Tentorial arteriovenous malformation presenting as an intracerebral hematoma

Case report

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A case of a tentorial arteriovenous aneurysm is reported in a 54-year-old man. This malformation, fed by branches of the external carotid artery, was drained exclusively by a parieto-occipital cortical vein. The rupture of this vein was responsible for the presenting intracerebral hematoma.

KEY WORDS • dural arteriovenous malformation • intracerebral hematoma • embolization

Dural arteriovenous malformations (AVM's) occur infrequently. Although the Cooperative Study did not mention any instances, Newton, et al.,5,6 found 15 cases in a series of 129 patients with angiomas. We would like to report an unusual case of a patient who presented with intracerebral hematoma in a purely dural fistula, and to discuss its physiopathology.

Case Report

This 54-year-old cabinet-maker was hospitalized for evaluation of the sudden onset of a headache while straining. He had a 5-year history of hypertension. On examination, he was conscious but had a stiff neck, moderate left hemiparesis, and left superior lateral homonymous quadrantanopsia. There were no bruits.

Computerized tomography (CT) (Fig. 1) before and after contrast enhancement demonstrated a right temporoparietal hematoma that had ruptured at the cortex. There was no apparent vascular abnormality. A right internal carotid angiogram (Fig. 2 left) confirmed the presence of an avascular space-occupying lesion without a visible vascular malformation. However, the right external carotid angiogram (Fig. 2 right) revealed an AVM situated on the tentorium cerebelli and fed by two dilated branches of the right occipital artery and a posterior branch of the middle meningeal artery. Venous drainage was by a voluminous cortical vein that followed a sinuous course over the temporoparietal cortex. Vertebral and left internal carotid angiograms were normal.

The right occipital and right maxillary arteries were selectively embolized with Gelfoam. The next day, a right parieto-occipital craniotomy was performed, and the draining cortical vein was immediately evident. A loop of this vein delimited the cortical surface of the intracerebral hematoma. The occipital lobe was lifted, and a venous ampulla, in contact with the right half of the tentorium cerebelli and site of the angioma, was seen. The AVM was clipped, along with its venous draining vessel.

There were no postoperative complications, and a follow-up angiogram showed complete eradication of the malformation (Fig. 3). The patient was seen a year later and had returned to work; the neurological examination was normal, with the exception of regressing quadrantanopsia.

Discussion

Dural AVM's occur most often in the posterior fossa; nine of the 15 cases reported by Newton, et al.,6 were in that location. The majority of AVM's are fed by branches of the external carotid artery (occipital and posterior twigs of the middle meningeal arteries) and are drained directly by the dural venous sinuses. The existence of parenchymatous AVM's, totally or
Intracerebral hemorrhage with tentorial AVM

FIG. 1. Preoperative computerized tomography scan showing a right temporal intracerebral hematoma ruptured at the cortex.

FIG. 2. Left: Right internal carotid angiogram showing a right parietotemporal avascular mass, with no vascular malformation visible. Right: Right external carotid angiogram showing the feeding arteries (open arrows), branches of the right occipital artery, and a posterior branch of the right middle meningeal artery. The venous drainage is indicated by small black arrows. Two confluent veins form an ampulla (two black arrows) which empties into a cortical vein, whose loop (large black arrow) marks the site of rupture.

FIG. 3. Postoperative common carotid angiogram showing absence of the malformation.

Our patient falls midway between the two varieties, partially fed by the external carotid artery, has been reviewed by Dahl and Kline. They are slightly less common than purely dural AVM's. The venous drainage is by way of cortical veins. Our patient falls midway between the two varieties, having a purely dural angioma with afferent supply exclusively from the external carotid artery, but with an efferent cortical vein and no hemispheric extension. This case can be categorized as Type 3 as described by Schisano, et al. (dural arteriovenous...
aneurysm with cortical drainage). Schisano’s first case and that of Legrè, et al.,\textsuperscript{4} concern dural occipital angiomas drained by cortical veins into the superior longitudinal sinus. The dural AVM in Case 2 of Pecker, et al.,\textsuperscript{7} was situated in the posterior fossa dura and was drained by veins of the brain stem.

Anastomosis between cortical and meningeal veins is well known, and as Kaplan\textsuperscript{2} states, “from the cerebral and cerebellar hemispheres veins lying superficial to the arteries join the meningeal venous system.” As for the existence of arteriovenous anastomosis between meningeal arteries and cortical veins, Ramamurthi and Balasubramanian\textsuperscript{8} reported two cases, without specifying the cortical or dural location of the angioma.

The most striking point of this case is that the patient presented with symptoms of a spontaneous intracerebral hematoma. When a dural AVM is associated with bleeding, it is usually as a subarachnoid hemorrhage. The origin of our patient’s hemorrhage was the rupture of an efferent vein, which is a very rare cause of intracerebral bleeding. Kosnik, et al.,\textsuperscript{3} have suggested that highly elevated venous pressure alone can cause such a rupture. In addition, this pressure also can later result in communicating hydrocephalus.

This rare complication of a dural angioma calls for a comment on the indications for angiography in patients with cerebral hemorrhage. It is generally accepted that angiography is justified if a vascular malformation is suspected as the cause of the hemorrhage, especially if the hematoma is located near the cortical surface and the patient is young and normotensive. These last conditions are perhaps too rigid, as our 54-year-old patient was hypertensive.

The malformation, which was not seen on the CT scan, would not have been detected without an external carotid angiogram. We feel justified, then, in recommending selective opacification of the internal and external carotid arteries. This approach is particularly recommended if patients are to be treated conservatively (without initial surgical exploration), with the possibility of missing an angioma and the risk of recurrent hemorrhage.

References


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