International Journal of Contemporary Pediatrics
Bhosgi R et al. Int J Contemp Pediatr. 2022 Jan;9(1):109-111
http://www.ijpediatrics.com
pISSN 2349-3283 | eISSN 2349-3291

DOI: https://dx.doi.org/10.18203/2349-3291.ijcp20214802

Original Research Article

Clinical profile of acute kidney injury in neonates with perinatal asphyxia

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Received: 15 November 2021
Revised: 28 November 2021
Accepted: 29 November 2021

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ABSTRACT

Background: Acute kidney injury (AKI) is one of the most common complication observed in perinatal asphyxia. Early recognition is required for appropriate treatment and improve the outcome.
Methods: It is a hospital based retrospective study conducted from august 2019 to December 2019. Total 85 full term neonates with perinatal asphyxia were included in the study. Renal functions were assessed by monitoring urine output, serum creatinine and ultrasonography. Acute kidney injury assessed by pRIFLE criteria and HIE staging is done by modified Sarnat and Sarnat staging. Severity of AKI is correlated with stages of HIE. AKI is managed as per unit protocol.
Results: Total 85 perinatal asphyxia neonates were included in the study. Out of total 85 neonates, 25 (29.4%) neonates had evidence of acute kidney injury. Among 25 neonates with acute kidney injury, higher percentage was observed in male neonates which was 14 (56%) against 11 (44%) among female neonates. Predominantly, non oligouric acute kidney injury was observed among acute kidney injury neonates which accounted to 20 neonates (80%) (p-0.258). Serum creatinine between 1.5-2 mg/dl was observed in 18 (21.1%) neonates and 7 (8.2%) neonates had creatinine between 2-3 mg/dl. Sonological abnormality was noted in 2 (2.3%) neonates. Among neonates with non oligouric AKI, 3 (12%) neonates had HIE stage 1, 15 (60%) had HIE-2 and 7 (28%) had HIE-3. However, neonates with non oligouric AKI were higher among HIE 2 when compared to neonates with oligouric renal failure who were higher in HIE 3. No mortality occurred among these neonates.
Conclusions: Majority of the neonates with perinatal asphyxia had non oliguric AKI which responded well to conservative treatment. AKI is most commonly seen in HIE stage 2 babies. Since non oligouric renal failure was a predominant finding among asphyxiated neonates, Serum creatinine monitoring remains main stay of diagnosis.

Keywords: Perinatal asphyxia, AKI, Oliguria, Neonates, Hypoxic ischemic encephalopathy

INTRODUCTION

Perinatal asphyxia is common cause of morbidity and mortality in neonates.[1] The incidence of asphyxia is estimated to be between 1 and 8 per 1000 live births. This wide range can be largely attributed to problems in selecting indicators to identify children with perinatal asphyxia. Perinatal Asphyxia ranks as the second most important cause of neonatal death. Asphyxia leads to diving reflex which causes redistribution of blood to vital organs such as heart and brain.[2,3] This compromises the blood supply to other organs such as gastrointestinal tract and kidneys. Kidneys are more susceptible to hypoxic insult. If asphyxia is prolonged, can lead to acute kidney injury such as renal tubular and cortical necrosis.[4,5] It is therefore not surprising that acute kidney injury (AKI) is common in asphyxiated neonates. Kidneys are sensitive to oxygen deprivation and renal insufficiency may occur within 24 hours of hypoxic ischemic episode which if

prolonged may even lead to irreversible cortical necrosis. In human beings, glomerulogenesis begins at 5 weeks gestational age and develops and peaks at the second trimester. In the fetus, glomerular filtration rate (GFR) has a correlation with both gestational age and body weight. Prenatal GFR, even corrected for body weight is lower in neonates than in adults.[6] Asphyxiated neonates can present both as oliguric or non-oliguric renal failure. The pRIFLE scale, for early detection and classification of the AKI severity is currently widely accepted, (p: pediatric, R: rick, I: injury, F: failure, L: loss, E: end stage). Current study is conducted to know the clinical profile of acute kidney injury in perinatal asphyxia and to correlate the severity of AKI with perinatal asphyxia stages.[6]

Aims and objectives

To determine the clinical profile of acute kidney injury (AKI) in perinatal asphyxia. To correlate the severity of AKI with HIE (hypoxic ischemic encephalopathy) staging.

