Pediatric skull fracture with injury and thrombosis of the superior sagittal sinus: illustrative case

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BACKGROUND Cerebral venous sinus thrombosis (VST) is a complication of head injury and can be secondary to sinus compression by depressed skull fractures. Fracture elevation is a treatment option for VST secondary to extrinsic compression, but conservative management may also be effective. Venous sinuses can also be lacerated from skull fractures, resulting in epidural or subdural hematomas. The authors presented a case of sagittal sinus injury and thrombosis from a depressed skull fracture that caused a subgaleal hematoma. The injury was successfully managed conservatively.

OBSERVATIONS A 14-year-old boy presented after a head injury with a diastatic, depressed parietal bone fracture. Computed tomography venogram showed disruption and occlusion of the superior sagittal sinus with a subgaleal hematoma in continuity with the injured sagittal sinus. Because of concern for hemorrhage if tamponade on the sinus was removed, the patient was treated nonsurgically. At follow-up, the sinus had recanalized and the fracture had healed.

LESSONS Skull fractures with underlying sinus thrombosis can be managed conservatively with good outcome. Careful assessment for venous sinus injury should be made before undertaking fracture elevation to relieve sinus compression.

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Cerebral venous sinus thrombosis (VST) is a potential consequence of head trauma. VST is present in approximately 4% of patients with penetrating head trauma.1 There is an unclear prevalence following blunt head trauma, with recent data suggesting that prevalence may be as high as 26% when skull fractures are present near a sinus.2 There is currently no definitive guideline or recommendation for the management of these injuries in children.3

The American Heart Association and American Stroke Association recommend that VST be treated with anticoagulation, even in the setting of hemorrhages resulting from venous congestion.4 However, patients with VST following traumatic injuries often have hemorrhages stemming from other etiologies; therefore, anticoagulation has an unclear role. Furthermore, there has been unclear efficacy of anticoagulation in the setting of traumatic VST.5

In situations of sinus occlusion caused by sinus compression from a depressed skull fracture, elevation of the fracture has been described to treat symptomatic vascular occlusion.6 However, small retrospective analyses suggest that many patients with VST secondary to extrinsic compression of the vessel may have resolution of sinus occlusion without fracture elevation.3

Illustrative Case

An otherwise healthy 14-year-old boy was admitted to our hospital following a fall from a moving vehicle. He experienced transient loss of consciousness that resolved before arrival to our hospital. On presentation, he reported only a headache.

His initial neurological examination was without focal deficit, and Glasgow Coma Scale score was 14 because of mild confusion. His physical examination was significant for a swollen and boggy scalp with a palpable skull fracture near midline. There were no open lacerations.

Laboratory evaluation revealed normal coagulation studies and normal electrolytes. Computed tomography (CT) of the head demonstrated
Diastatic and depressed fracture of the left parietal bone near midline, overlying the superior sagittal sinus (SSS). This study also demonstrated a large subgaleal hematoma overlying the fractured segment that spanned a large portion of the subgaleal space (Fig. 1A). CT venogram demonstrated thrombosis of the SSS in the region of the fracture (Fig. 1B and C). Magnetic resonance imaging and magnetic resonance venogram (MRV) revealed injury to the SSS in continuity with the subgaleal hematoma (Fig. 1D).

The patient was treated nonoperatively because of concern that if the subgaleal space were entered, severe bleeding could ensue from release of tamponade on the injured SSS. He was admitted to the pediatric intensive care unit for neurological monitoring and aggressively hydrated with normal saline to reduce the chance of clot propagation. Anticoagulation was deferred because of inconclusive evidence of efficacy in traumatic VST.

The patient’s symptoms improved, intravenous fluids were discontinued after 7 days, and he was discharged home from the hospital. At the 7-month follow-up, MRV demonstrated recanalization of the SSS and there was no palpable skull deformity (Fig. 2).

Discussion

VST is a well-known sequela of depressed skull fractures. VST has potentially catastrophic complications, including intracranial hemorrhage secondary to venous infarction. Anticoagulation is the treatment of choice for spontaneous VST, but in traumatic sinus thrombosis there is unclear benefit, with the additional risk of hemorrhage due to bleeding from additional injuries.

When secondary to extrinsic compression, fracture elevation has been described as a treatment for VST. However, there is evidence that conservative management may allow for clinical improvement and recanalization of an occluded sinus.

Observations

Skull fracture can cause both sinus occlusion and injury, resulting in intracranial hematomas. This case demonstrates a patient with a venous sinus injury and thrombosis in the setting of a depressed, diastatic skull fracture with a resultant subgaleal hematoma. This subgaleal hematoma was clinically apparent on physical examination and raised concern that elevation of the fracture could remove the tamponade on the underlying injured SSS and lead to severe bleeding.

The subgaleal space is a potential closed space overlying the skull and is separated from the epidural space by the skull. Therefore, in the setting of a skull fracture, blood can track between the epidural and subgaleal spaces. Consequently, venous sinus injury may produce a subgaleal hematoma if the skull is fractured, as it did here.

Lessons

Although depressed skull fractures may require operative intervention, clinical context must be considered. In this case, close clinical assessment and nonsurgical management was used out of concern that operative intervention would release the tamponade of the injured SSS.

Given the extension of hemorrhage arising from the SSS injury into the subgaleal space, with both imaging and physical examination findings for a large hematoma, initiating an operation in this area may have provoked a high-volume venous hemorrhage due to disruption of venous tamponade.

In this case, we demonstrate that fluid resuscitation alone without anticoagulation or fracture elevation was sufficient for management of the patient’s SSS injury. Even in a child with a mature
skull, the fracture healed with satisfactory cosmesis. Clinicians must be vigilant for concomitant sinus injury in cases of traumatic sinus occlusion and weigh the risks and benefits of operative intervention.

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Disclosures
The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions
Conception and design: Miller, Zoerner. Acquisition of data: Zoerner. Analysis and interpretation of data: Zoerner. Drafting the article: all authors. Critically revising the article: all authors. Reviewed submitted version of manuscript: Zoerner, Reardon. Study supervision: Miller.

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