SNI
SURGICAL NEUROLOGY INTERNATIONAL
SNI: Neurovascular
OPEN ACCESS
For entire Editorial Board visit : http://www.surgicalneurologyint.com
Editor: Kazuhiro Hongo, M.D., Shinsui University, Matsomoto, Japan

Case Report

Successful treatment of a case of tentorial dural arteriovenous fistula causing subarachnoid hemorrhage with invagination of the brainstem by huge and multiple venous pouches

Natsumi Fujii, Makoto Ideguchi, Takafumi Nishizaki, Norio Ikeda, Taichi Shimabukuro, Tomomi Okamura, Machiko Oono

Department of Neurosurgery, Ube-kohsan Central Hospital, 750 Nishikiwa, Ube, Yamaguchi, Japan

E-mail: Natsumi Fujii - fujii.ynt@gmail.com; *Makoto Ideguchi - mideguchi-nsu@umin.ac.jp; Takafumi Nishizaki - nishiza@jeans.ocn.ne.jp; Norio Ikeda - ikedanor@orange.ocn.ne.jp; Taichi Shimabukuro - white-owl.1000@outlook.jp; Tomomi Okamura - okam123@lime.ocn.ne.jp; Machiko Ohno - machiko5estow@hotmail.com
*Corresponding author

Received: 11 September 18 Accepted: 13 November 18 Published: 15 January 19

Abstract

Background: We present a case of tentorial dural arteriovenous fistula (TDAVF) causing subarachnoid hemorrhage with mass effect of large venous pouches, which was struggling to diagnosis and management due to complex vasculature and severe general condition.

Case Description: A 43-year-old man was transferred to our hospital due to sudden consciousness disturbance. A neurological examination revealed tetraparesis and pupil dilatation with no light reflex. Imaging findings showed a large lesion in the brainstem with subarachnoid and intraventricular hemorrhage. Since there were multiple feeding arteries and large and multiple venous pouches on vascular imaging, we diagnosed the patient with TDAVF. Because of a high-flow arteriovenous shunt and the presence of large venous pouches, it appeared to be very difficult to approach the shunting point by direct surgery. Therefore, we first performed transarterial endovascular treatment with 25% *n*-butyl-2-cyanoacrylate to shrink the venous pouches and to reduce the pressure of the posterior fossa, followed by direct radical interruption of the shunting point using the craniotomy maneuver. Postoperative vascular imaging revealed disappearance of abnormal feeding arteries, draining veins, and venous pouches. The patient was discharged and transferred to a rehabilitation hospital with a modified Rankin Scale Score of 3. Accurate interpretation of the detailed vasculature preoperatively and an appropriate treatment strategy using endovascular and direct surgical technique are required to achieve a satisfactory outcome for difficult-to-treat dural arteriovenous fistulas.

Access this article online
Website: www.surgicalneurologyint.com
DOI: 10.4103/sni.sni_317_18
Quick Response Code:

How to cite this article: Fujii N, Ideguchi M, Nishizaki T, Ikeda N, Shimabukuro T, Okamura T, *et al*. Successful treatment of a case of tentorial dural arteriovenous fistula causing subarachnoid hemorrhage with invagination of the brainstem by huge and multiple venous pouches. Surg Neurol Int 2019;10:2.
http://surgicalneurologyint.com/Successful-treatment-of-a-case-of-tentorial-dural-arteriovenous-fistula-causing-subarachnoid-hemorrhage-with-invagination-of-the-brainstem-by-huge-and-multiple-venous-pouches/

Conclusions: This combined maneuver with endovascular embolism as complementary pretreatment for radical surgery is useful for a case with high-flow shunting and large venous pouches.

Key Words: Combined treatment with endovascular and direct surgery, multiple venous pouches, subarachnoid hemorrhage, tentorial dural arteriovenous fistula

INTRODUCTION

Dural arteriovenous fistula (DAVF) accounts for 10–15% of all intracranial vascular malformations.[8,14,16] A tentorial dural arteriovenous fistula (TDAVF) is rare and constitutes less than 2.9–8% of DAVFs.[1,8] Because the risk of cerebral hemorrhage is 60–74%, DAVF must be treated appropriately.[1,3,12,15] Here, we report a case of ruptured TDAVF with multiple and large venous pouches invaginating the brainstem that was successfully treated with a combination of endovascular embolization and surgical intervention. We describe herein a rare and hard-to-treat case of TDAVF with large and multiple venous pouches which was satisfactory treated by direct surgery with complimentary endovascular maneuver.

