# 153 Middle Meningeal Artery Aneurysm: Non-traumatic Incidental Aneurysm of a Middle Meningeal Artery Supplying a Pial Arteriovenous Malformation; Endovascular Occlusion of the Aneurysm Using nBCA During the Preoperative Embolization of the AVM

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**Abstract**

A 48-year-old male patient presented with a single generalized epileptic seizure and a history of migraine with a left superior quadrantanopia. Diagnostic imaging revealed a large brain arteriovenous malformation (AVM) of the right occipital lobe. A treatment concept of preoperative embolization followed by microsurgical excision was offered to and accepted by the patient. During the first endovascular treatment session, an incidental aneurysm of the right middle meningeal artery (MMA) was found. The right MMA was supplying the pial AVM. Both the right MMA and the aneurysm were occluded by the injection of n-butyl cyanoacrylate diluted with Lipiodol. After a total of 16 embolization sessions, the large AVM was sufficiently devascularized to allow for a microsurgical excision of the AVM, 27 months after the first embolization. Both the endovascular and the microsurgical treatment were carried out without any issues and the complete interruption of the AV-shunt was confirmed by angiography. Occurrence and management of non-traumatic MMA aneurysms is the main topic of this chapter.

**Keywords**

Middle meningeal artery · Arteriovenous malformation · dAVF · Preoperative embolization · nBCA

## Patient

A 48-year-old, male patient, otherwise healthy, presenting with a first-ever generalized epileptic seizure and a history of migraine headache and a partial visual field cut to the left.

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## Diagnostic Imaging

MRI showed a large AVM of the right occipital lobe without signs of a previous hemorrhage. DSA allowed for a detailed analysis of the angioarchitecture, with supply of the AVM from the right posterior cerebral artery, the right external carotid artery (ECA), namely the right MMA with the aneurysm, and the right occipital artery, and the right middle cerebral artery (MCA). The left anterior circulation was not directly involved in the supply of the AVM, but the injection of the left internal carotid artery (ICA) confirmed the large arteriovenous shunt volume exerted by the AVM. Venous drainage was primarily via superficial cortical veins and there was no evidence of venous stenosis. The localization in an eloquent brain area, the deep venous drainage, and the maximum diameter of the AVM nidus of >3 cm but <6 cm were equivalent to a Spetzler Martin grade of IV, indicating an increased risk of surgery (Fig. 1).

## Treatment Strategy

The main goal of the treatment was the elimination of the brain AVM in order to avoid an intracranial hemorrhage from said AVM. A secondary indication was the prevention of future epileptic seizures. Epilepsy may develop in patients with brain AVMs over time, and the elimination of a brain AVM reduces not only the risk of an intracranial hemorrhage but also the likelihood of symptomatic epilepsy.

## Treatment

***Procedure, 12.06.2007***: preoperative embolization of a large right occipital brain AVM
*Anesthesia*: general anesthesia
*Premedication*: none
*Access*: right femoral artery, 6F sheath (Cordis); *guide catheter*: 6F Heartrail II (Terumo); *microcatheter*: Echelon-14 straight (then ev3, now Medtronic); *microguidewire*: SilverSpeed14 (then ev3, now Medtronic)
*Embolic agent*: Histoacryl (B. Braun)/Lipiodol (Guerbet), 1:3, 0.4 cc
*Postmedication*: none
*Course of treatment:* the aneurysm of the right MMA had been identified during the diagnostic DSA. The right ECA was selectively catheterized and injected using a 6F guide catheter. Under road map conditions, the right MMA, supplying transduraly the right occipital brain AVM, was catheterized with an Echelon-14 catheter. With the tip of the microcatheter about 2 cm proximal to the aneurysm, said microcatheter was rinsed with a 40% glucose solution (B. Braun). Thereafter, nBCA diluted with Lipiodol was slowly injected with minimal venous passage through the AVM nidus. Polymerizing of nBCA/Lipiodol started distal and progressed from there to proximal, forming a solid cast inside the MMA, occluding the aneurysm and the parent vessel distal and proximal to said aneurysm. The first treatment session was terminated thereafter (Fig. 2).
*Duration*: 1st–14th DSA run: 41 min; fluoroscopy time: 8.8 min
*Complications*: none
*Postmedication*: none

## Clinical Outcome

The patient remained without a new neurological deficit (mRS 1, GOS IV) after the first embolization session. During the following 2 years from June 2007 to September 2009, when a total of 16 embolization sessions had been carried out, the initial quadrantanopia developed to an almost complete hemianopia to the left (Fig. 3). No other neurological deficit occurred and the patient had no more epileptic seizures.

