Case Report

Perinatal asphyxia with hypoxic-ischemic encephalopathy stage I in a late preterm neonate: A case report

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Abstract

Background: Perinatal asphyxia is a major cause of neonatal morbidity and mortality worldwide. It results from impaired gas exchange during the perinatal period, leading to hypoxemia, hypercapnia, and metabolic acidosis. A significant complication is hypoxic-ischemic encephalopathy (HIE), which may cause long-term neurodevelopmental deficits. Early recognition and intervention are critical for preventing progression to severe neurological damage.

Keywords: Perinatal asphyxia; Hypoxic-ischemic encephalopathy; CPAP; Neonatal hyperbilirubinemia

Case Presentation: We present the case of a 36-week late preterm male infant weighing 2.771 kg, born via normal vaginal delivery with a tight nuchal cord. Apgar scores were 8 and 9 at 1 and 5 min, respectively. The infant cried after 5–6 seconds but developed respiratory distress within minutes of birth, requiring CPAP support. Neurological examination revealed increased tone, irritability, and exaggerated reflexes—consistent with HIE Stage I. Investigations showed mild bilateral periventricular flare on cranial ultrasound, normal blood glucose, and rising bilirubin levels necessitating phototherapy. Empirical antibiotics were initiated and discontinued after negative cultures. Feeding support and parental counselling were provided.

Outcome: The infant was successfully weaned from oxygen within 7 hours, feeding was established, bilirubin levels normalized, and the baby was discharged on day 3 in stable condition with follow-up planned for neurodevelopmental monitoring.

Conclusion: This case highlights the importance of prompt diagnosis, comprehensive NICU care, and multidisciplinary collaboration in the management of perinatal asphyxia with Stage I HIE, leading to a favourable prognosis.

1. Introduction

Perinatal asphyxia is defined as the failure to initiate and sustain breathing at birth, often accompanied by metabolic acidosis and hypoxic injury to vital organs. It is one of the leading causes of neonatal morbidity and mortality, particularly in developing countries, with an incidence ranging from 2 to 10 per 1000 live births in term infants and higher rates in preterm infants. Hypoxic-ischemic encephalopathy (HIE) is the neurological manifestation of perinatal asphyxia. It results from an acute or subacute brain injury caused by impaired cerebral blood flow and oxygen delivery. The severity of HIE is typically classified into three stages based on the Sarnat and Sarnat grading system, with Stage I representing the mildest form and generally associated with a favourable outcome.

SARNAT CLASSIFICATION OF HIE

	STAGE 1 - MILD	STAGE 2 - MODERATE	STAGE 3 - SEVERE
Level of Consciousness	Hyperalert	Lethargic or obtunded	Stuporous
Neuromuscular Control			
Muscle tone	Normal	Mild hypotonia	Flaccid
Posture	Mild distal flexion	Strong distal flexion	Intermittent decerebration
Stretch reflexes	Overactive	Overactive	Decreased or absent
Segmental myoclonus	Present	Present	Absent
Complex Reflexes			
Suck	Weak	Weak or absent	Absent
Moro	Strong; low threshold	Weak; incomplete; high threshold	Absent
Oculovestibular	Normal	Overactive	Weak or absent
Tonic neck	Slight	Strong	Absent
Autonomic function	Generalized sympathetic	Generalized parasympathetic	Both systems depressed
Pupils	Mydriasis	Miosis	Variable; often unequal; poor light reflex
Heart rate	Tachycardia	Weak; incomplete; high threshold	Variable
Bronchial & salivary secretions	Sparse	Profuse	Variable
Gastrointestional motility	Normal or decreased	Increased; diarrhea	Variable
Seizures	None	Common; focal or multifocal	Uncommon (excluding decerebration)

This case report aims to illustrate the presentation, diagnostic approach, and management of a late preterm infant with Stage I HIE secondary to perinatal asphyxia, correlating clinical features with investigations, nursing care, and eventual outcome. By highlighting the rapid recognition and multidisciplinary approach in this case, we seek to emphasize strategies for improving neonatal survival and neurodevelopmental prognosis.

