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Original Article

Spectrum of remote site extradural hematomas following decompressive craniectomy: Does fracture always co-exist?

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ABSTRACT

Background: Remote-site extradural hematomas (EDHs) after decompressive-surgeries for traumatic brain injury (TBI) are rarely encountered. Typically, they form contralateral to the injured side, with an overlying fracture. We present a subset which developed EDH immediately after decompressive-hemi-craniectomy for TBI, most without an evidence of fracture, and not limited to contralateral location.

Methods: Nine such patients were retrospectively identified. Plausible mechanisms, management issues and outcomes have been discussed.

Results: All nine patients were victims of severe-TBI. Six did not have any skull-fractures. Eight showed hemispheric-injuries while one had bifrontal-contusions. In hemispheric-injuries, midline-shift was at least 8 mm except one with midline-shift of 6 mm. The EDH was straddling the midline in 2 (bifrontal-1, bi-occipital-1), and juxtaposed to the previous craniectomy in 1, apart from a contralateral-bleed in 6; all, except one, needed evacuation. In most patients, venous-source of bleed was identified. All had improved from their preoperative Glasgow coma scale (GCS) at follow-up.

Conclusion: A fracture need not always co-exist in EDH following decompressive craniectomy. However, an extra-caution is suggested in its presence. Given the need for surgical-evacuation in most patients and an inability to assess immediate postoperative-GCS in severely head-injured, a routine postoperative-computed tomography is recommended to avoid overlooking such potentially treatable condition.

Keywords: Decompressive hemicraniectomy, Extradural hematoma, Head injury, Traumatic brain injury

INTRODUCTION

Development of remote site extradural hematoma (EDH) following decompressive hemicraniectomies for traumatic brain injury (TBI) is a rare but potentially devastating complication with reported incidence in the range of 5–12%.[5,13,14] These hematomas are usually seen on the contralateral side, and in association with an overlying fracture. The related literature is limited to mostly sporadic case reports, and few small series, the latter comprising predominantly those with fractures.[1,5,13,14]

We present the clinical experience of a series of patients with remote site EDH post decompressive status, the majority of which did not show any fracture and not strictly contralateral, yet developed such complication. In this article, we attempted to analyze the following features of this infrequently encountered entity by keeping in mind three research questions: (1) do the hematomas always occur contralateral to the craniotomy site? (2) Is presence of an overlying fracture mandatory for such bleeds? and (3) Do all patients need evacuation?

MATERIALS AND METHODS

Setting

Ours is a retrospective, hospital-based, and observational study. After obtaining institutional ethical clearance, the data of patients who developed remote site EDH after decompressive hemicraniectomies for various TBIs between the period 2015 and 2019 were retrieved from the hospital records.

Data collection

Clinical and radiological data

Patients' demographic details, modes of injury, preoperative Glasgow coma scale (GCS), and injury pattern for which the decompressive surgery was performed were noted. These patients neither had any associated coagulopathy or thrombocytopenia nor were on anticoagulant/antiplatelet medications. Furthermore, they did not carry any other co-morbidity with bleeding diathesis.

Their preoperative computed tomography (CT) was evaluated for location and type of injury, extent of mass effect (midline shift and effacement of cisterns), Rotterdam score and presence of fracture at a remote site.[16]

Surgical data and postoperative protocols

The operative records of patients were looked for the presence of any significant brain bulge during surgery. All the patients underwent surgery with their head positioned on a head-ring; no skull clamps were used. A fronto-temporo-parietal craniectomy with loose duraplasty was performed, and the size of the bone flap was almost similar (approximately 15*12 cm) in all cases.

As a standard operating protocol, we routinely performed postoperative CT scans within an hour in all the patients. Furthermore, those with severe TBI or significant brain edema were shifted unreversed to our intensive care facility and electively ventilated. Hence, the interval GCS of these patients was not known.

