

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

ROMANIAN  
NEUROSURGERY

Vol. XXXVIII | No. 3

September 2024



THE OFFICIAL JOURNAL OF  
"Romanian Society of Neurosurgery"

- Est. 1982 -

LONDON ACADEMIC PUBLISHING

ROMANIAN NEUROSURGERY

---

# EDITORIAL AND ADVISORY BOARD

## EDITOR-IN-CHIEF

Assoc. Prof. Vicentiu Saceleanu, MD, PhD  
vicentiu.saceleanu@ulbsibiu.ro

## ASSISTANT EDITORS

Andrei Marinescu  
marinescu.andrei.alex@gmail.com

Cosmin Cindea  
cindea.cos@gmail.com

Titus Fagarasi  
fagarasi.titus@gmail.com

---

## ADVISORY BOARD – ROMANIA

Prof. Horia Ples  
Prof. Stefan I. Florian  
Prof. Mircea Gorgan  
Assoc. Prof. Lucian Eva  
Prof. G.B.I. Iacob  
Prof. I. Poeta  
Prof. Aurel Mohan  
Assoc. Prof. Adrian Balasa  
MD Marius Dabija

## ADVISORY BOARD - INTERNATIONAL

Prof. M.A. Arraez, Spain  
Prof. H. Bertalanffy, Germany  
Prof. J. Brotchi, Belgium  
Prof. Y. Kato, Japan  
Prof. U. Kehler, Germany  
Prof. Christopher M. Loftus, USA  
Dr M.R. Mahmud, Nigeria  
Prof. P. Mertens, France  
Prof. B.K. Misra, India

Prof. D.F. Muresanu, Romania  
Prof. S.C. Robertson, USA  
Prof. M. Samii, Germany  
Prof. J. Schramm, Germany  
Prof. M. Sindou, France  
Prof. M. Tatagiba, Germany  
Prof. M. Buchfelder, Germany  
Prof. I. Solaroglu, Turkey  
Prof. T.T. Wong, Taiwan

## EMERITUS EDITORIAL BOARD

Prof. A.V. Ciurea,  
Honorary Member of the Romanian Academy

Assoc. Prof. Habil. H. Ples, Romania,  
Former *Editor-in-Chief*

Assoc. Prof. St. M. Iencean, Romania,  
Former *Editor-in-Chief*

Prof. Al. Constantinovici  
Former *Editor-in-Chief*

## FOUNDING EDITOR

Prof. Constantin Arseni

---

## EXECUTIVE EDITOR

Madalin Onu, PhD

ROMANIAN

# NEUROSURGERY

---

Vol. XXXVIII | No. 3

September 2024



London  
Academic Publishing

Copyright © 2024 Romanian Society of Neurosurgery &  
London Academic Publishing

All rights reserved. This book or any portion thereof may not be reproduced or used in any manner whatsoever without the express written permission of the Romanian Society of Neurosurgery or the publisher except for the use of brief quotations in a book review or scholarly journal.

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

First Printing: September 2024  
London Academic Publishing Ltd.  
27 Old Gloucester Street  
WC1N 3AX  
London, United Kingdom  
Email: [contact@lapub.co.uk](mailto:contact@lapub.co.uk)

[london-ap.uk](http://london-ap.uk)  
[lapub.co.uk](http://lapub.co.uk)  
[journals.lapub.co.uk](http://journals.lapub.co.uk)  
[journals.lapub.co.uk/index.php/roneurosurgery](http://journals.lapub.co.uk/index.php/roneurosurgery)

Company Reg. No. 10941794  
Registered in England and Wales

The opinions expressed in the published articles are the sole responsibility of the authors and do not reflect the opinion of the editors or members of the editorial board.

# CONTENTS

- 285 **Does vascularized adipose tissue enhance nerve regeneration?  
An experimental study on the sciatic nerve in rats**  
Bogdan Ioncioaia, Andrei-Nicolae Coseriu, Alex-Victor Oradan, Luciana-Madalina  
Gherman, Dan Gheban
- 291 **Armoured brain. An unusual presentation of chronic subdural  
haematoma**  
Chhitij Srivastava, Awdhesh Kumar Yadav, Mitrajit Sharma
- 294 **Our institutional experience with lateral extra-cavitary  
approach for posterior mediastinal neurofibroma. A case report**  
Shashank Nahar, Piyush Panchariya, Prashant Raj Singh, Prashant Lakhe
- 297 **Massive extra dural collection post cranioplasty causing rapid  
deterioration of the patient**  
Muhammad Mohsin Khan, Muhammad Mujahid Sharif, Bipin Chaurasia.
- 300 **Manifestation and outcome analysis of chronic subdural  
haemorrhage patient in a tertiary care centre**  
Sajag Gupta
- 310 **An unusual case of choroid plexus papilloma in infancy.  
Diagnostic and management challenges**  
Abhishek Kumar, Raj Kumar

- 314 **Schwannomatosis. A rare case report**  
Younes Dehneh, Hamid Khay, Mohamed Khouali, Moufid Faycel
- 317 **Skull base-cranio-facial trauma with dura mater repair after subsequent CSF leaking, diagnosis treatment and outcomes. A systematic review of the literature**  
Daniel Encarnacion, Gennady Chmutin, Bipin Chaurisia
- 328 **Technical nuance. Total endoscopic removal of third ventricle colloid cyst**  
Arvind Verma, Jaimin Modh, Krushi Soladhra, Dharmik Velani
- 332 **Primary bone non-Hodgkin lymphoma with vertebra involvement**  
Muhammed Erkam Yuksek, Gulsum Arslan Karagoz, Densel Arac, Fatih Keskin
- 337 **Spinal cord compression secondary to metastatic invasion of breast phyllodes tumour. Case report**  
Julián David Moreno Cavanzo, Juan Felipe Alvarado Riascos, Lorena García Agudelo, Laura Carolina Navarro Martínez
- 341 **A comparison of subperiosteal vs subdural drainage in the treatment of chronic subdural haemorrhage**  
Sajag Gupta
- 348 **Neuronavigational assisted ventriculo-peritoneal shunt surgery in the idiopathic intracranial hypertension (IIH) patients. Institutional experiences**  
Poonia Nemi Chand, Jain Surendra, Poonia Hardika, Poonia Dev, Kumar Chitresh, Poonia Sania
- 353 **Assessment of visual function in patients with posterior fossa tumours. A retrospective study**  
Raghav Jindal, Vijendra Bijarniya, Mayank Purohit, Vinod Sharma, Sanjeev Chopra, Ashok Gupta

360 **Delayed post traumatic CSF rhinorrhoea. Two rare cases and review of literature**

Jadhav Anil, Katyal Abhishek, Jagetia Anita, Srivastava A.K, Singh Daljit

364 **Guidelines for authors**





# Does vascularized adipose tissue enhance nerve regeneration? An experimental study on the sciatic nerve in rats

Bogdan Ioncioaia<sup>1</sup>, Andrei-Nicolae Coseriu<sup>2</sup>, Alex-Victor Oradan<sup>3</sup>,  
Luciana-Madalina Gherman<sup>4</sup>, Dan Gheban<sup>5</sup>

<sup>1</sup> Plastic Surgery and Burns Division, Clinical Department of General Surgery No.1, Clinical Emergency Hospital, Cluj-Napoca, ROMANIA

<sup>2</sup> Plastic Surgery Department, Clinical Rehabilitation Hospital, Cluj-Napoca, ROMANIA

<sup>3</sup> Plastic Surgery Department, Clinical Rehabilitation Hospital, Cluj-Napoca, ROMANIA;  
University of Medicine and Pharmacy "Iuliu Hatieganu", Cluj-Napoca, ROMANIA

<sup>4</sup> Experimental Centre of University of Medicine and Pharmacy "Iuliu Hatieganu", Cluj-Napoca, ROMANIA

<sup>5</sup> Department of Pathology, University of Medicine and Pharmacy "Iuliu Hatieganu", Cluj-Napoca, ROMANIA

## ABSTRACT

**Background.** The use of adipose tissue in reconstructive procedures has become popular in the last decades due to the adipose tissue properties of stem cells and mechanical protection. In acute nerve lesions, there have been experimental reports on the use of non-vascularised adipose tissue as an adjuvant for nerve recovery but to our knowledge there is no published research that describes the effect of the pedicled vascularised adipose tissue (pVAT) on acute nerve injuries. Therefore we decided to study the effect of pVAT on nerve regeneration when wrapped around an epineurial coaptation site and for a 5 mm defect of the sciatic nerve from the rat.

**Methods.** The effect of pedicled vascularized adipose tissue (pVAT) on nerve regeneration was studied on the sciatic nerve injury model in twenty-four Wistar rats divided into four groups: epineurial repair; epineurial repair and pVAT; a 5 mm nerve gap bridged by an autograft; a 5 mm nerve gap bridged by a conduit-pVAT (C-pVAT). Automatization injuries, walking track analysis, postoperative extraneural fibrosis and histological analysis were performed three months after the intervention.

**Results.** Although histological and functional nerve regeneration was present in various degrees in all the studied groups, nerve regeneration was not enhanced by the use of the pVAT or C-pVAT. The harvesting of the pVAT flap caused postoperative fibrosis when used as a conduit. Automutilation was not decreased by the use of the pVAT nor a correlation between automutilation and postoperative fibrosis in the studied groups could be established. The postoperative fibrosis did not influence neural regeneration in the pVAT and autograft group.

**Conclusion.** Vascularized adipose tissue did not enhance neural regeneration when used as an adjunct procedure for primary nerve repair and or even as a novel conduit for 5 mm nerve defects in the rat sciatic nerve.

## Keywords

adipose flap,  
nerve regeneration,  
pedicled adipose transfer,  
nerve repair



Corresponding author:  
**Bogdan Ioncioaia**

Clinical Emergency Hospital,  
Cluj-Napoca, Romania

bogdanioncioaia@gmail.com

Scan to access the online version



## INTRODUCTION

The use of adipose tissue in reconstructive procedures has become popular in the last decades due to the adipose' tissue properties of stem cells import and mechanical protection. Adipose tissue was successfully used in cases of recurrent compressive neuropathies as a graft<sup>1</sup>, as a pedicled flap<sup>2</sup> or even as free tissue transfer<sup>3</sup>.

Vascularized tissue has the advantage of carrying its own blood supply thus being independent from the state of the recipient site providing in theory a faster therapeutical response. Depending upon the size of the gap between the ending parts of a severed nerve, the surgical treatment of acute nerve injuries consists of end-to-end coaptation, nerve grafting, nerve conduits or nerve transfer for cases where nerve regeneration takes longer to reach the motor end-plate<sup>4</sup>.

In acute nerve lesions there has been experimental reports on the use of non-vascularized adipose tissue (fat graft) as an adjuvant for nerve recovery<sup>5,6</sup> but to our knowledge there is no published research that describes the effect of the pedicled vascularized adipose tissue (pVAT) on acute nerve injuries. Therefore, we decided to study the effect of pVAT on nerve regeneration when wrapped around an epineurial coaptation site and for a 5 mm defect of the sciatic nerve from the rat.

## MATERIAL AND METHOD

This study was performed in line with the principles charta of animal's care and use. Approval was granted by the Ethics Committee of University of Medicine and Pharmacy Iuliu Hațieganu (23.02.2021/251).

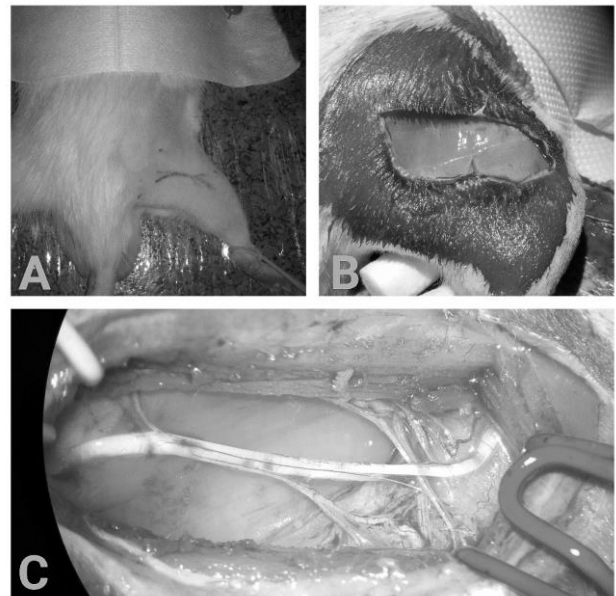
Twenty-four Wistar rats with an average weight of 300 g were divided randomly into four groups (n=6) as seen in table 1: group 1- transection and epineurial coaptation of the nerve (N) ; group 2 – transection, epineurial coaptation and wrapping of the coaptation site with pVAT ; group 3 – nerve defect of 5 mm length and defect bridging with nerve autograft from the contralateral sciatic nerve (A); group 4 – nerve defect of 5 mm length and defect bridging with a p-VAT conduit (C-pVAT).

Anesthesia was given intramuscular (40-90 mg/kg ketamine and 5-10 mcg/kg xylazine) before the commencement of the procedure. After preparation of the incision site (hair trimming and Betadine cleansing of the skin) an intermuscular approach was

chosen for sciatic nerve exposure. The sciatic nerve was exposed over its entire length in the hindlimb starting from its proximal entry location until its distal bifurcation point around the knee area (Figure 1).

**Table 1.** Distribution of the animal subjects into four study groups.

Group 1 N	Group 2 pVAT	Group 3 A	Group 4 C-pVAT
(n=6)	(n=6)	(n=6)	(n=6)
Transection + epineurial coaptation	Transection + epineurial coaptation + pVAT	5 mm nerve defect + Autograft	5 mm nerve defect + C-pVAT



**Figure 1.** Preparation of the incision site and exposure of the sciatic nerve.

A. The approach for the sciatic nerve is marked having the rat placed in a dorsal position.

B. Exposure of the intermuscular septum after skin preparation.

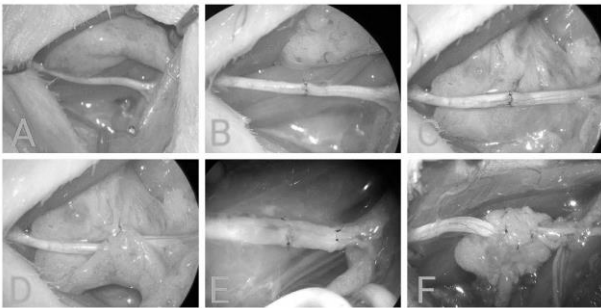
C. Exposure of the sciatic nerve through an intermuscular approach. The left of the image depicts the proximal part of the sciatic nerve while the right part of image depicts the distal sciatic nerve with its bifurcation around the knee area.

The pVAT was harvested from the vicinity of the sciatic nerve either anterior or posterior to it using a microsurgical dissection technique with the aid of an

operating microscope. The dissected pVAT was mobilized in order to cover the recipient site without tension. Neural coaptation and pVAT wrapping was achieved with 9-0 or 10-0 nylon sutures (Figure 2).

**Table 2.** Scoring system used for the postneurotomy lesions.

Lesions	Score
No lesions	1
One finger injured	2
Two fingers injured	3
Three fingers injured	4
Four fingers injured	5
Five fingers injured	6



**Figure 2.** Various procedures performed on the sciatic nerve. A. Mobilization of the adipose flap (pVAT) superior (anterior) to the uninjured sciatic nerve. B. Epineurial coaptation of the sciatic nerve with the pVAT on "stand-by". C and D. Wrapping the the pVAT around the coaptation site. E. Autograft for 5 mm defect from the contralateral hindlimb. F. Wrapping the pVAT as a conduit for a 5 mm defect using an inferior (posterior) approach.

After the conclusion of the procedure the rats were housed in appropriate conditions, with access to food and water ad libitum following a circadian rhythm with daily visits of the veterinary personnel. After a period of 3 months, the operated limb was examined for automutilation injuries which were and graded according to the criteria displayed in table 2, subjected to walking track analysis (WTA) followed by intraoperative nerve inspection under general anesthesia for postoperative extraneural fibrosis (table 3), concluding with a nerve sampling distal to the manipulated area of the nerve for histologic analysis using microscopy under haematoxylin eosine and trichrome Masson staining. Histological regeneration was quantified using a two-step

approach. First the presence of axons distal to the procedural site was sought and their presence were suggestive for positive regeneration. Secondly the degree of regeneration was graded using a qualitative scale established by the authors: absent, weak, moderate, good (table 4) using the average values of each studied group. The WTA values were processed in order to obtain the sciatic functional index (SFI), which was further used to quantify the nerve regeneration in a qualitative manner (table 5) based on a modification of the scale used by Amniattalab et al<sup>7</sup>.

At the end of the experiment the Wistar rats were euthanized according to the experimental protocol. The collected data was processed and statistically analyzed using Wilcoxon matched pair test and Pearson correlation test. Experimental results were displayed as percentage or/and as mean  $\pm$  standard deviation (SD); differences were considered significant when  $P < 0.05$  and correlation (R) could be established (negative, absent and positive for R negative, R null and R positive values).

**Table 3.** Postoperative extraneural fibrosis severity scale (after Petersen et al, 1996).

Scale	Definition
1	No dissection required/ mild blunt dissection
2	Vigorous blunt dissection required
3	Sharp dissection required

**Table 4.** Qualitative scale of histological nerve regeneration were regeneration was present.

Average value interval	Significance
Less than 0.25	Absent
0.25 – 0.50	Weak
0.51 – 0.75	Moderate
0.76 – 1.00	Good

**Table 5.** Qualitative scale of functional nerve regeneration based on SFI values interval.

Sciatic Functional Index	Significance
0 to - 25	Very good
-25 to -50	Good
-50 to -75	Moderate

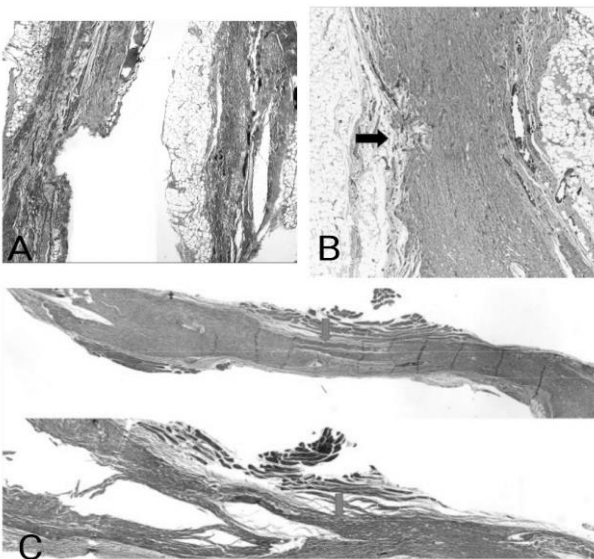
-75 to -99	Weak
Greater than - 99	Absent

**RESULTS**

Results are displayed in table 6 and 7. Histological nerve regeneration was present in various degrees in all the studied groups (Figure 3): group 1 – moderate regeneration, group 2 – weak regeneration, group 3 – good regeneration and 4 – moderate regeneration, but neural regeneration was not enhanced by the use of the pVAT (p=0.31) neither by the C-pVAT (p=0.56). Functional nerve regeneration quantified through the SFI was deemed as good for the coaptation (-42.11±29.70), moderate for the pVAT group (-52.36±34.36), while the autograft (-85.11±13.32) and C-pVAT group (-76.11±19.17) exhibited poor regeneration. Both pVAT (p=0.52) and C-pVAT (p=-1.15) group did not enhance the regeneration of the sciatic nerve.

Postoperative extraneural fibrosis (Figure 4) was present in each of the studied groups, but to a lesser degree in group 1 and 2 (p=0.04) compared to groups 3 and 4 (p=0.05). Postoperative extraneural fibrosis in the C-pVAT group had a negative effect on functional muscle regeneration (R=0.96, P=0.02).

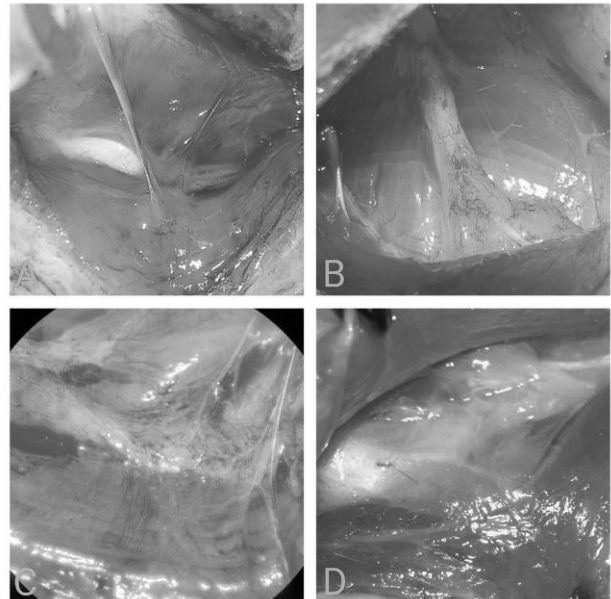
Automutilation was limited to one finger and was not decreased by the use of the pVAT (p>0.05) neither a correlation between automutilation and postoperative fibrosis in the studied groups could be established (p>0.05).



**Figure 3.** Histological analysis of the nerve samples collected at the repair zone depicting various degrees of nerve

regeneration (hematoxylin eosine and Masson trichrome staining).

- A. Nerve fibers crossing the coaptation site in subject no.1 from group 1.
- B. Nerve fibers display continuity in subject no. 7 from group 2.
- C. Thin nerve fibers displayed in continuity in subject no. 13 from group 3.



**Figure 4.** Various degrees of postoperative perineural fibrosis after Petersens' classification:

- A. Group 1 – Grade 1 fibrosis: mild dissection required;
- B. Group 2 - Grade 2 fibrosis: vigorous blunt dissection required;
- C. Group 3 – Grade 3 fibrosis: sharp dissection required;
- D. Group 4 - Grade 3 fibrosis: sharp dissection required.

**Table 6.** Statistical analysis of the studied groups.

Postneurotomy lesions	Mean ± SD	P value
Group 1	1.33 ± 0.81	0.26
Group 2	2.16±1.47	0.71
Group 3	2.83± 2.04	0.10
Group 4	1.16±0.40	0.65
<b>Postoperative extraneural fibrosis</b>		
Group 1	2.0±0.63	0.04
Group 2	1.83±0.75	
Group 3	2.66±0.51	0.05
Group 4	2.66±0.51	
<b>Nerve regeneration</b>		
Group 1	0.66±0.51	0.31

Group 2	0.50±0.54	
Group 3	0.83±0.4	0.56
Group 4	0.66 ±0.51	
<b>WTA</b>		
Group 1	-42.11±29.70	0.52
Group 2	-52.36±34.36	
Group 3	-85.11±13.32	-1.15
Group 4	-76.11±19.17	

**Table 7.** Statistical correlation between extraneural fibrosis, automutilation, histological nerve regeneration and functional muscle regeneration.

	Fibrosis and automutilation	Fibrosis and histological regeneration	Fibrosis and functional muscle regeneration
<b>Group 1</b>	R=0; P=1	R=0; P=1	R=-0.27; P=0.60
<b>Group 2</b>	R=0.03; P=0.95	R=- 0.24; P=0.64	R=-0.17; P=0.74
<b>Group 3</b>	R=0.31;P=0.54	R=- 0.31; P=0.54	R=0.05; P=0.92
<b>Group 4</b>	R=0.31; P=0.54	R=0.25; P=0.63	R=0.96; P=0.02

## DISCUSSION

Owing it not only to the presence of adipose-derived stem cells (ASC) and its growth factors<sup>8,9</sup>, but also to its mechanical properties adipose tissue serves as a promising add-on to the reconstructive armamentarium<sup>10</sup> with beneficial results in treating chronic nerve lesions<sup>1-3</sup>. The experimental work of Tuncel et al concluded that autologous fat graft improved the regeneration of the sciatic nerve in rats when used as an adjunct to primary coaptation, autograft and venous conduit filled with fresh or denaturated skeletal muscle<sup>6</sup>. Considering the reported<sup>11</sup> broad spectrum of fat graft take (between 20 and 80%), increased survival depends upon the optimal blood supply of the adipose tissue, which is ideal as vascularized tissue (pedicled or free tissue transfer). The vascularized adipose tissue transfer settles the issue of donor tissue preparation as in fat graft harvesting and recipient site viability, but requires careful flap dissection, maintaining the nutrient vessel in its substance. VAT in the form of

pedicled or free fat flaps are being used in chronic peripheral nerve injuries with good results<sup>1-3</sup>.

In our experiment we studied whether pVAT had a positive influence on the regeneration of an acute nerve injury model on the sciatic nerve after epineurial coaptation following complete transection and as a novel adipose conduit for a 5 mm nerve gap (C-pVAT) and its influence on postoperative fibrosis and automutilation injuries.

The p-VAT plays a similar role to the nerve wrapping procedures used in chronic neuropathies<sup>2,3</sup>. The C-pVAT on the other hand is a novel procedure used for small nerve gaps (5 mm) and is designed as a vascularized conduit using a similar technique as for nerve wrapping except its proximal and distal ends are secured to the nerve stumps in such a way that a tube which should facilitate the transition of axons to the target area is created. It is a technically demanding procedure that requires a patent lumen while wrapping the adipose flap around the defect and may finally cause the obstruction of regenerating nerve fibers due to its bulkiness, as was noticed in some cases.

Histological nerve regeneration was established in a qualitative manner by localizing the presence of axons in the distal stump and assigning a grade to the average value of each of the studied group in order to estimate the quality of nerve regeneration. Despite the fact that axons could be noticed distal to the procedural stump in various degrees, the use of VAT did not enhance nerve regeneration. As a caveat to the applied method we mention that the quantity of axons measured distally was not appreciated, thus the exact number of axons could not be estimated. Functional muscle regeneration was quantified by SFI values measured at 3 months postoperative that were compared to a scale developed by the authors based on other published research. The least extensive procedure presented the best outcome regarding WTA as seen in group 1 and 2 compared to group 3 and 4. Given that normal gait was compromised in the donor hindlimb for the autograft group, WTA values may have been altered and the functional nerve regeneration may have not been precisely determined in this instance.

The extent of flap dissection was direct proportional to the severity of the postoperative fibrosis (Figure 4), causing less fibrosis in the pVAT group compared to the C-pVAT group. Therefore harvesting pedicled fat from the vicinity of a nerve,

may not yield the best results for treating a nerve injury when both flap and nerve share the same location as it is known that postoperative fibrosis remains a cause of recurrence after peripheral nerve surgery<sup>1-3</sup>.

Automutilation of the rat's distal limb following a proximal nerve injury, indicates the loss of protective sensibility. Interestingly, the C-pVAT group exhibited fewer automutilation injuries than its counterparts, suggesting that fat may play a protective role in skin integrity during nerve regeneration although the mechanism is not understood and perhaps even play a similar role to the regenerative peripheral nerve interface for the treatment of painful neuromas<sup>12</sup>.

Although histological and functional nerve regeneration was present in different degrees in the p-VAT and the C-pVAT groups they did not improve nerve regeneration, showing no benefit of VAT when used as an adjunct for epineurial coaptation or as a novel adipose conduit for a 5 mm nerve gap. We would like to highlight some technical aspects that may have negatively influenced our outcomes.

One limitation of our study was the difficulty in maintaining a consistent pVAT harvesting location and nerve wrapping technique so as to avoid tension and devascularization of the flap at the injury site. Although flap preparation and VAT wrapping was performed with minimal tension, after 90 days we noticed in some cases that the sciatic nerve was elongated which may have had negatively influenced the outcomes. This may have been the explanation for the poor results obtained in the pVAT and C-pVAT groups as seen in histological and functional nerve regeneration values. There was also a learning curve in the process of flap raising and inseting. The difficulty in precisely determining the main blood supply of the flap, could have made some parts of the flap non-viable, especially the distal part. Last but not least, given its volume the pVAT was difficult to tailor as an adipose conduit as to maintain a patent lumen for the advancement of nerve fibers and therefore we think that it is not suitable for bridging nerve defects in small nerves.

## CONCLUSION

Vascularized adipose tissue did not enhance neural regeneration role when used as an adjuvant

procedure for primary nerve repair or as a novel conduit for 5 mm nerve defects in the rat sciatic nerve.

## REFERENCES

1. Cabrejo R, Podsednik A, Rosen J. Treatment of recurrent carpal tunnel syndrome with fat grafting as an adjunct. *Plastic and Aesthetic Research*. 2023; 10(1):1.
2. Adani R, Tos P, Tarallo L, Corain M. Treatment of painful median nerve neuromas with radial and ulnar artery perforator adipofascial flaps. *J Hand Surg Am*. 2014 Apr;39(4):721-7.
3. Jones NF. Treatment of chronic pain by "wrapping" intact nerves with pedicle and free flaps. *Hand Clin*. 1996 Nov;12(4):765-72.
4. Tan RES, Jeyaratnam S, Lim AYT. Updates in peripheral nerve surgery of the upper extremity: diagnosis and treatment options. *Ann Transl Med*. 2023;11(11):391.
5. Podsednik A, Cabrejo R, Rosen J. Adipose Tissue Uses in Peripheral Nerve Surgery. *Int J Mol Sci*. 2022 Jan 7;23(2):644.
6. Tuncel U, Kostakoglu N, Turan A, Çevik B, Çaylı S, Demir O, Elmas C. The Effect of Autologous Fat Graft with Different Surgical Repair Methods on Nerve Regeneration in a Rat Sciatic Nerve Defect Model. *Plast Reconstr Surg*. 2015 Dec;136(6):1181-1191.
7. Amniattalab A, Mohammadi R, Functional, Histopathological and Immunohistochemical Assessments of Cyclosporine A on Sciatic Nerve Regeneration Using Allografts: A Rat Sciatic Nerve Model. *Bull Emerg Trauma*. 2017 Jul;5(3):152-159.
8. Mazini L, Rochette L, Malka G. Adipose-Derived Stem Cells (ADSCs) and Growth Differentiation Factor 11 (GDF11): Regenerative and Antiaging Capacity for the Skin. Available at: <https://www.intechopen.com/chapters/71246>. Accessed January 5, 2024.
9. Rhode, SC, Beier, JP, Ruhl, T. Adipose tissue stem cells in peripheral nerve regeneration—In vitro and in vivo. *J Neurosci Res*. 2021; 99: 545–560.
10. Tan SS, Ng ZY, Zhan W, Rozen W. Role of Adipose-derived Stem Cells in Fat Grafting and Reconstructive Surgery. *J Cutan Aesthet Surg*. 2016 Jul-Sep;9(3):152-156.
11. Xue EY, Narvaez L, Chu CK, Hanson SE. Fat Processing Techniques. *Semin Plast Surg*. 2020 Feb;34(1):11-16.
12. Senger JL, Thorkelsson A, Wang BY, Chan KM, Kemp SWP, Webber CA. 'Inlay' Regenerative Peripheral Nerve Interface (RPNI) is superior to 'Burrito' RPNI for successful treatment of rat neuroma pain. *Plast Reconstr Surg*. Published online July 4, 2023.



# Armoured brain. An unusual presentation of chronic subdural haematoma

**Chhitij Srivastava, Awdhesh Kumar Yadav,  
Mitrajit Sharma**

Department of Neurosurgery, King George's Medical University,  
Lucknow, Uttar Pradesh, INDIA

## ABSTRACT

Calcified chronic subdural hematoma is a rare but known entity, estimated to represent 0.3-2.7% of chronic subdural hematomas. Although surgical treatment is unanimous for chronic subdural hematomas, therein lies some debate on surgical evacuation of calcified chronic subdural hematomas. We report a case of an 18-year-old male presenting after 17 years of VP shunt with calcified chronic SDH. He presented with only mental retardation and no other neurological deficit and as such he was managed conservatively and discharged with close follow-up. We believe that surgical treatment should only be used in circumstances when it is symptomatic, essential, and viable, and will eventually lead to the patient's neurological improvement based on the cases mentioned in the literature. Our personal experience with this case is that the non-surgical procedure sometimes is able to provide good outcomes, especially when the extracranial problems are managed carefully and the patient doesn't complain of any neurological deficit.

## INTRODUCTION

Chronic calcified subdural hematoma was originally described in 1884, though uncommon it is a well-known condition that accounts to 0.3-2.7% of chronic subdural hematomas.<sup>1,2,3,4</sup> The mechanism of calcified chronic subdural hematoma, sometimes known as "armoured" brain, is poorly understood. The ideal surgical technique for patients with armored brains has not been determined because these patients have a thick calcified inner membrane.<sup>5</sup> Additionally, it is challenging to get a successful re-expansion of the brain following surgery.<sup>6</sup> There have been about 120 cases of chronic calcified subdural hematoma published, and most cases of calcified chronic SDH are managed on an individual basis.<sup>4</sup> The need for surgery may be indicated by signs of increased intracranial pressure, headache, or neurological decline.<sup>1,2</sup> The surgical approaches depend on the extent and thickness of the calcification. Here, we present a case study of an 18-year-old male with an armored brain who was conservatively handled.

---

## Keywords

brain injury,  
calcification,  
chronic subdural haematoma

---



Corresponding author:  
**Mitrajit Sharma**

Department of Neurosurgery, King  
George's Medical University,  
Lucknow, India

mitrajitsurgery@gmail.com

Scan to access the online version



### CASE REPORT

The patient is an 18-year-old male, who has a history of Dandy Walker Malformation and underwent right sided parietal MPVP shunt at the age of 1 year. Following shunt surgery, he was lost to follow up, and he presented again in 2023 for follow-up with mental retardation. NCCT brain was done and it was suggestive of right sided calcified chronic SDH. (Figure 1). Even though there was significant midline shift, it did not cause any neurological symptoms. As such he was managed conservatively. He has continued to do well after 8 months of post discharge.



**Figure 1.** NCCT Brain showing the calcified membranes with shunt with left deviated lateral ventricles with midline shift.

### DISCUSSION

Chronic subdural hematoma is one of the common pathologies encountered in neurosurgical practice, but its calcification or ossification has been rarely published in the literature. Although the exact incidence of calcified or ossified CSDHs is unknown, it has been reported to range from 0.3 to 2.7%.<sup>1,2</sup> The calcified or ossified CSDHs due to various etiologies occur more frequently in children and young adults. They generally present with seizures, headache, memory deficit, difficulty in walking, or deficit in the level of consciousness.<sup>7</sup> In our case, he presented with only mental retardation and no other complain.

Calcified chronic SDH has been observed as late complication of head injury or late sequel of post meningitis subdural effusion. It has also been reported as delayed complication of VP shunt. Systematic review studied by Turgut et al. showed that there were 78 men and 29 women from 25 countries, ages ranging from 4 months to 86 years (mean 33.7 years), with etiologies of head trauma in 33.3%, shunting for hydrocephalus in 27.2%, or following cranial surgery in 4.4%. The duration of

symptoms ranged from acute onset to 20 years, with a mean of 24.1 months.<sup>8</sup>

The pathogenesis of calcification in CSDH is not very well understood. Some popular postulates regarding its formation are<sup>9,10,11</sup>

- 1) Dense collagen depositions occur on the membranes forming fibrotic capsule, eventually calcifies from progressive mineralization
- 2) Inadequate circulation and lack of absorption in the subdural cavity
- 3) Thrombosis in the bloodstream, clotting blood in the subdural cavity
- 4) Poor circulation and poor venous return
- 5) Thick layers of connective tissue prone to easy calcification.

Even though surgical treatment for chronic SDHs is universal, there is some question over whether it can be used for calcified chronic SDHs. Due to the restricted reexpansion of the brain following surgery, the ideal surgical treatment of "armored brain," has not been determined. This is most likely connected to the existence of a thick, calcified inner membrane that is frequently adhered to the cortical surface. Surgical removal of the calcification is difficult, and it could damage the underlying cortex.<sup>12, 13</sup> For calcified chronic SDH that is increasing in size, surgical intervention is preferred. Various surgical methods for calcified chronic SDH have been documented. Twist drill aspiration, burr hole aspiration, or microsurgical dissection are techniques frequently used.<sup>12, 13</sup> Brain contusion, bleeding, and the development of fresh neurological deficits can occur as a result of micro surgically removing a calcified hematoma from the surface of the brain. On the other hand, other authors argue that by reducing the detrimental effects of compression and inflammation as well as normalizing cerebral blood flow, evacuation of the hematoma will enhance neurological status.<sup>5</sup> According to our experience; patient management is decided on an individual basis. Patients who have no symptoms, are elderly, or don't have any neurological deficits can be handled conservatively with constant monitoring.

### CONCLUSIONS

Despite the rarity and indolent character of chronic calcified subdural hematomas, which can have remarkable radiologic features but no clear clinical

correlate, these rare entities are well tolerated. We describe a case of known DWM who underwent VP shunt and then developed chronic calcified membranous SDH 18 years later. This patient was managed conservatively because surgery is useful in only a small number of patients with a steadily worsening neurological status. We believe that surgical treatment should only be used in circumstances when it is symptomatic, essential, and viable, and will eventually lead to the patient's neurological improvement based on the cases mentioned in the literature. Our personal experience with this case is that the non-surgical procedure sometimes is able to provide good outcome, especially when the extra cranial problems are managed carefully and the patient doesn't complain of any neurological deficit.