2. Case Presentation

A 36-year-old G2 P1 L1 mother presented for delivery at 36 weeks' gestation. Antenatal history was notable for an amniotic fluid index (AFI) of 6.6 cm and otherwise normal Doppler and anomaly scans. Maternal serology (VDRL, HIV, HBsAg) was negative, and her blood group was B positive. Delivery occurred at 12:15 am via normal vaginal delivery in cephalic presentation. The infant was male, weighing 2.771 kg, appropriate for gestational age. Apgar scores were 8 at 1 minute and 9 at 5 min. A tight loop of umbilical

cord was noted around the neck. The infant cried 5–6 seconds after birth but soon developed tachypnoea, grunting, and mild subcostal retractions. The baby was initiated on delivery-room CPAP (PEEP 5 cm H_2O) and transferred to the NICU. Facial duskiness, bruises, subconjunctival haemorrhages, and jitteriness were noted. Neurological assessment revealed increased muscle tone, irritability, and exaggerated startle reflex, consistent with Stage I HIE. Newborn reflexes were intact. Respiratory distress improved over the next several hours, and CPAP was discontinued after 7 hours. Empirical intravenous amoxicillin-clavulanate was started pending sepsis work-up. Early jaundice appeared at 22 hours of life, attributed to facial bruising, with bilirubin levels in the phototherapy range per NICE guidelines. Phototherapy was given for 32 hours in total. Feeding was initially provided via paladai with IV fluids due to respiratory distress; breastfeeding was initiated once the infant's condition stabilized. Lactation counselling was given to the mother. The infant tolerated feeds well, maintained adequate urine output, and had stable hemodynamic throughout the stay. The baby was discharged on day 3, stable, feeding well, with instructions for neurodevelopmental follow-up.

3. Anatomy and Physiology

3.1 Central Nervous System (CNS)

The neonatal brain is highly metabolic, consuming significant oxygen and glucose for neuronal activity. Its immature autoregulatory mechanisms make it vulnerable to hypoxia. Grey matter regions, especially the basal ganglia, thalamus, and hippocampus, are highly susceptible to oxygen deprivation. In perinatal asphyxia, interruption of cerebral perfusion leads to neuronal energy failure, excitotoxicity, and, if prolonged, irreversible cell death.

3.2 Respiratory System

The neonatal lungs transition from a fluid-filled to an air-filled state at birth. Surfactant production reduces surface tension, aiding alveolar stability. In the event of hypoxia, impaired respiratory effort, poor gas exchange, and increased work of breathing can occur. This leads to reduced arterial oxygen content and worsens tissue hypoxia.

3.3 Cardiovascular System

At birth, closure of fetal shunts (ductus arteriosus, foramen ovale) redirects blood through the lungs for oxygenation. Hypoxia can delay or reverse this transition, causing persistent fetal circulation and reduced systemic oxygen delivery. Myocardial depression may result from prolonged hypoxia.

4. Pathophysiology

Perinatal asphyxia initiates a cascade of events

- **Hypoxic-Ischemic Insult:** Tight nuchal cord compromises umbilical blood flow, reducing oxygen delivery.
- **Anaerobic Metabolism:** Inadequate oxygen shifts cellular metabolism to anaerobic pathways, producing lactic acid and causing metabolic acidosis.
- **Primary Energy Failure:** ATP depletion disrupts ion gradients, leading to neuronal swelling and dysfunction.
- **Reperfusion Injury:** Restoration of blood flow generates reactive oxygen species, causing oxidative stress and inflammation.
- **Neuronal Injury:** Excitatory neurotransmitters (e.g., glutamate) cause calcium influx, activating cell death pathways.

In **Stage I HIE**, the injury is mild and largely reversible, characterized by hyper alertness, increased tone, exaggerated reflexes, and absence of seizures.

