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Case Report Open Access

Bilateral Thalamic Oedema: An Unusual Case of Reduced Speech Output in a 16 Year Old Patient

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Abstract

History and Background: A previously fit and well 16 year old school girl presented to the medical admissions unit with an acute onset of strange behaviour and reduced speech output over the previous few days. Collateral history from her mother revealed gradually worsening headaches over the previous 2 weeks. The headaches were worse on lying down and in the morning. Past medical history was unremarkable. She was on no regular medication and illicit substance use was denied.

Keywords: Bilateral thalamic oedema; Meningism; Neurological deficit

Introduction

Examination and investigations

On examination, the patient was dysphasic with limited speech output and ability to obey commands. Glasgow coma scale on admission was 15/15. There were no signs of meningism and only mild papilloedema on fundoscopy. Otherwise, there was no focal neurological deficit. She was afebrile and normotensive. Routine blood tests were within the normal range.

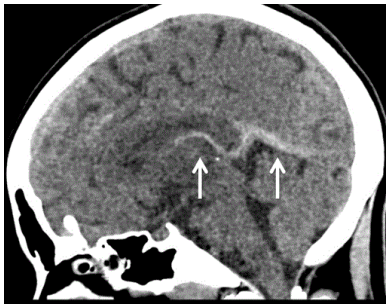

Figure 1: Non-contrast CT-Note the hyper dense straight sinus and inferior sagittal sinus secondary to thrombosis.

Differential Diagnosis and Initial Management

The differential diagnosis of an acute presentation of a young girl with speech and behavioural changes in the context of longer standing positional headache and mild papilloedema was diverse. This included central nervous system infection (i.e., herpes simplex encephalitis), a space-occupying lesion, undisclosed illicit substance use, a possible psychiatric cause and sinus venous thrombosis. The patient was empirically treated with intravenous aciclovir while brain imaging was arranged.

Management and Outcome

Initial computer tomography (CT) imaging (as shown in Figures 1 and 2) demonstrated hypoattentuation of the thalamus bilaterally as well as hyper dense straight sinus, indicative of sinus venous thrombosis. It was unclear at this stage if this represented infarction or thalamic oedema.

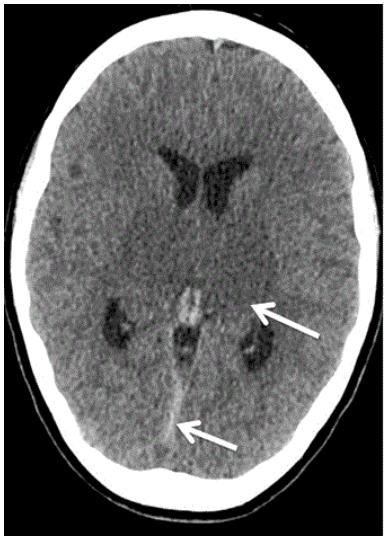

Figure 2: Non-contrast CT-bilateral thalamic hypo attenuation as well as the hyper dense straight sinus.

Further magnetic resonance imaging (MRI) again confirmed this and the thrombus can actually been seen in Figure 3. The patient was then anti-coagulated on heparin and her symptoms improved

significantly, she then was continued on warfarin (international normalised ratio target 2-3) for 6 months.

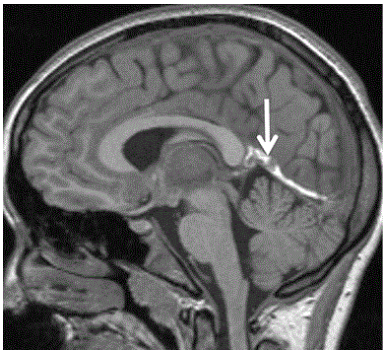

Figure 3: MRI 3D FLAIR-Note the signal change in the straight sinus likely indicating blood products.

Further assessment by haematology did not reveal any prothrombotic risk factors. MRI brain imaging 9 days later revealed complete resolution of the signal changes in both thalami, as shown in Figure 4. Neuropsychology follow-up confirmed improvement in cognitive impairment and she has since returned to school.

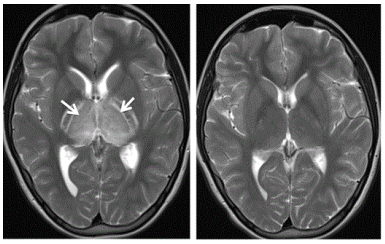

Figure 4: T2 Weight MRI-The image on the left is at presentation with the right being 9 days after. Note the marked diffuse abnormal signal from the thalamus and its subsequent resolution after anticoagulation.

Discussion

This case highlights the importance of rapid diagnosis and management of sinus venous thrombosis particularly in view of the impending infarction of both thalami.

Sinus venous thrombosis is a life threatening condition, if investigated and treated promptly there can be full reversal of symptoms [1]. There should be a high index of suspicion for sinus venous thrombosis in those presenting with headache, in this case symptoms were initially wide ranging with a number of differentials [1].

What is key about this case is that her symptoms completely resolved meaning that her imaging more likely demonstrated thalamic oedema rather than infarction. It does however mean that if management had been delayed much further the patient could have been potentially left with an irreversible neurological deficit.

Classically sinus venous thrombosis presents with cortical infarction and haemorrhage.1 The presentation of sinus venous thrombosis can be extremely diverse with bilateral thalamic involvement being extremely rare [2,3]. This patient's pattern of neurological deficit is an example correlating clearly with thalamic involvement. The mechanism of which is due to venous outflow obstruction to the thalamus secondary to thrombosis [3].

Non-contrast CT normally has low sensitivity for demonstrating changes in sinus venous thrombosis however subtle signs (i.e., hyper dense straight sinus) are useful markers and require specialist radiological review. Because of this, CT Venogram and MRI imaging should be sought as these are the most sensitive modalities to accurately diagnose this condition [4].

This case demonstrates a rare presentation of sinus venous thrombosis but highlights the importance of considering this diagnosis in patient with headache as delay in diagnosis can have significant consequences.

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