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Brief Report

Contralateral Expansion of Hemorrhagic Contusion in Patients Undergoing Decompressive Craniectomy: A Series of Two Patients

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Abstract

Contralateral formation and expansion of hemorrhagic contusion is a significant and rare complication following decompressive craniectomy. Decompressive craniectomy is an important surgical tool for management of raised intracranial hypertension secondary to various pathologies including trauma. These uncommon events are reported in the literature along substantive explanations and theories. We present two cases of road traffic accident (RTA) who following decompressive craniectomy developed expansion of contralateral hematoma. In this article, we are focus on the appearance and expansion of contralateral intraparenchymal contusion following decompressive craniectomy with a deep dive into the existing literature.

Keywords
- hematoma
- craniectomy
- decompressive
- contusion
- subdural

Introduction

Hematoma formation in the contralateral half of the brain is an uncommon event and has been reported earlier in several publications. Several theories and various mechanisms have been postulated regarding the pathophysiology leading to the development of the hematoma in the contralateral part of the brain in the form of acute extradural hematoma (EDH), subdural hematoma, and contusion. The current study focuses on the appearance and expansion of intraparenchymal bleeding in the form of contusion, which has expanded and led to significant morbidity in the patient following decompressive craniectomy.

Case Presentation

The summary of the cases is tabulated in ►**Table 1** and images are shown in ►**Fig. 1** (case1) and ►**Fig. 2** (case 2).

Discussion

Decompressive craniectomy is recommended for potentially life-threatening intracranial hypertension, and the mortality and morbidity following decompressive craniectomy after severe traumatic brain injury (TBI) are pretty high.[1] Bor-Seng-Shu E et al observed 50% mortality in 170 consecutive decompressive craniectomies over 5 years. The majority of the complications of decompressive craniectomies arise due to alterations in intracranial pressure (ICP), cerebral blood flow, and cerebrospinal fluid (CSF) circulation after the removal of a large area of the calvarium (►**Fig. 3**). Decompressive craniectomy induces augmentation of cerebral blood flow and cerebral metabolism, which transcranial Doppler and brain tissue oxygen probes have proven.[2]

Impairment of autoregulation in the early period of decompressive craniectomy leads to a state of maximal

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Table 1 Clinical summary of the two patients managed at our center

Sl. no.	Age/gender	RTA history	GCS at presentation	Initial CT	Surgery	Post-op CT	GCS at discharge
1	45/male	Two-wheeler rider: skid and fall	E1M3V2 with pupillary asymmetry (3 h posttrauma)	Left side acute subdural hematoma	Emergency left side decompressive craniectomy with evacuation of subdural	Adequate decompression with new onset right frontal contusion	E2M5Vt
2	32/male	Two-wheeler pillion rider: skid and fall	E1M4V2 with pupillary asymmetry (6 h posttrauma)	Left side temporal contusion	Emergency left side decompressive craniectomy with evacuation of contusion	Adequate decompression with new onset right frontal contusion	E3M5Vt

Abbreviations: CT, computed tomography; GCS, Glasgow coma scale; road traffic accident.

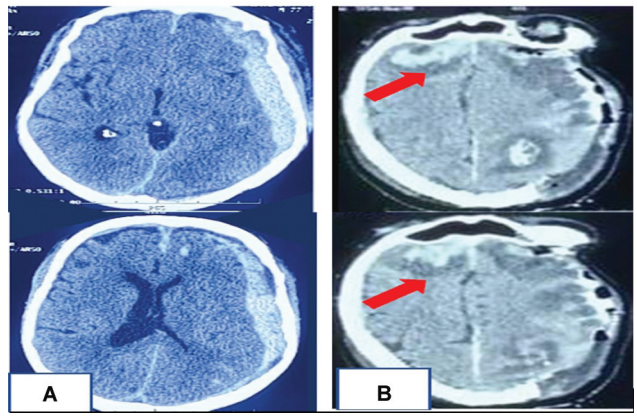

Fig. 1 (A) Preoperative computed tomography (CT) scan images suggestive of thick left frontotemporoparietal subdural hematoma with midline shift. (**B**) Postoperative CT scan images are suggestive of evacuation of the left subdural hematoma with blossoming of the right frontal contusion along with surrounding edema (*red arrow*).