Management of postoperative remote site EDH

A decision to operate upon the postoperative EDH depended on its size and the mass effect it produced. Patients who did not require an immediate clot evacuation were followed-up with serial scans and close observation of their clinical status; routine monitoring of intracranial pressure (ICP) was not a part of TBI management protocol in our set-up and was not performed. Their details such as location of bleed on CT, operative findings at second surgery, GCS at discharge, and Glasgow outcome score (GOS) at follow-up were noted.

RESULTS

A total of 9 (0.4%) were found to have remote side EDH after decompressive surgery performed for 2108 patients with TBI. [Table 1] shows the compiled data of the study patients. The operative indications for the primary decompressive craniectomy were subdural hematomas (SDH) and/or contusions with mass effect.

Baseline preoperative findings

The age varied between 22 and 55 years (mean, 34 years). There were seven males and two females. All patients had severe head injury (GCS ≤8) secondary to motor vehicular accidents.

Preoperative imaging

Of the nine patients, eight sustained hemispheric injuries, and one had bilateral frontal contusions. Among the patients with hemispheric injuries, seven showed complete effacement of cisterns (midline shift of 8–10 mm) and one had partially effaced cisterns (midline shift of 6 mm). The patient with bilateral frontal contusions showed chinking of ventricles with effacement of the basal cisterns. The Rotterdam score was measured as 5 in 7 (77.8%), and 4 in 2 patients (22.3%).

Of the nine patients, 6 (66.7%) had no associated skull fractures whereas 3 (33.3%) had evidence of fracture. In the latter, two patients had fracture at contralateral site, and one adjacent to the site of contusion.

Intra-operative findings at initial surgery

At surgery, persistent brain bulge was noticed in all even after removal of the post traumatic lesions (SDH/contusions).

Postoperative CT findings

Of 9 patients, the EDH was contralateral to the craniotomy site in six patients [Figures 1–3], bilateral and straddling across the midline in 2 (bifrontal-1, bi-occipital-1) [Figures 4 and 5], and adjacent to the craniectomy site in 1 [Figure 6].

Table 1: Baseline, operative, and outcome data of study patients.

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient 7	Patient 8	Patient 9
Age (years)	22	28	24	44	35	55	48	22	28
Gender	M	F	F	M	M	M	M	M	M
Type of injury	Left FTP* acute SDH† with contusions	Left FTP acute SDH with contusions	Bilateral frontal contusions	Left FTP acute SDH with contusions	Left TP contusions with SAH‡	Right FT contusions	Right FT acute SDH with contusions	Right FT acute SDH with SAH and contusions	Left FTP acute SDH with contusions
CT[a] findings	Thick SDH, cisterns effaced, midline shift of 9mm	Thick SDH, cisterns effaced, midline shift of 10 mm	Cisterns effaced, chinking of ventricles	Thin SDH, contusions, cisterns effaced, midline shift of 10mm	Thin SDH with SAH, cisterns effaced, midline shift of 8 mm	Thin SDH with contusions, cisterns partially effaced, midline shift of 6 mm	Thin SDH with contusions, cisterns effaced, midline shift of 9mm	Thick SDH with contusions, cisterns effaced, midline shift of 10 mm	Acute SDH with contusions, cisterns effaced, midline shift of 9 mm
Rotterdam score	5	5	4	5	5	4	5	5	5
Admission GCS[b]	E1 V1 M4	E1 V1 M2	E1 V1 M3	E1V1 M4	E1 VI M3	E1 V1 M4	E1 V1 M3	E1V1M3	E1V1M4
Intra-operative findings	Contusions not evacuated, brain bulge+	Contusions evacuated, brain bulge+	Contusions evacuated, brain bulge+	Contusions not evacuated, brain bulge+	Contusions evacuated, brain bulge+	Contusions evacuated, brain bulge+	Contusions not evacuated, brain bulge+	Contusions not evacuated, brain bulge+	Contusions not evacuated, brain bulge+
Postoperative site of EDH[c]	Right frontal	Bifrontal R>L	Left TP	Right TP	Bilateral occipital	Left TP	Left TP	Left TP	Right FTP
Evidence of fracture	Nil	Nil	Present adjacent to EDH	Nil	Nil	Present overlying the EDH	Nil	Present overlying the EDH	Nil
Discharge GCS	E2 VT M5	E2 V2 M5	E1 VT M5	E2 VT M5	E2 VT M4	E2 VT M5	E1 VT M4	E1 VT M4	E2 VT M5
GOS[d] at 6 months	3	4	3	3	2	3	2	2	3

