DOI: 10.7860/JCDR/2017/23398.9327

Original Article

Paediatrics Section

Clinical Profile and Outcome of Newborns with Acute Kidney Injury in a Level 3 Neonatal Unit in Western India

SATVIK CHAITANYA BANSAL[1], ARCHANA SOMASHEKHAR NIMBALKAR[2], AMIT R KUNGWANI[3], DIPEN VASUDEV PATEL[4], ANKUR RAJINDER SETHI[5], SOMASHEKHAR MARUTIRAO NIMBALKAR[6]

ABSTRACT

Introduction: Acute Kidney Injury (AKI) is a serious condition in neonatal care. It complicates the management necessitating the restrictive use of medications.

Aim: To evaluate clinical profile, identify associated and prognostic factors in newborns with AKI.

Materials and Methods: This was a case control study done between January 2008 to January 2010. Total 1745 newborns were admitted, of which 74 babies had AKI. It was defined as serum creatinine >1.5mg/dl. Control group was selected randomly from the hospital numbers of the newborns derived from the electronic registry with serum creatinine below 1.5 mg/dl. Demographic variables like birth weight, gender, gestational age, admission age, growth restriction, Apgar scores, electrolyte levels; and common clinical conditions like asphyxia, sepsis, meningitis, persistent pulmonary hypertension, Necrotizing Enterocolitis (NEC), mechanical ventilation, congenital heart disease; were compared amongst the two groups. Information was obtained from the admission register, admission files, labor register of obstetrics and gynaecology department and electronic registry. Chi square/independent sample t-test as applicable and logistic regression were used to establish an association of various factors and outcome with AKI.

Results: The incidence of AKI in our study was 4.24%. Demographic variables more common in AKI group were inborn (p=0.011), male gender (p=0.032), term gestation (p=0.001), Appropriate for gestational age (0.001), higher birth weight (p<0.001), full term (p<0.001), sepsis (p<0.001), NEC (p=0.042), low ApGAR scores at one minute (p=0.011) and five minute (p=0.003). However, on multivariate logistic regression only male gender [Odds Radio (OR)=2.84, Confidence Interval (CI)=1.12-7.21] and Sepsis (OR=14.46, CI=4.5-46.46) were associated with AKI. Respiratory distress syndrome was more prevalent in the control group (p<0.003). No need of mechanical ventilation and absence of shock, improved the survival.

Conclusions: AKI continues to be of clinical significance in neonatal intensive care. Further studies are needed to evaluate newer associations (like male gender and low APGAR scores).

Keywords: Acute Kidney Injury, Creatinine, Mortality, Sepsis, Shock

INTRODUCTION

Acute Kidney Injury (AKI) is defined as the inability of the kidneys to excrete nitrogenous waste products and maintain fluid and electrolyte homeostasis [1]. It is fairly common in newborn population and is a major contributor of neonatal mortality and morbidity [2,3]. The precise prevalence in the newborn population is still unknown, but the available data shows variable incidence of AKI in the Newborn Intensive Care Units (NICUs) around the globe; ranging from 6-24 % [4,5]. Different retrospective single center studies have shown higher incidence and significant impact on mortality of AKI in different subgroups of newborn population: Extremely Low Birth Weight (ELBW), Very Low Birth Weight (VLBW), asphyxia, sepsis, and sick term/near term newborns [6-9].

Use of serum creatinine is the simplest method for diagnosing AKI, but it is not the most accurate. During the first 14 days of life, plasma creatinine concentration drops from 1.1 mg/dl at birth (for preterm neonate: 1.3 mg/dl) to 0.4 mg/dl [10]. Unlike in pediatric and adult population, there is no consensus definition for diagnosing AKI in newborns. Even in the currently published large single center observational study did not compare various definitions of AKI for newborns [11].

The data on AKI from the Indian subcontinent is very limited. More importantly there are several gaps including the use of appropriate definition, risk factors, demographic profile and association with other co-morbidities which remain unanswered. We did a retrospective analysis delineating the epidemiology, associated factors and clinical profile of the newborns suffering from AKI.

