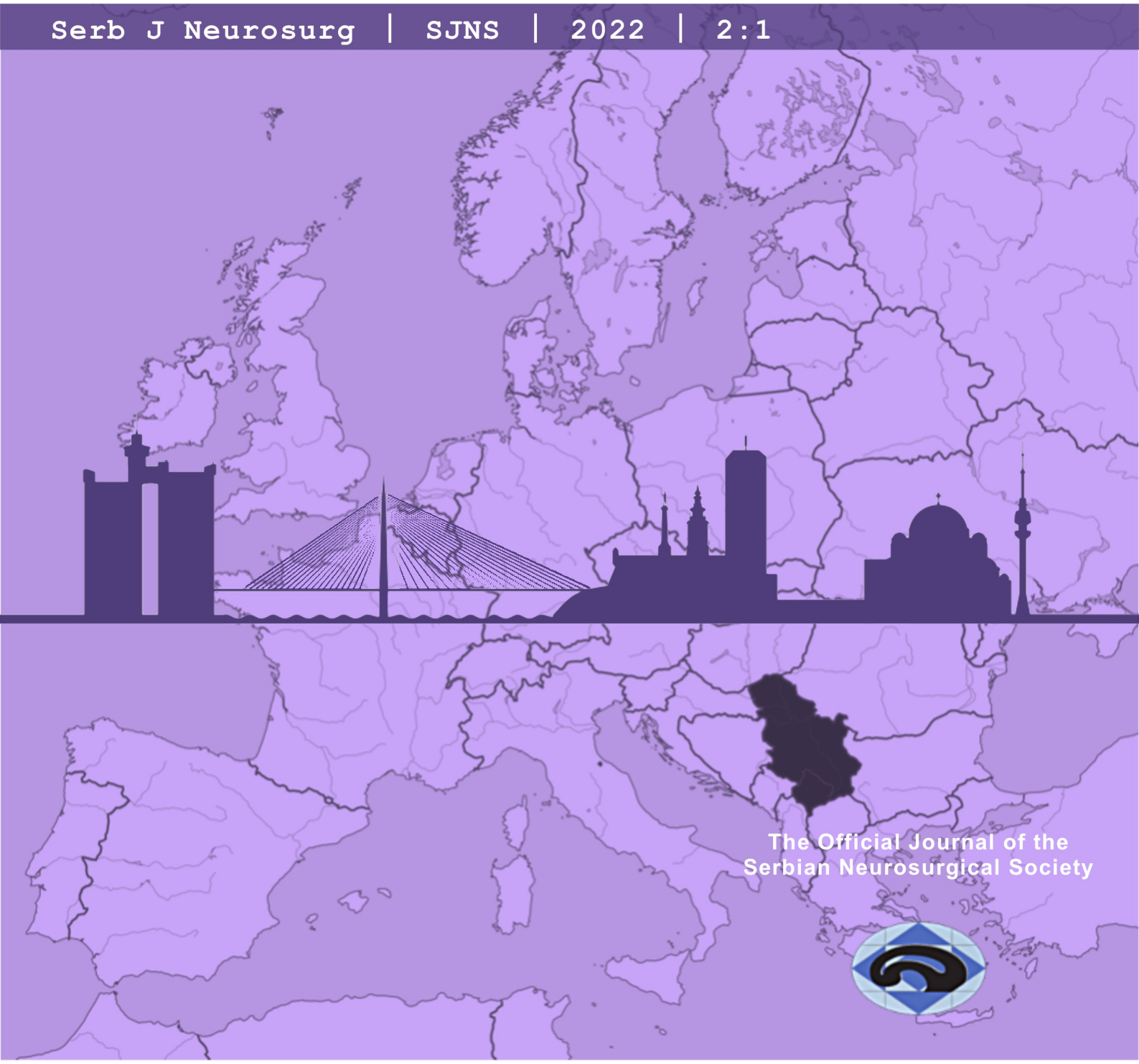
ISSN 2955-9162

НЕУРОХИРУРГИЈА

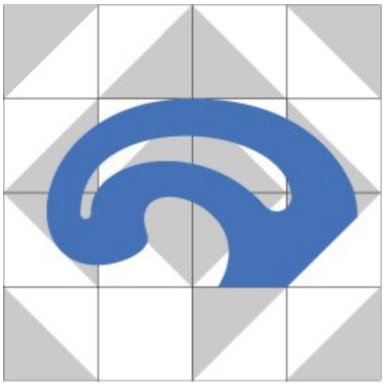
The Serbian Journal of Neurosurgery

Serb J Neurosurg | SJNS | 2022 | 2:1



The Official Journal of the
Serbian Neurosurgical Society

neurohirurgija.com



НЕУРОХИРУРГИЈА

The Serbian Journal of Neurosurgery

Publisher

Media Education Centre, Obilićev Venac 21, 11000 Belgrade, Serbia

Print

BAST, Kumodraška 44, Belgrade, Serbia

Contents

EDITORIAL

Why another journal? Well, why not?

Andreas Demetriades, MB.BChir. (Cantab), M.Phil. (Cantab), FRCSEd(Neuro.Surg), FEBNS[1,2]

1. European Association of Neurosurgical Societies (EANS)
2. Department of Neurosurgery, New Royal Infirmary, Edinburgh, UK

DOI: https://doi.org/10.55005/sjns.v2i1.1

As President of the European Association of Neurosurgical Societies (EANS), it is both an honour and a privilege to have been invited to write this editorial for the inauguration of *Neurohirurgija – The Serbian Journal of Neurosurgery*.

This new journal represents yet another venture from the neurosurgical community in Serbia, whose dedication and positive energy have made a mark over the last ten years and more in European neurosurgery, while expanding our professional family, and culminating in Belgrade being our host city for the EANS Annual Congress in October 2022.

Why another journal? one might ask. Well, why not?

Our professional field has continued to expand in both clinical and basic science and the medium of scientific communication continues to evolve. Our way of thinking or sharing knowledge ought not be subject to any 'monopoly' from any few established journals, whose contributions and quality remain important, but it should be open to innovation, competition, disruptive ideas and overall, to opportunities for our community - from younger to older - to communicate and share our thoughts, work, and insights.

Within a year from now the Serbian Neurosurgical Society will be celebrating its centenary. It is very relevant, therefore, that this journal has recently been launched. With its open access profile, and no charges to authors nor readers, this represents both progressive thinking and action.

Serbian and Balkan culture are a great mix of East and West, North and South, traditional and modern; a vibrant style that will hopefully translate into a strong and successful contribution to our clinical science. It is not easy to launch a journal and it is much harder to maintain one. But if it were easy, who would be interested.

I wish *Neurohirurgija – The Serbian Journal of Neurosurgery* the best of luck and great success; and look forward to reading and learning about the future of neurosurgery's art, craft, and science.

TECHNICAL NOTE

Temporalis muscle reattachment by using transosseus running suture along superior temporal line: technical note

Dinko Štimac, MD, PhD[1], Dragan Janković, MD[2,3]

1. Private Clinic "Ruđer-Medikol" Zagreb, Croatia
2. Department of Neurosurgery, University Medical Center of the Johannes Gutenberg University of Mainz, Mainz, Germany
3. Faculty of Medicine, Josip Juraj Strossmayer University, Osijek, Croatia

Abstract

Introduction: After reattachment of the temporalis muscle, atrophy of the temporalis muscle may occur, which is associated with difficulty in chewing function. To prevent this, numerous surgical modifications have been made to allow reattachment of the temporalis muscle with minimal damage.

Methods: We describe the technical details of surgical modification for reattachment of the temporalis muscle in 12 cases treated surgically in our department.

Results: We used a transosseous continuous suture along the superior temporal line as a base for reattachment of the muscle. The temporalis muscle was successfully reattached in all observed cases. No infections, dislocations, muscle tears, or significant temporal atrophy with depression occurred in any of the observed cases. In the author's technique, the temporalis muscle is reconstructed anatomically at the level of the superior temporal line. At follow-up after approximately 24 months, all patients were satisfied with the cosmetic result.

