Depressed skull fracture overlying the superior sagittal sinus as a cause of benign intracranial hypertension

Case report

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The use of surgical treatment for depressed skull fractures that are located over major venous sinuses is a matter of controversy. However, if clinical and radiological findings of sinus obliteration and related intracranial hypertension are present, surgical decompression is indicated. The authors present the case of a 38-year-old man who had a depressed skull fracture overlying the posterior one-third portion of the superior sagittal sinus. The lesion was initially treated conservatively and the patient was readmitted 1 month later with signs and symptoms of intracranial hypertension. The role of radiological investigation in the detection of venous sinus flow and indications for surgical treatment are discussed. If venous sinus flow obstruction is revealed in the presence of signs and symptoms of intracranial hypertension, surgery is indicated as the first line of treatment.

**Key Words •** depressed skull fracture • intracranial hypertension • cerebral angiography • magnetic resonance imaging

To reduce the risk of infection, open depressed skull fractures are generally treated by surgical approaches.\(^5,6\) However, if the depressed bone fragments are located over major venous sinuses, one may choose more conservative management. In these cases, to prevent massive sinus bleeding, the generally accepted approach is simple wound debridement, irrigation, and skin closure without bone elevation.\(^1,4\) In their series of 400 cases of depressed skull fractures, Miller and Jennett\(^7\) reported venous sinus involvement in 11% of cases. In 18 of these cases the depressed bone fragments were not elevated. By obliterating the sinus lumen and disrupting normal flow, bone fragments can cause an acute or chronic increase in intracranial pressure, and patients may be readmitted to the hospital with related symptoms.\(^4\)

Here we present the case of a man who had a depressed skull fracture located over the superior sagittal sinus (SSS). Initially the case had been managed conservatively and the patient was readmitted to the hospital with symptoms and signs of benign intracranial hypertension. Treatment choices and indications for surgery in such a case are discussed.

**Case Report**

**History.** This 38-year-old man was struck on his head by a pistol grip and admitted to the emergency department complaining mainly of headache. His physical examination revealed a wound located in the midline occipital region. He was otherwise neurologically intact. Skull x-ray films and computerized tomography (CT) scans revealed a depressed skull fracture located over the posterior one-third portion of the SSS (Fig. 1).

Because of the location of the fracture, treatment was limited to simple wound debridement and primary skin closure, and the patient was discharged 24 hours later. Ten days after trauma occurred, the patient presented at the outpatient clinic suffering from headache, nausea, and vomiting. These symptoms were treated by analgesic and antiemetic medications. Thirty days after trauma occurred, he was readmitted to the emergency department complaining of gradual visual loss.

**Examination.** The patient’s physical examination demonstrated homonymous hemianopsia and bilateral papilledema that was more pronounced on the left side. His visual acuity was 8/10 in the right eye and 2/10 in the left. No fever or sign of meningeal irritation was detected. Cranial CT scanning showed nothing other than the depressed skull fracture. Magnetic resonance (MR) imaging and MR angiography revealed flow disturbance in the posterior one-third portion of the SSS located under the depressed fracture (Figs. 2 and 3). Venous phase angiography showed severe flow disturbance in the posterior one-third portion of the SSS and bilateral congestion of the ophthalmic veins (Fig. 4). The benign intracranial hypertension was thought to be caused by obliteration of the posterior one-third portion of the SSS by depressed bone fragments. Surgical treatment was indicated.

**Operation and Postoperative Course.** The depressed
bone fragments were elevated and the SSS was decompressed. The patient’s visual acuity was partially improved and he experienced no headache or vomiting by the 2nd postoperative day. He was discharged from the hospital on the 3rd postoperative day. Magnetic resonance angiography performed 1 month after the patient was discharged showed normal flow in the sagittal sinus at the level of the skull fracture (Fig. 5).

At follow-up examination performed 6 months later, the patient’s visual field was intact and his visual acuity was 10/10 in the right eye and 8/10 in the left.

**Discussion**

Because of the high risk of uncontrollable bleeding and the subsequent risk of mortality, the treatment of choice for depressed skull fractures located over venous sinuses is a conservative one. The bone fragments can cause complete or incomplete obliteration of major venous sinuses and symptoms of intracranial hypertension.

To the best of our knowledge, there are only four reports in the medical literature of cases of intracranial hypertension due to sinus compression secondary to depressed skull fractures. Steinbok, et al., reported the case of a patient in whom papilledema developed on the 4th posttraumatic day; however, no details were given about treatment modality. In the case reported by du Plessis the patient suffered from partial compression of the posterior one-third portion of the SSS caused by depressed bone fragments; this was documented by venous phase angiography. After surgical removal of bone particles, the patient’s intracranial pressure decreased within 1 to 2 min-

The patient suffered from a depressed fracture located over the sagittal sinus. A partially obstructed SSS was documented by venous phase angiography. The patient experienced headache, left inferior hemianopsia, and papilledema, which were thought to be due to benign increased intracranial pressure resulting from venous compression. The patient was treated by multiple lumbar punctures.

In our case the patient experienced headache and vomiting, which began 10 days after trauma. Despite rapid progression of these symptoms, his visual loss developed gradually. Magnetic resonance angiography demonstrated a flow disturbance, but no complete occlusion in the posterior one-third portion of the SSS under the depressed skull fragments. Venous phase angiography revealed a severe flow disturbance in the posterior one-third portion of the SSS. Following elevation of the depressed bone fragments and decompression of the SSS, there were notable improvements in the patient’s visual field and acuity. At his last follow-up examination performed 6 months after the injury, his vision was 10/10 in the right eye and 8/10 in the left.

Radiological evidence of flow in the affected segment of the venous sinus and the presence or absence of thrombus inside the lumen are of great clinical importance. The importance of MR imaging and its advantage over conventional angiography in the diagnosis of dural sinus thrombosis are expressed by some authors. Magnetic resonance techniques including MR angiography can be used to obtain detailed information about the compressed segment of the sinus, the degree of flow, and the presence or absence of thrombosis inside the lumen. By means of conventional angiography, one may obtain evidence of compression and the presence or absence of flow. However, secondary evidence for venous flow obstruction can best be documented using MR angiography. As a result, MR techniques are advocated as the first choice of method for investigation of venous sinuses in these cases. Despite the compression of the SSS observed in our patient, evidence of minimal flow and the absence of thrombosis were demonstrated by MR angiography. Conventional angiography did not demonstrate the flow sufficiently.

The indication for surgical treatment was based on evidence that external compression was the major cause of the flow disturbance. This fact was confirmed on control MR angiography postoperatively.

Conclusions

A case of depressed skull fracture located over the major venous sinuses in which there is an unexplained worsening of the patient’s neurological condition should suggest the possibility of obstructed venous sinus flow caused by depressed bone fragments. Magnetic resonance techniques and conventional angiography are recommended diagnostic studies. When flow disturbance by external compression is documented in the presence of clinical signs and symptoms, surgical decompression is the first choice of case management.

References


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