"Parenchymal Arteriovenous Malformation In Parietal Lobe Presenting With Orbital Symptoms."

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Intracranial parenchymal arterio-venous malformations are the commonly encountered lesions in either sex. They frequently present at 20 to 40 years of age. About half of all arterio-venous malformations (AVMs) manifest with hemorrhage and 25% have seizures as the presenting symptom. The remainder have symptoms of mass effect, headaches, vascular steal phenomena or focal neurological deficit. The orbital symptoms like proptosis and visual deficit are one of the uncommon manifestations of intracranial parenchymal arterio-venous malformations. At times the orbital symptoms are the first clinical signs which points towards the possibility of an underlying intracranial arterio-venous malformation (AVM) [1].

Figure 1. 30 years man with a parenchymal AVM in left parietal lobe. (A) CECT shows enlarged and congested left cavernous sinus with lateral convexity. Few dilated venous channels can be visualized anterior to the left temporal lobe (straight arrow). Right sigmoid sinus is normal. (Curved arrow) (B) Arterial-phase CECT shows a parenchymal AVM in left parietal lobe with minimal perilesional mass effect. (C) Arterial-phase CECT (lower cut) shows multiple early-draining cortical veins draining the AVM, anteriorly and lateral to the left temporal lobe. Note the regurgitation of contrast into the left superior ophthalmic vein. (straight arrow).

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A 30-year-old man presented to ophthalmic outpatient department for symptoms of retro-orbital pain on the left side, since last 6 months. On clinical examination, there was mild proptosis with sclero-conjunctival congestion in left eye and ophthalmoscopy examination of the patient showed mild macular edema on the left side. The CT study of brain revealed a large lesion with intense post-contrast enhancement in the left parietal lobe. The left cavernous sinus was enlarged, congested and showed lateral convexity. [Figure 1A.] The cavernous sinus showed intense uniform enhancement and there was no filling defect within. The right cavernous sinus was normal. CT angiography was performed to further evaluate the lesion. Arterial-phase CECT showed a large arterio-venous malformation lesion in the left parietal lobe, involving the cortical-subcortical areas with minimal perilesional mass effect. [Figure 1B.] There were multiple early-draining cortical veins draining the AVM, anteriorly and laterally to the left temporal lobe. There was regurgitation of contrast into the left superior ophthalmic vein in the arterial-phase CT. [Figure 1C.] The parietal AVM had arterial feeders from the insular and opercular branches of left middle cerebral artery. Thus, the left cavernous sinus was a predominant route of venous drainage of this parietal lobe AVM and consequently the patient had presented with symptoms and clinical signs of unilateral orbital congestion.

The orbital drainage of parenchymal cerebral AVMs is rare. Volpe NJ et al, studied 100 adult patients with cerebral AVMs and found only three patients with orbital drainage; during a 4-year period [2]. Manifestations may include anterior visual pathway compression, dilated conjunctival veins, orbital congestion and asymmetrical disc swelling. Other uncommon mechanism of visual loss is ischemic optic atrophy associated with a steal phenomenon and direct compression of the right optic radiation. [3] Arteriovenous malformation in the left parieto-occipital area may manifest with symptoms of migraine, involving changes of visual fields indicative of impaired functions of symmetrical compartments of the other brain hemisphere. [4] At times, occipital dural arteriovenous malformation (AVM) can cause signs of a carotid-cavernous sinus fistula and consequent orbital congestion. Bilateral transverse sinus occlusion associated with the AVM produced these signs by rerouting intracranial venous drainage anteriorly through the cavernous sinuses and superior ophthalmic veins. [5]

In the present case, the parenchymal AVM in the parietal lobe did not cause any mass effect over the orbital apex or cavernous sinus. Apart from this, there was no dural sinus obstruction or thrombosis to cause re-routing of the intracranial venous drainage, anteriorly through the cavernous sinus. The orbital congestion developed only because the left cavernous sinus was a predominant route of the venous drainage for the AVM. To the best of our knowledge, none of the reported cases of parenchymal AVM in the literature had manifested with orbital symptoms, solely because the cavernous sinus was a predominant route of the venous drainage. The present case further illustrates the point that an intracranial parenchymal AVM in the parietal lobe can manifest with orbital symptoms.

REFERENCES: