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CORRESPONDENCE

Dural Arteriovenous Fistula Presenting as Tentorial Subdural Hemorrhage

Case Report and Review of the Literature

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Summary

Subdural hemorrhage is frequently caused by trauma and is commonly seen in older people; however, subdural hemorrhage has been reported after ruptured intracranial aneurysms and in relation to dural arteriovenous fistulas as well as arteriovenous malformations. This article presents a case of pure subdural hemorrhage of the tentorium cerebelli secondary to a posterior fossa dural arteriovenous fistula, later treated with endovascular embolization and subsequently radiotherapy.

Introduction

Intracranial dural arteriovenous fistulae (DAVF) are a frequently encountered pathology believed to be related to venous thrombosis [1]; however, the exact pathophysiological mechanisms are still unknown and likely to be multifactorial [2]. The two classification systems currently in use focus on the venous drainage pathways and particularly on whether there is flow into the cortical veins [3–5]. Cortical venous reflux and anterograde flow from the DAVF into cortical veins are the main reasons for aggressive clinical presentation defined as either intracranial hemorrhage or non-hemorrhagic neurological deficits [6]. Approximately 20% of intracranial DAVF present with intracranial hemorrhage which can be intraparenchymal, subarachnoid, subdural or a combination [6, 7]; however, presentation of patients with pure subdural hemorrhage is rare [8–12]. This article presents a case of DAVF presenting with pure subdural hemorrhage of the tentorium cerebelli.

Case Report

A 75-year-old male patient, with no significant past medical history, presented to the emergency department with a sudden onset severe headache. He reported that the headache began while he was performing push-ups and that shortly after the onset of the headache he developed dizziness and bilateral central scotoma as well as bilateral lower limb weakness. While still in the emergency department the lower limb weakness and visual deficit resolved. On examination he had full motor power, no altered sensation and the Glasgow coma scale (GCS) was 15. He was afebrile and the electrocardiogram (ECG) showed sinus tachycardia. An acute subarachnoid hemorrhage was suspected and therefore, an emergent computed tomography (CT) scan of the head with CT angiogram was performed. This demonstrated subdural hemorrhage of the tentorium cerebelli and multiple abnormal vessels that were suggestive of a DAVF (Fig. 1). There was no evidence of subarachnoid or intraparenchymal hemorrhage.

The following day the patient underwent diagnostic catheter angiography that demonstrated two DAVFs, a posterior fossa DAVF near the midline that was believed to be the cause of the subdural tentorial hemorrhage and an anterior cranial fossa DAVF that was clinically asymptomatic. The arterial supply to the posterior fossa lesion was principally via the middle meningeal artery (bilaterally), the left meningohypophyseal trunk (MHT) and the artery of the falx cerebelli from the posterior inferior cerebellar artery

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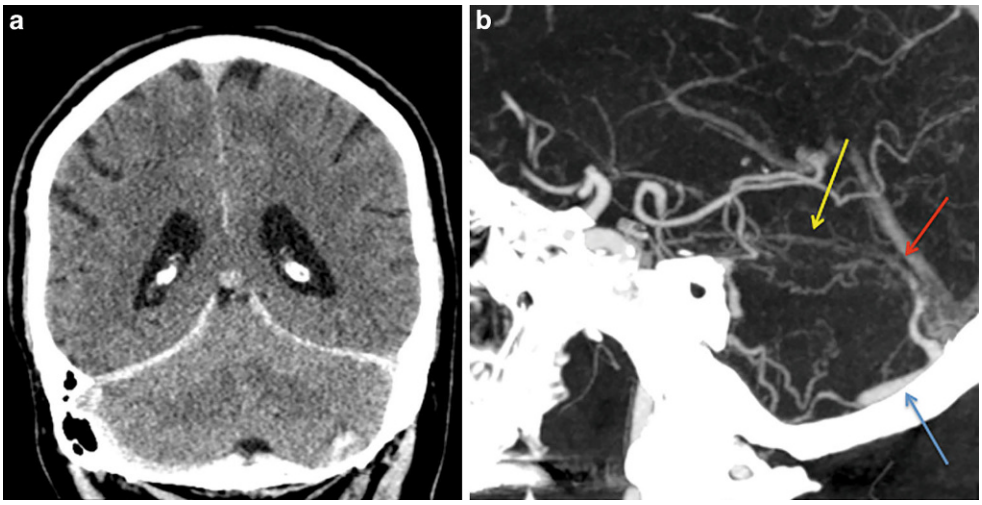

