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Case Report

Shaken baby syndrome in neonate: a clinical masquerade

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ABSTRACT

Abusive head trauma, which is a subset of Shaken baby syndrome, refers to a type of brain injury that occurs when the baby's head is subjected to undesirable forces. The characteristic lack of contributory history often leads to misdiagnosis, delayed treatment and suboptimal clinical outcome. We reported a neonatal presentation of abusive head trauma which posed significant diagnostic challenges. This manuscript depicts our journey to the correct diagnosis and also a review of current literature on abusive head trauma.

Keywords: Shaken baby syndrome, Abusive head trauma, Intracranial haemorrhage, Central fever, Hypoglycorrhachia

INTRODUCTION

Shaken baby syndrome refers to a form of physical abuse typically resulting in multi system injuries with serious long-term sequelae. Abusive head trauma (AHT) which is a subset of shaken baby syndrome, is a type of traumatic brain injury that occurs when a baby's head is subjected to undesirable forces such as a violent jerk/shake. It is characterised by a constellation of neurological findings, which includes cognitive decline, seizures and multiple intracranial haemorrhages, with a peculiar sparsity of external injuries.[1] Since the history of head trauma is not forthcoming in most instances, the diagnosis of abusive head trauma is often missed in as much as 30% of cases, leading to suboptimal treatment and clinical outcome.[2] To date, very few cases of abusive head trauma have been reported from India, particularly in the neonatal age group. We are presenting a case of neonatal abusive head trauma, which posed significant diagnostic challenges at the outset.

CASE REPORT

A term female neonate presented on day 7 of life with poor feeding, decreased activity, lethargy and abnormal involuntary movements suggestive of seizures of 3 days duration and high-grade fever of 1 day duration. She was born to a 33-year-old third gravida mother by elective LSCS with a birth weight of 2.76 kg following an uneventful antenatal period. No history of any instrumentation during delivery. Baby had a normal perinatal transition and was sent home on day 4 on exclusive breastfeeding. At admission, the baby was lethargic with poor cry, tone and activity with a temperature of 101.4 °F. Head circumference was 34.5 cm with bulging anterior fontanelle. She had incomplete Moro and poor sucking reflex. Baby was maintaining saturation in room air and had no respiratory distress. Peripheral pulses were palpable and perfusion was normal. Investigations revealed neutrophilic leukocytosis and thrombocytosis with normal electrolytes and blood sugar. Bedside EEG was normal. Neurosonogram was obtained which did not show any features of intracranial haemorrhage. Diagnostic lumbar puncture was performed which revealed blood-stained CSF with low sugar, elevated protein and polymorphonuclear leukocytosis. Based on the clinical presentation, a strong suspicion of meningitis/late onset neonatal sepsis was kept in mind and the child was initiated on IV antibiotics, anti-epileptics, and other supportive medications.

Subsequently, her general condition improved along with seizure control, however she continued to have intermittent high spiking fever.

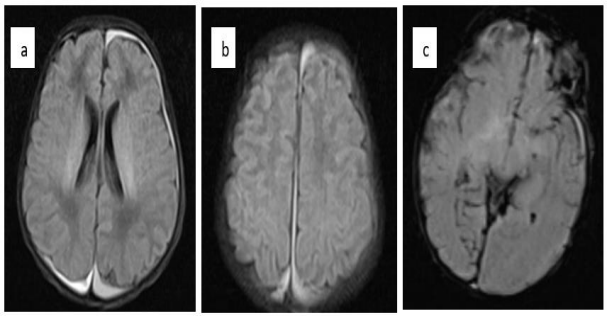

Figure 1: MRI brain images; (a) Axial T2 FLAIR image showing bilateral parietal and left frontal convexity subdural hematoma, (b) Axial T2 FLAIR showing interhemispheric subdural haemorrhage, (c) SWI sequence showing blooming along sulci in right occipito-temporal and left temporal lobe suggestive of subarachnoid haemorrhage; Small foci of blooming in bilateral lateral ventricles suggestive of intraventricular haemorrhage.

In view of absent inflammatory markers and sterile cultures, she was evaluated for other causes of neonatal seizures including metabolic work up (urine GCMS and TMS) and karyotyping which were normal. Repeat lumbar puncture on day 14 showed xanthochromic CSF with low sugar and high protein, which implicated the possibility of remote intracranial bleed. Following this, an MRI brain was performed which revealed subacute subdural hematoma over the left cerebral convexity, interhemispheric subdural haemorrhage, subarachnoid haemorrhage and intraventricular haemorrhage (Figure 1). On further probing, parents revealed the possibility of probable rough handling of the baby by an inexperienced caretaker in the immediate newborn period who was subsequently fired due to inappropriate behaviour. Thus, diagnosis for the confusing clinical scenario was clinched as abusive head trauma, which could explain the neurological findings, blood-stained CSF, hypoglycorrhachia and elevated protein levels.

