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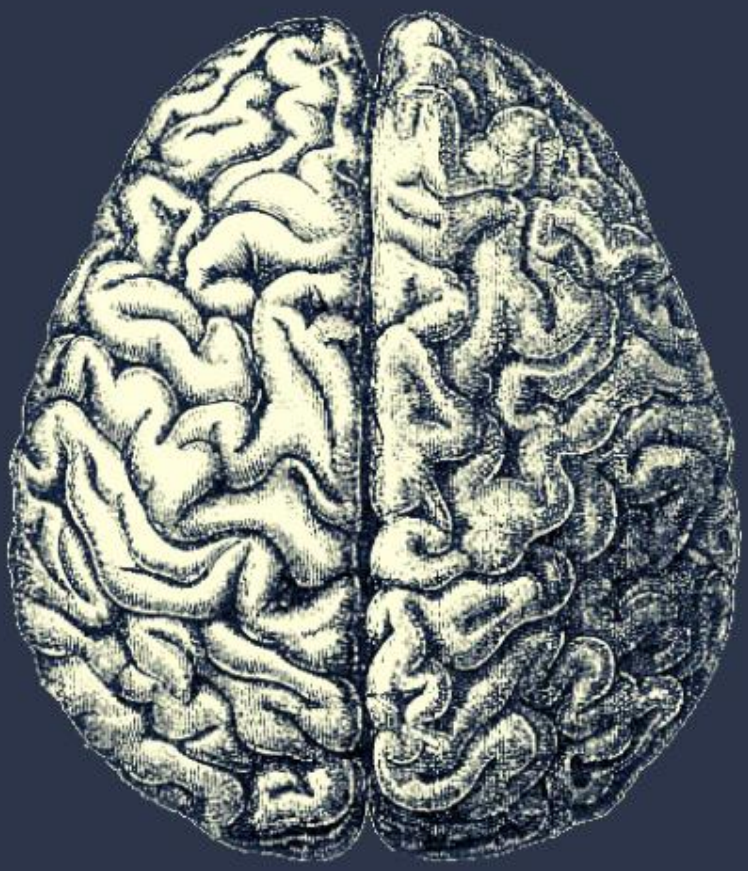

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Endovascular management of recurrent anterior communicating aneurysm previously embolized. Case presentation

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ABSTRACT
Although endovascular coiling treatment has been widely accepted as the method of choice for intracranial aneurysms, concerns about its durability are still discussed. Attention was largely focused on aneurysm recurrence after coil occlusion with possible unfavourable evolution to a new bleeding episode. We present our experience of a patient with a ruptured anterior communicating artery aneurysm previously treated by endovascular coil embolization that presented over a 4-year period for aneurysm recurrence.

Keywords
aneurysm recurrence, rebleeding, coil embolization

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INTRODUCTION

Coil embolization for intracranial aneurysms is currently the most accepted modality of treatment for patient with this vascular pathology. This is due to numerous studies which demonstrated its effectiveness in preventing rebleeding after aneurysmal rupture and a better outcome in terms of disability-free survival compared with aneurysmal neck clipping. Today, the main concern in endovascular treatment is represented by its durability due to aneurysm recurrence with disastrous potentially for the patient.

The purpose of this study was to describe a clinical situation requiring repeat embolization in a patient with a ruptured anterior communicating artery aneurysm previously treated by endovascular coil embolization, and discuss different aspects of aneurysm recurrence.

CASE PRESENTATION

A 50-year-old woman was referred for evaluation of an embolized anterior communicating aneurysm that demonstrated small coil compaction by fundus migration and aneurysm regrowth on serial angiography control. Four years before this referral, she had undergone an endovascular coil occlusion for a ruptured anterior communicating aneurysm. The patient made a good recovery.

Postoperative angiography control at 12 months revealed a small aneurysm neck repermeabilization due to partial coil compaction. The angiography control at 2 and 3 years showed a slow development of the remnant neck portion by aneurysm regrowth. Initially, a conservative management by imaging monitoring was decided. The angiography control at 4-year interval revealed a clear aneurysm sac enlargement with a 1:1 ratio of occluded aneurysm versus the permeable portion of aneurysm sac. A new session of coil embolization was decided for complete occlusion of the residual aneurysm (Fig.1).

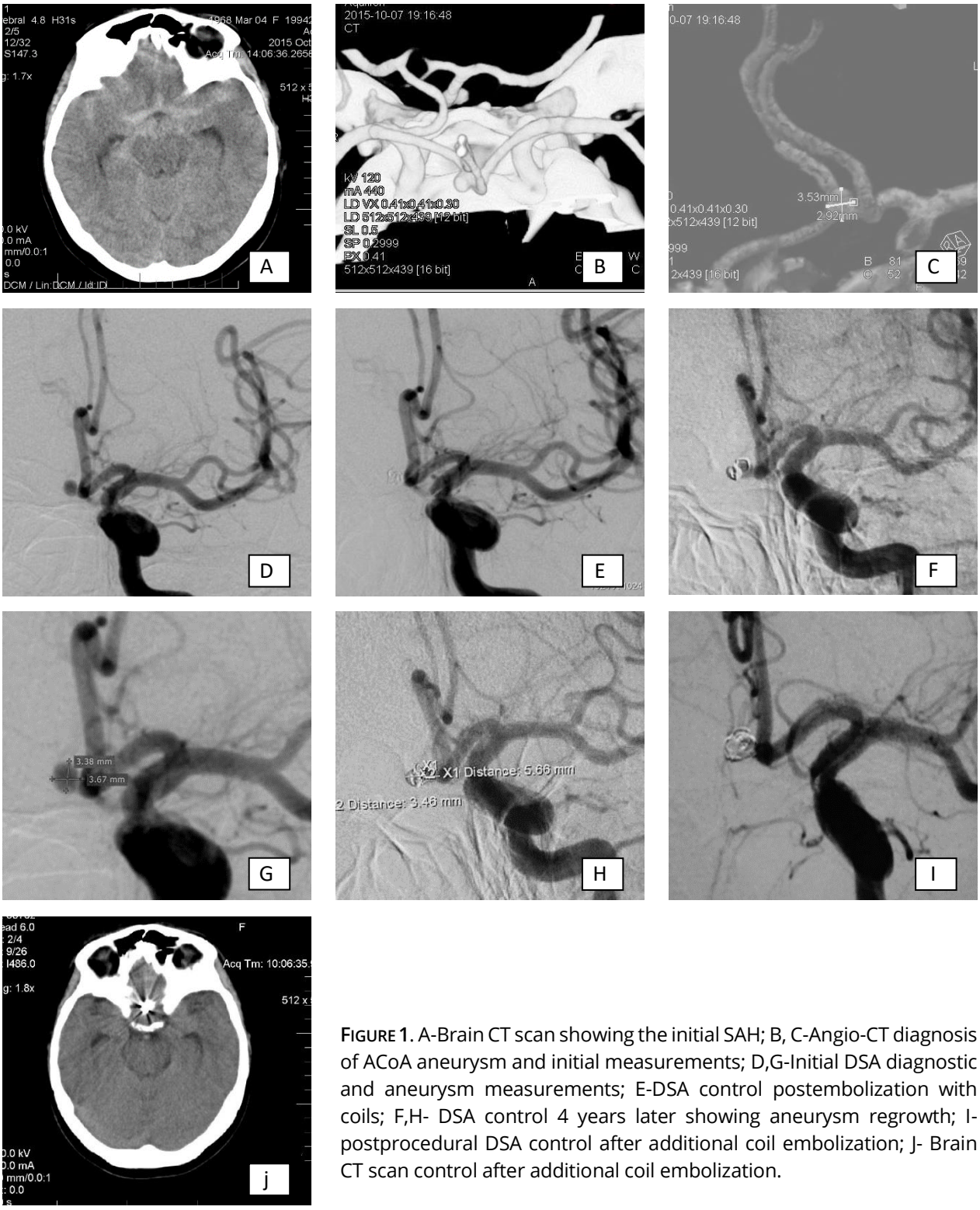

Figure 1. A-Brain CT scan showing the initial SAH; B, C-Angio-CT diagnosis of ACoA aneurysm and initial measurements; D,G-Initial DSA diagnostic and aneurysm measurements; E-DSA control postembolization with coils; F,H- DSA control 4 years later showing aneurysm regrowth; I-postprocedural DSA control after additional coil embolization; J- Brain CT scan control after additional coil embolization.

Endovascular Procedure

The interventional neurosurosurgeon team performed the endovascular treatment with the patient under general anaesthesia in a monoplane angiosuite. Treatment approach was performed through the right femoral access route with a 6F introduction sheath. Anticoagulation with 5000 IU heparin was used from the beginning of the procedures according to our centre protocol for unruptured aneurysm. The systemic administration of heparin is continued to entire period of the procedure (perfuzable solution of 1ml Heparin/500ml NaCl). A 6F guiding catheter (Ghider Softip 40XF Boston Scientific) was placed in the left internal carotid artery that provided arterial supply to the aneurysm remnant. Multiple DSA series were obtained in different degrees of obliquity. Also, a three-dimensional rotational angiography was finally performed to choose the working projection. Once an appropriate angle of obliquity was obtained on 3D PostProcessing unit that optimally highlight the relationship of neck of the aneurysm remnant and the parent vessel, the position was automatically send it to angiograph C-Arm. On this projection a road-mapping acquisition is performed and maintained throughout the duration of the procedure. A microcatheter (Excelsior SL-10, Stryker) was then advanced over a microguidewire (Transend 0.0014, Boston Scientific) under road-mapping guidance into the aneurysm remnant. Two GDCs-10 (Stryker) were then advanced and detached in the aneurysm remnant. The attempt to introduce the third GDC was not technically possible due to its distal end protruding into the parent vessel. Serial DSAs were performed to verify and to monitor the progress of aneurysm remnant occlusion. Once the aneurysm remnant was deemed to be satisfactorily occluded, the microcatheter was gently removed. This was followed by the guiding catheter removal through the femoral sheaths after obtaining a final DSA. The femoral sheaths were then removed from the femoral artery and hemostasis was obtained by prolonged compression. The patient was awakened from general anaesthesia and then admitted to the neuro–intensive care unit for 24-hour observation and monitoring. Patient was discharged home 48 hours later. Antiplatelet medication was maintained for the period of hospitalization.

Follow-Up Protocol

The imaging control of intracranial aneurysms postembolization was represented by conventional angiography. Flow-up angiography was performed during the early years of our endovascular activities, and was completed by MR angiography in the later years. Thus, in case of a completely embolized aneurysm conventional cerebral angiography, Dyna-CT and magnetic resonance angiography with three-dimensional reconstruction (3D MRA) were recommended at 1.5, 3, 6, 12 months, post-embolization, respectively. When a coil mass compaction, change configuration or migration was suspected in noninvasive studies, conventional angiography was performed immediately to check the exact state of the aneurysm and to decide the necessity of further treatment. If a stable occlusion is documented angiographically, follow-up conventional angiography was recommended after another 12 to 36 months. If stable occlusion was confirmed in post-embolization angiography performed after 1 to 2 years, follow-up was continued by noninvazive imaging studies [1,2].

In cases with unsatisfactory (low grade of coil packing) or incomplete aneurysm occlusion after initial coil embolization, we performed control conventional angiography at 3 months post-embolization. The next follow-up protocol depended on the stability of the coil mass and configuration of the embolized aneurysm. If a minor aneurysmal recanalization by minimal coil compaction at the aneurysmal neck occurs during the follow up, another 6-month MRA and 1-year angiography is performed. Instead, an early additional embolization treatment we performed when a major recanalization, with significant coil loosening, coil compaction or coil mass extrusion beyond the edge of a coil basket, or contrast filling within an aneurysm sac, occurs [2,3].

Discussion

Most of the largest studies from literature demonstrated that in patients treated for ruptured cerebral aneurysm, the risk of recurrence and rebleeding is significantly higher with endovascular coil embolization in comparison with surgery. Also, a direct relationship was shown between endovascular treatment and/or aneurysmal rupture, with an increased rate of incomplete occlusion of aneurysms.

The aneurismal recurrence was defined in the literature as being residual larger than 20% of the primary aneurysm, unstable progressing neck

remnants, aneurysm regrowth with or without coil compactation, and outgrowth of new aneurysmal sac. The evaluation of aneurysmal recurrence was usually performed by angioarchitecture comparing of angiographic controls with immediate postcoiling angiograms based on aneurysm sac size, neck morphology, dome-to-neck ratio and initial coil packing. Dorfer and all showed that 15,2% of the aneurysm smaller than 10mm and 38,2% of the aneurysm larger than 10mm had relevant recurrences and similarly 17,2% of aneurysm with neck <4mm and 27,3% with neck >4mm presented with recurrences. They also reported that a threshold of 25% packing density and 30% volumetric aneurysm filling has been correlated with significantly stable angiographic results [2,5,6].

Different mechanisms of aneurysm recurrence after endovascular treatment were described in large series. The most common mechanism of aneurysmal recurrence is residual aneurysm due to coils compaction. This is the most common situation in the cases of large and giant aneurysms with simple coils embolization. Lower rates of repermebilisation were reported for those aneurysms when the stent-assisted or balloon technique was associated with coil occlusion. Coil migration into the intraluminal thrombus was also frequently reported especially in large partial thrombosed aneurysm. If initially this situation was considered by specialists as a separate entity, it is currently included in the category of compaction aneurysms. Nevertheless, coil mass compactation was not constantly associated with aneurysm recurrence. There have been many situations where the coil compaction phenomenon has been stable over time without any evolution to an aneurysm recurrence that would impose a type of reintervention [1,2,3].

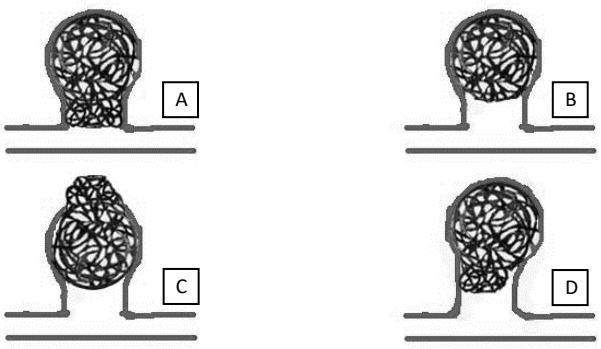

Figure 2: Mechanisms of aneurysm recurrence: A-Coil complete occlusion; B-Coil compactation; C-Coil Fundal migration; D-Coil regrowth.

In the situations of aneurysmal enlargement without signs of coil compactation, the mechanisms of aneurysm recurrence were defined as regrowth.

The third entity of aneurysmal recurrence mechanism defined by the last publications was fundal migration of the coil package through the wall of the aneurysmal sac. Also, some authors have mentioned as an aneurysm recurrence mechanism a combination of regrowth and fundal migration or compactation and fundal migration. All of these situations imposed an additional treatment.

Aneurysm recurrence could benefit from both endovascular and surgical treatment. The decision, regarding which type of treatment must be used, should be individualized as much as possible for each individual case. Most of the studies demonstrate that additional treatment is usually associated with a low complication risk and results in satisfactory stable occlusion in most patients. The main indication for initiating additional treatment was made because of the reported increased risk of further haemorrhage associated with unstable remnants and incompletely occluded aneurysms. The reported rebleed rate after endovascular therapy indicated in ISAT1 was 0.2% per patient year with a follow-up from 1 to 8 years (mean, 4 years) and 1.3/100 patient years in the CARAT study, with no haemorrhage occurred after 2 years[..]. The main factors to be considered when deciding on the method of treatment used in the aneurysmal recurrence were represented by the aneurysm size and location, patient age and clinical condition, size of aneurysm remnant and its relation with adjacent vessels, presence of coils in the aneurysm neck, mechanism of recurrence and history of SAH manifestation [2,6].

The time interval between aneurysm coil occlusion and angiographic control that demonstrate a clear evolution to aneurysm recurrence which require additional occlusion treatment was named the recurrence latency. It was demonstrated that the first control angiogram often performed at approximately 6 months is not sufficient to detect all aneurysms that may require additional treatment. Raymond et al have also reported progressive deterioration with aneurysms recurrences that were angiographically occluded at 6 months. Progressive neck remnant enlargement has been demonstrated in 14.8% of aneurysms in the first year after treatment and a major recurrence in

20.7% at a mean of 16.48 ± 15.93 months. The other authors detected 46.9% of all recurrences by 6 months and 96.9% by 36 months. [1,2,5]

The complication rate following aneurysms retreatment by endovascular retreatment ranged from 0% to 11% in previous reported series. Some of these series included patients with multistage treatment. Thromboembolic events were the major complication mentioned but only 3.2% of patients had a permanent neurologic deficit as Henkes et al described [2].

Conclusions

The coil mass instability due to compactation, migration or aneurysm regrowth that impose additional retreatment remains a major deficiency of endovascular treatment. The great challenge in managing patients with aneurysm recurrences remain the ability to early recognition of unstable aneurysm residuals with rehemorrhage potential by benign nonprogresive one. In case of relevant aneurysm recurrences both endovascular and surgical techniques could be successfully applied.

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Pathways of metastatic spread in meningiomas

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Abstract
Meningioma is a common intracranial neoplasm derived from meningothelial cells, and it is generally associated with a benign clinical course. In spite of this, the malignant behaviour of these tumours as the occurrence of extracranial meningioma metastases in different organs is described in literature: lung and pleura, spine and other bones, abdominal organs, lymph nodes or even skin. The aim of this review is to analyse the pathways of metastatic spread of the intracranial meningioma tumour cells towards different organs.

Keywords
meningioma, metastasis, malignant, WHO classification

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Introduction

Meningiomas are the most common central nervous system tumours in adults, making up approximately 30% of all the intracranial tumours, with an increasing incidence (14, 57). According to the mitotic activity and tumour differentiation, the World Health Organization (WHO) grading meningiomas as it follows: grade I, grade II and grade III, the last two being characterized by a more aggressive behaviour, a high risk of recurrence (12, 39) and even metastasis. These extracranial meningioma metastases (EMM) are more frequently associated with atypical meningiomas (Table 1) or anaplastic meningiomas (20, 27, 59). Although typical meningiomas are benign solitary intracranial neoplasms, they can cause extracranial metastasis, a rare phenomenon found only in 0.1% of cases (15, 23, 26, 35, 37). According to epidemiological data, about one in 1000 cases of meningiomas metastasize (35, 56).

The most frequent sites of EMM are: the lung (60%-70%), abdominal viscera (the liver most frequently) (30-40%), pleura (23%), lymph nodes (14-20%) and bones (10%) (3, 6, 21, 38, 42, 50, 60, 66). Other rare localizations were identified in the parotid gland (17), skin (32, 51), deep soft tissue (9, 18, 44, 48, 64, 70), kidney, spleen, thyroid and adrenal gland (64).

Due to the rare nature of EMM, this is a challenging diagnosis for physicians and pathologists (25). Because the treatment has not yet been standardized and a management protocol has not been developed, the prognosis of these patients remains unknown (3, 42, 50).

TABLE 1. Review of publications on extracranial metastasis of atypical meningiomas

Author	Gender, Age	Intracranial Localization	Metastasis Localization
Andric *et al.* (5)	F 52	parasagittal	lung
Baek *et al*. (9)	M 44	nasal septum	chest wall, parietal region
Drummond *et al.* (19)	M 66	frontal	lung
Doxtader *et al*. (18)	M 8	parasagittal	subcutaneous tissue of neck, lymph nodes
Kim *et al.* (41)	M 68	parietal	skull bone
Lanfranchi *et al.* (43)	M 74	not specified	lung, liver
Pinsker *et al*. (55)	M 76	frontal	cervical spine

METASTATIC SPREAD

Although all types of meningioma, including the grade I meningiomas can metastasize, EMM is more common in the atypical (5%) and anaplastic (30%) histopathological subtypes (7). Nevertheless, some authors believe that these percentages are overestimated, firstly because some of the reported cases probably are metastatic angioblastic meningiomas and not genuine meningioma metastases. Moreover, the angioblastic meningioma is now classified as haemangiopericytoma (25, 28, 34, 46). Thus, considering all these modifications, the 5% rate of metastasis in atypical meningiomas does not coincide with the current WHO classification (25). However, the true prevalence of EMM is unknown at present (64).As for the time interval between the initial diagnosis of the meningioma and that of extracranial metastasis, it varies from 3 months to 30 years (40).

Although throughout time, some authors emphasized tumour necrosis, blood vessel invasion, high cellularity, cellular heterogeneity, high mitosis rate and nuclear pleomorphism in the occurrence of EMM (1, 20, 54), these criteria apply to malignant tumours and do not explain metastatic disease in benign meningiomas (64). In spite of this, the exact etiology of metastatic spread in meningioma is still unknown (7, 22).

Initially, the surgical resection was reported as one of the most important risk factors for iatrogenic metastasis of histologically aggressive meningiomas (2). Hence, it was hypothesised that surgery causes the seeding of the lymphatics and vascular channels (including that of the scalp), resulting in extracranial metastatic disease via haematogenous or lymphatic route (31). Besides, other studies showed that tumour cells can propagate with no previous surgery (25, 36), considering that the invasion of adjacent venous sinuses can lead to extracranial spread of meningioma, especially when there is no history of surgery (23).

Lung metastasis. As mentioned before, the lung is the most common site of the EMM. In these cases, the seeded tumour cells access dural venous sinuses and cranial veins, then diffuse through the azygos system into pulmonary circulation and then they can reach either the lung or the pleura (21, 32, 40). Most EMM were found along the jugular vascular drainage, such as in the cervical lymph nodes, parotid and thyroid gland and finally in lung and pleura (64).

Bone metastasis. Bone is one of the least common sites of meningioma metastasis (38, 60). In a review of meningioma metastasis in the bone, Khan et al. found out that half of the patients had multiple areas of bone metastatic disease, while the

other half had solitary bone involvement (40). The most common region was the axial skeleton, namely the thoracolumbar spine and sacrum (40), while metastases in the extra-axial bone were rarely identified (22). The most common involvement of the spine can be explained by the connection between the dural venous sinuses and the vertebral venous system (10). This route via the paravertebral venous plexus may also play a role in the metastatic spread across the kidney or adrenal glands (64).

Of the non-axial skeleton, the femur was the most common long bone site of involvement (40), and this metastasis location is more difficult to explain, even if some authors suggested the extension of the tumour via arterial spread (68). The bone metastatic disease is a lytic bone lesion and may result in pathological fractures or collapse of the vertebral bodies (40).

Liver metastasis. As far as the dissemination pathways for hepatic metastasis are concerned, some authors believe that this lies in the dissemination through the vertebral venous system connected with the veins of the thoracoabdominal wall or dissemination of tumour cells of the bone metastasis to the liver (24, 25). The liver can be involved whether the metastases pass through the right atrium towards the inferior vena cava and further into the hepatic veins (64). In literature, cases of hepatic metastasis of meningioma via a ventriculoperitoneal shunt through the cerebrospinal fluid route were cited (51).

Scalp metastasis. Surgical seeding of tumour cells in meningioma surgery is a rare entity and it usually involves soft tissues near the craniotomy site (8). In literature, 19 such cases of scalp metastasis of intracranial meningiomas were reported (8).

Scalp metastasis of meningioma were firstly reported by Harvey Cushing, with an incidence of 1.21% cases in a cohort of 313 patients, which results in 4 patients suffering from this condition. In all of these cases, scalp metastases were found in surgical scar many years after the first surgery (4, 30, 49, 69). Different series reported percentages of 3% in a cohort of 119 patients with WHO grade II and III meningiomas (53) and others of 1.2% (8).

The suggested mechanism is intraoperative seeding and it may apply to all histological grades of meningiomas. Avecillas-Chasin et al. considers that immunosuppression, radiation therapy, CSF fistulae and multiple reoperations with subsequent surgical wounds problems (8). Nevertheless, the reason for extracranial tissue invasions is still unclear.

Carrying out multiple interventions can be a risk factor for the extracranial spreading of intracranial tumours, in that surgical bone deteriorates the natural barriers for intracranial tumour dissemination, providing access to the blood vessels and lymphatic system (8, 63). The higher-grade histopathological meningioma also contributes to this in case of tumours which have the tendency to produce greater levels of vascular endothelial growth factor and angiogenesis, thus favouring vascular invasion and metastatic behaviour (29).

Piecemeal resection was also presented as a risk factor of extracranial dissemination of tumour cells in WHO grade III meningiomas (8). Although initially, it was suggested that the latency period of meningioma metastasis is influenced by the histological grade (2), the significant variability over this la-tency period proved that this is not related only to the histological grade of meningioma (2, 16, 30, 45, 47, 52, 61, 62, 65, 67, 69).

With regard to preventing the spread of tumour cells, some authors recommended: air-tight closure of dura mater, changing the gloves and surgical instruments for wound closure after the intracranial phase, replacing the bone flap and saline irrigation, wound abundance before the closure (4, 33).

Treatment strategies. The treatment for meningioma metastasis has yet to be set out. In spite of this, metastectomy seems to improve the prognosis, especially when the tumour is a low-grade meningioma. As for the chemotherapy with hydroxyurea, vincristine, cyclophosphamide and doxorubicin, this has a limited efficiency with progression under chemotherapy (25). Moreover, some immunotherapies with interferon-α and somatostatin analogue were tested lately, but with limited effects (11, 58). Nonetheless, the choice treatment in meningiomas remains the gross total resection and adjuvant radiotherapy (13).

Conclusions

Because of the fact that EMM were rarely reported, there are no guidelines regarding the staging or treatment of EMM. Metastatic spread remains a therapeutic challenge and the treatment must be multidisciplinary evaluated. In addition, this data highlights the fact that metastatic dissemination of meningiomas is possible even under conditions of

benign histopathological grades and thus organ donation should be considered.

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Underlying histopathology of peripheral nerve injury and the classical nerve repair techniques

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ABSTRACT

A much-debated subject in the last 20 years, the recovery after peripheral nerve injury still remains one of the most researched themes of our days. Although the central nervous system has not exhibited any ground-breaking discoveries in matters of healing through surgical procedures, this is not the case for the peripheral nervous system (PNS). The PNS recovery after injury has improved over the years so we now speak of time and percentage of rehabilitation. The increased interest for this subject is a result in the development of the medical technique, that allowed the creation of new molecules capable to improve the regeneration rate. Furthermore, the evolution in diagnostic parameters, as well as the possibility of a thorough follow-up, contributed to the ascending research of this field. One must not forget that all experimental studies have as endpoint obtaining safe and reproducible solutions which can be applied in treating patients with peripheral nerve injury. We will briefly present the microscopic events that occur following a peripheral nerve injury, the key factors which influence their regeneration as well as the classical techniques used to repair them. However, the most intriguing topic in nerve regeneration is not related to the surgical procedure (considered to be the Gold Standard in whole nerve injury), but rather the helping substances that facilitate a faster and better recovery.

Keywords
peripheral nerve,
nerve graft,
Wallerian degeneration,
nerve fascicle

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INTRODUCTION

The first world war was the trigger that put peripheral nerve injuries under the microscope.[1] The lack of motility/sensitivity generated a major distress that affected the quality of life of war veterans. This resulted in a greater study of the anatomy, physiology and histopathology of peripheral nerves, Platt describing a series of 26 patients who underwent surgery for nerve repair.[2]

Several authors came up with a series of classification, taking into account the degree of injury and the histologic aspects, the most known being the Seddon classification (3 degrees of injury – neuropraxia, axonotmesis, neurotmesis) [2] and the Sunderland classification (5 degrees, splitting the axonotmesis degree into 3 different categories).

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[4] Mackinnon introduces a 6th type of nervous injury – a combination of different degrees of nerve injuries.[5]

Neuropraxia represents a block in the conduction of the nerve impulse without any histologic lesions. Its recovery is self-limited in 6-12 weeks. The second degree injury (axonotmesis) involves the interruption of the axon keeping the integrity of the neural tube. This leads to anterograde Wallerian degeneration as well as a short retrograde nerve degeneration. The intact endoneural tube consisting of epinerve, perinerve, endonerve and the sheath of Schwann cells ensures the premises of a good recovery.

