



Hypoxic Ischemic Encephalopathy with Cervical Spinal Cord Injury: A Diagnostic Dilemma

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Abstract

Perinatal spinal cord injury is a relatively uncommon, but a frequently misdiagnosed disorder. Improvements in obstetric care have certainly led to a decrease in the incidence of birth related spinal cord trauma but unfortunately the incidence of hypoxic-ischemic encephalopathy is still very high. The exact incidence of spinal cord trauma is difficult to determine because the spinal cord is not routinely examined in far and few neonatal autopsies done in India. Here, authors present a neonate who received treatment for birth asphyxia and then had extubation failure which made the clock tick towards cervical cord injury. This baby had a hemorrhagic contusion of cervical spinal cord.

Keywords Birth asphyxia · HIE · Spinal cord contusion · Cervical cord

Introduction

Perinatal spinal cord injury is a relatively uncommon, but a frequently misdiagnosed disorder. Here, authors present a neonate who received treatment for birth asphyxia and then had extubation failure which made the clock tick towards cervical cord injury. This baby had a hemorrhagic contusion of cervical spinal cord.

Case Report

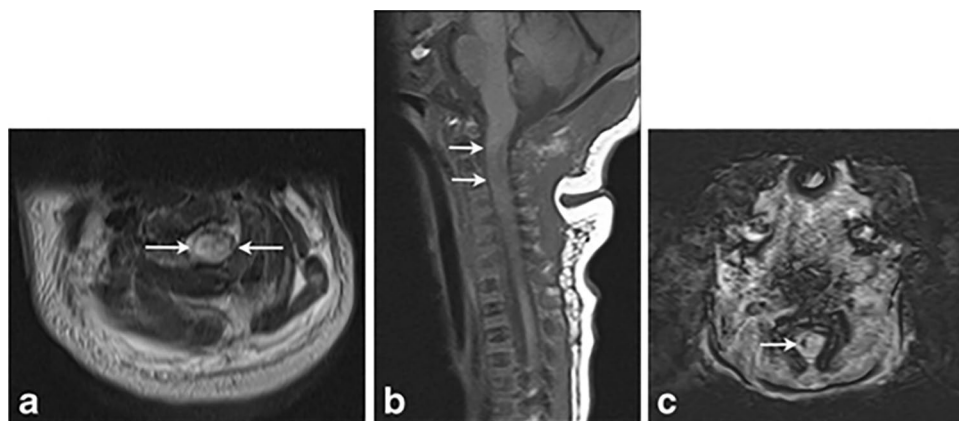
A term male baby born to a primigravida mother with uneventful antenatal history, was delivered by forceps assisted vaginal delivery with birth weight of 2.640 kg. The baby did not cry immediately after birth, required resuscitation, was ventilated and then referred at 4 h of life for cooling. At admission, the baby was hypotonic with sluggish upper & lower limb reflexes and had features of mild-moderate encephalopathy. The first arterial blood gas showed metabolic acidosis (pH: 7 and Base excess -18). Baby was fitting into both biochemical and neurological criteria for total body cooling, hence therapeutic hypothermia was

initiated after securing umbilical lines. Blood tests revealed elevated liver enzymes with normal coagulation profile. Renal function showed borderline creatinine and the baby never had oliguria. Although there were no clinical seizures, conventional EEG showed epileptiform discharges on left side for which levetiracetam was started. Baby had hypotension on day 2, which was managed with dobutamine infusion. 2D Echo showed large patent ductus arteriosus with severe pulmonary arterial hypertension (PAH). The baby was cooled to 33.5°C for 72 h followed by gradual rewarming at 0.5°C per hour over 6 h. The baby improved neurologically following total body cooling and had good sucking and facial expressions. MRI Brain was done on day 4 which showed two acutely restricting foci in right anterior and left posterior centrum semiovale, mild gyral restriction in bilateral parieto-occipital lobes (more on right side) likely suggestive of hypoxic-ischemic encephalopathy. The baby had failed extubation during which it was observed that baby had normal cry with no spontaneous movements below the level of neck. On detailed neurological examination, the baby had hypotonia and poor upper limb and lower limb reflexes, however facial movements, spontaneous eye opening, gag & sucking and pupillary reflex was intact. This led to a strong suspicion for spinal cord injury. Accordingly, MRI spine was done on day 9 which showed bulky spinal cord with T2/STIR hyperintensity (edema) and areas of T1 hyperintensity and few blooming spots on GRE-T2 weighted imaging (bleed) at cervicomedullary junction extending till C2

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Fig. 1 MRI spine of the baby showing (a) contusion and edema in cross-sectional view at the junction of brainstem and upper spinal cord, (b) hemorrhage and edema in sagittal view of the spinal cord, and (c) hemorrhagic area at C2 level (arrows)



vertebral body- suggestive of Hemorrhagic Cord Contusion (Fig. 1). Neurosurgery opinion was taken and prolonged ventilation and pulse steroid therapy was advised. Parents were counselled in depth regarding the prognosis and the continued requirement of intensive care. As the baby was referred from city outskirts, upon parents' request, he was transferred to a hospital close to family residence for the need of prolonged ventilation, however succumbed the very next day.

Discussion

Birth injury to the spinal cord secondary to traction forces during delivery is a common but frequently undiagnosed disorder. High cervical spinal cord injury in neonates is a rare but specific complication of forceps rotation. The index patient had a hemorrhagic lesion of cervical cord, which is vulnerable due to complexity of the vascular supply and because cervical cord segments also have highest metabolic rates and greatest oxygen demands [1]. Diagnosis and management are difficult and there are long-term serious medical, legal and ethical implications. Accurate localization and assessment of the severity of the lesion is of vital importance in planning the management strategies and long-term ventilation with tracheostomy is needed for prolonged ventilation [2, 3]. Sadly, the diagnosis of spinal cord injury is often missed early in the neonatal period as neurological signs may be either altered/overshadowed by accompanying hypoxic-ischemic damage or may even be falsely attributed to hypoxic-ischemic encephalopathy [4]. Even at this stage, a primary neuromuscular disorder may be difficult to differentiate but a history of good fetal movements will render this

diagnosis less likely. It is only late in the clinical course that autonomic involvement with disturbance in bladder function and development of progressive spastic paraplegia or quadriplegia make the diagnosis of spinal cord damage obvious.

In an acutely unwell term infant with symptoms of flaccid paralysis from spinal cord damage, hemorrhagic etiology needs to be considered in the differential diagnosis [5].

Authors' Contributions SG, PC, DMS: Patient management; LK, MV: Patient management and manuscript preparation. PC will act as guarantor for this manuscript.

Declarations

Conflict of Interest None.

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