ORIGINAL RESEARCH PAPER

Volume-8 | Issue-3 | March-2019 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

SPOT URINE URIC ACID LEVEL COMPARISON AMONG ASPHYXIATED AND NON-ASPHYXIATED PRETERM NEWBORNS

Paediatrics

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ABSTRACT

Introduction- Perinatal asphyxia is a condition defined as hypoxemia, hypercapnea and acidosis in neonate. Cellular hypoxia leads to increased excretion of uric acid.

Aims & objectives- To compare urinary uric acid levels within 48 hours of birth among asphyxiated and non-asphyxiated preterm newborns.

Material and methods-

Study design- Prospective observational cohort study

Settings- Tertiary level hospital in central India

Duration- July 2017 to June 2018.

Participants- Due to financial constraints 100 neonates were enrolled.

Statistical analysis was performed by IBM SPSS version 22 and Microsoft Excel. Test of significance by Mann-Whitney U test.

Results- The mean rank of urine uric acid (13.13vs 11.40) was not statistically significant in preterm's as per asphyxia indicator (p=0.561).

Conclusions- urine uric acid levels were not consonant with birth asphyxia.

KEYWORDS

Urine uric acid, birth asphyxia, Early marker.

INTRODUCTION

Perinatal asphyxia is a condition where impaired gas exchange leads to hypoxemia, hypercapnea, and acidosis in fetus or neonate. The incidence of perinatal asphyxia is 2 to 10 per 1000 term newborns (1-5.6 % of all live birth) In India 0.5-1 million cases of birth asphyxia are seen per year and it comes out to be the main cause of mortality (28.8%), morbidity and chief cause of stillbirth (45.1%).[1] Birth asphyxia can involve any organ i.e. kidney (50%), heart (25%), or Brain (28%) and hence can lead to multisystem failure. As the severity of birth asphyxia increases, the chances of having kidney injury also increase.

Brief hypoxia damages cerebral oxidative metabolism leading to an anaerobic glycolysis, yielding only 2 molecules of Adenosine Triphosphate as compared to 32 molecules of ATP during aerobic conditions [2]. Lack of ATP and increased cellular destruction will cause an accumulation of Adenosine Monophosphate (AMP) and Adenosine Diphosphate (ADP), which will then get catabolised to its constituents of adenosine, inosine and hypoxanthine [3,4]. Continuous tissue hypoxia and consequent reperfusion injury will result in hypoxanthine being oxidized to xanthine and uric acid in presence of xanthine oxidase. Increased excretion of uric acid caused by metabolic changes, reflecting the cellular hypoxia has been reported by number of studies [5, 6]. Urine uric acid/ creatinine ratio have been found to be raised in asphyxia in many studies but no study was relating urine uric acid with kidney injury.

Hence in this study, we tried to assess the value of urine uric acid in asphyxiated and non-asphyxiated preterm newborns in first 2 days of life.

Material and methods-

Study design- Prospective observational cohort study

Settings- Neonatal Intensive Care Unit in tertiary level hospital in central India

Duration- July 2017 to June 2018.

Inclusion criteria-

Newborns of both sexes irrespective of gestational age or birth weight having :-

1. Persistence of apgar score less than 3 at 5 minutes.

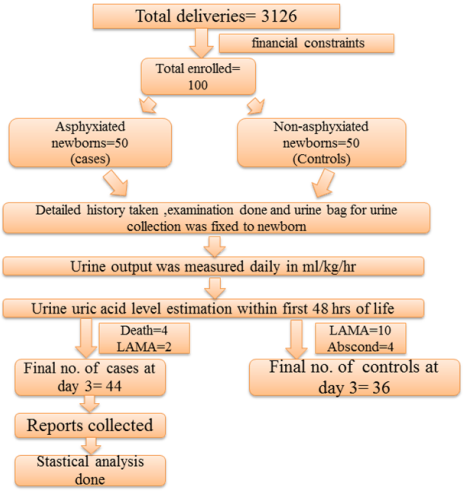
And/or

2. Newborns requiring resuscitation with positive pressure ventilation for >1 minute before achieving stable spontaneous respiration.

