Pial Arteriovenous Fistula: A Brief Review and Report of 14 Surgically Treated Cases

Atul Goel, Sonal Jain, Abhidha Shah, Survendra Rai, Sandeep Gore, Pralhad Dharurkar

■ OBJECTIVE: **The authors report their successful experience of treating 14 cases of pial arteriovenous fistula (PAVF) by direct surgery.**

■ METHODS: **During the period January 2010 to April 2017, 14 patients with PAVF were treated by surgery. Only those patients were selected who had a single arterial feeding channel. There were 9 male patients and 5 female patients, and their ages ranged from 5 to 53 years (average, 19 years). Ten patients were younger than 20 years of age. Five patients presented clinical and radiologic features that suggested hemorrhage from the PAVF. Ten patients had seizures. Two patients had hemispheric symptoms or neurologic deficits at the time of presentation. In 12 patients, there were no gross neurologic deficits. The diagnosis was made on the basis of digital subtraction angiography in all patients and computed tomographic angiography in 8 patients. Angiography revealed that the PAVFs in 8 patients were supplied by the middle cerebral artery, in 5 patients by the anterior cerebral artery, and in 1 patient by branches of the basilar artery. Surgical procedures involved identification of the site of fistula, obliteration of the feeding artery, and resection of the entire venous varix.**

■ RESULTS: **The PAVF was successfully excluded from circulation in all patients. There were no neurologic deficits related to the surgical procedure.**

■ CONCLUSIONS: **Direct surgical resection of the entire PAVF is a safe, effective, and probably curative method of treatment.**

INTRODUCTION

Pial arteriovenous fistula (PAVF) is a discrete clinical entity that differs from arteriovenous malformations (AVM) and from dural arteriovenous fistula (DAVF) in morphologic, clinical, radiologic, and management issues. PAVFs are relatively rare and constitute approximately 1.6% of all vascular malformations.[1-5] Fewer than 160 cases have been reported in the literature. A single and sometimes more than 1 arterial feeding vessel and a large and lobulated venous varix or venous aneurysmal formation characterize PAVF. Our experience suggests that the surgical treatment of PAVF is safe and relatively easy when compared with both AVMs and DAVFs, and the outcome is gratifying. We discuss this entity and detail our experience.

METHODS

During the period January 2010 to April 2017, we surgically treated 14 patients with PAVF (**Figures 1–6**). There were 9 male patients and 5 female patients in the series. Their ages ranged from 5 to 53 years, the average being 19 years. Ten patients were younger than 20 years in age. The clinical presenting features are elaborated in **Table 1**. Five patients presented with an acute clinical event or ictus, and the imaging suggested hemorrhage from the PAVF. Digital subtraction angiography was done in all cases. CT angiography that included 3-dimensional (3D) reconstruction was possible in the later part of the series in 8 patients. In 1 patient a 3D printed model showing the arteriovenous fistula was made (**Figure 5C**). The location of the PAVF is detailed in **Table 1**. PAVF drained into the superior sagittal sinus in 12 patients, into both the superior and inferior sagittal sinus in 1 patient, and into the deep venous system in 2 patients (**Figures 1–6**).

Key words
- Arteriovenous malformation
- Dural arteriovenous fistula
- Pial arteriovenous fistula
- Venous varix

Abbreviations and Acronyms
3D: 3-dimensional
AVM: Arteriovenous malformation
DAVF: Dural arteriovenous fistula
PAVF: Pial arteriovenous fistula