Fig. 1 Thick subdural hemorrhage of the tentorium cerebelli (**a** coronal unenhanced CT scan). There was no intraparenchymal hemorrhage or subarachnoid hemorrhage. On the multiplanar reformats (MPR) of the CT angiogram (**b** CT angiogram with sagittal reconstruction) a prominent ectatic and dilated inferior cerebellar vein was noted (*blue arrow*) with a dilated lateral tentorial branch from the meningohypophyseal trunk (*yellow arrow*). It was also believed that the likely fistulous point could also be seen (*red arrow*).

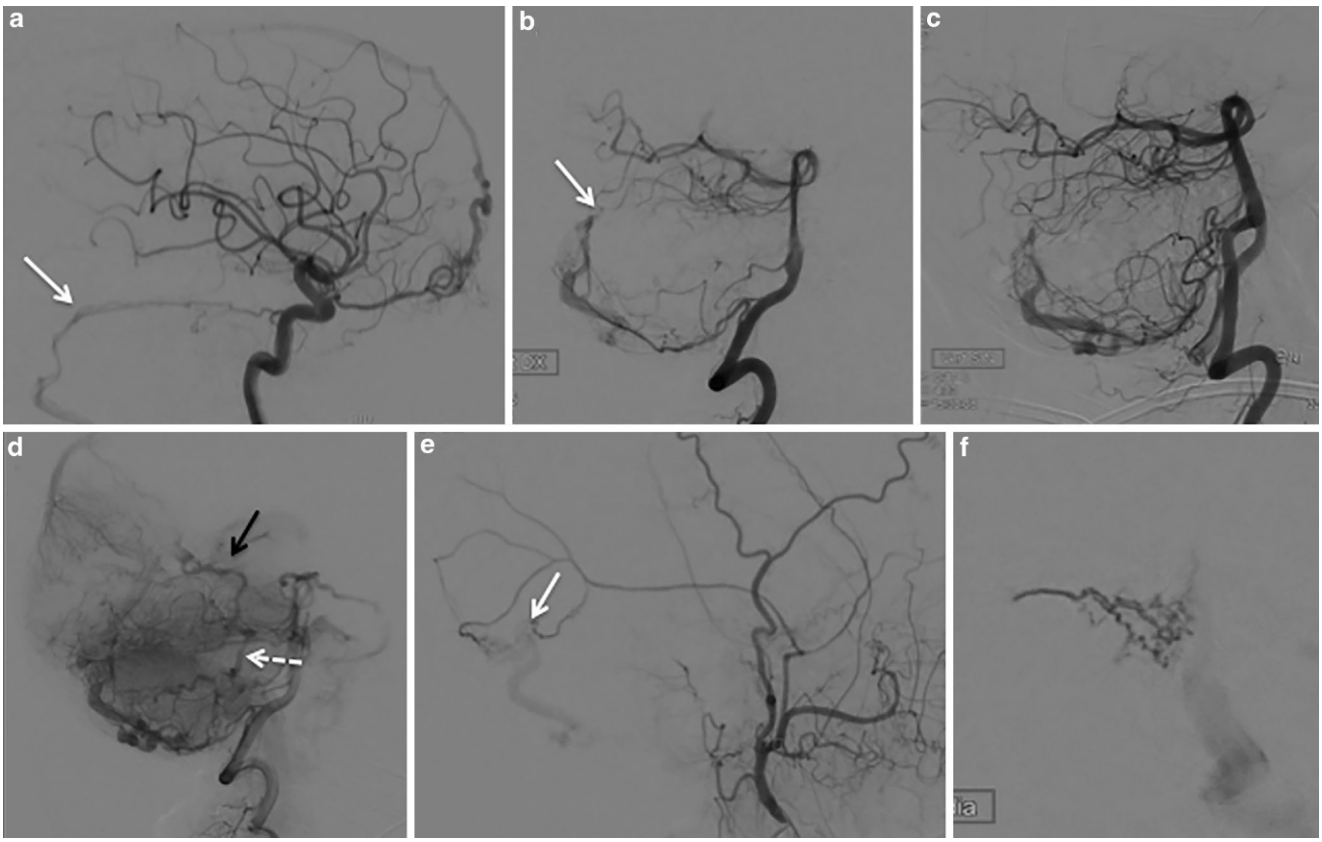

Fig. 2 Angiography demonstrated two DAVFs, one in the posterior fossa thought to be the cause of the tentorial subdural hemorrhage and another lesion in the anterior cranial fossa (**a**). Supply to the posterior fossa lesion was derived from the lateral tentorial branch of the MHT *(white arrow)* (**a**), a dural branch of the PICA (*white arrow*) (**b**, **c**), the middle meningeal artery and two branches derived from the distal petrosquamosal branch (*white arrow*) (**e**). The venous drainage was retrograde into a dilated inferior cerebellar vein, the vein of the great horizontal fissure and then into the anastomotic lateral mesencephalic vein (**d**, *white dashed arrow*) and distal basal vein of Rosenthal (**d**, *black arrow*). The fistula could be more clearly seen on super selective catheter angiography in the distal middle meningeal artery (**f**). *MHT* meningohypophyseal trunk

