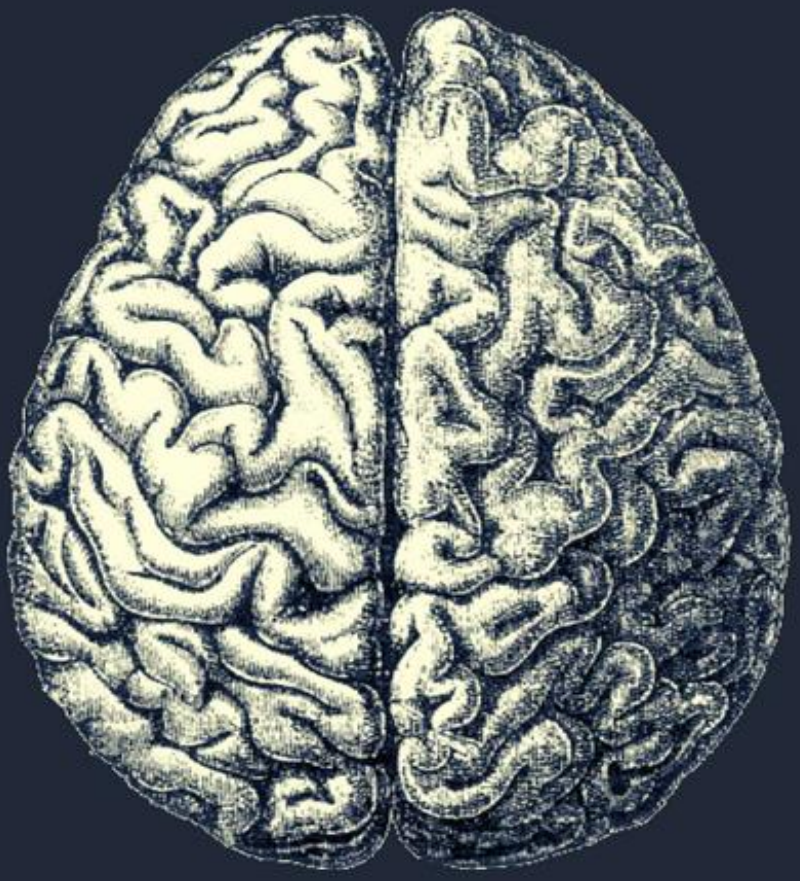
ISSN 1220-8841 (Print)
ISSN 2344-4959 (Online)

ROMANIAN NEUROSURGERY

Vol. XXXIV | No. 3 September 2020



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

LONDON ACADEMIC PUBLISHING

Editorial and Advisory Board

ROMANIAN

NEUROSURGERY

Vol. XXXIV | No. 3 September 2020

London
Academic Publishing

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

First Printing: September 2020
London Academic Publishing Ltd.
27, Old Gloucester Street
WC1N 3AX
London, United Kingdom
Email: contact@lapub.co.uk

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Contents

Romanian Neurosurgery (2020) XXXIV (1): pp. 373-378
DOI: 10.33962/roneuro-2020-058
www.journals.lapub.co.uk/index.php/roneurosurgery

The 3D printed models technology for the management of intracranial aneurysms

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ABSTRACT

Management of intracranial aneurysms is still a therapeutic challenge, especially in cases of complex lesions. Thus, the improvement of the study and intervention planning possibilities correlated with the access to continuous professional training based on simulation and clinical diversity represent optimal conditions for the efficient solution of this pathology. The development of three-dimensional printing technology offers a new opportunity in the modern treatment of intracranial aneurysms. The aim of this study is to present some aspects related to the materials and methods of manufacturing simulation models of individual 3D printed aneurysms and their influence in the optimal management of these lesions.

Keywords
intracranial aneurysm,
3D printed models

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INTRODUCTION

Intracranial aneurysms are increasingly complex vascular lesions, both in terms of their shape and relationships with parent and adjacent vessels. These will require a much more elaborate interventional treatment, based on a much more careful anatomical study and a much clearly interventional planning.

The three-dimensional (3D) printing technology is a promising technique with more and more applications in the field of medicine. The development of 3D printing technology provides a new perspective for the treatment of intracranial aneurysm. The intracranial aneurysm 3D printing simulation models created on the basis of 3D angiographic imaging acquisitions offers technical, practical and educational support to both neurointervention specialists and young residents in professional training.

The aim of the present study was to presents our experience concerning the materials and methods of producing 3D printed individual aneurysm model and its significance in the treatment of intracranial aneurysm.

ANEURYSM MODELING

Angiographic imaging data generation and post-processing

CTA and 3D DSA images data acquisition were used for intracranial aneurism investigation and therapeutically management. Dynamic CTA images were acquired using a Toshiba Aquilion 32 CT scanner (Canon Medical Systems USA, Inc.). The scanning parameters used are: scan range - 16 cm, gantry rotation time 0.75, slice thickness 0.5 mm, field of view (FOV) 240 mm, tube potential, 120 kV and tube current 218 mA. A mean total scanning time was 21 s and DLP 930mGy. The 3D DSA images acquisitions were obtained on a clinical biplane C-arm System Toshiba Infinix (Canon Medical Systems USA). A 5 s conventional mask and fill run protocol (70 kVp, 0.36mGy/Fr, 200° and 133 images were used. The injection parameters were 2.5 mL/s for 7 s with 2 s X-ray delay using 100% contrast concentration.

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Anatomical 3D computer model reconstruction

The original 3D CTA or DSA imaging dates, in DICOM format, are extracted through the Picture Archiving and Communication System (PACS; eRAD, China) and imported into Mimics (Materialise 18, Leuven, Belgium), a medical reconstruction software suite. Mimics allowed the reconstruction of only the intracranial arteries or both arteries and the skull in the case of CTA after format conversion and threshold extraction. The image threshold must be carefully adjusted to expose the image of the vessels as clearly as possible. The method of threshold segmentation is combined with manual segmentation to obtain the most important region of interest (ROI). Also, surrounding tiny branches or interfering bony structures could be removed for a better ROI exposure (aneurysm region). Then, we used the 3D calculate function to reconstruct the computer model. It is then stored and sent out as STL format files.

Aneurysm 3D printing model fabrication

The STL format file is input into the rapid prototyping 3D printer machine (Objet Connex350 3D printer Objet Technologies Ltd, Rehovot, Israel) for virtual model fabrication. If plaster was initially used to make a solid model including the skull, blood vessels and aneurysm, they proved too rigid and fragile to simulate human blood vessels. Thus, to create a suitable model of aneurysm it is necessary to use three different materials for the reconstruction of the solid skull, flexible blood vessels and eventually the empty aneurysm. The new types of 3D printers Objet Connex500, offer the possibility to work with a mixture of rigid and flexible materials. A light-cured resin material for bone reconstruction and a different flexible resin material for vessels and aneurysm modelling were used. Both types of 3D models were manufactured with this method.

Interventional planning

All manufactured 3D models were studied by the team of vascular neurosurgeons in order to establish the most appropriate therapeutic approach. The shape, dimensions, orientation and relationships between the aneurysmal sac, its neck, the carrier vessel and the adjacent branches were carefully analyzed to establish the optimal interventional planning for each lesion. A microsurgical clipping or endovascular occlusion was decided. In case of microsurgical clipping the 3D model was used to choose the type of clips, to simulate access and clipping direction, and in more complex lesions to establish the optimal clips arrangement for a complete closing of the aneurysmal neck. For endovascular treatment, the printed 3D models helped both to choose the coils and the proper endovascular technique of aneurysms occlusion.

Images were exported in standard digital imaging and communication in medicine (DICOM) format to the 3D calculation software, Soft Mimics17.0 (Materialise, Belgium). Soft tissue and brain tissue around the skull was removed. The vascular and bone area was segmented by the software, and the virtual 3D angiogram was generated. The skull, intracranial artery and aneurysm were distinguished by different colors. Data from the segmented area was transformed into a STereoLithography (STL) format, which was used in the rapid prototyping machine.

CASE PRESENTATION

A 43-years-old female patient was addressed to our Emergency Unit for sudden violent headache followed by a short episode of loss of consciousness 24 hours ago. At the neurological examination the patient was evaluated with GCS of 13 points presenting nuchal rigidity, confusion, somnolence, and no motor deficits. She was immediately investigated by CT scan that showed a subarachoidian hemorrhage in the basal cistern and both Sylvian fissures. The patient also presented intraventricular hemorrhage in the fourth and third ventricle (Figure 1).

After that an angio-CT was performed that revealed an anterior communicating artery aneurysm as a source of bleeding. (Figure 2). A 3D printed simulation model that comprised of entire vascular tree of aneurysm and parent artery and its branches was immediately manufactured. The 3D model was carefully studied by the neurovascular team and an endovascular aneurysm occlusion was decided for the next day after admission (Figure 3).

The patient was placed under general anesthesia by endotracheal intubation. Right femoral approach using 6F introducer sheath was performed and the right internal carotid artery catheterization was achieved with a 6F guiding catheter (Imager™ II, Boston Scientific). Different angulations biplane DSA series were obtained for an optimal working position. A road-mapping was used throughout the duration of the procedure for aneurysm occlusion. A microcatheter was then advanced over a microguidewire into the aneurysm dome. If the aneurysm remnant had a wide neck, the balloonassisted neck remodeling technique was used. 6 GDCs were then advanced and detached in the aneurysm until a complete agiographical occlusion was achieved (Figure 5). All catheters and the femoral sheaths were then removed from the femoral arteries and hemostasis was obtained by manual compression. Control cerebral CT scan was performed 5 days later. Anticoagulation was routinely continued 14 days after the procedure. The postoperative course was uneventful, with a progressive improvement of neurological status. After 14 days of hospitalization the patient was transferred to the Neurological department to continue the medical treatment.

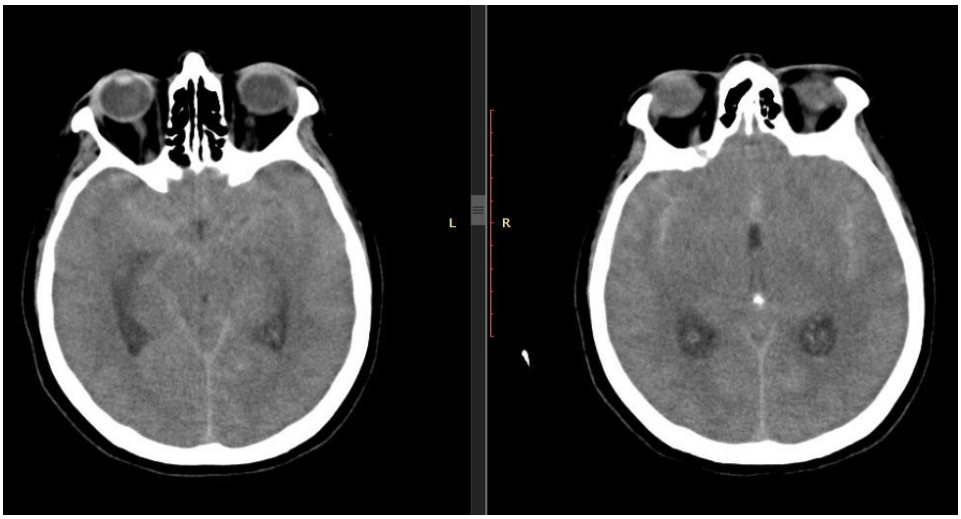


Figure 1. Diagnostic cerebral CT scan showing a SAH

Figure 2. Cerebral Angio-CT scan showing an anterior communicating artery aneurysm (arrow)

Figure 3.

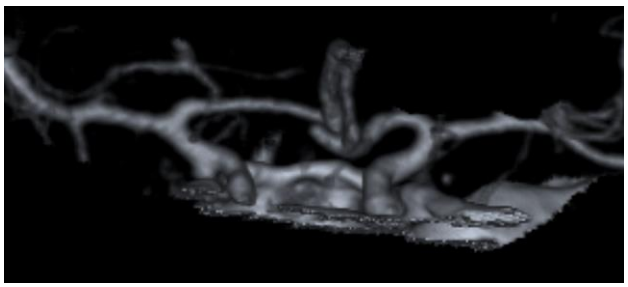
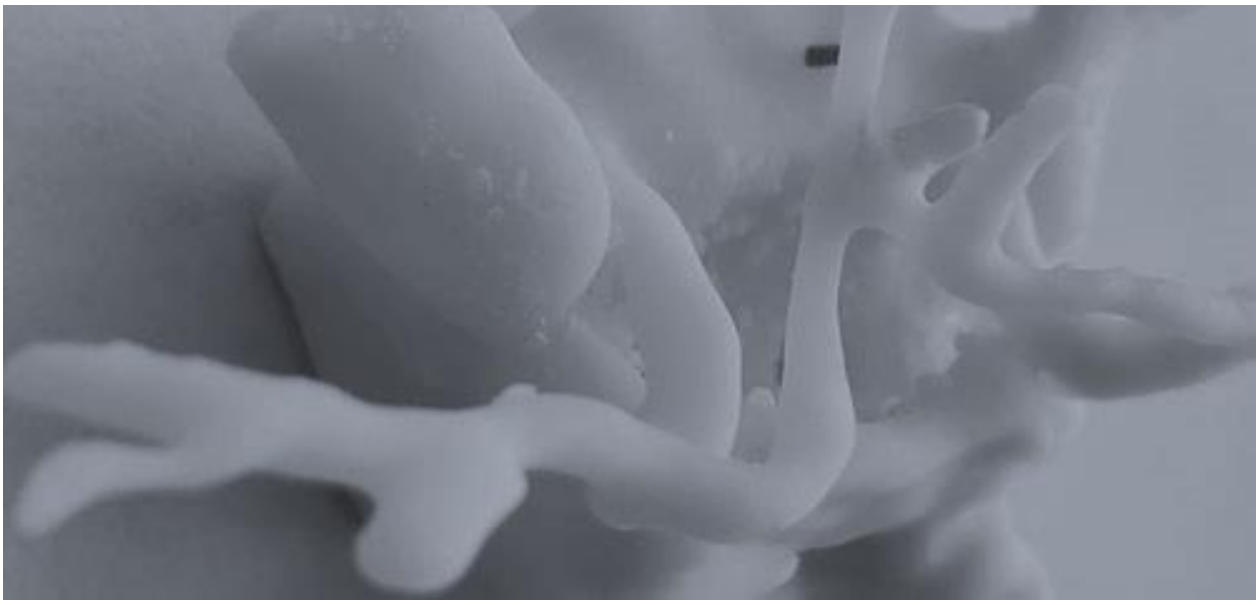


Figure 3. Different view of the ACoA aneurysm on 3D-printed simulation models



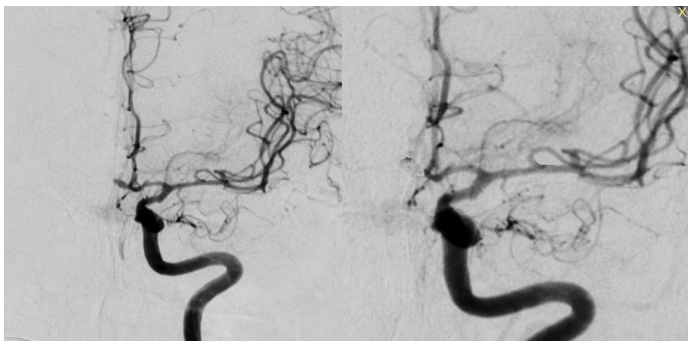


Figure 4. DSA images with the ACoA aneurysm pre and post coils embolization

Figure 5. Cerebral Ct scan control postembolizati

DISCUSSIONS

The development of materials and manufacturing technology through 3D printing has allowed the realization of vascular anatomical models as accurate as possible, with lower and lower costs. Gradually, the limitations of 3D printing technology represented by the creation of flexible vascular models of tubular type (hollow inside) were overcome allowing the production of high-precision anatomical articles with optimal haptic properties. Thus, 3D-printed simulation models based on DSA imaging can perfectly replicate the geometries of aneurysms, parent vessels, and adjacent vascular branches. The reconstruction and 3D printing of implants / models specific to each patient were based on the correlations between digital images and finite element analysis. The data of the computer models thus obtained are finally transferred to a 3D printing unit for the realization of the simulation model. The use of modern printers that allow the simultaneous use of various materials, sequential multilayer modeling technique or rotary model printing technique have contributed greatly to the introduction of this technique both in the current practice of treatment of intracranial aneurysms and

in the professional training of vascular neurosurgery specialists[2,3,4,6].

Although major advances have been made in the treatment of cerebral aneurysms with or without subarachnoid haemorrhage, therapeutic management remains one of the most challenging decisions, especially in complex anatomical situations. Numerous studies have shown that the main determinants in establishing the therapeutic strategy are the angioarchitecture of the lesion (size, location and direction of the aneurysm itself), the anatomy of the pathway and preprocedural clinical status (WFNS or Fisher's degree of the patient) [8]. Ripley et al [9] reported that 3D printed simulation models make a major contribution both to decision-making and to the completion and efficiency of traditional techniques for the treatment of intracranial aneurysms.

With the development of the 3D printed simulation models that allow realistic reconstruction of the shape of the aneurysm, important changes influence the traditional aneurysm treatment not only on the fields of microsurgical clipping but also on the endovascular coiling.

If the microsurgical clipping treatment strategy was in the past exclusively based on an imaging analysis with or without 3D computer reconstruction, at present, the possibility of clip model selection (as shape, size and curvature), the clipping direction and clips successive arrangement for aneurysmal neck reconstruction are favoured by the realization of 3D printed simulation models.

In the case of endovascular interventions, the realization of simulation models by 3D printing can be a valuable tool both for the optimal choice of the implant type (whether we are talking about coils or stents) and for predicting possible vascular deformations due to endovascular implant insertion. This can help improve the intervention plan, avoiding possible complications by using inappropriate and unnecessary materials [6, 7]. In the case of difficult interventions such as stent-assisted coil occlusion in the basilar trunk or complex vascular bifurcations, their planning and vascular reconstruction are difficult, accurate mounting and positioning of implants are essential to allow complete aneurysmal occlusion while maintaining permeable its branches [5,6].

A retrospective study on the efficient use of endovascular materials showed an increase in costs of up to 30% when it was necessary to perform immediate emergency interventions compared to cases in which they were performed after a thorough study on 3D imaging and simulation model by 3D printing. The use of oversize or undersized implants, of additional types of devices not initially anticipated compared to those used in a standard procedure, lead to an increase in the average cost of some classic interventions. The authors concluded that an adequate preoperative planning based on an imaging and model study, correlated in certain situations on practical simulation on the model lead both to a minimization of costs and to an improvement and efficiency of the interventions results [1,4]. The cost of producing 3D printed models seems much more reasonable especially compared to the cost of an endovascular material.

Improving the planning of interventions based on 3D printed simulation models has led to the development of neurovascular training procedures (microsurgical clipping and endovascular embolization) for specialists in their professional training period. The growing limitation of access to cadavers or to a sufficient number of neurovascular intervention procedures was perfectly offset by the development of increasingly high-performance vascular models both as a material and as a design possibility.

Conclusions

3D printed simulation models offer the possibility of optimal visual exposure of intracranial aneurysms to help neurointerventionists for application of a more appropriate therapeutic strategy. Also, creation of anatomi¬cally accurate 3D printed aneurysm models demonstrated its utilities for continues professional training. Access to fast-printing 3D machines and materials with advanced properties at lower and lower costs will certainly lead to the increasing use of this technology in the therapeutic management of intracranial aneurysms.

Acknowledgment

This study about intracranial aneurysm 3D printing simulation models applications in vascular neurosurgery is the subject of the grant: "New diagnostic and treatment methodologies: current challenges and technological solutions based on nanoparticles and biomaterials", that won the 2017 complex projects completed in consortia CDI, grant number: PN-III-P1-1.2-PCCDI-2017-0062, funded by CNCS –UEFISCDI Romania.

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Romanian Neurosurgery (2020) XXXIV (1): pp. 379-385
DOI: 10.33962/roneuro-2020-059
www.journals.lapub.co.uk/index.php/roneurosurgery

Organizing a microsurgery workshop for residents

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Keywords
experimental microsurgery, training, laboratory

ABSTRACT
Microsurgery represents an important branch in Plastic and Reconstructive Surgery. It involves fine skills which doctors need to repair nerves, vessels and thus being able to perform replantation and transplantation of different types of tissue. After traumatic injuries, a plastic surgeon is capable of either coverage of the exposed noble tissue or can perform the replantation of the amputated limb using microsurgery.
This field can be very challenging at the beginning, but very rewarding in the end. The utility microsurgery is quite vast; however, the skills to perform such surgery require a lot of training beforehand. Before doing any replantation or other tissue transplant in humans, it would be recommended that a surgeon should have a basic microsurgical course completed and afterwards several hours of practice in front of the microscope. Last but not least, one should also test the skills acquired in vivo, in order to improve and perform the correct manoeuvres from the beginning.
In order to do this, a plastic surgery trainee must therefore have a dedicated laboratory where he/she can practice this art. This place should be quiet, equipped with microscopes and microsurgery instruments and authorized to perform experiments on live animals.

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INTRODUCTION

A growing interest in the field of microsurgery has led to the development of training facilities, where residents can practice microsurgery. [1] The development in technology has led to new ways of doing microsurgery, such as using a smartphone to use magnification. [2]

Although it is mainly addressed to the plastic surgery trainees, microsurgical skills are also useful in neurosurgery, ophthalmology,

cardiovascular surgery, orthopedic surgery and even general surgery or urology (for liver and kidney transplant).[3],[4],[5]. A formal curriculum of microsurgery in the plastic surgery program proved to be very useful according to Mueller et. al. [6]

For this reason, there are several microsurgery centers in Romania, each having a similar basic program focused mostly on nerve and vessel repair. In time and with experience, these initial programs are developed and divided into more complex courses – involving the dissection of smaller vessels (lymphatic vessel or perforating vessel), with direct applicability in the clinical practice.

MedWorkshops Center

Materials and methods

In order to begin organizing such course, a suitable location is required. The location needs to have a veterinary employed in that facility (which is a compulsory condition for the other requirements). It would also be more comfortable for the participants if the location were far from loud noises and distraction, as microsurgery requires attention and calm.

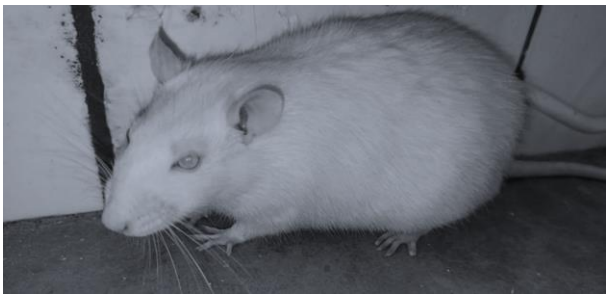
Due to the legislation, there are several approvals needed for research or medical education involving living animals. Every project has to pass the ethics committee and afterwards get the approval from the Veterinary Department. In Romania, this process may take up to 4 months and should be planned carefully before the beginning of the course.[7] Every major project which involves a teaching activity should be performed under the supervision of the Medical University or a recognized professional association.

For the first edition of the microsurgical program, there was collaboration between 3 associations – the Romanian Multidisciplinary Residents Association, the Romanian Plastic Surgery Association and the Romanian Aesthetic Surgery Association. The course took place in 2 distinct locations – the non-living tissue experiments were performed in a private facility (MedWorkshops), while the second part which involved live animal surgery took place in CEMT –The Excellence Centre in Translational Medicine, part of the Fundeni Hospital. The course duration was 4 days (8 hours/day), over 2 consecutive weekends.

Excellence Centre in Translational Medicine

Once all the approvals were obtained, a curriculum of the course was sketched and the dates for the course established. The lecturers for the theoretical part of the course were invited with a 2 months' notice (at least) and asked for the name of their presentation. A number of 10 participants per course was considered to be suitable for a microsurgery course. The selection of the animal to be practiced on was done taking into consideration several criteria – suitable size and type of the tissue (nerves, vessels), affordable costs, easy for manipulation, easy to induce anesthesia, accessible to purchase from special laboratories. For all the previous

mentioned reasons, the Wistar rat is the ideal candidate. [8]



Wistar rat

The announcement of the course was done with one and a half months in advance. There were 3 channels by which the marketing was done – internet (official web sites of the associations involved, social media pages as well as personal pages), direct speech among colleagues, and posters in the hospitals which prepared residents in plastic surgery.

Poster

A list of all materials needed for the 4-day workshop was created. The list included instruments for the participants and trainer, other materials, logistics for the theoretical part, the material on which the participants train and food and drink supplies for catering. Based on the presumed amount of the materials used, a budget was established to support the expenses for renting of the microscopes, the facilities and acquiring all materials needed.

Sutured silicone tube

Table 1. List of requirements for organizing the course

Instruments & sanitary	Other materials	Logistic theory	Practical workshop
Insulin syringes	Black bags for waste	Laptop	Latex gloves
Syringes 10/20ml	Cages	Projector	Silicone tubes
Disinfectants (alcohol)	Cleaning paper	Folders & pens	Cherry tomatoes/grapes
Gloves	First aid kit	Diplomas	Leaves and petals
Tampons/Dressing	Cotton swabs		Chicken legs
Yellow cannulas	Cork plates		Wistar rats
Blue dye	Plastic wrap		
Anesthetics	Pins and paper clips		
Euthanasia solution	Rubber band		
Sutures 8.0-10.0	Tape		
Sutures 3.0-5.0	Electronic scale		
Microsurgical instruments (scissors, needle holder, forceps, dilator, clamps and approximator)	Yellow bucket for sharp materials		
Macrosurgery instruments (forceps, small scissor)	Yellow bags for contaminated waste		
Scalpel blades	Hair clipper		
Serum	Background material		
Hand sanitizers	Cups		
Microscopes	Adjustable chairs		

Another important aspect which needed to be taken into consideration when organizing such a workshop was the fact that acquiring live animals could be done only in special conditions and from designated institutions. These institutions allow the purchase of these animals only based on the previously approved documentation.

Being an initiation microsurgery course, it needed to remain at an entry-level, both the theory and especially the practice. The selection of the participants was based on prior experience in the microsurgery training as well as year of residency. Taking into consideration the entry-level of the course, the curricula was designed to meet the experience of these participants. The program was thought to have 1,5-2 hours of theory and 6-6,5 hours of practice daily. The level of difficulty of the procedures performed was also designed to increase daily.

Day 1	**Day 2**	**Day 3**	**Day 4**
Theory	Theory	Theory	Theory
Introduction in microsurgery	Vascular anastomosis	Nerve anastomosis	Clinical experience in peripheral nerve repair
Microsurgical instruments. Surgical technique.	Free flaps – clinical experience	Ethics in animal experimentation	
Practical part	Practical part	Practical part	Practical part
Latex glove sutures	Silicon tube sutures	Anastomosis of the femoral artery	Anastomosis of the femoral vein
Grape/Cherry tomato sutures	Vascular anastomosis on chicken leg		
Leaves/Petals sutures		Sciatic rat nerve repair	Anastomosis of the aorta
Elastic band suture			Anastomosis of v. cava

Table 2. Basic microsurgery training program

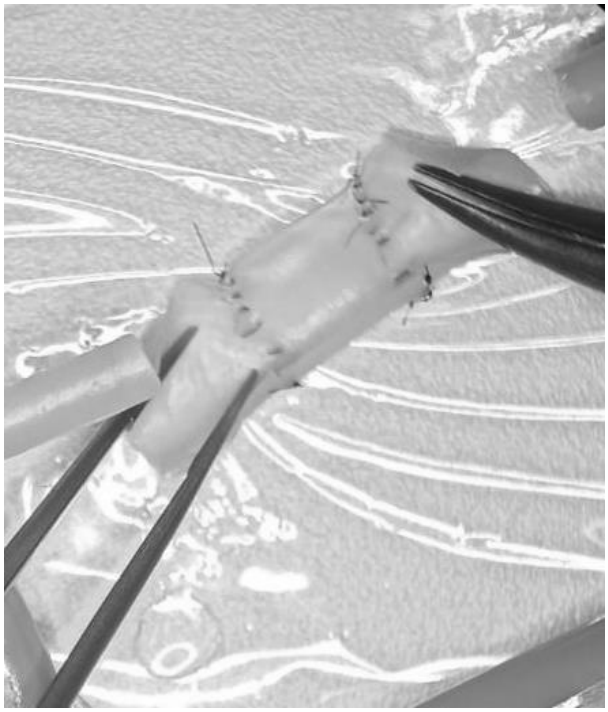
Another bureaucratic aspect of the workshop consisted of making individual folders for the participants. These folders contained the program, the list of equipment they received and for which they were responsible during the day, a file regarding work safety which they had to sign before the beginning of the course. At the end of the course the participants received a diploma and were requested to complete a feedback file.

Results

After making the formal announcement of the course, 20 plastic surgery residents sent an application for one of the 10 available places. 10 participants were selected based on their microsurgical experience and the other 10 were promised a place in the next course.

The residents were in different years of training (from the 1st to the 3rd), all but one having no former experience with microsurgery (one resident having previously participated to a 2-day microsurgery course). All participants attended all 4 days of the course, except one who could not come on the 4th day.

There were 4 lecturers who presented their clinical experience in the theoretical part of the workshop and the official organizer of the course who presented and demonstrated the practical experiments.



