

Case Report

Extradural Hematoma Following Decompressive Craniectomy for Acute Subdural Hematoma: Two Case Reports Illustrating Different Mechanisms

Abstract

The occurrence of extradural hematoma (EDH) after decompressive craniectomy (DC) for traumatic brain injury is uncommon. We report two cases, one developing ipsilateral EDH and another developing contralateral simultaneous EDH and subdural hematoma after DC. The strategies to anticipate the occurrence of such concurrent hematomas (CH) are highlighted. We propose a subclassification of CH into “immediate” and “delayed,” based on their difference in clinical presentation, image findings, pathogenesis, and surgical management.

Keywords: *Complication, concurrent hematoma, head injury, surgery*

Introduction

Traumatic brain injury (TBI) is the major cause of mortality and morbidity worldwide, with an estimated 10 million affected annually. TBI, according to the World Health Organization, will surpass many diseases as the major cause of death and disability by the year 2020. TBI resulting in acute subdural hematoma (SDH) carries the maximum mortality and morbidity. Majority of literature acknowledge decompressive craniectomy (DC), as a method of choice, to combat raised intracranial pressure, in TBI. The occurrence of extradural hematoma (EDH), following DC for SDH, is rare with about 50 reported cases.^[1-11] We report two cases of EDH, one developing ipsilateral and the other contralateral, following DC and evacuation of SDH. The differences in clinical presentation, pathogenesis, image findings, and outcomes are highlighted.

Case Reports

Case 1

A 30-year-old male, presented after an hour following trauma, with a Glasgow Coma Scale (GCS) score of 6/15 and symmetric and reacting pupils. Computed tomography (CT) scan of the brain showed an acute SDH in the right fronto-temporo-parietal region with mass effect and midline shift [Figure 1a]. There were linear fractures of the ipsilateral

parietal, temporal, and occipital bones with pneumocephalus, in the vicinity, of the right transverse sinus [Figure 1b]. He underwent emergency craniotomy and evacuation of the SDH. A lax duroplasty, was done and the bone flap not replaced, as the brain was tense. He remained neurologically stable on ventilator support, with symmetric and reacting pupils. A routine follow-up CT scan of the brain after 24 h showed an ipsilateral EDH straddling the transverse sinus, causing mass effect and herniation of the brain through the craniectomy defect [Figure 1c]. He underwent emergency craniotomy and evacuation of the hematoma. His extended glasgow outcome score (GOS-E) was 8 (upper good recovery), at 6 months.

Case 2

A 30-year-old male, was admitted 1.5 hrs following trauma, with a GCS score of 7/15 and asymmetric but reacting pupils. A CT scan of the brain showed an acute SDH in the right fronto-temporo-parietal region with mass effect and midline shift [Figure 2a]. There was also a thin EDH and a speck of extradural air in the contralateral temporal region, underlying an undisplaced fracture of the parietal bone, and diastasis of the squamosal suture [Figure 2a and b]. He underwent emergency surgical evacuation of the SDH. A lax duroplasty, was performed, and the bone flap was not replaced, as the brain was tense. He

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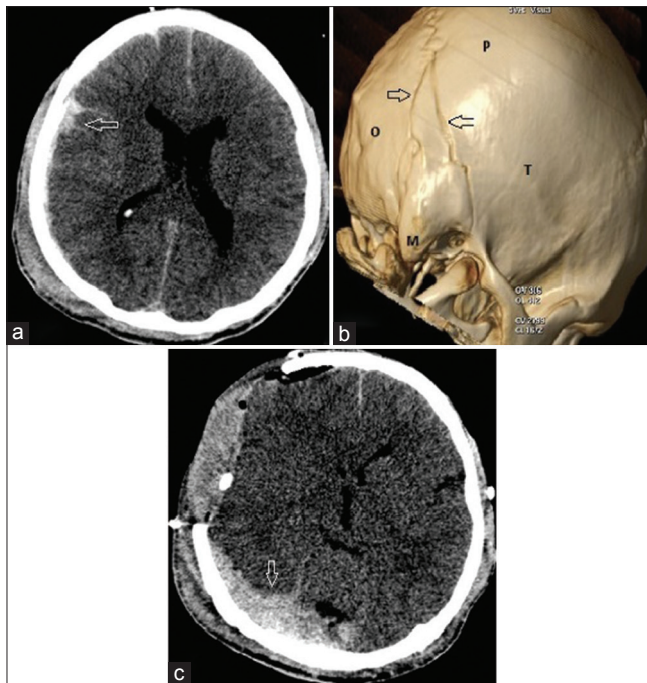