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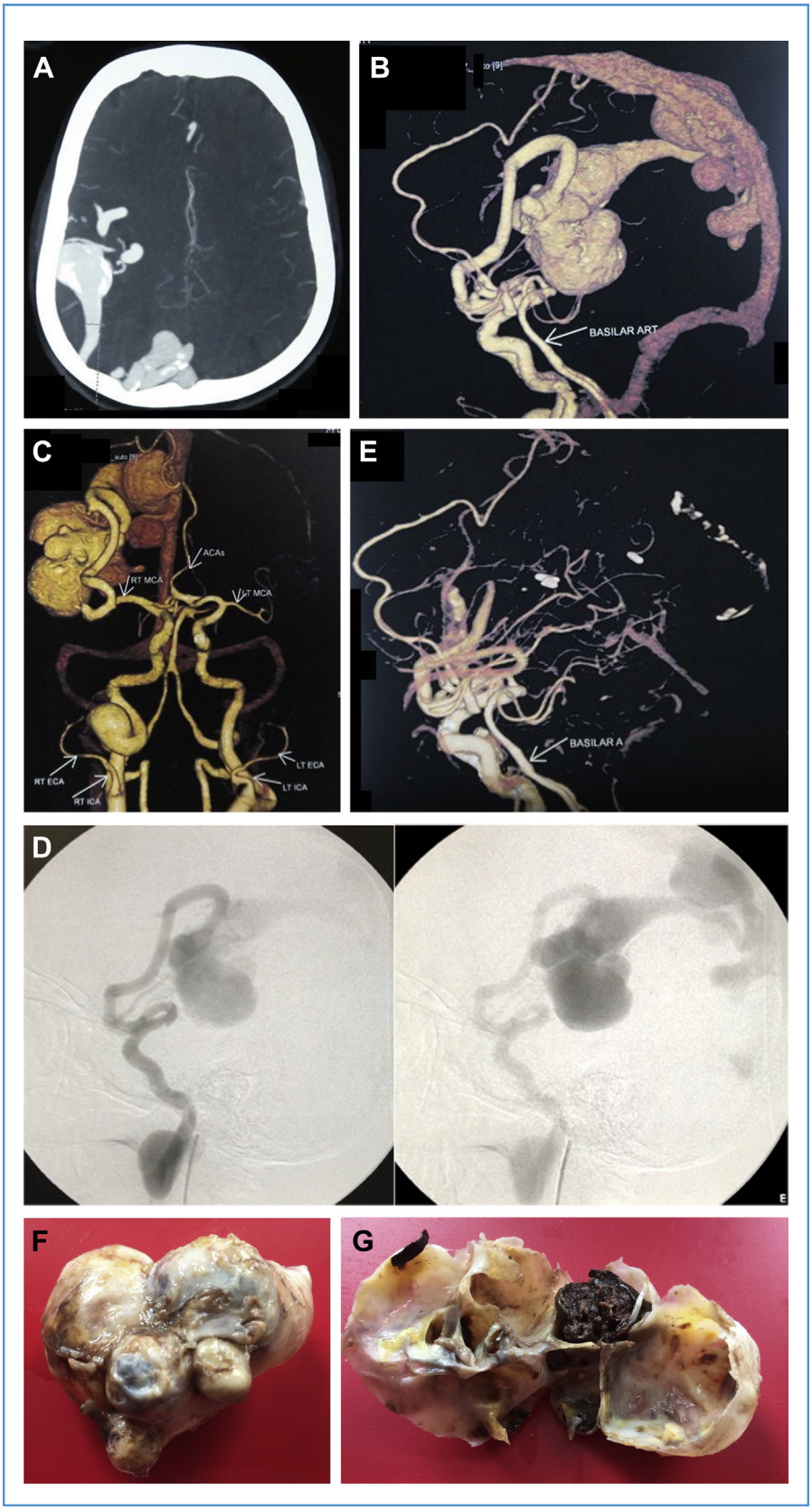
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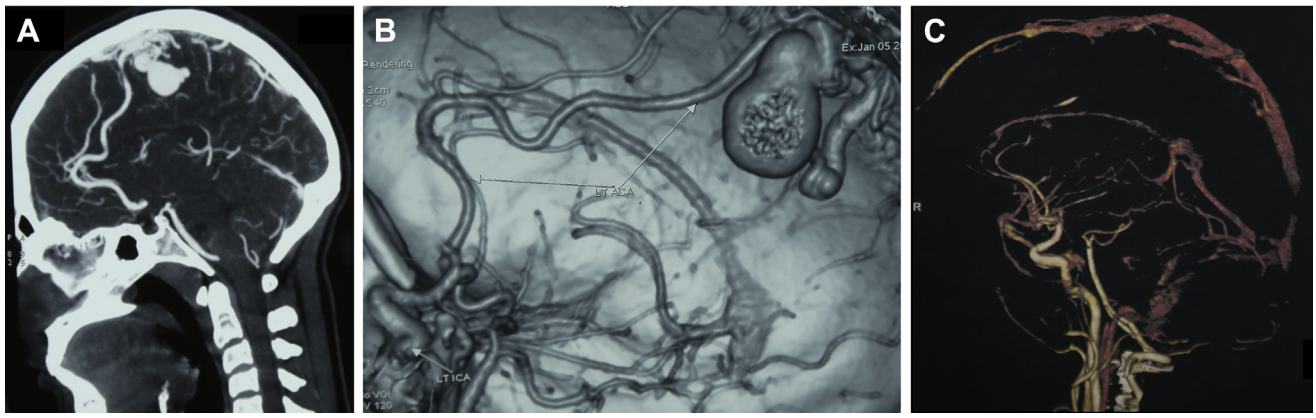

Figure 2. Images of an 18-year-old woman (case 2). (**A**) Sagittal image of computed tomographic (CT) angiogram showing a pial arteriovenous fistula (PAVF) fed by the anterior cerebral artery and draining into the superior sagittal sinus. (**B**) 3-dimensional reconstructed image showing the PAVF. (**C**) Postoperative CT angiogram showing excision of the PAVF.

