Depressed fractures overlying the major venous sinuses, in the absence of a CSF fistula or an associated hematoma, are often managed nonsurgically to avoid the risk of major sinus bleeding or sinus occlusion and venous infarction. Local wound debridement and closure are often sufficient in these cases.3,8 Distortion of the sinus lumen can occur, however, and results in the development of delayed, persistent elevated ICP5,12,13.

We report on the case of a previously neurologically healthy child in whom signs and symptoms of intracranial hypertension and cervicomedullary junction compression developed several days after the boy had sustained a depressed skull fracture over the posterior third of the SSS. Possible causes as well as diagnostic and treatment options are reviewed.

### Case Report

#### History and Examination

This previously healthy 9-year-old boy was struck on the back of the head, causing him to fall from a height of several feet to the ground. After a brief loss of consciousness, he was alert, neurologically intact, and suffering only from headache. Results of physical and neurological examinations were normal, with the exception of a horizontally oriented occipital scalp laceration and a heart rate of approximately 60 bpm associated with a normal blood pressure. Computerized tomography scans revealed a depressed occipital fracture overlying the SSS (Fig. 1). No intra- or extraaxial blood was noted on CT scanning. The wound was explored and closed after irrigation and culture.

### Neuroimaging Findings

The patient remained neurologically intact until 72 hours after the injury, when he experienced headache, neck pain, and diplopia. His heart rate remained higher than 60 bpm; his blood pressure remained normal. On examination, new onset papilledema and bilateral, complete sixth cranial nerve palsies were noted. Emergency MR images were obtained, which revealed significant compression of the posterior third of the SSS and displacement of the cerebellar tonsils into the cervical spinal canal (Fig. 2). High signal on T2-weighted images was noted in the region of the deep cerebellar nuclei and fourth ventricle. The superficial cerebellar tissues appeared normal. An MR venogram demonstrated significantly impaired blood flow through the SSS beneath the fracture (Fig. 3).

#### Operation

The patient was taken to the operating room, and a ventriculostomy was performed, revealing elevated ICP. The depressed fracture fragments overlying the SSS...
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were then elevated. On removal of the bone fragment, no disruption of the dura mater was seen, and no abnormal or excessive bleeding was encountered. Intraoperative Doppler ultrasonography confirmed flow through the sinus distal to the level of compression after the fragments were removed. In addition, the herniated tonsils were decompressed via a suboccipital craniectomy and C-1 and C-2 laminectomies. The ligamentum flavum and dura appeared normal at the C-1 and C-2 levels. The dura was opened, and necrotic cerebellar tonsils were revealed, with fragments of necrotic tonsillar tissue floating free in the CSF. Intraoperatively, the tonsils were noted to extend to the level of the superior edge of the C-2 lamina. A duraplasty with the pericranium was then performed. The remaining tonsillar tissue was not removed; the duraplasty was believed to provide adequate decompression to eliminate any further tonsillar compression.

Postoperative Course. Postoperatively, the patient’s heart rate increased from 60 to 70 to 90 to 100 bpm, with a normal blood pressure, and remained in this range for the remainder of his hospitalization. The ICP, elevated intraoperatively, rapidly returned to normal postoperatively and remained normal for the remainder of the monitoring period. A digital subtraction angiogram was obtained to assess flow through the SSS. The venous phase demonstrated normal contour and flow through the previously compressed region of the SSS (Fig. 4). The patient’s pain, papilledema, bilateral sixth cranial nerve palsies, and diplopia rapidly resolved after surgery.

Discussion

Depressed fractures are often associated with an underlying dural injury that, when associated with a major dural venous sinus, can result in significant blood loss intraoperatively. Attempts to repair sinus damage can cause thrombosis, stenosis, or complete occlusion of the sinus. In the region of the posterior third of the SSS, this will often result in venous hypertension and venous infarction. In most cases, nonoperative treatment is therefore widely considered the preferred therapy, in the absence of associated hematoma or CSF fistula. This treatment method is complicated by the possible impairment of normal sinus blood flow by fracture fragment compression, particularly when distal

![Fig. 1. Axial bone window CT scan demonstrating a depressed midline occipital skull fracture overlying the posterior third of the SSS.](image1)

