Angiographic visualization of fatal hemorrhage from a cerebral arteriovenous malformation

Case report

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The authors report and discuss a rare angiographic demonstration of extravasation of contrast material from a ruptured arteriovenous malformation.

KEY WORDS • cerebral hemorrhage • arteriovenous malformation • angiography

The angiographic diagnosis of a ruptured cerebral aneurysm is usually based on indirect evidence such as adjacent mass effect, spasm of local or distant vessels, irregularity of the dome of the aneurysm, increased circulation time, and hydrocephalus. Direct angiographic evidence of a ruptured aneurysm is rare but has been reported.5,9,10,12,16,17,20,22,24,27,30

This report we believe represents the first angiographic demonstration of an active hemorrhage from an arteriovenous malformation (AVM).

Case Report

A previously healthy, nonhypertensive 66-year-old man was involved in a minor automobile accident immediately preceding which he had experienced mild nausea and headache. When seen right after the accident he was initially alert, but had weakness of the left arm. His blood pressure was 200/130 mm Hg and pulse 85/min. Although he ignored the left side of his body, his sensorium was clear. He had no external evidence of trauma. During the next 15 minutes he became increasingly drowsy and began to vomit; left-sided hemianopsia, sensory loss, and upper extremity and facial paralysis rapidly evolved. Fifteen minutes later he was bilaterally decerebrate and had dilated fixed pupils. He was intubated and given intravenous mannitol and dexamethasone. Emergency right common carotid angiography was performed to rule out a treatable lesion; within 1 hr of his arrival at the hospital, the first angiographic series had been concluded and showed massive extravasation of the contrast medium from an AVM at the anterior end of the Sylvian fissure (Fig. 1). The patient was given supportive therapy only and died 24 hrs after admission.

At autopsy the brain weighed 1710 gm;
Angiographic demonstration of hemorrhage from AVM

there was extensive subarachnoid hemorrhage. The first (anterior temporal) major branch of the right middle cerebral artery entered a tangle of enlarged vessels about 2.5 cm in diameter deep to the orbital cortex. The malformation showed moderate to severe atherosclerosis with focal, but nonocclusive, thrombus formation. An intact saccular aneurysm, 0.9 cm in diameter, was located on the major feeding artery of the angioma (Fig. 2). A 60 cc clot in the right frontal lobe extended into the ventricular system.

**Fig. 1.** Right common carotid angiography. *Upper Left:* Frontal projection showing irregular tangle of vessels (arrowheads) with contralateral bowing of the pericallosal artery (small arrows). ICA = internal carotid artery. *Upper Right:* Arterial phase 1 second later. Extravasated contrast material (tailed arrow) is in contiguity with residual contrast in the vascular tangle (arrowheads). The cavernous sinus (Cav. S.) is prematurely opacified. *Lower Left:* Lateral projection of arterial phase showing vascular malformation (arrowheads) with its feeding vessel (open arrows) and coexisting aneurysm (black arrows). The basilar venous plexus (BVP) is opacified early while contrast material is still visible in the internal carotid artery (ICA). OR = orbital roof, ANT = anterior. *Lower Right:* Very early venous phase showing extravasated contrast material located between the temporal lobe anterosuperiorly and the sphenoid bone of the middle cranial fossa (black arrows). A second globular collection of dye is present in the temporal lobe (open arrow). Fr S = frontal sinus, OR = orbital roof, BVP = basilar venous plexus.
FtG. 2. Gross pathology. Right-sided brain dissection at postmortem examination demonstrating an unruptured aneurysm (large black arrows), 0.9 cm in diameter on the artery (open arrow) supplying the vascular malformation (arrowhead). Small arrows indicate clot in ventricle.

Discussion

Over the past 40 years, cerebral angiography has become the prime method for definitive diagnosis of aneurysms and angiomas. An insufficient number of angiographic studies cannot account for the rarity of documented extravasation when the Cooperative Study from 19 institutions reported 5484 patients who had undergone cerebral angiography in the investigation of recent subarachnoid hemorrhage.

Several factors may be relevant in explaining the rarity of angiographic demonstration of acutely leaking aneurysms and angiomas. Very ill patients, in the absence of suspected life-threatening hematoma collection (subdural or accessible intracerebral clots), are not studied acutely because the surgical mortality and angiographic morbidity are prohibitive. Patients initially classified as clinical Grade 4 or 5 include those whose cerebral hemodynamics constitute a threat of massive and persistent hemorrhage; they are also those least likely to undergo cerebral angiography early in the course of their vascular catastrophe. Progressive clot accumulation and brain swelling from persistent hemorrhage rapidly produce counterpressures which halt ongoing extravasation. A major exception allowing for prolonged leakage of blood without significant counter-pressure is rupture of a subependymal vascular anomaly or dissecting hematoma into the cerebral ventricles; this occurred in at least seven of the 20 or so documented cases of leaking aneurysm, allowing visualization of this dramatic event.

Subarachnoid hemorrhage patients in the progressively severe clinical Grades 3 through 5 carry an increasing incidence of arterial spasm of the cerebral vessels, decreasing the likelihood of cerebral angiography. Arterial spasm in the region of active hemorrhage probably plays an important role in reducing ongoing extravasation, although the exact relationship between cerebral blood flow, visible arterial spasm, and clinical status is still controversial. No arterial spasm was recognized in our patient.

Despite the opinions of several previous authors that, with coexistent aneurysms and AVM the angioma usually bleeds, the Cooperative Study shows a roughly equal incidence of hemorrhage from each type of lesion. When a saccular aneurysm is located on the major feeder of a coexisting AVM the hemodynamic influences, such as the physical laws of Bernoulli and Poiseuille and the variable effect of turbulence, must interact in complex fashion to regulate lateral pressure in the dome of the aneurysm; indeed, it may be that aneurysms in this location are relatively protected from rupture.

Our patient experienced a mild ictus followed within an hour by massive hemorrhage documented angiographically. Despite scattered reports attributing rebleeding from an aneurysm to an angiographic procedure, careful analysis of 7933 carotid and vertebral studies performed during the Cooperative Study revealed only seven such episodes during the procedure. In particular, Vines and Davis suggest that low-pressure small bolus injections and more peripheral angiographic routes be used to preclude this catastrophe. These technical modifications may result in suboptimal visualization and false negative studies. Conversely, there is evidence indicating no significant increase in cranial in-
travascular pressure following common carotid injections of contrast media.

Leakage of contrast material from cerebral vessels at the time of angiography has been documented in hypertensive basal ganglial hemorrhage,18 cerebral arteritis,8 cerebral contusion, torn meningeal vessels,14,21 traumatic disruption of intracranial arteries,16,38 and subdural hematoma.13 It has been recently recognized that there is frequently active leakage (or secretion) of contrast material from the falx cerebri and tentorium in patients without cerebral vascular lesions.19

We suggest that existing evidence indicates a purely coincidental relationship between rebleeding and radiographic documentation; this suggestion is based on the natural history of aneurysms and AVM's.

References


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