#### REFERENCES

- Kaplan M, Akgun B, Seçer HI. Ossified Chronic Subdural Haematoma with Armored Brain. *Turk Neurosurg* 2008;4:420-4.
- Park JS, Son EI, Kim DW, Kim SP. Calcified Chronic Subdural Haematoma Associated with intracerebral haematoma. *J Korean Neurosurg* 2003;34:177-8.
- Pruna V, Bucur N, Neacsu A, Voina A, Andrei G, Sandu A, et al. Calcified chronic subdural haematoma – Case report. *Roman Neurosurg* 2010;15:22-5.
- Yan HJ, Lin KE, Lee ST, Tzaan EC. Calcified chronic subdural haematoma: Case report. *Changgeng Yi Xue Za Xhi* 1998;21:521-5.
- Niwa J, Nakamura T, Fujishige M, Hashi K. Removal of a large asymptomatic calcified chronic subdural haematoma. *Surg Neurol* 1988;30:135-9.
- Oda S, Shimoda M, Hoshikawa K, Shiramizu H, Matsumae M. Organized Chronic Subdural Haematoma with a thick calcified inner membrane successfully treated by surgery: A case report. *Tokai J Exp Clin Med* 2010;3:85-8.
- P. Salunke, A. Aggarwal, K. Madhivanan, S. Futane, Armoured brain due to chronic subdural collections masking underlying hydrocephalus, *Br. J. Neurosurg.* 27 (2013) 524–525.
- M. Turgut, A. Akhaddar, A.T. Turgut, Calcified or ossified chronic subdural hematoma: a systematic review of 114 cases reported during last century with a demonstrative case report, *World Neurosurg.* 134 (2020) 240–263.
- Afra D. Ossification of subdural hematoma. Reports of two cases. *JNeurosurg.* 1961;18:393-7. DOI: 0.3171/jns.1961.18.3.0393.
- Ide M, Jimbo M, Yamamoto M, et al. Asymptomatic calcified chronic subdural hematoma – A report of 3 cases. *Neurol Med Chir (Tokyo).* 1993;33(8):559–63. DOI: 10.2176/nmc.33.559.
- Friede R. Incidence and distribution of neo membranes of the dura mater. *J Neurol Neurosurg Psychiatry.* 1971;34:439–46. DOI: 10.1136/jnnp.34.4.439.
- Sakamoto T, Hoshikawa Y, Hayashi T, Taguchi Y, Sekino H. Inner membrane preservation surgery for organized or calcified chronic subdural hematoma. *Jpn J Neurosurg (Tokyo)* 2000;9:541-6.
- Yamada K, Ohta T, Takatsuka H, Yamaguchi K. High field magnetic resonance image of a huge calcified chronic subdural haematoma, so called “armoured brain”. *Acta Neurochir (Wien)* 1992;114: 151-3.



# Our institutional experience with lateral extra-cavitary approach for posterior mediastinal neurofibroma. A case report

**Shashank Nahar, Piyush Panchariya, Prashant Raj Singh, Prashant Lakhe**

MGM Medical College, Indore (M.P), INDIA

## ABSTRACT

The LECA to the thoracic spine can be used for the removal of posterior mediastinal neurofibromas which causes anterior and lateral spinal cord compression. It provides lateral exposure to the thoracic and lumbar vertebrae without entering the pleural cavity (extra-cavitary) and enables the surgeon to visualize the anterior dural surface better than with other posterior lateral exposures. In this case report, we describe the challenges we face while operating the posterior mediastinal tumour using this surgical approach. This approach provides better access to ventral and lateral thoracic and upper lumbar spine pathologies.

## INTRODUCTION

The lateral extra-cavitary approach (LECA) to the thoracic spine was originally developed by Alexander (1) for the treatment of tuberculous spondylitis. (2) This procedure was later modified to expand its application to other anterior spinal column pathologic findings (e.g., fractures, infection, thoracic disk disease,) and it allows placement of posterior instrumentation. (3,4) The LECA to the thoracic spine can be used for the removal of posterior mediastinal neurofibromas which causes anterior and lateral spinal cord compression. It provides lateral exposure to the thoracic and lumbar vertebrae without entering the pleural cavity (extra-cavitary) and enables the surgeon to visualize the anterior dural surface better than with other posterior lateral exposures. (5) Access to the thoracolumbar junction using an anterior lateral approach (thoracotomy or thoracoscopy) requires the takedown of the diaphragm, which is completely avoided using the LECA. In the treatment of metastatic spine disease, the LECA is ideal for patients requiring resection of one vertebral segment for spinal cord decompression, reconstruction, and stabilization. It can also be used for patients with involvement of up to three vertebral segments.

## Keywords

neurofibromas,  
lateral extra-cavitary  
approach,  
mediastinal tumour,  
thoracic spine



Corresponding author:  
**Shashank Nahar**

MGM Medical College,  
Indore (M.P), India

naharshashank@gmail.com

Scan to access the online version



## CASE SUMMARY

A 45 years old female presented to us with chief complaints of pain in left mid back region for 2 months. Pain was continuous type throughout the day, progressive in nature, increases while resting posture and was not relived on medications. Patient was admitted and underwent MRI dorsal spine and screening of whole spine.

On MRI – well defined heterogenous altered signal intensity lesion noted in posterior mediastinal on right side extending from D-8 to D-11 vertebra. It is seen as heterogenous hyperintense on T-2 sequence and hypointense on T-1 sequence with contrast enhancement on T1 contrast MRI.

Lesion was seen abutting the adjacent vertebra. No bony erosion was seen. No intra-spinal extension was noted.

## OPERATIVE TECHNIQUE

### A. Positioning

The patient is placed into the prone position after the placement of intravenous and arterial access and an indwelling Foley catheter with chest rolls.

### B. Skin incision

We took T shaped incision; vertical part of T was based on D8- to D11 spinous process and horizontal part of T was extending from midline of D-10 till about 5to 6 cm lateral.

### C. Operative procedure

We have performed gross total excision of right paravertebral (D-8 to D-11) posterior mediastinal mass lesion by lateral extra-cavitary approach without opening pleura, by taking right paravertebral D-8 to D-11 T shaped skin incision as mentioned above.

### D. Steps and Intra-operative findings

At right D-8 to D-11 region, transverse process and part of lamina was excised and the part of the attachment of ribs on lateral part of vertebrae was removed safely (costotransversectomy was done)

Dorsal mediastinal encapsulated mass encountered beneath the ribs pushing right lung laterally and mass was firmly adherent to medial pleural wall.

Small pleura tear was present while separating the tumor from medial pleural wall, while rest of the

tumor was found to be separable from medial pleura wall.

Mass was greyish pink in colour, firm to hard in consistency moderately vascular with well -defined plane from surrounding.

Gross total excision of tumor was done and repair of pleura followed by placement of intercostal drainage tube inside the pleural cavity.

### E. Post operative events

Post-operatively, pt's HRCT chest was done showing mild amount of pleural effusion on right side with complete removal of mass lesion. ICD was removed on 3rd post operative day after resolution of pleural effusion. Suture was removed on 10th post-operative day. Pt was self-ambulatory with no post operative neurological deficit. On biopsy report, the tumour was found to be neurofibroma.

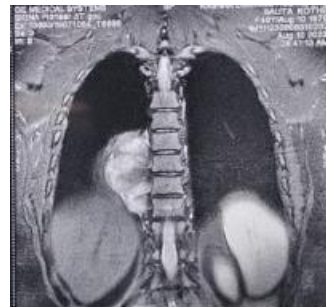


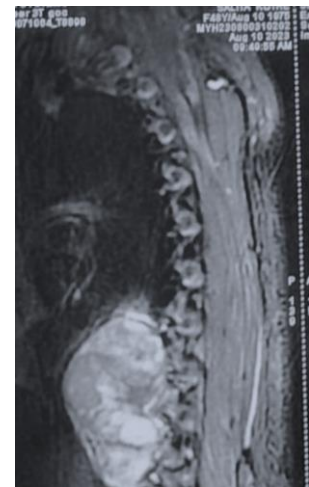
Figure 1.1. Coronal view.



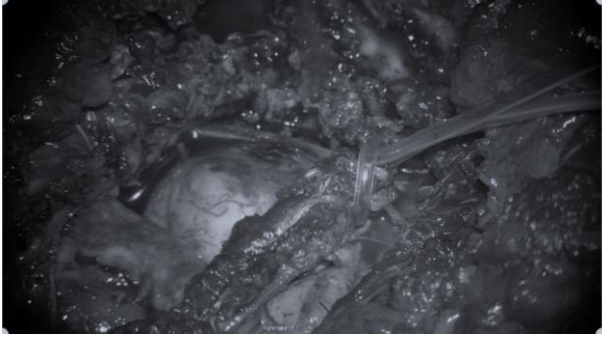
Figure 1.2. Axial view



Figure 1.3. Lateral view.



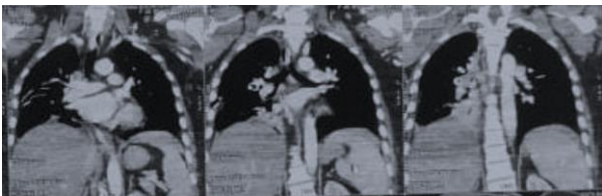
**Figure 1.** Shows coronal, axial, and lateral view MRI of chest. The tumour is present in posterior mediastinum at right paravertebral D8 to D11 region (pointed by red arrow).



**Figure 2.** Intra-operative image showing tumour after costotransversectomy and separating it from medial pleural wall.



**Figure 3.** Shows the separation of tumor capsule from the medial wall.



**Figure 4.** Post - Operative HRCT Chest Showing Rt sided mild pleural effusion with complete resection of posterior mediastinal tumour.

#### CONCLUSION

The LECA is a technically challenging procedure for a Neurosurgeons with a steep learning curve. Removing the posterior mediastinal tumor, staying extra cavity and without breaching the pleural surrounding wall is technically challenging procedure and needs much experience and expertise. In these procedures, the backup team of

thoracic surgeons is also many times essential as there can be risk of damage to pleura leading to pneumothorax or hemothorax, lung parenchyma and other vital structures in posterior mediastinum. Many times, it is one of the most versatile approaches to the spine with better visualization of anterior dural wall. Being a dorsolateral approach, it provides better access to ventral and lateral thoracic and upper lumbar spine pathologies.

#### Abbreviation

LECA - lateral extra-cavitary approach

MRI - Magnetic resonance imaging

CT - Computed tomography

#### Acknowledgement

The Author would like to thanks Dr Rakesh Gupta, Prof and Head dept of Neurosurgery and Dr Prashant Raj Singh, Associate Professor, Department of Neurosurgery, MGM Medical College, Indore for his valuable guidance and encouragement. I express my humble gratitude to all our faculty members Dr Piyush Panchariya, Assistant prof and Dr Prashant Lakhe, Assistant Prof, MGM Medical College, Indore for their constant support, encouragement, and valuable suggestions in conduct of this case report.

#### REFERENCES

1. Alexander GL. Neurological complications of tuberculosis. Proc R Soc Med 1946;39:730.
2. Lifshutz J, Lidar Z, Maiman DJ. Evolution of the lateral extra-cavitary approach to the spine. Neurosurgery Focus 2004;16(1): Article 12, 1-3.
3. Stillerman CB, Chen TC, Couldwell WT, Zhang W, Weiss MH. Experience in the surgical management of 82 symptomatic herniated thoracic discs and review of the literature. J Neurosurg 1998;88(4):623-33.
4. McCormick PC. Surgical management of dumbbell and paraspinal tumors of the thoracic and lumbar spine. Neurosurgery 1996;38(1):67-74.
5. Meic H. Schmidt, Sanford J. Larson, Dennis J. Maiman. The lateral extracavitary approach to the thoracic and lumbar spine. Neurosurgery Clinics of North America. 2004;15:437-441.



# Massive extra dural collection post cranioplasty causing rapid deterioration of the patient

Muhammad Mohsin Khan<sup>1</sup>, Muhammad Mujahid Sharif<sup>2</sup>,  
Bipin Chaurasia<sup>3</sup>

<sup>1</sup> Hamad General Hospital, QATAR

<sup>2</sup> PIMS, PAKISTAN

<sup>3</sup> Neurosurgery Clinic, Birgunj, NEPAL

## ABSTRACT

**Introduction.** Cranioplasty is a standard neurosurgical procedure these days where surgeons repair a defect or deformity of a skull. This procedure can lead to fatal experiences after surgery. We encounter a similar experience we want to share.

**Case report.** we present a case of 37 years 37-year-old patient who developed epidural fluid collection after cranioplasty. The patient deteriorated after surgery which we managed carefully and recovered later.

**Conclusion.** Post-cranioplasty patients should be under observation and neurological deterioration should be kept in mind. Surgical intervention is not always needed and can be managed conservatively.

## INTRODUCTION

Cranioplasty is a standard neurosurgical procedure these days where surgeons repair a defect or deformity of a skull. Cranioplasty, contrary to popular belief, is an ancient procedure. We have proof that surgeons in the 14th and 15th centuries carried out cranioplasty using metals like gold and copper. Contrary to popular belief, Cranioplasty is not just a cosmetic repair of cranial defects; cosmetics is only one part of its wide-ranging benefits. It returns the intracranial environment back to its normal physiology and helps in healing and repairing of neurological deficits. It is part of the rehabilitation process following a patient's neurological injury. Substantial enhancements in neuropsychological deficits, prevention of convulsions and partial prevention of cerebral atrophy are also achieved. New literature coming up shows that cranioplasty may improve the patient's psychological status, social performance, and neurocognitive functioning.<sup>1</sup> But the procedure comes with its own sets of complications. They include needing more operations, seizures, infections, hydrocephalus, Epidural fluid collection and even death. Epidural fluid collection (EFC) that occurs after cranioplasty is not a common side effect, and alot of them EFC regress

## Keywords

cranioplasty,  
extradural haematoma,  
road traffic accident



Corresponding author:  
Bipin Chaurasia

Neurosurgery Clinic,  
Birgunj, Nepal

trozexa@gmail.com

Scan to access the online version



naturally if given time, so we expect that the true incidence is miscalculated. 2,3 This complication of fluid collection in the epidural space is not described comprehensively in the literature, its importance is yet to be established, and there is not much data available on this issue. We report this case as extradural collection post cranioplasty leading to neurological deterioration.

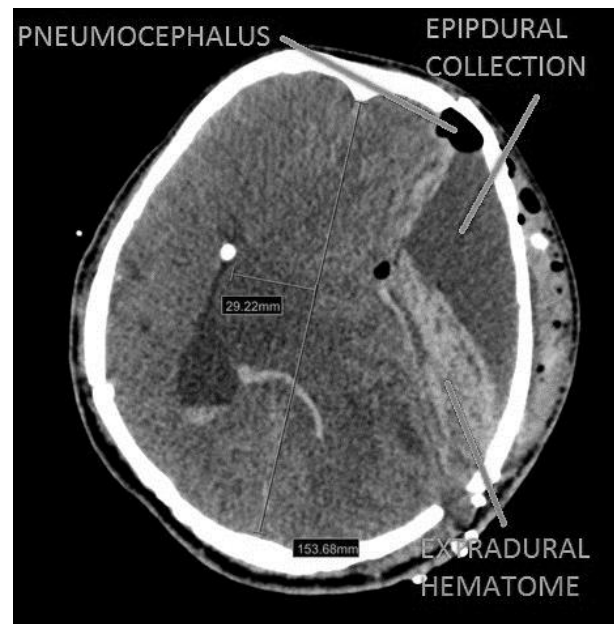
#### CASE PRESENTATION

37-year-old patient developed epidural fluid collection after cranioplasty. This patient presented with the history of road traffic accident, GCS at the scene 6/15, was intubated in the emergency department, CT scan done and showed acute subdural haemorrhage. Left side decompressive craniectomy and evacuation of subdural hematoma was done. Patient shifted toward later on, tracheostomy was done for the patient, patient condition was static and did not improve. He was planned for cranioplasty. After cranioplasty patient was shifted to recovery room, where he developed fixed pupil, CT SCAN was repeated which showed large underlying intracranial collection of CSF density containing haemorrhagic component (Figure 1). There was significant mass effect and midline shift to the right side by approximately 3 cm. Emergency surgery and evacuation of left parietal collection was done. Post op GCS was E1 M2 Vt. patient stayed in medical ICU for monitoring purposes his stay in ICU was uneventful and was transferred back to ward.

#### DISCUSSION

Cranioplasty nowadays is a very common neurosurgical procedure, and is viewed as safe and straight forward. But, even with cutting-edge techniques, a considerably higher complication rate is associated with cranioplasty as compared .4,5 Among minor complications, EFC is known to be very uncommon. Chang et al 2 in a series of 213 patients, stated that 13 of the patients (6.1%) faced fluid collection complications. Lee et al.3 examined incidence and factors that predict the fate of Extra axial Fluid Collection after Cranioplasty. Of the 59 patients studied in the series, EFC was occurred in 22 patients (37.3%).but these studies don't give us the reason or any pathophysiology of development of this fluid collection. The exact mechanism after cranioplasty why this collection in the epidural space happens is not known but the hypothesis is that this

is not a single mechanism but a complex results of several different mechanisms. cerebrospinal fluid (CSF) can leak from the Dural tear that can be formed during the surgery. calcification of the dura can cause it to get stiff and in turn prevent the brain to expand, which in turn would cause the epidural dead space to develop. One other important mechanism that has been postulated that is pneumocephalus, which is the air trapped inside the brain , this air bubble in the extradural space acts as a predecessor to inflammatory response , which leads to exudate forming up . Aoki<sup>6</sup> stressed the significance of air bubble(pneumocephalus) in acute epidural hematoma.



**Figure 1.** Post cranioplasty CT scan showing extradural collection and massive midline shift.

#### CONCLUSION

Post cranioplasty patient should be under observation and neurological deterioration should be kept in mind, early detection of fluid and blood collection can be life saving for the patient .surgical intervention is not always needed , can be managed conservatively.

#### REFERENCES

1. Agner C, Dujovny M, Gaviria M: Neurocognitive assessment before and after cranioplasty. *Acta Neurochir (Wien)* 144:1033-1040, 2002.
2. Chang V, Hartzfeld P, Langlois M, Mahmood A, Seyfried D. Outcomes of cranial repair after craniectomy. *J Neurosurg* 112: 1120-1124, 2010.

3. Lee JW, Kim JH, Kang HI, Moon BG, Lee SJ, Kim JS : Epidural fluid collection after cranioplasty : fate and predictive factors. J Korean Neurosurg Soc 50: 231-234, 2011.
4. Moreira-Gonzalez A, Jackson IT, Miyawaki T, Barakat K, DiNick V. Clinical outcome in cranioplasty: critical review in long-term follow-up. J Craniofac Surg 14:144-153, 2003.
5. Park JS, Lee KS, Shim JJ, Yoon SM, Choi WR, Doh JW. Large defect may cause infectious complications in cranioplasty. J Korean Neurosurg Soc 42:89-91, 2007.
6. Aoki N: Air in acute epidural hematoma. Report of two cases. J Neurosurg 65: 555-556, 1986.
7. Epidural Fluid Collection after Cranioplasty: Fate and Predictive Factors Jung Won Lee, M.D., Jae Hoon Kim, M.D., Hee in Kang, M.D., Byung Gwan Moon, M.D., Seung Jin Lee, M.D., Joo Seung Kim, M.D, J Korean Neurosurg Soc 50: 231-234, 2011.
8. Tiesler Blos, Vera (2003). Cranial Surgery in Ancient Mesoamerica. Mesoweb.



# Manifestation and outcome analysis of chronic subdural haemorrhage patient in a tertiary care centre

Sajag Gupta

UPUMS SAIFAI, INDIA

## ABSTRACT

**Aim.** To evaluate the results of surgical care in chronic subdural haemorrhage patients.

**Design of the study.** Prospective

**Study location.** Department of Neurosurgery, tertiary care centre

**Methodology.** The study comprised 65 individuals of both genders with chronic subdural haemorrhages who were above the age of 18. Patients' full medical histories, including age, gender, and place of residence, were documented. The Markwalder Grading System was used to classify patients. A CT scan was performed pre and post-surgery, as well as at the time of discharge. The Glasgow coma scale was used to record the outcomes.

**Results.** There were 57 (87.6%) male individuals and 8 (12.3%) female individuals. majority of them presented with headache (61.5%), followed by extremity weakness/paresis (52.3%). As Per, Markwalder's grade on admission, 33 (50.7%) people were in grade 1, and 30 (46.1%) people were in grade 2. The clinical picture at discharge was evaluated according to the Markwalder grade: 52 people (80%) were grade 0, 2 people (3%) were grade 1, 9 people (13.8%) were grade 2

**Conclusion:** The major risk factors for the development of CSDH in our set-up are male sex, mild head trauma, old age and alcohol intake. CSDH is more common on the left side. majority of patients had duration of trauma in between 4-12 weeks Majority of patients had grade 1 on Markwalder grading on admission. The majority of patients had a Thickness of CSDH is 10-20 mm MLS in CSDH is 5-10 mm on admission. The symptoms that present most frequently are headache and motor weakness, and the result is related to the person's pre-operative neurological condition, G.C.S., and concurrent systemic disorders. Surgical procedures should be individualised according to the radiological characteristics of chronic subdural haematoma.

## INTRODUCTION

Chronic subdural haemorrhage (CSDH) is an encapsulated accumulation of old blood between the dura layer and arachnoid layer that is produced by bridging vein injuries. When the haemorrhage is more than 3 weeks old, it is called chronic subdural haemorrhage. It is a rather frequent illness, particularly among the elderly, had incidence ranging from 1.72 to 7.35 per 100,000 population with a male majority.<sup>1,2,3</sup> Co-morbidities are common in the elderly population and

## Keywords

chronic subdural  
haemorrhage,  
markwalder,  
Glasgow coma scale



Corresponding author:  
Sajag Gupta

UPUMS SAIFAI, India

sajag.gupta@yahoo.com

Scan to access the online version



can have an influence on immediate postoperative prognosis and life expectancy. Although the majority of persons have a history of minor trauma, certain instances may be the result of a coagulation abnormality, cerebral hypotension, or the use of anticoagulants and antiplatelet drugs. Headaches, disorientation, sleepiness, vomiting, and seizures are frequent symptoms. On examination, the persons has a low G.C.S. hemiparesis/hemiplegia, ocular palsy, and other neurological abnormalities. The diagnosis is generally verified by an NCCT scan of the head; however, MRI is recommended for improved imaging of multiloculated membranes and septations.

This ailment is usually treated surgically; however, some people have been treated conservatively with steroids. Steroids have been used to treat mild headaches and individuals who are unable to undergo surgery <sup>4</sup>.

**MATERIALS AND METHODS**

**Study population**

The study population consists of all > 18 year age group peoples who underwent surgery for chronic subdural haemorrhage in Department of Neurosurgery in tertiary care hospital.

**Study design**

The study design was prospective cohort study. Peoples who fulfilled inclusion criteria, post-operative outcomes were assessed at discharge, then at one month and three months afterwards in the neurosurgery OPD.

**Sample size**

Sample size (n) was calculated using the underlying formulae for prevalence.

$$n = \frac{Z^2 * (p) * (1-p)}{m^2}$$

Where:

Where n = sample size required

p = estimated prevalence found between 1.72 to 7.35/100,000 based on previous publication

m = Precision with which to measure the prevalence of the study (margin of error) (confidence interval), set at ± 5%

The Z value is 1.96 for 95% confidence interval.

Substituting in the above formula, the sample size ranged 26-104 and since the local incidence was unknown, we used the average sample size of (26+104)/2=65.

**Inclusion criteria**

Peoples with chronic subdural haemorrhages who were over the age of 18 based on radiological and clinical presentation and had undergone surgery.

**Methodology**

A comprehensive history, medical checkup, and CT/MRI scans were performed to confirm the diagnosis. Before enrolling individuals in the study, written informed consent was obtained in / English/ Hindi/ from the patient, or from a blood relative, preferably a first degree relative, in persons who were comatose or unable to give consent, after explaining both surgical options, i.e. BHC and mini craniotomy as required by CT scan or MRI findings. The ethical approval was obtained from the Institutional Ethics Committee.

A detailed history was gathered, including a history of head injury during the past 3 months or before, use of anticoagulants or antiplatelet medicines within the last 1 week, drinking, a history of high blood pressure, and hyperglycaemias. The persons had a thorough clinical and neurological evaluation, including the Glasgow Coma Scale (GCS) and Markwalder's Grading Scale (MGS).

Routine blood tests and coagulation tests were performed. Any coagulation disorders corrections and blood or blood product transfusion requirements were reported. CT information on the following parameters: maximal haemorrhages thickness, density, midline shift, and septation in the haematoma. When a CT brain scan revealed a possibility of septation, an MRI was performed.

All persons were placed under general anaesthesia (G.A) or monitored anaesthesia care (MAC) for surgical evacuation. At one month and three months following discharge, all persons were seen in the outpatient clinic. The Markwalder Neurological Grading System for CSDH was utilised to compare preoperative, postoperative, one-month, and three-month outcomes.

A total of 65 persons were hospitalised to the tertiary care centre's Neurosurgery Department. Every individual got surgery for chronic subdural haemorrhage.

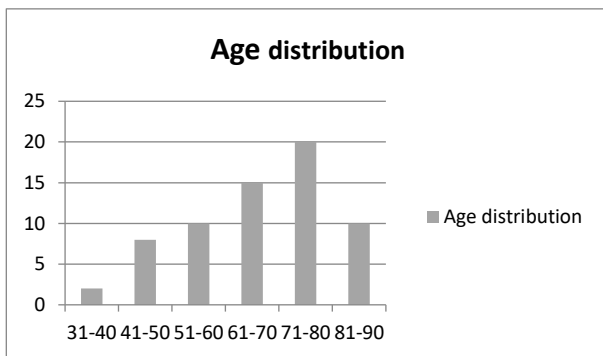
## RESULTS

### Demographic Characteristics

Out of 65 patients, the majority of them were in 8th decade, (71-80) (30.7%) followed by 7th decade (61-70) (23.0%) (Table-1, Figure- 1). There was male gender predominance with preponderance of the cases being men (87.6%), which shows to male to female ratio 9:1 (Table -2).

**Table 1.** Age distribution.

Age (years)	No of Patients (%)
31-40	2 (3.0%)
41-50	8 (12.3%)
51-60	10 (15.3%)
61-70	15 (23.0%)
71-80	20 (30.7%)
81-90	10 (15.3%)
Total	65 (100%)



**Figure 1.** Bar chart showing age distribution.

**Table 2.** Gender distribution.

Sex	No of Patients (%)
Male	57 (87.6%)
Female	8 (12.3%)
Total	65 (100%)

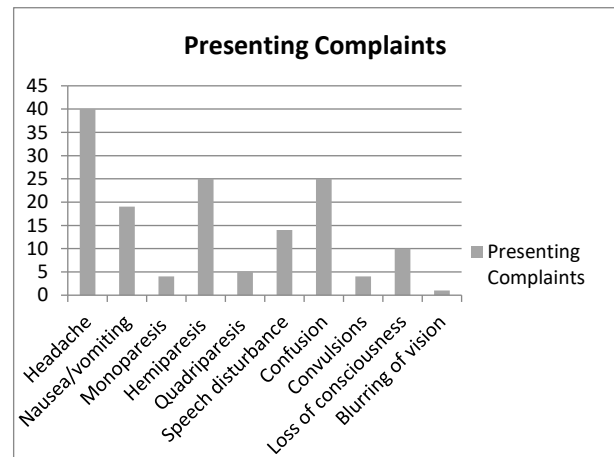
### Clinical presentation

Out of 65 patients, majority of them presented with headache (61.5%), followed by extremity weakness/paresis (52.3%). Other presenting

symptoms were confusion (38.4%), nausea/vomiting (29.2%), speech disturbance (21.5%), convulsions (6.15%), blurring of vision (1.5%) respectively (Table-3, Figure- 2).

**Table 3:** Presenting complaints.

Presenting Complaints	No of Patients (%)
Headache	40 (61.5%)
Nausea/vomiting	19 (29.2%)
Monoparesis	4 (6.15%)
Hemiparesis	25 (38.4%)
Quadriparesis	5 (7.6%)
Speech disturbance	14 (21.5%)
Confusion	25 (38.4%)
Convulsions	4 (6.15%)
Loss of consciousness	10 (15.3%)
Blurring of vision	1 (1.5%)



**Figure 2.** Bar chart showing incidence of symptoms.

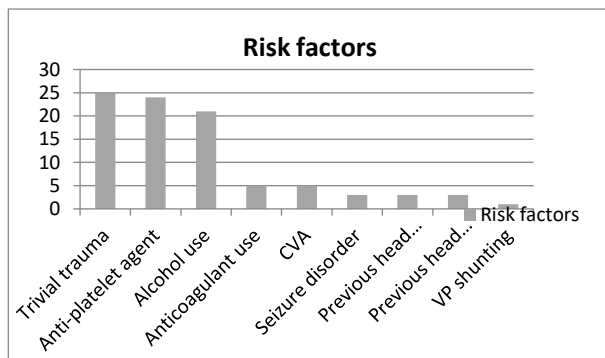
### Risk factors

Trauma was most common risk factors with (38.4%) history of trivial trauma in recent past, followed by, use of anti platelet agents (36.9 %) in which aspirin and clopidogrel were mainly used, history of alcohol consumption (32.3%), anticoagulant use, mainly warfarin was found in (7.6%) range of INR from 2 to

5.5, history of CVA (7.6%), history of seizure disorder (4.6%), previous head surgery (4.6%), previous severe head injury (4.6%) and VP shunting (1.5%) respectively (Table- 4, Figure-3).

**Table 4:** Risk factors

Risk factors	No of patients (%)
Trivial trauma	25 (38.4%)
Anti-platelet agent	24 (36.9%)
Alcohol use	21 (32.3%)
Anticoagulant use	5 (7.6%)
CVA	5 (7.6%)
Seizure disorder	3 (4.6%)
Previous head surgery	3 (4.6%)
Previous head injury	3 (4.6%)
VP shunting	1 (1.5%)



**Figure 3.** Bar chart risk factors for CSDH.

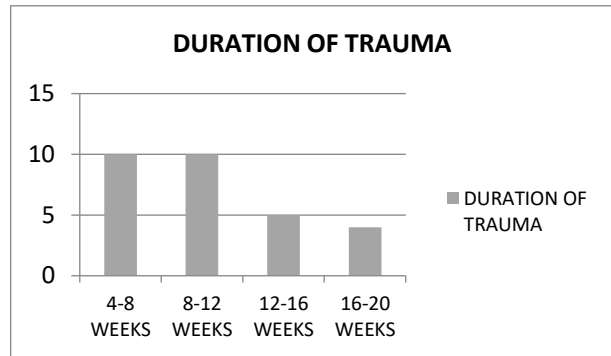
**Duration of trauma**

Duration of trauma in chronic subdural haematoma varied from the 4-20 weeks, majority of patients had duration of trauma in between 4-12 weeks (Table- 5, Figure- 4).

**Table 5:** Duration of trauma in chronic subdural haematoma (n= 26).

Duration (in weeks)	No of patients (%)
4-8	10 (38.4%)
8-12	10 (38.4%)

12-16	5 (19.2%)
16-20	1 (0.38%)



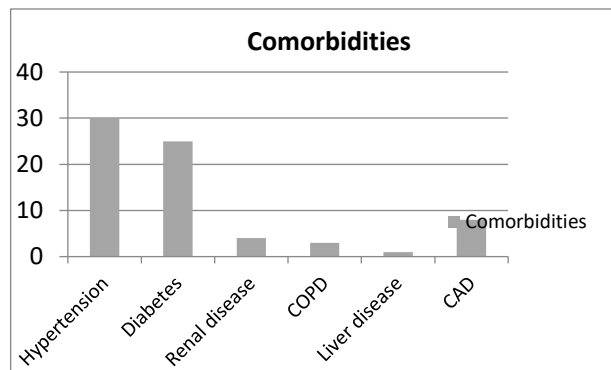
**Figure 4.** Bar chart duration of trauma in CSDH.

**Comorbidities**

In terms of comorbidities, hypertension was commonest at 46.1% followed by Diabetes was 38.4%, CAD was 12.3%, Renal disease was 6.1%, Chronic obstructive pulmonary disease was 4.6% and lastly Liver disease was 1.5% (Table-6, Figure 5).

**Table 6:** Comorbidities

Co-morbidities	No of patients (%)
Hypertension	30 (46.1%)
Diabetes	25 (38.4%)
Renal disease	4 (6.1%)
COPD	3 (4.6%)
Liver disease	1 (1.5%)
CAD	8 (12.3%)



**Figure 5.** Bar chart showing co morbidities in CSDH.

### On admission neurology

Mostly patients 54 (83.0%) had Glasgow Coma Scale of 15/15, 9 patients were in altered sensorium and 2 patients were in coma. (Table- 7)

**Table 7:** On admission GCS score.

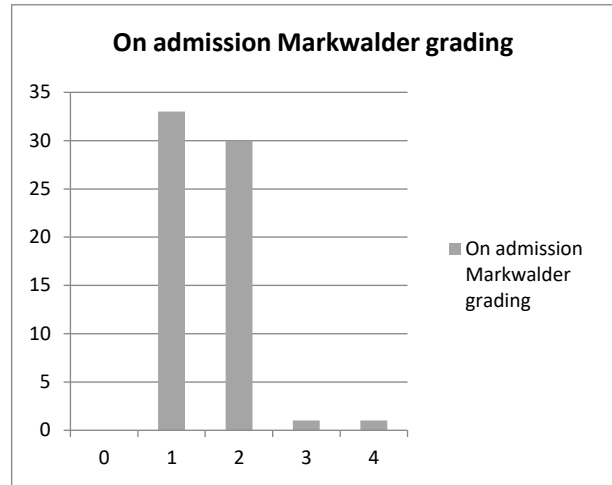
Glasgow Coma Scale Score	No. of patients (%)
9	2 (3.0%)
10	0
11	0
12	0
13	3 (4.6%)
14	6 (9.2%)
15	54 (83.0%)

### Markwalder grading on admission

As Per, Markwalder grade- no patient (0%) was grade 0, 33 (50.7%) patients were in grade 1, 30 (46.1%) patients were in grade 2, 1(1.5%) patient was in grade 3 and 1 (1.5%) patient was in grade 4 (Table- 8, Figure - 6).

**Table 8:** Markwalder grading on admission .

Grade	Markwalder grading	No. of patients (%)
0	Neurologically normal	0
1	Alert & oriented, mild symptoms such as headache or mild neurological deficit such as reflex asymmetry.	33 (50.7%)
2	Drowsy or disoriented or variable neurological deficit such as hemiparesis.	30 (46.1%)
3	Stuporous responding appropriately to noxious stimuli, several focal signs such as hemiplegia.	1 (1.5%)
4	Comatose with absent motor response to painful stimuli, decerebrate or decorticate posturing.	1 (1.5%)



**Figure 6.** Bar diagram showing Markwalder grading on admission.

### Radiological investigation

Out of 65 patients, pre-op investigation, only CT scan was done in 17 patients and only MRI was done in 24 patients. In rest 24 patients both CT and MRI were done. (Table 9)

**Table 9.** Radiological investigation done in CSDH patients.

Radiological investigation	No of patients (%)
CT scan only	17 (26.1%)
MRI only	24 (36.9%)
CT +MRI	24 (36.9%)

**SITE:** The haematoma was fronto-parietal in 35 patients, fronto-parietotemporal in 24 patients, fronto-parietotempocipital in 5 patients respectively. (Table 10).

**Table 10.** Site of CSDH.

Site	No. of patients (%)
Frontoparietal	36 (55.3%)
Frontoparietotemporal	24 (36.9%)
Frontoparietotempocipital	5 (7.6%)

**Laterality of CSDH** In 27 patients (41.5%), the haematoma was left sided, 20 patients (30.7%) it was right-sided, and 18 patients (27.6%) showed bilateral chronic haematomas. (Table 11).

**Table 11:** Laterality of CSDH

Side	No. of patients (%)
Right CSDH	20(30.7%)
Left CSDH	27(41.5%)
Bilateral CSDH	18(27.6%)

**Type of anaesthesia**

60 patients were operated under GA, but 5 patients were operated under MAC. All of them were burr hole evacuation surgery. (Table 12)

**Table 12.** Type of anaesthesia.

Type of anaesthesia	No. of patients (%)
GA	60 (92.3%)
MAC	5 (7.6%)
TOTAL	65 (100%)

**Type of surgery**

47 patients had unilateral CSDH, out of which 27 patients had burr hole evacuation surgery and 20 patients had minicraniotomy. In all patients of burr hole evacuation surgery double burr holes were made, none of the patient had single burr hole made. In 2 patients minicraniotomy with endoscopic membranectomy was done. 18 patients had bilateral CSDH out of which 15 patients had bilateral burr hole evacuation surgery and 3 patients had bilateral minicraniotomy, none of the patients had one side burr hole evacuation surgery and one side minicraniotomy. So, total numbers of procedures done were 83. Total number of Burr hole evacuations performed were 57 and Total number of mini craniotomies performed were 26. (Table-13) In patients, preoperative CT scans showed membranes within the haematoma cavity, which made craniotomy the treatment of choice. MRI was done to confirm the membranes in CSDH. Burr hole was done in rest of the patients where there were no membranes.