Surgery

The surgical procedures were adopted according to general principles of treating a large AVM[6] and involved wide and circumferential exposure of the PAVF. The main and critical issue was to identify the feeding arterial vessel early in the operation. In general, such a procedure can be relatively straightforward because this vessel stands out from the adjoining normal vascular tree in size, girth and thickness, and configuration. Although not used, neuronavigation assistance can be of help to locate the feeding artery. Isolation of the feeding artery, coagulation with or without proximal control, and sectioning formed a major step in the surgical procedure. The venous varix collapsed instantly after the feeding artery was blocked. The venous varix or aneurysm with a well-defined plain of dissection was then dissected off the adjoining cerebral parenchyma. The dissection of the venous aneurysm was remarkably simpler when compared with dissection, isolation, and resection of the AVM nidus. The large venous structure was isolated and sectioned close to the final draining site by coagulation/thread or clip ligation.

RESULTS

The clinical outcome after the surgical resection was satisfactory, and the PAVF was excluded completely from the circulation in all patients. There was no neurologic worsening in any patient. The follow-up times ranged from 4 to 92 months (average, 32 months). There were no delayed neurologic symptoms or events related to PAVF in any patient. Two patients who had neurologic deficits reported improvement in their symptoms at their follow-up visits. During the follow-up period, none of the patients had any focal or generalized seizures.

DISCUSSION

Lasjaunias et al.[7,8] and Weon et al.[9] recently identified pial AVF as a distinct clinical entity. Pial AVFs are relatively rare and account for approximately 1.6% of all vascular malformations.[10] During the same period of study, we surgically treated 170 cases of AVMs. In large AVMs, in general, there are multiple feeding arteries, nidus, and multiple draining veins. The nidus of the AVM is a "packed" malformation of small, medium, and large blood vessels. Flow-related arterial aneurysms along the large feeding arterial channel are frequent. Aneurysmal dilatation of blood vessels within the nidus is common, more particularly in AVMs with high-volume and high-intensity blood flow and with large nidus. The general understanding of PAVFs is that these vascular malformations are single-channel arteriovenous fistulas with 1 feeding artery and large, aneurysm-like dilatated or multiloculated venous drainage, without the presence of nidus. The large ectatic venous dilatation or venous varices may be in the form of multiple pouches resembling a nidus with intranidal aneurysms. The nidus in such a situation is a "false" nidus. In patients with more than 1 feeding artery and large draining venous channels, it is sometimes difficult to

Figure 1. Images of a 53-year-old woman (case 1). (**A**) Axial computed tomographic (CT) angiographic image showing the parietal pial arteriovenous fistula (PAVF). (**B**) 3-dimensional (3D) reconstructed sagittal CT angiographic image showing the large PAVF fed by the right middle cerebral artery. Note the large venous varices draining into the superior sagittal sinus. (**C**) 3D reconstructed coronal image showing the formidable-looking PAVF. Note the dilated right internal carotid artery. (**D**) Catheter angiogram showing the PAVF with the venous varix. (**E**) Postoperative CT angiogram showing excision of the PAVF and the venous varices. Also note that the circulation in the adjoining cerebral blood vessels is more voluminous and rapid when compared with the circulation before operation. (**F**) Postoperative specimen of the PAVF showing multiple daughter lobules. (**G**) Pathologic specimen showing the venous varix, which has several thin-walled and thick-walled loculations. Thrombus within a portion of the varix can be seen.

