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CASE REPORT

Spontaneous Regression of Subdural Haematoma Due to Redistribution in a Young Child: a Case Report

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INTRODUCTION

Acute subdural haematoma refers to the presence of blood between the dura mater and outer layer of the arachnoid mater. It occurs in almost 21% of all severe traumatic brain injuries and almost 11% of mild and moderate injuries. Mortality ranges from 30% to 90%.[1] The possible mechanisms of injury leading to subdural haematoma include trauma, rupture of cortical bridging veins, damage to the surface cortical vasculature, bleeding from underlying injury to the brain parenchyma, intracranial hypotension and defective anticoagulation, among which trauma is the most common cause in children. Various factors related to a poor outcome in such patients include low Glasgow Coma Scale (GCS) score on admission, sluggishly reactive pupils, hypotension and bilaterality of the haematoma.[1,2]

Management of subdural haematoma varies depending on the neurological status and radiological parameters, for example, thickness of bleed and mass effect leading to midline shift and herniation. The general consensus is that in all cases of haematoma with thickness >10 mm or midline shift >5 mm, drainage of the collection is required irrespective of GCS score. Cases of lesser severity can be conservatively managed. Surgical options include craniotomy with or without bone flap removal.[3,4]

In cases where conservative management is sought, spontaneous resolution may take weeks to months, depending on the size of bleed. Few cases in the literature describe rapid spontaneous resolution. We describe a young girl in whom subdural haematoma sufficiently large on initial scans to warrant surgical intervention showed a significant decrease in size with redistribution to other sites, simultaneously linked to rapid improvement in neurological status.

CASE PRESENTATION

In January 2021, a 5-year-old girl was admitted to our emergency department after a fall while playing. She became disoriented and lost consciousness over the course of 30 minutes, after which she also developed tonic, posturing of the limbs that lasted for a few minutes

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but was not associated with deviation of the angle of the mouth or frothing at the mouth. Neurological examination revealed a GCS score of 10/15.

Subsequently, the patient was referred to the radiology department for computed tomography (CT) scan of head. The CT scan performed nearly 60 minutes after her injury revealed a large concavo-convex (crescentic), extra-axial hyperdense collection overlying the right frontoparietal cortex, measuring approximately 12.5 cm anteroposteriorly, 10 cm craniocaudally and 14.5 mm in maximum thickness (Figure 1). There was an associated midline shift of 7 mm with uncal and subfalcine herniation to the left side. The underlying cerebral sulci and the right lateral ventricle were effaced due to compression by the subdural haematoma. There was a small extension of the haematoma into the anterior interhemispheric fissure but no damage to the cranial bones or sign of cerebral contusion.

On the basis of the GCS score and CT findings, neurosurgical intervention was planned and routine blood investigations performed. The patient was commenced on intravenous 3% hypertonic saline. After an hour, the young girl began to regain consciousness and GCS score improved slightly to 11/15. New CT scan 6 hours after the first revealed highly unexpected findings. The anteroposterior extent of the crescentic collection had reduced to 9 cm, and the thickness had shrunk by almost two thirds to nearly 5.8 mm with a now reduced midline shift of 3 mm (Figure 2). A separate thin new subdural collection of thickness 3.5 mm was noted in the left high-parietal region. Another new subdural collection was noted at the cervicomedullary junction, indenting the posterior subarachnoid space but not compressing the cervicomedullary junction (Figure 3). A smaller subdural collection was noted extending into the posterior interhemispheric fissure and tentorium cerebelli. Essentially, the large collection over the right cerebral cortex had become decompressed and been redistributed. Clinically too, the patient's GCS score had improved to 13/15.

