

## Original Research Article

# Clinical profile of neonates with acute renal injury in neonatal intensive care unit at GMERS Medical College and General Hospital, Gotri, Vadodara, Gujarat, India

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### ABSTRACT

**Background:** Acute kidney injury (AKI) is defined as an acute deterioration in ability of the kidneys to maintain homeostasis of body fluids and electrolytes leading to retention of wasted and toxic metabolic end products. It is fairly common in newborn population and is a major contributor of neonatal mortality and morbidity. The aim was to study the incidence of renal failure in high risk neonates and risk factors for renal failure.

**Methods:** A prospective observational study was done to evaluate renal profile in high risk neonates admitted to neonatal intensive care unit, GMERS Medical College and General Hospital, Gotri, Vadodara, Gujarat, India over a 1-year period. nRifle criteria was used for classification of acute kidney injury.

**Results:** The incidence of AKI in high risk newborns admitted in this study was 52 (37.14%). The male to female ratio in current study was 2.46:1. Majority of neonates with AKI were out born 44 (84.6%). The incidence of AKI was higher in term newborns. Mean weight in AKI group was 2048 grams. The highest incidence of AKI was found in AFD newborns (57.69%). nRifle criteria was used to diagnose AKI in this study. Out of 52 neonates who had AKI, 27 (51.9%) were in risk category, 21 (40.4%) were in injury group and 4 (7.7%) were in failure group. Mortality in these groups were 5 (18.51%), 7 (33.33%) and 3 (75%) respectively. Highest correlation of risk factors for AKI was found with birth asphyxia 18 (34.9%) followed by sepsis 12 (23.1%) and shock 15 (28.5%). 29 (55.76%) neonates had non oliguric AKI. 28 (53.8%) neonates with AKI developed dyselectrolytemia.

**Conclusions:** Early recognition and management of risk factors can help in reducing the occurrence and improve outcomes in them.

**Keywords:** AKI, Neonate, nRIFLE, Risk factors

### INTRODUCTION

Acute kidney injury (AKI) is defined as an acute deterioration in ability of the kidneys to maintain homeostasis of body fluids and electrolytes. It is associated with acute decrease in the rate of glomerular filtration (GFR) that leads to retention of wastes and toxic

metabolic end products. Most reports estimate the incidence of AKI in hospitalized neonatal population to be 8-24%. The incidence of intrinsic oliguric AKI in newborn infants admitted to the neonatal intensive care unit (NICU) ranges between 1-6% in retrospective studies and 6-8% in prospective studies. The incidence of acute kidney injury in foreign studies done in infants and

children varies between 3-10%.<sup>1</sup> Neonatal AKI is defined as the sudden severe derangement of glomerular function and is diagnosed when serum creatinine is greater than 1.0 mg/dl regardless of the rate of urine output or rising by 0.3 mg/dl/day or faster.<sup>2</sup> Evaluating acute kidney injury in infants and neonates is a challenge as renal function, serum creatinine and eGFR vary by the growth of the baby.<sup>3</sup> The change in renal function that defines AKI should be thought as the result of a combination of susceptibility factors and exposures with special consideration to risk factors inherent to neonatal renal development and physiology. Currently, the diagnosis of AKI is dependent on a rise in serum creatinine (S. creatinine) or decrease in urine output. Novel biomarkers such as urine neutrophil gelatinase associated lipocalin, cystitis C, kidney injury molecule-1 and others allow for the earlier identification of neonates with AKI, up to 48 hours prior to S. creatinine rise.<sup>4,5</sup>

### **Glomerular filtration rate**

The postnatal development of GFR has been assessed in premature neonates at different gestational ages and in term neonates. From a value of 20 ml/min x 1.73 m<sup>2</sup> at birth, GFR doubles in the first 2 weeks of life in term neonates.<sup>6</sup> Elevated concentration of creatinine at birth reflects the maternal levels while GFR develops at a lower velocity in premature infants. This explains why the specific dosage recommendations are tailored to the given gestational and postnatal ages for medications eliminated primarily via glomerular filtration e.g. aminoglycosides, vancomycin, digoxin.

