

Importance of Calvaria in Cerebrospinal Fluid Dynamics: A Case of Ventriculomegaly and Sinking Flap Syndrome after Decompressive Craniectomy

Abstract

Sinking flap syndrome and hydrocephalus are well-known complications of decompressive craniectomy. The underlying pathogenesis and management of these complications are still unclear. We present a case of hydrocephalus and recurrent sinking flap syndrome following decompressive craniectomy. We highlight the pivotal role of calvaria in the management of these complications.

Keywords: Craniectomy, cranioplasty, hydrocephalus, sinking flap syndrome

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Introduction

Decompressive craniectomy (DC) is a common procedure in the neurosurgical field for intracranial hypertension. It is occasionally associated with complications such as subdural collections, sinking flap syndrome, and ventriculomegaly (VP). Sinking flap syndrome or “syndrome of trephined” is a rare complication of decompressive craniectomy which may present with severe headache, deterioration of neurological status, focal deficits, and seizures.^[1]

Cranioplasty is the cornerstone of treatment for sinking flap syndrome and hydrocephalus. We present a case of recurrent sinking flap syndrome following bone flap removal and it reiterates the role of bone flap in cerebrospinal fluid (CSF) dynamics.

Case Report

A 55-year-old male presented with sudden onset loss of consciousness and left hemiparesis. On evaluation, he was found to have large right frontotemporal hematoma [Figure 1a] secondary to ruptured right middle cerebral artery aneurysm. The patient underwent decompressive craniectomy with aneurysm clipping and partial evacuation of hematoma. After initial improvement for 2 weeks, the patient deteriorated in clinical status in the 3rd week and computed tomography [Figure 1b]

showed ventriculomegaly. A medium pressure VP shunt was inserted on the left side. Following shunt, the patient improved clinically and repeat CT head [Figure 1c] revealed decompressed ventricles. A week later, the patient started worsening and became drowsy. CT scan at this time showed sinking flap syndrome with midline shift toward the contralateral side for which he underwent cranioplasty with autologous bone. The patient improved clinically as well as radiologically [Figure 1d]. Approximately, 3 months later, he got admitted with persistent discharge from the wound site and contrast-enhanced computed tomography head [Figure 1e] suggested underlying infection. During wound exploration, there was evidence of osteomyelitis, so the bone flap was discarded. CT head [Figure 1f] was satisfactory at this stage. Ten days following this, patient's clinical condition deteriorated. CT head [Figure 1g] revealed sinking flap and midline shift to the contralateral side. Attributing it to shunt over drainage, the shunt was immediately tied and then changed to high pressure [Figure 1h]. Although patient made gradual recovery with [Figure 1i] the correction of ventricle size, he never regained same neurological status. The patient was planned for artificial cranioplasty but succumbed to a different illness at home.

Discussion

Complications of DC include hydrocephalus and sinking flap. The exact pathophysiology

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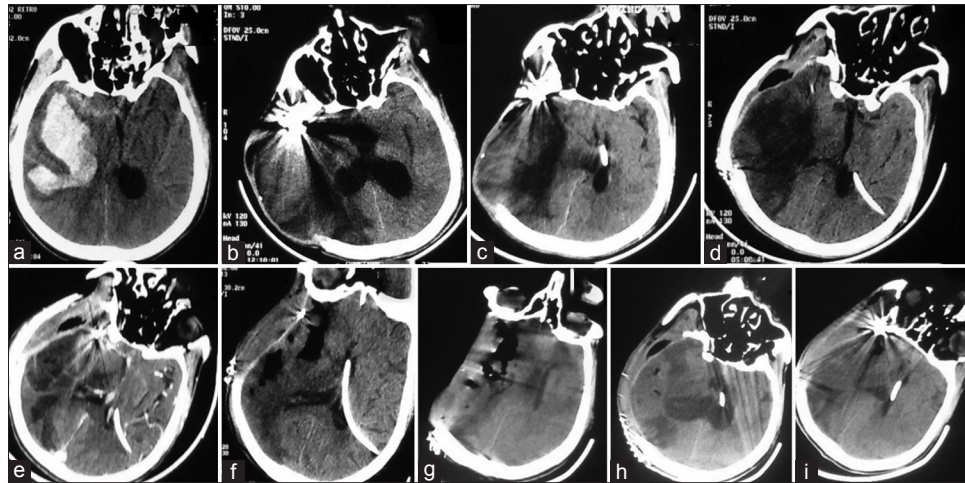


Figure 1: (a) Noncontrast computed tomography head (NCCT) (on the day of ictus): Showing intraparenchymal bleed in the right frontotemporal region. (b) NCCT head (25 days after decompressive craniectomy) shows increased brain bulge and ventriculomegaly. (c) NCCT head (41 days after decompressive craniectomy) shows settled brain bulge and decompressed ventricles after ventriculopleural shunt. (d) NCCT head (48 days after decompressive craniectomy) shows decompressed ventricles post cranioplasty and residual brain edema in the right frontotemporal region. (e) Contrast-enhanced computed tomography head (150 days after decompressive craniectomy) shows contrast enhancement along the dural margin at the site of craniotomy. (f) NCCT head (151 days after decompressive craniectomy) shows decompressed ventricles and no brain bulge immediately after the removal of bone flap. (g) NCCT head (160 days after decompressive craniectomy) shows sinking brain and chinked ventricles (after which shunt was ligated). (h) NCCT head (162 days after decompressive craniectomy) shows brain bulge and ventriculomegaly after shunt ligation. (i) NCCT head (165 days after decompressive craniectomy) shows settling brain bulge and decompressed ventricles after conversion from medium pressure to high-pressure ventriculopleural shunt

of these complications is not very clear.^[1] With an intact skull, CSF pressure of 2–5 cm H₂O is required to open the arachnoid villi for CSF absorption in superior sagittal sinus. A craniectomy defect may lead to a loss of the normal dirotic intracranial pressure (ICP) waveform required to keep the arachnoid villi open leading to failure of CSF absorption. This lack of CSF resorption is probably responsible for the development of hydrocephalus following decompression.^[2,3]

Another complication reported following DC is sinking flap syndrome. The suggested mechanism is a direct transmission of atmosphere pressure to intracranial cavity causing an inward shifting of the scalp over craniotomy defect. Others have proposed a negative gradient between atmospheric and ICP, aggravated by CSF hypovolemia after craniectomy as a cause sinking flap syndrome.^[4]

In the present case, the hydrocephalus was a result of DC and loss of the pressure required to keep the villi open. Probably, early cranioplasty would have sufficed, and CSF diversion (shunt) could have been avoided. Removal of the bone flap (because of infection) with shunt *in situ* aggravated the negative gradient between atmospheric and ICP leading to sinking flap syndrome. That the cranioplasty remains the cornerstone of treatment for sinking flap syndrome that has been amply proved in the present case as removal of the bone flap led to recurrence of the syndrome which was further exaggerated by drainage of CSF.

Unfortunately, the patient succumbed before we could hope to restore the pressures with artificial cranioplasty.

Management of post-DC hydrocephalus remains a controversial topic with proponents of shunt surgery both before and after cranioplasty.^[5] The balance is gradually tilting toward cranioplasty followed by the shunt, if required, thus highlighting the pivotal role of cranioplasty in re-establishing the disturbed CSF flow dynamics.

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Conflicts of interest

There are no conflicts of interest.

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