

Dural Sinus Thrombosis after Minor Head Injury in a Child

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Abstract

Introduction: Dural sinus thrombosis following minor head injury is rare. We report such a case in a child after mild head injury. **Clinical Picture:** A 4-year-old child presented with giddiness and vomiting after a fall. Clinical examination was unremarkable. Magnetic resonance venogram revealed thrombosis of the right sigmoid and transverse sinuses. **Treatment:** The patient was managed conservatively. **Outcome:** Repeat scans 10 weeks after injury showed recanalisation of the thrombosis. **Conclusion:** Dural sinus thrombosis should be excluded in children presenting with persistent giddiness and vomiting after minor head injury.

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Key words: Head injury, Lateral sinus thrombosis, Sigmoid sinus, Trauma

Case Report

A healthy 4-year-old Chinese girl was admitted to the hospital after a fall from a one-metre-high chair. She sustained a minor head injury to her right temporal region. There was no loss of consciousness, visual disturbances or motor dysfunction after the fall. She had symptoms of vomiting a few hours after the fall. Skull X-rays at the emergency department did not show any fractures.

Clinically, she was alert and afebrile. Her pupils were equal and reactive, and the rest of her central and peripheral neurological examination was unremarkable. There were no cerebellar signs. The patient was right-handed. Ophthalmoscopy revealed no papilloedema. There was slight tenderness over the right temporal region. Examination of both ears, mastoid, nose and throat examination did not reveal any abnormality.

She continued to have vomiting and giddiness in the ward. A computed tomography (CT) scan of the head and brain was done 12 hours after her injury and revealed no vault fractures. There was an increased density in the region of the right sigmoid sinus. This did not enhance with contrast (Fig. 1). The rest of the brain parenchyma was normal. There was no evidence of cerebral haematoma or oedema. A separate fine cut CT scan of the temporal bones showed no fracture or disease in the middle ear. Dural sinus

thrombosis was suspected. Further magnetic resonance imaging (MRI) scan and venogram (MRV) was performed. T1-weighted magnetic resonance (MR) images demonstrated an isointense area and T2-weighted imaging showed a hyperintense area in the right sigmoid sinus and the transverse sinuses. MRV confirmed occlusion of the right transverse and sigmoid sinus (Fig. 2a). There were no other intracranial lesions.

Her coagulation profile (antiphospholipid antibodies, prothrombin time and partial thromboplastin time) was normal. She did not show any neurological deterioration in the hospital. Her symptoms subsided on the third day of admission and she was discharged.

At follow-up 10 weeks after sustaining her head injury, the patient was asymptomatic with no neurological deficits. She had returned to her normal activities. Repeat MRI/MRV of the brain done at 10 weeks showed recanalisation of the sigmoid and transverse sinuses (Fig. 2b).

Discussion

Dural sinus thrombosis is usually associated with an otogenic source such as infection or tumour. However, dural sinus thrombosis has a reported incidence of 4% after a penetrating head trauma.¹ Although common when skull fractures cross the sinus, thrombosis can occur in association

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Fig. 1. Axial non-contrast computed tomography scan shows a hyperdense sigmoid sinus sign on the right, consistent with thrombus within the sinus (arrow).

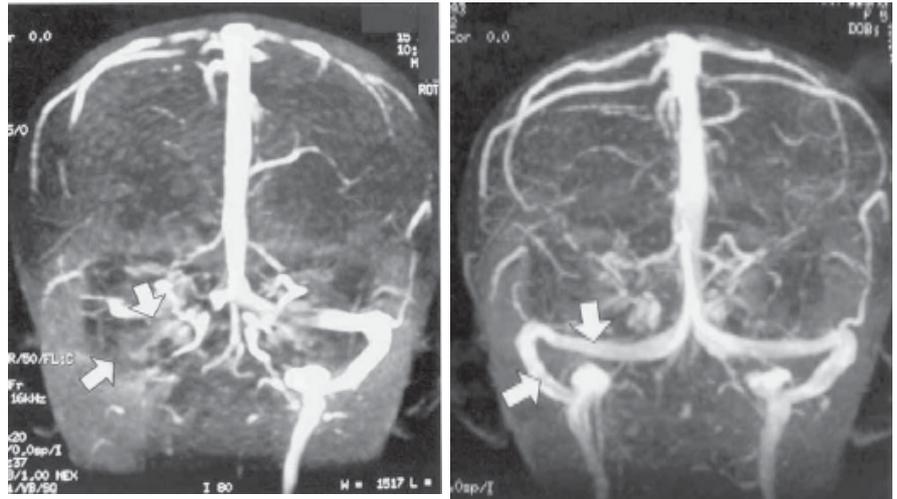


Fig. 2. Coronal view of magnetic resonance venography demonstrating complete flow void in the right lateral sinus and sigmoid sinus at presentation (left) and recanalisation at 10 weeks (right) (arrows).