Sunderland's 3rd degree lesion associates an endonerve injury, which implies a minimum fibrosis; for this reason, the recovery will be slightly deficitary. 4th degree lesion implies keeping only the epinerve intact, a surgical intervention being absolutely necessary. Neurotmesis (5th degree lesion) consists in the complete interruption of the nerve. With the exception of the 1st degree injury, all others will suffer processes of degeneration followed by regeneration.

Overview on evolution and complications in nerve injuries

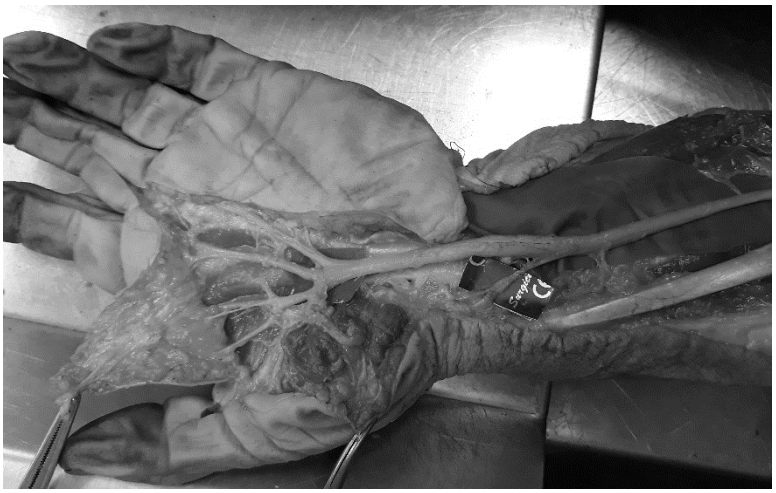
Nervous degeneration. The disruption of the connection between axon and neural body triggers the initiation of axonal disintegration distal to the level of injury. This is called Wallerian degeneration and it is an autolytic process of cleansing the distal stump. The axoplasma and the myelin sheath will be disintegrated, followed by a phagocytic process performed by macrophages and Schwann cells. The Wallerian degeneration takes place strictly in the myelinated fibers of the distal nerve. The Schwann cells will proliferate, changing their fenotype and reducing the synthesis of myelin. The cells will then align to form the Bugner bands (endoneural tubular structures capable of directing nerve fibers). Their role is to guide the neural sprouts from the proximal end on the right path towards the original end-organ. This guidance occurs under certain neurotrophic factors, as well as neurotropic agents such as: NGF – nerve growth factor, glial maturation factor-b, GFAP - glial fibrillary acidic protein, NCAM – cellular adhesion molecules, GGF – glial growth factor, BDNF – brain derived neurotrophic factor, IGF-1 insulin growth factor.[6]

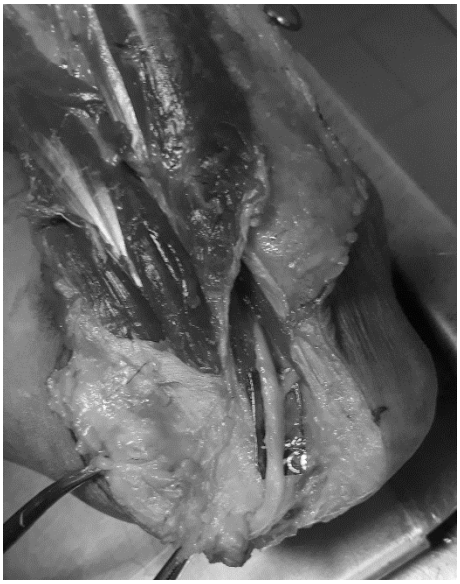
There are also a series of agents which modulate the inflammatory process or intervene in the cellular change: cytokines - IL1, IFNγ, IL6 si IL12, TNF-α, the latter being responsible for macrophage recruitment.

Aside from the Wallerian degeneration, there is also a certain degree of retrograde degeneration in the proximal segment. The axon proximal to the injury depends on the state of the cellular body – it either survives and regenerates or it dies. The last surviving Ranvier node modifies his structure and biochemistry, preparing for axonal regeneration.

Nervous regeneration. There is a de novo restoration of the axon through a process called axonal sprouting. This is realized through the synthesis of the cellular components and the renewal of the original axonal architecture. At the proximal site the neural sprouts advance distally and form together with the axon a regenerative unit. The distal area of each axonal sprout presents a tumescence called growth cone, which contributes to guiding the nervous regeneration. The axonal sprouts cross the lesion area, reaching the distal end. Once in contact with a Bugner band, the axonal sprouts will be covered by the cytoplasm of the Schwann cells and advance through the endoneural tubes to reach the target organs.[7] In case of a misalignment, these will advance towards other organs, different from the one they innervated initially. If these sprouts don't advance correctly, they may end their elongation process by forming a neuroma.

The advancement of the growth cone is done through contact guiding (the local environment playing an important role), as well as neurotropic factors. Tissue specificity implies preferential growth of the axonal sprouts towards nervous tissue structure, and inhibiting the paths towards other structures (muscle or tendon). The nervous fibers also posses the capacity to differentiate between motor and sensitive nerves in mixt nervous trunks. Topographic specificity implies correct re-innervation of the right muscle (motor fibers) or cutaneous area (sensitive fibers). End organ specificity refers to re-innervation of the correct sensitive organ (Paccini, Krause, Ruffini) or muscle fiber (fast or slow).





CADAVER DISSECTIONS – median nerve in forearm and hand (above) and ulnar nerve at elbow region (right).

Factors which influence the regeneration process

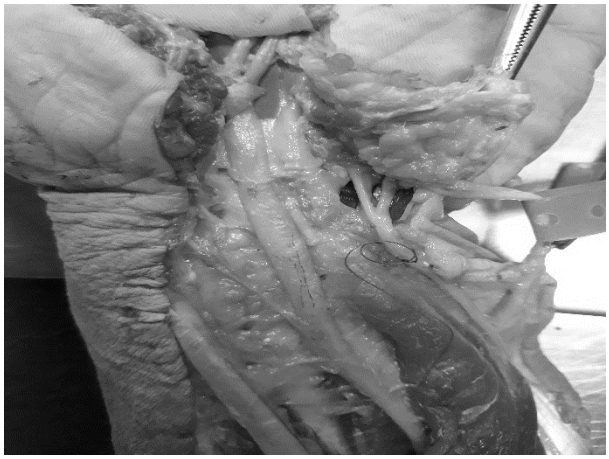
The division of the axon determines complex processes which aim the nervous restoration. The surgical repair creates favorable conditions for nervous re-innervation. The stages for nerve repair are: debridement of the injured area, approximation and nerve suture.

The factors that influence the final outcome are: factors relating to the patient (age, habits such as smoking), timing for nerve repair, the degree of injury, injury mechanism, level of injury, type of nervous repair, surgical technique (atraumatic, following the tension free principle), as well as the experience of the surgeon and the availability of proper microsurgery instruments as well as a microscope.

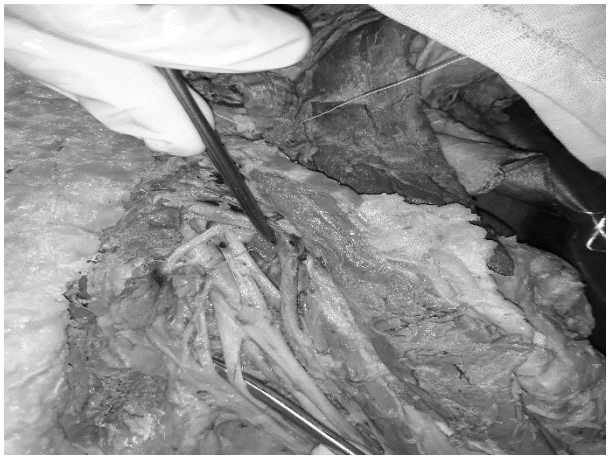
Young patients have a much better capacity of recovery compared to older patients. In model rat experiments, it has been proven that smoking slows down the regeneration of peripheral nerves. Other medical conditions such as diabetes, hypothyroidism or peripheral vascular diseases interfere with the nervous regeneration.

The timing is an important issue in nerve repair. Primary repair is done in the first 72 hours. Between 72 hours and 7 days, there is a delayed primary repair and after this time – secondary repair. The sharp object injuries with minimal contamination benefit from primary repair with favorable results. For crush injuries or avulsions where viable and non-viable tissue is more difficult to set apart - a wait-and-watch approach is the appropriate therapy. In case no improvement is seen 3 months from injury, a surgical intervention to prevent denervation and muscle loss is recommended.

The degrees of injury were previously presented. 1st and 2nd degree injuries have more favorable results compared to the higher degrees of injury. The mechanism of injury is a predictive factor – cutting injuries have better results than crush injuries. The level of injury constitutes another factor – proximal lesions have poorer results compared to distal ones. The explanation is that it takes a longer time for the axon to reach its end organ when the lesion is proximal; furthermore, the more proximal the injury, the more nervous fibers that the nerve contains (sometimes both motor and sensitive), which requires a perfect alignment so that each fiber finds the right way.



Cadaver dissection: median and ulnar nerve at the wrist region (representation of the ulnar nerve bifurcation at this site).



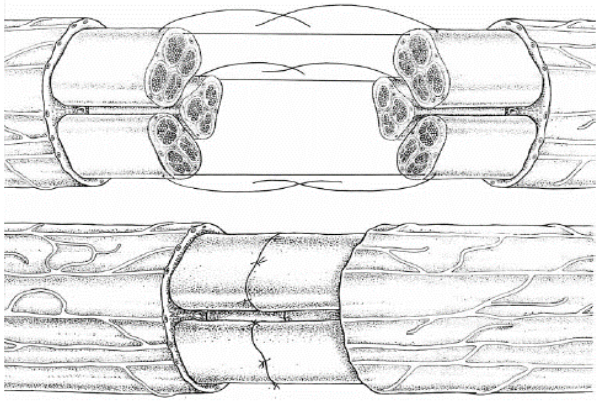
Cadaver dissection: median and ulnar nerve at the wrist region (representation of the ulnar nerve bifurcation at this site);

Classic repair techniques

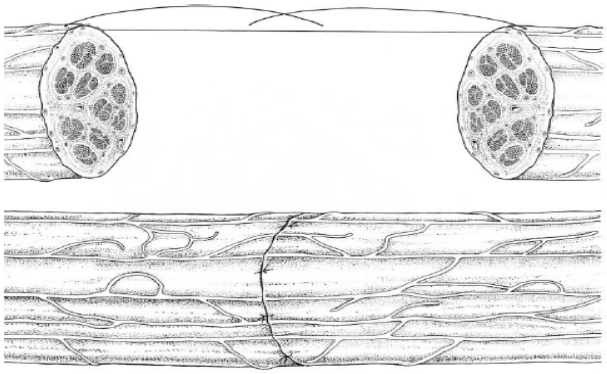
There are 3 classic nervous sutures: epineural, perineural and epiperineural.

The epineural technique requires nervous endings isolation, placing a contrasting background behind them, excision of the injured tissues. The nervous endings are trimmed for a suitable match and are then arranged so that the inner fascicles correspond to one another. The sutures are place at 180o and the rest are placed at half the distance. Main disadvantage is that the nerve is connected only on the outside, the fascicles being able to slide and follow wrong regeneration paths. On the other hand, this suture has the advantage of a minimal fibrosis.

Repairing groups of fascicles is a more precise technique in terms of alignment but it has the disadvantage of transferring the tension from the epinerve directly to the fascicles. It also requires more sutures for a well distributed tension, which implies more fibrosis.



GROUP FASCICLE REPAIR



EPINEURAL REPAIR

The fascicular repair is done by suturing each fascicle individually and may be indicated in mixt nerves where there is a high probability of unsuitable axonal growth (sensitive nerves growing in the motor nerves' path). In the case of fascicular suture, the internal epinerve is dissected and the fascicles are sectioned at different sites and repaired by 1-2 sutures. Despite an almost perfect alignment, there is a great degree of fibrosis so the outcome doesn't bring better results.

Nerve grafting

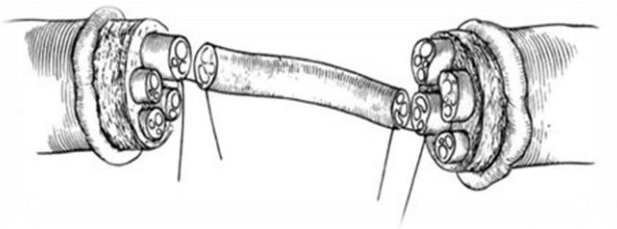
When the nervous defect exceeds the length of a direct suture, the gap must be filled by a structure that can guide the nervous regeneration. The nerve graft is the Gold Standard in this case. It acts as a path that the new axon passes, having the trophic support of the Schwann cells.

There are several types of grafts: autografts (from the same person), isografts (from identical twin), allografts (from the same species) and xenografts (from different species). When talking about allografts and xenografts, an important issue is the immune reaction of the organism and the immunosuppression used to prevent graft vs host reaction.

Allografts can be obtained from cutaneous nerves or nerve trunks. The harvest doesn't determine a high degree of functio laesa in case of cutaneous nerves but nerve trunks are harvested only in the case of a fully compromised muscle (complete muscle denervation). Graft diameter is of great importance in a successful transfer because in grafts with a diameter above 3mm there is a great risk of ischemia with possible central necrosis.

The shortcomings in using an autologous graft is that it requires a donor site sacrifice. Furthermore, all

grafts will suffer the same changes as in the distal end of an injured nerve and the axon will be forced to pass through 2 different sutures. Sometimes, in the distal suture, the fibrosis can be so great it can hinder the axon from running his course. This is the case for long grafts and this can be prevented by sectioning distal end of the graft in a second intervention. Some authors recommend only the proximal end of the graft to be sutured in a first stage and the distal in a second stage, once the neurotisation is complete. If the block exists, resection and resuture is mandatory.



Another important issue is the difference in calibre. There are 5 types of fascicle distribution which should be handled differently. The single fascicle arrangement (the nerve containing one big fascicle) or the arrangement with 2-4 big fascicles – the graft should cover the whole cross section of the injured nerve. In the case with 5-12 fascicles nerves, these contain a higher amount of connective tissue. Therefore, the suture is best done for each fascicle individually.

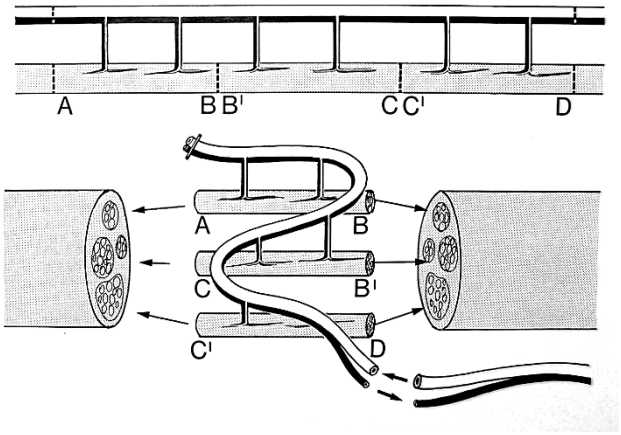
The polifascicle nerves contain a high level of connective tissue which should be excised to prevent graft adherence to connective tissue. Single suture similar to the previous type is the proper nerve repair in this case. The last type is the polifascicle arrangement in which the fascicles are randomly distributed and has a high risk of misalignment. It doesn't allow a fascicle group identification – therefore the interfascicle dissection is futile. Grafts similar in diameter and sectional topography are sutured to this type of nerve. If the nerve diameter exceeds the one of the graft, a longitudinal dissection of the graft is performed and only a part of the graft is used.

There are several types of nerve grafts: free transferred, pedicled, cable grafts, trunk grafts, fascicle and interfascicle grafts.

Free transferred grafts don't contain any vascular source, the inosculation process taking place 3-4 days after the nerve suture. These first days, the nerve is vascularized through diffusion. The large caliber grafts may suffer from ischemia, existing a risk of central necrosis.

The pedicle graft is used when there are simultaneous injuries to 2 nerves in proximity one to another (median and ulnar nerve). The proximal ends of the 2 nerves are sutured, while the ulnar nerve is sectioned as proximal as possible. The ulnar nerve however isn't deprived of it's vascular supply. After neurotization, the ulnar nerve graft is transposed inferiorly to restore the trajectory of the median nerve. At this time, the ulnar graft is sutured at the distal end of the median nerve.

The free vascularized nerve graft represents the transfer of the nervous tissue alongside his vascular supply. This technique can be used when the receiving site is not favourable for regeneration (high degree of fibrosis). This operation is reserved for extremities or fingers because the intervention can be quite laborious.



The interfascicle graft is used for groups of fascicle cooptation. It implies excision of the external epinerve at the proximal end, dissection on fascicle groups, each group being individualized at different distances to increase suture resistance. Same technique is used in the distal end of the graft. The individual fascicle graft is used for the repair of terminal nerves (digital nerves or the ulnar nerve at the elbow region which contains a small number of fibres.

The cable nerve graft is formed by harvesting several cutaneous small nerves, which are placed parallel to cover a surface equal to the diameter of the injured nerve. This was replaced by the interfascicle nerve graft because the cable nerve grafts involve putting larger amounts of connective tissue in contact with nervous tissue, thus inhibiting the nervous regeneration. The trunk graft is no

longer in use because of its large diameter that caused multiple central necrosis.

Conclusions

This review aims to present the most important aspects of peripheral nerve regeneration, being a foundation stone for a very complex subject. Having described the underlying processes involved in peripheral nerve degeneration, as well as the classical repair techniques, it is obvious that progress in this field has been done. However, the technological progress as well as the development of new molecules that can aid nerve regeneration show that there is always room for improvement in the future.

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Surgical management of middle cerebral artery aneurysms

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ABSTRACT

Background. The middle cerebral artery (MCA) harbors approximately 14% to 30% of all ruptured cerebral aneurysms. They can occur at multiple sites throughout the course of the middle cerebral artery, but most often are found at the bifurcation of the first segment (M1).

Methods. A retrospective review of 116 consecutive patients with an MCA aneurysm treated by surgical clipping, by two senior neurosurgeons, was performed. The data of all our consecutive patients were searched to obtain patient characteristics, details of the aneurysm size and orientation, treatment details, complications and follow up. At admission, the clinical condition of all patients was classified according to the Hunt and Hess scale. Clinical outcome was graded according to the modified Rankin scale. The follow-up period varied widely from 2 to 72 months (mean 30 months).

Results. Surgical clipping was performed for 113 ruptured MCA aneurysms; only in 3 cases the aneurysm was unruptured. Fourteen patients presented with significant hematoma which required the evacuation of the clot. Post-operative control angiography was performed in 32 patients (27.5%), from which we reported a full occlusion of the aneurysm in 32 patients (93.75%). Perioperative mortality was 5.2% (6 patients), due to neurological (4 patients) or systemic causes (2 patients). The outcome was graded mRankin 0–2 in 72.5% of the cases (84 patients) at the end of the first postoperative months, and 78.5% (91 patients) at six months follow-up. The most important improvement was recorded for patients graded mRankin 1-2 at the first month follow-up. All 3 patients with a surgically treated asymptomatic MCA aneurysm had an excellent outcome (mRS 0) at both follow-up, 1 months and respectively 6 months.

Conclusions. For experienced neurovascular team, MCA aneurysms currently make microsurgical treatment the preferred treatment modality for most MCA aneurysms.

Keywords
MCA aneurysm, surgical clipping, endovascular treatment, postoperative results

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INTRODUCTION

The middle cerebral artery (MCA) originates at the internal carotid artery (ICA) bifurcation and courses laterally within the sylvian cistern. MCA aneurysms are common, representing approximately 14% to 20% of all intracranial aneurysms (13,24). They can occur at multiple sites throughout the course of the middle cerebral artery, but most often are found at the bifurcation of the first segment (M1) and projects laterally

in the plane of the M1 segment (31). Because they project into the adjacent brain parenchyma, MCA ruptured aneurysms are more likely to present with intraparenchymal rather than subarachnoid haemorrhages. Thus, intracerebral haemorrhage rate is 43% versus 11% for aneurysms in other locations (13,24).

METHODS

A retrospective review of our neurovascular database was performed for patient diagnosed and surgically treated for MCA aneurysm in the period from January 2012 until May 2018, by two senior neurosurgeons. In this period, 116 consecutive patients with an MCA aneurysm were treated by surgical clipping, from a total of 384 patients operated for cerebral anterior circulation aneurysms. From this series of 116 patients, 26 patients harboured multiple intracranial aneurysms and at least one MCA aneurysm. Of our patients with multiple intracranial aneurysms, 12 had bilateral MCA aneurysms (mirror aneurysms).

TABLE 1 - Distribution of carotid system aneurysms in a series of 116 patients

Distribution of carotid system aneurysms	
Aneurysm location	*Number of patients*
Anterior communicating artery aneurysm	168 (44%)
Posterior communicating artery aneurysm	75 (19.5%)
Right medial cerebral artery aneurysm	65 (17%)
Left medial cerebral artery aneurysm	51 (13%)
Anterior cerebral artery aneurysm	4 (1%)
Internal carotid artery aneurysm	4 (1%)
Anterior choroidal artery aneurysm	6 (1,5%)
Pericallosal artery aneurysm	5 (1.5%)
Ophthalmic artery aneurysm	6 (1.5%)
Total	384 (100%)

After clinical assessment, each patient performed cerebral computed tomography (CT) scan and conventional cerebral angiography with digital subtraction. If treatment options, clipping or coiling, were considered equal, in MCA aneurysm surgery had priority.

The data of all our consecutive patients, surgically treated for MCA aneurysms, were searched to obtain patient characteristics, details of the aneurysm size and orientation, treatment details, complications and follow up. At admission, the clinical condition of all patients was classified according to the Hunt and Hess scale. Clinical outcome was graded according to the modified Rankin scale.

Only 3 patients with an unruptured MCA aneurysm (Hunt&Hess 0) were included in this series. Most of the patients were diagnosed as a cause of haemorrhagic stroke, subarachnoid haemorrhage or intracerebral hematoma. Patients with a ruptured aneurysm of a good grade (Hunt&Hess 1-3) were, as a rule, treated within 48-72 h after the diagnosis of the aneurysm. In a minority of symptomatic patients, the treatment of the ruptured aneurysm was postponed because of co-morbidity, late referral or poor clinical condition of the patient (Hunt&Hess 4-5). In these cases, acute hydrocephalus or clinical vasospasm, were treated before making the final decision about the timing of aneurysm clipping. In case of multiple aneurysms, treatment was at first selectively aimed at the ruptured aneurysm; additional unruptured aneurysms were not treated in the same session, unless in the same surgical field.

RESULTS

The patient characteristics are presented in Table 2.

TABLE 2 - Characteristics of patient with MCA aneurysms in our series

Characteristic	No of patients (%)
Male	42 (36.2%)
Female	74 (63.8%)
Hunt &Hess scale	
Grade 0	5 (4.2%)

Grade 1 and 2	81 (70%)
Grade 3	20 (17.3%)
Grade 4	8 (6,8%)
Grade 5	2 (1.7%)
Modified Fischer Grading Scale	
No SAH present	5 (4.3%)
Focal or diffuse thin SAH	62 (53.5%)
Focal or diffuse thick SAH	42 (36.2%)
Intraventricular haemorrhage	7 (6%)

There were 116 patients with MCA aneurysms operated in our series. Most of the patients were women (63.8%). At the time of surgery, 70% of the patients were in grade 1 or 2 on Hunt&Hess scale.

The aneurysm characteristics are summarized in Table 3. Most MCA aneurysms were larger than 5 mm (70%).

TABLE 3 - Aneurysms characteristics

Characteristics	No of patients (%)
Left MCA	51 (44%)
Right MCA	65 (56%)
Location	
MCA bifurcation	109 (94%)
M1 (pre-bifurcation segment)	5 (4.3%)
M2/M3 (post-bifurcation)	2 (1.7%)
Size	
≤ 5 mm	37 (31.9%)
5-10 mm	67 (57.75%)
≥ 10 mm	12 (10.35%)

Surgical clipping of an MCA aneurysm was performed following a subarachnoid haemorrhage in most of the patients, only in 3 cases the aneurysm was unruptured. The majority of the patients, 83 representing 71.5%, was treated within 96 h following the haemorrhage. Five patients had postponed clipping of ruptured aneurysm, due to re-bleeding, important medical co-morbidities and acute hydrocephalus that needed external drainage. Seven patients harboured a coincidental non-ruptured posterior communicating artery aneurysm (3 cases) and anterior communicating artery aneurysm (4 cases), which were clipped in the same surgical procedure. Fourteen patients presented with significant hematoma which required the evacuation of the clot. Post-operative control angiography was performed in 32 patients (27.5%), from which we reported a full occlusion of the aneurysm in 32 patients (93.75%).

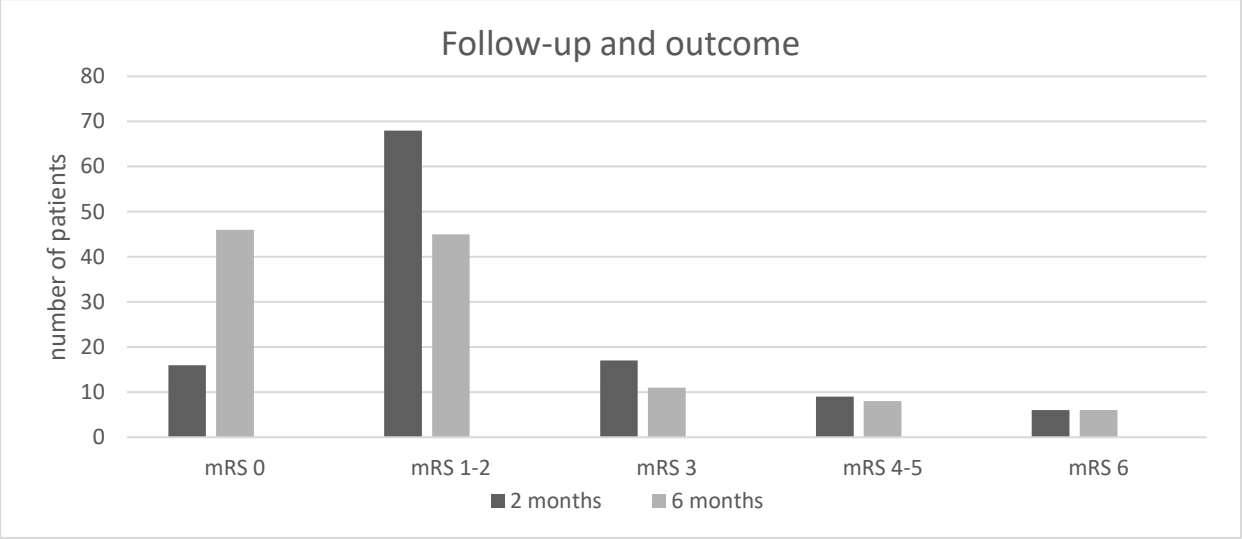
TABLE 4- Procedural and perioperative complication

Complications		No of cases
Intraoperative rupture		18 (15.5%)
Re-ruptured before surgery (waiting)		1 (0.85%)
Post-operative rupture		1 (0.85%)
Pre-operative vasospasm	angiography	14 (12%)
	clinic	6 (5%)
Post-operative vasospasm	angiography	15/32 (46.85%)
	clinic	23 (20%)
Post-operative subdural hematoma		2 (1.7%)
Meningitis		3 (2.5%)
Hydrocephalus		8 (7%)

VP shunt (within 30 days from surgery)	4 (3.5%)
Epilepsy	2 (1.7%)

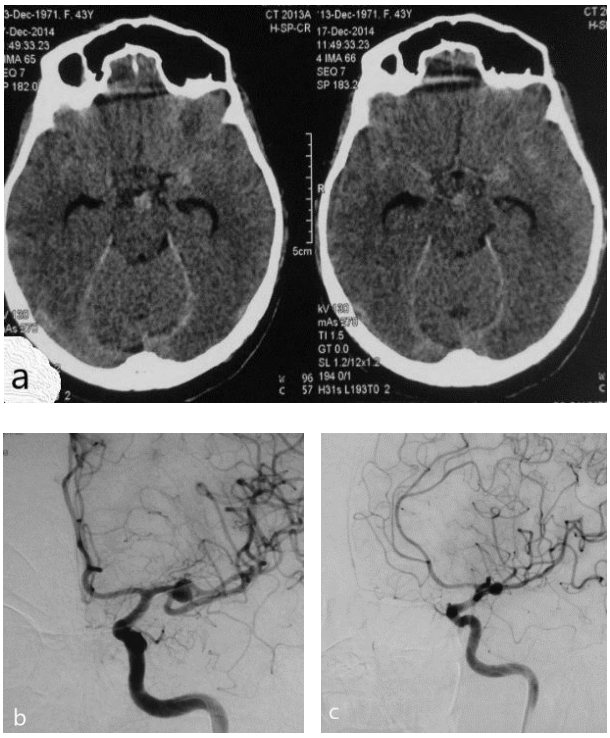
Preoperative vasospasm was demonstrated on cerebral angiography in 14 patients (12%) and clinically was manifested in 6 patients (5%). As postoperative event, 23 patients, including the ones with preoperative neurological signs, presented clinical symptoms of vasospasm including insidious onset of a decreasing level of consciousness, affected speech and/or motor deficits. On control angiography, cerebral vasospasm was detected on near half of the patients (46.85%).

