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Hypoxic Ischemic Encephalopathy Grade II with Severe Respiratory Distress due to Coexisting Laryngomalacia and Gastroesophageal Reflux Disease in a Term Newborn

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Abstract
Hypoxic ischemic encephalopathy (HIE) is a major cause of neonatal morbidity. Respiratory distress in neonates with HIE is often multifactorial and may be exacerbated by associated airway and gastrointestinal conditions. We report a case of a term newborn presenting on day of life three with HIE grade II, severe respiratory distress with recurrent desaturation, and coexisting mild laryngomalacia and gastroesophageal reflux disease (GERD). Early recognition of contributing factors and multidisciplinary management resulted in favourable short-term outcome.

Keywords: Hypoxic ischemic encephalopathy, HIE grade II, neonatal respiratory distress, laryngomalacia, GERD

Introduction
Hypoxic ischemic encephalopathy occurs due to perinatal asphyxia and remains an important cause of neonatal mortality and long-term neurodevelopmental impairment. HIE grade II is characterised by altered sensorium, hypotonia, seizures, and autonomic instability.
Laryngomalacia is the most common congenital cause of stridor in infants and may present with respiratory distress and desaturation. Gastroesophageal reflux disease (GERD) is frequently associated with laryngomalacia and may aggravate airway obstruction. In neonates with neurological compromise, even mild airway abnormalities may lead to significant respiratory distress.

Case Report
A term neonate, born at 39 weeks of gestation, presented on day of life 3 with poor feeding, lethargy, abnormal movements, and severe respiratory distress.
The baby was delivered by emergency cesarean section for fetal distress. Birth weight was 3.1 kg. Apgar scores were 5 at 1 minute and 7 at 5 minutes. The neonate required brief positive pressure ventilation at birth.

Clinical Findings
On admission, the neonate was lethargic with:

- Tachypnea (RR 72/min)
- Severe subcostal and intercostal retractions
- Nasal flaring and grunting
- Recurrent oxygen desaturation episodes (SpO_2 85–88% on room air)

Neurological examination revealed hypotonia, weak primitive reflexes, and intermittent subtle seizures. A high-pitched inspiratory stridor was noted, worsening during feeding and crying. The baby also had frequent regurgitation and choking during feeds.

Investigations

- Arterial blood gas: Metabolic acidosis
- Blood glucose and electrolytes: Normal
- Sepsis screen: Negative
- Chest X-ray: Normal lung fields
- EEG: Abnormal background activity with seizure discharges
- MRI brain: Findings consistent with moderate hypoxic ischemic injury involving basal ganglia and thalami
- Flexible laryngoscopy: Omega-shaped epiglottis with inspiratory supraglottic collapse suggestive of mild laryngomalacia

Diagnosis

- Hypoxic ischemic encephalopathy – Grade II (Sarnat staging)
- Severe respiratory distress with recurrent desaturation
- Mild laryngomalacia
- Gastroesophageal reflux disease

Management

The neonate was managed in the neonatal intensive care unit.

Respiratory Support

- Oxygen supplementation via nasal prongs
- Escalation to CPAP due to persistent respiratory distress and desaturation
- Propped-up positioning

Neurological Management

- Supportive care and maintenance of normothermia
- Seizure control with intravenous anticonvulsants
- Continuous neurological monitoring

Laryngomalacia

- Conservative management due to mild severity
- Close monitoring for airway compromise

GERD

- Small, frequent feeds
- Upright positioning after feeds
- Thickened feeds
- Proton pump inhibitor therapy

Outcome

The neonate showed gradual improvement in respiratory status with reduction in desaturation episodes. CPAP was successfully weaned. Seizures were controlled and feeding tolerance improved. Stridor decreased over time.

At discharge, the baby was maintaining oxygen saturation in room air and feeding adequately. Neurodevelopmental and ENT follow-up was advised.

Discussion

Respiratory distress in neonates with HIE is often multifactorial. Central respiratory dysregulation, hypotonia, and autonomic instability may worsen airway patency. Laryngomalacia, though mild anatomically, can cause significant respiratory compromise in neurologically affected infants. GERD further exacerbates symptoms by increasing laryngeal irritation and aspiration risk.

This case emphasizes the importance of evaluating airway and reflux pathology in neonates with HIE presenting with severe or disproportionate respiratory distress.

Conclusion

Neonates with HIE may present with severe respiratory distress due to coexisting laryngomalacia and GERD. Early diagnosis and multidisciplinary management are essential for improving outcomes.

References (Vancouver Style)

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