**Table 13.** Type of surgery.

	Burr hole 27	Mini craniotomy 20
Right Unilateral	10	10
Left Unilateral	17	10

	Bilateral burr hole ( no of procedure)	Bilateral mini craniotomy ( no of procedure)	One side burr hole evacuation surgery and one side minicraniotomy
<b>Bilateral CSDH</b>	<b>15 (Total 30)</b>	<b>3 (Total 6)</b>	<b>0</b>

**Thickness of CSDH**

Total numbers of unilateral CSDH were 47 and bilateral CSDH were 18, so total numbers of thickness measured were 83. CSDH thickness were 5-10 mm were 2, 10-20 mm were 53, 20-30 mm were 27, and more than 30 mm was 1 respectively. (Table 14)

**Table 14.** Thickness of CSDH.

Thickness	No. of CSDH 83
5-10 mm	2
10-20 mm	53
20-30 mm	27
>30 mm	1

**MLS of CSDH**

MLS in CSDH patients were 0-5 mm in 13 patients ,5-10 mm in 36 patients, 10-15 mm in 14 patients, and more than 15 mm in 2 patients. (Table -15, Figure-21,22)

**Table 15.** MLS of CSDH

MLS	No. of patients (%)
0-5 mm	13 (20%)
5-10 mm	36 (55.3%)
10-15 mm	14 (21.5%)
>15 mm	2 (3.0%)

**Post op scans**

All patients underwent surgery in post op scan showed some pneumocephalus and residual extra-axial fluid. (Figure- 23, 24, 25) 20 patients got resolution of CSDH within 1 month, 29 patients got resolution of CSDH with in 2 month and 7 patients took up to 3 month to resolve. One of our patient took 150 days for resolution of hematoma (Figure-26).7 patients (10.7%) had recurrence and one patient expired. (Table -16)

**Table 16.** Time taken to resolve CSDH.

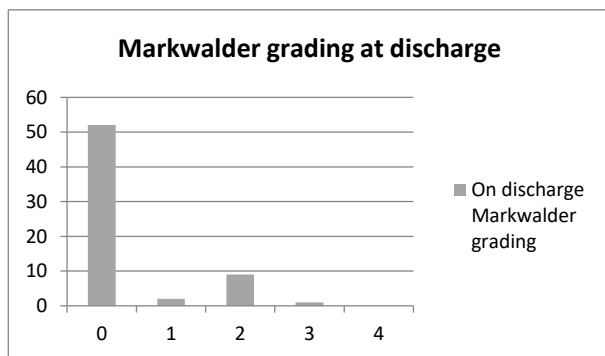
Time taken to resolve CSDH	No. of patients (%)
<1 month	20 (30.7%)
1-2 month	29 (44.6%)
2-3 month	07 (10.7%)
>3month	01 (1.5%)

### Markwalder grading on discharge

The clinical picture at discharge was evaluated according to the Markwalder grade: 52 patients (80%) were grade 0, 2 patients (3%) grade 1, 9 patients (13.8%) grade 2, one patient (1.5%) grade 3 and no patient (0%) grade 4. (Table-7, Figure- 7)

**Table 17.** Markwalder grading on discharge.

Grade	Markwalder grade	No of Patients (%)
0	Neurologically normal	52 (80%)
1.	Alert & oriented, mild symptoms such as headache or mild neurological deficit such as reflex asymmetry.	2 (3.0%)
2.	Drowsy or disoriented or variable neurological deficit such as hemiparesis.	9 (13.8%)
3.	Stuporous responding appropriately to noxious stimuli; several focal signs such as hemiplegia.	1 (1.5%)
4.	Comatose with absent motor response to painful stimuli, decerebrate or decorticate posturing.	0 (0%)

**Figure 7.** Bar diagram showing Markwalder grading at discharge.

In analysis of pre operative neurological condition, it was found that 54 (83.0%) of the patients, had G.C.S score of 15/15. 33 of our patients presented with the MGS 1 and 30 were MGS 2 at the time of admission and 1 patient was having MGS of 3 and 1 patient of MGS 4. On post operative outcome, we had 52 patients (80%) with favourable outcome of MGS 0 at discharge. Improvement up to MGS 0 in MGS progressed to 63(96.9%) patient at 3 months follow up. One patient expired and one had mild neurological deficit at 3 month follow up.

### DISCUSSION

Chronic subdural haemorrhage is a common condition in neurosurgical field. Because it mostly affects the elderly, its prevalence will continue to rise as the population ages.<sup>2-4</sup> The reason for its high prevalence in the elderly is due to growing brain atrophy and the corresponding higher baseline stretch on the bridging veins, which renders them more sensitive to minor head injury, which is one of the most common causes of CSDH.<sup>5,6,7</sup> Due to the advancing age of patients and their co-morbid conditions it is critical to find a surgical method which offers optimal efficiency in reducing possibly fatal haemorrhage-related mortality and morbidity while also demonstrating a lower recurrence rate and surgical complications. However, it is as gentle and safe as feasible for the often comorbid individuals.<sup>8</sup>

The mean age of the research sample in our study was 66.73 years, which matches to other published research<sup>9,10,11,12,13</sup>. Male preponderance (87.6%) is seen in our study, which is consistent with prior studies.<sup>9,11,14,15</sup>

In our research, the majority of patients (61.5%) reported with headaches, followed by extremities weakness/paresis (52.3%). Other presenting symptoms included confusion (38.4%), nausea/vomiting (29.2%), speech disorder (21.5%), convulsions (6.15%), and blurring of vision (1.5%). The most common presenting symptoms, according to Santarius et al.,<sup>16</sup> are abnormal gait (57%), impaired consciousness (35%), hemiparesis (35%), and headache (18%). The most common presentation in the elderly (50%-70%) according to the literature was disturbed mental condition.<sup>17,18</sup> In one series, hemiparesis was identified in 58% of the

patients.<sup>19</sup> The incidence of headache ranged from 14% to 50% in several series.<sup>20,21</sup> Epilepsy is an uncommon presentation and has been described as an early symptom in up to 6% of patients.<sup>19</sup> Our research is also consistent with this. The incidence of CSDH with transient neurologic impairments ranges from 1% to 12%.<sup>22</sup> Our patient population lacked transient neurologic impairments characteristics.

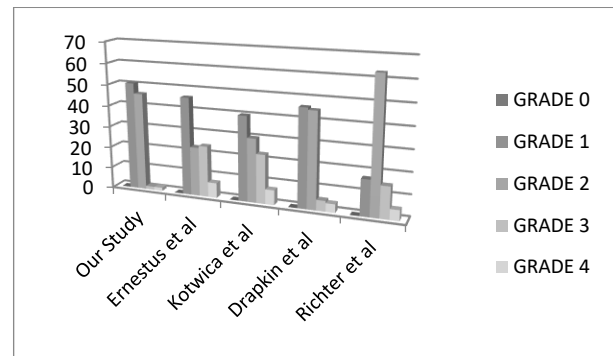
The most major risk factor (40%), trivial trauma, was generally minor and old. The average time from trauma to the start of manifestations was 7.9 weeks (4 to 20 weeks). Given the recall bias and the fact that major patients arrived with uncertainty, the history of trauma might be considerably greater; trauma was old and often minor, particularly in the elderly. Minor head injury has been proven as a common factor by several series<sup>23,24,25,26,27</sup>. Stroobandt<sup>28</sup> discovered it to be as high as 80% in his research of 100 patients, compared to aspirin (16%), coagulopathy (6%), and alcoholism (11%). Trauma is the most common cause of death in our research, accounting for 40%, followed by antiplatelet agents (35.3%) and alcohol intake (32%). Despite the fact that anticoagulant usage was 7.6% and antiplatelet agent use was 35.3% in our research. Because of the liver damage, chronic alcoholic consumption produces coagulation malfunction.<sup>29</sup> Other risk factors for CSDH include renal illness, dialysis therapy, liver failure, epilepsy, and chemotherapeutic drugs, as mentioned by Sim et al.<sup>29</sup>.

INR was abnormal in 5 patients, which was resolved before surgery, however 1 patient (1.5%) had persistent coagulopathy, which recurred after surgery. Despite the fact that certain studies have found a significant incidence of coagulation disorder ranging from 10% to 42%<sup>30,31</sup>. Berwerts and Webster discovered<sup>32</sup> oral anticoagulant medications, raised blood pressure, INR >4.5, and anticoagulation duration to be significant risk factors in cerebral haemorrhage in a multivariate study.

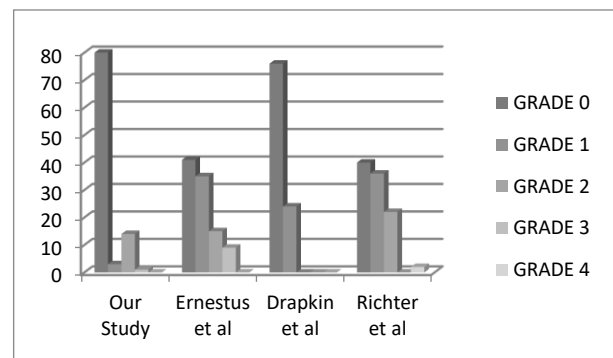
Another observation in this study, was the correlation between the surgical procedure and required anaesthesia with data suggesting that BHC are more likely to be performed under MAC while craniotomies require more invasive and physically demanding general anaesthesia. Patients over 60 years of age, who usually have concomitant diseases and risk factors and may not be fit enough to undergo general anaesthesia. Though we did not

find any such limitation but we preferred MAC if patient had multiple co morbidity and patient is going to be operated BHC if radiologically suitable for procedure.

The Markwalder grading was used to assess the neurological picture upon admission and at discharge. A comparison of our work with the series of Ernestus et al<sup>33</sup>, Kotwica et al<sup>34</sup>, Drapkin et al<sup>35</sup>, and Richter et al<sup>36</sup>, who all used the same technique of evaluation, shows that our study fits the results of Drapkin et al<sup>35</sup>. (Figures 8, 9)



**Figure 8.** Markwalder grading on admission comparison of present research with other research.



**Figure 9.** Markwalder grading at discharge comparison of present research with other research.

In summary, Considering increasing age and the often presence comorbidities of elderly neurosurgical patients who have to be treated for chronic subdural hematoma, the less invasive and physically demanding BHC should be the preferred if possible, but mini-craniotomy is required in patients in whom membranes are found within haematoma or there are solid blood clots, therefore there is need to individualize the surgical procedure on case to case basis depending on imaging study

characteristic of CSDH and to reduce recurrence and improve outcome.

### CONCLUSION

- The major risk factors for development of CSDH in our set up are male sex, mild head trauma, old age and alcohol intake.
- CSDH is more common on left side and fronto parietal convexity.
- majority of patients had duration of trauma in between 4-12 weeks.
- Majority of patients had grade 1 on **Markwalder grading on admission.**
- Majority of patients had Thickness of CSDH is 10-20 mm MLS in CSDH is 5-10 mm on admission
- The most frequent presentation signs are headache and motor weakness.
- High blood pressure, diabetes, antiplatelet medication, and anticoagulants are all potential risk factors.
- The patient's pre-operative neurology condition, G.C .S, and related systemic disorders all have an impact on the end result.
- Surgical procedure should be individualised according to radiological characteristics of chronic subdural haematoma.

### Recommendations

- Fall prevention in old age.
- Promotion of health and education helps in eradication of habits like alcohol consumption.
- Irrational use of antiplatelet agent should be relooked in view of high prevalence of CSDH in these patients.
- We should individualize the surgical procedure on case to case basis depending on imaging study characteristic to improve outcome reduce morbidity.

### REFERENCES

1. Scott G, Terbrugge K, Melancon D, Belanger G. Evaluation of the age of SDH by CT Scan. *J Neurosurg* 1977;47:311-315.
2. Foelholm R, Waltimo O. Epidemiology of chronic subdural haematoma. *Acta Neurochir.* 1975;32:67
3. Karibe H, Kameyama M, Kawase M, Hirano T, Kawaguchi T, Tominaga T. Epidemiology of chronic subdural hematoma. *No Shinkei Geka.* 2011; 39:1149-53.
4. Shapely J, Glancz LJ, Brenan PM. Chronic subdural haematoma in the elderly: Is it time for a new Paradigm in management. *Current Geriatrics Reports* 2002, 5: 71-77
5. Ducruet AF, Grobelny BT, Zacharia BE, Hickman ZL, Derosa PL, Anderson K, et al. The surgical management of chronic subdural hematoma. *Neurosurg Rev.* 2012;35:155-69
6. Stanicic M, Lund-Johansen M, Mahesparan R. Treatment of chronic subdural hematoma by burr-hole craniostomy in adults: influence of some factors on postoperative recurrence. *Acta Neurochir (Wien)* 2005; 147(12):1249-56; discussion 1256-7.
7. Sim Y, Min K, Lee M, Kim Y, Kim D. Recent changes in risk factors of chronic subdural hematoma. *J Korean Neurosurg Soc* 2012; 52(3):234-9.
8. Krieg SM, Aldinger F, Stoffel M, Meyer B, Kreutzer J. Minimally invasive decompression of chronic subdural haematomas using hollow screws: efficacy and safety in a consecutive series of 320 cases. *Acta Neurochir* 2012; 154(4):699-705.
9. Mondorf Y, Abu-Owaimer M, Gaab MR, Oertel JM. Chronic subdural hematoma: Craniotomy versus burr hole trepanation. *Br J Neurosurg.* 2009; 23:612-6.
10. Gelabert-González M, Iglesias-Pais M, García-Allut A, Martínez-Rumbo R. Chronic subdural haematoma: surgical treatment and outcome in 1000 cases. *Clin Neurol Neuro-surg* 2005; 107(3):223-9.
11. Muzii VF, Bistazzoni S, Zalaffi A, Carangelo B, Mariottini A, Palma L. Chronic subdural hematoma: comparison of two surgical techniques: Preliminary results of a prospective randomized study 2005; 49(2):41-7
12. Ahmed MA. Chronic subdural haematoma in Sudanese patients. Clinical Fellowship thesis, Sudan Medical Specialization Board, October 2002
13. Sakho Y, Kabre A, Badiane SB, Gueye M. Chronic SDH of adults in Senegal: A propos of 188 cases. *Dakar Med J* 1991; 36(2): 94-104.
14. Sreedharan PS, Rakesh S, Sajeev S, Pavithran K, Thomas M. Chronic SDH with spontaneous resolution: Rare manifestation of ITP. *J Assoc Physicians India* 2000; 48(4): 432-4
15. Salahaddin T. Management of chronic SDH: A review of 23 cases. *J PMA J Pak Med Assoc* 1996 Feb; 46(2): 32-33.
16. Santarius T, Kirkpatrick PJ, Ganesan D, Chia H L, Jalloh I, Smielewski P, et al. Use of drains versus no drains after burr-hole evacuation of chronic subdural haematoma: A randomised controlled trial. *Lancet.* 2009;374:1067-73
17. Potter JF, Fruin AH. Chronic subdural haematoma—"the great imitator". *Geriatrics* 1977; 32:61-6.
18. Cameron MM. Chronic subdural haematoma: a review of 114 cases. *J Neurol Neurosurg Psychiatry* 1978; 41:834-9.
19. Luxon LM, Harrison MJG. Chronic subdural haematoma. *Q J Med* 1979; 189:43-53.
20. Lesoin F, Destee A, Jomin M, et al. Quadriparesis as an unusual manifestation of chronic subdural haematoma. *J Neurol Neurosurg Psychiatry* 1983;46:783-5
21. Fogelholm R, Heiskanen O, Waltimo O. Chronic subdural haematoma in adults; influence of patient's age on symptoms, signs, and thickness of hematoma. *J Neurosurg* 1975;42:43-6
22. Moster ML, Johnston DE, Reinmuth OM. Chronic

- subdural haematoma with transient neurologic deficits: a review of 15 cases. *Ann Neurol* 1983;14:539-42
23. Mondorf Y, Abu-Owaimer M, Gaab MR, Oertel JM. Chronic subdural hematoma: Craniotomy versus burr hole trepanation. *Br J Neurosurg.* 2009; 23:612-6.
  24. Mori K, Maeda M. Surgical treatment of chronic subdural hematoma in 500 consecutive cases: Clinical characteristics, surgical outcome, complications, and recurrence rate. *Neurol Med Chir (Tokyo)* 2001;41:371-81
  25. Jennett B, Teasdale G. CSDH in management of Head injuries, *Contemporary Neurology series.* Davies, Philadelphia, 1983; 184-187
  26. Hamilton M.G, Frizzell J.B, Tranmer, The role of craniotomy reevaluated. *Neurosurgery.* 1993;33:67-72
  27. Ramzi Hadani M, Spielgeleemann R. Continuous irrigation-drainage of the subdural space for treatment of CSDH. A prospective clinical trial. *Acta neuro Chir(Wien)* 1993;120:40-23
  28. G. Stroobandt, P. Fransen, C. Thauvoy, and E. Menard. Pathogenetic Factors in Chronic Subdural Haematoma and Causes of Recurrence After Drainage. *Acta Neurochir (Wien)* 1995; 137:6-14
  29. Sim Y, Min K, Lee M, Kim Y, Kim D. Recent changes in risk factors of chronic subdural hematoma. *J Korean Neurosurg Soc* 2012; 52(3):234-9.
  30. Raymond et al. Aspirin as a risk factor for haemorrhage in patient with head injury. *Neurosurg review.* 1992 15:21-29
  31. Weir B, Gordon P: Factors affecting coagulation, fibrinolysis in CSDH fluid collection. *J Neurosurg.* 1983; 58:242-245
  32. Berwaerts J, Webster J: Analysis of risk factors involved in oral anticoagulation-related intracranial haemorrhage. *QJ Med.* 2000;93:513-521
  33. Ernestus R-I, Beldzinski P, Lanfermann H, Klug N. Chronic subdural hematoma: Surgical treatment and outcome in 104 patients. *Surg Neurol* 1997; 48:220-5.
  34. Kotwica Z, Brzezinski J. Chronic subdural haematoma treated by burr holes and closed system drainage: Personal experience in 131 patients. *Br J Neurosurg* 1991; 5:461-5.
  35. Drapkin AJ. Chronic subdural hematoma: Pathophysiological basis for treatment. *Br J Neurosurg* 1991; 5:467-73.
  36. Richter H P, Klein H J, Schäfer M. Chronic subdural hematomas treated by enlarged burr-hole craniotomy and closed system drainage. Retrospective study of 120 patients. *Acta Neurochir (Wien)* 1984; 71:179-88.



# An unusual case of choroid plexus papilloma in infancy. Diagnostic and management challenges

Abhishek Kumar, Raj Kumar

Ram Manohar Lohia Institute of Medical Sciences, Lucknow, INDIA

## ABSTRACT

Choroid plexus papillomas (CPPs) are rare tumours having bimodal distribution. Pediatric CPPs are commonly present in the supratentorial compartment and most commonly located in the lateral ventricle and usually present at 16-18 months. In the present case authors illustrate an atypical case of CPP in a 6-month-old infant who was admitted to our hospital after a trivial fall with no features of hydrocephalus or raised intracranial pressure. Quick surgery keeping in mind the Maximal allowable blood loss (MABL) and low physiological reserve of infants with quick decompression of cysts and devascularizing the tumor from all around and removing the tumor in toto plays a key role in operating the pediatric tumor. MRI features play a key role in preoperative diagnosis; nevertheless, it is difficult to distinguish atypical CPT from other lesions. Surgical resection is the cornerstone of this treatment.

## INTRODUCTION

Choroid plexus tumors (CPTs) infrequently occur with an intracranial derivation from the epithelium of the ventricular system's choroid plexus and are of neuroectodermal origin<sup>1</sup>. In adults, CPTs represent 0.3%–0.6% of all brain tumors, whereas in children, they comprise between 2% and 5%<sup>3</sup>. The World Health Organization (WHO) categorizes CPTs as choroid plexus papillomas (CPPs, WHO I), atypical choroid plexus papillomas (ACPPs, WHO II), and choroid plexus carcinomas (CPC WHO III)<sup>6</sup>.

CPTs normally occur in areas with a choroid plexus, usually in the ventricular system<sup>2,3</sup>. These tumors are more common in supratentorial locations, including the lateral ventricles among children and the fourth ventricle in the elderly<sup>7,8</sup>. The usual presentation is associated with increased intracranial pressure due to hydrocephalus secondary to CSF overproduction and/or obstruction of CSF flow. There are several causes of hydrocephalus, including direct mechanical obstruction of the flow of cerebrospinal fluid (CSF), hemorrhagic blockage of arachnoid granulations, and excessive production of CSF<sup>4,5</sup>.

The median age of diagnosis is 18 months in the pediatric population<sup>14</sup>. In 1927, Walter Dandy managed to perform the initial successful removal of pediatric CPT from the left lateral ventricle of a

## Keywords

choroid plexus papilloma,  
lateral ventricle,  
maximal allowable blood loss



Corresponding author:  
Abhishek Kumar

Ram Manohar Lohia Institute of  
Medical Sciences,  
Lucknow, India

kumar.abhishek595@gmail.com

Scan to access the online version



14-year-old girl, whereas Van Wagenen did the same in an infant aged 3 months<sup>15</sup>. Surgical extirpation is the best therapeutic option and significantly curtails the possibility of regrowth if gross total resection (GTR) is performed<sup>16</sup>. A majority of children who undergo treatment for these tumors are often very young; hence, their physiological reserve and maximal allowable blood loss (MABL) are low; hence, the need for meticulous surgery, especially from experienced neurosurgeons, becomes paramount. Profuse bleeding during surgery may be life-threatening among children with plexus tumors because CPTs are highly vascular and most children treated for plexus tumors are very young with low minimal allowable blood loss (MABL). Hence, it is crucial that feeding arteries are identified soon enough to reduce blood loss<sup>17</sup>.

## CASE REPORT

### History and physical examination

This 6-month-old male infant, born out of a non-consanguineous marriage, presented to our institute with a minor head injury after falling off the bed and accidentally hitting his head. He was taken to a nearby hospital for evaluation. The prenatal and antenatal periods were uneventful, and the child had a normal vaginal delivery with a normal cry immediately after birth. Before admission, the child had a history of episodes of excessive inconsolable crying for the past 2 months. The child was taking normal feeds and had attained normal developmental milestones as per age. At the time of examination, the child was active and playful. The anterior fontanelle was open and lax, and the head circumference (HC) was 44 cm (50<sup>th</sup> centile). There was scalp swelling in the left parietal region with no skin discoloration or underlying fracture. He underwent computerized tomography (CT) scan of the brain (Fig 1) and was referred to our center because of a brain tumor. There were no features suggestive of increased intracranial pressure (ICP) such as papilledema.

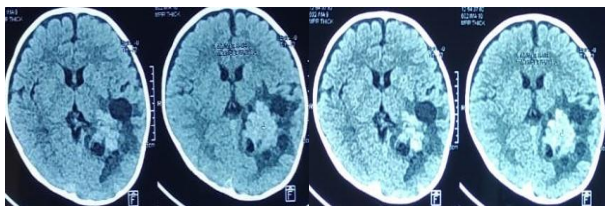


Fig 1: Pre-operative CT brain of the infant showing a lobulated hyperdense lesion in left parietal lobe, solid cystic in consistence and solid part enhancing on contrast with no evidence of hydrocephalus or mass effect.

### Imaging findings

CT scan showed a large lobulated, hyperdense solid cystic lesion located in the left parieto-occipital region, with the solid part of the tumor enhancing on contrast (Fig. 1). On MRI, the lobulated solid cystic lesion of size approximately 55mm x 45 mm x 50 mm, located in the left parieto-occipital region. The lesion solid part was isointense on both T1 and T2. The cystic part of the tumor was hypointense on T1 and hyperintense on T2 and bright contrast enhancement of the solid part was observed. There was no evidence of hydrocephalus, mass effect, or peri-lesional edema.

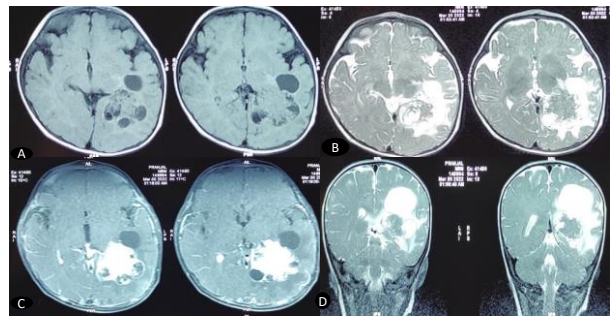


Fig. 2. Pre-operative T1 axial (A), T2 axial (B), T1 axial contrast (C), T2 Coronal brain MR imaging of infant showing lobulated, solid cystic lesion in left parietal lobe with solid part brilliant contrast enhancement. No evidence of hydrocephalus, mass effect, perilesional edema, mass effect.

### Management and surgical technique

Our working diagnosis was ependymoma, and other differential diagnosis was high grade glioma and choroid plexus papilloma (CPP). The absence of hydrocephalus blinded us to CPP diagnosis. We planned for surgical excision. The patient was given general anesthesia and positioned prone, and left-sided parieto-occipital osteoplastic craniotomy was performed. The brain was lax on removing the bone flap. Subsequently, the duramater was opened and transcortical-transventricular approach was taken to the left occipital horn. The tumor was soft to firm in consistency, yellowish-white in color, and appeared cauliflower-like. Considering that pediatric patients have very low MABL and these tumors are pose significant bleeding risks, microscopic circumferential dissection, coagulation, and disconnection of tumor vascular supply from the lateral ventricle was performed.

The feeders from the choroidal arteries were identified under a high-magnification microscope and coagulated. The stalks of the attachments were coagulated and cut using bipolar electrocautery. Gross total excision of the tumor was achieved, and

the lesion was excised in-toto (Fig 3). Intraoperative blood loss was 200 cc, which did not exceed the calculated MABL of 250 cc, and no blood transfusion was required. The choroid plexus of the left lateral ventricle was clearly visible and coagulated. Ventricular drain placed and maintained for 3 days in the left lateral ventricle.

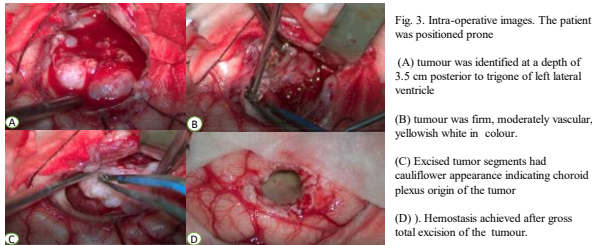


Fig. 3. Intra-operative images. The patient was positioned prone

(A) tumour was identified at a depth of 3.5 cm posterior to trigone of left lateral ventricle

(B) tumour was firm, moderately vascular, yellowish white in colour.

(C) Excised tumor segments had cauliflower appearance indicating choroid plexus origin of the tumor

(D) Hemostasis achieved after gross total excision of the tumour.

### Post operative course

Post-operative period was uneventful. Post-operative CT scan of the brain showed complete excision of the lesion (Fig 4). Patient was extubated next day. The baby was discharged on post-operative day 7 and he was taking oral feeds. The infant was doing well at the last follow-up at 6 months post-operation. A post-operative Magnetic Resonance Imaging (MRI) brain revealed no evidence of residual lesion.

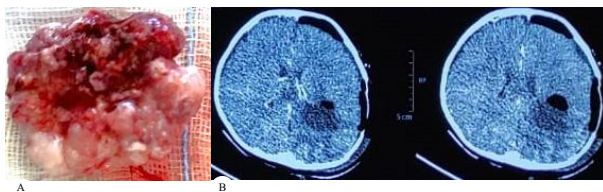


Fig 4 Tumor segments had cauliflower appearance indicating choroid plexus origin of the tumor(A) Post operative CT scan showing gross total excision of the tumor

### DISCUSSION

CPP accounts for 2-4% of all pediatric brain tumors and is of neuroectodermal origin<sup>14,16</sup>. In the pediatric population, CPTs usually occur in the lateral ventricles. They commonly appear as papillary or lobulated intraventricular lesions and are usually associated with hydrocephalus<sup>8,9</sup>. The usual presentation of CPTs in the pediatric population is an enlarged head size associated with clinical presentation suggestive of increased intracranial pressure. In our case, the child had no clinical features suggestive of raised ICP (anterior fontanelle was normal and absent papilledema) and presented with mild head injury after falling from bed. After imaging evaluation, there was a diagnostic dilemma between ependymoma and choroid plexus

papilloma. CPT is usually associated with hydrocephalus in 40% of patients<sup>12,14</sup>, and some articles reported that moderate or severe hydrocephalus was seen in almost all cases<sup>2,6,8,14,17</sup> which was absent in this case. Note that hydrocephalus was not associated in this study, which is one of the most significant differences between it and typical CPT. The absence of hydrocephalus as a marker poses a diagnostic dilemma for radiologists and neurosurgeons.

These tumors can be challenging to treat because they most often present in childhood and are highly vascular. During surgery due to low MABL, excessive blood loss can become life threatening. Physiological reserve in infants is very low; hence, attention should be paid to blood loss right from the skin incision and subcutaneous tissue dissection because by the time we reach the tumor, blood loss should not limit us in excision of the tumor.

Experienced neurosurgeons who have a lot of experience in operating pediatric cases are required along with an expert team of neuroanaesthetists who are experts in managing pediatric neurosurgery cases. In the pediatric age group and especially infants, one major challenge is the risk of hypothermia, which has to be tackled from the beginning by the neuroanaesthesia team. In planning surgery, the goal should be quick timed decompression of the cyst. The approach is to remove the tumor in toto by devascularizing the tumor from all around. The cyst proves to be beneficial and helps in the surgery as quick decompression of the tumor helps in reaching and excision of the tumor. Early identification of feeding arteries is therefore important to reduce blood loss. Gross total resection (GTR) has become the standard of care for avoiding tumor recurrence.

### CONCLUSION

In contrast to typical lesions, hydrocephalus was not found in our case of CPP, which posed a major diagnostic challenge. A team of experienced pediatric neurosurgeons and a neuroanaesthesia team experienced in managing pediatric neurosurgery cases is required to operate a pediatric choroid plexus tumor. Quick surgery keeping in mind the MABL and low physiological reserve of infants with quick decompression of cyst and devascularizing the tumor from all around and removing the tumor in toto plays a key role in

operating the pediatric tumor. MRI features play a key role in preoperative diagnosis; nevertheless, it is difficult to distinguish atypical CPT from other lesions. Our study showed that atypical CPT cases were treatable by radical excision. Surgical resection is the cornerstone of this treatment.

## REFERENCES

1. Safaee M, Clark AJ, Bloch O, Oh MC, Singh A, Auguste KI, Gupta N, McDermott MW, Aghi MK, Berger MS, Parsa AT. Surgical outcomes in choroid plexus papillomas: an institutional experience. *Journal of neuro-oncology*. 2013 May;113:117-25.
2. Dudley RW, Torok MR, Gallegos D, Liu AK, Handler MH, Hankinson TC. Pediatric choroid plexus tumors: epidemiology, treatments, and outcome analysis on 202 children from the SEER database. *Journal of neuro-oncology*. 2015 Jan;121:201-7.
3. Boström A, Boström JP, Von Lehe M, Kandenwein JA, Schramm J, Simon M. Surgical treatment of choroid plexus tumors. *Acta neurochirurgica*. 2011 Feb;153:371-6.
4. Bettegowda C, Adogwa O, Mehta V, Chaichana KL, Weingart J, Carson BS, Jallo GI, Ahn ES. Treatment of choroid plexus tumors: a 20-year single institutional experience. *Journal of Neurosurgery: Pediatrics*. 2012 Nov 1;10(5):398-405.
5. Buxton N, Punt J. Choroid plexus papilloma producing symptoms by secretion of cerebrospinal fluid. *Pediatric neurosurgery*. 1997 Mar 7;27(2):108-11.
6. Louis DN, Ohgaki H, Wiestler OD, Cavenee WK, Burger PC, Jouvet A, Scheithauer BW, Kleihues P. The 2007 WHO classification of tumours of the central nervous system. *Acta neuropathologica*. 2007 Aug;114:97-109.
7. McCall T, Binning M, Blumenthal DT, Jensen RL. Variations of disseminated choroid plexus papilloma: 2 case reports and a review of the literature. *Surgical neurology*. 2006 Jul 1; 66(1):62-7.
8. Nagib MG, O'Fallon MT. Lateral ventricle choroid plexus papilloma in childhood: management and complications. *Surgical neurology*. 2000 Nov 1;54(5):366-72.
9. Bian LG, Sun QF, Wu HC, Jiang H, Sun YH, Shen JK. Primary choroid plexus papilloma in the pituitary fossa: case report and literature review. *Acta neurochirurgica*. 2011 Apr;153:851-7.
10. Duke BJ, Kindt GW, Breeze RE. Pineal region choroid plexus papilloma treated with stereotactic radiosurgery: a case study. *Computer Aided Surgery: Official Journal of the International Society for Computer Aided Surgery (ISCAS)*. 1997;2(2):135-8.
11. Pillai A, Rajeev K, Chandi S, Unnikrishnan M. Intrinsic brainstem choroid plexus papilloma: Case report. *Journal of neurosurgery*. 2004 Jun 1;100(6):1076-8.
12. Greene RC. Extraventricular and intra-cerebellar papilloma of the choroid plexus. *Journal of Neuropathology & Experimental Neurology*. 1951 Apr 1;10(2):204-7.
13. Carter AB, Price DL, Tucci KA, Lewis GK, Mewborne J, Singh HK. Choroid plexus carcinoma presenting as an intraparenchymal mass: Case report. *Journal of neurosurgery*. 2001 Dec 1;95(6):1040-4.
14. Jaiswal S, Vij M, Mehrotra A, Kumar B, Nair A, Jaiswal A, Behari S, Jain V. Choroid plexus tumors: A clinico-pathological and neuro-radiological study of 23 cases. *Asian Journal of Neurosurgery*. 2013 Mar;8(01):29-35.
15. Menon G, Nair SN, Baldawa SS, Rao RB, Krishnakumar KP, Gopalakrishnan CV. Choroid plexus tumors: an institutional series of 25 patients. *Neurology India*. 2010 May 1;58(3):429-35.
16. Strojjan P, Popovic M, Surlan K, Jereb B. Choroid plexus tumors: a review of 28-year experience. *Neoplasma*. 2004 Jan 1;51(4):306-12.
17. Kumar R, Singh S. Childhood choroid plexus papillomas: operative complications. *Child's Nervous System*. 2005 Feb;21:138-43.



# Schwannomatosis. A rare case report

Younes Dehneh<sup>1,2</sup>, Hamid Khay<sup>1,2</sup>, Mohamed Khouali<sup>1,2</sup>,  
Moufid Faycel<sup>3</sup>

<sup>1</sup> Neurosurgery Department, University Hospital Centre Mohammed VIth Oujda, MOROCCO

<sup>2</sup> Faculty of Medicine and Pharmacy, University Mohammed Premier Oujda, MOROCCO

<sup>3</sup> Neurosurgery Department, Hôpital Universitaire International Mohammed VI Bouskoura MOROCCO

## ABSTRACT

Schwannomatosis is characterized by a predisposition to develop multiple schwannomas and rarely meningiomas. People with schwannomatosis are most commonly present between the second and fourth decades of life. The most common feature is localized or diffuse pain or an asymptomatic mass. Schwannomas most commonly involve peripheral and spinal nerves. We report the case of a woman who was initially referred to our department because of suspected neurofibromatosis type 2.

## INTRODUCTION

Schwannomatosis is distinguished by its inherent tendency to manifest multiple schwannomas in affected individuals [1]. It could be familial or sporadic [2]. Differentiating schwannomatosis from both type 1 and particularly type 2 neurofibromatosis is crucial due to their distinct genetic origins, disease progression, and associated impacts.