![Fig. 2. Left: Midsagittal T1-weighted MR image demonstrating significant compression of the SSS by the overlying fracture. Right: Midsagittal T2-weighted image revealing downward displacement of the cerebellar tonsils to the level of the lamina of C-1 and signal change consistent with edema in the cerebellum.](image2)
portions of the venous sinuses are involved. Six prior reports in the literature describe the delayed development of intracranial hypertension and subsequent neurological decline associated with a depressed skull fracture. In all cases, the SSS was involved. In three cases, the patients underwent surgical elevation of the fracture and experienced a rapid postoperative reduction in ICP. There was no report of significant intraoperative bleeding or postoperative sinus-related complications. In one case, the refractory ICP was treated with serial lumbar punctures. No treatment data were provided in the fifth case. In the sixth case, a 34-year-old man was initially treated conservatively with acetazolamide. Its failure to resolve his symptoms prompted surgical elevation of the fracture on the 12th day after his injury, with subsequent resolution of symptoms. In the four cases for which clinical outcome data were provided, all patients recovered without long-term sequelae. The time to clinical presentation of elevated ICP ranged from 3 days to 1 month.

The unique feature of this case is the associated symptomatic tonsillar herniation. The supratentorial injury and subsequent intracranial hypertension either caused a displacement of tonsillar tissue through the foramen magnum or exacerbated an asymptomatic, underlying Chiari malformation Type I. None of the previously reported cases have involved tonsillar herniation as a complicating factor. The
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presence of edema within the deep cerebellar structures may have increased the degree of tonsillar displacement. Direct cerebellar trauma with associated edema could cause or exacerbate downward displacement of the tonsils due to enlargement of the cerebellum. This mechanism may be at least partially responsible for the tonsillar herniation seen in the present case due to the location of the injury and the presence of cerebellar, periventricular edema. It is unlikely, however, to be solely responsible, given the limited amount of edema detected by MR imaging on the T2-weighted sequences. The lack of abnormal MR imaging signal in the superficial cerebellar tissues indicates that a direct coup–contrecoup mechanism of injury is not primarily responsible for the development of this edema. Couldwell, et al.,4 have previously reported a similar case of supratentorial trauma in the context of a Chiari malformation Type I. In their report, a right temporoparietal skull fracture (not involving a major venous sinus) resulted in significant cerebellar injury documented on CT scanning and MR imaging.

The risk of acquired elevated ICP and delayed neurological deterioration supports an early role for MR imaging and venography in the evaluation of the trauma patient with injuries potentially impinging on a major venous sinus. Additionally, in this case, the presence of sixth cranial nerve palsies (although not uncommon in the setting of trauma and increased ICP), in association with new-onset neck pain and a persistent bradycardia, prompted a more thorough evaluation of the cranio cervical junction. In the acute setting, when neurological distress requires the treatment of a fracture in this highly dangerous location, then a CT scan is adequate as a quick, emergent, preoperative study. In this case, however, and in others in which the deterioration is subacute and the patient continues to be stable globally, the additional information provided by MR imaging can help clarify the precise reasons for the patient’s delayed decline. Without the MR image in the present case, the associated tonsillar herniation would not have been appreciated. Without the addition of the cervico medullary decompression, the symptoms may not have resolved or may have resolved more slowly or less completely and a second procedure to decompress the region may have been necessary. Magnetic resonance imaging can provide use ful data regarding the presence and degree of sinus compression as well as determining if there is thrombus within the sinus. The decision to proceed with MR venography or contrast angiography can be based on the initial MR imaging findings. In most cases, MR venography is reliable in determining the degree of blood flow through the compressed region;7,8,10 however, Binder, et al.,1 have described a residual abnormality in the sinus on a postoperative MR venogram. Persistent abnormal flow was also noted in the case presented by MR venography. In both cases, venous phase angiography confirmed normal sinus contour and blood flow. Therefore, although MR venography is a useful first test, venous phase angiography should be used if any question regarding contour or flow in the sinus remains.

Conclusions

In cases in which depressed skull fractures overlie major dural venous sinuses, the risk of delayed intracranial hypertension and neurological deterioration is increased. In addition, the presence of underlying tonsillar herniation (as in this unique case, presumably related to a Chiari malformation Type I) can predispose a patient to tonsillar contusion or compression and subsequent tonsillar necrosis. Magnetic resonance imaging, as soon as the patient is stabilized, can help the surgeon evaluate the presence of sinus compression. In cases in which blood flow is potentially compromised, MR venography and/or venous phase angiography should be considered. If significant flow alterations exist, then close neurological observation and surgical elevation of the fracture are warranted.

References


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