

Original Research Article

Comparative study of clinico-biochemical profile and outcome of acute kidney injury in outborn and inborn neonates

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ABSTRACT

Background: Acute Kidney Injury (AKI) is one of the major clinical problem in hospitalised neonates having variable outcomes. Prognosis depends on early diagnosis, associated risk factors and type of renal failure. The present study was undertaken to evaluate and compare risk factors, biochemical derangements and outcome of AKI in outborn and inborn neonates.

Methods: For this hospital based prospective study 100 neonates were enrolled who were admitted in the NICU, diagnosed as AKI who had serum creatinine >1.5mg/dl. Study was done for 1 year from June 2016 onwards.

Results: A large majority (72.3%) cases were outborn neonates (extramural) whereas (27.7%) cases were inborn neonates (intramural). Most of (79.8%) cases were term and were admitted during summer months. In outborn, type of AKI in descending order was prerenal (64.7%), renal (33.8%) and postrenal (1.5%) while in inborn neonates, cases were equally (50%) divided in between renal and prerenal. Among outborn neonates risk factors for AKI was dehydration (44%), sepsis (28%) and shock (16%) whereas in inborn, perinatal asphyxia (31%), dehydration (27%), shock (23%) and sepsis (11.5%) were risk factors. In outborn 36.8% cases were oliguric whereas in inborn 53.9% cases were oliguric.

Conclusions: The maximum cases of AKI were outborn neonates in which outborn dehydration was the commonest cause while in inborn neonates perinatal asphyxia was the commonest cause. Sepsis and shock were other causes in both groups. Presence of oliguria, intrinsic AKI and shock carried poor prognosis.

Keywords: Acute Kidney Injury (AKI), Outborn neonates, Inborn neonates

INTRODUCTION

Acute Kidney Injury (AKI) is one of the major clinical problems in hospitalised neonates. There is wide variation in the incidence of AKI across studies.

It affects 8-24% of critically ill neonates and mortality rates vary between 10 to 61%.¹ The common conditions contributing to kidney injury in neonates according to various studies are perinatal asphyxia, neonatal sepsis, respiratory distress syndrome, dehydration, heart failure, nephrotoxic drugs, medication-phototherapy and

urological anomalies with asphyxia and sepsis being the most common.

Causes of neonatal AKI are multiple and can be divided in²

- Pre renal AKI
- Renal (Intrinsic) AKI
- Post renal AKI

Acute renal failure (ARF) is characterized by sudden (within 48 hours) impairment in kidney function that

results in the retention of nitrogenous waste products, e.g. urea and alters the regulation of ECF volume, electrolytes and acid base homeostasis.³

The old terminology of Acute Renal Failure (ARF) has been replaced now by the term Acute kidney injury (AKI). The initial definitions were based on Blood Urea Nitrogen (BUN) but BUN does not reflect the real status of kidney injury as it is affected by many other factors such as dehydration, steroid usage, increased catabolism, etc.

The Second International Consensus Conference of Acute Dialysis Quality Initiative (ADQI) group in 2004 formulated a multilevel classification system which was known as the RIFLE criteria for ARF.⁴ It was based on creatinine clearance, serum creatinine and oliguria.

The serum creatinine level is the simplest and most commonly used indicator of neonatal kidney function. The plasma creatinine (Pcr) concentration immediately after birth reflects the maternal creatinine concentration. The Pcr level gradually decreases from 1.1mg/dl in term infants (1.3 mg/dl in preterm infants) to a mean value of 0.4 mg/dl within first two weeks of life. In general, each doubling of the serum creatinine level represents a 50% reduction in GFR. For the sake of uniformity for different gestational age of neonates we have taken ≥ 1.5 mg/dl creatinine value as AKI.⁵

Passage of urine in term babies is quite variable but normally occurs within the first 48 hours. Oliguria in neonates is generally defined as urine flow less than 1ml/kg/hr. However, up to 1/3rd of the cases of AKI in neonates present with normal urine output especially in asphyxiated neonates.⁶

Prognosis and outcome of patients of renal failure are quite variable and depend on early diagnosis of the condition, the underlying pathology and type of renal failure whether oliguric or non oliguric.

