CASE REPORT

Traumatic Epidural Hematoma Crop up Following Decompressive Craniectomy for contrecoup Acute Subdural Hematoma

Baha Eldin Adam, M. Emin Eminoğlu

Department of Neurosurgery, Medeniyet University, Göztepe Education and Research Hospital, Istanbul, Turkey

ABSTRACT

We report a case of a Syrian refugee patient with head trauma after falling backward from a chair accidentally, presented in emergency unit with loss of consciousness. He was found to have subdural hematomas (SDHs) on the right frontoparietotemporal region of the brain. He underwent operation for the evacuation of the SDH and early post-operative computed tomography (CT) showed 15 mm contralateral epidural hematoma (EDH) in the left occipital region and the posterior fossa regions which was not clearly detected in pre-operative cranial CT. The EDH thought to be due to dural sinus bleeding that unexpectedly crop up after the evacuation of the SDH and subsequently decrease in intracranial pressure and ease the tamponade pressure effect of SDH on contralateral left occipital might prepare the ground for EDH. The second operation is immediately done that the EDH was evacuated. In this case, we discussed the possibility of developing a combined EDHs on the opposite side of the evacuated hematoma, detection of SDH preoperatively which is unusual phenomenon in relation to EDHs which usually caused by tears in arteries, and it progresses dramatically very fast with first traumatic impact. Also to draw attention to skull trauma especially in the occipital region, because unexpected late epidural hematoma can be detected surprisingly at this region, as result of hemorage due to fractured skull bone and dural sinuses lacerations.

Key words: Contrecoup acute subdural hematoma, Late epidural hematoma, Traumatic dural sinus laceration

INTRODUCTION

A subdural hematoma (SDH) and epidural hematoma (EDH) are a type of hematomas usually associated with traumatic brain injury (TBI). In 2009, the US Centers for Disease Control and Prevention estimated that at least 2.4 million emergency department visits, hospitalizations, or deaths were related to a TBI, either alone or in combination with other injuries.[1] In SDH, blood gathers between the dura mater and the brain. Usually resulting from tears in bridging veins which cross the subdural space, may cause an increase in intracranial pressure (ICP), compression, and damage to delicate brain tissue. Traumatic acute SDHs are the most lethal of all head injuries and have a high mortality rate, if they are not rapidly treated with surgical decompression, the mortality rate was found to be predominantly influenced by the pre-operative state of consciousness, associated brain lesions, and the duration of the time interval between onset of coma and surgical decompression. When this interval exceeded 2 h, mortality from SDH rose from 47% to 80%.[2] Chronic SDHs, however, have better prognosis if properly managed. In contrast, EDHs are usually caused by tears in arteries, resulting in a buildup of blood between the dura mater and skull. The condition is potentially deadly because the buildup of blood may increase pressure in the intracranial space, compress delicate brain tissue, and cause brain shift and herniations. The mortality rate of acute EDHs is 17% if intervented within an interval under 2 h after coma and 67% of good recoveries compared to 65% mortality and 13% of good recoveries after an interval of >2 h. They are most severe if associated with cerebral contusions, age, and concomitant injuries of other body regions.[2]

Address for correspondence:
Baha Eldin Adam, Department of Neurosurgery, Medeniyet University, Göztepe Education and Research Hospital, Istanbul, Turkey. E-mail: dradamsud@yahoo.com

However, EDH following decompressive surgery for acute SDH is an uncommon situation with only few cases previously reported in the medical literature.[3-5] We report a patient with SDH who have been operated and postoperatively found to have EDH in early post-operative control brain computed tomography (CT). The EDH was discovered incidentally and surprisingly because normally what we expected, acute subdural bleedings are usually venous and therefore develop in slow fashion than the typically arterial bleeding of an epidural hemorrhage. We comment on the why SDH detected first, the rare location of the EDH in the occipital and the posterior fossa regions, the tamponade effect of SDH that blocked the bleeding tendency by pressing the dural sinus veins against overlying calvarial bone on the contralateral side on the left occipital area and whether the left occipital EDH is of artery or sinus source of origin.