METHODS

The study was hospital based retrospective study. The study was conducted at Gulbarga institute of medical sciences, Kalaburagi. The study was conducted from August 2019 to December 2019.

Inclusion criteria

Full term neonates admitted with perinatal asphyxia.

Exclusion criteria

Preterm neonates. Septicemia. Congenital anomalies of kidney. Consent not given by parents of neonate.

Procedure

Full term neonates with perinatal asphyxia admitted in neonatal intensive care unit (NICU)were included in the study. Perinatal asphyxia definition and criteria was followed as per American academy of pediatrics criteria. Asphyxia staging was done by using Sarnat and Sarnat staging for hypoxic ischemic encephalopathy.4 Renal functions were assessed by measuring urine output, serum creatinine, blood urea and renal sonography. AKI staging was done by pRIFLE criteria.[3,4] Neonates were managed as per institutional protocol. Ethical approval was obtained from institutional ethical committee. Consent for was taken from parents of neonates before the study.

Statistical analysis

Data was analysed by Statistical package for social sciences (SPSS) 17. Fisher exact test was applied for the study.

RESULTS

Total 85 perinatal asphyxia neonates were included in the study. Out of total 85 neonates, 25 (29.4%) neonates had evidence of acute kidney injury. Among 25 neonates with acute kidney injury, higher percentage was observed in male neonates which was 14 (56%) against 11 (44%) among female neonates (Table 1 demographic profile of neonates with AKI).

Table 1: Demographic profile of neonates with AKI.

Sex	N=25 (Total no of neonates with AKI)
Male neonates	14
Female neonates	11

Table 2: Oliguric and non-oliguric AKI.

Stage of asphyxia	Neonates with oliguric AKI (n=25)	Neonates with non oliguric AKI (n=25)
	N (%)	**N (%)**
Stage 1	0	3 (12)
Stage 2	2 (8)	13 (52)
Stage 3	3 (12)	4 (16)
Total	5 (20)	20 (80)

Table 3: Serum creatinine levels in AKI.

Serum creatinine (mg/dl)	Number of neonates (%)
1.5-2	18 (72)
2-3	7 (28)

Table 4: AKI parameters in relation to HIE staging and sonologic abnormalities.

Neonates with AKI	25 (29.4%)
Neonates with AKI according to HIE stages	HIE stage 1: 3 (12%) Stage 2: 15 (60%) Stage 3: 7 (28%)
Neonates with sonologic abnormalities	2 (8%)

Predominantly, non oligouric acute kidney injury was observed among acute kidney injury neonates which accounted to 20 neonates (80%) (p-0.258) (Table 2 oliguric and non-oliguric AKI).

Serum creatinine between 1.5-2 mg/dl was observed in 18 (21.1%) neonates and 7 (8.2%) neonates had creatinine between 2-3 mg/dl (Table 3. serum creatinine levels in AKI). Sonological abnormality was noted in 2 (2.3%) neonates. Among neonates with non oligouric AKI, 3 (12%) neonates had HIE stage 1, 15 (60%) had

HIE-2 and 7 (28%) had HIE-3 (Table 4 AKI parameters in relation to HIE staging and sonologic abnormalities).

No mortality occurred. However, neonates with non oligouric AKI were higher among HIE 2 when compared to neonates with oligouric renal failure who were higher in HIE 3.

DISCUSSION

Neonates are more susceptible to AKI due to low glomerular filtration rate and immaturity of tubule for electrolyte resorption. Asphyxia further enhances the susceptibility of AKI.[3,7] Amongst the recognized complications of asphyxia such as renal tubular necrosis, renal vein thrombosis, AKI is more common and can even result in permanent renal damage.[8,9]

Our study showed AKI in 29.4% neonates as compared to the 64% in Girish gopal et al. study and 54% in Medani et al study.10,11 This shows that AKI is common in perinatal asphyxia. More sample size is required to confirm this in our study. Study by Alaro et al showed 51.6% neonates with AKI had HIE stage-2 as compared to our study which had 60% neonates in HIE stage-2.[12] This show that AKI is more common in stage 2. Non oliguric AKI (80%) was more common than oliguric AKI (20%) in perinatal asphyxia in our study. Study by Agrawal et al13 also shows common occurrence of non-oliguric AKI (67.6%) than oliguric AKI (32.3%) in perinatal asphyxia.