5. Investigations

- **Blood Group:** Baby O positive, mother B positive.
- **CBC at birth:** Total Count 21,130/mm^3; Neutrophils 67%.
- **CRP**: 1.3 mg/dl at 22 hours; reduced to 0.55 mg/dl.
- **Blood Culture**: No growth.
- **VBG at 1.5 hours**: pH 7.33 (mild acidosis).
- **Cranial Ultrasound:** Mild bilateral periventricular flare; no intracranial hemorrhage.
- **Bilirubin:** 9.1 mg/dl at 22 hours → 7.2 mg/dl at 41 hours → 12.1 mg/dl pre-discharge (normal for age).
- **Renal Function:** Serum creatinine 1.1 mg/dl at 22 hours → 0.82 mg/dl pre-discharge; electrolytes normal.
- **Screening Tests:** OAE pass; red reflex present bilaterally; CCHD screening normal (SpO_2 98% upper limb, 99% lower limb).

6. Management and Medications

- **Respiratory Support:** DR CPAP at PEEP 5 cm H_2O; weaned after 7 hours once distress resolved.
- **Antibiotics:** Empirical IV amoxicillin-clavulanate for 3 days, stopped after negative cultures.
- **Phototherapy:** Initiated at 22 hours for bilirubin 9.1 mg/dl; continued for 32 hours, discontinued after safe levels reached.
- **Feeding:** Initially formula by paladai with IV fluids; transitioned to direct breastfeeding with top-up feeds as tolerated.

- **Monitoring:** Continuous SpO_2, heart rate, temperature; neurological checks; bilirubin trends.
- **Parental Support:** Lactation counseling; education on infant care and warning signs.

7. Nursing Diagnosis

- Impaired Gas Exchange related to altered alveolar-capillary membrane function secondary to perinatal asphyxia.
- Risk for Altered Neurological Function related to hypoxic insult.
- Imbalanced Nutrition: Less than Body Requirements related to difficulty initiating breastfeeding.
- Risk for Hyperbilirubinemia related to bruising and red blood cell breakdown.
- Parental Anxiety related to NICU admission and uncertain outcome.

Nursing Interventions and Rationales

Intervention	Rational
Maintain CPAP and monitor oxygen saturation	Prevents hypoxia and supports alveolar ventilation
Perform regular neurological assessments	Detects early signs of deterioration or seizure activity
Provide lactation support and feeding assistance	Ensures adequate nutrition and promotes maternal-infant bonding
Administer and monitor phototherapy	Reduces serum bilirubin levels and prevents kernicterus
Educate and reassure parents	Reduces anxiety and enhances participation in care

8. Outcome and Follow-Up

The infant was weaned off CPAP within 7 hours, remained hemodynamically stable, and exhibited no seizure activity. Feeding was well established with adequate weight gain potential. Bilirubin levels normalized, and the infant was discharged on day 3 in good condition. Follow-up was arranged for neurodevelopmental monitoring to ensure early identification of any subtle deficits.

9. Discussion

This case demonstrates that Stage I HIE following perinatal asphyxia can have an excellent prognosis with early recognition and timely intervention. The infant's tight nuchal cord likely caused transient hypoxia, but rapid initiation of CPAP and close neurological monitoring prevented progression to more severe HIE stages. The literature indicates that Stage I HIE has a high rate of complete recovery if managed promptly. CPAP provides non-invasive respiratory support, reducing the work of breathing and improving oxygen delivery without the complications of mechanical ventilation. Nursing care is central to recovery, encompassing vigilant monitoring, feeding support, phototherapy management, and parental education. Similar studies highlight that coordinated neonatal team efforts reduce morbidity and improve neurodevelopmental outcomes in mild HIE cases. Long-term follow-up is essential because even mild HIE can occasionally result in learning difficulties or motor delays. This case reinforces the importance of early stabilization, multidisciplinary care, and ongoing surveillance in infants at risk of hypoxic brain injury.