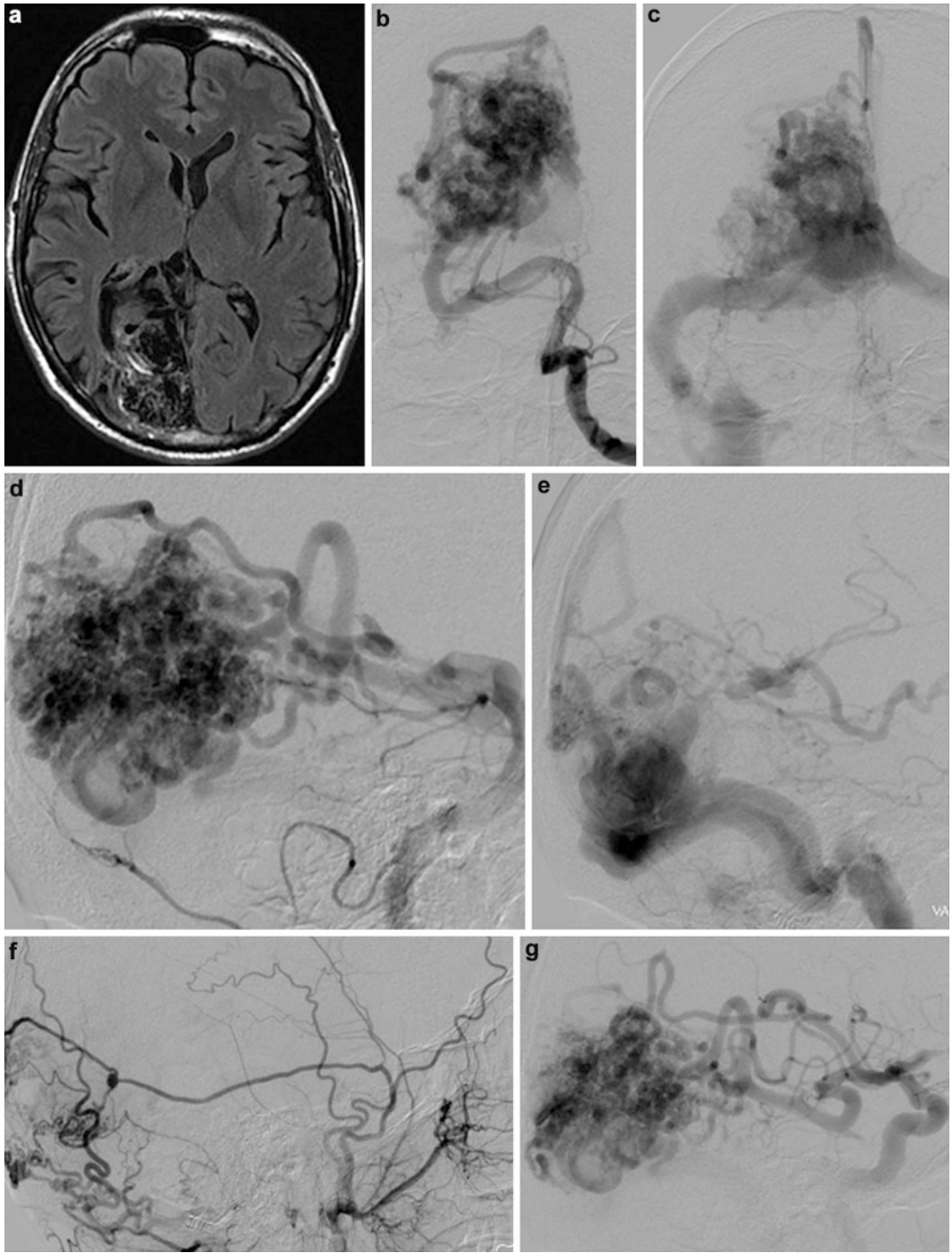


**Fig. 1** (continued)

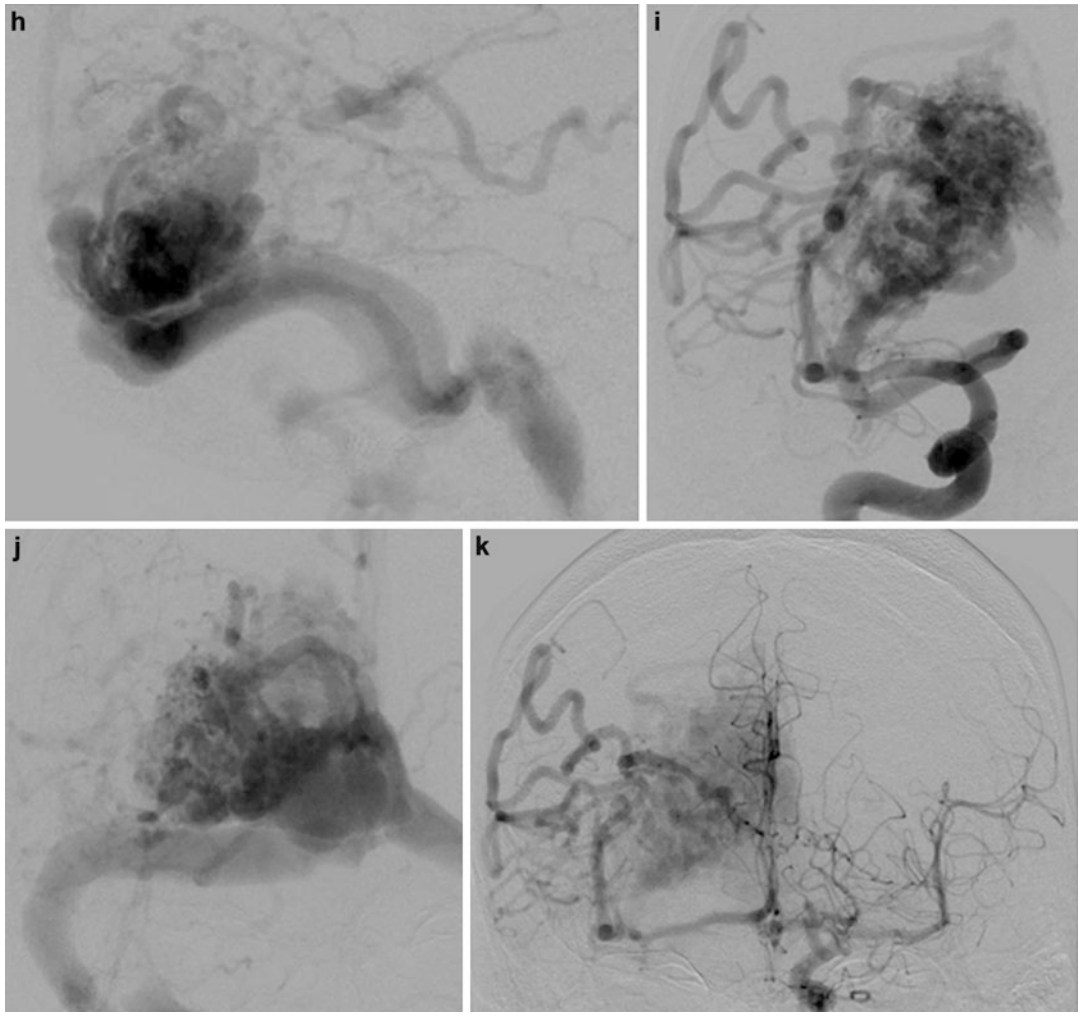


**Fig. 1** Diagnostic imaging carried out in a patient with a generalized epileptic seizure and a history of migraine headache and a partial visual field cut to the left. T2WI MRI (FLAIR) shows a large AVM of the right occipital lobe (**a**). The AVM was mainly supplied by the right posterior (**b–e**) and middle cerebral artery (**g–j**). Transdural supply came from the right middle meningeal artery, which carried an aneurysm (**f**). The injection of the left ICA confirmed the large volume of the arteriovenous shunt (**k**)

## Follow-Up Examinations and Further Treatment

Between June 2007 and September 2009, a total of 16 embolization sessions had been carried out, achieving a significant reduction of vascularity, size, and shunt volume