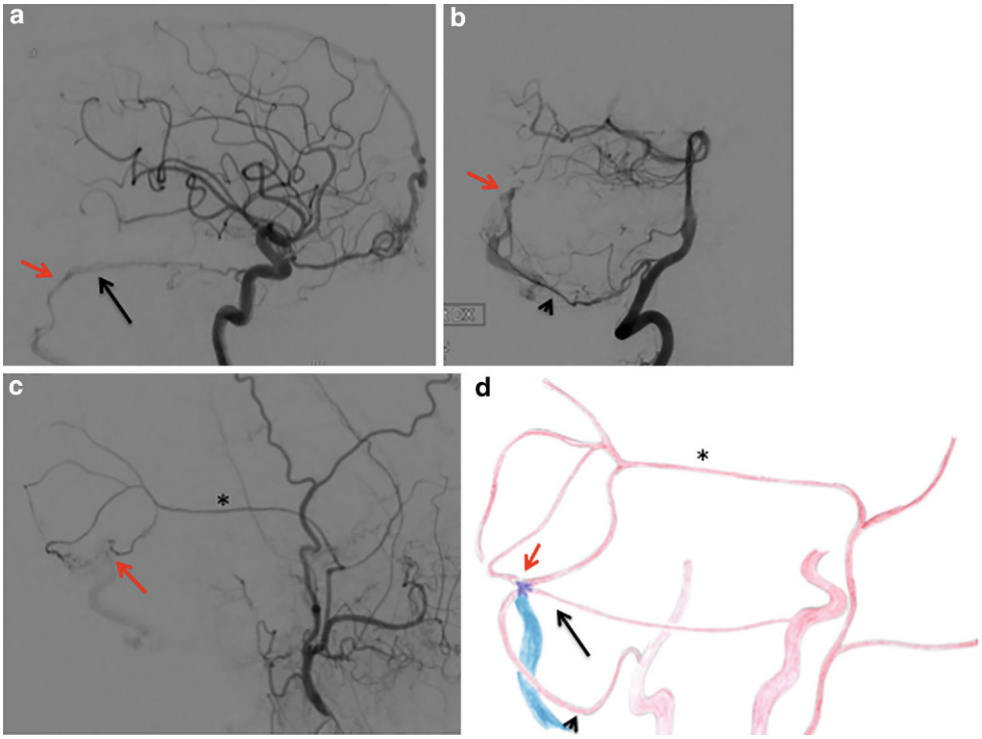

Fig. 3 The arterial supply from the lateral tentorial branch of the MHT (**a**, *black arrow*), the dural branch of the PICA (**b**, *black arrow head*), and the petrosquamosal branch (**c**, *black asterisk*) can also be seen to converge onto the same fistulous point (*red arrow*). A schematic drawing of the fistula is shown in **d**

(PICA) on the right. The venous drainage was retrograde through an ectatic inferior cerebellar vein and then subsequently via the vein of the great horizontal fissure to the lateral mesencephalic vein and the distal basal vein of Rosenthal (Borden 3 and Cognard 4; Figs. 2 and 3). The DAVF seen in the anterior cranial fossa was a Borden 3, Cognard 3 lesion.

After a multidisciplinary team discussion and obtaining informed consent, endovascular embolization was attempted with a liquid embolic (Onyx 18; Medtronic, Dublin, Ireland). The initial embolization procedure via the petrosquamosal branch of the middle meningeal artery did not penetrate to the fistulous point (Fig. 4a–c) and resulted in loss of access to the fistula via the medial meningeal arteries with continued supply from the right MHT and the PICA. A second embolization via the PICA was attempted 6 days later with further obliteration of the arterial supply using 25% histoacryl glue (Fig. 4d–f). At the conclusion of the procedure there was persistent supply from the MHT but as some of the histoacryl had penetrated into the draining vein it was thought that the fistula would occlude and further embolization was not attempted. There were no complications and the patient was discharged at baseline neurology.