## CASE REPORT

A 47-year-old female patient with a history of hypothyroidism. She was first operated on for uterine fibroids at age 34. Six years later, she underwent surgery for pleural schwannoma. She came to our emergency department because of back pain for 10 years and radicular pain in both legs. Her clinical examination revealed strabismus and multiple achromatic spots. MRI of the spine showed intra dural extra medullary lesion at L3, iso-intense in T1WI, with contrast enhancement after gadolinium injection and hyper-signal in T2WI (Figure 1). Cranial MRI was normal. The patient underwent surgery for spinal decompression and resection of the tumor. Anatomic pathologic examination revealed tumor composed of Schwann cells, which are seen as streams of elongated cells with tapering nuclei. Cellular organization shows loosely arranged cells with areas of vacuolation and

## Keywords

schwannomatosis,  
spine,  
schwannoma,  
neurofibromatosis



Corresponding author:  
Younes Dehneh

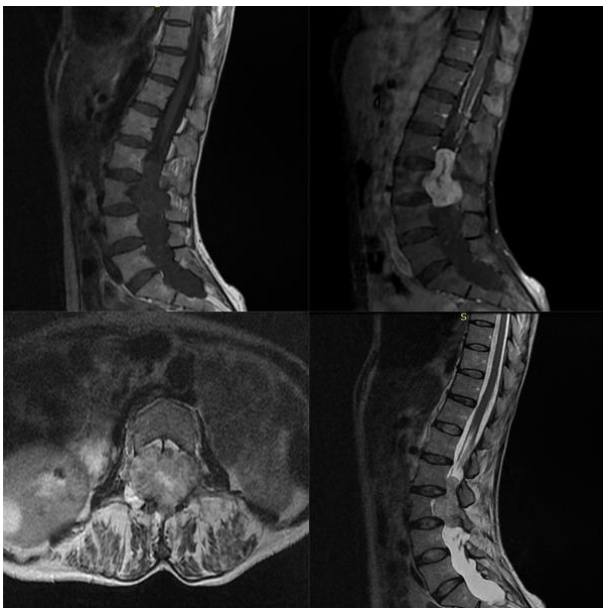
Faculty of Medicine and Pharmacy,  
University Mohammed Premier  
Oujda, Morocco

younesjo1@gmail.com

Scan to access the online version



lipidisation (Antoni B). Mitoses are sparse and the appearances are of a conventional Schwannoma (WHO grade I). (Figure 2). Postoperative evolution showed no back pain. Genetic counseling was performed, and the karyotype showed no gene mutations. There was no family history of neurofibromatosis. The diagnosis of schwannomatosis was made according to Kehrer-Sawatzki criteria (more than two non-intradermal schwannomas and no evidence of bilateral vestibular schwannomas) One year later, the patient is asymptomatic.

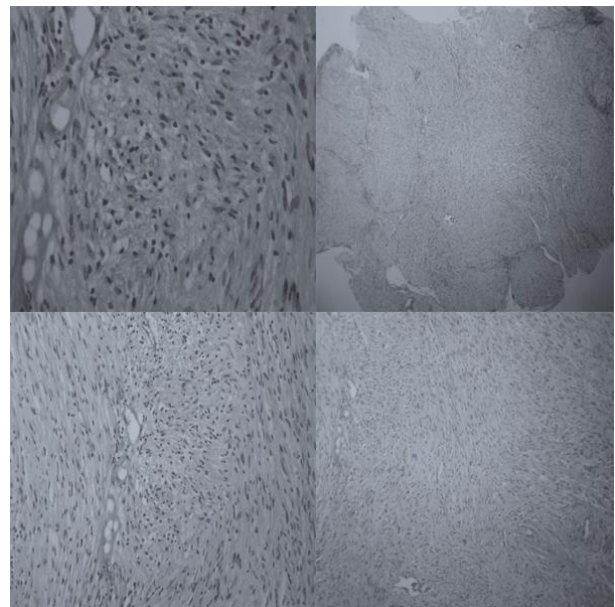


**Figure 1.** MRI of the spine showed intra dural extra medullary tumor at L3, iso-intense in T1WI, with contrast enhancement after gadolinium injection and hyper-signal in T2WI.

## DISCUSSION

The diagnosis of schwannomatosis is determined by either clinical criteria alone or a combination of molecular and clinical criteria [3]. The majority of schwannomatosis cases are considered sporadic, meaning they arise in individuals without a family history of the disorder [2]. While some individuals with sporadic schwannomatosis exhibit mutations in the SMARCB1 or LZTR1 gene, the underlying cause of the condition remains unknown in other cases [4]. Schwannomas mainly affect peripheral nerves (90%) and spinal nerves (75%). Within spinal nerves, the lumbar spine is most commonly involved [5]. Involvement of cranial nerves is rare, but the trigeminal nerve is the most commonly affected cranial nerve in such cases. In symptomatic

schwannomas causing pain or neurological deficits, surgical intervention is recommended. The principles of surgical resection of peripheral nerve tumors in such cases are similar to those for removal of sporadic nerve sheath tumors. Although there is a theoretical concern that radiation exposure may increase the risk of malignant transformation, this association has not been demonstrated in individuals with schwannomatosis [6]. The exact role of radiation therapy in this context is not yet clear and requires further investigation.



**Figure 2.** Histological examination reveals a tumor composed of Schwann cells, which are seen as streams of elongated cells with tapering nuclei. Cellular organization shows loosely arranged cells with areas of vacuolation and lipidisation (Antoni B). Mitoses are sparse and the appearances are of a conventional Schwannoma (WHO grade I).

## CONCLUSION

Schwannomatosis can be diagnosed based on clinical or combined molecular and clinical criteria. Surgical intervention is recommended for symptomatic schwannomas causing pain or neurological deficits, following similar principles as sporadic nerve sheath tumors.

## REFERENCES

1. Dhamija R, Plotkin S, Asthagiri A. Schwannomatosis Summary Genetic counseling Suggestive Findings. GeneReviews. 2018.
2. Patil S, Perry A, MacCollin M, Dong S, Betensky RA, Yeh TH, et al. Immunohistochemical analysis supports a role

- for INI1/SMARCB1 in hereditary forms of schwannomas, but not in solitary, sporadic schwannomas. *Brain Pathol.* 2008;18(4):517–9.
3. Kehrer-Sawatzki H, Farschtschi S, Mautner VF, Cooper DN. The molecular pathogenesis of schwannomatosis, a paradigm for the co-involvement of multiple tumour suppressor genes in tumorigenesis. *Hum Genet.* 2017;136(2):129–48.
  4. Plotkin SR, Messiaen L, Legius E, Pancza P, Avery RA, Blakeley JO, et al. Updated diagnostic criteria and nomenclature for neurofibromatosis type 2 and schwannomatosis: An international consensus recommendation. *Genet Med.* 2022;24(9):1967–77.
  5. Merker VL, Esparza S, Smith MJ, Stemmer-Rachamimov A, Plotkin SR. Clinical Features of Schwannomatosis: A Retrospective Analysis of 87 Patients. *Oncologist.* 2012;17(10):1317–22.
  6. Evans DGR, Birch JM, Ramsden RT, Sharif S, Baser ME. Malignant transformation and new primary tumours after therapeutic radiation for benign disease: Substantial risks in certain tumour prone syndromes. *J Med Genet.* 2006;43(4):289–94.



# Skull base-cranio-facial trauma with dura mater repair after subsequent CSF leaking, diagnosis treatment and outcomes. A systematic review of the literature

Daniel Encarnacion<sup>1</sup>, Gennady Chmutin<sup>1</sup>,  
Bipin Chaurisia<sup>2</sup>

<sup>1</sup> Department of Neurosurgery of People of Friendship University, Moscow, RUSSIA

<sup>2</sup> Department of Neurosurgery, Bhawani Hospital and Research Centre, Birgunj, NEPAL

## ABSTRACT

**Background.** Since there is a chance of serious neurological consequences, it is critical to demonstrate the diagnosis and treatment of craniofacial trauma with aetiologies such as extreme sports, motor vehicle accidents, and social trauma. Examining the effectiveness of methods for restoring the Dura after severe craniofacial and skull-base damage made worse by rhinorrhea and CSF leakage is our goal.

**Methodology.** In accordance with PRISMA (Preferred Reporting Items for Systematic Reviews) criteria, a systematic review was conducted. The following search terms were used: "Articles criticizing the analysis of open penetrating craniofacial injury with concurrent CSF leakage from the cranial cavity were examined and included, and associated pathologies, especially severe trauma of the upper and middle regions of the anterior cranial fossa." Rayyan - Intelligent Systematic Review Software A thorough search was conducted in databases like ScienceDirect and PubMed/MEDLINE before employing IBM SPSS Statistics for Windows, Version 26.0 (Released 2020; IBM Corp., Armonk, New York, United States), and EXCEL criteria for the statistical analysis.

**Results.** A total of 1584 patients with cerebrospinal fluid leaks were included in this investigation. Additionally, 698 patients were shown in Table 1 figure 2 and 886 patients in Table 2, figure 3-4-5. Rhinorrhea following trauma to the base of the skull, or craniofacial, was corrected by transnasal endoscopy in most cases and extracranial approach or craniotomy in fewer cases.

**Conclusion.** A thorough analysis of the outcomes within the context of healthcare reveals: 1. The efficacy of polyoxybutyrate membrane-based multilayer D-plasty. 2. This evaluation clarified that the inferred further planning for surgical suture material development was made.

## Keywords

skull base fracture, cerebrospinal fluid (CSF), CSF fistula, subarachnoid haemorrhage, diagnosis and management of skull-base-craniofacial trauma, rupture of the dura mater



Corresponding author:  
Daniel Encarnacion

Department of Neurosurgery of  
People of Friendship University,  
Moscow, Russia

Danielencarnacion2280@gmail.com

Scan to access the online version



## INTRODUCTION

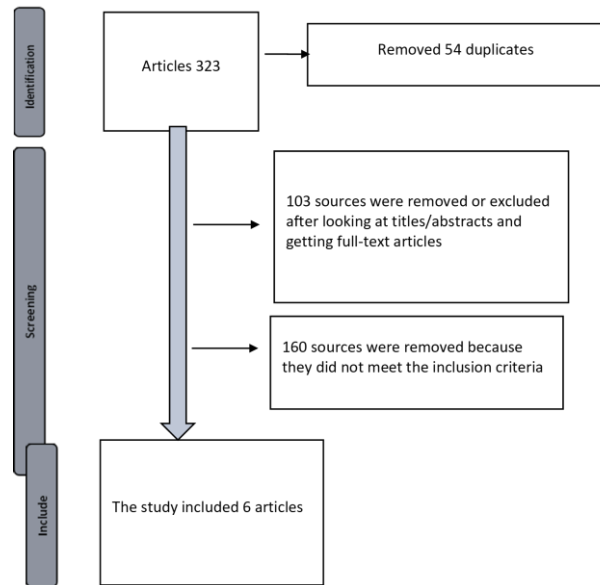
The dura mater (DM), which encircles the brain and spinal cord, is the outermost layer of the meninges. The DM protects a number of other brain membranes, such as the pia and arachnoid mater. According to trauma characteristics, it is clarified that 38% of patients with frontobasal fractures suffered an injury as a result of an accident (17% of the 38% were caused by falls from substantial heights). Ten percent of patients have an unknown cause for the damage. Three percent of patients had injuries from playing contact or severe sports. [1].

Walter E. Dandy reported the ideal transcranial procedure in 1926 after achieving excellent Dura mater restoration using a bifrontal craniotomy for access and an autograft of the fascia lata for remodeling. With this craniotomy technique, one can access the ethmoidal roof and cribriform plate. A wide craniotomy is necessary to reconstruct or repair sphenoid sinus damage caused by trauma. Retraction of the cerebrum is necessary to locate the lesion, and reconstruction may be accomplished after surgery. [2].

Preferred biological or synthetic materials include fascia lata, pedicled galea, and pericranial flaps; sealants, such as fibrin glue, are used to keep flaps in place for a few weeks. Despite documented success rates of approximately 32.8%, there were nevertheless reported failure rates. Some writers argue that it is better to use sutures to seal the dura mater away from the defect. Because there is a risk of serious neurological consequences, it is critical to diagnose and treat cases of craniofacial trauma with aetiologies based on motor vehicle accidents, physical abuse in society, or intense sports as soon as possible. [3]. When it comes to reducing neurological sequelae, medical and physiotherapy are ineffective and do not stabilize patients' circumstances as well. In short, surgical intervention can be used to treat pathological abnormalities of the spine, spinal cord, and brain that are acquired, congenital, or the result of trauma. [4]. Surgical procedures such as resection of benign and malignant neoplasms, cerebrovascular interventions including aneurysmal repair, spinal procedures involving compression fractures, functional neurosurgery including epileptogenic zone obliteration seizures, and stroke interventions like thrombectomy are all performed by a sufficient number of departments within the neurosurgical

and spinal surgical disciplines. All of these activities demand the utmost qualification and accuracy throughout execution. [5]

The aims to demonstrate the efficacy of the techniques designed to restore the Dura after severe skull base injury exacerbated by rhinorrhea and cerebrospinal fluid (CSF) leakage.



**Figure 1.** PRISMA Flowchart Traumatic Tear of the Dura Mater and Subsequent CSF Leak after Skull base, Craniofacial trauma.

### Search strategy and mesh terms

Mesh terms related to Traumatic Tear of the Dura Mater, Craniofacial and Subsequent CSF Leak were incorporated into the search strategy. The selection criteria emphasized English-language journals published through 2024. Rigorous criteria were applied to ensure the relevance and quality of publications on various types of Traumatic Tear of the Dura Mater, Craniofacial and CSF Leak Injuries with associated pathologies.

("Cerebrospinal Fluid Leak/cerebrospinal fluid"[Mesh] OR "Cerebrospinal Fluid Leak/classification"[Mesh] OR "Cerebrospinal Fluid Leak/complications"[Mesh] OR "Cerebrospinal Fluid Leak/diagnosis"[Mesh] OR "Cerebrospinal Fluid Leak/diagnostic imaging"[Mesh] OR "Cerebrospinal Fluid Leak/drug therapy"[Mesh] OR "Cerebrospinal Fluid Leak/epidemiology"[Mesh] OR "Cerebrospinal Fluid Leak/etiology"[Mesh] OR "Cerebrospinal Fluid Leak/history"[Mesh] OR "Cerebrospinal Fluid Leak/immunology"[Mesh] OR "Cerebrospinal Fluid

Leak/mortality"[Mesh] OR "Cerebrospinal Fluid Leak/pathology"[Mesh] OR "Cerebrospinal Fluid Leak/physiopathology"[Mesh] OR "Cerebrospinal Fluid Leak/prevention and control"[Mesh] OR "Cerebrospinal Fluid Leak/rehabilitation"[Mesh] OR "Cerebrospinal Fluid Leak/surgery"[Mesh] OR "Cerebrospinal Fluid Leak/therapy"[Mesh]

### Comprehensive search strategy keywords

In addition to the Mesh terms, keywords such as "Traumatic Dura mater ", "CSF Leak", "Skull trauma", "craniofacial trauma", "CSF Treatment and Outcomes.

### Inclusion criteria

- Age range: from 18 years to 75.
- Traumatic and spontaneous Cerebrospinal fluid leak at the base of the anterior skull.
- MRI Strategies in Skull Base Injuries after Skull Base Trauma with Suspected Cerebrospinal Fluid Leak.
- Spontaneous cerebrospinal fluid after trauma to the skull base of the middle fossa associated with otorrhea patients with skull base trauma after dural repair and with CSF control and with suspicion of meningitis.

### Exclusion criteria

Patients without cerebrospinal fluid leak after skull base trauma and without alteration of the Glasgow scale

Patients diagnosed by imaging without injured adjacent structures and without CSF leak patients under 18 years of age with craniofacial trauma but without injury to the entire length of the skull bas

### Data extraction and analysis

Standardized search methods and a careful review of pertinent research publications were used to obtain data from eligible studies. Obtaining comprehensive data on research on CSF leak injuries, craniofacial trauma, and traumatized tear of the dura mater with related diseases was part of the data gathering process. The authors' information, the parameters utilized for control in comparative studies of the injury, the adult population studied's demographics, and the specifics of the interventions were all considered. the sort of study and the year it was published.

### Analytical statistics

The data analysis was conducted using SPSS software version 26.0 from IBM Corporation located in Armonk, NY, USA. The results were presented as mean  $\pm$  standard deviation values. The fracture classification test Leford kinds 2-3 was applied. Using craniofacial-skull based injuries as the basis for statistical comparisons in CSF confirmation, a p value of less than 0.05 was deemed significant.

### RESULTS

A total of 1584 patients with cerebrospinal fluid leaks were included in this investigation. Additionally, 698 patients were shown in table 1 figure 2 and 886 patients in table 2, figure 3-4-5. Rhinorrhea following trauma to the base of the skull, or craniofacial, was corrected by transnasal endoscopy in most cases and extracranial approach or craniotomy in less cases.

In a research conducted from 2013 to 2022, FCL repair using hydroxyapatite cement was performed on 55 patients with a total of 60 faults and only 5 bilateral repairs. The favorable outcomes included 91.6% with 55 corrections. The tegmen tympani region was the most often repaired area. Acetazolamide was provided to these individuals prior to a second successful surgical intervention. In addition, five of these patients experienced headaches during the recovery phase, and three (5.4%) had VP shunts to lower intracranial pressure. 34 research publications covering the years 2000–2023 focused primarily on post-traumatic cerebrospinal fluid leaks, which are primarily the result of car accidents. where men made up 20% of the injured and women made up 80%. where bifrontal craniotomy and endonasal endoscopy were the most often used techniques, both of which produced excellent results following the intervention. It is more susceptible to longitudinal ruptures because a larger number of sheath components permit longitudinal orientation. Patients with upper and middle facial zone fractures involving the anterior cranial fossa were distributed as follows in terms of local cranial injuries. Twenty percent of patients experienced hemorrhage as a result of blood clots or accumulation in the intracranial subthecal space, while fifty-six percent of patients suffered contusion brain lesions. (Figure 1). [6,8,9, 10, 51]

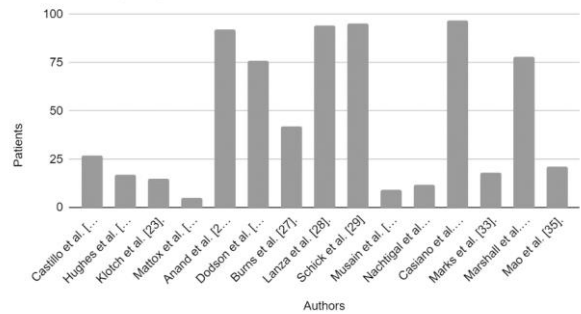
In an additional study conducted between 2012 and 2016, clinical data were gathered prospectively and retrospectively in a sequential manner to analyze potential risk factors for postoperative leakage of the multilayer base layer after a dural suture. utilizing a modification of the repair technique based on the degree of intraoperative CSF leak; among the 388 procedures performed, 10 EEAs were employed to account for 2.6% of the postoperative CSF leak cases following suture repair. [12, 13, 14, 15, 16, 52]. This study examines CFL leaks caused by trauma or spontaneous events that were successfully repaired endoscopically using a combination of endoscopic and trepanation techniques between 2015 and 2019. The study found no significant problems related to the patent frontal sinus trip. Most of the time. and just two individuals with synechiae development had a revision procedure. and there was a 22.7-month follow-up, with a range of 7-41 months. [17, 18,19, 20, 53].

**Table 1.** After trauma involving skull base defects, CSF is repaired using endoscopy in a transnasal, sphenoidal, and extracranial manner.

Authors	Type of Study	Year	Pt.	Management repair	Surgical approach	Follow up
Castillo et al. [21].	Clinical trial	1999	27	CSF rhinorrhea	Endonasal approach under optical guidance	24.7 months.
Hughes et al. [22].	Retrospective	1997	17	CSF leak/ Anosmia	primary endoscopic r	32 months
Klotch et al. [23].	Review	1998	15	CSF leak	Endoscopic approach /extracranial approach	
Mattox et al. [24].	Case report	1990	5	Cerebrospinal fluid leaks/ nasal cephaloceles.	Endoscopic sinus surgery	N/A
Anand et al. [25].	Research	1995	92	CSF leak /sinusitis	Functional endoscopic sinus surge	N/A
Dodson et al. [26].	Retrospective	1994	76	Recurrent CSF rhinorrhea	Functional endoscopic sinus surgery	3 to 43 months.
Burns et al. [27].	Retrospective	1996	42	Cerebrospinal fluid rhinorrhea / cephaloceles	Endoscopic procedure	5 to 68 months
Lanza et al. [28].	Retrospective	1996	94	CSF leakage	Endoscopic repair of skull base defects	2 to 57 months.
Schick et al. [29]	Retrospective	1997	95	Cerebrospinal fluid (CSF) rhinorrhea	Endoscopic	6 weeks to 4 Months-13 years

Musain et al. [30]	Retrospective	2003	9	CSF	Neuroendoscope	5 Months-8 years
Nachtigal et al. [31].	Retrospective	1999	12	Cerebrospinal fluid rhinorrhea	Intranasal endoscopic approach	N/A
Casiano et al. [32].	Retrospective	1999	97	CSF rhinorrhea	Endoscopic repair	29 months
Marks et al. [33].	Research	1998	18	CSF leaks	Endoscopic approach	N/A
Marshall et al. [34].	Research	2001	78	Sphenoid CSF leaks	transnasal endoscopic techniques	N/A
Mao et al. [35].	Research	2000	21	CSF leaks	Endoscopic approach	N/A

CSF Leaking Repair



**Figure 2.** Integrative Research Projects CSF repair following transnasal endoscopy technique, dura mismatch, and skull base trauma.

**Table 2.** Study group for repairing cerebrospinal fluid leaks using various methods and strategies.

Authors	Types of study	Year	Patients	Method	Techniques	Follow up	Journal
Chin et al. [36].	Retrospective	2003	37	CSF repair	Transnasal endoscopic closure of anterior skull base fistulas		Wiley
Castellon et al. [37].	Review	2001	31	CSF repair/ rhinorrhea	Endoscopic approach	1-year minimum follow-up.	American Journal of Rhinology & Allergy
Lund et al. [38].	Retrospective	2002	36	CSF repair/	Endoscopic	N/A	American Journal of

				rhinorrhea	approach		Rhinology & Allergy
McMains et al. [39].	Retrospective	2004	92	CSF rhinorrhea	Endoscopic approach	25 months	Wiley
Lee et al. [40].	Retrospective	2004	39	CSF rhinorrhea	Endoscopic approach	N/A	Wiley/The Laryngoscope
Lindstrom et al. [41].	Retrospective	2004	53	CSF rhinorrhea	Endoscopic approach	N/A	Wiley/The Laryngoscope
Husain et al. [42].	Retrospective	2003	9	CSF rhinorrhea	Endoscopic approach	5 months to 8 years	Skull Base: an Interdisciplinary Approach
Tosun et al. [43].	Retrospectively	2005	26	CSF repair	endonasal endoscopic approach/ craniotomy	N/A	Minimally Invasive Neurosurgery
Landeiro et al. [44].	Research	2004	10	CSF repair	Endoscopic repair - skull base defects	recurrence 14 months later	Minimally Invasive Neurosurgery
Briggs et al. [45].	Clinical trial	2004	52	CSF repair	Endoscopic approach	27 months	Clinical Neuroscience
Gendeh et al. [46].	Retrospective study	2005	16	sphenoid leak repair.	Endoscopic approach		J Laryngol Otol
Kirtane et al. [47].	Retrospective	2005	267	CSF repair	Transnasal endoscopic approach	N/A	Wiley/Otolaryngol Head Neck Surg
Araujo et al. [48].	Retrospective	2005	44	CSF rhinorrhea	Intranasal endoscopic approach	2 to 102 months	Braz J Otorhinolaryngol.

Bernal-Sprekelson et al. [49].	Retrospective chart study	2005	39	CSF repair	Endoscopic skull base surgery	65 months	Rhinology
Locatelli et al. [50].	Clinical trial	2006	135	CSF repair	endoscopic endonasal technique	N/A	Operative neurosurgery

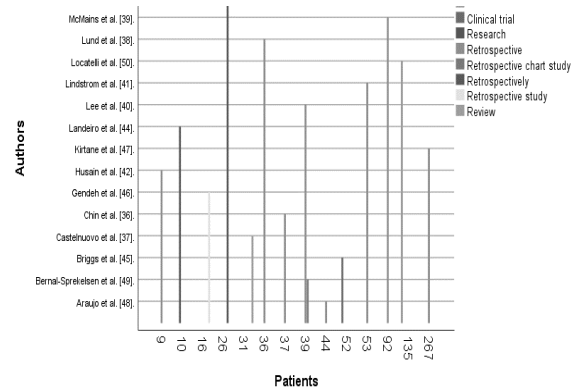


Figure 3. An illustration of the study group's strategy for fixing a cerebrospinal fluid leak using several methods and strategies.

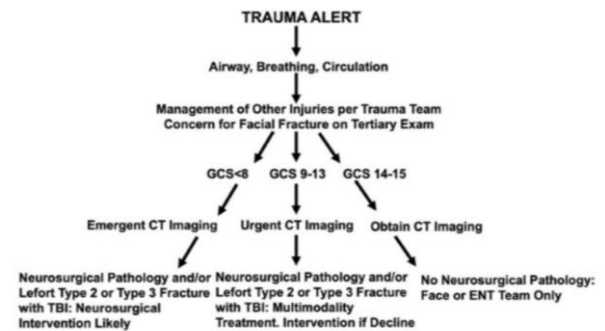
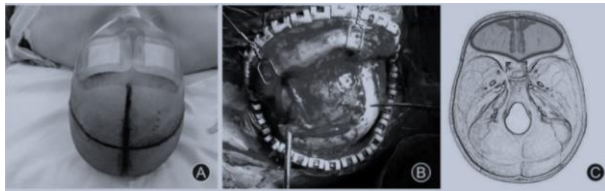


Figure 4. Flow diagram of the Management of craniofacial-skull base trauma per Leford types 2-3 fracture classification.

Fifteen per cent of patients had a haemorrhage in the cranial cavity, and nine per cent of people had an abnormal accumulation of air in the cranial cavity. Detailed data can be found in Figure 5.

Among patients with Frontobasal fractures, which are complicated by the outflow of cerebrospinal fluid (this aspect requires an operation), the frequency of detection of one-time injuries is as follows:

nasoethmoidal complex - seventeen and a half per cent; a paired cavity in the skull containing the eyeball with its appendages - 35.6 per cent; the lower wall of the orbit - 9 per cent; complex, including the zygomatic bone, lower, lateral wall of the orbit - 28.4 per cent; upper, lower jaw (8 per cent; 2.5 per cent).The status, which reflects the state of the nervous system, and cerebral, focal symptoms, was assessed by specialists after the initial intake, as well as in dynamics.[54]. In this case, focal symptoms can be divided into several categories.



**Figure 5.** The incision in the pericranial membrane and revision of the anterior base of the skull (A) enlarged bifrontal coronal incision originating from the zygomatic level to the contralateral zygomatic level. (B) A wide subgaleal dissection of the pericranial membrane was performed to the edge of the orbital rim to harvest the anterior-based vascularized graft. (C) The area outlined by the red line from the scan for lesions of the dura mater and skull base.

**Craniofacial symptoms due to the specifics of the injury**

**Focal symptoms associated with trauma to functionally important brain areas**

The first category of symptoms (discussed in the first table) involves dysfunction of the nerves extending from the brain. To localize the fistula, layer-by-layer scanning X-ray visualization of the CSF spaces of the brain was performed for thirty-six patients. It has been found in various places. This aspect could be observed at the same time with the problem of the bones of the skull, covering the brain from above, as well as from the sides; cranial base defect. [55].

**Table 3.** The frequency of localizations of CSF fistula in the anterior cranial fossa.

Craniobasal symptoms	%	CSF Fistula localization	%
Hyposmia, Anosmia	42	Ethmoid plate and roof of ethmoid cells	72,2
Oculomotor disorders	33	Frontal sinus	19,4

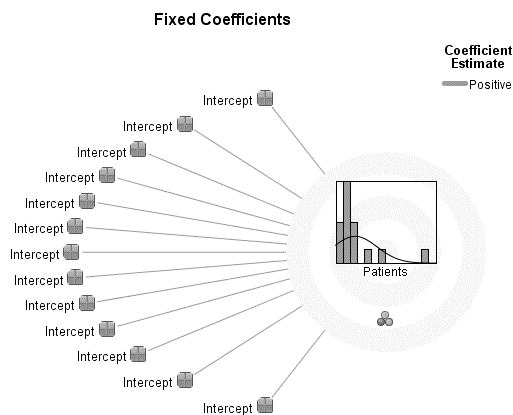
Violation of the function of the first and second branches of the V nerve	21	Site of the sphenoid bone	8,4
Violation of visual acuity (II pair of Craniocerebral insufficiency)	8		

The criteria for carrying out such activities are as follows: local outflow of Cerebrospinal fluid from the cranial cavity due to trauma to the bones of the base of the skull, the dura mater (it requires neurosurgical care); the same symptoms with significant defects in the bones of the vault, the cranial base. The most common symptom directly related to CSF leakage through defects in the dura mater is headache due to hypotension. Tension of the neck muscles, pathology of photophobia, and nausea. The pain in the head became greater in the upright position. Also, coupled with the indicated problems, fractures of the posterior walls of the paranasal sinuses located in the frontal bone can be observed. In case of complications of craniofacial damage, the goal of the operation is to remove contusion foci. surgeons treat a depressed fracture; eliminate the specifics of air accumulation inside the cranium. To neutralize the influence of excessive accumulation of fluid in the brain cells, bifrontal decompressive trepanation is performed. [56].

**A standard extended bifrontal craniotomy was performed at each procedure**

Typically, when harvesting biregular-type pericranial flaps, the bifrontal flap is lifted, and the frontal sinuses are remodeled by exposure without using the entire retractor in skull base trauma.when advancing to extradural dissection while using the microscope. The comminuted pieces of the anterior sinus should be removed with the utmost caution and using the high-speed craniotome to remove the sinus mucosa in the procedure up to the frontal nasal canal and careful look under the microscope, the dura mater was opened widely in terms of the location of the defect where it extended to the sellae tubercle, thus durotomy simplified the repair performed and through meticulous elevation through the durotomy to allow the dura to be moved at will without lacerating it. For the defects, a synthetic Dural graft was obtained as free tissue and

this material was used for the Dural graft. and so, in the flap, we place an additional piece in the epidural space. Thus, we can use clips on the sutures, securing the pericranial flap, and reducing its postoperative migration. note that 38% with an indication for urgent surgical intervention for a subgroup can be called local cranial pathology, in which brain dislocation is observed; skull fracture; other injuries. 6% development of meningitis. 5% were declared dead. [15, 57]



**Figure 6.** Covariance parametros Patients Subjects, Specifications for Residual and Radom Effects by CSF leaking.

### Dura defects are sutured (when possible) and then closed

Therefore, medical professionals conduct plastic surgery of the cranial base fistula when there is a major post-traumatic defect in the depression in the cranial base, which is home to the projecting frontal lobes of the brain, as well as liquorrhea. In this case, a vascularized flap is employed. The success rate for free flap materials is greater than 95% ( $p < 0.05$ ) when there is concurrent cerebrospinal fluid leakage from the cranial cavity as a result of bone trauma and a minor anterior fossa defect (less than 0.5 cm). According to the analysis under review, there is consensus regarding the general order of events while using surgical suture material, taking the defect's size into account. It was feasible to diversify the entire list of efficient materials utilized to seal the neurocranium in this instance since biodegradable material was used for Dural plasty. In another, he performed frontal sinus surgery to address chronic inflammation; he also removed the posterior wall of the sinus; he performed dural revision and D-plasty;

and he performed D-initial bone plasty of the cranial vault. A 2% patient developed meningitis. There have been two patient deaths. Twenty patients had a functional outcome based on a quality-of-life scale. Within a span of one to three days, 39% of patients had extradural dura mater, skull base plasty, and cranial trephination with soft tissue and bone flaps. In 1% of cases, a localized buildup of pus was discovered in the brain's material. [58].

### DISCUSSION

Traumatic brain injury, per craneocephalic trauma severe and skull base fracture, cause CSF fistula of cerebrospinal fluid is an important problematic aspect for study. By statistics increase in the frequency of severity of such injuries, without craniofacial reconstructions accord neurosurgeons and maxillofacial surgeons sometimes in combinations of orthopaedic department without the normal response of the situation can increase the level of not survival, in the section of surgery that considers the treatment of acute craniocerebral, spinal cord diseases - all these aspects affect the demand for a detailed analysis of the problem. Often there is a pathology in which the skull, brain, and skeleton of the face are damaged at a time. Such pathologies are difficult to treat. If we compare this symptomatology with damage to the lower facial region, damage to the brain at the same time as the upper, middle facial zone can lead to the not survival of the patient. There may be a symptom of CSF leakage from the cranial cavity; pathological accumulation of air in it. [59].

Various products were used in the D-plasty process of the canal through which the cerebrospinal fluid circulates (including a periosteal flap, adipose tissue, comb cue, and artificial dura mater). Perform a transcranial extradural plasty of the channels through which the cerebrospinal fluid circulates. The specialists used different materials. Often the plastic was 3-layer. Cerebrospinal fluid leakage in 8%. In 5% this symptomatology ceased as part of non-surgical medical care. external lumbar drainage was performed. The combination of materials "periosteal flap + Tachocomb 14% was used. When using the combination of materials "connective tissue + Tachocomb + artificial dura mater", no recurrence of the problems was observed either. Using a set of materials "connective tissue, cue comb, artificial dura", repeated manifestations of CSF leakage were

observed in 17%. To apply the clips, we use a series made up of a rotating piece of using approximately 8 cm curved that leaves a better field of vision. and each piece contains 25 clips. When using the "larger size" clips, their interior is about 3 mm for surgical use of each. but the most suitable would be 2-3 mm clips. [60].

In the last 10-15 years, there has been a general increase in the number of injuries to the lower parts of the cranial vault within the overall structure of the analyzed injuries by one and a half to two times. These trends are caused by a change in the specificity of aspects leading to injury (for example, indicators of damage due to road accidents, violent injuries), an improvement in the quality of the diagnostic activity of physicians, the creation and integration of advanced protocols for X-ray computed tomography of the skull and adjacent departments [5, 6, 60].

The Polyoxybutyrate membrane in some cases is used for dura mater defect repair. The material absorbs water and is durable. It does not need to be additionally fixed with surgical glue. Specialized multilayer plasty of cerebrospinal fluid outflow

channels was performed for nineteen per cent of patients to avoid many complications. The membrane made of polyoxybutyrate (thickness - two hundred microns) is suitable for plastics of the dura mater [7, 9]. [19]. [60]

Simultaneous use of adipose tissue, tacho-comb, and wide femoral fascia did not allow complex sealing of the cranial cavity. Complications in less than thirty-six per cent of cases. [20].

Multilayer D-plasty of the CSF fistula with the use of a specialized flap showed overall effectiveness in significant (more than five-tenths of a centimeter) post-traumatic defects of the ACF. There were no recurrences of pathology in the considered subgroup. Within small ACF defects (less than 0.5 cm), the success rate of free flap products exceeds ninety-five per cent ( $p < 0.05$ ) For patients with PBL, a significant post-traumatic defect for the cranial bases, to reduce the risk of complications, doctors use multilayer D-plasty. In the context of open injuries in the region of the lower parts of the cranial vault, when the periosteal flap is damaged, perifascial tissue can be used as an autograft. [11]. [60].

**Table 4.** Probability distribution: Multinomial Link function: Generalized logit a. Target: Patients

*Fixed Coefficients<sup>a</sup>*

Patients Model Term	Coefficient	Std. Error	t	Sig.	95% Confidence Interval		Exp (Coefficient)	95% Confidence Interval for Exp (Coefficient)	
					Lower	Upper		Lower	Upper
9 Intercept	2.137E-15	1.4142	.000	1.000	-6.085	6.085	1.000	.002	439.163
10 Intercept	2.026E-15	1.4142	.000	1.000	-6.085	6.085	1.000	.002	439.163
16 Intercept	2.026E-15	1.4142	.000	1.000	-6.085	6.085	1.000	.002	439.163
26 Intercept	2.082E-15	1.4142	.000	1.000	-6.085	6.085	1.000	.002	439.163
31 Intercept	2.137E-15	1.4142	.000	1.000	-6.085	6.085	1.000	.002	439.163
36 Intercept	2.137E-15	1.4142	.000	1.000	-6.085	6.085	1.000	.002	439.163
37 Intercept	2.137E-15	1.4142	.000	1.000	-6.085	6.085	1.000	.002	439.163
39 Intercept	.693	1.2247	.566	.628	-4.577	5.963	2.000	.010	388.697
44 Intercept	2.026E-15	1.4142	.000	1.000	-6.085	6.085	1.000	.002	439.163
52 Intercept	2.026E-15	1.4142	.000	1.000	-6.085	6.085	1.000	.002	439.163
53 Intercept	2.137E-15	1.4142	.000	1.000	-6.085	6.085	1.000	.002	439.163
92 Intercept	2.137E-15	1.4142	.000	1.000	-6.085	6.085	1.000	.002	439.163
135 Intercept	2.054E-15	1.4142	.000	1.000	-6.085	6.085	1.000	.002	439.163

## CONCLUSION

A comprehensive study of the results obtained in the framework of medical care indicates:

1. the effectiveness of multilayer D-plasty when using a polyoxybutyrate membrane.
2. As part of the analysis, information was obtained that it is possible to develop a strategy for the selection of surgical suture material.
3. In the context of the algorithm of actions, the factor of the magnitude of damage to the

cranial base is taken into consideration.

