Intracranial hypertension caused by a depressed skull fracture resulting in superior sagittal sinus thrombosis in a pediatric patient: treatment with ventriculoperitoneal shunt insertion

Case report

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**Object.** Intracranial hypertension resulting from compression of the superior sagittal sinus (SSS) by an overlying depressed calvarial fracture is a rare condition. Primary surgical treatment for the symptomatic patient in this setting traditionally involves elevation of the fracture, which often carries significant associated morbidity.

**Methods.** The authors report a case involving a 6-year-old boy who suffered a closed, depressed, parietooccipital fracture as the result of an unhelmed all-terrain vehicle accident. This fracture caused compression and subsequent thrombosis of the SSS, which resulted in CSF malabsorption and progressive intracranial hypertension. Initially headache free following the injury, he had developed severe and unremitting headaches by postinjury Day 7. A CT angiography study of the head obtained at this time exhibited thrombosis of the SSS underlying the depressed calvarial fracture. Subsequent lumbar puncture demonstrated markedly elevated intrathecal pressures. Large volumes of CSF were removed, with temporary improvement in symptoms. After medical management with anticoagulation failed, the decision was made to proceed with image-guided ventriculoperitoneal shunt insertion.

**Results.** The patient’s headaches resolved immediately following the procedure, and anticoagulation therapy was reinstituted. Follow-up images obtained 4 months after the injury demonstrated evidence of resolution of the depressed fracture, with recanalization of the SSS. The anticoagulation therapy was then discontinued. To the authors’ knowledge, this report is the first description of ventriculoperitoneal shunt insertion as the primary treatment of this infrequent condition.

**Conclusions.** This report demonstrates that select patients with this presentation can undergo CSF diversion in lieu of elevation of the depressed skull fracture—a surgical procedure shown to be associated with increased risks when the depressed fracture overlies the posterior SSS. The literature on this topic is reviewed and management of this condition is discussed. (DOI: 10.3171/2010.3.PEDS09441)

**Key Words** • sagittal sinus thrombosis • depressed skull fracture • ventriculoperitoneal shunt

Thrombosis of the cerebral venous sinuses caused by an overlying skull fracture in the pediatric patient is a relatively infrequent event that has been sparsely described in the literature.10,15 Regarding cerebral venous sinus thrombosis, 2 different pathophysiological mechanisms have been discussed.12 The first of these two mechanisms is characterized by occlusion of cerebral veins and subsequent venous insufficiency. The second is related to obstruction of the venous sinuses and the ensuing development of intracranial hypertension. Occlusion of cerebral veins often leads to venous insufficiency, edema, and venous infarction when collateral pathways for drainage are limited. In contrast, obstruction of the venous sinuses—where some degree of collateral drainage is present—more commonly manifests in proximal increases in venous pressure with malabsorption of CSF resulting in progressive intracranial hypertension. In adult patients who suffer spontaneous, nontraumatic thrombosis of the cerebral veins and sinuses, these phenomena have been studied in detail. In the largest study to date involving this popula-
tion, approximately 77% of patients with sinus thrombosis were classified as suffering from some degree of venous insufficiency based on the focality of their neurologic exam. It is worth noting that outcomes in this group were significantly worse than in the remaining 23% of patients designated as having isolated intracranial hypertension. In patients who suffer depressed skull fractures resulting in occlusion of underlying venous structures, no formal analyses have been conducted regarding the incidence of subsequent venous insufficiency.

Assessing whether venous insufficiency and/or infarction are present is important in the management of the injury and in triage of patients with depressed skull fractures that result in cerebral venous sinus obstruction. In those individuals with sinus obstruction and clinical or radiological evidence of cerebral venous insufficiency, elevation of the depressed fracture must be contemplated. This also holds true in individuals in whom neurological deterioration is a significant concern, and in those with open and/or contaminated fractures. However, in those individuals who present with a clinically stable neurological examination who later develop symptoms relating to CSF malabsorption and progressive intracranial hypertension, it is likely that VP shunt insertion alone is a reasonable measure. To our knowledge, this case report is the first to discuss CSF diversion accomplished using VP shunt insertion as the primary treatment of patients with intracranial hypertension caused by a depressed skull fracture resulting in venous sinus obstruction.

Case Report

History and Examination. This 6-year-old boy initially presented to the Vanderbilt University Children’s Emergency Department following an unhelmeted all-terrain vehicle accident in which he was ejected after the machine struck a barbed-wire fence. He landed on his back, struck his head forcefully against the ground, and briefly lost consciousness. The patient was noted to be neurologically intact on arrival at the ED, with evidence of moderate soft-tissue swelling and tenderness in the region just superior to the inion, but exhibited no evidence of direct overlying laceration on physical examination. Admission CT imaging of the head demonstrated a partially comminuted parietooccipital skull fracture measured to be depressed approximately 0.9 cm below the calvarial surface. The depressed fragment exerted mass effect on the posterior third of the SSS. Although minimal traumatic subarachnoid hemorrhage was associated with the depressed fracture, no gross evidence of intracranial hemorrhage was found (Fig. 1).