Latex tube anastomosis

Dissection on chicken leg

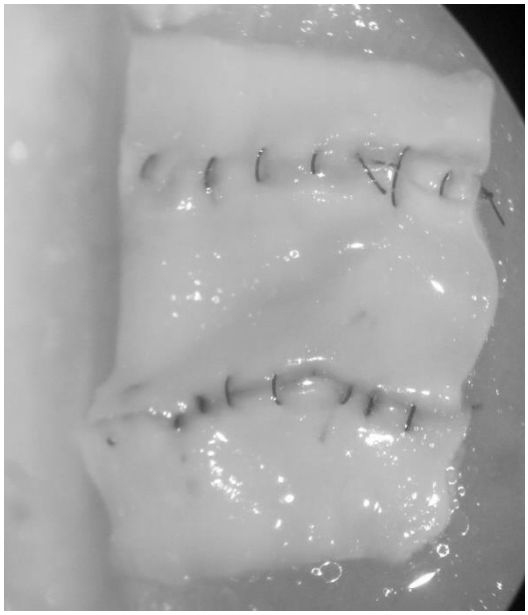
Sciatic nerve exposure

The theoretical lectures permitted the participants to learn the basics about the structure of a peripheral nerve and vessel [8],[9],[10] and also about the possibilities in the reconstruction of nerve defects, either with nerve grafts or conduits. [11],[12] Participants were also taught about the pitfalls in nerve microsurgery [13] as well as the basics in how to perform rat anaesthesia. [14]

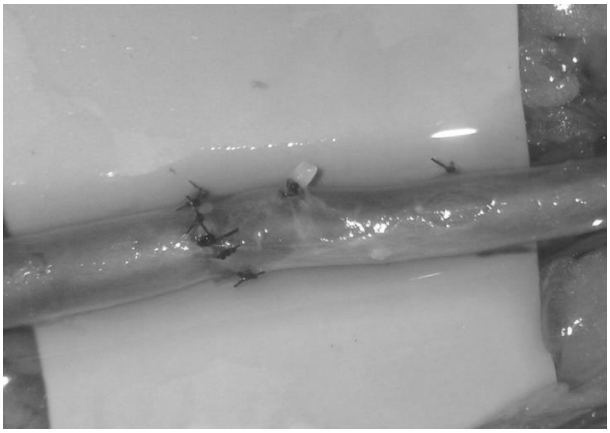
In the practical part, the residents were also taught some tricks which they could use in order to improve their outcomes. [15]

All participants improved their microsurgical technique between the first day and the final day of the course. All participants managed to complete the tasks required in different time-frames. [16] 8/10 participants managed to achieve a patent vascular anastomosis but all participants were able to perform a correct nerve anastomosis by the end of the program.

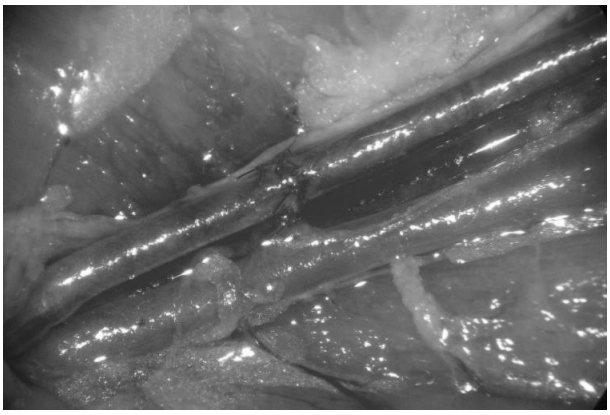
Dissection for rat aorta



Femoral artery in chicken leg (cut open)



Anastomosis for femoral artery



Participants during sciatic nerve repair on Wistar rat

Discussions

Having little experience, all the participants showed great interest in microsurgery, most of them staying the full 8 hours every day (some wanting to stay even longer). However, working long hours under the microscope was strenuous (some participants complaining about minor problems: back pain or headaches associated to eyesight problems).

Since the workshop was splint over 2 consecutive weekends, the more zealous participants could also come for an extra 2 days during the week to have more practice outside the formal course. This was a great opportunity to improve their surgical skills (especially in those who had no prior experience). In the formal feedback, some participants mentioned that they would have liked an extra day of practice on live animals, so that they would be able to achieve all perfect results.

Overall, the feedback was positive and the participants said they feel more confident with their microsurgical skills, thus having no issue in performing these procedures in their clinical practice. Furthermore, they mentioned that they would gladly attend a more advanced workshop if this was to be planned.

Although this was an entry-level course, more elaborated course could be developed in time, depending on the interest of the participants.

Conclusions

Both theoretical and practical knowledge is needed when it comes to learning microsurgery skills. These should first be learned in the safe environment of a laboratory, during a microsurgery course and then applied in clinical practice.

Organizing a microsurgery course requires special equipment, designated facilities and many approvals, but such courses are absolutely needed for the training of the medical residents.

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Romanian Neurosurgery (2020) XXXIV (1): pp. 386-390
DOI: 10.33962/roneuro-2020-060
www.journals.lapub.co.uk/index.php/roneurosurgery

Intracisternal papaverine toxicity in anterior circulation aneurysm clipping surgery. A literature review

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Keywords
Intracisternal papaverine toxicity,
anterior circulation aneurysm,
clipping surgery

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ABSTRACT

Introduction. Cerebral vasospasm is a major cause of mortality in patients with subarachnoid haemorrhage. Irrigation of intracisternal papaverine has been adopted as a strategy to reduce the incidence of aneurysm-surgery-associated vasospasm.

Aim. The aim of this literature review is to summarize the reported complications associated with intracisternal papaverine administration.

Patients and Methods. We searched the following databases: PubMed, Google Scholar, Cochrane Library, Clinical Key, Embase, Emerald, Health Business Elite, MEDLINE at OVID, EBM Reviews and Research Gate. The following keywords were used: Intracisternal papaverine, topical papaverine, direct papaverine, a vasodilator for aneurysm surgery, papaverine in aneurysm clipping, papaverine complications and papaverine side effects. The search criteria included all articles published between 1980-2019, in the English language.

Results. Our search yielded a total of 19 articles describing 43 cases. The most common reported complication was ipsilateral oculomotor nerve palsy. Other local complications included: Bilateral oculomotor nerve palsy, ipsilateral facial nerve palsy, and monocular blindness. Although less common, reports pointing to papaverine systemic toxicity did exist. Examples of such complications included: Profound hypotension, bradycardia, hypertension and tachycardia, hyperthermia and metabolic acidosis, cardiac arrest and even death.

Conclusion. Intracisternal papaverine irrigation is an effective strategy in reducing peri-operative vasospasm associated with aneurysm surgery. Although uncommon, both local and systemic side effects have been linked to papaverine use, calling for careful dosing and close monitoring to enhance its safety profile.

INTRODUCTION

The use of intracisternal papaverine to prevent vasospasm during aneurysm surgery was first described in the fifties of the 19th century (1). Papaverine is a vasodilator that can be installed directly onto the

the vessels in the exposed cisterns during aneurysm clipping. Papaverine's main role is to prevent intraoperative and postoperative vasospasm and to alleviate its deleterious consequences (1). The application of intracisternal papaverine is considered to be a safe procedure as compared to other routes of administration such as the intra-arterial, and intravenous routes along with the slow release pellets as these routes often entail several serious direct side effects (2-4). To date, there are few reports that describe the complications associated with the installation of intracisternal papaverine.

In this article, we reviewed the available literature regarding the adverse effects related to the intracisternal papaverine use during aneurysm surgery.

Patients and methods

We conducted a literature review of all articles published on PubMed, Google Scholar, Cochrane Library, Clinical Key, Embase, Emerald, Health Business Elite, MEDLINE at OVID, EBM Reviews and Research Gate from 1980-2019. The following keywords were used: Intracisternal papaverine, topical papaverine, direct papaverine, vasodilator for aneurysm surgery, papaverine in aneurysm clipping, papaverine complications and papaverine side-effects. The reported cases were analyzed in relation to the location of the aneurysm, the association with subarachnoid hemorrhage (ruptured or unruptured aneurysm), the dose of papaverine used, the reported side effects, the resolution time of these effects and the fenestration status of the lamina terminals. The demographic data and the surgical approaches were excluded from the results as these parameters were not mentioned in the majority of the reported cases.

Results

Our search yielded 19 articles, with a total of 43 aneurysms. As for the types of articles; there were two original articles (5,6), two case reports and reviews (7, 8), one case series (9), nine case reports (10-18), and five letters to the editor (19-23). The two most common aneurysm locations that were associated with topical papaverine complications were the middle cerebral artery (MCA) and the anterior communicating artery (AcomA) with 18 and 17 cases, respectively. Other reported locations included posterior communicating artery aneurysm (6 cases), internal carotid artery bifurcation aneurysm (one case), and ophthalmic artery aneurysm (one case). There was one report where the location of the aneurysm was not specified. Notably, amongst the 43 reported papaverine toxicity cases, 42 cases were associated with ruptured aneurysms while only one case was associated with an un-ruptured aneurysm (13) while the remaining cases did not specify the status of aneurysm rupture.

This literature review showed that intracisternal papaverine can cause ipsilateral (6, 9, 11-13, 18), contralateral (6, 7, 21), and even bilateral oculomotor nerve palsy (7-10, 19). The ipsilateral oculomotor palsy in the form of mydriasis with a non-reacting pupil was the most common reported complication of intracisternal papaverine installation (18 out of 43 patients). Temporary facial nerve palsy was the only other reported cranial nerve paresis after the oculomotor nerve (13).

Hemodynamic and metabolic adverse effects were reported in a total of 15 cases. These effects included profound hypotension (5, 20, 22), bradycardia (5), both hypotension and bradycardia (14, 16, 23), and hypertension and tachycardia (17). Two of the three reported cases of intracisternal papaverine complications in the form of both hypotension and bradycardia progressed to cardiac arrest and death (16, 23); these were the only reported deaths related to cisternal papaverine. McLoughlin et. al described the unique toxicity of papaverine as hyperthermia and metabolic acidosis (12). One other rare complication was permanent monocular blindness resulting from choroidal infarction with an unknown mechanism of causation (15). (Table 1)

Out of all the reviewed reports on the complications of the intracisternal papaverine, only three reports by Rath et al. 2006 (14), Singla et al. 2009 (22), and Baltaci et al. 2010 (16) included the fenestration of the lamina terminalis within the parameters under study; the authors described serious complications related to intracisternal papaverine installation after the fenestration of the lamina terminalis; namely, profound hemodynamic changes, severe hypotension, and cardiac arrest respectively.

The resolution time of the adverse effects of papaverine was variable. The oculomotor-related complications usually resolved within the first day

with the majority of patients recovering within the first 5 hours. However, in three cases the resolution extended beyond 4-7 days and even up to 23 days in one report (8, 10, 12). As compared to cranial nerve paresis, the hemodynamic complications of intracisternal papaverine were more abrupt with the majority of reports charting a resolution time of few minutes; only two cases, that reported hypotension, recorded a resolution time of more than one hour (20, 22).

Our review revealed that papaverine hydrochloride ampule was used in all cases. The concentration used ranged between 30 and 300 mg of 3% papaverine, diluted in 10 up to 100 ml of warm 0.9% normal saline or Ringer lactate with only a few cases reporting the use of undiluted (60 mg)of papaverine. The recommended papaverine regimen, based on our review, was 2 cc of 3% papaverine (60 mg) diluted in 10-20 ml of warm 0.9% normal saline or Ringer lactate at room temperature (35-37 °C).

Table 1: Review of the reported complications of intracisternal papaverine

Adverse effects	Number of reported cases	Percent of the total reported cases with toxicity
Ipsilateral oculomotor nerve palsy	18	42%
Bilateral oculomotor nerve palsy	7	16%
Contralateral oculomotor nerve palsy	3	7%
Ipsilateral facial nerve palsy	1	2%
Monocular blindness	1	2%
Profound hypotension	7	16%
Bradycardia	3	7%
Severe hypotension and bradycardia*	3	7%
Hypertension and tachycardia	1	2%
Hyperthermia and metabolic acidosis	1	2%

*Two of the cases end with cardiac arrest and death.

Discussion

The topical papaverine installation on the dissected cisterns after aneurysm clipping and just prior to dural closure can cause rare but diverse adverse effects ranging from temporary cranial nerve paresis to cardiac arrest and even death.

The association of these complications with papaverine use was confirmed in all reports after the exclusion of all possible anesthetic and surgical causes. Furthermore, the temporal association between the application of papaverine and the development of side effects confirmed that the primary cause was the drug itself rather than other factors. Also, the type of papaverine formula used had no neurotoxic solvent in its composition (14). It is worth noting that although the distribution of the reported aneurysm locations was found to follow the classic pattern of incidence of the surgically managed intracranial aneurysms, one cannot deny the possibility of papaverine related complications may increase with both AcomA and MCA aneurysms as the surgery for such locations entails more arachnoid dissection and involve the opening of more subarachnoid cisterns than surgeries in other proximal locations. Additionally, we suggest that aneurysm rupture may be one of the factors that contribute to papaverine toxicity as subarachnoid hemorrhage may render the brain and the cranial nerves more sensitive for the direct effect of papaverine, increasing the rate of complications.

The most commonly cited theory for the reason behind papaverine-associated cranial nerve paresis is that papaverine likely exerts direct chemical toxicity, working in synchrony with the subarachnoid blood to irritate the already-sensitized cranial nerves. The hemodynamic and metabolic changes were explained in the literature by the possible effect of papaverine while in contact with vital centers in the hypothalamus and brainstem.

As for the three reports that linked papaverine toxicity to the anterior third ventriculostomy (opening of lamina terminalis), the authors of these reports hypothesized that the introduction of papaverine into the ventricular cavity through the

fenestrated lamina terminalis may have resulted in direct chemical irritation to the hypothalamus and midbrain in the walls of the third ventricle and the vagal nucleus in the walls of the fourth ventricle (14, 16, 22). These complications can occur when papaverine is installed in the cistern after the fenestration of lamina terminalis (anterior third ventriculostomy or anterior ventriculo-cisternostomy). Thus, papaverine can easily enter the third ventricle and become in direct contact with the hypothalamic nuclei in the ventricular wall. Papaverine can also reach the fourth ventricle through the aqueduct of Sylvius and exert its effect on the brainstem nuclei in the floor of the fourth ventricle. These complications would, inevitably, be more evident when papaverine is used in a non-diluted form or in a relatively high dose. Considerable variability was found in the description of the used doses (concentration and amount) of papaverine to be installed into the cisterns during aneurysm clipping surgery. Whether to dilute the papaverine or not and what is the best diluting fluid are still debatable issues and the decisions currently based on the surgeon's personal experience.

Conclusion

Intracisternal papaverine use is an effective method for the prevention of perioperative vasospasm during intracranial aneurysm clipping surgery. However, this is not a completely safe intervention and may be associated with toxicity, mainly in the form of temporary paresis to the adjacent cranial nerves or variable hemodynamic consequences. Thus, precautions regarding dosing and monitoring must be contemplated when introducing topical papaverine into the surgical field.

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Romanian Neurosurgery (2020) XXXIV (1): pp. 391-399
DOI: 10.33962/roneuro-2020-061
www.journals.lapub.co.uk/index.php/roneurosurgery

Can routine biochemical tests be a short-term prognostic biomarker in patients operated for chronic subdural hematoma?

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ABSTRACT

Objectives: The effect of routine blood biochemistry parameters on the short-term prognosis of patients with chronic subdural hematoma (CSDH) has not been evaluated in literature before. In this study, it was aimed to establish markers for determination of short-term prognosis using data of patients who were operated for CSDH.

Methods: During admission to hospital, data of patients including age, sex, antiaggregan and/or anticoagulant drugs usage, comorbidity, Glasgow Coma Scale (GCS) and Glasgow Outcome Scale scores were evaluated. Location and thickness of CSDH were recorded using brain CT or MR images. Blood leukocyte, neutrophil, lymphocyte, eosinophil, basophil, platelet count results, neutrophil-lymphocyte ratio and platelet-lymphocyte ratio results, activated prothrombin time and INR values, serum glucose, aspartate aminotransferase, alanine aminotransferase, C-reactive protein, sodium, potassium, blood urea nitrogen and creatinine level values were also recorded. Patients were divided into two groups according to CSDH located "unilaterally (n=19)" and "bilaterally (n=12)". In addition, patients with unilateral CSDH were divided into two groups as CSDH located at the "right hemisphere (n=6)" and "left hemisphere (n=13)".

Results: It was concluded that short-term prognosis of patients with unilateral or bilateral CSDH was similar. Correlation analysis showed no correlation between short-term prognosis and demographic, clinical and laboratory findings. However, *Likelihood Ratio* test revealed that GCS score could be a biomarker in order to predict short-term prognosis of these patients, albeit weak (X^2=6.138, p=0.046).

Conclusion: It was thought that GCS scores could be effective in predicting short-term prognosis in patients with CSDH but routine biochemistry laboratory parameters could not predict short-term prognosis of these patients.

Keywords
biochemistry,
chronic subdural hematoma,
prognosis

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INTRODUCTION

Chronic subdural hematoma (CSDH) is characterized by the presence of fluid trapped in the capsule or membrane in the subdural space its annual incidence is 5-8.2 / 100000 and generally seen in patients older than 65 years. [1,2]

Several studies have reported that the cause of bleeding is often previous head trauma, brain atrophy, advanced age, alcohol abuse and anticoagulant use.[2,3] The rate of surgical treatment in CSDH patients is 2.7-33% and burr-hole drainage is the most commonly used surgical technique.[4] However, there is still some controversial in literature in deciding the short-term prognosis in CSDH patients. It has been suggested in some studies that midline shift thickness and volume of fluid accumulated in the subdural space may be prognostic markers.[5] In addition, it has been emphasized that the lower the modified Rankin Scale scores may make the prognosis worse.[6,7] It has been also demonstrated in studies in which CSDH cases were evaluated immunologically that interleukin-6 and "*Vascular Endothelial Growth Factor (VEGF)*" values measured in hematoma fluid were found to be higher than serum values.[8-10] However, to the best of our knowledge, there is no study in literature to demonstrate the effect of routine blood biochemical parameters on short-term prognosis of patients with CSDH.

Therefore, in this study, demographic, clinical and routine laboratory biochemistry results of patients operated for CSDH were investigated and markers were established to determine short-term prognosis.

Materials and methods

Materials

This retrospective study was conducted after the approval of the "Local Ethics Committee for Clinical Trials".

The hospital records were scanned using "*The International Statistical Classification of Diseases and Related Health Problems* (ICD-10)" coding (I62: Intracerebral hemorrhage (non-traumatic), other hemorrhage) I62.1: Extradural hemorrhage (non-traumatic); I62.9: Intracranial hemorrhage (non-traumatic), undefined; S06.5: Traumatic subdural bleeding). Patients who were treated surgically (i.e. burr-hole evacuation) between January 2017 and June 2019 after the determination of the chronic subdural hematoma on brain computed tomography (CT) and / or magnetic resonance imaging (MRI) were included in this study.

Patients with intracranial mass, patients with acute and / or subacute subdural hematoma, patients whose CSDH was evacuated by craniectomy or patients whose CSDH was not treated surgically but followed-up were excluded from the study. The patients were also excluded from this study if data were incomplete, if they had another intracranial bleed secondary to the trauma (e.g., epidural hematoma, subarachnoid hemorrhage etc), if they had been incorrectly coded with ICD-10, did not have subdural hematoma, or were in the pediatric age group (<16 years)

Patients were divided into two groups according to the subdural hematoma located "unilaterally (n = 19)" or "bilaterally (n = 12)". In addition, patients with unilateral subdural hematoma were divided into two groups as subdural hematoma located at the "right hemisphere (n = 6)" and "left hemisphere (n = 13)".

Patients were also divided into two groups according to age (<70 years and <70 years) or gender (female = 6, male = 25).

Methods

During admission to hospital, age, sex, antiaggregan and anticoagulant drugs use, history of comorbidity, Glasgow Coma Scale (GCS) scores and Glasgow Outcome Scale (GOS) scores of the patients were recorded. The location and thickness of the chronic subdural hematoma were evaluated using by brain CT or MR images. Blood leukocyte, neutrophil, lymphocyte, platelet, eosinophil, basophil count results, neutrophil-lymphocyte ratio (NLR) and platelet-lymphocyte ratio (PLR) results, activated prothrombin time and "*International Normalized Ratio*" (INR) values, serum glucose, aspartate aminotransferase (AST), alanine aminotransferase (ALT), C-reactive protein (CRP), sodium (Na), potassium (K), blood urea nitrogen (BUN) and creatinine level values were recorded.

The scales used in this study are listed below:

- Glasgow Coma Scale (GCS): This scale is used to determine and simply define the consciousness level and neurological status of patients.[11] The scale consists of three subscales (eye findings, speech content, motor response) and is evaluated over 15 points. The higher the patient's score, the better the state of well-being.
- Glasgow Outcome Scale (GOS): It is evaluated over 5 points and is used to identify patients' current neurological levels, levels of help / care, and awareness at the time of discharge from hospital.[12] As the

scale score increases, the patient's level of well-being increases.

Surgical procedure

After sedation anesthesia, frontal and parietal skin incisions of approximately 3 cm were performed in the supine position. Subsequently, burr-holes were opened to the cranium using a drill (Midas Rex®, USA). First, the cross-shaped incision was made to the dura mater in the frontal localized burr-hole and after the chronic hematoma content started to discharge, then cross-shaped incision was made to the dura mater in the parietal localized burr-hole and the hematoma content was completely removed. Subdural space was then irrigated with saline solution using foley catheter and then suction drain was left to the frontal region from the parietal burr-hole and the galea and skin were sutured anatomically before the operation was terminated (Figure 1). In patients with bilateral CSDH, a similar procedure was performed on the other side (Figure 2).

Biochemical analysis

Biochemical data of the study were obtained from the analysis of patients' venous blood samples which were taken during their admission to the hospital. Serum glucose (reference range 74-109 mg / dL), CRP (reference range 0.15-5 mg / dL), ALT (reference range 5-41 u / L), AST (reference range 5-40 u / L), creatinine (reference range 0.84-1.24 mg / dL) and BUN (reference range 17-43 mg / dL) level values were measured of the "immunoturbidimetric method" and levels of serum sodium (reference range 136-146 mmol / L) and potassium (reference range 3.5-5.1 mmol / L) were determined by ion selective electrode (ISE) method using commercial kits (Roche) and an analyzer device (Roche Diagnostic COBAS c501). Blood leukocyte (reference range 4400-11300 uL), neutrophil (reference range 1,100-9600 uL), lymphocyte (reference range 500-6000 uL), eosinophil (reference range 0-1000 uL), basophil (reference range: 0-300 uL) and platelets (reference range 150000-500000 uL) count values were determined using an analyzer (Mindray BC-6800, Shenzen, China). Activated prothrombin time (reference range 24.0-39.2 minutes) and INR (0.8-1.2) values were measured using an analyzer (ACLTOP700, USA).

Statistical analysis

Mann-Whitney U test was used to compare the nonparametric data between the groups. *Independent Samples t* test was used to compare the parametric data between the groups ($p < 0.05$). Spearman's rho *Correlation* test was used to determine the relationship between the parameters ($p < 0.05$). *Likelihood Ratio* test was used to find the best parameter to predict the short-term prognosis of the patients ($p < 0.05$).

Results

A total of 31 patients (female = 6, male = 25) were included in this study. Demographic, clinical and laboratory findings of all patients are summarized in Table 1 and Figure 3.

At the end of the medical past history, it was found that 3 patients had coronary artery disease, 5 had essential hypertension, 4 had diabetes mellitus, 4 had chronic obstructive pulmonary disease, 3 had a history of cerebral stroke, and 1 had atrial fibrillation. Six of the patients were using antiaggregant and two of the patients were taking anticoagulant drugs, but activated prothrombin time and INR values were within normal limits. It was observed that new onset left hemiparesis was detected in 5 patients, new onset right hemiparesis in 5 patients, new onset dysphasia in 3 patients, and new onset stupor in 4 patients at their admission to the hospital. At the end of the correlation analysis of all parameters of all patients, there was found no correlation between short-term prognosis and demographic, clinical and laboratory findings. However, at the end of *Likelihood Ratio* test, it was seen that GCS scores could be a good parameter in predicting the short-term prognosis of patients with CSDH ($X^2 = 6.138$, $p = 0.046$).

When patients were divided into two groups according to unilateral or bilateral localization of chronic subdural hematoma, comorbidity was found to be higher in patients with unilateral CSDH ($X^2 = 4.288$, $p = 0.038$). In addition, the thickness of right-sided hematoma was higher in patients with bilateral CSDH ($Z = -4.169$, $p < 0.001$). Other demographic, clinical and laboratory data was not different between the groups (Table 2). At the end of the correlation analysis applied to the parameters of each group, no study parameter was found to be correlated with the patient's neurological status and post-treatment short-term prognosis.

In addition, when the patients with unilateral CSDH were divided into two groups according to the hematoma located on the right side hemisphere or the left side hemisphere, the GOS scores of the patients with right side hemisphere hematoma were found to be lower (Z = - 2.156, p = 0.031), while platelet count values were higher (t = 2.243, p = 0.040). However, there was no statistical difference between the groups in terms of other demographic findings and laboratory values (Table 3, Figure 2). At the end of the correlation analysis of the parameters belonging to each group, no study parameter was found to be correlated with the patient's neurological status or postoperative short-term prognosis. However, at the end of the *Likelihood Ratio* test, it was concluded that GCS score (X^2 = 12.079, p = 0.002) and PLR values (X^2 = 8.578, p = 0.014) could be a biomarker for predicting the short term prognosis of the patients with unilateral CSDH.

When the patients were divided into two groups according to age distribution and gender, the demographic, clinical and laboratory findings of the patients was not statistically different between the groups and it was observed at the end of the correlation test and *Likelihood Ratio* test that these parameters could not be a predictive biomarker in determining the short-term prognosis of these patients.

VARIABLE		**Mean±SD/ Number (%)/ Median (min-max)**
Age (year)		72.19±14.62
Gender	Female	6 (19.4)
	Male	25 (80.6)
Comorbidity	No	16 (51.6)
	Yes	15 (48.4)
Drug usage	No	23 (74.2)
	Yes	8 (25.8)
Convulsion	No	29 (93.5)
	Yes	2 (6.5)
Glasgow Coma Scale score		15 (8-15)
Glasgow Outcome Scale score		5 (1-5)
Right sided hematoma thickness (mm)		15.61±11.21
Left sided hematoma thickness (mm)		12.39±11.59
Leukocyte (uL)		8726.79±3517.66
Neutrophil (uL)		6686±3199.51
Lymphocyte (uL)		1460±739.55
Monocyte (uL)		495±257.46
Eosinophil (uL)		121±121.42
Basophil (uL)		57±86.75
Platelet (uL)		244035±88146.97
Neutrophil-lymhocyte ratio		5.79±4.04
Platelet-lymphocyte ratio		195.75±91.86
C-reactive protein (mg/dL)		
Glucose (mg/dL)		130.04±44.14
Blood urine nitrogen (mg/dL)		52.80±29.53
Creatinine (mg/dL)		1.13±0.89
Sodium (mmol/L)		134.93±17.25
Potassium (mmol/L)		4.36±0.86
Alanine aminotransferase (u/L)		12.28±3.80
Aspartate aminotransferase (u/L)		23.93±13.73
International Normalized Ratio (INR)		1.15±0.23
Activated protrombine time		28.29±11.91

Table 1. It shows the demographic data and blood biochemistry findings of the patients (SD: standard deviation, min: minimum, max: maximum).

VARIABLE		UNILATERAL HEMATOMA Mean±SD/ Number (%)/ Median (min-max)	BILATERAL HEMATOMA Mean±SD/ Number (%)/ Median (min-max)	t/X²/Z	p
Age (year)		74.32±15.12	68.83±13.75	1.017*	0.317
Gender	Female	4 (12.9%)	2 (6.5%)	0.091†	0.763
	Male	15 (48.4%)	10 (32.3%)		
Comorbidity	No	7 (22.6%)	9 (29.0%)	4.288†	**0.038**
	Yes	12 (38.7%)	3 (9.7%)		
Drug usage	No	14 (45. 2%)	9 (29.0%)	0.007†	0.935
	Yes	5 (16.1%)	3 (9.7%)		
Convulsion	No	18 (58.1%)	11 (35.5%)	0.115†	0.735
	Yes	1 (3.2%)	1 (3.2%)		
Glasgow Coma Scale score		15 (8-15)	15 (8-15)	-0.709‡	0.478
Glasgow Outcome Scale score		5 (1-5)	5 (4-5)	-0.983‡	0.326
Right sided hematoma thickness		6.84±10.77	21.17±6.25	-4.169*	**<0.001**
Left sided hematoma thickness		16.42±13.09	14.33±7.71	0.499*	0.622
Leukocyte (uL)		8532±3892.01	9027±3001.62	-0.358*	0.723
Neutrophil (uL)		6535±3302.91	6918±3176.02	-0.304*	0.764
Lymphocyte (uL)		1415±872.74	1530±499.34	-0.392*	0.698
Monocyte (uL)		469±296.07	534±189.44	-0.647*	0.523
Eosinophil (uL)		140±126.54	91±112.14	1.047*	0.305
Basophil (uL)		53±69.02	62±112.43	-0.266*	0.792
Platelet (uL)		240823±110146.06	249000±39020.51	-0.235*	0.816
Neutrophil-lymhocyte ratio		5.99±3.64	5.49±4.76	0.312*	0.758
Platelet-lymphocyte ratio		203.06±100.54	184.45±79.89	0.516*	0.610
C-reactive protein (mg/dL)		14.50 (2.00-65.00)	2.58 (1.14-71.00)	-1.791‡	0.073
Glucose (mg/dL)		138.75±50.03	116.50±30.84	1.191*	0.247
Blood urine nitrogen (mg/dL)		57.85±32.12	44.21±23.56	1.167*	0.254
Creatinine (mg/dL)		1.20±1.03	1.02±0.61	0.497*	0.623
Sodium (mmol/L)		133.09±21.38	138.06±5.47	-0.716*	0.481
Potassium (mmol/L)		4.34±1.01	4.39±0.57	-0.145*	0.886
Alanine aminotransferase (u/L)		12.46±4.29	12.03±3.18	0.269*	0.790
Aspartate aminotransferase (u/L)		26.90±16.25	19.47±7.44	1.347*	0.191
International Normalized Ratio		1.18±0.18	1.12±0.29	0.666*	0.512
Activated protrombine time		30.39±16.48	25.98±1.49	0.841*	0.397

Table 2. This table shows the results of the demographic, radiographic and biochemical analyses of the patients according to the subdural hematoma located "unilaterally" or "bilaterally" (t: t score, X2: chi-square, Z: Z score, SD: standard deviation, min: minimum, max: maximum).

