ISSN 2822-2032 (Print) 2822-2040 (Online)

Medical Journal of Eastern Nepal

Volume 01, Number 02, Issue 02, December 2022, 52-55

Case Report

CASE REPORT AND REVIEW: AN UNUSUAL CASE OF FETAL TRAUMATIC BRAIN INJURY

***Ashraya Kadariya, Pragya Bharati Rimal, Bikash Sharma Poudel, Chandra Shekhar Bhagat, Unnati Khadka, Rupak Acharya**

Department of Pediatrics, B & C Medical College Teaching Hospital and Research Centre, Birtamode Jhapa, Nepal

Submitted: 05-June-202, Revised: 11-September-2022, Accepted: 04-October-2022

DOI: https://doi.org/10.3126/mjen.v1i02.51165

ABSTRACT

We herein report an unusual case of maternal road traffic accident with blunt trauma to the abdomen at 34 weeks of gestation followed by delivery of severely encephalopathic baby and also a brief review of similar previous reports. The baby had near normal transition, then progressively developed severe encephalopathy and multiple organ dysfunction attributed mainly to the intrauterine traumatic injury. The baby had multiple intra and extra axial bleeds, cerebral edema and diffuse axonal injury, as revealed by the CT scan. The baby was managed conservatively with various neuroprotective strategies similar to management in older children and thus cerebral edema/ hematomas subsided. The baby required ventilatory support, prolonged assisted ventilation/oxygen and the metabolic derangements were managed accordingly. The baby was discharged on oral Levetiracetam, multivitamins and nasogastric tube feeds. Baby was followed up sequentially till day 74 and on physical examination had poor head growth and increased muscle tone of the extremities. Neuroimaging revealed diffuse multicystic leukoencephalomalacia, hydrocephalus ex vacuo with cortical thinning.

Intrauterine fetal brain injury is an uncommon incidence which causes mortality and significant morbidity in the child. Neuroimaging of the fetus after trauma to the mother can lead to early diagnosis and anticipation. Early vigorous neuroprotective measures and management may prevent mortality and reduce further brain insult in these neonates. There is paucity of information on management of such cases thus they should be reported in detail for study and future references.

Keywords: Case report, Neonatal encephalopathy, Neonatal TBI, Traumatic fetal brain injury

INTRODUCTION

Traumatic brain injury (TBI), a form of acquired brain injury, occurs when a sudden trauma causes damage to the brain. Car accidents is the most common form of trauma in pregnant women which occur in 2.8% of pregnant women in the USA.(1) The incidence of such road traffic accidents in our context is not known.

Symptoms of a TBI can be mild, moderate, or severe, depending on the extent of the damage to the brain. Various TBIs include concussion, edema, diffuse axonal injury, hematomas, second impact syndrome, skull fracture and penetrating injuries.(2)

Fetal trauma in pregnant women who have suffered from blunt trauma is very rare but if there is trauma to the fetal head, is usually fatal.(3) Fetal traumatic brain injury can result in severe brain atrophy, intraparen-

*Corresponding author:
Ashraya Kadariya
Email: meet.ashraya@gmail.com
ORCID: 0000-0003-3422-8380

Citation
Kadariya A, Rimal P B, Poudel B S, Bhagat C S, Khadka U, Acharya R, Case Report and Review: An Unusual Case of Fetal Traumatic Brain Injury, MJEN. 2022 December; 1(2): 52-55.

chymal and intraventricular hemorrhages, subdural hematoma and skull fractures leading to neonatal developmental delay, hypotonia, blindness, oropharyngeal dysphagia and seizures.(4) The survival of fetus who suffered from head injuries depends on the type of injury, intrauterine asphyxia, maturity and postnatal period. Long term follow-up of the fetus with brain trauma has shown to develop encephalopathy, cerebral palsy, development delay.(4) Severe TBI besides death can lead to serious physical and psychological symptoms and coma.(2)

The severity of maternal injury correlates imperfectly with fetal injury, as fetal trauma can occur without direct uterine injury and may also have a delayed presentation. Acute trauma to the mother can lead to preterm birth, direct fetal injury, maternal hypotension/hypoxia, uterine hypoperfusion which can all insult a developing brain and are speculated to cause cerebral palsy later in the child.(5)

The management of cerebral edema and diffuse axial injury in neonates is still debatable. The occurrence of intrauterine TBI and the benefit of antiedema measures, the use of encephalopathy scoring systems in near term neonates with non-asphyxia (TBI) related cerebral edema is still unknown requiring further studies, which makes the evidence-based approach more difficult. This report may shed light in management of such recurrences and future studies.

