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Renal function estimation in critically ill newborns in Erbil.

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Abstract

Background and objectives: Acute kidney injury is a common in critically ill neonates all over the world, characterized by an acute decline in renal function, the result of which ranges from minimal alteration in serum creatinine to an a uric renal failure our objective is to estimate renal function and aetiologies of renal function deteriorated in critically ill neonates. **Methods:** This study was done in Rapareen teaching hospital in the neonatal care unit from the period of first January 2015 until first of January 2016. Two hundred cases collected including term and preterm babies, term baby defined as any labour 37 weeks, preterm labour defined as any labour between 28 weeks and 37 weeks. **Results:** The total number of the studied sample was 200 neonates. The mean age ± SD of the sample was 8.6 ± 9.9 days, ranging from one to 30 days. The median was 3 days. Two thirds 64.5% of the sample were less than seven days old. More than half 58.5% of the sample was males, and 57.5% of the neonates born by Cesarean section. Acute kidney injury was associated with significantly longer intensive care unit and hospital stay, making it a major burden on the healthcare system. **Conclusions:** Acute kidney injury in critically ill pediatric was more common in female neonate than males' neonate; high blood urea was more common in female than in male. Lower age and cardiac disease were independent risk factors for acute kidney injury.

Keywords: Acute kidney injury; Blood urea; Serum creatinine.

Introduction

Acute kidney injury (AKI) is defined as the rapid elevation in the concentration of blood urea nitrogen (BUN), creatinine, and other cellular waste products in the blood resulting from diminished glomerular filtration rate (GFR) in the kidney[1]. Frequently, it involves abnormal tubular function, including reduced sodium resorption and increased loss of bicarbnate, as well as diminished excretion of water. Unlike in older patients, it is difficult to define neonatal AKI on the basis of a specific serum creatinine value, in part because concentrations measured immediately after birth reflect the maternal rather than infant's renal function. Normal creatinine values are dependent on gestational age, with higher values normally seen in more immature babies, and on postnatal age. In term babies, the concentration normally rises somewhat in the first 24 to 36 hours after birth, subsequently decreasing and stabilizing at about 0.4 mg/dL (35.4 mcmol/L) by 5 days of age[2]. In preterm infants, the peak value occursbetween 2 and 3 days after birth, and stabilization typically is delayed until about 6 days of age. It is important to consider these normal patterns for creatinine in assessing the meaning of any particular value, which makes it very difficult to use a single value to diagnose renal failure, except that a clearly elevated value beyond the normal range indicates decreased glomerular function. Often, acute kidney injury (AKI) can be diagnosed by monitoring changes in the creatinine value because during a period of intrinsic renal failure, it usually increases between 0.5 and 1.0 mg/dL (44.2 and 88.4 mcmol/L) per day[3]. Urine output is another key indicator of renal function. Commonly, AKI is suspected when oliguria is present, defined as a period during which urine output is less than 0.5 mL/kg per hour. This is not a universal finding, however, and the urine output remains normal or even may be increased during acute kidney injury (AKI) in many patients[4- 5]. The underlying process leading to renal failure may be categorized as: 1) prerenal, due to a diminution of systemic circulation to the kidney as a result of a contraction in the intravascular volume or failure of the cardiac output; 2) renal, which results from damage or necrosis of the parenchyma of the kidney; or 3) post renal due to distal obstruction to urine flow in

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the kidney or collecting system. These distinctions are some what artificial, and some processes include elements from more than one of these general categories[6] .

Patients and method

This study was done in Rapareen teaching hospital in the neonatal care unit from the period of first January 2015 until first of January 2016. Two hundred cases are collected including term and preterm babies, term baby defined as any labour 37 weeks, preterm labour defined as any labour between 28 weeks and 37 weeks. Acute kidney injury (AKI) was defined as plasma creatinine concentration 1.5 mg⁄dl and blood urea nitrogen (BUN)>20mg/dl and on two separate test at least 24 hours apart. Oliguria was defined as urine output less than 1ml/kg/h. After taking a complete history and full physical examination 200 case were enrolled in this study. Inclusion criteria were: birth asphyxia, congenital heart disease (CHD), acute gastroenteritis with dehydration, respiratory distress syndrome (RSD) and sepsis.

Patients were excluded if more than 30 days of age. For all neonates admitted in our neonatal care unit, we did these investigations: Blood urea and serum creatinine measured by creatinine jaffe gen.2. Cobas Integra/Cobas System, made in Germany. Blood urea and serum creatinine measured by creatinine jaffe gen.2. Cobas Integra/ Cobas System, made in Germany.

Serum urea measured by Ureal, Urea/Bun, Cobas Integra/ Cobas System. Complete blood count (CBC) measured by MEDONIC –SERIUS DILUNT, made in Sweden. Statistical methods done using the statistical package for social sciences for windows (SPSS 12), p value <0.05 was considered statistically significant.