MATERIALS AND METHODS

This case control study was conducted at Level III NICU of Shree Krishna Hospital (SKH) in Anand District of Gujarat, India. It is a referral center for government and private establishments of the surrounding districts. The NICU is a well-equipped, 20 bedded state of the art facility with average newborn: nurse ratio of 2:1. The hospital has an electronic registry system for all admissions and central diagnostic laboratory. Screening criteria of serum creatinine >1.5mg/dl was used to identify the study population from the online registry of the central diagnostic laboratory. The medical records of all the newborns admitted, between January 2008 to December 2010, who had the diagnosis of AKI were reviewed. These were compared with the medical records of 100 NICU admissions without AKI during the same period. Controls were randomly selected from the list of hospital numbers showing serum creatinine <1.5mg/dl. Matching was not possible as rest of the details were not available in the online registry.

The diagnosis of AKI was made on the basis of serum creatinine levels >1.5 mg/dl [12-14]. The diagnosis of Respiratory Distress Syndrome (RDS), Meconium aspiration Syndrome (MAS), birth asphyxia, clinical sepsis, Culture proven sepsis, NEC, hyperbilirubinemia were as per World Health Organization (WHO) [15]. The babies with AKI were managed by fluid adjustment, adjusting dose of medications based on renal function, peritoneal dialysis and supportive care as per need. Data regarding clinical and demography profile and laboratory investigations were obtained from the admission register of NICU, admission files, labor register of obstetrics and gynaecology department and electronic registry.

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. Approval was taken from the Institutional Human Research Ethics Committee (HREC) of Shree Krishna Hospital (SKH).

RESULTS

During the study period, out of 1745 NICU admissions (819 inborn; 926 outborn) in the hospital, 74 (4.24%) were diagnosed having AKI. Male: female ratio of 1.69:1 was observed in the admissions during the study period (1096 males; 649 females). Out of 74 neonates, 20 were inborn, 61 were male and 52 were born through vaginal delivery.

Distribution of various factors across both the groups are given in [Table/Fig-1a,b]. The difference between mean birth weight and gestational age of AKI and non-AKI babies was statistically significant (2.35 kg v/s 1.94 kg, p-value < 0.001; 37.44 weeks v/s 34.96 weeks, p=0.001).

The female gender was less associated with AKI (28.9% v/s 47.3 %; p=0.032). AKI was more common in term babies (54.1% v/s 28.4 %; p=0.001), outborn babies (p=0.011) and low ApGAR score at one minute and five minute Appropriate for Gestational Age (AGA) newborns had more chances of AKI than Small for Gestational Age (SGA)(53.7% v/s 28.9%; p=0.001). One baby out of 22 babies <1500 gm had AKI, 30 babies out of 84 babies between birth weights 1500 - <2500 gm had AKI, and 39 out of 59 babies with birth weight 2500 gm or more had AKI. Birth weights of four babies were not available. There were no cases of congenital structural anomaly of urinary tract in either group.

On multivariate logistic regression analysis, variables found significantly associated with AKI were male gender (OR=2.84, CI=1.12-7.21) and sepsis (OR=14.46, CI=4.5-46.46) [Table/Fig-2]. RDS, NEC were not included in analysis because of collinearity. ApGAR scores were not included because it reduced the sample size.

Out of those 74 neonates, 21(28.4%) were discharged, 38(51.3%) newborns went Discharge against Medical Advice (DAMA) and 15(20.3%) newborns died. Survival (discharged babies) amongst AKI was significantly associated with no requirement of mechanical ventilation and absence of shock. DAMA were considered as non survivors as being a tertiary care center, non-affordability with poor prognosis were the reasons for DAMA [Table/Fig-3].