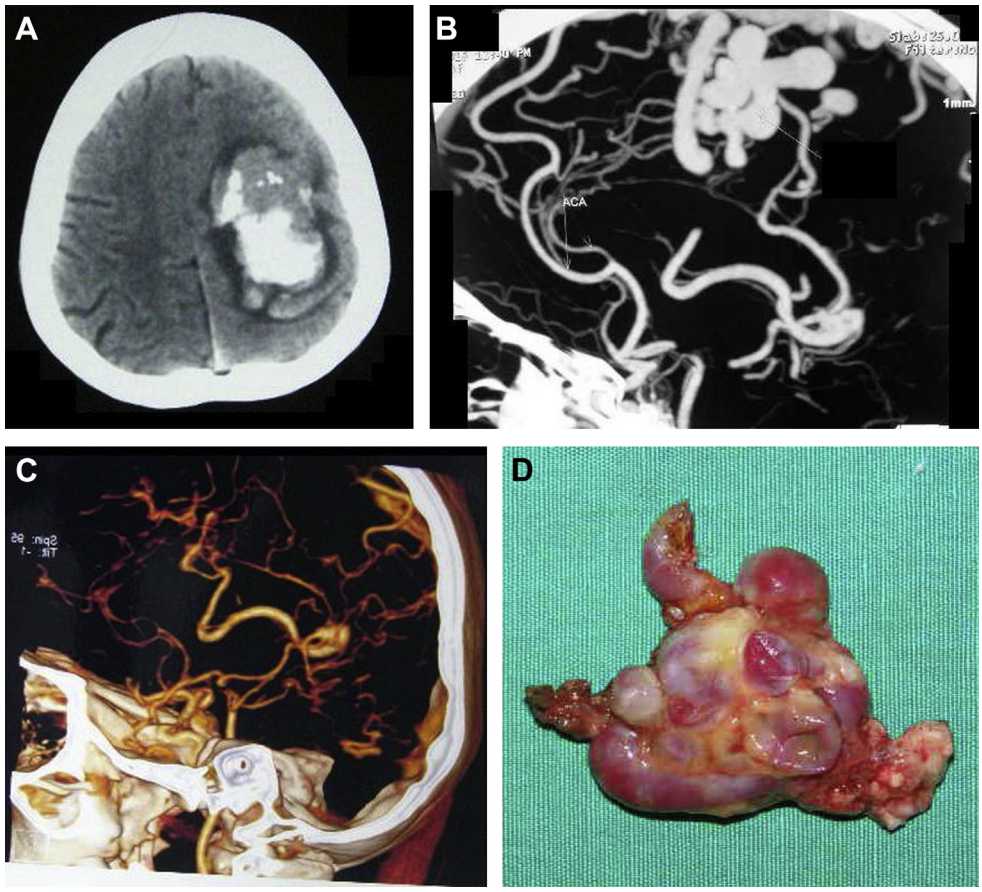

Figure 3. Images of a 15-year-old adolescent boy (case 3). (**A**) Axial computed tomographic (CT) image showing a large intraparenchymal clot in the left frontoparietal region. (**B**) Sagittal image of CT angiogram showing the left frontoparietal pial arteriovenous fistula (PAVF) fed by the anterior cerebral artery and draining into the superior sagittal sinus and the Galenic venous system. (**C**) Postoperative CT angiogram showing excision of the PAVF. (**D**) Excised specimen showing the feeding artery and the dilatated venous channels.

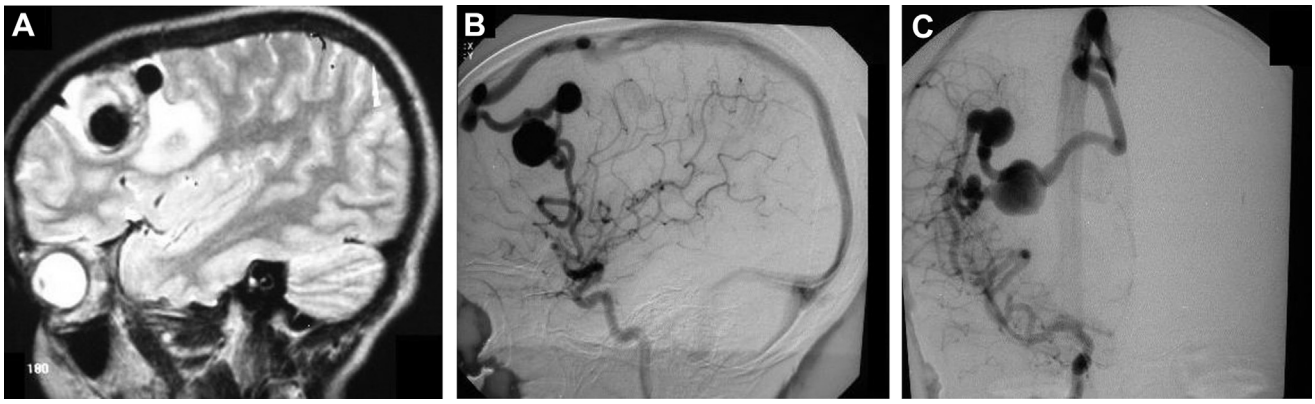

Figure 4. Images of a 16-year-old adolescent girl (case 4). (**A**) Sagittal T2-weighted image showing a large venous varix with perilesional edema. (**B**) Lateral view of catheter angiogram showing the pial arteriovenous fistula (PAVF) being fed by the right middle cerebral artery and draining into the superior sagittal sinus. (**C**) Anteroposterior view of catheter angiogram showing the fistula.