Results

The total number of the studied sample was 200 neonates. The mean age ± SD of the sample was 8.6 ± 9.9 days, ranging from one to 30 days. The median was three days. Table 1 shows that around two thirds 64.5% of the sample were less than seven days old. More than half 58.5% of the sample was male, and 57.5% of the neonates born by Cesarean section. The weight for age Z score was low in 43.5% of the neonates, Table 1.

Table (1): Distribution of sample by some basic characteristics.

Variables	Categories	No.	%
Age (days)	< 7	129	64.5
	7-13	16	8.0
	14-20	20	10.0
	>21	35	17.5
Sex	Male	117	58.5
	Female	83	41.5
Mode of delivery	NVD	85	42.5
	C/S	115	57.5
Z score	Low	87	43.5
(weight for age)	Normal	113	56.5
Total		**200**	**100.0**

Around half 47% of the patients had RDS, 27.5% had sepsis, and 11% had birth asphyxia. Only 5% had CHD alone, and 2% had CHD with other medical conditions (sepsis and RDS) as shown in Table 2.

Table (2): Distribution of patients by diagnosis (risk factors).

Diagnosis	No.	%
RDS	87	43.5
Sepsis	55	27.5
Birth asphyxia	22	11.0
Congenital heart disease	10	5.0
Acute gastroenteritis	16	8.0
Sepsis + acute GE	6	3.0
Sepsis + CHD	3	1.5
RDS + CHD	1	0.5
Total	**200**	**100.0**

The prevalence of high blood urea levels was 51% of the whole sample as mentioned in Table 3. It shows no significant association between gestational age, sex, and weight for age Z scores with the blood urea levels. The same table shows increased levels of blood urea among those with CHD 90%, birth asphyxia 81.8% and sepsis 67.3%, whereas it was 23% among neonates affected with respiratory distress ($p < 0.001$).

The same pattern is observed in table 4 regarding levels of serum creatinine, except for RSD, where only 2.3% of those with RSD, had high serum creatinine; and the prevalence of high serum creatinine was 21.5% of the whole sample.

Table (3): Levels of blood urea by gestational age, sex, weight for age, and clinical diagnosis.

Variables	Categories	Blood urea				Total (N)	p value
		Low/Normal		High			
		No.	%	No.	%		
Gestational	< 37	46	51.1	44	48.9	90	0.589
age	37	52	47.3	58	52.7	110	
Sex	Male	64	54.7	53	45.3	117	0.056
	Female	34	41	49	59	83	
Z score,	Low	42	48.3	45	51.7	87	0.857
Wt. for age	Normal	56	49.6	57	50.4	113	
Diagnosis	RDS	67	77	20	23	87	< 0.001
	Sepsis	18	32.7	37	67.3	55	
	Birth asphyxia	4	18.2	18	81.8	22	
	CHD	1	10	9	90	10	
	Acute GE	7	43.8	9	56.3	16	
	Sepsis +acute GE	1	16.7	5	83.3	6	
	Sepsis + CHD	0	0	3	100	3	
	RDS + CHD	0	0	1	100	1	
Total		**98**	**49**	**102**	**51**	**200**	

Table (4): Levels of serum creatinine by gestational age, sex, weight for age, and clinical diagnosis.

Variables	Categories	Blood urea				Total (N)	p value
		Low/Normal		High			
		No.	%	No.	%		
Gestational	< 37	67	74.4	23	25.6	90	0.207
age	37	90	81.8	20	18.2	110	
Sex	Male	96	82.1	21	17.9	117	0.147
	Female	61	73.5	22	26.5	83	
Z score,	Low	66	75.9	21	24.1	87	0.426
Wt. for age	Normal	91	80.5	22	19.5	113	
Diagnosis	RDS	85	97.7	2	2.3	87	< 0.001
	Sepsis	43	78.2	12	21.8	55	
	Birth asphyxia	10	45.5	12	54.5	22	
	CHD	3	30	7	70	10	
	Acute GE	13	81.3	3	18.8	16	
	Sepsis +acute GE	2	33.3	4	66.7	6	
	Sepsis + CHD	0	0	3	100	3	
	RDS + CHD	1	100	0	0	1	
Total		**157**	**78.5**	**43**	**21.5**	**200**	

Table 5 shows that the prevalence of high blood urea (after 24 hours) was 39.5%. Still no significant association was noticed between blood urea levels with gestational age, sex, and weight for age. Regarding the diagnoses (risk factors), the same table shows high rates of increased blood urea levels (after 24 hours) among those with CHD, birth asphyxia, and sepsis, and lower rate among RSD ($p < 0.001$).

