Traumatic Coronal Suture Diastasis and Contre-Coup Epidural Hematoma

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Acute epidural hematoma involves blood accumulating between the inner table of the skull and the dura mater, and complicates 1-2% of head injured patients.[1] The pathophysiology underlying this lesion involves trauma to vessels in proximity to a skull fracture with consequent bleeding, hence the hematoma is most frequently ipsilateral to the trauma site. While these lesions have been described to enlarge and cause delayed neurological sequelae following an initial concussive injury, the lucid interval, this is not the typical mode of presentation.[2] A consistent level of consciousness, either persistent coma from the time of injury or alertness without deterioration, is far more typical of this pathology. Indications for surgical evacuation include size of the epidural hematoma, extent of subfalcine herniation, patient Glasgow Coma Scale (GCS), and the presence of neurological deficit.[3] Owing to the coagulated nature of the blood, a craniotomy is normally required to evacuate the hematoma and reapproximate the dura to the bone flap to obliterate the epidural space and prevent reaccumulation. A recent institutional review by Tallon and coworkers[4] of 70 patients presenting with extra-axial hematoma revealed isolated epidural blood in 33%, isolated subdural blood in 49%, and both in remainder. Their study included 23 patients with isolated epidural hematoma, of whom two died and three experienced moderate to severe disability. A contemporary literature review to establish guidelines for epidural hematoma management by Bullock and coworkers[3] describes traffic-related accidents as the majority cause (53%), with falls (30%) and assaults (8%) contributing lesser frequency. They found overall mortality among patients undergoing surgical evacuation for these lesions to be approximately 10%, with the initial computed tomography (CT) scan features of clot thickness, hematoma volume, and midline shift prognosticating outcome.

Case Report

A 28-year-old, previously healthy female bicyclist presented to the emergency department after a non-helmeted fall onto the right side of her head. She did not lose consciousness or seize, and ambulated independently after the incident. Clinical history included severe bilateral headache and nausea without vomiting. Physical examination demonstrated GCS 13 (eyes open to voice, verbalizing comprehensible words but incoherent sentences, and moving purposefully and to command), pupils that were equal and reactive at 4 mm, and no focal neurological deficit. The right-sided injury site demonstrated a subcutaneous hematoma, with the right eye showing significant periorbital edema and a supraorbital laceration.

Emergent CT scan (Figure 1) demonstrated a 9 mm left frontal epidural hematoma with 4 mm of midline shift. The scout film (Figure 2) and bone window (Figure 3) both showed coronal suture diastasis crossing the midline. No other fracture was seen.

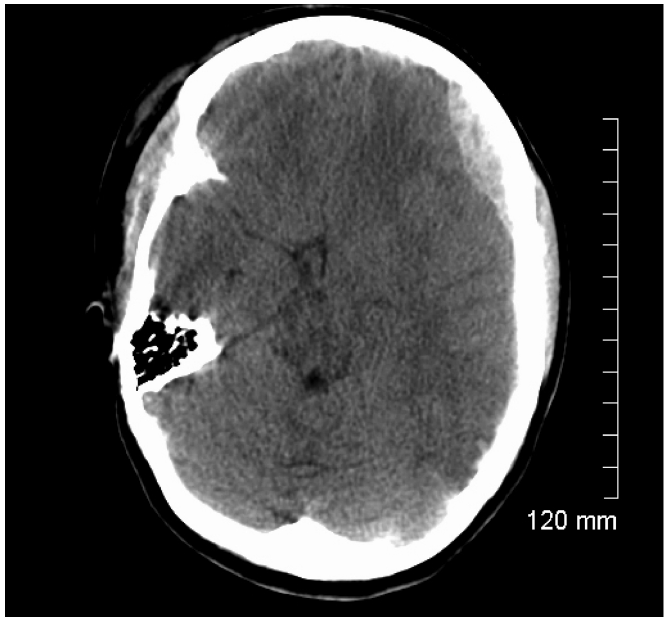

Figure 1: *Axial soft-tissue windowed CT scan at time of presentation demonstrates the right sided extracranial soft-tissue injury with associated left sided epidural hematoma.*

The patient underwent emergent craniotomy for evacuation of the frontal epidural hematoma. Coronal suture diastasis was demonstrated at the time of operation, underneath which an epidural hematoma was centered over a small, injured anterior branch of the middle meningeal artery. Hemostasis was achieved by bipolar electrocoagulation to the proximal middle meningeal artery as well as the putative branch. There were no post-operative complications and CT scan demonstrated effective hematoma evacuation.