Preoperative subarachnoid rebleeding, while waiting surgery, occurred in one patient. Postoperative rebleeding of a clipped MCA aneurysm occurred once, leading to a poor outcome.



Outcome of the surgically treated MCA aneurysms after 2 months and after 6 months follow-up. Excellent and good=mRankin 0–2, fair= mRankin 3, poor=mRankin 4-5, death=mRankin 6

The follow-up period varied widely from 2 to 72 months (mean 30 months). Best results were obtained in patients who preoperatively were included in 1st and 2nd grade of Hunt & Hess scale. Follow-up of the surgically treated symptomatic patients at 2 months was complete for all but 6 patients who died of neurologic or systemic causes. Perioperative mortality was 5.2% (6 patients), due to neurological (delayed cerebral ischemia-3 patients and meningitis–1 patient) or systemic causes (pulmonary embolism -2 patients). The mean follow-up was 30 months, obtained in 94.8% of the patients. The outcome was graded mRankin 0–2 in 72.5% of the cases (84 patients) at the end of the first postoperative months, and 78.5% (91 patients) at six months follow-up. The most important improvement was recorded for patients graded mRankin 1-2 at the first month follow-up. All 3 patients with a surgically treated asymptomatic MCA aneurysm had an excellent outcome (mRS 0) at both follow-up, 1 months and respectively 6 months.



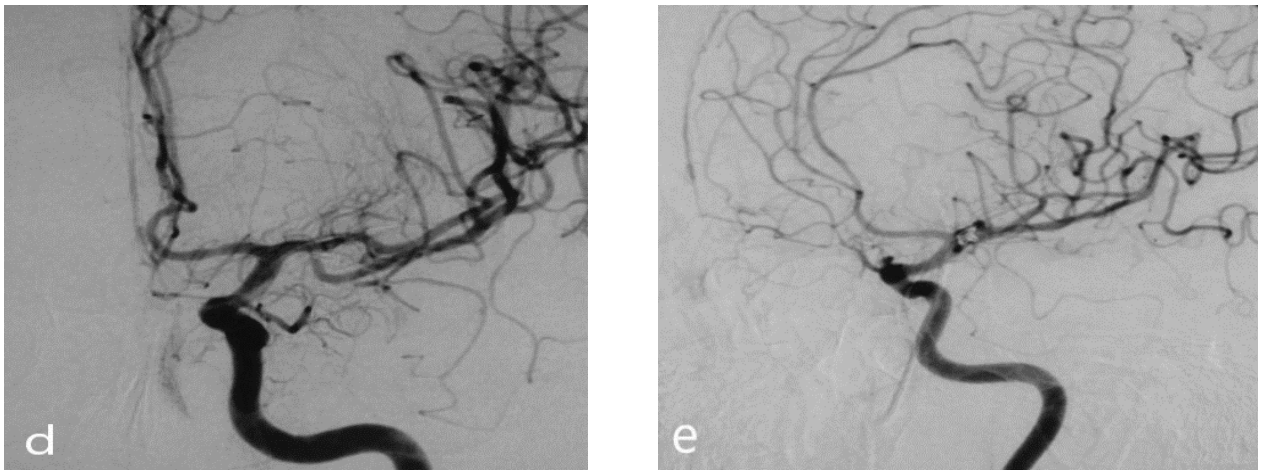


FIGURE LEGEND: (a) Preoperative native cerebral computed tomography (CT) showing subarachnoid haemorrhage, mainly in the left sylvian fissure; (b,c) Preoperative four vessels cerebral angiography showed a 8 mm length left middle cerebral artery bifurcation aneurysm; (d,e) Postoperative control cerebral angiography showed the correct clipping of the aneurysm and the preservation of both M2 branches.

DISCUSSIONS

The middle cerebral artery (MCA) is the largest of the cerebral arteries. The diameter of the MCA at its origin is about 4 mm (range, 2.4–4.6 mm). The MCA starts as the larger and the more direct branch at the internal carotid artery bifurcation and courses laterally below the anterior perforated substance within the sylvian fissure, where it gives rise to the lateral lenticulostriate arteries and cortical branches and, then, divides into its main trunks (23,28). The MCA is classically subdivided into four segments: the sphenoidal (M1) segment extending from the internal carotid artery bifurcation to the main MCA bifurcation where insular trunks (M2) begin and course over the insula until the peri-insular sulcus, where the opercular (M3) segments start and continue until the lateral surface of the brain in the sylvian fissure and, then, continue as cortical (M4) segments (23,31).

The middle cerebral artery (MCA) harbors approximately 30% of all ruptured cerebral aneurysms and 36% of all unruptured cerebral aneurysms (7,10). MCA aneurysms are classically divided into three groups: proximal middle cerebral artery aneurysms (M1 aneurysms), main middle cerebral artery bifurcation aneurysms (bifurcation aneurysms) or distal middle cerebral artery aneurysms (M3 or M4 aneurysms) (7,15). Most of them are located at the MCA bifurcation and often project laterally in the plane of the M1 segment (11). Each location raises different surgical considerations, but the anatomic features of MCA aneurysms render them amenable to microsurgical clipping as the primary treatment of choice for most patients. In addition, MCA aneurysms are often broad necked and incorporate branches at the neck, which challenges endovascular coiling more than surgical clipping (15,21).

Around 40% of patients with MCA aneurysms have multiple aneurysms, in contradistinction to approximately 20% of those with aneurysms in other locations (25). Moreover, mirror aneurysms can be seen in 13% of patients with MCA aneurysms (25,29).

Microsurgical clipping of MCA aneurysms is still the preferred treatment modality in most centers due to the relatively straightforward surgical approach to these relatively superficial aneurysms (2,11). Surgery provides effective and durable exclusion of MCA aneurysms, and, also, allows the reduction of intracranial pressure through evacuation of related intracerebral hematoma. The different locations of MCA aneurysms pose distinct challenges to the neurosurgeon and require specific surgical strategies.

Proximal MCA aneurysms (M1 aneurysms) are especially challenging lesions because of their intimate relation to lateral lenticulostriate artery. These perforators arteries may arise from the M1 segment or from the aneurysm neck itself. These branches are quite easily damaged during dissection, included with the aneurysm during clipping or compressed by the clip with subsequent thrombosis (2,12). Lateral lenticulostriate arteries irrigate eloquent areas of the brain and, thus,

damage to them could result in a poor clinical outcome.

The M1 bifurcation is the most common location for aneurysms in the MCA. In a large series, 60-83% of middle cerebral artery aneurysms are located at the bifurcation (24,31). The bifurcation of the MCA typically lies medial to the junction of the horizontal and anterior portions of the Sylvian fissure near the anterior edge of the island of Reil or limen insulae. Clipping of MCA bifurcation aneurysm must be precise with careful attention paid to the origins of the M2 branches. After M1 is exposed the dissection proceeds distally along the anterior and inferior aspects of M1 to reach the aneurysm neck at the MCA bifurcation (9). Anteriorly pointing aneurysms allow M1 exposure along their posterior pole, and, posteriorly pointing aneurysms allow M1 exposure along their anterior pole. Some mobilization of the M2 trunks may be necessary for creating enough space to place a temporary clip on M1 (8,16).

Temporary clipping is very important, because it softens the aneurysm sac during difficult and risky dissection maneuvers and is particularly helpful for isolating branching and perforating arteries adherent to the dome. Anterior temporal artery and both M2 branches should be completely dissected free, before clip application. The aneurysm neck should usually be clipped along the plane of the M2 branches and perpendicular to the M1 (14). After clip application, the tips of the blades should be inspected to ensure the safety of all branching and perforating vessels. Care must be taken not to kink the frontal or temporal M2s. "Perfect" clip application is dangerous and often leads to stenosis of the M2 outlets (14,16).

Multilobed aneurysms often require more than one clip and more complex clip reconstruction. They often also have calcified or atheromatous walls, all this requiring higher closing pressure or grater occlusion surfaces for full obliteration. In these situations, tandem- clipping techniques applying fenestrated clips with higher closing pressure to the distal neck supplemented by proximal neck occlusion with non-fenestrated clips are useful (6,14). Sometimes, in large ruptured MCA aneurysms, temporary clipping of the dome will provide sufficient control of the bleeding site to facilitate final dissection of the neck (6,17).

The main challenge during **surgery for distal MCA aneurysm** is to localize it, particularly when they are small and distal to the M2–M3 junction or when the Sylvian fissure is filled with subarachnoid hemorrhage or intracerebral hematoma (7). Localization of distal MCA aneurysms requires more experience and careful study of the preoperative angiography. The distance from the MCA genu, the location in relation to the associated ICH if present, the depth of the aneurysm from the surface of the brain are some of the data that can be obtained from CTA and which can help during surgery (2,7). Neuronavigation, intraoperative DSA or intraoperative ultrasound might also be considered.

Giant and fusiform aneurysms of the bifurcation may require bypasses techniques. When performing the craniotomy for a large MCA aneurysm, it is important to preserve at least one branch of the superficial temporal artery (STA) in case an unanticipated STA-MCA bypass is needed. High-flow extracranial-intracranial bypasses using saphenous vein or radial artery grafts may needed to be considered for fusiform aneurysms involving more than one branch (16).

The implementation of adjunctive tools including somatosensory evoked potential (SSEP) monitoring, indocyanine green (ICG) angiography or intraoperative digital subtraction angiography (DSA) improve surgical outcomes by providing critical information regarding optimal clip placement (5).

Efficacy of endovascular approach was evaluated in multicenter randomized control trials (RCT) as the International Subarachnoid Aneurysm Trial (ISAT) started in 2002 or the Barrow Ruptured Aneurysm Trial (BRAT) started in 2003 (18,19). In both trials, the early results at 1-year follow-up suggested superiority of coiling compared to clipping. But, long-term follow-up outcomes in both studies, evaluated at more than 3 years, demonstrated an attenuation of the benefit gain achieved by endovascular treatment to the point where no significant difference of neurological outcome was observed between the two treatment modalities (18,27). In the endovascular group, at long term follow-up, higher re-bleeding risk, lower obliteration rate and higher retreatment rate were noticed. But there were no specific randomized control trials to compare safety and efficacy of MCA aneurysm treatment between the two modalities. Regli et al. (22) described the first series of comparison between endovascular and surgical treatment in a single cohort; the surgical results were excellent with only 3% morbidity,

compared with a failure rate of 85% in coiling, with only two patients successfully treated by coiling with a "first coil" policy.

A most recent comparison study (2014) was conducted by Diaz and co-workers (9) but with the selection criteria bearing a moderate disadvantage towards surgery, where for all ruptured MCA aneurysm a "coil first" strategy was implemented and for unruptured MCA aneurysms only large ones were recommended to undergo clipping. The authors reported comparable results in poor outcome for coiling (10%) and clipping (5.9%) with a slight advantage toward surgery. They also observed a 14% recanalization of the aneurysm in endovascular group in comparison to none in the surgery group in a 9-months follow-up period (9). All these results were supported by evidence from systematic reviews and meta-analysis of the current literature that showed slight to moderate advantage of surgery over endovascular embolization in treatment of MCA aneurysms (1,30).

For ruptured MCA aneurysms, the outcome after endovascular treatment is reported to be good in 48–100% of cases and poor in up to 52% of cases with a mortality rate of up to 14% (3,4). The outcome after the surgical treatment of ruptured MCA aneurysms is recorded to be good in 55–95% of cases and poor in 5–45% with a mortality rate of up to 13% (20,26,27). For unruptured MCA aneurysms, the outcome after endovascular treatment is reported to be good in 93–99% of cases and poor in up to 5% of cases with a mortality rate of up to 3% (3,22). The outcome of surgical treatment is recorded to be good in 88–100% of cases and poor in 4–12% of cases with a mortality rate of up to 2% (2,8,26).

Conclusions

A good anatomic understanding of MCA branching patterns and sylvian fissure compartments, and experience with standard management strategies are required to effectively treat these, often, complex aneurysms. All attempts to treat MCA aneurysms endovascularly, despite ample progress in endovascular techniques and devices has been made, are unjustified in a situation where an excellent surgical solution is at hand. For experienced neurovascular team, MCA aneurysms currently make microsurgical treatment the preferred treatment modality for most MCA aneurysms.

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Neuromodulation devices nowadays

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Abstract

Introduction. Neuromodulation devices have known a great progress in the past years being used in treatment of drug resistant neurological diseases such as epilepsies and migraines. A neuromodulation device can stimulate profound or superficial neural pathways in order to balance chronic drug-resistant disorders that involve disturbances of cellular electrical potentials.

Material. Cranial neuromodulation devices implants used until now usually determined skull irregularities, implant site infection, resorption of the bone flap or osteomyelitis. In order to solve these problems, it was needed a customized cranial implant that integrates the neuromodulation device.

We report the first description of a fully integrated neuromodulation device within a customized cranial implant, publicised in 2018 by Gordon et al., that demonstrates the utility of a computerized neurostimulation device combined with clear custom-designed cranial implant.

Conclusion. The new approach of neurotechnology confines a better solution for neuroimplants devices with less follow-up complications and great patient's satisfaction.

Keywords
neuromodulation devices,
neurostimulation,
drug resistant epilepsy,
customized cranial implant

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Introduction

Neuromodulation devices stimulate profound or superficial neural pathways in order to balance chronic drug-resistant disorders that involve disturbances of cellular electrical potentials, such as epilepsies and migraines. Most of the cranial neuromodulation device implants used until now had adverse reactions like skull irregularities, implant site infection, resorption of the bone flap or osteomyelitis [1-5].

Material

We report the first description of a fully integrated neuromodulation device within a customized cranial implant, publicised in 2018 by Gordon et al., that demonstrates the utility of a computerized neurostimulation device combined with clear custom-designed cranial implant.

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Modern day treatment for drug resistant neurological diseases has known a great progress during past years. In 2018, Gordon et al. from Johns Hopkins Hospital, Maryland, USA, reported first in-human experience using a neuromodulation device within a cranial implant. The neuromodulation was used before in drug resistant epilepsies and migraines [1,2]. It is well known that one in three patients with focal seizures develops a drug resistant form of epilepsy. Patients with focal seizures who have failed multiple drug associated treatment that do not fit neurosurgical approach need vagus nerve stimulation or responsive neurostimulation [1,3,4,5].

The responsive neurostimulation system, approved by the FDA in 2013, is used for patients that have drug resistant epilepsy with less than 2 epileptogenic foci and it has a cortical stimulator that detects and respond to the electroencephalographic events [1,6]. Even though it has a great efficacy, there has been reported a lot of complications about the montage of the device, such as: scalp dehiscence, device exposure, contamination of hardware, infections, contour irregularities, bone flap osteomyelitis, visual deformities that affect quality of patient's life [1,7,8].

In order to solve all these problems and to reduce the number of re-interventions that increases the risk of infections, Gordon et al. started to plan a new improved implant design. They changed the opaque material used before with a transparent one for a better visibility and accuracy of the positioning to avoid the electrocorticographic signal interference. All the components of the device were integrated below the implant as an incorporated piece, fact that prevents the obvious deformities of the skull and scalp and the migration of the device into the scalp [1, 9-11].

Discussion

Gordon et al. used a 54 years old drug resistant epilepsy patient who needed responsive neurostimulation device with cranioplasty. A multidisciplinary team formed by neurosurgeons and plastic surgeons produced a perfect size 3D printed mold of the patient's skull that was computer-laser modified with a 5-axis robot laser cutter and after that presterilised [1,12,13]. The responsive neurostimulation device with the leads were placed under the clear cranial implant making possible a perfect visualization of the connections and possible inadvertent device interference. The patient was fully recovered with no complications. This was the first description of a fully integrated neuromodulation device within a customized cranial implant [1,13,14].

The method allows the avoidance of visible irregularities, scalp dehiscence, device extrusion and lead migration identified as complications of standard procedures above skull. The technology of cranial implants is expected to develop and treat other brain pathologies such as tumours and movement disorders and maybe it can be implicated more in intelligent cognition devices for memory diseases [1].

Conclusion

We can make a conclusion that the new approach of neurotechnology confines a better solution for neuroimplants devices with less follow-up complications and great patient's satisfaction. It is a great demonstration that can inspire new research in other brain pathologies that can use computerized neurostimulation device combined with clear custom-designed cranial implant [1, 15, 16].

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New technics for removal of intradural spinal tumours

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Abstract

Introduction. Neuronavigation is a computer-assisted technology based on pre- and intraoperative images that permit neurosurgeons to have a better approach of the brain and intradural spinal tumors. The neuronavigation systems have been a significant progress in neurosurgery. These systems allow neurosurgeons to evaluate surgical risks, select the best interventional method, localize better the tumors in order to improve the accuracy of the resection and decide on the optimal trajectory for the surgical procedure, resulting in decreased patient morbidity and mortality.
Material: Spinal cord tumors are rare and uncommon lesions. Their growth result in compression of the spinal cord, which can cause severe neurologic deficits such as limb dysfunction, motor and sensation loss with the possibility of leading to death.
We present o short report of a study publicated by Stefini et al. in 2018 regarding the use of neuronavigation for removal of intradural spinal tumors.
Conclusion: The benefits of using neuronavigation in resection of the intradural spinal tumors include decreased risk of bad localization of the tumor, minimal invasive surgery technique and reduction of bone removal.

Keywords
neuronavigation,
intradural spinal tumours

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Introduction

Neuronavigation is a computer-assisted technology used by neurosurgeons in resection of the tumours to evaluate surgical risks, select the best interventional method, localize better the tumours in order to improve the accuracy of the resection and decide on the optimal trajectory for the surgical procedures. The technique is based on pre- and intraoperative images that permit neurosurgeons to have a better approach of the brain and intradural spinal tumours. The neuronavigation systems have been a significant progress in neurosurgery, their use resulted in decreased patient morbidity and mortality [1-4].

Material

The use of neuronavigation for the removal of spinal tumours has been described since 1990's, with good detailed notes in 2000 by Haberland

et al. [1,2]. Nowadays, spinal navigation is used to create 3D images in order to position the pedicle screws. A good microsurgical resection of spinal intradural tumours needs an accurate hemilaminectomy or laminectomy, that sometimes can be more excessive than necessary and leads to chronic pain and acquired deformities of the spine [3,4]. In 2018, Stefini et al. described the for first time the possibility to use the merging of the 3D fluoro images obtained intraoperative with spinal navigation with MRI preoperative images, in order to make a better approach of the spinal intradural or intramedullary tumours [3,5-7].

Stefini et al. published in 2018 a report regarding the use of neuronavigation for removal of intradural spinal tumours. They made a study between January and July 2016 that included 10 navigated procedures for intradural spinal tumours with the technique of merging 3D fluoro images obtained intraoperative with spinal navigation with MRI preoperative images. Initially, all the patients underwent contrast-enhanced MRI with volumetric acquisitions and after that there were obtained the 3D fluoro images with neuronavigation. All these images were merged automatically or manually, verified if the association was correctly done on the sagittal, coronal and axial planes and used to perform all the steps of the required procedure using the navigated probes. The studied patients had all completed the fusion procedures, there were no errors detected and the intervention lasted about 20 min for each person [3].

Discussion

Intraoperative navigation, considered an essential tool for cranial surgery, became in the last years an advantageous technique for spinal surgery in degenerative disc disease, spondylolisthesis, tumours and traumatic lesions. It reduces the neurological complications and system failure for the patient and the radiation exposure for the neurosurgical team [3,8]. The idea of merging the 3D fluoro images with the volumetric-enhanced MRI images made spinal navigation more accurate. This procedure resolved also problems like the centering of the tumours with a good indication of the precise level of the lesion, length of the skin incision, muscle strip and extent of bone removal, incidence of instability after the surgery, blood loss and postoperative pain [3,9].

The technique was found very useful in case of small and intramedullary tumours, especially for the thoracic lesions because it reduces the need of radiation. The limitation of the procedure is that this it was not studied extensively [3,10].

Conclusions

In our opinion this technique provides numerous advantages and it is simple to learn by the neurosurgeons. Intradural spinal tumours resection using neuronavigation is a minimal invasive technique that has numerous benefits including decreased risk of bad localization of the tumour, minimal invasive surgery technique and reduction of bone removal. The use of neuronavigation is in continuous progress of research that promises to be an excellent neurosurgery tool.

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The surgical procedure of syringomyelie with Chiari I in adults regarding the intrapial aspiration of cerebellar tonsils: does this procedure improve symptoms with less complication?

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ABSTRACT

Introduction. Cranio-vertebral decompression remains the common denominator for the treatment of syringomyelia associated with Chiari I. On the other hand, the details of the procedure, remains controversial. The success of the surgery is to restore the circulation of cerebro-spinal fluid at the level of the foramen magnum. How is this circulation restored to the level of foramen is the question? We offer our attitude towards the treatment of syringomyelia with Chiari I.

Material and method. Consecutive series of 32/121 patients benefiting from cranio-vertebral decompression associated with intrapial aspiration of cerebellar tonsils treated for syringomyelia with a Chiari I malformation in adults.

Result. Motor deficits were present in 20 / 22 patients, representing 90% of the entire patient group. these motor deficits are improved in 16 out of 22 cases, and remained unchanged in 06cas. no motor aggravation occurred. in our study, bone decompression and intra pial aspiration of cerebellar tonsils (sub arachnoids manipulation) were found to be associated with favourable results on clinical signs and symptoms. However, sub arachnoids manipulation and intra pial aspiration of cerebellar tonsils showed a little more complication compared with bone decompression with dural plasty.

Conclusion. The bone decompression with dural graft and intradural dissection of adhesions and reduction by intra pial aspiration or resection of the tonsils is indicated on the MRI aspect of cerebellar tonsils of considerable size totally obstructing the foramen Magnum, the intraoperative finding, through the arachnoid, of the absence of passage of the cerebro-spinal fluid because of the bulging of the cerebellar tonsils.

Keywords
syringomyelia,
Chiari I,
intra pial aspiration of cerebellar tonsils

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INTRODUCTION

The understanding of the pathophysiology of the development of syringomyelia with Chiari malformation formulated in many series [1,12,20,32,34,38,54,60,67,85]. explains the use today more and more of cranio-vertebral decompression technique is therefore the common denominator of syringomyelia treatment associated with Chiari I, on the other hand the details of the procedure, the technique of closure of the dura mater, and whether a drainage system should be placed or not in the syringomyelia cavity remains controversial. The maintenance of restored cerebrospinal fluid flow, however, is often problematic.

Many different surgical techniques are utilized to treat syringomyelia with Chiari type I malformation, and there is no consensus. Surgical technique may include bony decompression of the posterior fossa with or without duraplasty, arachnoid dissection, or shrinking of the cerebellar tonsils. The goal of any of these operations is to restore adequate cerebro spinal fluid CSF flow at the level of the foramen magnum and reconstruction of the Cisterna magna.

Is the reduction of the volume of the cerebellar tonsils by intra pial aspiration or resection of cerebellar tonsils or finally tonsilar shrinking associated with osteo dural decompresionis the best choice of traitment. we will discuss the results of our series of intra pial aspiration of cerebellar tonsils by comparing it with the different publications cited in the literature and propose our opinion.

Materials and Methods

Preoperative Population Characteristics

In our consecutive series of 32/121 patients with cranio-vertebral decompression associated with intrapial aspiration of the cerebellar tonsils or resection of the cerebellar tonsils treated for syringomyelia associated with a Tonsillar herniation (Chiari I malformation) in adults, 10 of them were however excluded from the study for lack of fully exploitable files. Our series therefore includes 22 patients.The mean age of the patients was 30 years. There were 13 female (59%) and 09 male (40%) patients.

Preoperative Clinical Symptoms

Signs and symptoms are summarized in Table I. Sensory disturbances were present in the upper limbs in 59%. Motor weakness was present together in the upper limbs and in the lower limbs in 90,9% of patients. Headaches were noted in 45,4%, lower cranial nerves palsy in 13,6% and Balance disorders or Gait ataxia in 08 cases.

Table I. Symptoms/Signs in 22 Patients

Symptom / Sign	Preoperative Status Syringomyelie+Chiaril (No. of patients)	Postoperative result (No. of patients)
Weakness & muscular atrophy	20	15
Sensory disturbance	15	13
Balance disorders, Gait problems or Gait ataxia	08	08
Pain headache	10	10
Cranial nerv dysfonction	03	03

The presence or absence of clinical signs was noted in all of these patients and motor disorders were assessed according to Mc Cormick's classification (Table II). All our patients were adults. The distribution by sex shows a female preponderance, which corresponds to what has been reported in some series [57,91,3,93,45,28 , 66,79 ,17, 51,49,14]. Motor disorders were the main symptom of the majority of patients in this series, followed by the sensory disturbances and headaches. We noted ataxia in 08 cases and cranial nerve deficits in 03 cases.

Table II. Mc Cormick's classification.

Functional grade	Clinical prerequisites
I	Intact neurologically, normal ambulation, minimal dysesthesia
II	Mild motor or sensory deficit, functional independence
III	Moderate deficit, limitation of function, independent w/external aid
IV	Severe motor or sensory deficit, limited function, dependent

Table III. Summary of Preoperative Status of motor disorders and Postoperative results Grade in 22 Patients I: motor disorders were assessed according to Mc Cormick's classification: Pré-opérative stading and after surgery.

Pré-opérative Status ChiariI+Syringomyelie (No. of patients)	Post opérative Status ChiariI+Syringomyelie (No. of patients)
09 cases Grade III	08 Grade II (improvement) 01 cases (unchanged)
08 cases Grade II	08 Grade I (improvement)
03 cases Grade IV	Unchanged

02 cases Grade I	Unchanged
22	Total

Preoperative Imaging

All patients underwent magnetic resonance imaging (MRI) studies including sagittal and axial spin-echo T1 and T2 sequences. We analyzed the size of the ventricles, studied the anatomy of the occipito-vertebral junction, namely the position of the tonsils, the posterior arch of C1, the height extension of the syringomyelia cavity. Preoperative MRI showed that the syrinx predominantly involved the cervical or the cervicothoracic spinal cord (28% and 50% of the patients respectively) and occupied from 30% to 80% of the spinal canal according to the Vaquero's index [81].

