation, as appropriate. Significance tests in all bivariate analysis were conducted without assumptions about underlying distributions. Given the small sample size, multivariate regression analysis was conducted with exact logistic regression and was limited to no more than three parameters.

Results

Nineteen patients with BSI+ and 19 patients with BSI− were evaluated. The mean age for all patients was 11.05 years (standard deviation 3.17). Of 38 total patients, 31 patients evaluated were men (81.6%) and 7 were women (18.4%).

Table 1. Comparison of the distribution of patients with and without BSI

		Groups						
		Bsi +		Bsi -		Total		P value
		N	%	N	%	N	%	
Age	5-10 years	5	26.3%	7	36.8%	12	31.5%	.485
	11-15 years	14	73.7%	12	63.2%	26	68.4%	
	Total	19	100.0%	19	100.0%	38	100.0%	
Sex	Male	16	84.2%	15	78.9%	31	81.6%	0.675
	Female	3	15.8%	4	21.1%	7	18.4%	
	Total	19	100.0%	19	100.0%	38	100.0%	
Loc	Present	19	100.0%	19	100.0%	38	100.0%	1.000
	Absent	0	.0%	0	.0%	0	.0%	
	Total	19	100.0%	19	100.0%	38	100.0%	
Seizure	Present	3	15.8%	3	15.8%	6	15.8%	1.000
	Absent	16	84.2%	16	84.2%	32	84.2%	
	Total	19	100.0%	19	100.0%	38	100.0%	
Ent bleed	Present	2	10.5%	2	10.5%	4	10.5%	1.000
	Absent	17	89.5%	17	89.5%	34	89.5%	
	Total	19	100.0%	19	100.0%	38	100.0%	
Vomiting	Present	1	5.3%	1	5.3%	2	5.3%	1.000
	Absent	18	94.7%	18	94.7%	36	94.7%	
	Total	19	100.0%	19	100.0%	38	100.0%	
Type of head injury	Moderate	5	26.3%	7	36.8%	12	31.6%	0.485
	Severe	14	73.7%	12	63.2%	26	68.4%	
	Total	19	100.0%	19	100.0%	38	100.0%	
Hypoxia	Present	12	63.2%	16	84.2 %	28	73..6%	0.14
	Absent	7	36.8%	3	15.8%	10	26.3%	

	Total	19	100.0%	19	100.0%	38	100.0%	
Hypotension	Present	5	26.3%	5	26.3%	10	26.3%	1.000
	Absent	14	73.7%	14	73.7%	28	73.7%	
	Total	19	100.0%	19	100.0%	38	100.0%	
Hemoglobin	< 9 gm/dl	6	31.6%	4	21.1%	10	26.3%	0.461
	> 9 gm/dl	13	68.4%	15	78.9%	28	73.7%	
	Total	19	100.0%	19	100.0%	38	100.0%	
Hematocrit	<21	3	15.8%	5	15.8%	8	15.8%	0.426
	>21	16	84.2%	14	73.7%	30	78.9%	
	Total	19	100.0%	19	100.0%	38	100.0%	

Statistical comparison by Fisher's exact/ Mann–Whitney U-tests as appropriate.

Table 2. Outcome analysis between the two groups

	Group						
	Bsi positive		**Bsi negative**		**Total**		**P value**
	Mean	Sd	Mean	Sd	Mean	Sd	
Length of stay	7.00	3.40	5.53	2.01	6.26	2.85	0.271
Gcs score	5.89	2.21	6.84	2.01	6.37	2.14	0.166
Gos e	4.37	2.99	5.21	2.30	4.79	2.66	0.674

Table 3. Outcome analysis in BSI positive group based on mean GOSE

		GOS E		
		Mean	**Standard deviation**	**P-value**
Lesion type	Brainstem contusion	5.6	2.5	0.214
	Duret	4.0	4.2	
	Tai	2.7	2.9	
Hypoxia	Absent	5.3	3.0	0.125
	Present	3.8	3.0	
Hypotension	Absent	4.8	3.0	0.2596
	Present	3.2	3.0	
Hemoglobin	< 9 gm/dl	3.8	3.1	0.354
	> 9 gm/dl	4.6	3.0	
Hemocrit%	< 21	3.0	3.5	0.958
	>21	1.0		
Impact site	Infratentorial	4.3	2.9	0.325
	Supratentorial	4.6	3.4	
Skull fracture	Absent	4.3	3.0	0.256
	Present	6.0		
Lesion volume (cm 3)	< 1	5.4	2.8	0.001
	> 1	3.0	2.8	
Location of lesion	Anterior	7.0	0.0	0.01
	Both	3.4	2.9	
	Posterior	4.4	3.3	
Traumatic sah	Absent	5.4	2.8	0.007
	Present	3.6	3.0	
Traumatic ivh	Absent	5.1	2.8	0.001
	Present	2.2	2.7	

Table 4. Predicting favorable outcome after TBI in patients with brainstem lesions using exact logistic regression