Conclusions: The use of running sutures along the superior temporal line is a safe, simple, and successful alternative for reattachment of the temporal muscles in patients undergoing surgery for intracranial pathology. The surgery takes slightly longer but does not require additional costs. This technique minimizes the risk of atrophy of the temporal muscles. With this technique, muscle tension was maintained with good stabilization and the cosmetic result is also satisfactory.

Keywords: craniotomy; operative technique; temporalis muscle; superior temporal line

DOI: https://doi.org/10.55005/sjns.v2i1.2

Introduction

Temporal hollowing and cosmetic deformity often follow after the temporalis muscle has been reattached from the underlying skull[1]. Once the temporalis muscle is repositioned, bony remodeling at the deep surface aids its reattachment[2]. Because the temporalis muscle lifts the mandible when it contracts and closes the teeth when chewing, dysfunction of the temporalis muscle can affect the patient's quality of life.

A viable muscle flap can be obtained with subperiosteal dissection and elevation of the temporalis muscle[3]. Although the temporal fascia is divided into superficial and deep layers, the temporalis muscle must be elevated along with the fascia from the superior temporal line[4].

There are numerous different techniques for reattachment of the temporalis muscle after pterional and orbitozygomatic craniotomy[5-7]. In this article, the authors describe the technical aspect of modified reattachment of the temporalis muscle.

Methods

Operative technique

Recent advances in neurosurgery include satisfactory postoperative cosmetic results. To achieve this, bone, muscle, fascia and skin must be as close as possible to the ideal anatomical reconstruction.

Detachment of the temporalis muscle

The skin incision is made in the regular fashion for pterional or orbitozygomatic craniotomy in all patients. The skin flap is retracted with fishhooks. The pericranium is separated from the skull in the frontal region. The temporalis fascia and intact temporalis muscle are sharply incised along the anterior 2/3 of the superior temporal line and elevated using the retrograde dissection technique. A vertical incision is then made through the muscle toward the pina. To minimize muscle atrophy, we did not use monopolar cauterization for hemostasis or dissection. A standard pterional craniotomy is then performed with or without fronto-orbital extension.

Reattachment of the temporalis muscle

Once the intracranial surgery is completed, small drill holes are made along the superior temporal line on the bone flap, at 5-8 mm apart (***Figure 1***) or along the craniotomy margins if the craniotomy is performed along the superior temporal line (***Figure 2***). A running suture Vicryl 2-0 (Ethicon Inc., Raritan, NJ) is placed through these holes. At the time of closure, the bone flap was replaced and fixed with titanium miniplates and screws or the FlapFix system. The temporal muscle flap and temporalis fascia are rotated backward and reattached with interrupted single Vicryl 2-0 (Ethicon Inc., Raritan, NJ) sutures at the running suture along the superior temporal line (***Figure 1***). Thus, the transosseous running suture mimics the "muscular cuff" and serves as a base for muscle fascia reattachment.

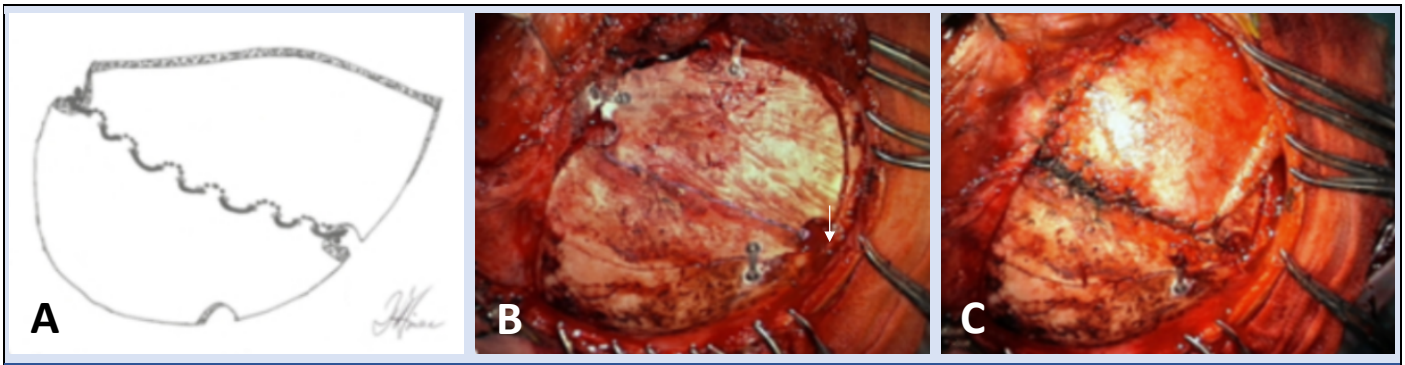


Figure 1. **A.** Artist's depiction shows a running stich alongside superior temporal line. **B.** After fixation of the bone flap with the microplate reattachment of the temporalis muscle can start. **C.** The temporalis muscle is tighty reattached to the running stich alongside superior temporal line (based on anatomical reconstruction).

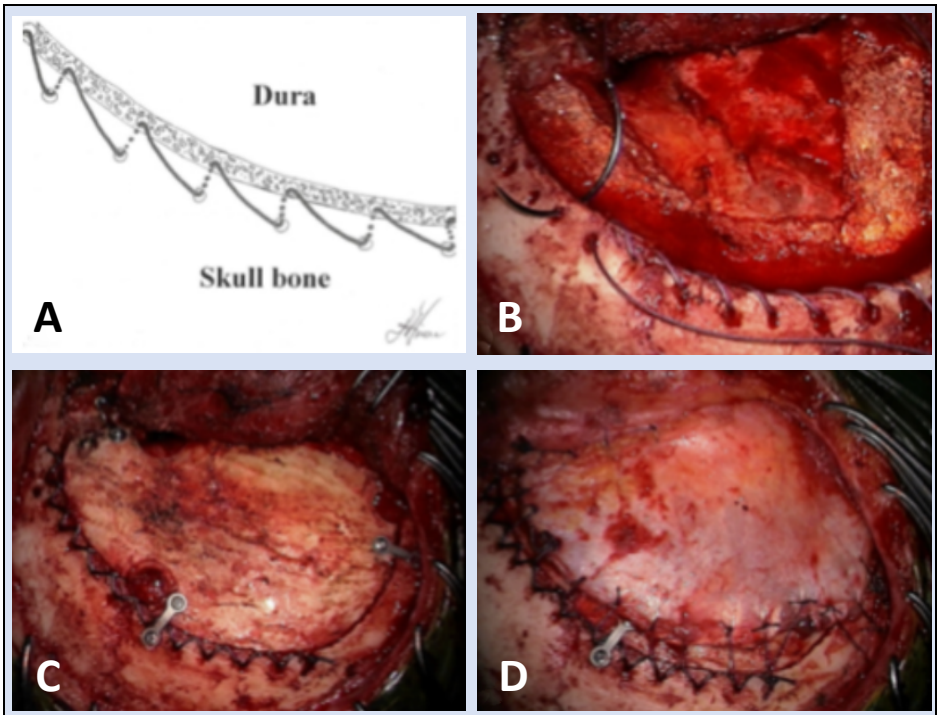


Figure 1. **A.** The drawing shows small holes through the bone edge which have been made by the high-speed drill **B**. Running stich through the small holes on the edge of the skull bone. **C.** Bone flap have been fixed with the 3 titanium microplates and screws. **D.** Temporalis muscle, fascial and galeal tissue have been tightly reattached to the running stich alongside craniotomy edge.

When a craniotomy is performed along the superior temporal line, a running suture can be made along the superior temporal line to reattach the temporalis muscle (***Figure 2***). The muscle incision perpendicular to the skull base is closed in the usual fashion. It is important to achieve the best possible anatomic reconstruction of the temporalis muscle and fascia toward the bony flap.

T1 sequences of brain MRI were used to measure the temporal muscle. Temporal muscle thickness was measured manually using DICOM Viewer (version 4.6.9). The baseline of the temporalis muscle was measured perpendicular to the long axis of the temporalis muscle at the level of the orbital roof. The Sylvian fissure was used as an anatomical reference point in the anterior-posterior direction. The temporalis muscle was measured on the side on which the surgery was performed, as shown in Figure 3. Measurements were performed by both authors (D.S. and D.J.), and discrepancies were resolved by consensus.

