How to Cite:
Sivuni, S., Venkata, P. C. B., Narayana C, S. ., & Chintha, L. P. (2022). A study on etiological profile of acute kidney failure in neonates admitted to neonatal intensive care unit at a tertiary care centre. *International Journal of Health Sciences*, *6*(S7), 1195–1214.
https://doi.org/10.53730/ijhs.v6nS7.11437

A study on etiological profile of acute kidney failure in neonates admitted to neonatal intensive care unit at a tertiary care centre

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***Abstract*---**Introduction: Acute kidney failure (AKF) / Acute kidney injury (AKI) Is defined as sudden deterioration in kidney function that leads to the accumulation of nitrogenous waste products (e.g.urea) and alters the regulation of extra cellular fluid volume, electrolyte and acid-base homeostasis. Renal dysfunction due to acute kidney failure is common in high-risk newborn babies and its incidence varies depending upon the criteria used for diagnosis. Materials and Methods: A 18 months study involving all those neonates who admitted in NICU, GGH Kurnool with manifestations of acute kidney injury. The study population comprised of all the neonates suspected or diagnosed with AKF were admitted as per the inclusion and exclusion criteria's mentioned earlier in NICU, Govt General Hospital, Kurnool between the time period of 18 months from 2019 to 2021. A detailed birth history involving resuscitation, detailed maternal history was recorded as precoded proforma was available. All the inborn deliveries were attended by the pediatric resident. Gestational age was assessed by Modified Ballard scoring system. The physical examination of the baby at the time of admission was recorded in the proforma. Sarnat and Sarnat classification was used for HIE. Results: Among 100 neonates 67 (67%) were male, 33 (33%) were female. Among 100 neonates 51 (51%) were Inborn, 49 (49%) were Out born. Among 100 neonates 22 (22%) were pre term, 78 (78%) were term. Among 100 neonates 48

Manuscript submitted: 9 March 2022, Manuscript revised: 18 May 2022, Accepted for publication: 27 June 2022

(48%) neonates are >2500 gms, 40 (40%) neonates are between 2000-2500 gms, 12 (12%) neonates are <2000gms. Among 100 neonates 20 (20%) have APGAR <5, 19 (19%) have APGAR 6-8, 61 (61%) have APGAR >8. Among 100 neonates with AKI 40% neonates had the history of birth asphyxia. Among 100 neonates with AKI 80 (80%) had evidence of sepsis. Among 100 neonates with AKI 3% had respiratory distress syndrome. Among 100 neonates with AKI 32% had evidence of shock. Among 100 neonates with AKI 24% had evidence of disseminated intravascular coagulation. Among 100 neonates with AKI 49% neonates needed mechanical ventilation. Among 100 neonates with AKI only 4% neonates had evidence of necrotising enterocolitis, 96% neonates had no evidence of necrotising enterocolitis. Conclusion: Though various studies showed varied incidence of AKI in term and preterm neonates, present study showed AKI more commonly in term neonates when compared to preterm neonates. A number of lab parameters proposed to help to differentiate pre renal failure from renal failure but none of them has therapeutic advantage as well as diagnostic reliability of fluid challenge and diuretics. There is need for many further studies to define and enumerate the use of these indices, and their use in management of AKI. Acute kidney failure is fatal yet reversible in early phase, needs prompt and accurate monitoring, diagnosis for good recovery and prevention of complications.

***Keywords*---** Acute kidney failure, NICU, Gestational age.

Introduction

Acute kidney failure (AKF) / Acute kidney injury (AKI) Is defined as sudden deterioration in kidney function that leads to the accumulation of nitrogenous waste products (e.g.urea) and alters the regulation of extra cellular fluid volume, electrolyte and acid-base homeostasis. Renal dysfunction due to acute kidney failure is common in high-risk newborn babies an d its incidence varies depending upon the criteria used for diagnosis.[1] Although there are varied criteria to define acute kidney failure in neonates, more frequently used definition is serum creatinine level of more than 1.5 mg/dL or rise by 0.3 mg/dL /day.[1] Oliguric acute kidney failure is characterized by a urine flow rate of less than 1 ml/kg/hour, whereas in non oliguric acute kidney injury flow rate is maintained above this level.2 In 2004, the acute dialysis quality initiative (ADQI) proposed a classification for AKF called Risk, Injury, Failure, Loss, End-stage kidney disease (RIFLE) for a consistent definition of AKI.[3]