		AKI			p-value
		Yes (n=74)	No(n=100)	Total	
Place of birth	Inborn	20	46	66	0.011
	Outborn	54	54	108	
Gender	Male	61	68	129	0.032
	Female	13	32	45	
Gestational age	Full term	53	45	98	0.001
	Pre term	21	53	74	
	NR*	0	2	2	
Growth restriction	SGA	22	54	76	0.001
	AGA	51	44	95	
	LGA	0	0	0	
	NR	1	2	3	
Outcome	Discharged	21	38	59	0.368
	DAMA	38	42	80	
	Expired	15	19	34	
	NR	0	1	1	
Sepsis	Yes	68	61	129	< 0.001
	No	6	37	43	
	NR	0	2	2	
Meconium aspiration syndrome	Yes	9	9	18	0.498
	No	65	91	156	
Asphyxia	Yes	24	20	44	0.062
	No	50	80	130	
Persistent pulmonary hypertension	Yes	2	0	2	0.098
	No	72	100	172	
Congenital heart disease	Yes	6	3	9	0.133
	No	68	97	165	
Congenital structural anomaly	Yes	4	3	7	0.425
	No	70	97	167	
Hyperbilirubinemia	Yes	6	3	9	0.133
	No	68	97	165	
Meningitis	Yes	5	3	8	0.242
	No	69	97	166	
Necrotizing enterocolitis	Yes	3	0	3	0.042
	No	71	100	171	
Respiratory distress syndrome	Yes	2	16	18	0.004
	No	72	84	156	
Birth weight (gm)	ELBW(<1000)	0	3	3	< 0.001
	VLBW(1000 to <1500)	1	18	19	
	LBW(1500 to <2500)	30	54	84	
	NBW (>2500)	39	20	59	
	NR	4	5	9	
Ventilation	Yes	40	44	89	0.211
	No	34	55	8	
	NR	0	1	1	

[Table/Fig-1a]: Factors associated with AKI.
NR: Not Recorded (not included in analysis), LGA: Large for gestational age, NBW: Normal birth weight

DISCUSSION

We provided a descriptive overview of AKI in newborns that were admitted to NICU. The incidence of AKI in our unit was 4.24 % during the study period. A previous study from India found the incidence of AKI in newborns to be 3.9 in 1000 live births and 34.5 in 1000 newborns admitted in the NICU [16]. A Turkish study using criterion of serum creatinine >1.5 mg/dl showed an incidence of AKI as 3.4% [12].

The wide variability of incidence of AKI in the available data from different units can be attributed to demographic characteristics of population studied, and secondly no consensus definition of AKI was used. There have been two recent studies in similar population (critically ill neonates); one using urine output with serum creatinine as the criteria and the other one only using serum creatinine. The incidence of AKI was 20% and 6.3% respectively; highlighting the importance of having fixed definitions of AKI [17,18].

Most of the published studies, especially older, have used arbitrary definitions of AKI; one frequently used is 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, which are not meant for neonatal population [13]. Recently, Jetton JG and Askenazi [19] proposed a new definition in 2012 [Table/Fig-4]. It graded severity of AKI using changes in serum creatinine and urine output. Subsequently, in April 2013 the group of neonatologists and paediatric nephrologists at the National Institute of Health (NIH) neonatal AKI workshop recommended the use of this definition [14]. However, the group stressed on the need of large multicenter cohort studies to validate its ability to predict clinical outcomes; Assessment of Worldwide Acute Kidney Injury Epidemiology in Neonates (AWAKEN) trial [20]. We could not use the new definition as we used retrospective data collection and could not find quantitatively documented urine output for the babies.

