Improvement in venous outflow following superior sagittal sinus decompression after a gunshot wound to the head: case report

Daniel M. Birk, MD,1 Matthew K. Tobin, BS,1 Heather E. Moss, MD, PhD,2,3 Eric Feinstein, MD,2 Fady T. Charbel, MD,1 and Ali Alaraj, MD1

Departments of 1Neurosurgery, 2Ophthalmology and Visual Sciences, and 3Neurology and Rehabilitation, University of Illinois at Chicago, Illinois

The most commonly described indications for surgical management of closed depressed skull fractures are hematoma evacuation and repair of extensive cosmetic deformity. Venous sinus injury, which occurs in a subset of depressed skull fractures, is not typically listed as an indication for surgical treatment due to the potential for major venous hemorrhage associated with surgery near these structures. However, if patients exhibit signs and symptoms of intracranial hypertension and radiographic findings demonstrate sinus compromise, surgical elevation of the depressed skull fragments is indicated. The authors present the case of a 25-year-old woman with a depressed skull fracture secondary to a gunshot wound with symptomatic compromise in venous outflow of the posterior one-third of the superior sagittal sinus. The patient was treated with surgical decompression via bilateral craniectomy along with intracranial pressure–lowering medical therapy and had almost full resolution of her presenting symptoms with documented improvement in flow through the superior sagittal sinus. While the use of surgical treatment for these types of injuries is highly debated, the authors demonstrate here that safe, effective surgical management of these patients is possible and that surgical decompression should always be considered in the case of symptomatic venous sinus flow obstruction.

http://thejns.org/doi/abs/10.3171/2014.10.JNS141349

KEY WORDS superior sagittal sinus; intracranial hypertension; gunshot wound; skull fracture; craniectomy; traumatic brain injury

While dural venous sinus thrombosis is a well-recognized cause of elevated intracranial pressure (ICP), little attention has been given to nonthrombotic causes of occlusion to venous sinuses. Furthermore, depressed skull fractures occur over venous sinuses in 11%–18% of cases, representing a fairly large proportion of these cases.16,19 Nonetheless, very few cases of intracranial hypertension due to venous sinus compression have been reported.12 Traditionally, conservative management of depressed skull fractures that lie over venous sinuses has been preferred over surgical treatment because of the risk of hemorrhage associated with operating over a venous sinus.16,19 However, in cases in which neurological deficits are present with elevated ICP, surgical treatment is preferred.3,14,15 In this article, we describe the case of a 25-year-old woman who presented with a 3-week history of worsening headache, blurry vision, and diplopia following removal of bullet fragments from a gunshot wound (GSW) to the head. We report on the successful surgical management of a depressed skull fracture compromising flow through the superior sagittal sinus (SSS).

Case Report

History and Examination

This 25-year-old woman presented with progressive headaches, visual field loss, transient visual obscurations, pulsatile tinnitus, and horizontal double vision developing in the weeks following removal of bullet fragments after a GSW to the posterior head. Urgent ophthalmologi-
d. m. birk et al.

The computerized axial tomography (CAT) evaluation revealed mildly decreased central vision of 20/20-3 in each eye, inferior temporal visual field defect in the right eye, and inferior temporal and nasal visual field defects in the left eye, abduction deficits in both eyes with intermittent esotropia, and Grade 4 optic disc edema in both eyes. The patient was referred to our neurosurgery team for emergency evaluation given suspicion for intracranial hypertension as a cause of her symptoms. Medical therapy with acetazolamide 1000 mg twice daily was initiated, with minimal change in symptoms.

Neuroimaging

CT scans demonstrated a depressed skull fracture in the parietooccipital skull overlying the distal one-third of the SSS (Fig. 1A and B), which was consistent with the history of GSW. Digital subtraction angiography demonstrated compromised venous flow in the posterior one-third of the SSS (Fig. 2A and B), and volumetric flow analysis measured via quantitative MR venography (Q-MRV) and quantitative MR angiography (Q-MRA) (NOVA, VasSol Inc.) showed reduced flow through the SSS of 214 ml/min, total jugular venous outflow of 397 ml/min (Fig. 3A and B), and a large difference between large vessel arterial inflow and large vessel venous outflow (301 ml/min).

