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Original Research Article.

A Prospective Study of AKI by Using Koralkar Criteria in Hospital Born Preterm Babies

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ABSTRACT

Background: Acute kidney injury (AKI) is a clinical condition often seen in the neonatal intensive care units. The incidence of AKI in neonates treated at the NICU ranges from 2.4 to 56 %. Many etiological factors predispose development of AKI in neonates. AKI has a significant impact on survival rates, especially in preterm infants and Neonates with AKI have very high mortality rates (4.5–78 %). Our understanding of AKI in LBW newborns is mostly limited to retrospective studies. So we planned a prospective study in preterm babies with AKI diagnosed by Koralkar criteria.

Objectives: To find proportion of preterm babies (<37 weeks of gestation) with acute kidney injury born in the hospitals and to find out demographics, co-morbidities, clinical presentation, risk factors, and outcome in preterm newborns with AKI.

Materials & Methods: A Prospective study on 215 preterm babies was conducted at Neonatal units attached to SMS Medical College, Jaipur during Feb 2015 to March 2017. The study variables were analyzed using Epi-Info7 software with application of Mean, Proportion, Chi-square, t- test, regression analysis and Kaplan-Meier Survival analysis.

Results and Conclusion: Out of 215 pre-term infants 36 (16.7%) had AKI with maximum patients in cat.1 (11.1%). Out of 36 preterm infants with AKI 13 died and no statistically significant association was found between AKI and mortality among preterm infants. Statistically significant association was found between birth weight, sepsis, HMD, MV, NEC and mortality among preterm infants. Logistic regression was performed to eliminate potential confounders. Our final model included variables with p<0.10. After regression analysis only birth weight was associated with mortality among preterm infants with p=0.028. On comparing survival among preterm with AKI and without AKI the Average Hazard Rate was more among preterm infants with AKI but on applying Log Rank Test no statistically significant difference was found between the survival probabilities of the two groups.

Key words: AKI, LBW, Koralkar Criteria, Kaplan Meier.

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INTRODUCTION

Up to 3 days of life, serum creatinine levels in newborns are a reflection of maternal serum creatinine. Thereafter, creatinine levels gradually drop and within the next 2–3 weeks are normalized in 93 % of neonates.[1-5] Acute kidney injury (AKI) is a clinical condition often seen in the neonatal intensive care units (NICU). The incidence of AKI in neonates treated at the NICU ranges from 2.4 to 56 %.[6-11] There is no universally adopted definition of AKI in the literature for neonates, different cut-off values (1.5 mg/dl; 1.13 mg/dl, 1.3 mg/dl, 2 mg/dl, etc) of serum creatinine (S Cr) levels on the second or third day of life are proposed to define AKI in neonates.[6,7,12-15] The Acute Kidney Injury Network (AKIN) and modified pediatric RIFLE (pRIFLE) classification recommend criteria for diagnosis of AKI based on the increase in S Cr levels and/or reduction of urine output.[3,16-18] Koralkar et al. proposed modification of the AKIN criteria for diagnosis and staging of AKI. These classifications and staging systems and their modifications allow more accurate diagnosis and staging of AKI. Many etiological factors predispose development of AKI in neonates. These factors include a low APGAR score, sepsis, hypothermia, nephrotoxic drugs, various therapeutic interventions (catheterization, intubation, mechanical ventilation, etc.), dehydration, hemodynamically significant patent ductus arteriosus (PDA) and severe intracranial hemorrhage.[3,4,7,15,19,20] AKI has a significant impact on survival rates, especially in preterm infants. Neonates with AKI have very high mortality rates (4.5–78%).[2,8,9,19,21] Our understanding of AKI in

LBW newborns is mostly limited to retrospective studies. The present prospective study is planned in preterm babies with the hypothesis that preterm with AKI will have decreased survival compared with those without AKI.

