Case Report: Spontaneous Thrombosis of a Cerebral Arteriovenous Malformation

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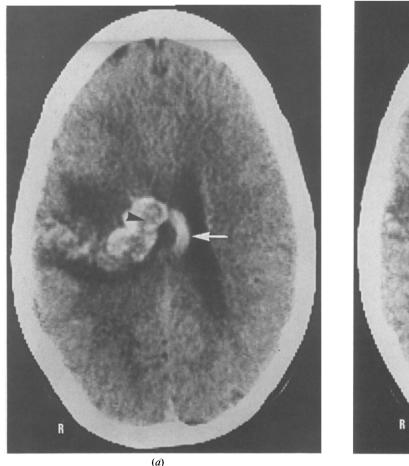
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We report a patient who presented acutely to a neurosurgical unit with a mild left hemiparesis following mild trauma and an episode of hypotension. Computed tomography of the brain showed fresh partial thrombosis of a right parietal arteriovenous malformation. Subsequent angiography confirmed partial obliteration of an arteriovenous malformation.

The malformation had completely thrombosed on follow up computed tomography, and disappeared angiographically. Barker, C.S. & Anslow, P.L. (1990). *Clinical Radiology* 42, 130–132. Case Report: Spontaneous Thrombosis of a Cerebral Arteriovenous Malformation

CASE REPORT

A 23-year-old right handed female cut her hand, and while washing the hand in the bathroom she fell, striking her left temporal area. On standing up she fainted, losing consciousness for about 15 seconds. She was then well until 4 h later when she experienced sudden onset of severe headache and vomiting. On admission to hospital she was found to have a mild left hemiparesis.

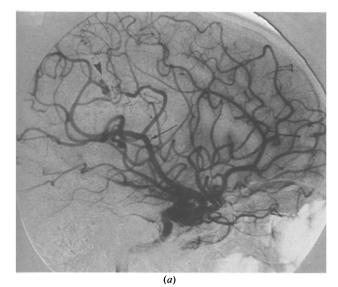


Five days later computed tomography of the brain was performed. The unenhanced scan (Fig. 1*a*) showed a 5 cm diameter mass in the right parietal region, of mixed density, although predominantly hyperdense, with a high density serpiginous structure arising from it and passing along the wall of the lateral ventricle. There was surrounding oedema. On other sections fresh thrombus was seen within the straight sinus (Fig. 1*b*). Following intravenous contrast medium an enlarged branch of the right middle cerebral was seen passing towards the mass, but little



Fig. 1 – Day 6. Unenhanced CT. (a) Presumed older thrombus within the nidus of the arteriovenous malformation (black arrowhead) and fresh thrombus within the draining vein (white arrow). (b) Fresh thrombus within the straight sinus (black arrow).

Correspondence to: Dr C. S. Barker, Department of Radiology, Radcliffe Infirmary, Woodstock Road, Oxford OX2 6HE. enhancement of the mass itself occurred. This was interpreted as a partially thrombosed arteriovenous malformation containing older thrombus within the nidus and fresh thrombus in the draining vein. A right carotid angiogram performed on day 8 (Fig. 2) showed an



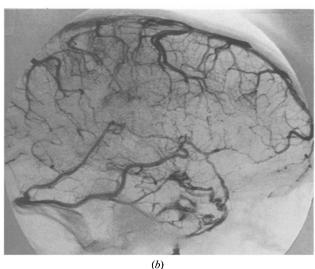


Fig. 2 – Day 8. Right carotid angiogram. (a) Arterial phase: enlarged branch of middle cerebral artery (small black arrow) and apparently small arteriovenous malformation (black arrow head) (b) Venous phase: there is no filling of deep veins.

enlarged branch of the middle cerebral artery with very low flow into a small arteriovenous malformation in the parietal lobe, an appearance consistent with a malformation into which the blood flow has recently been markedly reduced. The venous phase showed drainage via superficial veins with no filling of the deep veins.

Computed tomography was again performed on day 19 (Fig. 3), before and after contrast enhancement. The unenhanced scan showed mature isodense thrombus within the nidus and draining vein. Other sections revealed fine calcification within the nidus of the arteriovenous malformation. The scan with contrast medium showed some marginal enhancement of the malformation. There was still moderate surrounding oedema. Angiography on day 42 (Fig. 4) showed disappearance of the malformation and reduction in the size of the middle cerebral artery.

the malformation and reduction in the size of the middle cerebral artery. Further computed tomography has shown progressive calcification of the nidus and reduction in the size of the draining vein. The most recent examination, $2\frac{1}{2}$ years after the ictus, shows merely a small focus of calcification in the right parietal lobe (Fig. 5). There has been no radiographic evidence of reopening of the arteriovenous malformation nor of haemorrhage from it. The patient's hemiparesis gradually improved and she has remained well over 3 years apart from occasional headaches.