CASE REPORT

A 33-year-old man was brought to the emergency department said to be fell backward from a chair and hit the back of the head on the ground. He had abrasions on the left occipital but not on the right temporofrontoparietal regions of the scalp where there was SDH. On examination, his Glasgow coma score (GCS) was E1 M4-5 V1 (6-7/15). He had paucity of the right-sided limb movements to painful stimuli; his left pupil was larger than the right and not reacting to light. This was clinically suggestive of left uncal herniation or as result of primary injury to its parenchyma. The injury ascertained to be occurred approximately 1–2 h before. He was investigated with an urgent CT scan of the head. The CT scan showed a right temporofrontoparietal SDH causing significant mass effect on the lateral ventricles associated with severe diffuse cerebral edema and effacement of all the basal cisterns, hence, 1.5 cm midline shift on pre-operative brain CT of the head [Figure 1a-d]. He was shifted to the operating room directly from the emergency unit within 50 min of being brought to the hospital (an analysis of the records revealed that this was the time taken for doing the CT scan, drawing blood for cross-match, and an urgent head shave). He underwent emergency right frontotemporoparietal craniotomy. During surgery, a linear fracture of the temporal bone was noted. On elevating the bone flap, dura was opened a 1.5–2 cm thick SDH was encountered and was evacuated. The source of the bleeding was a laceration of the small cortical veins, which was controlled with gelatin sponge tamponade and meticulous coagulation. However, he remained comatose, with a GCS of E2 M5 V2 (9/15) and the pupillary asymmetry reverted. A post-operative control CT axial image showed complete evacuation of the SDH with reversal of the midline shift [Figure 2a-d]. However, cerebral edema persisted but surprisingly a 2–2.5 cm thick acute EDH was detected on the left occipital lobe with extension into the posterior fossa, the bleeding was expected to be leaked from the left transverse sinus or the posterior cerebral artery territories. The second surgery was decided and the patient immediately underwent the second operation and the EDH was evacuated. During the operation, we observe that bleeding is of the left transverse sinus origin. After intensive care unit and service follow-up the patient consciously and fed orally, the patient discharged with a right hemiparesis. In follow-up, the patient neurological deficit recovers completely with the craniotomy bone flap safeguarded in abdominal subcutaneous tissue which is deserved to be used for the cranioplasty flab 6 months later.

In Figures 3a-d and 4a-e, post-operative CT showing resolution of midline shift, no edema, or left occipital epidural hematoma.

DISCUSSION

The most common cause of intracranial EDH is traumatic, although uncommonly spontaneous hemorrhage is known to occur, it is caused by adjacent infections, dural vascular malformations, tumors, and disorders of blood coagulation.[6] Traumatic hemorrhages commonly result from acceleration-deceleration trauma and transverse forces.[7] The majority of bleeds originate from pterion region which overlies the middle

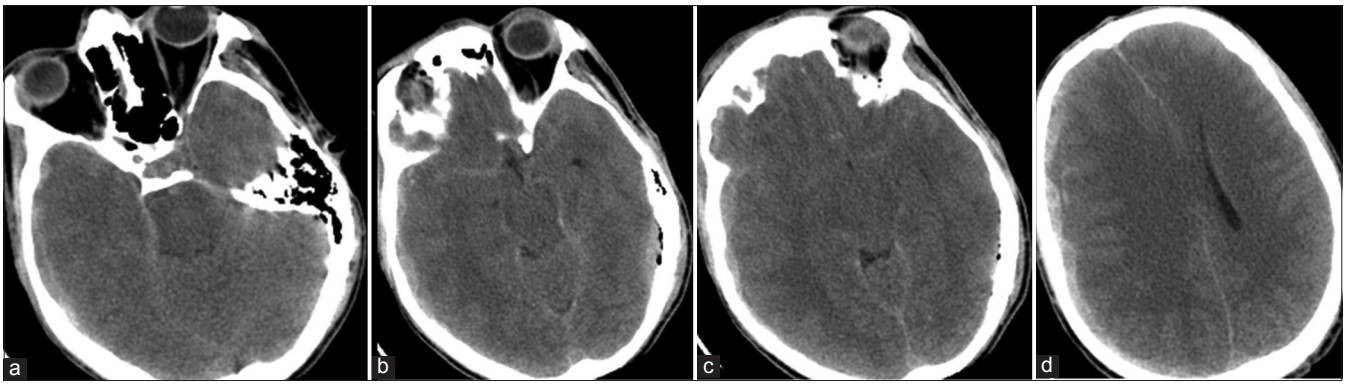

Figure 1: (a-d) Pre-operative axial plan cranial computed tomography was taken in the emergency service, showing early preoperative traumatic acute subdural hematoma diffuse edematous brain parenchyma and midline shift, no clear evidence of epidural hematoma on the left occipital region or on the left side of the posterior fossa