These are several studies which demonstrate that 40-50% of neonates who recovered from acute renal failure are being left with residual renal damage and manifest by structural, glomerular or tubular abnormalities or hypertension later in life.

METHODS

A prospective study was conducted on 100 neonates diagnosed as AKI, admitted in the NICU of JLN Medical College and Hospital and associated Rajkiya Mahila Chikitsalaya, Ajmer, for 1 year from June 2016 onwards. The Institutional ethical committee approved the study.

Those neonates who had serum creatinine ≥ 1.5 mg/dl were included in the study. Six cases were excluded from the study because 3 neonates died within 24 hours of admission and another 3 leave against medical advice

(LAMA). So remaining 94 neonates were analysed and followed for the study purpose to evaluate and compare risk factors, biochemical derangements and outcome of AKI in outborn and inborn neonates.

- (Outborn Neonates – Neonates who were born on outside of JLN Hospital, Ajmer)
- (Inborn Neonates - Neonates who were born in Rajkiya Mahila Chikitsalaya, JLN Hospital Ajmer)

Signed informed consent was taken from parents. A detailed history and clinical examination was done and relevant clinical and laboratory findings, demographic features, gestational age (term or preterm assessed on the basis of physical characteristics), birth weight/weight on admission, antenatal, perinatal history was recorded on predesigned proformas for this study along with any associated contributing conditions including perinatal asphyxia (newborn presenting with history of delayed cry or having sign and symptoms of hypoxic ischaemic encephalopathy, sepsis, respiratory distress syndrome, dehydration, heart failure, meconium aspiration syndrome and history of surgical operation.

In all neonates with AKI relevant investigations like blood urea, serum creatinine, serum electrolytes (sodium and potassium), urine output, urine examination, urinary sodium and creatinine were performed on every day until renal parameters became normal or the patient became unavailable for follow up.

FeNa⁺ value ≥ 2.5 or < 2.5 in newborns with renal failure, was considered to have intrinsic renal or prerenal AKI respectively.

Oliguria was defined as urine output < 1.0 ml/kg/hour and anuria are defined as absence of any urine output within 24 hours after birth.⁶ Twenty-four hours urine output was measured by collecting urine in baby urobags.

An ultrasound imaging of kidney was carried out for size, echotexture and cortico - medullary differentiation, any congenital malformation and/or abnormality of urinary tract.

Statistical analysis

Appropriate statistical tools were used to test significance and to compare data. Categorical variables were presented as number and percentage. Following statistical methods were used wherever applicable

- Independent 't' test
- Chi-Square test

p value of < 0.01 was considered statistically significant and p value of < 0.001 was considered highly significant. Microsoft word and excel have been used to generate tables.

RESULTS

In present study Out of total 94 cases, a large majority 68 (72.3%) cases were outborn neonates (extramural) whereas 26 (27.7%) cases were inborn neonates (intramural). Out of 68 outborn cases 52 neonates were male and 16 were female. Out of 26 inborn cases 17 neonates were male and 9 were female. In this study males outnumbered females and there is no statistically significant difference between outborn and inborn neonates according to sex.

Table 1: Distribution of cases in relation to the type of delivery.

	Outborn neonates (n = 68)	Inborn neonates (n = 26)	Total
Normal delivery	65	9	74 (78.7%)
Caesarean section (CS)	3	17	20 (21.3%)
	68	26	94 (100%)

$\chi^2 = 41.7466$; p value = 0.00001

Majority (78.7%) of cases had normal vaginal delivery and remaining (21.3%) were caesarean born (CS). But in inborn neonates of AKI about 2/3rd cases were delivered by CS. Statistically this difference was highly significant. (p = 0.00001) (Table 1).

Authors found that there was preponderance of AKI in term neonates (79.8%) as compared to preterm neonates (20.2%). (p = 0.88345). Most of neonates (55.9% outborn and 69.2% inborn) with AKI were admitted during summer months (i.e. April, May, June).

