Unilateral thalamic oedema secondary to venous sinus thrombosis

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DESCRIPTION

A man in his 40s presented with a 6-week history of fatigue, unsteadiness and difficulty with fine motor control of the left hand. His medical history included several years of right-sided otalgia and vertigo attributed to eustachian tube dysfunction, and one episode of Guillain-Barré syndrome. His body mass index was >35 and he was a heavy snorer.

The patient was alert and orientated, with no abnormalities on testing cranial nerves. In the limbs, there was pastpointing, dysdiadochokinesis and heel-shin ataxia on the left with impaired tandem walking, but there was no nystagmus, and strength and sensation were relatively preserved bilaterally. Both plantar responses were flexor.

A CT head showed a low-density region in the right thalamus, posterior limb of the internal capsule and adjacent lentiform nucleus (figure 1A). It extended to the upper brainstem, compressing the right third and right lateral ventricles, with a minor midline shift. On MR imaging, the lesion returned a high signal on T2-weighted images (figure 1B) with relatively unrestricted diffusion on DWI (diffusion-weighted imaging, figure 1C). There was also a low signal on T1-weighted images, with surrounding vasogenic oedema and enhancement with contrast (figure 1D), giving an initial impression of glioma or lymphoma. This was communicated to the patient and they were referred to the neuro-oncology multidisciplinary team.

However, on later review of MR spectroscopy images, the low creatine peak was not supportive of malignancy (figure 2A). The possibility of deep venous thrombosis was raised based on the spectroscopy data. A CT venogram confirmed non-filling of the straight sinus, the vein of Galen (both midline, singular veins) and the internal cerebral veins (figure 2B,C).

The patient was anticoagulated, first with heparin and then warfarin, and the presenting symptoms improved over the following months. A thrombophilia screen was negative at presentation and following withdrawal of warfarin therapy. Occasional difficulty with fine motor control of his left-hand remains the only ongoing symptom. A plain CT scan a year later showed only a minor low density in the right thalamus (figure 2D).

Thalamic oedema and/or infarction secondary to sinus thrombosis is usually bilateral,[1] due to the symmetry of venous drainage, but infrequently it is unilateral with reported cases suggesting a predisposition for left thalamic damage.[2] Initial imaging can suggest a glioma or intracerebral abscess,[3 4] and some patients have undergone biopsy before the diagnosis of venous thrombosis was considered.[5] Misdiagnosis can be distressing for patients and could lead to delayed and inappropriate management.

Despite a wide symptom spectrum including headaches, lateralised weakness and sensory loss, disorders of speech and semantic memory, patients often recover well from unilateral thalamic oedema following anticoagulation.[6] An apparent predilection for left thalamic infarction could be explained by the asymmetry in venous anatomy or a tendency for left thalamic infarction to produce more clinically obvious symptoms than right.[2]

Obstructive sleep apnoea is associated with hypercoagulability[7] and altered internal jugular vein blood flow,[8] which may contribute to cerebral thrombosis.[9 10] Interestingly, this patient's

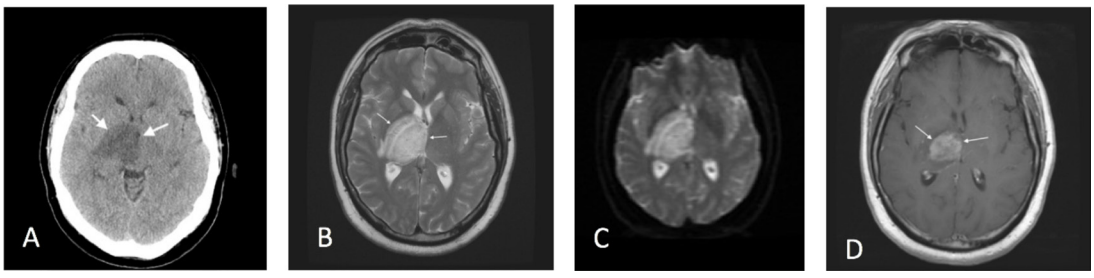

Figure 1 (A) Axial CT scan without contrast. There is low density and swelling in the right thalamus (arrows) compressing the third ventricle. (B) Axial T2-weighted MRI showing abnormal signal with mass effect in the right thalamus (arrows). (C) Apparent diffusion coefficient parametric map, showing relatively unrestricted diffusion in the right thalamus. (D) Postcontrast axial T1-weighted image showing bright enhancement in the right thalamus (arrows) indicating blood barrier breakdown.

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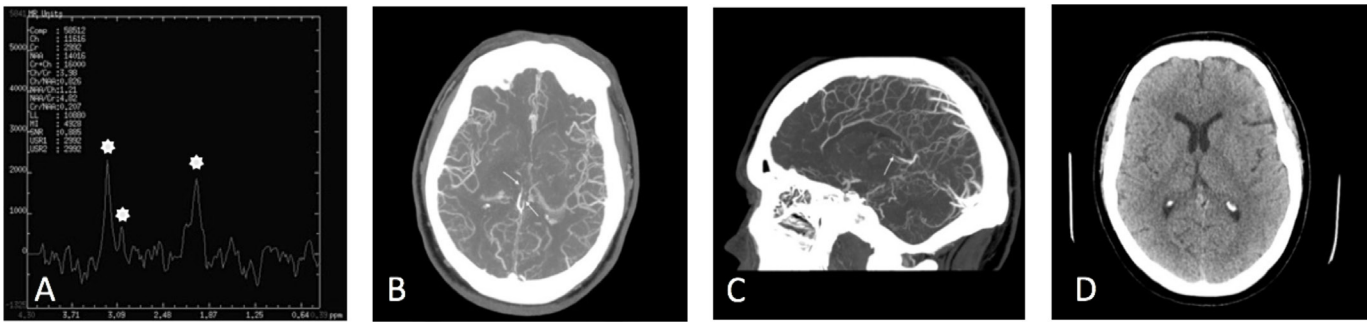

Figure 2 (A) MR spectroscopy data from a voxel centred on the right thalamus. The three important peaks are for N-acetyl aspartate (right star), choline (left star) and creatine (middle star). The low creatine peak would be atypical for malignancy. Axial (B) and sagittal (C) images from CT venogram show truncation/occlusion of the internal cerebral veins. (D) Axial image from a CT scan taken 1 year after admission, showing minor low density in the right thalamus.

jugular veins were particularly narrowed on imaging. In this case, we postulate that obesity with possible undiagnosed obstructive sleep apnoea may have contributed to slowly progressive venous sinus thrombosis, crossing a threshold to manifest symptoms and signs.

Learning points

- Thrombosis of the deep cerebral veins can present with unilateral thalamic oedema and/or infarction, and should be considered in patients who present with a unilateral thalamic mass lesion on imaging.
- Lengthy diagnostic processes can lead to undue distress for patients and their families, and delay appropriate management.
- The mainstay of treatment is anticoagulation, and prognostic outcome is better for unilateral than bilateral thalamic infarction.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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