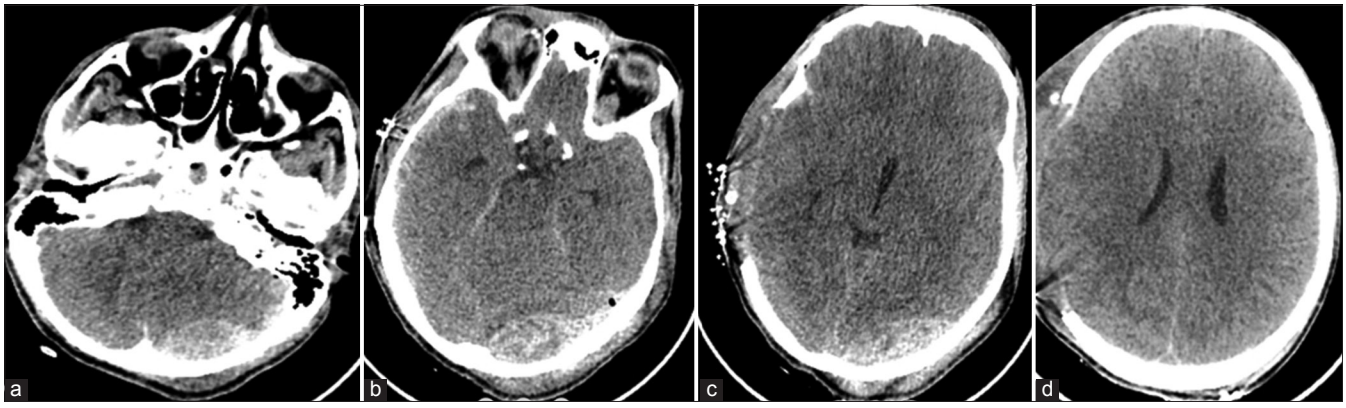

Figure 2: (a-d) Early control post-operative axial plan cranial computed tomography showing evacuation of the subdural hematoma on the right frontoparietotemporal region of the brain and resolution of midline shift, but there is epidural hematoma on the left occipital region of the brain extended into the posterior fossa detected postoperatively

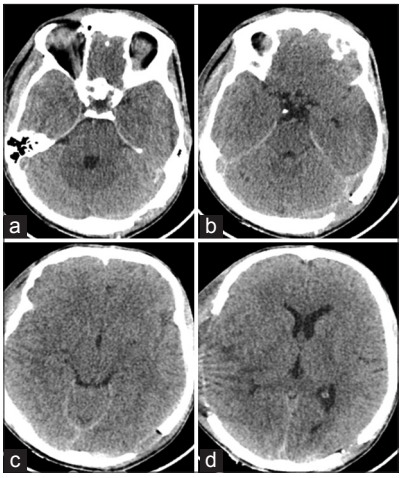

Figure 3: (a-d) Post-operative axial plan cranial computed tomography showing evacuation of the epidural hematoma after the second operation on the left occipital region of the brain and on the left side of the posterior fossa

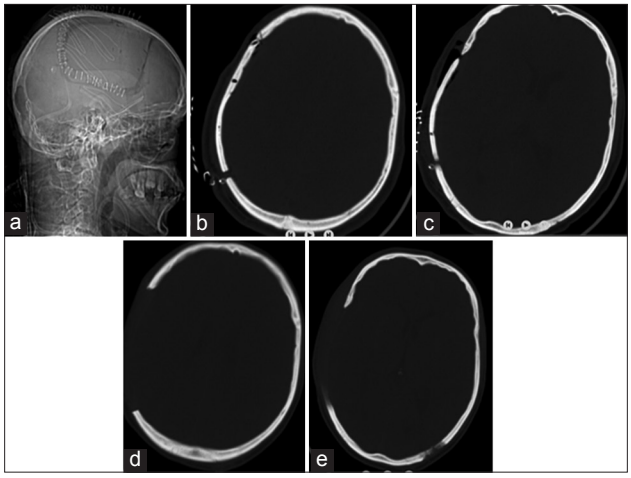

Figure 4: (a-e) Post-operative axial plan cranial computed tomography showing late cranioplasty operation (6 months after the first subdural and the second epidural hematomas intervention), we fix up the bone graft which was embedded subcutaneously in the right lower quadrant of the abdomen

meningeal artery where is relatively weak and prone to injury in 75% of cases.[8] However, posterior fossa bleeds associated with occipital trauma are more commonly secondary to dural venous sinus bleeding and are account for 0.3% of all head injuries and 10% of all epidural bleeds.[9] EDHs are usually coup lesions and are caused either by seepage of blood from a calvarial fracture or injury to the dural arteries. An acute SDH, on the other hand, is generally contrecoup in location and venous in origin. It is unusual, but not uncommon to find both an EDHs and acute SDH on the same side in patients with severe head trauma.[10] The cause of the EDH is thought to be direct trauma and the SDH is probably due to brain recoil causing damage to the cortical bridging veins.[11] Shen *et al*., in 2013, estimated that the percentage of the occurrence of EDH contralateral to the site of drainage of acute SDH was 2.4% according to the published data. Most of the patients were male, with an average age of 35 years and with a main mechanism of head trauma.[3] Our case EDH developed on same side of the blow but crop up incidentally postoperatively following the evacuation of the contrecoup SDH.

