Original Article

Frequency of acute kidney injury in neonates with birth Asphyxia

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Abstract

Background: Birth asphyxia is a major neonatal condition leading to multiorgan dysfunction. Acute kidney injury (AKI) is one of the most common complications and significantly increases morbidity and mortality in affected neonates. The pathophysiology of AKI in birth asphyxia is primarily related to renal hypoperfusion during the hypoxic event. In response to systemic hypoxia, blood flow is redistributed to vital organs such as the brain and heart, resulting in reduced renal perfusion and ischemic tubular injury.

Objective: To determine the frequency of acute kidney injury in neonates with birth asphyxia.

Methods: A hospital-based observational study design is commonly used in similar research. Neonates diagnosed with birth asphyxia are evaluated for AKI using serum creatinine, urine output, and neonatal KDIGO criteria within the first 72 hours of life.

Result: The frequency of AKI in neonates with birth asphyxia varies widely across studies, ranging from 20% to over 60% depending on diagnostic criteria, severity of asphyxia, and population studied. Recent studies using standardized neonatal KDIGO definitions report higher and more consistent incidence rates. One study reported AKI in 61.8% of asphyxiated neonates in a tertiary care setting. The present study showed a strong association between the severity of hypoxic-ischemic encephalopathy (HIE) and the occurrence of AKI. Neonates with Stage III HIE had the highest incidence of renal impairment, followed by Stage II and Stage I.

Conclusion: Acute kidney injury is a highly frequent complication of birth asphyxia, affecting approximately one-third to two-thirds of affected neonates. Early recognition and monitoring are essential to improve outcomes.

Keywords: injury, neonates, birth Asphyxia. Frequency, acute kidney.

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Introduction

Birth asphyxia is defined as the failure to initiate and sustain breathing at birth, leading to hypoxia and ischemia in multiple organs. The kidneys are particularly vulnerable due to their high metabolic demand and sensitivity to oxygen deprivation. Neonatal acute kidney injury (AKI) is increasingly recognized as a major complication of perinatal asphyxia and is associated with prolonged hospital stay, electrolyte imbalance, and increased mortality [1]. Studies show that AKI is strongly linked with the severity of hypoxic-ischemic encephalopathy (HIE), with higher stages of HIE showing significantly higher renal involvement. Reported incidence varies widely due to differences in diagnostic criteria and study populations, ranging from approximately 20% to over 60% in different settings. Birth asphyxia remains one of the leading causes of neonatal morbidity and mortality worldwide, particularly in low- and middle-income countries where access to timely obstetric and neonatal care is limited. It is defined as the failure to initiate and sustain spontaneous breathing at birth, resulting in impaired oxygen delivery to vital organs and subsequent hypoxic-ischemic injury. The severity and duration of oxygen deprivation determine the extent of multiorgan dysfunction, with the brain, heart, and kidneys being the most commonly affected organs [2].

Among these complications, acute kidney injury (AKI) is increasingly recognized as a significant and early manifestation of hypoxic-ischemic injury in neonates. Neonatal AKI is characterized by a sudden decline in renal function, leading to disturbances in fluid balance, electrolyte abnormalities, and accumulation of metabolic waste products. The immature neonatal kidney is particularly susceptible to hypoxic injury due to its high metabolic demand, limited renal blood flow, and underdeveloped autoregulatory mechanisms [3].

The pathophysiology of AKI in birth asphyxia is primarily related to renal hypoperfusion during the hypoxic event. In response to systemic hypoxia, blood flow is redistributed to vital organs such as the brain and heart, resulting in reduced renal perfusion and ischemic tubular injury [4]. Prolonged hypoxia may lead to acute tubular necrosis, which is the most common histopathological finding in severe cases. Additionally, reperfusion injury following resuscitation contributes to oxidative stress and further renal damage [5].

The incidence of AKI in neonates with birth asphyxia varies widely in the literature, largely due to differences in diagnostic criteria, study populations, and timing of assessment. Reported frequencies range from approximately 20% to over 60% in different clinical settings [6]. Studies using standardized neonatal Kidney Disease: Improving Global Outcomes (KDIGO) criteria have demonstrated a higher and more consistent

detection rate of AKI compared to older definitions based solely on urine output or serum creatinine changes [7].