### **Assessment of renal injury**

Assessment of glomerular filtration rate is done by Inulin clearance determination and serum creatinine levels.<sup>3</sup> Inulin clearance determination is "gold standard" for assessing GFR in both the immature and the mature kidney as it is freely filtered even in the preterm neonate with a gestational age as low as 27 weeks. S. creatinine is commonly used to assess GFR in neonates. Creatinine clearance correlates with inulin clearance and creatinine clearance studies have confirmed the rapid development of GFR during the first postnatal weeks. In clinical practice, sequential determination rather than a single value of serum creatinine gives the most valuable information on glomerular filtration rate. In term babies serum creatinine is approximately 0.8 mg/dl at birth and thereafter gradually decreases to approximately 0.5 mg/dl and 0.4 mg/dl by 1 week and 4 weeks respectively. The most common S. creatinine cut point used to define AKI has arbitrary been set as 1.5 mg/dl or greater, independent of day of life and regardless of the rate of urine output. In addition to serum creatinine, blood urea nitrogen has been widely used in clinical practice to assess renal function. However, as BUN is also influenced by the state of hydration, protein intake, liver disease, catabolic states including infections, it is a much less reliable marker of renal function than serum creatinine.

For prenatal evaluation, antenatal ultrasound is most widely used and effective diagnostic tool in the prenatal evaluation of the fetal kidneys and urinary tract. The most common indication for an ultrasound survey of the fetal genitourinary tract is the presence of oligohydramnios followed by presence of positive family history of renal disease. The fetal kidneys can be identified early in the second trimester, but a better evaluation is done usually between 28-30 weeks of gestation.

The biomarker of renal injury for clinical use till date is serum creatinine it, rises after almost 50% reduction of renal function and is affected by factors like age, sex, muscle mass and state of hydration. Therefore, diagnostic panel with sequential elevation of different proteins after renal injury is developed.

The earliest among these to rise exponentially after injury is plasma and urinary NGAL (neutrophil gelatinase associated lipocalin) plasma cystatin C, urinary IL-18 (interleukin 18) and KIM-1 (kidney injury molecule 1). Elevated urinary B2 M appears in 90% sick neonates with apparently normal renal parameters and indicates subclinical proximal tubular dysfunction especially in neonates with asphyxia, sepsis and congenital malformations. These investigations will enable an earlier diagnosis, differentiate between prerenal and renal failure and guide for different etiologies of renal injury.

### **Acute kidney injury criteria in neonates**

Over the last few decades, more than 35 different definitions have been used to define acute kidney injury (AKI).<sup>3</sup> Many of those definitions were complex, however, the more commonly used were based on urine output (UO) and/or serum creatinine (S. creatinine) criteria. The two most common classification systems for severity of AKI are the Risk, Injury, Failure, Loss, and End-Stage Renal Disease (RIFLE) and the Acute Kidney Injury Network (AKIN) classifications. In children, Akcan-Arikan et al, suggested a modified pediatric RIFLE (pRIFLE) classification with a lower cutoff of S. creatinine to achieve the failure (F) category, thereby reflecting the fact that children have a lower baseline S. creatinine.<sup>7</sup> The patient should be classified using the criteria (S. creatinine and/or UO) which leads to the worst classification (maximum RIFLE), Similar classification definitions of AKI are greatly needed to better describe the incidence and outcomes of AKI in different populations of critically ill neonates. Despite these working classification systems, the diagnosis of AKI is problematic, because current diagnosis relies on two functional abnormalities: functional changes in S. creatinine (marker of Glomerular Filtration Rate (GFR)) and oliguria. Studies report more than 50% of AKI cases to be non-oliguric, which highlights the insensitivity of oliguria in predicting AKI in neonates. Both of these measures are late consequences of injury and not markers of the injury itself. The ideal marker to detect AKI should be upregulated shortly after an injury and be independent

of the GFR (1). Creating AKI definitions using early injury biomarkers, which can ultimately predict morbidity and mortality accurately.