Septa were present in 30% of patients.All patients showed tonsillar herniation reaching the inferior rim of C1. None showed spinal dysraphism or basilar invagination

Surgical technique

Regarding the surgical technique, all patients of our series of 121 cases were operated in the ventral position which allows the easy visualization of the tonsils, since in the sitting position the tonsils are stretched down under the effect of the universal attraction so they will be less visible than ventral, and also the brainstem and cerebellum have a tendency to migrate downward in the sitting position. In this series, the craniocervical decompression procedure was chosen based on the different flow of the cerebro spinal fluid dynamics at the cervico vertebral junction. After a small craniectomy, opening the foramen magnum, removing the small lower part of the posterior fossa, and removing the posterior arch of C1. The extent of bone decompression is also important, if the craniectomy is too wide in height, the patient is at risk of ectopy of the cerebellum and recurrence of symptoms. Some authors suggest that the craniectomy should be wide [21] in addition to the craniectomy, Isu [47] is satisfied with the removal of the outer layer of the dura as a treatment for syringomyelia occurring with a Chiari I malformation for him is enough. and others, like us, prefer a small craniectomy in order to avoid the sagging of the cerebellum (cerebellar ptosis), which is sometimes responsible for the patient's death, as reported after a wide craniectomy [43,83,84]. Zhang et al. [92] retrospectively analysed 132 patients in a comparison of small and large craniectomy. As noted above, the success rate was 78.2% in the large craniotomy group, with nearly half of patients developing complications, the most common of which was cerebro spinal fluid fistula (11.5%), whereas in the small craniotomy group, the success rate was 82.5% with only three cases of complications. This study demonstrated the superiority of smaller craniotomies.

But according to Ellenbogen [29] a limited craniectomy can lead to inadequate decompression and persistence or recurrence of symptoms, I totally agree with Ellenbogen if we do not realize an intra pial aspiration of cerebellar tonsils. In 2003, Milhorat and Bolognese reported a good result in their experience in tailoring the craniectomy based on the extent of the compressed subarachnoid space on pre-operative magnetic resonance imaging.

We advocate a suboccipital craniectomy limited to enlarging the occipital foramen and allowing both decompression and maintenance of the cerebellum in the posterior fossa. For some authors occipito-vertebral decompression alone is enough Di Lorenzo N [26]. then the opening of the dura with keeping the arachnoid intact and an intraoperative exploration that will show if there is a good passage of the cerebro spinal fluid by visualization or observation of the bulge of the arachnoid or not (Figure1). If there is a bulge of the arachnoid it means that there is a good passage of the cerebro spinal fluid so this procedure is sufficient. according to this finding the decision of the realization of the intrapial aspiration of the cerebellar tonsils will be taken. all of the 21 patients in our series have benefited from the intrapial aspiration of the cerebellar tonsils, the extent of intra pial aspiration or resection was ended when the tonsils were reduced and Magendie foramen with the obex of the fourth ventricle are seen.

So even in our therapeutic attitude the decision to use either bone decompression or sub arachnoid manipulation was routinely made intraoperatively, before the dural opening was made. at last duraplasty was also done to all patients using fascia lata graft (Figure2).

During the whole procedure, careful hemostasia isperformed with bipolar forceps under saline irrigationto keep the field bloodless, to prevent adhesions that canbe induced around the spilled blood.

Results

Immediate postoperative result and Complications

In our series no mortality was seen, this are similar to those reported by [7,5,16,93] who reported no mortality in their series. But we noted, some cases of postoperative complications, such as cerebro spinal fluid leakage (04/22, 18.1%) who were treated with the use of repeated lumbar puncture with complete closure of the cerebro spinal fluid leakage, three patients 13.6% have intracranial infection who were treated medically. We have noted, hiccups in one case, who was traited only with the use of a synthetic drug as a tranquilizer, sedative, and antiemetic (chlorpromazine). The most serious complication was aseptic meningitis (02/22, 9.09%) with cerebro spinal fluid leakage (04/22, 18.1%).

Based on the results of this series, headache responds very well to decompression surgery with intra pial aspiration of cerebellar tonsils, on 10 of 22 patients with preoperative headache experienced relief complete immediately after surgery. Motor deficits were present in 20 / 22 patients, representing 90,9% of the entire patiens. No motor and sensory aggravation occurred. but the follow-up of the patients shows us that the motor improvement occurs slowly. Other symptoms, such as ataxia in 08 cases and cranial nerve deficits in03 cases, are improved in imediat postoperatively in our series, similar with findings reported by other authors. [14,17,30,33,37,16] series, which adopts the same surgery technique of craniovertebral decompression with tonsillectomy used in this study.

MRI Follow-up

On the neuro-radiological level, the MRI was performed successively at one month, six months and 12 months then every 02 years, found a normalization of the position of the cerebellar tonsils in all cases with a reconstruction of a new large cisterna magna. The syrinx cavity was clearly or markedly regressed in 09 case, and Slightly regressed in 13cas.

Syrinx cavity clearly regressed in 09 caseFig2 Figure 3. Preoperative MRI scan reveals syrinx cavity with tonsillar herniation extending below the foramen magnum (ChiariI). Post op Postoperative MRI scan reveals the regression of the syrinx cavity and absence of cerebellar tonsil herniation. (A) Slightly regressed in 13cas Fig3:

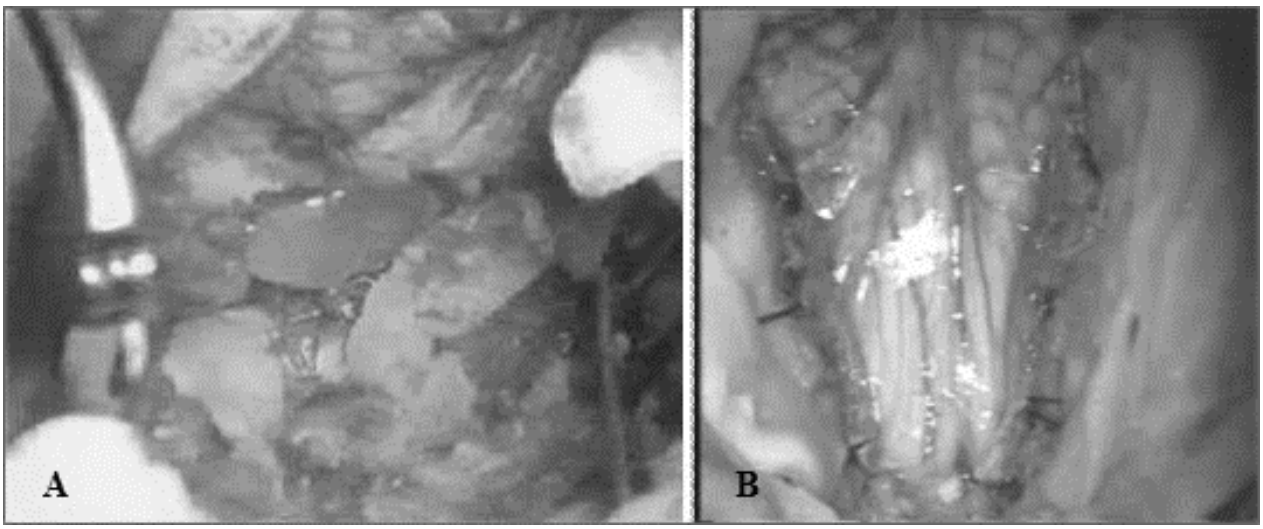

Figure 1. Preoperative picture of Dural opening with preservation of intact arachnoid
(A) showed a bulge of the arachnoid which confirms the good passage of the cerebro spinal fluid
(B) shows the large tonsils that occupy a large space and prevent the normal passage of the cerebro spinal fluid

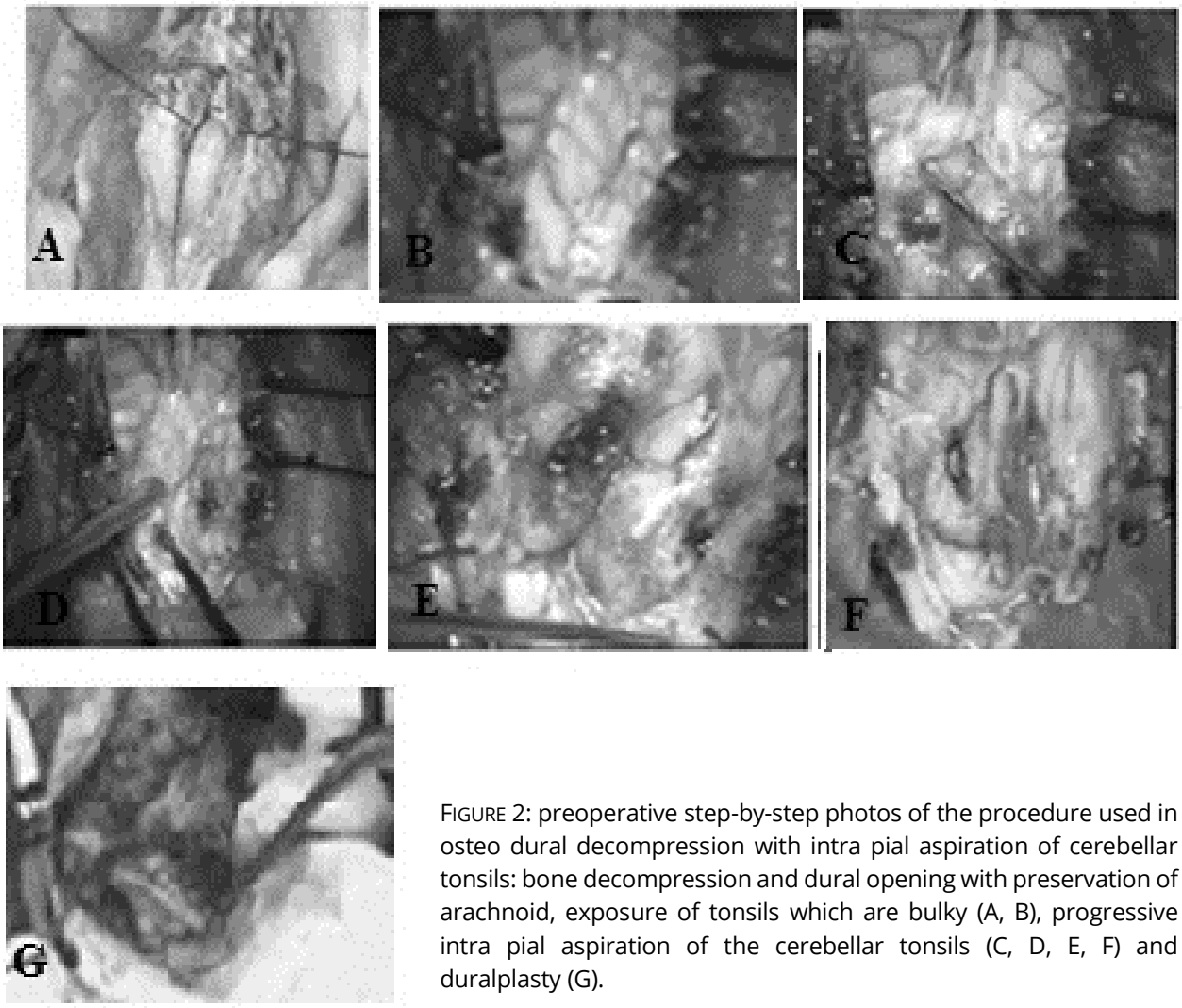

FIGURE 2: preoperative step-by-step photos of the procedure used in osteo dural decompression with intra pial aspiration of cerebellar tonsils: bone decompression and dural opening with preservation of arachnoid, exposure of tonsils which are bulky (A, B), progressive intra pial aspiration of the cerebellar tonsils (C, D, E, F) and duralplasty (G).

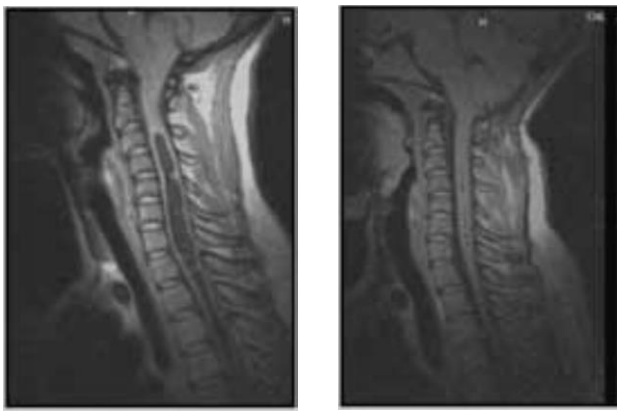

FIGURE 3.
Pre-operative (A) syringomyelia cavity with Chiari I
Post-operative(B) clearly regression of the syringomyelia cavity

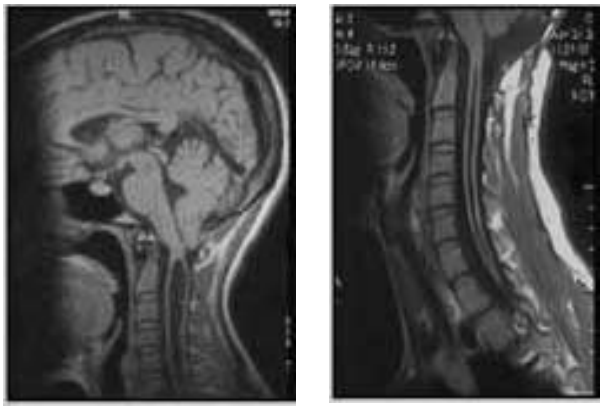

FIGURE 4.
Syringomyelia cavity pre op with Chiari I (A) Slight postoperative regression of the syringomyelic cavity (B)

The reoperation was not performed in our series because the result of long-term follow-up of our patients showed an improvement especially on the motor level. among patients who improved 15.79% remained stable in their initial improvement with an average follow-up over 8 years. These are similar to those in the series of reports on the best prognosis of the literature summarized and reported by Nozar Aghakhani [2].

Literature review

A literature review was performed with the PubMed search engine of the National Library of Medicine of the National Institutes of Health (http://www.ncbi.nlm.nih.gov/pubmed) and on the research gate site, using the following Keywords "Chiari malformation," "Chiari malformation type I with syringomyelia,"," "posterior fossa decompression with or without duraplasty," and "Craniometrical decompression with duraplasty and cerebellar tonsillectomy". The search was restricted to English-language publications without date limitations.

DISCUSSION

First, we will say what are the circumstances that led us to opt for this method.

Secondly, we analyse the various publications through the literature, is this method seen our retreat is effective or not, based on the following parameters: the immediate postoperative and the long-term results.

Third is that this method can be proposed in some patients.

To discuss the first issue, as you know that the key point of surgery in syringomyelia with Chiari imalformation is to allow a flow of cerebrospinal fluid at the level of Magendie foramen and foramen magnum. For our part, we have opted for the 22 patients a osteo-dural decompression associated with intra pial aspiration of the cerebellar tonsils for several reasons:

- when the opening of the dura with maintenance of the arachnoid intact and an intraoperative exploration will show us intraoperatively, through the arachnoid, the absence of passage of cerebrospinal fluid due to the large volume of the cerebellar tonsils. As there is no bulge of the arachnoid, this means that there is not a good passage of cerebrospinal fluid in sufficient quantity.
- it has not been demonstrated that the resection of the cerebellar tonsils does not lead to neurological disorders. the function of which is not yet clearly determined.
- Visualization or appearance of large cerebellar tonsils and considerable size completely obstructing the foramen Magnum causes neurological disorders.depending on the presence of these 03 parameters, the decision to perform intrapital aspiration of the cerebellar tonsils will be taken.

In our experience, the intra pial aspiration or resection of the tonsils does not lead to a noticeable neurological deficit. according to Asgari S and al [9] during removal of the cerebellar tonsil, the integrity of the pia mater of the cerebellar tonsil should be preserved to reduce postoperative adhesions, causes aggravation or reappearance of neurological disorders. we take into account the analysis of the literature publication and the study of Arnautovic A [6], and Aghakhani N [2], Royo Salvador [71] to evaluate of surgical treatment options in the ChiariI Malformation Type I with syringomyelia and concerning the occurrence of complications during the realization or not of the intrapial aspiration of the cerebellar tonsils, we found that the debate still exists as to whether, once the dura mater is open, arachnoid dissection should be performed or not and if the cerebellar tonsils are resected or retracted or not?. for some neurosurgeons the manipulation of the tonsils can lead in the long term to arachnoid adhesions between the cerebellar tonsils and the dura mater which prevents or hinders the subsequent flow of the cerebro spinal fluid at the foramen Magnum, with worsening of syringomyelia and symptoms according to Asgari S [9]. to avoid this postoperative arachnoiditis Sindou et al [76] and others authors such Hyun Seok L [44], Lee HS [56], Oldfield EH [67], Perrini .P[69] advocated or suggested opening of the dura with preservation of the arachnoid membrane and adding duraplasty, and reported favourable results. while in our series on an average of 8 years of follow up , we did not have this type of problem since we did not have any post-operative neurological aggravation in medium and long term (after minimum 1 year of follow-up) which eliminates the eventual arachnoiditis following intra pial aspiration of cerebellar tonsils which is confirmed in the study of Junpeng Ma1[49]and Zuev A.A[93] that the Cerebellar tonsillectomy with

suboccipital decompression and duraplasty can provide long-time cure for most Chiaril malformation cases, and some authors reported, the good results with this procedure[55, 91]and they have good results. Williams also recommended to remove part of the tonsils to ensure that the pathways are maximally opened. Many papers have supported arachnoid opening and/or tonsillectomy [5,16,7,93]. However, a successful surgical outcome (100% normalization of hindbrain herniation) was reported without arachnoid opening by Heiss et al [39] what was mentioned also by Royo Salvador [71] in his article. Klekamp [52]and Lee HS [56] showed that combined arachnoid pathology was a strong risk factor for symptom recurrence, because any operation undertaken to improve a problem related to arachnoid scarring may create new arachnoid scars, the lesser the extent of dissection and the lesser the contamination of the surgical field with blood, the better the chance of achieving good long-term results

Alden [4] in his article explains that, tonsillar shrinkage or resection has been advocated "as a way to improve the volume mismatch and to increase communication between the fourth ventricle and the spinal compartment". Fisher [30] recommends subpial resection of the cerebellar tonsils, While Batzdorf U [16] and others many neurosurgeons recommend tonsil shrinkage by coagulation or other means. and other authors Gonçalves da Silva J, Arruda J AM [7], Galarza M [30], Raftopoulos C [70] have another way to achieve sub arachnoid manipulation, and claim that this technique improves decompression in the region of the foramen magnum. In his article José Alberto Gonçalves da Silva1and al [22], observed a Blockage of the foramen of Magendie in 55 (52.8%) patients, mainly caused by the presence of a dense membrane or adhesions between the cerebellar tonsils. In our opinion this is a valid argument for the exploration of the cerebellar tonsil and foramen of Magendie. On the other hand, some of the neurosurgeons [63,21, 39. leave them intact, performing especially the opening of the fourth ventricle. On the other side, Batzdorf [16] recommends lightly diathermying the pia mater over the surface of the tonsils. for Raftopoulos[70] it is enough just to open the arachnoid and then remove the adhesions.

Secondarily in the past, decompression treatment of the occipito cervical junction has been a dangerous operation, with a significant mortality rate [Di Lorenzo]. With the development of neurosurgery, occipito vertebral decompression has become a relatively low-risk procedure. In our series, there have been no cases of death, or serious infections requiring revision of the wound, Badie et al. [10] retrospectively evaluated 20 cases treated via decompression technique with duraplasty and tonsillectomy. The success rate was 85%, with no mortality. Complications were not described. The 04/22 cases of cerebro spinal fluid leakage in our series is slightly high but if we take all our series of 121 patients who have benefited from occipito cervical decompression with dural plasty, the leak rate of cerebro spinal fluid is similar to that reported in other series [28,35,40,46,48,49]. The incidences of CSF-related complications and hydrocephalus in studies about occipito cervical decompression with dural plasty were 4–10% and 3–4% respectively [79].

No appearance of a a pseudomeningocel in our series because we use a small suboccipital decompression, this makes it possible to avoid the appearance of a a pseudomeningocel. Recently, Jeffrey S et al [17], performing a small bone decompression report in his article on minimally invasive subpial tonsillectomy for Chiari I decompression significantly reduced the risk of pseudo-meningocele formation.

The incidence of Motor deficits and sensory disorder in our series is higher than in other series [8,10,68]. In our series, these motor deficits are improved in 16 out of 22 cases after functional rehabilitation, and remained unchanged in 06cases, we agree with Bălaşa. A, Gherasim.D.N[11] who belives that preoperatory longtime neurological deficit is a predictor of poorer outcome, making early surgery. this explains why in our series we had 06 patients who remained unchanged postoperatively and this is probably due to late diagnosis in our patients who only consult at the late stage.

Depreitere et al. [25] retrospectively analysed 22 cases operated used tonsillectomy. On initial follow-up, 16 patients (76%) were improved. In late follow-up, the success rate was 68%. Alfieri et al, reported that surgical decompression with durotomy, arachnoid opening, tonsillar shrinkage, and recreation of the cisterna magna was a safe and effective procedure. Prognosis was excellent, with

global clinical and radiological improvement in more than 90% and 80% of patients, respectively Stanko et al[77] suggested that tonsillar cautery might provide an extra benefit in the resolution of the syrinx compared with bone-only decompression alone or in combination with dural opening. Another previous study reported on 22 adult patients, with the conclusion that sub arachnoid manipilutaion may improve the symptoms in Chiari I malformation [30],In an older study, seven of eight patients who underwent sub arachnoid manipulation showed very good clinical outcomes [36,37] for some neurosurgeons sub arachnoid manipulation are generally accepted as the effective surgical procedures in the treatment of Chiari I malformation [67,70,72], and their effects have been confirmed by clinical practice.

In our series postoperative, MRI scan reveals clearly regression of the syrinx cavity in 09 case, and Slightly regression of the syrinx cavity in 13 cases, based on the Vaquero index and absence of cerebellar tonsil herniation in all cases. Wetjen [82] and coauthors have estimated the rate of syringomyelia resolution by examining the postoperative images in patients after ChiariI malformation decompression. Based on the largest anteroposterior diameter of the syringomyelia, the authors concluded that the median time to greater than 50% narrowing of the syringomyelia was 3.6 months postoperatively, whereas the mean time was 6.5 months. In a follow-up study by Wu et al. [86], syringomyelia was found to be obviously reduced in all patients examined and completely eliminated in eight patients. in the study of Bao and al [14], the re-examination by MRI in 44 patients showed successful construction of the cis¬terna magna, with eliminated or obviously reduced syringomyelia in 42 patients. According to de Lotbinière ACJ [58] The tonsillectomy or cerebellar tonsil exeresis proposed by some authors probably adds to the mortality and morbidity rates. It can be an unnecessary and maiming surgical manoeuvre. Alden [4] states that "no neurological deficit has been demonstrated as a result of tonsillar resection; however, the exact function of this structure is largely unknown we agree with Alden effectively, we have practiced it since 1999 and we have not had neurological disorders following their resection including cognitive disorders. taking into consideration that, the role of the cerebellum in learning and cognition began to be considered. In 1997 and 1998 Schmahmann and Sherman [75] named dysfunction of the cerebellums contribution to cognition and behavior the cerebellar cognitive affective syndrome. The posterior lobe of the cerebellum and the vermis contribute to verbal fluency, short-term memory, abstract reasoning, and spatial cognition, functions that may underlie the frequent complaint of "brain fog" in patients the Chiari I malformation. According to Royo Salvador MB [71] the tonsillectomy for syringomyelia with Chiari I,is not indicated, given that no benefit is obtained and it involves an amputation of a part of the cerebellum that can only contribute to sequelae, situations of permanent instability and vertigo.

In our study of clinical presentation in 32 patients with the Chiari I malformation with syringomyelia, we did not consider the psychological and or congenital state during the preoperative clinical examination.

Finally, and generally, the results of this procedure are favourable and differ little between the series. This was confirmed by the present study that the results were good, the complications limited in number .in 2005 we presented our comparative study of 40 patients between bone decompression with dural plasty (20 patients) and bone decompression with dural plasty followed by intrapial aspiration of cerebellar tonsils (20 patients) we found that the clinico-radiological results of bone decompression with dural plasty followed by intrapial aspiration of cerebellar tonsils are comparable and sometimes better than the bone decompression with dural plasty [18].

We believe that one of the advantages of arachnoid dissection, in the context of Syringomyelia with Chiari I malformation, is that it allows the surgeon to release the adhesions especially at the foramen of Magendie which could contribute to the obstruction of the cerebro spinal fluid flow from the fourth ventricle to the spinal canal. Whereas the subpial resection of the cerebellar tonsils, especially when they are bulky, we recommend it as a means of improving particularly the passage volume of the cerebro spinal fluid flow and increasing the communication between the fourth ventricle and the spinal cord.

Our experience that patients treated by tonsils resection had a good outcome with no increased operative risk and no additional surgery required surgical technique. I totally agree with Arruda and al []in his study Arruda conclude that craniocervical

decompression with tonsillectomy and duramater graft proved an effective method for the treatment of Syringomyelia and Chiari malformation.

Limitations

First, the present study was a retrospective analysis of 22 patients and the small number may have resulted in a lower statistical power. Second a prospective multicentre study with a large and equal number of patients in the occipito cervical decompression and occipito cervical decompression with intra pial aspiration of cerebellar tonsils or tonsillectomy groups might provide sufficient data for an adequate comparison of these 2 techniques to better define the indications and benefits.

CONCLUSIONS

For the treatment of symptomatic patients, various approaches may be used: bone decompression with dural graft, or bone decompression with dural graft and intradural dissection of adhesions and reduction by intrapial aspiration of the cerebellar tonsils or resection of tonsils because the key point of surgery in syringomyelia with ChiariI malformation is to allow a cerebro spinal fluid flow at the level of foramen of Magendie and foramen magnum. For our part, we have opted for a certain category of patients for osteo-dural decompression associated with subpial resection of the cerebellar tonsils for several reasons: -no neurological disturbance has been demonstrated to result from the intra pial aspiration resection of ectopic cerebellar tonsils,- the function of which is not yet clearly determined - the attribution of the neurological disorders, noted in the pre-surgical phase, to the malformation of Arnold Chiari, the MRI appearance of cerebellar tonsils of considerable size totally obstructing the foramen Magnum. -the intraoperative finding, through the arachnoid, of the absence of passage of the cerebro spinal fluid due to the bulging of the cerebellar tonsils. according to Beecher. J.S[17] the indications for subpial resection of the cerebellar tonsils surgery are the presence of one or more of the following criteria:

1. Karnofsky score of 70 or less secondary to Chiari malformation stereotypic constellation of symptoms
2. An expanding syringomyelia on consecutive MRI scans, syringomyelia cavities in excess of 75 % of the transverse cord diameter on the index MRI, or eccentric appearance of the syringomyelia cavity with intraparenchymal blebs
3. Severe, rapidly progressive neurological deficit

However, it should be acknowledged that this is an observational study and ultimately, we can say that the technique can be used in particular cases. It would be interesting to pay more attention to the effectiveness / inefficiency of the indication of intra pial aspiration of the cerebellar tonsils in the syringimyelie surgery with tonsils herniation (Chiari I malformation).

For these reasons, I believe long-term follow-up, with periodic measures, is required to really understand the success of surgery.

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Primary cerebellopontine angle glioblastoma in a child. A rare entity

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ABSTRACT

Cerebellopontine angle extraaxial glioblastoma (GBM) is extremely rare at any age but especially in children. We reported a case of 14-year-old girl, who presented with nausea, vomiting and ataxia. She was evaluated with computed tomography (CT) and magnetic resonance imaging (MRI). Imaging demonstrated irregular ring enhancing right CP angle mass. The atypical findings of irregular ring enhancement, CP angle location and presentation in childhood, combine to make the prospective diagnosis of GBM a difficult one. This combination of findings has been reported very rarely.