Operation

To decompress the SSS and decrease ICP, we performed bilateral occipital craniectomy (Fig. 4) and cranioplasty with titanium mesh (Fig. 1C). The patient was placed prone, and, following skin incision, 4 bur holes (2 bur holes on each side of the SSS) were made. The craniectomy was completed using a high-speed power drill (diamond tip), at which time the depressed bone fragments were removed. However, due to the risk of venous sinus bleeding, the bone fragments penetrating the sinus were not completely removed, and a small fragment of bone was left in place and was drilled to an eggshell thickness (Fig. 1C). No sinus bleeding was encountered during the decompression. Dural matrix was then placed in the bony defect and was covered with a piece of GelFoam followed by a preformed titanium mesh, and the incision was closed.

Postoperative Course

The procedure was performed without complications, and the patient woke up with improvement in many symptoms, including headaches and double vision. Follow-up Q-MRV performed 1 day postoperatively showed improvement in the flow through the SSS measured at 380 ml/min, with total venous outflow to 541 ml/min (Fig. 3C and D). Acetazolamide was discontinued due to rash. The patient was discharged the day after surgery, and at 2 weeks postoperatively she had improvement in headaches but persistence of transient visual obscurations and papilledema (Fig. 5A). Visual field showed improved loss in both eyes, sparing part of the inferotemporal field in the left eye. She received topiramate and by 1 month postoperatively had improvement in visual fields in both eyes as well as resolution of papilledema (Fig. 5B). Q-MRV and Q-MRA performed 2 weeks postoperatively showed additional improvement in total venous outflow to 599 ml/min and a decrease in the difference between arterial inflow and venous outflow to 185 ml/min.

Discussion

Signs and symptoms of intracranial hypertension often include headache, diplopia, loss of peripheral vision, blurred central vision, and nonspecific symptoms, including nausea, vomiting, and dizziness. Intracranial hypertension caused by impaired cerebral venous outflow due to compression of the SSS is a very rare event, with few re-
The presentations are heterogeneous, some with acute symptoms within 48 hours, and others with delayed symptoms often several weeks after the initial traumatic injury. The compression of the SSS occurred across various parts of the sinus from anterior to posterior. Overall morbidity and mortality is affected by the location of sinus compression, where injury across the anterior one-third of the sinus is associated with the lowest mortality while injury to the posterior one-third is associated with the highest. While Meier et al. reported death in all of their patients with compression of the posterior one-third of the SSS, demonstrating the significant difficulty in managing these injuries, our patient and previous reports of similar injuries illustrate successful operative management of posterior SSS compression in the setting of depressed skull fractures.

Typically, conservative management of depressed skull fractures overlying venous sinuses is advocated rather than a surgical approach because of the high risk of hemorrhage associated with surgery around a venous sinus. However, in patients in whom evidence of elevated ICP is present, as was the case with our patient, surgery to elevate the depressed skull fractures and remove the cause of sinus compression can be safely performed to restore blood flow in the sinus. Furthermore, when deciding whether surgical intervention is appropriate, one must distinguish between SSS thrombosis causing a flow obstruction and SSS stenosis causing intracranial hypertension. While surgical intervention and elevation of the depressed fracture is often curative in patients with SSS stenosis, surgery alone will not cure SSS thrombosis unless the thrombus is removed. This approach, however, drastically increases the risk of major hemorrhage and is not normally considered. In cases of nonthrombotic SSS stenosis, significant injuries and neurological deficits are unlikely to resolve without surgical intervention, especially when the posterior one-third of the sinus is involved. Therefore, elevation of the depressed fracture must be considered in the management of these patients. While this has been the method most