- The Incidence of skull base fracture provides round-the-clock findings of patients involved in these types of emergencies, pathologies, and complications that should be treated.

## REFERENCES

- Eichberg D, Ali S, Buttrick S. The use of dehydrated amniotic membrane allograft for augmentation of dural closure. 2018 Oct;32(5):516-520.doi: 10.1080/02688697.2018.1490943. <https://pubmed.ncbi.nlm.nih.gov/29989435/>
- Alekseev, D.E. Plasty of supratentorial defects of the dura mater during operations on the brain / D.E. Alekseev, D.V. Svistov, E.D. Alekseev, A.V. Fedorenkov // Proceedings of the XVI All-Russian Scientific and Practical Conference "Polenov Readings". - St. Petersburg: Man and his health, 2017. - S. 14-15. DOI: 10.17116/neiro20188205148/ <https://pubmed.ncbi.nlm.nih.gov/30412156/>
- Barthelemy E.J., Park K.B., Johnson W. Neurosurgery and sustainable development goals. (2018) 120:143-52. doi: 10.1016/j.wneu.2018.08.070.
- Bolton V, Aruparail N., Bundu I. et al. Technology Diffusion in Global Surgery. *Brj Surg.* (2019) 106:e34-43. doi:10.1002/bjs.11036.
- Díaz M, Martínez J, Guerrero P. et al. Techniques for Blunt-Trauma Fractures of the Skull-Base With Cerebrospinal-Fluid Leaks. 2021 Oct 1;32(7):2500-2507. doi: 10.1097/SCS.0000000000007932.
- Prosser J, Vender J, Solares C. et al. Traumatic Cerebrospinal-Fluid Leaks; 2011 Aug;44(4):857-73, vii.doi: 10.1016/j.otc.2011.06.007
- Dempsey R.J., Buckley N.A. Education-based solutions to the global-burden. (2020) 140: e1-6. doi: 10.1016/j.wneu.2020.01.057.
- Phang S, Whitehouse K, Lee L. et al Management of CSF leak in base of skull fractures in adults. 2016 Dec;30(6):596-604. doi: 10.1080/02688697.2016.1229746.
- Le C, E Strong B, Luu Q. et al Management of Anterior Skull Base C. Fluid Leaks. 2016 Oct;77(5):404-11.doi: 10.1055/s-0036-1584229.
- Omura K, Nomura K, Mori R. et al. Optimal Multiple-Layered Anterior-Skull-Base Reconstruction.-2022 Jan 1;22(1):e1-e6. doi: 10.1227/ONS.0000000000000013.
- Hentati F, Kocharyan A, Ruthberg J. et al. Anterior and Lateral Skull-Base Spontaneous CSF-Leaks. 2023 Sep;132(9):1102-1109. doi: 10.1177/00034894221134368.
- Nix P, Alavi S, Tyagi A. et al. Endoscopic repair of the anterior skull- 2018 Aug;32(4):407-411.doi: 10.1080/02688697.2018.1478062.
- Archer J, Sun H, Bonney P. repair techniques using combined vascularized tissue flaps. 2016 Mar;124(3):647-56.doi: 10.3171/2015.4.JNS1528.
- Ziu M, Savage J, Jimenez D. et al. Diagnosis and treatment of cerebrospinal fluid rhinorrhea following. 2012 doi.org/10.3171/2012.4.FOCUS1244.
- Milton C, Andrews B, Baker C. et al. Primary Repair of Posteriorly Located Anterior-Skull Base-Dural Defects Using Nonpenetrating Titanium-Clip in Cranial Trauma. 2020 Nov 26;83(2): 116-124.doi: 10.1055/s-0040-1718765.
- Lucke-Wold B, Pierre K, Aghili-Mehrzi S. Facial Fractures;; 2248-9614. June 1, 2022, DOI <https://doi.org/10.1055/s-0042-1749068>.
- Zhang F, Zeng T, Gao I. et al. Treatment extended extradural anterior skull base approach. Volume 24, Issue 5, September 2021, Pages 280-285, <https://doi.org/10.1016/j.cjtee.2021.06.002>.
- Fritz C, Harris J, Ravin E. et eal. Epidemiology of Anterior and Lateral Basilar Skull Fractures with CSF Leak: A National Trauma Data Bank Analysis. 2023 Jul-Aug;34(5): 1393-1397.doi: 10.1097/SCS.0000000000009279.
- Milton C, Andrews B, Baker C. et al. Primary Repair of Posteriorly Located Anterior-Skull in Cranial Trauma. 2020 Nov 26. doi: 10.1055/s-0040-1718765.
- Wang E, Zanation M, Gardner P. et al endoscopic skull-base surgery. Volume 9, pages S145-S365 2019. <https://doi.org/10.1002/alr.22326>.
- Castillo L, Jaklis A, Paquis P, Haddad A, Santini J. Nasal endoscopic repair of cerebrospinal fluid rhinorrhea. *Rhinology.* 1999 Mar;37(1):33-6. PMID: 10229980.
- Hughes RG, Jones NS, Robertson IJ. The endoscopic treatment of cerebrospinal fluid rhinorrhoea: the Nottingham experience. *J Laryngol Otol.* 1997 Feb;111(2):125-8. doi: 10.1017/s0022215100136643. PMID: 9102436.
- Klotch DW, Patel N, Dussia E. Evolution of the extracranial approach for repair of CSF leaks: review of the literature and clinical cases. *J Craniomaxillofac Trauma.* 1998 Fall;4(3):30-5. PMID: 11951424.
- Mattox DE, Kennedy DW. Endoscopic management of cerebrospinal fluid leaks and cephaloceles. *Laryngoscope.* 1990 Aug;100(8):857-62. doi: 10.1288/00005537-199008000-00012. PMID: 2381261.
- Anand VK, Murali RK, Glasgold MJ. Surgical decisions in the management of cerebrospinal fluid rhinorrhoea. *Rhinology.* 1995 Dec;33(4):212-8. PMID: 8919214.
- Dodson EE, Gross CW, Swerdloff JL, Gustafson LM. Transnasal endoscopic repair of cerebrospinal fluid rhinorrhea and skull base defects: a review of twenty-nine cases. *Otolaryngol Head Neck Surg.* 1994 Nov;111(5):600-5. doi: 10.1177/019459989411100510. PMID: 7970798.
- Burns JA, Dodson EE, Gross CW. Transnasal endoscopic repair of cranionasal fistulae: a refined technique with long-term follow-up. *Laryngoscope.* 1996 Sep;106(9 Pt 1):1080-3. doi: 10.1097/00005537-199609000-00007. PMID: 8822709.
- Lanza DC, O'Brien DA, Kennedy DW. Endoscopic repair of cerebrospinal fluid fistulae and encephaloceles.

- Laryngoscope. 1996 Sep;106(9 Pt 1):1119-25. doi: 10.1097/00005537-199609000-00015. PMID: 8822717.
29. Schick B, Weber R, Kahle G, Draf W, Lackmann GM. Late manifestations of traumatic lesions of the anterior skull base. *Skull Base Surg.* 1997;7(2):77-83. doi: 10.1055/s-2008-1058612. PMID: 17170993; PMCID: PMC1656597.
  30. Husain M, Jha D, Vatsal DK, Husain N, Gupta RK. Neuroendoscopic Transnasal Repair of Cerebrospinal Fluid Rhinorrhea. *Skull Base.* 2003 May;13(2):73-78. doi: 10.1055/s-2003-820561. PMID: 15912162; PMCID: PMC1131833.
  31. Nachtigal D, Frenkiel S, Yoskovitch A, Mohr G. Endoscopic repair of cerebrospinal fluid rhinorrhea: is it the treatment of choice? *J Otolaryngol.* 1999 Jun;28(3):129-33. PMID: 10410342.
  32. Casiano RR, Jassir D. Endoscopic cerebrospinal fluid rhinorrhea repair: is a lumbar drain necessary? *Otolaryngol Head Neck Surg.* 1999 Dec;121(6):745-50. doi: 10.1053/hn.1999.v121.a98754. PMID: 10580231.
  33. Marks SC. Middle turbinate graft for repair of cerebral spinal fluid leaks. *Am J Rhinol.* 1998 Nov-Dec;12(6):417-9. doi: 10.2500/105065898780707900. PMID: 9883298.
  34. Marshall AH, Jones NS, Robertson IJ. CSF rhinorrhoea: the place of endoscopic sinus surgery. *Br J Neurosurg.* 2001 Feb;15(1):8-12. doi: 10.1080/02688690020024319. PMID: 11303672.
  35. Mao VH, Keane WM, Atkins JP, Spiegel JR, Willcox TO, Rosen MR, Andrews D, Zwillenberg D. Endoscopic repair of cerebrospinal fluid rhinorrhea. *Otolaryngol Head Neck Surg.* 2000 Jan;122(1):56-60. doi: 10.1016/S0194-5998(00)70144-6. PMID: 10629483.
  36. Chin GY, Rice DH. Transnasal endoscopic closure of cerebrospinal fluid leaks. *Laryngoscope.* 2003 Jan;113(1):136-8. doi: 10.1097/00005537-200301000-00025. PMID: 12514397.
  37. Castelnovo P, Mauri S, Locatelli D, Emanuelli E, Delù G, Giulio GD. Endoscopic repair of cerebrospinal fluid rhinorrhea: learning from our failures. *Am J Rhinol.* 2001 Sep-Oct;15(5):333-42. PMID: 11732821.
  38. Lund VJ. Endoscopic management of cerebrospinal fluid leaks. *Am J Rhinol.* 2002 Jan-Feb;16(1):17-23. PMID: 11895190.
  39. McMains KC, Gross CW, Kountakis SE. Endoscopic management of cerebrospinal fluid rhinorrhea. *Laryngoscope.* 2004 Oct;114(10):1833-7. doi: 10.1097/00005537-200410000-00029. PMID: 15454781.
  40. Lee TJ, Huang CC, Chuang CC, Huang SF. Transnasal endoscopic repair of cerebrospinal fluid rhinorrhea and skull base defect: ten-year experience. *Laryngoscope.* 2004 Aug;114(8):1475-81. doi: 10.1097/00005537-200408000-00029. PMID: 15280729.
  41. Lindstrom DR, Toohill RJ, Loehrl TA, Smith TL. Management of cerebrospinal fluid rhinorrhea: the Medical College of Wisconsin experience. *Laryngoscope.* 2004 Jun;114(6):969-74. doi: 10.1097/00005537-200406000-00003. PMID: 15179197.
  42. Husain M, Jha D, Vatsal DK, Husain N, Gupta RK. Neuroendoscopic Transnasal Repair of Cerebrospinal Fluid Rhinorrhea. *Skull Base.* 2003 May;13(2):73-78. doi: 10.1055/s-2003-820561. PMID: 15912162; PMCID: PMC1131833.
  43. Tosun F, Gonul E, Yetiser S, Gerek M. Analysis of different surgical approaches for the treatment of cerebrospinal fluid rhinorrhea. *Minim Invasive Neurosurg.* 2005 Dec;48(6):355-60. doi: 10.1055/s-2005-915636. PMID: 16432785.
  44. Landeiro JA, Lázaro B, Melo MH. Endonasal endoscopic repair of cerebrospinal fluid rhinorrhea. *Minim Invasive Neurosurg.* 2004 Jun;47(3):173-7. doi: 10.1055/s-2004-818451. PMID: 15343435.
  45. Briggs RJ, Wormald PJ. Endoscopic transnasal intradural repair of anterior skull base cerebrospinal fluid fistulae. *J Clin Neurosci.* 2004 Aug;11(6):597-9. doi: 10.1016/j.jocn.2003.09.011. PMID: 15261227.
  46. Gendeh BS, Mazita A, Selladurai BM, Jegan T, Jeevanan J, Misiran K. Endonasal endoscopic repair of anterior skull-base fistulas: the Kuala Lumpur experience. *J Laryngol Otol.* 2005 Nov;119(11):866-74. doi: 10.1258/002221505774783421. PMID: 16354338.
  47. Kirtane MV, Gautham K, Upadhyaya SR. Endoscopic CSF rhinorrhea closure: our experience in 267 cases. *Otolaryngol Head Neck Surg.* 2005 Feb;132(2):208-12. doi: 10.1016/j.otohns.2004.09.004. PMID: 15692527.
  48. Araujo Filho BC, Butugan O, Pádua FG, Voegels RL. Endoscopic repair of CSF rhinorrhea: experience of 44 cases. *Braz J Otorhinolaryngol.* 2005 Jul-Aug;71(4):472-6. doi: 10.1016/s1808-8694(15)31202-7. Epub 2005 Dec 15. PMID: 16446963; PMCID: PMC944196.
  49. Bernal-Sprekelsen M, Alobid I, Mullol J, Trobat F, Tomás-Barberán M. Closure of cerebrospinal fluid leaks prevents ascending bacterial meningitis. *Rhinology.* 2005 Dec;43(4):277-81. PMID: 16405272.
  50. Locatelli D, Rampa F, Acchiardi I, Bignami M, De Bernardi F, Castelnovo P. Endoscopic endonasal approaches for repair of cerebrospinal fluid leaks: nine-year experience. *Neurosurgery.* 2006 Apr;58(4 Suppl 2):ONS-246-56; discussiom ONS-256-7. doi: 10.1227/01.NEU.0000193924.65297.3F. PMID: 16582647.
  51. Shah R, Kveton J, Schwartz N. Hydroxyapatite Use in Repair of Lateral Skull Base CSF Leaks Via Transmastoid Approach: When Does It Work? *Otol Neurotol.* 2023 Sep 1;44(8):804-808. doi: 10.1097/MAO.0000000000003973. Epub 2023 Aug 3. PMID: 37550871.
  52. Hara T, Akutsu H, Tanaka S, Kino H, Miyamoto H, Ii R, Takano S, Ishikawa E. Risk Factors for Postoperative Cerebrospinal Fluid Leak after Graded Multilayer Cranial Base Repair with Suturing via the Endoscopic Endonasal Approach. *Neurol Med Chir (Tokyo).* 2023 Feb 15;63(2):48-57. doi: 10.2176/jns-nmc.2022-0132. Epub 2022 Nov 25. PMID: 36436977; PMCID: PMC9995146.
  53. Gâta A, Trombitas VE, Albu S. Endoscopic management of frontal sinus CSF leaks. *Braz J Otorhinolaryngol.* 2022 Jul-Aug;88(4):576-583. doi: 10.1016/j.bjorl.2020.08.002. Epub

- 2020 Sep 20. PMID: 33012703; PMCID: PMC9422493.
54. Soudry E, Turner JH, Nayak JV, Hwang PH. Endoscopic reconstruction of surgically created skull base defects: a systematic review. *Otolaryngol Head Neck Surg.* 2014 May;150(5):730-8. doi: 10.1177/0194599814520685. Epub 2014 Feb 3. PMID: 24493791.
  55. Gandhi J, DiMatteo A, Joshi G, Smith NL, Khan SA. Cerebrospinal fluid leaks secondary to dural tears: a review of etiology, clinical evaluation, and management. *Int J Neurosci.* 2021 Jul;131(7):689-695. doi: 10.1080/00207454.2020.1751625. Epub 2020 May 13. PMID: 32242448.
  56. Gilat H, Rappaport Z, Yaniv E. Endoscopic transnasal cerebrospinal fluid leak repair: a 10 year experience. *Isr Med Assoc J.* 2011 Oct;13(10):597-600. PMID: 22097227.
  57. Oh JW, Kim SH, Whang K. Traumatic Cerebrospinal Fluid Leak: Diagnosis and Management. *Korean J Neurotrauma.* 2017 Oct;13(2):63-67. doi: 10.13004/kjnt.2017.13.2.63. Epub 2017 Oct 31. PMID: 29201836; PMCID: PMC5702760.
  58. Dong RP, Zhang Q, Yang LL, Cheng XL, Zhao JW. Clinical management of dural defects: A review. *World J Clin Cases.* 2023 May 6;11(13):2903-2915. doi: 10.12998/wjcc.v11.i13.2903. PMID: 37215425; PMCID: PMC10198091.
  59. Nishioka H, Izawa H, Ikeda Y, Namatame H, Fukami S, Haraoka J. Dural suturing for repair of cerebrospinal fluid leak in transnasal transsphenoidal surgery. *Acta Neurochir (Wien).* 2009 Nov;151(11):1427-30. doi: 10.1007/s00701-009-0406-2. Epub 2009 Jun 5. PMID: 19499173.
  60. Chen PF, Yip CM, Lin YH. Endoscopic transpterygoid approach to repair lateral sphenoid recess cerebrospinal fluid leak by multilayered reconstruction. *Eur Ann Otorhinolaryngol Head Neck Dis.* 2021 Nov;138 Suppl 4:135-136. doi: 10.1016/j.anorl.2021.06.010. Epub 2021 Jul 8. PMID: 34246589.



# Technical nuance. Total endoscopic removal of third ventricle colloid cyst

Arvind Verma, Jaimin Modh, Krushi Soladhra,  
Dharmik Velani

NHL Medical College and SVP Hospital, INDIA

## ABSTRACT

**Background.** Colloid cyst treatment with purely endoscopic surgery is considered to be safe and effective. Complete capsule removal for gross total resection is usually recommended to prevent recurrence but may not always be safely feasible. Our objective was to go for complete endoscopic surgery using mainly aspiration, manipulation and coagulation with complete capsule resection and discuss the rationale for the procedure.

**Methods and materials.** A case report with a third ventricle colloid cyst was surgically treated with a complete endoscopic excision using the proper technique.

**Results.** Our patient underwent Transforaminal endoscopic surgery and the cyst was excised completely and the capsule was removed intentionally. Cyst remnants were absent on postoperative MRI. Mild Intraventricular haemorrhage was an intraoperative complication. Surgery was statistically associated with cyst volume and ventricular size reduction. There were no serious complications postoperatively. Follow-up did not show any recurrence or remnant growth that needed further treatment.

**Conclusion.** Gross total resection may be the main objective for selected cases and seems to be safer while preserving good results, especially in a limited resource environment. Surgical planning allows the surgeon to choose among the different resection routes and techniques available. Decisions are predominantly based on preoperative imaging and intraoperative findings. Full-endoscopic approach for third ventricle colloid cyst removal is a feasible technique. Cyst aspiration followed by grasping and rotational manoeuvre for the cyst wall provides total removal with the resolution of the obstruction if present and relief of symptoms.

## INTRODUCTION

Intraventricular colloid cysts are a rare, benign cystic lesion originating from embryonic remnants of the brain endoderm. The most common location is in the rostral portion of the third ventricle near the opening of interventricular foramen of Monro. Obstructive hydrocephalus is the common presentation in symptomatic patients due to blockage of the CSF pathways around the foramen of Monro [2]. This makes surgical resection mandatory and treatment of choice for large (>7 mm) symptomatic cysts with hydrocephalus.

There are various surgical options such as ventriculoperitoneal shunt, pure aspiration of the cyst material, microsurgical or complete

## Keywords

third ventricle,  
intraventricular colloid cyst,  
endoscopic surgery,  
complete excision,  
foramen of monro



Corresponding author:  
Arvind Verma

NHL Medical College and SVP  
Hospital, India

dr.arvindverma8886@gmail.com

Scan to access the online version

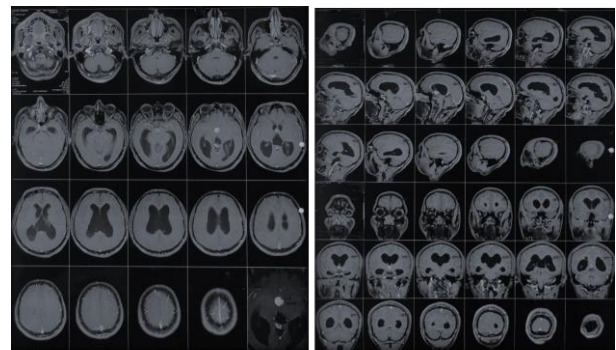


endoscopic resection of the cyst. The newer endoscopic approach is a well-known and established option for surgical candidates. It offers shorter ICU and hospital stays, fewer complications, lower costs and shorter duration of surgery compared to microsurgery [3, 4]. The endoscopic view reveals better and complete anatomy of the cyst, its exact location and attachments to the roof of the third ventricle and relations to the surrounding structures [1].

Neurosurgeons regularly use the transforaminal (TF) route for the cysts localisation. It leads to easy access to the cyst, followed by opening of its capsule, aspiration of its mucinous contents and coagulation of the base with complete resection of. Route variations such as transseptal or interforaminal are required for proper aspiration, coagulation and removal of the cysts in some difficult cases with complex anatomy. In this article, the author has presented a case report to show that the total endoscopic technique is effective and has a low recurrence and complication rate.

#### CASE PRESENTATION

A 29 years old male patient presented to us with complaint of progressive dimness of vision in right eye followed by in left eye associated with headache (on and off) since 2 months.



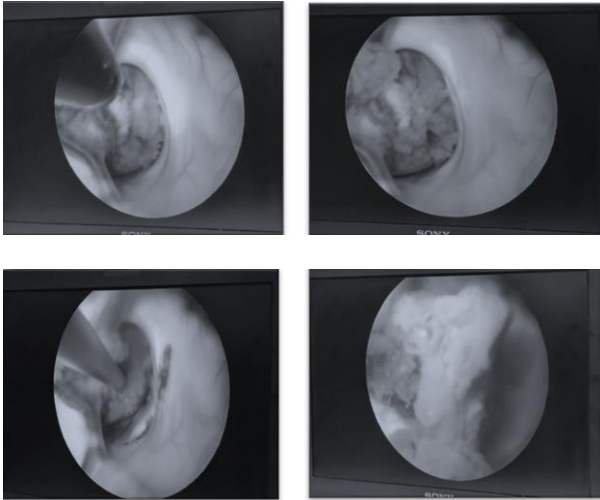
**Figure 1.** Preoperative MRI plain with contrast scans.

On examination he was E4 V5 M6 with no perception of light on left eye and counting fingers at 3 feet on left eye. Right pupil was non reacting to light and Left sluggishly reacting to light with rest Neurology was normal. MRI scans were suggestive of third ventricular lesion which was hyperintense on T1W and hypo to isointense in T2W images with no contrast enhancement (Fig a,b,c,d). Surgery was planned and he underwent endoscopic removal of tumor. During intraoperative period there was mild oozing of blood from tumor bed which was managed by saline irrigation and electrocauterization. Postoperative period was uneventful and patient is doing well.

#### OPERATIVE TECHNIQUE

A single skin incision was made, five cm anterior to the coronal suture and four cm lateral to the midline

followed by a large burr-hole measuring about 14 mm in diameter was created. We think that this entry point into the lateral ventricle provides a good enough view of the third ventricle cyst. In most cases, the side with the bigger ventricle was selected, and the right side was used to stay in the non-dominant hemisphere if both ventricles were equal.



**Figure 1-4.**



**Figure P.** (Post-operative CT scan).

Ventricular puncture was performed initially by needle insertion 5-7 cm deep and the CSF flow was obtained. Next puncture was made using a trocar introducer. The trocar was removed and the sheath

was stabilized with the assistant's help. The 0°-angled neuroendoscope which have an internal working channel diameter of 3.1 mm and a length of 122 mm, was inserted through the sheath, and the surgical operation was initiated under visual control. At the level of the foramen of Monro, the cyst was found (Figures 1 and 2). Before excising the cyst wall, we opted to aspirate the cyst's constituent parts. If the cyst is removed without aspirating its constituent parts, it may rupture, causing subtotal resection of the cyst wall that remains and perhaps impairing eyesight. The cyst's attachments were coagulated following the cyst's mobilization and emptying of its contents (Figures 3 and 2).

Little oozing from the ventricular wall was noticed during the procedure and this was managed by compression, continuous saline irrigation, and electrocauterization. The cyst was completely excised by firmly holding the cyst wall with small forceps and rotating (swiveling) the cyst wall to accomplish complete resection. Following the procedure, a standard external ventricular drainage (EVD) was left in place before being closed. Following surgery, the patient was shifted to the neurosurgical intensive care unit for postoperative monitoring and an assessment of any minor bleeding. When the CT brain plain was obtained on the first postoperative day, there was not much IVH, and it was treated conservatively (Figure P). When not even the cyst capsule was seen in the pictures, total resection was declared.

#### DISCUSSION

Surgery is necessary for symptomatic patients with hydrocephalus since there is a 10% chance of abrupt death. There is disagreement concerning the optimal surgical therapy modalities. In contemporary neurosurgery, methods like stereotactic cyst aspiration or ventriculoperitoneal shunting are often abandoned. While safe, effective, and minimally invasive, stereotactic cyst aspiration simply removes the cyst's substance. Small or dense cysts might be problematic. High recurrence rates result from the cyst capsule being left behind (12). The most often used surgical techniques are endoscopic removal and microsurgical excision, according to a meta-analysis. The surgeon's experience and personal preference play a major role in selecting between the two techniques. The reason ETV has become so popular is because it is successful procedure,

provides shunt free period and a lifelong dependency on shunt surgery can be avoided. This operation was most suited for our patient, who had hydrocephalus and a third ventricular cyst. After undergoing transforaminal endoscopic surgery, the cyst was removed completely. One intraoperative complication was mild intraventricular bleeding. Conservative management was observed for the minimal number of post-operative problems. Surgery is statistically linked to ventricular size and cyst volume. Following surgery, there were no significant problems. There was no recurrence or residual growth that required medical attention, according to the follow-up. This technique can serve as a good alternative for VP shunting and open microscopic excision.

### CONCLUSION

When compared to microsurgical technique, complete excision by endoscopic technique appears to be safer while maintaining best results and safety, particularly in a limited area of exposure. It should be the primary goal in every case. The surgeon can select from a variety of resection routes and techniques for surgical planning. Preoperative imaging and intraoperative findings are the main sources of decision-making. It is very possible to remove a third ventricle colloid cyst using a full-endoscopic method. If an obstruction is present, it can be resolved and symptoms can be relieved by aspirating the cyst and then gripping and rotating the cyst wall. Our paper is a case report that demonstrates the effectiveness of the whole endoscopic procedure with minimal complications and no recurrence.

### REFERENCES

1. Azab WA, Najibullah M, Yosef W. Endoscopic colloid cyst excision: Surgical techniques and nuances. *Acta Neurochir (Wien)* 2017;159:1053-8. [PubMed], [Google Scholar].
2. Brun A, Egund N. The pathogenesis of cerebral symptoms in colloid cysts of the third ventricle: A clinical and pathoanatomical study. *Acta Neurol Scand* 1973;49:525-35. [PubMed], [Google Scholar].
3. Horn EM, Feiz-Erfan I, Bristol RE, Lekovic GP, Goslar PW, Smith KA, et al. Treatment options for third ventricular colloid cysts: Comparison of open microsurgical versus endoscopic resection. *Neurosurgery* 2007;60:613-8; discussion 618-20. [PubMed], [Google Scholar].
4. Samadian M, Ebrahimzadeh K, Maloumeh EN, Jafari A, Sharifi G, Shiravand S, et al. Colloid cyst of the third ventricle: Long-term results of endoscopic management in a series of 112 cases. *World Neurosurg* 2018;111:e440-8. [PubMed], [Google Scholar].
5. Agha RA, Borrelli MR, Farwana R, Koshy K, Fowler AJ, Orgill DP, et al. The PROCESS 2018 statement: Updating consensus preferred reporting of case series in surgery (PROCESS) guidelines. *Int J Surg* 2018;60:279-82. [PubMed], [Google Scholar].
6. Armao D, Castillo M, Chen H, Kwock L. Colloid cyst of the third ventricle: Imaging-pathologic correlation. *AJNR Am J Neuroradiol* 2000;21:1470-7. [PubMed], [Google Scholar].
7. Boogaarts HD, Decq P, Grotenhuis JA, Le Guerinel C, Nseir R, Jarraya B, et al. Long-term results of the neuroendoscopic management of colloid cysts of the third ventricle: A series of 90 cases. *Neurosurgery* 2011;68:179-87. [PubMed], [Google Scholar].
8. Brostigen CS, Meling TR, Marthinsen PB, Scheie D, Aarhus M, Helseth E. Surgical management of colloid cyst of the third ventricle. *Acta Neurol Scand* 2017;135:484-7. [PubMed], [Google Scholar].
9. Burhan Janjua M, Reddy S, El Ahmadih TY, Ban VS, Hwang SW, Ozturk AK, et al. Inchoate guidelines of endoscopic resection of colloid cysts. *J Clin Neurosci* 2020;71:1-8. [PubMed], [Google Scholar].
10. Connolly ID, Johnson E, Lamsam L, Veeravagu A, Ratliff J, Li G. Microsurgical vs. endoscopic excision of colloid cysts: An analysis of complications and costs using a longitudinal administrative database. *Front Neurol* 2017;8:259. [PubMed], [Google Scholar].



# Primary bone non-Hodgkin lymphoma with vertebra involvement

**Muhammed Erkam Yuksek, Gulsum Arslan Karagoz, Densel Arac, Fatih Keskin**

Necmettin Erbakan University Faculty of Medicine, Department of Neurosurgery, Konya, TURKEY

## ABSTRACT

NHL accounts for less than 5% of all adult cancers. Bone lymphomas, mostly NHL, may cause extradural cord compression. Non-Hodgkin lymphomas (NHL) represent a group of heterogeneous neoplasms originating from the bone marrow or lymph nodes. NK cell tumours with CD56 positive immunophenotype are a very rare and heterogeneous group of diseases among bone lymphomas presenting with nonspecific clinical and radiological findings. Although there is no gold standard treatment for this disease, there are various forms of treatment such as chemotherapy, immunotherapy, surgery or radiotherapy. In this report, a primary bone lymphoma with vertebral and femur involvement, which is treated with chemotherapy, radiotherapy and autologous transplantation after surgical treatment, is presented.

## INTRODUCTION

Non-Hodgkin lymphomas (NHL) accounts for less than 5% of all adult cancers. Lymphoma is considered as primary bone lymphoma (PBL) when it does not involve in distant lymph nodes or non-osseous regions and affects the bones. PBL constitutes only 7% of all malignant bone tumors and is extremely rare among all lymphomas.<sup>28</sup>

NK cell tumors with CD56 positive immunophenotype are a very rare and heterogeneous group of diseases among bone lymphomas presenting with nonspecific clinical and radiological findings. Early diagnosis of the disease is important because its stage affects the prognosis. The rarity of malignant lymphomas involving the spine may complicate early diagnosis.

In this report, a primary bone lymphoma with vertebral and femur involvement, which treated with chemotherapy, radiotherapy and autologous transplantation after surgical treatment, is presented.

## CASE

A 22-year-old male patient presented with low back pain, swelling in the right groin and pain in the right thigh for 1 month. On examination, there was a palpable 4x4 cm mass in the right groin and hypoesthesia on the right L3 dermatome. In the superficial tissue ultrasonography,

## Keywords

lymphoma,  
natural killer cell,  
non-Hodgkin,  
spinal



Corresponding author:  
**Gulsum Arslan Karagoz**

Necmettin Erbakan University  
Faculty of Medicine, Department of  
Neurosurgery, Konya, Turkey

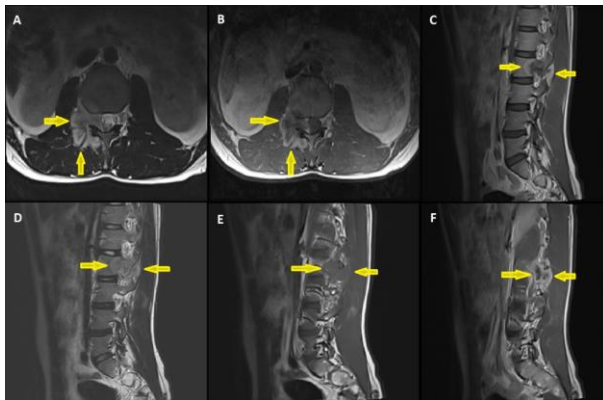
drgulsumarslang6@gmail.com

Scan to access the online version

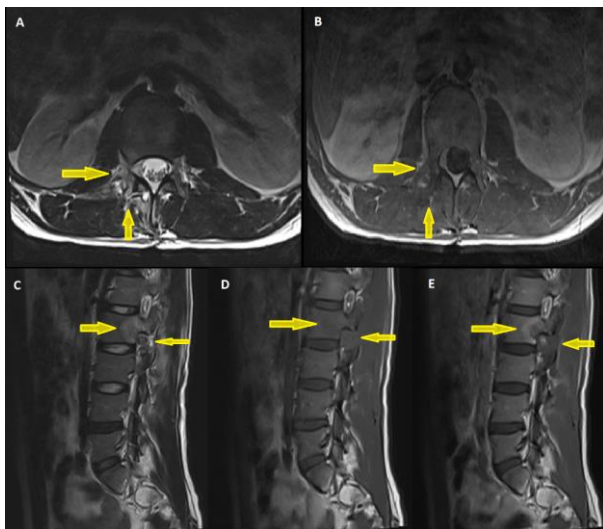


42 mm in diameter round lymphadenopathy was observed in the right iliac region. Contrast-enhanced lumbar MRI showed a hypo-isointense lesion on T1-weighted images (Figure 1a), and a heterogeneous hyperintense lesion on T2-weighted images (Figure 1b). Contrast-enhancing extradural lesion involved the L2 corpus and right pedicle and protruded beyond the bone (Figure 1c,d). Lymphoma was considered in the preliminary diagnosis and excisional biopsy was performed. The biopsy was reported as a cytotoxic and aggressive T-cell lymphoma.

When preparations were completed, the patient was taken into operation.



**Figure 1.** Lesion seen on preoperative contrast-enhanced lumbar MRI (Yellow Arrow).



**Figure 2.** Sections showing that adequate decompression was achieved on postoperative contrast-enhanced lumbar MRI.

During the operation, an extradural tumoral lesion involving the L2-3 facet and extending to the transfer

process was observed. A gray-colored heterogeneous mass with soft consistency surrounding the right L2 root and filling the foramen was grossly excised. Right L2, L3 roots were decompressed. In immunohistochemical examination, CD2, CD3, CD4, CD7, CD8, CD30, CD45, CD56, Granzyme B, Bcl-6, Kappa and Lambda positive, Epstein Barr Virus, CD5, CD10, CD20, CD21, CD23, CD1A, BCL-2, HHV-8 was ALK negative. The pathological diagnosis of the patient, whose Ki-67 index was measured around 95%, was reported as high grade NHL with NK/T cells. CD56 positivity showed NK origin. In addition, T cell markers CD30 and Granzyme B expressions supported an aggressive and cytotoxic lymphoma.

Postoperative contrast-enhanced MRI showed that spinal canal decompression was achieved (figures 2A,2D) and there was a decrease in contrast-enhancing areas (figures 2B,2C,2E,2F). Patient's complaints regressed after surgery. Granulocytic cell dominance and hypercellular bone marrow with 90% cellularity were observed in pathological examination of the bone marrow. Then, chemotherapy and radiotherapy were planned for the patient by medical oncology and radiation oncology consultants. The patient received 8 cycles of CHOP (Cyclophosphamide, Adriamycin, Vincristine and Prednisolone) in a post-operative 6-month period. 15 sessions of radiotherapy were applied to the lesion on the right inguinal and femoral head. Bone marrow TRU-CUT biopsy which was performed 1 month after the completion of chemotherapy and radiotherapy was found normocellular and the patient was completely cured. In control PET CT, it was reported that the involvement of the right femoral head and lumbar vertebra regressed and the lymph nodes were non-metabolic. Stem cells were collected 1 month after complete cure and autologous transplantation was performed. A case of NK/T cell primary bone lymphoma with complete cure was discussed in the light of the literature.

## DISCUSSION

Nearly 370,000 patients are diagnosed with NHL each year, and extra-nodal NHL accounts for approximately 10-20% of all NHL<sup>8</sup>.

In order to reduce the inconsistencies in the definition of PBL Ostrowski et al. developed guidelines. According to these guidelines lymphoma is considered as primary bone lymphoma (PBL) when

it does not involve in distant lymph nodes or non-osseous regions and affects the bones. The presence or absence of primary lymph node involvement does not affect the diagnosis<sup>17</sup>. Primary bone lymphoma is a rare disease 5% of all extranodal non-Hodgkin lymphomas<sup>4</sup>. Most of bone lymphomas are NHL<sup>1</sup>. The spine and pelvis are the most frequently involved regions after the femur<sup>23</sup>. Thoracic (69%) and lumbar (27%) spine involvements were reported most frequently among all spinal cord involvement<sup>3</sup>. They originate from the paravertebral lymph nodes or the vertebral body and may cause extradural cord compression(%16)<sup>13</sup>.