Figure 1: (a) Right subdural hematoma (arrow). (b) Right postero-lateral view of skull (three-dimensional reconstruction) showing the linear fractures (arrows); P – parietal; T – temporal (squamous); O – occipital; M – mastoid bones. (c) Right extradural hematoma straddling the transverse sinus (arrow)

remained under ventilator support, with the pupils equal and reacting to light. An hour later, his left pupil became dilated and nonreacting. A CT scan of the brain showed a large contralateral EDH and a thin SDH with midline shift, subfalcine, and uncal herniation [Figure 3a]. The EDH showed mixed densities and “swirl sign” suggesting active hemorrhage. He underwent an emergency craniotomy, evacuation of the left EDH, and bipolar coagulation of the actively bleeding posterior branch of the middle meningeal artery (MMA). The dura was not opened, as the SDH was thin and the dura was lax after the evacuation of the EDH. Postoperatively, the pupils were again equal and reacting to light. A follow-up CT scan of the brain showed no residual EDH and a thin acute SDH [Figure 3b]. His GOS-E was 7 (lower good recovery), at 6 months.

Discussion

The incidence of contralateral EDH, secondary to DC for TBI, ranges from 1.3% to 10%.^[2,12] Skull fractures, rupture of blood vessels, surgical decompression, coagulopathy, aggressive anti-edema measures, intracranial hypotension, cerebrospinal fluid fistula, and defective autoregulation are the various factors implicated.^[10] The absence of contusional hemorrhage and presence of remote skull fracture are independent risk factors for the development of EDH.^[1] Linear fracture in line with the axial CT scan images may be undetected [Figure 2c], but indirect evidence like pneumocephalus, thin EDH or SDH are useful pointers [Figure 2b]. Clinical indicators of a

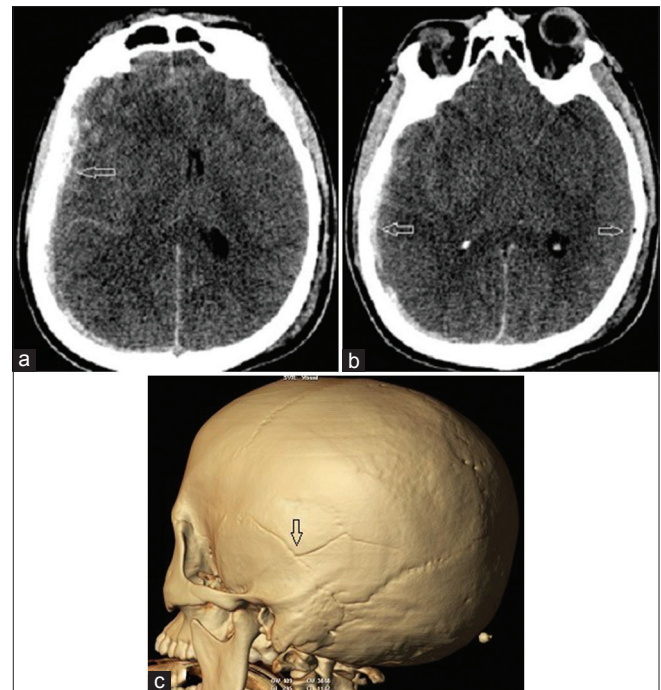


Figure 2: (a) Right subdural hematoma (arrow) causing mass effect and midline shift. (b) Right subdural hematoma (large arrow) and left thin extradural hematoma with speck of air (small arrow). (c) Three-dimensional reconstruction showing linear fracture of left parietal bone and diastasis of squamosal suture (arrow)

remote hematoma are progressive intra-operative brain swelling (76%) and new-onset asymmetry of pupils in the postoperative period.^[2]