VARIABLE		RIGHT SIDED HEMATOMA Mean±SD/ Number (%)/ Median (min-max)	LEFT SIDED HEMATOMA Mean±SD/ Number (%)/ Median (min-max)	t/X²/Z	p
Age (year)		81.17±9.60	71.15±16.44	1.374*	0.187
Gender	Female	0.0%	4 (21.1%)	2.338†	0.126
	Male	6 (31.6%)	9 (47.4%)		
Comorbidity	No	3 (15.8%)	4 (21.1%)	0.652†	0.419
	Yes	3 (15.8%)	9 (47.4%)		
Drug usage	No	4 (21.1%)	10 (52.6%)	0.223†	0.637
	Yes	2 (10.5%)	3 (15.8%)		
Convulsion	No	6 (31.6%)	12 (63.2%)	0.487†	0.485
	Yes	0 (0.0%)	1 (5.3%)		
Glasgow Coma Scale score		15 (8-15)	15 (9-15)	-0.203‡	0.839
Glasgow Outcome Scale score		4.50 (1-5)	5 (4-5)	-2.156‡	**0.031**
Hematoma thickness (mm)		21.67±5.68	24.00±7.75	-0.657*	0.520

Leukocyte (uL)	9916±4933.55	7955±3456.79	0.943*	0.361
Neutrophil (uL)	7372±4350.28	6187±2921.63	0.662*	0.518
Lymphocyte (uL)	1640±754.62	1322±931.92	0.672*	0.512
Monocyte (uL)	628±280.93	403±287.41	1.477*	0.160
Eosinophil (uL)	198±146.70	116±115.43	1.232*	0.237
Basophil (uL)	52±45.50	54±78.58	-0.062*	0.952
Platelet (uL)	323800±127609.56	206250±85391.21	2.243*	**0.040**
Neutrophil-lymhocyte ratio	5.27±2.37	6.29±4.12	-0.514*	0.615
Platelet-lymphocyte ratio	225.66±130.63	193.6±590.38	0.586*	0.567
C-reactive protein (mg/dL)	16.00 (5-65)	13.00 (2-53.14)	-0.570‡	0.569
Glucose (mg/dL)	154.31±77.67	132.52±38.13	0.723*	0.484
Blood urine nitrogen (mg/dL)	67.47±34.68	53.85±31.69	0.787*	0.444
Creatinine (mg/dL)	1.01±0.17	1.28±1.23	-0.486*	0.634
Sodium (mmol/L)	140.04±3.54	130.19±25.09	0.858*	0.404
Potassium (mmol/L)	4.77±0.48	4.16±1.13	1.155*	0.266
Alanine aminotransferase (u/L)	14.42±5.54	11.68±3.74	1.087*	0.299
Aspartate aminotransferase (u/L)	21.28±8.03	28.95±18.25	-0.798*	0.439
International normalized ratio	1.24±0.26	1.16±0.14	0.772*	0.455
Activated protrombine time	29.27±5.74	30.81±19.44	-0.132*	0.898

Table 3. This table shows the results of demographic, radiographic and biochemical analyses of the patients according to the subdural hematoma located "right hemisphere" or "left hemisphere" (t: t score, X2: chi-square, Z: Z score, SD: standard deviation, min: minimum, max: maximum).

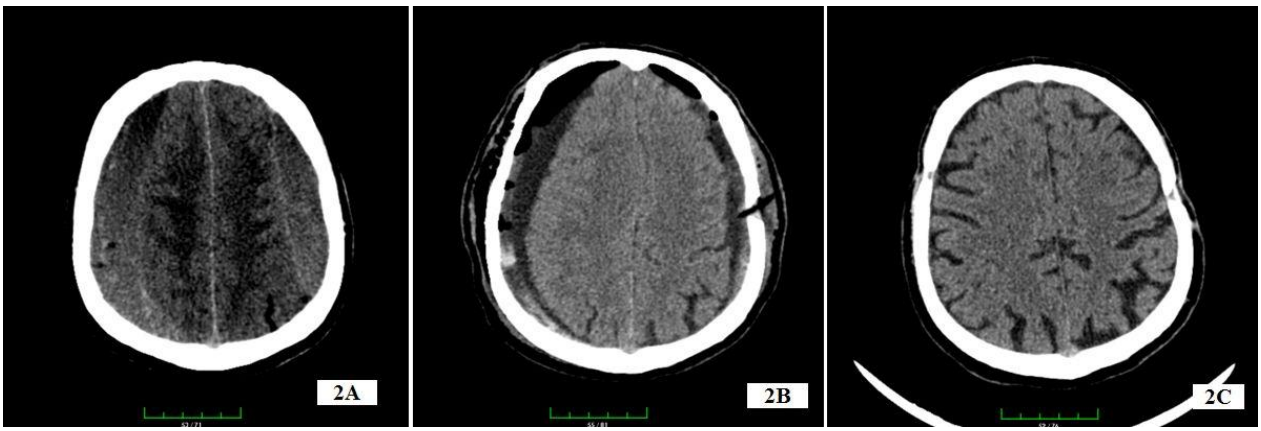
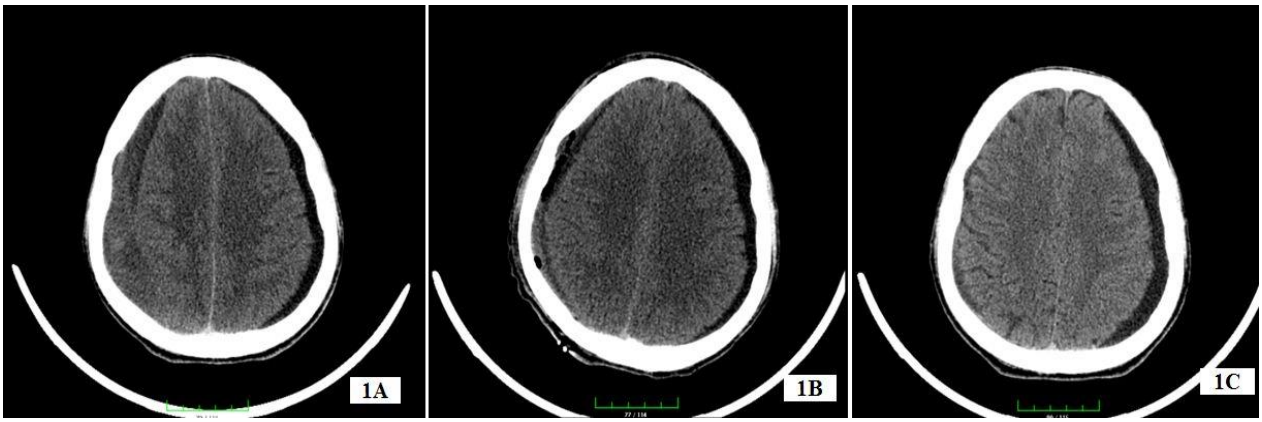


Figure 1. CT images of a patient with chronic subdural hematoma unilaterally; (1A) on admission to hospital; (1B) early postoperative image.

Figure 2. CT images of a patient with chronic subdural hematoma bilaterally; (2A) on admission to hospital, and (2B) early postoperative image and (2C) at discharge from the hospital.



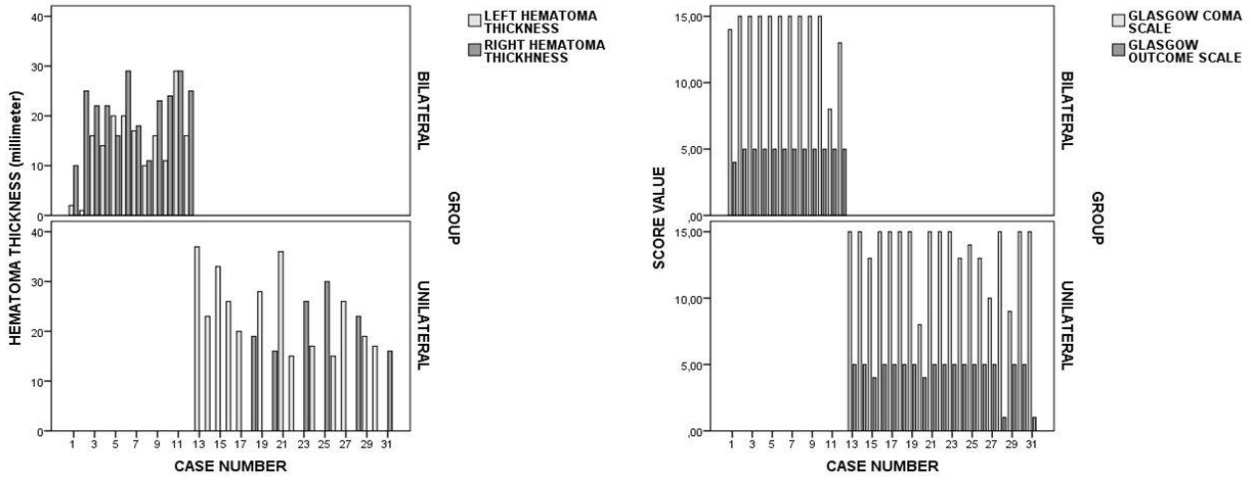


Figure 3. Graphics show the "Glasgow Coma Scale" and "Glasgow Outcome Scale" scores, chronic subdural hematoma thickness located at the left and right-side hemisphere of all patients.

Discussion

The aim of this study was to determine how its surgical treatment affected the short-term prognosis of the patient using their demographic, clinical and laboratory findings. For this purpose, GOS scores of patients included in the study were used to establish the short-term prognosis of these patients. Furthermore, in this study, it was aimed to determine short-term prognostic indicators in the patients with CSDH using simple, inexpensive and easily applicable blood biochemistry tests in almost every health institution.

It has been reported in the literature that the prognosis of the patient with intracranial hemorrhage who has a GCS score of 7 or less is worse.[13-15] In the studies conducted on this subject, the mean GOS score was found to be 1 point in cases with GCS score of 7 and below, while the GOS score was found in the range of 4-5 points in cases with GCS score above 7.[16-19] In addition, it has been shown that the advanced patient age and comorbidity such as vascular pathology, coagulation disorder and diabetes mellitus adversely affects the prognosis in patients with chronic subdural hematoma.[20] The most commonly preferred surgical treatment method in patients with chronic subdural hematoma is the burr-hole evacuation technique.[21-23] It has been reported that the mortality rate after surgical treatment is 10% in patients with CSDH, but in some studies, this rate can reach up to 32% and morbidity may occur in up to 20% of these patients.[23] In our study, it was found that the additional diseases detected in the study patients had no effect on the short-term prognosis but bilateral subdural hematoma was more common in patients with additional disease. In addition, it was found that anticoagulan and / or antiaggregant drug use did not affect short-term prognosis in these patients. With these findings it could be said that comorbidity and / or anticoagulan and / or antiaggregant drug use may not affect on the short term prognosis in surgically treated patients with CSDH. On the other hand, short-term prognosis was similar in patients with unilateral or bilateral CSDH.

Therefore, the presence of unilateral or bilateral CSDH had no effect on the short-term prognosis of these patients. However, in patients with unilateral CSDH, right-sided subdural hematoma was thought to adversely affect to the short-term prognosis. On the other hand, in our study, no significant correlation was found between the GCS scores of the patients at the time of hospital admission and GOS scores at the end of the treatment. In addition, no direct or indirect relationship was found between these GOS scores and other parameters including demographic or laboratory parameters. However, *Likelihood Ratio* test applied to the data of all patients revealed that the GCS scores of patients could be a parameter in predicting prognosis of these patients in the short-term period, albeit in a weak character.

As a matter of fact, 2 operated patients died in the hospital (mortality rate of 6.4%) despite the GCS score of 15/15 while 3 operated patients whose GCS scores were 8, 13, 14 were discharged from the hospital with the help of daily life (GOS score was 4, morbidity of 10.34%). Furthermore, all of the

remaining patients left the hospital with a GOS score of 5 regardless of GCS scores.

On the other hand, routine blood biochemistry results have been examined in studies conducted to determine the prognosis in patients with CSDH and it has been argued that hsCRP, albumin, prealbumin, INR elevations may be associated with poor prognosis but other routine blood biochemistry values have no effect on prognosis.[24] In our study, unfortunately, none of the simple routine blood biochemistry value had any effect on the short-term prognosis of the patients, postoperatively. However, it was concluded at the end of the *Likelihood Ratio* test that GCS scores and PLR values may be a biomarker in predicting the prognosis of these patients in the early postoperative period. As a matter of fact, two of the patients with unilateral subdural hematoma died postoperatively. One of these 2 patients had no additional disease and laboratory values were within normal limits. The other patient had chronic obstructive pulmonary disease and high leukocyte and lymphocyte count values. Both patients had no history of anticoagulant and / or antiaggregant drug use. Therefore, no parameter could be found to correlate the existing mortality with CSDH. In addition, GCS values were found to be similar in patients with CSDH which located on the right or left hemisphere and it did not show any statistical relationship with any study parameter. However, unilateral CSDH was thought to cause a relative increase in platelet count in patients and this could lead to an increase in PLR values. However, this increase in platelet count remained within the normal range of laboratory values.

Furthermore, GOS scores, platelet and lymphocyte count and PLR values were not different between groups and there was no direct or indirect relationship with the GOS scores and platelet and lymphocyte count and PLR values. Therefore these parameters could not be a biomarker in predicting the short-term prognosis of patients with CSDH.

Limitations

This study had some limitations. Firstly, the study was of a retrospective character and the number of patients included in the study was not sufficient because the patients' data was obtained from single health center. Secondly, patients with chronic subdural hematoma followed without performing surgery was not included in this study. Therefore, in this study, it could not be determined how chronic subdural hematoma itself affected the short-term prognosis of these patients. Thirdly, because ultra-structural methods such as histopathological and biochemical investigation of the hematoma and / or cerebrospinal fluid could not be included in this study, the effects of these investigation findings on short term prognosis could not be evaluated in this study. Finally, patients with chronic subdural hematoma evacuated by craniotomy were not included in the study. Therefore, the differentiation of the short term prognosis between the patients performed burr-hole evacuation and patients treated using craniotomy could not be discussed.

Conclusion

In conclusion, GCS values of patients with chronic subdural hematoma which were measured during the admission to the hospital could be weakly effective in predicting the short-term prognosis after surgical treatment of these patients. However, it was found that routine biochemical laboratory parameters were not successful in determining the short-term prognosis of these patients.

Conflict of interest and financial disclosure statements

The authors declare that they have no conflict of interest. There no any funding. They also declare that they have not engaged in any financial relationship with any company whose product might be affected by the research described or with any company that makes or markets a competing product.

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Romanian Neurosurgery (2020) XXXIV (1): pp. 400-404
DOI: 10.33962/roneuro-2020-062
www.journals.lapub.co.uk/index.php/roneurosurgery

Chondrosarcoma in petroclival synchondrosis without visual change. A case report

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Keywords
tomography, x-ray computed, diagnostic imaging, chondrosarcoma

Abstract

Chondrosarcomas are a heterogeneous group of malignant bone tumours that share the production of the chondroid matrix in common. At the base of the skull, they are most commonly found in the region of the various synchondroses with an affinity for the petroclival fissure, they are locally invasive tumours, with little capacity to perform metastasis. The age group affected is variable, however, they frequently occur in middle-aged adults. Its clinical manifestation depends on the location and local extent; headache or paralysis of cranial nerves, particularly of the VI nerve is a frequent sign. As the petrous apex cannot be viewed directly, imaging studies such as computed tomography and magnetic resonance imaging play an important role in the evaluation of injuries. We present a case of a 36-year-old patient with chondrosarcoma of petroclival syndromes without visual changes. For the identification of this pathology, a battery of imaging tests was used and the diagnosis was made assertively, preserving the best choices for the treatment of the patient.

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Introduction

Chondrosarcomas (CS) is a heterogeneous group of malignant bone tumors that share in common the production of the chondroid (cartilaginous) matrix. Cartilaginous tumors are almost always found in bones that arise from endochondral ossification, noting in the growth plate, the proliferation and hypertrophic cell differentiation of chondrocytes, which undergo apoptosis, followed by subsequent local invasions by vessels and osteoblasts, thus initiating the matrix formation, which consequently leads to longitudinal bone growth. Chondrosarcomas of the skull are rare and have a higher incidence in

male patients in a 2:1 ratio. They are usually diagnosed around the third and fourth decades of life(1,2).

At the base of the skull, they are most commonly found in the region of the various synchondroses that remain after ossification of the embryonic chondroid matrix, with an affinity for the petrooccipital (petroclival) fissure. Therefore, they present as para-sagittal tumors. These tumors are locally invasive, with little capacity to metastasize. They spread through the petroclival fissure, involve the clivus, the petrous portion of the temporal bone, and are more prominent in cisterns or soft tissues at the base of the skull (5,6).

Case report

A 36-year-old female patient enters a private clinic in the city of Dourados-MS-Brazil with headache, otalgia, runny nose, dysphonia, hyposmia, postural vertigo, weight loss of 5 kg and without visual changes. Rhinoscopy procedure was performed after clinical examination, which detected a "vegetating" lesion in the nasopharynx. A biopsy of the rhino pharynx tumor was then requested, with the removal of numerous irregular fragments of tissues with a light-brown surface, finely granular and fibroelastic, sent for anatomopathological analysis and initial imaging investigation with multislice computed tomography of the skull and Magnetic Resonance (MR) of the brain. The anatomopathological result suggested cartilaginous neoplasia with hypercellularity and nuclear hyperchromasia, concluding a histological aspect that could correspond to grade 1-2 chondrosarcoma depending on the correlation with clinical and radiological data. The multislice computed tomography of the skull (Figures 1 and 2) showed an expansive, heterogeneous and predominantly hypodense lytic lesion, with foci of calcification in the aspect of "popcorn" in-between, with irregular contours and defined limits, which presents a slight enhancement and predominantly peripheral after the infusion of the contrast medium, with an epicenter in the right petroclival synchondrosis. This formation invades the petrous region of the temporal bone and part of the sphenoid wing anterolaterally, without invading the orbital accumulation; extends medially and inferiorly through the jugular foramen, widening it, to the cerebellar point angle, right lateral wall of the cavum, part of the clivus and without cleavage planes with the internal carotid artery, sphenoid sinus, and lateral retropharyngeal space, with consequent obliteration of the corresponding parapharyngeal space; it later compromises part of the cerebellum and the occipital condyle; finally, it affects the locoregional temporal lobe superiorly. The result of Brain Nuclear Magnetic Resonance Figure (3 and 4) suggested an expansive lesion whose epicenter is in the right petroclival synchondrosis, with erosion and bone destruction, characterized by an intermediate signal in T1, marked hypersignal in T2-weighted sequences, with some foci hypointense inside (calcification) and intense impregnation utilizing paramagnetic contrast. This lesion exhibits a component extending anteriorly and inferiorly to the retropharyngeal region and carotid space, maintaining close contact with the internal carotid artery and with the internal jugular vein, displacing the posterior wall of the rhino pharynx. Subsequently, the lesion occupies the cistern of the cerebellar-cerebellar and cerebellar-bulbar angles, causing an impression on the right anterolateral face of the bridge and bulb and the anterior face of the cerebellar hemisphere, extending further to the jugular foramen, petrous apex and part of the condyle occipital rights. Superiorly it extends to the parasellar region where it probably involves the cavernous sinus and circumferentially the internal carotid artery, also determining the impression on the medial aspect of the corresponding temporal lobe.

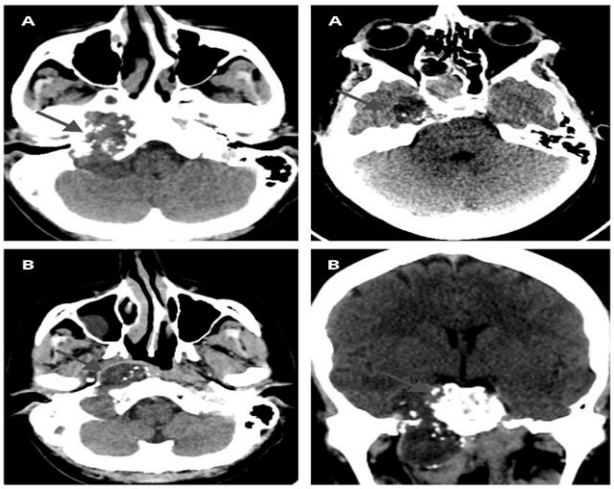


Figure 1. A. Axial CT scan using computed tomography, showing an expansive lytic lesion predominantly hypodense, with an epicenter in the right petroclival synchondrosis. B. Post-contrast axial and coronal section CT, showing slight and heterogeneous contrast to the contrast medium.

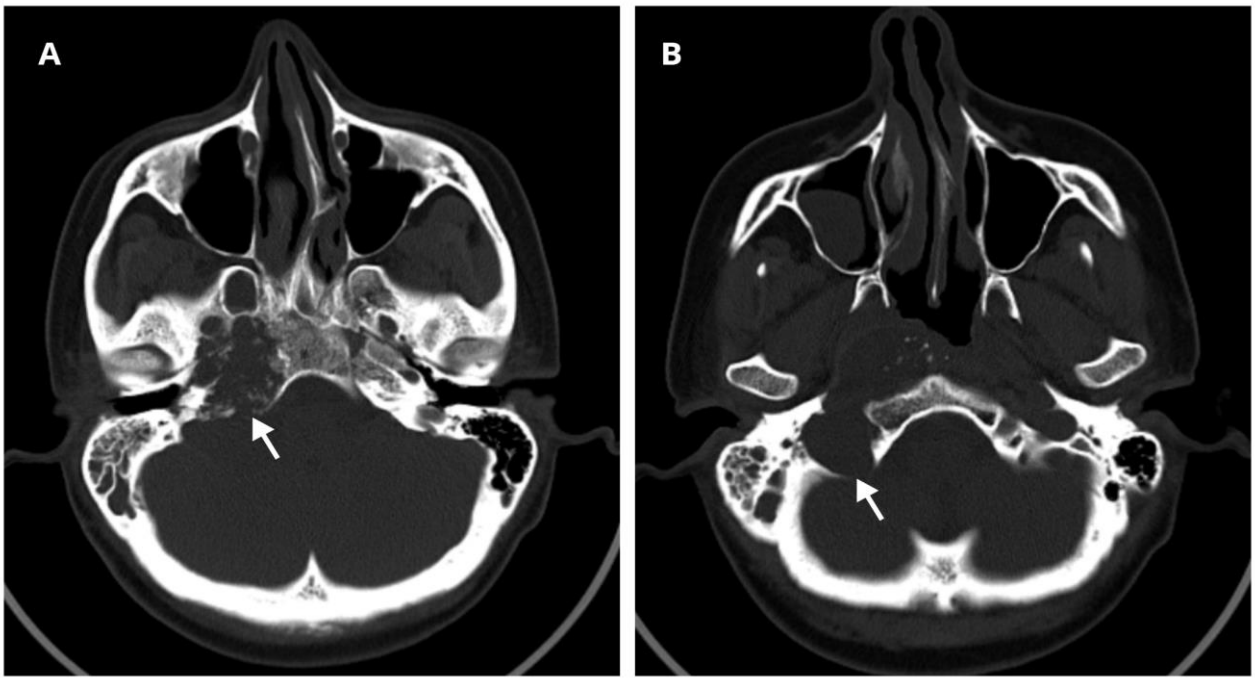
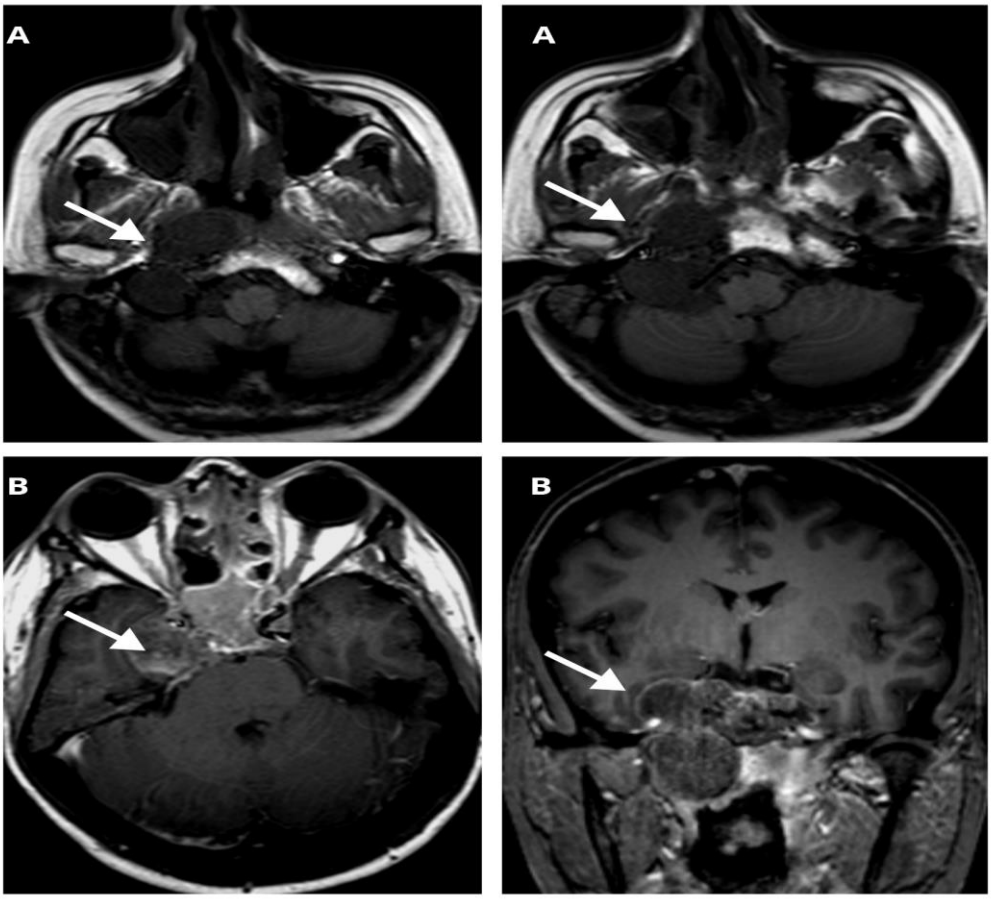


Figure 2. A, B- CT axial axial section in bone window, showing the lytic aspect of the lesion, and calcifications in "popcorn".

Figure 3. A-axial T1-weighted MRI showing an intermediate sign of the expansive lesion in petroclival synodrosis. B- Axial and coronal MRI, weighted in T1 after contrast, showing enhancement by gadolinium.



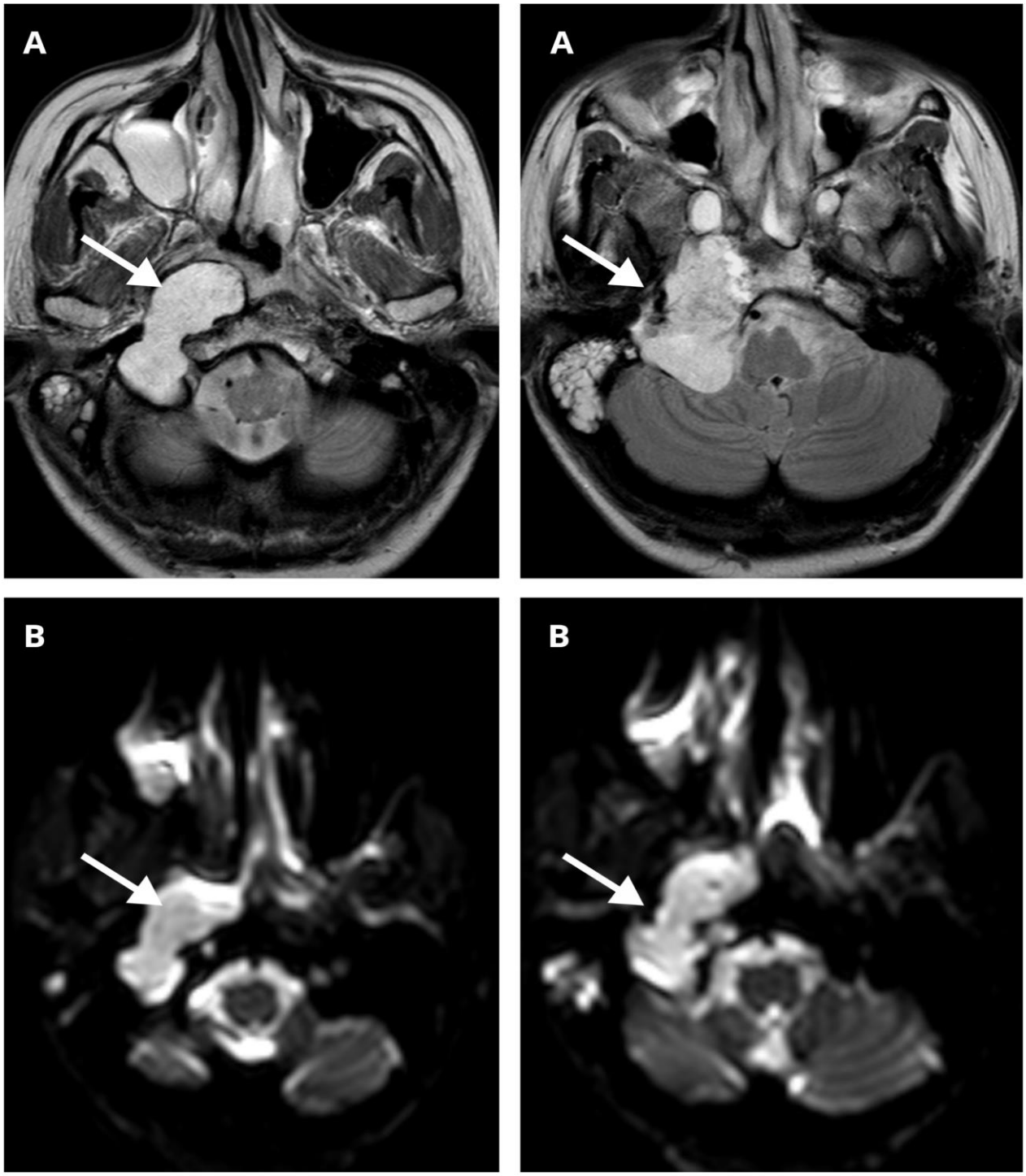


Figure 4. A-axial T2-weighted MRI, showing a high sign of the referred lesion. B- Diffusion-weighted axial MRI showing a high signal in the ADC values.

Discussion

Chondrosarcoma is a cancer of chondral origin, rare, slow-growing, locally advanced and with aggressive behavior, constituting 0.15% of all intracranial neoplasms, making up the third most common cause of primary bone malignancy, after multiple myeloma and the osteosarcoma. Approximately 25% of all cranial chondrosarcomas occur at the base, representing 6% of all neoplasms at this site (1). This type of tumor has a slow growth, constituting locally

aggressive neoplasms that makeup 0.15% of all intracranial tumors, being capable of generating bone, cartilage or even tissues without cartilage constituents (1,2).