CASE REPORT

An informed consent was obtained from the father for publication of this case report and images.

ANTENATAL AND BIRTH HISTORY

A 26-year-old woman at 34 weeks of pregnancy, who had suffered a road traffic accident-causing crush injuries over left calf followed by fall to solid ground sustained blunt trauma to the lower abdomen. The mother was operated for fracture/ avulsion of the left lower leg and stabilized in the ICU. Antibiotic prophylaxis and ANS were given. CTG tracings done and were assuring. On the 2-3rd hospital stay mother started having labor pains, which was not augmented and baby was delivered spontaneous vaginally as mother was unfit for second surgery.

A live male preterm baby was born via Vaginal Delivery in the ICU. The APGAR score was 6/10 and 8/10 at 1 and 5 minutes respectively.

AFTER ADMISSION

At the NICU, baby had mild respiratory distress and thus placed on nasal CPAP. On examination the baby's limbs had extension with strong distal flexion, edema was noted over both lower limbs and multiple hematomas over body. All relevant investigations were sent. ABG done within the 1st hour was normal.

Baby started developing seizures on the first day which became refractory by day 5 and were finally controlled on Inj Phenobarbitone, Phenytoin, Valproate, Levetiracetam and midazolam infusion. Baby became progressively encephalopathic with Thomason's score on day 5 and 17 was 19 and 8 respectively. required mechanical ventilation. CT head revealed IVH-3/4, SAH, IPH, cerebral edema and features of DAI (Figure 1). It was managed conservatively with Inj Mannitol, dexamethasone, furosemide, head elevation, hypertonic saline and hyperventilation episodes for 5-7 days. Repeat CT head showed decreased edema and hematoma (Figure 2). Baby's sensorium and respiratory drive gradually improved and thus was extubated to CPAP on day 10 with periextubation caffeine.

Baby had hyponatremia with polyuria (CSW), AKI- 2 (hypoxia/drugs), transaminitis (hypoxia/direct trauma), hyperglycemia (steroid/stress), metabolic acidosis, anemia (drugs/sampling), NNH which were managed accordingly by titration of fluid/ electrolytes/drugs, bicarbonate infusion, insulin infusion, PRBCs transfusion and phototherapy.

Baby initially had negative septic screen, blood culture and CSF but later developed NEC- 1, VAP and urine KOH positive for fungal elements. Broad spectrum IV antibiotics and oral fluconazole were provided.

OG feeding was restarted by day 9, reached full feeds by day 14 and further fortified with HMF. Baby was discharged on day 23 on OG feeds, multivitamins, anti-reflux measures, oral Levetiracetam and oral fluconazole (Figure 3). Baby had static HC, hypotonia with poor suck and reflexes.

Follow up on day 42 baby had improved suck/ oropharyngeal coordination, increased tone, static HC. ROP screening was normal. Spoon and bottle feeds were commenced and physiotherapy of limbs advised. By day 74 baby had poor head growth (+0.5cm), generalized hypertonia, accepting bottle feeds with strong suck/grasp/cry and no breakthrough seizures (Figure 5). CT head showed diffuse cystic encephalomalacia, loss of deep gray and white matter, cortical thinning, hydrocephalus ex vacuo and bilateral thalamic calcifications (Figure 4). BERA and EEG was refused and prognosis was explained.

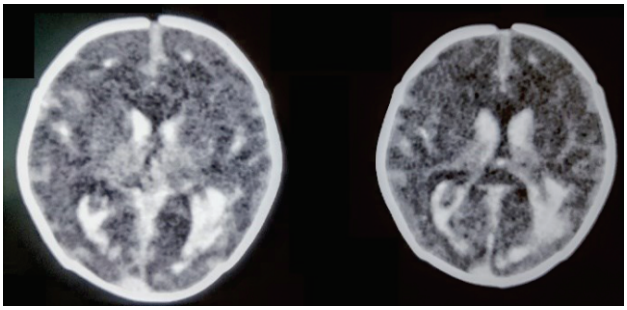

Figure 1: Cranial CT scan on 3rd day showing intraventricular, subarachnoid and intraparenchymal hemorrhage