Early recognition of renal failure is important in neonates to facilitate appropriate fluid and electrolyte management as a stable biochemical milieu is vital to prevent permanent renal damage. The evaluation involves evaluating pre renal, intrinsic and post renal causes.

Prerenal AKI is the most common form of neonatal AKI. It occurs due to renal hypo-perfusion due to either systemic hypotension or to selective decrease in renal blood flow in response to tissue hypoxia without systemic hypotension but GFR is maintained.

About 6 to 8% of admitted newborns in Neonatal Intensive Care Unit (NICU) develop intrinsic renal failure with most common cause being severe perinatal asphyxia with irreversible renal damage. Other important causes include acute tubular necrosis, renal vascular causes, toxins induced, infections, intra renal obstruction.

Obstructive renal failure can be caused by variety of congenital condition of kidneys and urinary collecting system.

### **Clinical features and management<sup>8,9</sup>**

The chief clinical features of acute kidney injury are oliguria, edema if excessive fluid has been administered, hyperkalemia with cardiac arrhythmias, acidotic breathing, vomiting and poor feeding. The general approach in management consists of maintenance of fluid and electrolyte balance, avoidance of life-threatening complications, adequate nutritional support and treatment of the underlying cause.

Fluid administration should be electrolyte free 10% dextrose and limited to estimated insensible water losses plus the urine output. Infants with renal failure should be weighed every 12 hours and fluid administration adjusted accordingly. Hyperkalemia is managed with nebulized salbutamol, calcium gluconate, glucose insulin drip, Na bicarbonate, resins, I.V. frusemide and dialysis.

Symptomatic hyponatremia or serum Na <120 mmol/l (correct to 125) has to be treated. In addition to the treatment of hyperkalemia, metabolic acidosis should be corrected if the plasma bicarbonate concentration falls below 16 meq/L or the arterial ph is less than 7.20 by administering sodium bicarbonate. Phosphorus intake should be restricted in infants taking enteral feedings by using a formula low in phosphorus.

Symptomatic hypocalcemia should be corrected by infusing 10% calcium gluconate at a dose of 1-2 ml/kg over 5-10 minute under cardiac monitoring. Nutritional support is essential for infants with AKI and should consist of a minimum of 25 kcal/kg. Infants who are able

to take enteral feedings should be given a formula that has a low renal solute load and low phosphate content. However, the need for fluid restriction makes it difficult to meet the caloric needs of an oliguric infant. As a result, daily loss of 0.2 to 1 percent of body weight usually persists beyond the first week of age.

### **Renal replacement therapy**

should be considered if appropriate fluid and electrolyte balance and adequate nutrition cannot be maintained because of persistent oliguria or anuria. Available renal replacement modalities for the management of AKI in newborns include hemodialysis, peritoneal dialysis, and hemofiltration (with or without dialysis). The choice of modality is influenced by the clinical presentation, the presence or absence of multisystem failure, and the indication for renal replacement therapy.

Peritoneal dialysis is performed in newborns, even in low birth weight. It is safe and technically simpler and less expensive than hemodialysis and hemofiltration, requiring only minimal equipment. It can be initiated immediately after the dialysis catheter is placed and can be done as soon as three days after major abdominal surgery. Morbidity and mortality are high in newborns undergoing peritoneal dialysis and are related to the infant's underlying diagnosis and clinical condition.

Complications include peritonitis, obstruction of the dialysis catheter, hyperglycemia, bleeding and perforation of abdominal viscera are common. Use of continuous veno-venous hemodiafiltration (CVVHD) is increasing in newborns as it allows more precise fluid and metabolic control, decreases hemodynamic instability, and, enhances the removal of cytokines in patients with sepsis or multiorgan system failure.

## **METHODS**

Present study was observational and prospective study, hospital based conducted at Neonatal Intensive Care Unit (NICU) at GMERS Medical College and General Hospital, Gotri, Vadodara, Gujarat, India from June 2017 to June 2018.

