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# Case Report

# Midline depressed skull fracture presenting with quadriplegia: A rare phenomenon

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#### **Abstract**

**Background:** Midline depressed skull fractures (MDSFs) deserve a special mention among skull fractures and should always be treated with caution. Here, an extremely unusual clinical presentation of a case of MDSF is highlighted along with its successful surgical management.

Case Description: A 26-year-old male presented with quadriplegia following assault on the head with sharp weapons. The patient had multiple lacerated wounds on the scalp with underlying cranial fractures. On evaluation, computerized tomography (CT) of the brain showed a midline depressed skull fracture compressing the superior sagittal sinus (SSS) causing bilateral frontoparietal venous infarction. CT venogram showed a filling defect of the SSS due to the penetrating bone fragment. He underwent elevation of the depressed fracture and repair of the sinus with pericranial graft. Patient improved neurologically, and follow-up magnetic resonance venogram showed a patent SS.

**Conclusion:** MDSF can present with quadriparesis/quadriplegia due to middle one-third SSS obstruction/thrombosis leading to bilateral motor cortical venous infarction. Such MDSFs may require emergent surgical elevation of the depressed bone fragment for restoration of the patency of the sinus.

**Key Words:** Compound depressed skull fracture, midline depressed skull fracture, quadriparesis, quadriplegia, superior sagittal sinus thrombosis, surgical intervention



#### **INTRODUCTION**

Depressed skull fracture is a type of traumatic brain injury often seen secondary to assault. It can lead to infection, cosmetic deformity, intracranial hematoma, or a local mass effect on underlying brain tissue, leading to neurological deficits with possibility of early or delayed seizures. However, a midline depressed skull fracture (MDSF), in addition to the abovementioned complications, can lead to compression of the superior sagittal sinus (SSS), which may lead to stenosis, obstruction, or thrombosis of the SSS with subsequent

venous ischemia and infarction, with or without features of raised intracranial pressure. [1] Patients with depressed

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fracture are known to present with monoparesis and rarely with paraparesis. [6] However, patients with MDSF presenting as quadriparesis/quadriplegia, secondary to SSS injury, is an extremely rare phenomenon. Here, we report a case of MDSF indenting the middle one-third of SSS, leading to bilateral frontoparietal venous ischemia, and presenting with quadriplegia (MRC grade 0/5). The patient underwent surgical decompression of the depressed bone segment and eventually had a good clinical outcome with magnetic resonance venography (MRV) at 6-month follow-up showing a patent SSS.

## **CASE REPORT**

# History and clinical findings

A 26-year-old male was allegedly assaulted by multiple unknown assailants with sharp weapons (knives and sickle) on his head. At presentation, patient had a Glasgow Coma Scale (GCS) of E3M6V5. His pupils were equal, bilaterally 3 mm in diameter, and briskly reacting to light. On motor system examination, the patient had quadriplegia, with Medical Research Council (MRC) grade 0/5 power in all four limbs along with decreased tone. On sensory examination, the patient had intact crude touch, pain, and temperature sensations in all four limbs. Loss of motor function in all four limbs in conjunction with intact sensations all over the body pointed towards a cranial cause for the deficit and ruled out any spinal cord or peripheral nerve involvement. Multiple sutured lacerated wounds were observed over the scalp: Large midline vertical wound extending from occipital protuberance to forehead (~20 cm in length), transverse wound on right posterior frontal scalp (~5 cm), vertical wound on the left frontal scalp (~8 cm), and vertical wound on the left parietal scalp (~7 cm).

# Investigations and management

Computerized tomography (CT) scan of the brain showed midline parietal depressed fracture with bilateral frontoparietal mixed density lesions and surrounding edema suggestive of venous infarction [Figure 1a-d]. A CT venogram showed absence of filling of middle one-third of SSS under the depressed fracture fragment [Figure 1c]. CT and magnetic resonance imaging (MRI) of the cervical spine were normal [Figure 1b], thus ruling out a spinal cause for the quadriplegia.

The patient was diagnosed to have a compound depressed skull fracture over the midline parietal bones, with the depressed bone fragment causing SSS obstruction. In view of SSS obstruction and presence of progressive venous infarction, patient underwent bilateral parietal parasagittal craniotomy, elevation of depressed fracture, repair of the tear in the SSS with pericranial graft, and repair of adjacent dural tear. Intraoperatively, after elevation of the depressed fracture, a tear was noted on the superior surface of SSS with brisk bleeding. Normal

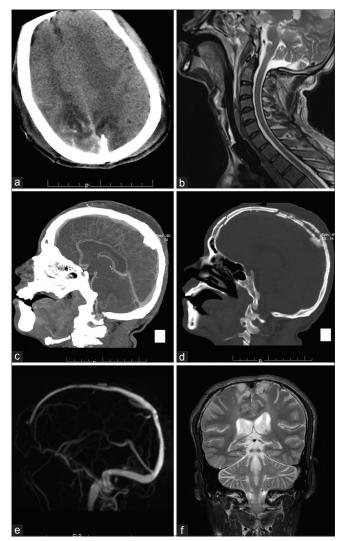


Figure 1: (a) Computed tomography scan showing midline depressed fracture compressing the SSS and causing bilateral posterior frontal venous infarctions, (b) MRI cervical spine showing normal study, (c) CT venography (preoperative) showing a defect in the SSS, (d) Bone window of the same CT venography image as in c showing the depressed bone fragment in the midline, (e) MRV done at 6 months follow-up showing filling of the SSS, (f) MRI scan of the brain in coronal section (posterior frontal region) showing resolved venous infarctions

proximal and distal sinus was exposed and the defect was repaired with pericranial graft overlaid with a muscle patch. Bone fragments were discarded and thorough wash was given followed by wound closure in a single layer.

