In the ED, he was conscious and had a tender closed wound with an obvious palpable defect in the skull topography; a depression of 1.5 cm bone fragment pushed to a depth of approximately 1 cm from the skull silhouette. Noncontrast computed tomography (NCCT) scan of the brain and skull showed a depressed fracture in the left frontal region with underlying hypodense area showing edema [Figure 1a and b]. No underlying hemorrhagic contusion, extra- or sub-dural bleed was, however, visible.

Surgery was offered to the patient and his family; however, the family was reluctant and denied any surgical intervention. Despite multiple counseling sessions by many health-care consultants and neurosurgeons, the family left the hospital against medical advice 2 days after admission. We tried to follow-up with the family 1-month after the admission, but they said that the patient was asymptomatic and did not need any medical attention. Subsequent follow-up attempts failed because of unresponsive family members.

In 2014, the patient was readmitted at our institute with complaints of two episodes of seizures in the previous 12 h. There was no history of injury or any neurological symptoms in the intervening 2 years. A repeat NCCT scan of the brain and skull showed no defect in the bony topography of the skull [Figure 1c]. There were, however, bifrontal hypodense areas, more on the left side, suggestive of gliosis [Figure 1d]. No underlying hemorrhagic contusion, extra- or sub-dural bleed was, however, visible.

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Discussion

Pond fractures and ping-pong ball type depressed fractures of skull in younger children have been previously reported to elevate spontaneously over months, with few segments reducing over hours to 3 days. A 13-year-old skull is, however, more stiff and unyielding. It seems that the constant pressure of the brain over the depressed fragment was responsible for its elevation, and the broken fragment gradually rising, fit in the skull profile such as segments of a jigsaw puzzle. Nonsurgical management of depressed bone in infants by breast pump or manually has been previously reported. We are yet to come across reports of spontaneous resolution of depressed fracture in adolescents. Most cases of spontaneously resolving depressed fracture report good result on follow-up, with no neurological deficit or seizures. Our patient, being older than those previously reported, had firmer skull bone, and the depressed bone must have taken more time to elevate when continually pressed by the underlying brain. This must also have kept the brain under continued counter-pressure from the bone, hence leading to a large gliotic area, which served as epileptogenic focus. There was no evidence of raised intracranial pressure. Moreover, the depressed broken fragment was displaced such that it fit back in the skull topography such as the pieces of a jigsaw puzzle, hence the term, “jigsaw” depressed fracture.

Conclusion

Although this case is a good example, even if only rare and odd, of how “jigsaw” depressed fracture may spontaneously elevate in older children as well, leaving no sign of previous depression, the patient suffering from seizures is a warning that depressed fractures if not surgically reduced well in time, may lead to generation of epileptogenic focus in the form of gliosis. Also, there is no evidence in literature that may suggest that surgical reduction of the depressed segment prevents late presentation of seizures.

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References


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