OBJECTIVES

1) To find proportion of preterm babies (<37 weeks of gestation) with acute kidney injury (AKI), born in the hospital
2) To find out demographics, co-morbidities, risk factors, and outcome in preterm newborns with AKI

MATERIALS AND METHODS

It was a prospective study carried out in neonatal units attached to SMS Medical College, Jaipur from 2015 to March 2017. Permission from ethical committee was taken prior to the study. Sample size was calculated using 26% of AKI among preterm babies and assuming a 6% absolute error and 95% confidence interval. The calculated sample size was 214 and we included 215 patients in the study. Preterm (<37 weeks of gestation) of either sex born in hospitals attached to SMS medical College Jaipur and who stayed in the hospitals for minimum of 7 days were included in the study. Preterm discharged to another facility within 72 hrs and preterm with congenital structural disease or other major anomalies or with genetic syndromes, as well as those died or left hospital within first 72 hrs of life and for whom S. Creatinine level was not determined were excluded from the study. Infant and maternal demographic data were collected after getting consent. Basic serum creatinine was determined on the 3 day of life after that the serum creatinine value was analyzed every other day, for the first 7 days. Serum creatinine levels were determined in hospital laboratory by autoanalizer (Randox imola). AKI was determined by using Koralkar criteria. Acute Kidney Injury was defined as an increase of serum creatinine levels ≥0.3 mg/dl compared to basal values. Based on Koralkar criteria, AKI was classified into three stages as follows-

0 No change or rise <0.3 mg/dl
1 Increase of S. Cr 0.3 mg/dl or increase of S. Cr 150–200 % from previous trough value
2 Increase of S. Cr 200–300 % from previous trough value
3 Increase of S. Cr of 300% from previous trough value or 2.5 mg/dl or receiving dialysis

Descriptive statistics were applied to differentiate preterm infants with or without AKI. Normally distributed continuous variables were compared using Student t test. Categorical variables were analyzed using Chi square test. P value <0.05 was considered statistically significant for the descriptive statistics. Logistic regression was performed to eliminate potential confounders. Our final model included variables with p<0.10. Epi-info 7 software was used for statistical analysis.

Table 1: Comparison of infant characteristics among those with and without AKI

Infant Characteristics	No AKI (n=179)	AKI (n=36)	Test value	P value
GA (wk)	32.0±1.94	31.1±2.16	t=2.491	0.013**
Birth Weight(Grams)	1.5±0.32	1.4±0.41	t=1.627	0.105
Male Sex	108 (60.3%)	23 (63.8%)	χ2=0.159	0.690
APGAR at 1 min	5.8±0.69	5.4±1.18	t=2.766	0.006**
APGAR at 5 min	7.0±0.45	6.8±0.82	t=2.070	0.040**
NEC	15 (8.3%)	3 (8.3%)	χ2=0.0001	0.992
CHD/PDA	13 (7.2%)	4 (11.1%)	χ2=0.609	0.434
HMD	25 (14.9%)	8 (22.2%)	χ2=1.572	0.209
ICH	1 (0.5%)	0 (0%)	χ2=0.2011	0.653
Sepsis	71 (39.6%)	12 (33.3%)	χ2=0.5069	0.476
MV	59 (32.9%)	15 (41.7%)	χ2=1.0064	0.315