#### Postoperative course

Postoperatively, patient was managed with anti-edema measures, anti-epileptics, and broad spectrum antibiotics. At discharge, he had MRC grade 2/5 power in left upper limb with MRC grade 0/5 power in all other limbs. At 6-month follow-up, patient had MRC grade 4/5 power in all four limbs. MRV imaging showed a patent SSS [Figure 1e], with areas of residual gliotic changes on both sides of the SSS [Figure 1f].

### **DISCUSSION**

Depressed skull fractures occur due to a high energy impact on a small area of the skull leading to indentation of the affected segment. [6] Depressed skull fractures are classified as closed or open depending upon the status of the overlying skin. Closed depressed skull fractures (skin intact) are usually treated conservatively unless significant cosmetic deformity, underlying hematoma, or venous sinus injury leading to increased intracranial pressure warrant surgical repair.[8] On the other hand, open depressed skull fractures (skin breached) are usually managed surgically because of the possible risk of infection due to dural tear and contamination.<sup>[5]</sup> Among the depressed skull fractures, midline depressed skull fractures warrant a special mention for the possible risk of SSS injury. Holmes and Sargent first emphasized the possibility of venous sinus injury due to depressed fracture.[1] It has been reported that approximately 11.5% of depressed fractures involve a dural venous sinus.[3]

MDSFs with underlying SSS injury and resulting thrombosis can lead to venous hypertension or intracranial hypertension due to impairment of CSF absorption. [1,8] A CT or MRV is essential to look for the patency of the superior sagittal sinus in all MDSF cases. Clinical presentation may vary from being asymptomatic to monoparesis, paraparesis, or raised intracranial pressure. [2,6] Quadriparesis or quadriplegia is an extremely rare presentation, and has not been reported previously. We postulate that, in our case, the penetrating skull fragment impinged on the SSS, thus resulting in SSS obstruction or thrombosis, which in turn caused venous edema or infarction of the bilateral motor cortices or subcortical pathways (located in the parasagittal region), and eventually the patient presented with quadriplegia.

Management of a midline depressed fracture can be a dilemma for neurosurgeons because of the risk of massive hemorrhage from SSS intraoperatively. [8] Traditional approach to midline depressed skull fractures has been conservative. Textbooks have often mentioned that even a compound depressed fracture over the SSS is a contraindication for surgery. However, the concepts regarding this theory are changing. Recent literature shows that depressed fractures over venous sinuses may lead to early or delayed benign intracranial hypertension due to venous obstruction, as well as elevation of the depressed fragment restores patency of the venous flow and resolution of symptoms due to raised intracranial pressure. [1,2,7,8] Patients presenting with features of benign intracranial hypertension without any focal neurological deficits have been managed conservatively with anti-edema measures or repeated lumbar punctures. [7] However, some authors have reported persistence of features of benign intracranial hypertension (BIH) with conservative measures, ultimately requiring

surgical intervention (elevation of bone fragment) in their patients. [4] In addition, if untreated, the thrombosis can extend proximally or distally leading to venous infarction, which is irreversible. [8] However, it should be borne in mind that surgical intervention should be individualized for each patient's clinical condition and weighed against its risks. Moreover, reports have documented spontaneous recanalization of SSS thrombosis without surgical intervention.

Depressed fracture leading to monoparesis is well-known due to compression of underlying motor cortex by the depressed fragment. Patients of MDSFs presenting at a later stage with BIH have been described. [2,7] Patients of MDSF presenting with paraparesis has also been documented. [6] To the best of our knowledge (after a thorough PubMed search), we could not find any documented case of midline depressed fracture presenting with quadriparesis or quadriplegia.

The elevation of the depressed fracture led to flow restoration through the sinus, which was demonstrated by MRV. In our case, the the depressed bony fragment causing obstruction of the SSS was removed, which revealed a rent in the SSS extending onto the adjoining dura. We repaired the rent with a pericranial graft so that we could achieve a watertight closure of the dura. A partial or full-thickness pedicled dural graft was considered, however, the presence of tear extending onto the adjoining dura and presence of dural venous lakes in the vicinity precluded its use in our patient. Opening up of the SSS may have resulted in decrease in the size of the eventual venous infarct and could be validated by the neurological improvement seen in our patient at follow-up. Hence, radiological evidence of SSS obstruction/thrombosis in MDSF, along with corresponding neurological deficits may be considered as an indication for surgery and can help in the restoration of venous blood flow in the SSS leading to neurological improvement, as seen in our case. This surgical approach is perilous; hence, should be undertaken by experienced microneurosurgeons trained in vascular anastomotic techniques and under stringent neuroanesthesia monitoring.

# **CONCLUSION**

MDSFs deserve a special mention among skull fractures and should always be treated with caution. Presentation of MDSF can be varied and rare presentation such as quadriparesis/plegia should be noted. An MR or CT venogram is imperative in the evaluation of MDSFs to look for SSS stenosis, obstruction, or thrombosis. Emergency surgical elevation of depressed fragment for flow restoration through the venous channel should be considered if filling defect is noted on imaging and the patient is symptomatic for the same. In lieu of good

postoperative outcome, as seen in our case, surgical intervention for symptomatic midline depressed skull fracture merits consideration in the present neurosurgical era

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**Informed Consent:** The patient and his relatives have consented to the submission of this case report to the journal.

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#### **Conflicts of interest**

There are no conflicts of interest.

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