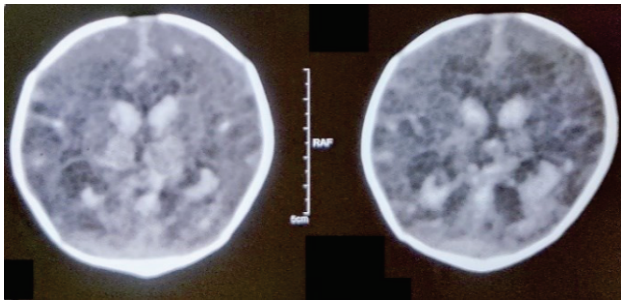

Figure 2: Cranial CT scan on day 8 revealing decrease in hematomas and edema

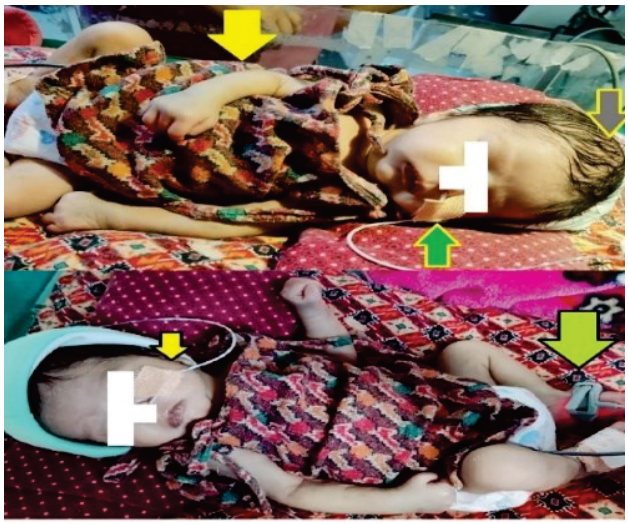

Figure 3: Baby on day 20, note the bulging Anterior Fontanelle, strong distal flexion and NG tube in situ

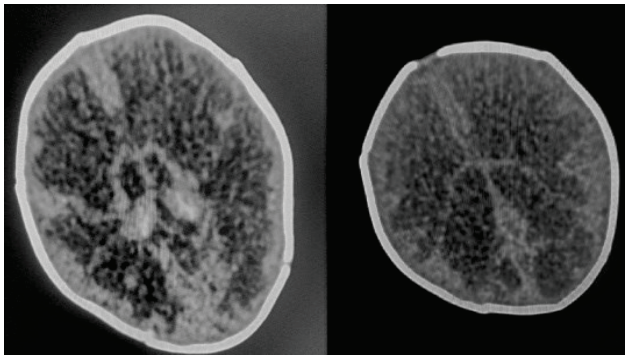

Figure 4: Plain Head CT scan on day 74, note the diffuse cystic encephalomalacia, cortical thinning and deep grey matter calcifications.

Figure 5: Baby on follow up (74th day of life) sucking and coordinating bottle feed well

DISCUSSION

Intracranial hypertension due to cerebral edema is generally managed with dehydration, corticosteroids, osmotic agents, hyperventilation and antiepileptics. There have been minimal studies on the medical management of cerebral edema in the severely asphyxiated neonates. The management of non-asphyxiated/ trauma related cerebral hemorrhage and edema in neonates is even more unknown.

The Thompson HIE score consists of 9 sets of clinical signs associated with central nervous system dysfunction in neonates.(6) It has not been used in non-asphyxiated encephalopathy but as it encompasses more clinical parameters than others, we used it in our case. More studies are required to find significant association.

The routine use of dexamethasone in severe perinatal asphyxia with edema added no benefit.(7) Previous studies have showed that Mannitol seemed to be more beneficial than dexamethasone in the management of severe perinatal asphyxia associated with cerebral edema and intracranial hypertension (8). But concerns have surrounded the adverse effects of mannitol causing hypovolemia due to diuresis and the association with renal failure.

Hyperventilation produces cerebral vasoconstriction, decreased cerebral blood flow and intracranial pressure but may induce brain ischemia. Prophylactic severe hyperventilation to P_aCO_2 < 30 mmHg should be avoided in the initial 48 hours after injury and is associated with higher rates of mortality(9). Data on hyperventilation for neonatal TBI is scarce.

