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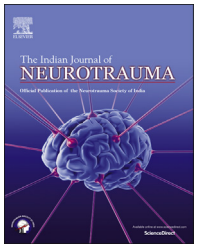

Case Report

Spontaneous resolution of traumatic acute subdural hematoma with good clinical outcome: A report of two cases

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ABSTRACT

Post-traumatic acute subdural hematoma (ASDH) requires immediate surgical evacuation in most cases. We present two cases of ASDH which resolved spontaneously. The first patient had 9 mm thick ASDH in left fronto-temporo-parietal region with initial GCS of $E_1V_1M_3$ which improved to $E_2V_tM_4$ within 7 h. Repeat CT of the brain showed a decrease in the size of ASDH. Hence, patient was conservatively managed with anti-edema measures and elective ventilation. The patient improved and ASDH also resolved completely. The second patient also had 8 mm thick ASDH in the right fronto-temporo-parietal region with GCS of $E_1V_2M_5$ which 5 h later improved to $E_3V_4M_5$. With conservative measures, there was clinical and radiological improvement. Based on previous reports, certain characteristic features have been shown to favor spontaneous resolution of ASDH; absence of underlying contusion and the presence of a low-density band between the skull and the hematoma on imaging were features common to our patients also.

1. Introduction

Acute subdural hematoma (ASDH) is a common sequel of traumatic brain injury. Immediate surgical evacuation of clot is required for most cases except in patients with poor grade of Glasgow coma scale, irreversible brain stem injury or patients who have poor general condition.[1] Acute SDHs which are larger than 10 mm or those which are 5–10 mm thick in a patient with low GCS generally require immediate surgical

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intervention.[2] However, there are instances where spontaneous resolution of SDH has been noted within first 72 h. Here, we present and discuss two cases of acute SDH which resolved spontaneously.

2. Case 1

A 40-year-old lady presented with a history of fall from a moving two wheeler followed by immediate loss of and two episodes of vomiting. She was immediately taken to a local hospital where the patient's admission GCS was $E_1V_1M_3$ and pupils were asymmetrical (left bigger than right) and sluggishly reacting to light. Patient's trachea was immediately intubated and Computed Tomography (CT) scan of the brain showed diffuse brain edema with 9 mm thick acute SDH in the left fronto-temporo-parietal region with a midline shift of 10 mm (Fig. 1A). She was immediately transferred to our hospital, for further management. Seven hours after the trauma, her current admission GCS was $E_2V_tM_4$ with unchanged pupillary reaction. In view of her improving GCS, a repeat CT scan was done which showed a decrease in the size of SDH to 2 mm (Fig. 1B). Therefore, she was shifted to ICU for elective mechanical ventilation and anti-edema measures (20% mannitol 100 ml 6th hourly, 3% hypertonic saline at 15 ml/h) were started. A repeat scan on day three revealed complete resolution of the hematoma (Fig. 1C). The patient was $E_3V_tM_5$ and pupils became equal and reacting to light. Patient was extubated on day 6 and made a full neurological recovery (Fig. 1D).

3. Case 2

A 55-year-old lady presented with an alleged history of fall from a two wheeler, while getting down from it. She had loss of consciousness for 10–15 min, with multiple episodes of projectile vomiting. However, there was no external bleeding or any other systemic injuries. Patient reached a local hospital 1 h after the head injury, where her GCS on admission was $E_1V_2M_5$. CT of the brain revealed subdural hematoma in the right fronto-temporo-parietal region measuring 8 mm, with mass effect and midline shift of 9 mm (Fig. 2A). After resuscitation, the patient was referred to our institution for further management. Five hours after the injury, patient was hemodynamically stable and her GCS had improved from initial score of $E_1V_2M_5$ to $E_3V_4M_5$. Hence, non-surgical management was planned. Patient was shifted to ICU and anti-edema measures were initiated with mannitol 100 ml q6h and 3% hypertonic saline at 15 ml/h infusion. A repeat CT done after 6 h showed resolving SDH which decreased from 8 mm to 2 mm with reduction in the midline shift (Fig. 2B). In view of the improving clinical and imaging status, medical management was continued. Six hours later the patient's GCS improved to $E_4V_5M_6$. On the 2nd day, patient was conscious, oriented with no neurological deficits and was discharged.