Results

Table 1 shows the group of patients with all measurements. In 12 patients, the gender distribution is equal (6 men and 6 women). The median age was 52.5 years (range, 36 to 68). All patients underwent extended pterional craniotomy. Intracranial aneurysm was diagnosed in 6 patients (2 anterior communicating artery aneurysm, 2 ACI aneurysm, 1 anterior cerebral artery aneurysm, 1 posterior communicating artery aneurysm), whereas tumor was present in the other 6 patients. The most common tumor was a meningioma of the sphenoid wing (in 3 patients), followed by a meningioma of the tuberculum sellae (in 1 patient), a clinoidal meningioma (in 1 patient), and a suprasellar pituitary adenoma (in 1 patient).

Regarding temporal muscle measurements, the preoperative median of the temporal muscle measurement was 6.95 mm, whereas at follow-up it was 6.95 mm at 3 months and 6.92 mm at 6 months (***Table 1***)

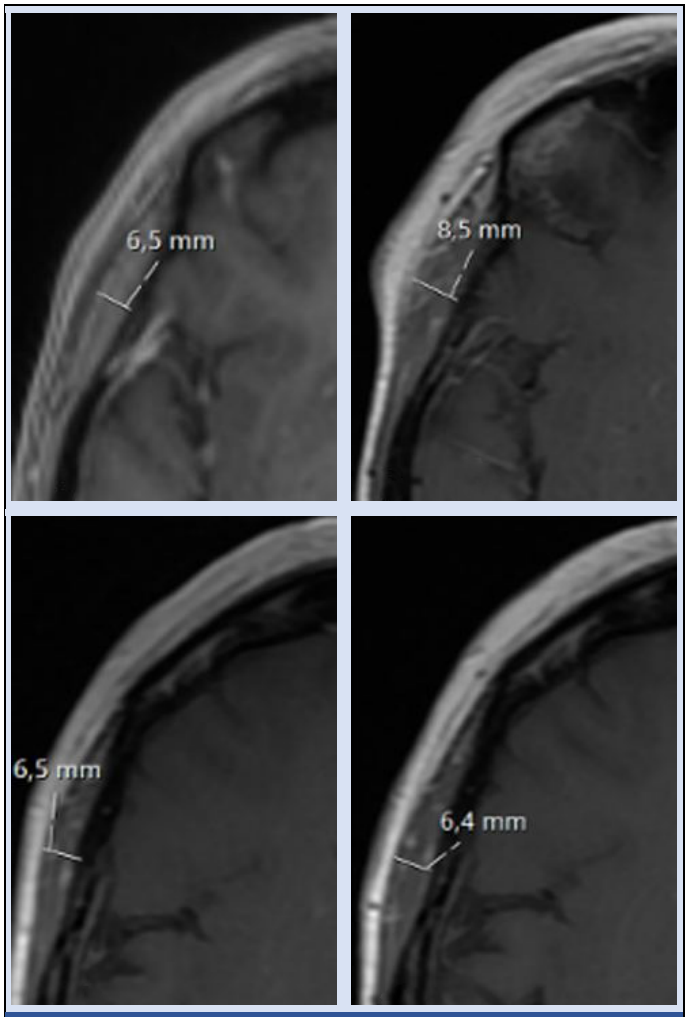


Figure 3. Temporal muscle thickness (TMT) measurement on cranial MRI. A. preoperatively, B. at 1 week postoperatively, C. at 3 months postoperatively, D. at 6 months postoperatively

Table 1. Patient characteristics

No.	Age/ Gender	Diagnosis	Measurement of Temporalis muscle thickness (TMT) (mm)			
			preoperatively	at 1 week postoperatively	at 3 months postoperatively	at 6 months postoperatively
1	54/F	Anterior communicating artery aneurysm	7,1	9,3	7,0	7,0
2	38/M	Sphenoid wing meningiomas	7,8	8,9	7,9	7,8
3	55/F	Clinoidal meningioma	6,5	8,5	6,5	6,4
4	56/F	Anterior communicating artery aneurysm	7,2	9,1	7,2	7,1
5	62/M	ACI aneurysm	6,6	7,9	6,6	6,6
6	45/M	Tuberculum sellae meningioma	7,8	9,6	7,7	7,6
7	42/M	Anterior cerebral artery aneurysm	7,3	8,9	7,4	7,4
8	35/F	Sphenoid wing meningioma	7,0	8,9	7,2	7,0
9	68/F	Suprasellar pituitary adenoma	5,6	7,8	5,7	5,7
10	57/M	ACI aneurysm	7,6	9,4	7,5	7,6
11	51/F	Posterior communicating artery aneurysm	7,0	8,9	6,8	6,8
12	67/M	Sphenoid wing meningioma	5,9	8,1	6,0	6,0

Discussion

Several methods have been described for the temporalis muscle reattachment to its site of origin. Wells and Kirn describe suturing the muscle to a cuff of the temporalis muscle left attached to the superior temporal line[8]. Frequently, suture tears the muscle cuff either during the suturing or soon after the surgery, because of the contractions of the temporalis muscle. Reattachment of the muscle to the aponeurotic galea frequently tears on the side of the galea, and the muscle becomes loosely attached and pulled down toward the zygomatic arch, with a subsequent bump on that region, and functional and cosmetic results far from the ideal.

Our study showed that after the postoperative swelling of the temporal muscle (measured one week after surgery), the muscle tissue gradually recovered to the preoperative condition.

In addition, the thickness of the temporal muscle decreased slightly at the 6-month follow-up (Table 1).

Hoenig et al. described a technique for anchoring the detached temporalis muscle[9]. Using the V-tunnel drill system, a V-shape tunnel is made through which suture is pulled and the temporal muscle is attached to the bone by consecutive stiches[10]. Thick drills can cause a sulcus instead of a tunnel, while thin drills create too narrow a tunnel for the suture needle to pass through.

There are several methods of positioning microscrews. In the technique described by Stechison, the microscrews were placed directly above the superior temporal line[7].

Webster et al. described a similar technique using microscrews, but in their technique, the microscrews are placed beneath the muscle[11]. The advantage of adsorbable plates and screws has led many authors to suggest that they should be a method of choice for muscle fixation in pediatric patients[12,13]. Another advantage of these plates and screws is avoiding complications with titanium plates, which may cause dose enhancement and shielding of the adjacent tissue[14].

In an animal study comparing self-reinforced polylevolactide and metal miniplates, it showed the same efficacy, while rapidly degradable self-reinforced polyglycolide showed less effective consolidation than on the contralateral side treated with titanium[15]. To minimize the risk of atrophy of the temporalis muscle before craniotomy, subperiosteal elevation of the muscle must be performed, avoiding monopolar cauterization[6].

Conclusion

Reconstruction of the temporalis muscle is a challenging task for neurosurgeons. The technique described achieves an almost anatomical approximation between the bony and muscular flaps. In addition to good stabilization, an excellent cosmetic result was also achieved. By attaching the temporalis muscle to the superior temporal line, a good, nearly ideal anatomic reattachment of the temporalis muscle is achieved. Further studies should focus on prolonged patient follow-up, with emphasis on measuring the thickness and surface area of the temporalis muscle.

Disclosures

Conflict of Interest: All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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

Funding: No funding was received for this research.

Acknowledgement

The first author would like to thank Professor Laligam Sekhar, his great teacher and friend, former Director of the World Skull Base Association, and Professor Rudolf Fahlbusch for his guidance, helpful suggestions and the review of this article, drawings and charts.

