Original Article**A Cohort Study of Acute Kidney Injury in Egyptian Neonates; Etiology, Risk Factors, Diagnosis and Three Months Follow Up.****Mohammad Abdelmonaem Sharaf¹, Wafaa Osman Ahmed²****1-Department of Pediatrics, Nephrology Unit, Faculty of Medicine, Ain Shams University, Cairo, Egypt.****2-Department of Pediatrics, Neonatology Unit, Faculty of Medicine, Ain Shams University, Cairo, Egypt.****Abstract**

Introduction: Acute kidney injury (AKI) is a major contributor of neonatal mortality and morbidity. Understanding the different anatomy of the neonatal kidney could help physician understand pathology of AKI and consequently could treat it efficiently.

Aim of the study: This one year duration prospective Cohort study involved twenty seven admitted neonates who had acute kidney injury (AKI).

Methods: Laboratory and radiologic diagnosis of AKI, further AKIN and pRIFLE classification and follow up after one and three months.

Results: AKI had an incidence of 1.35%, mainly in full-term (59%). Hypertension was presented in full-term (75%) and preterm (45.5%). Hematuria presented by 18.8% (full-term) and 27.3% (preterm). Proteinuria occurred in full-term (46.7%) and preterm (36.4%). Urinary tract infection was more among (45.5%) preterm than full term (25%). Oliguric AKI was found in full term (68.8%) and preterm (72.7%). Anuric AKI present in full-term (31.1%) and preterm (9.1%). Post renal AKI were present 18.8 % (full term) and 9.1 % (preterm). Perinatal asphyxia (18.8%) and sepsis (27.3%) were the most common risk factor. Both full-term (37.5%) and preterm (18.2%) needed peritoneal dialysis. Ultrasonography showed bilateral Grade 2 (50%) and 1(25%) nephropathy and bilateral back pressure (18%). pRIFLE was inversely correlate with age and urine output and positively correlated with hypertension at baseline. At one month; 43.8% full-term, 18.2 % preterm were found hypertensive, 18.8% fullterm had proteinuria, pRIFLE was inversely correlated with urine output and proteinuria and eCCL was significantly improved after one month in both preterm and full-term. At three month, chronic kidney disease was 12.5% and 9.1% in full-term and preterm respectively. Total recovery was higher in preterm neonates than in full-term ones.

Conclusion: Sepsis and asphyxia are considered the most common causes of AKI.

Limitation: A longer follow up is mandatory. Further follow up studies of AKI in preterm neonates are mandatory.

Key words: AKI, pRIFLE, AKIN, Neonate, Critically ill, NICU

Running title: A short term follow up of AKI in neonates

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Introduction

Acute kidney injury (AKI) is a major contributor of neonatal mortality and morbidity. Understanding the different anatomy of the neonatal kidney could help physician understand pathology of AKI and consequently could treat it efficiently. Failure of renal function adaptation could precipitate AKI, for example; the kidney must excrete low sodium contained filtrate to preserve sodium as the maternal milk contain small sodium content [1].

Many factors could precipitate AKI in neonates. The decreased renal blood share of the cardiac output that increases to normal level by 2 years of age, the reduced medullary tonicity that renders a hypotonic urine(max 700 mOsm), the low glomerular filtration rate (GFR) that increases 8 folds during first week and increases further till the age of two months. Also the impaired tubular function is present initially and is responsible for the initially occurring positive potassium balance needed for growth of the preterm neonate. The accelerated diuresis that occurs later that if failed to occur, may render AKI [2].

Serum creatinine is not the ideal marker for assessment of kidney function due to its delayed rise after kidney injury and it initially reflects maternal creatinine [3]. It could help if serial measurements are taken, but this also may cause anemia in preterm babies due to multiple sampling [4].

In 2000 Gouyon and Guignard defined renal insufficiency of very preterm babies as a daily increase in serum creatinine of >0.50 mg/dL from day 0 to 1 and 0.30 mg/dL/24 h during the remainder of the first week of life [5].

Oliguria was defined as urine volume of <1 mL/kg/h over a period of 24 h; severe oliguria was defined as urine volume of <0.5 mL/kg/h. Subsequent AKI definitions have also used varying combinations of rising serum creatinine, oliguria, and elevated blood urea nitrogen (BUN) levels, with the most widely used definition between 1995 and 2005 as an absolute serum creatinine of ≥ 1.5 mg/dL. Estimated creatinine clearance (eCCL) is used to more understanding of the AKI incidence. Treatment of AKI is directed towards fluid restriction, electrolyte balance and renal replacement therapy [6].