CASE HISTORY

A 43-year-old man was transferred to our hospital due to sudden loss of consciousness. Neurological examination revealed tetraparesis and pupil dilatation with no light reflex. The Glasgow Coma Scale Score was 4 (E1, V1, M2). His condition immediately deteriorated to ataxic respiration, and he then underwent intratracheal intubation.

Computed tomography (CT) revealed a very large high-density mass lesion on the pons, midbrain, and cerebellopontine (CP) angle with subarachnoid and intraventricular hemorrhage and acute hydrocephalus [Figure 1a–c]. Three-dimensional CT angiography (3D-CTA) revealed large and multiple venous pouches invaginating the brainstem [Figure 1d]. A cerebral angiogram revealed a DAVF fed by multiple feeding arteries draining into the right petrosal vein and reversed to the basal vein of Rosenthal. A right external carotid angiogram showed multiple feeders, including the middle meningeal artery (MMA), accessory meningeal artery (AMA), maxillary artery (Max. A), and ascending pharyngeal artery [Figure 2a]. A right vertebral angiogram also showed multiple feeders, including the right superior cerebellar artery (SCA), right anterior inferior cerebellar artery (AICA), and right posterior inferior cerebellar artery [Figure 2b]. A bilateral internal carotid artery angiogram revealed tentorial arteries from the meningohypophyseal trunk [Figure 2c and d], and large and multiple venous pouches invaginating the midbrain, pons, and right CP angle. The simplified schema was showed in Figure 2e in order to comprehend the relationship among feeding arteries, draining veins, and the shunting point on dura matter of cerebellar tent. Because there was no blood flow to any sinuses, including the superior petrosal sinus, we finally diagnosed a TDAVF, Borden type III and[2] Cognard type IV.[3] The shunting point was located at the right cerebellar tent near the petrosal bone [Figure 1e].

The patient underwent immediate lateral ventricular drainage for severe hydrocephalus on the day of admission. Because of a complication related to neurogenic cardiomyopathy, he was in a serious condition. We first attempted endovascular treatment under local anesthesia 2 days after admission. To shrink the venous pouches, transarterial embolization (TAE) of feeders was performed via the right MMA, AMA, and Max. A using 25% *n*-butyl-2-cyanoacrylate without the penetration of embolus to shunting point. The venous pouches of the midbrain became smaller after embolization, but the size of the pouches in the operative route of the CP angle did not change [Figure 2f]. However, with shrinkage of the venous pouches of the midbrain, his level of consciousness improved to Glasgow Coma Scale 7 (E3, V1, M3) and his general condition was getting more stable due to a decrease in the hard compression toward brainstem. Because the cardiopulmonary function of the patient recovered enough to endure general anesthesia, open surgery was performed 10 days after embolization.

Because the large venous pouch at the CP angle did not allow for an approach at the shunting point [Figure 3a], and an intraoperative rupture could have been fatal, the feeding arteries in the cerebellar tent, for which we could not perform the TAE used for other meningeal arteries, were coagulated to shrink the size and pressure of venous pouches to the maximal extent before reaching the shunting point. This maneuver was able to reduce the pressure of the venous pouch and then resulted to visible the shunting point, which was just rostral to a portion of the trigeminal nerve [Figure 3b]. An interruption procedure was performed, and successful obliteration of the shunting point was achieved [Figure 3c]. Postoperative 3D-CTA and cerebral angiograms showed no abnormal vasculature [Figure 4].

After surgery, the patient underwent tracheotomy and ventriculoperitoneal shunt for hydrocephalus. His consciousness began to recover and he was withdrawn