In high risk neonates, AKI is more common and occurs mainly in the first week of life, mainly due to hypovolemia, hypertension, ischemia, and less commonly due to primary kidney disease.[4] More over the confirmation of AKI in the first place in neonatal period is cumbersome due to immature renal physiology and unreliability of dialysis.4 In children, Akcan-Arikan et al,[5] suggested a modified paediatric RIFLE (pRIFLE) classification with lower cut-off of serum creatinine to achieve the failure category, thereby reflecting the fact that children have lower baseline serum creatinine. Acute kidney failure is an under recognized morbidity in neonates, the

incidence is not accurate due to the absence of single definition of AKF in neonates and because prior studies have shown variations in screening for AKI with serum creatinine and urine output. AKF can cause chronic kidney disease (CKD), and preterm neonates with AKF maybe at higher risk for long-term renal issues. AKF in neonates is most of the times multifactorial and may be due to prenatal, perinatal, or postnatal insults individually or in a combination.[6]

Aims and Objectives

1. To find out the etiological factors of acute kidney failure in neonates
2. To identify the maternal risk factors for acute kidney failure in neonates
3. To study the incidence of different types of acute kidney failure in neonates

Materials and Methods

Source of data: A 18 months study involving all those neonates who admitted in NICU, GGH Kurnool with manifestations of acute kidney injury.

Methods of collection of data

Inclusion Criteria:

1. Gestational age >28weeks, birth weight >1000 gms
2. Neonates brought with complaints of oliguria, anuria after first 48 hours of life.
3. Neonates admitted in NICU with evidence of renal failure and maternal risk factors.

Exclusion Criteria

1. Neonates whose attenders are not willing to give consent
2. Neonates who brought with complaint of not passed in urine in first 48 hours of life.
3. Neonates with ambiguous genitalia, genital malformations.

Study Design: Prospective observational study

Sample Size: Sample size was calculated using open Epi software Version 2.3.1 . Based on the study conducted by Hossain Emad Momtaz et al[48] incidence of sepsis among AKF/AKI Neonates in NICU is 77.5% with 95% confidence limit and 11% relative precision.

Statistical Analysis: Data was entered in the Microsoft excel and analysed using SPSS version 21.results were expressed in terms of no of percentage, quantitative data expressed as mean and standard deviation. Chi- square test used to test association between variables. P values <0.05 are considered significant at 95% confidence interval.

Methods of collection of data

The study population comprised of all the neonates suspected or diagnosed with AKF were admitted as per the inclusion and exclusion criteria's mentioned earlier

in NICU, Govt General Hospital, Kurnool between the time period of 18 months from 2019 to 2021.

A detailed birth history involving resuscitation, detailed maternal history was recorded as precoded proforma was available. All the inborn deliveries were attended by the pediatric resident. Gestational age was assessed by Modified Ballard scoring system. The physical examination of the baby at the time of admission was recorded in the proforma. Sarnat and Sarnat classification was used for HIE.

Basic investigations like complete blood picture and septic screen were done at the time of admission. Subsequent follow up of the baby regarding the analysis of the renal system involvement were done and the samples for the estimation of serum electrolytes, creatinine, blood gas analysis and blood urea were collected in heparinized containers from the peripheral vein at 48-72 hours and at subsequent intervals. Samples were immediately sent to the lab. Blood cultures were drawn from the peripheral vein under aseptic precautions before starting antibiotics whenever suspecting the sepsis.

Neonates subsequently followed for renal involvement by measuring urine output with the help of paediatric urine collection bag with adhesive tape fixedaround the external genitalia of the baby or by weighing the diapers at fixed intervals (every 6hours). Abdominal ultrasonography was done for renal anomalies by the radiologist if suspected. Daily follow up of babies with urine output, renal parameters,electrolytes were done till the discharge or death.
Oliguric AKI confirmed if following criteria present

1. Urine output less than 1ml/kg/hr for 24 hrs,
2. Blood urea more than 20mg/dl,
3. Serum creatinine rise>0.3 mg/dL or value >1.5mg/dL .

Non oliguric AKF was confirmed if following criteria present

1. Urine output more than 1 ml/kg/hr for 24 hours,
2. Blood urea more than 20mg/dL,
3. Sr.creatinine rise >0.3mg/dL or value> 1.5mg/dL .

Whenever baby had oliguria a fluid challenge with 20 ml/kg with normal salinewas given. If there was no improvement in urine output, diuretic furosemide 1mg/kg/dose IV given provided adequate hydration was maintained or another fluid challenge was given. If still urine output did not improve, fluids were restricted. Antibiotics were used for treatment of sepsis. The electrolyte disturbances were treated as per standard protocol.