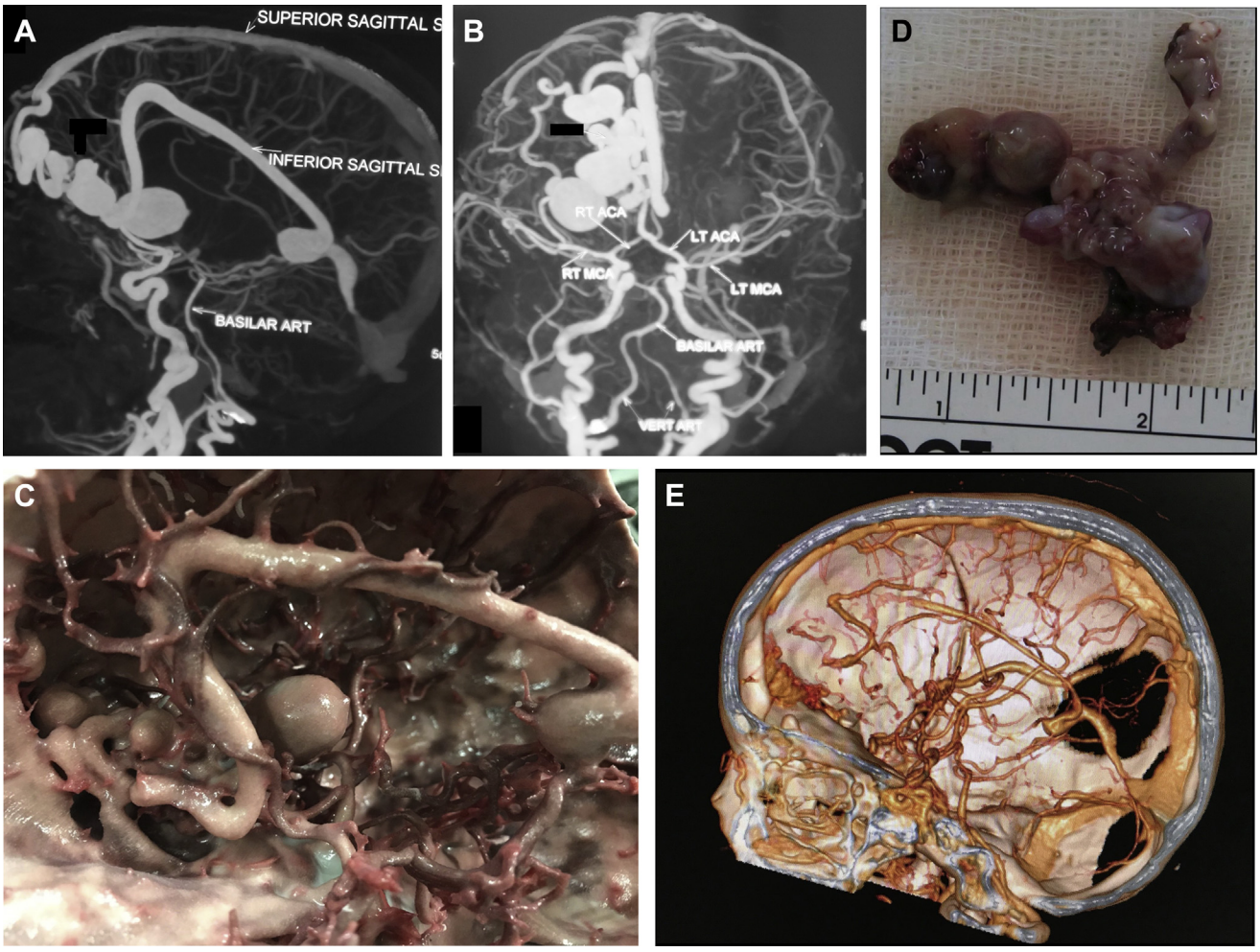

Figure 5. Images of a 11-year-old boy (case 5). (**A**) Sagittal image of a 3-dimensional (3D) reconstructed computed tomographic (CT) angiogram showing a large multiloculated pial arteriovenous fistula fed by the anterior cerebral artery and draining into both the superior and the inferior sagittal sinus. (**B**) Coronal image showing the arteriovenous fistula being fed by the A2 segment of the right anterior cerebral artery. (**C**) 3D printed model showing the location of the arteriovenous fistula. (**D**) Postoperative specimen showing the "false nidus" of the arteriovenous fistula. (**E**) Postoperative CT angiogram showing removal of the arteriovenous fistula.