Considering the new findings, a more conservative approach was adopted and the child was kept under observation with regular monitoring of GCS score. An

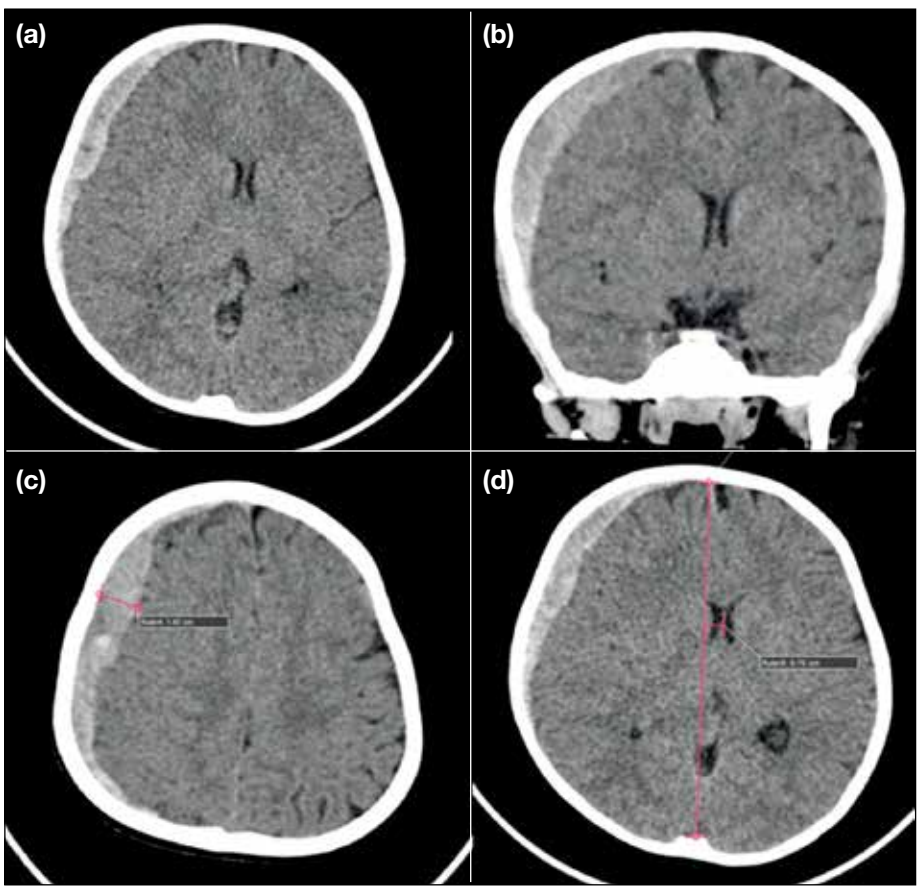

Figure 1. Axial and reconstructed coronal images of the initial computed tomography brain reveal a hyperdense crescentic subdural collection along the right frontoparietotemporal convexity (a, b) with maximum thickness of 14.5 mm (c) and midline shift towards the left side by 7 mm (d).

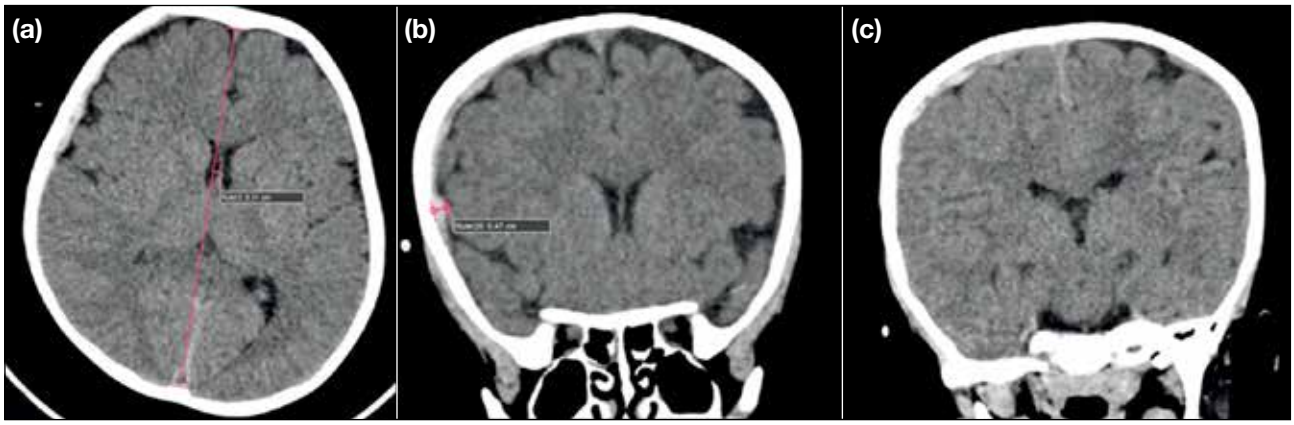

Figure 2. Axial and reconstructed coronal images of the follow-up computed tomography brain, conducted 6 hours after the first, showing a decrease in the midline shift to 3 mm (a) consequent to the decrease in size of the subdural haematoma with maximum thickness measuring 4.7 mm (b, c). Thin subdural haematoma is seen in the left parietal region (c) due to redistribution.

