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Original Research Article

Utility of Urinary Uric Acid–Creatinine Ratio as a Marker of Neonatal Asphyxia: A Comparative Retrospective Study

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Abstract:

Background: Particularly in underdeveloped nations, perinatal hypoxia continues to be a significant contributor to neonatal death, morbidity, and complications. The incidence of hypoxic ischemic damage is 0.3 to 2 per 1000 term infants, whereas the prevalence of perinatal asphyxia is 1 to 6 per 1000 live births. There was a need to detect infants with asphyxia at risk for hypoxia ischemic encephalopathy and multi-organ damage utilizing simple bedside testing. The study's goals were to determine the effectiveness of the uric acid to creatinine (UA/Cr) ratio in early spot urine samples as a diagnostic tool for perinatal asphyxia and to determine the correlation between the severity of HIE and the urinary UA/Cr ratio.

Methods: This study was conducted on 60 neonates having asphyxia (cases) and 60 neonates without asphyxia (controls) born at Patna medical college and hospital throughout 12 months. Spot urine samples were taken within 24 hours of life and forwarded for anal

Results: Infants that were asphyxiated had a greater urine UA/Cr ratio (2.83) than the control group (1.14). The severe asphyxia was observed in 15 neonates with the mean UUA/Cr ratio of 2.58 ± 0.72.

Conclusions: Neonates with congenital defects and premature babies typically have lower Apgar scores. Therefore, in order to make an early choice regarding the baby's level of care, it is desirable that the clinical diagnosis of hypoxia based on APGAR scores be supported by additional studies. For the early detection and evaluation of neonatal hypoxia, the urine uric acid/creatinine ratio was found to be a good, early, straightforward, and reliable screening test. The UUA/Cr ratio is a simple and reliable screening test for early detection and assessment of perinatal hypoxia.

Keywords: Urinary Uric Acid/Creatinine Ratio, Hypoxic Ischemic Encephalopathy, Perinatal Asphyxia.

Introduction

Perinatal asphyxia continues to be a major contributor to infant morbidity and mortality, especially in low- and middle-income nations [1]. It is linked to hypoxic–ischemic encephalopathy (HIE), multi-organ dysfunction, long-term neurodevelopmental damage, and mortality [2]. Prompt diagnosis of new-borns who have undergone substantial hypoxia is essential for appropriate intervention, risk assessment, and prognosis [3].

Standard diagnostic instruments for neonatal asphyxia encompass Apgar ratings, umbilical cord blood gas analysis, and clinical evaluation of neurological condition. Nonetheless, these criteria possess limitations: Apgar scores are subjective and affected by resuscitative interventions, whereas blood gas analysis may not consistently be accessible or practical, particularly in resource-constrained environments [4]. Consequently, there remains an ongoing pursuit for straightforward, cost-effective, and dependable biochemical markers indicative of hypoxia damage.

Hypoxia causes defective oxidative phosphorylation, which accelerates the degradation of adenosine triphosphate (ATP) and results in heightened synthesis of purine metabolites, including hypoxanthine, xanthine, and finally uric acid [5]. Uric acid is eliminated in urine, and its content increases after hypoxic stress. The urinary uric acid to creatinine (UUA/Cr) ratio has been suggested as a reliable and non-invasive indicator of fetal hypoxia to account for fluctuations in urine concentration [6].

This retrospective study seeks to assess the efficacy of the urine uric acid–creatinine ratio as a biochemical indicator of neonatal asphyxia and to compare its levels in asphyxiated versus non-asphyxiated neonates.

Materials and Methods

Study Design and Setting: This retrospective comparative study was conducted in the neonatal unit of Patna medical college and hospital, Bihar over 1 year. The medical records and laboratory data of new-borns admitted within a specified study period were examined.

Study Population: A total of 120 neonates participated in the study, which was divided into two groups:

- Cases (n = 60): Neonates diagnosed with perinatal asphyxia.
- Controls (n = 60): Healthy neonates without evidence of perinatal asphyxia.

Inclusion Criteria

- Term neonates (gestational age ≥37 weeks).
- For cases: clinical diagnosis of perinatal asphyxia based on low Apgar score at 5 minutes (<7), need for resuscitation, and/or clinical features suggestive of hypoxic–ischemic injury.
- For controls: normal Apgar scores (≥7 at 5 minutes) and absence of perinatal complications.