Keywords
glioblastoma,
paediatric,
CP angle

INTRODUCTION

Most lesions involving the cerebellopontine angle (CPA) are located extra-axially. The common differentials at this location include acoustic schwannomas, meningiomas, epidermoid tumours, metastases, and arachnoid cysts. Glioblastomas are usually intraxial and mostly in supratentorial compartments and usually in elderly age groups. GBM rarely present in cerebellopontine angle (CPA). Most of cases reported are primary intraaxial GBM arising from cerebellar hemisphere or brainstem, with exophytic extension into CPA. Pubmed advanced search only single of primary extraaxial GBM in the CPA has been reported, arising from the region of root entry zone of the eighth cranial nerve1. Regardless of the site of origin, tumours in the CPA represent with sign and symptoms resulting from compression of fifth, seventh, and eighth cranial nerves and pons and cerebellar peduncle. Because these lesions have different treatment modalities, prognosis, and outcome, so it is important to make differential diagnosis of these lesions. Appropriate diagnosis is important for the management of these lesion, as clinical manifestations of these are similar.

CASE REPORT

A 14-year-old girl, presented to Neurosurgery OPD with a several week histories of progressively increasing frontal headaches, projectile vomiting and excessive drowsiness and recent onset left sided weakness. Neurological examination revealed GCS-E3V2M6 with left

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sided hemiparesis and right sided 5, 6, 7th and lower cranial nerve palsies.

Computed tomography (CT) demonstrated right CP angle mass lesion of size 5x3.5 cm with effacement of fourth ventricle with obstructive hydrocephalous, with irregular ring enhancement. Magnetic resonance (MR) imaging demonstrated the lesion to be essentially isointense on T1 and hyperintense on T2 weighted sequence (Fig.1), and restricted diffusion on DWI. Post contrast T1 weighted images demonstrated irregular peripheral enhancement. There were areas of peripheral blooming on SWI in periphery of mass lesion in pons area (suggestive of hemorrhage). Patient underwent emergent ventriculoperitoneal shunt to relieve hydrocephalus. But patient condition did not improve and further deteriorated after VP shunt and emergency right retromastoid suboccipital craniectomy with tumour decompression was done. Intraoperatively the tumour mass was situated extraxially in cp angle, extending across 7, 8th cranial nerves complex to lower cranial nerves and medially extending to brainstem. Morphologically it was grayish pink, highly vascular, soft and CUSA amenable. Pathologic examination was conclusive for Glioblastoma Multiforme. Patient did not improve neurologically even after tumour decompression and finally expired 2 months after surgery. The diagnosis of GBM could be made after histopathological examination.

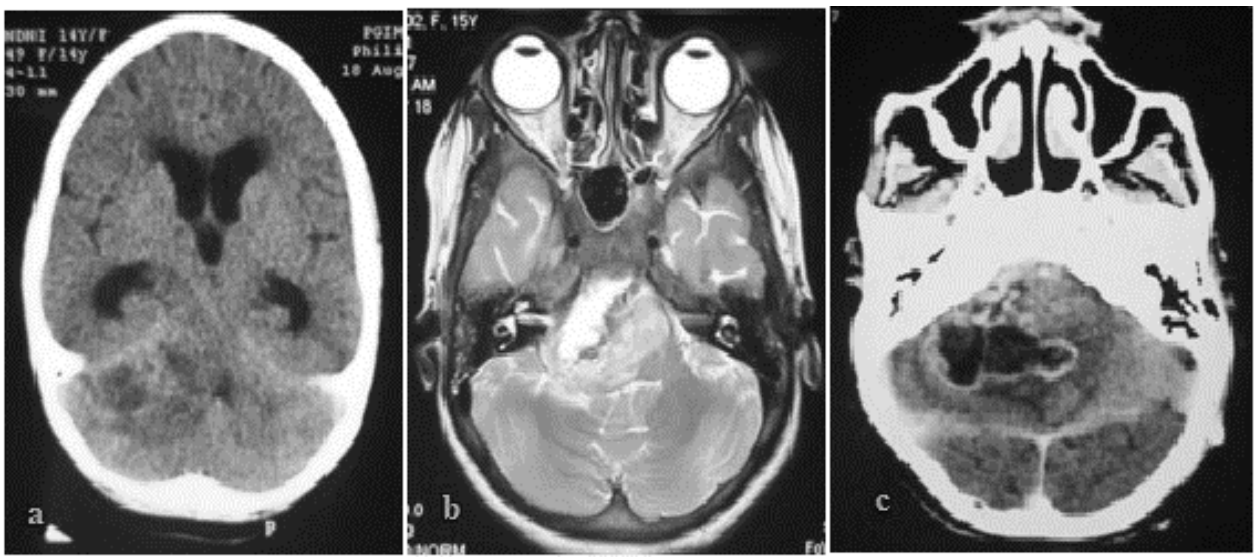

FIGURE 1. (a) Axial Noncontrast CT showing an iso to hypodense right CP angle mass lesion with obstructive hydrocephalous. (b) T2 weighted MRI image showing lesion heterogeneously hyperintense with a linear hypointensity. (c) Axial contrast CT image showing lesion enhancing heterogeneously with cystic component.

DISCUSSION

Most of the gliomas including GBM occur in the cerebral hemispheres, and posterior fossa glioma is uncommon, estimating as 1.5% in the cerebellum and 4.1% in the brainstem [2]. However primary GBM arising in the CPA is further rare with a few reported cases [1], [3]-[7] depending on the origin of the tumour primary CPA glioblastoma can be divided into two types. The first type of the CPA glioblastoma is an intraaxial tumour originated from brain stem or cerebellum with an exophytic growth into the CPA [4] –[6], [8]. This type of GBM is rare, and especially cerebellar GBM with the exophytic growth pattern is very rare, and up to date, four cases have been reported in the literature [3], [4], [8]. Two cases showed exophytic growth into CPA, and the other two cases were in the crural/quadrigeminal cistern and cisterna magna, respectively.

The second type is an extraaxial CPA glioblastoma. There has been one case of primary extraaxial GBM in the CPA, arising from the proximal portion of cranial nerve VIII [7]. Few possible mechanisms were documented regarding the origin of primary extraaxial GBM in the CPA . One is that the tumour arose from cranial nerve system tissue within proximal cranial nerve itself, and the other is that the tumour originated from heterotopic neuroglial cells in the leptomeninges covering the proximal cranial nerve or brainstem [1], [7].

In patients with cerebellopontine GBM the clinical features are similar to those of other aggressive fast growing infratentorial tumors. Signs and symptoms include headache, nausea, vomiting, and cerebellar dysfunction including ataxia, imbalance and unsteady gait, ipsilateral cranial nerves [9]-[11].

The radiological features of posterior fossa GBM are nonspecific [10]-[12]. Lesions occur laterally in the cerebellopontine angle. The lesions are typically infiltrating with indistinct margins. Signal characteristics are heterogenous, often with necrotic and cystic components. A thick and irregular wall is commonly seen. Irregular peripheral enhancement occurs following contrast administration. Edema is usually present and obstructive hydrocephalus is common.

Other features as histology and biology of cerebellar GBM are similar to that of cerebral GBM.11 This includes malignant tumour cells, mitoses, hypercellularity, pleomorphism and neoangiogenesis. The presence of necrosis helps differentiate GBM from anaplastic astrocytoma or from well-differentiated astrocytoma [9].

Cerebellar GBM has poor prognosis as with any GBM and any paediatric malignant brain tumour. This is attributed to rapid tumour progression, locally aggressive behaviour as well as the common findings of CSF pathway spread [12]. Early intervention has been advocated to increase the disease-free interval and to prolong survival includes, aggressive surgery as well as aggressive radiation and chemotherapy [9], [12]. Despite these measures; however, survival of children with CP angle GBM is very poor. However, the optimal management for the CPA glioblastoma is to be defined because of its rarity.

Conclusions

Primary glioblastoma arising in the CPA in paediatric age group although very rare and cannot always be differentiated from other usual benign tumours at this site radiologically, we should keep GBM in the differential diagnosis of an atypical lesion of the cerebellopontine angle, when patient presented with rapid clinical deterioration due to fast progression of the lesion.

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Ossification of the yellow ligament in thoracic spine: a case report

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ABSTRACT
Ossifications of yellow ligament (OYL) and calcification of yellow ligament are relatively rare clinical entities, and can make sever morbidity surgical evaluation can relived the sign and symptom and improve life quality we present a patient with ossification of yellow ligament in level of T9 and T10 of thoracic spin.

Keywords
yellow ligament,
thoracic spin,
canal stenosis

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CASE PRESENTATION
A 54 years old woman with chief complaint of back pain and lower limbs paraesthesia from 9 month ago, past medical history was negative and in drug history only she used pain relief agent like NSAIDs or acetaminophen in clinical evaluation upper motor neurons sign is positive (plantar reflex double extensor , deep tendon reflex in knee and achile reflex significantly increased +4) and no episode of bladder or anal dysfunction , in MRI of thoracic we found stenosis in level of T9-T 10 from posterior element compression ,extradural lesion which was isosignal in T1 hypo signal in T2 without enhancement with gadolinium(figure 1) , for future evaluation CT of vertebral column with 3d reconstruction show a tiny ossification in interlamina space which bulged to central canal (figure 2) ,patient admitted to operation room in prone position under general anaesthesia standard laminectomy was done bony yellow ligament was removed thecal sac was decompressed , 2 days after surgery patient discharged back pain and paraesthesia was improved after 3 month lower limbs muscles strong before and after surgery was 5/5 upper motors sign improve immediately a day after surgery

DISCUSSION
A Symptomatic OYL usually is located at the lower thoracic spine (38.5%) and the lumbar spine (26.5%) and is rare at the cervical spine (0.9%) (3). The detailed mechanism of OYL is unclear. There are several

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There are several reports that support the relationship between the growing factors of these ossification diseases and static and dynamic factors. (4,5) The pathophysiology of OYL is similar to that of YL hypertro¬phy. Degeneration of the YL is due to hyper mobility of the poste¬rior column, which results in collagen hyperplasia and hypertro¬phy of the YL (5). Subsequent deposition of calcium pyrophosphate dehydrates and calcium hydroxyapatite occurs in the ligament, resulting in OYL (6). The pathology of YL hypertrophy includes fibro cartilaginous changes due to proliferation of type II collagen, ossification, and calcium crystal deposition, degeneration of collagen and elastic fibers, and chondroid metaplasia of ligament fi¬broblasts .(6,7) Ossification of the spinal ligament is characterized by heterotrophic bone formation in the spinal ligaments, which are normally composed of fibrous tissue Chondroid metapla¬sia in YL hypertrophy appears to play a pivotal role in ligament ossification, as cartilage differentiation, hypertrophy, and cell death are followed by bone formation in the bone morphogenetic pathway(7). CT and MRI are useful tools for diagnosis, surgical planning, and evaluation of surgical prognosis. Reconstructed 2- dimensional and 3-dimensional CT images visualize the ossified lesion in all directions, which clarifies the actual shape and extent of OYL and OPLL and contributes to surgical planning Surgical treatment for symptomatic OYL and OPLL is recommended. Posterior decompression by partial laminectomy or laminoplasty with removal of OYL is effective for cases. (8,9). in this case posterior decompression of spinal canal with standard laminectomy was done patient pain was improved immediately after the surgery and muscle force was improver after 3 month and tendon reflex and upper motor singe disappeared after surgery, the location of the pathology in lower thoracic similar to usual site of this pathology.

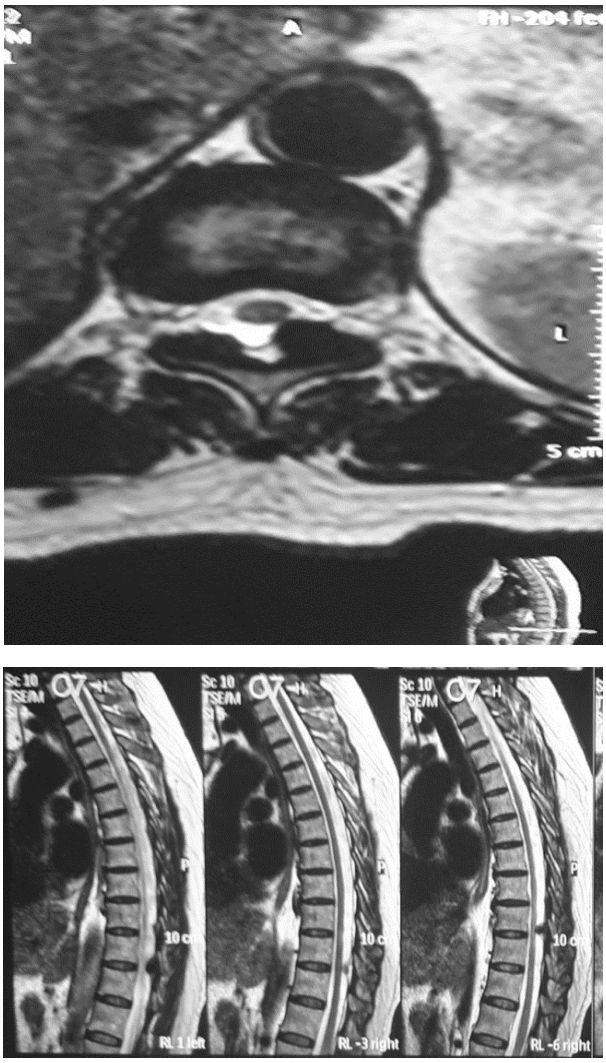
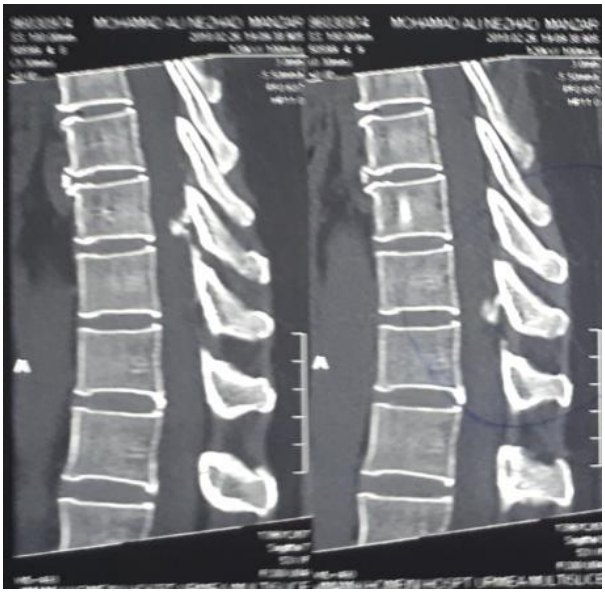


FIGURE 1. T 2 sequence of thoracic MRI sagital and axial view, hypo signal extradural lesion which compress the thecal sac from posterior



Ethical Considerations

Compliance with ethical guidelines All steps of this research were reviewed by Urmia University of Medical Sciences, ethical committee, with ethical code of 43256/43269.

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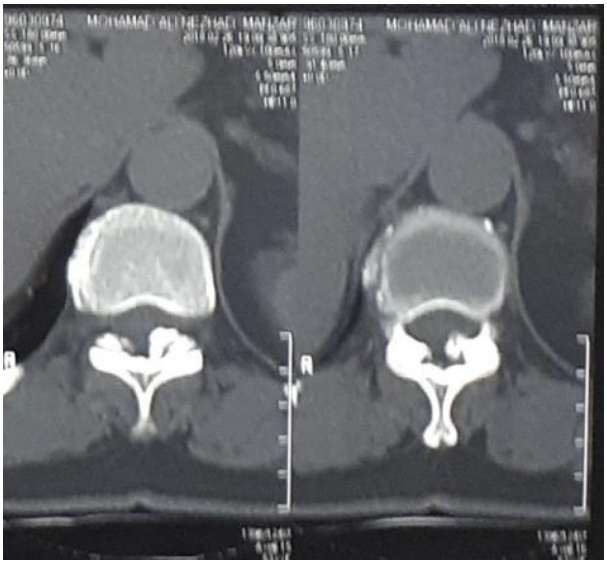

FIGURE 2. CT of vertebral column in sagital and axial view: high density lesion from lamina projected to central canal

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Comparison of the predictive strength of total white blood cell count within 24 hours on the outcome of traumatic brain injury with cranial computed tomography scan in a resource-limited tertiary health centre in sub-Saharan Africa

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ABSTRACT

Background: The enormous disease burden of patients with traumatic brain injury (TBI) remains a huge source of concern to the patient and caregivers. Computed tomography (CCT) scan is a valuable investigative tool in patients with traumatic brain injury which can be used to predict the outcome of TBI. The use of total white blood cell as a predictive parameter in patients with TBI is still at a primordial stage. This study aimed to compare the predictive strength of total WBC count within 24 hours of TBI with cranial computed tomography scan.

Methods: This research was done over one-year period at the Lagos University Teaching Hospital, Lagos. One hundred and fifty-eight patient who met the inclusion criteria were studied and the male to female ratio of 3.6:1.

Results: The mean total WBC count was 14,279.94 and the area under the curve of total WBC count and CCT scan was 0.633 and 0.855 respectively.

Conclusion: Our conclusion was that despite both parameters been a predictor of the outcome of TBI, the total white blood cell is a weaker predictor of outcome compared to cranial computerize tomography scan.

Keywords
TBI,
WBC count,
CT scan

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BACKGROUND

Traumatic brain injury (TBI) is likened to an epidemic and it will be the third leading cause of death in the developing world by 20201. It is defined as an alteration of brain function or other evidence of brain pathology caused by an external mechanical force2. It is a time bomb almost happening if left unattended to, with the male productive sector of the population affected1. It causes a huge drain on socioeconomic status of the affected individual, family, and country at large. Total white blood cell (WBC) count have been known to be elevated due to varied reasons in traumatic brain injury3,4,5,6,7 and this have been found to correlate with poor outcome.

Cranial tomography (CCT) scan have been known to predict outcome of traumatic brain injury. Our aim was to establish if the predictive strength of total WBC count can be compared with another known outcome model such as CCT scan.

METHODOLOGY

This is a hospital based prospective study of 158 patients who presented with isolated TBI within 24 hours of injury over a year period ranging from October 2014-September 2015. 5mls of blood sample was obtained in an Ethylene Diamine Tetra acetic Acid (EDTA) bottle and sent for full blood count analysis at a specific reference laboratory in Lagos University Teaching Hospital (LUTH) during which the total white blood cell count was analysed using auto-analyser (MEK-6400 haematology analyser). The patients with traumatic brain injury (TBI) meeting the inclusion criteria were reviewed. Cranial computed tomography (CCT) scan was performed at the radiological department of LUTH using Toshiba Aqillol 128 slice CT scanner. Outcome was determined using standard scale such as the GOS-E at 6 months post injury.

Inclusion criteria

Patients with clinical and radiological features of isolated TBI presenting within 24 hours of injury to the Neurosurgical unit of LUTH after obtaining informed consent.

Exclusion criteria

1. Patients with TBI who present to the hospital after 24 hours of injury.
2. Patients with TBI who are diagnosed clinically to be brain dead at presentation.
3. Patients with evidence of confirmed/established ongoing infectious processes before injury.
4. Patients with confirmed diseases that may alter white blood cell count such as haematological disorders like leukaemia and lymphoma, and uncontrolled diabetes mellitus.
5. Patients with open wounds and other systems injuries other than TBI.
6. Patients not consenting to be part of the study.

Data analysis. All statistical analyses were done using descriptive and inferential statistics.
P value <0.05 was taken as significant. Data collected were collated using statistical package for social science (SPSS) Illinois Chicago version 21. Receiver operating characteristic (ROC) curve was constructed to compare predictive strength of the variables.

Results

Age and sex distribution

A total of one hundred and ninety-nine patients were recruited into the study. 41 (20.6%) of these patients were excluded from analysis due to incomplete data and lost to follow. Altogether 158 patients met the inclusion criteria with complete data and were analysed.
Age of patients ranged between 5-83 years with a mean age of 37.04 + 18.37 years.
Most of the patients were in the age range of 31-40 years and 20-29 years representing 21.5% and 20.9% respectively. This is closely followed by those between 40-49years and 60-69years representing 16.5% and 12.7% respectively. One hundred and sixteen (73.4%) of these patients were males, while 42(26.6%) were females, with a male: female ratio of 3.6: 1. Table 1 shows the age group distribution and Figure 1 shows the gender distribution.

TABLE 1: Distribution of patients' age group

Age group in year(s)	Frequency (%)
0-9	16(10.1)
10-19	9(5.7)
20-29	33(20.9)
30-39	34(21.5)
40-49	26{16.5)
50-59	15(9.5)
60-69	20(12.7)
70-79	4(2.5)
>80	1(0.6)
Total	158(100)

Table 1 shows the age distribution of patients with 20.9% and 21.5% between the third and fourth decade respectively.

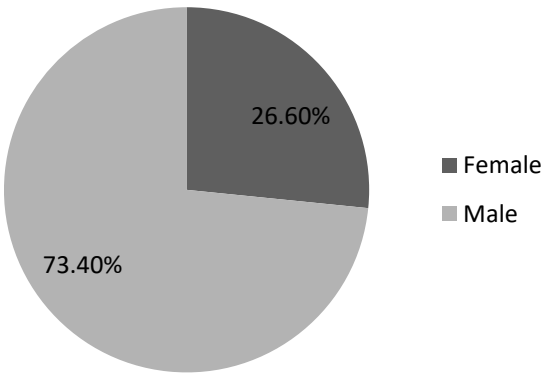

FIGURE 1 showing sex distribution
Male accounted for 73.4% of the total patients studied as shown in the pie chart in Figure 1, while 26.6% were female.

TABLE 2: Showing the relationship between the mean total WBC count and CCT scan findings 24 hours post-injury

Marshall CCT scan grading	N (%)	Mean WBC count	Standard deviation
1. Normal findings	23(14.56)	12,672.61	3,923.81
2. Cistern present	63(39.87)	14,552.54	3,773.85
3. Cistern present	21(13.29)	13,585.24	4,049.10
4. >5mm midline shift	3(1.90)	13,266.67	5,262.45
5. Surgically correctable lesion	27(17.09)	15,064.07	4,786.97
6. Non-surgically correctable lesion	21(13.29)	15,053.81	5,531.47
Total	**158(100)**	**14,279.94**	**4,312.06**

FIGURE 2 showing the area under the curve of various TBI outcome predictors

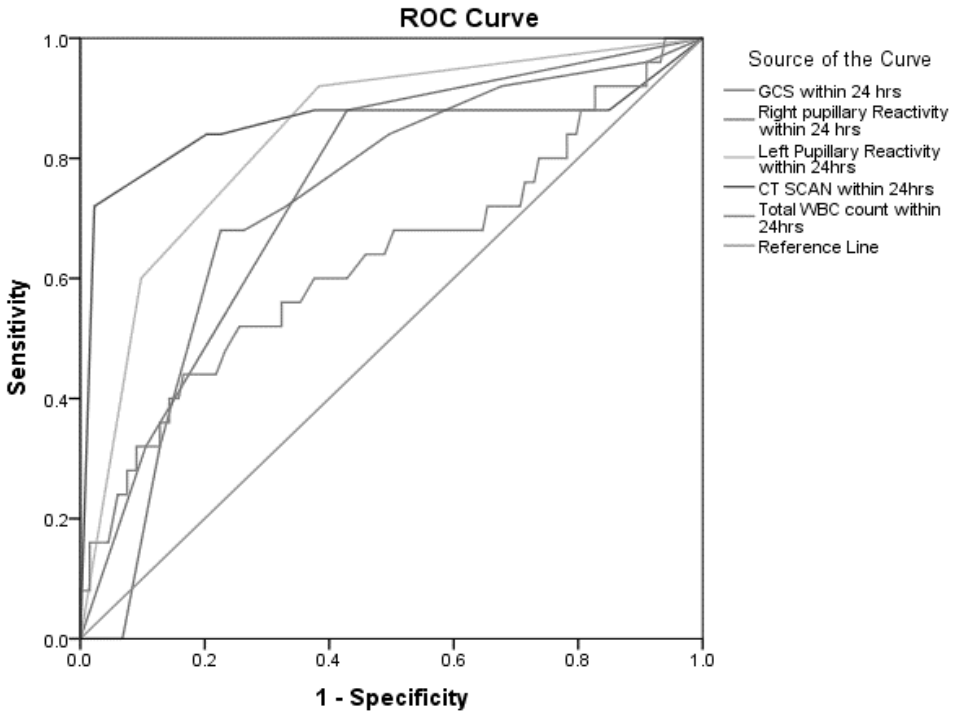

TABLE 3 - Showing area under the curve of the predictive strength of both outcome parameters

Test result variable(s)	Area under curve	Sensitivity	Specificity	P value
CCT Scan within 24 hours	0.855	72	97.74	<0.001
Total WBCC within 24 hours	0.633	44	83.36	<0.001

TABLE 3 revealed area under the curve of total WBC count as 0.633 with a sensitivity and specificity of 44% and 83.33% respectively. While, CCT scan within 24hours has the highest capacity to predict outcome with sensitivity and specificity of 72% and 97.74 % respectively.

Discussion

Studies have shown that the total white blood cell count increases with severity of traumatic brain injury, several pathophysiological processes have explained these processes responsible for elevated total WBC count3,4,5,6,7. Radiological outcome model such as CCT scan have been identified to help predict the outcome of TBI, however studies comparing the predictive strength of total WBCC is sketchy.

Cranial computed tomography (CCT) scan is a radiological tool which is useful in predicting the outcome of patients with TBI. It is a reproducible, relatively available, although expensive radiological investigative tool which helps to identify the type and severity of TBI9,10.

Studies have shown a higher predictive value with CCT scan in patients with TBI when compared to PR and GCS score in predicting short-term outcome of TBI. Findings on CCT scan such as compressed basal cistern and presence of a mass lesion are predictive of poor outcome. Studies by Van Dongen et al11 and Teasdale G et al12 confirmed strong association between GCS score, PR and CCT scan. These studies showed that predictive strength of CCT scan alone was about 48%.

The CCT scan done within 24 hours has a high area under the curve of 0.855 which was statistically significant $p<0.001$. Sensitivity and specificity of the CCT scan (done within 24 hour) predictive strength was 72% and 97.74% respectively. This showed that the CCT scan has a strong discriminative capability for outcome prediction in traumatic brain injury.

Few studies have predicted the strength of total WBCC. Gunkalar et al13 showed that predictive value of WBC count exceeding 17.5 x106/L has a predictive value for poor outcome at $p < 0.001$. In this study the predictive value of total WBCC was weak, evident by area under the curve of 0.633 at statistically significant $p<0.001$ as shown in figure 2 and table 3. Therefore, the predictive strength of these parameters to predictive outcome of TBI is strongest with CCT scan findings, and weakest with total WBC counts assessed within 24 hours of TBI.

Conclusion

It can be concluded that the predictive strength of total white cell count in patients with traumatic brain injury is weaker compared to radiological tool (CCT) used to predict outcome in TBI.

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Isolated fourth ventricle haemorrhage: "think beyond intracranial source'' unusual presentation of lumbosacral spine arteriovenous malformation presentation

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Keywords
spinal arteriovenous malformations (SAVMs), fourth ventricle, intraventricular hemorrhage (IVH), Cerebral SAH

ABSTRACT
Spinal arteriovenous malformations (SAVMs) are rare vascular lesions and account for about 4% of primary intraspinal masses. Since SAVMs can involve any location along the spinal column and produce a host of different problems, the symptoms are extremely variable. There are few reports of simultaneous cerebral SAH and intraventricular hemorrhage (IVH) following rupture of a spinal AVM (SAVMs). Herein, we present a rare case of Lumbo Sacral spine arteriovenous malformation, which clinically manifests as sudden onset of severe headache and vomiting due to isolated fourth ventricle Hemorrhage (IVH) without cerebral subarachnoid hemorrhage.

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INTRODUCTION

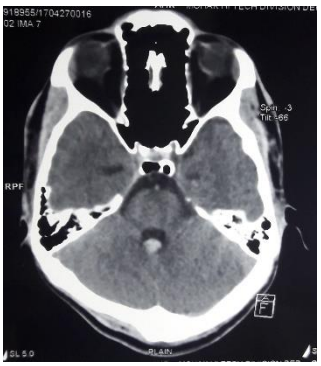
Being a rare category, spinal vascular malformations (SAVMs) account for about 4 % of primary intraspinal masses (3). Rosenblum and coworkers described two major types of spinal AVM intradural versus dural on the basis of location of nidus: intradural versus dural. Intradual AVMs were classified as intramedullary and dural AVFs (Arteriovenous fistula). They usually present with pain, numbness, and weakness, loss of bowel/bladder control, incoordination, and impotence is few of the issues. In majority of cases SAVMs first come to medical attention by bleeding - this usually presents as acute, severe back pain, followed by sudden onset weakness, numbness, incontinence; severity ranges from no neurologic dysfunction to complete paralysis, depending on location and extent of bleeding. The natural history of spinal vascular malformations (SAVMs) is unpredictable and varies from acute subarachnoid hemorrhage of spine to venous congestion of cord.

