Pial Arteriovenous Fistula Occurring Secondary to Chronic Cerebral Venous Sinus Thrombosis

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Abstract

An arteriovenous fistula (AVF) involving pial circulation is a rare pathology. It comprises of a single or multiple arteries draining into the venous channels without a nidus as in AV malformations. These lesions make 1.6% of all intracranial vascular abnormalities. Unlike dural AVFs, 80% of pial AVFs are supratentorial and typically lie adjacent to the ventricular ependyma or just beneath the brain surface. This report describes the occurrence of pial AVF, predominantly in the region of prepontine cistern, secondary to chronic cerebral vein thrombosis. Pial AVFs are rare lesions however they do exist and one should be wary with adequate knowledge of imaging findings and clinical picture to make the diagnosis in time. Diagnosis at the earliest is of utmost importance to save the patient from the devastating complications which are part of the natural course of the disease.

Keywords: Pial Arteriovenos Fistula; Cerebral Vein Thrombosis; Vascular Lesion


Introduction

An arteriovenous fistula (AVF) involving the pial vasculature is an extremely rare intracranial vascular pathology accounting for only 1.6% of intracranial vascular malformations from 1977-2009 [1]. It consists of single or multiple arteries directly draining into a vein without an intermediate nidus of supply, the latter being diagnostic of an arteriovenous malformation (AVM). A fistula between pial arteries and cortical veins is usually congenital in origin. Acquired pial fistulae are very rare. Pial AVFs are different from dural AVFs in terms of blood supply i.e., they take their blood supply from pial or cortical arteries and are located outside the dural leaflets. These lesions should be identified at the earliest and treated immediately to prevent from the high mortality (with a rate of 63%) if left untreated or from the complications which are a natural part of the disease [2]. We report an acquired pial arteriovenous fistula developing after a cortical vein thrombosis.

Case Report

A 55-year-old gentleman presented with a slow growing left parotid swelling and a suspicion of space occupying lesion in brain. MRI brain showed partial re-canalization of the superior sagittal sinus due to chronic cerebral venous sinus thrombosis resulting in diffuse bilateral pial venous collateral formation which is predominantly concentrated in the region of prepontine cistern. However no acute or chronic established infarction or intracranial hemorrhage

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was seen. Chronic deep white matter ischemic changes were identified in bilateral centrum semiovale. No diffusion restricted infarction was noted. There was no hydrocephalus, mass effect or shift of mid line structures.

Consequent MR Angiography revealed multiple enlarged vessels throughout brain in the CSF spaces (Figure 1) and diffuses bilateral pial venous collateral formation which is predominantly concentrated in the region of prepontine cistern (Figure 2). Diagnostic angiography was performed which showed complete occlusion of superior sagittal sinus, straight sinus and the Torcula (Confluence of sinuses). Complete occlusion left transverse, left sigmoid sinus and proximal right transverse sinus was also noted. Numerous abnormally dilated cortical veins were noted draining through right transverse sinus. A dural AV fistula was also seen in the left temporal region with both left internal and external carotid supply. It had a nidus measuring 13 x 10 mm which was draining into multiple cortical veins (Figure 3).

**Figure 1:** MRI Brain; Axial T1 (a) T2 (b) and Post Contrast T1 (c) images showing multiple enlarged vessels throughout brain in the CSF spaces.

**Figure 2:** MRI Brain; Midline Sagittal T2 (a) and Post contrast T1 (b) section show diffuse pial collateral venous channels predominantly in the pre-pontine cistern, there is Superior Sagittal sinus thrombosis seen as loss of normal flow void (a) and filling defects (b).
boy who presented to the Emergency Room with sudden onset headache, vomiting, and brief loss of consciousness. Computed tomography (CT) scan of the brain showed diffuse subarachnoid hemorrhage and digital subtraction angiogram showed infratentorial pial AVF with arterial feeder from the left posterior cerebral artery and enlarged venous varices draining into vertebral venous plexus.

Another case was reported in year 2007 of a 33-year-old woman who presented with a rare intracranial pial arteriovenous fistula manifesting as monoparesis and hypesthesia of the right lower extremity. Computed tomography demonstrated an approximately 10-mm diameter subcortical hematoma in the left postcentral gyrus. Two months after suffering the ictus, angiography demonstrated a pial arteriovenous fistula in the late arterial phase fed by the left paracentral artery and drained into the left precentral vein. The authors suggest that pial arteriovenous fistula causing mild symptoms should be treated by flow disconnection because the direct arteriovenous shunt and attendant high blood flow usually results in huge venous varices. To determine whether direct surgery or endovascular treatment is appropriate, the position and shape of the lesion must be known [8]. The unique presentation in our case was, however, was the occurrence of a pial arteriovenous fistula secondary to chronic cerebral venous sinus thrombosis.

Conclusion

An acquired arteriovenous fistula involving pial circulation is a rare entity and its occurrence secondary to chronic venous sinus thrombosis is even rarer. Early diagnosis is essential for prompt treatment and improved outcome.

References