The patient defaulted follow-up appointments for 2 years before attending the clinic with no new symptoms. An angiogram showed that the fistula remained patent. After discussion in the multidisciplinary meeting and with the patient it was decided to treat the lesion with gamma-knife radiosurgery, as access to the fistula via the remaining arterial vessel (MHT) was deemed impossible.

Discussion

Intracranial DAVFs are relatively common lesions and their propensity to bleed is related to the venous drainage pattern. Those lesions with drainage into cortical veins, Cognard 3 and 4, most often present with hemorrhage and carry a substantial risk for hemorrhage and repeat hemorrhage [13–15]. In most patients, an intraparenchymal hemorrhage (ICH) or subarachnoid hemorrhage (SAH) occurs, with pure subdural hemorrhage (SDH) being extremely rare. Since 1990 only 8 cases of DAVF with pure SDH have been published with a further 12 cases of SDH ± ICH or SAH. Of the 20 cases the Cognard classification was unknown in 3, 1 was type IIa, 6 were type III and 10 were type IV (Table 1).

The anatomical disposition of these lesions may explain why they can bleed into the subdural space. In Cognard III and IV lesions the dural arterial supply fistulizes directly with the bridging veins. These bridging veins, as their name suggests, have a short dural course; however, they must also traverse the subdural space since they originate in the subarachnoid space where they form after the confluence of multiple leptomeningeal veins. The lack of a dural envelope and arterialized veins may account for the high risk of hemorrhage [27]. The Cognard I and IIa lesions are con-