*F: Frontal, T: Temporal, P: Parietal; †SDH: Subdural hematoma, ‡SAH: Subarachnoid hemorrhage, aCT: Computed tomography, bGCS: Glasgow coma scale, +: Present, cEDH: Extradural hematoma, dGOS: Glasgow outcome scale

Intra-operative findings at 2nd surgery

All the patients required surgical evacuation except one who had a small frontal EDH which was managed conservatively [Figure 3]. They were operated within 3 h of detection of hematoma. In most patients (77.8%), the EDH was secondary to a venous source. In the 2 patients who had initial CT evidence of skull fracture, an arterial spurter was the cause. Operative records revealed no evidence of additional fracture lines during surgery in the remaining patients.

Postoperative course and outcome

At discharge, all the patients showed improvement from their preoperative status. At the 6-month follow-up, three patients remained in persistent vegetative state (GOS, 2), five had severe disability (GOS, 3), and one was moderately disabled (GOS, 4).

DISCUSSION

Formation of remote site hemorrhage has been described after intracranial procedures, performed for both traumatic and non-traumatic causes.[3,4,10,15,16] For such event occurring after surgeries of non-traumatic indications, the pathophysiology postulated is sudden release in cerebrospinal fluid (CSF) pressure coupled with cerebral venous over drainage.[3] The bleed can occur anywhere (supra or infratentorial) irrespective of the site of the primary surgery. Here, we focus on a subset of patients who developed EDH after surgery for traumatic lesions such as acute SDH or contusions.

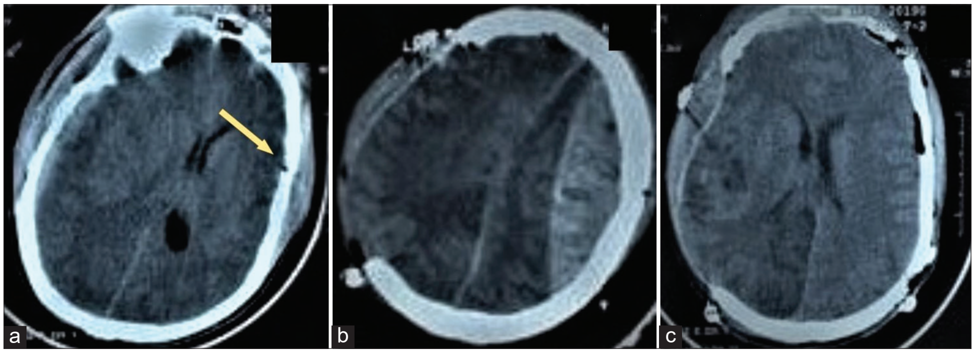

Figure 1: (Contralateral EDH with fracture) (a) Preoperative CT shows right frontotemporal acute SDH with midline shift, and contralateral frontotemporal linear fracture (arrow). (b) Postoperative CT shows huge contralateral EDH underneath the fracture. (c) Repeat CT postevacuation; however, the patient developed right posterior cerebral artery infarct. EDH: Extradural hematomas, SDH: Subdural hematomas, CT: Computed tomography.