of the pial AVM. Eventually, this preparation allowed for the microsurgical extirpation of the AVM in September 2009. In late December 2009, a disturbance of the wound healing required a revision surgery. Intraoperative DSA examination, as well as DSA 4 months later and 2 years later confirmed the complete removal of the AVM (Figs. 4, 5, and 6).

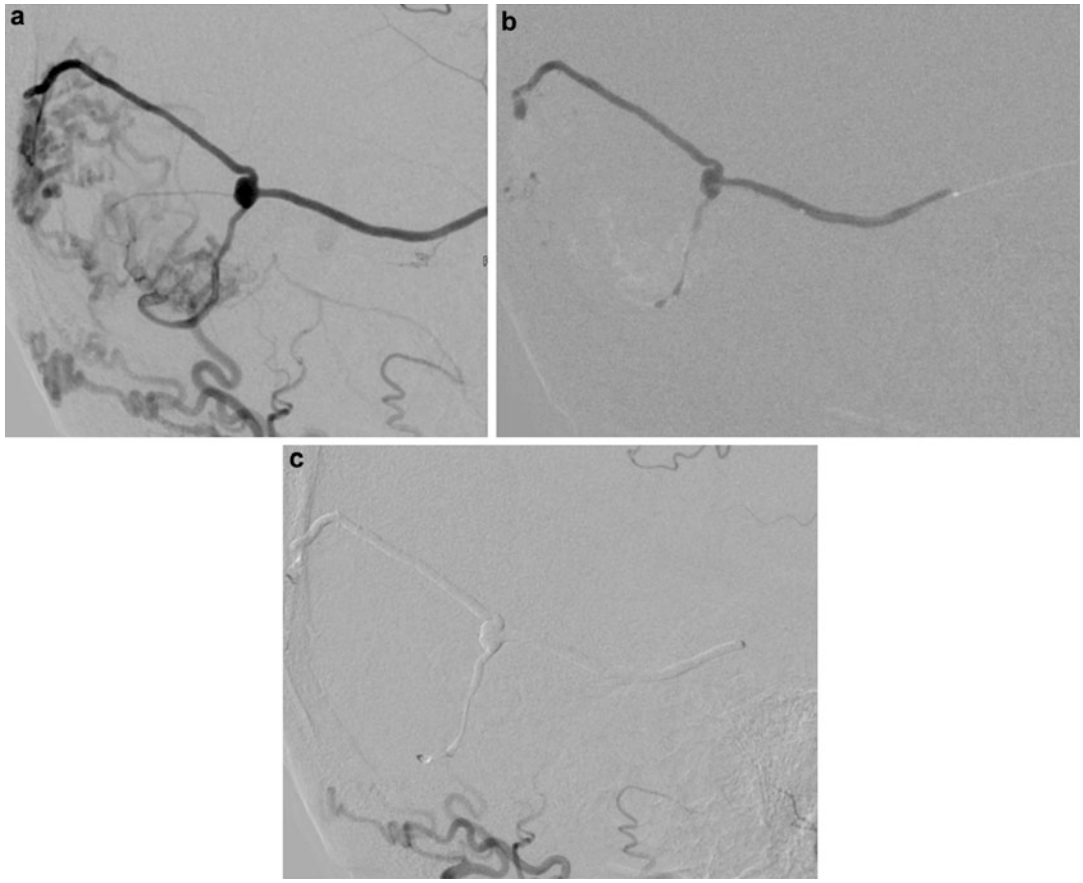


**Fig. 2** DSA with injection of the right ECA in lateral projection (**a**) shows an aneurysm in a bifurcation of the right MMA, which is supplying a brain AVM of the right occipital lobe. Both the aneurysm and the MMA distal and proximal to it were occluded by the injection of Histoacryl and Lipiodol in a 1:3 mixture, creating a continuous cast inside the MMA (**b**). The injection of the right ECA eventually confirmed the occlusion of the aneurysm (**c**)