The study population includes all high-risk newborns (<28 days of life) including inborn and outborn admitted at neonatal division of Department of Paediatrics at GMERS Medical College and General Hospital, Gotri, Vadodara, Gujarat, India. Sample size was 140 newborns.

### **Inclusion criteria**

High risk neonates admitted to neonatal intensive care unit, GMERS Medical College and General Hospital, Gotri, Vadodara, Gujarat, India.

- Birth weight <1500 grams,
- Gestation <32 weeks,

- Birth weight  $\geq 1500$  grams or Gestation  $\geq 32$  weeks with any of the,
- Below mentioned associated risk factors
  - Intrauterine growth restriction weight  $< 3^{\text{rd}}$  centile,
  - Hypoxic ischemic encephalopathy stage 2 or higher,
  - Received mechanical ventilation,
  - Meningitis,
  - Inborn errors of metabolism/intrauterine infections,
  - Major malformation,
  - Symptomatic hypoglycaemia,
  - Hyperbilirubemia requiring exchange transfusion,
  - Abnormal neurological examination/seizure,
  - Major morbidities such as chronic lung disease, IVH grade III or more.

**Exclusion criteria**

Babies admitted to neonatal division but not fitting in inclusion criteria. Parents unwilling to give informed consent.

**Procedure**

High risk neonates were selected according to inclusion criteria. Informed consent was taken from parents. Renal profile (blood urea and serum creatinine) was done after 12 hours of life (or at the time of admission for outborn

babies) and later as and when necessary. 24-hour urine output was measured. Neonates who developed renal failure were managed as per standard hospital protocol. ABG or VBG, CRP, urine routine and microscopy, urine culture, blood culture were done when indicated. Renal profile was repeated after appropriate treatment in those babies having abnormal renal function. Gestational age, birth weight, findings on general examination and systemic examination, urine output, investigations, treatment given, and outcome were recorded on predesigned proforma.

Data was analysed using Microsoft excel sheet computer program. Statistical analysis of the data was performed using the chi-square or Fisher exact test. Significance was defined as  $p < 0.05$ . By means of this study, author tried to assess incidence, risk factors and the effect of rapid effective treatment of contributing condition in reducing the incidence of acute kidney injury and long-term adverse effects.

**RESULTS**

A prospective observational study was conducted from June 2017 to June 2018 at tertiary level neonatal intensive care unit of GMERS Medical College and General Hospital, Gotri, Vadodara, Gujarat, India.

All high-risk newborns admitted to Neonatal Intensive Care Unit (NICU) fulfilling the inclusion criteria were enrolled for the study. Total numbers of high-risk neonates included in this study were 140 out of which AKI developed in 52 (37.14%) of the newborns.

**Table 1: Criteria adopted for the study.<sup>7</sup>**

Class	Serum Creatinine	Urine output and duration		
		RIFLE	pRIFLE	nRIFLE
Risk (R)	SCr X 1.5	$\leq 0.5$ ml/ kg/hr (6h)	$< 0.5$ ml/kg/hr (8h)	$< 1.5$ ml/kg/hr (24h)
Injury (I)	SCr X 2.0	$\leq 0.5$ ml/ kg/hr (12h)	$< 0.5$ ml/kg/ hr (16h)	$< 1.0$ ml/kg/hr (24h)
Failure (F)	SCr X 3.0 or $\geq 4$ or Acute rise $> 0.5$ mg/dl	$\leq 0.5$ ml/kg/hr (24h) or Anuric (12h)	$< 0.3$ ml/kg/hr (24h) or Anuric (12h)	$< 0.7$ ml/kg/hr (24h) or Anuric (12h)

Data was analysed using Microsoft excel sheet computer program. Statistical analysis of the data was performed using the chi-square or fisher exact test. Significance was defined as  $p < 0.05$ . To assess the degree of correlation between each independent variable and abnormalities, a ratio of prevalence was determined with a confidence interval of 95%.