Mohammad Safdari et al., 2018, reported a baby born microcephalic with global hypotonia by a mother who suffered seat belt trauma at 28 weeks but delivered at term. By 4 moths the baby had developmental delay, blindness and partial seizures. CT head cerebral hypoplasia and ventriculomegaly. (10) Similar maternal trauma at 24weeks followed by delivery at 37 weeks, with normal APGARS and microcephaly at birth had later developed porencephaly with epilepsy, developmental delay and spastic quadriparesis by 3 years(11).

A lady at 35 weeks had delivered preterm due to blunt trauma in an RTA, a live infant with Apgar scores of 1, 1, 1, and 1 in contrast to our case who had a near normal transition during birth. After complete resuscitation, the infant expired 13 min later. On autopsy, there was IVH, SAH, IPH, SGH and fracture of the overlying bone. (3). Similar traumatic born neonate suffered severe HIE and death by day 6.(12)

Fetal fractures may be visualized on CT scans performed through the gravid uterus in the second and third trimester pregnancies. (3). CT scan must be done at once because damage due to radiation is much less than the damage caused by the delay in diagnosis but moreover so MRI would be much safer and more

detailed.(13). CT scan of the fetal head was not done antepartum in our case, which might have revealed intracranial bleeds earlier.

ACKNOWLEDGMENT

We thank the baby's parents and B & C Medical College Teaching Hospital and Research center for allowing to publish this case report and images. We would also like to thank Department of Neurosurgery and Department of Radiology, B&C Hospital for valuable exert opinion for this case.

Conflict of interest: None

REFERENCE

1. Hyde LK, Cook LJ, Olson LM, Weiss HB, Dean JM. Effect of motor vehicle crashes on adverse fetal outcomes. Obstet Gynecol. 2003;102(2):279–86.
2. Maxwell WL. Traumatic brain injury in the neonate, child and adolescent human: An overview of pathology. Int J Dev Neurosci [Internet]. 2012;30(3):167–83. Available from: http://dx.doi.org/10.1016/j.ijdevneu.2011.12.008
3. Sadro CT, Zins AM, Debiec K, Robinson J. Case report: Lethal fetal head injury and placental abruption in a pregnant trauma patient. Emerg Radiol. 2012;19(2):175–80.
4. Leroy-Malherbe V, Bonnier C, Papiernik E, Groos E, Landrieu P. The association between developmental handicaps and traumatic brain injury during pregnancy: An issue that deserves more systematic evaluation. Brain Inj. 2006;20(13–14):1355–65.
5. Redelmeier DA, Naqib F, Thiruchelvam D, R Barrett JF. Motor vehicle crashes during pregnancy and cerebral palsy during infancy: a longitudinal cohort analysis. BMJ Open. 2016 Sep 20;6(9):e011972.
6. Thompson CM, Puterman AS, Linley LL, Hann FM, Elst CW Van Der, Molteno CD, et al. The value of a scoring system for hypoxic ischaemic encephalopathy in predicting neurodevelopmental outcome. 1997;(Table 1).
7. Levene MI, Evans DH. Medical management of raised intracranial pressure after severe birth asphyxia. Arch Dis Child. 1985;60(1):12–6.
8. Hossain K, Islam M, Das P. A comparative study on the use of mannitol and dexamethasone in severe perinatal asphyxia among newborn babies conducted in Ad-din Sakina Medical College, Jessore. Mediscope. 2018;5(2):16–20.
9. Curry R, Hollingworth W, Ellenbogen RG, Vavilala MS. Incidence of hypo- and hypercarbia in severe traumatic brain injury before and after 2003 pediatric guidelines. Pediatr Crit Care Med. 2008;9(2):141–6.
10. Safdari M, Safdari Z, Pishjoo M. Intrauterine Fetal Traumatic Brain Injury Following Motor Vehicle Accident; A Case Report and Review of the Literature. Vol. 6, Bulletin of emergency and trauma. 2018. p. 372–5.
11. Matsushita H, Harada A, Sato T, Kurabayashi T. Fetal intracranial injuries following motor vehicle accidents with airbag deployment. J Obstet Gynaecol Res. 2014;40(2):599–602.
12. Nishida N, Ina S, Hata Y, Nakanishi Y, Ishizawa S, Futatani T. Fetal closed head injuries following maternal motor vehicle accident. Med (United States). 2018;97(44).
13. Kopelman TR, Bogert JN, Walters JW, Gridley D, Guzman O, Davis KM, et al. Computed tomographic imaging interpretation improves fetal outcomes after maternal trauma. J Trauma Acute Care Surg. 2016;81(6):1131–4.