Embryology findings suggest the hypothesis that cranial-based chondrosarcomas may originate from multipotential mesenchymal cells or remnants of embryonic cartilage from cranial synchondrosis. The various plaques that remain after ossification of the chondroid fissures remain as growth sites for these tumors, and the most common tumor origin sites described were petroclival, petro-occipital, spheno-cecipital, and spheno-petrous synchon-droses, with a propensity for the first, that is, constituting parasagittal tumors in the majority. Other locations of expected impairment are the midline, more specifically in the basisphenoid/basioccipital, which are structures related to sphenoccipital cleft, and less commonly the junction between the nasal septum and the sphenoid face (1,3).

Histologically, chondrosarcomas are divided into conventional subtypes (myxoid and hyaline type), differentiated, clear cells and mesenchymal (1). The conventional form is the most common type at the base of the skull, being further subdivided into three classes: well-differentiated (grade I), moderately differentiated (grade II) and poorly differentiated (grade III) (1). The age range affected is variable; however, they often occur in middle-aged adults. The presentation depends on the location and the local extension; headache or paralysis of cranial nerves, particularly of the VI (abducent) nerve is a frequent sign. However, the patient, in this case, showed no signs and symptoms of visual impairment despite being diagnosed with petroclival chondrosarcoma (5).

As the petrous apex cannot be viewed directly, the radiological image plays a crucial role in the evaluation of injuries (4). Computed tomography (CT) presents a varied pattern, depending on the amount of the chondroid matrix. Generally, there are components of soft parts with a dense appearance in the non-contrast phase and enhanced by the iodinated agent. Calcifications are characteristic, but not always present. A magnetic resonance imaging (MRI), these tumors usually present an intermediate signal at T1, a high signal at T2, with heterogeneous impregnation after the gadolinium injection (5,6). The main differential diagnoses include cholesteatoma of the petrous apex, calcified meningioma, chondromyxoid fibroma, chordoma, plasmacytoma, nasopharynx carcinoma, and metastases.

Conclusion

Imaging examinations such as computed tomography and magnetic resonance become an important means of evaluation in this type of injury due to its difficult anatomical presentation. The indicated treatment combines surgery with several types of radiation; eventually, it consists exclusively of radiotherapy. Complete surgical excision is usually not feasible due to its location and proximity to neurovascular structures. However, as the growth pattern is slow, the prognosis is good (99% in 10 years). Although the VI cranial pair is affected in this type of injury, no characteristic visual symptoms were observed in the studied patient, highlighting the importance of the case report presented.

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Romanian Neurosurgery (2020) XXXIV (1): pp. 405-410
DOI: 10.33962/roneuro-2020-063
www.journals.lapub.co.uk/index.php/roneurosurgery

Ventriculoperitoneal shunt occlusion and cranioplasty. A case report

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Abstract

Decompressive craniectomy (DC) is an urgent neurosurgical procedure, effective in the reduction of intracranial pressure (ICP) in patients with elevated ICP and in complications of brain infarction that do not respond to clinical treatment; traumatic brain injury (TBI); intracerebral haemorrhage (ICH) and aneurysmal intracerebral haemorrhage. Symptomatic hydrocephalus is present in 2 to 29% of patients who undergo craniectomy. They may require a ventriculoperitoneal shunt (VPS). The literature does not yet show standard management of cranioplasty in patients who have previously undergone a shunt, showing evidence of sinking skin flap syndrome. This case shows parenchymal expansion after VPS occlusion and cranioplasty in the patient's profile. The 23-year-old male patient, right-handed, went to the hospital in January 2017 due to severe traumatic brain injury following multiple traumas. The patient underwent urgent DC surgery for the management of elevated ICP. The patient developed hydrocephalus. hydrocephalus. It was decided to perform the VPS implant. After 2 years, and with quite a sunken flap, the patient was submitted to cranioplasty procedure after shunt occlusion was performed. The patient left the hospital receiving outpatient care with no more complaints. In spite of the favourable outcome, new studies are fundamental to decide upon the best approach.

Keywords
cranioplasty,
VPS,
VPS Occlusion

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Introduction

Decompressive craniectomy (DC) is efficient in reducing intracranial pressure (ICP) in patients with intracranial hypertension in complications of brain infarction that do not respond to clinical treatment, traumatic brain injury (TBI), intracerebral hemorrhage and aneurysmal intracerebral hemorrhage.[1,4]

Hydrocephalus is present in 2 to 29% of those who undergo craniectomy, possibly requiring ventriculoperitoneal shunt (VPS) after the subacute stage. Most of the patients needing craniectomy accompanied by VPS develop sinking skin flap syndrome. This particular factor may be challenging during cranioplasty due to the difficulty in parenchymal expansion because of the *shunt* and the increased

chances of material gathering in the vacant space created between the implant and brain parenchyma.[12]

The literature does not yet show a standard management of cranioplasty in patients who have previously undergone a shunt, showing evidence of sinking flap syndrome.

The reported case demonstrates parenchymal expansion after VPS occlusion and the cranioplasty that ensued for this patient. In spite of this favorable outcome, new studies are necessary for the best approaches to benefit these types of patients.

Case study

A right-handed, 23-year-old male patient entered hospital in January 2017 due to a diagnosis of TBI accompanied by multiple traumas, which required decompressive craniectomy on the right side to control raised ICP. Hydrocephalus evolved in the patient and a ventriculoperitoneal shunt was performed. After a positive medical evaluation, he was able to leave the hospital for the cranioplasty to occur at a later stage. Due to demanding workloads and loss of *follow up* procedures during 2018, the cranioplasty procedure was left until January, 2020.

Upon his second admission to hospital, the patient presented a quite sunken flap with a concave aspect (Figure 1). In order to plan the surgery, a pre-operative tomographic exam took place. The exam showed up parenchyma from the right side sheering off towards the side that had not been fractured, with structural deviances in the midline greater than 1 cm. The catheter of the VPS was normally placed in the frontal position of the right lateral ventricle (Figure 2).

In the wake of parenchymal expansion after cranioplasty, caused by the existing VPS, plans were made (after consulting the neurosurgery and plastic surgery departments) for shunt occlusion before performing cranioplasty during the same surgical period.

Electively, the patient was then submitted to occlusion of the distal end of the ventriculoperitoneal shunt catheter through clavicular incision. Within the same surgical period, the cranioplasty proceeded with the customization of the polymethylmethacrylate (PMMA) prosthesis created from a sterile mold based on the reconstruction of bones in the gaps with the help of a 3D printer (Figures 3 and 4). No complications arose during the time of the procedure.

Immediately after the operation, the tomography of the cranium showed cerebral parenchyma that had not expanded and the presence of its gathering in the subdural area (Figure 5).

The patient was directed to the neurological intensive care unit (ICU), with rigorous observations on level of consciousness and serial neurological exams. Through the evolution of the patient's recovery, the exams showed no alteration in level of consciousness, nor any physical changes. On the fourth and ninth days of the post-operative period, controlled observation by tomography showed gradual expansion of cerebral parenchemy until the point of total expansion in nine days (Figure 5). The patient, in spite of ventricular ectasia, recovered with no additional complaints. After one-week of observation, he was released from hospital (Figure 6). An outpatient *follow-up* was performed 3 times a week for 4 weeks. Clinically, the patient remained stable.

Outpatient care has continued without any additional complaints.

Figure 1.

Figure 2.

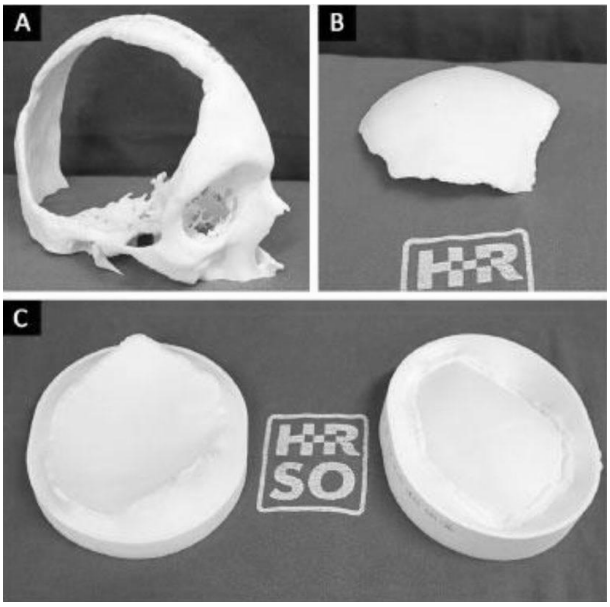

Figure 3.

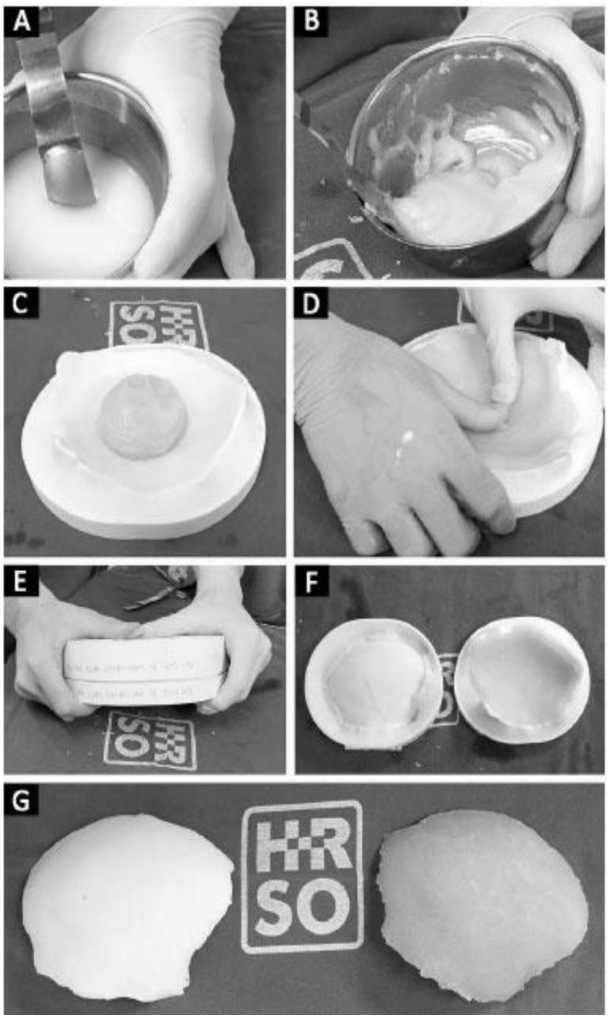

Figure 4.

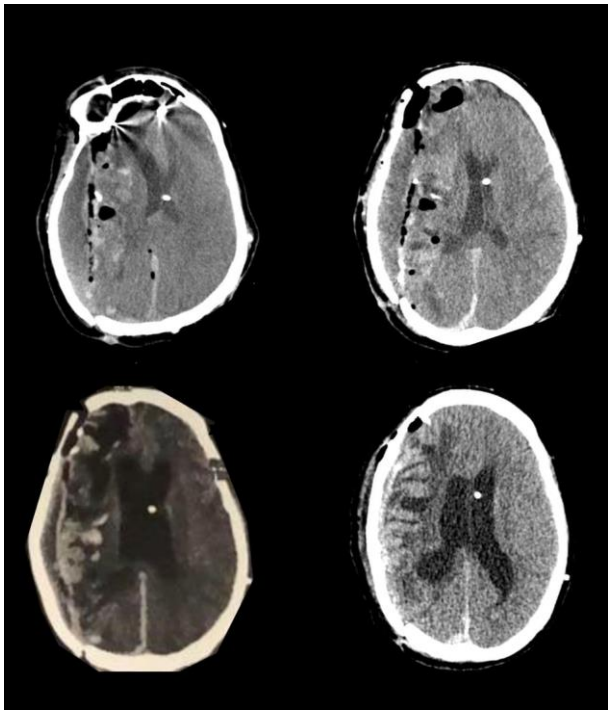

Figure 5.

Figure 6.

Discussion

DC is indicated for the treatment of elevated intracranial pressure in grave situations of traumatic injury to the brain. This procedure consists of the removal of a significant part of the skullcap. The removal entails parts of the frontal, temporal, parietal, and part of the sphenoid from the affected side, permitting the free expansion of cerebral edema without exceeding limits inside the cranial vault. Even though this procedure saves lives, in many cases it leaves behind grave aesthetic and functional disadvantages for the patient. Even after the decrease in cerebral edema and when the

patient has achieved a favorable clinical profile, cranial reconstruction is recommended. The surgery seeks to recover cerebral protection against traumas, recover the cranial contour and improve neurological symptoms by re-establishing physiological intracranial pressure. The restoration of the anatomic barrier between intracranial structures and the environment normalize the dynamics of CSF and the blood flow inside the brain.[1,4,5]

Even though the ideal moment for performing cranioplasty remains uncertain, recent studies show that it should be done between 3 to 6 months later, in order to allow for significant motor skill and cognitive recovery.[4,12]

Cranioplasty after DC for management of elevated intracranial pressure is a neurosurgical procedure that seeks to restore stasis, improve the dynamic of CSF and establish the conditions for protecting the brain. Under these circumstances, the procedure can facilitate neurological rehabilitation and potentially improve neurological recovery.[11,15] Otherwise, cranioplasty can be associated with complications and even morbidity.[2,13]

A significant cranial defect after DC can alter the dynamics of the circulation of CSF and turn itself into a risk factor in terms of hydrocephalus In relation to complications after DC in patients suffering TBI, hydrocephalus is present in between 2 to 29% of patients. In this context, patients submitted to DC may need cranioplasty and VPS after the subacute stage.[1,8,12]

Diagnostic of hydrocephalus in people who have had hemicraniectomy surgical procedures done is necessarily subjective, since criteria based on measuring intracranial pressure or details on ventricular structure are generally not reliable in the scenario of an open cranial vault. However, the almost universal disposition towards the progressive accumulation of CSF in these individuals, frequently manifested as an increase in extra-axial gatherings over the hemispheric convexity, indicates an incapacity for adequately balancing the production of CSF by draining the venous sinuses.[3,14]

The physiopathology of patients operated by craniectomy has still not been well established. However, it is believed that the absence of skull bone coverage near the arachnoid granulations modifies the hydrodynamics of fluid absorption. Besides this, it is possible for other factors to contribute, besides alteration in the dynamics of intracranial pressure – for example, mechanical blockage or inflammation of arachnoid granulations because of post-surgical remains. Furthermore, these patients could present other isolated risk factors for hydrocephalus – for example, subarachnoid hemorrhage.[3,10]

The management of the dynamic of fluids after hemicraniectomy can be quite a challenge as a result of problems in hemispheric change or compartmentalization, emphasizing that the definitive resolution for the accumulation of CSF is a great priority. In patients with a bulging scalp flap and ventriculomegaly (VM), some authors indicate temporary management until cranioplasty by way of frequent lumbar puncture or the placement of a ventricular or external spinal tap. Some studies demonstrate that cranioplasty, performed as early as possible, can promote an immediate solution for the problem of hydrocephalus. In spite of this, many patients possess a persistent hydrocephalus, especially when cranioplasty occurs at a late stage. This can make them predisposed for the necessity of a previous shunt, as described in this case study.[8,10,12,14]

In the patients who have gone through craniectomy and whose hydrocephalus is persistent, the literature's point of view is still controversial as to the management of and the adequate time for applying shunt and cranioplasty. Recent data suggest that patients submitted for cranioplasty procedures and VPS by stages can benefit from less complicated results when compared to patients who go through the two procedures at the same time. Some authors will defend the shunt for managing hydrocephalus and cranioplasty at a later stage.[3,6,8,9]

Cranioplasty in patients with VPS may be challenging, mainly in those patients presenting a sunken flap due to the shunt. In patients with the sunken flap, there is a technical difficulty at the time of operating during the separation of the cutaneous layer, dura mater and encephalic tissue. Besides this, in these patients, due to the presence of the VPS, a major difficulty can occur in the expansion of cerebral tissue, facilitating the gathering of material in the vacant space between prosthesis and encephalic tissue that has not expanded. Until now, there are no randomized studies that can guide one through the knowledge in handling these situations. In the case described, what was chosen was the

closing of the VPS in order to permit parenchymal expansion after the cranioplasty procedure.[3,8,10,12,14]

As in the majority of hydrocephalus cases, after decompressive craniectomy there is a spontaneous resolution with cranioplasty. VPS occlusion was maintained after cranioplasty, and a rigorous post-operative observation was conducted with regard to expansion inside the brain and the necessity for a shunt. After one week of observation, involving more than one hospital, with good clinical improvement, a follow up was done on an outpatient basis, three times per week for 4 weeks. No worsening in the patient's condition was observed.[3,8,9]

The literature is yet to offer a standard for managing cranioplasty in patients with previous shunt and sunken skin flap. In spite of the success in the case related, there is no relevant sample or previous works available to compare such a case. New studies are fundamental to pave the way for a better approach to these patients.

Conclusion

Therefore, the path chosen for cranioplasty procedure, associated with occlusion of the VPS, is quite rare. We still do not know much about the best approach for cranioplasty in patients who have a previous shunt and sunken skin flap. In spite of the case's favorable outcome, new studies are fundamental in order to discover the best way to approach the problem

Disclosure

The authors report no conflicts of interest

Abbreviations and Acronyms

CSF = cerebrospinal fluid; DC = Decompressive craniectomy; ICH = intracerebral hemorrhage; ICP = intracranial pressure; PMMA = polymethylmethacrylate; TBI = traumatic brain injury; ICU = intensive care unit; VM = centriculomegaly; VPS = ventriculoperitoneal shunt.

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Romanian Neurosurgery (2020) XXXIV (1): pp. 411-415
DOI: 10.33962/roneuro-2020-064
www.journals.lapub.co.uk/index.php/roneurosurgery

The Idiopathic Hypertrophic Spinal Pachymeningitis. A case report and review of literature

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Keywords
chronic inflammatory spinal pachymeningitis,
idiopathic,
paraplegia,
cord compression

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ABSTRACT

Idiopathic hypertrophic spinal pachymeningitis (IHSP) is a rare inflammatory condition characterized by chronic inflammatory hypertrophy of the dura mater. It can involve the entire spine. However, most cases are reported in the cervical and thoracic spine. It can progress from local pain to radiculopathy and eventually develop myelopathy. The aetiology of IHSP is not known. However, it has been suggested to be associated with many diseases. Here we report a case of IHSP in 21-year-old female who presented with paraplegia. The diagnosis was made on MRI Spine and histopathological examination. It was treated with surgical decompression, steroid therapy and patient improved gradually.

INTRODUCTION

Spinal IHP is a rare disease which causes chronic inflammatory hypertrophy of the duramater. Its etiology is still unknown. It most commonly involves cervical and thoracic spine1,2,3. Commonly presents in the age of 6th and 7th decade of life4. Symptoms arises due to progressive compression of adjacent structures. We report a rare case of IHSP in a 21 year old women who presented with paraplegia.

CASE REPORT

A 21 years old female presented with history of progressive weakness of bilateral lower limb since 2½ months, was admitted in department of neurosurgery, SMS hospital jaipur. On examination, modified Ashworth scale of spasticity was grade 2 in bilateral lower limb, Medical Research

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Council power grade was 0/5 in both lower limb, bilateral Knee and ankle reflex was +3. Bilateral Babinski sign was positive and superficial abdominal reflex was absent. 70-80 % sensory loss of all modality below T2 dermatome level was present bilaterally. Other systemic examinations were normal. MR imaging of the thoracic spine demonstrated an intradural extramedullary lesion extending from C7-D4 level that was hyperintense on T1 and hypointense on T2 and nonenhancing on contrast . The lesion was compressing the spinal cord with cord edema. All routine hematological investigations were within normal limit. All the relevant investigations related to the cause of spinal pachymeningitis were negative. After D1–D5 laminectomy, duramater was found to be thickened compressing spinal cord. Posterior excision of duramater was done. A Durafoam was placed posteriorly to expand the thecal sac and specimen was sent for histopathological examinations. Chronic inflammation with lymphoplasmacytic infiltrate and fibrosis was found on histology. Postoperative steroid therapy was given. patient was under follow up and after 8 months, patient developed some sensation in both lower limb and power of lower limb improved from 0/5 to 2/5.

Figure 1. MRI Cervico-dorsal Spine sagittal preoperative (A) T1 image- A hyperintense intradural extramedullary lesion present at C7-D4, compressing the spinal cord. (B) T2 image- A hypointense intradural extramedullary lesion present at C7-D4, compressing the spinal cord.

Figure 2. MRI Cervico-dorsal Axial image post contrast: (A) Minimal enhancing intradural extramedullary lesion present posterior to spinal cord (B) T2 weighted image- Hypointense lesion present posterior to spinal cord.

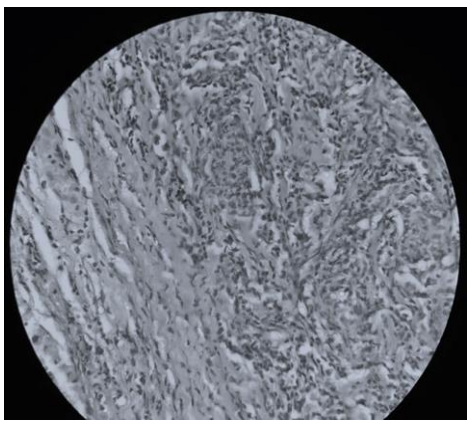

Figure 3. (A) Intraoperative image showing a grossly thickened dura (B) histopathological examinations showing fibrocollagenous tissue with dense infiltrate of lymphocytes and plasma cells.

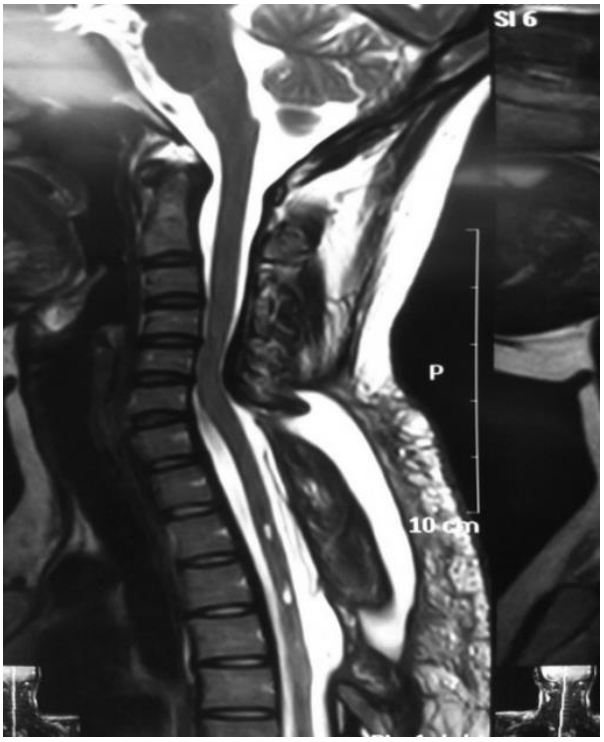

Figure 4. Postoperative MRI of the cervico-dorsal spine, sagittal view: showing the expanded cord.

Discussion

This disorder is rare and usually found intracranially and spinal form is extremely rare [5-8]. Friedman D et al found most cases of IHP intracranially[2]. It usually involves the cervical and thoracic dura or occurs as a craniospinal form [5,9,10]. The first case of spinal IHSP was reported by Charcot and Joffroy named as "Pachymeningitis hypertrophica cervicalis" in 1869[11,12]. Spinal IHSP is a rare cause of nerve root and spinal cord compression. It has been implicated in variety of inflammatory and infectious processes like tuberculosis, sarcoidosis, rheumatoid arthritis, wegener granulomatosis etc.[5-8]. So, other possibilities need to be ruled out as IHSP is a diagnosis of exclusion [5-7]. In our case, we could not find any predisposing illnesses such as infectious diseases or autoimmune diseases in spite of thorough investigations. Joffroy and Rosenfeld et al, described three stages; local and radicular pain in first stage. Signs of nerve root compression in second stage and spinal cord compression was described in third stage [12,13]. Haobin chen et al, reported age of the patients ranges from 28 years to 68 years (median age 56 years), and female being more commonly affected (male/female ratio: 6:9). Thoracic spine being most commonly affected followed by cervical and lumbar spine[14]. Friedman D and Flanders described peripheral enhancement on MRI which was present in all 3 patients as highly suggestive of hypertrophic pachymeningitis, which was also noted in another report[2]. Dumont AS and S.Pai et al, proposed extramedullary mass extending over multiple vertebral levels, strongly hypointense signal on T2- weighted images, and variable peripheral margin enhancement are suggestive of the IHSP[15,16]. In our case, extramedullary lesion was present in C7-D4 level which was hypointense on T2-weighted image and hyperintense on T1 weighted image. spine. Martin N et al reported good result of methotrexate treatment in cranial pachymeningitis[7]. Treatment choices for IHSP include surgical decompression, administration of corticosteroids, radiation therapy, and immunosuppressive agents but relapses are common [5,17]. Surgical decompression provides some relief, and early surgical intervention can successfully alleviate neurologic sequelae[1,9,10]. Naffziger and Rosenfeld et al, recommended surgical decompression by laminectomy and excision of the involved dura for its management [13,15,18]. Dumont AS and Kitai Ret al reported that biopsy with steroid therapy can reduce the thickness of the dura and can improve neurologic deficit[15,19].Naffziger and Dumont et al suggested laminoplasty instead of extensive laminectomy because it reduce back pain and increase spinal stability[15,18].In our case C7-D4 laminectomy with dural excision was done and post-operative steroid therapy was administered. After 8 months, power of lower limb improved from 0/5 to 2/5.

Conclusion

We reported a rare case of cervico-dorsal compressive myelopathy caused by IHP. Definite diagnosis needs thorough blood investigations, radiological and histopathological findings as IHSP is a diagnosis of exclusion. Surgical decompression along with steroid therapy can be considered as an effective mode of treatment. Early diagnosis and treatment can lead to better neurological outcomes.

Conflict of interests

The authors declare no conflict of interests.

Abbreviations and Acronyms

IHSP: Idiopathic hypertrophic spinal pachymeningitis; IHP: Idiopathic hypertrophic pachymeningitis; MRI: Magnetic resonance imaging.

Table 1. Idiopathic hypertrophic pachymeningitis – Age, location, mode of treatment and its response.

Author [Ref no.]	Age	Sex	location	Treatment	Outcomes
Pai et al [16]	68	F	Spinal: C6, C7	Laminectomy, steroid	spontaneous temporary resolution of symptoms, recurrence after surgery
Takahashi et al [20]	67	M	Spinal C3, C7	Corticosteroid	At the 2-year follow-up, the patient could walk independently
Ranasinghe et al [21]	65	M	Spinal T7, T8	Laminectomy, Corticosteroids	MRI improvement at 57 months
Yasuda et al[22]	28	M	Spinal: T1, T4, then L1, L3	Laminectomy, Corticosteroids	Recovered
Lai et al[23]	41	M	Spinal T2, T4	Laminectomy, dura excision	Unavailable
Present study	21	F	Spinal C7-D4	Laminectomy, dura excision	Partial Recovery

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Romanian Neurosurgery (2020) XXXIV (1): pp. 416-420
DOI: 10.33962/roneuro-2020-065
www.journals.lapub.co.uk/index.php/roneurosurgery

A thoracic intradural intramedullary epidermoid in a 12-years old female without any evidence of spinal dysraphism. A rare case report and review of literature

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ABSTRACT

The spinal epidermoid lesion is an extremely rare benign condition, seen in < 1% of all spinal tumours and are most commonly associated with spinal dysraphism. They are more common in young children and have female preponderance. They can be either congenital or acquired with congenital being more common. They are most commonly located in the thoracic region. They grow slowly and present with back pain and progressive neurological deficit with or without bladder bowel involvement. We present a case of a 12-year-old female child with gradually progressive neurological deficit in the form of spastic paraparesis and decreased sensation with early bladder involvement. Her contrast MRI dorsolumbar spine showed a well-defined intradural intramedullary lesion hypointense on T1 image, hyperintense on T2 image with no contrast enhancement at D10-D11 level. The patient was managed by surgical intervention with D9-D10-D11 laminectomy with total excision of the mass. Postoperatively on follow up patient had gradually improved motor and sensory symptoms with no improvement in bladder symptoms. Her histopathological study was confirmative of an epidermoid cyst.

Keywords
ectodermal cells, spinal epidermoid, spinal dysraphism, intramedullary, intradural

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INTRODUCTION

Spinal epidermoid tumors are rare occurrence found in < 1% of all spinal tumors in adults and 3% in childrens .[1-3] They are benign lesion with slow and indolent growth. They are derived from epidermal elements and can be congenital with entrapment of ectodermal cells during development and acquired by iatrogenic injury after surgery, trauma (penetrating injuries), lumbar puncture, myelography .[4-5] They are most commonly associated with spinal dysraphism. It was first reported by chiari in 1833. [6-7] There most common location is thoracic followed by lumbosacral region. Most of these lesions are found extramedullary and intramedullary location is a rarity .[8-9]

CASE REPORT

A 12 year old female child presented with complaints of gradual progressive ascending weakness and numbness of both lower limbs for 6 months with difficulty in walking and bladder involvement in the form of intermittent retention of urine for last 3 months .There was no history of back pain ,fever, trauma , any previous spinal procedure , cutaneous manifestation of spinal dysraphism .Her neurological examination revealed spastic paraparesis with increased tone (grade 3 modified ashworth scale)in both lower limbs , power 3/5 in both lower limbs . Reflexes were +3 in both knee, ankle. DTR were brisk and planter was bilateral extensor. Sensory examination showed decreased pain, touch and temperature sensation of around 60% below L2 spinal segments with decreased perianal sensation. Her systemic, general, local, spine and upper limbs were normal in examination.

MRI dorsolumbar spine revealed a well-defined, homogenous, oval, cystic intramedullary lesion seen at D10-D11 level. It was hypointense on T1 image (figure 1a), hyperintense on T2 image (figure 1b,1c,) with no contrast enhancement (figure 2a,2b). D9-D10-D11 laminectomy was performed with bulging dura seen. After opening of dura, a 1.5 x .8 x .5 cm intramedullary lesion, bulging out from the cord, and attached to cord on right lateral aspect was seen, it was well encapsulated, pearly white in color, smooth, soft to firm, avascular in nature. The cord was thinned out and shifted to left side. Total excision of mass was done with watertight closure of dura was performed.

