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Case Report

A rare complication of remote bi-frontal acute extradural hematoma following craniotomy: a case report and literature review

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ABSTRACT

This article presents a rare case of a 26-year-old male admitted to the hospital following a mob assault and was initially diagnosed with an acute subdural hematoma. Following a sizable decompressive craniectomy, a subsequent complication of bi-frontal acute extradural hematoma was identified. We discuss this rare occurrence's pathophysiology, diagnosis, awareness, and management, shedding light on the challenges and considerations in such cases.

INTRODUCTION

Acute extradural hematomas (EDHs) are well-recognized complications following traumatic brain injury. Postoperative acute EDH is a severe, well-known complication that usually occurs at the operation site after cranial surgery. The reported incidence of postoperative EDH is approximately 1%.(1–4)

However, the occurrence of a remote bi-frontal acute EDH after craniotomy for evacuation of an acute subdural hematoma (SDH) is a rare entity. Remote EDH has been reported after a ventriculoperitoneal shunt operation (0,4%), resection of brain tumours (0,15%), or following a decompressive craniectomy (5%–12%).(2–5) They usually develop with an overlying fracture contralateral to the injured side, though they are not restricted to that location.(5,6)

This case study explores the unique case of a 26-year-old male who developed bilateral frontal extradural haematomas remote from his original craniectomy site. We review the existing literature to understand better the pathophysiological mechanisms, diagnostic nuances, awareness, and optimal management strategies for this atypical presentation.

CASE PRESENTATION

A 26-year-old male, following a mob assault, presented to the hospital five days later with a Glasgow Coma Scale (GCS) of 15/15 and complained of severe headache. Otherwise, he was neurologically intact. After one day of observation, the headache worsened, and he started vomiting. He became somnolent, and his GCS dropped to 13/15. A CT brain scan revealed a right-sided acute SDH with a 6mm midline shift to the left and mass effect. There was no associated skull fracture. (Figure 1). He was taken to the theatre emergently for clot drainage.

Mannitol infusion was initiated preoperatively and continued intraoperatively. He was placed in a supine position over a horseshoe headrest without rigid pin fixation. After craniotomy, the dura was hitched to the parent bone. The subdural clot was successfully removed in its entirety. However, there was significant brain swelling and cerebral herniation, requiring a higher dose of mannitol infusion. The dura could not be closed, and the bone was not replaced.

Subsequently, in the ICU, dilated pupils were noted, prompting an urgent CT brain scan that revealed a large bifrontal acute EDH away from the initial surgical field. (Figure 2) He was immediately taken to the theatre, where a bi-frontal craniectomy was done, and the hematomas were evacuated. (Figure 3) The coagulation parameters were within the normal range.

Following this second operation, he improved to a GCS of 8/10 (intubated). He died two weeks later of a chest infection. Because he could not be extubated, a complete neurological assessment to establish the extent of his neurological recovery beyond the GCS could not be done.