DISCUSSION

There have been about 50 reported cases of spontaneous thrombosis of cerebral and dural arteriovenous malfor-

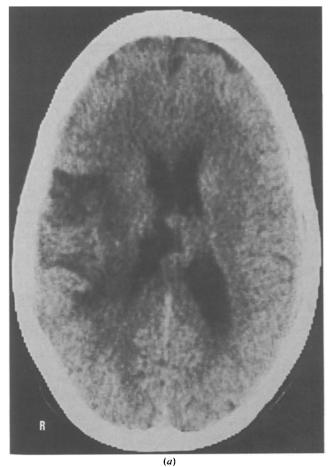




Fig. 3 – Day 19. (a) Unenhanced CT: mature thrombus throughout the malformation. (b) Enhanced scan at same level shows a little marginal enhancement (black arrowheads).



Fig. 4 - Day 42. Carotid angiogram: no evidence of arteriovenous malformation.

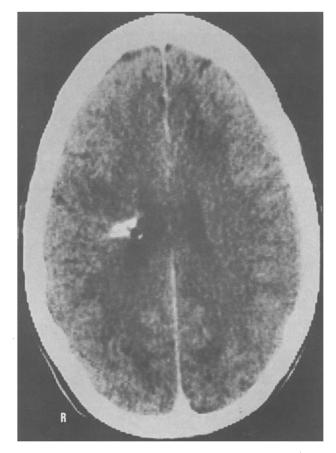


Fig. 5 – Computed tomography two and a half years after initial presentation shows a small focus of calcification in the right cerebral hemisphere.

mations over the past 40 years (Yasargil, 1987). Regression has been documented by angiography, radionuclide studies, computed tomography, or a combination of these. Most reports have been of isolated cases, and many have shown only partial thrombosis. However, several authors have collated the data from these case reports in order to draw general conclusions (Wakai *et al.*, 1983).

Patients in whom thrombosis is demonstrated are usually young or middle aged adults. Spontaneous thrombosis is more likely in a small arteriovenous malformation drained by a single vein (Conforti, 1971; Pasqualin *et al.*, 1985). In most cases regression has occurred acutely. The mechanisms of spontaneous thrombosis are not certain, but in the majority of cases it is associated with a_n episode of intracranial bleeding, either intracerebral or subarachnoid (Wakai *et al.*, 1983; Yasargil, 1987). Acute thrombosis is thought to occur due to compression from haematoma or oedema, or to reduced flow secondary to vasospasm. Less commonly thrombosis may occur sub. acutely due to increased blood coagulability or alterations in general cerebral perfusion. Other postulated mechanisms include occlusion of feeding vessels by atheroma or embolism (Sukoff *et al.*, 1972).

In our patient there was no radiographic evidence of intracranial haemorrhage. However, she did have a short period of hypotension after cutting her hand and this may have initiated subacute thrombosis within the nidus of the arteriovenous malformation.

In an assessment of future risk of haemorrhage from a thrombosed arteriovenous malformation it is important to know whether the occlusion is partial or complete. Angiography may be misleading in this respect: Norlén (1949) showed that partial thrombosis may so diminish the flow within the malformation that the abnormal vessels do not fill with sufficient contrast medium to appear radio-opaque. The greater contrast resolution of computed tomography should allow this small flow to be detected on a contrast enhanced scan but extravasation into damaged tissues also contributes to the enhancement shown by some of these malformations (Golden and Kramer, 1978). Magnetic resonance imaging has demonstrated persistence of an arteriovenous malformation in angiographically complete occlusion (Yasargil, 1987). However, magnetic resonance imaging may not always demonstrate patent vessels within such a lesion, particularly if flow is slow or there is local haemosiderin deposition from past haemorrhage (Needell and Maravilla, 1988).

We suggest that the marginal enhancement demonstrated in our patient (Fig. 3b) was due to extravasation of contrast medium and that thrombosis was complete at that time. The patient has remained well, but should recanalization and bleeding occur, reassessment as regards the suitability of the lesion for surgery, endovascular embolization, or radiosurgery would be required.

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