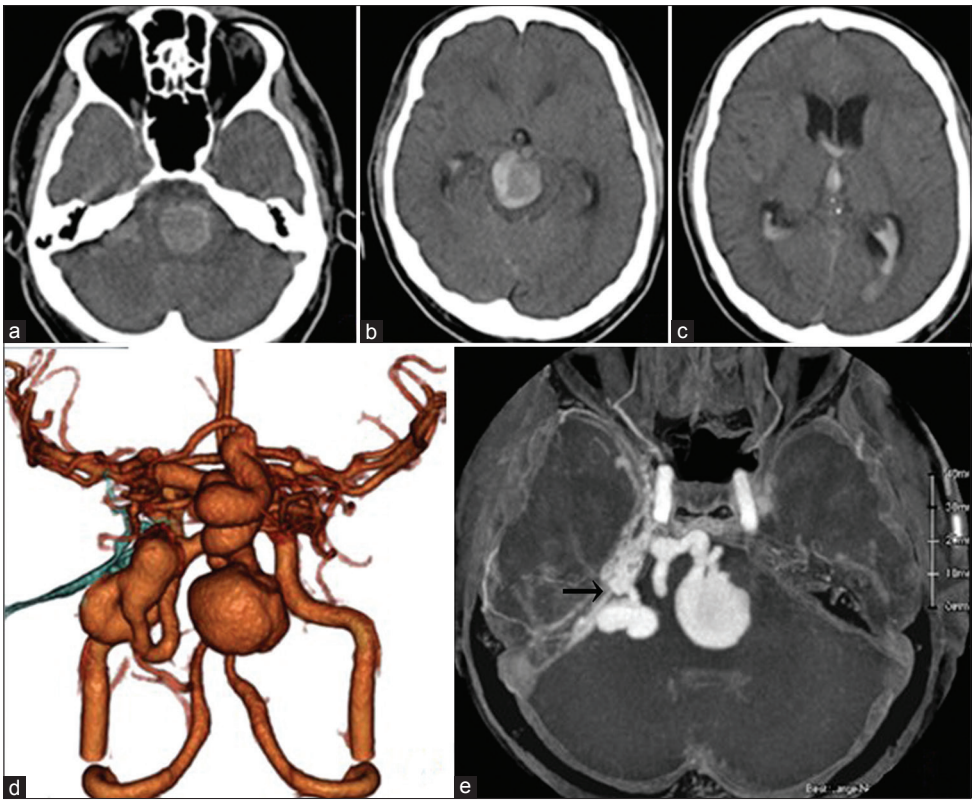

Figure 1: (a–c) CT showed a hematoma at the pons and midbrain with intraventricular hemorrhage and acute hydrocephalus. (d) 3D-CTA showed a large and multiple venous ectasia. (e) The shunting point was at the right cerebellar tent near the petrosal bone (black arrow)

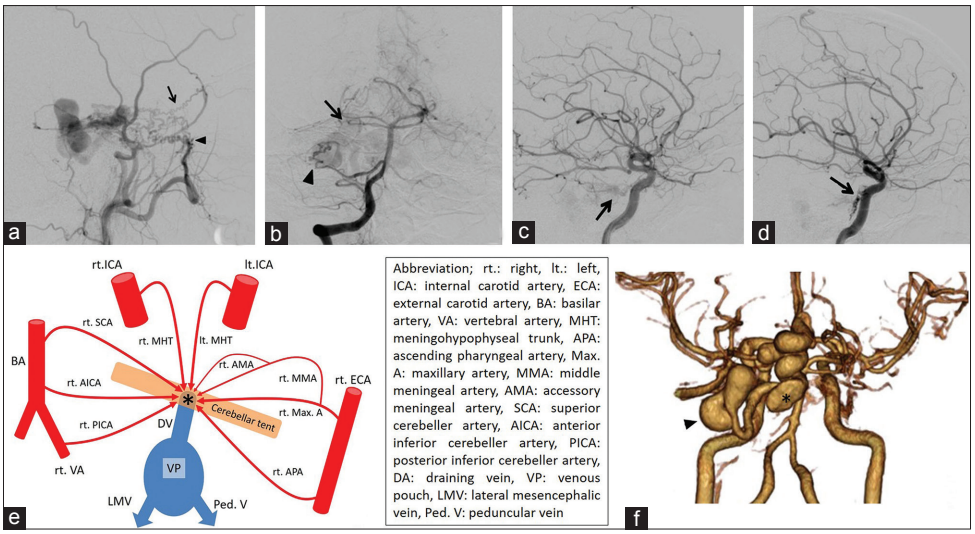

Figure 2: (a) Lateral view on a right external carotid angiogram. The MMA (arrow) and Max. A (arrowhead) are feeding the TDAVF. (b) Anteroposterior view on a right vertebral angiogram. The SCA (arrow) and AICA (arrowhead) are feeding the TDAVF. Lateral view on carotid angiogram (c: right, d: left) showing dilated tentorial arteries (arrows). (e) Simplified scheme relationship among feeing arteries (red), draining veins (blue) and shunting point (asterisk). (f) 3D-CTA after endovascular treatment. The venous pouches of the midbrain became smaller (asterisk). The venous pouch of the CP angle did not change (arrowhead)