Investigations

1. Basic hematological investigations including Hb%, total leukocyte count, platelet count, blood grouping.
2. C-reactive protein
3. Blood culture and sensitivity, urine culture and sensitivity.
4. Urine albumin, sugar, microscopic examination
5. Serum electrolytes
6. Serum creatinine, blood urea
7. Ultrasound abdomen for renal anomalies
8. Arterial blood gas analysis
9. Serum albumin

Results

Table 1
Sex Distribution of Neonates with AKF

Sex		**No. of neonates**	**Percentage (%)**
Male		67	67.00%
Female		33	33.00%
Total		100	100.00%

Table shows among 100 neonates 67 (67%) were male, 33 (33%) were female

Graph 1:- Pie Diagram Showing Sex Distribution of Neonates with AKF

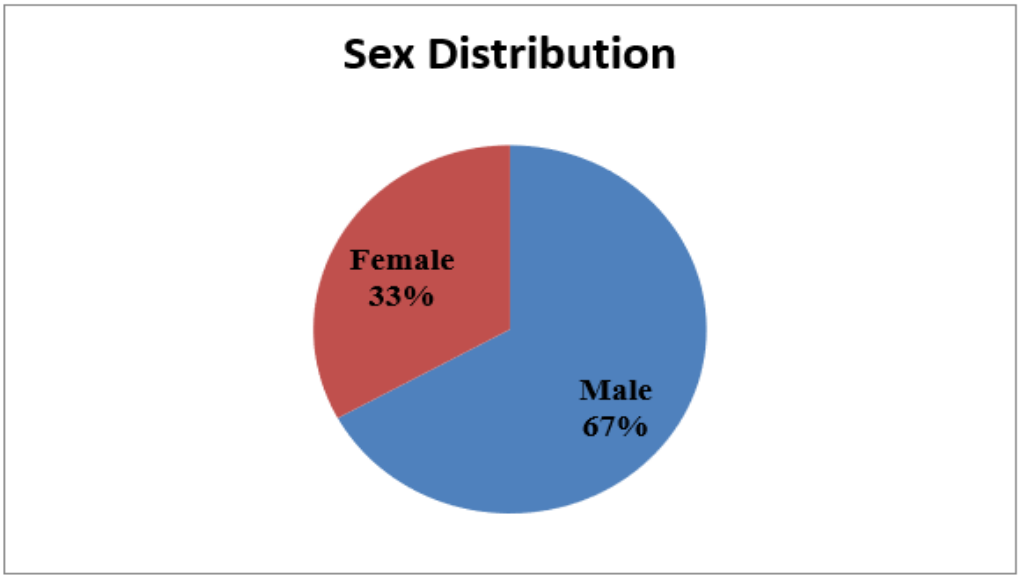

Table 2

Birth Status of Neonates with AKF

	No. of neonates	percentage
IN BORN	51	51.00%
OUT BORN	49	49.00%
Total	100	100.00%

Among 100 neonates 51 (51%) were Inborn, 49 (49%) were Out born.