Figure 1a. T1 weighted MRI axial view showing hypointense lesion.

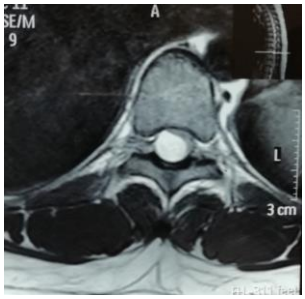

Figure 1. T2 weighted MRI b) sagittal view c) axial view showing single, cystic, hyperintense intradural lesion at D10 –D11 level, with cord shifted anteriorly and towards left side.

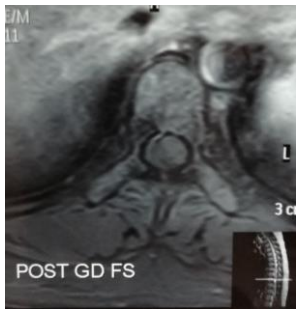

Figure 2. Post gadolinium contrast image a) sagittal, b) axial view with no contrast enhancement.

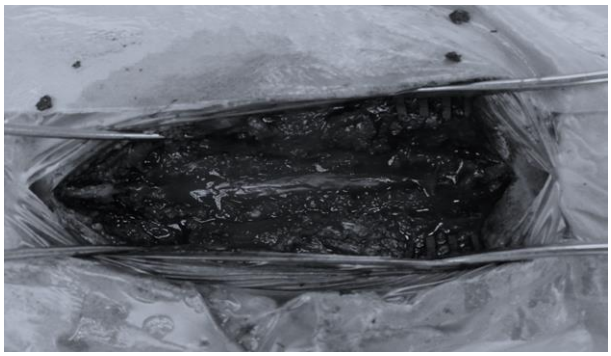

Image 1. Intraoperative image with dural bulge (arrow head).

Post-operative period was uneventful with mild improvement in power and tone. Post op MRI was done and revealed complete excision of mass (figure 3a, 3b, 3c). Pt was discharged and followed up at 3 and 6 months. Her power improved gradually to 4+/5 in both lower limbs and tone was decreased, sensory symptoms also improved but there was no

improvement in bladder symptom. Her histopathological study showed a fibrous capsule with stratified squamous epithelium with degenerated epithelial tissue (figure 4) confirming it to be epidermoid cyst.

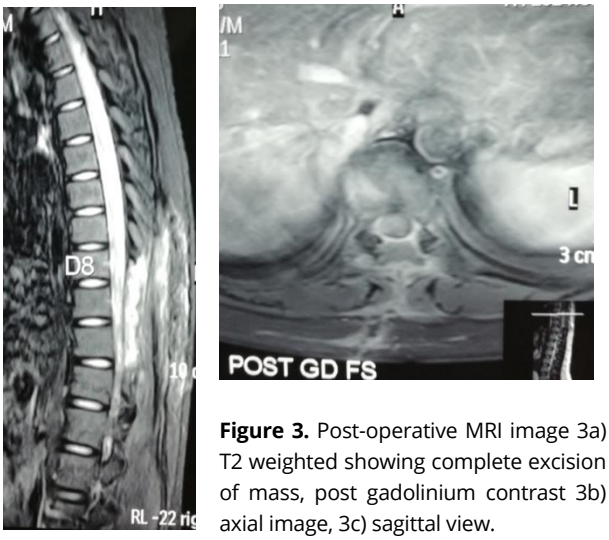

Figure 3. Post-operative MRI image 3a) T2 weighted showing complete excision of mass, post gadolinium contrast 3b) axial image, 3c) sagittal view.

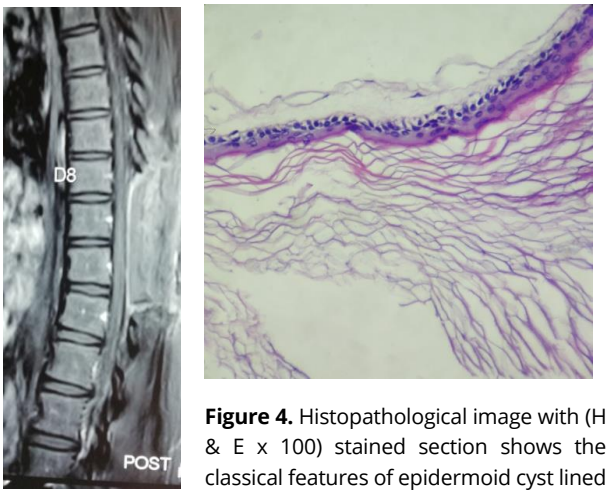

Figure 4. Histopathological image with (H & E x 100) stained section shows the classical features of epidermoid cyst lined with keratinizing stratified squamous epithelium with anucleate squamous debris.

Discussion

Spinal epidermoids are uncommon lesions and very rare without any evidence of spinal dysraphism. Its first description was given by cruveilhier in 1835 , who described them as tumors perlees (pearly tumors) .[10] Its first surgical removal was done by Gross in 1934 .[11] An incidence of .7% was reported by Guidetti and Gagliardi .[12] In 1956 Choremis *et al* described 6 case in childrens receiving subarachnoid injections of streptomycin for treatment of TBM . Roux *et al* in 1992 presented 47 cases of intramedullary epidermoid .[13] Manno et al reported a total of 90 cases, of which 39 were acquired and 51 congenital .[14] These are most commonly seen in thoracic spine in D4-D6 and D11-D12 region followed by lumbosacral region.

They are benign lesion which arise when epidermal cells are trapped into the thecal sac. This can occur congenitally or acquired. Congenital causes are spectrum of spinal dysraphism including spina bifida , myelomeningocele , split cord malformation- diastematomyelia ,dermal sinus ,syringomyelia , hemivertebrae .[15 -17-18] Love and kernohan in 1936 described them as congenital epithelial tumors .[16] They are believed to arise from displaced ectodermal inclusions in early development resulting from defective closure of neural tube between 3rd to 5th week of fetal life .[17] Dias and Walker corroborated a defect at the gastrulation stage .[18] On the other hand acquired lesions develop after iatrogenic displacement of epithelial cells after trauma , lumbar puncture ,previous surgery .[14,19] Gibson and Norris found epidermal tissue in needle bevels after skin puncture and needles with well fitted stylet did not carry any fragments with production of intramuscular dermoid in rats after injection of these skin fragments .[20] Oblu experimented development of intraspinal epidermoid cyst by introduction of dermoepidermal fragment into subrachnoid space of dogs by lumbar puncture .[21] A study by Van Gilder and Schwartz in rats showed , 89% of young rats develop intraspinal epidermoid and they failed to develop in adult rats .[22] Repeated punctures are thought to increase the risk of developing intraspinal epidermoid cyst . Since 1970 incidence has decreased because of use of styletted needles lumbar puncture.

The signs and symptoms are dependent on location of tumor and size of lesion. Slow and indolent growth causes the usual delay in diagnosis. They can range from back pain, painful radiculopathy to UMN signs below the level of lesion including weakness, spasticity, increased tone from involvement of corticospinal tract to sensory deficit, numbness and bladder -bowel dysfunction. Rarely they can also present with chemical meningitis .[23] Malignant transformation has not been documented so far in intraspinal epidermoid.

Radiologically, the X-ray of these patients are usually normal but may show the evidence of spinal

dysraphism, and scalloping of the vertebral bodies, and scoliosis. On CT spine they show widening of spinal canal, scalloping of the vertebral bodies, thinning of lamina. MRI of spine usually shows a well-defined homogenous lesion that is hypointense on T1 image , hyperintense on T2 image , shows intense diffusion restriction on DWI image and no significant enhancement on T1 gadolinium image [6,24,25,26,27,28,29]. Occasionally their margin may look "shaggy" due to leak of content causing chronic inflammatory response and gliosis along the margin .

Diagnosis is primarily done by proper history and clinical examination and established with preoperative radiological investigation and confirmed by postoperative histopathological findings. The differential diagnosis of these tumors includes dermoid, neurofibroma, meningioma, lipoma.

On HPE examination stratified squamous epithelium and an outer layer of collagenous tissue. With progressive desquamation, keratin breakage the inner part of tumor is filled with soft white material, rich in cholesterol .[13,28,30]

Treatment*:* In symptomatic patients. Total excision of mass without any neural damage is the primary goal. In asymptomatic cases found incidentally, it can be managed conservatively. Mostly their capsule is attached to the cord element, so some authors (including Rand and Rand [31]) believe in subtotal resection of tumor with preservation of neural structures. Subtotal resection frequently results in early relapse of symptoms and leak of contents causing more distressing meningitis. Total microsurgical excision with intact capsule is the best possible treatment option available [13,32,33], but may lead to some neurological injury. This can be prevented by using intraoperatively neuro-physiological monitoring guided by EMG and somatosensory evoked potential .[34] These lesions have high recurrence rate because of its dense adherence to neural tissue and the risk of recurrence is approximately equal to patients age plus 9 months .[35,36] Surgery remains the treatment of choice for recurrent lesions also. Radiotherapy was given to 1 patient with repeated relapses with good result .[37] Radiotherapy can be offered to multiple recurrence .[37] So, it can be considered an option for pts not fit to undergo surgery. Despite all this most pt treated surgically enjoy a normal life with good neurological function.

Conclusion

Spinal epidermoid tumors are extremely rare slow growing lesions with non-specific presentation. Most commonly these are congenital lesions seen in children. Mostly they present with gradually progressive neurological deficit. Diagnosis is predicted MRI with DWI features and are confirmed by histopathological evidences. Early diagnosis and prompt treatment limit the neurological deterioration. Surgical excision is the treatment of choice. The preoperative neurological status is the prime determinant of functional outcome. Maximal safe resection results in cessation of clinical progression and remission of symptoms. Subtotal resection results in tumor recurrence and surgery is the treatment option. Radiotherapy can be offered to multiple recurrence.

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Romanian Neurosurgery (2020) XXXIV (1): pp. 421-423
DOI: 10.33962/roneuro-2020-066
www.journals.lapub.co.uk/index.php/roneurosurgery

Spinal cord injury without radiologic abnormalities in a 4-years old boy. A case report

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Keywords
trauma,
spinal cord,
children

ABSTRACT
In this study, we describe a 4-year-old boy with a head, cervical, and left shoulder injury following the reception of a heavy metallic door on his left side that resulted in SCIWORA.
SCWORA is defined as spinal cord injury without x rays or CT scans lesions but can be detectable on RMI scans. SCIWORA follows trauma, sometimes trivial. The management consists of immobilization and nursing. The clinician should be aware of this entity. We are reporting the management of a four years old boy.

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INTRODUCTION

The term SCIWORA (spinal cord injury without radiographic abnormality) was coined by Pang and Wilberger in 1982 [1].

It is defined as objective signs and symptoms of myelopathy with no evidence of fracture or ligamentous instability on plain spine radiographs and tomography. This definition excluded all MRI findings and any injuries from open or penetrating trauma, electric shock and all related to congenital spinal anomalies. Ergun et al [2] highlighted the patho-physiological basis of SCIWORAs. The anterior spinal artery receives its main blood supply from vertebral arteries. Hyper motion effect of the spinal cord during trauma may provoke a temporary occlusion or spasm of the vertebral arteries, the anterior spinal artery

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or the distal branches of the central sulcus arteries leading to a spinal cord infarction.

Other anatomic factors considered by Ergun include the vertebral alignments of cervical spine; the planes of the facet joint which are more horizontal, the anterior portions of vertebral bodies are weighed forward, the uncinate processes are flat, the under developed musculature of children's neck and the heavy head. These above predispose the cervical column of children to flexion, extension or longitudinal distraction injuries. Trigylidas et al [3] conclude that the innate ligamentous laxity of the pediatric spine may protects it from injury in minor trauma, but these same protective features expose them to permanent injuries in high intensity trauma. Here we are reporting a case of SCIWORA in a 4years old boy.

Observation

A 4 years old boy polytraumatism referred from Poudriere general hospital with head injury with initial loss of consciousness, cervical trauma and left shoulder trauma. The patient was stamped by a heavy metallic door on the head, neck and the left shoulder. At admission the Glasgow scale was normal, left mydriasis, bilateral palpebral oedema. He was tetraplegic spontaneous mobilization of the cervical spine, lost of sensation of the right hemi body and reduced sensation of the left side. The ROT are absents in the 4 limbs, no Babinski sign, cremasteric sign is abolished and the anal sphincter was not tonic. The lesionnel level was the mammary line. The standard radiograph of the head, the cervical spine was normal [fig1]; the left shoulder X ray revealed a mid-shaft clavicle fracture. A cerebrospinal CT scan was performed, but was normal.

Figure 1. Lateral X ray of the cervical spine, no lesion observed.

We requested a neurologic view. The patient returned to us with same examination findings. The diagnosis of SCIWORA was made the patient placed in ICU, nursing and resting was consigned and a cerebrospinal RMI was performed. It revealed no cerebral lesion but there was thickening of the spinal cord from C3 to C7 with narrowing of the perimedullar space [fig 2].

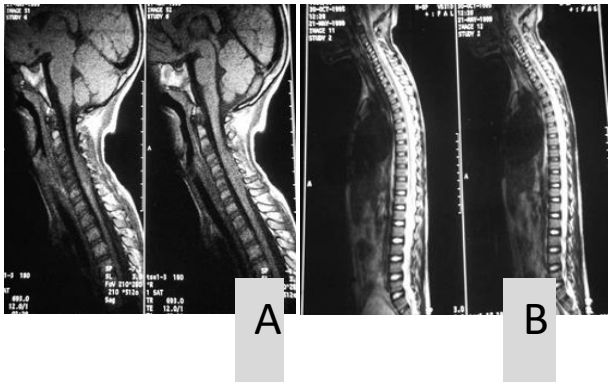

Figure 2. Cervical spine RMI Showing cord thickening from C3 to C7; no other lesion was recorded. A: Sagital T1; B: Sagital T2.

Discussion

SCIWORA accounts for 1-10% of the spinal cord injury in children. Gregory et al [5] reported 0.08%cases in a multicentric study on about 34069 patients., SCIWORA is more common in children than adult with literature reports of 13 to 19% and 10 to 12% of children and adult spinal cord injuries respectively [6, 8, 9]

The trauma causing SCIWORAs may not be of high intensity only, it may be trivial also. This is widely illustrated in the literature. Trigylidas [3] found 51% cases of sport related injuries, 36% of RTA and 13% of falls. Ergun et al reported a 12-year-old girl who slipped on her way home from a volley ball match and developed SCIWORA. Our patient was stamped by a heavy door. Injury to the cervical spine is much more common than the thoracic and lumbar spine. The upper cervical spine (C2, C3) is more prone to flexion injuries. Snoek [4] reported that traction injuries may be common in thoracic and lumbar spine.

Clinical features classically include neurological deficit (decrease reflexes and power, reduced sensations below the level of the lesion, reduced anal tone and loss of bladder control), torticolis [4]. These findings correlate with our patient. The IRM findings ranged from spinal cord contusion [2], central disc

herniation [2, 5], paramedian disc herniation [5], spinal cord stenosis [5], ligamentous injury spinal cord edema [4] and hemorrhage [4]. The corresponding standard radiographs and CT scans were all normal. We found cord edema in IRM reports, Lammertse et al [7] coined IRM as the gold standard in diagnosis of spinal cord injuries. The outcome of SCIWORA may be good or devastating to the patients and their families'. The patient in our report sustained permanent injury. Snoek reported a good prognosis in a child who sustained bifocal SCIWORA following RTA. The outcome is independent the intensity of the trauma. It would rather be influenced by the patient age and the level of the lesion. The development of the cervical spinal musculator reaches it fulcrum by the age of 8. Lesions below 8 years of age may have poor prognosis especially associated with upper cervical injuries [2]. Above 8 years the spinal architecture is approaching that adult, thus lesions occur in lower spine under high velocity trauma.

Conclusion

SCIWORA is a disease entity of children and adult. Following injury, a high index of suspicion is necessary in injured patients in whom movement of all limbs is not seen. MRI is standard diagnosis and the outcome depends on the spinal injury.

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Romanian Neurosurgery (2020) XXXIV (1): pp. 424-426
DOI: 10.33962/roneuro-2020-067
www.journals.lapub.co.uk/index.php/roneurosurgery

Fever as an independent prognostic factor in traumatic brain injury

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ABSTRACT

Traumatic Brain Injury (TBI) patients more often than not develop fever within the first few days of their hospitalization. Studies report that causes are variable and according to the pathogenesis, fever may be harmful or protective. The study was conducted to correlate the development of fever with clinical prognosis. Throughout the study spanning 6 months, a total of 98 patients of TBI were included. In the first 48 hours, 54 patients did not develop fever (temperature >37°C), 20 patients recorded temperatures between 37°C and 39°C; and 24 patients developed high fever (39°C). On regular temperature monitoring and follow up, it was found that patients developing fever relatively early during hospitalization were more likely to end up with a poor outcome (Glasgow outcome scale 4 to 5). Therefore, fever is independently a predictor of poor prognosis in TBI patients and should be managed diligently in the first few days.

Keywords
fever,
prognostic factor,
traumatic brain injury

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INTRODUCTION

A major proportion of neurosurgical patients are victims of traumatic brain injury (TBI). TBIs are a leading cause of morbidity, mortality, disability and socioeconomic losses in India and other developing countries. Road traffic accidents are the leading cause of TBIs followed by falls and violence.[1] According to indianheadinjuryfoundation.org India has the unfortunate distinction of having highest rate of head injury in the world.[2]

A large proportion of patients with any type of acute brain injury will develop fever within the first few days of their ICU or hospital stay. The causes are variable. Often the patient gets non-infectious fever which is a direct consequence of brain injury itself in addition to the high risk of infections in brain-injured patients.[3]

We aim at studying the effect of hyperthermia in patients admitted with moderate to severe head injury and try to correlate the outcome of these patients with hyperthermia.

MATERIALS AND METHODS

This study was conducted at a tertiary health care centre (IPGMER and Bangur Institute of neurosciences, Kolkata, India) between July 2019 to December 2019.

All patients with Glasgow Coma Scale of 12 or below admitted with history of head injury were included.
Exclusion criteria:

- All patients with normal radiological findings (CT scan or MRI)
- All patients requiring surgical intervention (except minor procedures like repair of open wound, tracheostomy etc). The study was limited to patients managed conservatively.

All patients fulfilling the above criteria were included in the study. The temperature was recorded 6 hours. According to the reading, the patients were divided into three groups upto 37℃, temperature 37℃-39℃ and temp >39℃. All patients were managed for head injury appropriately and patients requiring surgical intervention at a further stage (due to failure of conservative management) were dropped from the study.

The outcome of the patients was divided into two: good outcome (Glasgow outcome scale score 4 to 5) and poor outcome (Glasgow outcome scale score 1 to 3).[4]

Results

A total of 98 patients were included in the study. The most common mode of injury was road traffic accidents (66 patients), fall from height (18 patients) and assault (10 patients). (Table 1) Of these, 62 patients were males and 36 patients were females. The average age of male patients was 39.4 years and that of females was 42.2 years.

All these patients were regularly monitored for hyperthermia. As hyperthermia within 48 hours of TBI is more significant, so the patients were divided into three groups at the end of 48 hours. The result are tabulated in Table 2.

The patients who did not develop hyperthermia within the first 48 hours were followed up for any subsequent development of hyperthermia and were again categorized into three groups. The data obtained was tabulated in Table 3. The patients who developed temperature >39℃ in any time period were treated vigorously with antipyretics, sponging and suitable antibiotics. The patient who developed fever >37℃ but less than 39℃ were managed with sponging and antibiotics but no antipyretics.

Mode of injury	Male	Female
RTA	40	26
Fall from height	12	6
Assault	7	3
Others	3	1

Table 1. Mode of injury and gender wise distribution.

Temperature within 48 hours	Good outcome	Poor outcome	Death
Upto 37℃	36	12	6
37-39℃	8	6	6
>39℃	2	3	19

Table 2. Proportion of patients developing hyperthermia within first 48 hours and outcome ratio.

Temperature after first 48 hours	Good outcome	Poor outcome	Death
Upto 37℃	18	4	1
37-39℃	14	7	3
>39℃	4	1	2

Table 3. Proportion of patients developing hyperthermia after first 48 hours and outcome ratio.

Discussion

Fever occurs with an incidence of up to 70% in neurologically injured patients and typically is not an isolated event but rather a sustained response seen for as long as 2 weeks following injury. Only half of the febrile patients are attributable to infection. In one fifth to one third of cases, fever remains unexplained despite extensive diagnostic workup.[5]

Early fever (within 24 hour) has been associated with an increased relative risk of a poor outcome by 2.2 fold with every 1℃ increase and even a 0.5℃ increase may lead to a series of secondary injuries and neuron death.[4,6]

What adds to the already complicated situation is that after major brain injury, brain temperature is higher than and can vary independently of systemic temperature. In such scenario, the brain is extremely sensitive and vulnerable to small variations in temperature.[7] There is growing evidence that elevated body temperature may be detrimental in patients with acute neurological disorder. Many work done till date shows that elevated body temperature is associated with increased mortality rate and poor functional outcome.[3,8]

The epidemiological and etiological spectrum of traumatic brain injury revealed in our study agrees with the already available data.[2]

Our study reveals that temperature >39℃ within first 48 hours of traumatic brain injury after

admission is significantly associated with higher death rate which concurs with other studies like that of Bao L et al.[4] The net effect of fever is reduction in internal perfusion pressure with diminished oxygenation of brain tissue resulting in cerebral oedema. The association of early development of fever with poor outcome is probably because already compromised brain tissue is more susceptible to oxygenation deficit.[9] In addition, fever causes a generalized increase in metabolic rate (7-10% increase per ℃ increase in core temperature), with corresponding increase in minute ventilation and oxygen consumption which can be detrimental.[3]

Our study does not establish a significance to delayed development of hyperthermia and poor outcome. This needs to be evaluated further.

According to some studies, the hospital stay is likely to be increased in patients who develop hyperthermia.[10] It remains to be studied and should be evaluated further in the backdrop of a developing country like India where resources are limited.

Targeted temperature management like therapeutic hypothermia has been touted as a valid candidate of neuroprotective treatment but the same has not been proven in larger randomized controlled trials. While overwhelming majority of TBI injury patients may benefit from fever control, the patients with severe infection who need an inflammatory response might not benefit and may even suffer adverse consequences. Additionally, in a small subset of patients both conditions may coexist.[11]

Conclusively, fever acts as an independent prognostic factor in patients of TBI in first few hours of admission and is associated with a poor outcome. It should be carefully watched for and vigorously treated. Further studies are required to assess the relation between delayed fever development and its impact on outcome. The pathophysiology of hyperthermia (infective or non-infective) also plays an important role in making a clinical decision.

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Romanian Neurosurgery (2020) XXXIV (1): pp. 427-433
DOI: 10.33962/roneuro-2020-068
www.journals.lapub.co.uk/index.php/roneurosurgery

MRI spectrum and prevalence of lumbar spinal degenerative disease in patients with non-traumatic low back pain

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Keywords
degenerative changes,
disc bulge,
facet hypertrophy,
intervertebral disc,
low back pain,
sciatica

ABSTRACT

Background. Low back pain (LBP) is a frequent cause of global disability and activity limitation. In the majority of cases, LBP is nonspecific, yet diagnostic confirmation is required to rule out serious underlying pathologies such as infection, tumour, fracture or degenerative disease. It can be done by a number of imaging techniques. Of all available techniques, MRI is currently the imaging modality of choice owing to lack of radiation, multiplanar reformation capabilities and high contrast resolution.

Objectives. To determine various MRI patterns and the common sites of spinal degenerative lesions among patients with LBP.

Method. This study was conducted on 622 patients suffering from non-traumatic LBP, referred for MRI of the lumbar spine. MRI database of the study population were analysed using axial T2-weighted, sagittal STIR, T1and T2-weighted and coronal STIR images. After excluding patients with h/o prior surgery and MR findings suggesting infective or neoplastic etiologies, 598 patients constituted the sample size of our study.

Results. A review of 598 patients with LBP revealed that degenerative changes in intervertebral disc were the most common abnormality detected. Among these, Disc bulge was the most common abnormality followed by disc desiccation, protrusion, extrusion, HIZ/annular tear, reduced IVD space and Schmorl's nodes. Other non- disc degenerative findings were Modic endplate changes, facet joint arthropathy, osteophytes, Spinal canal stenosis and Ligamentum Flavum hypertrophy.

Conclusions. Results reported the common occurrence of lumbar disc degenerative disease in patients with low backache. Research efforts should attempt to trim down risk factors and perk up the quality of life.

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INTRODUCTION

Low Back Pain (LBP) is one of the most common causes of hospital visits and is the leading cause of activity limitations and work absences in many parts of the world.[1,25,9]In the 2016 Global Burden of Disease study, musculoskeletal conditions were the second highest contributor to global disability, and lower back pain remained the single leading cause of disability.[32] LBP poses a considerable monetary menace to

the individual, family, workplace and society. Patients presenting with LBP frequently need imaging investigations to determine the cause. Standard radiography isusually the first investigation to perform, with MRI or CT only usedfor further workup [10]. Despite technical advancements inimaging, the specificcause of the pain can only be determined in less than 50% of cases [7]. This study was conductedto determine various MRI patterns and the common sites of spinal degenerative lesions among patients with LBP.

Materials and methods

This study was conducted on patients with LBP referred to Radiology department of Dr Ram Manohar Lohia Institute of Medical Sciences, Lucknow for MRI of lumbosacral spine. It was a cross sectional observational study conducted from June 2017 to May 2018. Permission for study was taken from the ethics committee of our Institute. Consent from all patients was taken before their enrolment in the study. Patients name, age, sex and detailed history were obtained. MRI of the lumbar spine was performed with a 3 T (GE) MR imager using spine phased array coils. The scans consisted of axial T2-weighted, sagittal STIR, T1and T2-weighted and coronal STIR images with slice thickness of 4.0mm for each plane. A field of view of 30x30mm for sagittal and coronal images and 18x18mm for axial images were used. The images were stored directly as DICOM files in the workstation.

Inclusion Criteria:
- All patients with non-traumatic LBP referred for MRI lumbosacral spine.

Exclusion Criteria:
- Patients with history of recent trauma;
- prior lumbar spine surgery;
- metallic implants and pacemakers; and
- cases with MR findings s/o infective or neoplastic etiology.

Statistical Analysis -Statistical analysis was done using SPSS 15 software. Percentages were calculated for the various categories.

Results

There were 622 patients of LBP referred for MRI to Radiodiagnosis department. Out of these 24 were excluded from the study as 18 had radiological diagnosis of infective pathology and 6 had neoplastic etiology. A total of 598 patients constituted the sample size of the study. Out of the total patients there were 278 males (46.49%) and 320 (53.51%) females. The age of the patients ranged from 18 years to 80 years. Most common age group was between 31 to 40 years (33.76%). Distribution of various degenerative spinal abnormalities detected on MRI is shown in Table 1.

Type of Abnormality	Frequency (%)
Disc Bulge	**72**
Disc dessication	**69.56**
Disc Protrusion	**27.42**
Disc extrusion	**4.34**
Disc sequestration	**0.83**
HIZ /Annular tear	**28.12**
Schmorl's nodes	**12.67**
Osteophytes	**51.83**
Facetal arthropathy	**30.43**
Modic changes	**23.21**
Flaval hypertrophy	**13.04**
Spinal stenosis	**53.54**
Vertebral collapse	**6.5**
Transitional vertebra	**6.8**
Spinal Listhesis	**5.2**

Table 1. Distribution of various degenerative spinal abnormalities detected on MRI.

Degenerative changes in intervertebral disc were the most common abnormality detected. Among these, Disc bulge was most common abnormality, constituting 72% of the total study population. It was followed by disc dessication (in 69.56%), disc protrusion (in 27.42%), disc extrusion (in 4.34%) (Fig-1), HIZ/annular tear (in 28.12%), reduced IVD space (in 20.54%) and Schmorl's nodes in 12.67% of the patients (Fig-2).

Figure 1. T2W sagittal and axial images of LS spine show disc bulge (a, b), disc protrusion (c, d), disc extrusion (e, f) and sequestration (g, h) marked by arrows.

Figure 2. T2W sagittal images show multiple level disc desiccation with grade 5 changes at L5/S1 level (arrow in fig a), multiple Schmorl nodes (b) and posterior high intensity zone at L3/4 level (c, d).

Other non- disc degenerative findings were Modic end plate changes (in 23.21%), facet joint arthropathy (in 30.43%), osteophytes (in 51.83%), (Fig-3).

Spinal canal stenosis in 53.54% and Ligamentum Flavum hypertrophy (in 13.04%) of patients (Fig-4).

Other less commonlyseen but important findings included: vertebral collapse (in 9.1%), transitional vertebral complex (in 6.8%) and spinal listhesis (in 5.2% of the population) (Fig-5).

In our study, disc bulge was the most common abnormality (72%) seen in patients with low back ache. It was most commonly seen at L4-L5 (in 39.30%) followed by L5-S1 (in 28.14%) and L3/4 (in 24.18%) levels. Single level bulge was seen in less than a quarter of patients, (in 24.18%) while multiple level involvement was a more frequent finding (in 75.82%). Posterocentral disc bulge was most commonly seen followed by paracentral, forminal and extraforaminal types.

Figure 3. a) Sagittal T1WI shows Modic 2 end plate changes. b) Sagittal T2WI shows marginal osteophytes. Axial T2WI show Ligamentumflavum hypertrophy (c) and facet joint hypertrophy (d).

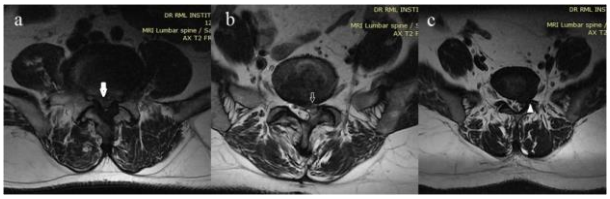

Figure 4. Axial T2WI show central canal stenosis (a), lateral recess stenosis (b) and neural foramina stenosis (c).

Disc dessication was the 2nd most common abnormality detected in 69.56% of total study population. Grade 3 degenerative changes were the most common pattern followed by grade 4 and 5 changes. All these changes were most commonly involving L4/5 and L5/S1 levels.

Figure 5. Sagittal T2WI show vertebral collapse (a), spondylolisthesis (b) and Transitional vertebral complex (c).