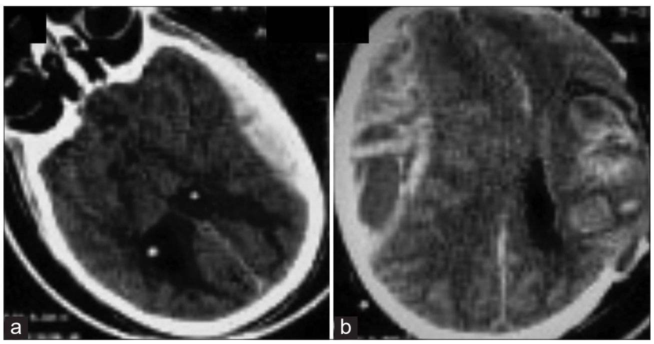

Figure 2: (Contralateral EDH without fracture). (a) Baseline CT shows left hemispheric SDH with mass effect. (b) Postoperative CT following decompressive craniectomy shows left sided blossoming contusions, and newly developed large contralateral EDH. EDH: Extradural hematomas, SDH: Subdural hematomas, CT: Computed tomography.

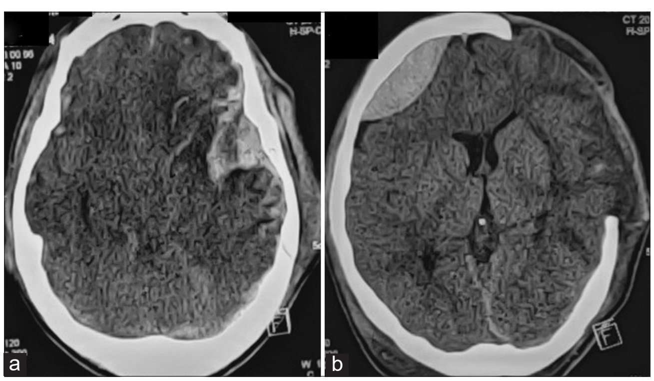

Figure 3: (Contralateral EDH without fracture) (a) Preoperative CT shows left fronto-temporo-parietal acute SDH and underlying contusions with mass effect and completely effaced cisterns. (b) Postoperative CT shows contralateral small right frontal EDH; the patient was managed conservatively. EDH: Extradural hematomas, SDH: Subdural hematomas, CT: Computed tomography.

Postoperative EDH after decompressive craniectomies for TBI can present immediately or in delayed fashion (hours to

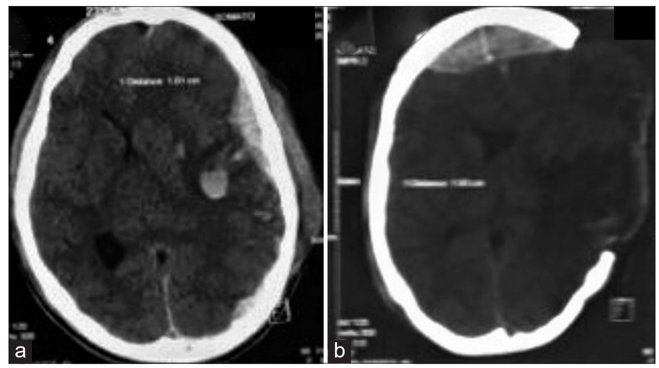

Figure 4: (Bifrontal EDH without fracture). (a) Preoperative CT shows left hemispheric SDH with underlying contusions, subarachnoid bleed and the presence of severe midline shift. (b) Postdecompression scan shows freshly formed bifrontal EDH. EDH: Extradural hematomas, SDH: Subdural hematomas, CT: Computed tomography.

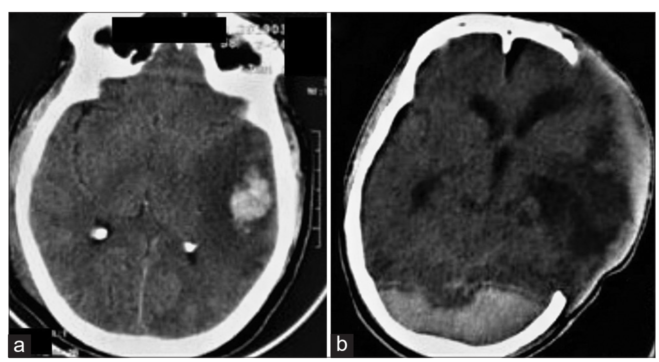

Figure 5: (Bi-occipital EDH without fracture). (a) Left temporal contusion with mass effect in baseline CT. (b) Postdecompression, patient developed bilateral occipital EDH. EDH: Extradural hematomas, CT: Computed tomography.