from the mechanical ventilator. Despite severe neurological deficits including bilateral oculomotor palsy, he had only mild weakness of the upper and lower extremities, and mild cognitive dysfunction. He could walk with limited assistance 6 months after treatment and was grade 3 on the modified Rankin Scale at discharge.

DISCUSSION

TDAVF is a relatively rare but potentially life-threatening condition, with a high risk of hemorrhage. The optimal therapeutic strategy is still debatable. Based on the hemodynamic status of each

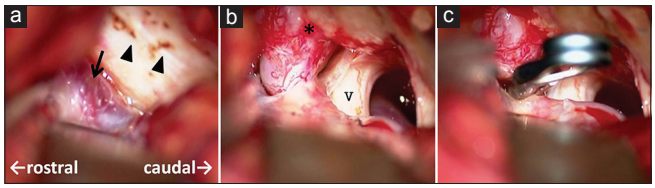

Figure 3: Intraoperative photographs show a venous pouch in the operative route (arrow) and coagulation of the cerebellar tent (arrowheads). (b) The shunting point (asterisk) was in the rostral portion of the trigeminal nerve (v). (c) After clipping of the draining vein

case, endovascular treatment, surgical disconnection, and stereotactic radiosurgery, or a combination of these procedures, can be used for treatment.[4,10,13] Because the shunting point of the TDAVF in our case did not have blood flow via any sinuses, we could not treat it with transvenous embolization. The TAE was also possible for TDAVF, the initial angiographical occlusion rate of which was 77% (before Onyx) and 91% (Onyx) as described previously.[5] On the other hand, some of the feeding arteries could not be embolized because they fed nerves or were related to an extra-intracranial anastomosis.[7,15] Thus, complete remission with TAE alone might be hard to achieve. Since treatment with stereotactic radiosurgery usually requires about 2–3 years,[6] it was not indicated due to massive hemorrhage onset. Therefore, direct surgical interruption of the draining vein was required in this case.

The retrosigmoid approach was an individualized surgical strategy because the draining vein was the right petrosal vein in our case.[11] However, large and multiple venous pouches with mass effect to brain stem are an exceedingly rare presentation of a TDAVF, and a large venous pouch at the CP angle increases the difficulty in reaching the vein.[9]

CONCLUSION

We encountered a severe case of TDAVF with massive hemorrhage, high-flow shunting, and large venous pouches with severe neurological deterioration, which was considered to be difficult to treat without causing serious complications. However, an accurate preoperative diagnosis and appropriate direct surgery with pretreatment of endovascular embolism achieved a satisfactory result. In such a case, proactive treatment is recommended to allow for the best possible outcome, even if the patient is in a moribund condition with hemorrhage.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

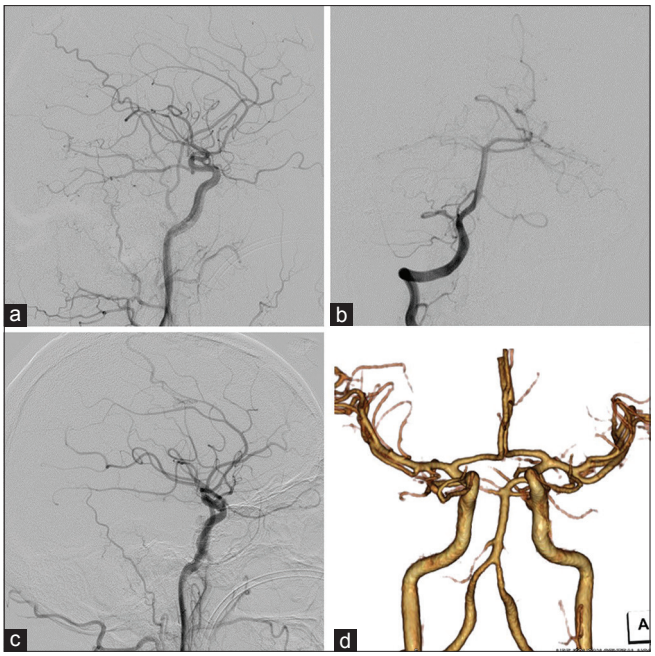

Figure 4: Lateral view on a right common carotid angiogram (a), anteroposterior view on a right vertebral angiogram (b), and lateral view on a left common carotid angiogram (c) showing no vascular abnormalities. (d) Postoperative 3D-CTA was also showing no vascular abnormalities