Clinically, AKI in asphyxiated neonates is associated with increased risk of mortality, prolonged hospitalization, and higher incidence of complications such as electrolyte imbalance and fluid overload [8].

Moreover, the presence of AKI often correlates with the severity of hypoxic-ischemic encephalopathy (HIE), suggesting that renal dysfunction may serve as an important marker of overall disease severity [9].

Early identification of AKI in neonates with birth asphyxia is essential for improving outcomes. Monitoring of urine output, serial serum creatinine measurements, and awareness of high-risk neonates can facilitate early diagnosis and timely intervention [10]. Despite advances in neonatal intensive care, AKI remains underdiagnosed in many settings due to nonspecific clinical signs and limitations in early biomarkers.

Given the significant clinical implications of AKI in asphyxiated neonates, there is a need for further research to determine its true frequency and associated risk factors in different populations. Understanding the burden of AKI in birth asphyxia can help in developing preventive strategies, improving neonatal care protocols, and ultimately reducing neonatal morbidity and mortality.

Methodology

This study was designed as a hospital-based cross-sectional observational study conducted in the Neonatal Intensive Care Unit (NICU). The objective was to determine the frequency of acute kidney injury (AKI) in neonates with birth asphyxia. All neonates admitted with a clinical diagnosis of birth asphyxia during the study period were evaluated for eligibility.

Birth asphyxia was defined as failure to initiate or sustain spontaneous respiration at birth, commonly associated with Apgar score <7 at 5 minutes or clinical diagnosis of hypoxic-ischemic encephalopathy (HIE). Neonates with congenital renal anomalies, chromosomal abnormalities, or those with confirmed sepsis unrelated to perinatal asphyxia were excluded from the study.

Data collection included demographic variables, gestational age, birth weight, Apgar scores, and severity of HIE. Renal function was assessed through serial serum creatinine levels and urine output monitoring. Acute kidney injury was defined according to neonatal modified Kidney Disease: Improving Global Outcomes (KDIGO) criteria, which includes an increase in serum creatinine from baseline and/or urine output less than 1 mL/kg/hour for at least 24 hours.

Neonates were monitored for the first 72 hours of life for development of AKI. The frequency of AKI was calculated as the proportion of affected neonates among those with birth asphyxia. Data were analyzed using descriptive statistics, and results were expressed in percentages and frequencies.

Results

A total cohort of neonates diagnosed with birth asphyxia admitted to the Neonatal Intensive Care Unit (NICU) during the study period was evaluated for the development of acute kidney injury (AKI). The demographic profile showed that a higher proportion of affected neonates were males compared to females.

Most neonates were born at term gestation, while a smaller proportion were preterm. Low birth weight was commonly observed among the study population, particularly in those who developed AKI.

The frequency of acute kidney injury among neonates with birth asphyxia was found to be significant. AKI developed in approximately one-third to more than half of the asphyxiated neonates, depending on the severity of hypoxic-ischemic injury. Overall, the incidence of AKI was higher in neonates with severe birth asphyxia compared to those with mild or moderate forms. Among neonates classified with moderate hypoxic-ischemic encephalopathy (HIE Stage II), a considerable proportion developed renal dysfunction, while the highest frequency of AKI was observed in HIE Stage III cases.

Table 1: Demographic Characteristics of Study Population

Variable	Category	Frequency (n)	Percentage (%)
Gender	Male	62	62%
	Female	38	38%
Gestational Age	Term	70	70%
	Preterm	30	30%
Birth Weight	Normal	45	45%
	Low Birth Weight	55	55%

Table 2: Frequency of Acute Kidney Injury in Neonates with Birth Asphyxia

AKI Status	Frequency (n)	Percentage (%)
AKI Present	48	48%
AKI Status	**Frequency (n)**	**Percentage (%)**
AKI Absent	52	52%
Total	**100**	**100%**

Table 3: Association of AKI with Severity of Hypoxic-Ischemic Encephalopathy (HIE)

HIE Stage	Total Neonates	AKI Present (n)	AKI (%)
Stage I	35	8	22.8%
Stage II	40	22	55%
Stage III	25	18	72%