differentiate a PAVF from a regular AVM. To avoid confusion, we have included only single-channel PAVFs or those that were fed by 1 large feeding artery. Whereas DAVFs are fed by meningeal blood vessels, major branches of the cerebral blood vessels feed PAVFs. Unlike a DAVF, the location of a PAVF is not within the dural leaves, but like an AVM it is located in the brain parenchyma. The characteristic feature is the presence of a large arterial feeder and a giant aneurysm-like dilatation of the venous draining vessels. As per the definition, in PAVF, there is a fistulous connection of the feeding artery with the venous drainage at a single "point," as is seen in carotiocavernous fistulas. The unnatural nature of blood flow leads to progressive dilatation of both the feeding artery and the venous draining compartments. Abnormal hemodynamic stress on the walls of the blood vessels may induce chronic endothelial alterations that induce increased endothelial permeability, intimal fibrous proliferation, and destruction of internal elastic lamina.[11]

The exact pathophysiologic mechanism of formation of PAVFs is not known.[7,9,12-16] Although AVMs have been speculated to have an embryogenic origin; PAVFs can even be acquired and related to trauma or to previous surgery. Hoh et al.[1] speculated that there is a possible misstep in the embryologic cerebrovascular development that produces these lesions. Lasjaunias et al.[7,8] and Weon et al.[9] proposed that structural congenital alterations are primarily responsible for vascular remodeling processes, affecting the endothelial cells at the venous side of the capillaries. PAVF can present as acute or chronic headache,[17,18] seizures,[19,20] focal neurologic deficits,[18,21,22] hemorrhage,[23,24] and rarely in neonates and infants with cardiac failure.[25] Enlarging head circumference, cranial erosions, and symptoms of increased intracranial pressure are among the possible presenting features.[9,22] They

