Image Diagnosis: Encephalopathy Resulting from Dural Arteriovenous Fistula

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CASE REPORT

A 69-year-old woman presented to the Neurology Department with 2 months of progressive psychomotor slowing, inability to concentrate, and periods of disorientation. Her past medical history was unremarkable, and she was taking no medication. There was no history of trauma. On neurologic examination she was alert but taking a long time to answer, apathetic, distractable, and hypophonic with right visual and sensitive hemiextinction and left hemiparesis. Montreal Cognitive Assessment Exam score was 11/30. A computed tomography scan of the brain (Figure 1) showed possible convexity subarachnoid hemorrhage that the brain magnetic resonance imaging (MRI)/MRI angiography (Figures 2 and 3) revealed to be engorged cerebral vessels. Hyperintensity in the deep white matter of the cerebral hemispheres was also present. Cerebral angiography (Figure 4) revealed a dural arteriovenous fistula (DAVF) of the superior sagittal sinus and torcula (Cognard classification IIb).

The patient underwent endovascular embolization, with combined transarterial (n-butyl-cyanoacrylate) and transvenous (coils) approach, resulting in proximal occlusion of the superior sagittal sinus, torcula, and transverse sinus (Figure 5). Posttreatment angiography revealed near complete DAVF occlusion (Figure 6). Control MRI revealed a marked decrease of the deep white matter hyperintensity and no engorged cerebral veins (Figures 7 and 8). The patient’s mental status improved post procedure (Montreal Cognitive Assessment Exam score, 20/30), and she progressively came back to her baseline.
DISCUSSION

DAVFs are abnormal arteriovenous communications within the dura, usually located within the walls of a dural sinus or an adjacent cortical vein, and account for 10% to 15% of all intracranial arteriovenous lesions. The initiating events which lead to their development are not clear, but the literature reports association with trauma, infection, recent surgery, and dural sinus thrombosis. A wide variety of signs and symptoms, which can vary because of lesion location and pattern of venous drainage, may arise from DAVFs, namely, pulsatile tinnitus, ophthalmoplegia, proptosis, chemosis, retro-orbital pain, decreased visual acuity, seizures, Parkinsonism, cerebellar symptoms, apathy, and dementia.

CONCLUSION

Our patient with DAVF presented with encephalopathy with diffuse white matter changes related to venous ischemia. Her symptoms partially reverted with endovascular treatment. DAVF should be considered in patients with encephalopathy. This relatively nonspecific clinical picture may delay the diagnosis and result in further deterioration. A high level of suspicion should be maintained in patients who present in the context of unexplained intracranial hemorrhage without significant risk factors (trauma, hypertension, or anticoagulation) or in possible subarachnoid hemorrhage in a nonaneurysmal pattern. This should prompt imaging with MRI and angiography, which are the gold standard for diagnosis. DAVF recognition is essential because these patients are potentially treatable.

Disclosure Statement

The authors have no conflicts of interest to disclose.

How to Cite this Article


References