The time between onset of symptoms and diagnosis is approximately eight months

Bone lymphomas occur with nonspecific radiological findings. Lytic or sclerotic image can be observed on direct X-Ray. Computed tomography (CT) is used to identify the extraosseous spread of the lesions. In magnetic resonance imaging (MRI), it is hypointense on T1-weighted images and hyperintense on T2-weighted images<sup>24</sup>. Gadolinium uptake is observed in contrast-enhanced examinations<sup>14</sup>. Positron emission tomography (PET) provides functional imaging and high FDG (Fludeoxyglucose) uptake is observed. The combined use of PET and CT scanning gives simultaneous functional and anatomical information and assists in determining response to therapy. PET - CT has been used for advanced staging in 42% of lymphoma patients<sup>22</sup>.

Immunohistochemistry analysis is used to determine the subtype of lymphoma. Electron microscopic studies and immunohistochemical studies have shown that these tumors most commonly originate from B cells. Diffuse Large B Cell Lymphoma (DLBCL) is the most common subtype in both primary bone lymphomas and secondary bone invasions<sup>15</sup>.

NK cell tumors are an extremely rare, heterogeneous group of diseases with a wide spectrum of morphological, immunophenotypic, and clinical features<sup>27</sup>. NK Cells have a CD56 positive immunophenotype. Mature and activated NK cells are negative for CD20, CD5 antigens and lack other T cell antigens such as surface CD3, CD4, CD5, CD57, CD16, CD20<sup>18</sup>. Tumor cells are rarely positive for the T-cell markers such as CD7, CD30, and CD45RO. Since NK cells do not contain CD3, expression of the CD3 antigen indicates that the tumor also contains T

lymphocytes<sup>9</sup>. In this case, CD3, CD7, CD30 expression was positive and CD5 was negative.

Staging of bone lymphomas is crucial as it determines the prognosis and treatment of the disease. Recommended staging is Ann Arbor modified staging criteria<sup>15</sup>. Patients with Ann Arbor Stage 1 and 2 has a higher rate of positive response to treatment and less relapse rates. This information draws attention to the importance of early diagnosis. There are many treatment options for this disease, such as chemotherapy, immunotherapy, surgery or radiotherapy. In the presence of spinal instability and progressive neurological deficit, surgical intervention is required to maintain stability and prevent neurological deterioration<sup>11</sup>. Adjuvant therapy should be a part of the treatment. Surgical treatment without adjuvant therapy has been shown to have no benefit in overall or progression-free survival<sup>6</sup>. CHOP is primarily recommended for treatment.

Radiotherapy is usually the first treatment for stage 1, 2 NK/T-cell lymphoma<sup>29</sup>. High-dose methotrexate-based chemotherapies are thought to have better survival than radiotherapy alone<sup>5</sup>.

## CONCLUSION

Treatment of spinal cord tumor should be based on diagnosis, staging, and awareness of tumor biology and behavior<sup>2</sup>. Post-surgical chemotherapy and autologous bone marrow treatment provide local and systemic control of lymphoma. Thus, early diagnosis can effect survival positively. In this case, surgical treatment was the primary choice because the characteristics of NK/T-cell lymphomas, tumor location, neurological status, and spinal stability were taken into account. The aggressive prognosis has led some physicians to consider bone marrow or peripheral stem cell transplantation as an alternative treatment option<sup>12</sup>.

## REFERENCES

1. Alencar A, Pitcher D, Byrne G, Lossos IS. Primary bone lymphoma--the University of Miami experience. *Leuk Lymphoma*. 2010;51(1):39-49. doi:10.3109/10428190903308007
2. Al-Hakeem DA, Fedele S, Carlos R, Porter S. Extranodal NK/T-cell lymphoma, nasal type. *Oral Oncol*. 2007;43(1):4-14. doi:10.1016/j.oraloncology.2006.03.011
3. Barz M, Aftahy K, Janssen I, et al. Spinal Manifestation of Malignant Primary (PLB) and Secondary Bone Lymphoma

- (SLB). *Curr Oncol*. 2021;28(5):3891-3899. doi:10.3390/curroncol28050332
4. Chisholm KM, Ohgami RS, Tan B, Hasserjian RP, Weinberg OK. Primary lymphoma of bone in the pediatric and young adult population. *Hum Pathol*. 2017;60:1-10. doi:10.1016/j.humpath.2016.07.028
  5. Deangelis LM, Iwamoto FM. An update on therapy of primary central nervous system lymphoma. *Hematol Am Soc Hematol Educ Progr*. Published online 2006:311-316. doi:10.1182/asheducation-2006.1.311
  6. D'Cruz J, Adeeb N, Von Burton G, et al. Diagnosis and management of intramedullary spinal cord lymphoma: A case illustration and review of literature. *Interdiscip Neurosurg*. 2020;19:100552. doi:https://doi.org/10.1016/j.inat.2019.100552
  7. Ferreri AJM, Abrey LE, Blay JY, et al. Summary statement on primary central nervous system lymphomas from the Eighth International Conference on Malignant Lymphoma, Lugano, Switzerland, June 12 to 15, 2002. *J Clin Oncol Off J Am Soc Clin Oncol*. 2003;21(12):2407-2414. doi:10.1200/JCO.2003.01.135
  8. Freeman C, Berg JW, Cutler SJ. Occurrence and prognosis of extranodal lymphomas. *Cancer*. 1972;29(1):252-260. doi:10.1002/1097-0142(197201)29:1<252::aid-cncr2820290138>3.0.co;2-#
  9. Jaffe ES, Chan JK, Su JJ, et al. Report of the Workshop on Nasal and Related Extranodal Angiocentric T/Natural Killer Cell Lymphomas. Definitions, differential diagnosis, and epidemiology. *Am J Surg Pathol*. 1996;20(1):103-111. doi:10.1097/00000478-199601000-00012
  10. Kim GE, Koom WS, Yang WI, et al. Clinical relevance of three subtypes of primary sinonasal lymphoma characterized by immunophenotypic analysis. *Head Neck*. 2004;26(7):584-593. doi:10.1002/hed.20015
  11. Laufer I, Rubin DG, Lis E, et al. The NOMS framework: approach to the treatment of spinal metastatic tumors. *Oncologist*. 2013;18(6):744-751. doi:10.1634/theoncologist.2012-0293
  12. Lee J, Au WY, Park MJ, et al. Autologous hematopoietic stem cell transplantation in extranodal natural killer/T cell lymphoma: a multinational, multicenter, matched controlled study. *Biol Blood Marrow Transplant J Am Soc Blood Marrow Transplant*. 2008;14(12):1356-1364. doi:10.1016/j.bbmt.2008.09.014
  13. Lizuka H, Nakamura T, Kato M. [Malignant lymphoma of the spinal epidural space. Report of three cases]. *Neurol Med Chir (Tokyo)*. 1989;29(4):307-311. doi:10.2176/nmc.29.307
  14. Mathew P, Todd N V. Intradural conus and cauda equina tumours: a retrospective review of presentation, diagnosis and early outcome. *J Neurol Neurosurg Psychiatry*. 1993;56(1):69-74. doi:10.1136/jnnp.56.1.69
  15. Messina C, Christie D, Zucca E, Gospodarowicz M, Ferreri AJM. Primary and secondary bone lymphomas. *Cancer Treat Rev*. 2015;41(3):235-246. doi:10.1016/j.ctrv.2015.02.001
  16. Ollila TA, Olszewski AJ. Extranodal Diffuse Large B Cell Lymphoma: Molecular Features, Prognosis, and Risk of Central Nervous System Recurrence. *Curr Treat Options Oncol*. 2018;19(8):38. doi:10.1007/s11864-018-0555-8
  17. Ostrowski ML, Unni KK, Banks PM, et al. Malignant lymphoma of bone. *Cancer*. 1986;58(12):2646-2655. doi:10.1002/1097-0142(19861215)58:12<2646::aid-cncr2820581217>3.0.co;2-u
  18. Quintanilla-Martinez L, Franklin JL, Guerrero I, et al. Histological and immunophenotypic profile of nasal NK/T cell lymphomas from Peru: high prevalence of p53 overexpression. *Hum Pathol*. 1999;30(7):849-855. doi:10.1016/s0046-8177(99)90147-8
  19. Ramadan KM, Shenker T, Sehn LH, Gascoyne RD, Connors JM. A clinicopathological retrospective study of 131 patients with primary bone lymphoma: a population-based study of successively treated cohorts from the British Columbia Cancer Agency. *Ann Oncol Off J Eur Soc Med Oncol*. 2007;18(1):129-135. doi:10.1093/annonc/mdl329
  20. Rao T V, Narayanaswamy KS, Shankar SK, Deshpande DH. "Primary" spinal epidural lymphomas. A clinicopathological study. *Acta Neurochir (Wien)*. 1982;62(3-4):307-317. doi:10.1007/BF01403638
  21. Sachdev RK, Sen A, Pathak A, Jangid DR. Primary extradural non-hodgkin's lymphoma. *Indian J Nucl Med IJNM Off J Soc Nucl Med India*. 2012;27(3):185-186. doi:10.4103/0972-3919.112727
  22. Schaefer NG, Strobel K, Taverna C, Hany TF. Bone involvement in patients with lymphoma: the role of FDG-PET/CT. *Eur J Nucl Med Mol Imaging*. 2007;34(1):60-67. doi:10.1007/s00259-006-0238-8
  23. Sharma A, Ahmed R, Agrawal N, et al. Primary Bone Lymphoma: A 13 Year Retrospective Institutional Analysis in the Chemo-Immunotherapy Era. *Indian J Hematol Blood Transfus an Off J Indian Soc Hematol Blood Transfus*. 2021;37(2):240-248. doi:10.1007/s12288-020-01327-3
  24. Shen G, Su M, Liu B, Kuang A. PET/CT Imaging for Solitary Primary Bone Lymphoma of Thoracic Vertebra. *Clin Nucl Med*. 2018;43(11):857-859. doi:10.1097/RLU.0000000000002275
  25. Su YJ, Wang PN, Chang H, et al. Extranodal NK/T-cell lymphoma, nasal type: Clinical features, outcome, and prognostic factors in 101 cases. *Eur J Haematol*. 2018;101(3):379-388. doi:10.1111/ejh.13126
  26. Wang Y, Li J, Wei R, Liu C, Nataraj A, Yan J. Prognostic Factors Associated With Bone Lymphoma Primarily Presenting in the Spine. *Spine (Phila Pa 1976)*. 2019;44(3):185-194. doi:10.1097/BRS.0000000000002844
  27. Weber MH, Burch S, Buckley J, et al. Instability and impending instability of the thoracolumbar spine in patients with spinal metastases: a systematic review. *Int J Oncol*. 2011;38(1):5-12.
  28. Wu H, Bui MM, Leston DG, et al. Clinical characteristics and prognostic factors of bone lymphomas: focus on the clinical significance of multifocal bone involvement by

- primary bone large B-cell lymphomas. *BMC Cancer*. 2014;14(1):900. doi:10.1186/1471-2407-14-900
30. Yao B, Li Y xiong, Fang H, Yu Z hao, Jin J, Liu X fan. [Prognostic factors and treatment outcome in early stage nasal NK/T cell lymphoma]. *Zhonghua Xue Ye Xue Za Zhi*. 2006;27(4):222-225.



# Spinal cord compression secondary to metastatic invasion of breast phyllodes tumour. Case report

Julián David Moreno Cavanzo, Juan Felipe Alvarado Riascos,  
Lorena García Agudelo,  
Laura Carolina Navarro Martínez

Health Research Department, Hospital Regional de la Orinoquía,  
COLOMBIA

## ABSTRACT

Spinal cord compression syndrome is one of the most frequent oncologic emergencies, in which early diagnosis and treatment are key factors to prevent severe and irreversible neurological damage. It is estimated to have a prevalence of 3.4% in oncologic patients and is a source of significant morbidity in cancer patients.

Spinal cord infiltration with hematogenous dissemination is the most common cause of spinal cord compression. In the present case, we present a clinical case of spinal cord compression syndrome secondary to a phyllodes tumour of the breast who was admitted to the emergency for lumbar pain with red flags and who suffered a spinal cord infarction with irreversible sequelae and poor prognosis.

## INTRODUCTION

Oncological emergencies are a group of complications that arise in the course of disease progression in cancer patients, and these complications increase morbidity and mortality, as well as sequelae. Dealing with these clinical situations requires a high level of suspicion, a correct approach and timely treatment in the emergency department, as it helps to decrease the mortality rate and the costs of hospital care.

There are multiple oncological emergencies; however, within this article we will address oncological emergencies of a neurological nature, specifically spinal cord compression syndrome, a common phenomenon in oncological patients. It occurs in 5% to 10% of patients with oncological pathology. Spinal cord compression syndrome is common in breast, lung and prostate tumors (about 60% of cases); however, about 20% of patients presenting with spinal cord compression syndrome did not have an official diagnosis of any oncological pathology. [1-2]

Generally, spinal cord compression occurs at the thoracic level in about 70% of cases, followed by involvement of the lumbosacral vertebrae (25%) and cervical vertebrae (up to 15%). However, the

## Keywords

spinal cord compression.  
paralysis vertebral  
metastasis.  
oncologic emergency.  
vertebral fracture



Corresponding author:  
Lorena García Agudelo

Health Research Department,  
Hospital Regional de la Orinoquía,  
Colombia

investigacion@horo.gov.co

Scan to access the online version



literature documents that in up to 40% of cases several segments are compressed, hence the importance of performing imaging studies that allow us to evaluate all compressed segments. [3-5]

It has been documented that patients with compression have a poor prognosis and there is evidence of a drastic decrease in 5-year survival, especially in cases where paralysis is present. However, the neurological prognosis will depend on the degree of neurological focality of the patient and the timeliness of intervention. The final prognosis depends on the type of tumour and the degree of invasion and malignancy of it. [6-7]

We describe the case of a patient with spinal cord compression syndrome secondary to metastatic invasion of the primary tumour and the presentation of low back pain with alarm flags. We present the article with the aim of providing tools for the management of low back pain in the ED and documenting a curious case.

#### CLINICAL CASE

A 41-year-old female patient with a history of malignant phyllodes tumor of the left breast with mesenchymal fibroepithelial component with ki 67 of 21%, managed with left radical mastectomy, chemotherapy and radiotherapy and under follow-up for 4 years, who three days previously consulted for severe headache associated with amaurosis fugax, in which the simple cranial tomography showed no alterations and it was decided to discharge her due to total resolution of the pain. She was admitted to the emergency department for a week of sudden onset of pain in the left lumbar region radiating to the ipsilateral flank associated with moderate to severe holocranial headache with no other associated symptoms.

The patient was found to be in good general condition, without neurological focality, and with no other abnormalities on physical examination. Laboratory tests on admission found no alterations suggestive of an active inflammatory or obstructive process (Table 1); Urinary tract computed tomography (CT) ruled out urolithiasis. In view of the clinical improvement, it was decided to discharge the patient again with analgesia and indication for outpatient magnetic resonance imaging of the lumbosacral spine.

She consulted again a week later due to persistent low back pain that intensified to severe

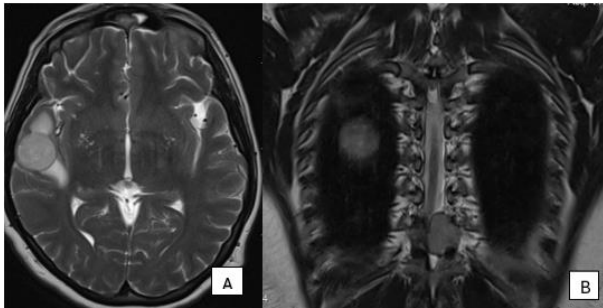
intensity; Lassegue and Bragard signs, present bilaterally. She brought an MRI of the lumbosacral spine which showed L4 - L5 and L5 - S1 disc protrusion without foraminal compromise. She was assessed by neurosurgery, who did not indicate surgical management and she was discharged once again. Four days later, she consulted for the third time due to exacerbation of pain that she was unable to control with medication and the appearance of paresthesia in both lower limbs. After two days of hospital stay, she suddenly presented with paraplegia with sensory level at T10. A new MRI of the lumbosacral spine revealed an extradural tumor lesion involving the left pedicle of T10 with a large mass effect on the spinal canal, causing critical narrowing (Figure 1).

Given this, urgent decompression was decided with osteotomy of the posterior arch of the thoracic spine in segments T9, T10 and T11 via the posterior approach and resection of the tumor lesion, without success.

Probably metastatic lesions were found in the brain and in the lower lobe of the right lung (Figure 2), so the therapeutic effort was redirected, and the patient was discharged with controls by the treating specialty and pain medicine and palliative care.



**Figure 1.** Tumour compression and invasion by metastasis of primary tumour at T9 level.



**Figure 2.** a. Shows brain metastasis in right temporal lobe and right occipital lobe; b. Metastasis of tumour in the middle lobe

## DISCUSSION

Breast phyllodes tumor is a rare fibroepithelial neoplasm that accounts for less than 1% of breast tumors. The average age of diagnosis is 45 years, being more common in women. [8] It has a highly metastatic component that predisposes to tumor seeding in places distant from the primary tumor, such as the spinal canal, predisposing patients like the one in this case to suffer from secondary spinal cord compression syndrome, which in most cases occurs through direct invasion of the primary tumor, increasing this risk by 20% if there are metastatic lesions in the spine. [9]

Regarding the incidence of spinal cord compression syndrome, it is estimated to have a prevalence of 3.4% of oncology patients; regarding the survival time of patients with this syndrome, it has been shown that it is greater in patients with breast cancer compared to other cancers, being around 114 days, compared to other cancers such as lung or prostate cancer, whose survival is estimated at around 32 days. [12] About 97% of metastatic lesions generate extradural compression, while intradural, intramedullary or leptomeningeal lesions represent only 3%. Regarding the relationship of the tumor with the medulla, this generates alterations with the epidural venous plexus, the vertebral body and the medullary canal, causing medullary oedema secondary to vascular leakage. This leads to a decrease in blood flow, which increases free radicals, inflammatory markers and prostaglandins, predisposing to spinal cord infarction and therefore to all the symptoms of neurological focalization, ischemia, irreversible tissue damage that the patient in this case presented, increasing her morbidity and mortality exponentially [8-9].

The main symptoms of spinal cord compression syndrome include low back pain in 80% of cases, as

happened with our patient; and claudication symptoms in up to 60% of cases. [10] Symptoms of lower limb weakness, sensory loss and altered bowel or bladder function, secondary to autonomic dysfunction, are also associated in 40% of patients in the literature, in most cases associated with severe motor weakness or paraplegia, as in the case in question [9-10].

In an oncology patient, low back pain should always be considered as a red flag pain (Table 2). Pathology that puts the patient's functionality and life at risk should be suspected, as treatment should be timely to avoid partially or totally irreversible spinal cord injury. [7] In general, a complete clinical history and neurological examination are useful for diagnosis. As for the use of simple vertebral radiography, this has not presented an adequate diagnostic performance, with a sensitivity of less than 70%, while magnetic resonance imaging of the spine has shown the best performance with a sensitivity of 93% and specificity of 97%, being useful for prognosis and for the selection of treatment.

Regarding the lumbar pain in our patient, the majority of metastatic infiltrates (60-80%) are located in the thoracic spine and are associated with signs of neurological focalization. [11-12] The rapidly progressive course of involvement in the case of the patient is interesting. In this case, computed tomography and magnetic resonance imaging are the indicated methods and it is suggested that in up to 46% of cases the management can be changed by magnetic resonance imaging, improving the approach and prognosis. [13-15]

In terms of management, it has been documented that patient who underwent surgery had an increased survival rate and significantly more functional preservation than patients who received radiotherapy or no intervention. In addition, the need for stronger analgesics after surgery was reduced. In addition, a recent study showed that surgery in addition to radiotherapy had a very similar outcome of improved motor function to that of patients who received radiotherapy alone. However, surgery-related complications can occur in 11-29% of patients. [16-17]

In patients with established paraplegia, the effect of surgery may be more significant, with up to four times the recovery rate to functional ambulation compared to radiotherapy. [9] In the case of a patient, surgery was attempted as an option to

decompress and avoid severe damage. However, the involvement was severe, and the spinal cord was infarcted at the thoracic level.

## CONCLUSION

Oncological emergencies are a group of frequent pathologies in patients with neoplastic disease, including spinal cord compression syndrome. It is important to systematically and timely search for differential diagnoses to the usual ones in oncological patients in order to perform interventions and timely diagnosis and early treatment to avoid neurological sequelae and decrease the survival rate. Treatment is still debated; however, surgery or radiotherapy is an important determinant of the patient's prognosis. Individualized management should also be given according to the prognostic factors of each patient.

## REFERENCES

1. Instituto Nacional del Cáncer. Estadísticas del cáncer [Internet]. 2015 [citado el 17 de julio de 2024]. Disponible en: <https://www.cancer.gov/espanol/cancer/naturaleza/estadisticas>
2. Zuluaga M, Roldan MD, Baena F, Upegui S. Generalidades de las principales urgencias en el paciente oncológico: Revisión de tema. *Med UPB*. 2022;41(1):38-50. DOI:10.18566/medupb.v41n1.a06
3. Lawton AJ, Lee KA, Cheville AL, Ferrone ML, Rades D, Balboni TA, et al. Assessment and management of patients with metastatic spinal cord compression: A multidisciplinary review. *J Clin Oncol* [Internet]. 2019;37(1):61-71. Disponible en: <http://dx.doi.org/10.1200/jco.2018.78.1211>
4. Halfdanarson TR, Hogan WJ, Madsen BE. Emergencies in hematology and oncology. *Mayo Clin Proc*. 2017;92(4):609-41
5. Paulino Pereira NR, McLaughlin L, Janssen SJ, et al: The SORG nomogram accurately predicts 3- and 12-months survival for operable spine metastatic disease: External validation. *J Surg Oncol* 115:1019-1027, 2017
6. Bautista-Hernández MY, Medina-Villaseñor EA. Síndrome de compresión medular por cáncer. *Gac Mex Oncol* [Internet]. 2011 [citado el 17 de julio de 2024];10(5):295-302. Disponible en: <https://www.elsevier.es/es-revista-gaceta-mexicana-oncologia-305-articulo-sindrome-compresion-medular-por-cancer-X1665920111894309>
7. Corti M, De Carolis L, Centioni A, Velásquez J, Rizzo C, Narbaitz M. Síndrome de compresión medular como forma de presentación de linfoma no Hodgkin epidural espinal primario. Descripción de un caso y revisión de la literatura. *Neurol Argent* [Internet]. 2017;9(2):120-4. Disponible en: <http://dx.doi.org/10.1016/j.neuarg.2016.04.006>
8. Parker SJ, Harries SA. Phylloides tumours. *Postgrad Med J* [Internet]. 2001 [citado el 24 de julio de 2024];77(909):428-35. Disponible en: <https://pubmed.ncbi.nlm.nih.gov/11423590/>
9. Romero P, Manterola A, Martínez E, Villafranca E, Domínguez MA, Arias F. Compresión medular. *An Sist Sanit Navar* [Internet]. 2004 [citado el 24 de julio de 2024];27:155-62. Disponible en: [https://scielo.isciii.es/scielo.php?script=sci\\_arttext&pid=S1137-66272004000600015](https://scielo.isciii.es/scielo.php?script=sci_arttext&pid=S1137-66272004000600015)
10. Sacoto García XE, Guillén Núñez R, Juárez Lemus A. Evolución de la lumbalgia en el paciente oncológico tratado con manejo intervencionista del dolor. *Rev Soc Esp Dolor* [Internet]. 2021 [citado el 25 de julio de 2024];28(2):76-81. Disponible en: [https://scielo.isciii.es/scielo.php?script=sci\\_arttext&pid=S1134-80462021000300005](https://scielo.isciii.es/scielo.php?script=sci_arttext&pid=S1134-80462021000300005)
11. Quint DJ. Indications for emergent MRI of the central nervous system. *JAMA*: 2000: 253-283.
12. Santos C, Donoso R, Ganga M, Eugenin O, Lira F, Santelices jp. dolor lumbar: revisión y evidencia de tratamiento. *Rev médica Clín Las Condes* [Internet]. 2020;31(5-6):387-95. Disponible en: <http://dx.doi.org/10.1016/j.rmclc.2020.03.008>
13. Spinal metastases and metastatic spinal cord compression. National Institute for Health and Care Excellence; 2023.
14. Boussios S, Cooke D, Hayward C, Kanellos FS, Tsiouris AK, Chatziantoniou AA, et al. Metastatic spinal cord compression: Unraveling the diagnostic and therapeutic challenges. *Anticancer Res* [Internet]. 2018;38(9):4987-97. Disponible en: <https://ar.iijournals.org/content/anticancer/38/9/4987.full.pdf>
15. Federspiel C, Morgen SS, Suppli MH, Kamby C, Kelsen J, Gehrchen M. Treatment of metastatic spinal cord compression. *Ugeskr Laeger* [Internet]. 2021 [citado el 17 de julio de 2024];183(33). Disponible en: <https://pubmed.ncbi.nlm.nih.gov/34477101/>
16. Kuah T, Vellayappan BA, Makmur A, Nair S, Song J, Tan JH, et al. State-of-the-art imaging techniques in metastatic Spinal Cord Compression. *Cancers (Basel)* [Internet]. 2022 [citado el 17 de julio de 2024];14(13):3289. Disponible en: <https://pubmed.ncbi.nlm.nih.gov/35805059/>
17. DePalma MG. Red flags of low back pain. *JAAPA* [Internet]. 2020 [citado el 26 de julio de 2024];33(8):8-11. Disponible en: <https://pubmed.ncbi.nlm.nih.gov/32740106/>



# A comparison of subperiosteal vs subdural drainage in the treatment of chronic subdural haemorrhage

Sajag Gupta

UPUMS SAIFAI, INDIA

## ABSTRACT

**Introduction.** Symptomatic chronic subdural haemorrhages (CSDH) continue to be one of the most common diagnoses in modern neurosurgery. The current standard procedure for symptomatic CSDH is a burr-hole craniostomy with irrigation and the installation of close-system drainage. The purpose of this study is to conduct a direct comparison of two surgical procedures for the treatment of symptomatic CSDH that are effective in prior studies. Our main goal was to compare the efficacy of placing a subperiosteal drain (SPD) and a subdural drain (SDD) after a single burr-hole craniostomy and irrigation and to show any significant differences in terms of overall surgical complications, functional outcome and mortality rate at three months, and complications at six months.

**Materials and methods.** From August 2022 to December 2023, the study was conducted in the Department of Neurosurgery at a tertiary care centre, with a total of 25 patients in both treatment groups.

**Results.** Overall, there was no statistically significant difference between the two groups in terms of patient general characteristics, pre-and post-operative symptoms, Markwalder grades, postoperative hematoma volume and recurrence, mortality, and functional outcome at discharge and at three months follow-up. Despite not reaching statistical significance, we found a decreased risk of surgical complications, particularly post-operative cerebral haemorrhage, with SPD system implementation.

**Conclusions.** According to our findings, both therapy modalities are extremely successful in the treatment of CSDH. therapy with a single burr-hole craniostomy, irrigation, and implantation of the SPD system, on the other hand, has a reduced overall surgical complication rate and can be regarded as a therapy of choice for the management of symptomatic CSDH.

## INTRODUCTION

The frequency of patients presenting with symptomatic chronic subdural haemorrhage (CSDH) has steadily increased in recent years, owing to increased life expectancy, particularly in developing nations (1, 6). There have only been a few class (II) evidence articles in the literature on the treatment of CSDH to yet. Burr-hole craniostomy coupled with irrigation and implantation of a closed-drainage system is the primary surgical treatment of choice for symptomatic CSDH (10, 12). Santarius et al. concluded in 2009 that placing a subdural drain (SDD) after burr-

## Keywords

subperiosteal,  
subdural drainage,  
chronic subdural  
haemorrhage



Corresponding author:  
Sajag Gupta

UPUMS SAIFAI, India

sajag.gupta@yahoo.com

Scan to access the online version



hole evacuation of CSDH was linked with decreased recurrence and death. Recent investigations have reported on a significantly less invasive approach that involves the insertion of a subperiosteal drain (SPD) rather than the traditional SDD. (3, 5, 14). This was looked at since placing an SDD on the cortical brain surface might result in problems including haemorrhage, seizures, and surgical-site infections like empyema. SPD installation has been indicated as the therapy of choice for patients with a predicted high risk of problems, particularly those over 80 years old, with a clear trend towards lower mortality and complications (3). Kaliaperumal et al.'s single-center prospective randomised trial comparing the results of SDD and SPD in 2012 found statistically significant modified Rankin Score (mRS) measures, with improved outcomes in the SPD group at three and six months of follow-up. The current study compared SDD and SPD implantation for the treatment of CSDH to examine recurrence rates and overall results in terms of surgical complications, functional outcomes, and death in both groups.

#### MATERIALS AND METHODS

The goal of this prospective interventional trial was to compare patients who received SDP versus SDD drain insertion for the treatment of symptomatic CSDH. The primary goal was to assess effectiveness and detect differences between the two groups in total surgical adverse events, functional outcomes, and death. We recruited 50 patients (25 each group) who met the inclusion and exclusion criteria between August 2022 and December 2023, and we performed the surgical operations using the standardised methodology specified below. Prior to surgery, general patient parameters such as age, gender, concomitant medical problems, medicines, and other risk factors were evaluated. Pre-operative symptoms, admission Glasgow Coma Scale (GCS) and Markwalder scores, as well as radiological data (hematoma volume) from pre-operative CT scans, were also examined. The XYZ/2 formula (7) was used to obtain haemorrhage size and volume estimates. The following variables were recorded: post-operative signs, neuroradiological abnormalities on post-operative CT at 24 hours, instances of surgical complications and repeat operations, Glasgow Outcome Score (GOS), Markwalder score, and mortality during hospitalization. GOS scores, repeat

CT results, and death rates were examined after three months of follow-up. A favourable result was defined as a decrease in pre-operative symptoms, a Markwalder grade of 0 or 1, minimal surgical morbidity and mortality, and a GOS score of 4 or above. In a symptomatic patient, a recurrent hematoma was defined as a thickness of more than 10 mm. Any patient who came with a recurrence haemorrhage within three months of the scheduled follow-up session was documented and treated accordingly.

#### STUDY INCLUSION AND EXCLUSION CRITERIA

This study's inclusion criteria were an age of 80 years, the presence of symptomatic CSDH on plain CT imaging, and an admission Glasgow Coma Scale (GCS) of 6 or higher (with a motor score of at least 4). Exclusion criteria included terminal disease, pregnancy, a history of prior CSDH surgery, or an entrance GCS of 6.

Outline of peri-, intra-, and post-operative management Each patient or their immediate family members or carers provided informed permission prior to surgery. Anticoagulants and antiplatelet medicines were stopped during surgery, and fresh frozen plasma with intravenous (IV) vitamin K was given to restore normal coagulation characteristics.

Patients who showed with seizures upon arrival were given antiepileptic medicines. At both research sites, these perioperative parameters were standardized for both patient groups. The patient was always positioned supine and given general anesthesia, with the head resting on a rubber horseshoe ring. The incision site was noted and cleansed with povidone iodine before being draped in sterile surgical drapes. Each patient received a single injection of antibiotic prophylaxis shortly before the skin incision. At the location of maximum blood thickness, a single burr hole was created, with the craniostomy measuring at least 10 mm× 10 mm in diameter. The dura mater was coagulated and expanded wide enough to accommodate the burr-hole. Intraoperative subdural irrigation with body temperature normal saline was performed until the effluent was reasonably clear but not completely eliminated. The closed drainage system was installed in accordance with normal procedure at each facility. A passive corrugated Redivac catheter was put across the burr-hole beneath the galea for SPD

systems. A Jacques catheter was navigated through the burr-hole and carefully positioned in the subdural area for SDD systems. Each drain was attached to a passive collection system by a minor skin incision posterolateral to the burr-hole, with no suction force used. To reduce intracranial air collection, the subdural space was filled with body-temperature saline before sealing the skin incision. The drainage system was installed beneath the head. After a repeat brain CT scan within 24 hours, the drain was removed.

### STATISTICAL EVALUATION

SPSS software for Windows version 21.0 was used to examine the data. Every variable was expressed as a mean standard deviation (X SD). The t-test and the chi-square test were used to assess the studied parameters. To establish statistical differences in total surgical complications, functional outcomes, and death, the determined sample size was 25 patients per group (power of 80%). The significance threshold was set at  $P < 0.05$ .

### RESULTS

**General demographics and patient characteristics** A total of 25 subjects per group were eligible for analysis. The SPD group included 18 males (72%) and 7 females (28%), while the SDD group contained 17 males (68%) and 8 females (32%). The mean age was 67 years in the SPD group and 71 years in the SDD group. As depicted in Table 1, there were no significant differences in mean age or gender between the groups ( $P > 0.05$ ). In the SPD group, the CSDHs included 10 right-sided, 14 left-sided, and one bilateral. The SDD group included 12 right-sided and 13 left-sided CSDHs. ONE patients (4%) in the SPD group and TWO (8%) in the SDD group were on chronic oral antiplatelet therapy. Only two subject (8%) in the SPD group was on anticoagulation. The most frequent associated comorbid conditions in the SPD versus SDD groups, respectively, were hypertension (60% versus 72%), type II diabetes mellitus (24% versus 24%), ischemic heart disease (16% versus 20%), chronic kidney disease (4% versus 4%), chronic liver disease (4% versus 4%), and chronic alcoholism (0% versus 4%). There were no significant differences regarding these clinical parameters between the SPD and SDD groups ( $P > 0.05$ ).

**Table 1.** General demographics and patient characteristics between both groups (chi-square test).

	Subperiosteal Drain	Subdural Drain	P-value
No. of Patients	25	25	
Age (mean)	67	71	NS
Sex Male	18 (72%)	17 (68%)	NS
Female	7 (28%)	8 (32%)	
Right SDH	10	12	NS
Left SDH	14	13	NS
Bilateral SDH	1	0	NS
on anti-platelet	1 (4%)	2(8%)	NS
on anti-coagulation	2 (8%)	0	NS
Co-morbidities			
HPT	15 (60%)	18(72%)	NS
IHD	4 (16%)	5 (20%)	NS
DM	6 (24%)	6 (24%)	NS
CKD	1 (4%)	1 ( 4%)	NS
CLD	1(4%)	1 (4%)	NS
Previous Stroke	1 (4%)	2 (8%)	NS
Chronic Alcoholic	0	1 (4%)	NS

**Pre- and post-operative symptoms** All patients in both study groups were assessed for presenting symptoms and signs on admission, and subsequently reassessed and documented to ascertain post-operative outcomes upon discharge. As shown in Table 2, a majority of patients presented with GCS scores of 13–15, including 22 patients (88%) in the SPD group and 23 (92%) in the SDD group. Two patients (8%) in the SPD group and one (4%) in the SDD group presented with a GCS of 9–12. One patient (4%) from each group presented with a GCS of 8. The most common symptoms at admission in the SPD versus SDD groups, respectively, included headache (88% versus 84%), altered sensorium (92% versus 88%), asymmetrical reflexes (96% versus 84%), hemiparesis (60% versus 64%), aphasia (32% versus 28%), hemiplegia (4% versus 0%), and seizures (4% versus 4%). Overall, there were no significant differences in pre-operative symptoms and signs between the two groups ( $P > 0.05$ ). All patients were re-examined postoperatively with regard to presenting symptoms. As shown in Table 2, all patients in both groups fully recovered post-operatively from the initial symptoms and signs of aphasia, altered sensorium, and asymmetrical reflexes. Both groups showed significant improvements in headache (from 88% to 20% in the

SPD group and from 84% to 12% in the SDD group) and hemiparesis (from 60% to 12% in the SPD group and from 64% to 4% in the SDD group) ( $P < 0.05$ ). No patients in the SPD group and SDD group developed persistent hemiplegia postoperatively. There were no detectable differences in seizure control post-operatively between the groups (0% versus 4%); all patients who presented with pre-operative seizures had residual focal seizures post-operatively, which were controlled by optimisation of antiepileptic drugs. Overall, no statistically significant differences were demonstrated in post-operative symptoms between the groups ( $P > 0.05$ ).

