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Case Report

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Split-Thickness Decompression in the Management of Intracranial Pressure

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Conflict of Interest
The authors have no financial conflicts of interest.

ABSTRACT

Surgical management of elevated intracranial pressures due to stroke or traumatic brain injury has classically been through decompressive craniectomy (DC). There is significant morbidity associated with DC including subdural hygromas, syndrome of the trephined, and the need for subsequent cranioplasty. Alternative techniques including the hinged and floating craniotomy have shown promise though can still suffer from complications associated with an unsecured bone flap. We report a case in which a patient who presented with an acute subdural hematoma and associated midline shift that was successfully treated with decompression via thinning and re-securing of the bone flap in a "split-thickness decompression."

Keywords: Decompressive craniotomy; Intracranial pressure; Split thickness decompression

INTRODUCTION

Decompressive craniectomy (DC) was first described by Kocher in the early 1900's for management of elevated intracranial pressures (ICPs). Recent trials have demonstrated its utility in the improved survival of carefully selected patients after large territory strokes and traumatic brain injuries.[8,18] However, DC is associated with significant morbidity secondary to prolonged exposure of the underlying cerebrum to atmospheric pressures leading to subdural hygromas and syndrome of the trephined.[15] It also necessitates subsequent surgery for re-implantation of the bone flap, which may be subject to resorption or contamination.[3,7,19] More recent techniques such as the hinge craniotomy (HC) and floating craniotomy (FC) attempt to combine the benefits of decompression and immediate replacement of the bone flap by incompletely securing the bone flap after decompression allowing some degree of cerebral expansion post-operatively.[12,17] Complications from an incompletely secured bone flap in these alternative techniques, however, have been described.[11,14] In this report, we discuss a unique case in which a patient with an acute subdural hematoma (SDH) in the setting of a hyperostotic skull benefited from a split-thickness decompression, in which the inner cortex of the bone flap was drilled, allowing for the full securing of the bone flap.

CASE REPORT

A 78-year-old female with history of atrial fibrillation on therapeutic warfarin presented 2 days after a fall. She was noted to have a progressive decline in consciousness and associated confusion. Computed tomography (CT) scan of the head (CTH) demonstrated an 8 mm right sided acute SDH with 10 mm of midline shift (MLS) measured at the septum pellucidum. Hyperostosis throughout the cranium was also noted. On neurological exam, the patient was noted to be somnolent with a Glascow Coma Scale (GCS) of 8 (E2V2M4). The patient was started on prophylactic anti-epileptics and reversed with prothrombin complex concentrate and fresh frozen plasma with a normalization of the international normalized ratio to 1.1. The patient was brought to the operating room for craniotomy and SDH evacuation. A 10×10 cm craniotomy was performed and the dura was opened in a stellate fashion. After SDH evacuation and adequate hemostasis was achieved the brain was noted to be relaxed, the dura was approximated with a single central suture and a collagen matrix graft (DuraGen; Integra Lifesciences, Plainsboro, NJ, USA) was placed as an onlay. Given the patient's age, low suspicion for significant brain injury, and visual confirmation of the relaxed brain, a hemicraniectomy was not performed. The bone flap was replaced and fixed with titanium plates and screws. A subgaleal drain was placed. Post-operatively the patient improved neurologically to a GCS of 15 on post-operative day 1. Post-operative CTH demonstrated adequate SDH evacuation with an improved MLS to 5 mm.

On post-operative day 2 the patient was noted to be more somnolent with a GCS of 10 (E2V2M6). Repeat CTH demonstrated trace re-accumulation of the SDH but with a significant worsening of the MLS to 10 mm. The patient was brought back to the operating room where the prior incision was re-opened and bone flap removed. Upon re-opening of the dura and evacuation of the re-accumulation, the underlying cerebrum appeared to be pulsatile and not rapidly expanding. Given the change of MLS out of proportion to the reaccumulation of SDH, however, we attempted to expand the cranial vault without a complete hemicraniectomy. Since the flap was noted to be hyperostosed, the inner cortex of the bone flap was thinned using a high-speed drill. Once the bone flap was thinned to the outer cortex, it was replaced, and circumferentially fixed with titanium plates and screws. Post-operative CT showed filling of the cerebrum into the expanded cranial space and improvement of MLS to 5.8 mm. Electroencephalogram post-operatively was negative for seizures. The patient improved neurologically to a GCS of 15 by post-operative day 2 and over subsequent sequential scans the cerebrum continued to fill the decompressed area and show continued improvement in the MLS (**FIGURE 1**). The patient was ultimately discharged to acute rehabilitation with mild left hemiparesis but was otherwise neurologically intact.

The patient was seen in follow up at 6 months with excellent neurological recovery. She was noted to be independent with a modified Rankin score of one. Incision site was completely healed with no evidence of breakdown or infection. Evaluation of final CT scans demonstrate that roughly 5 mm of bone was removed from the bone flap allowing a maximal ipsilateral axial expansion 7 mm greater than the maximal contralateral axial expansion (**FIGURE 2**). No evidence of bone resorption was appreciated at 6 months.