Graph 2:- Simple Bar Diagram Showing Birth Status of Neonates with

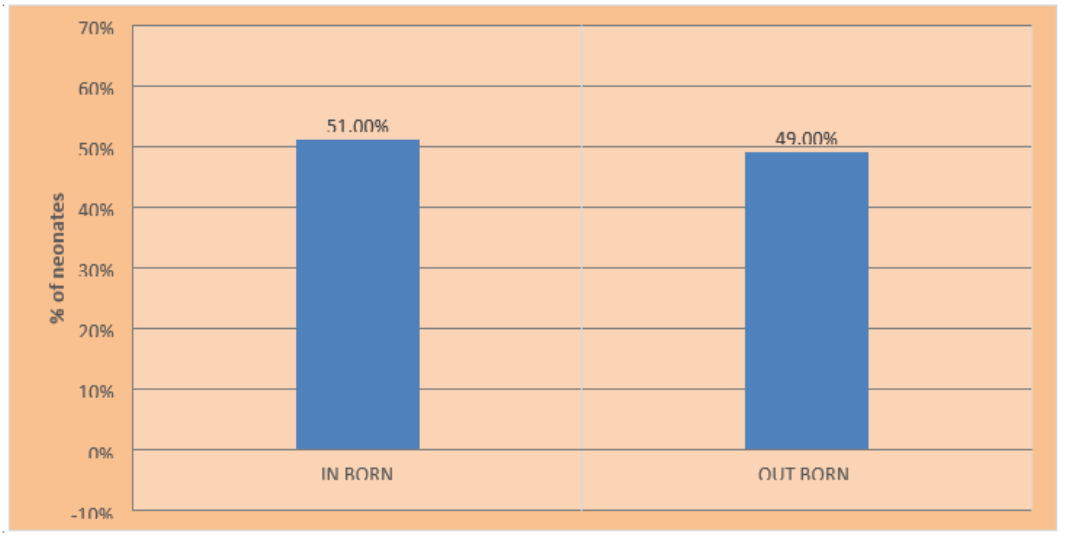

Table 3
Gestational Age of Neonates with AKF

	No. of neonates	percentage
PRETERM	22	22.00%
TERM	78	78.00%
Total	100	100.00%

Among 100 neonates 22 (22%) were pre term, 78 (78%) were term.

Graph 3: Simple Bar Diagram Showing Gestational Age of neonates with AKF

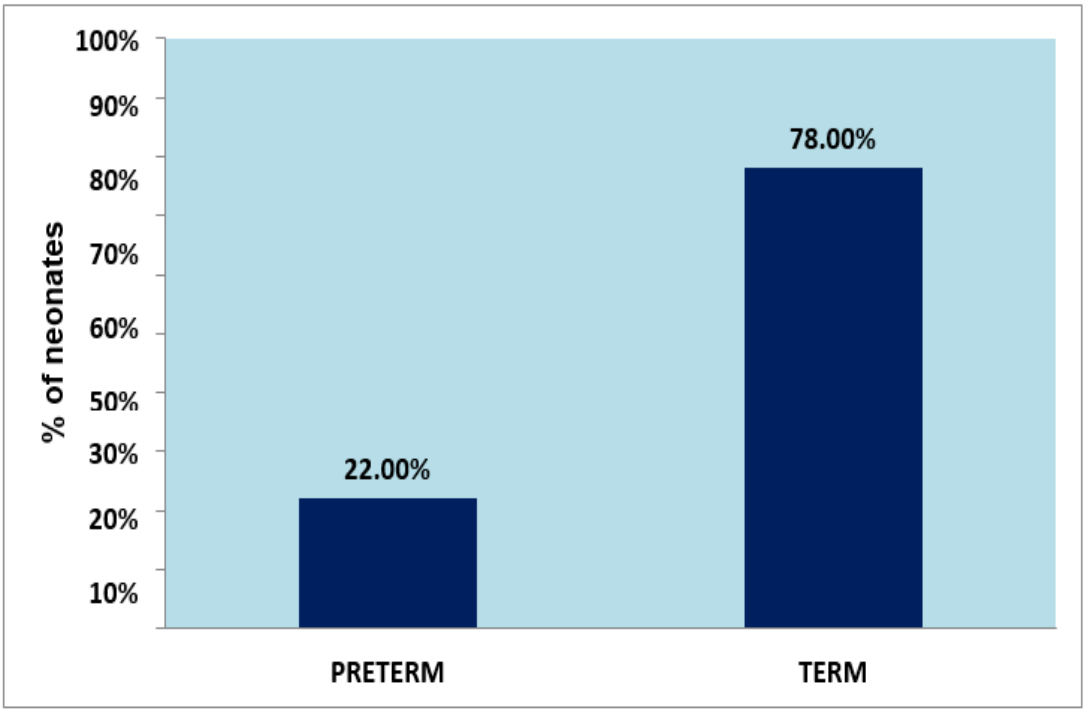

Table 4
Birth Weight of Neonates With AKF

	No. of neonates	**%**
< 2000 grams	12	12.00%
2000-2500 grams	40	40.00%
>2500 grams	48	48.00%
Total	100	100.00%

Among 100 neonates 48 (48%) neonates are >2500 gms, 40 (40%) neonates are between 2000-2500 gms, 12 (12%) neonates are <2000gms.

Table 5
APGAR score of neonates with AKF

	No. of neonates	**%**
< 5	20	20.00%
6-8	19	19.00%
9 -10	61	61.00%
Total	100	100.00%

Among 100 neonates 20 (20%) have APGAR <5, 19 (19%) have APGAR 6-8, 6161%) have APGAR >8.