**Table 2.** Pre-operative and post-operative symptoms and signs between both groups (chi-square test).

	Subperiosteal Drain	Subdural Drain	P-value (between groups)
<b>Pre-op Symptoms</b>			
GCS 13-15	22 (88%)	23 (92%)	
GCS 9-12	2 (8%)	1(4%)	
GCS 3-8	1 (4%)	1(4%)	
<b>Symptoms &amp; Signs on Admission</b>			
Hemiparesis	15(60%)	16(64%)	NS
Hemiplegia	1(4%)	0	NS
Aphasia	8(32%)	7(28%)	NS
Headache	22(88%)	21(84%)	NS
Seizures	0	1(4%)	NS
Altered Sensorium	23(92%)	22(88%)	NS
Reflex Asymmetry	24(96%)	21(84%)	NS
<b>Symptoms and Signs at Discharge</b>			
Hemiparesis	3(12%)	1(4%)	NS
Hemiplegia	0	0	
Aphasia	0	0	
Headache	5(20%)	3(12%)	NS
Seizures	0	1	
Altered Sensorium	0	0	
Reflex Asymmetry	0	0	

Markwalder grade Markwalder grades at admission and upon discharge were used to assess the clinical courses of patients in both groups. As shown in Table 3, the majority of patients presented with Markwalder grade 2 on admission, including 19 patients in the SPD group and 14 in the SDD group. Five subjects in the SPD group and 10 in the SDD group had Markwalder grade 1 on admission. One

patient (4%) in each group had Markwalder grade 3 on admission. Post-operatively, the patients in both groups were examined and reassigned post-operative Markwalder grades. All patients in each group with admission Markwalder grade 1 improved to Markwalder grade 0 by the time of discharge. Therefore, the majority of patients demonstrated good post-operative Markwalder scores (grade 0 or 1) at discharge, for a total of 23 patients (92%) in the SPD group and 24 (96%) in the SDD group. Table 5 shows that the calculated mean admission Markwalder score was 1.72 for the SPD group and 1.65 for the SDD group. Upon discharge, the mean Markwalder scores in both groups had improved significantly (from 1.72 to 0.49 in the SPD group and from 1.65 to 0.51 in the SDD group). These changes were statistically significant within each group, respectively ( $P < 0.05$ ). However, there were no significant differences in mean Markwalder scores between the SPD and SDD groups upon admission and at discharge ( $P > 0.05$ ).

**Table 3.** Markwalder grades on admission and upon discharge comparing mean grade within and between both groups (independent t-test).

Markwalder Grade	Subperiosteal Drain (SPD)	Subdural Drain (SDD)	P-value (between groups)
<b>On admission</b>			
MW 0/ MW 1 /MW 2/ MW 3 /MW 4	0/5/19/1/0	0/10/14/1/0	
Mean Markwalder Grade	1.72	1.65	NS
<b>On discharge</b>			
MW 0 /MW 1 /MW 2 /MW 3 /MW 4	20/3/2/0/0	21/3/1/0/0	
Mean Markwalder	0.49	0.51	NS

Hematoma volume The mean hematoma volume was estimated pre-operatively, within 24 hours post-operatively, and at three months of followup based on CT findings; Table 4 illustrates that there were no significant differences ( $P > 0.05$ ). However, within the respective groups, hematoma volumes were significantly reduced compared to pre-operative imaging ( $P < 0.05$ ). In the SPD group, the mean pre-operative hematoma volume decreased significantly from  $118.92 \times 10^3 \text{ mm}^3$  to  $9.10 \times 10^3 \text{ mm}^3$  during the

post-operative period ( $P < 0.05$ ). Significant changes were also noted in mean hematoma volume from the post-operative CT ( $9.10 \times 10^3 \text{ mm}^3$ ) to the three-month follow-up CT ( $3.87 \times 10^3 \text{ mm}^3$ ) ( $P < 0.05$ ). Similar results were found for the SDD group, in which the mean pre-operative hematoma volume significantly decreased from  $118.81 \times 10^3 \text{ mm}^3$  to  $8.30 \times 10^3 \text{ mm}^3$  in the post-operative period, with a further drop to  $3.62 \times 10^3 \text{ mm}^3$  on the three-month follow-up CT ( $P < 0.05$ ).

**Table 4.** Mean hematoma volume in both groups pre-operatively, post-operatively and at 3 months follow-up (independent t-test).

Mean Hematoma volume (* $10^3$ )	Subperiosteal Drain (SPD)	Subdural Drain (SDD)	P-value (between groups)
Pre-op	118.92	118.81	NS
Post-op (24 hours)	9.10	8.30	NS
Follow-up (3 months)	3.87	3.62	NS

**Overall surgical complications** The overall surgical complications were categorised into intracerebral hematoma (ICH), recurrent hematoma, surgical-site infection, and tension pneumocephalus. Table 5 shows that the overall complication rate in the SPD group was 8%, with two patients who developed recurrent hematoma during follow-up, requiring re-evacuation surgery via burr-hole craniostomy and SPD drainage. None of the patients developed ICH as a post-operative complication in this group. The two recurrent hematomas were a direct result of restarting anticoagulant treatment by the cardiology team within two weeks of surgery. In the SDD group, the overall complication rate was 8%, with two patients (8%) developing ICH while hospitalised, and one (3.3%). Both patients with ICH underwent craniotomy and clot evacuation while hospitalised, and a repeat burr-hole craniostomy was performed for the other patient with recurrent CSDH. However, as shown in Table 5, this did not translate into a significant statistical difference ( $P > 0.05$ ). There was also no detectable difference between the groups in post-operative recurrent hematomas ( $P > 0.05$ ). Additionally, in terms of the overall surgical complication rate, there was no significant difference between the SPD and SDD groups ( $P > 0.05$ ). None of

the patients in either group developed post-operative seizures, surgical-site infections, or tension pneumocephalus.

**Table 5.** Overall surgical complications and mortality between both groups (independent t-test).

	Subperiosteal Drain (SPD)	Subdural Drain (SDD)	P-value (between groups)
<b>Repeat surgeries while hospitalised</b>			
Re-evacuation	0	0	
Craniotomies	0	2(8%)	NS
<b>Repeat surgeries after discharge</b>			
Re-evacuation	2(8%)	0	NS
Craniotomies-	0	0	
Intracerebral Hematoma	0	2(8%)	NS
Recurrent Hematoma	2(8%)	0	NS
Surgical Site Infection	0	0	
Tension Pneumocephalus	0	0	
Seizures	0	0	
<b>Overall Surgical Complications</b>	2 (8%)	2(8%)	NS

**Functional outcomes and mortality** Functional outcomes were measured with the Glasgow Outcome Score and compared between the two groups. A good functional outcome was defined as a GOS of 4 or higher (Table 6). In the SPD group at discharge, a total of 22 patients had a GOS of 5, and 3 patients had a GOS of 4. The mean GOS at discharge was 4.8 in the SPD group. In the SDD group, 22 subjects had a GOS of 5, one had a GOS of 4, and two had a GOS of 3 at discharge. The mean GOS at discharge was calculated at 4.7 for the SDD group. There was no significant difference between the two groups in GOS scores at discharge ( $P > 0.05$ ). At three months of follow-up in the SPD group, 24 (96%) patients had a GOS of 5, while one (4%) had a GOS of 4. In the SDD group, 24 patients (96%) had a GOS of 5 and 1 (4%) had a GOS of 4. No significant difference was noted in mean GOS scores at three months. The mean GOS was 4.9 in the SPD group and 4.9 in the SDD group ( $P > 0.05$ ). There was no

mortality throughout the entire study duration in either group.

**Table 6.** Functional outcome assessment between both groups.

Glasgow Outcome Score	Subperiosteal Drain (SPD)	Subdural Drain (SDD)	P-value (between groups)
<b>Outcome at discharge</b>			
GOS 5 GOS 4 GOS 3 GOS 2 GOS 1	22/3/0/0/0	22/1/2/0/0	
Mean GOS (at discharge)	4.8	4.7	NS
<b>Outcome at 3 months follow-up</b>			
GOS 5 GOS 4 GOS 3 GOS 2 GOS 1	24/1/0/0/0	24/1/0/0/0	
Mean GOS (at 3 months follow-up)	4.9	4.9	NS
<b>Mortality</b>	0	0	

## DISCUSSION

There have been an increasing number of studies published in recent years on the efficacy of placing SPD devices following burr-hole craniostomy for the treatment of symptomatic CSDH. SPD insertion has been rated a technically simple solution that is both safer and more successful than traditional SDD insertion. The purpose of our study was to conduct a prospective direct comparison of these two surgical methods, with the realistic objective of evaluating their efficacy and discovering any variations in total surgical complications, functional outcomes, and death. There were no statistically significant changes in patient data, mean haemorrhage size, concomitant conditions, or pre-operative complaints. When compared to traditional SDD, we discovered that SPD implantation was similarly effective and resulted in a reduced risk of surgical complications. This was indicated by a considerable decrease in post-operative symptoms and an improvement in Markwalder grades within each group. We discovered a decreased risk of post-operative ICH, which can be induced by unintended insertion of the SDD into the brain, due to the small invasiveness of the SPD method, which includes no

contact with the brain parenchyma. However, this difference did not achieve statistical significance. As previously reported (3, 14), we found no significant differences in the decrease of postoperative seizures or recurrent haemorrhages in the SPD team. The comparatively small sample size in our study hampered these conclusions somewhat. We also discovered that recurring hematomas were closely associated to the resumption of anticoagulant therapy throughout the postoperative period. The authors of three distinct trials that yielded class (III) recommendations discovered that restarting oral anticoagulants 72 hours after surgery was safe and did not increase the incidence of post-operative cerebral haemorrhage (4, 9, 13). As a result, further research is needed to provide better recommendations for beginning anticoagulant therapy during the post-operative period. A GOS score of 4 or above at three months of follow-up indicated favourable functional results. Other trials with identical follow-up periods revealed comparable satisfactory results three months after surgery (3, 14). In line with these findings, our study found that after three months, there was no discernible difference between the two groups. Yet, Kaliaperumal et al. (2012) discovered that patients treated with the SPD system had considerably superior modified Rankin scores after six months. As a result, a longer period of follow-up would be required to show any significant changes in the overall functional results of the patients in our research.

## CONCLUSION

Our findings show that the SPD and SDD methods are both safe, technically simple, and very successful in the treatment of CSDH, with no statistically significant differences in terms of total surgical complications, functional results, or death. The SPD group had a lower risk of post-operative ICH due to unintentional subdural drain insertion in the brain parenchyma, but the difference was not statistically significant. Because our study had a limited sample size and a short follow-up time, we urge a future prospective, randomised, multicenter investigation with a bigger sample size and a longer follow-up period (at least six months) to further corroborate our findings .

## REFERENCES

1. Baechli H, Nordmann A, Bucher HC, et al. Demographics and prevalent risk factors of chronic subdural haematoma: results of a large single-center cohort study. *Neurosurg Rev.* 2004;27(4):263–266. <https://dx.doi.org/10.1007/s10143-004-0337-6>.
2. Belkhair S, Pickett G. One versus double burr holes for treating chronic subdural hematoma meta-analysis. *Can J Neurol Sci.* 2013;40(1):56–60. <https://dx.doi.org/10.1017/S0317167100012956>
3. Bellut D, Woernle CM, Burkhardt JK, Kockro RA, et al. Subdural drainage versus subperiosteal drainage in burr-hole trepanation for symptomatic chronic subdural hematomas. *World Neurosurg.* 2012;77(1):111–8. <https://dx.doi.org/10.1016/j.wneu.2011.05.036>.
4. Chari A, Clemente Morgado T, Rigamonti D. Recommencement of anticoagulation in chronic subdural haematoma: a systematic review and meta-analysis. *Br J Neurosurg.* 2014;28:2–7. <https://dx.doi.org/10.3109/02688697.2013.812184>.
5. Gazzeri R, Galarza M, Neroni M, Canova A, et al. Continuous subgaleal suction drainage for the treatment of chronic subdural haematoma. *Acta Neurochir (Wien).* 2007;149(5):487–493; discussion 93. <https://dx.doi.org/10.1007/s00701-007-1139-8>.
6. Gelabert-Gonzalez M, Iglesias-Pais M, GarciaAllut A, et al. Chronic subdural haematoma: surgical treatment and outcome in 1000 cases. *Clin Neurol Neurosurg.* 2005;107(3):223–229. <https://dx.doi.org/10.1016/j.clineuro.2004.09.01>
7. Hassan KS, Metin G, Fazil G. The value of XYZ/2 technique compared with computer-assisted volumetric analysis to estimate the volume of chronic subdural hematoma. *Stroke.* 2005;36:998–1000. <https://dx.doi.org/10.1161/01.STR.0000162714.46038.0f>.
8. Kaliaperumal C, Khalil A, Fenton E, et al. A prospective randomised study to compare the utility and outcomes of subdural and subperiosteal drains for the treatment of chronic subdural haematoma. *Acta Neurochir (Wien).* 2012;154(11):2083–2088. <https://dx.doi.org/10.1007/s00701-012-1483-1>
9. Kawamata T, Takeshita M, Kubo O, et al. Management of intracranial hemorrhage associated with anticoagulant therapy. *Surg Neurol.* 1995;44(5):438–442. [https://dx.doi.org/10.1016/0090-3019\(95\)00249-9](https://dx.doi.org/10.1016/0090-3019(95)00249-9).
10. Lee JK, Choi JH, Kik CH, Lee HK, et al. Chronic subdural hematomas: A comparative study of three types of operative procedures. *J Korean Neurosurg Soc.* 2009;46:210–214. <https://dx.doi.org/10.3340/jkns.2009.46.3.210>.
11. Santarius T, Kirkpatrick PJ, Ganesan D, et al. Use of drains versus no drains after burr-hole evacuation of chronic subdural haematoma: a randomised controlled trial. *Lancet.* 2009 Sep 26;374(9695):1067–1073. [https://dx.doi.org/10.1016/S0140-6736\(09\)61115-6](https://dx.doi.org/10.1016/S0140-6736(09)61115-6).
12. Weigel R, Schmiedek P, Krauss JK. Outcome of contemporary surgery for chronic subdural haematoma: evidence based review. *J Neurol Neurosurg Psychiatry.* 2003;74(7):937–943. <https://dx.doi.org/10.1136/jnnp.74.7.937>.
13. Yeon JY, Kong DS, Hong SC. Safety of early warfarin resumption following burr-hole drainage for warfarin-associated subacute or chronic subdural hemorrhage. *J Neurotrauma.* 2012; 29 (7): 1334–1341. <https://dx.doi.org/10.1089/neu.2011.2074>.
14. Zumofen D, Regli L, Levivier M, Krayenbuhl N. Chronic subdural hematomas treated by burr hole trepanation and a subperiosteal drainage system. *Neurosurgery.* 2009;64:1116–1121. <https://dx.doi.org/10.1227/01.NEU.0000345633.45961.BB>.



# Neuronavigational assisted ventriculo-peritoneal shunt surgery in the idiopathic intracranial hypertension (IIH) patients. Institutional experiences

Poonia Nemi Chand, Jain Surendra, Poonia Hardika, Poonia Dev, Kumar Chitresh, Poonia Sania

Neuro Care Hospital & Research Centre Pvt. Ltd. Jaipur, INDIA  
S.M.S. Medical College Jaipur, INDIA  
JNU, Jaipur, INDIA

## ABSTRACT

**Object.** Lumboperitoneal shunt are commonly used for idiopathic intracranial hypertension (IIH) because of the difficulty of insertion of Ventriculoperitoneal (VP) shunt into normal or small-sized ventricles. The authors showed their experience with VP shunts for IIH with the help of a Neuronavigation system.

**Methods.** This is a retrospective study of 16 patients of IIH in whom Neuronavigation was used to guide the insertion of a shunt in the ventricle for IIH at our institution. A trial of either acetazolamide or steroid therapy had failed in all patients.

**Results.** Shunt placement was successful and uncomplicated in each case except for one patient in which shunt failure occurred (6.25%) post-operatively. Outcomes were assessed at 12 months which were favourable regarding symptoms 13 cases (81.25%) got relief in headaches and 4 patients (25%) showed improvement in vision. Misplacement of the shunt occurred in one case postoperatively and Shunt failure occurred in 2 cases (12.50%) during the follow-up period due to ventricular end obstruction. These three patients required shunt revision. Progression of the visual deficit did not occur.

**Conclusion.** Our results suggest that the Neuronavigation-guided ventriculoperitoneal shunt is a good alternative to Lumboperitoneal shunt in IIH patients.

## INTRODUCTION

Benign intracranial hypertension (BIH) also known as Idiopathic intracranial hypertension (IIH) or Pseudotumor cerebri is a disorder of raised ICP in the absence of any secondary pathology in radiological imaging (1,2,3). Empty Sella syndrome, lateral sinus collapse, flattened globes and fully unfolded optic nerve sheaths are signs of increased intracranial pressure, as seen in imaging.

This disorder is more common occurred in young (mainly in childbearing age), obese and female sex (4,5).

## Keywords

hydrocephalus,  
idiopathic intracranial  
hypertension,  
neuronavigation,  
ventriculoperitoneal shunt



Corresponding author:  
Jain Surendra

S.M.S. Medical College Jaipur,  
India

simplesogani@gmail.com

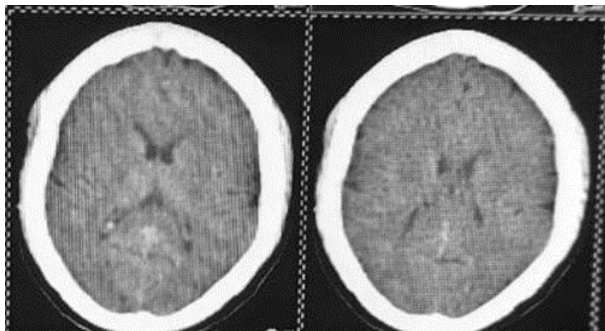
Scan to access the online version



The signs and symptoms of intracranial hypertension include headache, tinnitus (pulsatile tinnitus), transient visual obscurations and visual loss. Neurological examination is normal except for diplopia, sixth cranial nerve paresis and papilledema. Visual loss is the major morbidity in IIH.

There are multiple treatment options for IIH. Start as conservative to end as a surgical procedure. The initial treatment of choice is Acetazolamide which provides symptomatic relief (6,7,8,9). Steroid therapy is also used when patients do not get relief from medicine, conservative treatment fails, or significant visual loss, progressive visual loss or severe headache then surgical treatment is needed. Possible surgical modalities are Optic nerve sheath fenestration and CSF diversion procedure. Two CSF diversion methods exist (1) LP shunting and (2) VP shunt placement. LP shunt placement is a more commonly performed procedure than VP shunt because difficulty in the insertion of the catheter into small-size or normal-size ventricles (5,10,11,12,13,14).

However, after the development of IGN (image-guided Neuronavigation) the introduction of the catheter into the ventricle is not difficult. Our study is based on Neuronavigation-based VP shunt in IIH patients.



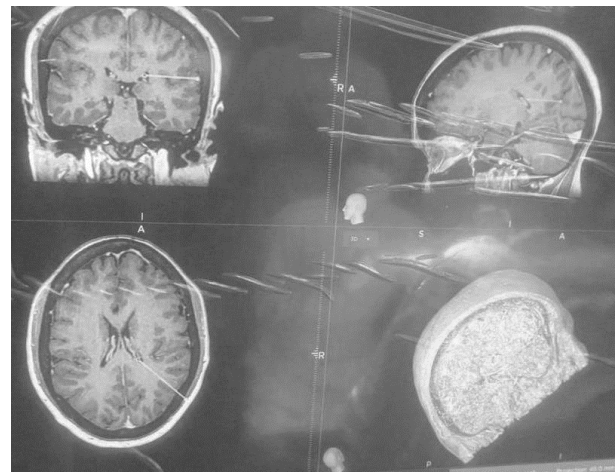
**Figure 1.** Pre-operative NCCT Head (A & B) and Post Operative NCCT Head (C & D).

## WHAT IS NEURONAVIGATION AND STEPS

Neuronavigation is a tool used during brain surgery to locate where in the brain the surgeon is working i.e. intraoperative orientation. The navigation is based on preoperative MR /CT images, which are merged to build a 3D view that is displayed on a computer workstation. Neuronavigation are techniques which help Neurosurgeons to precisely localize the different intracerebral pathology.

Each Neuronavigation system follows the same steps for the surgical procedure:

- Obtaining preoperative images (CT, MRI) and images can be transferred in the navigation system via computer network)
- Patient registration,
- Intraoperative localization;
- Intraoperative control;
- Obtaining intraoperative images and fusion with preoperative ones;
- Visualization and surgery



**Figure 2.** Planning of trajectory of shunt before surgery with help neuronavigation in axial, sagittal and coronal views.

## MATERIAL AND METHODS

This is a retrospective study of 16 patients of IIH in whom VP shunting was done with the help of Neuronavigation at Neurocare Hospital Jaipur from 2018 to 2023. The diagnosis of IIH was made by an author who is a neurosurgeon, based on the history, clinical symptoms, neurological examination and imaging studies. ICP measurements were also done in some cases when available. Fundus examination was done by an ophthalmologist in every case. Laboratory evaluation in IIH patients is normal except for increased intracranial pressure. All

patients had taken medical treatment including steroids and acetazolamide, but did not get relief. All shunts were placed under the guidance of the Neuronavigation system. Shunt placement grading is done in each case by CT scan.



**Figure 3.** The navigation stylet is used as the catheter trocar.

#### INCLUSION CRITERIA

All patients of IIH with failed response to medical treatment.

#### SURGICAL TECHNIQUE

After explaining the pros and cons, the patient is shifted to the operation theatre. After general anesthesia, patients are placed in a supine position with their heads tilted toward the opposite side. After system registration, the surgical plan was made from the entry site to the final tip position of the catheter (figure 2). After proper sterilization, the entry point is marked with the help of Neuronavigation. C-shaped skin flap raised and burr hole made. The peritoneal catheter is passed subcutaneously and placed in the abdomen. Dura is coagulated. The proximal end of the shunt was inserted with the help of a catheter stylet of the Neuronavigation system (figure 3) and confirmed in all planes (axial, coronal and sagittal). We are using the MEDTRONIC optical system. Once the proximal catheter is in place, it is connected to the peritoneal end. Both wounds closed in layers.

Patients attended a follow-up visit at 1 month, 6 month and 12 months and asked about vision, headache and any other symptoms. No patient was lost to follow-up.

#### RESULTS

In our study, we included 16 patients in whom this procedure was performed between 2018 and 2023. There were 12 females and 4 males; their mean age was 33.06 years (range 22–44 years). Most of the women were in the reproductive age group and the mean BMI was 26.42 that is most of the patients were overweight.

In our study symptoms and signs common in most of the patients were headache, blurred vision and tinnitus. Diplopia and visual loss were less common. Headache and blurred vision were usually presenting symptoms. Papilloedema and sixth nerve paresis are also a common finding in IIH.

#### ASSESSMENT OF EFFECTIVENESS

Postoperatively, CT brain was done in every case and assessment of the tip of ventricular catheter for grading of shunt placement was done.

Grade I; Catheter tip position free-floating in CSF, away from ventricular wall or choroid plexus (Figure 1).

Grade II; Catheter tip touching choroid plexus or ventricular wall.

Grade III; Tip within parenchyma or failure to reach the intraventricular space.

Grade 1 shunt placement was found in 11 (68.75%) patients, and grade II shunt and grade III shunt placement were found in 4 (25%) and 1 (6.25%) patients respectively. According to shunt placement the shunt failure rate was 6.25%. Shunt revision was done in grade III shunt placement.

Outcome- Outcomes were assessed at 12 months which were favorable regarding symptoms. 13 cases (81.25%) got relief in headache and 4 patients (25%) showed improvement in vision. Shunt failure occurred in 2 cases (12.50%) at 12 months due to ventricular end obstruction. (table 1)

Complication - There was no immediate or early complication seen (no ICH, no IVH, no infection) except misplacement of the ventricular end in one case. Proximal end obstruction was seen in 2 cases (12.50%) during the follow-up period.

#### DISCUSSION

There are multiple treatment options for IIH including drugs (steroids, acetazolamide, weight loss) optic nerve fenestration, lumbar peritoneal shunt and ventriculoperitoneal shunt.

Optic nerve sheath fenestration-related complications are peripapillary hemorrhage, papillary dysfunction, and strabismus. Death, worsening of visual field deficits, permanent loss of vision, and stroke are rarely reported complications of the Optic nerve sheath fenestration procedure (15,16,17,18). Shunts are more effective than optic nerve sheath fenestrations for relieving headaches

because shunt procedures lower ICP (19).

The use of LP shunts for IIH has been well-described in many studies (12,20,21,22).

According to the different literature, LP shunt surgery is associated with multiple complications such as a high rate of obstruction-related shunt failure (10,14). Intracranial hypotension resulting from excessive drainage of CSF (10,12,14,24).

**Table 1.** Table showing sixteen patients, with their symptomatology, grading of shunt and outcome at 1 year.

Case no	Age/ sex	Symptoms & sign	Fundoscopy (papilledema)	Grade of shunt placement	outcome		
					Headache	Vision	Obstruction
1	35/f	B/V, H/A	+ (Present)	GRADE 1	NO	IMPROVED	
2	32/f	B/V, H/A	+ (Present)	GRADE 2	YES	STABLE	Proximal end obstruction
3	44/f	B/V, H/A	+ (Present)	GRADE 1	NO	STABLE	
4	22/f	B/V, H/A	+ (Present)	GRADE 3	NO	STABLE	
5	44/m	B/V, H/A	+ (Present)	GRADE 1	YES	STABLE	
6	38/m	B/V, H/A	+ (Present)	GRADE 1	NO	IMPROVED	
7	26/f	B/V, H/A	+ (Present)	GRADE 1	NO	IMPROVED	
8	37/f	B/V, H/A	- (Absent)	GRADE 1	NO	STABLE	
9	37/f	B/V, H/A	+ (Present)	GRADE 1	NO	STABLE	
10	42/f	B/V, H/A	+(Present)	GRADE 1	NO	IMPROVED	
11	26/f	B/V, H/A	+(Present)	GRADE 2	NO	STABLE	Proximal end obstruction
12	28/f	B/V, H/A	+(Present)	GRADE 1	NO	IMPROVED	
13	32/m	B/V, H/A	- (Absent)	GRADE 1	NO	STABLE	
14	26/f	B/V, H/A	+(Present)	GRADE 1	YES	STABLE	
15	32/f	B/V, H/A	+(Present)	GRADE 2	NO	STABLE	
16	28/m	B/V, H/A	+(Present)	GRADE 2	NO	STABLE	

B/V = blurred vision, H/A = Headache

Lumbar radiculopathy (23, 24). and Tonsillar herniation is also reported following LP shunt placement (25).

According to some literature, the ventriculoperitoneal (VP) shunt was superior to the Lumboperitoneal (LP) shunt in IIH with better clinical outcomes. However, Ventricular shunts are infrequently used in IIH because of the difficulty in placement of a shunt in a small-sized or a normal-sized ventricle with a fear of misplacement of the catheter. VP shunt malfunction is often caused by

ventricular end obstruction (26,27). Hence, accurate placement is one of the most important predictors of shunt survival (28). The ventricular catheter insertion, in cases of IIH, has been considered technically challenging and this may require Neuronavigation insertion. In our study, we are using medtronic optical system navigation and the results are favorable regarding the effectiveness of shunt placement as well as resolving of symptoms. Our study aims to establish VPS surgery in the case of IIH with the help of Neuronavigation. The disadvantages

of navigation systems are time-consuming, expensive, and not easily available. The limitation of this study was that there were no comparison groups of VPS and LPS in cases of IIH.

## CONCLUSION

Our results support Neuronavigation-guided VP shunt placement as an effective and durable treatment option in cases IIH with a low incidence of complication rates.

## REFERENCES

- Dandy WE: Intracranial pressure without brain tumor: diagnosis and treatment. *Ann Surg* 106:492–513, 1937.
- Rush JA: Pseudotumor cerebri: clinical profile and visual outcome in 63 patients. *Mayo Clin Proc* 55:541–546, 1980
- Wall M: Idiopathic intracranial hypertension. *Neurol Clin* 9:73–95, 1991
- Durcan FJ, Corbett JJ, Wall M: The incidence of pseudotumor cerebri. Population studies in Iowa and Louisiana. *Arch Neurol* 45:875–877, 1988
- Johnston I, Paterson A, Besser M: The treatment of benign intracranial hypertension: a review of 134 cases. *Surg Neurol* 16:218–224, 1981
- Cinciripini GS, Donahue S, Borchert MS: Idiopathic intracranial hypertension in prepubertal pediatric patients: characteristics, treatment, and outcome. *Am J Ophthalmol* 127:178–182, 1999
- Gucer G, Bernstein L: Long-term intracranial pressure recording in the management of pseudotumor cerebri. *J Neurosurg* 49:256–263, 1978
- Johnson LN, Krohel BG, Madsen RW, et al: The role of weight loss and acetazolamide in the treatment of idiopathic intracranial hypertension. *Ophthalmology* 105:2313–2317, 1998
- Wandstrat TL, Phillips J: Pseudotumor cerebri responsive to acetazolamide. *Ann Pharmacother* 29:318, 1995
- Chumas PD, Kulkarni AV, Drake JM, et al: Lumboperitoneal shunting: a retrospective study in the pediatric population. *Neurosurgery* 32:376–383, 1993
- Cornblath WT, Miller NR: Pseudotumor cerebri treated with lumboperitoneal shunt. *Ann Neurol* 26:183, 1989
- Eggenberger ER, Miller NR, Vitale S: Lumboperitoneal shunt for the treatment of pseudotumor cerebri. *Neurology* 46:1524–1530, 1996
- Johnston I, Besser M, Morgan MK: Cerebrospinal fluid diversion in the treatment of benign intracranial hypertension. *J Neurosurg* 69:195–202, 1988
- Rosenberg ML, Corbett JJ, Smith C, et al: Cerebrospinal fluid diversion procedures in pseudotumor cerebri. *Neurology* 43:1071–1072, 1993
- Mauriello JA Jr, Shaderowfsky P, Gizzi M, et al: Management of visual loss after optic nerve sheath decompression in patients with pseudotumor cerebri. *Ophthalmology* 102:441–445, 1995
- Plotnik JL, Kosmorsky GS: Operative complications of optic nerve sheath decompression. *Ophthalmology* 100:683–690, 1993
- Rizzo JF III, Lessell S: Choroidal infarction after optic nerve sheath fenestration. *Ophthalmology* 101:1622–1626, 1994
- Spoor TC, McHenry JG: Complications of optic nerve sheath decompression. *Ophthalmology* 100:1432–1433, 1993
- Sergott RC, Savino PJ, Bosley TM: Modified optic nerve sheath decompression provides long-term visual improvement for pseudotumor cerebri. *Arc Ophthalmol* 106:1384–1390, 1988
- Burgett RA, Purvin VA, Kawasaki A: Lumboperitoneal shunting for pseudotumor cerebri. *Neurology* 49:734–739, 1997
- Guy J, Johnston PK, Corbett JJ, et al: Treatment of visual loss in pseudotumor cerebri associated with uremia. *Neurology* 40:28–32, 1990
- James HE, Tibbs PA: Diverse clinical applications of percutaneous lumboperitoneal shunts. *Neurosurgery* 8:39–42, 1981
- Eisenberg HM, Davidson RI, Shillito J Jr: Lumboperitoneal shunts. Review of 34 cases. *J Neurosurg* 35:427–431, 1971
- Selman WR, Spetzler RF, Wilson CB, et al: Percutaneous lumboperitoneal shunt: review of 130 cases. *Neurosurgery* 6:255–257, 1980
- Chumas PD, Armstrong DC, Drake JM, et al: Tonsillar herniation: the rule rather than the exception after lumboperitoneal shunting in the pediatric population. *J Neurosurg* 78:568–573, 1993
- Browd SR, Ragel BT, Gottfried ON, Kestle JR: Failure of cerebrospinal fluid shunts: part I: obstruction and mechanical failure. *Pediatr Neurol*. 2006;34:83–92. [PubMed] [Google Scholar]
- Tuli S, O'Hayon B, Drake J, Clarke M, Kestle J: Change in ventricular size and effect of ventricular catheter placement in pediatric patients with shunted hydrocephalus. *J Neurosurg*. 1999;45:1329–1333. [PubMed] [Google Scholar]
- Wan KR, Toy JA, Wolfe R, Danks A: Factors affecting the accuracy of ventricular catheter placement. *J Clin Neurosci*. 2011;18:485–488. [PubMed] [Google Scholar]



# Assessment of visual function in patients with posterior fossa tumours. A retrospective study

Raghav Jindal<sup>1</sup>, Vijendra Bijarniya<sup>1</sup>, Mayank Purohit<sup>2</sup>,  
Vinod Sharma<sup>1</sup>, Sanjeev Chopra<sup>1</sup>, Ashok Gupta<sup>1</sup>

<sup>1</sup> SMS Medical College, Adarsh Nagar, Jaipur, Rajasthan, INDIA

<sup>2</sup> MGM Medical College and Hospital, Panvel, Navi Mumbai, Maharashtra, INDIA

## ABSTRACT

**Introduction.** Posterior fossa tumours (PFTs) frequently present with ophthalmic complaints. The literature is mainly focused on PFTs affecting the pediatric age group, and the post-treatment visual outcomes (VOs) are scarcely reported.

**Objective.** To evaluate the VOs at 6 months following the index surgery in patients with PFTs.

**Materials and methods.** This retrospective study involved 50 patients of all age groups who underwent surgical resection of PFTs in the Department of Neurosurgery. The patients with PFTs, except those with low-grade tumours, received concurrent chemo-radiotherapy. Pre- and postoperative (6 months after the index surgery) ophthalmic examinations were done and compared. VOs included colour vision, night vision, visual acuity (VA), pupillary function (size, reactivity), papilledema grade, splinter haemorrhage, retinal venous dilatation, strabismus (esotropia and hypertropia), and nystagmus.

**Results.** The patients were mainly aged 1–10 years (22%) with slight female predominance (52%). The most common PFTs were brainstem glioma and pilocytic astrocytoma (each 18%). At 6 months, there was no significant change in colour and night vision, pupil size and reactivity, splinter haemorrhage, and hypertropia (all p-values>0.05). A significantly lesser proportion of patients had moderate VA (p-value=0.013), retinal venous dilatation (p-value=0.001), and grade 1 (p-value=0.005) as well as grade 4 papilledema (p-value=0.041). Moreover, a significantly greater proportion of patients had grade 0 papilledema (p-value<0.0001). While the incidence of nystagmus and esotropia increased significantly (both p-values<0.0001).

**Conclusion.** At 6 months, the majority of the patients had good VOs, including significant improvement in moderate VA, papilledema, and retinal venous dilatation. While nystagmus and esotropia increased significantly.

## INTRODUCTION

Brain tumors are the 19th most common malignancies, and the 12th leading cause of cancer-related mortality. [1] Among various brain tumors, those located in posterior fossa are more prevalent among children (54–70%) than adults (15–20%). [2] The posterior fossa

## Keywords

hydrocephalus,  
nystagmus,  
papilledema,  
posterior fossa tumours,  
strabismus,  
visual outcome



Corresponding author:  
Vinod Sharma

SMS Medical College, Adarsh Nagar,  
Jaipur, Rajasthan, India

neurovinod@gmail.com

Scan to access the online version



tumours (PFTs) are associated with grave consequences due to limited available space, and risk of brain stem nuclei involvement. [3]

PFTs often manifest with visual symptoms and signs that may adversely impact the visual afferent and efferent pathways. [4] The pressure of tumor on the visual pathway can result in reduced visual acuity (VA), loss of visual field, and impairment of eye movement. Additionally, increased intracranial pressure (ICP) may result from obstructive hydrocephalus, brain tumor mass effect, brain edema, and tumor infiltration into the leptomeninges. Raised ICP may result in papilledema and optic disc atrophy, leading to irreversible vision loss, even with hydrocephalus treatment. [4, 5]

Hydrocephalus can also result in abducens nerve paralysis, causing esotropia and horizontal diplopia. Trochlear nerve dysfunction can result in diplopia in the vertical or oblique direction, along with hypertropia. These tumors can interfere with the ability to maintain visual focus and affect the mechanisms responsible for vestibular and gaze stabilization, leading to skew deviation, nystagmus, and complex gaze palsies. [6]

Timely diagnosis of visual impairment is essential as vision loss caused by a brain tumor or its therapy is frequently permanent. [7] Gadgil *et al.* [4] and Peeler *et al.* [8] reported long-term (median 4-years and mean 20.5-months, respectively) visual outcomes (VOs) following surgical resection of PFTs in pediatric age group. Singh *et al.* described VOs in both pediatric and adult age groups; however, these were short-term outcomes (6-weeks). [6] Review of current literature suggested that further studies are required to evaluate the VOs, especially in both pediatric and adult age groups. Moreover, intermediate-term VOs have not been evaluated yet. Thus, the present study aimed to evaluate the VOs at 6-months following the index surgery in patients with PFTs.

#### **MATERIALS AND METHODS**

This retrospective study involved the review of electronic medical records of patients who had undergone surgical resection of PFTs, between August 2022 and January 2024, in the Department of Neurosurgery of a tertiary care hospital. The study included patients of all age groups, of either sex, and underwent surgical resection of PFTs with or without hydrocephalus. While, patients with recurrent PFTs,

visual and ocular motor dysfunction unrelated to the tumor, those receiving chemo-radiotherapy as primary treatment, and incomplete examination data were excluded. The study was approved by the Institutional Ethics Committee, and consent of the patients was waived off due to retrospective nature of the study.

#### **PARTICIPANTS AND INTERVENTION**

Over the period of 18-months, complete pre- and postoperative medical records were available in 50 patients fulfilling the eligibility criteria and thus, included in the study. The patients who presented with symptoms of hydrocephalus were subjected to magnetic resonance imaging (MRI) of brain to confirm the presence of posterior fossa mass as well as hydrocephalus, and subsequently underwent ventriculo-peritoneal (VP) shunt as emergency procedure. Based on the tumors, the patients underwent biopsy (< 25% of tumor volume [TV]), subtotal (25–90% of TV), near-total (>95% of TV), or gross total surgical resection, and the extent of resection was determined by postoperative MRI.