Exclusion criteria-

1. Newborns with any congenital urological anomaly.
2. Family h/o of genetic disorder (Disease running in families)
3. Newborn who could not be included due to researcher financial constraints.

Initially 250 newborns came as sample size but due to financial constraints only 100 patients were enrolled in the study. (Flow chart)



The urine sample was collected within first 48 hrs of life with all aseptic precautions and was assessed for uric acid by auto analyzer using spectrophometry uricase method.

Analysis was performed using the commercially available statistical Software-IBM SPSS version 22 and Microsoft Excel. The statistical analysis between variables was done using Mann whitney test. A p value of <0.05 was considered significant.

Results-

Out of 100 neonates enrolled in study 20 neonates couldn't complete study and hence 36 normal and 44 asphyxiated neonates completed the study. Out of 80 neonates, 55(68.75%) neonates were male. As per gestational age, 23(28.7%) neonates were pre-term, 2(2.5%) were

post-term and 55(68.8%) were term neonates (table-1).

Table 1- Showing distribution of sample as per gestational age and birth asphyxia

Asphyxia indicator	Total No. Of patients	Pre-Term	Term	Post-term
Yes	44	8	34	2
No	36	15	21	0

The mean rank of urine uric acid (13.13vs 11.40) was not statistically significant in preterm's as per asphyxia indicator (p=0.561) (table-2).

Table 2- Comparison of urine uric acid in preterm neonates as per asphyxia indicator

Asphyxia Indicator	No. of newborns	Urine Uric Acid (micromol/24 hrs) mean Rank		
Yes	8		13.13	
No	15		11.40	

- The p value in preterm=0.561, non-significant

DISCUSSION-

Perinatal asphyxia is a condition that can lead to alteration in normal functioning of various body organs but the most commonly affected organ is kidney. There are a no. of studies available that focused on urine UA/Cr ratio while considering asphyxiated and non-asphyxiated neonates.

In 2008, Pallab Basu [7] conducted a case control hospital based study over 12 months time on 31 asphyxiated and 31 normal newborn to see whether urinary uric acid and creatinine ratio can be used as a marker of perinatal asphyxia. . It was found that the ratios were significantly higher in cases than controls (3.1± 1.3 vs. 0.96± 0.54; P< 0.001) and among asphyxia patients.

In 2017, Kinjal Prahalad bhai Patel [8] conducted a case control study at a teaching hospital in Central Gujarat. 40 healthy newborns and 40 asphyxiated newborns were collected, the mean (UA/Cr ratio) (2.75±0.18 vs. 1.78±0.23) was significantly higher in asphyxiated group than in the control group (p<0.0001).

In our study we observed that the p value of 0.561 for comparing urine Uric acid level between preterm asphyxiated and preterm non-asphxiated neonates was more than the calculated p value at 95% confidence interval. Thus we concluded that urine uric acid was not raised in preterm asphyxiated as compared to preterm non-asphyxiated newborns .This study shows that urine uric acid levels were not consonant with birth asphyxia.

Conclusion-

In our study, urine uric acid was not statistically significantly in preterm asphyxiated and preterm non-asphyxiated newborns. Urine uric acid was not raised in preterm asphyxiated neonates.

Limitations-

We couldn't find significant correlation between urine uric acid in preterm asphyxiated newborns due to-

1. Sample size was small.
2. Time period boundation or shorter duration of study.
3. Due to cost factor, urine uric acid estimation of only few neonates was possible.

Recommendations-

Urine uric acid was not higher in preterm asphyxiated newborns. Thus it is recommended that a larger study with more cohorts needed to validate urinary uric acid in asphyxia newborns.

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