4. Discussion

Rapid spontaneous resolution of acute SDH is defined as neurological improvement within 24 h of presentation and

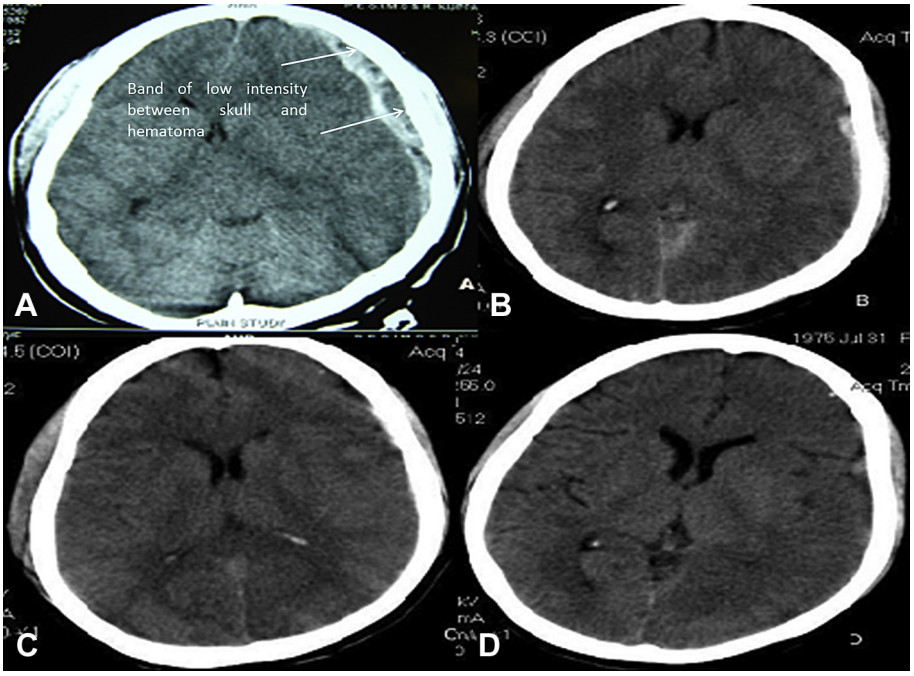

Fig. 1 – A) CT brain showing diffuse brain edema with a 9 mm thick acute SDH in the left fronto-temporo-parietal region with a midline shift of 10 mm. Arrow shows the band of hypodensity between the inner side of skull bone and the hematoma. B) Repeat CT scan showing decrease in size of SDH. C and D) Repeat CT scan at day 3 and 6 showing complete resolution of ASDH.