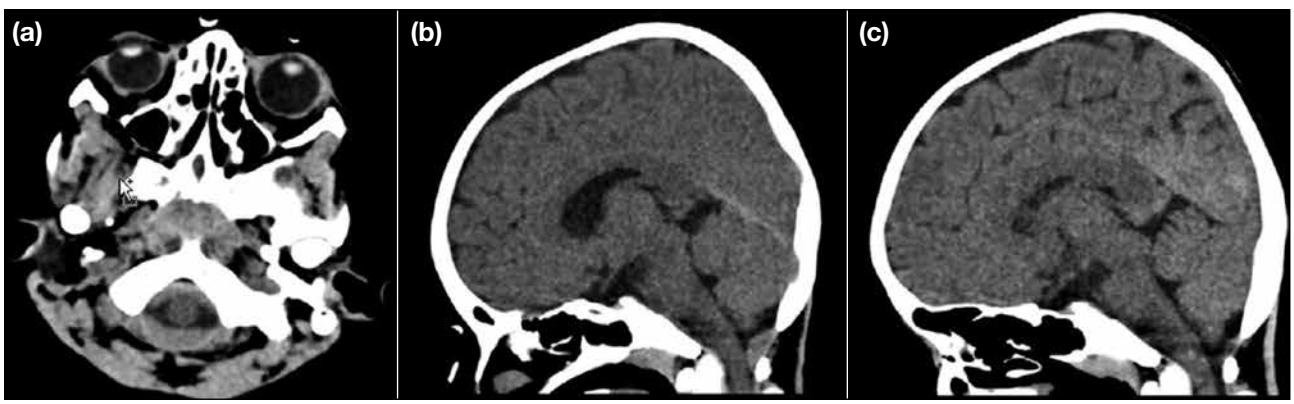

Figure 3. Axial and reconstructed sagittal images of the follow-up computed tomography brain, conducted 6 hours after that first, showing redistribution of the subdural collection at the level of the foramen magnum along the posterior aspect of the cervicomedullary junction (a, b [white arrow]). Compare this to the reconstructed sagittal image of the previous computed tomography (c) that reveals clear cerebrospinal fluid in the same region.

evaluation was sought by magnetic resonance imaging of the brain 40 hours after the first CT and subsequently revealed a small collection of maximum thickness 3.6 mm along the right frontotemporal region (Figure 4). Magnetic resonance angiography was also performed to exclude any vascular aetiology such as aneurysm and was normal.

DISCUSSION

Patients with traumatic subdural haematoma and poor GCS score on admission, as in our case, are considered candidates for craniotomy and drainage of haematoma to prevent further brain damage and reduce morbidity and risk of mortality.[3,5] However, spontaneous improvement in symptomatology and associated reduction in haematoma size mandates a more conservative approach. The first case of spontaneous resolution of subdural haematoma was documented in Tokyo in 1986 by Nagao et al.[6] Many cases have since been reported although few over such a short period of time, between serial CT scans. Even fewer such cases have been reported in children, less than 10.

