The Changing Angiographic Appearance of an Arteriovenous Malformation After Subarachnoid Hemorrhage

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Summary. The changing angiographic appearance of a cerebral arteriovenous malformation (AVM) illustrated hemodynamic changes that can occur following subarachnoid hemorrhage and antifibrinolytic therapy. Decreased size of this lesion suggested thrombosis of the AVM. This appearance actually represented a transient, vasospastic phenomenon which reversed with time. Although the AVM underwent significant changes acutely, little changed in the long term.

Key words: Arteriovenous malformation - Spontaneous thrombosis - Subarachnoid hemorrhage - Cerebral angiography - Epsilon-aminocaproic acid (EACA)

Cerebral arteriovenous malformations (AVM) account for approximately 8% of all subarachnoid hemorrhages and are the major cause of spontaneous intracerebral hemorrhage in young individuals [10]. When the AVM is associated with an aneurysm, it is usually the aneurysm that has bled. AVMs, when examined at various intervals following diagnosis, have been reported to increase in size, decrease in size, disappear, or not change significantly [2, 4, 9, 11, 17, 18]. Their natural history, however, has not been well delineated. Only a few reports in the literature have documented partial or complete spontaneous disappearance of intracranial AVMs by serial angiographic studies [5, 6, 9, 12, 17, 18]. Many of these patients have had proven hemorrhage, usually subarachnoid, in association with thrombosis of the AVM [4, 6, 7, 12, 18]. The place of "cryptic" or "occult" AVMs in the natural history or classification of AVMs is poorly understood. This report presents unusual dynamic changes of an AVM during the immediate postictal period as revealed by angiography and the computed tomographic (CT) scan.

Case Report

A 35-year-old woman was admitted to Stanford University Hospital with a history of progressive, non-lateralizing frontal headaches, blurred vision, slight dysarthria, and numbness of the right arm for 2 days. The patient acutely developed right hemiparesis and dysarthria, followed by transient loss of consciousness on the day of admission.

The initial CT scan on day 1 revealed blood in the basal cisterns, Sylvian cisterns, and sulci of the left hemisphere. Diffuse enhancement of the leptomeninges, most prominent in the basal cisterns, was noted after intravenous contrast infusion (Fig. 1). No enlarged vascular structures were identified on the CT scan. On day 3, a cerebral arteriogram (Fig. 3 a-c) demonstrated a left parieto-occipital AVM. The anterior portion of the AVM was fed by branches of the left anterior and middle cerebral arteries which exhibited mild vasospasm. The posterior cerebral artery fed a posterior segment of this AVM (not illustrated). Venous drainage was accomplished by the superior sagittal sinus and an enlarged vein of Labbé. Multiple aneurysms were identified and involved the anterior communicating artery, the left internal carotid artery, the trifurcation of the left middle cerebral arteries which exhibited mild vasospasm. The posterior cerebral artery fed a posterior segment of this AVM (not illustrated). Venous drainage was accomplished by the superior sagittal sinus and an enlarged vein of Labbé. Multiple aneurysms were identified and involved the anterior communicating artery, the left internal carotid artery, the trifurcation of the left middle cerebral artery, and the proximal right and left posterior cerebral arteries. Treatment was initiated with dexamethasone, methyldopa, phenobarbital, and epsilon-aminocaproic acid (EACA) following angiography on day 3.

The patient's hemiparesis progressed and the patient became mute on the tenth hospital day.
Fig. 1a–d. Day 1 CT scan without (a, c) and with (b, d) contrast demonstrated blood in subarachnoid space (a) and contrast enhancement of leptomeninges (c, d), most prominent in basal cisterns. Subarachnoid blood and contrast enhancement were most prominent in left Sylvian fissure, the site of bleeding aneurysm. Nodular density in region of left posterior communicating artery represents an aneurysm (a).

Fig. 2a–d. Day 10 CT scans without (a, c) and with (b, d) contrast demonstrate left parietal infarct (c, d). Linear and nodular areas of contrast enhancement represent infarct rather than AVM (d). Blood was no longer detectable on plain scan (a) and leptomeningeal enhancement had become less evident. Previously noted aneurysm is now more discretely visualized after contrast administration (a, b).

Evaluation of a cerebral AVM may be influenced by a sampling error in the timing of cerebral angiography, especially in the context of an acute event such as subarachnoid hemorrhage. Lack of visualization of part or all of a cerebral AVM on subsequent angiography has been interpreted as thrombosis, while an increase in size has been interpreted as growth of a lesion. In our patient, the AVM appeared to decrease in size from the first to the second angiogram, and to increase in size from the second to the third angiogram. If considered separately, either combination of angiograms could have led to erroneous conclusions. This type of sampling error could occur if an initial angiogram were obtained after subarachnoid hemorrhage, and a follow-up angiogram were performed months to years later. Serial angiography in our patient revealed that, despite significant short interval alterations, the scan at this time demonstrated a left parietal infarct (Fig. 2). Repeat cerebral arteriography on day 11 (Fig. 3d and e) showed marked vasospasm and apparent occlusion of the multiple feeding arteries. Transit time determined by angiography was markedly slowed. Several arteries never filled completely. This resulted in nonfilling of a major portion of the AVM. The previously demonstrated large vein of Labbé did not fill.

The aneurysm of the left middle cerebral artery, the source of the subarachnoid hemorrhage as determined at operation, and the aneurysm of the left posterior cerebral artery were clipped on the 20th hospital day. On day 26, 6 days after cessation of EACA therapy, a third cerebral angiogram (Fig. 3f and g) revealed restoration of the original pattern of blood flow to the AVM which was again visualized in its entirety. The enlarged vein of Labbé, however, was not demonstrated and was presumed to be thrombosed. CT scan demonstrated a resolving parietal infarct 47 days after admission.

Discussion

Evaluation of a cerebral AVM may be influenced by a sampling error in the timing of cerebral angiography, especially in the context of an acute event such as subarachnoid hemorrhage. Lack of visualization of part or all of a cerebral AVM on subsequent angiography has been interpreted as thrombosis, while an increase in size has been interpreted as growth of a lesion. In our patient, the AVM appeared to decrease in size from the first to the second angiogram, and to increase in size from the second to the third angiogram. If considered separately, either combination of angiograms could have led to erroneous conclusions. Less dramatic findings than in this patient could be misinterpreted. This type of sampling error could occur if an initial angiogram were obtained after subarachnoid hemorrhage, and a follow-up angiogram were performed months to years later. Serial angiography in our patient revealed that, despite significant short interval alterations, the