## Discussion

Non-traumatic aneurysms of dural arteries are rare. In the majority of cases, the MMA is the parent vessel. In their review of the literature, Hedjoudje et al. (2017) found 25 non-traumatic aneurysms of the middle meningeal artery published since 1930. Most aneurysms of the MMA are hemodynamically induced, for instance by the transdural supply of a brain AVM as in the case reported in this chapter. Other concomitant conditions include dural arteriovenous fistula (Kähärä 1999), Paget's disease (Berk 1961), Moyamoya disease (Koebbe and Horowitz 2004), meningioma (Maekawa et al. 2009), neurofibromatosis type 2 (Lesley et al. 2004), ICA occlusion (Zingesser et al. 1965), arterial hypertension (Kpelao et al. 2017), and diffuse calvarial metastases (Hedjoudje et al. 2017). An MMA aneurysm without any associated disorder is the rare exception (Bollati et al. 1980; Kpelao et al. 2017; Sandin et al. 1999). Akyuz and Tuncer (2010) described multiple MMA aneurysms in an adult patient with a Vein of Galen AVM. Due to the nature, location, and mode of treatment of MMA aneurysms, histological examination is

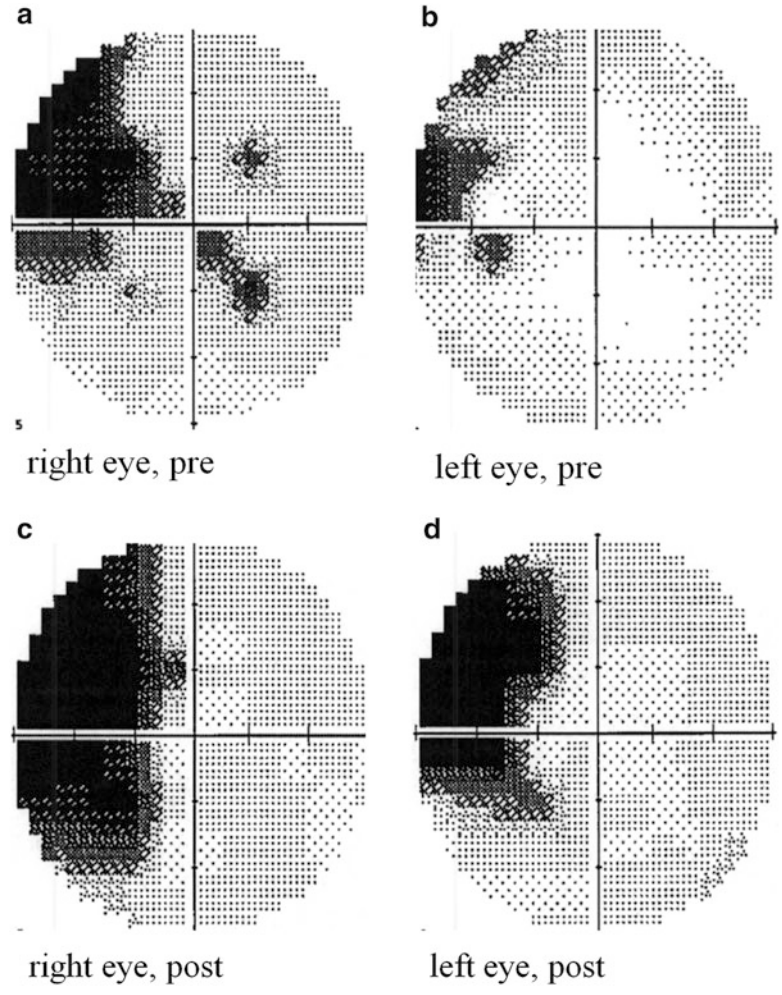


**Fig. 3** Perimetry results from June 12, 2007 (right eye (**a**) and left eye (**b**)) and from June 2, 2009 (right eye (**c**) and left eye (**d**)) before (**a**, **b**) and during (**c**, **d**) the endovascular treatment sessions as a preparation for the intended microsurgical extirpation. As expected from the localization of the AVM, the pre-embolization left superior quadrantanopia (**a**, **b**) progressed to an almost complete hemianopia to the left (**c**, **d**)