There are three main modalities of RRT in pediatric age of which the peritoneal dialysis is considered the most common used line of treatment in neonates. The choice of the modality of RRT largely depends on the center experience, availability of access, and the presence of certain contraindications to a particular modality [7]. The glomerular filtration rate (GFR) is much lower in neonates than the adult GFR. So the neonates especially prematurely delivered ones are more liable to develop AKI due to failure of extra uterine renal adaptation [8].

Methods

This prospective Cohort study was carried out in our children hospital NICU, from June 2019 to June 2020. Written informed consent was obtained from each parents/ care giver. The ethical committee had approved the search. A total of 27 sick neonates (aged 3-28 days) admitted and fulfilled the criteria of AKI were included in the study. Patients with lethal congenital anomalies were excluded. AKI

was suspected when urinary output was reduced <0.5 ml/ kg/hr and increasing serum creatinine with or without reduced output <0.5 ml/kg/hr, for 24 hours duration. In preterm neonates; baseline creatinine was recorded according to gestational age (GA); (GA 24–27: 1.6 mg/dL, GA 28–29: 1.1 mg/dL, GA 30–32: 1.0 mg/dL), so serum creatinine considered noted when raised 1.5-2 folds from the base line. By Schwartz formula; we estimated creatinine clearance (eCCL) ($eCCL = k \times \text{Length (cm)} / S. \text{Creatinine}$) where k for preterm = 0.27 and for term = 0.37}. Further recording of BUN, serum bicarbonate (HCO_3), potassium (K), complete blood count (CBC). According to pRIFLE criteria neonates with impaired renal function were classified as Risk, Injury, Failure group based upon eCCL and urine output. According to AKIN staging acute kidney injury classified as stage I, stage II, stage III on the basis of serum creatinine and urine output. AKI was divided into oliguria and non oliguric and the early outcomes were registered as death and discharge.

Urine collecting adhesive bags were used to collect urine, and was recorded as per kilogram of body weight per hour in admission sheets. Blood samples were collected as per routine evaluation for the admitted neonates and the lab data were further collected from the medical records.

Detailed history was taken including demographic characteristics, history and type of nephrotoxic drugs, history of associated diseases; respiratory distress, sepsis, dehydration, necrotizing enterocolitis, asphyxia and underlying congenital anomalies according to WHO definitions [10]. Clinical evaluation of patients entailed the monitoring of the

urine output, blood pressure, presence of hematuria, proteinuria and urinary tract infection. Further data collection involved the abdominal ultrasonography, history of assisted ventilation, blood transfusion, need for inotropic support. Different treatment modalities involved either sodium bicarbonate intake alone or conjoined with K exchange resin, glucose insulin infusion, intravenous albumin and phosphorus (P) binder. Further treatment by peritoneal dialysis was recorded. Two follow up visits were done for the patients; during which; clinical reevaluation including weight gain, length, presence of hypertension, hematuria, proteinuria and laboratory evaluation of BUN, creatinine, CBC and urine analysis. Hypertension is defined as systolic blood pressure (BP) of at least the 95th percentile (stage 1) for gestational age, birthweight, and sex on 3 separate occasions.

Severe hypertension (stage 2) defined as systolic BP >99 th percentile and was measured non-invasively, using the oscillometric technique, by Dragger; the Infinity Delta Series (Delta/Delta XL/Kappa), the studied groups had stage one to two hypertension. Assessment of treatment that was received by the patients (active vitamin D, NaHCO_3 , erythropoietin and K reducing medications). In the second follow up visit (two month later); same evaluation as the one month follow up visit, in addition, abdominal ultrasonography was done. The mean duration of hospitalization for each newborn was recorded. All available data were compared between the two groups.

Statistical analysis

Data was entered in Microsoft Excel

2007 and STATA 14 was used to analyze descriptive data. Chi square/independent sample t-test as applicable and logistic regression were used to establish an association of various factors and outcome with AKI. The p-value less than 0.05 were considered significant. Data were compared using SPSS statistical software version 16.0 (SPSS Inc., IL, USA). And through descriptive statistical and inferential statistical methods including Chi-square test, Pearson's correlation test and multiple linear regression analysis. $P < 0.05$ were considered as statistically significant.