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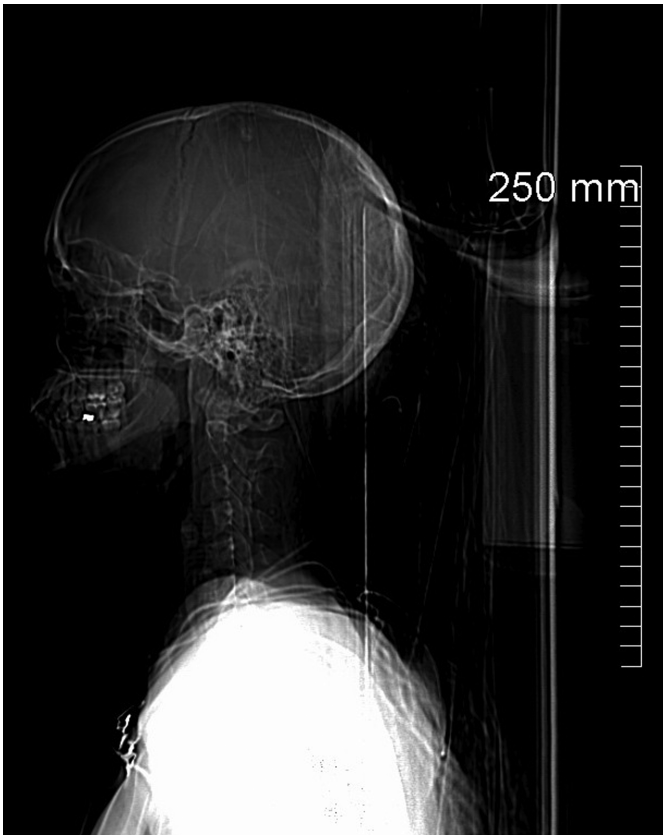

Figure 2: *Skull scout film at time of presentation demonstrates diastasis of the coronal suture.*

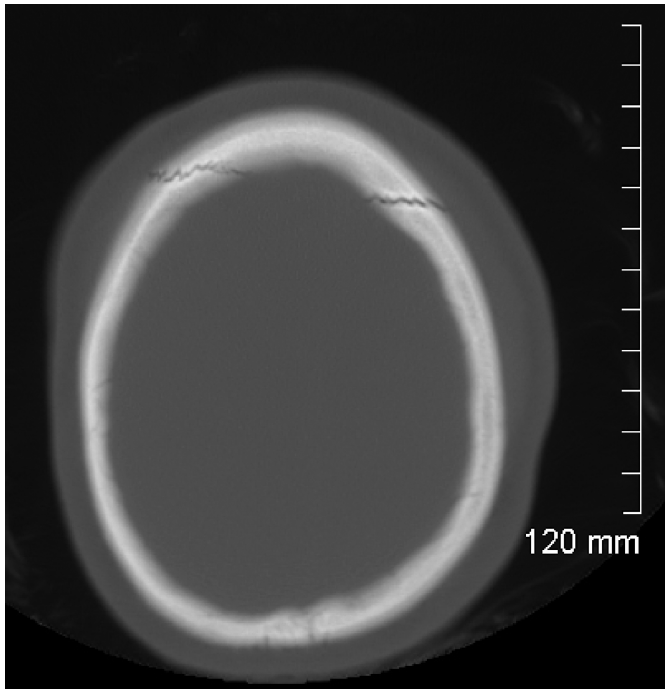

Figure 3: *Axial bone-windowed CT scan at time of presentation demonstrates diastasis of the coronal suture.*

Discussion

Acute epidural hematomas nearly always occur at the coup site of injury, with a proximate fracture site identified in the majority of cases.[5] The force of the initial trauma causes a transient loss of consciousness in approximately one third of patients. Enlargement of clot after the initial event has been reported with variable incidence, arising from high arterial pressure that strips the dura away from the bone. Progressive herniation classically leads to declining level of consciousness, ipsilateral oculomotor nerve palsy, and contralateral hemiparesis. Computed tomography imaging reveals a biconvex lens-shaped hyperdense lesion, ipsilateral to the side of injury, limited in extent by cranial sutures, that may rarely cross dural folds such as the falx or tentorium. The most common location is the temporoparietal region as a consequence of disrupting the middle meningeal artery under a fracture of the temporal squamosa. Venous bleeding can also lead to epidural hematomas in the frontal region or the posterior fossa, although their shape and acuity of course is far less typical.

