

Growth of an Arteriovenous Malformation: A Case Report

DAVID R. MINOR AND THOMAS GANDY

*Yale University School of Medicine, New Haven, Connecticut
and Mercy Medical Center, Redding, California*

Received April 9, 1977

A case of an intracerebral arteriovenous malformation that grew is presented. On the initial arteriogram only one early filling vein was seen in the region where a large arteriovenous malformation was present eight years later. The patient's headaches were relieved by antihypertensive medications. The authors speculate that distension of the AVM caused the headaches.

Cerebral arteriovenous malformations are believed to be developmental malformations and not true neoplasms [1]. In rare instances growth of these malformations has been documented [2]. In all previously reported cases of enlargement of an arteriovenous malformation (AVM) the lesion itself was clearly visible on initial angiographic studies. We are reporting a patient with surprising growth of her AVM and recurrent headaches of unusual character.

CASE REPORT

A 17 year-old female presented in 1967 with headache and lethargy, and lumbar puncture revealed bloody spinal fluid. Arteriograms at that time showed an aneurysm of the left internal carotid artery. In retrospect one early-filling vein in the left temporal region can be seen (Fig. 1). At surgery she had partial ligation of the left common carotid artery and intracranial exploration where two additional small daughter aneurysms were seen at the internal carotid-middle cerebral junction, one of which was clipped. The large aneurysm was packed with muscle and gelfoam. Several years later seizures began, and she was placed on phenytoin.

She was normotensive and well until January, 1974, when she was readmitted with a one day history of abdominal pain and vomiting, and headache for the previous hour. On admission the blood pressure was 190/110 and the spinal fluid was clear. The next day confusion and nuchal rigidity appeared and lumbar puncture revealed 30,000 RBC's and xanthochromia. All symptoms subsided with conservative care. As an outpatient her blood pressure was 110/80 and she was free of headaches.

In March, 1975, she was readmitted with headache, vomiting, and blood pressure of 180/100. The headache disappeared within minutes with intravenous nitroprusside although it recurred whenever the systolic pressure rose above 140. Lumbar puncture showed an opening pressure of 185 and clear fluid. Repeat cerebral arteriography showed a large superficial arteriovenous malformation in the left temporal-parietal region (Fig. 2). EEG demonstrated a left posterior temporal slow wave focus. Upper gastrointestinal series, intravenous pyelogram, and urine VMA were normal. She was discharged on phenytoin, methyldopa, and diuretics. In May, 1975, symptoms of increased intracranial pressure recurred. Spinal tap was traumatic with 2,000 RBC's

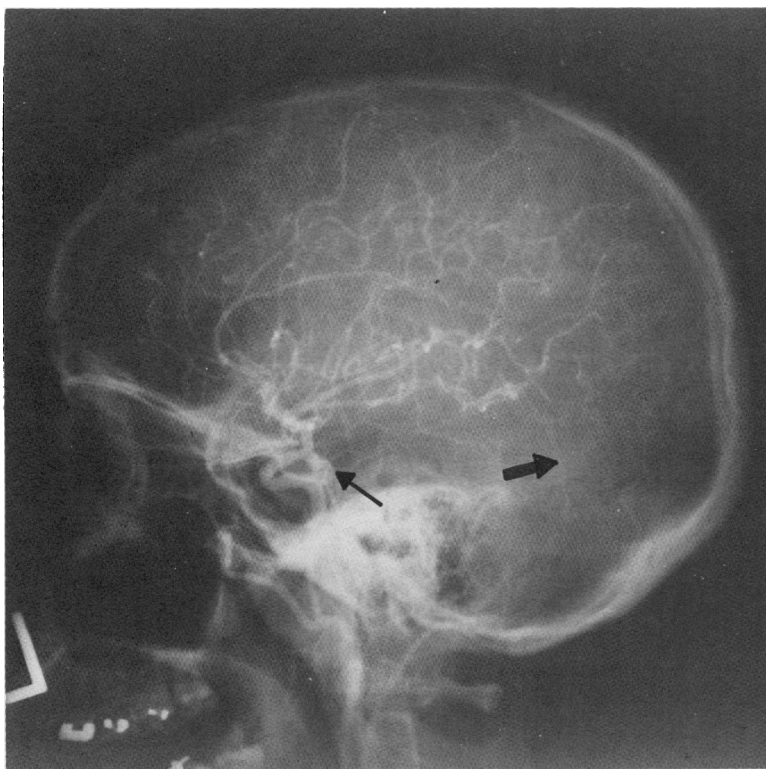


FIG. 1. Left carotid angiogram, July, 1967. The early filling vein draining the left temporal area is shown (*large arrow*). The AVM itself cannot be identified. The small arrow identifies the largest aneurysm.

and clear supernatant. The headache resolved but she was left with a fluent aphasia that partially cleared over the next few months. As an outpatient her blood pressure remained low on diuretics alone.

She was readmitted February, 1976, with a three hour history of headache, vomiting, and hypertension. Examination revealed an alert 25 year old woman with a severe fluent aphasia. Blood pressure was 150/90. A surgical scar and bruit were present over the left common carotid. There was no intracranial bruit. Although she was fluent, severe deficits in memory, comprehension, calculations, and vocabulary were present. She had mild right facial weakness, generalized hyperreflexia, and a right superior homonymous field defect. The headache was relieved within one hour by giving intravenous furosemide and methyldopa with subsequent fall of blood pressure to 90/60. Lumbar puncture was normal. Headache recurred three times on this admission, each time coincident with a rise in blood pressure as antihypertensive medications were being adjusted. During these episodes the pressure ranged from 150–185/90–110 and on each occasion the headache disappeared when the antihypertensive medications brought the systolic pressure below 130.