Various studies suggest that AKI is common in VLBW/ELBW newborns and is associated with poor prognosis [6,13,21]. Koralkar et al., reported incidence of AKI using modified KDIGO criteria to be 18% amongst 229 VLBW infants. They also reported higher mortality in the AKI group (p-value <0.001) [6]. Vishwanathan S et al., and Carmody JB et al., also reported similar findings [7,22]. A previous study from India showed that the percentage of babies

	AKI	N	Mean	SD	p-value
Weight on admission (Kg)	Yes	74	2.3539	0.53111	< 0.001
	No	97	1.9412	0.61106	
Gestational age (Weeks)	Yes	70	37.44	1.481	< 0.001
	No	98	34.96	3.395	
Age at admission (days)	Yes	73	5.22	6.961	0.085
	No	99	3.41	6.595	
Age at renal failure (days)	Yes	74	6.93	12.027	0.231
	No	4	14.50	15.927	
Sr Na	Yes	74	139.92	18.066	0.086
	No	45	135.07	6.604	
Sr K	Yes	74	5.9727	1.89711	0.004
	No	45	5.0476	1.21301	
Sr Ca	Yes	29	4.4224	11.98009	0.203
	No	25	1.3256	0.26785	
Apgar 1 min	Yes	19	3.89	1.853	0.011
	No	29	5.38	.1916	
Apgar 5 min	Yes	19	6.47	1.712	0.003
	No	29	7.86	1.356	

[Table/Fig-1b]: Factors associated with AKI (Independent sample t-test).
Sr=serum

AKI	Odds Ratio (OR)	p-value	95% Confidence Interval (CI)
Weight on admission	0.98	0.707	0.89, 1.08
Place of birth Inborn	0.49	0.084	0.22, 1.1
Gender Male	2.84	0.028	1.12, 7.21
Maturity preterm	0.51	0.168	0.2, 1.32
Growth restriction AGA	1.9	0.247	0.64, 5.59
Sepsis Yes	14.46	<0.001	4.5, 46.46
Asphyxia Yes	1.47	0.383	0.62, 3.52
Birth weight NBW	3.35	0.075	0.89, 12.67
Constant	0.02	<0.001	0.002, 0.12

[Table/Fig-2]: Multivariate logistic regression.

Factor	Non-Survivors	Survivors	p-value
Place of delivery Inborn Outborn	17 36	3 18	0.12
Sex Male Female	46 7	15 6	0.12
Mode of delivery cesarean Vaginal	17 36	5 16	0.77
Neonatal resuscitation Yes No	20 17	6 8	0.48
Birth weight ELBW VLBW LBW NBW	0 1 23 27	0 0 7 12	0.65
Maturity Preterm Full term	16 37	5 16	0.58
Appropriate for gestational age Small for gestational age	37 16	14 6	0.28
Mechanical ventilation Yes No	36 17	4 17	<0.001
Shock Yes No	39 14	3 18	<0.001
Sepsis Yes No	49 4	19 2	1
Asphyxia Yes No	17 36	7 14	0.92
Persistant pulmonary hypertension of newborn Yes No	1 52	1 20	0.49
Congenital heart disease Yes No	6 47	0 21	0.17
Congenital structural disease Yes No	3 50	1 20	1
Respiratory distress syndrome Yes No	1 52	1 20	0.49
Hyperbilirubinemia Yes No	2 51	3 18	1
Peritoneal dialysis Yes No	6 47	1 20	0.385

[Table/Fig-3]: Risk factors for mortality in babies with AKI.

Stage	Serum Creatinine	Urine output
0	No change in sCr or rise <0.3 mg/dl	> 1 ml/kg/h
1	sCr rise ≥0.3 mg/dl within 48 h or sCr rise ≥1.5–1.9× reference sCr within 7 days	> 0.5 ml/kg/h and ≤ 1 ml/kg/h
2	sCr rise ≥2–2.9× reference sCr	>0.3 ml/kg/h and ≤ 0.5 ml/kg/h
3	sCr rise ≥3× reference SCr or sCr ≥2.5 mg/dl or receipt of dialysis	≤ 0.3 ml/kg/h

[Table/Fig-4]: Modified KDIGO (Kidney Disease | Improving Global Outcomes) definition.
sCr- serum creatinine
Reference sCr will be defined as the lowest previous sCr value.

with birth weight of <2500 gm in AKI group was higher than in healthy neonates [9]. Interestingly, we observed higher incidence in term babies, this could be attributed to the fact that a major portion of full term neonates catered in our study were referred for sepsis or asphyxia, which also form a high risk group for AKI,

In current study, we observed predominance of male newborns (n= 61; 82.4%) in the AKI group, in accordance with previous study [23]. Another recent NICU study although reported higher prevalence of AKI among females [10].

