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diagnostic dilemma

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ABSTRACT

Acute subdural hematoma (ASDH) is one of the most common clinical entities encountered in daily neurosurgical practice. The advent of computed tomography (CT) has made a major impact on the radiological diagnosis of ASDH. Although most of them appear as crescentic, concavo-convex hyperdense lesions on CT, sometimes they may present atypically. Here we report such a case, where ASDH presented as acute EDH, causing a diagnostic dilemma.

INTRODUCTION

A hyperdense crescentic region that stretches over the majority of the subdural space and conforms to the underlying cerebral surface is the defining hallmark of acute subdural hematomas (ASDH) when they are situated over the convexity. These are most frequently caused by cortical bridging veins rupture. Because the subdural space is continuous, blood is free to diffuse throughout the cerebral hemispheres. [1] Majority of acute extradural hematomas (EDH) appears as a biconvex collection between the dura and inner skull bone. It frequently happens as a result of skull fractures that cause disruptions to the meningeal vein, dural sinus, or artery that overlies them. As a result, it typically has a focused nature with few extensions [2]. This is the primary radiological characteristic that distinguishes it from acute SDH which tends to have a diffuse extension over the hemisphere. We report a case wherein acute SDH has presented with a lentiform shape, resulting in a preoperative diagnosis of EDH, and a completely different intraoperative finding.

CASE REPORT

A 65-year-old man presented to emergency room with history of self fall followed by loss of consciousness. He was taken to a nearby hospital, where the first radiology (non contrast computed tomography) was done after 2 hours of injury and was suggestive of a hyperdense

Keywords

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biconvex lesion at right fronto parietal region with right parietal hemorrhagic contusion. It was diagnosed as acute EDH and was referred to our hospital (Figure 1). Another repeat scan was done at 4 hours post trauma when he arrived in our emergency room, which again showed similar findings (Figure 2). On arrival he was comatose and manifested bilateral alternating purposeful and decerebrate movements on painful stimuli. Blood studies showed hemoglobin 10.5 g/dl, leukocytes 13400, platelets 120000, and normal prothrombin and partial thromboplastin time.



Figure 1. Right fronto parietal biconvex hyperdense lesion with underlying small contusion (post trauma 2 hours).

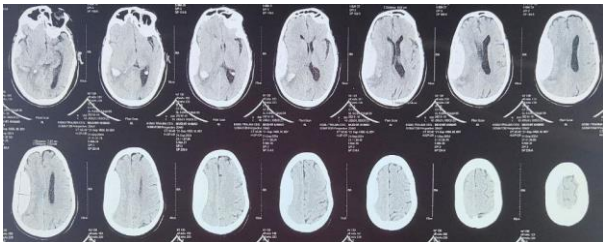


Figure 2. Right fronto parietal biconvex hyperdense lesion with underlying small contusion (post trauma 4 hours).

He was taken in emergency operation, a fronto parietal horseshoe incision was taken and a craniotomy flap raised. There was no evidence of acute EDH (Figure 3). The dura appeared bluish but without any significant bulge. Dura was incised and underlying acute SDH was seen and evacuated (Figure 4). The brain cortex showed scattered petechial hemorrhages. Postoperatively, the patient recovered neurologically and was discharged at a GCS of E4V4M6 and is presently in outdoor follow up.

DISCUSSION

Acute SDH is a common neurosurgical emergency. Although acute SDH is seen mainly following traumatic brain injury, other causes such as

postoperative hemorrhage, coagulopathy and arterial venous fistulas, are also encountered [3]. It is not difficult to differentiate acute SDH from EDH on CT scans. Subdural space is a potential space without barriers, as such whenever blood accumulates in the space it appears as panhemispheric and crescent-shaped lesions. Reed *et al.*, in a review of 71 cases of acute SDH, reported a 39% incidence of atypical features on CT scans [4]. Among them, about 14% of SDH had convex margins, mimicking typical features of acute EDH. This specific feature easily leads to misdiagnoses.



Figure 3. Bluish tinged dura without any evidence of acute EDH.



Figure 4. Acute SDH with sinus based dural reflection.

Pathophysiology involved in lentiform SDH

While the mechanism of focal collection of SDH is not well understood, most theories involve subdural blockage secondary to hemorrhage into this potential space. Braun *et al.* reported lentiform SDH in a patient with coagulopathy [5]. Another study by I-Chang Su proposed that blockage of the subdural space by adhesions, which occur due to previous asymptomatic bleeding secondary to coagulopathy might be a plausible explanation. This theory was again addressed by other authors [6].

Another explanation proposed by Braun *et al.*, may be the loss of brain turgidity [5]. A similar observation was also seen in the study of I-Chang Su wherein the brain was atrophied because of a previous severe parenchymal injury. In their report, parenchymal loss was obvious in post-traumatic cerebral atrophy, as such easy indentation of the atrophied brain made localized accumulation of SDH more possible. Although in our case, he did not provide any history of trauma or coagulopathy, but owing to his advanced age, cerebral atrophy was quite noticeable.

Radiographically, differentiation between acute lentiform SDH and EDH by their shape alone was not reliable. Some additional CT features were very useful in establishing correct diagnoses. From previous literature review we can summarize them as follows

1. Crescentic tail: Presence of a crescentic tail at the margin of the hematoma, which was concave toward the brain. [5] On retrospective analysis of our CT scan, we can notice this finding in the serial axial films towards the temporal base.
2. Change in shape: Hematomas eventually assume their crescent shape in the periphery of serial sections. This was regarded as a reliable feature in favor of acute SDH, which was also seen in our case. [5]
3. Angulation: Obtuse angle between the inner border of the hematoma and the skull bone. Obtuse angles resulted from indentation of the brain surface. Arachnoid membrane and brain parenchyma are easily indented than the dura mater, and as such obtuse angles at the margin might serve as an important clue to the location of hematomas. [6] Again, retrospectively this finding was very much evident in the repeat CT scan at 4 hours post trauma.

It is important to note that these distinguishing characteristics are not always present at the same time. Consequently, neurosurgeons should be informed about the potential for an unusual SDH by any patients presenting with a perplexing horizontal head CT study.

CONCLUSION

We highlighted in this report the diagnostic difficulty of CT imaging when lentiform focal SDH is present. When a biconvex-shaped hematoma is seen on CT, careful attention must be given, particularly if the patients have a history of intracranial injuries or neurosurgical procedures. This atypical presentation of SDH is most likely caused by blockage of potential subdural space. Consequently, the various radiographic features described with a proper clinical history can help in its diagnosis. Practicing clinicians should be aware of such pitfalls in diagnosis solely based on radiology to prevent inaccurate preoperative judgments.

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