The observations were made  $n=52$ . Out of 52 neonates who had AKI, 37 (71.1%) were males and 15 (28.9%) were females. So, male to female ration in current study was 2.46:1, out of 52 neonates with AKI, 8 (15.4%) were inborn and 44 (84.6%) were outborn. Occurrence of AKI

was observed to be significant in outborn neonates ( $p < 0.05$ ). In this study, the incidence of AKI was higher in term newborns. This finding can be explained by high risk conditions like birth asphyxia, sepsis, occurring more in term neonates. Among 85 neonates of  $\geq 37$  weeks, 40 (47%) had AKI. Occurrence of AKI increased with increasing gestational age ( $p < 0.05$ ). Mean weight of study was 2018 grams. Mean weight in AKI group was 2048 grams. In this study, only 4 VLBW neonates had AKI, while 18 neonates (34.61%) in weight band 1500-2500 developed AKI. The highest incidence was found in AFD newborns (57.69%). Incidence of AKI increased with increasing birth weight ( $p < 0.05$ ).

**Table 2: Demographic association with AKI.**

Gender	No.	%	P value
Male	37	71.1	<0.05
Female	15	28.9	
<b>Admissions</b>			
Inborn	8	15.4	<0.05
Outborn	44	84.6	
<b>Gestational age</b>			
<33 weeks	6	11.5	<0.05
33-37 weeks	6	11.5	
>37 weeks	40	76.9	
<b>Birth weight</b>			
=<1500 gms	04	07.69	<0.05
1500-2499 gms	18	34.61	
>=2500 gms	30	57.69	

**Table 3: Association of nRIFLE criteria with outcomes.**

Class	Total	Mortality	Survival
Risk	27(51.9%)	5 (18.5%)	22 (81.5%)
Injury	21(40.4%)	7 (33.3%)	14 (66.7%)
Failure	4(7.7%)	3 (75%)	1 (25%)
Total	52	15	37
<b>Pearson Chi-square</b>			
Value	5.760		
Df	2		
P value	0.05		

**Table 4: Risk factor association with AKI.**

Risk factor	AKI
Birth asphyxia	18 (34.9%)
Sepsis	12 (23.1%)
Shock	15 (28.5%)
Birth weight <1500 GRAMS	3 (5.8%)
Gestational age <32 WEEKS	4 (7.7%)

**Table 5: Clinical features in AKI.**

Clinical feature	N=52	%
Oliguria	23	44.23%
Non oliguric	29	55.76%
Hyperkalemia	23	44.23%
Metabolic acidosis	05	9.61%
Edema	03	5.76%
Seizures	03	5.76%

nRIFLE criteria was used to diagnose AKI in this study. Out of 52 neonates, who had AKI, 27 (51.9%) were in risk category, 21 (40.4%) were in injury group and 4 (7.7%) were in failure group. Mortality in these groups were 5 (18.51%), 7 (33.33%) and 3 (75%) respectively. Thus, author can conclude that there is strong correlation between the stage of nRIFLE criteria and percentage of mortality (p=0.05).

Among 52 neonates with AKI, 18 (34.9%) had moderate or severe birth asphyxia. 12 (23.1%) neonates with AKI had sepsis as primary risk factor. Shock was observed as risk factor in 15 (28.5%) of newborns with AKI. Highest correlation was found in birth asphyxia (p <0.001) followed by sepsis and shock.

Out of 52 neonates, who had AKI, 29 (55.76%) had adequate urine output whereas 23 (44.23%) had oliguria (urine output <1 ml/kg/hr for 24 hours). Non oliguric AKI was more common in this study (p=0.05), 28 (53.8%) neonates with AKI developed dyselectrolytemia.

