

Cortical blindness and status epilepticus following head injury in a child

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Abstract: A three year old boy sustained a depressed fracture of the parietal region when a 25 kg window frame fell on his head from a height of about a 100 feet. His GCS at presentation was $E_1V_2M_4$. He underwent surgical decompression within a few hours. The patient had echolalia followed by status epilepticus along with Cerebral Salt Wasting and Hyponatremia in the post operative period. He also had Occipital Blindness which resolved.

Keywords: cerebral salt wasting; depressed fracture skull; occipital blindness; seizures

INTRODUCTION

Depressed fracture of the skull or 'cave-in' injuries can occur with heavy objects falling on the head. In the presence of significant cortical injury, a decompressive craniectomy in addition to elevation of the depressed fragment debris and duraplasty may be necessary. Cerebral salt wasting and hyponatremia can occur in a severe traumatic brain injury setting. Occipital blindness can occur in the presence of damage or a conduction deficit of the optic radiation.

Case Details

A three-year-old boy was playing outside a high rise building when a 25 kg window frame fell from a height of 100 feet on his head.

The boy lost consciousness immediately. At the casualty he was intubated after his GCS was assessed at $E_1V_2M_4$. CT head revealed a depressed fracture of the left parietal and frontal regions. Child was prepared and taken to the operation theater Fig 1.

At surgery, the lacerated wound over the left parietal region was extended to facilitate elevation of the depressed fragment (Fig. 1).

The grossly contaminated edges of bone were discarded and the remaining bone was kept in the anterior abdominal wall for the later cranial reconstruction (Figs 2-4). The dural opening was extended. Clots, hair and debris were washed out with saline irrigation. The dura was closed loosely with a patch of synthetic dura.

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Fig. 1: Taken to the operation theater

Post cranial reconstruction photographs

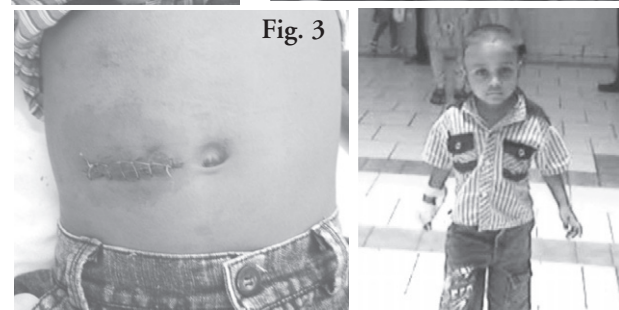
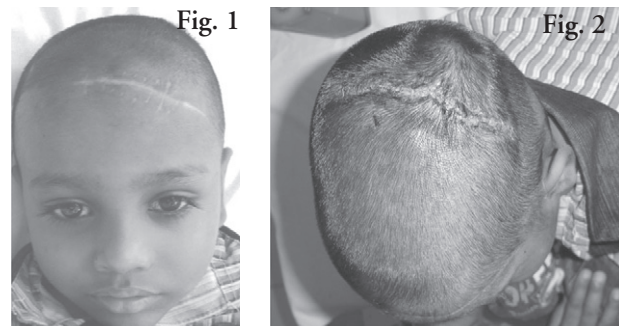


Fig 1: Frontal scalp wound

Fig 2: Parietal scalp wound

Fig 3: Bone pieces of fracture skull kept subcutaneously in anterior abdominal wall.

Fig. 4: Child walking after one month

The patient was electively ventilated in the postoperative period. He was extubated on third postoperative day as his neurological status improved. Medications administered included broad spectrum antibiotics, mannitol, phenytoin and ranitidine. A nasogastric tube was passed to initiate feeding.

The boy was obeying commands on 6th post operative day, but was noted to have echolalia. He demonstrated visual inattention in all fields and was unable to reach out and localize objects although both his papillary reaction and blink response were normal. The hemogram and electrolytes done on third postoperative day were within normal range.

On sixth day following surgery, child developed generalized seizures which persisted despite diazepam and phenytoin administration. A midazolam infusion was started before the seizures came under control in about one hour. The serum sample collected at this time showed hyponatremia (Na = 118 mEq/L). He was started on hypertonic saline infusion and the sodium level was corrected to near normal over a period of 48 hours. A second antiepileptic (clobazam) was added. The child remained seizure free over the next seven days and his sensorium returned to normal. He also started localizing objects by seeing and could count fingers. He was now on a normal diet with salt supplementation. A postoperative MR image did not reveal significant cortical damage.

Three weeks after the decompressive surgery, he was taken up for cranial reconstruction. The bone pieces placed in the anterior abdominal wall were placed back with vicryl sutures and Titanium miniplates, for reconstruction. The post operative recovery was eventful.

DISCUSSION

Depressed fractures of skull appear gruesome, but may have favourable outcomes with optimal management¹.

In this 3 year old boy there was a vertical impact to the calvarium. Brain swelling after elevating the fracture necessitated duraplasty. Conservation of healthy bone in the abdominal wall, after nibbling off grossly contaminated edges provided adequate autologous material for cranial reconstruction at a later date.

Cerebral salt wasting is a commonly underdiagnosed entity after severe brain insults². In this case, the resultant hyponatremia lead on to status epilepticus^{3,4}. The occurrence of echolalia prior to the onset of seizures suggest a common, if not sequential aetiopathogenesis⁵. The blindness manifested by the child for three to four weeks was cortical, as suggested by the presence of preserved papillary and blink reflexes. The blindness with recovery suggest axonal conduction failure in the optic radiation which would have been in the way of the transmitted shock waves. The persistence of occipital blindness for a prolonged period could also indicate subclinical seizure activity in the parieto-occipital cortex.

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