with mild head injuries in the absence of fractures as reported by Bagley¹ in 1934. Since then, there have been sporadic reports¹⁻⁴ in the literature. Clinical description of sigmoid sinus thrombosis, especially in association with transverse sinus thrombosis, after closed head trauma is rare. The exact mechanism is unknown although several theories have been proposed. These include compression of the sinus by intracranial oedema or bleeding,^{2,5} intramural haemorrhages,² extension of trauma from the scalp or injured emissary veins,² and trauma to the sinus endothelial lining.⁵ Systemic conditions such as coagulation disorders (protein C and S deficiency)⁵ and thrombophilia (antiphospholipid syndrome, metastatic malignancy)⁶ have been reported to be associated with dural sinus thrombosis.

Symptoms are frequently vague and delayed in onset. These include gait ataxia, giddiness and/or vertigo, vomiting, and focal neurological deficits.⁷ Most of these signs and extensions are common following septic thrombosis of sigmoid sinus.

The diagnosis is often made on imaging studies, as in our patient. CT and MRI are the usual investigations of choice.⁸⁻¹¹ Findings suggestive of dural sinus thrombosis on CT include an area of hyperdensity seen in the sigmoid sinus area – “dense sigmoid sign”,⁴ as seen in our case; presence of high-density clots within the cortical veins – the “cord sign”,⁹ and clots within the dural sinuses – “dense vein sign”.¹⁰ In addition, secondary signs such as congested cortical veins, tentorial or gyral enhancement, and white matter oedema may also be noted.⁸ However, signal abnormalities in the area of the sigmoid sinus could be

related to extracerebellar clot and may not be due to thrombosis of the sinus per se. As such, MRI/MRV can provide further information. MRI scans and magnetic resonance venography can non-invasively provide direct visualisation of the dural sinuses and an assessment of sinus flow abnormalities.¹² The thrombus within a sinus can show variable signal intensity on MRI that depends on clot age.⁴ Acute blood clot is usually isointense on T1-weighted and hypointense on T2-weighted images reflecting the characteristics of oxyhaemoglobin. As the clot ages, the signal intensity of blood increases, first on T1- and later on T2-weighted images. Post-contrast T1 coronal images demonstrate a filling defect within the sinus. On T2-weighted images, there is increased signal from the sinus and on MR venography, there is absence of flow in the sinus. The MR images and MR venography are useful for both early diagnosis and follow-up. MRI is also the ideal method for following up on the recanalisation of the sinus. MRI, as a non-invasive technique that does not use ionising radiation, should be considered the investigation of choice, especially in young patients.

The clinical course in these patients is unpredictable and can vary from benign to fatal, depending on the aetiology and involvement of the non-dominant or dominant sinus.^{4,13} Non-dominant sigmoid sinus thrombosis (usually the left) rarely produce any neurological deficits and is associated with a good outcome.¹³ Untreated sinus thrombus progression may be fatal due to venous congestion and infarction. Treatment options for septic thrombosis include anticoagulation, fibrinolytic therapy and sigmoid

thrombectomy.¹⁴ The treatment of non-septic dural sinus thrombosis has not been conclusively defined. It is generally agreed that treatment should be tailored to the individual cases. Commonly, patients require only close neurological observation and a good recovery with no residual deficits is expected. In the absence of progressive neurological decline, many authors feel that prophylactic medical or surgical intervention is not indicated.^{4,8} Sinus recanalisation is achieved in about 6 weeks^{4,15} without treatment. The outcome of our case supports the conservative approach in management.

The case history of our patient is notable for two reasons. The simultaneous occurrence of sigmoid and transverse sinus thrombosis, especially in the absence of a skull fracture or intracranial haematoma, is extremely rare. In addition, in spite of the extent of thrombosis, the patient remained relatively well, and there was good resolution and outcome with conservative management.

Paediatric closed head injury is common in clinical practice and paediatric patients with giddiness and vomiting after mild head injury are frequently encountered. The significance and implications of these symptoms remain unclear and does not seem to be related to any specific feature of the head injury.¹⁶ Dural sinus thrombosis may be a contributing factor in some of these cases. Our case illustrates this known but uncommon complication of minor head injury, which, if not suspected, can easily be missed. Overt neurological symptoms and signs may be absent. Diagnosis relies on a high index of suspicion in children who sustained blunt head injury, and have persistent symptoms of giddiness and/or vomiting out of proportion to the severity of the injury, without obvious parenchymatous lesions on imaging studies.

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