Hospital Course. Given that the fracture was closed and the patient was neurologically intact, after consideration of the significant risks associated with elevation of the depressed fracture overlying the posterior third of the sagittal sinus, the decision was made not to proceed with elevation of the fracture. Following evaluation in the ED, the patient was transferred to the pediatric intensive care unit for serial neurological examinations overnight.

The patient did reasonably well following the injury, initially complaining of focal occipital pain to palpation, but denying generalized headaches. He was transferred to the floor on postinjury Day 1 and plans were made to follow up with neurosurgery as an outpatient. On postinjury Day 4, the patient began complaining of a mild headache. By postinjury Day 7, the headaches had become severe and unremitting, and the patient had developed diplopia relating to a mild restriction in abduction of the left eye, which was consistent with new onset of a left abducent nerve palsy. The pediatric trauma team again consulted with the pediatric neurosurgical service at this time. Subsequent CT angiography of the head demonstrated thrombosis and occlusion of the SSS at the level of the depressed parietooccipital fracture (Fig. 2). An ophthalmological evaluation revealed bilateral papilledema. Lumbar puncture was then performed, and revealed an opening pressure of 36 cm H₂O. The CSF was drained to a closing pressure of 5 cm H₂O, and the patient’s headaches and diplopia resolved.

Treatment options were discussed with the family,
and the decision was made to proceed with anticoagulation therapy. A heparin drip was instituted per the recommendations of the neurology service, with a partial thromboplastin time of 60–85 seconds as a goal. The patient’s headaches gradually improved, and he was discharged home after 7 days of observation, on a regimen of enoxaparin injections (Sanofi-Aventis). The patient returned to the ED 2 days after discharge with recurrent, generalized headaches. The enoxaparin was withheld, and subsequent lumbar puncture demonstrated an opening pressure of 40 cm H₂O. Discussions were then held with the family regarding the risks and benefits of surgical elevation of the fracture versus VP shunt insertion. The family elected to proceed with VP shunt insertion.

Operation. Given the small size of his ventricular system, frameless stereotaxy and image guidance was used after a navigation protocol CT scan was registered to the patient by using the Tracer software included on a StealthStation (Medtronic, Inc.). Desired entry and target points were chosen. A peritoneal catheter was inserted and connected to a programmable valve (Codman) with an antisiphon device, which had previously been set at 150 mm H₂O. A higher than normal opening pressure was chosen in an attempt to prevent collapse of the ventricular system around the catheter. Following insertion, the ventricular catheter was connected to this system. The wounds were irrigated and closed.

Postoperative Course. The patient’s headaches resolved almost immediately following VP shunt insertion. He resumed anticoagulation therapy with enoxaparin and was discharged home on postoperative Day 2. Follow-up CT and CT angiography studies of the head obtained 4 months postinjury demonstrated evidence of resolution of the depressed calvarial fragment, with recanalization of the SSS (Figs. 3A, 3B, and 4). The decision was made to stop anticoagulation therapy at this time.

Discussion

Depressed skull fractures overlying major intracranial venous sinuses can result in obstruction of cerebral venous outflow. This in turn can result in localized edema of the brain and subsequent venous infarction. When elevated venous pressure is transmitted to the arachnoid granulations in communication with the SSS, absorption of CSF is impaired. Because no pressure gradients exist across intracranial compartments, intracranial hypertension in this setting is not as immediately perilous as it is with noncommunicating hydrocephalus or focal mass lesions. Symptoms in progressive intracranial hypertension—including progressive headaches, lethargy, and blurry vision—are nonspecific and often appear subacutely following time of sinus thrombosis. False localizing signs (including sixth cranial nerve palsy) can occur, as they did in the patient previously described. If left untreated, elevated intracranial pressures can be deleterious to vision over time.

A head CT scan in a patient with progressive intracranial hypertension resulting from CSF malabsorption will fail to demonstrate progressive ventriculomegaly in a brain with normal compliance, for reasons previously discussed (no pressure gradient between the intraventricular and cortical subarachnoid spaces, or between intracranial compartments). Lumbar puncture in this setting is helpful, and often demonstrates markedly increased intrathecal pressures. A CT or MR venography study is usually diagnostic and can demonstrate the site and degree of obstruction. Catheter angiography remains the gold standard for evaluation of flow through the underlying sinus, and has been used in previous studies to stratify surgical management (for example, if no flow is visualized through the sinus, operative elevation is strongly considered).