Correspondence

Dinko Štimac

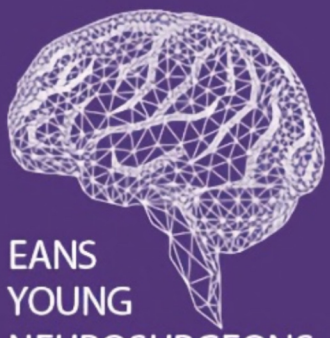
Private Clinic "Ruđer-Medikol", Kneza Branimira 119a
10000, Zagreb, Croatia

dinkostimac@hotmail.com

References

1. Oikawa S, Mizuno M, Muraoka S, Kobayashi S. Retrograde dissection of the temporalis muscle preventing muscle atrophy for pterional craniotomy. Technical note. J Neurosurg. 1996;84(2):297-299. doi:10.3171/jns.1996.84.2.0297
2. Chierici G, Miller AJ. Experimental study of muscle reattachment following surgical detachment. J Oral Maxillofac Surg. 1984;42(8):485-490. doi:10.1016/0278-2391(84)90006-5
3. Zager EL, DelVecchio DA, Bartlett SP. Temporal muscle microfixation in pterional craniotomies. Technical note. J Neurosurg. 1993;79(6):946-947. doi:10.3171/jns.1993.79.6.0946
4. Hochberg J, Kaufman H, Ardenghy M. Saving the frontal branch during a low frontoorbital approach. Aesthetic Plast Surg. 1995;19(2):161-163. doi:10.1007/BF00450252

5. Bowles AP, Jr. Reconstruction of the temporalis muscle for pterional and cranioorbital craniotomies. Surg Neurol. 1999 Nov;52(5):524-9.
6. Spetzler RF, Lee KS. Reconstruction of the temporalis muscle for the pterional craniotomy. Technical note. J Neurosurg. 1990;73(4):636-637.doi:10.3171/jns.1990.73.4.0636
7. Stechison MT. Temporal muscle fixation. J Neurosurg. 1995;82(4):701-702.
8. Wells MD, Kirn DS. Bone anchors: new application in craniofacial surgery. J Craniofac Surg. 1996;7(2):164-167.
9. Hönig JF. V-tunnel drill system in craniofacial surgery: a new technique for anchoring the detached temporalis muscle. J Craniofac Surg. 1996;7(2):168-169. doi:10.1097/00001665-199603000-00021
10. Castillo R, Zarate A, Chavez R. Reconstruction of the temporalis muscle. Journal of
Neurosurgery. 1992 Feb;76(2):336. DOI: 10.3171/jns.1992.76.2.0336.
11. Webster K, Dover MS, Bentley RP. Anchoring the detached temporalis muscle in craniofacial surgery. J Craniomaxillofac Surg. 1999;27(4):211-213. doi:10.1016/s1010-5182(99)80031-6
12. Eppley BL, Sadove AM, Havlik RJ. Resorbable plate fixation in pediatric craniofacial surgery. Plast Reconstr Surg. 1997;100(1):1-13. doi:10.1097/00006534-199707000-00001
13. Habal MB. Fixation, imaging, and resorption. J Craniofac Surg. 1996;7(5):325. doi:10.1097/00001665-199609000-00001
14. Rozema FR, Levendag PC, Bos RR, Boering G, Pennings AJ. Influence of resorbable poly(L-lactide) bone plates and screws on the dose distributions of radiotherapy beams. Int J Oral Maxillofac Surg. 1990;19(6):374-376. doi:10.1016/s0901-5027(05)80086-4
15. Peltoniemi HH, Tulamo RM, Toivonen T, Hallikainen D, Törmälä P, Waris T. Biodegradable semirigid plate and miniscrew fixation compared with rigid titanium fixation in experimental calvarial osteotomy. J Neurosurg. 1999;90(5):910-917. doi:10.3171/jns.1999.90.5.0910



EANS Young Neurosurgeons **Network**

Join** the Young Neurosurgeons Network **now** and take a seat in the **future of Neurosurgery!

youngneurosurgeons.eans.org/ynn/

CASE REPORT

Delayed cerebral vasospasm following traumatic acute subdural hematoma: case report

Kaima Suzuki, MD, PhD[1], Milan Lepić, MD, PhD[1],Hiroki Sato, MD, PhD[1], Shinya Tabata, MD[1], Tatsuki Kimura, MD[1], Hiroki Kobayashi, MD[1], Keijiro Yoshida, MD[1], Masayoshi Fukuoka, MD[1], Nobuyuki Yahagi[1], Takuma Maeda, MD[1], Hidetoshi Ooigawa, MD, PhD[1], Hiroki Kurita, MD, PhD[1]

1. Department of Cerebrovascular Surgery, Saitama Medical University International Medical Center, Hidaka, Japan

Abstract

Introduction: Post-traumatic vasospasm is a rare but known ischemic damage after severe traumatic brain injury that independently predicts patients' outcome. Although the pathogenesis and risk factors have not been elucidated, some reports describe relationship between the occurrence of vasospasm and traumatic subarachnoid hemorrhage. Here, we report a case of vasospasm in a patient with acute subdural hematoma in which traumatic subarachnoid hemorrhage was not recognized both surgically and radiologically.

Case report: A 60-year-old male was admitted for head trauma. Neurologically, he was somnolence and showed right-sided hemiparesis. Computerized tomography (CT) revealed large acute subdural hematoma in the left side associated with midline-shift. He underwent urgent craniotomy and hematoma evacuation. Postoperatively, he recovered well with resolution of neurological symptoms. Follow-up CT revealed complete removal of hematoma. However, his level of neurological status deteriorated on the 5th day after surgery. CT excluded ischemic lesion, but 3D-CT angiography revealed diffuse vasospasm in the left middle cerebral artery, and perfusion imaging confirmed a zone of altered cerebral blood flow in left frontotemporal region. Subsequently, his neurological condition recovered gradually and he was discharged ambulatory on the 9th day after the surgery. Follow-up angiography showed the spasm had disappeared completely.

Conclusion: Post-traumatic vasospasm without traumatic SAH was described. The etiology and pathogenesis of this fairly rare condition associated with head trauma is discussed.

Keywords: cerebral vasospasm; traumatic brain injury; subdural hematoma; delayed cerebral ischemia

DOI: https://doi.org/10.55005/sjns.v2i1.3

Introduction

Cerebral vasospasm is an arterial narrowing, usually following the subarachnoid and/or intraventricular hemorrhages in course of the aneurysm rupture [1]. Moderate or severe vasospasm develops in roughly two thirds, followed by the delayed cerebral ischemia in one third, followed by infarction in one sixth of patients with subarachnoid hemorrhage (SAH).

Post-traumatic vasospasm (PTV) is not common but it is a well-known complication related to the severe traumatic brain injury that independently comprises the outcomes thorough a delayed cerebral ischemia mechanism [2].

Although the pathogenesis and risk factors have not been elucidated, there are several current pathophysiological mechanisms based on the molecular level response, underlying the occurrence of the PTV, while the mechanical theories based on the direct stretching were abandoned [3]. These theories are all focused to describe this occurrence considering the presence of traumatic SAH.

Conversely, pure acute subdural hematoma of traumatic or spontaneous origin is considered to be a rare cause of the PTV [4]. We report a case of a delayed PTV after the surgery for traumatic acute subdural hematoma (ASDH) in which traumatic SAH was excluded both intraoperatively and radiologically.

Case report

A 60-year-old male was referred from the local hospital after a head trauma, and CT confirmed ASDH. On admission, the patient was somnolent - Glasgow coma scale (GCS): 12 (E2V4M6), with equal pupils and prompt light reflex, and showed mild right-sided hemiparesis. Computerized tomography (CT) revealed enlarged ASDH on the left side associated with moderate midline-shift compared to initial imaging. Traumatic SAH was not evident (***Figure 1A***). Three-dimensional CT angiography (3D-CTA) excluded underlying vascular pathology and cerebral vasospasm (***Figure 2A***).

The patient soon deteriorated neurologically and underwent urgent craniotomy and hematoma evacuation. Intraoperatively, Skull fracture or dural injury were not observed. The hematoma was localized entirely in the subdural space and SAH was not identified in subarachnoid space, basal cisterns or Sylvian fissure. Small laceration of cortical middle cerebral artery branch on the convexity was found to be the bleeding source, and it was completely coagulated. Postoperatively, he recovered well with resolution of neurological symptoms, and CT confirmed complete removal of ASDH (***Figure 1B***).