Table 2: Distribution of cases according to type of Acute Kidney Injury (AKI).

Type of AKI	Outborn neonates	Inborn neonates
Renal (Intrinsic)	23 (33.8%)	13 (50%)
Pre-renal	44 (64.7%)	13 (50%)
Post-renal	1 (1.5%)	0 (0%)
Total	68 (100%)	26 (100%)

Renal (Intrinsic) v/s Prerenal; $\chi^2 = 1.939$; P value = 0.163775

In outborn neonates, maximum cases (64.7%) were found in prerenal type of AKI followed by renal (intrinsic) type (33.8%) and postrenal type (1.5%), whereas in inborn neonates, cases were equally divided in between renal (intrinsic) and prerenal type of AKI. And no case was found in postrenal type. (p = 0.163775) (Table 2).

In current study, among outborn neonates the most common risk factor associated with developing AKI was dehydration (44%), followed by sepsis (28%) and shock (16%), whereas in inborn neonates most common risk factor associated with AKI was perinatal asphyxia (31%)

followed by dehydration (27%), shock (23%) and sepsis (11.5%).

In outborn 36.8% cases were oliguric (<1ml /kg /hr) whereas in inborn 53.9% cases were oliguric. There was no statistically significant difference in urine output of outborn and inborn neonates diagnosed with AKI (p >0.01).

Authors found that there was no statistically significant difference in urine output and serum biochemical analysis (e.g. serum creatinine, FeNa+, serum calcium, sodium and potassium) in between outborn and inborn neonates with prerenal AKI as well as neonates with renal (intrinsic) AKI.

Authors found that in prerenal AKI nobody had abnormal renal USG in both outborn and inborn neonates while in renal (intrinsic) AKI, sonographic abnormalities (i.e. renal parenchymal disease) were seen in 43.5% outborn and 46.2 % inborn neonates.

Table 3: Outcome of Acute Kidney Injury (AKI) in outborn and inborn neonates.

	Outborn neonates	Inborn neonates	Total
Expired	19 (28%)	9 (35%)	28
Discharged	49 (72%)	17(65%)	66
	68 (100%)	26(100%)	94

$\chi^2 = 0.4006$; p value = 0.526781

Out of 68 outborn neonates who had AKI, nineteen (28%) expired and forty-nine (72%) were successfully discharged with complete recovery of renal functions, while out of twenty-six inborn neonates who had AKI, nine (35%) expired and seventeen (65%) were successfully discharged, statistically no significant difference in outcome of outborn and inborn neonates (p = 0.5267) (Table 3).

Table 4: Outcome of outborn neonates according to type of AKI.

	Expired	Discharged	Total
Prerenal AKI	2 (4.5%)	42 (95.5%)	44 (100%)
Renal (Intrinsic) AKI	17 (73.9%)	6 (26.1%)	23 (100%)
	19	48	67

$\chi^2 = 35.7745$; p <0.00001

Authors found that the mortality was quite high in outborn cases of AKI due to renal (intrinsic) cause (73.9%) as compared to prerenal cause (4.5%), difference was statistically highly significant (p value <0.00001). In inborn neonates, cases expired of renal (intrinsic) cause (61.5 %) were statistically significantly higher than prerenal (7.7 %) cause in inborn. This difference was statistically significant (p value = 0.003906) (Table 4 and 5).

Table 5: Outcome of inborn neonates according to type of AKI.

	Expired	Discharged	Total
Prerenal AKI	1 (7.7%)	12 (92.3%)	13 (100%)
Renal (Intrinsic) AKI	8 (61.5%)	5 (38.5%)	13 (100%)
	9	17	26

$\chi^2 = 8.3268$; $p = 0.003906$

Table 6: Outcome of outborn neonates with AKI according to risk factors.