Results

This study involved 11 preterm neonates and 16 full-term ones, admitted to neonatal intensive care unit (NICU). All cases were diagnosed to have AKI according to Gouyon and Guignard, 2000. Mean age at presentation to the NICU were 7.2 ± 5.4 days and 8.9 ± 5.7 days in both the full-term and preterm neonates respectively. Mean birth weight was 2885 ± 519 gram (gm) and 1837 ± 310.6 gm in full-term and preterm respectively. Full-term group were divided as 8 males and 8 females, while preterm group were divided as 6 males

and 5 females. The male to female ratio was 1:1 in full-term and 1:1.19 in preterm respectively. (59%) were full-term neonates. Mean gestational age at presentation were 37.6 ± 1.2 and 33.5 ± 1.8 weeks in full-term and preterm neonates respectively. History of nephrotoxic drugs and dehydration were demonstrated in (Table 1). Oliguria and anuric AKI were more pronounced in full-term than preterm neonates. Post renal AKI were 18.8% and 9.1% in full-term and preterm respectively. Perinatal asphyxia (18.8%) and sepsis (27.3%) were the most common predisposing factor for AKI in full-term neonates respectively as shown in (Table 2).

Serum creatinine noted when raised more than 0.3 mg/dl or raised 1.5-2 folds from the base line within duration of 24 hours. At base line evaluation, all of our patients are of the stage III according to AKIN classification. While according to the pRIFLE; 3.7% were at risk, 11.1% had injury and 85.2% had renal failure. After one month follow up; 7.4% risk and 11.1% injury. While according to AKIN classification, stage I (59.3%), stage II (18.5%), stage III (3.7%). No sex predilection in affection of neonates according to pRIFLE ($P=0.15$).

Table 1: Comparison of baseline characteristics between different studied subgroups

			Group 1 (Full-term) n=16	Group 2 (Preterm) n=11	P value
Hx of nephrotoxic drugs	+ve	N	5	6	0.264
		%	31.3%	54.5%	
Hypoxic insult : (clinical + cranial U/S)	+ve	N	6	1	0.183
		%	37.5%	9.1%	
Sepsis	+ve	N	11	10	0.350
		%	68.8%	90.9%	
Hx of dehydration	+ve	N	5	4	0.782
		%	31.3%	36.4%	

Significant: $p < 0.05$

Table 2: Initial diagnosis of the studied groups

			Group 1 (Full-term) N= 16	Group 2 (Preterm) N=11	P value
Primary Diagnosis	Respiratory distress	N	0	4	0.038 *
		%	0.0%	36.4%	
	Congenital pneumonia	N	1	2	
		%	6.3%	18.2%	
	Meconium aspiration	N	2	0	
		%	12.5%	0.0%	
	Sepsis	N	2	3	
		%	12.5%	27.3%	
	Urinary Tract Infection	N	2	1	
		%	12.5%	9.1%	
	Hypoxic ischemic encephalopathy	N	3	0	
		%	18.8%	0.0%	
	Congenital Cyanotic heart disease.	N	2	0	
		%	12.5%	0.0%	
	Trache-oesophageal fistula	N	1	0	
		%	6.3%	0.0%	
	Hydrocephalus + Ventriculitis	N	0	1	
		%	0.0%	9.1%	
	Diaphragmatic hernia	N	2	0	
		%	12.5%	0.0%	

*Significant

Laboratory evaluation in our study revealed that (68.8%) full-term and (90.9%) preterm neonates had positive CRP and blood culture proved sepsis. Results of kidney functions are illustrated in (Table 3). Pelviabdominal sonography was done and revealed different degrees of nephropathy, where 48.1% and 18.5% had grade 2 and 3 respectively and 14.8% had bilateral back pressure changes. At one month and three month clinical evaluation were illustrated in (Table 4).

Medications used to treat base line AKI and follow up treatment were mentioned in (Table 5). While Outcome of AKI at three months follow up is shown in (Table 6).

Blood transfusion was needed in 68.8% of full-term and 54.5% of preterm babies. During treatment, all full-term patients needed respiratory support while 81.8% of preterm babies needed respiratory support. A higher percentage of full-term and preterm babies (87.5%

and 54.5%) respectively needed inotropic support ($p = 0.05$). Mean eCCL at one month was 34.4 ± 22.3 in full-term and 29.8 ± 12.3 in preterm. A high significant difference was found among full-term

neonates regarding the base line and follow up eCCL ($p < 0.001$). Also the same finding was found between base line and follow up eCCL in preterm neonates ($p < 0.001$).