** Statistically significant

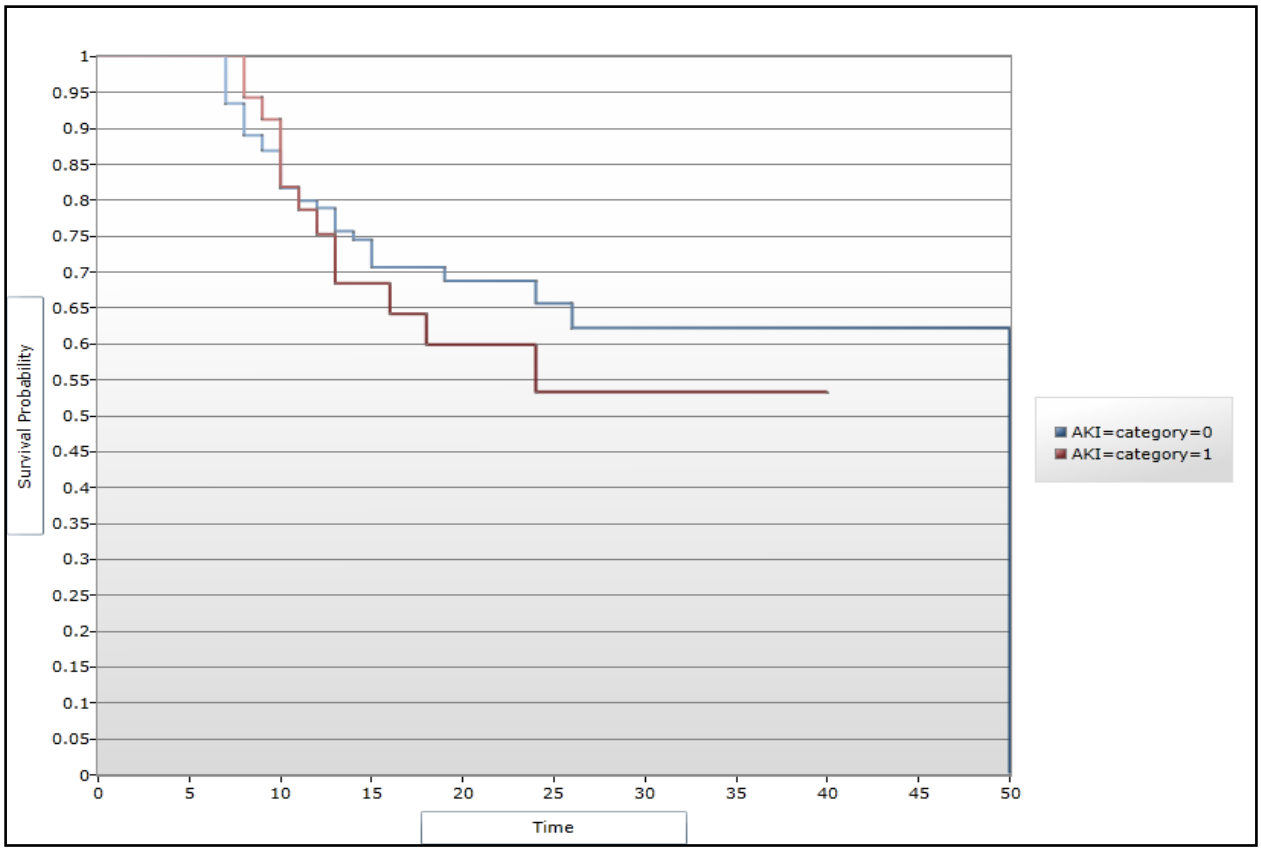
Table 2: Comparison of Maternal characteristics among those with and without AKI

Maternal characteristics	No AKI (n=179)	AKI (n=36)	Test value	P value
Age (yrs)	24.3±1.93	24.8±1.70	t=1.445	0.150
Height (cm)	159.6±11.34	161.2±3.38	t=0.838	0.403
Prenatal care	177 (98.8%)	35 (97.2%)	χ2=0.6006	0.438
HTN	3 (1.7%)	1 (2.8%)	χ2=0.1993	0.655
Steroid intake	3 (1.7%)	1 (2.8%)	χ2=0.1993	0.655
Drug intake	5 (2.8%)	1 (2.8%)	χ2=0.0000	0.995
Mode of delivery (LSCS)	45 (25.1%)	8 (22.2%)	χ2=0.1373	0.710

Table 3: Log rank test to compare survival probability of both the groups (with AKI and Without AKI)

Test	Statistics	DF	P value
Log Rank	0.3548	1	0.5514
Wilcoxon	0.0001	1	0.9908

Fig 1: Comparison of survival probability of both the groups (with AKI and Without AKI): Kaplan-Meier Survival Curve



(Here AKI-category=0 =no AKI and category=1 = AKI)

RESULTS

A total of 215 preterm infants were analyzed. AKI was found in 36 (16.7%) preterm infants out of 215. Maximum were in cat 1 (24, 11.1%), followed by cat 2 (7, 3.3%) and cat 3 (5, 2.5%).

Table 1 shows the comparison of infant characteristics among those with and without AKI. Statistically significant difference was found in gestational age, birth weight, APGAR at 1 and at 5 min between those with and without AKI. Preterm infants with AKI had lower mean GA and low mean birth weight and low mean APGAR at 1 and at 5 min as compared with preterm infants without AKI. Table 2 shows the comparison of maternal characteristics among those with and without AKI. No statistically significant difference was found in maternal characteristics of preterm infants with and without AKI. Of those preterm with AKI, 13 (36.1%) died and those without AKI, 43 (24%) died. No statistically significant association was found between AKI and mortality. Other variables like gestational age, birth weight, HMD, MV, NEC and sepsis were found significantly associated with mortality among preterm infants. After applying logistic regression, only birth weight was associated with mortality among preterm infants with p=0.028. On comparing survival among preterm with AKI and without AKI the Average Hazard Rate was more among preterm infants with AKI (Died/ Sum of the observed survival time=13 death /623days=0.0208 = 21 death/1000/day) as compared with those without AKI (43death /2445days=0.0175= 17 death/1000/day). Fig 1 shows that preterm with AKI have a lower probability of survival as compared with those without AKI. Log Rank test (Table 3) was used to compare the survival probability of both the groups. No statistically significant difference was found between the survivals of the two groups.