vasodilation, resulting in hyperemia following relief of prolonged severe compression.[3] These changes in cerebral compliance and disturbed autoregulation are the underlying pathophysiological events that lead to complications following decompressive craniectomy. Bleeding, being the most common complication noticed during decompressive craniectomy, may be because of elevated ICP with altered cerebral hemodynamics.

The Blossoming of Contusion

The expansion of hemorrhagic contusion is inherent and has been observed on subsequent computed tomography (CT) scans following TBI.[4] The expansion of hemorrhagic contusions have been correlated with severity of TBI, 42% in TBI with median GCS score of 8 and 58% of patients with TBI were noticed to be in coma.

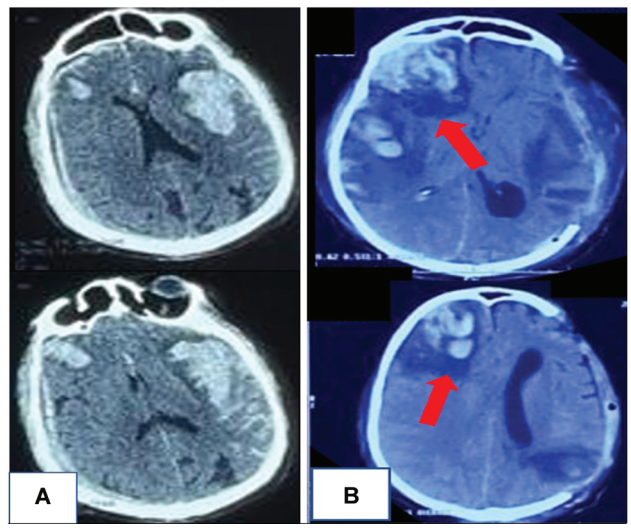

Fig. 2 (A) Preoperative computed tomography (CT) scan images suggestive of large left frontal contusion with midline shift. (**B**) Postoperative CT scan images are suggestive of evacuation of the left frontal contusion along with the appearance of fresh frontal and temporal contusions on the right side (*red arrow*).