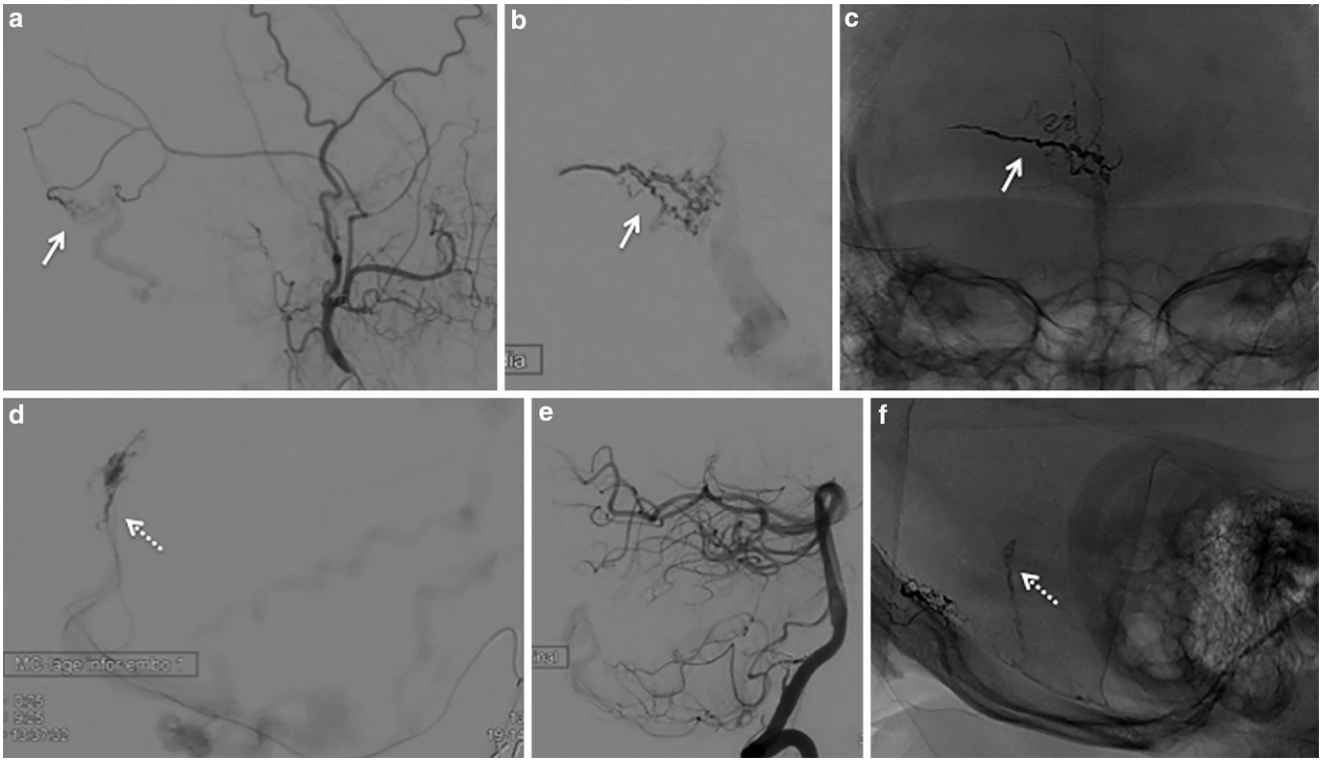

Fig. 4 The initial embolization attempt was performed using Onyx 18 via the middle meningeal artery after getting as close to the fistulous point via this route as possible (**a**, **b**, *white arrow*). The Onyx cast can be clearly seen on an unsubtracted image (**c**). There was persistent supply via the PICA and MHT and therefore a second embolization attempt was performed via the PICA (**d**, *dashed white arrow*). There was a significant reduction in the shunting but persistent shunting could be seen (**e**). The glue cast could be clearly seen on un-subtracted images (**f**, *dashed white arrow*)

fined to the venous sinus and protected by a dural envelope and therefore should not lead to subdural hemorrhage. It is also important to note that cortical venous reflux does not equate to pial venous reflux and pial venous reflux has been associated with an increased rate of hemorrhage compared to DAVFs with pure cortical vein reflux [28, 29]. Alternatively, it has been suggested that the rupture of a leptomeningeal vein may result in laceration of the arachnoid mater and hence cause a subdural hemorrhage [12]. Similar mechanisms have been put forward to explain the rare occasions that intracranial aneurysms can result in subdural hemorrhage rather than subarachnoid hemorrhage [30]. Ultimately it is likely that the rupture point will result in different patterns of hemorrhage.