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Awad IA, Little JR, Akarawi WP, Ahl J. Intracranial dural arteriovenous malformations: Factors predisposing to an aggressive neurological course. J Neurosurg 1990;72:839-50.
2. Borden JA, Wu JK, Shucart WA. A proposed classification for spinal and cranial dural arteriovenous fistulous malformations and implications for treatment. J Neurosurg 1995;82:166-79.
3. Cognard C, Gobin YP, Pierot L, Bailly AL, Houdart E, Casasco A, *et al.* Cerebral dural arteriovenous fistulas: Clinical and angiographic correlation with a revised classification of venous drainage. Radiology 1995;194:671-80.
4. Giller CA, Barnett DW, Thacker IC, Hise JH, Berger BD. Multidisciplinary treatment of a large cerebral dural arteriovenous fistula using embolization, surgery, and radiosurgery. Proc (Bayl Univ Med Cent) 2008;21:255-7.
5. Gross BA, Albuquerque FC, Moon K, McDougall CG. Evolution of treatment and a detailed analysis of occlusion, recurrence, and clinical outcomes in an endovascular library of 260 dural arteriovenous fistulas. J Neurosurg 2017;126:1884-93.
6. Guo WY, Pan DH, Wu HM, Chung WY, Shiau CY, Wang LW, *et al.* Radiosurgery as a treatment alternative for dural arteriovenous fistulas of the cavernous sinus. AJNR Am J Neuroradiol 1998;19:1081-7.
7. Halbach VV, Higashida RT, Hieshima GB, Wilson CB, Hardin CW, Kwan E.

Treatment of dural fistulas involving the deep cerebral venous system. AJNR Am J Neuroradiol 1989;10:393-9.
8. Hiramatsu M, Sugiu K, Hishikawa T, Haruma J, Tokunaga K, Date I, *et al*. Epidemiology of dural arteriovenous fistula in Japan: Analysis of Japanese registry of neuroendovascular therapy (JR-NET2). Neurol Med Chir (Tokyo) 2014;54:63-71.
9. Iwamuro Y, Nakahara I, Higashi T, Iwaasa M, Watanabe Y, Hirata E, *et al*. Tentorial dural arteriovenous fistula presenting symptoms due to mass effect on the dilated draining vein: Case report. Surg Neurol 2006;65:511-5.
10. Kopitnik TA, Samson DS. Management of dural arteriovenous malformations. Contemp Neurosurg 1995;17:1-8.
11. Lawton MT, Sanchez-Mejia RO, Pham D, Tan J, Halbach VV. Tentorial dural arteriovenous fistulae: Operative strategies and microsurgical results for six types. Neurosurgery 2008;62 (3 Suppl 1):110-24; discussion 124-5.
12. Lewis AI, Tomsick TA, Tew JM, Jr. Management of tentorial dural arteriovenous malformations: Transarterial embolization combined with stereotactic radiation or surgery. J Neurosurg 1994;81:851-9.
13. Natarajan SK, Ghodke B, Kim LJ, Hallam DK, Britz GW, Sekhar LN. Multimodality treatment of intracranial dural arteriovenous fistulas in the Onyx era: A single center experience. World Neurosurg 2010; 73:365-79.
14. Newton TH, Cronqvist S. Involvement of dural arteries in intracranial arteriovenous malformations. Radiology 1969;93:1071-8.
15. Picard L, Bracard S, Islak C, Roy D, Moreno A, Marchal JC, *et al*. Dural fistulae of the tentorium cerebelli. Radioanatomical, clinical and therapeutic considerations. J Neuroradiol 1990;17:161-81.
16. Soderman M, Pavic L, Edner G, Holmin S, Andersson T. Natural history of dural arteriovenous shunts. Stroke 2008;39:1735-9.