This study shows evidence of acute kidney injury among perinatal asphyxiated babies. It also shows higher evidence of non oligouric renal failure among perinatal asphyxiated neonates. This inference along with other studies suggest that normal urine output must not be a missed diagnosis of kidney injury in perinatal asphyxia neonates. Hence, lab evaluation of serum creatinine is a must in perinatal asphyxia neonates. However non oligouric renal failure in perinatal asphyxia does not predict poor prognosis in comparison to oligouric renal failure with hyponatremia among HIE 2 perinatal asphyxia neonates.[14]

Limitations of our study has been our inability to check residual renal tubular dysfunction, bp monitoring, evidence of RTA.

CONCLUSION

Majority of the neonates with perinatal asphyxia had non oliguric AKI which responded well to conservative treatment. AKI is most commonly seen in HIE stage 2 babies. Since non oligouric renal failure was a predominant finding among asphyxiated neonates, Serum creatinine monitoring remains main stay of diagnosis.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Cowan F, Rutherford M, Goenedaal F, Murcuri E, Bydder GM. Origin and timing of brain lesions in term infants with neonatal encephalopathy. Lancet. 2003;361:736-42.
2. Grow J, Barks JD. Pathogenesis of hypoxic ischemic brain injury in the term infant- current concepts. Perinat. 2004;29:585-602.
3. Cloherty JP, Eichenwald EC, Stark AR. Manual of neonatal care. 7th ed. Philadelphia,PA: Lippincot Williams and Wilkins. 2011;711-8.
4. Akcan A, Zappitelli M, Loftiz LL, Washbum KK, Jellerson LZ. Modified RIFLE criteria including children with Acute kidney injury. J Nephrol. 2001;71:1028-33.
5. Sarnat, Sarnat. Neonatal encephalopathy following fetal distress. Arch neurol. 1976;33:696-705.
6. Vanpee M, Blennow M, Linne T, Herin P, Aperia A. Renal function in very low birth weight infants: normal maturity reached during early childhood. J Pediatr. 1992;121:784-8.
7. Durkan AM, Alexander RT. Acute injury post neonatal asphyxia. J Pediatr. 2011;158(2):e29-33.
8. Anne M Durkan, Todd Alexander R. Acute kidney injury post neonatal asphyxia. J Pediatr 2011;158(2):e29-e33.
9. Aggarwal A, Kumar P, Chowdary G, Majumdar S, Narang A. Evaluation of renal functions in asphyxiated neonates. J Trop Pediatr. 2005;51(5):295-9.
10. Girish G. Acute kidney injury (AKI) in perinatal asphyxia. Indian J Pharm Boil Res. 2014;2(2):60-5.
11. Medani SA, Kheir AEM, Mohamed MB. Acute kidney injury in asphyxiated neonates admitted to a tertiary neonatal unit in sudan. Sudan J Pediatr. 2014;14(2):29-34.
12. Alaro D, Bashir A, Musoke R, Wanaiana L. Prevalence and outcomes of acute kidney injury in term neonates with perinatal asphyxia. African health sciences. 2014;14(3),682-8.
13. Agrawal S, Chaudhuri PK, Chaudhary AK, Kumar D. Acute kidney injury in asphyxiated neonates and its correlation hypoxic ischemic encephalopathy staging. Indian J Child Health. 2016;3(3):254-7.
14. Gupta BD, Sharma P, Bagla J, Parakh M, Soni JP. Renal failure in asphyxiated neonates. Indian pediatrics. 2005;42(9):928-34.

Cite this article as: Bhosgi R, Harwalkar K. Clinical profile of acute kidney injury in neonates with perinatal asphyxia. Int J Contemp Pediatr 2022;9:109-11.