Table 3: Comparison of basic numeric data between the studied subgroups

	Group 1 (Full-term) N=16		Group 2 (Preterm) N=11		P value
	Mean	SD	Mean	SD	
Basal S. Creatinine (mg/dl)	3.1	1.4	2.4	1.3	0.190
Basal BUN (mg/dl)	64.5	28.0	49.8	26.9	0.186
S. Potassium (mEq/L)	7.2	0.6	7.1	0.6	0.659
S.Bicarbonate (mmol/L)	10.5	2.3	11.4	1.9	0.256
Hemoglobin (g%)	10.3	3.0	10.3	1.3	0.977
Duration of Peritoneal Dialysis (Hrs)	27.0	38.2	16.4	36.7	0.477
NICU stay duration (days)	24.8	10.2	17.4	9.1	0.049*

*Significant

Table 4: Illustration of clinical data at one & three month after onset of AKI among studied groups.

		One month follow up				Three month follow up		
			Group 1 (Full-term) N = 16	Group 2 (Preterm) N = 11	P	Group 1 (Full-term) N = 16	Group 2 (Preterm) N = 11	P
Urine output	Normal	N	12	11	0.499	14	11	0.499
		%	80.0 %	100 %		87.%	100 %	
	Polyuria	N	3	0		2	0	
		%	20.0 %	0.0 %		12.%	0.0 %	
Hyper tension	+ ve	N	7	2	0.231	3	0	0.248
		%	43.8%	18.2%		18.8%	0.0%	
	-ve	N	9	9		13	11	
		%	56.3%	81.8%		81.3%	100 %	
Hematuria	- ve	N	16	11		16	11	
		%	100 %	100 %		100 %	100 %	
Proteinuria	+ ve	N	3	0	0.262	2	0	0.499
		%	18.8 %	0.0 %		12.5%	0.0%	
	- ve	N	13	10		14	11	
		%	81.3%	100.0%				

UTI	+ ve	N	2	1		2	1	0.782	
		%	12.5%	9.1%		12.5%	9.1%		
	-ve	N	14	10			14		10
		%	87.5%	90.9 %			87.5%		90.9%
Weight gain	+ ve	N	7	9	0.618	12	10	0.428	
		%	43.8%	81.8%		75.0%	90.9%		
	-ve	N	2	1			4		1
		%	12.5%	9.1%			25.0%		9.1%

Table 5: Illustration of base line and current drugs for AKI between different studied groups.

Initial treatment					Current treatment					
		Gp 1 (FT) N = 16	Gp 2 (PT) N = 11	P			Gp 1 (FT) N =16	Gp 2 (PT) N =11	P	
NaHCO3.	N	1	1	0.554	None	N	5	6	0.59	
	%	6.3 %	9.1%			%	31.3%	54.5%		
K exchange resin + Gluc. Insulin inf + NaHCO3.	N	3	1		Active vit D	N	4	4		
	%	18.8 %	9.1 %			%	25 %	36.4 %		
Gluc. Insulin inf + NaHCO3.	N	2	3		NaHCO3, +EPO,+ active vit D	N	1	0		
	%	12.5%	27.3%			%	6.3%	0.0%		
K exchange resin + IV albumin + NaHCO3.	N	2	1		P binder + NaHCO3, + EPO, + active vit D	N	1	0		
	%	12.5%	9.1%			%	6.3%	0.0%		
K exchange resin+ P binder + Gluc. Insulin inf + NaHCO3.	N	3	0		EPO,+ active vit D	N	1	0		
	%	18.8%	0.0%			%	6.3 %	0.0%		
K exchange resin +NaHCO3.	N	4	3		K exchange resin + P binder + NaHCO3, + EPO,+ active vit D	N	1	0		
	%	25.0%	27.3%			%	6.3 %	0.0%		
P binder + Gluc. Insulin inf + NaHCO3.	N	1	0		P binder + EPO, +active vit D, + Fe	N	1	0		
	%	6.3%	0.0%			%	6.3%	0.0%		
K exchange resin + Gluc. Insulin inf + IV albumin + NaHCO3.	N	0	2		P binder +active vit D, + =Fe	N	1	0		
	%	0.0%	18.2%			%	6.6%	0.0%		
					P binder + active vit D	N	1	0		
						%	6.3 %	0.0%		
					P binder+ NaHCO3+activ e vit D	N	0	1		
						%	0.0 %	9.1%		

p; phosphorus, EPO; erythropoietin, K : potassium, **NaHCO₃**: sodium bicarbonate, vit D : vitamin D

Table 6: Outcome of AKI at three months follow up among studied subgroups.