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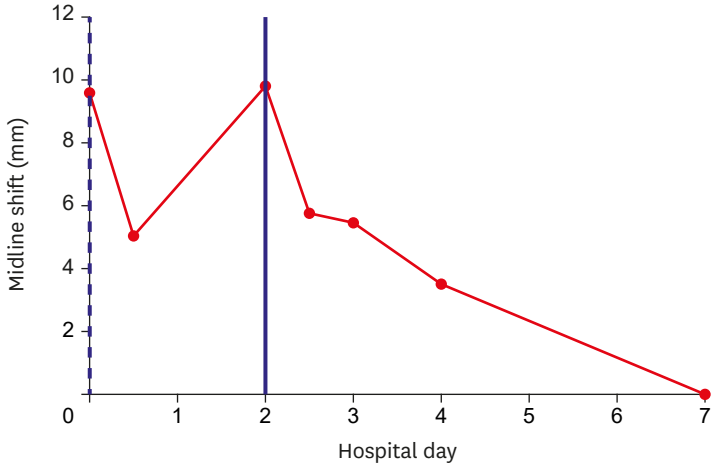

FIGURE 1. Midline shift trend throughout patient's hospital stay. Dashed vertical line: first craniotomy and subdural hematoma evacuation. Solid vertical line: second craniotomy and split-thickness decompression.