Risk factors of AKI	Expired		Discharged		Total
	Cases	%	Cases	%	
Shock	6	55	5	45	11(100%)
Dehydration	5	17	25	83	30 (100%)
Sepsis	4	21	15	79	19 (100%)
RDS	1	50	1	50	2 (100%)
Perinatal asphyxia	3	60	2	40	5 (100%)
Obstructive uropathy	0	0	1	100	1 (100%)

The causative mortality in AKI in outborn neonates was perinatal asphyxia (60%) followed by shock (55%), RDS (50%), sepsis (21%) and dehydration (17%) (Table 6), whereas in inborn neonates was perinatal asphyxia (63%) followed by shock (33%), sepsis (33%) and dehydration (14%) (Figure 1).

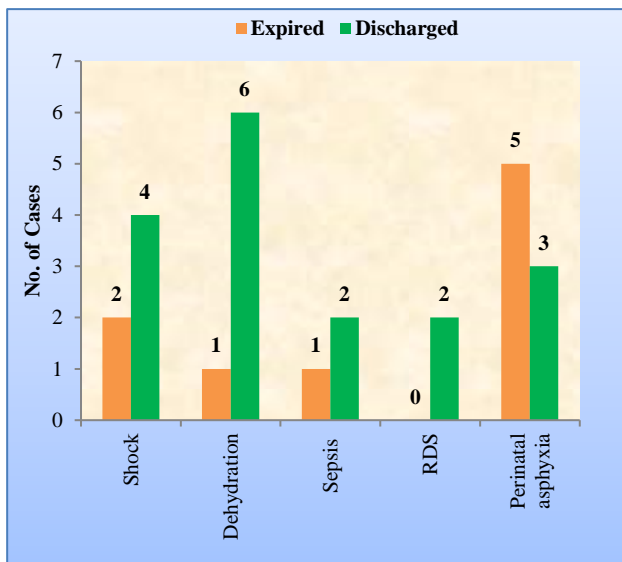


Figure 1: Outcome of Inborn neonates (n = 26) with AKI according to risk factors.

Authors found in the study that in outborn, majority (72%) of oliguric neonates expired and only 2.3% non-oliguric had fatal outcome. This was statistically highly significant ($p \leq 0.00001$), while in inborn neonates, majority (64.3%) of oliguric neonates expired and in non-oliguric none had fatal outcome.

DISCUSSION

Increased incidence of AKI amongst newborns in intensive care units has been attributed to prolonged survival of seriously ill neonates with improved resuscitative and ventilatory support, increased use of nephrotoxic drugs, increased incidence of sepsis and higher risk of renal failure in premature neonates due to physiological immaturity of renal function. The early recognition of renal dysfunction is important in critically sick neonates because it facilitates appropriate fluid and electrolyte management and modification in drug dosage if required. Thus, this study was conducted in extramural and intramural NICU, Department of Pediatrics, JLN Medical College and Hospital, Ajmer with the objectives to know determine and compare the etiology and type of renal failure in newborns and its associated risk factors for developing AKI, and to find out prognostic factors which affect ultimate outcome of patient with AKI in outborn and inborn neonates.

In present study total 100 neonates who were diagnosed as AKI admitted in extramural and intramural NICU in JLN Medical College and Hospital, Ajmer. Six cases were excluded from the study (three LAMA and three neonates died within 24 hours of admission). We found that most of cases (68) were outborn neonates whereas 26 cases were inborn neonates. Similarly, Airede et al studied 43 neonates with AKI and revealed majority (27) of neonates were outborn.⁷ The higher proportion of AKI amongst neonates in outborn NICU might be due to the fact that these newborn were brought in advanced stage of disease such as dehydration, sepsis and had either compensated or decompensated shock. Low proportion of AKI amongst newborns of inborn NICU might be explained by the fact that these neonates were delivered within our hospital, picked up at an early stage of disease and managed appropriately that must have taken care of hypoperfusion of various body organs particularly that of kidneys.

In present study 69 male and 25 female neonates were diagnosed as AKI in a simple randomized manner, though there was no statistically significant difference between outborn and inborn neonates on the basis of gender. Similarly Airede et al studied 43 neonates with AKI and observed male to female ratio was (M:F; 3.3:1).⁷ Higher number of male neonates in the study might be explained by the fact that there is always a significant social and cultural bias against the female child in community in general.