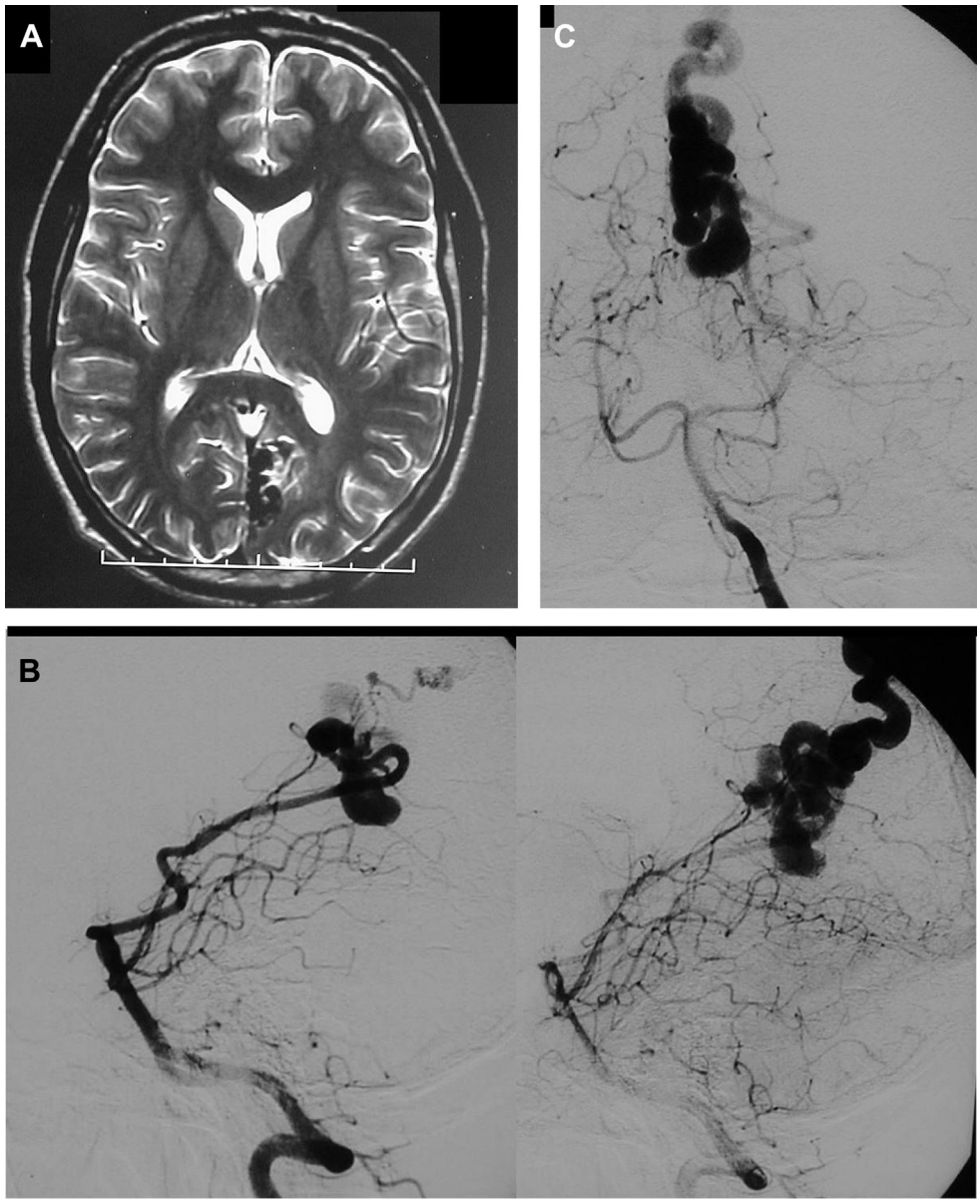

Figure 6. Images of a 19-year-old man (case 6). (**A**) T2-weighted axial magnetic resonance image showing a pial arteriovenous fistula in the left medial occipital region. (**B**) Catheter angiogram showing the fistula being fed by the left posterior cerebral artery and draining into the superior sagittal sinus. (**C**) Anteroposterior image of the fistula.

have also been identified as a part of syndromes such as Rendu-Osler-Weber or Ehler Danlos syndromes.[20,22,26,27]

The abnormality of the blood flow from artery to vein without the intervening "speed-buffer" or "speed breakers" provided by the capillaries subjects them to the risk of hemorrhage. Although it cannot be clearly hypothesized, the site of abnormal fistulous connection can be between the artery and the vein or may be between the artery and a capillary or an arteriole that subsequently dilates. In the long term, the absence of innumerable capillary channels that provide resistance and avoid quick drainoff of blood, the passage of blood from artery to vein becomes direct and with a low level of resistance. These events make the arteries progressively larger than normal and cause them to lose their muscular strength and become flabby and weak. The veins now function to accommodate the excessive amount of blood that should have been otherwise dispersed in the capillary system and to become large and ectatic in size. This leads to a progressively increasing high-blood volume flow through the fistula, contributing to

Table 1. Clinical and Radiologic Features of the Patients

Patient No.	Sex/Age (Years)	Presenting Symptoms	Neurologic Findings	Location	Radiologic Findings	
					Arterial Feeder	Venous Drainage
1	Female/53 (Figure 1)	Headache and multiple episodes of seizures	No deficits	Right temporoparietal	Right middle cerebral artery	Superior sagittal sinus
2	Female/18 (Figure 2)	Right-sided focal seizures	Right ankle dorsiflexion and plantar flexion 2/5	Left medial frontal	Left pericallosal artery	Superior sagittal sinus
3	Male/15 (Figure 3)	Focal convulsions of right-sided limbs and right-sided limb weakness	Right hemiparesis, grade 3/5	Left posterior frontal	Left middle cerebral artery	Superior sagittal sinus and deep venous system
4	Female/16 (Figure 4)	Headache and generalized tonic-clonic seizures	No deficits	Right frontal	Right middle cerebral artery	Superior sagittal sinus
5	Male/11 (Figure 5)	Episode of seizure with loss of consciousness and headache	No deficits	Right subfrontal region	A2 segment of right anterior cerebral artery	Superior sagittal sinus and inferior sagittal sinus
6	Male/19 (Figure 6)	Multiple episodes of seizures	No deficits	Left medial occipital	Left posterior cerebral artery	Superior sagittal sinus
7	Male/5	Episode of loss of consciousness	No deficits	Left posterior frontal	Left middle cerebral artery	Superior sagittal sinus
8	Male/21	Headache, loss of consciousness with altered sensorium	No deficits	Left posterior frontal	Middle cerebral artery	Superior sagittal sinus
9	Female/17	Headache and focal convulsions	No deficits	Right posterior frontal	Right pericallosalaretry	Superior sagittal sinus
10	Male/25	Generalized tonic-clonic seizures	No deficits	Left temporal	Left middle cerebral aretry	Superior sagittal sinus
11	Male/19	Headache and multiple episodes of seizures	No deficits	Right medial frontal	Right pericallosal artery	Superior sagittal sinus
12	Female/30	Episode of loss of consciousness	No deficits	Left frontal	Left middle cerebral artery	Superior sagittal sinus
13	Male/13	Episodes of seizures	No deficits	Left parietal	Left pericallosal artery	Superior sagittal sinus
14	Male/8	Headache and 1 episode of focal convulsion	No deficits	Left parietal	Left middle cerebral artery	Straight sinus