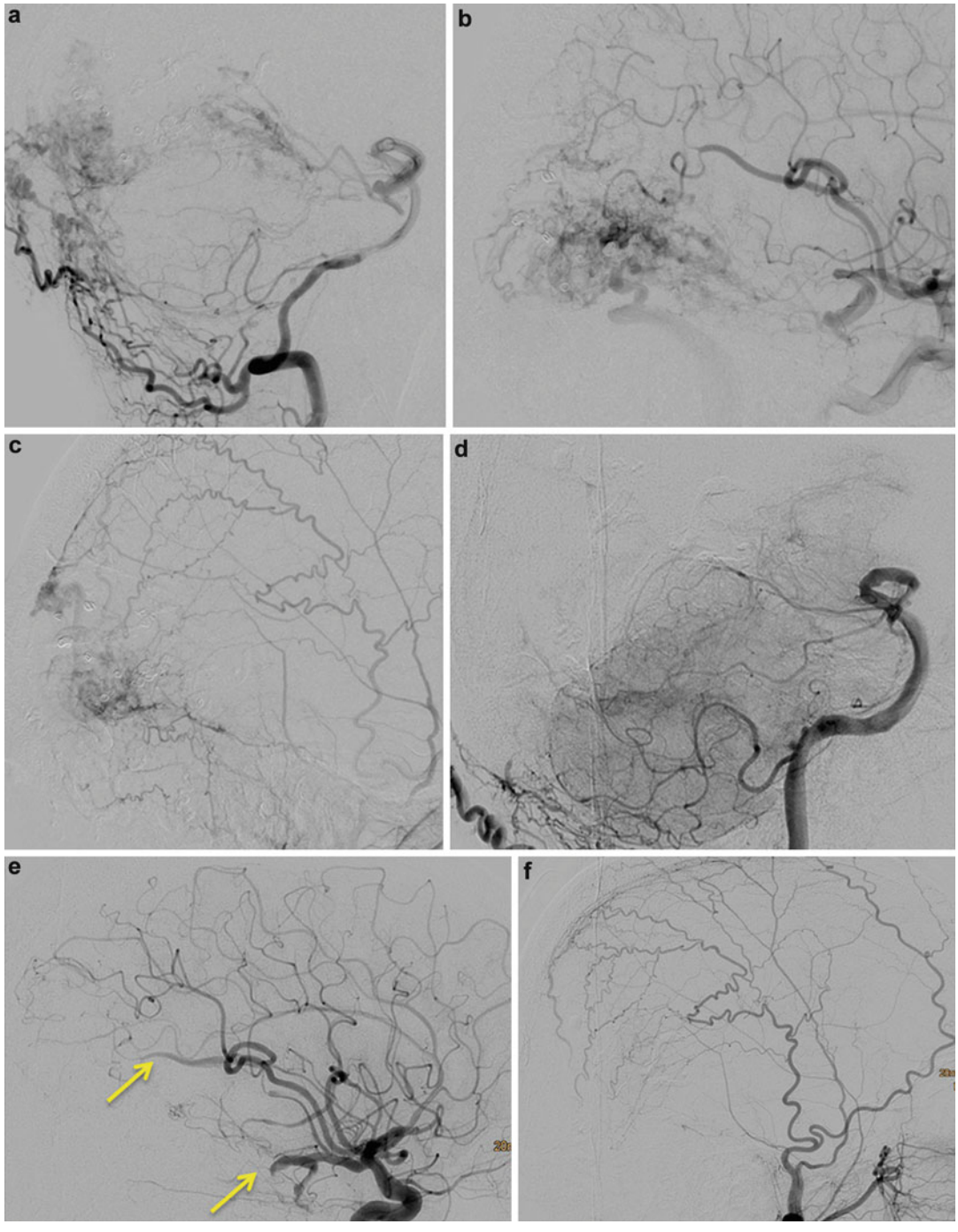


**Fig. 4** (continued)

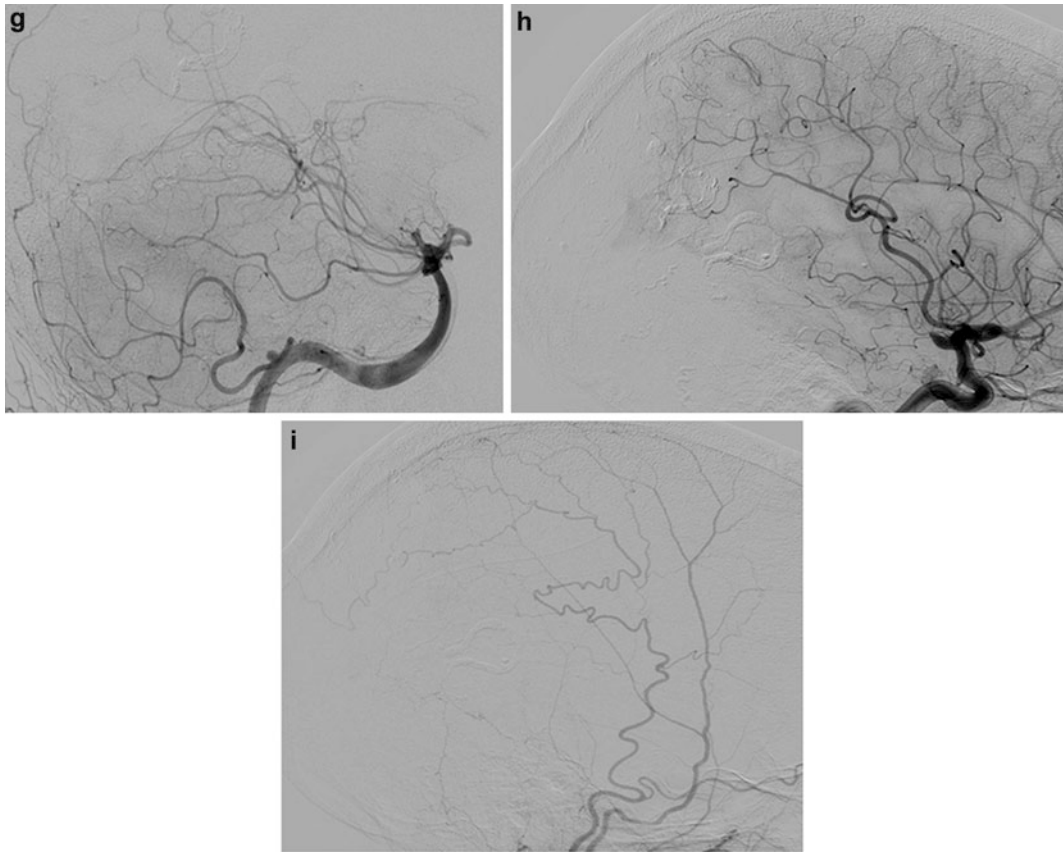


**Fig. 4** DSA after the endovascular treatment of a right occipital lobe brain AVM. The injection of the left VA (**a**), the right ICA (**b**), and the right ECA (**c**) shows significant devascularization of the AVM nidus, achieved after 16 embolization sessions. This preparation allowed the microsurgical extirpation of the AVM, which was considered inoperable at the beginning. An early postoperative DSA 2 days after surgery, again with injection of the left VA (**d**), the right ICA (**e**), and the right ECA (**f**) confirmed that the arteriovenous shunt in the right occipital lobe was entirely interrupted. Previous feeding vessels of the AVM from the right MCA and PCA appeared as stagnant arteries (*arrows*; **e**) and the ECA branches were hypertrophic. Another DSA examination 3 months later (**g**, **h**, **i**) illustrated the remodeling of the cerebral vasculature. The site where the AVM nidus had been located was avascular (*arrows*; **g**, **h**). The remaining pial and dural vessels had essentially returned to normal, including the persistent occlusion of the aneurysm of the MMA

