Stenosis of Afferent Vessels of Intracranial Arteriovenous Malformations

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Four women are described with stenoses of the feeding arteries to cerebral arteriovenous malformations. These stenoses were hemodynamically significant in that the nidus malformations filled late in the arterial phase of angiography and the draining veins did not fill much earlier than the usual venous phase. In two of these patients, who were referred for possible embolization, the stenosis served as an impediment to balloon navigation in the feeding vessel in one and served as a partial contraindication to embolization in the other. It is possible that if these stenoses progress, they may lead to thrombosis of the arteriovenous malformations.

Arteriovenous malformations (AVMs) are regarded as developmental malformations and not as neoplasms [1, 2]. Parkinson and Bachers [3] suggested a more descriptive definition, "congenital arteriovenous fistulous malformation." Essentially an AVM consists of one or more large afferent arteries, a coiled mass of vessels, and one or more large draining veins that empty into one of the venous sinuses. The arterial supply or afferent artery is usually a normally placed but enlarged tortuous channel [1, 4, 5]. Most reviews of the pathology have tended to concentrate on the core or nidus of the lesion, that is, the coiled mass of vessels, with the afferent or efferent vessels receiving very little attention.

The aim of our communication is to report four patients with supratentorial AVMs in whom the afferent arteries showed stenotic regions. Angiographic documentation of such stenoses in afferent vessels of AVMs is uncommon: development of such a stenosis has been reported in association with partial spontaneous regression of a cerebral AVM [6]. The pathogenesis of such lesions is speculative. Such stenoses may have clinical implications in the choice of treatment technique, particularly with regard to intraarterial treatment.

Case Reports

Case 1

A 24-year-old woman had a subarachnoid hemorrhage some months earlier. She was able to speak but suffered from word reversal and some jargon speech. She had no major weakness or other significant neurologic findings. Angiography showed an AVM in the region of the splenium of the corpus callosum fed by callosal branches of the left posterior cerebral artery (PCA). A left carotid angiogram months after the hemorrhage showed filling of the left PCA, whose branches fed the AVM. There was a short, smooth stenosis in the posterior perimesencephalic part of the PCA (fig. 1). The patient had been referred to our clinic for possible embolization by calibrated-leak balloon. This was denied because it was believed that the balloon would not be able selectively to get the posterior callosal branches supplying the AVM, and the stenosis would increase the difficulty of intraarterial navigation with the balloon.
Fig. 1.—Case 1. Left carotid angiogram. Filling of left PCA, which feeds splenium AVM. Stenosis (arrow) in posterior perimesencephalic segment of left PCA.

Fig. 2.—Case 2. Left carotid angiogram. Parietal AVM supplied by pericallosal artery. Short stenosis (arrow) of distal part of feeder. Some small vessel narrowing.

Case 2

A 25-year-old woman had a non-coma-producing hemorrhage 6 years after recognition of a nondominant parietal AVM that was fed by all three major circulations. Left carotid angiography demonstrated a stenosis of one of the anterior cerebral feeders of the AVM (fig. 2).

Case 3 (case 3 in [6])

A 49-year-old right-handed woman was discovered by her optometrist to have a left visual field defect. She also had headaches. Computed tomography (CT) revealed a large contrast-enhancing calcified mass lesion surrounded by a zone of low density in the right occipital region. Cerebral angiography revealed a right occipital AVM being fed by the right PCA with an aneurysm at the origin of the feeding vessel. The PCA was demonstrated to be large and tortuous (fig. 3A). She was managed conservatively. A follow-up angiogram 17 months later showed a decrease in caliber and multiple segmental stenoses of the feeding vessels (fig. 3B). The aneurysm at the origin of the feeding vessels was smaller and had a more irregular outline. It is important to note there was no change in the symptomatology at repeat angiography.
Case 4

A 34-year-old left-handed woman had an episode of right hemianesthesia with headache, nausea, and vomiting at age 18. She recovered after a few days without residual deficit. One year later, she started to have generalized seizures and sometimes transitory numbness of the right hemibody; these were partially controlled with Dilantin and phenobarbital. She continued to have an average of five or six seizures a year. When an investigation revealed a left frontal AVM, she was referred to our hospital for management. A left carotid angiogram showed the left frontal AVM fed mainly by the left middle cerebral artery (fig. 4). There was a tight stenosis of the middle cerebral artery. A calibrated-leak balloon was able to pass the stenosis. Partial embolization of the AVM was carried out with a calibrated-leak balloon for injection of isobutyl-2-cyanoacrylate [7]. The stenosis of the left middle cerebral artery proved to be an impediment to any additional balloon navigation beyond, and therefore no additional embolization was done. However, the partial embolization slowed the flow through the AVM. At no time in this patient’s course had there been evidence of hemorrhage.

Discussion

Known causes of vascular stenosis are spasm, arteriosclerosis, periarterial fibrosis from organizing hemorrhage, recanalized thrombus, embolic phenomenon, arteritis, and tumor encasement of vessels. Henderson and Gomez [8] stated that hemorrhages from AVMs do not cause arterial spasm. However, occasional vasospasm has been described with AVM [9]. In a report of 100 consecutive supratentorial AVMs, Parkinson and Bachers [3] documented only one instance of vasospasm with AVM hemorrhage, concluding and agreeing with previous writers that vasospasms was rarely seen in subarachnoid hemorrhage from AVMs either pre- or postoperatively. However, the angiogram in our second patient was obtained after a recent bleed, and since follow-up angiography was not done, spasm could not be completely excluded.

However, case 4 showed a short stenosis, not as smooth as in the other patients. This was probably arteriosclerotic in origin. Large irregular nodules of hyalinized intima and smooth muscle have been demonstrated histologically to project into the lumina of vessels in AVMs [10]. Walls of AVM vessels are usually degenerate and fibrosed with no elastic laminae [1, 2, 11].

In a previous communication [6], we postulated that the stenoses in case 3 could have been associated with emboli from the clot-filled aneurysm on the feeding vessels. This idea is supported by the fact that the aneurysm was irregular and smaller on repeat study, indicating considerable clot formation within. On the other hand, decreased flow through the AVM at the same time as the stenoses developed could have initiated turbulent flow changes that allowed the aneurysm secondarily to thrombose. There was no history of a recent bleed to suggest the possibility of spasm. The other causes of stenosis could not be applicable in these patients. There was no evidence of intracerebral hematoma or neoplasm, which could encase the vessels and thereby produce stenosis.

Embolization of the AVM was carried out in case 4. The stenosis proved to be an impediment to intraarterial balloon navigation after one injection of acrylate. A request for embolization was refused in case 1 partly because of experience with case 4 and because AVM was in a location where the risks of surgical approach were not unreasonable.

The potential hemodynamic effects of such stenoses include slowing down the rate of flow in the affected vessels, and progression of such stenoses may obliterate the AVM in unicompartamental lesions. Case 3 definitely showed partial regression of the AVM at the time the stenoses were demonstrated [6]. The other three cases showed the AVMs filling later in the arterial phase of the angiogram than the usual early filling in such lesions. A reduction in pressure across some stenotic lesions is expected and the risk of further hemorrhage in these patients is uncertain. However, a reversal of stenosis in those cases suspected to result from spasm would be expected.

Addendum

Since submission of this manuscript, another article on the same subject has been published [12].

REFERENCES

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