Table 6
Neonatal risk factors for neonatal acute kidney failure

		Number of neonates	%
Birth Asphyxia	Yes	40	40.00%
	No	60	60.00%
RDS	Yes	3	3.00%
	No	97	97.00%
FEVER	Yes	50	50.00%
	No	50	50.00%
SEPSIS	Yes	80	80.00%
	No	20	20.00%
NEC	Yes	6	6.00%
	No	94	94.00%
DRUGS	Yes	5	5.00%
	No	95	95.00%
SHOCK	Yes	32	32.00%
	No	68	68.00%

EDEMA	Yes	15	15.00%
	No	85	85.00%
DIC	Yes	24	24.00%
	No	76	76.00%
MV	Yes	49	49.00%
	No	51	51.00%
CHD	Yes	4	4.00%
	No	96	96.00%

Among 100 neonates with AKI 40% neonates had the history of birth asphyxia. Among 100 neonates with AKI 80 (80%) had evidence of sepsis. Among 100 neonates with AKI 3% had respiratory distress syndrome. Among 100 neonates with AKI 32% had evidence of shock. Among 100 neonates with AKI 24% had evidence of disseminated intravascular coagulation. Among 100 neonates with AKI 49% neonates needed mechanical ventilation. Among 100 neonates with AKI only 4% neonates had evidence of necrotising enterocolitis, 96% neonates had no evidence of necrotising enterocolitis.

Table 7
Maternal Risk Factors for Neonatal Acute Kidney Failure

Risk Factor		**Number of neonates**	**%**
GDM	Yes	3	3.00%
	No	97	97.00%
Pregnancy induced hypertension	Yes	7	7.00%
	No	93	93.00%
Preeclampsia	yes	3	3%
	No	97	97%
	yes	2	2%

Eclampsia	No	98	98%
Amniotic fluid	OH	3	3.00%
	PH	1	1.00%
	No	96	96.00%
FEVER	Yes	50	50.00%
	No	50	50.00%
Seizure disorder	yes	2	2%
	No	98	98%

Above table shows distribution of various maternal risk factors in neonatal AKI.3% mothers had GDM, 7% had PIH, 50% had history of fever, 3% had history of oligohydramnios. Among the 100 neonates 3 (3%) have oligohydromnios, 1(1%) havepolyhydromnios, 96 (96%) have normal amniotic fluid.

Table 8
Urine Output of Neonates with AKF

URINE OUTPUT	**NO OF NEONATES**	**PERCENTAGE**
NORMAL	**67**	**67%**
OLIGURIA	**26**	**26%**
ANURIA	**7**	**7%**

Among100 neonates with AKI 67% babies had normal urine output, 26% babies have oliguria, only 7% babies had anuria.

Table 9
Types of AKF In Neonates

TYPE OF AKI	**NO OF NEONATES**	**%**
PRE RENAL	**96**	96
RENAL	**3**	3
POST RENAL	**1**	1

Among100 neonates with AKI 96% have pre renal type of AKI, 3% had renaltype of AKI,1% had post renal type of AKI.

Table 10
Lab Investations in Neonates with AKF

		Number of neonates	%
HTN	Hypertension	3	3.00%
	Normal	97	97.00%
Haemoglobin	Anaemia	33	33.00%
	Normal	67	67.00%
PLT	Thrombocytopaenia	66	66.00%
	Normal	34	34.00%
S.CREAT	Raised	100	100.00%
	Normal	0	0.00%
Blood Urea	Raised	100	100.00%
	Normal	0	0.00%
SODIUM	Decreased	27	27.00%
	Normal	73	73.00%
POTASSIUM	Decreased	3	3.00%
	Increased	17	17.00%
	Normal	80	80.00%
Albumin	Decreased	12	12.00%
	Normal	88	88.00%
ABG[MA]	Yes	27	27.00%
	No	73	73.00%
USG	Not done	84	84.00%
	Normal	16	16.00%
Peritoneal Dialysis	Yes	7	7.00%
	No	93	93.00%

Among 100 neonates with AKI 3% had hypokalemia, 17% had hyperkalemia,

80% had normal potassium values. Among 100 neonates with AKI 67% neonates had anaemia, 66% neonateshad thrombocytopenia.