few days).[4,11,13] A number of pathomechanisms have been proposed in relation to the occurrence of EDH.[1,15,17] Sudden decrease in ICP with release of the tamponade effect has been suggested as the primary cause. The brain shift that happens after surgical decompression opens up the contralateral bleeding source, usually arterial or at times venous, which was contained by the initial brain edema or clot. Other factors implicated are aggressive anti-edema measures, CSF fistula, and systemic hypotension that can cause intracranial hypotension.[15,17]

In the previous studies, certain predictors of postoperative EDH have been reported, the most important of which is the presence of skull fractures [Table 2].[4,5,8,10,13,15,17-19] Most commonly, the hematoma occurs on the contralateral calvarium, usually with an overlying fracture, the most consistent finding. A younger age, probably related to easiness of dural stripping, and fractures involving multiple calvarial bone plates are identified as independent risk factors in the presence of fractures.[17] The severity of head injury and preoperative Rotterdam CT score have also been correlated with such remote side hematoma formation.[16] A craniectomy, particularly larger one, as compared to a craniotomy procedure increases the chances of EDH formation.[7,16] Noticeably, intraoperative brain swelling (76%) can also potentially hint such unforeseen complication.[15]

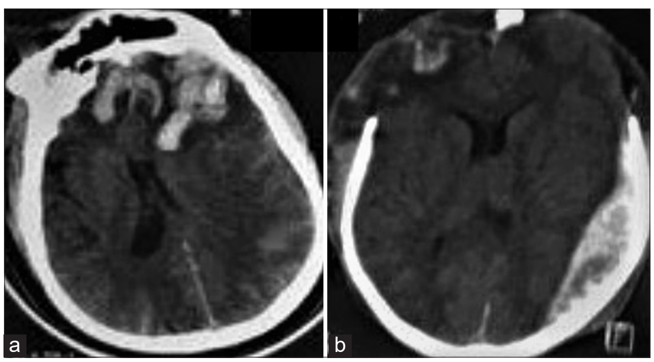

Figure 6: (Adjacent site EDH without fracture) (a) This patient was operated for frontal contusions (L>R) with chinked ventricles. (b) There is newly formed left temporo-parietal EDH contiguous with the previous craniectomy site. EDH: Extradural hematomas.

In prior reports, linear fracture in association with an EDH has been observed in about 80% of cases.[15] Contrary to that of the existing literature, we noticed fracture overlying a contralateral EDH only in two patients (22.2%), with arterial injury as the source of bleed. In approximately 20–30%, the associated fractures have been overlooked on the preoperative radiology, and detected only during craniotomies.[16] However, we did not encounter any such findings in our remaining patients.

As in previous studies, an evidence of an increasing mass effect on CT/Rotterdam score corresponded to development of postsurgical EDH.[14-16] We noticed, in seven of our eight patients with hemispheric SDH/contusions, imaging showed a midline shift of >8 mm with complete obliteration of the basal cisterns. However, these patients differed by the fact that they did not sustain any associated fracture lines. A plausible mechanism of contralateral EDH formation was a fresh venous tear/rupture secondary to dural stripping incited by sudden decompression and shift of an edematous brain. Another remote possibility is flaring up of a pre-existent but contained contra-coup venous injury, in-apparent on the initial scan.

A couple of patients in the present series developed midline EDH. The likely source of hemorrhage was venous rupture because of the stretch of the diploic/bridging veins at their entry into the dural venous sinuses. Dural sinus thrombosis as a cause of spontaneous EDH has also been reported, and can occur even in the absence of an overlying fracture.[9] A similar etiology could also possibly explain the occurrence of a midline EDH in our patient. The resultant venous hypertension could have manifested as bilateral extradural bleed after the release of brain tamponade. In the absence of definitive evidence of venographic studies, the latter mechanism, however, is only speculative.