As a result of unique neonatal renal physiology, maternal exposures and perinatal events can lead to AKI. Perinatal risk factors associated with neonatal AKI include intubation at birth, low ApGAR scores, low cord pH and asystole [13]. Recently Bolat F et al., confirmed association between intubation at birth and AKI in NICU population [24].

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 [10,18,24]. Another study from India by Mathur NB et al., shows that out of 200 newborns with sepsis, 26% developed AKI. The study also concluded that those with AKI had lower birth weight, and were prone for meningitis, disseminated intravascular coagulation and septic shock [9]. The newborns with sepsis are thought to be predisposed for AKI as a result of hypotension secondarily to sepsis and a direct damaging effect on renal microvasculature [13].

We found RDS to be more common in non-AKI group. This finding however, can also be due to more proportion of premature babies in the control group as compared to AKI group. The limited literature supports a positive association between AKI and RDS. A recent study by Momtaz HE et al., reported RDS as a third most common association with AKI (34.6%) after sepsis and dehydration [10].

We found significant association low ApGAR scores at one minute and five minutes with AKI. Surprisingly asphyxia did not show any association. Newborns with perinatal asphyxia have been associated

with a higher risk of AKI. As Apgar scores are dependent on asphyxia, low Apgar scores have also been shown to be associated with AKI [13]. Several previous studies have found birth asphyxia to be the most common cause of AKI of neonatal period [12,16,25]. Perinatal 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 for AKI [8,26]. Selewski DT et al., reported an incidence of 38% of AKI and Kaur S et al., reported 41.67%. The second study was conducted amongst neonates undergoing therapeutic hypothermia for perinatal asphyxia [8,26].

We observed an association of need of mechanical ventilation and shock with AKI for mortality. Several theories have been proposed in support of this hypothesis. Major mechanisms involved compromised renal blood flow because of hypercapnia or hypoxemia; and barotrauma induced pulmonary inflammatory reaction leading to secondary systemic inflammatory reaction [27,28]. A recent NICU study from Turkey also reported similar association [24].

In the study by Mathur NB et al., amongst various clinical factors, only shock was found to be as a significant predictor for mortality [9]. In the study by Tellier B et al., age <24 hours, underlying diseases, low urine output and multiorgan failure were shown as prognostic factors [29]. Another study by Agras PI [12], demonstrates that intrinsic AKI, need for dialysis and mechanical ventilation were associated with higher mortality rates and no significant correlation was found between mortality rate and prematurity, serum blood urea nitrozen and creatinine level and perinatal factors. In the current study peritoneal dialysis did not have any prognostic implication. This might be because of low sample size.

A recent study by Esfandier N et al., mentions hyaline membrane disease (HMD), using mechanical ventilation, the need to use surfactant, low Apgar score, high blood PCO_2, high serum creatinine level, and low birth weight being related to mortality [30].

LIMITATION

The inability to use the most accepted definition for neonatal AKI. Single center data reduces generalizability. The retrospective design of the study is a limitation that restricts our understanding to certain associations only. The control group was not matched with the cases; this may have restricted the clear delineation of associated factors. Absence of long term follow up precludes comments on delayed renal sequelae.

CONCLUSION

Male gender and sepsis came out as significant risk factors for AKI. Major factors associated with mortality in AKI were presence of shock and need for mechanical ventilation. As we move to gentler ventilation modes, better care, and smaller neonates, continued review of prospectively collected data are necessary to establish prognostic factors. Further studies are needed to assess the impact of low ApGAR scores on AKI.