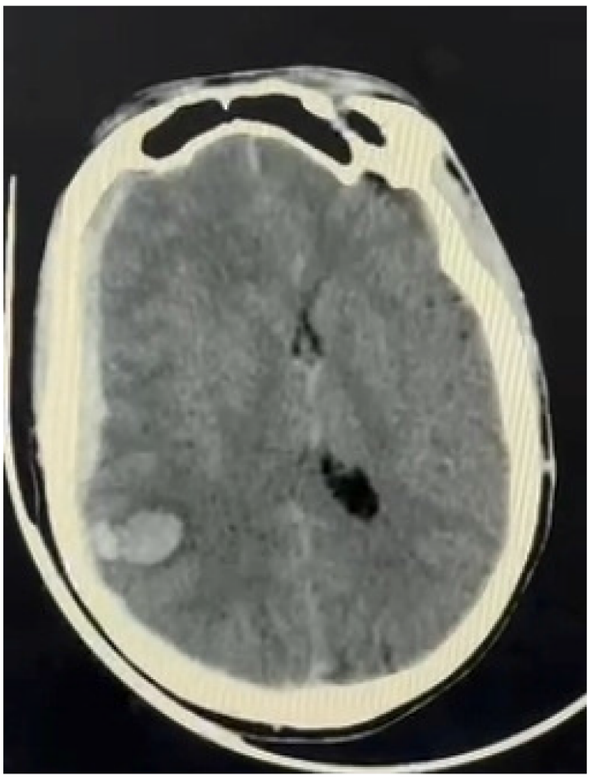

Figure 1: CT scan of the brain showing a right-sided acute subdural hematoma with midline shift and mass effect.

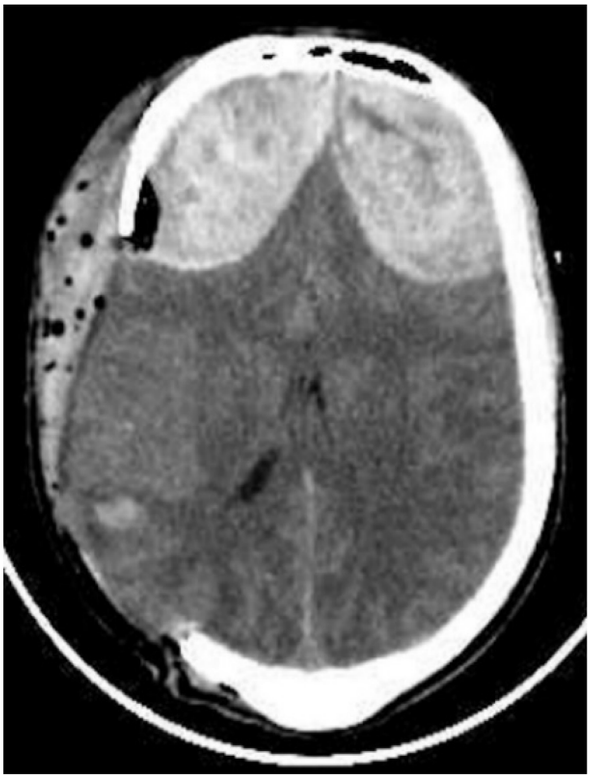

Figure 2: Initial postoperative CT scan of the brain showing bifrontal acute subdural hematoma.

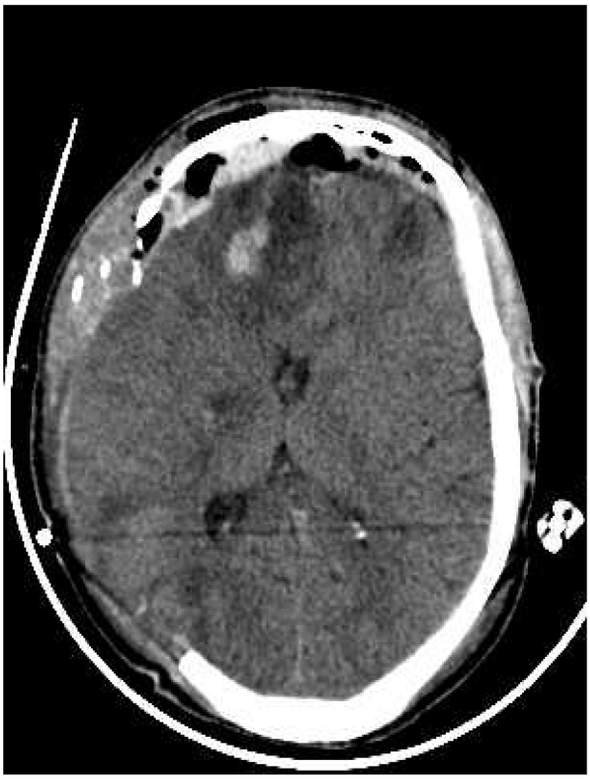

Fig 3: Postoperative CT scan following the evacuation of the bifrontal extradural hematoma.