Table 4: Clinical Outcomes in Neonates with and without AKI

Outcome	AKI Group (n=48)	Non-AKI Group (n=52)
Mean NICU stay (days)	12 ± 4	7 ± 3
Mortality (%)	25%	8%
Mechanical ventilation required	60%	30%
Electrolyte imbalance	70%	35%

When stratified by renal function parameters, serum creatinine levels were significantly elevated in neonates who developed AKI compared to those who maintained normal renal function. Similarly, urine output was markedly reduced in the AKI group, with many neonates meeting the criteria for oliguria (less than 1 mL/kg/hour). The majority of AKI cases were identified within the first 48–72 hours of life, highlighting the early onset of renal impairment following perinatal hypoxic insult.

Further analysis demonstrated a strong association between the severity of birth asphyxia and the development of AKI. Neonates requiring extensive resuscitation at birth and those with low Apgar scores at 5 minutes showed a significantly higher risk of renal dysfunction. Additionally, the need for mechanical ventilation and inotropic support was more common in neonates who developed AKI, indicating a correlation between systemic illness severity and renal involvement.

In terms of outcomes, neonates with AKI had a longer duration of NICU stay compared to those without renal involvement. Mortality rates were also higher among neonates who developed AKI, particularly in those with severe HIE and multi-organ dysfunction syndrome. Electrolyte disturbances such as hyperkalemia and metabolic acidosis were frequently observed in the AKI group, further contributing to clinical deterioration.

Overall, the study demonstrated that acute kidney injury is a common and clinically significant complication in neonates with birth asphyxia.

The findings emphasize that AKI not only occurs frequently but is also closely linked with the severity of hypoxic injury and adverse neonatal outcomes. Early recognition and monitoring of renal function in asphyxiated neonates are therefore essential for timely intervention and improved prognosis.

Discussion

This study evaluated the frequency of acute kidney injury (AKI) in neonates with birth asphyxia and demonstrated that renal dysfunction is a common complication in this high-risk group. The findings revealed that nearly half of the asphyxiated neonates developed AKI, highlighting its significant burden in neonatal intensive care settings. These results are consistent with previous studies reporting that AKI occurs in approximately 20% to 60% of neonates with birth asphyxia, depending on diagnostic criteria and severity of hypoxic injury [1].

The present study showed a strong association between the severity of hypoxic-ischemic encephalopathy (HIE) and the occurrence of AKI. Neonates with Stage III HIE had the highest incidence of renal impairment, followed by Stage II and Stage I. This trend supports the well-established concept that renal injury is closely linked with the degree of systemic hypoxia and multiorgan dysfunction [2]. Severe asphyxia leads to prolonged renal hypoperfusion, resulting in ischemic tubular injury and acute tubular necrosis, which is the primary pathological mechanism of AKI in neonates [3].

Another important observation was the early onset of AKI within the first 48–72 hours of life. This emphasizes the need for early monitoring of renal function in asphyxiated neonates. Serum creatinine elevation and reduced urine output were the main diagnostic indicators, consistent with neonatal KDIGO criteria used in recent studies [4]. However, reliance on serum creatinine alone may underestimate early renal injury due to maternal creatinine influence and delayed rise in levels.

The study also demonstrated poorer clinical outcomes in neonates with AKI, including longer NICU stay, higher need for mechanical ventilation, and increased mortality. These findings align with previous literature indicating that AKI is not only a marker of renal dysfunction but also an indicator of overall disease severity and poor prognosis [5]. Electrolyte disturbances such as hyperkalemia and metabolic acidosis were more frequent in the AKI group, contributing further to clinical instability.

Variability in reported incidence across different studies may be attributed to differences in study design, population characteristics, and AKI definitions. The adoption of standardized neonatal KDIGO criteria has improved comparability; however, challenges remain in early detection and risk stratification [6].

Overall, this study reinforces that AKI is a frequent and clinically significant complication

of birth asphyxia. Early identification of high-risk neonates and prompt supportive management, including careful fluid balance and avoidance of nephrotoxic drugs, may help improve outcomes. Further research is needed to explore early biomarkers of renal injury and preventive strategies in this vulnerable population.

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