Table 11
Significant Risk Factors in Neonates with AKF

		OUTCOME				n	Chi square test/yates corrected chi square test	Odds ratio (95%CI)
		Death		Improved				
		n	%	n	%			
Gender	Male	21	31.34%	46	68.66%	67	**χ^2=4.35 p=0.05* (S)**	**3.4 (1.1-10.9)**
	Female	4	12.12%	29	87.88%	33		
Fever	Yes	17	34.00%	33	66.00%	50	**χ^2=4.32 p=0.05* (S)**	**2.7 (1.1- 7.0)**
	No	8	16.00%	42	84.00%	50		
Mechanical Ventilation	Yes	18	36.73%	31	63.27%	49	**χ^2=7.06 p=0.01** (S)**	**3.7 (1.4- 9.8)**
	No	7	13.73%	44	86.27%	51		
5 min APGAR	≤ 5	9	45.00%	11	55.00%	20	**χ^2=5.33 p=0.05* (S)**	**3.3 (1.2- 9.2)**
	>5	16	20.00%	64	80.00%	80		
Album*in*	Decreased	6	50.00%	6	50.00%	12	**χ^2=4.54 p=0.05* (S)**	**3.6 (1.1- 7.0)**
	Normal	19	21.59%	69	78.41%	88		

The above table shows significant factors for leading to deaths among Acute kidney failure in neonates. Significance are analysed using chi square test

and risk are given odds ratio with 95% confidence interval.

Table 12
Outcome of Neonates with Acute Kidney Failure

Out Come	**No. of neonates**	**%**
Improved	75	75.00%
Death	25	25.00%
Total	100	100.00%

Among 100 neonates with AKI 75 (75%) neonates improved, 25 (25%) neonates expired

Table 13
Association between Outcome and Risk Factors In neonatal Acute Kidney Failure

		OUTCOME				n	Chi square test/yates corrected chi square test
		Improved		Death			
		n	%	n	%		
Gender	Male	46	68.66%	21	31.34%	67	**□2=4.35 p=0.05*(S)**
	Female	29	87.88%	4	12.12%	33	
IN/OUT	In	40	78.43%	11	21.57%	51	□2=0.65 p=0.42 (NS)
	Out	35	71.43%	14	28.57%	49	
TERM/PT	Preterm	18	81.82%	4	18.18%	22	□2=0.70 p=0.40 (NS)
	Term	57	73.08%	21	26.92%	78	
Birth weight	< 2000 gms	8	66.67%	4	33.33%	12	□2=0.50 p=0.48 (NS)
	>2500 grams	67	76.14%	21	23.86%	88	
GDM	Yes	2	66.67%	1	33.33%	3	□2=0.11 p=0.74 (NS)

	No	73	75.26%	24	24.74%	97	
Pregnancy induced hypertension	Yes	4	57.14%	3	42.86%	7	χ^2=0.46 p=0.50 (NS)
	No	71	76.34%	22	23.66%	93	
Amniotic fluid	OH/PH	4	100.00%	0	0.00%	4	χ^2=0.34 p=0.55 (NS)
	No	71	73.96%	25	26.04%	96	
Birth Asphyxia	Yes	28	70.00%	12	30.00%	40	χ^2=0.89 p=0.35 (NS)
	No	47	78.33%	13	21.67%	60	
HIE	Yes	3	100.00%	0	0.00%	3	χ^2=1.03 p=0.31 (NS)
	No	72	74.23%	25	25.77%	97	
RDS	Yes	1	33.33%	2	66.67%	3	χ^2=1.03 p=0.31 (NS)
	No	74	76.29%	23	23.71%	97	
FEVER	Yes	33	66.00%	17	34.00%	50	**χ^2=4.32 p=0.05*(S)**
	No	42	84.00%	8	16.00%	50	
SEPSIS	Yes	59	73.75%	21	26.25%	80	χ^2=0.33 p=0.56 (NS)
	No	16	80.00%	4	20.00%	20	
NEC	Yes	3	50.00%	3	50.00%	6	χ^2=0.95 p=0.33 (NS)
	No	72	76.60%	22	23.40%	94	
DRUGS	Yes	5	100.00%	0	0.00%	5	χ^2=0.63 p=0.42 (NS)
	No	70	73.68%	25	26.32%	95	
SHOCK	Yes	27	84.38%	5	15.63%	32	χ^2=2.20 p=0.14 (NS)
	No	48	70.59%	20	29.41%	68	
EDEMA	Yes	10	66.67%	5	33.33%	15	χ^2=0.65 p=0.42 (NS)