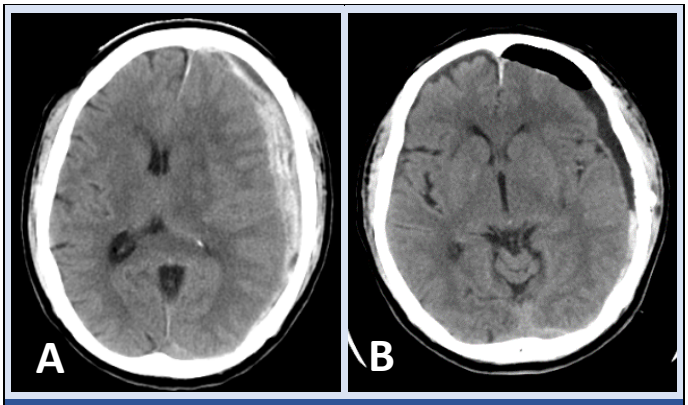


Figure 1. Computerized tomography A. Preoperative, showing left-sided ASDH without traumatic SAH. B. Postoperative, showing complete evacuation of the ASDH with decrease of the mid-line shift.

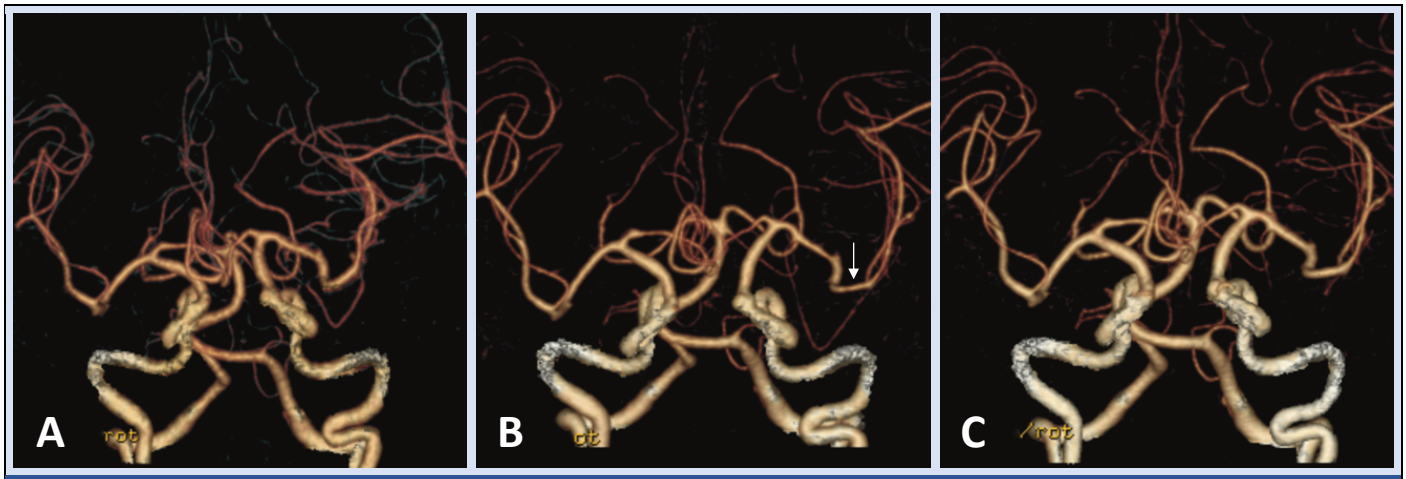


Figure 2. 3D CT angiography findings A. Initial angiography on day 0 showing no vasospasm; B. The arrow points at the vasospasm site on day 5; C. The vasospasm is relieved completely on day 9.

On the 5th day after surgery, the patients' neurological status started to deteriorate with initial dysarthria, which worsened to aphasia within several hours, followed by the development of the right-sided severe hemiparesis. A perfusion CT revealed signs decreased cerebral blood flow (CBF) in the left frontotemporal region, and 3D-CTA found vasospasm of the left sided middle cerebral artery stem (***Figure 2B and 3A***). Vasospasm treatment protocol was introduced with triple H therapy (hypertension, hypervolemia, and hemodilution), with only Atorvastatin 10mg/day as complete vasospasm treatment protocol was contraindicated due to the trauma.

The patient improved gradually over the following days. Hemiplegia improved on the following day, and completely resolved in two days, while the aphasia persisted for 3 days. On the 9th day after the surgery, the patient was discharged ambulatory, with complete symptoms resolution, and 3D-CTA confirmed complete resolution of the vasospasm of the left MCA, while the perfusion CT confirmed blood flow normalized (***Figure 2C and 3B***).

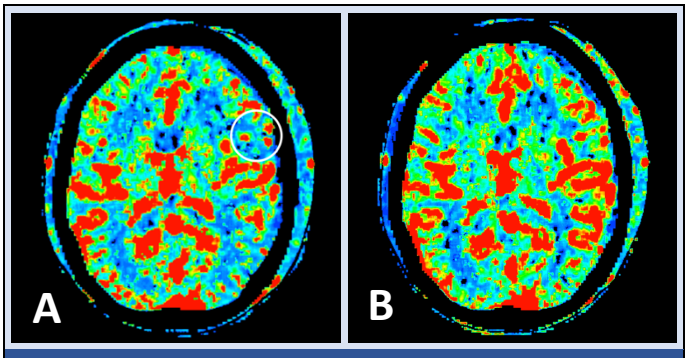


Figure 1. Postoperative perfusion CT findings showing A. altered cerebral blood flow in the frontotemporal region on day (the circle emphasizes the most severe frontal region insufficiency) 5; B. complete resolution on day 9.

Discussion

Post-traumatic vasospasm in our patient developed on the 5th day after the surgery for ASDH, leading to the neurological worsening and confirmed with 3D-CTA. Usual vasospasm treatment protocol led to the CT and symptoms resolution after 4 days.

Cerebral vasospasm is known to occur after head injury; reported incidence reaches up to 63%, however, the screening for PTV is not routine in these patients. Originally, angiographic studies reported PTV rates between 5% and 18.6%, with the introduction of transcranial Doppler sonography and improved imaging modalities, the rates of PTV have increased ranging from 27% to 63% [5].

The origin of PTV remains unknown. Presence of a subarachnoid clot, especially higher Fischer score SAH has been reported to be a major contributing factor in the development of PTV [6], but additional presence of intracerebral [7], subdural and epidural hematomas, SAH [8] are also thought to be causative. However, distinct feature in our patient is occurrence after acute, purely subdural hematoma.

In a wartime cohort, vasospasm was associated with the presence of pseudoaneurysm, hemorrhage, the number of lobes injured, contributing to the injury severity, and impacting the mortality [9, 10]. In our patient, there was no pseudoaneurysm, however, the origin of bleeding was a cortical artery, which may have induced pathophysiological cascade.

The knowledge on the pathophysiology of the PTV development is scarce, and it is most likely a combination of interrelated mechanisms following the SAH, including the altered vascular response to nitric oxide, impact of hemoglobin and its degradation products, inflammation, cortical spreading depolarization and microcirculation disturbance [11]. Hypothalamic dysfunction secondary to increased intracranial pressure and blood degradation products of ASDH might have played some role in the development of the vasospasm in our patient by triggering a sympathetic discharge and catecholamine release [12, 13].

The usual timing for the development of vasospasm after aneurysmal SAH is typically 4-14 days [14]. On the other hand, PTV is characterized by the earlier onset, most frequently in the first 3 days following the injury [4], and it usually lasts less than aneurysmal SAH related; rarely more than 6 days [5]. These characteristics were present in our patient, as the vasospasm occurred on 5th day after injury/surgery and lasted for the following 4 days. Clinically, fever on admission [6], and initial poor neurological status with GCS<9 [15] are also found to be a predisposing factor. All of which were not present in the presented case.

Although it was not possible to apply complete vasospasm treatment protocol due to the traumatic origin of the vasospasm, the treatment based on the triple-H therapy was used for the management with a favorable outcome. The reported clinical outcome of PTV is generally good, but may be also related to the bad prognosis and death [16]. Practical perfusion CT and 3D-CTA were helpful to detect this fairly rare condition, and response with the adequate treatment promptly, regardless of the usual dissociation of the PTV and ASDH.

Conclusions

Delayed PTV can develop even without traumatic SAH, after surgery for ASDH in rare cases. Perfusion CT and 3D-CTA is practical and effective method for early detection in suspicious cases, while the novel therapeutic modalities allow for an uneventful PTV resolution.

Disclosures

Conflict of Interest: All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent: Informed consent was obtained from the reported individual.

Funding: No funding was received for this research.

