Near Complete Resolution of Bilateral Thalamic Venous Infarct in the Absence of Superficial Venous Sinus Thrombosis

Case Description

A 45-year-old woman presented to the emergency department with a headache and progressive deterioration in sensorium since 2 days. On examination, her vitals were stable. Blood investigations were normal. Her Glasgow Coma Scale was 9/15. Bilateral plantar response was extensor. Magnetic resonance (MR) imaging of the brain demonstrated hypointense signals on T1 weighted images, hyperintense signals on T2 [Figure 1a white arrow], and fluid-attenuated inversion recovery (FLAIR) [Figure 1b white arrow] weighted images in bilateral thalamus. On diffusion weighted imaging (DWI), both the thalamus demonstrated isointense to hyperintense signals [Figure 1c white arrow] with corresponding hypointense signals on apparent diffusion coefficient imaging (ADC) [Figure 1d white arrow]. On susceptibility weighted imaging (SWI), blooming was evident in right thalamus [Figure 1e black arrow] and along the course of vein of Galen and straight sinus [Figure 1e white arrow]. On MR venogram, the vein of Galen and straight sinus was occluded [Figure 1f black arrow]. Superficial venous systems were patent. The imaging was consistent with the diagnosis of bilateral thalamic hemorrhagic venous infarct following occlusion of deep venous system. Her medical history was remarkable for multiple attacks of diarrhea before the onset of headache. On receiving anticoagulation, she showed gradual improvement in neurological status with near-complete recovery by 3 months. Investigations for coagulation disorder revealed normal protein C, protein S and antithrombin III levels. MR imaging (MRI) done at 3 months showed near complete reversal of signal changes in both the thalamic on T2 and FLAIR imaging [Figure 2a and b]. Few discrete bilateral thalamic signal intensity changes were seen on T2 weighted, FLAIR, and SWI imaging [Figure 2a-c white arrow].

Discussion

Isolated bilateral symmetrical infarction of the thalamus is an uncommon finding.\(^1\) It can occur following occlusion of either the artery of Percheron or deep venous system.\(^2\) Bilateral thalamic infarct following thrombosis of deep venous system in the absence of superficial venous sinus thrombosis is rarely reported.\(^3\) Thrombosis of vein of Galen or straight sinus results in venous hypertension and hemorrhagic venous infarct in both the thalami.\(^1\) This leads to combination of both vasogenic and cytotoxic edema to a varying extent in both the thalami.\(^3\) Vasogenic edema is evident by hyperintense signals on both T2, FLAIR and ADC sequences and isointense to hyperintense signals on DWI. Cytotoxic edema leads to high signal intensity on DWI and low signal intensity on ADC map.\(^3,4\) Advanced MRI sequences such as SWI and MR venogram demonstrates occlusion of the deep venous system thereby establishing correct diagnosis and optimizing early therapy to prevent permanent neurological deficit.\(^5\) The degree of neurological deficit is directly proportional to extent of cytotoxic edema and varies inversely to degree of vasogenic edema.\(^3\) Near complete reversal of signal changes in the present case on follow-up imaging is due to the resolution of vasogenic edema.
The overall neurological improvement following cerebral deep venous system occlusion depends on early diagnosis and extent of vasogenic edema on advanced MRI sequences and initiating early therapy.

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**Conflicts of interest**

There are no conflicts of interest.

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