The decision to operate is based on clinical neurological status including alertness, focal deficit, or progressive deterioration, hematoma size, and hematoma progression on sequential CT scans. There is literature support surgical evacuation of acute epidural hematomas in patients who are symptomatic or those with asymptomatic lesions of size greater than 15 mm or with associated effacement of the basal cisterns and midline shift.[3,5,6] Non-surgical management of epidural hematomas may require frequent clinical evaluations often furthered by radiographic follow-up. Bullock and coworkers[7] describe a series of 123 patients with acute epidural hematomas, of whom 10% were treated non-surgically. Inclusion to this group required non-comatose (GCS was between 12 and 15 at presentation), no neurological deterioration while admitted, hematoma volume of less than 40 mL (average was 26.8 mL), and less than 15 mm of midline shift on CT scan. Resolution with complete hematoma absorption occurred over 7.25 weeks, requiring a mean of 3.5 CT scans. Similarly, Cuccinello and coworkers[8] describe 57 patients managed conservatively for acute epidural hematomas, admission GCS of 14-15 in the majority with only four patients having GCS of between 10 and 14. The maximum thickness of these hematomas ranged from 6 to 12 mm, and midline shift of 4 mm was observed in only one. Resolution with complete hematoma absorption occurred between one and three months, utilizing a standard monitoring regiment of between five and six CT scans. In the setting of the acutely deteriorating patient, the role of emergent skull trephination remains disputed with regards to both safety and benefit.[9,10] This modality is purported to temporize high intracranial pressure while a patient may be transferred to a neurosurgical centre, but recent investigation has expanded to the application of negative pressure clot evacuation with success in avoidance of craniotomy for 11 of 13 patients in a small series of selected patients by Liu and coworkers.[11]

A literature review of isolated supratentorial contre-coup epidural hematomas without a coup lesion showed this

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Table: Literature review of trauma patients with isolated contre-coup epidural hematoma

Author	*Age, Sex*	*Injury*	*Site of EDH*	*Craniotomy*	*Bleeding Vessel Identified*
Okamoto, 1983[13]	51, F	R Occipital	L Frontal	Yes	No
Motohashi, 2000[12]	59, F	R Occipital	L Frontal	No	No
Current	25, F	R Frontal	L Frontal	Yes	Yes

EDH=epidural hematoma

pathology to be quite rare. Only two other cases satisfied these criteria and they are summarized in the Table with our case for comparison,[12,13] with two further cases involving isolated contre-coup posterior fossa epidural hematoma after forehead impact.[14,15] Conversely, there are several other cases in association with association with coup epidural hematoma[16,17], subdural hematoma[18], or contusion[19,20].

In the two previously reported cases, no clear mechanism was found to underlie the development of these lesions. In the patient who needed craniotomy, the author recounted only oozing from small, non-specific dural vessels. Theorized mechanisms have been predicated on the fact that frontal dura is only loosely attached to the inner table of the skull. Following occipital trauma in those cases, negative pressure in the frontal region may detach this dura and tear the interposed surface vessels.[21]

Our case is unusual with a clear mechanism found to explain injury to a branch of the contralateral middle meningeal artery. Traumatic diastasis of the coronal suture following right frontal trauma lacerated an anterior branch of left middle meningeal artery. The associated arterial bleeding resulted in an epidural hematoma that required craniotomy followed by coagulation of the lacerated branch and the proximal artery as a surgical remedy.

We report an unusual presentation of acute epidural hematoma contralateral to the site of external injury in association with traumatic coronal suture diastasis. While most literature surrounding isolated contralateral epidural hematoma has theorized mechanisms accounting for a small vessel bleeding source, our patient had clearly identifiable pathology with a putative mechanism. Traumatic coronal suture diastasis associated with an arterial laceration provided for a clinically significant epidural hematoma. Craniotomy, hematoma evacuation, and subsequent hemostasis provided our patient with excellent outcome.

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