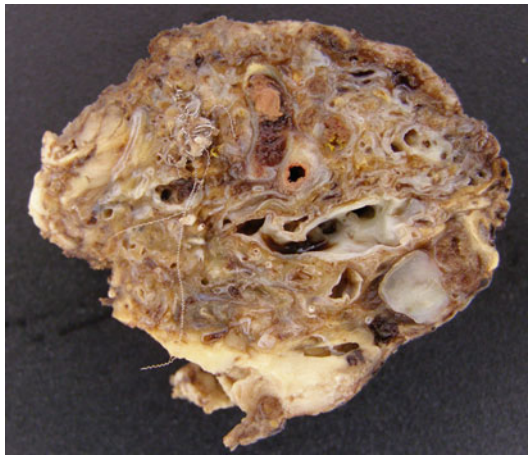

**Fig. 5** Macrophotography of the resected, 30 g weighing brain AVM. (Courtesy of Dr. Patricia Kohlhof-Meinecke, Institut für Pathologie, Klinikum Stuttgart)

usually not available. Dispersal of the tunica media and interruption of the external lamina elastica has been found (Ohta et al. 1991; Sandin et al. 1999). These aneurysms can cause an epidural (Holland and Thomson 1965), acute subdural (Koebbe and Horowitz 2004; Korosue et al. 1988), and subarachnoid hemorrhage (Kobata et al. 2001; Zubkov et al. 1998). Massive intracerebral hematomas have also been described (Kobata et al. 2001; Sandin et al. 1999).

As long as the parent artery allows access with a microcatheter, endovascular treatment of these aneurysms based on the use of liquid embolics, preferentially polymerizing glue, is usually straightforward (Lesley et al. 2004; Maekawa et al. 2009) (Fig. 7). Coil occlusion has also been advocated (Koebbe and Horowitz 2004). If the parent vessel does now allow access with a microcatheter, microsurgical clipping (Zubkov et al. 1998) or conservative management (Fig. 8) remain alternative strategies; although given the rarity of these lesions, the natural history and hence the potential risks of conservative management are unknown.