	No	65	76.47%	20	23.53%	85	
DIC	Yes	21	87.50%	3	12.50%	24	χ^2=2.63 p=0.10 (NS)
	No	54	71.05%	22	28.95%	76	
Mechanical ventilation	Yes	31	63.27%	18	36.73%	49	**χ^2=7.05 p=0.01** (S)**
	No	44	86.27%	7	13.73%	51	
CHD	Yes	4	100.00%	0	0.00%	4	χ^2=0.34 p=0.54 (NS)
	No	71	73.96%	25	26.04%	96	
5MIN APGAR	≤ 5	11	55.00%	9	45.00%	20	**χ^2=5.33 p=0.05*(S)**
	>5	64	80.00%	16	20.00%	80	

Male babies, fever having babies, Mechanical ventilation babies, ≤ 5 MIN APGAR babies are having more death than others. It was confirmed using chi square test

Among 100 neonates 31.34% of male neonates and 12.12% female neonates expired. Among 100 neonates with AKI, 34% of neonates with dehydration fever expired, 16% neonates with out dehydration fever expired.

Among 100 neonates with AKI, 36.73% of neonates kept on mechanicalventilation expired, where as 13.73% neonates expired among non ventilated babies.

Among 100 neonates with AKI, among the babies with <5 Apgar 45% expired where as only 20% neonates expired among those who have >5 APGAR.

Among 100 neonates among AKI, 50% of neonates with hypoaalbuminemia expired where as 21.59% neonates expired among those who have normal albumin values.

Table 18
Association between outcome and investigations IN AKF

		OUTCOME				n	Chi square test/yates corrected chi square test
		Improved		Death			
		n	%	n	%		
Haemoglobin	Anaemia	26	78.79%	7	21.21%	33	χ^2=0.37 p=0.54 (NS)

	Normal	49	73.13%	18	26.87%	67	
PLT	Decreased	47	71.21%	19	28.79%	66	□2=1.48 p=0.22 (NS)
	Normal	28	82.35%	6	17.65%	34	
S.CREAT	Raised	75	75.00%	25	25.00%	100	□2=0.00 p=1.00 (NS)
	Normal	0	0.00%	0	0.00%	0	
Blood Urea	Raised	75	75.00%	25	25.00%	100	□2=0.00 p=1.00 (NS)
	Normal	0	0.00%	0	0.00%	0	
Urine output	Anuria /Oliguria	25	75.75%	8	24.25%	33	□2=0.01p=0.90 (NS)
	Normal	50	74.62%	17	25.38%	67	
SODIUM	Decreased	19	70.37%	8	29.63%	27	□2=0.42 p=0.52 (NS)
	Normal	56	76.71%	17	23.29%	73	
POTASSIUM	Decreased /Increased	14	70.00%	6	30.00%	20	□2=0.33 p=0.56 (NS)
	Normal	61	76.25%	19	23.75%	80	
Albumin	Decreased	6	50.00%	6	50.00%	12	**□2=4.54 p=0.05* (S)**
	Normal	69	78.41%	19	21.59%	88	
ABG[MA]	Yes	18	66.67%	9	33.33%	27	□2=1.37 p=0.24 (NS)
	No	57	78.08%	16	21.92%	73	
USG	Not done	62	73.81%	22	26.19%	84	□2=0.40 p=0.53 (NS)
	Normal	13	81.25%	3	18.75%	16	
PeritonialDialysis	Yes	4	57.14%	3	42.86%	7	□2=0.46 p=0.50 (NS)
	No	71	76.34%	22	23.66%	93	

Decreased albumin is significantly associated with more number of deaths. Itwas confirmed using chi square test.

Discussion

The neonatal kidney is particularly vulnerable to the effects of hypo perfusion since the renal vascular resistance and plasma renin activity are high. Consequently renal blood flow is proportionately more reduced in neonates.[7]

Acute tubular necrosis has many parallels with the physiologic characteristics of neonatal kidney- the low GFR, decreased inter cortical perfusion, decreased proximal reabsorption of sodium and increased plasma renin activity. The neonatal kidney has been described as **'half way to acute renal failure'.**[8]

Many malformations and prenatal, perinatal and postnatal events may result in AKF.

The demographic distribution in neonates revealed that of the 100 neonates with AKI, male neonates 67 (67%) were commonly affected when compared to female neonates 33 (33%).

Mortazavi et al[7] reported male, female ratio of 2:1 and Airede et al, who reported male to female ratio of 3.3:1 in neonates with AKF, this was found similarly in present study with male to female ratio of 2.1:1.

The reason for such high incidence among males was primarily attributed to the higher number of male neonates admission during the period of study and in partly to the general population attitude of seeking more active care for male neonates as compared to female neonates.