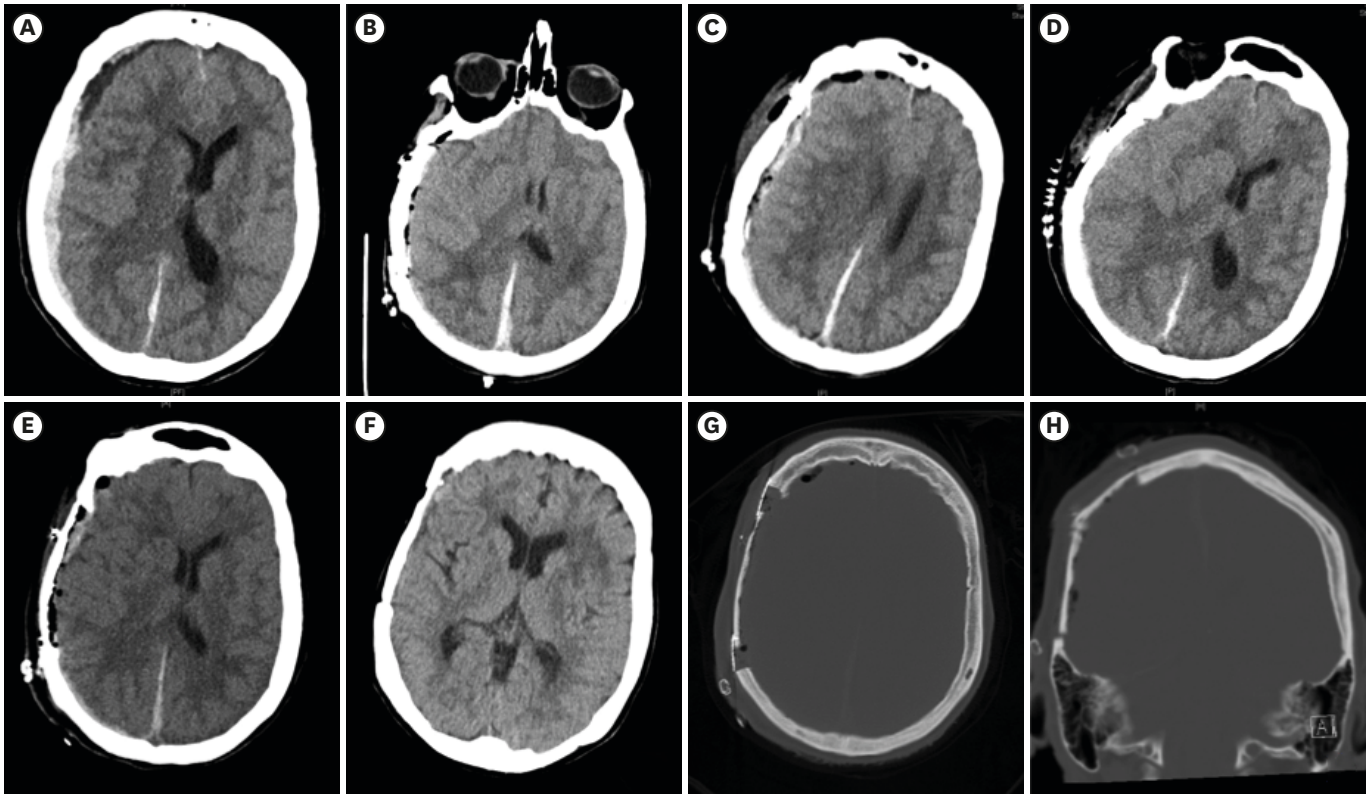

FIGURE 2. (A) CT on presentation demonstrating 8 mm right-sided acute SDH with 9.6 mm of MLS, (B) post-operative CT with evacuation of SDH and improvement of MLS to 5 mm, (C) mild re-accumulation of right-sided SDH with (D) 9.8 mm MLS on post-operative day 2. (E) Post-operative CT after re-evacuation and split thickness decompression showing immediate and (F) continued expansion on post-operative day 7 of the cerebrum into decompressed area. (G) Axial and (H) coronal bone window CT at 6 months demonstrating the volume of decompression allowed by drilling of the inner cortex.
CT: computed tomography, SDH: subdural hematoma, MLS: midline shift.

DISCUSSION

Decompression of the cranial vault has been a method of controlling elevated ICP in the setting of traumatic brain injury and stroke. In appropriately selected patients, decompression is a life saving procedure with potential improvement of neurologic

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outcomes.[8,18] Classically, DC was used to achieve adequate decompression. However, removal of the bone flap is not without morbidity. The rapid and largely unrestricted decompression of the cerebrum can lead to herniation through the defect causing further blossoming of contusions, as well as ischemic and hemorrhagic injury along the defect borders.[16] Subdural hygromas from altered cerebrospinal fluid dynamics can commonly occur after DC that may require surgical drainage.[20] Removal of the bone flap also necessitates a subsequent operation to replace the bone flap which involve potential morbidity including resorption, infection, and anesthetic risk.[3,7,19] Cranioplasty typically occurs 3–6 months after initial decompression during which the exposed cerebrum is subject to atmospheric pressure inducing neurologic changes and symptoms associated with syndrome of the trephined including headaches, hemiparesis, irritability, and vertigo.[15,16] Furthermore, though bone flap resorption is typically seen in the younger population, it remains a concern with post-DC cranioplasty as factors associated with resorption include delayed re-implantation, cryopreservation, and autoclaving prior to re-implantation.[2,4,13]

To address the associated morbidity of DC and subsequent cranioplasty, many alternative methods for decompression have been described most prominent of which are the HC and FC.[12] There are many variations to these approaches though the HC fundamentally involves the elevation of the bone flap, durotomy, followed by securing the bone flap in such a manner that allows an unsecured part of the bone flap to elevate as necessary with any continued cerebral swelling while the FC bone flap is loosely sutured circumferentially to the same effect.[1,5,6,11,14,17] Though these methods do not achieve the same amount of cerebral decompression nor do they allow for the same extent of cerebral herniation, they have been shown to achieve similar degrees of ICP control and overall outcomes when compared with DC. This may suggest that the amount of unrestricted cerebral expansion seen in DC is gratuitous and perhaps detrimental.[10,12] Replacing the bone flap often obviates the need for a second surgery for cranioplasty and its associated risks. By immediately reconstituting the cranial vault it also avoids the altered pressure dynamics associated with the development of subdural hygromas, syndrome of the trephined, and sunken flap. Nevertheless, there remain possible complications with the HC and FC approaches due to the incompletely secured nature of the bone flap including prolonged elevation or depression of the bone flap, prominence of intentionally proud cranial plating screws, and the need for a second procedure or operation, albeit smaller than post-DC cranioplasty, to fully secure the bone flap.[6,9,11,14]

Here we report a unique case in which preoperative and intraoperative information did not clearly demand a DC, but the postoperative course after the first surgery provided some indication that additional cranial volume might be necessary. In that context, we chose to perform a modified decompression that allowed the circumferential securing of the bone flap at the time of decompression and avoided the need for future cranioplasty. Drilling out the inner cortex provided adequate expansion of the cranial vault as a result of several factors. The uniform hyperostosis of the skull allowed for a greater potential volume expansion through thinning of the bone flap. Furthermore, though there was significant MLS and effacement of the sulci and gyri out of proportion to the re-accumulated SDH, rapid expansion of the cerebrum upon re-opening of the dura was not noted, indicating that maximal decompression may not be necessary. Postoperative serial imaging and neurological exam demonstrated expansion of the cerebrum into the decompressed area with improvement in consciousness and MLS suggesting that the extent of decompression was necessary and adequate. Though bone flap resorption was not seen in our follow up period, further investigation is necessary to determine specific risk factors for short and long term resorption in a split-thickness

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decompression. The split-thickness technique avoids the increased risk of bone resportion that is associated with explantation and re-implantation of DC, however, we nonetheless recommend mitigating techniques including obtaining maximal apposition of the bone flap with the native skull and avoiding fragmentation of the bone flap.

CONCLUSION

We describe a unique case in which a patient who presented with an acute SDH with hyperostosis of the skull benefitted from a split-thickness decompression simply through thinning of the bone flap. Immediate securing of the bone flap obviated the need for a subsequent cranioplasty and avoided the morbidity of a large cranial defect.

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