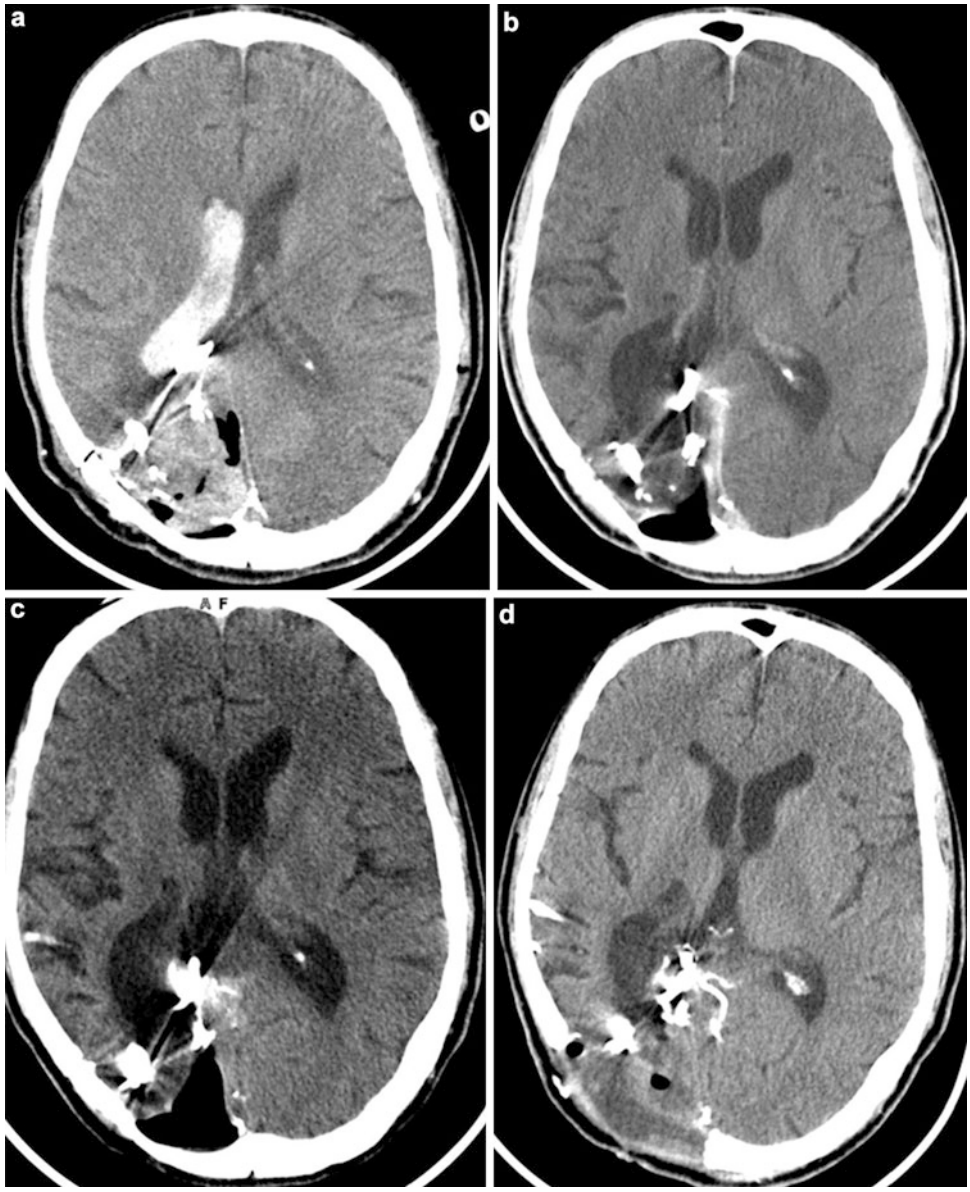


**Fig. 6** Postoperative CT examinations. The immediate postoperative CT (**a**) shows some blood in the previous location of the AVM nidus in the right occipital lobe, remaining hyperdense glue in non-resected and occluded MCA and PCA feeding arteries of the AVM and blood in the right lateral ventricle. One week later (**b**) there is no more blood visible, neither in the operative field nor in the ventricle but gas is trapped underneath the craniotomy. The amount of gas had increased almost 4 months later (**c**), when a wound-healing disturbance required revision surgery. Nine days after surgical wound revision the trapped gas is almost removed, with some local liquid collection (**d**)

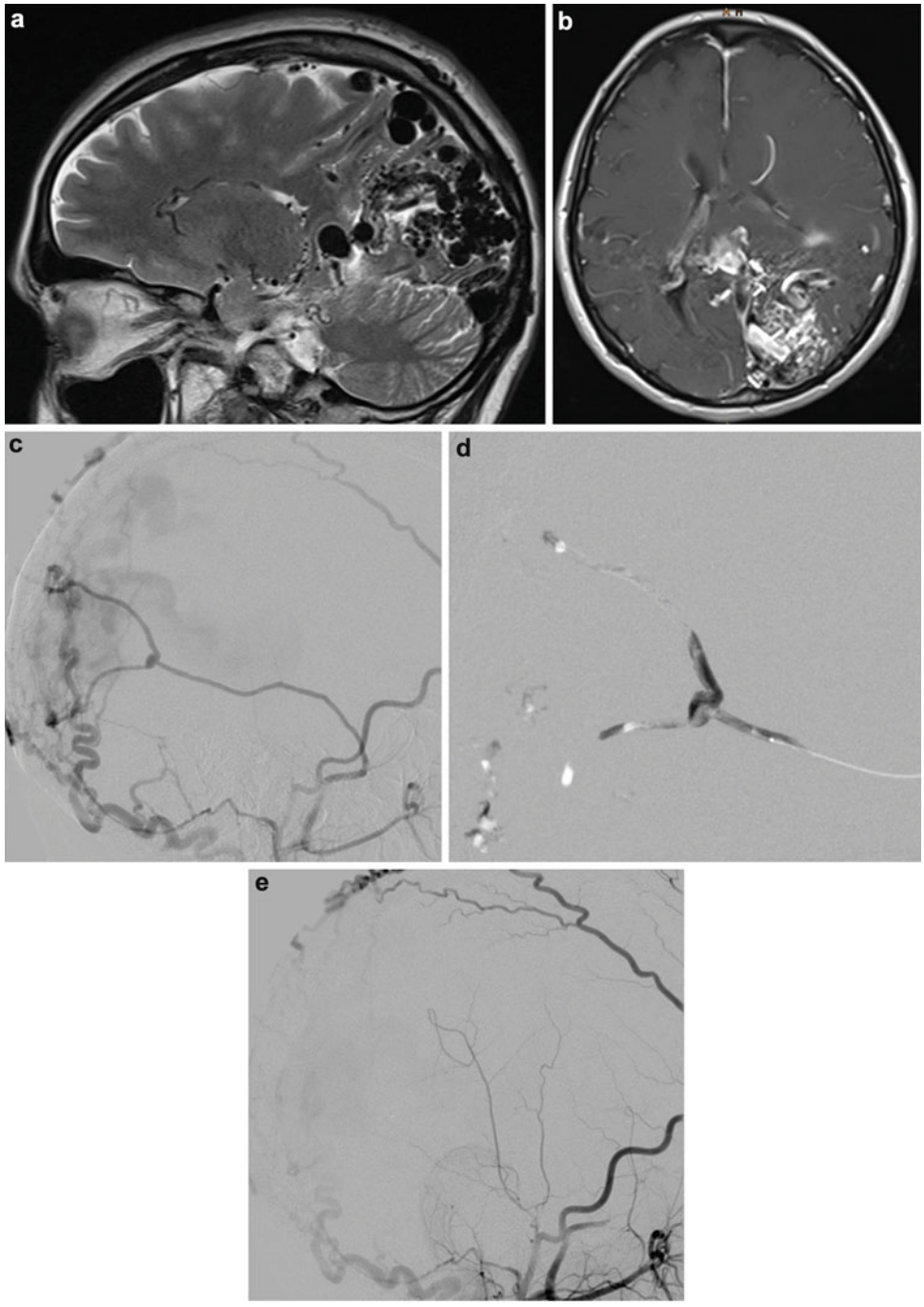


**Fig. 7** MRI and DSA images of a 28-year-old female patient who presented with recurrent headaches and occasional generalized epileptic seizures. T2WI (**a**) and contrast enhanced T1WI (**b**) MRI revealed a large AVM of the left occipital lobe. DSA with injection of the right ECA showed an incidental aneurysm of the posterior branch of the MMA with a diameter of 4 mm (**c**). The right MMA was catheterized with a Marathon (Medtronic) microcatheter, using a Mirage 0.008″ (Medtronic) microguidewire. After careful flushing of the microcatheter with a 40% glucose solution, a 1:3 mixture of Glubran2 (GEM) and Lipiodol ultra fluid was slowly injected. Venous passage through the nidus of the AVM was avoided. The injection of the liquid embolic agent was carried out in a way that ensured the obliteration of the MMA distal and proximal to the aneurysm (**d**). The final DSA run again with injection of the right ECA confirmed the occlusion of aneurysm and parent artery (**e**)