Correspondence

Kaima Suzuki

Department of Cerebrovascular Surgery and Stroke Center, International Medical Center, Saitama Medical University, 1397-1 Yamane, Hidaka, Saitama 350-1298, Japan.

+81429844111 (ext. 5579)

ks7121@5931.saitama-med.ac.jp

References

1. Findlay JM, Nisar J, Darsaut T. Cerebral Vasospasm: A Review. Can J Neurol Sci. 2016;43(1):15-32. doi: 10.1017/cjn.2015.288.
2. Baggott CD, Aagaard-Kienitz B. Cerebral vasospasm. Neurosurg Clin N Am. 2014;25(3):497-528. doi: 10.1016/j.nec.2014.04.008.
3. Perrein A, Petry L, Reis A, Baumann A, Mertes P, Audibert G. Cerebral vasospasm after traumatic brain injury: an update. Minerva Anestesiol. 2015;81(11):1219-28. doi:
4. Witten AJ, Ordaz JD, Alentado VJ, Bohnstedt B. Intracranial Vasospasm After Evacuation of Acute Spontaneous Subdural Hematoma. Cureus. 2021;13(5):e15284. doi: 10.7759/cureus.15284.
5. Al-Mufti F, Amuluru K, Changa A, Lander M, Patel N, Wajswol E, et al. Traumatic brain injury and intracranial hemorrhage-induced cerebral vasospasm: a systematic review. Neurosurg Focus. 2017;43(5):E14. doi: 10.3171/2017.8.FOCUS17431.
6. Shahlaie K, Keachie K, Hutchins IM, Rudisill N, Madden LK, Smith KA, et al. Risk factors for posttraumatic vasospasm. J Neurosurg. 2011;115(3):602-11. doi: 10.3171/2011.5.JNS101667.
7. Oertel M, Boscardin WJ, Obrist WD, Glenn TC, McArthur DL, Gravori T, et al. Posttraumatic vasospasm: the epidemiology, severity, and time course of an

underestimated phenomenon: a prospective study performed in 299 patients. J Neurosurg. 2005;103(5):812-24. doi: 10.3171/jns.2005.103.5.0812.
8. Zubkov AY, Lewis AI, Raila FA, Zhang J, Parent AD. Risk factors for the development of post-traumatic cerebral vasospasm. Surg Neurol. 2000;53(2):126-30. doi: 10.1016/s0090-3019(99)00178-0.
9. Armonda RA, Bell RS, Vo AH, Ling G, DeGraba TJ, Crandall B, et al. Wartime traumatic cerebral vasospasm: recent review of combat casualties. Neurosurgery. 2006;59(6):1215-25; discussion 25. doi: 10.1227/01.NEU.0000249190.46033.94.
10. Vitošević F, Milošević-Medenica S. Traumatic intracranial aneurysms associated with traffic accidents and endovascular management options. Serbian J Neurosurg. 2022;1(1):27-32. doi: 10.55005/sjns.v1i1.5.
11. Chugh C, Agarwal H. Cerebral vasospasm and delayed cerebral ischemia: Review of literature and the management approach. Neurol India. 2019;67(1):185-200. doi: 10.4103/0028-3886.253627.
12. Wilkins RH. Cerebral vasospasm in conditions other than subarachnoid hemorrhage. Neurosurg Clin N Am. 1990;1(2):329-34. doi:
13. Wilson JL, Feild JR. The production of intracranial vascular spasm by hypothalamic extract. J Neurosurg. 1974;40(4):473-9. doi: 10.3171/jns.1974.40.4.0473.
14. Bracard S, Schmitt E. Vasospasm and delayed consequences. Interv Neuroradiol. 2008;14 Suppl 1(Suppl 1):17-22. doi: 10.1177/15910199080140S105.
15. Al-Mufti F, Amuluru K, Lander M, Mathew M, El-Ghanem M, Nuoman R, et al. Low Glasgow Coma Score in Traumatic Intracranial Hemorrhage Predicts Development of Cerebral Vasospasm. World Neurosurg. 2018;120:e68-e71. doi: 10.1016/j.wneu.2018.07.143.
16. Armin SS, Colohan AR, Zhang JH. Vasospasm in traumatic brain injury. Acta Neurochir Suppl. 2008;104(13):421-5. doi: 10.1007/978-3-211-75718-5.

CASE REPORT

Dorsal hemangioblastoma with holocord syringomyelia: case report

Alejandra Arévalo Sáenz, MD[1], Manuel Pedrosa Sánchez, MD[2]

1. Department of Neurosurgery, Hospital Clinic San Carlos, Madrid, Spain
2. Department of Neurosurgery, University Hospital La Princesa, Madrid, Spain

Abstract

Introduction: Intramedullary hemangioblastomas are usually accompanied by syringomyelia. However, a holocord syringomyelia is rare. The most common cause of syringomyelia continues to be Chiari disease, and only 10 cases of hemangioblastomas with holocord syringomyelia reported so far.

Case report: We present a case of a 35-year-old patient with a two-month history of cervicobrachialgia at the C7-C8 root level, previously preceded by pain at the D1-D2 level. Cervico-dorso-lumbar MRI revealed a medullar tumor with hyper-uptake mural nodule at the conus medullaris level accompanied by an extensive syringomyelic cavity from C5 to L1 compatible with medullary hemangioblastoma. The patient underwent surgery for tumor resection with subsequent resolution of her painful symptoms.

Conclusion: It is important to note that the surgery is aimed at treating the origin of this syringomyelia and not the syringomyelia itself. Although the majority of patients with holocord syringomyelia have Chiari as its cause, the possibility of focal spinal intramedullary tumors as being responsible for syringomyelia should not be forgotten.

Keywords: hemangioblastoma; holocord syringomyelia; spinal neoplasm; Chiari malformation; microsurgery

DOI: https://doi.org/10.55005/sjns.v2i1.4

Introduction

Spinal hemangioblastomas comprise 1.6-2.1% of all primary spinal cord tumors. They are the third most common primary spinal cord tumor, following astrocytoma and ependymoma, and represent 2%–6% of all intramedullary tumors. They are preferably located in the cervical or thoracic region and typically present as a highly vascularized solid lesion with an associated cystic component. Although between 50-70% of these tumors are associated with syringomyelia, they very rarely present a holocord syringomyelic cyst (1-3) Hemangioblastomas and ependymomas are the most common tumors associated with syringomyelia, while astrocytomas are usually more diffuse and with less tendency to present cysts (4)

Most hemangioblastomas occur sporadically, but up to 32% are part of the von Hippel-Lindau syndrome. 80% of these tumors are solitary (5).

Case report

A 35-year-old woman, with no history of interest, who debuted with acute symptoms of intermittent right cervicobrachialgia in C7-C8 dermatome of two months´evolution. The pain was preceded by right dorsal pain at the level of the T1-T2 territory. On examination, there is no neurological focality

Whole spine magnetic resonance imaging (MRI) (Figure 1) diagnosed an intramedullary tumor with a hyperenhanced mural nodule accompanied by an extensive syringomyelic cavity from C5 to L1 compatible with spinal hemangioblastoma. The rest of the pre-operative tests were normal.

The patient underwent scheduled surgery for tumor resection under neurophysiological control (somatosensory and motor potentials and root control). A D10 laminectomy with complete microsurgical excision of a hypervascularized subpial spinal cord lesion with multiple pial vascular microafferents was performed (Fig. 2). The surgery was performed in the anti-Trendelenburg position to avoid the massive leakage of cerebrospinal fluid from the extensive syringomyelic cavity.

In the postoperative period, the patient had dysesthesia at the T1 level and symptoms of fluid hypotension that remitted with acetazolamide. The pathology showed a result of hemangioblastoma.