DISCUSSION

Proportion of AKI in our study was 16.7% which was within the range of AKI available in the literature. In our study male: female ratio was 2:1 among infants who developed AKI. This finding was similar with the study done by Fakhrossadat Mortazavi et al.[19] High frequency of AKI among male babies could be due to their more susceptibility to perinatal insults like sepsis and respiratory distress syndrome. We found maximum patients in cat 1 and this finding was similar with Askenazi et al[3] This might probably because in our study we determined SCr levels of all new born babies on the third day of life and then each or every other day during the first week and, in the cases where those values are elevated, until their normalization. This way, we were more likely to diagnose AKI in its early stages. Koralkar et al found maximum patients in cat 3 in their study; this finding was contrary to our study. Reason could be that Koralkar et al included very low birth weight babies in their study and VLBW might be more susceptible to severe acute kidney injury. Kolarkal et al[8] found that infants born by mothers who had pre-eclampsia and/or hypertension were protected from AKI whereas in present study maternal factors like age of mother, disease statuses of mother like hypertension and diabetes, antenatal steroid intake were not associated with AKI among preterm infants. This could be due to the fact that we had very few numbers of mothers who had hypertension (only 4) and diabetes (no mother had diabetes). Sepsis was observed by Mortazavi F et al[19] in 28.5% of their patients and in 22.2% of the patients studied by Agras and associates[9]. In our study we found sepsis among 38.6% of neonates. A variety of mechanisms, including shock, disseminated intravascular coagulation, hemorrhage, and cardiac failure may cause AKI in septic

neonates. In a study by Mathur and coworkers[22] in India, 26% of septic neonates developed AKI. In our study we found 14.5% of septic neonates developed AKI. Mortality of AKI among septic neonates is high, and reportedly 50% to 78% of the cases end up with death.[23] Like other studies, mortality of septic neonates was significantly higher than non-septic neonates in our study. In present study mortality was 36.1% among those with AKI and 24.0% among those without AKI. Similar mortality (39%) was observed by Prayong Vachvanichsanong.[10] Data on survival of preterm infants are extremely variable. In our study, the observed percentages are comparable to the results of other studies in Western countries.[24,25] We used Average Hazard Rate to determine the survival probability. Average Hazard Rate is a measure of non-survival potential rather than survival. A group with a higher Average Hazard rate will have a lower probability of survival as compared with those with a lower Average Rate. Neonates with AKI had higher Average Hazard Rate means low survival probability than those with no AKI but the difference between the two groups were not statistically significant. That means AKI is not normally a direct cause of death.[26] The cause of death for patients diagnosed with AKI may not be the same as the cause of AKI. The mortality rate depends on other associated conditions, e.g. other organ failure, particularly cardiac failure, birth asphyxia and serious infection.

CONCLUSION

AKI is not independently associated with mortality among preterm babies**.** Other variables like birth weight, sepsis, HMD, MV, NEC are associated with mortality among preterm babies. Preterm with AKI have a lower probability of survival as compared with those without AKI. But the difference was not statistically significant between the survivals of the two groups.