FIG. 3. A: Preoperative Q-MRV NOVA 3D reconstruction of volumetric flow analysis demonstrating reduced flow through the SSS as well as the compression of the sinus (arrows). B: Preoperative Q-MRV venous map demonstrating flow through the venous sinuses. C: Postoperative Q-MRV NOVA 3D reconstruction of volumetric flow analysis demonstrating the increased flow through the SSS following decompression as well as the absence of any noticeable postoperative compression (arrows). D: Postoperative Q-MRV venous map demonstrating flow through the venous sinuses following decompression. LJV = left jugular vein; LSIGMOID = left sigmoid sinus; LTRANS = left transverse sinus; RJV = right jugular vein; RSIGMOID = right sigmoid sinus; RTRANS = right transverse sinus; VOG = vein of Galen.
One report demonstrated the use of repeat lumbar puncture to reduce ICP while collateral venous drainage develops.25

One of the most important aspects of management of intracranial hypertension is appropriate and adequate imaging studies. These studies not only help guide treatment strategies, but also help to delineate distinctions such as SSS thrombosis versus SSS stenosis, as discussed above. Traditional imaging done in patients presenting with signs and symptoms of intracranial hypertension includes contrast-administered MR studies and cerebral angiography. Studies not traditionally done, and not previously reported for SSS compression due to depressed skull fracture, include Q-MRA and Q-MRV with volumetric flow analysis, as we report here. The role of Q-MRA in assessment of intracranial flow has been validated in animal models6 and established in ischemic stroke following intracranial stenting or carotid endarterectomy, and in the diagnosis of subclavian steal syndrome.2–4,13 We demonstrate quantitative improvement in cerebral venous outflow following surgical intervention and further improvement with additional medical management. In addition, we demonstrate a narrowing of the gap between measured arterial inflows and venous outflows following treatment, similar to Q-MRV findings in dural arterial venous fistula–associated intracranial hypertension17 and idiopathic intracranial hypertension.1 These Q-MRV results provide an objective measure of short- and long-term patient outcomes and could be a potential method with which to track progress in patient recovery over extended periods of time.

In closed depressed skull fractures, surgery is often only recommended for one of 3 reasons: repair of lacerated dura, evacuation of hematoma, or correction of cosmetic deformity.5,9,22,24,26 While this and previous case reports illustrate the efficacy of surgical intervention in depressed skull fractures overlying venous sinuses, most texts and current recommendations do not list venous sinus injury as an indication for surgical intervention. The most commonly cited reason for not operating is the associated risk of hemorrhage from operating near a venous sinus. The risk of hemorrhage is most often correlated with the complete removal of depressed bone fragments. Rather than remove the bone fragments, we decided that drilling them down with a high-speed drill to an eggshell thickness would not only decompress the SSS but would help avoid this dreaded complication. Furthermore, it is very possible that even with decompression of the sinus, hemorrhage could still occur, and thorough planning and preparation must take place to ensure adequate response should this happen. While the risk of hemorrhage is very real and is not one to discount, the long-term risk for permanent neurological deficit by not performing surgery in these patients far outweighs the risk of hemorrhage associated with surgery near venous sinuses. It is clear, though, that the best approach for the management of depressed skull fractures overlying venous sinuses must consider imaging studies (including quantitative venous flow studies), the patient’s overall clinical condition, and the risks and benefits of different treatment modalities. However, surgical intervention, as successfully demonstrated by our patient, should always been considered an option in similar cases.

Conclusions

Patients with venous sinus injury may present with various signs and symptoms due to intracranial hypertension, typically including visual disturbances and headache. They may present shortly after the initial injury, or may not develop symptoms for days to weeks afterwards. Patients presenting with signs of intracranial hypertension and possible venous sinus injury should be evaluated with Q-MRV (when available) and cerebral angiography, and they should be treated promptly to minimize intracranial hypertension–related morbidity. While conservative management is often advocated for closed depressed skull fractures, surgical intervention should always be considered in patients with symptomatic venous sinus injury, especially when the posterior aspect of the sinus is involved. We demonstrate here that successful surgical decompression of the injured sinus results in hemodynamic and clinical improvement, with complete resolution of headaches and significant improvement in visual deficits and papilledema.