This study revealed that 74 (78.7%) neonates out of 94 cases with AKI were delivered by normal vaginal delivery in comparison to 20 (21.3%) neonates with AKI were delivered by caesarean section. Statistically occurrence of AKI was highly significant in inborn caesarean delivered neonates ($p = 0.00001$). This can be attributed to multiple factors like higher antenatal risk factors such as fetal distress, prolonged hospitalization

and more occurrence of dehydration due to ineffective lactation. Yaseen H et al (2004) had reported that dehydration fever was associated with caesarean section.⁸

In present study, proportion of AKI was 20.2% amongst preterm neonates which was lower than that of term neonates (79.8%). Sumitra et al (2002) and Gharehbaghi et al (2007), Gupta SK et al (2010) also observed higher incidence of AKI in term neonates in their study.^{9,10,11} All the preterm neonates were also given intravenous fluid soon after admission in NICU. This might have resulted in lower proportion of AKI in preterm neonates in our study. Term neonates in our study had higher proportion of AKI (79.8%) because of complication like birth asphyxia and septicemia.

Authors found that most of neonates (55.9% in the outborn and 69.2% inborn) were admitted during summer months (i.e. April, May, June). In these months environmental temperature was above 40°C (41-47°C), and due to high environmental temperature in these months, most of cases of AKI were admitted due to dehydration. Similarly, Devina et al observed that majority of cases (69.2%) of dehydration fever were admitted in June and July months which were warm months of the year.¹²

In present study, in outborn neonates majority of cases (64.7%) were prerenal followed by renal (intrinsic) (33.8%) and postrenal (1.5%) and in inborn neonates prerenal and renal (intrinsic) AKI cases were equal (50%) (p value >0.01). In outborn neonates, most of cases were admitted due to dehydration and sepsis which were predisposing risk factors for prerenal AKI, while in inborn neonates most of cases were admitted due to perinatal asphyxia and dehydration.

In present study, in outborn neonates causative risk factors associated with AKI were dehydration (44%), sepsis (28%), shock (16%), perinatal asphyxia (7.5%), RDS (3%) and obstructive uropathy (1.5%), while in inborn neonates most common risk factor associated with AKI were perinatal asphyxia (31%) followed by dehydration (27%), shock (23%), sepsis (11.5%) and RDS (7.5%). Similarly, Kapil et al observed that in outborn neonates neonatal sepsis was the commonest cause of AKI followed by perinatal asphyxia, respiratory distress syndrome and genitourinary anomalies.¹³

In present study, among outborn neonates majority of cases (63.2%) had normal urine output while in inborn only 46.2% cases had normal urine output. (p value >0.01). This difference was due to the fact that more cases of AKI in inborn neonates were having intrinsic renal failure. Kapil et al observed oliguria in 66% of outborn neonates.¹³ Jaishree et al found oliguria in 16.6% of acute renal failure cases.¹⁴

In present study, on the basis of FeNa⁺ AKI was divided into prerenal and renal (intrinsic) AKI. Fiftyseven

neonates (44 outborn and 13 Inborn) had FeNa⁺ <2.5 and were considered as prerenal failure while 36 neonates (23 outborn and 13 Inborn) had FeNa⁺ ≥2.5 and were considered as renal (intrinsic) AKI. Mathew et al (1980) in a prospective study of 42 neonates with oliguria found FeNa⁺ was the most useful index to differentiate prerenal from intrinsic renal failure and in prerenal disease FeNa⁺ was constantly <2.5 in contrast to intrinsic renal failure where value were >2.5.¹⁵ Present study revealed that prerenal failure was responsible for 60.6% cases of AKI while 38.3% cases were because of renal and 1.1% of AKI was post renal. Norman and Asadi also found that 72% cases of AKI were prerenal failure while 28% had IRF. These studies revealed that prerenal failure accounts for approximately 2/3rd cases of acute renal failure in the neonates.¹⁶

In inborn neonates there were equal proportion of prerenal and renal (50% each) while in outborn 64.7% cases of AKI were because of prerenal and 33.8% cases of AKI were because of renal and 1.5% due to post renal.