REFERENCES

1. Gouyon JB, Guignard JP (2000) Management of acute renal failure in newborns. Pediatr Nephrol 14:1037–44.
2. Andreoli SP (2004) Acute renal failure in the newborn. Semin Perinatol 28:112–23.
3. Askenazi DJ, Ambalavanan N, Goldstein LS (2009) Acute kidney injury in critically ill newborns: what do we know? What do we need to learn? Pediatr Nephrol 24:265–74.
4. Jetton JG, Askenazi JD (2012) Update on acute kidney injury in the neonate. Curr Opin Pediatr 24:191–6.
5. Cuzzolin L, Fanos V, Pinna B, di MaryioM, PerinM, Tramontozzi P, Tonetto P, Cataldi L (2006) Postnatal renal function in preterm newborns: a role of diseases, drugs and therapeutic interventions. Pediatr Nephrol 21:931–8.
6. Lunn AJF, Shaheen I, Watson AR (2006) Acute renal insufficiency in the neonatal intensive care unit. Arch Dis Child Fetal Neonatal Ed 91:F388.
7. Aggarwal A, Kumar P, Chowdhary G (2005) Evaluation of renal functions in asphyxiated newborns. J Trop Pediatr 51:295–9.
8. Koralkar R, Ambalavanan N, Levitan EB, McGwin G, Goldstein S, Askenazi D (2011) Acute kidney injury reduces survival in very low birth weight infants. Pediatr Res 69:354–8.
9. Stapleton FB, Jones DP, Green RS (1987) Acute renal failure in neonates: incidence, etiology and outcome. Pediatr Nephrol1:314–20.
10. Hentchel R, Lodige B, Bulla M (1996) Renal insufficiency in the neonatal period. Clin Nephrol 46:54–8.
11. Gupta BD, Sharma P, Bagla J (2005) Renal failure in asphyxiated neonates. Indian Pediatr 42:928–34.
12. Cataldi L, Leone R, Moretti U, De Mitri B, Fanos V, Ruggeri L,Sabatino G, Torcasio F, Zanardo V, Attardo G, Riccobene F,Martano C, Benini D, Cuzzolin L (2005) risk factors for the development of acute renal failure in preterm newborn infants: a case–control study. Arch Dis Child Fetal Neonatal Ed 90:F514– F519.
13. Kungwani AR, Nimbalkar AS et al. (2012) 1,324 clinical profile and outcome of newborns with acute kidney injury in a level 3 neonatal unit in western India. Arch Dis Child 97:A377.
14. Agras PI, Tarcan A, Baskin E (2004) Acute renal failure in the neonatal period. Ren Fail 26:305–9.
15. Vachvanichsanong P, McNeil E, Dissaneevate S, Dissaneewate P, Chanvitan P, Janjindamal W (2012) Neonatal acute kidney injury in a tertiary center in a developing country. Nephrol Dial Transplant 27:973–7.
16. Mehta RL, Kellum JA, Shah SV, Molitoris BA, Ronco C, Warnock DG (2007) Acute Kidney Injury Network (AKIN): report of an initiative to improve outcomes in acute kidney injury. Crit Care 11:R31
17. Blinder JJ, Goldstein SL, Lee W, Baycroft A, Fraser CD, Nelson D, Jefferies JL (2012) Congenital heart surgery in infants: effects of acute kidney injury on outcomes.JThoracCardiovasc Surg143:368-74.
18. Akcan-Arikan A, Zappitelli M, Loftis LL, Washburn KK, Jefferson LS, Goldstein SL (2007) Modified RIFLE criteria in critically ill children with acute kidney injury. Kidney Int 71:1028–35.
19. Mortazavi F, Sakha SH, Nejati N (2009) Acute kidney failure in neonatal period. Iran J Clin Infect Dis 3:136–40.
20. Karlowicz MG, Adelman RD (1992) Acute renal failure in the neonate. Clin Perinatol 19:139–58.
21. Chevalier RL, Campbell F, Brenbridge AG (1984) prognostic factors in neonatal acute renal failure. Pediatrics 74:265–72.
22. Mathur NB, Agarwal HS, Maria A. Acute renal failure in neonatal sepsis. Indian J Pediatr. 2006;73:499-502.
23. Stoll BJ, Hansen N, Fanaroff AA, et al. Late-onset sepsis in VLBW neonates: the experience of the NICHD Neonatal Research Network. Pediatrics 2002;110:285–91.
24. Vesna Stojanović & Nenad Barišić & Borko Milanović & Aleksandra Doronjski: Acute kidney injury in preterm infants admitted to a neonatal intensive care unit: Pediatr Nephrol (2014) 29:2213–20.
25. Low birth weight premature infants: a retrospective case control study. Pediatrics 2003;111:e590.
26. Hoste EA, Kellum JA (2006) Acute kidney injury: epidemiology and diagnostic criteria. Curr Opin Crit Care 12:531–7.

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