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Spontaneous Regression of Large Bilateral Basal Ganglia Arteriovenous Malformations

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The complete or partial spontaneous regression of large arteriovenous malformations (AVM) is very rare [1–5]. In most cases, a hemorrhagic event or surgical manipulation will have contributed to the thrombosis of the AVM [2, 3].

We report here a patient with large bilateral basal ganglia AVMs who presented with headaches. She was neurologically intact and has remained so for 10 years. The patient recently developed new bifrontal headaches that were not associated with hemorrhage or ischemia as revealed by MR imaging. An angiogram demonstrated near complete regression of the large AVMs. This case is unique because of the bilateral location of the AVMs in the basal ganglia as well as their spontaneous regression.

Case Report

A 20-year-old, right-handed woman presented in 1980 with persistent mild headaches. She had no sudden, severe headaches suggestive of an intracranial hemorrhage, or any seizure activity. Her neurologic examination was normal. A cerebral angiogram demonstrated numerous large striate and thalamoperforate vessels supplying bilateral large basal ganglia AVMs. The feeding vessels originated from both A1 and M1 segments bilaterally, the apex of the basilar artery, both posterior cerebral arteries, and the choroidal vessels (Fig. 1). Since the patient had no hemorrhage and was neurologically intact, she was treated conservatively.

Over the next 10 years, the headaches recurred intermittently but were never severe enough to suggest hemorrhage. Frequent CT and MR scans ruled out bleeding associated with the headaches. Aside from the AVM, no other lesions or abnormalities were ever detected. An MR examination performed approximately 6 months prior to the most recent angiogram demonstrated signal indicative of abnormal vasculature in the basal ganglia bilaterally.

During the last 4 months of follow-up, the patient developed new bifrontal headaches. She remained neurologically unchanged, and a CT scan revealed no hemorrhages or infarcts. A follow-up angiogram was obtained as part of this investigation of new headaches. The study demonstrated almost complete regression of the AVM in both the right and left basal ganglia. There was no evidence of mass effect, hemorrhage, or early draining veins (Fig. 2).

Discussion

The natural history of AVMs includes enlargement and hemorrhage but rarely regression [3–10]. Factors predisposing an AVM to regression by thrombosis are most likely those that affect the hemodynamic states of the AVM. These include the vascular complexity of the AVM, the surgical manipulation of the lesion, or the compression of the AVM by surrounding mass lesions [2–4].

The bilateral location of the AVMs in this case is unusual. Cases of multiple cerebral AVMs in a single patient are rare, and Nakayama et al. [11], in a review of the literature, found 13 such cases, of which seven had bilateral representation but none was situated bilaterally in the basal ganglia. Willinsky et al. [12] found multiple brain AVMs in 18 of 203 patients with AVMs, of which 10 had no history or stigmata of Osler-Weber-Rendu or Wyburn-Mason syndrome. Our patient had no evidence of either of these genetic disorders.

In their study of 20 patients with AVMs, Minakawa et al. [4] found that smaller AVMs with single feeders had a greater propensity to thrombose and regress while larger AVMs with multiple arterial feeders tended to increase in size. Superficially located AVMs also had a greater tendency toward regression than did the deep AVMs [4]. In no series has there been a description of bilateral basal ganglia AVMs that have regressed.

It is generally accepted that hemorrhage can cause thrombosis of AVMs [1–4]. The mass effect of the blood clot may alter the dynamics of the AVM and decrease blood flow to the extent that thrombosis may occur. Gibb et al. [3] documented either hemorrhage or development of neurologic deficits in all patients whose AVM regressed, and believed this particularly true of patients with deep AVMs. Similarly, surgical intervention, including evacuation of a blood clot or placement of a shunt, has been associated with regression of AVMs. Again, a change in the dynamics of blood flow through the AVM may be anticipated as the surrounding brain environment is altered [4, 12].

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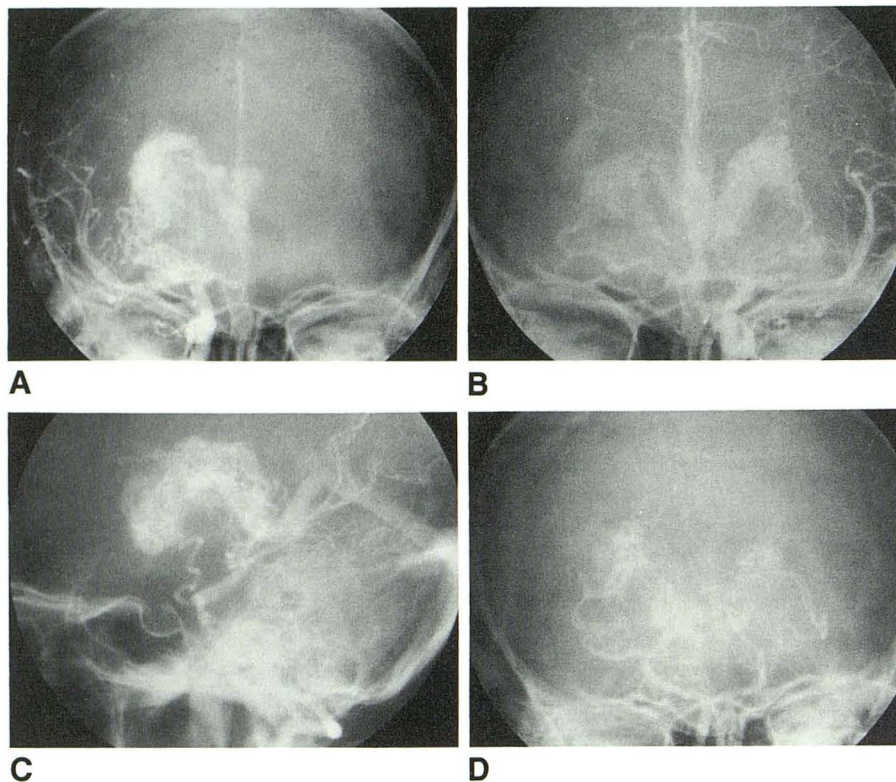


Fig. 1.—Case 1: Angiograms obtained 10 years prior to present admission.