One of our patients developed EDH who presented adjacent to the previous craniectomy defect. Excessive stripping of dura during its separation around the burr holes can predispose to such bleed at the proximity of the craniectomy.

Table 2: Major series reporting extradural hematoma following decompressive craniectomy.

Author, year	No. of cases	Skull fracture	Brain swelling	Avg. admission GCS[a]	Avg. GOS[b]
Piepmeier *et al.* (1982)	3	Present in all	Present in 1	4.3	2
Feuerman *et al.* (1988)	3	Present in all	Present in all	6.3	2
Matsuno *et al.* (2003)	4	Present in 1	Present in all	4	4.25
Su *et al.* (2008)	12	Present in all	Present in 10	5.8	2.58
Shen *et al.* (2013)	5	Present in all	Present in 3	3.8	2.6
Flordelís Lasierra *et al.* (2013)	11	Present in all but 1	NA	5.3	2.54
Kim *et al.* (2015)	24	NA	NA	11.3	NA
Su *et al.* (2016)	13	Present in all	NA	6.9	Favourable- 7 Unfavourable- 6
Present study	9	Present in 3	Present in all	5.4	2.7

[a]GCS: Glasgow coma scale, [b]GOS: Glasgow outcome scale, NA: Data not available

The commonly used hitch sutures can limit relatively small EDH between the adjacent bone and stripped dura. However, when the dura is inadvertently stripped to a large extent, these sutures *per se* can compartmentalize the hematoma which subsequently can evolve.

Rarely, EDH related to the pin site of the skull clamps has been reported.[20] However, this association is unlikely in our patients as they were operated only on a head ring.

Because abrupt decompression is considered the inciting event in the origin of contralateral EDH, previous authors have suggested certain precautionary measures for a gradual reduction in ICP.[2,12] An initial burr hole before proceeding with decompressive craniectomy, and netted incisions over the dura are some of them. The final outcome in these patients, to a large extent, depends on the initial GCS. Better outcome has been reported following immediate postoperative evacuation compared to a delayed surgery. It has been observed both in the present and previous reports that most patients with postoperative EDH need surgical evacuation.[4,15,17] This fact underscores the need for an early detection and appropriate intervention of a postoperative EDH. Although the initial primary impact is no longer modifiable, at least the secondary insult due to an EDH can be minimized.

A high index of suspicion is the only key to identify new-onset contralateral extradural bleed following decompressive craniectomy. Persistent raised ICP in the postoperative period indicated by a clinical examination (hypertension and bradycardia) can clue this unprecedented complication. Such contralateral EDH phenomenon reiterates the importance of postoperative routine ICP monitoring and a thorough neurological assessment in the neuro-intensive care unit for new onset anisocoria.[6,13] However, by the time, it is detected, the crucial time period might be missed. Furthermore, in a resource restraint set up, routine ICP monitoring may not be possible. The other viable option which allows early detection of the hematomas is performing an ultrasound intraoperatively in susceptible patients such as those with persistent brain bulge.[5] Given the fact that patients operated for severe TBI are invariably shifted unreversed and electively ventilated, as part of ICP reduction measures in many centers, we prefer obtaining routine immediate postoperative CT scans in these patients.

The study is limited by its retrospective nature. Another limitation is a less number of patients because of which statistical derivations could not be drawn.

CONCLUSION

In summary, the subset of patients in this series comprised TBI patients who developed postoperative EDH at various sites, predominantly without any associated fracture. Increased mass effect and brain bulge during surgery can possibly predict such a complication. Although the occurrence of remote site EDH may be infrequent, the only way to pick up these potentially treatable lesions is by performing immediate routine postoperative CT, at least in severely head injured patients.

Compliance with ethical standards

The procedures performed were in accordance with the ethical standards of the institutional ethics committee and with the 1964 Helsinki declaration and its later amendments. For this, type of study formal consent is not required.

Declaration of patient consent

Institutional Review Board (IRB) permission obtained for the study.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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