DISCUSSION

Remote EDH can be classified based on their location: Type 1 includes hematoma ipsilateral to the surgical area but not involving the surgical site; Type 2 includes hematoma contralateral to the surgical area; Type 3 includes remote EDH involving the bilateral side of the surgical area; and Type 4 includes supratentorial EDH with infra-tentorial surgery.(3)

Developing a remote bi-fontal acute EDH post-craniotomy involves intricate pathophysiological processes that are poorly understood. There is no single etiologic factor, and various hypotheses have been suggested.(1,3) These hypotheses include a sudden decrease in intracranial pressure (ICP) with loss of tamponade effect, massive drainage of cerebrospinal fluid (CSF), unequal distribution of ICP causing brain shift, underlying coagulopathies, and use of pin fixation. However, the association is unlikely in our patient as he was operated on a head-ring. Frequent usage of mannitol during surgery may also increase the chances of developing this complication.

It is commonly accepted that ICP decreases following craniotomy due to significant loss of CSF. This excessive loss of CSF during surgery causes brain shift and acute negative pressure in a remote area. Increasing the transmural pressure of the dural venous system causes the rupture of blood vessels, leading to epidural hematoma.(2–4)

Whatever the underlying mechanism post-surgery, low ICP is the crucial triggering factor.

In most patients, a venous source of bleeding was identified.(5) In cases with a midline epidural hematoma, stretching of the diploic/bridging veins at their entry into the dural venous sinuses can rupture the veins and cause bleeding.(5)

The resultant venous hypotension could have manifested as bilateral EDH after the release of brain tamponade. This mechanism is only speculated without definitive evidence from venographic studies.

Previous studies have reported specific predictors of postoperative epidural hematoma (EDH), the most important of which is the presence of two or more bone plates with a skull fracture.(7) A younger age was also noted, probably related to the ease of dural stripping.(7) Additional risk factors identified include intraoperative blood loss exceeding 800mls, maximum craniotomy length, and craniotomy area equal to or greater than 100 mm and 71.53 cm2.(7) Intraoperative brain swelling and external cerebral herniation can also potentially hint at the occurrence of this complication.(5,6). The prognosis of bilateral EDH seems more risky than a unilateral one, and most patients with postoperative EDH need surgical evacuation.(5)

CONCLUSION

This case highlights the importance of considering rare complications in the postoperative phase of traumatic brain injury (TBI), emphasizing the significance of timely diagnosis and appropriate management in improving patient outcomes.

Clinicians should be vigilant about the potential for delayed EDH formation following craniotomy. In patients with large tumour sizes and a significant loss of blood and cerebrospinal fluid, as well as a large craniectomy, there is a risk of developing remote EDH. Therefore, neurosurgeons need to maintain high suspicion for this condition. A fracture need not always coexist in EDH following decompressive craniectomy.(5)

To avoid complications, it is recommended to use slow and gradual decompression during surgery. Some authors have proposed a temporal burr hole before proceeding to actual craniotomy. Basic procedures should always be remembered, such as filling the normal saline in the intradural space before the last dural suture to prevent pneumocephalus.

Every patient with neurological deterioration in the early postoperative period should have a rapid and urgent brain CT scan because early detection and removal of postoperative acute EDH can be lifesaving.(1,2) Aggressive hematoma evacuation often results in satisfactory outcomes for patients.

Our patient had some risk factors for developing this complication: decompressive large craniectomy, pre and intra-operative mannitol infusion, intraoperative brain swelling, and brain contusion. However, exceptionally, pin-fixation was not used, the GCS was 15/15, and there was no skull fracture.

We are not aware of any previous report of the occurrence of postoperative remote bifrontal EDH in non-severe TBI. In our patient, the sizable decompressive craniectomy combined with mannitol infusion leading to a sudden decrease of ICP and loss of tamponade effect may be the cause of bilateral frontal acute EDH.

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