In the first case, the cause of ipsilateral EDH was bleeding from the right transverse sinus, due to the loss of tamponade effect after DC, as evidenced by the fracture crossing the sinus. In the second case, the cause of contralateral EDH was arterial hemorrhage secondary to loss of tamponade effect after DC. The common factor in both situations was the presence of a skull fracture which is present in 81% of contralateral EDH.^[2]

Concurrent EDH may be, considered to be, immediate or delayed, based on the speed of deterioration, image findings, and pathogenesis. The management and the neurological outcome differ in each situation. Immediate concurrent EDH presents within the first 6 h after DC. Here, the source of bleed is arterial and may be associated with intra-operative external cerebral herniation and rapid onset neurological signs like the asymmetry of pupils. This is likely to carry a less favorable prognosis due to secondary insult as illustrated by the second case. Immediate postoperative CT scan, in a patient with unusually tense brain during the closure, may reveal the EDH. On the other hand, delayed concurrent EDH may occur any time after 6 h, following DC. Here, the source of bleed is usually venous, due to the loss of tamponade, and may not be associated with any new neurological signs. Here, the diagnosis is evident only on a routine

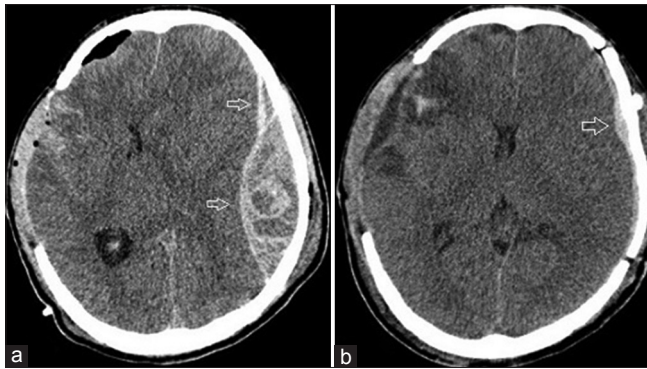


Figure 3: (a) Left subdural hematoma (upper arrow) and extradural hematoma (lower arrow) – Note the mixed densities in the extradural hematoma and “swirl sign” which are indicates active hemorrhage. (b) Postoperative computed tomography scan with no residual extradural hematoma - Note the more evident thin subdural hematoma (arrow)

postoperative CT scan after 24–48 h. This type probably carries a more favorable prognosis, as illustrated by the first case. Another unique feature demonstrated in Case 2, was the simultaneous presence of a contralateral acute SDH and EDH, after SDH evacuation.

Early complications of DC are the development of remote hematoma, contusion expansion, seizures, pseudomeningocele, external cerebral herniation, interhemispheric/contralateral subdural effusion and infection. Late complications are hydrocephalus and syndrome of the trephine. There are published literature citing the importance of guarded durotomy as an effective measure to reduce the incidence of immediate post-DC complications.^[2,13-15]

Another possible way of picking up an imminent contralateral EDH would be to extend the CT angiography study, commonly done as part of polytrauma evaluation, to include the brain, which may detect a leak from an MMA tear. This would enable a simultaneous exploration of the contralateral fracture segment to control the arterial bleed through a mini-craniotomy.

Conclusion

Strategies to anticipate the occurrence/progression of remote EDH is paramount in the management of TBI patients who undergo DC. A thorough evaluation of CT brain (with three-dimensional reconstruction) to identify all the skull fractures and potential bleeding sources, is valuable before embarking on surgery. Another strategy is gradual decompression of the brain by performing a guarded durotomy. A follow-up CT scan, after 24–48 h following DC, to look for delayed remote hematoma, is mandatory. An immediate concurrent EDH is primarily due to loss of tamponade over an injured meningeal artery, while a delayed concurrent EDH is probably due to loss of tamponade on an injured venous sinus or vein. An immediate concurrent EDH probably carries a poorer prognosis as compared to a delayed concurrent EDH.

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Conflicts of interest

There are no conflicts of interest.

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