ACKNOWLEDGEMENTS

Dr Amee Amin for English edits to the mansucript. Mr Ajay Phatak for Statistical Analysis. Dr Karishma Buch for assisting with data entry.

REFERENCES

[1] Martin RJ, Fanaroff AA, Walsh MC. Fanaroff and Martin's Neonatal- Perinatal Medicine - Diseases of the Fetus and Infant. 10th ed., Philadelphia, PA, 2015; pp 1683.
[2] Abdulkader RC, Liborio AB, Malheiros DM. Histological features of acute tubular necrosis in native kidneys and long-term renal function. Ren Fail. 2008;30:667-73.
[3] Coca SG, Singanamala S, Parikh CR. Chronic kidney disease after acute kidney injury: A systematic review and meta-analysis. Kidney International. 2012;81(5):442–48.
[4] Gouyon JB, Guignard JP. Management of acute renal failure in newborns. Pediatr Nephrol. 2000;14(10-11):1037-44.
[5] Drukker A, Guignard JP. Renal aspects of the term and preterm infant: A selective update. Curr Opinin Pediatr. 2002;14(2):175-82.
[6] Koralkar R, Ambalavanan N, Levitan EB, McGwin G, Goldstein S, Askenazi D. Acute kidney injury reduces survival in very low birth weight infants. Pediatr Res. 2011;69:354–58.
[7] Viswanathan S, Manyam B, Azhibekov T, Mhanna MJ. Risk factors associated with acute kidney injury in extremely low birth weight (ELBW) infants. Pediatr Nephrol. 2012;27:303–11.
[8] Selewski DT, Jordan BK, Askenazi DJ, Dechert RE, Sarkar S. Acute kidney injury in asphyxiated newborns treated with therapeutic hypothermia. J Pediatr. 2013;162:725–29.
[9] Mathur NB, Agarwal HS, Maria A. Acute renal failure in neonatal sepsis. Indian J Pediatr. 2006;73:499–502.
[10] Momtaz HE, Sabzehei MK, Rasuli B, Torabian S. The main etiologies of acute kidney injury in the newborns hospitalized in the neonatal intensive care unit. J Clin Neonatol. 2014;3:99-102.
[11] Sutherland SM, Byrnes JJ, Kothari M, Longhurst CA, Dutta S, Garcia P, et al. AKI in hospitalized children: Comparing the pRIFLE, AKIN, and KDIGO definitions. Clinical Journal of the American Society of Nephrology. 2015;10:554-61.
[12] Agras PI, Tarcan A, Baskin E, Cengiz N, Gürakan B, Saatci U. Acute renal failure in the neonatal period. Ren Fail. 2004;26:305-09.
[13] Selewski DT, Charlton JR, Jetton JG, Guillet R, Mhanna MJ, Askenazi DJ, et al. Neonatal Acute Kidney Injury. Pediatrics. 2015;136:463-73.
[14] Jetton JG, Askenazi DJ. Acute kidney injury in the neonate. Clin Perinatol. 2014;41:487-502.
[15] Working Definitions. South East Asia Regional Neonatal - Perinatal Database. World Health Organization (South-East Asia Region) Available from- http://www.newbornwhocc.org/pdf/database.pdf. Accessed - February 25, 2016.
[16] Aggarwal A, Kumar P, Chowdhary G, Majumdar S, Narang A. Evaluation of renal functions in asphyxiated newborns. J Trop Pediatr. 2005;51:295-99
[17] Bezerra CT, Vaz Cunha LC, Libório AB. Defining reduced urine output in neonatal ICU: importance for mortality and acute kidney injury classification. Nephrol Dial Transplant. 2013;28:901-09.
[18] Vachvanichsanong P, McNeil E, Dissaneevate S, Dissaneewate P, Chanvitan P, Janjindamai W. Neonatal acute kidney injury in a tertiary center in a developing country. Nephrol Dial Transplant. 2012; 27:973-77.
