



Brush Sign in Cortical Venous Sinus Thrombosis

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Case

A 23-year-old male patient, with no previous comorbidities, was brought to the emergency department with complaints of headache for 2 days, which was moderate to severe intensity, diffuse in nature, throbbing type associated with few episodes of vomiting from day 1. The patient also developed mild transient weakness of the left upper limb and lower limb associated with the inability to speak and a mild decrease in comprehension, 4 hours before arriving at the hospital. There was a history of full spontaneous recovery of left-sided weakness and motor speech within 15 minutes. However, altered comprehension and sensorium persisted since then. Examination revealed mild sensory aphasia with no focal limb weakness, normal cranial nerve, and sensory loss examination. Examination of the fundus was suggestive of bilateral grade III papilledema.

Magnetic resonance imaging (MRI) of the brain revealed replacement of normal flow-related signal void in the lumen of superior sagittal sinus. There was also evidence of loss of normal flow void in the vein of Galen on the right side, straight sinus, and right transverse sinus and bilateral sigmoid sinuses by T1 hyperintense signal. On corresponding contrast-enhanced T1, T2, susceptibility weighted imaging (SWI), and MR venography sequences, there is also absence of contrast opacification of the described sinuses suggestive of venous sinus thrombosis. Multifocal filling defects are also seen in the distal segment

of the transverse sinus on the left side and proximal segment of the sigmoid sinus suggestive of intraluminal clots. Dilatation with blooming artifacts is seen in the superficial cortical veins of both the cerebral hemispheres due to congestion, *brush sign* (BS). Fluid-attenuated inversion recovery (FLAIR) hyperintensity is seen in the cortical sulcal spaces likely because of congested veins (►**Fig. 1**).

Venous system of the brain consists of multiple deep medullary venous channels draining the white matter of the cerebral hemispheres. These medullary veins drain into subependymal veins, along the lateral ventricles, and under normal physiological circumstances are difficult to depict on T1 and T2 MR sequences. Paramagnetic-sensitive MR sequences (SWI and T2*-weighted imaging [T2*WI]) allow better visualization of these veins, particularly in the conditions associated with high intravenous deoxyhemoglobin.^{1,2}

The **BS** manifests as an abnormal decrease in signal intensity of the subependymal and deep medullary veins on paramagnetic-sensitive MRI sequences seen in few patients of cerebral venous sinus thrombosis (CVST) and that too with a high thrombus load. An alternative explanation of the BS is the development of collateral venous pathways between the superficial and deep venous systems of the brain, seen on postcontrast images. Additionally, the BS has also been described in other pathological conditions, such as the moyamoya disease or Sturge-Weber syndrome.^{3,4}

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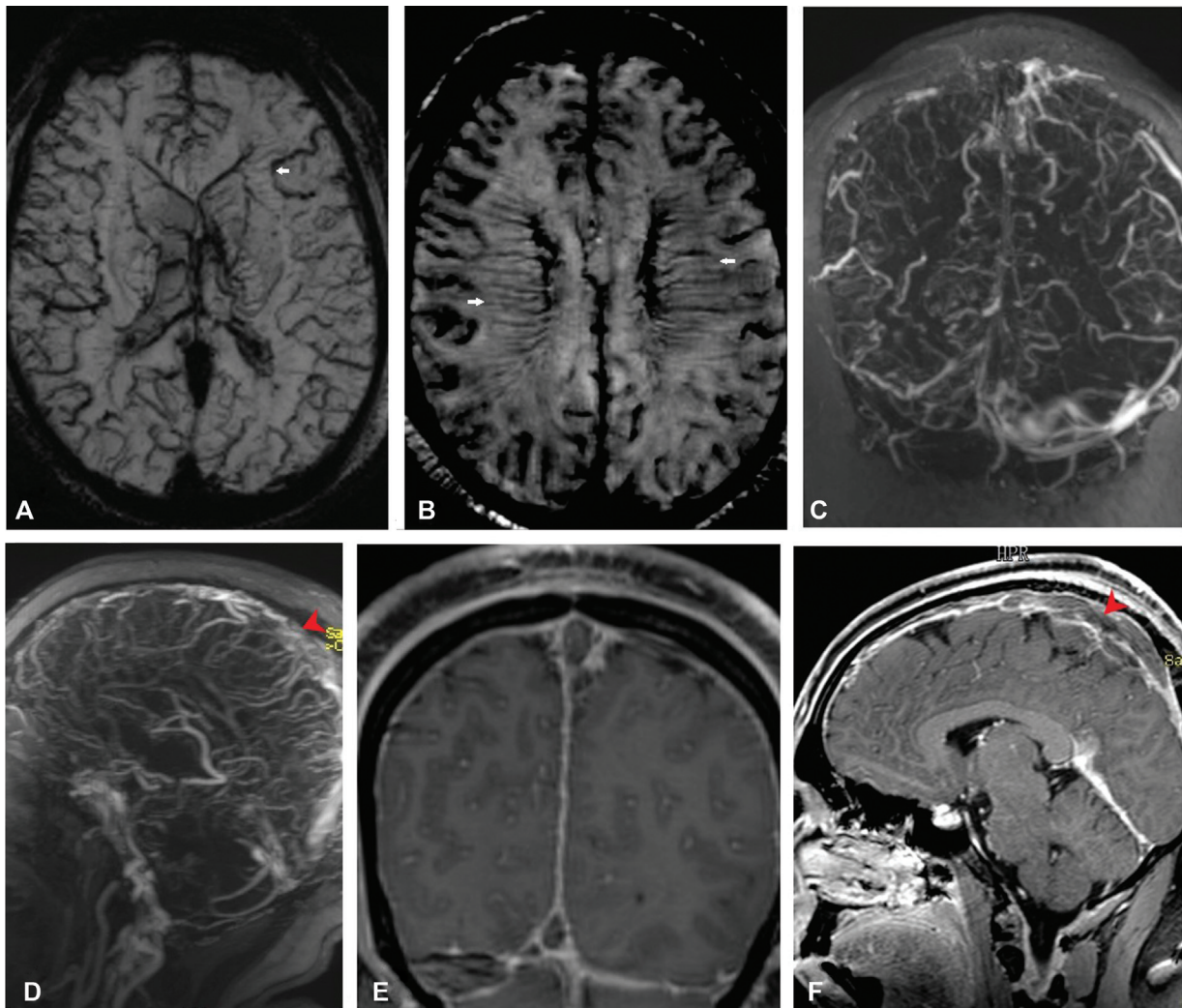


Fig. 1 (A, B) Axial gradient echo sequence (GRE) images show dilatation with blooming artifacts seen in the superficial cortical veins of both the cerebral hemispheres due to congestion (*small white arrows*), the brush sign. (C, D) Contrast magnetic resonance venography (MRV) coronal and sagittal images and (E, F) postgadolinium sagittal and coronal T1-weighted images show replacement of normal flow-related signal void and absence of contrast opacification of these segment of the dural venous sinuses suggestive of thrombosis involving superior sagittal sinus (*red pointed arrowheads*), torcular herophili, and right-sided transverse sigmoid sinus, proximal third of the left transverse sinus and straight sinus, and vein of Galen, also extending to the right internal jugular vein.

Although infrequent, follow-up imaging has shown that the BS usually disappears entirely, indicating that it is susceptible to rapid changes caused by improvements in tissue metabolic status.

Conflict of Interest
None declared.

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