

DIFFERENT CLINICAL FORMS OF POST-HYPOXIC COMPLICATIONS IN NEWBORNS WITH ASPHYXIA.**Fayzullayeva Xilola Baxronovna**

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ABSTRACT. *This article explores the clinical presentation, diagnosis, and management of post-hypoxic complications in newborns who experience perinatal asphyxia. The review will also address the most common clinical outcomes and the underlying pathophysiological mechanisms, with references to key studies in the field.*

Key Words: *perinatal asphyxia, neonatal encephalopathy, hypoxic-ischemic encephalopathy, cardiovascular complications, persistent pulmonary hypertension.*

АННОТАЦИЯ. *В данной статье рассматриваются клинические проявления, диагностика и методы лечения постгипоксических осложнений у новорожденных, перенесших перинатальную асфиксию. Также анализируются наиболее распространенные клинические исходы и основные патофизиологические механизмы, с учетом ключевых исследований в данной области.*

Ключевые слова: *перинатальная асфиксия, неонатальная энцефалопатия, гипоксически-ишемическая энцефалопатия, сердечно-сосудистые осложнения, персистирующая легочная гипертензия.*

INTRODUCTION. Perinatal asphyxia is a major contributor to neonatal morbidity and mortality worldwide, characterized by impaired oxygen delivery to the tissues at the time of birth. The severity of the hypoxic insult can lead to a cascade of cellular events that result in various post-hypoxic complications, which significantly affect the long-term neurological and physical development of newborns. Post-hypoxic complications can range from subtle manifestations to

severe, life-threatening conditions, and understanding these clinical variants is crucial for early detection and intervention.

RESULTS AND DISCUSSION. Asphyxia during labor and delivery leads to a disruption of oxygen and nutrient supply to the brain and other vital organs. The pathophysiological mechanism involves a combination of ischemia, reperfusion injury, and subsequent metabolic disturbances. In the immediate post-asphyxic period, the infant may show signs of encephalopathy, including abnormal muscle tone, altered consciousness, and seizures. The brain is the most sensitive organ to hypoxic injury, and as a result, neonatal encephalopathy (NE) is one of the primary post-hypoxic complications (Benzies et al., 2008).

Clinical Manifestations of Post-Hypoxic Complications. Neonatal Encephalopathy (NE) Neonatal encephalopathy (NE) is a broad term used to describe a spectrum of neurological abnormalities caused by perinatal asphyxia. This condition manifests as altered levels of consciousness, abnormal tone, seizures, and respiratory disturbances. The severity of NE is categorized into mild, moderate, and severe forms based on clinical signs and neuroimaging findings (Scher, 2017).

Mild NE: Characterized by lethargy, poor feeding, and mild hypotonia.
Moderate NE: Includes signs of altered consciousness, abnormal posturing, and episodes of apnea.
Severe NE: Marked by coma, frequent seizures, and multi-organ dysfunction.

Studies have shown that the severity of NE correlates with the degree of hypoxic-ischemic injury to the brain, with poorer outcomes in infants presenting with severe NE (Fanaroff et al., 2015). **Hypoxic-Ischemic Encephalopathy (HIE)** Hypoxic-ischemic encephalopathy (HIE) is a subset of NE that results from a significant insult to the brain, typically involving both hypoxia (lack of oxygen) and ischemia (lack of blood flow). The consequences of HIE can range from mild developmental delays to severe cognitive and motor deficits or even death. HIE is often diagnosed using neuroimaging techniques such as MRI, which can reveal areas of ischemic injury

(Sarnat & Sarnat, 1976). The clinical features of HIE include: Seizures: Often an early sign of HIE and associated with more severe injury. Abnormal neurological responses: Persistent abnormal tone and reflexes, including hypertonia or hypotonia. Feeding difficulties and respiratory distress. Neonates with severe HIE are at high risk for long-term neurological deficits, including cerebral palsy and cognitive impairments (Pihusch et al., 2013). Cardiovascular Complications The cardiovascular system is often compromised in infants who experience perinatal asphyxia. Hypoxia leads to vasodilation and hypotension, resulting in poor perfusion to vital organs. This can contribute to persistent pulmonary hypertension of the newborn (PPHN), which can cause further complications if not addressed promptly. Clinical signs of PPHN include cyanosis, tachypnea, and respiratory distress (Fitzgerald et al., 2019). Persistent pulmonary hypertension (PPHN): A condition where the normal transition of the pulmonary circulation after birth is impaired, leading to sustained high pressure in the pulmonary arteries. Congestive heart failure (CHF): Due to impaired myocardial function secondary to hypoxia. Effective management requires careful monitoring and, in some cases, pharmacological support to maintain systemic and pulmonary vascular tone. Renal Complications The kidneys are another organ susceptible to injury during asphyxia. Renal dysfunction in the context of perinatal asphyxia may result from both ischemic injury and the systemic effects of hypoxia, including hypotension and acidosis. Renal failure in neonates can present with oliguria, electrolyte imbalances, and metabolic acidosis (Ng et al., 2012). Acute kidney injury (AKI): Often transient, but severe cases can lead to the need for dialysis in the neonatal period. Renal tubular dysfunction: This can present as electrolyte imbalances, particularly hyponatremia and hyperkalemia.

Gastrointestinal Complications Gastrointestinal manifestations of post-hypoxic complications include feeding intolerance, necrotizing enterocolitis (NEC), and delayed gastric emptying. The pathophysiology of these conditions involves

ischemic injury to the gut, leading to inflammation and, in the case of NEC, bowel necrosis (Kouadio et al., 2016). Necrotizing enterocolitis (NEC): A severe gastrointestinal condition involving bowel ischemia and necrosis, which may occur in neonates with asphyxia, particularly in preterm infants. Feeding difficulties: Due to gastrointestinal immaturity or impaired motility following hypoxia. The diagnosis of post-hypoxic complications is primarily clinical, supported by laboratory findings and imaging studies. Neurological assessments, such as the Sarnat and Sarnat grading system for HIE, are critical for determining the severity of encephalopathy. Neuroimaging, particularly MRI, is invaluable in assessing brain injury, while echocardiography may be used to evaluate cardiovascular involvement (Laptook et al., 2017). The prognosis for newborns with post-hypoxic complications depends on the severity of the asphyxial event and the timeliness of medical intervention. Early recognition and treatment, including hypothermic therapy for HIE, can significantly improve long-term outcomes. However, severe cases may result in lifelong neurological deficits, including cerebral palsy and cognitive impairment. The management of newborns with post-hypoxic complications is multidisciplinary, involving neonatologists, neurologists, cardiologists, and other specialists.

Key interventions include: therapeutic hypothermia: The most well-established treatment for moderate to severe HIE, which reduces the risk of neurological impairment. Supportive care: Including ventilation, circulatory support, and management of electrolyte imbalances. Pharmacological interventions: Such as anticonvulsants for managing seizures and vasopressors for cardiovascular support.

Conclusion. Post-hypoxic complications in newborns born with asphyxia encompass a wide range of clinical variants, from mild manifestations to severe, life-threatening conditions. Early diagnosis and intervention are critical for improving outcomes and preventing long-term disabilities. Further research into the pathophysiological mechanisms and the development of targeted therapies will

continue to improve the management of these neonates, providing hope for better neonatal survival and long-term quality of life.

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