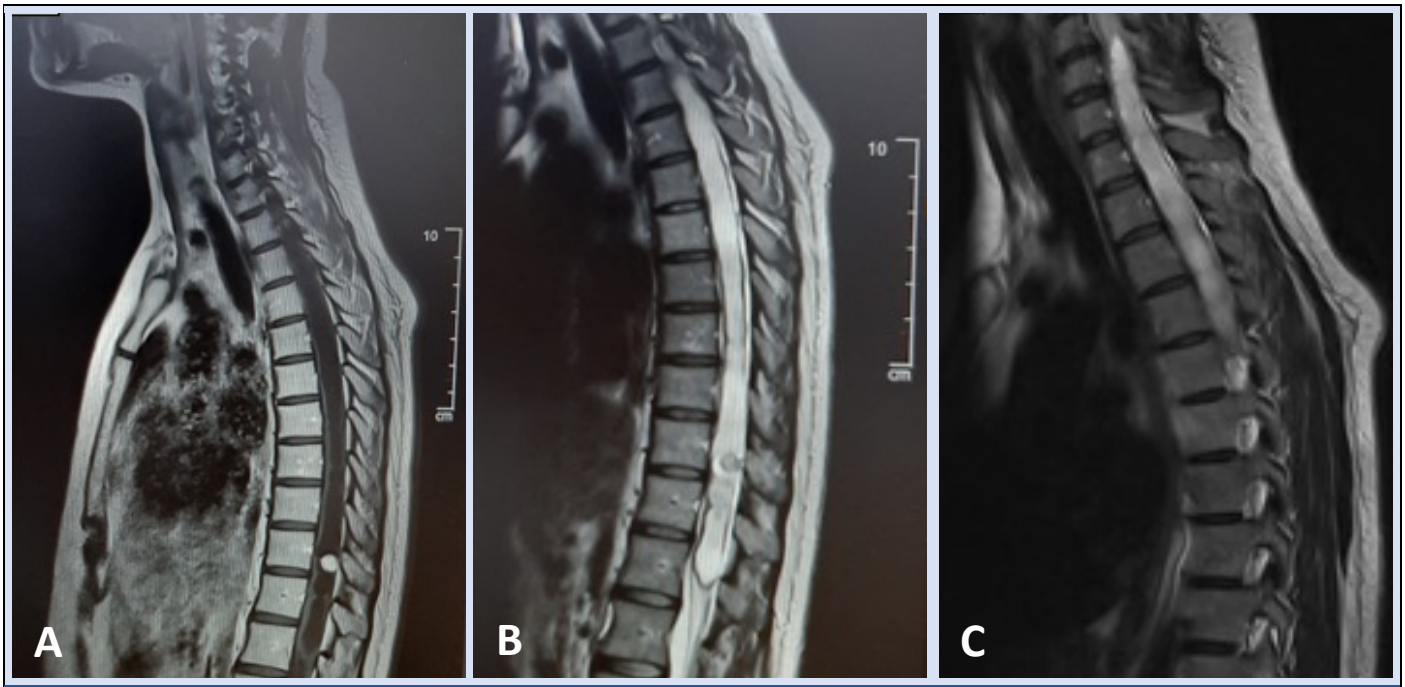


Figure 2. 3D Preoperative whole spine MRI. Sagittal slices on T1 (image A) and sagittal slices on T2 (images B and C). They demonstrate the existence of a well-defined intramedullary nodule at the level of D10, hypointense on T2, hyperintense on T1 with intense contrast enhancement surrounding it. It associates a prominent syringomyelic cavities from C5-C6 above to D12 below, occupying almost the entire cord.

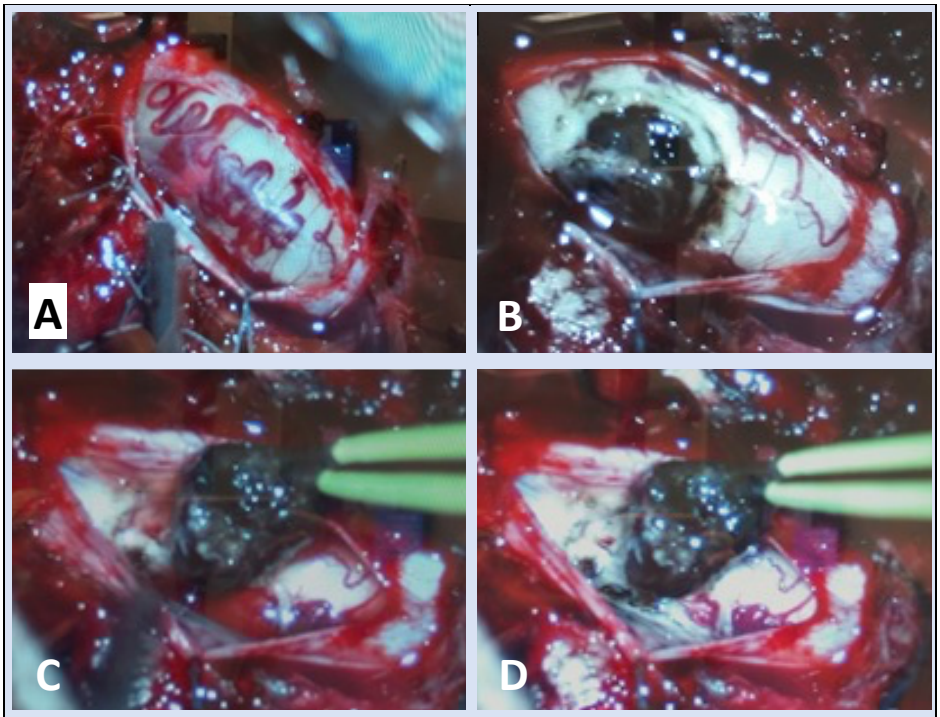


Figure 1. **A.** Intraoperative images after D10 dorsal laminectomy and dural opening showing the intramedullary nodule with pial vascular afferents (image A). In image B we can see the lesion once disconnected from the pial vascular supply. Images C and D detail the moment of total resection of the lesion.

Discussion

The appearance of a cyst or syrinx was reported in 53.94% of spinal hemangioblastomas, which most often appeared in the cervical and thoracic spine (3).

The theory behind the formation of syringomyelic cysts is that the high interstitial pressure of an intramedullary tumor leads to extravasation of plasma into the central canal of the spinal cord; that is, the fluid that expands the central canal comes directly from the tumor or its associated vascularization (4). Studies about high protein concentrations in the cerebrospinal fluid of tumoral intramedullary cysts support this theory (6). Other theories also combine the obstruction of the cerebrospinal fluid and/or the obstruction of the flow of perimedullary extracellular fluid (1,7).

Varying degrees of asymptomatic central spinal medullary stenosis, common in healthy adults based on autopsy studies (8), may limit further expansion of the central canal and explain the multiseptate appearance often seen in cysts associated with intramedullary tumors. It should be noted that the higher the tumor level, the higher the incidence of an accompanying cyst (6). However, there is no association between the levels of syringomyelia and severity of symptoms (3)

Syringomyelia is mainly associated with Chiari malformation and also with inflammatory pathologies, spinal attacks, trauma and intramedullary tumors of the spinal cord. Holocord syringomyelia is more frequently associated with Chiari malformation (9). In a patient with this diagnosis, an exhaustive evaluation of the entire spine should be performed to rule out other pathologies.

In the literature there are only 11 cases (1, 6, 10-15) of hemangioblastomas with holocord syringomyelia (Table 1). The main difference in this group of patients is the surgical treatment, since the massive leakage of cerebrospinal fluid from the cavity can lead to permanent neurological symptoms. The resolution of the cavity is carried out naturally once the cause of the cavity has been treated, either by tumor resection or Chiari treatment. Based on our experience and previous literature, we believe that intraoperative the opening of the syrinx is not necessary during surgery, as associated edema and syringomyelia usually disappear over time after complete surgery resection (3).