			Group 1 (Full-term) N = 16	Group 2 (Preterm) N = 11	P value
Outcome of Primary disease at 3 months	Total recovery	N	6	9	0.157
		%	37.5 %	81.8 %	
	Broncho-pulmonary dysplasia	N	2	1	
		%	12.5 %	9.1 %	
	Sequelae of asphyxia	N	4	0	
		%	25.0 %	0.0 %	
	Congestive heart failure	N	2	0	
		%	12.5 %	0.0 %	
	Chronic kidney disease	N	2	1	
		%	12.5 %	9.1 %	

Discussion

This study aimed at evaluation of causes of AKI, diagnosis, treatment, follow up at one and three month of age and defining residual kidney insults in a group of Egyptian neonates. AKI diagnosis was based on creatinine and urine output, and was further classified by pRIFLE and AKIN.

Out of 2000 neonate admitted to our NICU; incidence of AKI was 1.35% in a period from April 2019 to April in 2020. This was nearly similar to Hossein et al., Aggarwa et al., Satvik et al. And Masoumeh et al., 2015 who reported a percentage of 1.5%, 3.45%, 4.24% and 3.2%. Also, Rinaldo et al., 2004 reported that 1% to 25% of critically ill neonates had AKI. Other studies using criterion of serum creatinine >1.5 mg/dl showed an incidence of AKI as 3.4%, 6.3% [11, 6, 12-15].

Other study using urine output with serum creatinine as criteria for diagnosis, the incidence of AKI was 20%, highlighting the importance of having fixed definitions of AKI [16, 17]. Most

older studies, have used different definitions of AKI; one frequently used absolute serum creatinine >1.5 mg/dl [12–14], other studies have used risk, injury, failure, loss of kidney function, and End-stage kidney disease (RIFLE) and Acute Kidney Injury Network (AKIN) criteria [13]. Recently, Jetton JG and Askenazi [18] proposed a new definition in 2012. It graded severity of AKI using changes in serum creatinine and urine output. Subsequently, in April 2013 the group of neonatologists and pediatric nephrologists at the National Institute of Health (NIH) neonatal AKI workshop recommended the use of this definition [14].

Masoumeh et al., 2015 included 904 admissions in the NICU, where the rate of ARF was 3.2% by the old definition and 77.5% by the RIFLE criteria, reflecting underestimation of AKI by measurement of creatinine and urine output only [13].

A recent study by Momtaz HE et al., reported RDS as a third most common association with AKI (34.6%) after sepsis and dehydration [20].

Birth asphyxia is another common cause of AKI in newborn infants [16, 26, 31]. Other studies reported an association between asphyxia and AKI using modern definition for AKI [21, 22]. Selewski DT et al., reported an incidence of 38% of AKI and Kaur S et al., reported 41.67% among infants who suffered from asphyxia.

In our study, 59% neonates were full-term. Other studies reported the predominance of AKI in term neonates [21, 11]. AKI was more common in term babies (54.1% v/s 28.4 %; $p=0.001$) [12]. On the contrary, Jennifer et al., 2017 found that 45% were 29 weeks to younger than 36 weeks, and 41% were 36 weeks or older [23].

In our study, mean birth weight was 2885 ± 519 gram (gm) and 1837 ± 310.6 gm in full term and preterm respectively. While Hossein et al 2014 reported a higher average weight of AKI babies (3510 ± 680 g). In Mathur et al., study [24] the birth weight of <2500 g in AKI group was higher in sick than healthy neonates.

Our study showed no sex difference in incidence of AKI according to pRIFLE ($P=0.15$), where the male to female ratio was 1:1 in full-term and 1:1.19 in preterm respectively. Hossein et al found a higher female affection (87.8%). Other studies found that the boys had a higher prevalence of AKI [6, 11, 12]. Predominance of affection of male newborns were observed [12]. Another study although reported higher prevalence of AKI among females [25].

The mean age at presentation and evaluation was earlier in full-term than in preterm (7.2 ± 5.4 vs 8.9 ± 5.7) days respectively. This could be explained by delay or failure of adaptation of kidneys

of preterm babies. Also Hossein et al 2014 reported a similar mean age of affection (7.4 ± 6.2 days) [11].