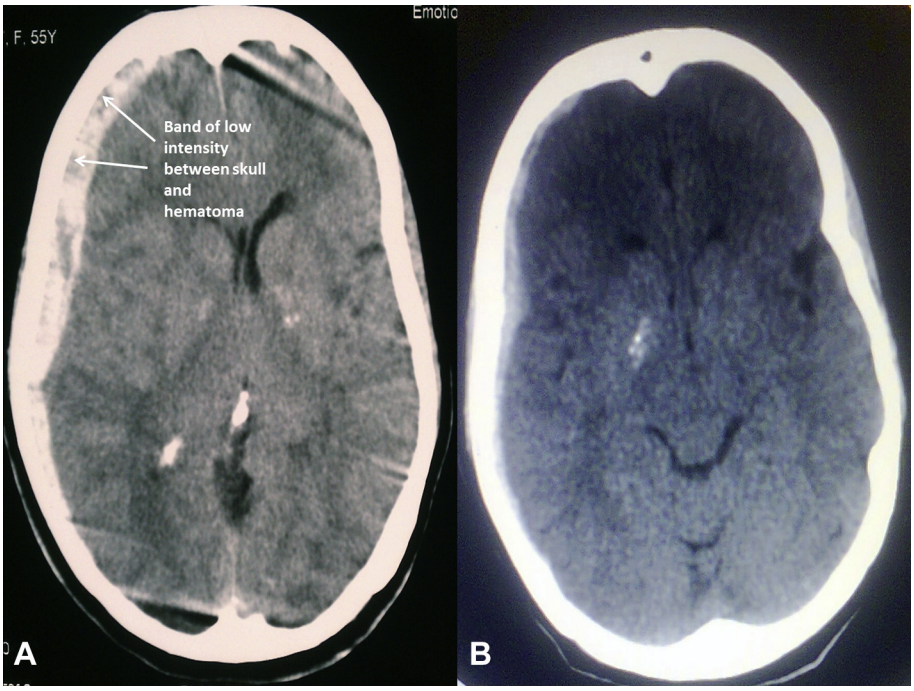

Fig. 2 – A) CT brain showing ASDH in the right fronto-temporo-parietal region measuring 8 mm and midline shift of 0.9 cm to the left. Arrows shows band of hypodensity between the inner side of skull bone and the hematoma. B) A repeat CT scan brain done after 6 h showed resolving acute SDH which decreased in thickness to 2 mm.

reduction of clot thickness by more than 5 mm within 96 h of initial CT scan.[3] Spontaneous resolution of acute SDH is a rare phenomenon in clinical practice, but few reports describing its occurrence have been published previously.[4–6] Various theories have been proposed for explaining this phenomenon.

- The hematoma is compressed by the pressure produced by acute brain swelling and redistributed.[4]
- In acute SDHs, there is a tear in the arachnoid permitting the entrance of cerebrospinal fluid (CSF) into the subdural space.[7] The circulating CSF then dilutes and liquefies the blood clot and is redistributed to the subdural and subarachnoid spaces. The hematoma is diluted by cerebrospinal fluid and is washed out.[8]
- Occasionally the subdural blood clot can be forced out into extra cranial spaces by the increasing brain edema in the presence of a meningeal tear and fracture skull.[9]

The incidence of spontaneous resolution of acute SDH is very rare and many investigators have tried to identify factors which help us in anticipating spontaneous resolution of acute SDH. Wen et al[10] reviewed previously described cases of spontaneous resolution of acute SDH and suggested the following five characteristics which were common in these patients;

- Transitory coma lasting no longer than 12 h
- Exclusion of cerebral contusion
- Band of low density between the skull and the hematoma on (CT) imaging
- Thin width which is widely distributed
- Glasgow Coma Scale >8 on admission.

Similarly Fujimoto et al[3] studied 366 consecutive patients with acute SDH and found that patients with a low-density band between the hematoma and the inner wall of the skull bone on initial CT scan and those patients who have been administered antiplatelet agents before head injury were more likely to experience rapid spontaneous resolution of acute SDH and neurological improvement.

Based on various predictive factors described by different authors, factors which were consistent between our patients were;

- No cerebral contusion
- Presence of a band of low density between the skull and the hematoma on CT imaging.

In our cases, the cerebral edema possibly caused the compression and redistribution of subdural blood leading to the resolution of SDH. Also, the presence of low-density band between the skull and hematoma suggests the dilution of hematoma by CSF flow through the subdural space. Our patients had a large hematoma with profound impairment of consciousness, yet made a complete recovery.

5. Conclusion

In patients with ASDH showing improvement in neurological status, a subsequent CT scan should be done before surgery. Absence of contusion and presence of low-density band on CT scan help in identifying the patients who are likely to have good prognosis without surgery despite poor neurological status at admission.

Conflicts of interest

All authors have none to declare.

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