Table 1. General characteristics of hemangioblastomas with holocord syringomyelia previously reported in the literature

Reference	Year	Number of patients	Age	Gender	Clinical presentation	Duration	Tumor location	Extension syringomyelia
Pai SB et al.	2003	2	17	M	Progressive quadriparesis and bladder involvement	1 month	Medullary conus	C2 - medullary conus
			35	M	Dysesthetic pain in both lower limbs with progressive paraparesis	6 months	D8	Cervicomedullary -T10
Wu et al.	2005	1	20	M	Intermittent right upper extremity numbness	3 months	T10-T11	Cervicomedullary -T11
Borkar SA et al.	2009	1	38	F	Spastic quadriparesis and hesitancy with urge incontinence	6 months	C7/T1	Cervicomedullary -conus
Cosgrove et al.	2015	1	50	M	Muscle wasting and weakness of both legs	6 years	T6/T7	Cervicomedullary -D12
Dutta et al.	2018	1	21	F	Progressive numbness/tingling of the face, hands and feet, gait instability, and hesitancy/ urge incontinence	6 months	T4	Cervicomedullaris - T12
Pojskic et al.	2018	1	30	M	Sudden onset urinary incontinence	Days	T11	C1 - T11
Maejima et al.	2019	3	42	M	Abnormal sensation in left upper extremity	2 months	T6	-
			43	F	Left hand numbness	3 years	T4	-
			42	M	Back pain	years	T7-T8	-
Knoop et al.	2019	1	37	M	Screening for von Hippel Lindau disease	-	T1-T2 and T11-T12	Cervicomedullary -T10

Conclusions

Although most patients with holocord syringomyelia are caused by Chiari malformation, others may have focal spinal intramedullary tumors. Therefore, it´s essential to assess the entire spine to avoid missing these injuries and missing the opportunity to properly treat these patients.

Disclosures

Conflict of Interest: All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent: Informed consent was obtained from the reported individual.

Funding: No funding was received for this research.

Correspondence

Alejandra Arévalo Sáenz

✉ Department of Neurosurgery, Hospital Clinic San Carlos, Calle Profesor Martín Lagos, s/n, 28040 Madrid, Spain

☏ +34913303000

@ praimale@hotmail.com

References

1. Pai SB, Krishna KN. Secondary holocord syringomyelia with spinal hemangioblastoma: a report of two cases. Neurol India. 2003 Mar;51(1):67-8.
2. Sarikaya S, Acikgöz B, Tekkök IH, Güngen YY. Conus ependymoma with holocord syringohydromyelia and syringobulbia. J Clin Neurosci. 2007 Sep;14(9):901-4. doi: 10.1016/j.jocn.2006.06.005.
3. Jankovic D, Hanissian A, Rotim K, Splavski B, Arnautovic KI. Novel Clinical Insights into Spinal Hemangioblastoma in Adults: A Systematic Review. World Neurosurg. 2022 Feb;158:1-10. doi: 10.1016/j.wneu.2021.10.105.
4. Stoodley M. The filling mechanism. In: Flint G, Rusbridge C, eds. Syringomyelia—a disorder of CSF circulation. Berlin Heidelberg: Springer-Verlag, 2014:87–101.
5. Chu BC, Terae S, Hida K, Furukawa M, Abe S, Miyasaka K. MR findings in spinal hemangioblastoma: correlation with symptoms and with angiographic and surgical findings. AJNR Am J Neuroradiol. 2001 Jan;22(1):206-17.
6. Wu TC, Guo WY, Lirng JF, Wong TT, Chang FC, Luo CB, et al. Spinal cord hemangioblastoma with extensive syringomyelia. J Chin Med Assoc. 2005 Jan;68(1):40-4. doi: 10.1016/S1726-4901(09)70131-5.
7. Samii M, Klekamp J. Surgical results of 100 intramedullary tumors in relation to accompanying syringomyelia. Neurosurgery. 1994 Nov;35(5):865-73; discussion 873. doi: 10.1227/00006123-199411000-00010.
8. Milhorat TH, Kotzen RM, Anzil AP. Stenosis of central canal of spinal cord in man: incidence and pathological findings in 232 autopsy cases. J Neurosurg. 1994 Apr;80(4):716-22. doi: 10.3171/jns.1994.80.4.0716.
9. Kumar J, Kumar A, Gupta S. Neurological pictures. Chiari I malformation with holocord syrinx. J Neurol Neurosurg Psychiatry. 2007 Feb;78(2):146. doi: 10.1136/jnnp.2006.099440.
10. Pojskic M, Arnautovic KI. Microsurgical Resection of Spinal Cord Hemangioblastoma: 2-Dimensional Operative Video. Oper Neurosurg (Hagerstown). 2018 Dec 1;15(6): E88-E89. doi: 10.1093/ons/opy123.
11. Dutta G, Singh D, Singh H, Srivastava AK, Jagetia A, Agrawal A. Dorsal hemangioblastoma manifesting as holocord syringomyelia. Surg Neurol Int. 2018 Apr 5; 9:73. doi: 10.4103/sni.sni_47_18.
12. Maejima R, Kawaguchi R, Aoyama M, Takeuchi M, Joko M, Takayasu M. [Three Cases of Small Hemangioblastoma with a Large Syringomyelia]. No Shinkei Geka. 2019 May;47(5):573-576. Japanese. doi: 10.11477/mf.1436203984.
13. Cosgrove J, Warren D, Derham C, Jamieson S. Holocord syrinx associated with haemangioblastoma. Pract Neurol. 2015 Dec;15(6):485-7. doi: 10.1136/practneurol-2015-001251.
14. Borkar SA, Kasliwal MK, Suri A, Sharma BS. Cervical Hemangioblastoma with holocord syrinx. Surg Neurol. 2009 Oct;72(4):437-8. doi: 10.1016/j.surneu.2008.11.014.
15. Knoop N, Seidel C, Frydrychowicz C, Meixensberger J. Combined Microsurgery and Radiotherapy for Multiple Spinal Cord Hemangioblastomas with Holocord Syrinx in von Hippel-Lindau Disease: A Case Report. J Neurol Surg Rep. 2019 Oct;80(4):e46-e50. doi: 10.1055/s-0039-3401808.

НЕУРОХИРУРГИЈА
The Serbian Journal of Neurosurgery

CALL FOR PAPERS

Dear collegues and friends,

As the Official Journal of the Serbian Neurosurgical Society, НЕУРОХИРУРГИЈА - The Serbian Journal of Neurosurgery is aimed at attracting neurosurgical and papers from related disciplines, clinical and experimental research, as well as reviews, reports, letters, and commentaries.

We announce the call for papers for the second issue, and we look forward to your contribution to the further development of our Journal!

The journal encourages Format-free submission - include everything necessary for review and use a consistent citation format, and you can submit your paper without needing to worry about formatting your manuscript to meet that journal's requirements.

On the other hand, a Journal Template is also available at the following link:

https://neurohirurgija.com/neurohirurgija/JournalSNSS-Template.docx

For any further information, please send the inquiry to office@neurohirurgija.com

With best regards,

Lukas Rasulić, Editor-in-Chief

SJNS Editorial Office and Media Education Centre

JOINT VENTURE WITH:

EANS - European Association of Neurological Societies

WANS - World Academy of Neurological Surgery

CAANS - Continental Association of African Neurosurgical Societies

SBNS - The Society of British Neurological Surgeons

SiNch - Italian Society of Neurosurgery

IANA - International Academy of Neurosurgical Anatomy

SeENS - Southeast Europe Neurosurgical Society

SERBIAN NEUROSURGICAL SOCIETY AND FRIENDS

FIRST ANNOUNCEMENT

October 30th – November 3rd, 2023

9th SNSS Annual Meeting 10th SNSS Congress

with international participation

UDRUŽENJE NEUROHIRURGA SRBIJE - UNHS

SERBIAN NEUROSURGICAL SOCIETY - SNSS

NEUROSURGERY IN SERBIA

EANS Individual Members 10% Discount

https://100yearsneurosurgerysrb.talkb2b.net/

October 31st, 1923 - October 31st, 2023

100 YEARS

FOCUS. DEDICATION. SPECIALIZATION.

CENTENNIAL ANNIVERSARY

OFFICIAL CEREMONY

October 31st, 2023, Belgrade, Serbia

31.10.1938-31.10.2023, 85th ANNIVERSARY CLINIC FOR NEUROSURGERY, UNIVERSITY CLINICAL CENTER OF SERBIA, BELGRADE

1956-2023, 67th anniversary Clinic for neurosurgery, Military Medical Academy, Belgrade
1962-2023, 61st anniversary Clinic for Neurosurgery, Clinical Center of Vojvodina, Novi Sad
1967-2023, 56th anniversary Clinic for Neurosurgery, University Clinical Center Nis, Nis
1977-2023, 46th anniversary Department of neurosurgery, Clinical Hospital Center Zemun, Belgrade
1984-2023, 39th anniversary Center for neurosurgery, Clinical Centre Kragujevac