Tentorial DAVFs are relatively rare and account for approximately 4–7% of all intracranial DAVFs [31–33]. Tentorial DAVFs are high-grade lesions that drain into leptomeningeal veins (Cognard grade III and IV) and thus carry a high risk of hemorrhage with the reported occurrence of hemorrhage ranging between 60% and 74% [3, 31, 34, 35]. In the publication by Lewis et al. 74% (40/54) of lesions presented with hemorrhage [34] and in the series of Awad et al. [31] 97% (31/32) of tentorial DAVFs presented with either hemorrhage or progressive neurological deficits. In some cases, hemorrhage in the posterior fossa from these lesions can be fatal [36, 37]. The clinical course of DAVF with long-term persistent leptomeningeal reflux was investigated by van Dijk et al. [38]. In this small cohort of patients not amenable to treatment or refusing treatment, the annual mortality rate was 10% with an annual risk of hemorrhage or non-hemorrhagic neurological deficits being 8% and 7%, respectively. Of the six patients with tentorial or torcular DAVF death occurred in four including the patient with a torcular DAVF that was partially treated. Moderate disability was seen in one patient and a good recovery was seen in one patient.

Picard et al. [35] divided the venous tributaries in the tentorium into three regions. The lateral tentorial sinus group is adjacent to the lateral sinus and receives supratentorial drainage from the lateral and inferior surfaces of the temporal and occipital lobes. The medial tentorial sinus group is located adjacent to the torcula and drains the torcula, the lateral or the straight sinus. This group primarily receives infratentorial venous drainage from the cerebellar hemispheres and vermis. Along the free edge of the tentorium lesions receive venous drainage from the basilar and lateral mesencephalic veins and can have infratentorial or supratentorial drainage and even drain into the spinal veins

Table 1 Summary of the previously reported cases of dural arteriovenous fistula in the literature

Case	Author	Sex	Age (years)	Presenting symptoms	CT findings	Cognard classification	Treatment	Outcome
1	Duffau et al. [8]	M	55	Headache + monoparesis	SDH	III	Endovascular + surgical	Improvement
2	Duffau et al. [8]	M	64	Headache + hemiparesis	SDH + temporal ICH	III	Endovascular + surgical	Died
3	Duffau et al. [8]	M	64	Headache	SDH + frontal ICH	IV	Surgical	Improvement
4	Duffau et al. [8]	F	56	Headache	SDH	III	Surgical	Died
5	Kohyama et al. [9]	M	60	Headache	SDH	IIa	Endovascular + surgical	Improvement
6	Kitazono et al. [10]	M	68	Headache	SDH + occipital ICH	IV	Surgical	Improvement
7	Ogawa et al. [16]	M	27	Headache	SDH	III	Surgical	Improvement
8	Kominato et al. [11]	F	42	Headache	SDH	Unknown	None	Died
9	Saito et al. [12]	M	56	Headache	SDH + occipital ICH	IV	Endovascular + surgical	Improvement
10	Baskaya et al. [17]	F	51	Headache + altered consciousness	SDH	Unknown	Surgical	Improvement
11	Nakagawa et al. [18]	M	60	Headache + altered consciousness	SDH + frontal ICH	IV	Surgical	Improvement
12	Sugawara et al. [19]	M	67	Altered consciousness + deficits	SDH + frontal ICH	IV	Surgical	Improvement
13	Tanei et al. [20]	M	75	Altered Consciousness	SDH + frontal ICH	Unknown	Surgical	Improvement
14	Kagawa et al. [21]	F	65	Headache + altered consciousness	SDH + SAH	IV	Surgical + Endovascular	Died
15	Dietrich et al. [22]	F	68	Altered consciousness + hemiparesis	SDH + ICH	III	Endovascular	Improvement
16	Kominato et al. [11]	F	42	Headache	SDH	Unknown	None	Died
17	Matsuzaki et al. [23]	M	66	Cognitive dysfunction	SDH + Temporo-occipital ICH	IV	Endovascular	Improvement
18	Kohama et al. [24]	M	58	Headache	SDH + SAH	IV	Surgical	Improvement
19	d'Angelo et al. [25]	F	60	Headache + altered consciousness	SDH	IV	Endovascular	Improvement
20	Suyama et al. [26]	F	61	Headache + altered consciousness	SDH + occipital ICH	III	Surgical + Endovascular	Improvement