There is a limited number of case reports and case series in the literature describing patients with depressed skull fracture resulting in obstruction of intracranial venous outflow and subsequent intracranial hyperten-

![Fig. 3. Head CT scans (brain windows) obtained without contrast approximately 4 months after image-guided VP shunt insertion.](image)

![Fig. 4. Sagittal reconstruction of CT angiography studies of the head obtained approximately 4 months after VP shunt insertion demonstrating recanalization of the SSS, with remodeling and partial resolution of depressed parietooccipital calvarial fracture.](image)
These manuscripts describe some variation of the following algorithm: if the patient with posttraumatic venous sinus thrombosis caused by a depressed skull fracture remains asymptomatic, conservative management is indicated. If the patient clinically deteriorates because of venous insufficiency or intracranial hypertension relating to communicating hydrocephalus, surgical elevation of the fracture is recommended. Endovascular measures have been described in cases in which sinus thrombosis and symptoms persist despite surgical elevation of the fracture.2

The morbidity associated with surgical elevation of a depressed skull fracture overlying a dural venous sinus has been well described in various populations. Ozer et al.11 reported a series of 17 patients—ages ranging from 7 to 48 years, with a mean age of 24.2 years—who required surgical elevation of a depressed skull fracture overlying a cranial venous sinus within 24 hours of presentation. The types of injuries in their series were various (motor vehicle accidents, assaults, falls, and so on) and included both high- and low-energy mechanisms. It is notable that 13 (76%) of the 17 patients had a GCS score of 13–15 on arrival. Although the decision-making process for operative intervention in this paper was not discussed, information regarding the rate of intraoperative hemorrhage was recorded. Specifically, 11 of the 17 patients were found to suffer profuse bleeding in the operating room, requiring a sum total of 35 U for transfusion. Two patients in this series deteriorated postoperatively and died.

The aforementioned report can be contrasted to a case series by Meier et al.,8 which described 39 patients who suffered a head injury that resulted in traumatic injury of the dural sinuses requiring operative management. This series involved a greater proportion of high-energy traumatic mechanisms, as illustrated by the fact that 27 (69%) of the 39 patients presented with a GCS score of 8 or less. The overall mortality rate in this series was 41%, with an intraoperative mortality rate of 20%. All patients with injury of the posterior third of the SSS died in this series (67% of whom died intraoperatively), in comparison with a 50% death rate in patients with traumatic injury of the middle third of the SSS, and a 17% death rate in patients with traumatic injury of the anterior third of the SSS. This case report is referenced to reinforce the notion that significant injury involving the posterior third of the sagittal sinus appears to be associated with increases in intra- and perioperative morbidity and/or mortality. On the contrary, injury involving the anterior third of the SSS—relating in part to the lower flow and feasibility of surgical ligation at this location—is often not as treacherous.

There are several caveats that should be mentioned before applying information gleaned from the previous 2 case series to the present case. First, the majority of patients included in the aforementioned reports were adults. Because the pediatric calvaria exhibits decreased mineralization and less overall thickness than with adults, skull fractures often occur following comparatively lower-energy mechanisms in children.9 Concomitant injury to underlying neural and venous structures would be expected to be less severe in skull fractures that occur following these relatively lower-energy injuries. Additionally, although it appears that a very high proportion of patients in both the Ozer and Meier series required urgent and/or emergency operative intervention, the patient presented in our case report was neurologically stable on arrival and was taken to the operating room in a semi-elective fashion. Of all case reports involving patients who underwent delayed elevation of depressed skull fractures that had resulted in compression of the posterior SSS and subsequent intracranial hypertension, the incidence of severe intraoperative hemorrhage appears to be much lower than in the reports previously discussed (see Table 1).

Whereas knowledge of the presence of an underlying dural sinus laceration would help to stratify preoperative risk, there is a paucity of data regarding the ability of radiological features to predict dural sinus laceration in patients with depressed skull fractures. In the anecdotal reports that have been reviewed (Table 1), the correlation between findings reported on preoperative neuroimaging and the presence of intraoperative hemorrhage relating to dural laceration has been suboptimal. Although the specific criteria were not described, it was the opinion of