**DISCUSSION**

Acute kidney injury in neonates and infants in literature has been described in special populations like post asphyxia low birth weight, post cardiac surgery, preterm etc.<sup>10-12</sup>

GMERS Medical College and General Hospital is a tertiary neonatal intensive care unit referral centre with wide drainage of patients from peripheral areas. In this study, the occurrence of AKI was 37.1%. This was slightly higher than that reported by Aggarwal A et al, 34.5%.<sup>13</sup> However, other studies have reported an incidence of acute kidney injury in range of 3.4-24%.<sup>14</sup> The wide variability in incidence of AKI in the available data from different units can be attributed to demographic characteristics of population studied as well as different criteria being used for diagnosis of AKI. In this study, there was male sex predominance and the male-female ratio was 2.46:1. Present study was in agreement with Pradhan SK et al, and Gharenbhaghi MM et al, who reported a male-female ratio of 2.03:1 and with Airede A et al, with a male-female ratio of 3.3:1 in neonates with AKI.<sup>15-17</sup>

Present study showed that the mean and standard deviation of gestational age in weeks was 37.37±3.6. Incidence of AKI increased with increasing gestational age (p <0.05). This is in agreement with study by Bansal SC et al, who also reported higher incidence in full term neonates. Similar correlation was found with birth weight.<sup>18</sup>

Previous study from India showed that the percentage of babies with birth weight of <2500 gram in AKI group was higher than in healthy neonates. Most studies suggest that AKI is more common in VLBW/ELBW neonates and is associated with poor prognosis. Koralkar R et al, reported incidence of AKI using modified KDIGO criteria to be 18% amongst 229 VLBW infants.<sup>19</sup> Vishwanathan S et al, and Carmody JB et al, also reported similar findings.<sup>20,21</sup> Interestingly, author observed higher incidence in term babies in this study, due to the fact that a majority of full term neonates in this study were referred for sepsis or asphyxia, that are also individual high risk factors for AKI. Majority of cases with AKI (57.6%) weighed more than 2500 gms. Non

oliguric AKI was more frequent (55.76%) than oliguric AKI, ( $p=0.05$ ). This finding is similar to the study done by Girish G with non-oliguric AKI observed in 62.5% cases.<sup>22</sup> Significant association was seen with low APGAR score at 1 minute with AKI ( $P < 0.001$ ). Neonates with birth asphyxia have been associated with higher risk of AKI. Several previous studies have found birth asphyxia to be the most common cause of AKI of neonatal period. Birth asphyxia is associated with acute tubular injury which is the most common cause of intrinsic AKI. Two recent studies reported an association between asphyxia and AKI using modern definition of AKI. AKI after perinatal asphyxia was noted in 42% of cases for Martin-Ancel A et al, 47% in Gupta B et al, 68% in Aggrawal A et al, 70% in Gluckman P et al, 17.2% by Nouri S et al.<sup>23-27</sup> Present study noted 34.9% prevalence rate hence lying within the range of most of the studies done. The available studies show that the prevalence rates were similar in both resource poor and resource rich areas proving that AKI in perinatal asphyxia is a global problem. The presence of perinatal asphyxia and its severity appears to correlate with increasing incidence of AKI. About 23.1% of neonates with AKI had sepsis. Sepsis has been consistently associated as a risk factor for development of AKI in various studies conducted around the world contributing to as high as 78% cases in some neonatal studies.<sup>28</sup> Another study from India by Mathur NB et al, shows that out of 200 newborns with sepsis, 26% developed AKI.<sup>29</sup> The newborns with sepsis are thought to be predisposed for AKI as a result of hypotension secondary to sepsis and a direct damaging effect on renal microvasculature. Shock has significant association with AKI in this study ( $p < 0.001$ ). It corresponds with the study done by Mathur NB et al.

Major limitation is small sample size and absence of long term follow up precludes comments on delayed renal sequelae.

## CONCLUSION

Neonatal AKI represents a rapidly evolving area in clinical research. There have been significant advancements in these understandings of AKI and its impact on outcomes. Major risk factors associated with AKI were male gender, birth asphyxia, shock and sepsis in this study.

## Recommendations

The first step for prevention and management of neonatal AKI is to identify infants at high risk for renal failure and oliguria. Renal profile monitoring is vital if any of these risk factors are present. Renal profile also helps to guide fluid therapy and drug dosage in AKI group. Early recognition of risk factors and rapid effective treatment of these contributing factors will reduce the morbidity and mortality in AKI Group.

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