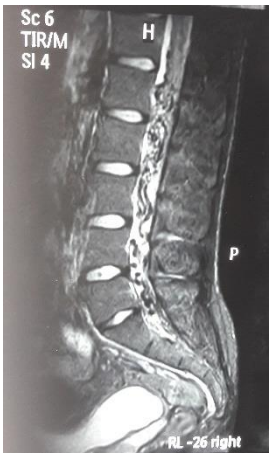
As far as cranial manifestation of SAVMs concerns cerebral sub arachnoid hemorrhage (SAH) has been reported in English literature, and seems to be caused by rupture of same intra ventricular bleed due

to SAVMs has been reported four times in last decade. We believe this to be the first reported case of a Lumbosacral SAVMs presenting as isolated fourth ventricle hemorrhage (IVH.) We also need a kind attention regarding left lower limb swelling; it might be due to raised venous pressure which was resolved spontaneously after definitive surgical management of SAVMs.

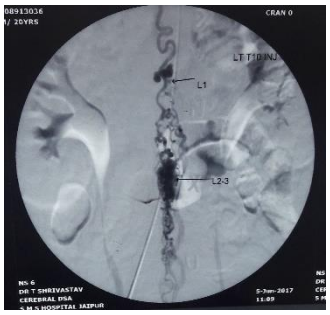
Case Report

A twenty-year-old adult male experienced sudden onset severe headache associated with episodes of vomiting in a morning during defecation, which was preceded by severe lower back pain. There was no history or signs of trauma, no previous history of back pain, radiculopathy, or myelopathy were reported. On examination it was revealed that patient was conscious, oriented and without any focal neurological sign. He had left lower limb swelling which was misdiagnosed as varicose vein of limb and was operated 6 months back for same, but swelling did not subsidize.





Non contrast computed tomography (CT) of head was done, which showed fourth ventricle hemorrhage with concomitant sparing of bilateral lateral and third ventricle, all cisterns were seen normally and had no evidence of cisternal bleeding and sub arachnoid hemorrhage (Figure 1). Magnetic Resonance imaging (MRI) brain, CT Angiography brain and digital subtraction angiography (DSA) brain did not reveal any intra cranial source of bleeding, but MRI Lumbo sacral region (Figure 2) and spine Digital subtraction angiography revealed spinal perimedullary AVM (SAVMs) at L1 to S1 level (conus medullaris) with feeding artery aneurysm and venous drainage from spinal vein to perimedullary vein and extending cranially(figure-3). The lesion was treated surgically as cauterization and disconnection of the vein. Till the writing of this report 12 month follow up has been completed and he is neurologically intact and most importantly lower limb swelling subside within first month of surgery (Figure 4).



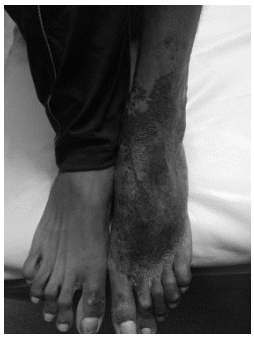


Figure 1. NCCT Head showed small hyper density in 4th ventricle suggestive of recent intraventricular bleed.

Figure 2. MRI L S Spine T2 weighted with GRE shows multiple flow void in intradural lumbo-sacral region extending from L1 to S1 levels. Subtle enhancement is seen in blood vessel with slow flow. No enhancement is seen with blood vessel with high flow suggestive of spinal dural AVF.

Figure 3. Spinal DSA showing Lumbo-sacral dural AVF with pre nidal aneurysms with feeder from left T 10 rediculomeningeal artery.

Figure 4. Swelling left leg, which reduced drastically after treating primary pathology along with hyper pigmentation on dorsum of foot.

Discussion

Spinal arteriovenous malformation (SAVM) is a rare, abnormal tangle of blood vessels on, in or near the spinal cord and account approximately 10% of CNS vascular malformations in all age groups. These lesions are directly supplied by radicular arteries and drained by spinal cord veins, although dural supply can occur as with dural arteriovenous fistulas. (3, 7)

On the basis of location these lesions are divided into either intradural or extradural, and intradural further subdivided into intramedullary or extramedullary. Most are thoracolumbar, posterior, and outside the cord (extramedullary). The rest are cervical or upper thoracic and often inside the cord (intramedullary). AVMs may be small and localized or may affect up to half the cord. (3,6)

In majority of patients the ultimate fate of spinal AVM is progressive neurological deficit in terms of sensory, motor or bladder/ bowel involvement. In

some situation rarely, high cervical AVMs rupture into the subarachnoid space, causing subarachnoid hemorrhage with sudden and severe headache, nuchal rigidity, and impaired consciousness (4). The natural history of SAVMs is characterized by venous congestion causing progressive neurological deficits in majority of patients. (3,8)

In the literature, a thoracolumbar SAVMs presenting with both SAH and IVH appears to be a rare occurrence. Although there are few reports of concomitant cerebral SAH and intraventricular hemorrhage (IVH) following rupture of a spinal SAVMs (2,3,5). There have been three case reports of SAVMs presenting in the adult population and two in pediatric age group with intraventricular hemorrhage (IVH) exist in the literature. In both groups, the clinical, radiographic, and surgical findings suggested that the SAVM was the source of the hemorrhage.

In 1999 P Bazro et al reported first case of intraventricular hemorrhage attributed by the rupture of conus medullaris AVM in a young patient. (1). ES Marlin et al reported a case of intraventricular hemorrhage in both lateral, third and fourth ventricle in a 2-year-old female child caused by rupture thoraco lumber SAVMs, who died in next few days probably due to re rupture. (3). Recognition of such cases in future may allow earlier diagnosis and treatment before catastrophic re hemorrhage. Masanori et al reported another case who presented with intraventricular hemorrhaging (IVH) into the fourth and third ventricles that was caused by a cervical intramedullary arteriovenous malformation. (4)

To the best of our knowledge, this is the first case report of an adult patient in whom initial imaging demonstrated isolated fourth ventricle hemorrhage (IVH) without Sub arachnoid hemorrhage (SAH) secondary to a ruptured low lumbosacral SAVMs. Till the date only five cases of Intraventricular hemorrhage is reported in literature.

TABLE 1. Patients presenting with IVH caused by rupture of Spinal AVMs

S.NO.	Author(s)	Year	Age/sex	Ventricular hemorrhage	Location of spinal AVM
1	H Baharvahdat et al (2)	2016	48 Year/Male	B/L Lateral, Third & fourth ventricle and SAH	Conus Medullaris AVM
2	E. S. Marlin et al. (3)	2014	2 Year/ Female child	B/L Lateral, Third & fourth ventricle	Thoraco lumber Spinal AVM
3.	Kenning. T.et al (5)	2009	1 Year 2 month /female childe	B/L Lateral, Third & fourth ventricle and SAH	Thoraco lumber spinal perimedullary AVM
4	Masanori Ito et al (4)	2007	33 Year /male	Third & fourth ventricle	Cervical intramedullary spinal AVM
5	P Barzó et al (1)	1999	28 Year /male	B/L Lateral, Third & fourth ventricle	Conus medullaris AVM
6	Present case	2017	20 Year/male	Isolated fourth ventricle	Lumbo sacral Spinal AVM

Although to kept Lumbosacral SAVMs as a differential diagnosis for isolated spontaneous fourth ventricle IVH is beyond the imagination particularly in adult patient, where the distance between the bleeding source and presenting site is significant. The present case suggests that MRI of the

entire spine with dedicated blood detected sequence GRE & SWI should be considered if cranial angiography does not reveal a source. (2,3). There must be some possible hypothesis of raised venous pressure behind the swelling of left leg due to SAVMs being wrongly operated for varicocities. So, such possibilities in these cases should also be considered.

Conclusions

The case reported raises necessity of complete spinal neuraxis evaluation especially in young group of patients presenting with angiographically negative intraventricular hemorrhage. Evaluation for thoracolumbar spinal vascular malformations must be included in the initial work up. A whole spinal workup should be considered, when bleeding from intracranial origin is carefully excluded.

This is reminder to treating neurosurgeon along with concern medical fraternity as neurointerventionalist for careful consideration this rare differential and it has to be kept in mind that presentation can varied from head to toe.

Abbreviations

IVH - Intra Ventricular hemorrhage
SAH - Sub arachnoid hemorrhage
SAVM - Spinal arterio vascular malformation
DAVF - Dural arterio venous fistula
MRI - Magnetic Resonance imaging (MRI)
DSA - Digital substraction angiography
SWI -Susceptiblity weighted imaging

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First report of two synchronous but separately placed intramedullary angiolipomas located in the dorsal spine causing progressive compressive myelopathy: management strategies and outcome review

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ABSTRACT

Spinal angiolipoma is a benign lesion and presents with compressive myelopathy. Typically, it is located in epidural compartment. However, intramedullary angiolipoma is extremely uncommon, and till date only eight cases are reported in the literature and all reported cases had isolated solitary lesion. Authors report an interesting case of intramedullary spinal angiolipoma (ISAL) in a - 48- years male, presented with compressive myelopathy, magnetic resonance imaging study revealed presence of two separate angiolipomas, which were located at D8-D9 and D10-D12 vertebral levels respectively, underwent successful near total surgical resection with good neurological outcome. Current case represents first of its kind in the western literature. Management of such rare pathology along with pertinent literature is briefly discussed.

Keywords
spinal angiolipoma,
intramedullary angiolipoma,
outcome,
surgical management

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INTRODUCTION

Angiolipoma is a rare benign tumour most commonly found in the subcutaneous tissue of the trunk and extremities but other sites are also reported. [1] Histopathologically, it consists of mature fatty tissue interspersed with abnormal vascular elements. [2]. Spinal angiolipoma represent a distinct clinical and pathological entity, which account for approximately 0.04–1.2 % of all spinal axis tumours. [3]. Spinal angiolipoma is mostly located in the epidural space and accounting for 2–3 % of spinal tumours. Only eight ceases of intramedullary lipoma are reported in literature. [4-10]. Due to rarity of lesion, these cases are usually missed as differential causes of compressive myelopathy, which have good prognosis following early surgical resection. Authors report a case of intradural spinal angiolipoma (ISAL), which was located in intramedullary- subpial located, who underwent successful surgical excision with good neurological recovery in the postoperative period.

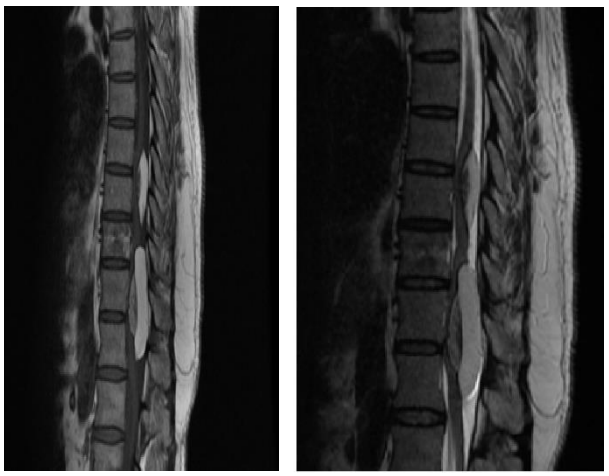
CASE ILLUSTRATION

A 48 –year- male presented to our neurosurgical outpatient services with complaint of progressive spastic paraparesis for last two years. He also noticed tingling and numbness in involving trunk below

umbilicus and both lower limbs. He had no associated history of diabetes mellitus, hypertension, coronary artery disease or pulmonary tuberculosis. Examination on admission, revealed healthy male with vital stable, had scissoring gait, increased tone with motor power of 4 /5 in bilateral lower limbs along with graded sensory loss below the level D10 dermatomes, which was comparatively more marked on left side. The deep tendon jerks were brisk with ill sustained patellar and ankle clonus with plantars was extensor response bilaterally.

MR Imaging of the spine revealed, presence of two separate intradural intramedullary mass lesions located at D8 to D9 level and D10 to D12 levels (Fig-1), showing heterogeneous hyper- intense signals on T1 and T2 weighted image, showing with characteristics suggestive of angiolipomas. Patient was diagnosed to two separate D8-D10 and D10-D12 level spinal angiolipoma (Fig-2, 3). He was advised for surgical management with intraoperative electrophysiological monitoring.

He was taken up for surgery in prone position under general anaesthesia, intraoperative level was localized with image intensifiers, and underwent D7-L1 laminectomy, after laminectomy bulging of dura sac was noted, however, no epidural mass was observed. But dura was bulging. Midline durotomy was carried out; two separate lesions were noted at D8-D9 and D10- D12 levels respectively. Eccentrically placed mass was observed, after pial incision, pale coloured fat with larger branching vessels were noted, near total decompression with Cusa was carried out. The lesions were intra medullary in location containing lipomatous mass interspersed with dilated veins. Subtotal excision was carried out as further resection attempt led to electrophysiological abnormality. After securing hemostasis, primary dural closure was carried out and the wound was closed in layers. He tolerated surgical procedure well with no appearance of fresh neurological deficits and discharged on fifth day following surgery. At last follow-up six months after surgery, has mild improvement in motor power, however sensory deficit was persisting.



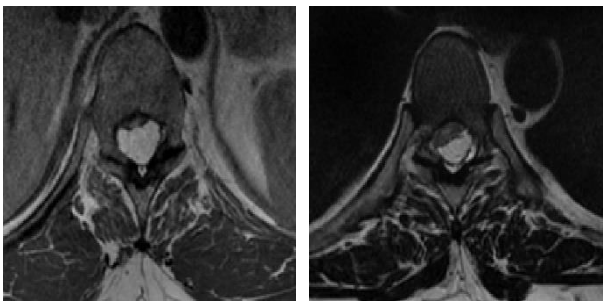


FIGURE 1. Magnetic resonance imaging, T1WImage, dorsal spine sagittal section showing two separate intradural intramedullary lesion at D8-D9 and D10-D12 causing severe distortion of spinal cord with secondary canal stenosis. Vascular component showing hypointense and fatty component with hyperintense signals.

FIGURE 2. Dorsal spine, Magnetic resonance imaging, sagittal section, T2WI showing heterogeneous mass, vascular component showing hyperintense signal on T2-weighted image.

FIGURE 3. Magnetic resonance imaging of dorsal spine, T1Weighted axial section image showing ventrally located angiolipoma.

FIGURE 4. Magnetic resonance imaging of dorsal spine, T2 Weighted image, axial section showing ventrally located angiolipoma with heterogeneous hyperintense signal.

DISCUSSION

Spinal angiolipoma represents a distinct, benign lesion and characterized by presence of mature fat cells interspersed with excessive abnormal vascular proliferations, commonly observed in the subcutaneous locations. However, spinal angiolipoma represents a separate entity with extremely rare occurrence, predominantly located in the epidural space but extremely rarely can also occur in intradural spinal compartment. Liebscher was credited to first describe the angiolipoma. [10]

Spinal angiolipoma may contain variable proportions of mature adipose tissue interspersed with abnormal vascular elements. The proportion of

adipose to vascular tissue is variable, being predominantly in lipomatous lesion to the very scanty dense mixed with vascular and stromal elements. [11] Benvenutti-Regato et al. carried out a detailed literature search in 2015 and could find out only a total of 177 patients suffering with spinal epidural angiolipoma. [12] the average age for epidural spinal angiolipoma was 46 years with predlection for female gender [59%]. The most common presenting symptom was paraparesis (30 %), others were thoracic or low back pain in (24. %). 12 The majority of epidural spinal angiolipoma occurred, in the order but and least involved site was cervical spine 2.3%. [12]

Histopathologically, ISAL lesions consist of mature adipocytes and network of branching capillary-sized vessels, which usually contain fibrin thrombi and usually positivity for CD31, Factor VIII and Factor XIII a [13].

The clinical feature of ISAL is compressive myelopathy related to spinal cord compression and usually presents early. Case with long standing history may show rapid or acute deterioration as result of enlarging vessels, engorged vein or vascular steal phenomenon, venous thrombosis or occurence of fresh haematoma. [14].

X-ray spine may show enlargement of spinal canal and moulding of adjoining lamina. Angiolipomas typically displays heterogeneous slight hypointense signal compared to typical epidural fat on T1-weighted images and inhomogeneous enhancement on contrast is observed in T1-weighted images using fat-saturation techniques. [2, 15]. A high vascular content correlates with the presence of large hypointense regions on T1-weighted images. ISALs are typically hyperintense on non-contrast T-1-weighted images relative to other spinal tumors or epidural lipomatosis. [16].

additionally, spinal lipomas occur most commonly in the midline of the lower. [15]

Surgery is the main treatment modalities; however, the extent of surgical resection is variable in reported cases, varying from simple biopsy, and subtotal resection, and near total resection and complete surgical resection have been carried out. Surgical resection may be augmented if surgery is carried out with electro-physiological monitoring. In our case we could only manage with subtotal resection, as this is limitation, as further attempt of resection led to neurophysiological alteration as, we had to stop resection.

Garg et al. reported two cases of ISAL who underwent near total surgical excision.2 Maggi et al. in 1996 reported an eight-year-old female child suffering with D11-L2 intramedullary angiolipoma, who underwent total surgical resection. [7]. Klisch el al. analysed in a 34- year -old female, with intramedullary angiolipoma at C6-D4 level, underwent partial resection and observed the clinical presentation is nonspecific, however, MRI findings was helpful in the preoperative diagnosis and helped in planning of surgical management. [5]. similarly, a 36-year female suffering with C6-D2 intramedullary angiolipoma, also underwent partial surgical resection. [9].

Prasad et al reported a -26-year- female harbouring D5-D9 ISAL, who underwent successful near total surgical excision. [4]. Preul et al. reported a 36-year female with ISAL at D7-D11 underwent only subtotal resection with improved postoperative outcome. [6]. However, Weil et al advised biopsy as treatment modality in a case with large non - resectable ISAL, and illustrated a case- 27-year-old female with extensive ISAL at C 5-D8 was managed with surgical biopsy for confirmation of diagnosis. [8].

TABLE 1. Review of Previously reported intradural angiolipoma.

S.no.	Author/ references	Y ear	Age/sex	location	Surgical management	Neurological outcome
1	Prasad and Sinha [4].	2014	26 year /male	D5-D9	Neat total excision	Incomplete recovery
2a	Garg et. al.[2]	2002	26 year/male	D3-D7	Subtotal resection	Incomplete recovery
b		2002	28year/male	C6-D2	subtotal	Incomplete recovery
3	Klischet al.[5]	1999	34 year/ female	C6-D4	Partial resection	Good outcome
4	Maggi et al. [7]	1996	8 year/ female	D11-L2	total resection	Good outcome

5	Preul et al. [6]	1993	36 year /female	D7-D11	subtotal	Good outcome
6	Weill et al [8]	1991	27 year/female	C5-D8	Only biopsy	No improvement
7	Palkovic et al.[9]	1988	27 year/male	C6-D2	partial	Not available
	Current case	2017	48year/male	D8-D9 and D10-D12	Subtotal resection	Incomplete recovery

Conclusion

Spinal angiolipoma is a benign lesion and surgery can provide good outcome. Spinal angiolipoma is mostly located in epidural compartment and only eight cases of intramedullary angiolipoma is reported. Preoperative MRI and detailed history and clinical evacuation are helpful in preoperative diagnosis. Current case is extremely rare as two isolated intramedullary angiolipoma were present separately, and underwent successful surgical resection, representing first case- report in the western literature. A high degree of suspicion for spinal angiolipoma should also be kept as differential of intramedullary pathology with typical neuroimaging findings.

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Cystotoperitoneal shunt surgery during infancy in porencepahalic cyst located in frontal region led to regaining of developmental milestone

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Keywords
porencephaly, raised intracranial pressure, cystoperitoneal shunt surgery.

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ABSTRACT

Porencepahalic cyst is considered as an extremely uncommon developmental disorder of the central nervous system, being characterized by the presence of a fluid-filled cysts or cavities located within the cerebral hemispheres. It can be associated with varied aetiology and can present with a spectrum of clinical presentation varying from asymptomatic to grossly spastic limbs, mental retardation, cognitive impairment and intractable seizure. Extensive Pubmed and Medline search did not yield any result when searched for term "infancy, porencepahalic cyst, cystoperitonreal shunt." However, clear guideline for management is still lacking. Authors report an interesting case of giant porencepahalic cyst located in the right frontal region in infancy and underwent cystoperitonreal shunt surgery, which lead to good outcome with remarkable recovery of delayed milestones with adequate scholastic performances along with marked diminution in the size of porencepahalic cyst.

INTRODUCTION

Porencepahalic cyst is considered as an extremely rarer encephalomalatic disorder of the central nervous system characterized by the presence of a fluid-filled cavities or cysts. [1] It usually represents the end result of various types of the cerebral parenchymal injury, but can also be congenital developmental disorder, or consequences of intracerebral hemorrhage. [2,3] Author reports an interesting case of porencepahalic cyst located in the right frontal region, who underwent cystotoperitoneal shunt surgery, led to recovery of regressive milestones and currently child is extremely well scholastically at four years following CSF diversion surgery.

CASE ILUSTRATION

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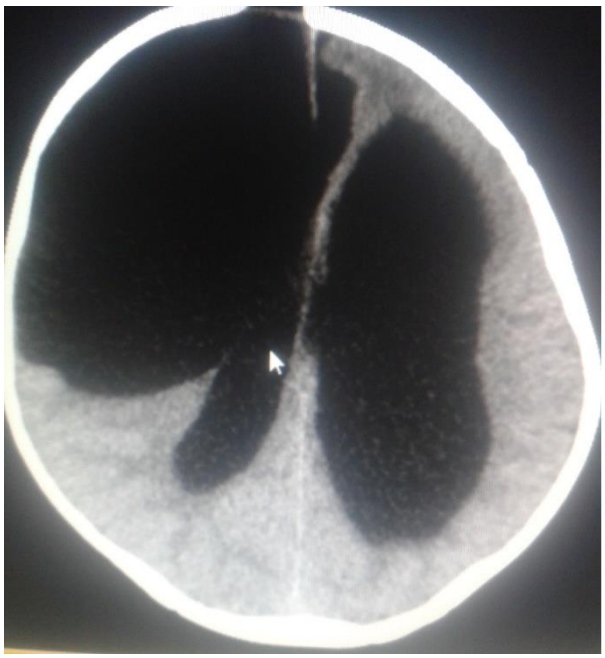
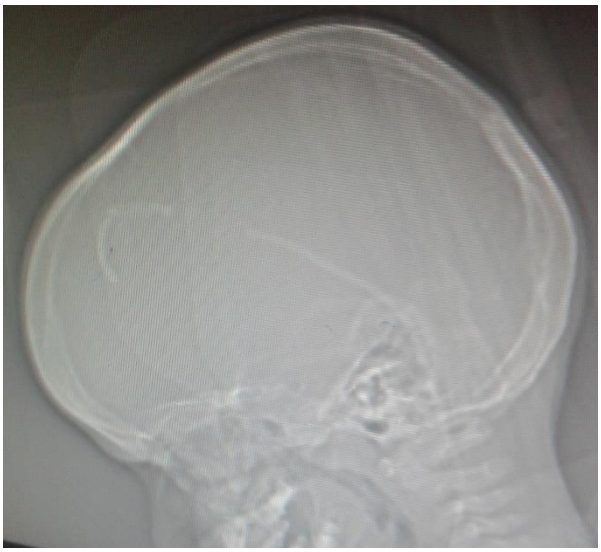
A one- year- old male infant was brought to our neuroscience emergency services with complaint of failure to thrive, delayed developmental milestones with repeated episodes of vomiting and progressive head enlargement for the last six months. On general physical examination at admission, vitals were stable, with bulging anterior frontanelle and head circumference was 54 cm. On neurological examination, he was alert, crying. Evaluation of milestones, he was unable to can sit up without support and not able to crawl and could not use the pincer grasp. He was not able to begin to make recognizable syllables like "ma" and also had regression of language development. Fundi showed bilateral papilloedema. He had bilateral up gaze paresis with presence of sunset sign.

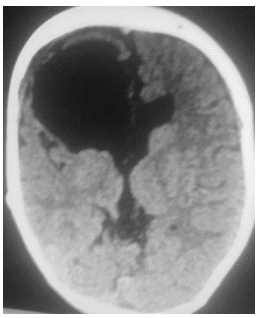
The routine hematological and biochemical parameters were normal limit. The computerized cranial tomography which was carried out after admission (Fig. 1) revealed presence of a giant porencepahalic cyst in the right frontal region crossing across the midline and causing mass effect obstruction along the CSF pathway leading to dilated lateral ventricles, the third ventricle and. The periventricular ooze was present. (Fig. 2)

He underwent cysto-peritoneal shunt surgery under general anaesthesia. CSF study revealed normal biochemical parameters with normal cell count and further, CSF culture did not show growth of microorganism. After surgery he regained milestones like crawling, pincer grip and started telling Monosyllabic words over two months after the surgery. (Fig. 3)

The computerized tomography carried out at follow-up at four years after shunt surgery revealed well decompressed ventricles (Fig- 4) with ventricular catheter well placed, (Fig. 5) and marked reduction in the size of porencepahalic cyst with well-functioning shunt. Follow-up at three year, he was doing extremely well with developmental milestone appropriate to age and the body weight also appropriate to the age.







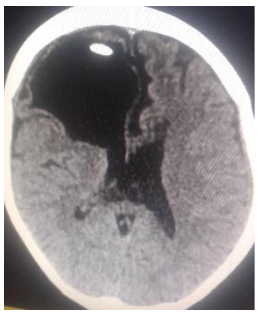


FIGURE 1. Noncontract cranial computed scan showing large porencepahalic cyst

FIGURE 2. Tomogram showing shut in situ

FIGURE 3. Noncontract cranial computed scan showing significant diminution in size of porencepahalic cyst

FIGURE 4. Noncontract cranial computed scan showing signicant diminution in size of porencepahalic cyst with shunt tip in situ

DISCUSSION

In 1859, Heschl coined the term "porencephaly" to describe presence of a cavity within the human brain. [2] The porencepahalic cavities can result from focal cerebral degeneration secondary to haemorrhages. [8] further de novo or inherited heterozygous mutations in COL4A1 coding the type IV α1 collagen chain, which is essential for vascular basement membranes structural integrity is observed in individuals with porencephaly by Yonder et al. [8] Yoneda et al postulated abnormalities of the α1α1α2 heterotrimers of type IV collagen causes porencephaly. [4] During embryonic life. The large craters develop on the brain surface and gets lined with smooth tissue, filled –up with fluid and causes local mass effect and focal neurological deficit and regression of milestones. [5,6] Usually caused by antennal or perinatal insult prior to development of the cerebral gyri due to various pathology i.e. Infective, infarction ischemic or intracerebral haemorrhage.

Depending on the location, size and approximate to the vital region and severity of the defects, symptoms are highly variable and may cause only minor neurological deficit to severe mental retardation and paralysis in extreme cases. The patients with a porencepahalic cyst also frequently suffer with seizure disorder. [4,7]

The diagnosis can be also made even during antenatal period with ultrasound and considered as preferred imaging modality during fetal life. In infant, trans-illumination of the skull can also aid in the diagnosis of the porencepahalic cyst. Cranial computed tomography reveals location, size; volume, degree of mass effect, associated hydrocephalus and associated developmental pathology and effect of previous treatment and very useful in serial follow-up in assessing the size and evaluating the effect of cystoperitoneal shunt and recurrence of porencepahalic cyst. [6] Currently. The magnetic resonance imaging is considered as modalities of choice as it clearly depicts the pathology and associated obstruction along the CSF pathway and associated congenital hydrocephalus, agenesis of corpus callosum or other developmental pathology.