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Pt Age (yrs), Sex</th>
<th>GCS Score on Arrival</th>
<th>Significant Hemorrhage on Initial CT</th>
<th>Appearance of SSS†</th>
<th>Date of Op</th>
<th>Bleeding Encountered</th>
<th>Degree of Recovery</th>
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<tr>
<td>du Plessis, 1993</td>
<td>20, M</td>
<td>7</td>
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<td>small amt</td>
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<td>Uzan et al., 1998</td>
<td>38, M</td>
<td>15</td>
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<td>PO</td>
<td>PID 30</td>
<td>none</td>
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<td>Meltzer et al., 2000</td>
<td>13, M</td>
<td>15</td>
<td>no</td>
<td>FO</td>
<td>PID 3</td>
<td>none</td>
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<tr>
<td>Binder et al., 2004</td>
<td>34, M</td>
<td>15</td>
<td>no</td>
<td>FO‡</td>
<td>PID 12</td>
<td>none</td>
<td>full</td>
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<tr>
<td>Donovan, 2005</td>
<td>35, M</td>
<td>11</td>
<td>no</td>
<td>PO</td>
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<td>15</td>
<td>no</td>
<td>FO</td>
<td>PID 3</td>
<td>none</td>
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<tr>
<td>Yokota et al., 2006</td>
<td>52, M</td>
<td>15</td>
<td>no</td>
<td>PO</td>
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* Pediatric case reports are listed in bold. Abbreviations: amt = amount; FO = full occlusion; PID = postinjury day; PO = partial occlusion; pt = patient.
† Patency of the SSS was assessed using MR venography, diagnostic angiography, or both.
‡ Magnetic resonance venography of the SSS demonstrated full occlusion on postinjury Day 4, and repeat MR venography on postinjury Day 11 demonstrated partial occlusion.
Placement of VP shunt for skull fracture–induced hypertension

Meier et al. that dural sinus laceration could be suspected approximately half of the time based on findings on pre-operative imaging. At present, no reliable radiological or clinical method exists to confirm or contradict this statement. Conclusively, although we hesitate to directly extrapolate the surgical risks reported in the case series described earlier to the patient described in this case report (who had suffered traumatic injury to the posterior third of his SSS), clearly the risks associated with elevation of a depressed skull fracture overlying a dural venous sinus merit consideration.

In contrast to those described above, the surgical risks of VP shunt insertion are much lower, with a documented mortality risk well under 1%. Although shunt dependency is a concern, the estimated 1-year complication-free shunt survival rate for the patient described in our report could be approximated at 75% based on previously published, age-matched evidence. Furthermore, given the well-documented precision and widespread acceptance of contemporary frameless stereotaxy, small ventricular size is no longer an obstacle to accurate ventricular catheter placement. In our case, the SSS was shown to have recanalized 4 months after VP shunt insertion, despite the fact that the depressed fracture was never surgically addressed. Based on this evidence of recanalization as demonstrated on CT venography, the patient is probably no longer “shunt dependent.”

Most practitioners agree that the morbidity involving elevation of a depressed skull fracture overlying a venous sinus is significant, and should be avoided when at all possible. With this in mind, it should be reiterated that anecdotal data suggest the risks associated with fracture elevation may be less in pediatric patients and in those who present in a delayed fashion. We believe that management of a depressed skull fracture resulting in compromise of the underlying venous sinus can be stratified based on symptoms and underlying pathophysiological mechanisms. In asymptomatic patients, conservative management with watchful waiting is indicated. In patients who become symptomatic because of local venous hypertension and/or venous infarction, elevation of the depressed fracture is advised. In patients who become symptomatic because of progressive intracranial hypertension, primary treatment with VP shunt insertion is a plausible option. In all populations, when the primary pathophysiological mechanism underlying neurological deterioration is uncertain, clinical examination, radiological evaluation, and diagnostic procedures—including lumbar puncture to measure opening pressure and to evaluate for resolution of symptoms following large-volume drainage of CSF—can assist with differentiation among these causes.

Conclusions

In this case report we describe a patient who initially presented following an all-terrain vehicle accident with a depressed parietooccipital fracture that resulted in occlusion of the posterior third of the sagittal sinus and the development of communicating hydrocephalus. After medical therapy with anticoagulation failed, the patient was successfully treated with VP shunt insertion.

Because of the significant risks relating to intraoperative hemorrhage associated with elevation of a depressed skull fracture over a venous sinus, other surgical methods for treatment should be contemplated in select patients. If clinical evaluation and neuroimaging studies indicate that symptoms are related to the development of CSF malabsorption, resulting in progressive intracranial hypertension, VP shunt placement with subsequent anticoagulation therapy should be considered a plausible option.

Following VP shunt insertion, our patient experienced full resolution of his symptoms. Follow-up CT angiography studies obtained 4 months after the procedure demonstrated recanalization of the SSS and partial resolution of the depressed calvarial fracture. To our knowledge, this case report is the first to discuss the use of CSF diversion via VP shunt insertion as the primary surgical treatment in a patient with intracranial hypertension caused by a depressed skull fracture, and resulting in venous sinus obstruction.

Disclosure

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

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