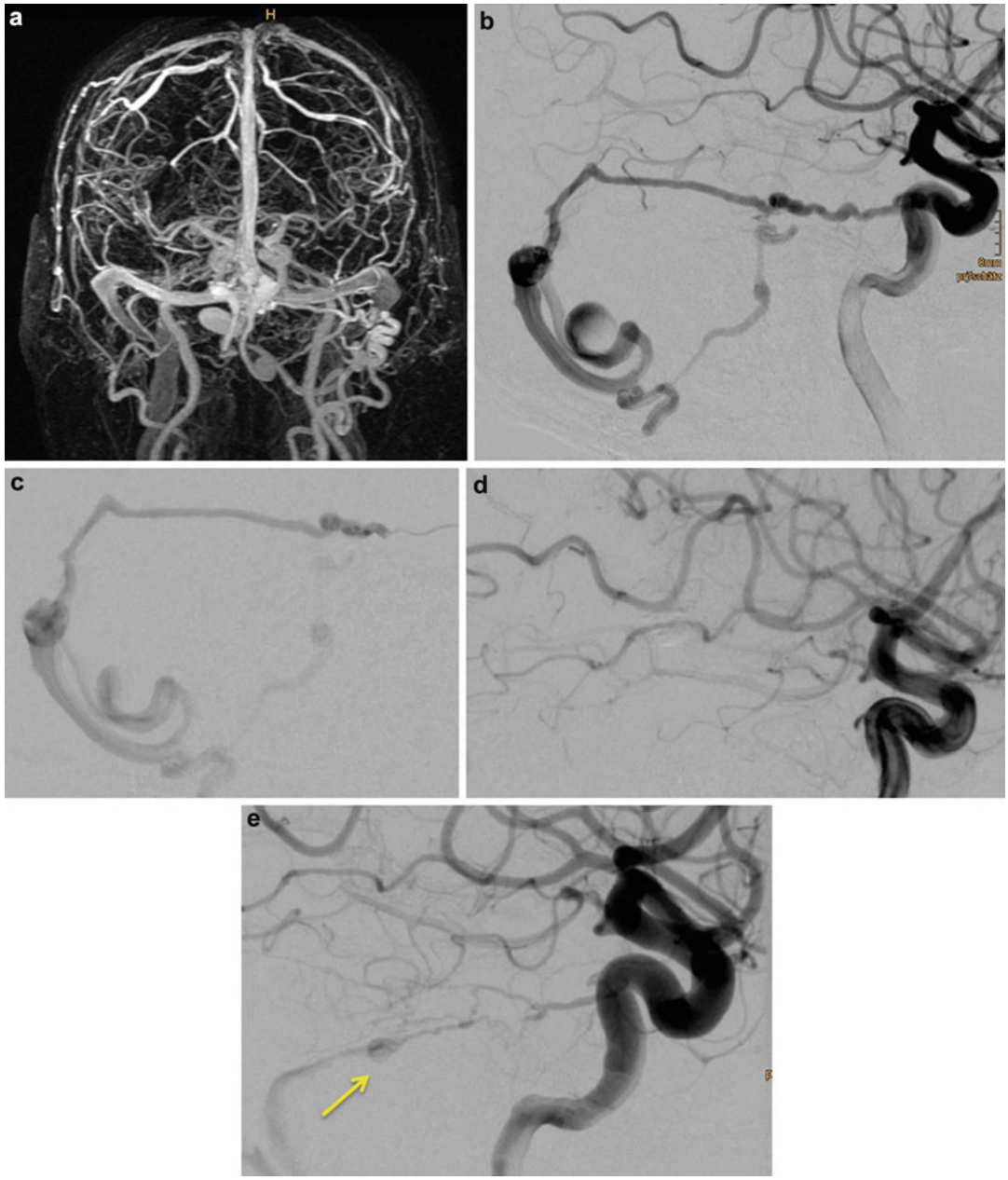


**Fig. 8** Dural arteriovenous fistula (dAVF) of the posterior tentorium and the torcular. Contrast enhanced venous MRA (**a**) shows the location of the dAVF and the varices. The hypertrophic tentorial artery from the right ICA was supplying the dAVF (**b**). Said artery was catheterized selectively (**c**) and occluded with Histoacryl and Lipiodol ultra fluid in a 1:2 mixture. The following injection of the right ICA confirmed the occlusion of the tentorial artery (**d**). DSA with injection of the right ICA 7 months later showed again a dural AV shunt with tentorial supply, now with a *de novo* aneurysm on this artery (*arrow*, (**e**)). This was the last AV shunt of the previously large dAVF. No attempt to catheterize the tentorial artery was undertaken, and the patient refused to have any other treatment. The last DSA (**e**) dates from February 2009 and through June 2018, the patient remained asymptomatic

## Therapeutic Alternatives

Coil Occlusion
Microsurgical Treatment
Onyx Embolization
PHIL Embolization

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