Porencepahalic cyst diagnosis requires detailed clinical and neurological evaluation, family's history, clinical observations, or based on neuroimaging findings. Neuroimaging establishes the diagnosis and further assessment of intelligence, memory and speech evaluation can further aid in the planning of the holistic management and rehabilitation measures. [4]

Currently, there is no curative therapy for porencepahalic cyst and various treatments currently offered included physiotherapy for spastic limbs, rehabilitation, providing antiepileptic medication for those suffering with seizures. Surgical procedure may include cerebrospinal fluid diversion surgery like cystoperitonreal shunt, VP shunt surgery and surgery for epilepsy with intractable epilepsy. [1] According to the location of porencepahalic cyst, extent of the mass effect, size of cavities, and severity of the disorder, combinations of treatment modalities of treatment are usually made tailor made to suit the case.

Prognosis of porencephaly depending on various factors i.e. The location of the cyst and extent of the damage of the brain. Cases with mild neurological deficit can lead normal lives and self-care is possible, however more seriously disabled cases require lifelong support in the nursing care and rehabilitator measures. [5] Early diagnosis, medication, participation in rehabilitation activity related to fine-motor control, and communication skill can significantly improve the symptoms and ability to cope up with the severe disability and prompt to lead to normal life. Infants, with proper treatment, can develop good locomotors control as our case, as he was offered surgical treatment at infancy continued antiepileptic medication, led to good recovery of milestones and improvement in cognitive and scholastic performance.

Conclusion

Early diagnosis and management of symptomatic raised intracranial pressure in case of porencepahalic cyst, appropriate surgical intervention at early age and antiepileptic medication and rehabilitation measure and regular follow-up cases may aid in not only good neurological recovery and acquiring good motor and cognitive skill and can lead independent life as our cases underwent Cystoperitonreal shunt at one-year age.

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Tibial nerve schwanoma: short review of surgical management

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ABSTRACT
Schwannoma is a benign, solitary nerve sheath tumour and accounting for about 5% of soft tissue tumours. It can occur along the peripheral nervous system in any part of body. It presents as a painless, swelling. We report an adult male presented with tibial nerve schwanoma underwent successful surgical excision. However, differentiation with neurofibroma is very important as surgical planning and prognosis is quite different. In lower limb usually incidence of neurofibroma is higher in contrast of upper limb. Pertinent literature and management are briefly discussed.

Keywords
tibial nerve schwannoma, painless mass, surgery, neuroimaging

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INTRODUCTION

Schwannoma represents a benign peripheral nerve tumour, originating from Schwann cells [1], [2]. It usually presents as a solitary, slow growing mass. It accounting for about 5% of all soft tissue tumours. Iot can present with pain, paraesthesia or rarely with neurological deficit [3], [4], [5].

CASE ILLUSTRATION

An adult male reported presented with complaints of painless mass with paraesthesia and difficulty in sitting on chair for two-years. Local examination showed presence of mass in rthe popliteal fossa, about size of 4cm X5 cm, no compressible, nonpulsatile being mobile along transverse axis burt no mobility in craniocaudal axis. A magnetic resonance imaging was carried out to ascertain the nature, revealed presence of a mass lesion causing expansion of tibial nerve. (Fig-1) A provisional diagnosis of peripheral nerve sheath tumour was made and planned for surgical excision. He underwent micro-surgical total excision, intraoperative expansion of the nerve was observed, and nerve fascicles were carefully separated from mass lesion, with electrophysiological nerve monitoring. He had relief in pain and

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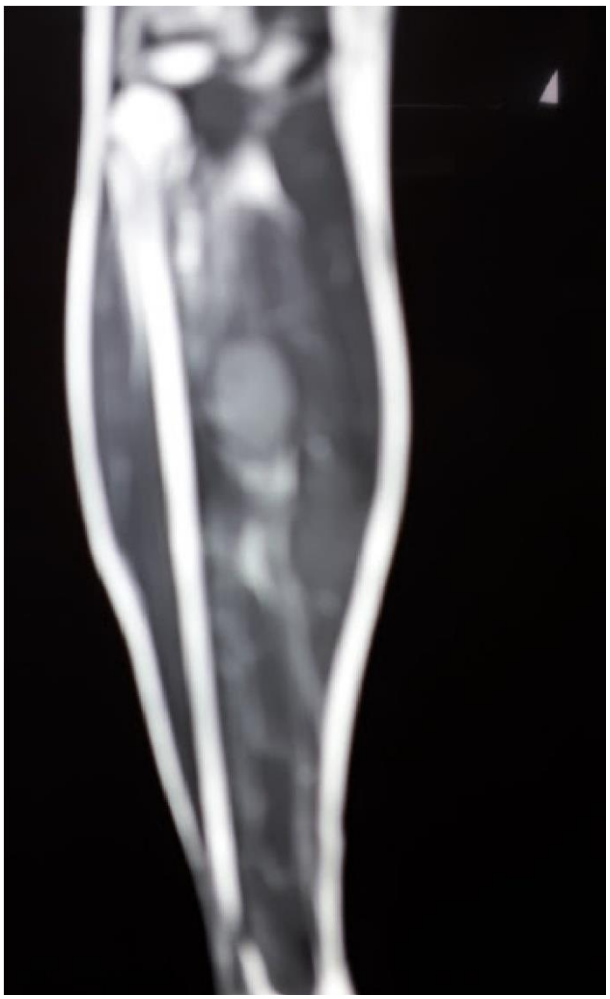
paraesthesia in post-operative period. Histopathology of resected specimen was suggestive of schwannoma.



Discussion

Tibia nerve is continuations of larger terminal branch of the sciatic nerve with root values of L4, L5, S1, S2, and S3 [1], [5], [6], [7] Tibial nerve usually lies superficial to the popliteal vessels, extending from the superior angle to the inferior angle of the popliteal fossa, crossing the popliteal vessels from lateral to medial side [8].

Peripheral nerve sheath schwannoma symptoms are related to alteration in the function of nerve and surrounding muscle and neurovascular bundles, and mostly commonly present with paraesthesia or pain of insidious onset and progresses slowly [2]-[4]. Pain is a much more common symptom than focal motor or sensory deficits. Physical examination may reveal the presence of a mass along the course of the nerve, tender, usually mobile along the transverse axis but limited along the longitudinal course of the nerve, and positive Tinel sign [6], [9].

However, pre-operative confirmatory diagnosis of schwanoma usually not possible in most cases but can help in delineating shape, size, location, extent and relation with parent nerve and adjacent neurovascular structures and muscle. Imaging plays a limited role in distinguishing among various types of peripheral nerve sheath tumours. Magnetic resonance imaging may show presence of fusiform mass with characteristic tapering cephalad and distal ends, fasciculation sign and split fat signs [3], [8], [9]. The mass is well-circumscribed and eccentrically placed, and showing isointense signal on T1-weighted images and T2 weighted images shows hyperintense signal and peripheral rim demonstrate hypo-intensity signal representing capsule [3], [5].

After confirmation of diagnosis management of peripheral nerve schwanoma is usually surgical except when the mass is very small and not causing any physical disfigurement.

Treatment of epineurium encapsulated tumour is microsurgical excision with careful preservation of the nerve fascicles. Histopathological examination of specimen provides definitive diagnosis [4], [5]. Kim et al. analysed 397 cases of peripheral nerve sheath tumour, out of which 91% were benign and the rest were malignant. A total of 251 were located in the brachial plexus region or upper limb. The peripheral nerve sheath tumor involving lower-limbs included 53 cases of neurofibroma and 32 cases of schwannomas [5]. Typically showing the incidence of schwanoma is less than neurofibroma.

Recurrence is uncommon following total surgical excision. Usually surgical excision provides good outcome in view of its benign biological nature and malignant transformation is extremely rare.

Conclusion

Tibial nerve schwanoma is raer entity compared to neurofibroma and prognosis and surgical planning should be discussed and prognosticated to patient as imaging may also may not definitely distinguish between neurofibroma, schwanoma. However electrophysiological monitoring is an important aid in preserving neurological outcome. Hence, every surgical team member should be always considering the possibility of neurofibroma, schwanoma.

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Toddler with repeated fall frequently visiting hospital presented with acute subdural hematoma on readmission with ultra-rapid evolution: surgical management strategy

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ABSTRACT

Repetitive fall producing head injury in children may lead to development of intracranial hematoma. The course of evolution may be rapid in case of repeated fall due to induction of sub-clinical coagulopathy caused by repetitive cranial injury. The awareness of such possibility is highly desired among the pediatrician and neurosurgeon and emergency team and quick diagnosis and pertinent imaging study is of immense value and appropriate surgical management for prompt and expediting the evacuation of intracranial hematoma evacuation should be attempted to preserve good neurological outcome. Authors reports a case, who had rapid neurological worsening, managed surgically with good neurological outcome, further various surgical management options along with pertinent literature are briefly reviewed.

Keywords
repeated fall,
cranial injury,
acute subdural hematoma,
surgery

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INTRODUCTION

Traumatic brain injury is a global epidemic affecting all age group and producing cognitive, emotional, psychological, and economic burden and various disability and huge cost to society on care of acute phase of head injury extending to the rehabilitative phase. Author presents a case, who had repeated fall in house due to carelessness and previously also visited hospital for fall, at current admission had rapid neurological worsening due to acute subdural hematoma. He was managed successfully.

CASE ILLUSTRATION

Author presents a case of two- year old male child with history of repeated falls. Patient had fall from first floor with loss of consciousness. Patient presented to trauma emergency, on evaluation GCS was E4V5M6 with no focal neurological deficit. Computed tomography head did not reveal presence of fracture or intracranial haematoma and discharged satisfactorily after observation. (Fig-1) However, due carelessness of parents, the child had again fell down and brought to emergency in unconscious state after three days after discharge from hospital following previous admission. On examination at current admission, vitals were stable with GCS was E3V3M5. His

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pupils were asymmetric, left pupil was dilated and not reacting to light. Urgent repeat computed tomography scan head was done, showed presence of acute subdural hematoma extending over the left frontotemporal with midline shift with subfalcine herniation. (Fig-2). Baby had further neurological deterioration while awaiting admission at emergency to E2V2M4 and hence taken up for emergency surgical evacuation of acute SDH under general anaesthesia. dura was tense and following opening the dura, thick dark coloured blood clot was observed in the subdural space. Post operatively child was electively ventilated for two days and then extubated and continued to receive decongestant therapy, antibiotics and antiepileptic medication continued. Following emergency evacuation of acute subdural hematoma, pupil anomaly improved. At the time of discharge, child was E4V5M6, accepting orally and playing actively with mother.

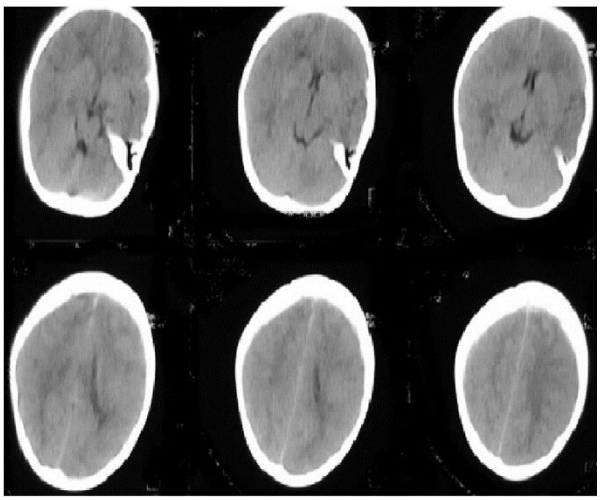

FIGURE 1. Cranial computed tomography scan following fall at previous admission showing no evidence of fracture or presence of extra-axial or intra-axial hematoma.

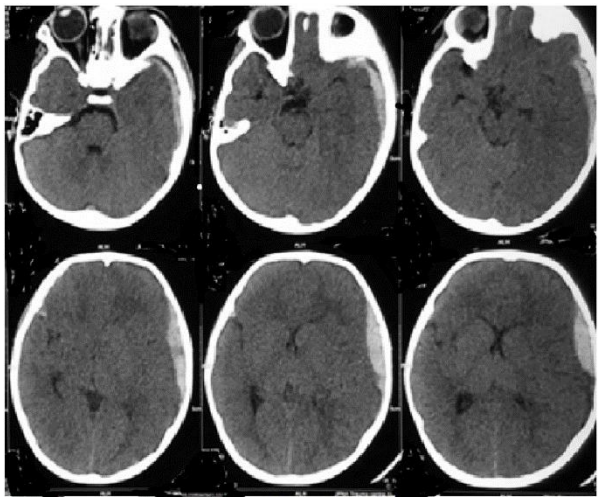

FIGURE 2. Computed tomography showing presence of large acute subdural hematoma over left fronto- temporal region with associated mass effect and subfalcine herniation, at current admission.

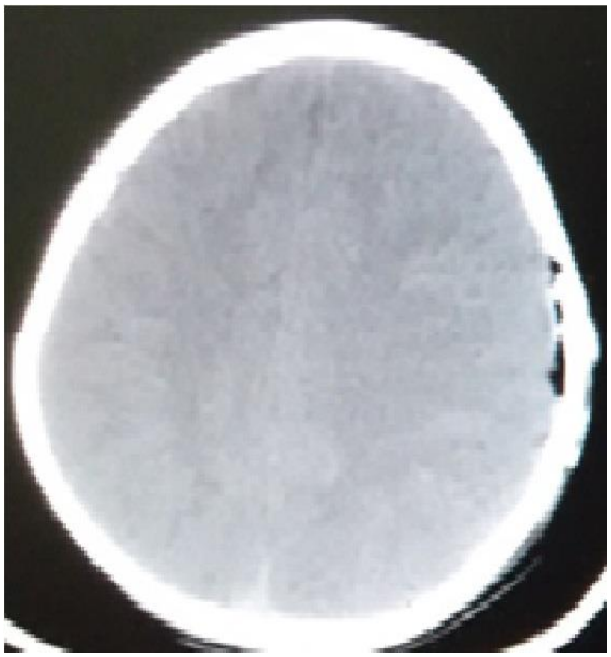

FIGURE 1. Non contrast computed tomography after six months following surgical evacuation of acute subdural hematoma.

DISCUSSION

Traumatic brain injury constitutes leading causes of acquired disability and death in the infants as well as children[1]. Subdural hematoma is most common intracranial pathology observed in neurosurgical practice following traumatic brain injury[2,3].

Falls and motor vehicle accidents are common non-inflicted causes, while child abuse among infants and young children are unfortunate inflicted causes of traumatic brain injury[2]. The patho-mechanism of fall from a short height in the very young children impart a predominantly linear force to the head. These forces can cause local deformity of skull or in cases forces being sufficient enough to produce skull fractures, and extradural hematomas. Subdural hematomas development commonly results from displacement of the brain relative to the dura, and may associated with rupture of the bridging veins courses from the brain's surface to the overlying larger draining venous sinuses[4,5]. However, extradural hematomas is commonly associated with

focal impact injuries, but subdural hematoma almost always results from angular head deceleration, in which the brain continues to rotate relative to the more stationary skull and dura, associated with diffuse parenchymal damage. Management of traumatic brain injury primary aims for limiting the progression of the primary brain injury and minimizing secondary brain injury[6-11].

Surgical management is the mainstay of management of acute subdural hematoma with mass effect. However, in acses with rapid neurological deterioration, surgical decompression needs to be expedited and, in many resource, scarce centre, operation theatre may not be available, larger burr hole craniostomy with evacuation of subdural hematoma with subdural drain placement can offer an option with burr hole being placed at the site of thickest component[9,11].

Satyarthee et al. reported burrhole evacuation of acute subdural haematoma is considered as a novel technique to reverse the worsening neurological state of patient[7,8].

Other traditional approach include craniotomy with evacuation of subdural hematoma, decompressive craniectomay and management of associated intracerebral hematoma. Decompressive craniectomy or other major intracranial procedure for evacuation of acute subdural hematoma can be done if patient GCS and neurological status remains stable[8-13].

Conclusion

A child with repeated fall may develop larger haematoma, although previous cranial CT scan may not show presence of any intracranial hematoma, and depending on history, detailed clinical assessment, appropriate neuroimaging study is advocated and further early and rapid neurological deterioration, emergent surgical management is must. Awareness of rare but important pathology is highly recommended for pediatrician, neurologist, neurosurgeon and emergency care team.

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Middle cerebral artery stroke following massive hornet sting: a case report

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ABSTRACT
Hornet stings are frequently encountered in practice in Nepal. Majority of patients sustain minor illness. However, complications like anaphylactic shock and the rare acute kidney injury, multiple organ dysfunction and acute myocardial infarction have been reported. Ischemic stroke following hornet stings has been reported infrequently in scientific publications. We report a case of fatal right Middle Cerebral artery territory ischemic stroke and acute kidney injury in a 40-year farmer.

Keywords
hornet, sting, middle cerebral artery stroke

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INTRODUCTION

Human encounter with hornets has been found in plantation, cultivation and forest areas. They are aggressive upon disturbance and attack in swarms causing victims to sustain multiple stings. The sting is excruciatingly painful and the insect leaves the stinger. Unlike typical bees, hornets and wasps do not die after stinging because their stingers are not barbed and are not pulled out of their bodies. Hornet stings are more painful to humans than typical wasp stings because hornet venom contains a large amount (5%) of acetylcholine.

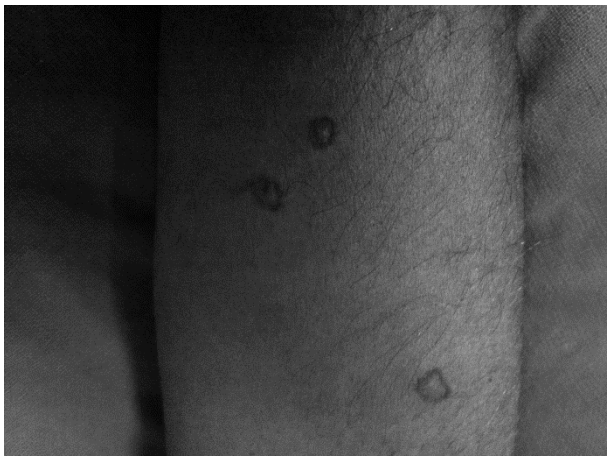
Hornets, like many social wasps, can mobilize the entire nest to sting in defense, which is highly dangerous to animals and humans. The attack pheromone is released in case of threat to the nest.

The majority of sufferers recover with minor illness. But fatal and non-fatal complications that arise from such stings include anaphylaxis, acute kidney injury (8), multiple organ dysfunction (7), myocardial infarction (3) and ischemic stroke (1,2,5,6). Vasoactive, inflammatory, and thrombogenic peptides and amines, including histamine, leukotrienes, and thromboxane are responsible for the end organ complications. The allergenic proteins such as phospholipases which elicit IgE responses, resulting in mast cell activation, underlie the anaphylaxis.

CASE REPORT

A 40-year-old patient was referred to the emergency department for decreased consciousness after sting by a swarm of hornets. The

previously healthy farmer from the Dang valley in western Nepal was stung by around 100 hornets when he was collecting maize crops in his field. He was immediately taken to local hospital where he received chlorpheniramine (antihistamine) and hydrocortisone. The patient had fall in sensorium for which he was referred to our center. The family's medical history did not reveal thromboembolic disease or significant neurological and autoimmune rheumatoid disease. Upon arrival, his GCS was poor (6/15). Physical examination revealed multiple stings in his body including his face and scalp. (Fig 1) He had acute kidney injury (creatinine level 9.5mg%). His serum LDH was 5211(normal range: 225-450) and CPK of 3652 (normal <170U/L). He had severe metabolic acidosis with Bicarbonate level of 15meq/l. His CT head revealed a large MCA (middle cerebral artery) ischemic stroke with mass effect and midline shift. (Fig 2) He underwent dialysis for acute kidney injury, however patient succumbed to his illness.



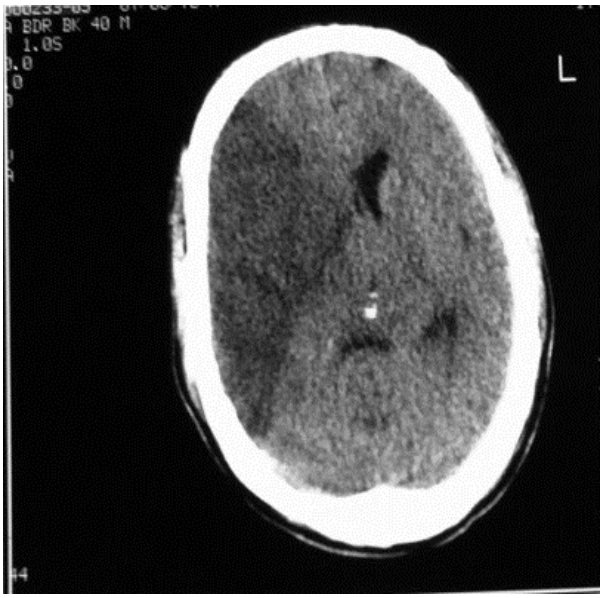


FIGURE 1: Hornet sting induced local erythematous changes in the forearm.
FIGURE 2: Plain CT head revealing large right MCA infarct with mass effect

DISCUSSION

Hornet venom contains vasoactive, inflammatory, and thrombogenic peptides and amines, including histamine, leukotrienes, and thromboxane. The venom also contains allergenic proteins such as phospholipases which elicit an IgE response, resulting in mast cell activation which is the hallmark of anaphylaxis.

Pain, wheal, flare, edema and swelling, which are generally self-limiting, constitute local disease. Multiple stings can lead to vomiting, diarrhea, generalized edema, dyspnea, and hypotension.

Severe systemic complications include anaphylaxis which constitutes the leading cause of death, acute kidney injury, multiple organ dysfunctions, disseminated intravascular coagulation, myocardial infarction, Rhabdomyolysis and neurological disease.

Numerous neurological complications include ischemic stroke, venous sinus thrombosis, ocular myasthenia gravis and thrombotic thrombocytopenic purpura. The proposed underlying mechanism of early ischemic stroke includes hypotension of anaphylaxis and vasospasm due to treatment with adrenaline. The delayed phenomena leading to infarctions appear to be spasms caused by vasoactive substances and thrombosis induced by thrombogenic factors in venom (1).

The other neurological complications of stings which have been reported are individual case reports of ocular myasthenia gravis, optic neuritis, limb numbness, trigeminal neuralgia and encephalopathy. Postulated mechanisms include both toxic effect of venom and hypersensitivity to venom (6).

In the case of acute myocardial infarction following hornet stings, the postulated mechanism is a combination of coronary vasoconstriction and platelet aggregation secondary to mediators released after wasp sting, aggravated by exogenous adrenaline given as part of the treatment.

In our patient, we postulate that the systemic immune mediated reaction to the bee sting caused vasoconstriction and a prothrombotic state with subsequent ischaemia leading to stroke.

Conclusions

Insect stings can lead to neurological complications and can be fatal. Timely intervention is required to prevent these.

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Epidemiological study of intracranial meningiomas in a tertiary care hospital

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ABSTRACT

Meningiomas are tumours that arise from the meningothelial cells. Most of these tumours are intracranial; some are intraspinal and few extra cranial. There are many histological variants classified into three grades depending on clinical behaviour. Classification is important for determining the modality of treatment. Objectives: To study the incidence, location, sex and age predilection, histological variants and grading of meningiomas based on WHO 2007 classification and recurrence if present. Materials and methods: All 200 cases of meningiomas. Based on Histological features, typing and grading of meningiomas was done as per the WHO 2007 classification of Meningiomas. Age, Sex incidence, Location of meningiomas were studied. Results: Meningiomas comprised 26.17% of all CNS tumours during the study period. Of 764 CNS tumours, 200 were meningiomas. Most of them were intracranial, predominantly involving the convexities of brain, females and the 41 – 50 age group. Of these, 180 were benign grade I tumours, 12 were grade II and 8 were grade III. The most common histological variant was fibroblastic and meningothelial. Grade II and Grade III tumours commonly recurred. Conclusion: Meningiomas are slow growing tumours arising from the meningothelial cells accounting for 26.17% of all CNS neoplasms showing a variety of histological patterns, more common in women, predominantly Grade I tumours. Recurrence of tumours depends on histological grade and extent of surgery.

Keywords
intracranial meningioma, epidemiological study, tertiary care hospital

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INTRODUCTION

A meningioma is a tumour that develops from the specialized meningothelial cell called as arachnoidal cap cells, the membrane that surrounds the brain and spinal cord, and located along the parasagittal sinus, over the cerebral convexity, sphenoid wing, around the pontocerebellar angle and along region of the spinal cord (1). Meningiomas constitute approximately a quarter of central nervous system (CNS) neoplasms. Most meningiomas (90%) are categorized as benign tumours, with the remaining 10% being atypical or malignant.

Harvey Cushing in 1922 coined the name "meningioma" for the most common dural based tumour, accounting for 15-30% of all primary intracranial tumours (2). These tumours can occur in any age, but commonly present in middle age and has a female preponderance,

with a female/male ratio of approximately 2:1 intracranial and 10:1 on the spine. Genetic factors also play a role in meningioma development and predisposition. Type 2 neurofibromatosis (NF2) is an autosomal dominant condition related to a mutation on chromosome 22q12 and is a common condition related to increased risk for developing meningiomas, among other neoplasms (3). Ninety percent of meningiomas are benign, 6% are atypical, and 2% are malignant tumours (4).

Meningiomas vary in their symptoms, cranial meningiomas may cause seizures, headaches, and focal neurological deficits. Diagnosis is made by a contrast enhanced CT and/or contrast MRI (magnetic resonance imaging) scan. While MRIs are in some ways superior, the CT-scan can be helpful in determining if the tumour invades the bone, cause hyperostosis of bone.

Most patients with meningioma undergo resection to relieve neurological symptoms. Complete resection is often curative. For incompletely resected or recurrent tumors not previously irradiated, radiotherapy is administered. Two of the most important factors that determine the prognosis in patients with meningiomas are the extent of the resection and the tumor's histological grade (5). Although as a group they are considered to be benign, symptoms, variability in recurrence frequency, life expectancy, histological appearance and prognosis exist.

Material and Methods

This study is a retrospective study conducted in the Department of Neurosurgery, G. R. Medical College and Jay Arogya Hospital, Gwalior, M.P. India, over a period of 5 years. Of all CNS tumours, only cases of meningiomas during the study period were included. Meningiomas in all age groups and both sexes were included in the study. Other CNS tumours were excluded. These cases were analysed for age, sex incidence, location and histopathological diagnosis. Statistical analysis was done by calculating the numbers and percentage for computing the incidence in various age groups, in sexes, location and HPE diagnosis.

Study design: A meta-analysis
Ethical approval: The study was undertaken after consent and clearance by the ethical committee of G.R. Medical College Gwalior
Inclusion criteria: Of all CNS tumours, only cases of meningiomas during the period 2012 – 2017 were included. Meningiomas in all age groups and both the sexes were included in the study.
Exclusion criteria: Other CNS tumours were excluded.
Sample size: Two hundred cases of meningiomas
Methodology: Based on Histological features, typing and grading of meningiomas was done as per the WHO 2007 classification of Meningiomas. Age, Sex incidence, Location of meningiomas were studied.
Statistical analysis: It was done by calculating number and percentage for computing the incidence in various age groups, in sexes, location and also comparison with other studies.