A, Anteroposterior view, right internal carotid angiogram, shows markedly enlarged and tortuous lenticulostriate arteries feeding a large right basal ganglia arteriovenous malformation. A similar image was seen on the lateral view. Drainage is primarily into the deep cerebral veins.

B, Anteroposterior view, left internal carotid angiogram, shows both anterior cerebral arteries supplying pathologically enlarged lenticulostriate arteries bilaterally. These arteries pass directly into large arteriovenous malformation niduses, which drain into the deep cerebral veins.

C, Lateral view, left vertebral artery angiogram, shows unusually enlarged and tortuous thalamoperforate artery feeding inferior portion of arteriovenous malformation nidus. The remainder is supplied by enlarged lateral posterior choroidal vessels and other branches of the posterior cerebral arteries.

D, Anteroposterior half axial view, left vertebral artery angiogram, clearly demonstrates the feeding vessels and the arteriovenous malformation niduses.

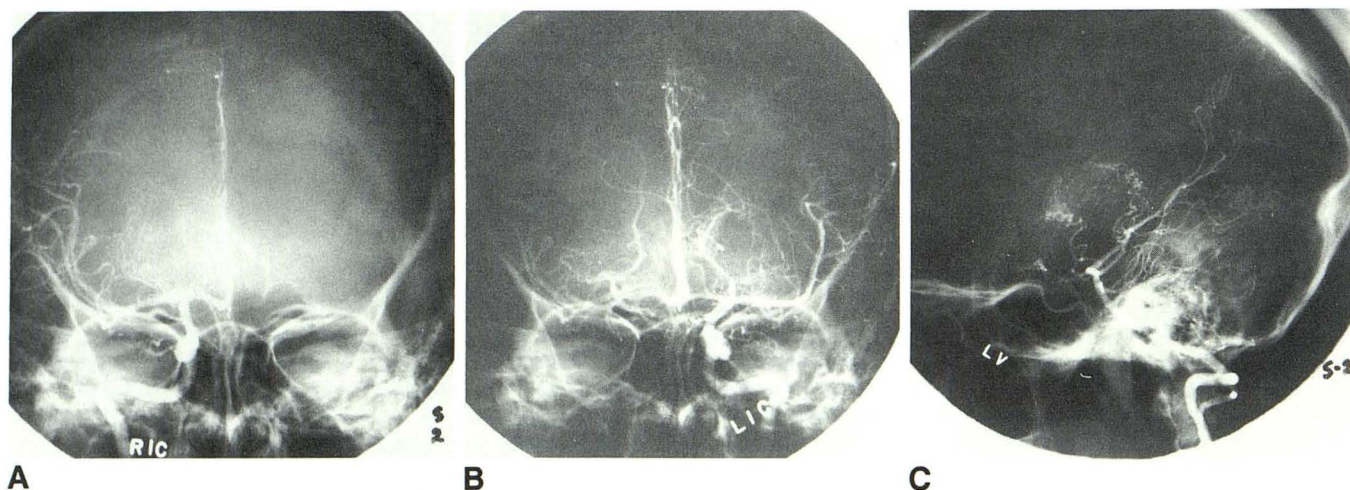


Fig. 2.—Case 1: Angiograms obtained 10 years after initial clinical presentation because of a change in headache pattern.

A, Right internal carotid angiogram shows no nidus or early draining vein, even with careful evaluation of the capillary phase (compared with Fig. 1A).

B, Left internal carotid artery angiogram again shows cross-filling of right carotid branches and opacification of both posterior cerebral arteries and a portion of the basilar artery. None of the arteriovenous malformation is visible (compare with Fig. 1B).

C, Left vertebral angiogram shows unusually tortuous and enlarged thalamoperforate and lateral posterior choroidal arterial branches. No early draining vein was visible throughout the filming.

In the 18 reported cases with angiographic documentation of AVM regression, six had undergone surgical intervention that included clot aspiration and partial excision of the AVM [3, 7]. Omojola et al. [10] reported a case in which a tumor was associated with the AVM. They postulated that the mass effect of the tumor altered blood flow through the vessels

with resultant thrombosis of the malformation. As spontaneous thrombosis occurs more frequently in patients over the age of 30, some have suggested atherosclerosis as a contributing factor [4, 13]. Dyck [2] described a case wherein a thrombosed AVM was discovered intraoperatively. There was no history of hemorrhage or previous surgery. Megison et al.

[5] angiographically documented the spontaneous resolution of an AVM without any history of hemorrhage, and Whitaker et al. [14] observed the spontaneous thrombosis of a vein of Galen aneurysm. These cases have similarities to our case.

In our patient there was no hydrocephalus, hemorrhage, or tumor documented on MR imaging studies, and she did not receive any surgical manipulation or radiation therapy. Thus, the reason for AVM regression is unclear. The finding that both AVMs receded suggests a diffuse process affecting the general cerebral circulation. Nevertheless, this case is unique because of the lack of any neurologic symptoms, the locations of these AVMs, their large size, and their spontaneous regression in the absence of hemorrhage or surgical intervention.

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