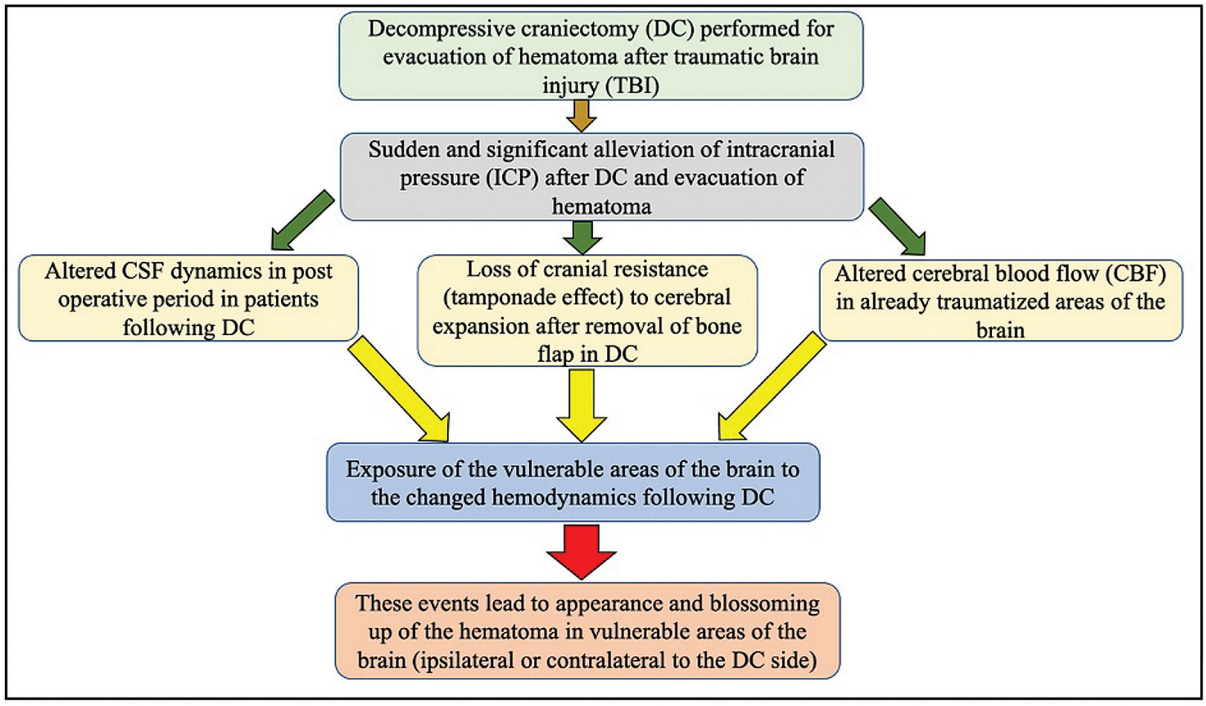

Fig. 3 Flowchart showing the pathophysiology and possible mechanisms responsible for the evolution and blossoming of the contusion after decompressive craniectomy. CSF, cerebrospinal fluid.

Flint et al, in a CT imaging study, noticed a remarkably high incidence of expansion of hemorrhagic contusion following decompressive craniectomy.[5] Posttraumatic contusions may be nonhemorrhagic, noticed as hypodense areas with foci of stippled hemorrhage without any measurable hematoma. On the contrary, hemorrhagic contusions have defined margins with measurable hematoma following TBI. De novo appearance or expansion of contusions was observed in 58% of the cases undergoing decompressive craniectomy, and several factors have been implicated in its evolution.

In the study conducted by Flint et al., 82% of the ipsilateral contusions blossomed, while 18% appeared on the contralateral hemisphere.[5] Rotterdam score provided an objective score regarding the risk associated with the expansion of hemorrhagic contusion. The volume of hemorrhagic contusion was correlated with the outcome of the patients who sustained TBI. The ROC analysis, with contusion volume >20 ml, demonstrated 57% mortality within 6 months of TBI, whereas the population having contusion volume <20 ml noticed only 15 enormously significant mortality ($p = 0.011$).[6]

In the present series, the presentation GCS scores were E1V2M3 (GCS score of 6) and E1V2M4 (GCS score of 7) without focal neurological deficits. The intraoperative period was uneventful; however, the contusion blossomed in the postoperative period, as observed on the first brain CT scan (done as a routine within 8–10 hours after surgery). Both patients were managed on antiedema and antiepileptic measures but had a prolonged hospital stay because of the appearance of a new contusion on the opposite side of the brain.

Evolution of Contralateral Mass Lesion

Reduction in ICP after decompressive surgery plays a vital role in the appearance of new mass lesions contralateral or remote in the decompressed hemisphere.[7]

External cerebral herniation through the craniectomy defect is among the early consequences of decompressive craniectomy. In the study by Yang et al, 26% of the patients developed external cerebral herniation following decompressive craniectomy. This cerebral herniation led to cortical venous compression, resulting in venous infarction.[8]

Conclusion

Anticipation of the occurrence/progression of contralateral hematoma is the most critical step in the management of TBI patients who undergo decompressive craniectomy. A thorough evaluation of the brain CT (with three-dimensional reconstruction) to identify all the skull fractures and potential bleeding sources is of utmost importance before embarking on surgery. Intraoperative guarded durotomy leads to sudden decompression of the brain, which prevents sudden loss of the tamponade effect. A follow-up scan also plays a critical role in managing TBI and early identification of rebleed.

Funding
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Conflict of Interest
None declared.

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