No sex predilection was noted in cases of disc prolapse. Most common age of presentation for both protruded and extruded disc was 31-40 years. Disc protrusion was seen in 27.42% of total sample size, most commonly identified at L4-L5 (48%) and L5-S1 (29%) levels. Posterocentral disc protrusion (68.2%) was most common type followed by paracentral and forminal protrusions. Disc extrusion was demonstrated in 4.34% of the population, most common level being L4/5 followed by L5/S1.Posterocentral type extrusion was most frequently seen. Disc sequestration was seen in only 5 patients constituting 0.83% of total study population.

Annular tear is characterised by T2W Focal hyperintensity, described as High Intensity Zone (HIZ) in posterior annulus of the disc. It was noted in 28.12% of the total study population and was most prevalent at L4/5 and L5/S1 levels. These changes were most commonly seen between 60-80-year age group. Schmorl's nodes were seen in 12.67% of the cases and were most commonly involving 21-40yr age group males.

Lumbar canal stenosis was noted in 53.54% of the study population without any sex predilection. Multilevel involvement and bi-laterality was the most common presentation in this study. Bilateral lateral recess stenosis was most common pattern (in 58.68%) followed by bilateral foraminal stenosis in 23.24% and central stenosis in 18.08%. These patterns can be seen unaccompanied or in amalgamation.

Spondylolisthesis was demonstrated in 5.2% of population. Most common level involved was L4 over L5 and L5 over S1. Vertebral collapse was noted in 6.5% of the cases with anterior wedge collapsebeing the most common type. Lumbosacral transitional vertebral complex (LSTV) was detected in 6.8% of the population. Majority of the cases were showing sacralization of L5 vertebra with occasional occurrence of Lumbarization of S1.

Our study demonstrated vertebral end plate changes in 23.21% of cases. Modic type I and type II changes were seen in 3.16% and 20.05% of the population respectively. These changes were most commonly seen after age of 50 years. Marginal osteophytes were reported in 51.83% of our study population.

Facetal arthropathy was seen in 30.43% of cases and was more common in elderly patients. Most common vertebral level involved was L4/5 followed by L5/S1. Ligamentumflavum hypertrophy was noted in 13.04 % of the study population.

Discussion

Low back pain (LBP) is an important public health problem with many possible etiologies and uneven distribution. As a result, the existingenormous literature on LBP is not only heterogeneous but also conflicting. According to a global review published in 2012, point prevalence of LBP was 11.9%+2.0%, overall mean prevalence was 31.0%+ 0.6%, and the lifetime prevalence was 39.9% +24.3%.[12] Studies on Indian population have shown the prevalence ranging between 6.2% in general population to 92% in heavy physical workers. Such great variation can be attributed to the heterogeneity of the population.[4] The diagnostic accuracy of MRI for degenerative conditions of spine is high. MRI is 75% sensitive and 77% specific in diagnosing nucleus pulposus herniation, resulting in a positive predictive value of 84% and a negative predictive value of 64%.[30] Similar studies have shown high sensitivity of 96% coupled with lower specificity of 75% in the identificationof spinal stenosis[2] and sensitivity of 92% tied with higher specificity of 100% in evaluation of nerve root compression [6].

Our study demonstrated female predilection for LBP seen in 53.51% females and 46.49% males. It was in accordance with systematic review of the global prevalence of low back pain [12]and studies conducted on Indian population [23,26].

Most common age group who presented with LBP was 31-40 years (33.76%), an age group that is usually involved in strenuous physical activity. These

results were in accordance with those found by Kopec [17].

In our study, disc bulge was the most common pathology seen in 72% of cases which correlated well with previous studies [34]. It was seen most commonly at L4-5 level followed by L5-S1 and L3-4 levels respectively. These findings correlated well with study by Ma D et al [20]. In our study, single level bulge was seen in less than a quarter of patients with multiple level involvements being a more frequent finding. This finding was in accordance with studies by Pokhraj Suthar et al [28] and Osman et al [24]. Posterocentral disc bulge was most common type followed by paracentral and forminal bulge. These findings were well supported by Pintu et al [5].

Disc desiccation was the 2nd most common abnormality detected in 69.56% of total study population with the last two lumbar levels clearly predominating.These findings were supported by study done by Jarvik JG et al [15].While the gradesof disc degeneration are not much taken into account in the literature, so-called 'discreet or grade 3' changes seem to be more common than 'moderate to severe (grade 4/5)' changes [31].

Disc protrusion was next most common abnormality detected in 27.42% of population. This is unlike the findings of Pokhraj et al [28] who found disc protrusions in 62.24% and disc bulges in 27.39% of population. This discrepancy could be owing to preponderance of younger individuals in our study. Study by Pintu Biswas demonstrated high incidence of disc bulge (71.59%) as compared to protrusions (8.8%). [5) Disc protrusions were mostly seen in 60-80-year-old individuals, most common level being L4-L5 (48%) and L5-S1 (29%).

Disc extrusion was seen in 4.34% of the population, most common level being L4/5 followed by L5/S1. These findings correlated well with study by Jacob et al [13]. Disc sequestration was seen in only 5 patients constituting 0.83% of total study population.

Annular tear was noted in 28.12% of the total study population. It corresponds to a previous study by Aprill and Bogduk, which reported 28% prevalence of annular tear in patients with back pain. This study also concluded that HIZ was highly specific and strongly predictive of a painful disc. [3]

Schmorl's nodes were seen in 12.67% of the cases and were most commonly involving 21-40yr age group males. These findings correlated well with study by Jagannath D et al who reported the prevalence of 9.2% and these features being most common in 4th decade. [14]

Lumbar canal stenosis was noted in 53.54% of the study population whereas study by Shobeiri E et al revealed lower number of cases seen only in 37% of the cases.[27] Bilateralism and multilevel involvementwas mostcommon presentation in our study. Spinal stenosis can be central, lateral recess or foraminal. Central stenosis is a result of hypertrophy of the inferior facet articular process of cephalic vertebra. Lateral recess and foraminal stenosis occurs due to hypertrophy of the superior facet articular process of caudal vertebra.[19] Bilateral lateral recess stenosis was most common pattern (in 58.68%) followed by bilateral foraminal stenosis in 23.24% and central stenosis in 18.08%. These patterns can be seen unaccompanied or in amalgamation.

Spondylolisthesis was found in 5.2% of study population, with obvious female predilection. In a study performed by Frennered et al. the prevalence of spondylolisthesis in patients with LBP was estimated to be 2.5% which is less than that in present study. [8] Recent studies like He et al. and Layegh M, Hejazian E. estimated higher prevalence [13%] than what was approved in our study [11,18]. Most common level involved was L4 over L5 followed by L5 over S1which correlated well with previous studies. [18]

Vertebral collapse was noted in 6.5% of the cases with anterior wedge collapse being the most common type seen in approximately 80% cases. It was higher as compared to the study byMustapha et al who found wedge collapse in just 1.97% of the cases. It can be explained by higher number of female patients in our study whereas the previous study was a male predominant study [22].

Lumbosacral transitional vertebral complex was seen in 6.8% of the population. Majority of the cases had sacralized L5 vertebra and only few cases had Lumbarized S1. Study by Layegh on Iranian population found the prevalence of LSTV to be 9.8%. Of all patients, 8.2% had sacralisation of L5 and 1.6% had lumbarisation of S1 [18].

Our study demonstrated vertebral end plate changes in 23.21% of cases. Systemic review by Tue Secher documented that median prevalence of end plate changes is 43% in patients with non-specific LBP [29]. Type I and type II changes were seen in

3.16% and 20.05% of the population respectively. These findings correlated well with the original study of Modic et al which demonstrated that type 2 changes were the most frequent and may account for up to 90% of Modic changes.[21]

Marginal osteophytes were reported in 51.83% of our study population. Nemoto found osteophytes in 46% of patients and there was no difference between patients with and without back pain. [24] Facetal arthropathy was seen in 30.43% of cases and was more common in elderly patients. Although study done by P Y Yong et al was correlating with our study as the prevalence was 29.8%. [33] Study by A.K. Kohat et al had shown very high prevalence (75%) of facet joint arthropathy in chronic low backache patients. [16] Most common vertebral level involved was L4/5 followed by L5/S1which is supported by both the previously mentioned studies.

Ligamentum flavum hypertrophy was noted in 13.04 % of our study population. It was in accordance with study by PY Yong et al (prevalence 14.0%) but very low as compared to study by Kohat (prevalence 70.8%). It was predominantly seen at lower lumbar levels (L4/L5 and L5/S1).

Conclusion

Degenerative disease of the lumbar spine is a common condition that radiologists will come across frequently. Role of diagnostic imaging in patients with low back pain is to provide precise anatomic information which in turn affects the management. MRI is a mainstay in the evaluation of low back pain and degenerative disease of the lumbar spine. This paper highlights a variety of degenerative patterns affecting the vertebral bodies, intervertebral discs, facet joints, and ligamentum flava, as well as the collective effects of these changes on the spinal canal and neural foramina.

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Romanian Neurosurgery (2020) XXXIV (1): pp. 434-436
DOI: 10.33962/roneuro-2020-069
www.journals.lapub.co.uk/index.php/roneurosurgery

An uncommon intracranial malign tumour which was misdiagnosed as Glioblastoma multiforme: Hemangiopericytoma

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ABSTRACT

Background. Hemangiopericytoma (HPC) which is mostly located at the lower extremity and visceral organs was found extremely rare in the central nervous system. Radiological images are not enough to differentiate HPC from other CNS tumours. The case was analyzed to determine presurgical features for diagnosis and the challenges during surgery.

Case. A 65-year-old male patient with headache was diagnosed as Glioblastoma Multiforme (GBM) considering the image findings. However, the intraoperative macroscopic shape and tendency to bleeding were not relevant to the GBM. The mass was reported as Hemangiopericytoma which is a malign tumour, originates from pericapillary bodies of veins, and commonly locates out of the CNS.

Conclusion. Even in advanced age and radiologically considered high-grade glial tumours, HPC should be considered in the differential diagnosis for preoperative preparation.

Keywords
hemangiopericytoma,
diagnosis,
central nervous system,
surgery

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INTRODUCTION

Hemangiopericytoma (HPC), which is mostly located in the musculoskeletal system and the skin, is rarely seen in the central nervous system. It mostly locates in the lower extremities, retroperitoneum and pelvis 1,2. It accounts for 2% of meningeal tumors, less than 1% of all central nervous system (CNS) tumors1,3. HPC, first reported by Begg 4, originates from the Zimmerman pericytes around the capillary and postcapillary veins 1. It is a highly malignant tumor with a tendency to local and distant metastases. It is very difficult to distinguish radiologically from the glial and meningeal tumors observed in the central nervous system 5.

Our aim is to present the difficulties encountered during intraoperative treatment of HPC, which is rarely located in the CNS and radiologically mixed with high grade glial tumor.

Case report

A 65-year-old male patient had a headache and dizziness for about 1 month. He was diagnosed with benign positional vertigo and received medical treatment, but did not recover. The patient, who started complaining of weakness and nausea 3 days ago, worsened gradually and started to vomit and prone to sleep for 1 day. He was admitted to the emergency room with unconsciousness and the occipital mass was detected in the CT scan (Figure 1). The patient underwent contrast MR imaging in intensive care unit and a preliminary diagnosis of Glioblastoma Multiforme (GBM) was made radiologically, which was presented with edema, peripheral contrast, necrotic areas in the center (Figure 2). When the mass of the patient was reached during the operation performed under general anesthesia, it was observed that the mass was very smooth and shiny surface, rather fragile, bleeding tendency, but easily dissected in the surrounding tissues. When frozen sample was sent to pathology, it was reported that the mass was extremely malignant, but the diagnosis could not be made before immunohistochemical staining. Mass excision was performed by performing difficult bleeding control. It was observed that the mass was removed close to total by postoperative imaging. HPC was diagnosed after further pathological examination of the removed mass. After histopathological diagnosis, radiotherapy was planned and the patient was discharged in good condition. The patient who presented with the same complaints after 3 months showed that the mass recurred as large as the preoperative volume. The patient was missing while preparing for reoperation.

Discussion

Intracranial HPC is a malignant tumor that makes up less than 1% of all CNS tumors. The location in CNS is extremely rare. It tends to locate in the lower limbs and intra-abdominal visceral organs 3. Unlike other meningiomas, it is more common in young men (38-44 years) and tends to be supratentorial 6.

It is very difficult to differentiate HPC with benign meningioma before surgery 5. Differential diagnosis can be made by observing multilobulate mass and dural connection in CT and MRI imaging. In addition, osteolytic areas can be observed in x-ray and CT imaging. However, in our patient, there was no multilobulate mass and osteolytic area images in preoperative imaging (Figure 1 and 2). There was an edematous mass image with peripheral contrast, necrotic areas in the center. Considering the current imaging findings and the patient's age, primarily GBM was considered and surgical planning was done accordingly. However, it was understood that the character of the tumor was different due to its intraoperative macroscopic shape and the mass tending to bleed. Since meningeal artery bleeding is common during HPC surgery, preoperative arterial embolization is recommended to prevent bleeding 7. Tumor embolization also reduces the operation-related mortality and morbidity rate 3.

HPC tends to recur, especially in primer surgical localization. Distant organ metastases are frequently observed 8. 5-year life after postoperative adjuvant radiotherapy has been reported as 57% 9. However, despite our patient's radiotherapy, relapse was observed after 3 months and was mortal.

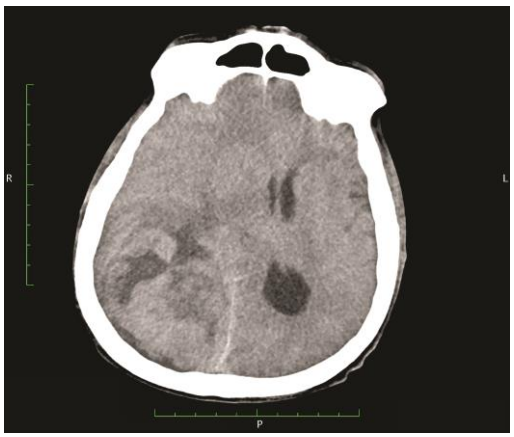
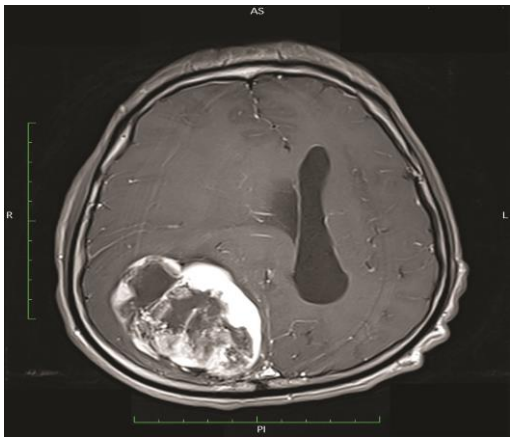


Figure 1. There was no osteolytic bone lesion in CT images.

Figure 2. Due to the peripheral contrast enhancing and the central necrosis, the preliminary diagnose was Glioblastoma multiforme.



Conclusion

HPC is a highly malignant tumor, which is more common in young men and rarely seen in CNS. It is quite difficult to make a preoperative diagnosis. Multilobulence in CT and MRI imaging and osteolytic bone lesions are valuable for preliminary diagnosis. Even in advanced age and radiologically considered high grade glial tumors, HPC should be considered in differential diagnosis for preoperative preparation. Unlike other glial tumors, preoperative tumor embolization should be planned since HPC tends to bleed more. In addition, since the distant metastasis of HPC is common, the whole-body scan must be performed.

Declaration of interest: None

Submission declaration: We confirm that this study is an original contribution, has not been published before and is not currently being considered for publication elsewhere.

Consent for publication: Science the patient died, the family were given detailed information of the procedure and informed written consent was obtained from them.

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Romanian Neurosurgery (2020) XXXIV (1): pp. 437-440
DOI: 10.33962/roneuro-2020-070
www.journals.lapub.co.uk/index.php/roneurosurgery

Craniopharyngioma and arteriovenous malformation operated using the same craniotomy. An unusual case

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ABSTRACT

Craniopharyngiomas (CPs) are rare benign epithelial tumours. Brain arteriovenous malformations (AVMs) are also rare lesions occurring in young adults. The appearance of both these lesions in the same patient is rare. A 42-year-old patient presented with headaches for 3 months and a progressive decrease in his visual acuity. Bitemporal hemianopsia was detected in the visual field. Magnetic resonance imaging (MRI) revealed a tumour with cystic and solid components located in the suprasellar region and AVM in the right temporal lobe. AVM (Martin–Spetzler grade III) was visualised using digital subtract angiography (DSA), which was fed from the right middle cerebral artery and drained through the sigmoid sinus via the inferior petrosal sinus. The patient was operated with enlarged right frontotemporal craniotomy. AVM nidus was totally removed at the first operation. Embolisation was not preferred before the AVM surgery. After 3 days, sylvian dissection was performed using the same craniotomy. The tumour was completely removed via the carotid cistern by making sharp dissection from the infundibulum. Post-operatively, the patient showed normal neurological examination and significant improvement in his visual field examination. There was no residual/recurrent tumour or AVM on contrast-enhanced MRI and DSA at post-operative 6 months. Histopathological examination revealed AVM in the first operative material and papillary-type CP in the second. The coexistence of these two rare pathologies has previously been reported in only one patient. This is the first case of surgical resection of CP and AVM using the same craniotomy.

Keywords
arteriovenous malformation, AVM, craniopharyngioma

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INTRODUCTION

Craniopharyngiomas (CPs) are rare benign epithelial tumours with slow growth. They account for 2%–5% of intracranial tumour primers, with an incidence of 0.5–2.0 billion per year.[7] The bimodal age distribution peaks between 0 and 19 and 40 and 79 years of age.[20] While complete improvement can be achieved with radical resection of CP, high recurrence rates have been reported for partial resection.[1]

Brain arteriovenous malformations (AVMs) are also rare lesions that usually occur in young adults. The prevalence of AVMs in the population is 10–18 per 100,000 adults.[11] Morphologically, in AVM, a vascular mass can be observed, with direct blood flow between the arterial and venous

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circulation without a true capillary bed. Generally, the high current feeder consists of the arteries, nidus and drainage veins.[3]

The coexistence of these two rare pathologies has previously been detected using radiology in only one patient. However, we presented the first case of simultaneous occurrence of CP and AVM in the same patient who was then treated with surgical resection.

Case report

A 42-year-old patient was referred to our clinic with persistent headache for 3 months and a time-lapse decrease in visual acuity. On magnetic resonance imaging (MRI), a 32 × 26 × 24-mm tumour with cystic and solid components extending from the supracellular region to the third ventricle was detected. At the same time, AVM of size 36 × 28 × 24 mm was observed in the right temporal lobe. AVM (Martin–Spetzler grade III) was seen in the patient's brain digital subtract angiography (DSA), which was fed from the right mild cerebral artery (MCA), with no aneurysm in the nidus, and sigmoid sinus was drained via the inferior petrosal sinus (Figure 1). Bitemporal hemianopsia was detected in the visual field of the patient. The patient then underwent an enlarged right frontotemporal craniotomy. The AVM nidus on the temporal basal was totally removed. Embolisation was not preferred before the surgery. As the duration of the first operation was relatively long, it was decided to perform CP surgery in the next session. After 3 days, sylvian dissection was performed using the same craniotomy. The cyst was aspirated by passing through the carotid cistern. The tumour was totally removed by making a sharp dissection from the infundibulum. After the surgeries, the patient was placed in intensive care for 1 day. His general condition was good, and his neurological examination was normal. However, diabetes insipidus developed after CP surgery. Nasal desmopressin therapy was therefore started but was discontinued after 1 month. The patient showed normal levels of pituitary hormones and no hypothalamic obesity. An improvement was noted in his visual field examination. There was no evidence of residual/recurrent tumour or AVM on contrast-enhanced MRI and DSA during the early post-operative period and at 6-month follow-up (Figure 2).

Histopathological examination

The resected tissue was fixed in 10% formalin, embedded in paraffin and stained with haematoxylin–eosin (H&E). In the first surgical material, vessels of different sizes and a lesion composed of the gliotic brain tissue was detected. Some of the existing vessels consisted of small-sized, thick-walled arteries, whereas others included larger lumens surrounded by hyalinised veins. These findings were compatible with those of AVM. The second operative material showed a well-differentiated, non-keratinised squamous epithelium papillary-type CP (pCP) around a partially oedematous fibrovascular stroma. Calcification was not observed (Figure 3).

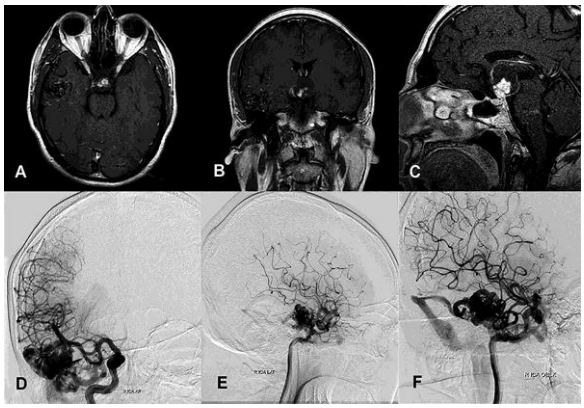

Figure 1. (a) Axial, (b) coronary contrast T1 and (c) sagittal T2 MRI sections showing cystic tumour in the midline, extending to the third ventricle, and a right temporal AVM. (d) A-P, (e) lateral (f) and oblique right internal carotid artery DSA images showing AVM from the right MCA, with no aneurysm in the nidus, and draining of the sigmoid sinus via the inferior petrosal sinus. AVM, arteriovenous malformation; DSA, digital subtract angiography; MCA, mild cerebral artery; MRI, magnetic resonance imaging.

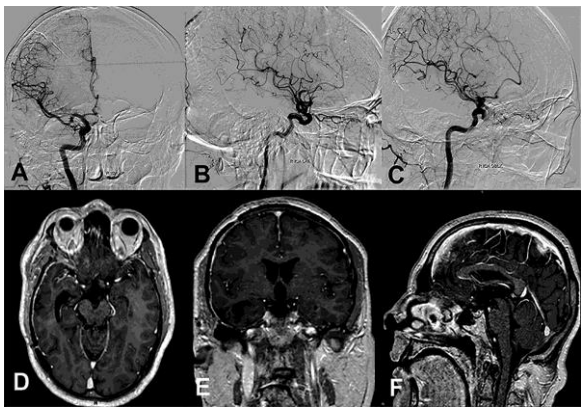

Figure 2. Post-operative (a) A-P, (b) lateral (c) and oblique right internal carotid DSA images. No AVM can be seen. Post-operative (a) axial, (b) coronary (c) and sagittal contrast T1 MR images showing total resection of both the tumour and AVM.

AVM, arteriovenous malformation; DSA, digital subtract angiography.

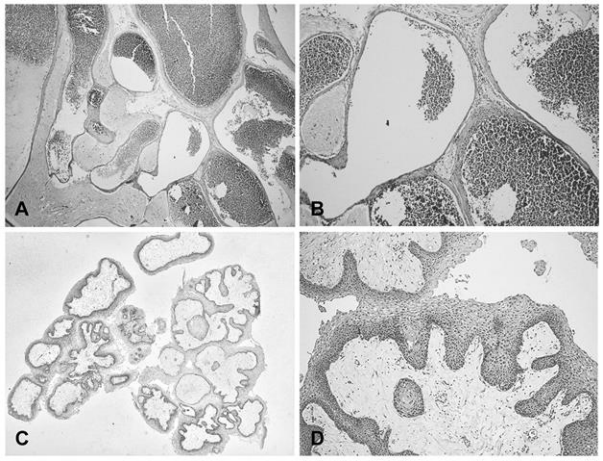

Figure 3. (a) Malformed, randomly distributed vessels, both in the veins and arteries, separated by variable amounts of intervening brain parenchyma (H&E, ×40). (b) H&E, ×100.and (c) papillary, cauliflower-like appearance, with surface epithelium covering the fibrovascular cores (H&E, ×20). (d) Non-keratinising, well-differentiated squamous epithelium surrounding the fibrovascular stromal cores (H&E, ×100). H&E, haematoxylin and eosin staining.

Discussion

Vascular malformations of the abnormal arteries and veins are usually congenital. They can appear at any age but are often diagnosed between 20 and 40 years of age. The clinical symptoms of AVM depend on its location. The most common symptoms include headache and seizures, but at least 15% of patients are asymptomatic. Every year, more than half of AVM cases are referenced with intracranial haemorrhages, constituting to approximately 2% of the total haemorrhagic stroke cases.[2,6,8] The methods used for the specific treatment of AVM include endovascular embolisation and surgery or gamma knife treatment.[4] In this study, we preferred to employ only surgical resection as the treatment approach.

It is assumed that CPs are histologically of two subtypes: adamantinomatous (aCP) and papillary (pCP). aCP is the more common type and is characterised by necrotic debris, cystic and/or solid components, fibrous tissues and calcification (more frequent in children). Infiltration is also common in the neighbouring brain tissue. pCP mostly occurs in adults, with 14%–50% of adult cases and only 2% of paediatric cases. pCP is characterised by the presence of a solid component or a combination of solid and cystic components. The cystic content is usually yellow and viscous, with rare calcification. pCP is generally well-defined, and infiltration into the neoplastic epithelium of the neighbouring brain tissue is minimal or even absent.[10, 13] The patient's pathological findings indicated pCP, and its dissection from the surrounding tissues was easy during the surgery.

The clinical symptoms of CPs are related to hypothalamic/pituitary deficiencies, visual impairment and increased intracranial pressure. The preferred treatment is complete resection which is carefully performed so as to preserve the optic and hypothalamic functions.[15] Post-operatively, in our patient, visual impairment decreased and hypothalamic dysfunction was not observed. In their meta-analysis, Dandurand et al.[7] compared the gross total resection of CPs with subtotal resection followed by adjuvant radiotherapy and found that the lowest recurrence rate was achieved with gross total resection. In our patient, gross total resection of CP was performed, and no recurrence was observed at the first 6-month follow-up.

Endoscopic trans-sphenoidal approach is the most preferred method for CP surgeries, which involves a pterional approach.[18] We prefer endoscopic trans-sphenoidal approach only if the patient is a candidate for CP surgery. However, an extended pterional approach was preferred in this case, considering that both the lesions could be resected using a single craniotomy flap.

Whether there exists any association between the formation of vascular malformations and development of intracranial meningiomas needs to be discussed. Rare cases of vascular malformations with primary brain tumours have been previously reported. In the literature, AVM has been reported mostly in association with oligodendrogliomas and astrocytomas.[12,17,19]Pleomorphic xanthoastrocytoma [16], meningioma[9] and ganglioneuroma[5] have also been reported with AVM. Mori et al.[14] were the first to report AVM and CP in patients, which were detected radiologically. However, these patients had only CP resections and were following AVM. Our patient is the first case in whom both the lesions were completely excised.

The occurrence of primary brain tumour in association with cerebral AVM has been reported.

However, the simultaneous occurrence of CP and AVM in a patient is extremely rare.

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Romanian Neurosurgery (2020) XXXIV (1): pp. 441-443
DOI: 10.33962/roneuro-2020-071
www.journals.lapub.co.uk/index.php/roneurosurgery

Very late recovery of vision after removal of giant pituitary tumour

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ABSTRACT
Visual impairment is the most common clinical presentation of the pituitary tumour. Visual recovery usually occurs within days to months after surgical removal of the tumor. We report a case of a giant pituitary tumour where preoperatively there was severe visual impairment. Postoperatively he recovered vision in one eye within three months and the other eye remained completely blind for 5 years, then it began to recover very slowly to a serviceable vision in the next six years.

Keywords
very late recovery of vision, giant pituitary tumour

A 22 years old young man presented with progressive loss of vision in both eye for last 2 years. He was near totally blind on right side and could see with to some extent by the left eye. Clinical examination showed, there were only PL and PR on right side where as visual acuity was 6/36 on left side. Confrontation test revealed blind right eye with temporal hemi field cut on left side, which was confirmed by perimetry. Fundoscopy revealed early atrophic changes in left eye and atrophic changes in right eye. Contrast MRI showed giant pituitary adenoma extending into suprasellar and subfrontal region. His hormonal study was normal. He under-went transnasal transsphenoidal removal of tumor first but it failed to remove the tumor satisfactorily. So second surgery was done within few days to remove the tumor through right lateral supraorbital frontal craniotomy. Postoperatively his vision on left side began to recover quickly. By the end of three month after operation, visual acuity and visual field on left side recovered completely whereas on the right side it remained as preoperative without any improvement. He was on regular follow up. Five year after operation his vision on right eye began to recover very slowly. It continued to recover for next four years and perimetry showed central and nasal field recovery on the left side [Figure1]. By the end of 09 years after operation, His visual acuity was 6/6 on left side where as on the right side it was 6/20. MRI showed small residual tumor without further growing for last 09 years [Figure2].

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Many factors influences in visual recovery after pituitary surgery such as types of tumor, size of tumor, consistency of tumor, extension and location of tumor, preoperative duration and severity of symptoms, preoperative state of fundus, type and approach of surgery, type of resection, optic canal and optic apparatus decompression etc. 1

Most of the visual recovery occurs with 3-6 month but it may continue several years.2 Although a previous study has reported that the recovery of visual field progresses over several years and that most of the recovery (>50%) occurs in the first 3 to 6 months after surgery, the time course of visual function recovery after transsphenoidal surgery for pituitary adenomas is unclear.3,4 But after scleral buckling procedure of rhegmatogenous retinal detachment involving the macula the vision may continue to recover up to 10 years.5

To best of our knowledge there is no report (of post pituitary surgery) where visual recovery started 5 years after surgery and that continued for another four years. In our case in early postoperative period less severely affected eye recovered with in three month but blind eye remained blind for five years after operation and there after it went for slow recovery. How and why such thing happened, it's a big question.