In this study mean urine output in prerenal failure in outborn was 1.73±0.55 ml/kg/hr and in inborn was 1.39±0.78 ml/kg/hr and in renal (intrinsic) AKI in outborn was 0.49±0.32 ml/kg/hr and in inborn was 0.74±0.41 ml/kg/hr (p >0.01). This study revealed that in outborn most of cases of AKI were due to dehydration and after fluid therapy urine output normalised.

In present study mean serum creatinine in prerenal AKI in outborn was 2.18±0.60 mg/dl and inborn was 2.13±1.11 mg/dl and in renal AKI in outborn neonates was 3.11±1.73 mg/dl and in inborn was 2.29±1.00 mg/dl. (p >0.01). Likewise, the mean blood urea in prerenal AKI in outborn was 197.55±88.88 mg/dl and inborn was 126.25±71.97 mg/dl (p value = 0.0107). While in intrinsic renal AKI mean blood urea in outborn was 197.32±153.73 mg/dl and in inborn was 120.4±125.91 mg/dl (p value >0.01).

In this study the mean serum sodium level in prerenal AKI in outborn neonate was 156.91±12.10 mEq/L and in inborn neonate was 151.85±9.9 mEq/L and in intrinsic renal AKI in outborn neonate was 155.35±13.99 mEq/L and in inborn neonate was 145.46±11.35 mEq/L. Likewise the mean potassium level in prerenal AKI in outborn neonate was 5.27±0.86 mEq/L and inborn neonate was 5.85±0.39 mEq/L and in intrinsic renal AKI in outborn neonates was 5.52±0.74 mEq/L and in inborn neonate was 5.65±0.43 mEq/L. Hyperkalemia and dilutional hyponatremia are usual electrolyte changes in AKI. Though in this study, we observed slightly higher level of serum sodium in some neonates which might be due to the fact that these neonates were brought in relatively dehydrated condition.

In present study outborn neonates with AKI 28 % cases expired while inborn neonates with AKI had 35% case fatality. Contrary of this, Norman and Asadi et al

reported that 45 % newborns of acute renal failure expired.¹⁶ In outborn neonates mortality due to renal (intrinsic) AKI (73.9%) was quite higher than pre renal AKI (4.5%), this was statistically highly significant (p value <0.00001).

Similarly, in inborn neonates mortality due to renal (intrinsic) AKI (61.5%) were higher than prerenal cause (7.7%), this was also statistically significant (p value-0.004). Outcomes depend on oliguria, presence of shock and ischemic damage to vital organ (brain, heart, kidney) due to perinatal asphyxia.

Authors found that in outborn, majority (72%) of oliguric neonates were expired and in non – oliguric only one case (2.3%) expired. This outcome was statistically highly significant (p ≤0.00001), while in inborn neonates majority (64.3%) of oliguric neonates expired and in non – oliguric none had fatal outcome. Similarly, Norman and Asadi et al and Jaishree and Saili et al observed that oliguric renal failure carried a poorer prognosis than non oliguric renal failure.^{16,17}

In current study authors observed that in outborn neonates mortality higher with perinatal asphyxia (60%) followed by shock (55%), respiratory distress syndrome (50%), sepsis (21%) and dehydration (17%), while in inborn neonates mortality higher with perinatal asphyxia (63%) followed by shock and sepsis (each 33%) and dehydration (14%).

Similarly, Chaitanya S et al observed that outcome of AKI was associated with perinatal asphyxia and presence of shock.¹⁸ Afros S et al also observed in inborn neonates that 27% of neonates diagnosed as AKI died and 57% improved with normal renal function.¹⁹

CONCLUSION

In conclusion, findings of present study suggest that AKI contribute significantly to alarmingly high neonatal morbidity and mortality. But the heartening fact is that most of these cases are prerenal in nature which carries better prognosis. Thus, a high index of suspicion for presence of AKI in sick neonates, whether oliguric or non-oliguric; thorough workup of these cases to find out type and cause of AKI; prompt and appropriate management of these cases will go a long way in reducing high neonatal mortality rate prevalent in our country.

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