Two principal mechanisms are ascribed to spontaneous resolution of haematoma, both of which involve redistribution of blood at their core rather than actual regression. One is associated with intrinsic redistribution that involves acute swelling of the brain. The other, an extrinsic one, is linked to a dural or arachnoid tear that results in redistribution of blood to other intra- or extra-cranial spaces, thereby releasing pressure from the primary site.[7,8] The mixing of blood with cerebrospinal

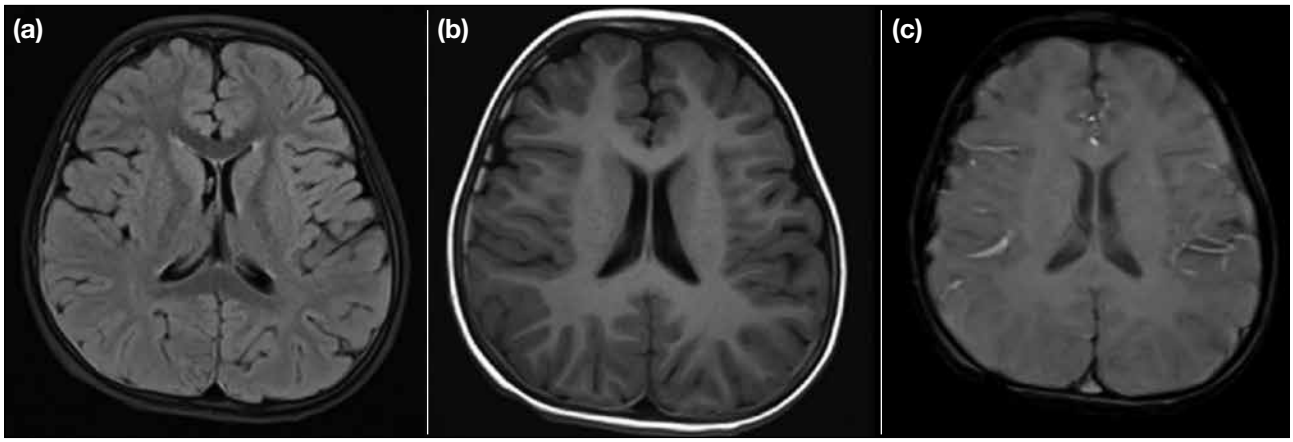

Figure 4. Magnetic resonance imaging brain conducted approximately 40 hours after the injury revealed a sliver of small crescentic subdural collection on the right side in the fluid attenuation inversion recovery (a) and T1-weighted (b) images with corresponding blooming on susceptibility weighted image (c).

fluid and subsequent washout serves to dilute the bleed and carry it into the subarachnoid, subdural or spinal subdural space.[9,10] The presence of cerebral atrophy with prominent subarachnoid spaces may help the dilution of subdural haematoma.[11] Rarely, the haematoma can drain into the subgaleal or diploic space in the presence of skull fracture and associated dural injury.[12] In our case, there was redistribution of subdural bleed to newer sites, to the interhemispheric fissure and also to the spinal subdural space, evidenced by the presence of blood in the spinal space at the cervicomedullary junction. Nonetheless there was no subarachnoid extension of the haemorrhage.

Various factors have been linked to and studied in resolving acute subdural haematoma,[10,13,14] namely instances where subdural haematoma is located at the frontotemporoparietal, frontotemporal, or frontoparietal convexities; where a patient presents with transient deterioration in neurological status with subsequent improvement[15]; or a patient prescribed anticoagulants where the lack of clotting factors prevented formation of a platelet plug and thus enabled redistribution.[16] Other observations in similar cases have included sustained trauma with GCS score >8; and presence of a band of low density between the haematoma and the interior table of the skull bone.[10] The extent of their significance has not been completely determined.

Our patient presented with various factors favouring spontaneous resolution — frontal location of the bleed, thickness of the haematoma close to the mean thickness seen in other cases with spontaneous resolution of subdural haematoma. The subdural haematoma was located over the right frontoparietal cortex, with thickness of 14.3 mm at admission. The patient had a low GCS score at admission, which subsequently improved over a period of 6 hours with redistribution of the subdural bleed and consequent spontaneous resolution with symptomatic improvement. The patient was able to be managed conservatively and avoided major surgery and associated risks.

CONCLUSION

The present case describes the rare occurrence of spontaneous regression of subdural haematoma with redistribution of blood to other sites and symptomatic improvement. Close monitoring of a patient with subdural haematoma by GCS score and serial non-contrast CT brain can help identify spontaneous regression and avoid the need for surgical intervention.

PATIENT PERSPECTIVE

In the child's words, "I know I'm at a hospital right now [well oriented] and I feel better now. Though I still feel some pain over my head but I want to go home now".

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