Of the 100 neonates with AKI 51 are out born, 49 are inborn. This study revealed high incidence of AKF in out born neonates 51 (51%) when compared to inborn cases 49 (49%), the reason may be due to fact that GGH, Kurnool is a tertiary care hospital and most of neonates were referred and out born admission.

Neonatal Risk Factors

Present study shows 78 (78%) neonates are term babies, whereas 22 (22%) neonates are preterm. Mortazavi et al[7] reported that AKI associated with 70.2% of term neonates, 29.8% in preterm neonates. Present study results are similar to this study. This can be explained by the fact that term perinatal asphyxia and sepsis being the strong predictive factors for neonatal acute kidney failure when compared to preterm gestation.

In present study of the 100 neonates 12 are of less than 2000 gms, 40 neonates are of 2000- 2500 grams, 48 neonates are of more than 2500 gms weight there is varied distribution of AKF in neonates in different birth weights, more incidence in neonates having birth weight >2500 gms, followed by 2000-2500 gms, and <2000gms.

In current study highest incidence of AKI found in babies with birth weight

>2500gms, this is attributed primarily to increased association of neonatal acute kidney failure with term risk factors like perinatal asphyxia, poor APGAR , neonatal sepsis, the more no of admissions of babies in particular birth weight range.[9]

In a study conducted by shalaby et al it was reported that incidence more in

smaller and sicker infants. In a study carried out by Unni et al found that highest incidence was in birth weight 1500-1999gms, similarly Griffin et al also found maximum prevalence of AKI between 1000-2000gms birth weights due to perinatal asphyxia, however the incidence related to associated factors like septicemia, peri natal asphyxia rather than low birth weight.

In present study 61% neonates have APGAR 9 or 10, where 19% neonates have APGAR 6-8, 20%neonates have APGAR of <5. Apart from increased AKI incidence in perinatal asphyxia, there is stronger association of incidence of AKI with poor APGAR score in the first 5 minutes of life. As APGAR score indirectly reflects perfusion of several vital organs in the body, it is good indicator of renal perfusion in the perinatal period and subsequent renal dysfunction.[10]

Several studies showed varied importance of various risk factors in neonatal AKI. In present study 40% neonates with AKI had history of perinatal asphyxia. 60% neonates does not have history, evidence of perinatal asphyxia.

Conclusion

Neonatal AKI/AKF emerged as new concern for neonatologists and paediatricians, reason being improved quality of care leading to the prolonged survival. Recovery rate in present study is 75% which implies importance of early and antenatal detection of renal anomalies and close monitoring of renal parameters in sick neonates.

Etiological factors identified for neonatal AKI/AKF in this study was maternal factors like pregnancy induced hypertension, GDM, oligohydromnios, maternal sepsis during or before the labor.

Neonatal factors observed responsible for AKI were sepsis, perinatal asphyxia, hypoxic ischemic encephalopathy, dehydration fever, necrotizing enterocolitis, nephrotoxic drug administration, hemodynamic instabilities such as shock, disseminated intra vascular coagulation, respiratory distress syndrome, need of mechanical ventilation.

All urine output variations normal output, oliguria, anuria were found to be associated with acute kidney failure in neonates.

All three types of acute kidney injury pre renal, renal, post renal found in neonates with pre renal being the most common type of acute kidney injury. Reason being perinatal asphyxia and sepsis are most important etiologies.
Though various studies showed varied incidence of AKI in term and preterm neonates, present study showed AKI more commonly in term neonates when compared to preterm neonates.

A number of lab parameters proposed to help to differentiate pre renal failure from renal failure but none of them has therapeutic advantage as well as diagnostic reliability of fluid challenge and diuretics. There is need for many further studies to define and enumerate the use of these indices, and their use in management of AKI.

Acute kidney failure is fatal yet reversible in early phase, needs prompt and accurate monitoring, diagnosis for good recovery and prevention of complications. The following conclusions can be drawn from the present study:

Limitations

Though detailed observations conducted there are few limitations for present study.

- As the study centre is a tertiary care centre and all the neonates referred are sick and high risk cases. So present study findings are not representative of accurate prevalence of etiological of AKF in community.
- Early predictors of acute kidney failure like nGAL, KIM, IL-18 could not beassessed due to u availability.
- Only the immediate outcome of acute kidney failure was studied in the form of survival versus mortality. Neonates who progressed to chronic kidney disease could not be followed up, also included under the category of improved neonates.
- Peritoneal dialysis could not be performed due to institutional constraints.

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