Exclusion Criteria

- Preterm neonates.
- Neonates with congenital anomalies.
- Neonates with suspected or proven inborn errors of metabolism, renal disease, or sepsis.
- Maternal history of drug intake affecting neonatal renal function or purine metabolism.

Sample Collection and Biochemical Analysis: Spot urine samples were collected within the first 24 hours of life. Urinary uric acid was quantified utilizing an enzymatic uricase method, whereas urinary creatinine was assessed through the Jaffe reaction. The urine uric acid to creatinine ratio was computed for each sample.

Statistical Analysis: Data were examined utilizing conventional statistical software. Continuous variables were represented as mean ± standard deviation (SD). Comparison between cases and controls was performed using the Student's t test. A p-value less than 0.05 was considered statistically significant.

Results

A total of 120 new-borns were assessed, with an equal distribution between cases and controls. The average urine uric acid–creatinine ratio was markedly elevated in asphyxiated infants relative to the control group.

- **Cases:** Mean UUA/Cr ratio = 2.83 ± 0.75
- **Controls:** Mean UUA/Cr ratio = 1.14 ± 0.38

The mean UUA/Cr ratio between the two groups exhibited a statistically significant difference ($p < 0.001$). Asphyxiated neonates exhibiting more severe clinical manifestations demonstrated comparatively elevated ratios, indicating a connection between the extent of hypoxia and the disruption of purine metabolism.

Table 1: Comparison of Urinary Uric Acid–Creatinine (UUA/Cr) Ratio Between Asphyxiated Neonates and Controls

Study Group	Number of Neonates (n)	Mean UUA/Cr Ratio	Standard Deviation (± SD)	p-value
Cases (Asphyxiated neonates)	60	2.83	0.75	< 0.001
Controls (Non-asphyxiated neonates)	60	1.14	0.38	

Asphyxiated neonates had a considerably higher mean UUA/Cr ratio than controls.

Table 2: Association Between Severity of Birth Asphyxia and Urinary Uric Acid–Creatinine Ratio

Severity of Asphyxia (Clinical Features)	Number of Neonates (n)	Mean UUA/Cr Ratio
Mild asphyxia	26	1.54± 0.23
Moderate asphyxia	19	1.98± 0.32
Severe asphyxia	15	2.58 ± 0.72

Discussion

Increased uric acid synthesis and faster ATP breakdown are two of the metabolic abnormalities brought on by perinatal hypoxia. The kidneys discharge excess uric acid, making urine an accessible medium for measuring hypoxic stress [7]. Urine concentration and hydration status changes can be mitigated by normalizing uric acid levels to creatinine.

The findings of this study reveal a significantly higher urine uric acid–creatinine ratio in neonates with perinatal hypoxia compared to healthy controls.

In this study, the mean UUA/Cr ratio was found to be 2.83 ± 0.75 in asphyxiated neonates compared to control (1.14 ± 0.38). The severity of asphyxia was defined as mild, moderate, and severe. The mild asphyxia was observed in 26 neonates with the mean UUA/Cr ratio of 1.54± 0.23, moderate asphyxia in 19 neonates with the mean UUA/Cr ratio of 1.98±

0.32, whereas the severe asphyxia in 15 neonates with the mean UUA/Cr ratio of 2.58 ± 0.72. These findings are in line with earlier research that found elevated UUA/Cr ratios in neonates after hypoxic–ischemic episodes.

Compared to conventional markers, the UUA/Cr ratio offers several advantages: it is non-invasive, cost effective, and straightforward to perform, especially in resource constrained circumstances. Additionally, urine collection spares vulnerable new-borns from repeated blood samples. The lack of long-term neurodevelopmental follow-up and correlation with severity grading, such as phases of hypoxic-ischemic encephalopathy, limits the current analysis, though, as it is a retrospective study [8].

Conclusion

A straightforward, non-invasive, and accurate biochemical indicator for detecting neonates with perinatal hypoxia is the urine uric acid–creatinine ratio. Its markedly higher levels in infants who have been asphyxiated demonstrate its potential value as an additional diagnostic tool, especially in situations when sophisticated tests are not easily accessible. Prospective studies with bigger sample sizes and association with clinical severity and outcomes are recommended to further validate its therapeutic applicability.

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