SDH subdural hemorrhage, *SAH* subarachnoid hemorrhage, *ICH* intracranial parenchymal hemorrhage

(Cognard V). Lawton et al. differentiated these fistulae into 6 types (1. Galenic DAVF, 2. straight sinus DAVF, 3. torcular DAVF, 4. tentorial sinus DAVF, 5. superior petrosal sinus DAVF and 6. incisural DAVF) and proposed surgical strategies for each type [39].

In a review of 86 cases of tentorial DAVFs from the English language literature 94% (81/86) were drained solely by leptomeningeal veins for the thrombosed dural venous sinus. The arterial supply to these lesions can also be complex with supply coming from both the external carotid artery, particularly the MMA and occipital artery, but also from the ICA via the MHT. Indirect pial supply to tentorial DAVFs has also been noted and can be derived from the PICA via the artery of the falx cerebelli or posterior meningeal artery, the SCA via the medial tentorial artery, and the PCA via the artery of Davidoff and Schechter [40–44]. In addition, direct piodural supply may develop de novo from PCA or SCA branches. In the retrospective review of 31 patients treated surgically Lawton et al. demonstrated a pial supply from the PCA and/or SCA in 26% ($n=8$) of tentorial DAVFs [39]. The role pial arterial supply plays in DAVF is yet to be fully elucidated; however, the presence of pial supply to DAVFs may confer an increased risk of complications from treatment. Sato et al.

[45] recently published a case of acute subdural hemorrhage and temporal lobe hemorrhage after embolization of a tentorial DAVF with pial supply from the PCA. Wu et al. [46] showed a significantly higher rate of complications in patients with pial arterial supply compared to those without pial supply (33% vs. 2%, $p=0.03$). In this study 11% of patients were found to have pial supply and all the DAVFs with pial supply were Cognard III or IV lesions. More recently Hetts et al. [47] showed an increase in the rate of ischemic complications for patients with DAVF and piodural supply compared to those without piodural supply (14% vs. 2%, $p=0.04$). The authors suggested that embolization of a liquid embolic and thrombosis within piodural arteries may be responsible for the increase in thromboembolic complications. Therefore, it appears that piodural supply to lesions may carry an increased risk of both hemorrhagic and thromboembolic complications although this was not shown in the publication of Osada et al. [48].

Tentorial DAVFs can be endovascularly treated via an arterial, venous or combined approach. The arterial approach, via the MMA, is often favored since the drainage of these lesions may be purely via cortical veins as mentioned earlier. Huang et al. [49] reported complete cure of tentorial DAVF in 78.6% (11/14) by transarterial embolization using Onyx with Kortman et al. [50] reporting successful transarterial embolization in 82% (9/11). In selected cases a transvenous approach may be feasible and has been reported previously [35, 51, 52] and a careful examination of the anatomy is required. Venous access to the fistula can be difficult and although liquid embolic agents have been used transvenously [53] they are not ideal given the direction of flow. Improvements in catheter technology have made transvenous navigation and embolization more feasible [54] allowing the fistulous point to be coiled.

Conclusion

This case serves to demonstrate that subdural hemorrhages can occur secondary to rupture of a dural arteriovenous fistula. Although rare, this cause of bleeding should be kept in mind and may in selected cases warrant investigation with catheter angiography.

Compliance with ethical guidelines

Conflict of interest P. Bhogal, L.L. Yeo and M. Söderman declare that they have no competing interests.

Ethical standards Ethics committee approval was not required for this publication. 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 1975 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study

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