increasing sizes of both the feeding artery and the vein. Owing to the limited energy required for the blood flow over long periods of time, the arteries and veins lose their turgor and become enlarged, The veins dilate more than the artery in the process. Essentially, the volume of flow of blood is higher, and the intensity or pressure of the blood flow is less than normal arterial flow but higher than normal venous flow. The flow dynamics appear to be different from that of DAVF, wherein the dural arteries struggle to find adequate venous drainage. In DAVF, the arteries are larger than normal, and the pressure of flow of blood is also significantly higher than the normal arterial blood flow. From the morphologic assessment and in the absence of capillary blood flow, it does not appear that PAVFs have any functional role in vascular feeding of the cerebral parenchyma. Because of low perfusion in the normal brain tissue and intracranial venous congestion, these lesions may also affect normal brain development and cause cognitive impairment.[7] Such clinical events are more commonly seen in infants or young adults. Direct arterial pulsations and formation of a giant venous varix can cause focal neurologic deficits or seizures from mass effect. In general, PAVFs in younger patients present with symptoms related to the blood shunting effect, whereas older patients present with symptoms related to mass effect or those related to hemorrhage.[15] Unfortunately, in our series, no neurophysiologic monitoring or assessment of perfusion pressures was done during the surgical procedure.

The exact pathogenesis of PAVF is unclear. Eight patients in our series were in the pediatric age group. Some authors identify the role of error in the embryologic development in the pathogenesis of PAVF. The natural history of PAVF is unclear and is not known because of the rarity of these lesions. However, the general impression from published reports is that the clinical course is significantly worse than that of AVM. Some authors report that the

mortality rate after conservative treatment can be above 60%.[28] In 1 report, 5 of the 8 conservatively treated patients died.[29] In general, aggressive treatment has been recommended.[9,30] Review of the literature suggests that approximately 65% of patients with untreated PAVF succumb to disease and its hemorrhagic consequence.[1] Spontaneous thrombosis of the pial AVF is also known.[31,32] Thrombosis of a portion of a multiloculated venous varix was observed in 1 of our patients (**Figure 1G**). The exact weak point in the fistula that can lead to hemorrhage is unclear. However, as seen in **Figure 1G**, the venous varix has several thin-walled and thick-walled loculations that could be prone to rupture by a persistent and unusual blood flow. The natural history and growth of the size of venous varix has not been documented over the period of clinical observation.

In general, a multidisciplinary approach has been identified to be essential for the treatment of AVMs in general and PAVFs is particular. Angiography of PAVFs presents a dangerous or formidable appearance for surgical maneuvering, and "safer" endovascular procedures are generally preferred. The majority of current reports describe the adoption of an endovascular form of treatment. However, surgery on PAVFs is remarkably simpler than surgery on large AVMs or DAVFs. Whereas surgery on an AVM involves painstaking circumferential dissection and resection of the nidus, surgery on a PAVF essentially involves disconnection of the fistula by blocking the arterial feeding vessel at the point of fistulous connection. Although disconnection of the fistula by ligating/clipping of the feeding artery is a recognized method of treatment, the need for resection of the venous varices, like resection of an AVM nidus, is a debated issue. However, the venous varix was resected in all our patients. The resection of a venous varix is a straightforward surgical exercise because there is a well-defined and avascular plain of dissection. A review of literature reports from 1977 to 2009 indicated that the success rate of surgical treatment (96.8%) was comparatively higher than the rate for endovascular treatment (86.5%).[15]

Disconnection of the arteriovenous shunt, either surgically or by endovascular techniques, represents the possible modes of treatment. Obliterating the venous drainage or packing off of venous varices by endovascular techniques that involve use of glue, coils, balloons, and similar materials are other modes of treatment that are under evaluation. However, high blood flow in the PAVF has been associated with migration of the embolic material into the major sinuses and heart and even into other regions of the brain. Considering that surgery is relatively straightforward, it forms the primary and accepted form of treatment. Embolization of the PAVF supplied by deep located feeding vessels or draining veins could be considered as treatment. All the patients in our series underwent a successful resection of the entire PAVF. Some authors have identified that sudden occlusion of the PAVF during surgery or by embolization can lead to alteration of the cerebral blood flow dynamics that can be a cause of intracerebral hemorrhage, a phenomenon that is similar to "normal-pressure breakthrough" recognized after surgery on large AVMs. Such an event was not encountered in our series.[33,34]

Although it cannot be convincingly appreciated, resection of the PAVF normalized the blood flow in the adjoining cerebral arteries (**Figure 1E**). The role of radiation treatment has not been conclusively clarified.

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