Following surgical resection, all patients with PFTs, except those with the World Health Organization grade 1 tumors, received concurrent chemo-radiotherapy. Chemotherapy included capsule temozolomide (75 mg/m<sup>2</sup> of body size, once daily for Days 1-5, per oral) given for 6-weeks and later adjuvant for 6-months. Radiotherapy was given in a dose of 60 Gy over six weeks in 30–33 fractions (2 Gy/day for 5-days and then rest for 2-days).

#### **ASSESSMENT**

Preoperatively, the patients were assessed for color vision (on Ishihara test, present/absent), night vision (present/absent), VA (on Snellen chart), pupillary function (size, reactivity), papilledema grade, retinal findings (splinter hemorrhage [present/absent] and venous dilatation), strabismus (esotropia and hypertropia), and nystagmus (present/absent). Papilledema was graded from 0 to 5, by the modified Frisén Scale, with grade 0 and 5 suggesting normal optic disc and severe degree of edema, respectively. [9] Strabismus was described as any visible misalignment of the eyes associated with the tumor or treatment. The ellipsoid volume formula was used to calculate the TV (length × height × width × [π/6]). Postoperatively, at 6-months, all the above-mentioned parameters were assessed, and

improvement or deterioration in visual parameters were evaluated.

### STATISTICAL ANALYSES

The data was analysed with SPSS (IBM, Armonk, NY, USA) version 23.0 for Windows. The categorical and continuous variables were represented as frequency (percentages) and mean (standard deviation), respectively. The association between categorical and continuous variables were assessed with Chi-square test and independent sample t-test, respectively. A two-tailed p-value <0.05 was regarded as statistically significant.

### RESULTS

The mean age of the study population was 31.64±19.97 years. The patients were mainly aged 1–10 years (22%) with slight female predominance (52%). The most common PFTs were brainstem glioma and pilocytic astrocytoma (each 18%). Of 50 patients, 40 (80%) had hydrocephalus and all of them underwent VP shunt procedure (Table 1).

**Table 1.** Demographic and disease characteristics.

Characteristics	n (=50)	%
Age, years		
1–10	11	22
11–20	8	16
21–30	7	14
31–40	6	12
41–50	8	16
51–60	6	12
>60	4	8
Sex		
Male	24	48
Female	26	52
Pathological diagnosis		
Brainstem glioma	9	18
Pilocytic astrocytoma	9	18
Medulloblastoma	7	14
Hemangioblastoma	6	12
CP angle vestibular schwannoma	5	10
Ependymoma	4	8
Posterior fossa metastasis	4	8
CP angle epidermoid cyst	3	6
CP angle meningioma	2	4
Anaplastic astrocytoma	1	2
Hydrocephalus	40	80
Shunt procedure	40	80

Preoperatively, color and night vision were present in 78% and 68% patients, respectively. Examination of VA revealed that patients mostly had moderate (38%) and normal vision (30%). The pupils were mainly 3 mm in size (84%) and reactive to light (74%). Grade 1 papilledema, splinter hemorrhage, and retinal venous dilatation were observed in 30%, 46%, and 76% patients, respectively. Moreover, nystagmus, esotropia, and hypertropia were present in 12%, 10%, and 10% patients, respectively. The mean TV was 40.48±10.26 mm<sup>3</sup> (Table 2).

**Table 2.** Comparison of pre- and postoperative characteristics.

Characteristics	Preoperative (n=50)	Postoperative (n=50)	p-value
Color vision	39 (78%)	39 (78%)	1.000
Night vision	34 (68%)	29 (58%)	0.300
Visual acuity			
Normal Vision (6/6)	15 (30%)	23 (46%)	0.099
Good Vision (6/9 to 6/12)	9 (18%)	11 (22%)	0.617
Moderate Vision (6/18 to 6/60)	19 (38%)	8 (16%)	0.013
Poor Vision (<6/60)	7 (14%)	8 (16%)	0.779
Pupil size, mm			1.000
3	42 (84%)	42 (84%)	
4	8 (16%)	8 (16%)	
Pupil reactivity			
Normal	37 (74%)	39 (78%)	0.640
Non-reactive	8 (16%)	8 (16%)	1.000
Sluggish	5 (10%)	3 (6%)	0.461
Papilledema			
Grade 0	9 (18%)	27 (54%)	<0.0001
Grade 1	15 (30%)	4 (8%)	0.005
Grade 2	8 (16%)	7 (14%)	0.779
Grade 3	6 (12%)	4 (8%)	0.564
Grade 4	4 (8%)	0 (0%)	0.041
Grade 5	8 (16%)	8 (16%)	1.000
Splinter hemorrhage	23 (46%)	20 (40%)	0.545
Retinal venous dilatation	38 (76%)	22 (44%)	0.001
Nystagmus	6 (12%)	23 (46%)	<0.0001
Strabismus			
Esotropia	5 (10%)	23 (46%)	<0.0001
Hypertropia	5 (10%)	7 (14%)	0.538
Tumor volume, mm <sup>3</sup>	40.48±10.26	8.35±1.89	<0.0001

Comparison of pre- and postoperative characteristics revealed no significant change in color and night vision (both  $p$ -values  $>0.05$ ). Similarly, there was no significant change in pupil size and reactivity (both  $p$ -values  $>0.05$ ). VA categories were comparable (all  $p$ -values  $>0.05$ ), except significantly lesser proportion of patients with moderate vision observed postoperatively ( $p$ -value = 0.013). Similarly, grades of papilledema did not change significantly postoperatively (all  $p$ -values  $>0.05$ ), except significantly greater and lesser proportion of patients with grade 0 ( $p$ -value  $<0.0001$ ) and grade 1 ( $p$ -value = 0.005) as well as grade 4 papilledema ( $p$ -value = 0.041), respectively. While, proportion of patients with splinter hemorrhage did not change significantly ( $p$ -value = 0.545), significantly lesser proportion of patients had retinal venous dilatation postoperatively ( $p$ -value = 0.001). Furthermore, postoperative incidence of nystagmus and esotropia increased significantly (both  $p$ -values  $<0.0001$ ). Finally, the TV decreased significantly ( $p$ -value  $<0.0001$ ) (Table 2). At 6-months, 16 (32%) patients had recurrence of PFTs.

## DISCUSSION

The principal findings of the present study suggested that PFTs were predominantly present in pediatric age group and included brainstem glioma as well as pilocytic astrocytoma. At 6-months, moderate VA, grades of papilledema, retinal venous dilatation, and TV improved significantly, while nystagmus and esotropia worsened significantly. Other outcome measures, including color vision, night vision, pupil size, pupillary reactivity to light, and splinter hemorrhage, were comparable with respect to preoperative status.

PFTs are reported in both adults and children with around two-third pediatric brain tumors originating from the posterior fossa. [10] In childhood, the predominant PFTs include pilocytic astrocytomas, ependymomas, and medulloblastoma, while tumors including lymphomas, metastatic lesions, and hemangioblastoma are more prevalent in adulthood. [11] Moreover, several PFTs occur in the cerebellopontine angle (CPA), consisting of arachnoid tissue, cerebrospinal fluid (CSF), facial and vestibulocochlear nerves, and the anterior inferior cerebellar artery. CPA tumors account for less than 10% and less than 1% of all intracranial tumors in

adults and children, respectively. [12] Similarly, we observe comparable pattern of PFTs distribution in pediatric and adult age groups. Moreover, all the CPA tumors were diagnosed in adults. However, contrary to the literature, 70% PFTs were diagnosed in adults.

Complete surgical resection of the PFTs is the goal, to be followed by concurrent chemo-radiotherapy based on the histopathological findings. Over the last several years, better management of PFTs has reduced mortality, thereby increasing the number of long-term survivors. However, the survivors have visual deficit and decreased quality of life. [13] Thus, assessment of the VOs is critically important to allow for the best possible vision to survivors to better cope with the sequelae.

Mechanistically, the visual symptoms depend on the severity and duration of hydrocephalus and papilledema. [13] More than a quarter of the patients (80%) were diagnosed with hydrocephalus and an emergency VP shunt procedure was performed. Moreover, a quarter of the patients (24%) had grade 4 and 5 papilledema. Following the surgical excision of the tumor, none of the patients experienced hydrocephalus during the study period. At 6-months, grade 5 papilledema persisted, while none of the patients had grade 4 papilledema. Thus, excision of the tumor mass and placement of VP shunt resulted in reduction of raised ICP and significantly decreased severity of papilledema, thereby improving the visual outcomes. Furthermore, persistence of the grade 5 papilledema highlights the irreversible nature of the neuronal injury.

At 6-months, there was an overall improvement in VA. The number of patients with normal and good vision increased from 30% to 46% and 18% to 22%, respectively, though this increase was not statistically significant. This increase in normal and good vision was associated with significant decrease in number of patients with moderate vision. However, the patients with preoperative poor vision persisted. Similar results were reported by other studies involving pediatric, [4, 8] as well as both pediatric and adult age groups. [6]

Various authors have reported VOs in benign PFTs, particularly low-grade gliomas. A study involving 51 patients with low-grade posterior fossa glioma reported that none of the patients had VA worse than 20/200, though 5.9% had visual field defect 15-years after initial diagnosis. [14] Another

study showed that none of the 34 survivors of low-grade glioma had VA worse than 20/200, although 5.9% had visual field defects at 10.7-years. [15] Peeler et al. demonstrated a low rate of visual impairment (4.7%) in patients with low-grade glioma at 20.5-months. [8] Similarly, we observed that only 2 of 20 patients with low-grade PFTs had poor vision <6/60, both pre- and postoperatively, thereby suggesting no deterioration in vision among patients with low-grade of PFTs.

PFT type is reported to be a significant determinant of VO. We found that patients with poor VA (n=8) had medulloblastoma (n=4), brainstem glioma (n=3), and posterior fossa metastasis (n=1). Similarly, patients with medulloblastoma and ependymoma have been demonstrated to have significantly worse VOs compared to those with juvenile pilocytic astrocytoma. [8] Medulloblastoma, brainstem glioma, and posterior fossa metastasis have aggressive growth patterns. Moreover, these tumors require more aggressive surgical resection as well as adjuvant chemo-radiotherapy. [16-18] More prolonged increases in ICP may put patients at greater risk of developing optic atrophy. Supporting these findings, all eight patients with poor VA had optic atrophy.

Additionally, visual impairment can be induced iatrogenically. The surgical resection of the tumor can result in impaired vision either through direct surgical damage to the optic pathway or due to perioperative visual loss resulting from a sudden decline in ICP or disruption of blood flow to the visual pathway. [19, 20] Radiation therapy may result in radiation-induced optic neuropathy and/or necrosis affecting the visual pathway. [21-23] Lastly, chemotherapy may lead to papilledema, optic neuritis, maculopathy, optic neuropathy, and cataracts. [24, 25]

A study reported that treatment of PFTs led to an increase in number of patients with nystagmus (from 5% to 23%), and strabismus (from 23% to 42.4%). [8] In another study, the authors observed that small esotropia mostly resolved spontaneously (80%), while 53% patients with moderate esotropia and 100% with large esotropia required surgical intervention. In these patients, the symptoms resolved, thereby suggesting an excellent prognosis for patients with post-operative esotropia. Hypertropia was observed in 14% patients, of which around half resolved spontaneously and remaining

half required surgery resulting in correct eyes alignment in 40% patients, while 60% had continued symptomatic hypertropia with persistent diplopia despite multiple surgeries, thus suggesting a poorer prognosis for patients with post-operative hypertropia relative to those with esotropia. [4] Similarly, we observed that number of patients with nystagmus (from 12% to 46%), and esotropia (from 10% to 46%) increased significantly (both p-values<0.0001). Though patients with hypertropia increased as well (from 10% to 14%), it did not statistically significant level (p-value=0.538). However, we did not evaluate the outcome of strabismus surgery, as it was not performed in any of the patients during the study period. Increase in postoperative incidence of nystagmus and strabismus could be ascribed to more aggressive posterior fossa surgery and use of concurrent chemo-radiotherapy.

The present study had certain limitations. First, the study was retrospective in nature and control group could not BE used. Second, sample size was relatively small. Third, data related to visual fields was not available in all the patients, and thus not evaluated. Fourth, esotropia was not categorized into small, moderate, and large.

## CONCLUSION

To conclude, surgical resection of PFTs followed by concurrent chemo-radiotherapy resulted in good VOs in majority of the patients, including significant improvement moderate VA, papilledema, retinal venous dilatation, and TV. While, color and night vision, pupil size and reactivity, hypertropia, and splinter hemorrhage did not change significantly. However, nystagmus and esotropia increased significantly. Adverse VOs were mainly related to hydrocephalus and papilledema, leading to optic atrophy.

## ABBREVIATIONS

CPA	Cerebellopontine angle
CSF	Cerebrospinal fluid
ICP	Intracranial pressure
MRI	Magnetic resonance imaging
PFTs	Posterior fossa tumors
TV	Tumor volume
VA	Visual acuity
VOs	Visual outcomes
VP	Ventriculo-peritoneal

## REFERENCES

- Ilic I, Ilic M (2023) International patterns and trends in the brain cancer incidence and mortality: An observational study based on the global burden of disease. *Heliyon* 9(7):e18222. <https://doi.org/10.1016/j.heliyon.2023.e18222>
- Bose A, Prasad U, Kumar A, Kumari M, Suman SK, Sinha DK (2023) Characterizing Various Posterior Fossa Tumors in Children and Adults With Diffusion-Weighted Imaging and Spectroscopy. *Cureus* 15(5):e39144. <https://doi.org/10.7759/cureus.39144>
- Kakar J, Ashraf J, Khan AA, Imran M, Rehmani MA, Ghori SA, et al (2020) The satisfactory surgical outcome of posterior fossa brain tumors in children at civil hospital, karachi. *Asian J Neurosurg* 15:377–81. [https://doi.org/10.4103/ajns.AJNS\\_56\\_19](https://doi.org/10.4103/ajns.AJNS_56_19)
- Gadgil N, Edmond J, Stormes K, Lam S, Shah V (2019) Visual Complications of Pediatric Posterior Fossa Tumors: Analysis of Outcomes. *Pediatr Neurol* 92:48–54. <https://doi.org/10.1016/j.pediatrneurol.2018.09.016>
- Nuijts MA, Degeling MH, Stegeman I, Schouten-van Meeteren AYN, Imhof SM (2019) Visual impairment in children with a brain tumor: a prospective nationwide multicenter study using standard visual testing and optical coherence tomography (CCISS study). *BMC Ophthalmol* 19:220. <https://doi.org/10.1186/s12886-019-1225-8>
- Singh DK, Agrahari VK, Kaif M, Kumar R, Yadav K (2021) Visual outcome analysis in patients with posterior fossa tumours undergoing surgical treatment. *Romanian Neurosurgery XXXV(2)*:174–9. <https://doi.org/10.33962/roneuro-2021-027>
- Moreno L, Bautista F, Ashley S, Duncan C, Zacharoulis S (2010) Does chemotherapy affect the visual outcome in children with optic pathway glioma? A systematic review of the evidence. *Eur J Cancer* 46(12):2253–9. <https://doi.org/10.1016/j.ejca.2010.03.028>
- Peeler CE, Edmond JC, Hollander J, Alexander JK, Zurakowski D, Ullrich NJ, et al (2017) Visual and ocular motor outcomes in children with posterior fossa tumors. *J AAPOS* 21(5):375–9. <https://doi.org/10.1016/j.jaapos.2017.05.032>
- Scott CJ, Kardon RH, Lee AG, Frisén L, Wall M (2010) Diagnosis and grading of papilledema in patients with raised intracranial pressure using optical coherence tomography vs clinical expert assessment using a clinical staging scale. *Arch Ophthalmol* 128(6):705–11. <https://doi.org/10.1001/archophthalmol.2010.94>
- Loevner LA (1999) Imaging features of posterior fossa neoplasms in children and adults. *Semin Roentgenol* 34(2):84–101. [https://doi.org/10.1016/s0037-198x\(99\)80024-8](https://doi.org/10.1016/s0037-198x(99)80024-8)
- Santos de Oliveira R, Jucá CEB, Valera ET, Machado HR (2008) Hydrocephalus in posterior fossa tumors in children. Are there factors that determine a need for permanent cerebrospinal fluid diversion? *Child's Nervous System* 24:1397–403. <https://doi.org/10.1007/s00381-008-0649-x>
- Bertot B, Steele WJ, Boghani Z, Britz G (2017) Diagnostic Dilemma: Cerebellopontine Angle Lipoma Versus Dermoid Cyst. *Cureus* 9(11):e1894. <https://doi.org/10.7759/cureus.1894>
- Peeler CE (2017) A Review of Visual and Oculomotor Outcomes in Children With Posterior Fossa Tumors. *Semin Pediatr Neurol* 24(2):100–03. <https://doi.org/10.1016/j.spen.2017.04.007>
- Armstrong GT, Conklin HM, Huang S, Srivastava D, Sanford R, Ellison DW, et al (2011) Survival and long-term health and cognitive outcomes after low-grade glioma. *Neuro Oncol* 13(2):223–34. <https://doi.org/10.1093/neuonc/noq178>
- Sonderkaer S, Schmiegelow M, Cartensen H, Nielsen LB, Muller J, Schmiegelow K (2003) Long-term neurological outcome of childhood brain tumors treated by surgery only. *J Clin Oncol* 21(7):1347–51. <https://doi.org/10.1200/JCO.2003.08.009>
- Iyer S, Ismail M, Tamrazi B, Salloum R, de Blank P, Margol A, et al (2022) Novel MRI deformation-heterogeneity radiomic features are associated with molecular subgroups and overall survival in pediatric medulloblastoma: Preliminary findings from a multi-institutional study. *Front Oncol* 12:915143. <https://doi.org/10.3389/fonc.2022.915143>
- Eisele SC, Reardon DA (2016) Adult brainstem gliomas. *Cancer* 122(18):2799–809. <https://doi.org/10.1002/cncr.29920>
- Sunderland GJ, Jenkinson MD, Zakaria R (2016) Surgical management of posterior fossa metastases. *J Neurooncol* 130(3):535–42. <https://doi.org/10.1007/s11060-016-2254-2>
- Peragallo JH (2018) Effects of brain tumors on vision in children. *Int Ophthalmol Clin*. 58(4):83–95. <https://doi.org/10.1097/IIO.0000000000000237>
- Ahn Y, Cho BK, Kim SK, Chung YN, Lee CS, Kim IH, et al (2006) Optic pathway glioma: outcome and prognostic factors in a surgical series. *Childs Nerv Syst*. 22(9):1136–42. <https://doi.org/10.1007/s00381-006-0086-7>
- Saha A, Salley CG, Saigal P, Rolnitzky L, Goldberg J, Scott S, et al. (2014) Late effects in survivors of childhood CNS tumors treated on head start I and II protocols. *Pediatr Blood Cancer* 61(9):1644–72. <https://doi.org/10.1002/pbc.25064>
- Mayo C, Martel MK, Marks LB, Flickinger J, Nam J, Kirkpatrick J (2010) Radiation dose-volume effects of optic nerves and chiasm. *Int J Radiat Oncol Biol Phys*. 76(3):S28–35. <https://doi.org/10.1016/j.ijrobp.2009.07.1753>
- Donahue B (1992) Short- and long-term complications of radiation therapy for pediatric brain tumors. *Pediatr Neurosurg* 18(4):207–17. <https://doi.org/10.1159/000120664>
- Al-Tweigeri T, Nabholtz JM, Mackey JR (1996) Ocular toxicity and cancer chemotherapy. *Cancer* 78(7):1359–73. [https://doi.org/10.1002/\(SICI\)1097-](https://doi.org/10.1002/(SICI)1097-)

- 0142(19961001)78:7<1359::AID-CNCR1>3.0.CO;2-G.
25. Schmid KE, Kornek GV, Scheithauer W, Binder S (2006) Update on ocular complications of systemic cancer chemotherapy. *Surv Ophthalmol* 51(1):19-40. <https://doi.org/10.1016/j.survophthal.2005.11.001>.



# Delayed post traumatic CSF rhinorrhoea. Two rare cases and review of literature

Jadhav Anil, Katyal Abhishek, Jagetia Anita, Srivastava A.K,  
Singh Daljit

<sup>1</sup> GB Pant institute of Postgraduate Medical Education & Research  
(GIPMER), New Delhi, INDIA

## ABSTRACT

Cerebrospinal fluid (CSF) leak can occur immediately or several years after traumatic skull base injury which may present merely as a CSF leak or may even present with recurrent meningitis. Around 2% of all cases of head trauma, and 12%–30% of all basilar skull fractures may develop CSF leak. Posttraumatic CSF rhinorrhoea usually occurs within the first 48 hours, and the majority of them occur in the first 3 months, whereas delayed CSF leak beyond 3 months is rare.

We encountered two such cases of delayed post-traumatic CSF rhinorrhoea about a decade after the head injury. We have reviewed and discussed previous studies on delayed post-traumatic CSF rhinorrhoea which have shown CSF leaks occur after months, years, or even after trauma. Such cases may help clinicians to be aware of the possibility of delayed CSF rhinorrhoea which may occur years after traumatic head injury.

## INTRODUCTION

Cerebrospinal fluid (CSF) leak is not an unknown entity after traumatic brain injury (TBI). Around 2% of all cases of head trauma, and 12%–30% of all basilar skull fractures may develop CSF leak. In two third of patients, post-traumatic CSF rhinorrhea usually occurs in first 48 hours whereas majority of them occur in the first 3 months. The delayed CSF leak that occurs beyond 3 months is rare but not un-common. However delayed post-traumatic CSF rhinorrhoea have been reported to occur even decades after TBI. Such patients after detail evaluation found to have CSF leak for the cause of recurrent meningitis. [1,2]The following report describes two such cases of delayed CSF rhinorrhoea about a decade after TBI.

## CASE REPORT

### Case 1

A 26 years old young male with no known history of Asthma, Tuberculosis or allergic rhinitis was referred to us for treatment of watery discharge from right nostril which was there for 2 years. The

## Keywords

cerebrospinal fluid (CSF),  
traumatic brain Injury (TBI),  
rhinorrhoea,  
anterior cranial fossa (ACF)



Corresponding author:  
Anil Jadhav

Department of Neurosurgery,  
GIPMER, New Delhi., India

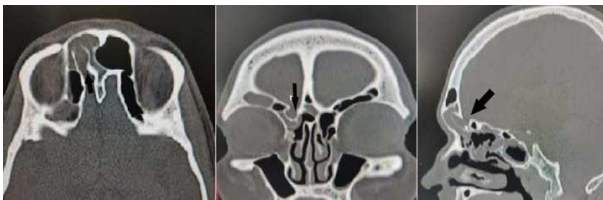
aniljadhav7@gmail.com

Scan to access the online version



watery discharge used to exacerbate on lying supine and on sitting position. The leak was intermittent in nature, clear non sticky, non blood stained, no history of headache, fever and vomiting and neck rigidity. The patient had not undergone any previous surgery and was not on any medications. He had fall from height (10 feet) and had sustained head injury 2 years ago. Patient subsequently had nose bleeding but patient could not recollect whether blood was mixed with any watery discharge or not. There was no history of loss of consciousness or seizure or vomiting. He did not seek medical advice following the incident and had no documents regarding the same. General examination was unremarkable. However, patient had anosmia with no other neurological deficits.

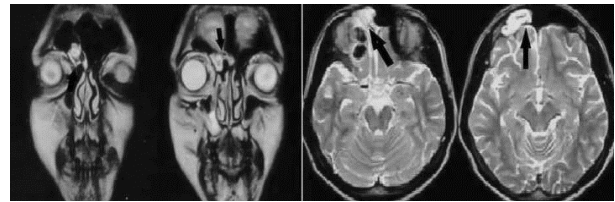
CSF rhinorrhea was also evident on clinical examination. Routine hematological and biochemical profile was normal. Well demarcated bone defect in the floor of the anterior cranial fossa into posterior wall of frontal air sinus with invaginated bone with everted margins on right side was evident on CT Head (Fig 1). Similar defect with herniation of thin gliotic tissue in the right frontal air sinus with CSF collection was revealed on MRI (Fig 2). Hence a diagnosis of post traumatic CSF rhinorrhoea with defect in the anterior cranial fossa (ACF) base at frontal air sinus probably due to growing fracture was made based on clinical and radiological findings.



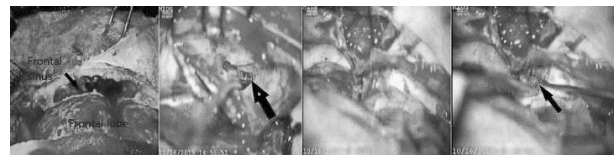
**Figure 1.** Demarcated bone defect in the floor of the anterior cranial fossa into posterior wall of frontal air sinus (Arrows).

A bifrontal craniotomy and intradural-extradural repair of CSF fistula was done. On retracting the right frontal lobe, a thin gliotic tissue and arachnoid was seen going through the bone defect with evidence of bony invagination into frontal air sinus on right side (Fig 3). As gliotic tissue and arachnoid was resected, bone defect of 4.4 X 5 mm was found. Frontal air sinus was exteriorized and packed with gelfoam soaked in betadine solution and galea tissue. The bony defect was sealed with a bone graft harvested from inner table of frontal bone. The base of ACF was

covered with fascia pericranium tissue. Postoperative period was uneventful and patient had no leak in follow up.



**Figure 2.** MRI showing defect with herniation of thin gliotic tissue in the right frontal air sinus with CSF collection (arrows).



**Figure 3.** Intra-operative view showing gliotic tissue herniating through the bony defect (arrows).

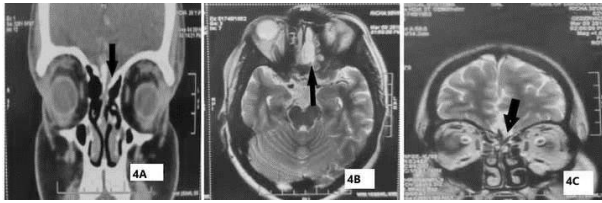
### Case 2

A 30 years old young female was referred to us with watery discharge from left nostril for last 4 years. The leak was exacerbated on sitting, was intermittent in nature, clear non sticky, not blood stained. Patient has history of being treated for meningitis episode 4 years back after which she developed CSF leak. Patient is not a known case of asthma or allergic rhinitis, had not undergone any previous surgery. She had sustained head injury at age of 19 years due to fall from height following which was managed conservatively in view of grossly normal CT brain findings. There was no history of CSF leak during the last 4 years. General examination revealed two scars on forehead. She had anosmia on left side with no other neurological deficit.

The routine blood investigations were normal. CT Head revealed a well demarcated bone defect in the floor of the anterior cranial fossa involving the cribriform plate on left side (Fig 4A). MRI brain revealed the same along with herniation of gliotic brain tissue with CSF collection (Fig 4B and 4C). Hence diagnosis of post traumatic CSF rhinorrhoea with defect in the anterior cranial fossa (ACF) base at left cribriform plate was made based on clinical and radiological findings.

A bifrontal craniotomy and repair of defect with sub-frontal intra and extradural approach was performed as in previous case. Postoperative period

was un-eventful and patient had no further leak in subsequent follow up.



**Figure 4.** CT Head revealed a well demarcated bone defect in the floor of the anterior cranial fossa involving the cribriform plate on left side (Fig 4A). MRI of brain revealed.

## DISCUSSION

A total of 19 cases of delayed posttraumatic CSF rhinorrhea including the present cases are described in Table 1. CSF leaks commonly occurs following nonsurgical trauma (80%–90% of cases), 16% occurs due to surgical procedures and the remaining 4% are non-traumatic. It is also evident in 12%–30% of all basilar skull fractures.[1] CSF leak has 10% risk of developing meningitis per year.[1,2] Traumatic CSF rhinorrhea has been classified into immediate that occurs within 48 hours and delayed CSF rhinorrhea.

Literature has shown that 50% of traumatic CSF rhinorrhea occurred in first 48 hours and almost all of them present in first 3 months. The delayed CSF leaks that are occurs beyond 3 months are seen in the 5% of patients whereas CSF leak beyond a year is very rare. [3,4,5,6,7,8,9,10] However leaks occurring up to a prolonged period of 44 years has been reported in literature.[14] Generally post traumatic CSF leak subsides spontaneously. These happens either due to sealing by blood clot or, inflammation, or due to brain tissue herniation. The natural healing that occurs as per above mentioned mechanisms are not perfect and hence patients may present again with CSF leak in few month time. The re-leak may be due to resolution of edema or, due to resolution of inflammation or due to absorption of blood clot resulting into reopening of bony defect. If CSF leak does not occur within months of TBI, the incidents are often forgotten till patients get recurrent meningitis or CSF leak at later date.

The possible mechanisms for re-leak may be raised intracranial pressure, another trauma, growing skull fracture (frontal/ ethmoid), necrosis of scar and bone or due to craniofacial remodeling with advancing age and ascending infections. CSF leak usually occurs through dural tearing and associated

fracture of anterior cranial fossa involving cribriform plate and posterior wall of the frontal sinus and sphenoid sinus [3,4,5,6,7,8,9,10,11,12,13,14,15,16].

Table 1

Cases of delay post traumatic cerebrospinal fluid leak

Author (Year)	Age	Trauma	Interval from trauma to CSF leak in years	Meningitis	Operative approach
Linell and Robinson (14)(1)	-	Unknown	14	Yes	Unknown
Schneider and Thompson (10)(4)	37	Traffic	Unknown	Yes	Unknown
Uemura and Makino (17)(7)	33	Gun shot	9	Yes	Unknown
Kamerer and Caparosa (18)	-	Unknown	17	-	Unknown
Merelli et al., 1982(2)	35	Traffic accident	12	No	Intradural
Russell and Cummins (1984)(5)	43	Falling down	34	No	Intradural
Okada et al., 1991(6)	44	Traffic accident	13	Yes	Intradural
Pandya and Keogh (1991)(8)	52	Head trauma	30	Yes	Intradural
Pandya and Keogh (1991)(8)	58	Traffic accident	35	Yes	Unknown
Stewart and kaye (1992)(11)	38	Traffic accident	14	Yes	Intradural
Crawford et al., 1994 (10)	40	Traffic accident	35	Yes	Unknown
Salca and Danaila (1997)(13)	54	Traffic accident	27	No	Extradural
Rao et al., 2010(16)	57	Falling down	44	No	Intradural
Kamochi et al., 2013(15)	66	Traffic accident	20	Yes	Intradural
Guyar and Turner (2015)(16)	62	Traffic accident	5	No	Intradural
Guyar and Turner (2015)(16)	61	Traffic accident	12	No	Intradural
Case 1	26	Fall from height	12	No	Intradural Extradural
Case 2	30	Fall from height	07	Yes	Intradural Extradural

**Table 1.** Reported cases of delayed post traumatic CSF rhinorrhea.

## CONCLUSION

Delayed post traumatic csf rhinorrhoea although rare but are not unreported complications of traumatic injuries involving the skull base. A high index of suspicion in cases of spontaneous csf leaks or recurrent meningitis with history of remote head trauma can lead to adequate diagnosis & timely management.

## REFERENCES

1. Friedman JA, Ebersold MJ, Quast LM. Post-traumatic cerebrospinal fluid leakage. *World J Surg.* 2001;25:1062–6.
2. Banks CA, Palmer JN, Chiu AG, O'Malley BW, Jr, Woodworth BA, Kennedy DW, et al. Endoscopic closure of CSF rhinorrhea: 193 cases over 21 years. *Otolaryngol Head Neck Surg.* 2009;140:826–33.
3. Linell EA, Robinson WL. Head injuries and meningitis. *J Neurol Neurosurg Psychiatry.* 1941;4:23.

4. Schneider RC, Thompson JM. Chronic and delayed traumatic cerebrospinal rhinorrhea as a source of recurrent attacks of meningitis. *Ann Surg.* 1957;145:517.
5. Uemura K, Makino H. Operative indication and operative method of frontal base skull fracture. Lewin's intradural patch. *Shuju.* 1972;26:733-42.
6. Kamerer DB, Caparosa RJ. Temporal bone encephalocele – Diagnosis and treatment. *Laryngoscope.* 1981;92:878-82.
7. Merelli E, Merli GA, Sola P. An unusual method for diagnosing spinal fluid rhinorrhea in a case of delayed post-traumatic fistula. *Ital J Neurol Sci.* 1982;3:249-50.
8. Russell T, Cummins BH. Cerebrospinal fluid rhinorrhea 34 years after trauma: A case report and review of the literature. *Neurosurgery.* 1984;15:705-6.
9. Okada J, Tsuda T, Takasugi S, Nishida K, Tóth Z, Matsumoto K, et al. Unusually late onset of cerebrospinal fluid rhinorrhea after head trauma. *Surg Neurol.* 1991;35:213-7.
10. Pandya PM, Keogh AJ. Traumatic cerebrospinal fluid rhinorrhoea: A timely reminder *Injury.* 1991;22:492.
11. Stewart BT, Kaye AH. Delayed cerebrospinal fluid rhinorrhoea: A case report. *Aust N Z J Surg.* 1992;62:818-20.
12. Crawford C, Kennedy N, Weir WR. Cerebrospinal fluid rhinorrhoea and haemophilus influenzae meningitis 37 years after a head injury. *J Infect.* 1994;28:93-7.
13. Salca HC, Danaila L. Onset of uncomplicated cerebrospinal fluid fistula 27 years after head injury: Case report. *Surg Neurol.* 1997;47:132-3.
14. Rao K, Shukla D, Indira Devi B. Unusually delayed posttraumatic CSF rhinorrhea. *Indian J Neurotrauma.* 2010;7:171-2.
15. Kamochi H, Kusaka G, Ishikawa M, Ishikawa S, Tanaka Y. Late onset cerebrospinal fluid leakage associated with past head injury. *Neurol Med Chir (Tokyo)* 2013;53:217-20.
16. Guyer RA, Turner JH. Delayed presentation of traumatic cerebrospinal fluid rhinorrhea: Case report and literature review. *Allergy Rhinol (Providence)* 2015;6:188-90.

# Guidelines for authors

## 1. ETHICS

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

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

**Acceptable percentage of resemblance - 5%.**

Duplicate content in research papers shall be considered only up to 5% of the total content.

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

### Links:

Committee on Publication Ethics

(COPE): <http://www.publicationethics.org>

World Association of Medical Editors

(WAME): <http://www.wame.org>

International Committee of Medical Journal Editors

(ICMJE): <http://www.icmje.org>

## 2. ENCLOSED LETTER

In addition to the manuscript, the Editorial Board should receive an enclosed letter containing the exclusive reservation of copyright guaranteed by all authors whose manuscripts have already been accepted. If the paper was completely or partially published or exposed previously, a copy or a photocopy of it should be also sent. The technical reports should contain a declaration concerning the financial sources that cover the costs necessary for instruments and methodology acquisition.

In order to illustrate different cases, photos of identifiable patients will not be published without their legal consent or that of their legal representative. The letter containing this consent together with the manuscripts should be sent to the editorial office.

If the author wishes his unpublished manuscripts returned, please note this in the enclosed letter.

## 3. SENDING OF MANUSCRIPTS

The manuscript sent for publishing must be submitted in English.

**Authors shall ensure that the article has been spell and grammar checked prior to the submission.**

The manuscript will be typed without formatting, with 1-line space. Please enclose one copy of the manuscript, tables, graphics and photos. After publishing, the paper and pictures become the property of Romanian Neurosurgery.

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

## 4. MANUSCRIPT ELABORATION

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

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

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

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

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

## 5. REFERENCES

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

#### **6. TABLES AND PICTURES**

There should be 1 copy sent. Each table with its own title should be submitted on separate pages. The photos, radiographies, CT scans should be labelled on the back with the number of each picture, corresponding with the number included inside the text, and the author's name. The label will be placed on the top of the picture. The drawings may be sent on vellum paper, tracing paper or transparent paper. The use of pictures belonging to other publications is accepted, only if mentioning the original source. Further, the partial use of any previously published text is accepted with the approval of its author and editor. All pages should be consecutively numbered, starting with the title page.

#### **7. ABBREVIATIONS**

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

#### **8. SUBMISSION**

Manuscripts should be sent using the online submission platform or to the Editor:

**Vicentiu Saceleanu, MD Dr.**

**([vicentiu.saceleanu@ulbsibiu.ro](mailto:vicentiu.saceleanu@ulbsibiu.ro))**

#### **9. DISCLAIMER**

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

#### **10. THE REVIEW PROCESS**

After the manuscript submission, the peer review process is broken down into the following steps:

1. The Editor assigns Reviewers to the manuscript.
2. The Reviewers review the manuscript.
3. The Editor drafts a decision to be sent to the author/authors.

The review process takes between three weeks and two months.