Observation and Results

Table 1: Age wise distribution of patients

S.No.	Age (yrs)	No. of patients	Percentage
1.	< 20	9	4.5%
2.	20-40	75	37.5%
3.	41-60	96	48%
4.	> 60	20	10%

Table 3: Presenting complaints

S.No.	Clinical presentation	No. of patients	Percentage
1.	Headache	178	89%
2.	Seizure	96	48%
3.	Raised ICP	80	40%
4.	Ptosis	20	10%
5.	Hemiparesis	69	34.5%
6.	Behaviour problem	15	7.5%
7.	Memory difficulties	40	20%
8.	Visual problem	27	13.5%

TABLE 2. Gender wise distribution of patients

S.No.	Gender	No. of patients	Percentage
1.	Male	92	46%
2.	Female	108	54%

TABLE 4: Distribution of patients according to location of tumour

S.No.	Location of tumour	No. of patients	Percentage
1.	Falx or parasagital	40	20%
2.	Convexity	80	40%
3.	Sphenoid wing	20	10%
4.	Olfactory groove	13	6.5%
5.	Petroclival	3	1.5%
6.	Posterior fossa & CP angle	22	11%
7.	Tentorial	9	4.5%
8.	Pterional	2	1%
9..	Tuberulam sellae	2	1%
10	Intraventricular	5	2.5%
11	Diploic	2	1%
12	Foramen magnum	2	1%

TABLE 5. Type of Craniotomy

Location of tumour	No. of patients	Percentage
Fronto-Temporo-Parietal craniotomy	20	10%
Fronto- Parietal craniotomy	62	31%
Frontal	33	16.5%
Temporo-parietal	19	9.5%
Tempro-parieto-occipital	9	4.5%
Bifrontal	15	7.5%
Parietal	5	2.5%
Sub-occipital	24	12%
Parieto-occipital	13	6.5%

TABLE 6. Distribution of patients according to according to surgical excision

S.No.	Simpson grade	No. of patients	Percentage
1.	I	30	15%
2.	II	135	67.5%
3.	III	17	8.5%
4.	IV	16	8%
5.	V	2	1%

TABLE 7. Distribution of patients according to size of the tumour

S.N.	Size of tumour	No. of patients	Percentage
1	1-3 cm	0	0
2	3-4 cm	138	69
3	4-5 cm	42	21
4	>5 cm	20	10

TABLE 8. Distribution of patients according to grade

S.No.	Grade	No. of patients	Percentage
1.	I	180	90%
2.	II	12	6%
3.	III	8	4%

TABLE 9. Post op complications

S.No.	Post op complications	No. of patients
1.	Infection	38
2.	Seizure	23
3.	Hemiparasis	86
4.	Visual loss	1
5.	Behavior change	28
6.	Memory deficit	43
7.	Raised (ICP)	13

TABLE 10. Patient follow up data given as frequency

S.No.	Follow up	No. of patients with recurrence	%
1.	< 2 year	5	2.5%
2.	2-5 years	12	6%

FIGURE 1. Pre op and post op CT of tuberculum sellae meningioma. FIGURE 2. Pre op and post op CT of olfactory groove

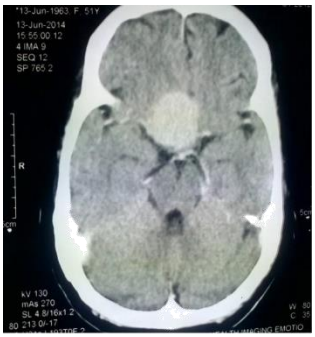
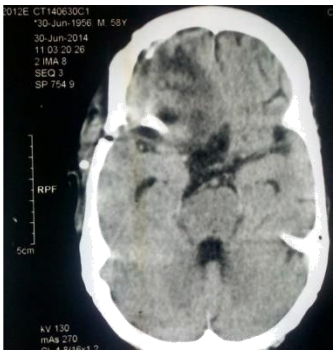
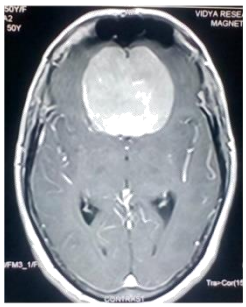
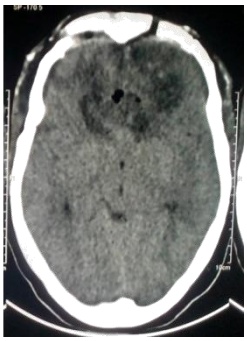





FIGURE 3. Pre op and post op CT of posterior fossa meningioma FIGURE 4. Pre op and post op CT of sphenoid wing meningioma

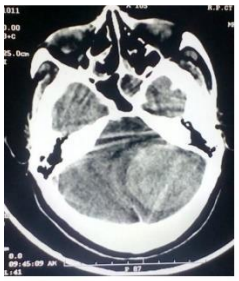
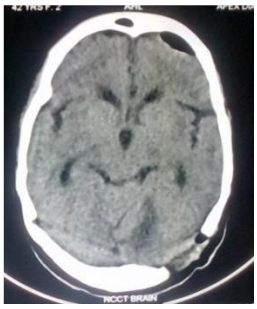
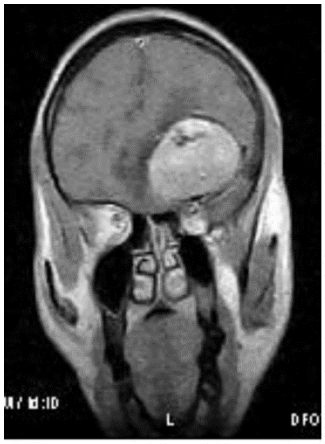
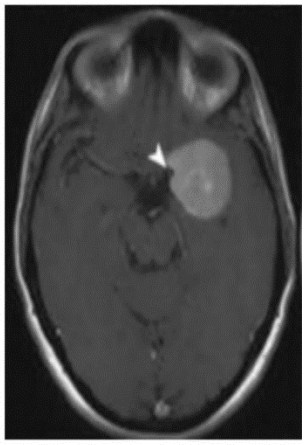





FIGURE 5. Pre op and post op CT of frontal convexity meningioma

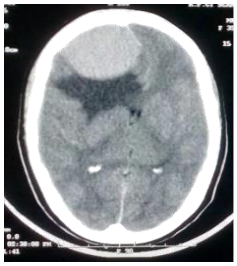
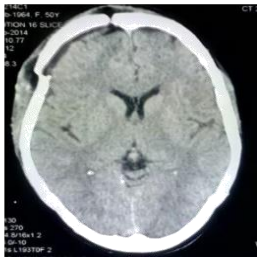
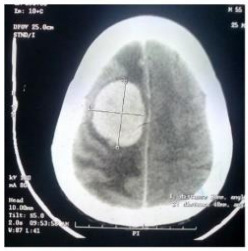
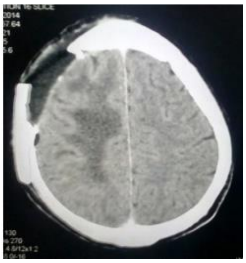
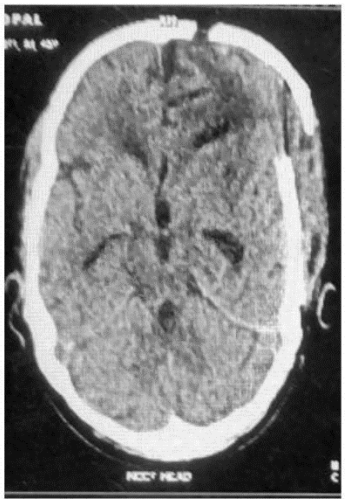



FIGURE 6. Pre op and post op CT of para saggital meningioma



Discussion

Meningiomas constitute 25 - 30% of all CNS tumours and are the most common tumour arising from the meninges (6). In our centre out of 764 cases of CNS tumours, Meningiomas constituted 200 (26.17 %), similar to studies by AB, Shah et al (7), Ruberti R F (8), Intisar SH Patty et al (9), Shrilakshmi 25.25% and Ejaz Butt et al (10). Women are more likely to develop a meningioma, (5) as in our study, females were more commonly affected 108 cases (54%) compared to males 92 (46%). A female preponderance for meningioma correlates with an endogenous hormone level and exogenous hormone replacement in postmenopausal women (in whom an increased incidence of meningioma is seen) as compared with postmenopausal women who have not taken exogenous hormone replacement therapy.

The present study revealed that the incidence of meningioma was common in the age group 41-60 years 48% of patients. The mean age was 48.54 years. In the studies done by A B Shah et al (7), Shrilakshmi (2), the most common age group involved were also 40-50 years.

Meningiomas in children are less common (11), and in our study, there were only 9 cases of meningiomas in children of age group 11-20 years. The intracranial location of meningiomas were distributed as to be the convexities were commonly involved 40%, in which frontal was more common, 45.45%, followed by the parasagittal and falcine meningioma were 20%, 10% were in sphenoid wing, 11% in CP angle and posterior fossa. In a study by shrilakshmi et al, 61.11% of tumours were located in convexity. The clinical presentation of meningiomas, depends on tumour location (12). The symptoms at presentation are rarely precipitous, but often insidious. Onset of slowly evolving headache is common and usually not associated with other symptoms suggestive of raised intracranial pressure, reflecting the slow growth of these tumours. A history of partial seizures is common for convexity meningiomas and an insidious personality change that is confused with dementia or depression is common in patients with large inferior frontal meningiomas (4). In our study, the most common clinical symptoms were headache, seizures and vomiting. The common radiological findings were mass lesions with pressure effect on adjacent structures and peritumoral edema.

Meningiomas divided in wide variety of histological patterns. Our present study revealed that the most common histologic type was meningothelial (38.89%), similar to studies by Nasrin Samadi et al (13) Sangamithra et al (14), Thomas Backer et al (15), followed by atypical meningiomas (16.67%). The other variants were fibroblastic (11.11%), transitional (11.11%), psammomatous variant, angiomatous, lympho-plasmacytic and fibrous (5.56%) each. According to WHO (5) atypical meningiomas have more than three of the following features – increased cellularity, smaller cells with high N/C ratio, greater than 4 mitotic figures/ 10HPF, prominent nucleoli and geographic necrosis. In our study (16.67%) of atypical meningiomas were reported. Singh Avninder et al (16) reported that papillary meningiomas and anaplastic meningiomas are rare and constitute 1 – 2.5% of all meningiomas. In the studies done by S Hoon et al (17) and Gottfried et al. (18) Histological analysis reveals that 80–90% of meningiomas are benign [World Health Organization (WHO) Grade I], 5–15% are atypical (WHO Grade II) and associated with a marked increase in recurrence. Only 1–3% of the cases become anaplasic or malignant (WHOGrade III), developing a high tendency to invade brain structures, metastasize, and recur. In our study, 16.67% of atypical meningioma was observed. Though meningiomas are considered to be benign tumours, recurrence is frequently observed (19). Benign meningiomas can recur following incomplete resection, if large and associated with monosomy14 and del (1p36). The extent of surgical resection depends on the size of the tumour, site, and its relation to vital structures. The best accepted system for prediction of recurrence is the Simpson grading system for completeness of resection (20), which evaluates the invasion of the venous sinuses, tumour nodules in adjacent dura, and infiltration of unresected bone by meningothelial cells. The recurrence rates that Simpson refers to 9% for grade I, 16% for grade II, 29% for grade III, 39% for grade IV, and 100% for grade V, respectively.

Simpson's scale of grading divides the extent of resection into 5 grades:

Grade I: Complete removal
Grade II: Complete removal with coagulation of dural attachment
Grade III: Complete removal without coagulation of dural attachment or resection of involved sinus or hyperostotic bone

Grade IV: Subtotal resection
Grade V: Decompression biopsy.

For patients with resection grades IV and V, endpoint for recurrence was enlargement of the remaining tumour, shown on MRI or CT. In addition, histological characteristics of malignancy such as peritumoral brain edema, cellular pleomorphism, nuclear atypia, presence of macronuclei, atypical mitoses, increase of neovascularization, brain invasion and necrosis, favour recurrence rate of meningiomas (20).

The treatment in grade I meningioma is total resection. In grade II and grade III meningiomas (2), surgery and adjuvant radiotherapy are the treatment of choice. Extent of surgical resection is one of the most important factors in predicting recurrence along with histological grading.

Bone flap removal was done for 2 cases due to intraoperative brain swelling. Immediate complication was haematoma in 2 cases (3.84%), for which reexploration was done. Major post-operative complications in our study were convulsions 21.1%, wound infection 17.3%, CSF leak in 9.62%, meningitis in 11.53%, of cases. All the patients before surgery were adequately treated with anti-convulsive therapy. Postoperatively 15% of cases developed convulsions within 48 hrs after surgery. They were controlled with increase in the dose of anti-epileptics or addition of another antiepileptic drug. The major morbidity in our series was post-operative infection, in the form of wound infection, CSF leak, and meningitis.

Follow-up period was 6 months to 5 years. Cases were followed up with CT brain in symptomatic patients. Twelve cases of recurrence are noted on follow-up for which incomplete resection was done. Anyhow follow-up period was not enough to assess the recurrence as meningiomas are slow growing tumours.

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Introduction of triage. An experience of a triage nurse in a tertiary centre in Japan

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ABSTRACT

Emergency medical treatment in Japan is subject to jurisdiction by the Fire Department. Triage, by definition, is a dynamic process, as the patient's status can change rapidly. Triage is very important for Japan, where emergency patients are on the rise. The role of triage nurse is also important. That will improve the life-saving rate of emergency patients and improve the reversion to society.

Keywords
emergency,
role of triage nurse,
tertiary centre in Japan

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INTRODUCTION

Emergency medical treatment in Japan is subject to jurisdiction by the Fire Department. Patients being transported by ambulance will be treated as soon as they arrive at the hospital, Japanese ambulance can be requested for free. In recent years, the rate of emergency vehicle dispatch and emergency patients tend to increase year by year.Triage was introduced to Japan so that medical treatment can be provided to patients who really need urgent.

HISTORY OF TRIAGE

Triage, by definition, is a dynamic process, as the patient's status can change rapidly. Patients may enter the triage stream at any point1. Urgency scales of the world are being developed in the United States in 1994, Australia in 1993 and Canada in 19953. Since around 2012, operation of JTAS became popular, and it was introduced in many hospitals. Our hospital is an emergency designated hospital.

JTAS (JAPAN TRIAGE AND ACUITY SCALE) [4]

Japan focused on CTAS (Canadian Triage and Acuity Scale)2 in Canada. CTAS is a highly credible scale that is progressing in North America, Asia and Europe. Japan developed and introduced JTAS based on CTAS. Emergency patients such as brain surgery diseases and cardiovascular diseases are 8507 in one year in Japan [5]. We aimed at improving the

the life-saving rate of our hospital. Therefore, from 2018 triage was introduced.

Components of JTAS

Pre-hospital treatment

As a pre-hospital treatment, there are doctors, car and doctor helicopter. The emergency system in Japan, ambulance and walking patients are treated in the same place. The emergency medical system in Japan is different for each facility

Patients who go to an emergency hospital are patients with mild to severe cases and urgent cases. Among them, as many as 1% of the patients who walked into the hospital waiting for a sudden change are hidden. There is a danger of missing the patient's life crisis if you are consulting in order of acceptance. Moderate emergency patients are accepted for 24 hours.

Triage method [6]

Triage will walk to the patient who come to the hospital, the triage nurse will receive the patient.

1. Triage fast touch. After reception, the nurse first responds, at that time, she judges the severity and urgency is necessary or not. If it is an emergency, she starts the treatment immediately. In case of infectious diseases, patients should be isolated.
2. We ask for chief complaints, followed by vital sign measurements, interview, objective findings, etc and the patient is judged according to JTAS level. Patient is asked to wait in the waiting room or monitoring room if monitoring is necessary.
3.After triage, I will consult the patient, when the turn comes

Triage nurse

A nurse performing a triage is called a triage nurse there are 30 nurses involved in triage. Nurses' years of experience ranged from 5 to 30 years. In order to introduce triage, it was necessary to confirm the ability of the nurse. What is sought are medical knowledge, first-aid skills, cooperation with other occupations, judgment.

Training for triage

In introducing triage, I confirmed the triage knowledge of the nurse many nurses do not know hospital triage. Depending on the years of experience, there is a difference in ability to do triage. Training was given based on:

What is TRIAGE
⇩
Urgent degree and severity
⇩
Merit by the triage
⇩
Perform in the triage
⇩
About Qsofa [7]
(Quick sepsis related organ failure assessement)

Conclusions

Triage is very important for Japan, where emergency patients are on the rise. Among the medical staff, fast touch to the patient is triage nurse, who will improve the life-saving rate of emergency patients and improve the reversion to society.

Table 1

	Work site	Job Description
Intensive care type	ICU	Intensive care of severely hospitalized patients
ER type	ER	All emergency medical examination
Each department-type collaborative type	From ER to ward	Treatment from initial care to ward

Table 1

Level 1	Resuscitation: immediate treatment
Level 2	Emergency: Rapid treatment
Level 3	Semi-Emergency: First-aid measures may be necessary
Level 4	Low Emergency: Treatment within 1 hour to 2 hours
Level 5	Non-Emergency: No Urgency

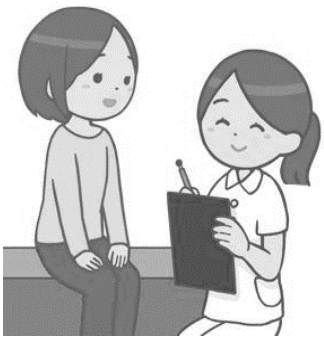

Figure 1
Fast touch.
Assessment of severe complaints

FIGURE 2
Vital sign measurement
Medical interview

FIGURE 3
Medical examination

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Surgeon specialist branding or what do surgeon specialists need to know about their brand

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Abstract

As the world has widely adapted the principle of searching for relevant information online, many practicing physicians, especially those who are involved in specialty - must pay a close attention to their brand and their reputation on the World Wide Web. There are numerous web pages available to the general public about particular physician and where he/she practices. Most of those pages are either managed by a third-party company or created for promotional use. Some physicians are not even aware of this and are found by surprise when their patients bring up the information, they found on them online. Physician's name is a brand that he or she carries throughout entire medical career, and it might make or break the legacy of academic or private physician on the long run.

Keywords
physician branding,
physician leadership,
healthcare management

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Introduction

The modern physician leadership skills have tremendously shifted from academia to patient advocacy within the hospital walls. Nowadays, modern physician leaders, particularly surgeon specialists are required to have a proper information about their brand online, if they want to actively engage in the patient advocacy, improvement of medical techniques, drive better brand awareness about their practice or ultimately raise a better return on investment for their department.

By owning and optimizing physician's brand image online, physician showcases his/her professionalism exactly the same way, as it would be presented in the real world. A first impression is an exact trademark – how patients as well as colleagues are going to perceive a particular physician, whether it's online or in-person.

Method

This research publication demonstrated the latest techniques and practices that available for surgeon specialists to implement on their

behalf - in order to improve their branding or reputation online. The strategy employed in this paper is widely used by marketing professionals and could be applied to any surgical subspecialty to improve physician's brand awareness and recognition.

Basic online search

A very first step in identifying your presence online is to perform a simple online search. By searching your name in any popular search engines such as Google, Bing or Yahoo will bring the results associated with your name. By doing so, a physician can see what comes up, what hospitals or educational institutions he or she is associated with, physician ratings and comments, social media profiles etc.

This is a very important first step that any physician should take in order to see where he/she stands online, as this helps to understand where the work needs to be done and how to improve or make it better. During a quantitative online research of specialist's name, it's advisable to make priority notes on troubling websites and negative reviews that come up on the first 3 pages of search engines. The theory strongly suggests performing a search for up to 3 pages, as less than 10% of people online go beyond 3rd page of Google [1]. By creating notes on troubling websites first, a doctor creates a visual picture where he/she needs to do some work. As a rule of thumb, most of the troubling websites where most of the physicians find surprising ratings are on physician rating websites such as Google Business, Yelp, RateMDs, Vitals, HealthGrades, Facebook, ZocDoc, CareDash, Angie's List and more. When we surveyed surgeon specialists whether they were aware of their online reputation score on the rating websites, most of them didn't even know that their name was associated or mentioned there. That's why this very first step of identifying your online weakness, is a crucial step in your journey on better branding in general.

The next step after identifying your brand vulnerabilities online is to make notes on the websites that provide accurate and positive information about doctor's name. There will be information that is most likely associated with your residency ties, medical school or current/past employment webpages. If these pages still show up on the first three pages of search engine, it's a good indicator of brand awareness online.

Name/brand ownership

Once the troubled as well as properly informative websites were noted, it's time to visit those websites and understand who owns these websites, what comments prevails there and how you can get a full ownership of the given information. It was interestingly noted, that some websites incorrectly identify physician's specialty and association with the hospital. Those misinforming websites should be contacted directly for correction or reported for misleading information. What every physician needs to understand is, that if his or her information is found online and they do not own it, then somebody else does. If this situation persists and it's not corrected right the way, it can potentially ruin physician's reputation, as random negative content could be fed directly to the search engines on daily basis. That's why it's very crucial to own your personal information online.

Dealing with incorrect information and negative reviews

Once the list of good and bad websites is established, a physician should proactively engage with the bad ones first. Generally, every legitimate website should have a "contact us" page where any technical errors could be addressed. This is where you need to email or sometimes-even call to get your information corrected. Once it's done, the next step will be addressing negative reviews.

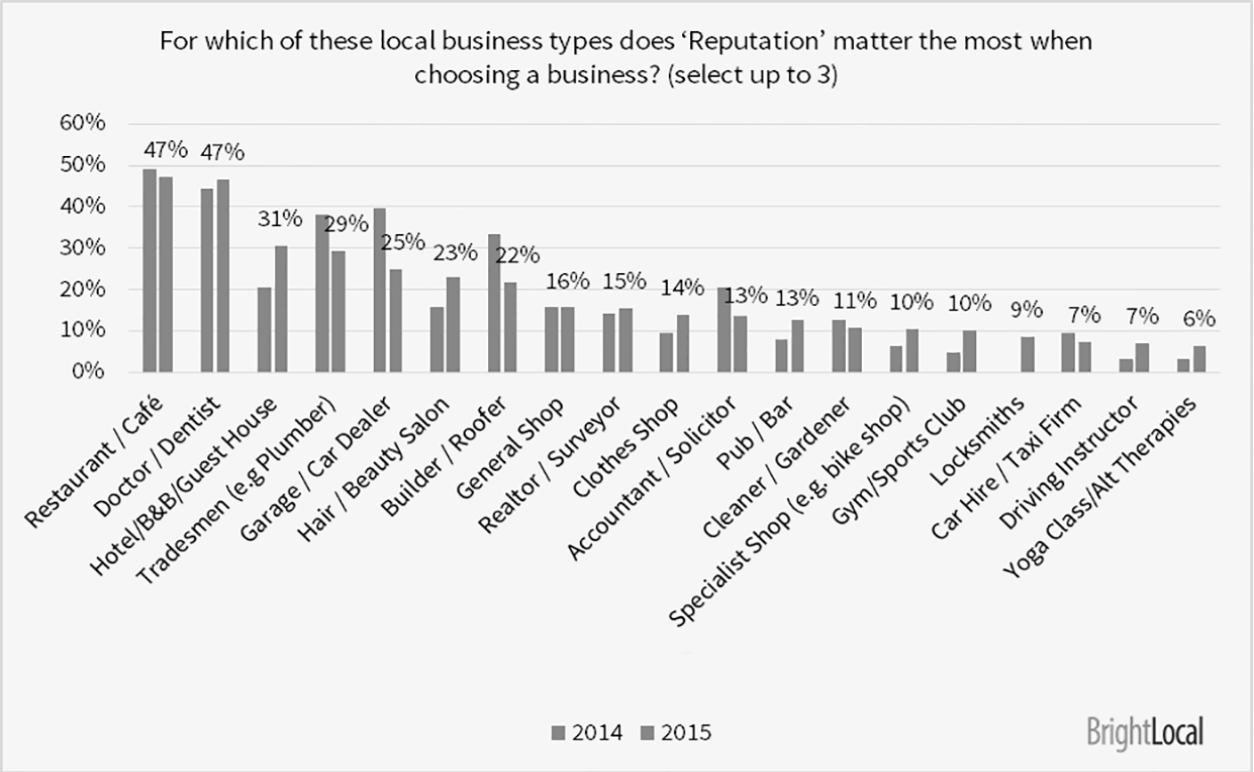
To address negative reviews, a physician should clearly understand why a negative review occurred. On some review websites such as Yelp, a doctor can message a reviewer directly about given feedback online, in order to smooth the situation and potentially turn a negative review into a positive. However, there are some situations, where you get no response from the user at all. In this case, it's advisable to leave a public reply/feedback with thorough explanation why the misunderstanding has occurred. This way a professionalism and constructive criticism should be taken into consideration before leaving a public comment. Being professional in person and online is an essential tool for any respected physician of the modern world, this is where a parallel between virtual and real world crosses the path.

If physician struggles with the negative reviews online, it's advisable to fully optimize social media networks. A logical reason to optimize or sometimes

even create more social media profiles is to push negative websites further down to the 3rd, 4th pages of search engine. Social media has one of the highest online traffics out of the entire Internet; therefore, social media websites are the ones that usually show up on the top pages of any search engine.

By optimizing those social media profiles, physician can create more positive brand awareness, regardless of the prior negative reviews. To support statement on importance of online reviews; a study that was conducted by BrightLocal - demonstrated that out of all businesses online, 47% of general populations look at the reputation of doctor before making a decision on primary visit (FIGURE 1).

FIGURE 1



The anatomy of a good medical brand

Any reputable healthcare brand online has five main points, these points are:

1. Professionally done website optimized for mobile devices.
2. A unified (consistent) high definition profile and cover picture throughout all websites, including social media.
3. Academic CV or biography.
4. A website or information website link on how to contact physician.
5. Shared relative information associated with physician's specialty and accepted insurance.

Ideally, these top 5 points must be present throughout all the active websites where physician's information is found online.

Outside of those points, it's advisable to establish an online physician's niche, just like a specialty that physician specializes in. Whether a physician would like to be found a thought-provoking leader or just share the latest news from the industry or his/her own practice – but it has to be consistent [2]. Any physician who is actively engaged with his/her own brand sees a phenomenal potential for growth not only academically but also in the professional world of medicine [3]. An understanding the physician's strength and capability will certainly create a great brand awareness and recognition for physician's career on a long run.

Personal vs. professional use of social media network

Some physicians who experienced social media probably understand there are 2 main differences between personal and professional use of social media networks. For example, a Personal profile on Facebook is completely different from Business

profile. This is where many physicians fail to separate two. A business Facebook page allows physician to see engagement with audience, advertise a promoted content, see statistics etc [4]. While personal profile is mostly used to connect with friends and family. So, it's very important to separate these two and the same rule applies to the widely used physician networks such as LinkedIn and Instagram.

Results

Optimization and ownership of surgeon specialist's identity online can make or break the outlook on physician's long-term career. It's important to own and correct misleading information online right the way, in order to prevent further damage. A simple research and advocating for your own brand name online, as it was shown here, can tremendously increase surgeon specialist's reputation in the eyes of the patients and the entire world.

Discussion

In the competitive world of modern healthcare, where physicians take an active role

of advocating for their patients or simply would like to be connected with potential patients or colleagues, brand image plays a huge role in the era of Internet. A shift from the old-school word of mouth to the new way of searching for information about particular doctor, forced many physicians to pay closer attention to the marketing techniques in order to be properly recognized online for their identity and work. [5,6]

With open and free accessibility of the Internet [7], any person can now build his or her own brand, however, the same way this brand can be misrepresented or misleading to the general public. Any practicing physician should take this particular issue seriously, as it can trigger a multitude of unwanted results in terms of personal branding. A modern surgical specialist should be an active advocate of his or her own brand online as well as in-person. In this research paper we showcased how any surgical specialists can own their brand identity and what steps they should take in order to be successful. There are some limitations to this study, as evolving marketing techniques are constantly changing, however the basic principles of marketing strategy on branding and brand awareness remained the same for the last 5 years.

Conclusion

As Internet plays a huge role in everyday life, owning and protecting your brand identity is becoming a crucial step for any well-respected physician specialist.

A full optimization and ownership of physician's brand name showcases a better correlation to a positive image online and drives better return on investment on the long run. Surgeon specialists who are actively sought online by patients [8] and professional medical colleagues will have an ability to correctly represent who they are and what they stand for in the most professional and positive way.

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