Lateral radionuclide flow studies were performed. On the first study while the patient was free of headache a hyperperfused area in the left temporal-parietal area was clearly shown. A repeat flow study three days later during a headache and while the patient was hypertensive showed an even greater increase in perfusion to the same area. A computerized axial tomographic scan showed the left temporal AVM as well as left hemisphere atrophy and dilatation of the left lateral ventricle. On subsequent

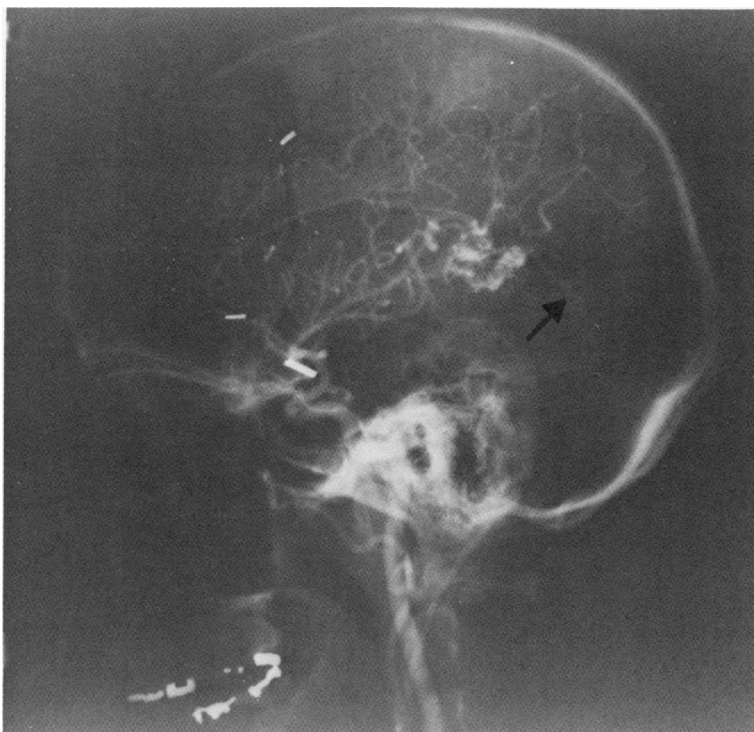


FIG. 2. Left carotid angiogram, March, 1975. A large AVM is clearly visible. The same draining vein is now enlarged (arrow). Both angiograms were performed injecting 60% Renografin through 5-F catheters taking 10 films over 10 seconds (2 films/sec x 2, then 1 film/sec x 3, then one film every other second for 6 seconds).

occasions her blood pressure has ranged from 90 to 160 systolic but headaches have not recurred. At present the patient is normotensive on a regimen of propranolol, methyldopa, diuretics, and phenytoin.

DISCUSSION

Potter[3] first reported enlargement of an intracerebral AVM in a patient with an intracerebral hemorrhage. He proposed that local increased venous pressure could lead to dilatation of neighboring venules and increase in the size of the lesion. Hamby[4] suggested that the rupture of small veins, forming an aneurysmal varix, might produce a larger shunt and enlargement of an AVM. Spetzler[5] showed enlargement of an AVM that appeared to be due to progressive dilatation of pre-existing abnormal vessels. In the case presented here the vessels making up the AVM were not visible on the initial angiogram. Only one early filling vein, a hallmark of an AVM, was present at that time. More than enlargement of pre-existing vessels seems to have occurred in this case, but the mechanism of its growth remains unexplained.

Headaches are a common symptom of an AVM in an adult. However, the pathophysiology of a headache due to an AVM in the absence of intracerebral or subarachnoid hemorrhage is unclear. Our patient had two episodes of gross subarachnoid hemorrhage and probably suffered hemorrhage or infarction in the area around the AVM in May, 1975, when her aphasia appeared. At other times she had severe generalized headaches accompanied by symptoms of increased intracranial pressure. The headaches always responded to reduction of the systolic pressure to

below 130. Migraine headaches are commonly believed to arise from dilatation of intracranial vessels. We propose that in this patient transient increased cerebral blood flow would lead to engorgement of the AVM, in turn increasing intracranial pressure with reflex increased blood pressure. Reduction of systemic blood pressure would decrease distension of the AVM and relieve the headache. The radionuclide flow studies appeared to show distension of the AVM occurring during the headache. Growth of the AVM did not seem to produce headaches; between the arteriograms shown here the patient complained of headache only once, at a time at which subarachnoid hemorrhage had occurred.

The coexistence of cerebral aneurysms and AVMs has been well documented in the past[5]. In some series they tended to occur on the same side as they did in this patient. One may only speculate on what role the aneurysms and the surgically created stenosis of the common carotid artery have played in the hemodynamics involved in the growth of this AVM and the recurrent headaches.

REFERENCES

1. McCormick WF: The pathology of vascular ("arteriovenous") malformations. *J Neurosurg* 24:807-816, 1966
2. Spetzler RF, Wilson CB: Enlargement of an arteriovenous malformation documented by angiography. *J Neurosurg* 43:767-769, 1975
3. Potter JM: Angiomatous malformations of the brain: their nature and prognosis. *Ann R Coll Surg* 16:227-243, 1955
4. Hamby WB: The pathology of supratentorial angiomas. *J Neurosurg* 15:65-75, 1958
5. Perret G, Nishioka H: Report on the cooperative study of intracranial aneurysms and subarachnoid hemorrhage: arteriovenous malformations. *J Neurosurg* 25:467-490, 1966

David R. Minor, M.D.
Oncology Fellow
Yale University School of Medicine
New Haven, Connecticut 06510

Thomas Gandy, M.D.
Radiologist
Mercy Medical Center
Redding, California 96001