



Case Report

Rapid development and resolution of sinking skin flap syndrome in a patient following decompressive craniectomy

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ABSTRACT

Sinking skin flap syndrome (SSS) is a serious complication following decompressive craniectomy (DC). Although CSF hypovolemia and exposure to atmospheric pressure have been attributed as causative factors, the definite etiologies remain elusive. We present a case where a patient had a rapid deterioration without any warning sign following a diagnostic and therapeutic lumbar puncture after DC. The patient had rapid fluctuation of symptoms with change of position. She was managed conservatively and finally achieved a full neurologic recovery without cranioplasty. This case report shows the heterogeneity of SSS and that cranioplasty may not be required in acute cases.

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1. Introduction

Decompressive craniectomy (DC) is a widely performed procedure to reduce uncontrollable intracranial pressure.^{1,2} The DC converts the physiology of cranium from a 'closed box' to an 'open box.' In some craniectomy patients, the forces of atmospheric pressure and gravity can thus overwhelm intracranial pressures, leading to paradoxical brain herniation. CSF (Cerebrospinal fluid) diversion, dehydration, and position change may aggravate this phenomenon.^{3,4} The terms paradoxical brain herniation and sinking skin flap syndrome (SSS) are alternatively used, but the pathophysiology is still not fully explained and there is no established management guideline. We present a patient who developed paradoxical herniation after a lumbar puncture following hemicraniectomy and who was managed successfully with Trendelenburg position and intravenous fluids alone. She achieved full neurologic recovery without cranioplasty.

2. Case Report

Fifty-eight-year-old hypertensive lady presented with left hemiparesis and altered sensorium. She was found to have a right putaminal bleed with para and intraventricular extension (NIHSS of 16 at baseline). On 3rd day evening, she became more drowsy (GCS 11/15) and CT brain revealed mid-line shift. She underwent urgent decompressive craniectomy with hematoma evacuation. Extubation was done next day morning with GCS of 15. On 12th day, she had an episode of fever and next morning she became drowsy with a bulge over the hemicraniectomy site. The CT scan of brain showed pseudomeningocele with hydrocephalus. Lumbar puncture was planned to rule out meningitis and also to reduce the pseudomeningocele and 40 ml of CSF was drained. She was more alert next day morning but had a sudden deterioration of sensorium with pupillary asymmetry (right 3.5 mm, left 2.5 mm, both sluggish). She was rushed for CT Scan suspecting a rebleed after a bolus dose of 200 ml of Mannitol. While being transferred to scanner, she improved immediately after she was slightly turned towards the hemicraniectomy side. We initially thought

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the improvement could be due to the bolus mannitol but absence of any new bleed and her prompt improvement with posture change made us think about the possibility of “Paradoxical herniation” (when brain herniation occurs in presence of low intracranial pressure in a patient with a craniectomy defect where atmospheric pressure supersedes the ICP leading the brain shift and herniation). So we started her on intravenous fluid replenishment, and all anti-edema measures were stopped. Her craniectomy site was still a bit swollen and there was no evidence of sinking skin flap in the CT scan. She became responsive, with normal pupillary reflexes when kept on the hemicraniectomy side and in Trendelenburg position. But she immediately became drowsy with pupillary asymmetry when we reverted her back to the same position as before. We continued to keep her in Trendelenburg position and the final scan was done on 18th day. Considering bulge of the brain at hemicraniectomy site, cranioplasty (definitive management of the “sinking flap syndrome”) was postponed. She was discharged home with a “helmet” (a plastic cap) over the hemicraniectomy site and advised to maintain the above posture with close monitoring of vitals and to follow up after two weeks. As she revisited us on 36th day, there was a persistent bulge over the hemicraniectomy site and she continues to be asymptomatic even after assuming supine position.

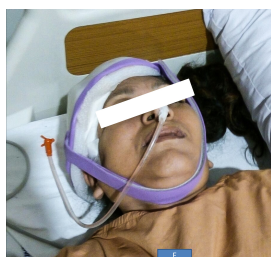


Fig. 1: Patient after “sinking skin flap syndrome” with the protective “helmet” on

3. Discussion

Sinking skin flap syndrome (SSS) is a well-known entity since its publication in the year of 1939. Although many investigators have tried to explain the mechanism, the exact pathophysiology is yet unknown. Several authors have proposed that a negative gradient between atmospheric and intracranial pressure, exacerbated by CSF hypovolemia is the likely mechanism of neurological deterioration after craniectomy.^{5,6}The development of this syndrome usually takes several months after DC, but it is variable. However, there are some differences between the syndromes those develop acutely than late. While decreased blood flow and the long duration for which the underlying brain parenchyma remains under a considerable pressure are the key factors for development of the chronic subtype; altered

CSF hydrodynamics allowing atmospheric pressure to shift the brain underneath is the pathophysiology of the acute counterpart.⁷ Our case highlights the heterogeneity of this condition and that it can develop acutely as well. The following points are unique features in our case. First of all, a much lesser amount of CSF drainage (only 40ml) was carried out (as compared to earlier studies like that of Zhao et al where 200-300 ml /24 hours were drained)⁷ that resulted to this unfortunate clinical consequence. Secondly, there was a very rapid deterioration as well as recovery of symptoms with alteration of patient’s posture including pupillary changes. Thirdly, there was absence of warning signs like headache, vomiting or other autonomic disturbances of SSS mentioned in the literature.⁸ Our patient did not actually improve with anti-edema measures, and it rather had worsened the situation causing further reduction of ICP and more herniation. Thus, the use of anti- edema measure and the reason to discontinue it at a particular juncture is an experience worthy of mentioning. This again underscores the importance of positioning, intravenous fluids in the acute crisis period to maintain CSF hydrodynamics.⁸Another uniqueness of our case was use of an artificial helmet over the defect at the time of emergency. This approach has not been mentioned so far to our knowledge. As management of pseudomeningocele, we should be more cautious about therapeutic lumbar puncture in the early few weeks of stroke where the affected brain is still edematous and also probably about the amount of CSF that may be drained. Patient has to be monitored closely, keeping in mind the possibility of developing the above mentioned complications.

4. Conclusion

To summarize, we should be careful about post DC paradoxical herniation at any point of time. The CSF accumulated over the craniectomy site acts as a buffer preventing atmospheric pressure to supersede the intracranial pressure and thereby prevents paradoxical herniation. Hence it’s wiser to perform CSF diversion or drainage in absolute necessity, and the amount of CSF drainage should be kept as minimum to prevent this phenomenon to develop.

5. Source of Funding

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6. Conflict of Interest

None

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