In our study; the leading cause of AKI in full-term neonates was asphyxia, while in preterm neonates was neonatal sepsis. 37.5% and 9% of the studied full-term and preterm neonates experienced perinatal asphyxia, and this is a much lower percentage than that was found by Tahmina et al., 2017. Their study showed that 68.2% of the neonates had perinatal asphyxia stage II and stage III [28]. On our side; 68% and 81.8% rate of sepsis was documented in both preterm and full-term respectively. Also Tahmina et al., study showed that septicemia occurred in a percentage of (59%). Other cases that developed AKI in our study were associated with respiratory distress syndrome, meconium aspiration, congenital pneumonia, urinary tract infection, diaphragmatic hernia, ventriculitis (Table 2). The primary diagnoses documented study of Masoumeh et al., 2015 were respiratory distress syndrome (RDS) 56%, gastrointestinal obstruction 19%, sepsis 6%, prematurity 6%, convulsion 5% and others 8%. Hossein et al., 2014 documented that the most common leading cause of AKI was sepsis (77.5%), followed by hypovolemia secondary to dehydration (46.9%), hypoxia secondary to respiratory distress syndrome (RDS) (34.6%), patent ductus arteriosus (PDA) (8.1%), posterior urethral valves (PUVs) (6.1%), asphyxia (4%), and renal venous thrombosis (RVT) (2%) [11, 13].

Csaicsich et al. study [27] also showed that the most common predisposing factor for AKI was sepsis and ischemic events. Other studies,

prenatal asphyxia has been considered as the most prevalent cause of AKI higher than sepsis which was not consistent with the present observation [11, 12, 13]. Furthermore, in a study by Abu-Haweleh [30], the perinatal asphyxia with the prevalence of 42% was the most common predisposing factor for AKI in neonates and the one most associated with mortality. Other study of AKI in sixteen neonates, perinatal asphyxia, renal anomalies and congenital heart diseases were risk factors for AKI [16].

At base line evaluation, all of our patients are of the stage III according to AKIN classification. While according to the pRIFLE; 3.7% were at risk, 11.1% had injury and 85.2% had renal failure. pRIFLE classification was inversely correlate with age and urine output and positively correlated with hypertension at baseline.

After one month follow up; 7.4% risk and 11.1% injury. While according to AKIN classification, stage I (59.3%), stage II (18.5%), stage III (3.7%) were documented. Masoumeh et al., 2015 found that 77.5% developed AKI by RIFLE criteria and among them 43% met the risk (R), 51% the injury (I) and about 6% the failure (F) criteria [13]. In our study; pRIFLE was inversely correlated with urine output and proteinuria and eCCL was significantly improved after one month in both preterm and full-term. Oliguric AKI was found in (68.8%) and (72.7%) in fullterm and preterm neonates respectively in our studied groups. A similar percentage was found by Hossein

et al., 2014 who reported oliguria in 38 patients (77.6%) [29]. In similar studies, oliguric AKI was also more prevalent [24, 26].

Mathur et al. [24] indicated that AKI was developed in 26.0% of newborns from which 15% were oliguric. In 2007 for the first time Akcan-Arikan et al determined the modified pediatric RIFLE (pRIFLE) criteria and reported the pediatric patients with a maximum RIFLE score for R, I and F had an adjusted odds ratio for death of 2.9, 3 and 3 respectively [31].

Conclusion

Preterm neonates are more affected by AKI due to sepsis and liable for a faster recovery. Full-term neonates are more prone to asphyxia as a common cause of AKI and frequently have a progressive grade of nephropathy. Follow up data after 3 month are deficient in neonates especially in preterm neonates; hence further studies are required to follow the patients further.

Study limitations

Relatively larger studies are needed to assess the long term consequences for better configuration of the nature of AKI in neonates especially preterm ones.

List of abbreviations

AKI :Acute kidney injury
AKIN: Acute Kidney Injury Network
BUN : blood urea nitrogen
CBC:Complete blood count
eCCL : Estimated creatinine clearance
GFR : glomerular filtration rate
HCO3: bicarbonate

K: potassium
pRIFLE: Pediatric Risk ,Injury ,Failure, loss,
End stage renal disease
p: phosphorus
PDA : patent ductus arteriosus
RDS: respiratory distress syndrome

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Statements

Ethics approval and consent to participate

The local ethical committee permitted the study under the Helsinki declaration of Bioethics and its later amendments. Informed consent (written form) was

obtained from all participants or their caregivers.

Consent for publication

The contents and material of the manuscript have not been previously reported at any length or being considered for publishing elsewhere.

Availability of data and material

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