There were no long bone fractures on skeletal survey and fundus examination ruled out vitreous and retinal haemorrhages. Neurosurgery consultation was sought and conservative management with antiepileptics and supportive care was recommended, along with serial head circumference measurements. She was initiated on early stimulation and physiotherapy. She was eventually noted to have rapidly increasing head circumference associated with bulging anterior fontanelle. A diagnosis of communicating hydrocephalus was confirmed with neuro-sonogram (Figure 2). Owing to progressive ventriculomegaly in the ensuing weeks, she underwent permanent CSF diversion. The possibility of negative cognitive sequelae and developmental delay was explained to the parents and close follow up was recommended.

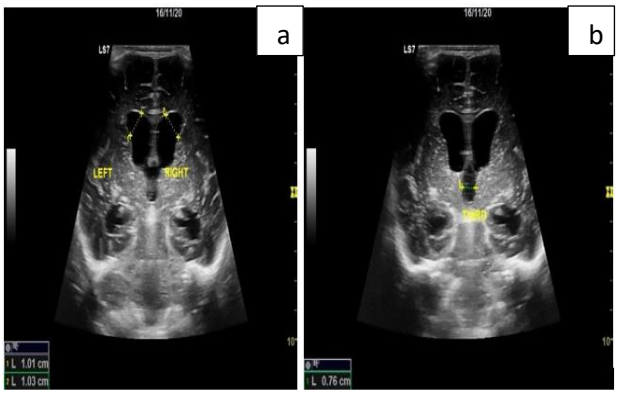

Figure 2: Coronal neurosonogram image at the level of foramen of Monro; (a) symmetrical dilation of frontal horns and temporal horns of both lateral ventricles; (b) Showing dilation of third ventricle.

DISCUSSION

Shaken baby syndrome refers to a form of abuse, in which the child is subjected to whiplash forces, often inadvertently, resulting in an array of multisystem injuries manifesting as intracranial bleeds, retinal haemorrhages and bony injuries. The history of shaking is characteristically absent, as the perpetrator inflicts the injury unknowingly out of anger or even during rough play. The term shaken baby syndrome was coined by Caffey to describe a group of infants with this typical picture where a nursemaid admitted to roughly shaking the babies by the arm or trunk.[3] Abusive head trauma, which is the most serious component of shaken baby syndrome typically manifests as fever, lethargy, poor feeding and vomiting and has been postulated to result from the effects of non-impact acceleration deceleration forces causing tearing of the bridging veins resulting in subdural hematoma, intracerebral haemorrhage and rarely death. The relatively large size of infant's head, weakness of the neck musculature, softness of the skull, relatively large subarachnoid space and high-water content of the brain are deemed to contribute to the pathogenesis of brain injuries in infants.[4]

It may be noted that the clinical findings in AHT are non-specific and can be easily confused with other commoner entities such as neuro-infection, metabolic abnormalities and toxic encephalopathy.[5] The presence of fever has been known to mislead most clinicians, and often leads to excessive workup and suboptimal treatment. It must be understood that central fever, unlike hyperpyrexia in other pathologies, is due to damage to any of the structures involved in temperature homeostasis pathways including spinal cord, midbrain and hypothalamus.[6] The occurrence of Intraventricular haemorrhage, subdural haemorrhage and subarachnoid haemorrhage, typically

seen in abusive head trauma, is now known to contribute to the pathogenesis of central fever.

The presence of associated hypoglycorrhachia in these children, resulting from cerebral ischemia and resultant diffusion of glucose from CSF to the brain, also often leads to a false diagnosis of neuro-infection. In various studies, hypoglycorrhachia is found to be a good index of meningeal haemorrhage and predictor of future post haemorrhagic hydrocephalus.[7] In the index case, even though a neuro-sonogram was obtained initially, the diagnosis was missed. This is because ultrasound visualisation of structures adjacent to bone is relatively poor. MRI has to be performed whenever there is a strong clinical suspicion of AHT, as diffusion weighted magnetic resonance imaging is the most sensitive and specific method of confirming abusive head trauma. The finding of intracranial bleeds in multiple planes along with hypoxic ischemic injury and cerebral edema are notably associated with AHT.[8] As in the current case, there are ample reports of initial misdiagnosis of AHT, due to the various factors mentioned above.

Jenny et al reported a cohort of 173 abused children, in which 54 cases were missed on initial presentation.[9] In their study of abusive head injuries, Benzel and Hadden found that 9 of 23 abused children with head injuries had seen multiple physicians before a final diagnosis of child abuse was made.[10] Laurent-Vannier et al also found that out of the 100 infants diagnosed with AHT, 75 percent underwent medical consultations wherein the diagnosis of abuse was missed.[11,12] It must now be exceedingly clear that diagnosis depends on a high index of suspicion, thorough elicitation of history and the physical findings of a raised ICP and features of unexplained encephalopathy. The finding of blood-stained CSF from a lumbar or subdural tap is also highly corroborative.

CONCLUSION

AHT is a clinical entity that is often missed by most clinicians owing to its obscure presentation. Often, the diagnosis is made in a retrospective fashion after several days, due to a low index of suspicion. Appropriate investigations must be sought at the outset, in order to clinch the diagnosis and to initiate treatment at the earliest for optimal clinical outcome. Management is mainly conservative, unless there are clinical manifestations of raised ICP secondary to intracranial haemorrhage or subsequent hydrocephalus. AHT generally has a very poor outcome with frequent major long-standing sequelae, and this has to be appropriately conveyed to the caretakers.

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