Univariate Analysis							
		Gos E Score				**Or (95% Ci)**	**P Value**
		Favourable (5-8)		Unfavourable (1-4)			
		N	%	N	%		
Lesion Type	Brainstem Contusion	8	72.7%	2	25.0%	Ref	
	Duret	1	9.1%	1	12.5%	4.00 (0.17-95.76)	0.392
	Tai	2	18.2%	5	62.5%	10.00 (1.05-95.46)	**0.045**
Hypoxia	Present	6	54.5%	6	75.0%	Ref	
	Absent	5	45.5%	2	25.0%	0.40 (0.06-2.93)	0.367
Hypotension	Present	2	18.2%	3	37.5%	2.70 (0.33-21.98)	0.353
	Absent	9	81.8%	5	62.5%	Ref	
Hemoglobin	< 9 Gm/Dl	3	27.3%	3	37.5%	1.60 (0.23-11.27)	0.637
	> 9 Gm/Dl	8	72.7%	5	62.5%	Ref	
Hemocrit%	<21	1	9.1%	2	25.0%	3.33 (0.25-45.11)	0.365
	>21	10	90.9%	6	75.0%	Ref	
Impact Site	Infratentorial	7	63.6%	5	62.5%	Ref	
	Supratentorial	4	36.4%	3	37.5%	1.05 (0.16-6.92)	0.960
Lesion Volume (Cm 3)	<1 Cm^3	8	72.7%	3	37.5%	Ref	
	>1 Cm^3	3	27.3%	5	62.5%	4.44 (0.63-31.29)	0.134
Traumatic Sah	Present	5	45.5%	6	75.0%	3.60 (0.49-26.40)	0.208
	Absent	6	54.5%	2	25.0%	Ref	
Traumatic Ivh	Present	1	9.1%	4	50.0%	0.100 (0.008-1.193)	0.069
	Absent	10	90.9%	4	50.0%	Ref	
Multivariate Analysis							
		Gos E Score				Or (95% Ci	P Value
		Favourable (5-8)		Unfavourable (1-4)			
		N	%	N	%		
Lesion Type	Brainstem Contusion	8	72.7%	2	25.0%	21.33 (2.94 – 154.56)	0.0025
	Bsi Negative	3	15.5%	16	84.5%	Ref	
	Duret	1	9.1%	1	12.5%	5.33 (0.25 – 110.80)	0.279
	Bsi Negative	3	15.5%	16	84.5%	Ref	
	Tai	2	18.2%	5	62.5%	2.133 (0.27 – 16.6)	0.469
	Bsi Negative	3	15.5%	16	84.5%	Ref	

There was no significant difference in age, sex, presenting symptoms, severity of head injury, hypotension, hypoxia, hemoglobin and hematocrit among the two groups with p ranging from 0-14 to 1.00.

Outcome, as evidenced by GOSE score, did not identify a significant difference between BSI+ and BSI− groups, although there was a trend toward the BSI+ group having less favorable outcome (BSI+ mean GOSE score 4.37, BSI− mean GOSE score 5.21, p = 0.64).

This was also the case with length of stay (LOS) wherein there was no significant difference but the BSI + cases had a mean stay of 7 days in comparison to 5.53 days of the BSI – group (p=0.271).

Subgroup analysis of BSI + cases

To examine this further, we next completed subgroup analysis of the BSI+ group. In the group we tried to analyse the different factors resulting in any significant difference in outcome within the same group (Table 3). To begin with the lesion type did not affect the outcome, although TAI tend to have a poorer outcome with mean GOSE of 2.7. by lesion size, lesion location, and lesion type. Assessment of patients with BSI+ by lesion volume above or below 1 cm3 revealed significant differences in outcome. Assessment of presenting symptoms, hypotension, hypoxia, hemoglobin and hematocrit did not reveal any significant factor for outcome, although the presence of hypoxia, hypotension, hemoglobin less than 9 gm/dl and hematocrit of <21 is associated with a poorer outcome. Infratentorial impact and presence of skull fracture is again associated with poorer outcome but of not statistically significant.

Eight cases had lesions more than 1 cm3 achieving mean GOSE scores of 3, whereas 11 patients with brainstem lesions lesser than 1 cm3 made it 5.4 (p = 0.001), signifying that a lesion size of more than 1 cm3 is associated with poorer outcome. Assessment of patient outcome by brainstem lesion location did identify significant difference in outcome among those subgroups, with lesion extending both sides associated with a poorer outcome (mean GOSE 3.4), followed by posterior (mean GOSE 4.4) and then anterior (mean GOSE 7, p = 0.01). Presence of SAH and IVH is associated with poorer outcome, evident by a mean GOSE score of 3.6 and 2.2, respectively (p=0.001).

Exact logistic regression analysis was then used to evaluate the predictive ability of brainstem injury characteristics in an exploratory fashion compared to other clinical parameters. First, univariate exact logistic regression was employed to test the predictive ability of BSI volume, and BSI type. Subgroup of BSI + specifically TAI (OR 10, CI 1.05-95.46, p = 0.045), and BSI volume greater than 1 cm3 (OR 4.4, CI 0.6- 31.2, p = 0.13) is associated with an unfavourable outcome. Given these univariate findings, we last explored whether multivariate modeling of the significant univariate elements would strengthen the overall prediction of favorable outcome and whether there would be a unique contribution of brainstem lesions. Multivariate modeling of brainstem lesion type and volume identified a significant and unique contribution of brainstem lesion type, brainstem contusion to less favorable outcome (OR 21.33, CI 2.94 – 154.56, p = 0.002).