The pathophysiology involved in the formation of delayed contralateral EDH following decompressive surgery is not fully understood but may include loss of tamponade effect, vasomotor mechanisms, and coagulopathy, with the main cause appearing to be the upsetting of the equilibrium of the damaged vessels and the reactive ICP.[12] In the present case, the cause of the EDH was probably thought to be due to direct trauma to the head, causing occipital bone fracture and laceration of the left transverse sinus. It is familiar in traumatic epidural hematomas to occur early at the time of the accident as result of arterial injury, especially the middle meningeal artery, where the epidural hematoma developed much more rapid than the usually contracoup and venous in origin subdural hematomas. This fact is exact opposite in contrast to our case where the

epidural hematoma remain silent and detected incidentally in early post-operative control brain BT. In our case although is actually "coup" type of epidural hematoma that occur as result of left transverse sinus laceration at the moment of the blow, we thing the reasons for the late development of epidural hematoma are because of slow transverse sinus bleeding and the temponate effect on the dural sinus created by increase intracranial pressure (ICP) caused by effect of contrecoup acute subdural hematoma. Interestingly, the epidural hematoma thought to be transpire following evacuation of contracoup acute subdural hematoma, as a result of relief of ICP and ease the temponate effect on the transverse sinus which was compressed against the skull bone preopratively.

CONCLUSION

In this case, there are many critical lessons to be learned; we have to stress the importance of early post-operative brain CT or follow-up the patient with ICP monitoring device during the 1st day after surgery to reduce the morbidity and the mortality rates in patients that can develop post-operative contralateral EDH. Like the case in our patient, where the left occipital EDH found incidentally after evacuation of the right temporofrontoparietal SDH, it is unusual to detect EDHs later where a progressive SDHs have been detected in early pre-operative brain CT at the patient admission to the emergency service.

REFERENCES

1. Centers for Disease Control and Prevention (CDC). CDC grand rounds: Reducing severe traumatic brain injury in the United States. MMWR Morb Mortal Wkly Rep 2013;62:549-52.
2. Haselsberger K, Pucher R, Auer LM. Prognosis after acute subdural or epidural haemorrhage. Acta Neurochir (Wien) 1988;90:111-6.
3. Shen J, Pan JW, Fan ZX, Zhou YQ, Chen Z, Zhan RY, *et al*. Surgery for contralateral acute epidural hematoma following acute subdural hematoma evacuation: Five new cases and a short literature review. Acta Neurochir (Wien) 2013;155:335-41.
4. Saberi H, Meybodi AT, Meybodi KT, Habibi Z, Mirsadeghi SM. Delayed post-operative contralateral epidural hematoma in a patient with right-sided acute subdural hematoma: A case report. Cases J 2009;2:6282.
5. Su TM, Lee TH, Chen WF, Lee TC, Cheng CH. Contralateral acute epidural hematoma after decompressive surgery of acute subdural hematoma: Clinical features and outcome. J Trauma 2008;65:1298-302.
6. Zheng FX, Chao Y. Spontaneous intracranial extradural hematoma: Case report and literature review. Neurol India 2009;57:324-6.
7. McCaffrey P. Traumatic brain injury: Effects of closed head injury. In: CMSD 636 Neuropathologies of Language and Cognition. Ch. 11. Chico: CSU.
8. Irie F, Le Brocque R, Kenardy J, Bellamy N, Tetsworth K, Pollard C, *et al*. Epidemiology of traumatic epidural hematoma in young age. J Trauma 2011;71:847-53.
9. Khwaja HA, Hormbrey PJ. Posterior cranial fossa venous extradural haematoma: An uncommon form of intracranial injury. Emerg Med J 2001;18:496-7.
10. Bhateja A, Shukla D, Devi BI, Kolluri VR. Coup and contrecoup head injuries: Predictors of outcome. J Neurotrauma 2009;6:115-8.
11. Madhugiri VS, Arimappamagan A, Chandramouli BA. Traumatic epidural and subdural hematomas and extensive brain infarcts in a patient with pial arteriovenous malformation: Mechanisms underlying clinical and radiological findings. Asian J Neurosurg 2012;7:210-3.
12. Lesoin F, Viaud C, Pruvo J, Redford H, Jomin M. Traumatic and alternating delayed intracranial hematomas. Neuroradiology 1984;26:515-6.

How to cite this article: Adam BE, Eminoğlu ME. Traumatic Occipital Epidural Hematoma Detected Post-operatively Following Decompressive Craniectomy for Acute Subdural Hematoma. Clin J Surg 2018;1(2):1-4.