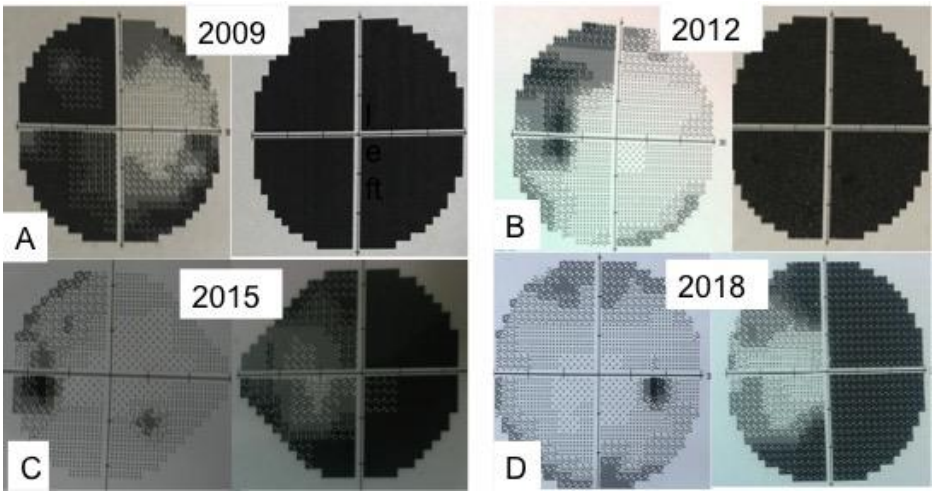

Figure 1. Visual fields analysis (VFA). A- before pituitary surgery, left nasal field was the only remaining field of vision. B- 04 years after operation, VFA showing left eye fields recovered whereas right eye was totally blind. C- 07 years operation, VFA showing recovery of left sided central and nasal field of vision. D-9 years after operation VFA showing recovery of left sided central and nasal field of vision.

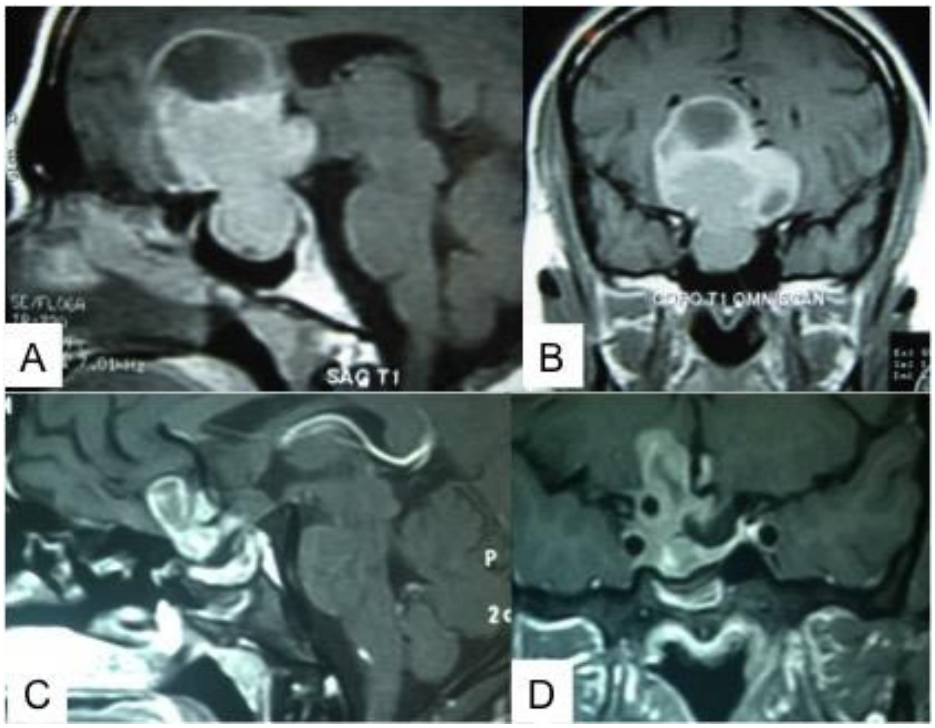

Figure 2. Contrast MRI of brain. A&B –Preoperative MRI; sagittal and coronal view showing giant pituitary tumor. C&D- Post operative MRI 09 years after operation; sagittal and coronal view showing small residual tumor.

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Romanian Neurosurgery (2020) XXXIV (1): pp. 444-451
DOI: 10.33962/roneuro-2020-072
www.journals.lapub.co.uk/index.php/roneurosurgery

Optic Nerve Sheath Fenestration (ONSF). Indications, techniques and results

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Keywords
optic nerve sheath fenestration,
ONSF,
IIH,
CVST,
TB,
visual impairment

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ABSTRACT

Objectives. Optic nerve sheath fenestration (ONSF) is commonly used in idiopathic intracranial hypertension (IIH). Here we will present our experiences of ONSF in 26 patients with special attention to indications, surgical techniques and results

Methods. The recorded data of patient management (with the result) who underwent ONSF were reviewed and studied retrospectively.

Results. The total number of patients who underwent ONSF was 26. The male-female ratio was 1:12.

Indications of ONSF were: 1. Idiopathic Intracranial Hypertension (IIH)-23 cases; 2. Cerebral Venous Sinus Thrombosis (CVST)-02 cases; 3. CNS Tuberculosis-01case.

All patient underwent bilateral ONSF with post-operative continues lumbar CSF drain for 04 days. After fenestration gush of CSF came out with force in all-first operated eyes whereas 13-second operated eyes showed very little CSF flow after fenestration. Vision improved in different grades in all cases at discharge except in three cases. Preoperatively, visual acuity was either PL&PR or hand movement in 40 eyes where 04 eyes were preoperatively total blind (no PL&PR). Visual acuity improved in 48 eyes (92.3% eyes) where the patient can do his/her daily life activities including self-care. Improvement in IIH is 100% (23 cases i.e-46 eyes) whereas 01 case out of 02 cases in CVST. Though vision was improved dramatically fundal appearances changes very slowly and very less frequently returned to normal appearance.

Conclusion. Due to the delicate and technically demanding nature of the surgery, safety is a major concern of the ONSF. Our experience showed ONSF is a technically safe operation with very good results where indicated.

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INTRODUCTION

Orbit contains eyeball, optic nerve (ON), extraocular muscles, fat, lacrimal gland, vessels and nerves (Figure 1). Actually, the optic nerve is an optical system projection white matter tract of the CNS. It is covered by pia mater, arachnoid mater, and dura mater. ON has intracranial, foraminal and orbital part. The subarachnoid space (SAS) of the CNS is continues with SAS of optic nerve. So increased intracranial pressure is transmitted to the optic nerve head and causes papilledema.[1]

Optic nerve sheath fenestration (ONSF) was first used by De Wecker in 1872 as an incision in the dura and arachnoid surrounding the optic nerve in order to relieve increased intracranial pressure. 2,3The technique is commonly applied in idiopathic intracranial hypertension (IIH) with rapid and/or progressive vision loss.4

But this technique can also be used in other conditions where CSF pressure in optic nerve SAS is increased locally, compartmentally or generally i.e. cerebral venous sinus thrombosis, CNS infection such as tuberculosis.

Here we will present our experiences of ONSF in 26 patients with special attention to indications, surgical techniques and results.

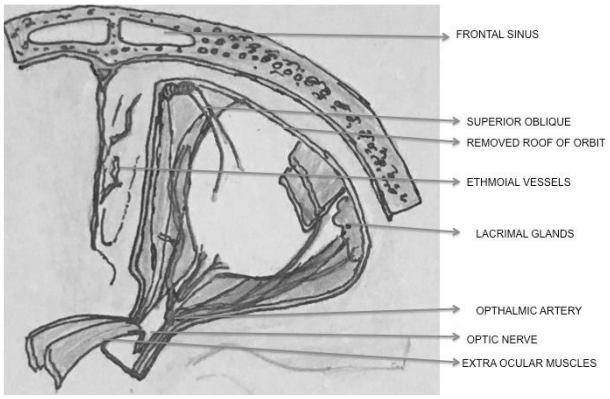

Figure 1. Schematic hand drawing showing the orbital structures including optic nerve after removal of orbital roof.

Methods

From January 2011 to December 2018, the patients who underwent ONSF in National Institute of Neurosciences & Hospital and some other private Hospital in Dhaka, Bangladesh were studied. The recorded data of patient management along with follow up were reviewed retrospectively. Clinical pictures, investigations, medical and other failed surgical management/s, ONSF procedures and follow up (clinical and investigations) were carefully studied and presented as results. Post-operative follow ups were done on 1st POD, 3rd POD, at discharge, after one month, at the end of six month and then 01 yearly. If patient failed to attend for follow up then follow up was achieved through voice or videophone call. Perimetry and fundal photograph were done six months after operation. Total follow up period was ranging from 06 months to 72 months (average 22.4 months) except one patient where patient recovered vision from PL&PR to hand movement at discharge on 7th POD and then she lost from follow up even over telephone.

Results

The total number of patients who underwent ONSF was 26. Male female ratio was 1:12.

Age range was 11 years to 38 years (average-22.5years). Build of patients was average and no one was obese or morbidly obese patient. Two patients were pregnant and were in 2nd trimester. In all cases bilateral ONSF was done i.e. total number of eyes were 52. No one was taking any drugs, steroid or contraceptive. Indications of ONSF were: 4. Idiopathic Intracranial Hypertension (IIH)-23 cases; 5. Cerebral Venous Sinus Thrombosis-02 cases; 6.CNS Tuberculosis-01case.

Clinical profile (Table 1)

All IIH patients were diagnosed as IIH in Neurology department according to IIH diagnostic criteria. All underwent MRI of brain and eyeball (Figure 2) including MRV where all ventricles were normal with normal MRV.

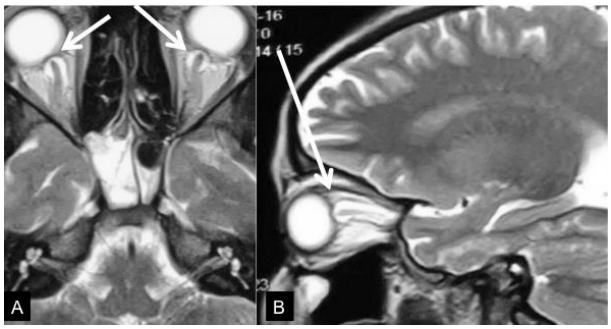

Figure 2. A&B- MRI of brain and orbit (T2W axial and sagittal image) showing elongated & curvy ON with increased CSF spaces and depression on globe by ON head (arrows marked) in IIH.

When there was severe visual deterioration even after having adequate medical management including serial lumbar puncture and/ continues lumbar CSF drainage for three days. Opening pressure was measured in all cases where CSF pressure was above the normal level ranging from 280 to 330 mm of CSF (Average 295mm). CSF study was normal in all cases except three (one with CNS TB, 01 cases with IIH where CSF protein was just above the normal another case with high CSF

without any known cause) Preoperative visual status of patient was shown in Table 1.

No patient underwent lumbo-peritneal (LP) or ventriculo-peritoneal (VP) shunt procedure. Preoperatively (Just before ONSF) no patient had nausea or vomiting and in 08 patients (30%) there was only mild to moderate (not severe) headache. Four patient had diplopia but clinical 6th nerve palsy was found in two cases.

Pre-operatively fundoscopy and fundal photograph was taken in all cases and perimetry was done in cases where vision is not badly impaired. Fundal photographic and perimetric findings are shown in Table 1.

Table 1. Particulars of patients with pre and postoperative Visual acuity and papilledema outcome.

Age (years), sex & Diagnosis	Pre operative-VA	Post operative last F/U-VA	Pre operative papilledema (Frisén scale)	Post operative papilledema (after six month)
22,F,IIH	Rt-PL&PR, left -HM	6/18 bilaterally	G-4	G-3
31,F IIH	PL&PR bilaterally	Hand movement-bilaterally (at discharge)	G-4	--
11, M, IIH	PL&PR-bilaterally	6/6 bilaterally	G-4	G-2
35, F, IIH	Finger count- bilaterally	6/6 bilaterally	G-4	G-2
19, F, IIH	PL&PR- bilaterally	6/12 bilaterally (only central field of vision recovered)	G-5	G-4
27,F, IIH	PL&PR- bilaterally	6/6-Rt 6/12-Lt	G-4	G-3
19,F,IIH	Rt-Pl&PR Lt-finger count	Rt-6/24 Lt-6/12	G-5	G-4
23,F, IIH	Hand movement-bilaterally	6/18 bilaterally	G-4	G-4
26,F, IIH	Hand movement-bilaterally	6/6 Rt 6/12 Lt	G-4	G-4
15,F,IIH	Left- total blind, Rt-PL&PR	Rt-6/36 Lt-6/24	G-5	G-4
22,F,IIH	PL&PR-Bilaterally	6/36- bilaterally	G-5	G-5
25, F ,IIH	Hand movement-bilaterally	6/6-bilaterally	G-4	G-3
16,F, IIH	PL&PR-bilaterally	6/6-Rt 6/12-Lt	G-4	G-4
26,F,IIH	Hand movement-bilaterally	6/18 -bilaterally	G-4	G-3
33,F, CVST)	PL&PR-bilaterally	Rt-6/18 Lt-6/24	G-4	G-4
27, F, IIH	Hand movement-bilaterally	6/6-bilaterally	G-4	G-3
26, F ,IIH	Rt-hand movement Lt-6/24	Rt-6/12 Lt-6/6	G-4	G-3
14, F, CVST	Blind(No PL&PR)-bilaterally	Blind (no improvement)	Early optic atrophy	Optic atrophy
30, F, IIH	Rt-6/60 Lt-6/36	Rt-6/12 Lt-6/6	G-4	G-4
38, F,IIH	Hand movement-bilaterally	Rt-6/12 Lt-6/6	G-4	G-4
26,M, IIH	Hand movement –Rt Lt- 6/24	Rt-6/12 Lt-6/6	G-4	G-3
20, F, IIH	PL&PR -bilaterally	Total recovery 6/6-bilaterally	G-4	G-2

32, F, IIH	Hand movement-bilaterally	6/18 -bilaterally	G-5	G-4
14, F, IIH	Rt-6/18 Lt-PL&PR	Rt-6/6 Lt-6/12	G-4	G-3
26, F, IIH	Hand movement-bilaterally	6/6-bilaterally	G-4	G-3
30, F, TB,	Rt eye vision loss (no PL/PR) Lt-PL&PR	Rt-No improvement Lt-PL&PR lost (blind)	Early optic atrophy	Optic atrophy

Operation (ONSF)

All patient underwent bilateral ONSF with post-operative continues lumbar CSF drain for 04 days.

Surgical techniques (Figure 3 and Figure 4)

Under general anaesthesia with endotracheal intubation patient was positioned in supine position. Eyelid and periorbital skin was painted carefully (with protection of cornea and conjunctiva) by diluted povidone Iodine solution. Then eyelids, corneal and conjunctiva were irrigated with normal solution. Keeping both eye exposed patient was draped. More severely affected eye was operated first. Universal eye speculum was placed to retract the eyelids. Then rest of the procedure was done with operating neurosurgical microscope under high magnification. A perilimbal conjunctival incision was given 12 to 3 o'clock position on right side and 9-12 o'clock position on left side. Scleral insertion of superior and medial rectus muscle were identified and a 2-0 silk was passed under the tendons of SR and MR muscles near its insertion to sclera for controlling the movement of eyeball. The eyeball was rotated downward and outward to make the optic nerve superficial and accessible. By dissecting under the bulbar conjunctiva in between SR and MR ON was reached. Conjunctiva and upper eyelid was retracted upward and medially. ON was identified by its position, color and continuity with the posterior eyeball. Vortex veins and cilliary nerves also help to identify the ON to some extent. If orbital fat prolapse in the field then the situation becomes difficult and more number of spatula's retraction may be needed. Undue pressure on globe was avoided. During globe rotation with MR &SR stay sutures corneal injury was carefully avoided. A linear, parallel to optic nerve incision was given to dural sheath and gush of CSF was noted and aspirated. Then retractor, MR & SR sutures were removed. Conjunctiva was re-sutured with 7-0 vicryl.

Same procedure was done on the opposite eye and antibiotic eye ointment was given.

The patient was positioned lateral to insert lumbar CSF drain and 200ml/day CSF was drained for the next four days. Tab. Acetazolamide continued for next six months.

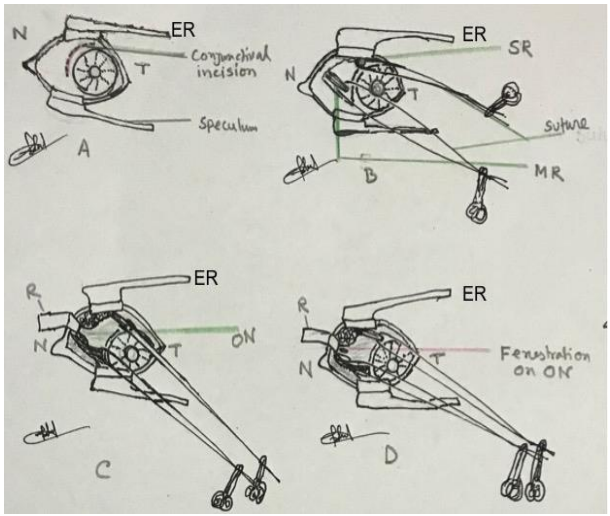

Figure 3. Sequential (A, B, C &D) schematic hand drawings of medial trans-conjunctival ONSF on left side. N-nasal side, T-temporal side, ER-eyelids retractor, R-retractor, Sutures-controlling sutures on superior and medial rectus tendon, SR-superior rectus, MR-medial rectus, ON-optic nerve.

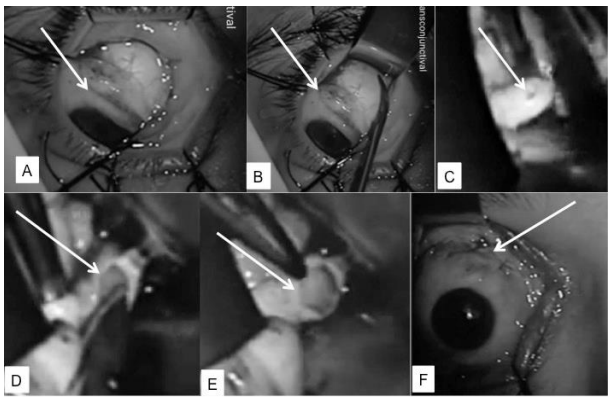

Figure 4. A, B, C, D, E & F - per operative sequential images showing stages of ONSF on right side. A&B- conjunctival incision and trans conjunctival dissection (arrow marked). C, D&E-Fenestration on sheath (arrow marked). F-Conjunctival closure after ONSF.

OBSERVATIONS

The total operation time was ranging from 45 minutes to 90 minutes (average 55 minutes). Peroperative corneal abrasion occurred in 02 cases. Single vortex vein injured in one case that needed to be coagulated. Peroperative minor venous bleeding occurred in two cases that needed to be coagulated. In six cases peroperative difficulties occurred due to orbital fat prolapse in the targeted zone. After fenestration gush of CSF came out with force in all-first operated eyes whereas 13-second operated eyes showed very little CSF flow after fenestration. There was no scleral injury or bulbar perforation or rupture.

Post-operative transient upper eyelid swelling occurred in three cases. Transient anisocoria with abnormal light reflexes occurred in 18 eyes that recovered within three days. There was no proptosis, retrobulbar hematoma or no ocular cranial nerve palsy occurred in any of the cases.

Headache cured in all cases after the operation. Vision improved in different grades in all cases at discharge except in three cases:

1. Case with CNS TB
2. One CVST case &
3. One case of IIH where at discharge there was no visual improvement but by the end of three month her vision recovered to 6/12 bilaterally.

Case with high CSF protein began to improve from 1st POD.

By the end of three month all sixth nerve palsy recovered.

In one case patient's vision recovered from hand movement to near normal but at the end of six month she developed left sided sixth nerve palsy without any return of IIH symptoms. This abducent nerve palsy recovered with steroid therapy for three weeks.

Pre-operative and postoperative (last follow up) visual status is shown in Table1. In all cases vision were severely affected preoperatively. Preoperatively, visual acuity was either PL&PR or hand movement in 40 eyes where 04 eyes were preoperatively total blind (no PL&PR). Visual acuity improved in 48 eyes (92.3% eyes) where the patient can do his/her daily life activities including self-care. In one case (two eyes), vision recovered only in central part of visual fields where peripheral part did not recovered even after one year (Figure 5). Improvement in IIH is 100% (23 cases i.e-46 eyes) whereas 01 case out of 02 case in CVST.

Pre and postoperative fundoscopic findings are also shown in the Table 1. Though vision improved dramatically fundal appearances changes very slowly and very less frequently returned to normal appearance (Figure 6 & 7). In our series, six months after ONSF papilledema improved in 23 cases (88.4%).

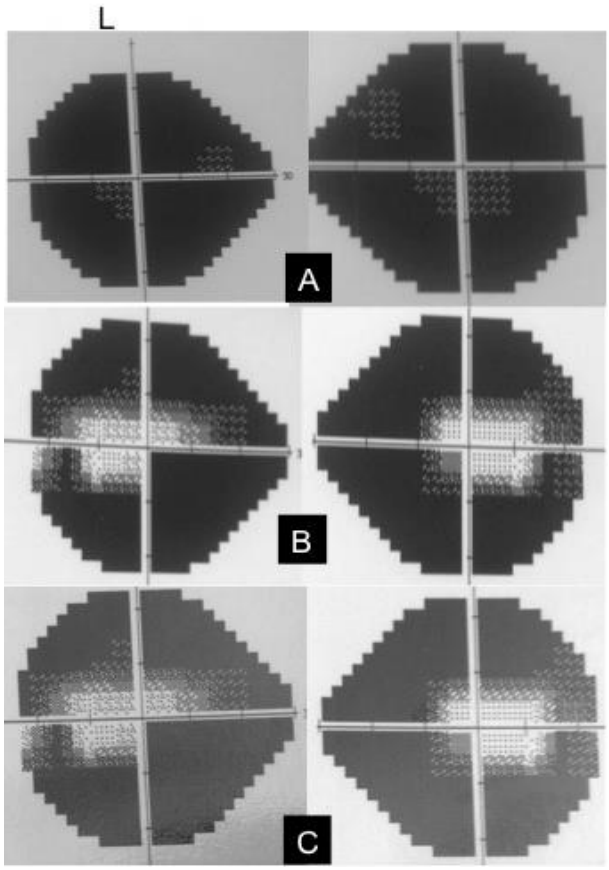

Figure 5. Visual field analysis (in IIH). A-preoperative. B &C-consecutive postoperative visual field analysis 06 and 12 after operation showed only central fields recovery (though patients' visual acuity was 6/6 bilaterally).

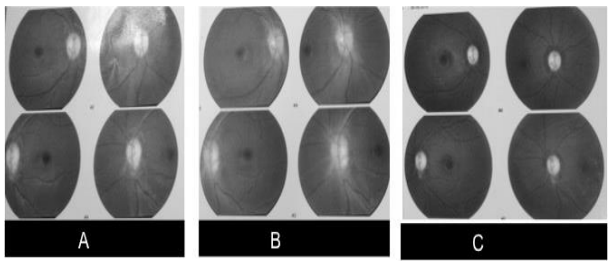

Figure 6. A-preoperative fundal photograph, B-post operative (03 months after ONSF) fundal photograph and C- post operative (06 months after ONSF) fundal photograph.

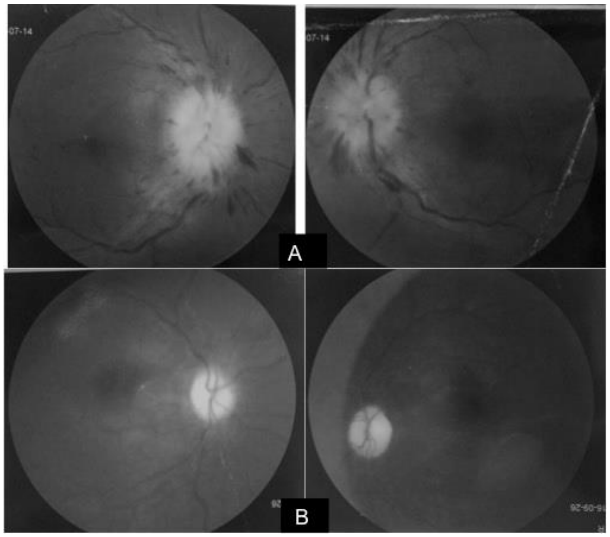

Figure 7. A-pre operative fundal photograph and B-post ONSF fundal photograph (after six month).

Discussion

Pathogenesis of raised intracranial pressure includes increased production of CSF, reduced drainage to circulation, intracranial space occupying lesions, traumatic brain injury, cerebral venous outflow obstruction, CVST and idiopathic intracranial hypertension (IIH).6,7,8 IIH is a special pathology, a diagnosis of exclusion, diagnosed by papilledema without any identifiable CNS pathology, and usually occurs in obese women of child bearing age.9 IIH is also referred to as pseudotumor cerebri. It is a disorder of unknown etiology that must meet the following criteria:

- Signs and symptoms of increased intracranial pressure (headache, nausea, transient visual obscurations lasting seconds, double vision, dizziness, emesis);
- Elevated cerebrospinal fluid (CSF) pressure (>200 mmHg in nonobese adults and >250 mmHg in obese adults);
- Normal neuroimaging studies;
- Normal neurologic examination (with the exception of papilledema and/or cranial nerve palsies);
- No other identifiable cause such as medications (including vitamin A, tetracycline, oral contraceptive pills, nalidixic acid, lithium, steroid use, or withdrawal)[5].

The effects of raised ICP are responsible for disruption of the axoplasmic flow, swelling of axons, leakage of water and proteins resulting papilledema.10,11Papilledema in its severe form (if left untreated), causes significant and irreversible loss of vision, with Visual Field (VF) defects and color vision.12

Papilledema treatment is primly focused on treating identifiable etiologies of raised ICP. IIH is treated by medical or surgical options as indicated. Medical treatment includes Acetazolamide, Steroids, Topiramate, Frousemide Whereas surgical options include Optic Nerve Sheath Fenestration (ONSF) or shunt procedures (lumboperitoneal shunts, ventriculo-peritoneal shunts, or ventriculo-atrial shunt).9,13,14

ONSF, first described by DeWecker in 1872 for treatment of papilledema.14 Hayreh described the blood supply of optic nerve, and thus also described the efficacy of ONSF in resolution of papilledema.15

The ONSF is principally used for the treatment of IIH. The ONSF is also used less frequently for the management of other pathological conditions that adversely affect the vision such as progressive non-arteritic ischemic optic neuropathy, and optic disc drusen,16 cryptococcal meningitis,17 CVST, and intracranial masses causing raised ICP that causes visual deterioration.

The three main surgical approaches for ONSF are superior eyelid, lateral orbital, and medial transconjunctival approach. In medial transconjunctival approach scleral insertion of MR is cut and after fenestration it is reattached.1 In our series we used the medial transconjunctival approach but we performed the nerve fenestration without cutting the MR tendon.1

Primarily visual acuity (VA) is the function of fovea, and does essentially provide the idea into preservation of central or paracentral visual field (VF).18 Incresed ICP essentially affects the macula, as a result of swelling of retinal nerve fibre layer as well as exudates and subretinal fluid. So resolution of papilledema is expected to improve vision after ONSF.

Analysis the histological features of IIH have shown that vision loss from outer retinal layer changes in the macula is more reversible than vision loss from optic neuropathy and inner retinal layer change.19 Improvement in VA does not depend upon pre-operative papilledema stage and thus explains the macular function is independence of optic disc swelling alone. It also means change in VA is independent from pre-operative VA, explaining

that poor vision before surgery doesn't necessarily contra-indicates the performance of ONSF.

The range of improvement in VA after ONSF is a debatable and variable subject. Studies have shown wide range in improvement in VA, from as low as 14% to as high as 100%.20,21In our series improvement in VA in 24 cases (92,3%), non-improvement in 2 cases (7.7%). In IIH visual improvement is 100% (23cases out of 23 cases of IIH. In one of the largest studies conducted on 578 eyes of 331 patients, improvement or stability was seen in 94.4% and worsening in 5.6% of eyes.16

ONSF principally means the improvement of vision. But it has efficacy in improvement of other symptoms also. Published data shows wide range in headache improvement, with as low as 13%, and as high as 90% patients with headache.22,23 our series also showed the same. So there is indirect evidence that ONSF has role is in controlling chief symptom of IIH or raised ICP.

Post ONSF improvement of papilledema ranges of 71 to 100%. 24,25It is logical to mention that improvement in papilledema stage does not necessarily mean complete resolution of papilledema. After ONSF complete resolution of papilledema is rare. In our series, six months after ONSF papilledema improved in 23 cases (88.4%). In the largest meta-analysis done on result of ONSF with follow up of 20 months, improvement in headache, visual acuity and papilledema was seen in 26%, 42% and 92% respectively.9

The complication rate of ONSF is ranging from 5 to 45% in the literature.26 Complications of ONSF including visual deterioration (<1%),22 permanent atonic pupil, retrobulbar hemorrhage and sixth nerve palsy have been reported in the literature.27 Other less frequently seen complications are transient blindness, choroidal infarction, diplopia and orbital infections.16,28In our series we face no major permanent complication.

Conclusion

Due to the delicate and technically demanding nature of the surgery, safety is a major concern of the ONSF. Our experience showed, ONSF is a technically safe operation with very good results where indicated.

Declaration

Ethics approval and consent to participate – Not applicable (NA)
Consent for publication - Taken from the patients/patient's party.
Availability of data and materials - NA
Competing interests - None
Funding - None

Abbreviations

CVST-cerebral venous sinus thrombosis
CNS-central nervous system
CSF-cerebro spinal fluid
ICP-intracranial pressure
IIH-idiopathic intracranial hypertension
LP-lumbo-peritoneal
MR-medial rectus
MRV-magnetic resonance venography
ON-optic nerve
ONSF-optic nerve sheath fenestration
POD-post operative day
PL-perception of light
PR-projection of rays
SR-superior rectus
SAS-subarachnoid space
TB-tuberculosis
VA-visual acquity
VF-visual fields
VP-ventriculo-peritoneal

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Romanian Neurosurgery (2020) XXXIV (1): pp. 452-454
DOI: 10.33962/roneuro-2020-073
www.journals.lapub.co.uk/index.php/roneurosurgery

Supratentorial PNET in a geriatric patient. A rare differential diagnosis leading to diagnostic dilemma

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Keywords
PNET, geriatric, diagnostic

ABSTRACT
Supratentorial PNETs are most commonly seen in children and rarely seen in adults. PNET show a proliferation of undifferentiated or poorly differentiated neuroepithelial cells and, thus, are histologically similar to medulloblastomas. They account for approximately 2.5% of brain tumours in children and only 0.4% in adults. Prognosis is poor in the pediatric age group while it shows favourable prognosis in adults. In literature, less than 100 cases of adult PNET have been reported till date with mean age of 35years. PNET in the geriatric age group is rarely been reported.