Discussion

This study provides evidence that pediatric patients with brainstem lesions evident on acute head CT done within 48 hours of injury, have the potential for favorable outcome and do not differ significantly when compared directly with patient with TBI with similar injury severity that do not have brainstem lesions. Outcome, as evidenced by mean GOSE score, did not identify a significant difference between BSI+ and BSI− groups, although there was a trend toward the BSI+ group having less favorable outcome (BSI+ mean GOSE score 4.37, BSI− mean GOSE score 5.21, p = 0.64). This was also the case with length of stay (LOS) wherein there was no significant difference but the BSI + cases had a mean stay of 7 days in comparison to 5.53 days of the BSI – group. Although no specific previous study has been conducted, solely to find out the significance of BSI in pediatric age group, but similar results was gained by John R. Williams et al, [15] who showed no significant difference in 6-month GOSE scores in patients with BSI+ (mean 2.7) compared with patients with similar but only cerebrum injuries (mean 3.9). In a study conducted by C Wedekind, et al [16] concluded that brainstem involvement in survivors of severe traumatic brain injury conveys a negative impact on long-term outcome. Similarly another study found that poor prognosis is more common in those with brainstem injury. The study concluded that understanding the anatomy and extent of brainstem injury, as well as its relationship to supratentorial abnormalities, and early use of MRI brain, would help assist in prognosticating and counseling of families. [17]

A lower GOSE profile was observed in our BSI+ cohort with lesions above a volume threshold of 1 ml. BSI volume greater than 1 ml was associated with a mean GOSE score of 3.0, implying most patients in that group did not survive. Conversely, patients with hemorrhage volume less than 1 ml had a significantly better mean outcome score of 5.4 (p = 0.001), and a reasonable chance of gaining functional independence. The lesion type did not affect the outcome significantly, although TAI tend

to have a poorer outcome with mean GOSE of 2.7. In univariate analysis TAI was found to significantly predict less favorable outcome (OR 10, CI 1.05-95.46, p = 0.045). Assessment of patient outcome by brainstem lesion location did identify significant difference in outcome among those subgroups, with lesion extending both sides associated with a poorer outcome (mean GOSE 3.4), followed by posterior (mean GOSE 4.4) and then anterior (mean GOSE 7, p = 0.01). Presence of SAH and IVH is associated with poorer outcome, evident by a mean GOSE score of 3.6 and 2.2, respectively (p=0.001). Another study conducted by A Hilario, et al showed 66% of BSI cases in their study had poor outcome. He also found that bilateral involvement was strongly associated with poor outcome (P < .05). Posterior location showed the best discriminatory capability in terms of outcome (OR 6.8, P < .05) and disability (OR 4.8, P < .01). [18]

In our study pediatric patients with brainstem injury have a reasonable chance for functional recovery, and for this reason, traumatic BSI should not be interpreted as a categorical entity from a mechanistic, pathologic, or prognostic standpoint. Multiple studies indicate added precision in prognostication when brainstem lesions are included in models, even when controlling for other clinical, demographic and imaging factors [15]. Although MRI acquisition in the early stages of post-TBI care is often not feasible in our setup, a study designed to compare early CT with early MRI in patients with traumatic BSI would allow more insight into the best characterization of BSI subtype and other TBI lesions outside the brainstem that may be contributing to observed long-term outcomes.

This study's findings should be balanced by its limitations, which include a small sample size, restricting statistical analysis and confidence, lack of further patient follow-up. Another drawback is not using dedicated volumetric analysis of lesions in our study. Furthermore, the data were analyzed in a retrospective and unblinded fashion, and as such, we acknowledge that the findings of this study may be heavily influenced by confirmation bias. Further prospective study of BSI lesions on early head CT in their relation to additional imaging, clinical parameters, and long-term outcome is needed before they can be incorporated into widely used prognostic models.

Conclusion

Early evidence from the current study suggests that certain TBI patients with BSI can have positive outcomes. It appears that early head CT can identify two different groups of patients with acute traumatic brain stem injury (BSI): a group with larger lesions consistent with duret hemorrhage or contusion with a lower chance of functional independence recovery, and a group with smaller lesions consistent with TAI. Brainstem lesions with volume of more than 1 ml have been found to have a poorer outcome. Similarly, lesions in brainstem spanning both anterior and posterior tend to have a worse prognosis. Although there was no significant difference in outcome when compared with BSI – cases, we conclude that the data support the notion that newer CT imaging classification systems must include these subsets of injury pattern, which may augment traditional clinical measures and provide a better individualized care to the patients and bring in limelight that patients with TBI, especially brainstem injuries stand a higher chance of favorable outcome.

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