Cerebral oedema and dural sinus thrombosis in an adolescent with diabetic ketoacidosis

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DESCRIPTION

Diabetic ketoacidosis (DKA) is a serious complication of type 1 diabetes mellitus (T1DM) and can be complicated by injury of the central nervous system (CNS). The most common CNS injury in DKA is cerebral oedema, diagnosed clinically in approximately 0.5%–0.9% of children with DKA.^{1–6} Subclinical cerebral oedema has been identified by MRI in up to 50% of cases of DKA.⁷ Intracranial thrombosis of arterial/venous origin can also occur during DKA. As cerebral oedema and CNS thrombosis can present similarly, timely neuroimaging is essential to identify pathology and direct therapies if the patient is not clinically improving after treatment for suspected cerebral oedema.

We describe an adolescent female who developed nausea and vomiting, headache and confusion at home. She was minimally responsive and was diagnosed with new-onset T1DM and severe DKA on hospital admission. Given clinical concern for cerebral oedema, she was treated with mannitol with mild neurological improvement. She had respiratory failure requiring intubation. Because of this minimal response to treatment in the setting of clinically declining neurological status, imaging was ordered and a non-contrast CT of the brain suggested mild cerebral oedema. CT-venogram demonstrated dural sinus thrombosis within the torcula and left transverse sinus (figure 1). DKA management was continued, mannitol and hypertonic saline

Figure 2 MRI/MRV images (T1, post gadolinium). Thrombus within the torcula (A) and progression of thrombus into right transverse sinus (B, white arrows). MRV, magnetic resonance venography.

were administered, anticoagulation therapy was initiated, and an external ventricular drain was placed to monitor intracranial pressure. Follow-up imaging with contrast-enhanced MRI and venography revealed progression of thrombus to include the right transverse sinus (figure 2). After 6 days, she was extubated, and neurologic exam slowly improved without deficits. She was transitioned to warfarin for anticoagulation, and follow-up MRI 10 months later showed full resolution of the thrombosis.

The most common neurologic abnormality diagnosed via imaging in youth with DKA is cerebral oedema. Cerebral oedema in children with DKA has a 20%–30% mortality rate.⁴ The pathophysiology of cerebral oedema involves multiple pathways.⁸ Hyperglycaemic and metabolic acidosis converge to produce hypoperfusion and subsequent hypoxic insult contributing to overall cytotoxic injury.^{8–13} Systemic and neuroinflammatory processes, impaired cerebrovascular autoregulation

Figure 1 Coronal CT venogram showing filling defects demonstrating area of thrombosis within the torcula (confluence of straight and transverse sinuses) and left transverse sinus (black arrows).

Learning points

- ▶ This case highlights the importance of rapid treatment when cerebral oedema is suspected, and the importance of neuroimaging when patients with diabetic ketoacidosis (DKA) and neurologic compromise do not respond to appropriate therapy for suspected cerebral oedema.
- ▶ Although the vast majority of central nervous system injury in children in the setting of DKA is related to cerebral oedema, it is important for clinicians to consider ischaemic prothrombotic pathology in the differential diagnosis.

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and reperfusion injury lead to the disruption of the blood-brain barrier and contribute to vasogenic injury.^{8–13} Clinically apparent cerebral oedema is more likely to occur with severe acidosis, severe hypocapnia and an elevated level of blood urea nitrogen.^{4 13 14} Other cerebral complications of DKA are less common and can include haemorrhagic or ischaemic infarction of arterial/venous origin.^{15 16} Stroke in children accounts for 10% of the neurologic complications seen with DKA.¹⁶ Multiple mechanisms predispose children with DKA to ischaemic stroke: cerebral oedema, increased blood viscosity due to hyperosmolality, severe dehydration, increased systemic inflammation, vascular endothelial injury and vasoconstriction.^{17 18} Focal deficits are rare and identification of DKA-associated thrombosis requires a high index of suspicion.¹⁹ Urgent diagnosis is critical because paediatric stroke is also associated with a high rate of lasting neurologic deficits.¹⁹ Cerebral oedema and intracranial thrombosis are CNS injuries that have high mortality and morbidity rates, making it critical to monitor children with DKA for changing neurologic symptoms.

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