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INTRODUCTION

The term of primitive neuroectodermal tumor (PNET) was coined by Hart and Earle in 1973 for small round cell tumors with high malignant potential and affecting both the central and peripheral nervous systems. PNETs show a proliferation of undifferentiated or poorly differentiated neuroepithelial cells and, thus, are histologically similar to medulloblastomas. World Health Organization (WHO) described supratentorial PNETs as cerebral or suprasellar embryonal grade IV tumor with capacity to display differentiation along neuronal, astrocytic, ependymal, muscular or melanocytic lines. These tumors are most commonly seen in children and rarely seen in adults. They account for approximately 2.5% of brain tumors in children and only 0.4% in adults. Prognosis is poor in pediatric age group while in shows favorable prognosis in adults. [(1)] In literature less than 100 cases of adult PNET have been reported till date with mean age of 35years. PNET in geriatric age group is rarely been reported. [(2)]

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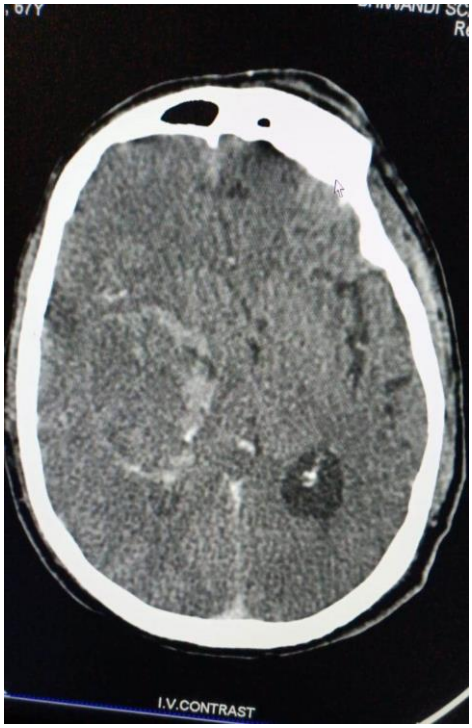
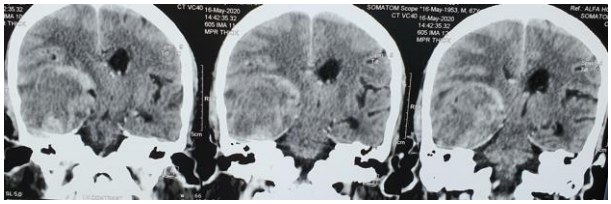
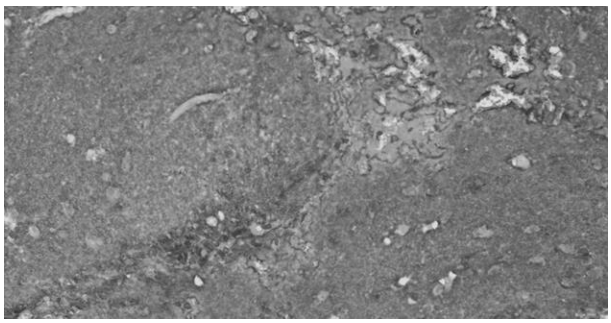
Case

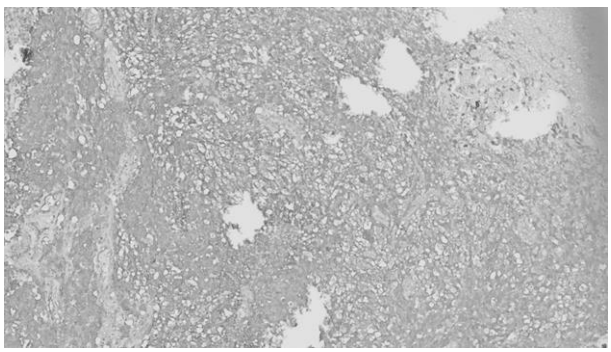
We had a 67-year-old male patient presented to us with sudden onset severe headache and vomiting for 1 day, patient was drowsy on examination. His CT scan of brain with contrast was suggestive of a large ,5*4*3 cm, well circumscribed lesion in right temporal lobe with perilesional edema. Lesion was minimally enhancing on contrast with isodense centre. As patient was drowsy, he underwent emergency craniotomy with near total excision of lesion. Small part of lesion which was invading the basal ganglia was left behind as seen on post-operative CT scan. Post operatively patient improved in consciousness and was discharged on 7th post-operative day without any deficits.

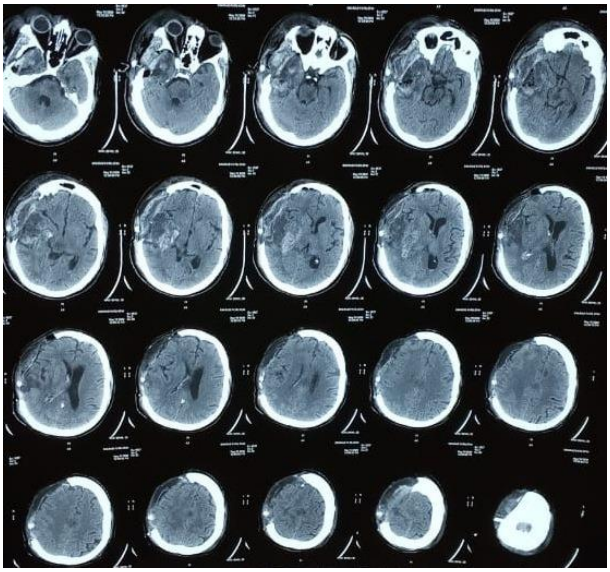
Histopathology was suggestive of highly cellular lesion with nests, cords and sheets of tumor cells with high N/C ratio. Hyperchromatic nuclei, inconspicuous nucleoli and brisk mitoses was seen. On IHC, tumor cells were positive for CD99, CD 56 and vimentin, GFAP negative, Ki-67 ratio was high (60-70%), suggestive of PNET (Intracranial central). Patient was advised post-operative radiotherapy and chemotherapy.

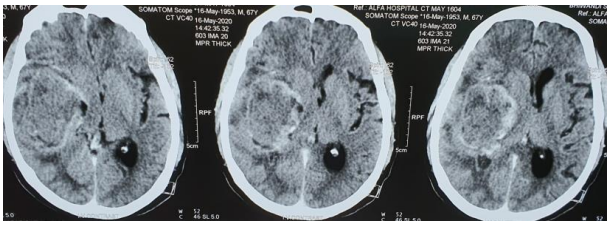


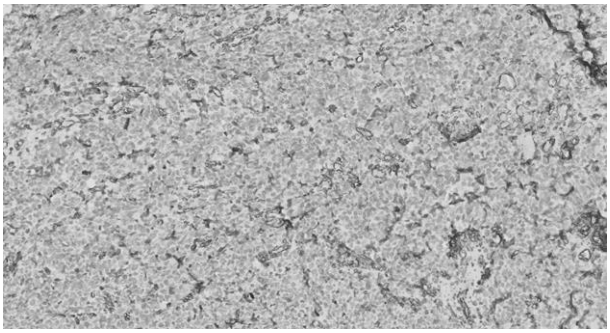


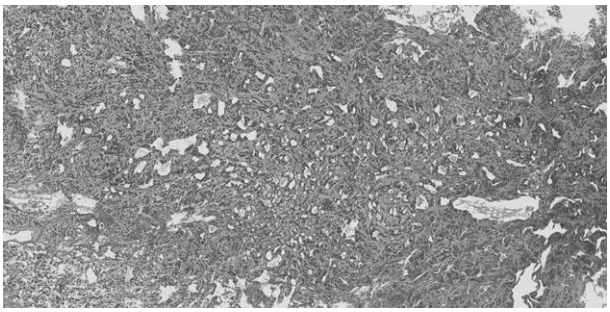












Discussion

Primitive neuroectodermal tumors (PNETs) include a group of tumors, thought to originate from undifferentiated neuroepithelial cells, that are commonly seen in pediatric patients and rare in adults. Hart and Earle described the term of primitive neuroectodermal tumor – PNET – in 1973 originally for cerebral high-grade undifferentiated neuroepithelial tumor of childhood, rarely demonstrating focal differentiation along glial and neuronal lines. This term was soon generalised for undifferentiated embryonal tumors of all CNS sites and all ages by Rorke in 1983. PNETs may occur in almost any location within or outside the central nervous system. PNETs seen outside the CNS are termed as peripheral PNETs (pPNET). CNS PNET and pPNET are two different entities with different immunohistochemical profiles and genetics. Clinically both are aggressive tumors, but show different local manifestation and metastatic spread. CNS PNETs can be further divided into 2 types: infratentorial tumors (medulloblastoma) and supratentorial tumors (sPNETs). All these tumor types are rare in adults. [1,2]

Intracranial PNETs are uniformly distributed in the frontal lobe, temporal lobe, and parietal lobe and they are more than 6cm in size. Intracranial PNETs are further divided into central and peripheral according to their location with peripheral variety carrying a better prognosis. Accordingly, it is essential to differentiate between the two types. Chromosomal translocation of chromosome (11;22) is unique to central and not seen in peripheral PNETs. Immuno-histochemical assay for CD99 and fluorescence in situ hybridization (FISH) assay for the (11;22) translocation are specific for central PNET.[2,3]

The exact protocol for treatment of PNET is not clearly defined yet. So as per patient's clinical status, complete tumor excision, chemotherapy, and radiotherapy are performed as standard procedures like any other intracranial tumors. Prognosis of patients with PNET differs according to age. Pediatric age group carries poor prognosis with older age appears to be have favorable prognosis. Unfortunately, the 5-year survival in case of CNS PNET remains to be less than 50% in all age groups. [1,2]

Conclusion

The diagnostics and treatment protocol of primitive neuroectodermal tumors does not differ from other types of the central nervous system tumors. Complete tumor excision, chemotherapy, and radiotherapy are performed as a treatment standard with better survival prognosis in old age. So even though very rare in older age group PNET should be kept as one of differential diagnosis as it carries better prognosis in this age group. A CD99 and FISH assay for the (11;22) translocation (unique to central and not peripheral PNETs) should be conducted in order to distinguish between intracranial peripheral and intracranial central PNETs, as peripheral PNET carries better prognosis.

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Romanian Neurosurgery (2020) XXXIV (1): pp. 455-458
DOI: 10.33962/roneuro-2020-074
www.journals.lapub.co.uk/index.php/roneurosurgery

Enlarged anterior communicating artery masquerading as intracranial aneurysm. Case report

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Keywords
A1 hypoplasia, ACoA complex, anatomic variations, enlarged ACoA

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Abstract

Background. The anterior communicating artery (ACoA) complex consists of the ACoA, the pre-and post-communicating segments of the anterior cerebral artery, and the recurrent artery of Heubner. It is the most common site for anatomical variations in the circle of Willis. Such variations can mimic intracranial aneurysms.

Case description. A 30-year-old female presented with recurrent episodes of extreme headache and bilateral tinnitus. A brain computed tomography (CT) scan showed no significant lesions, while her CT-angiography (CTA) showed an enlarged vascular lesion at the ACoA, raising the suspicion for an ACoA aneurysm. A repeated CTA revealed a rare anatomical variation with a pattern of cross dominance in the ACoA complex; the left A1 and right A2 were dominant-enlarged, resulting in an enlargement of the ACoA. The presence of an ACoA aneurysm was hence excluded and the patient was managed conservatively. At 6-month follow-up, CTA showed no new findings.

Conclusion. ACoA enlargement can result from unequal hemodynamics around the ACoA complex, which may be mistaken for an aneurysm. A thorough study of the imaging data is of pivotal importance and may change the management strategy.

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Introduction

The anterior communicating artery (ACoA) connects the two anterior cerebral arteries, dividing them into pre-communicating (A1) and post-communicating (A2) segments. ACoA, A1, A2 segments, and the recurrent artery of Heubner are collectively known as the AcoA complex [1]. Anatomical variations in this complex are common and are detected

in 58-85% of all patients with ACoA aneurysms [2]. A range of different arterial variations was indexed, including fenestration, duplication, trifurcation, hypoplasia, and aplasia [3]. Several efforts have been made to classify the ACoA complex variations as they may influence the surgical approach of any co-existing vascular disease [1].

Also, these anatomical variations can mimic intracranial aneurysms by the superimposition of a duplicated or a fenestrated vessel [2]. Furthermore, the net-result of the hemodynamic changes in these variations plays a considerable role in the development of several vascular pathologies, such as aneurysms and ischemic strokes [3]. Moreover, ACoA complex is the most common site for intracranial aneurysms, constituting up to 40% of all aneurysms, which can further ambiguate identifying these variations [4]. In this report, we present a case report of an enlarged ACoA masquerading initially as an ACoA aneurysm. To our knowledge, this is the first such case report of its kind in the literature.

Case scenario

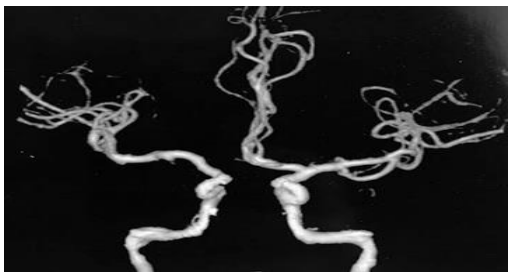
An otherwise healthy, 30-year-old, female typist was referred by her general physician with a suspension of an unruptured ACoA aneurysm. She had a history of recurrent attacks of severe non-localized headaches associated with episodes of bilateral tinnitus, more on the left side, for two weeks. Each attack lasts for more than one hour with no exacerbating factors. The headache was rated as 8/10 by the patient, and it impacted her daily activities. The patient had an intact neurological examination. Her brain computed tomography (CT) scan revealed no significant lesions apart from a suspicious anterior interhemispheric lesion. The CT-angiography showed a pouched-out vascular lesion at the ACoA, raising suspicion for an ACoA aneurysm (Fig. 1A). As we had no endovascular facility at our institute, we opted to perform another CTA, aiming to obtain better-qualified images and more clear views. The new CTA showed a dominant left A1 and a hypoplastic right A1 (0.4 mm) segments of the anterior cerebral artery, which is a common variation especially encountered with ACoA aneurysms. Besides, a noticeable enlargement of the ACoA at its junction with the right anterior cerebral artery was found, most probably resulting from the inverted hemodynamics between the two sides of ACoA. The left A2 was normal, while the right A2 was enlarged. No intracranial aneurysms were detected (Fig. 1B-D). The patient was informed that she had a normal variation in the circulation rather than an ACoA aneurysm. Simple analgesics were prescribed, which helped the headache. The patient continued to have tinnitus. Therefore, a brain magnetic resonance imaging (MRI) was done to exclude the possibility of an arteriovenous fistula, which then came back negative. The patient was referred to an otolaryngologist, who diagnosed her with mild otitis media. At her 6-month follow-up visit, the patient's headache and tinnitus had resolved, and she had resumed her daily activities. The follow-up CTA and MRI studies revealed no new findings.

Figure 1. Imaging of an ACoA variation mimicking an aneurysm. (A): A cerebral CT angiography, 3D reconstructed image showing a suspected ACoA aneurysm. (B, C) different views of a second CTA, confirming the absence of an intracranial aneurysm. Instead, an enlarged ACoA was noted accompanied by an enlargement of both the left A1 and right A2. (D) An artistic depiction of the ACoA arterial complex demonstrating the inverted hemodynamics. The vascular configuration showing an enlarged ACoA (*), LA1, and RA2 with a hypoplastic RA1 and a normal-sized LA2 with no evidence of aneurysm formation.

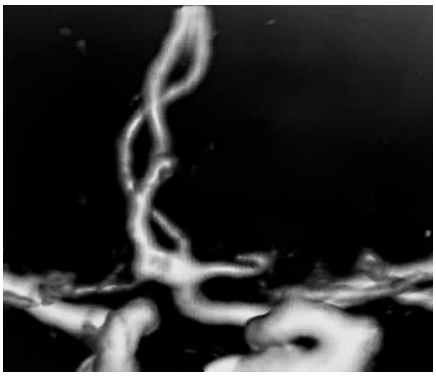
ACoA; Anterior communicating artery, ICA; Internal carotid artery, MCA: middle cerebral artery, R; Right, L; Left. M1: sphenoidal or horizontal segment of the MCA. A1: Pre-communicating segment of the anterior cerebral artery. A2: Post-communicating segment of the anterior cerebral artery.

A.

B.



C.



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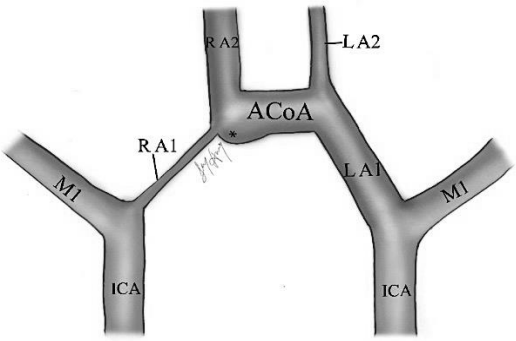

Discussion

The anterior cerebral artery plays a vital role in the blood supply to the anterior collateral circulation as it gives off penetrating striatal arteries that supply the anterior hypothalamus, septum pellucidum, and parts of the corpus striatum [5]. Vascular anomalies of the ACoA complex are not uncommon and are mostly encountered in the A1 segment [6]. A unilateral hypoplastic A1 segment is one of the most common anomalies in the circle of Willis with an incidence of 3% [3,7]. Marinković et al. described two types of A1 hypoplasia based on carotid angiogram namely, mild and extreme hypoplasia [8]. Mild hypoplasia is diagnosed when the diameter of the A1 segment is more than 1 mm, whereas extreme hypoplasia is defined by an A2 segment diameter of less than 1 mm [8]. Hypoplasia of one A1 segment is compensated by the dominance of the contralateral A1 segment [6]. These variations are often asymptomatic or found incidentally, chronic headache is, however, a possible presentation in patients with arterial hypoplasia [9]. The A1 segment is a rare site for ischemic strokes, representing only 0.6-3% of all stroke etiologies [10]. The risk for ischemic stroke is further aggravated by the presence of such hypoplasia [5,10]. Furthermore, the presence of A1 hypoplasia increases the risk of aneurysm formation; a phenomenon that can be explained, in part, by alteration in ACoA complex hemodynamics [1]. In this case, the extreme hypoplastic right A1 segment was compensated by the dominance of the left A1 segment, which supplied the right A2 segment through the ACoA. These variations manifested radiologically as an enlarged ACoA with a pouched-out appearance leading to a suspicion of an ACoA aneurysm. Moreover, the presence of such inverted hemodynamics around the ACoA complex may render the distal circulation more prone to ischemic stroke or aneurysm formation [7]. Thereafter, follow-up by annual imaging is important in determining patient's prognosis and could confirm or exclude the stability of such anomaly [10]. Knowing such variations is also essential in planning surgical and endovascular approaches, as these variations can require a different management strategy [3]. The purpose behind presenting this case is to highlight their propensity to pose a diagnostic dilemma, especially when catheter angiography is not feasible. Moreover, our case demonstrates how cautious the surgeon should be in reviewing the patient's radiology while planning the appropriate management.

Conclusion

Anterior communicating artery enlargement can result from unequal hemodynamics around the anterior communicating complex, which may be mistaken for an aneurysm. A thorough study of the imaging data is of pivotal importance and may change the management strategy.

Abbreviations

ACoA - Anterior communicating artery
CT - Computed tomography
CTA - Computed tomography angiography
MRI - Magnetic resonance imaging.

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Romanian Neurosurgery (2020) XXXIV (1): pp. 459-462
DOI: 10.33962/roneuro-2020-075
www.journals.lapub.co.uk/index.php/roneurosurgery

Cerebrospinal fluid dynamics with its surgical implications

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Keywords
cerebrospinal fluid, hydrocephalus, ventricles

ABSTRACT
Cerebrospinal fluid (CSF) is largely (70-80%) produced by the choroids plexus of the ventricles and is considered as the plasma ultrafiltrate. While CSF formation, circulation, and composition appear to be physiological and physical, its absorption appears to be mainly physical. The formation, composition, circulation, absorption, and changes in pathological conditions of CSF are discussed briefly in this review article. The CSF pressure dynamics studies provide information about the tightness, elastance, or outflow resistance of the CSF in the CNS. We believe that the present study shall help to provide essential details of CSF physiology which are important to many disciplines including radiology, neurology, and neurosurgery.

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INTRODUCTION

Thorough knowledge of CSF dynamics is essential for understanding the intracranial -intraspinal changes due to pathologic conditions. Perhaps, the first time described was by Dandy in 1919 (1). However, years later three components of CSF dynamics was stablished by Czosnyka et al, wich are CSF formation, circulation, and composition. This, appears to be physiological and physical, and its absorption may be mainly physical. Since most of the surgical treatments involve manipulating the physical principles, it is important to understand the studies, which are emerged on the dynamics of CSF (2). Studies in patients with hydrocephalus and traumatic brain injury has contributed to understand the strong association between CSF dynamics, physical

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and surgical components (3). The main objective of this review is to review CSF dynamics and the association with surgical components.

Physiology of CSF dynamics

Physiological principles include vascular, neuro-humoral, and sub-cellular regulation, while physical principles include all the fluid dynamics. Some of the well-known physiological principles include Cushing's reflex, which affects the cerebrovascular system. Pressure on ventricular walls affects the secretion of CSF at the aquaporin level of the choroid plexus. Sco-spondin, the peptide secreted by the SCO (subcommissural organ) will regulate the flow of CSF across the aqueduct of Sylvius. However, Sco-spondin is rudimentary in humans. There are approximately 150 ml of CSF, with a rate of 0.3-0.6 ml/min of production in an adult, distributed between 120 and 30 ml in cranial and spinal subarachnoid spaces, with daily volume produced between 500-600 ml (4). The extracellular space is about 15% of the brain volume and about 1500 ml of adult intracranial space consists of 1100 ml of intracellular space, 200 ml extracellular space, 140 ml of cerebrospinal fluid, and about 60 ml of blood at a given time (5). The choroid plexus in the ventricles is the main production site of CSF formation. However, there is a strong association between resistance of CSF and formation, which in many studies has shown to be the first parameter to consider in patients with intracranial hypertension (6–8). Perhaps, Large amounts of CSF are drained primarily into the blood through the arachnoid villi by penetrating the sinus, mainly into the superior sagittal sinus. Understanding this process, may contribute to be the key to comprehend surgical components. As surgery component, according to Monro-Kellie theory; a disbalance between CSF reabsorption and resistance may be the key to develop every illness related to CSF dynamics (9,10).

Physiological responses

Hypothermia decreases the CSF volume by 11%, with each one-degree reduction in the temperature and hyperthermia increases the CSF volume. In general, the drugs that increase the cerebral blood flow consequently increase the CSF production; on the other hand, the aging process and some infections cause reduce in the CSF formation (11–14). However, the total CSF volume increases with age in both sexes, mainly due to the contribution by the cortical sulcal volume. These pressure gradients which are being created by continuous CSF secretion and facilitated by the arterial pressure pulsations have an important role for CSF circulation and absorption through the venous system (15). The respiratory variations and vascular pulsations emanating from the choroid plexus and cerebral arteries cause the ventricles to pulsate providing the additional movement to CSF. Although trans-mural migration of CSF across the pial arteries has been demonstrated in the animals, these do not contribute to the bulk of CSF absorption (16).

Functions of CSF

This blood- CSF barrier maintains a chemically precise environment, which is necessary for the neurotransmitter and removes the metabolic products, unwanted drugs and pathological substance that may result in CNS injury (17,18). Changes in CSF Ca, Mg, K produce changes in the sympathetic and autonomic response, ventilation, muscle tone, and emotional state. In continuity with the brain interstitial fluid, CSF provides a stable supply of substrates, primarily glucose, even though the plasma concentration of the substrate is continuously changing. Transport of endorphins, hormones of the hypothalamus and pineal gland is facilitated by the CSF. Specialized ventricular cells and brain parenchymal neurons secrete neuroendocrine factors (19,20).

Laboratory Investigation of CSF

Important information on CSF can be derived from the following parameters, opening pressure, gross appearance, total and differential cell count, bacterial culture and sensitivity, protein and glucose, analysis of immunoglobulins (to detect chronic CNS inflammatory conditions) and cytology (to detect malignant cells). Increased neutrophils in the CSF indicate bacterial meningitis (21). Other causes of increased neutrophil count include a cerebral abscess, seizures, and CNS hemorrhage. Increased lymphocytes in the CSF indicate viral meningitis. Lymphocyte counts are also elevated in meningitis, tuberculosis, syphilis, fungal and parasitic infections. Increased plasma cells is a feature of TB meningitis and chronic inflammatory disorders like multiple sclerosis. The presence of leukemic cells in the CSF indicates meningeal infiltration by leukemic cells (22).

Leukemic cells typically appear in the CSF after several remissions have been achieved by chemotherapy. Tumor cells can be detected by cytological studies and the sources of tumor cells can be from primary CNS tumors like medulloblastoma, or metastatic CNS tumors from lung, breast, GI tract, and melanoma. It may be necessary to determine if a nasal or ear fluid is found as a content in the CSF. CSF fluid contains a modified transferrin protein called β2-transferrin (tau protein), which is not present in plasma or other fluids. The presence of β2-transferrin in a fluid strongly suggests that the fluid is CSF, which may be used, by instance, to identify CSF leak after sinus surgery (22,23).

Studies of CSF dynamics

In clinical practice, external lumbar drainage (Tap-test) is widely used to evaluate normal pressure hydrocephalus (NPH). Infusion tests, bolus injections, and isotope dilution methods are employed most often in experimental studies. ICP monitoring in acute conditions such as head injury and chronic conditions such as NPH is widely used. In the study on CSF dynamics by Ramesh et al. a simple, safe, and cost-effective method of infusion method to predict the VPS effectiveness was devised (24). Not only in NPH and PTH but also postmeningitic hydrocephalic patients seems to correlate and benefit from the pre-operative saline infusion studies. CT cisternography consists of the injection of radioactive agents into the subarachnoid space and serial CT scans are taken at different times depending on the clinical problem, such as CSF fistulae, NPH, etc. It is also used to evaluate the shunt function. It can also help in the evaluation of patients receiving intrathecal chemotherapy, evaluation of brain fluid-filled structures (arachnoid cysts, etc.), evaluation of CSF pumps for continuous delivery of medication. CT and MRI are useful to estimate intracranial compliance (25).

Conclusion

This review briefs about the formation, composition, circulation and absorption of the CSF. The changes in CSF due to pathological conditions are highlighted. It is believed that the present study will offer the details about the physiology and dynamics of CSF, which is essential to the broad specialties including radiology, neurology, and neurosurgery.

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Romanian Neurosurgery (2020) XXXIV (1): pp. 463-464
DOI: 10.33962/roneuro-2020-076
www.journals.lapub.co.uk/index.php/roneurosurgery

Global warming, neurosurgery and neurocritical care

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Keywords
global warming, traumatic brain injury, cerebral stroke, subarachnoid haemorrhage, prevention

ABSTRACT
The changing temperatures are making an impact on health-related mortality outcomes with many studies on the role of temperature and mortality risks in cardiovascular and respiratory illnesses. Global warming a real phenomenon, progressing rapidly and producing changes in the ecosystem and have economic, social and public health implications.

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The changing temperatures are making an impact on health-related mortality outcomes with many studies the role of temperature and mortality risks in cardiovascular and respiratory illnesses[1]. Global warming a real phenomenon, progressing rapidly and producing changes in the ecosystem and have economic, social and public health implications. Climate change is causing warmer and more variable temperatures as well as a physical flux in natural populations, will affect the ecology and evolution of infectious disease epidemics. The greenhouse effect has increased the temperature by more than 0.5 ° C and it is estimated that there will be another increase of 0.5 ° C in the next coming decades. [2, 3] Despite the alarming rise we have turned a blind eye to these problems and now we may face the consequences of this phenomenon in every field. Ali et al [4] analyzed 111 patients to assess the impact of the lunar cycle and season on the incidence of aneurysmal subarachnoid hemorrhage and noted incidence peak for aneurysm rupture was observed during the phase of new moon, which was statistically significant, however no seasonal variation in the

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incidence of overall total subarachnoid hemorrhage caused by various etiology was observed. Authors concluded lunar cycle affect the incidence of intracranial aneurysm rupture, with the new moon being associated with an increased risk of aneurysmal subarachnoid hemorrhage. [4] Chyatte et al. [5] analyzed 1487 patients with a primary diagnosis of aneurysmal subarachnoid hemorrhage. Men showed a single large peak in late fall, and late spring in women Changing climatic conditions precede aneurysm rupture in men but not in women, which suggests that weather is causally related to aneurysm rupture in men and that factors that lead to aneurysm rupture in women may be different from those in men. These data do not explain why weather fronts or gradients are associated with aneurysm rupture in men. [5]

Li et al [6] estimated temperature-related mortality projection for acute ischemic heart disease and ischemic and hemorrhagic stroke with concomitant climate warming. The median number of projected annual temperature-related deaths for hemorrhagic stroke had virtually no change compared with the 1980s, and for acute ischemic heart disease Authors projected temperature-related mortality associated with ischemic stroke can increase dramatically as an effect of climate warming. However, projected temperature-related mortality pertaining to acute ischemic heart disease and hemorrhagic stroke should remain relatively stable over time. [6]

It is anticipated that the incidence of traumatic brain injury is likely to rise as level of industrialization will continue to increase. Increases number of natural disasters like cyclones and floods will lead to greater load of trauma. Many centers now have fully functional round-the-clock operation theaters and cooling them needs large amounts of power. Again, most of the Computed tomography machines and Magnetic resonance imaging machines need super-cooled magnets which against needs electricity. Thus, global warming will lead to greater health care expenditures which will trans late to increased health care costs and thus non-affordability will rear its ugly head again. Changes made before the problem becomes unreversible is the only solution. [7] The global warming and ecological changes may produce myriad of health hazard including cerebral stroke, aneurysmal subarachnoid hemorrhage. The real inconvenience shall be in the mangement of diseases that will arise dur to this and will push us back in terms of our advancement adn health care delivery. It is best to control global climate changes and warming with the help of international co-operation further health hazard.

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