[19] Jetton JG, Askenazi DJ. Update on acute kidney injury in the neonate. Curr Opin Pediatr. 2012;24:191–96.
[20] Jetton JG, Guillet R, Askenazi DJ, Dill L, Jacobs J, Kent AL, et al. Neonatal Kidney Collaborative. Assessment of Worldwide Acute Kidney Injury Epidemiology in Neonates: Design of a Retrospective Cohort Study. Front Pediatr. 2016;19(4):68.
[21] Askenazi DJ, Griffin R, McGwin G, Carlo W, Ambalavanan N. Acute kidney injury is independently associated with mortality in very low birthweight infants: A matched case-control analysis. Pediatr Nephrol. 2009;24:991–97
[22] Carmody JB, Swanson JR, Rhone ET, Charlton JR. Recognition and reporting of AKI in very low birth weight infants. Clin J Am SocNephrol. 2014;9:2036–43.
[23] Mortazavi F, HosseinpourSakha S, Nejati N. Acute kidney failure in neonatal period. Iran J Kidney Dis. 2009;3:136–40.
[24] Bolat F, Comert S, Bolat G, Kucuk O, Can E, Bulbul A, et al. Acute kidney injury in a single neonatal intensive care unit in Turkey. World J Pediatr. 2013;9:323–29
[25] Gupta BD, Sharma P, Bagla J, Parakh M, Soni JP. Renal failure in asphyxiated neonates. Indian Pediatr. 2005;42:928–34.
[26] Kaur S, Jain S, Saha A, Chawla D, Parmar VR, Basu S, et al. Evaluation of glomerular and tubular renal function in neonates with birth asphyxia. Ann Trop Paediatr. 2011;31:129–34.
[27] Vieira Jr JM, Castro I, Curvello-Neto A, Demarzo S, Caruso P, Pastore Jr L, et al. Effect of acute kidney injury on weaning from mechanical ventilation in critically ill patients. Crit Care Med. 2007;35:184-91.
[28] Kuiper JW, Plötz FB, Groeneveld AJ, Haitsma JJ, Jothy S, Vaschetto R, et al. High tidal volume mechanical ventilation- induced lung injury in rats is greater after acid instillation than after sepsis-induced acute lung injury, but does not increase systemic inflammation: An experimental study. BMC Anesthesiol. 2011;11:26.
[29] Tellier B, Jouvet P, Hubert P, Niaudet P. Prognostic factors in neonatal acute renal failure. Annales de paediatrie. 1999;46:216-22.
[30] Esfandiar N, Mohkam M, Afjeii A, Kompani F, Shahrazad I, Naderi M, et al. Prognostic factors and mortality rate in neonates with acute renal injury in NICU. J Ped Nephrology. 2013;1:32-36.

PARTICULARS OF CONTRIBUTORS:
1. Senior Resident, Department of Paediatrics, Pramukhswami Medical College, Karamsad, Gujarat, India.
2. Associate Professor, Department of Physiology, Pramukhswami Medical College, Karamsad, Gujarat, India.
3. Senior Resident, Department of Paediatrics, Pramukhswami Medical College, Karamsad, Gujarat, India.
4. Associate Professor, Department of Paediatrics, Pramukhswami Medical College, Karamsad, Gujarat, India.
5. Assistant Professor, Department of Paediatrics, Pramukhswami Medical College, Karamsad, Gujarat, India.
6. Professor, Department of Paediatrics, Pramukhswami Medical College, Karamsad, Gujarat, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Somashekhar Marutirao Nimbalkar,
Department of Paediatrics, Pramukhswami Medical College, Karamsad-Anand-388325, Gujarat, India.
E-mail: somu_somu@yahoo.com

FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Aug 07, 2016**
Date of Peer Review: **Aug 31, 2016**
Date of Acceptance: **Oct 04, 2016**
Date of Publishing: **Mar 01, 2017**