Table (5): Levels of serum creatinine by gestational age, sex, weight for age, and clinical diagnosis.

Variables	Categories	Blood urea				Total (N)	p value
		Low/Normal		High			
		No.	%	No.	%		
Gestational	< 37	54	60	36	40	90	0.896
age	≥37	67	60.9	43	39.1	110	
Sex	Male	76	85	41	35	117	0.126
	Female	45	54.2	38	45.8	83	
Z score,	Low	53	60.9	34	39.1	87	0.915
Wt. for age	Normal	68	60.2	45	39.8	113	
Diagnosis	RDS	78	89.7	9	10.3	87	< 0.001
	Sepsis	27	49.1	28	50.9	55	
	Birth asphyxia	4	18.2	18	81.8	22	
	CHD	1	10	9	90	10	
	Acute GE	10	62.5	6	37.5	16	
	Sepsis +acute GE	1	16.7	5	83.3	6	
	Sepsis + CHD	0	0	3	100	3	
	RDS + CHD	0	0	1	100	1	
Total		**121**	**60.5**	**79**	**39.5**	**200**	

Table 6 shows that the prevalence of high serum creatinine (after 24 hours) of the whole sample was 23.5%. No significant association was detected between serum creatinine levels with gestational age, sex, and weight for age. The prevalence was only 2.3% among those with RSD, while higher levels were detected among those with CHD 80%, birth asphyxia 63.6%, and sepsis 23.6%.

Table (6): Levels of serum creatinine after 24 hours by gestational age, sex, weight for age, and clinical diagnosis.

Variables	Categories	Blood urea				Total (N)	p value
		Low/Normal		High			
		No.	%	No.	%		
Gestational	< 37	65	72.2	25	27.8	90	0.197
age	≥37	88	80	22	20	110	
Sex	Male	91	77.8	26	22.2	117	0.613
	Female	62	74.7	21	25.3	83	
Z score,	Low	64	73.6	23	26.4	87	0.390
Wt. for age	Normal	89	78.8	24	21.2	113	
Diagnosis	RDS	85	97.7	2	2.3	87	<0.001
	Sepsis	42	76.4	13	23.6	55	
	Birth asphyxia	8	36.4	14	63.6	22	
	CHD	2	20	8	80	10	
	Acute GE	14	87.5	2	12.5	16	
	Sepsis +acute GE	2	33.3	4	66.7	6	
	Sepsis + CHD	0	0	3	100	3	
	RDS + CHD	0	0	1	100	1	
Total		**153**	**76.5**	**47**	**23.5**	**200**	

***By Fisher's exact test**

Table 7 shows significant decrease of mean blood urea levels after 24 hours (53.1 to 50.6) (p = 0.008). The mean creatinine level after 24 hours was higher than that on admission but the difference was not significant (p = 0.302)

Table (7): Comparison between mean blood urea and serum creatinine readings on admission and after 24 hours.

	N	Mean	SD	P value
Urea	200	53.10	42.73	0.008
Urea after 24 h.	200	50.64	47.61	
Creatinine	200	1.00	.93	
Creatinine after 24 h.	200	2.03	14.18	0.302

Discussion

In this study, 200 neonates were enrolled and about 51%of them were affected by AKI. The total number of the studied sample was 200 neonates. The mean age + SD of the sample were 8.6 + 9.9 days, ranging from one to 30 days. The median was three days. Table 1 shows that around two thirds 64.5% of the sample were less than seven days old. More than half 58.5% of the sample was males, and 57.5% of the neonates born by Cesarean section. The weight for age Z score was low in 43.5% of the neonates. The prevalence of AKI in the female neonates were more than the male neonates while in other studies the prevalence of AKI in the boys were more than the female 20% of newborns were products of term pregnancy, which is similar to other researches7. In this study, AKI was more prevalent mainly because more common etiologies of AKI in our patients such as sepsis and congenital heart disease and asphyxia. The most common cause of AKI in this study was CHD 90%, birth asphyxia 81.8%, and sepsis 67.3%. In 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 6, 8.

This may be due to more precise documentation of immediate prenatal, delivery room and postnatal condition of the fetus and newborn in other studies, which has revealed prenatal asphyxia as Abu-Hawley study 9. The worst prognosis of patients in this study was associated with asphyxia and the best prognosis with medical treatment as in Csaicsich et al study10. Also, it showed that the most common predisposing factor for AKI was sepsis and ischemic events. And this is was commitment with our study in which the most common risk factor was CHD 90% from the total number in another study, of 16 AKI neonates, in nine cases the perin atal asphyxia, in four cases renal anomalies and in three cases, congenital heart diseases were risk factors for acute kidney injury AKI11 & this is also was commitment to our study in which CHD was the most common cause.

Conclusions

Acute renal injury was more common in female neonate than males' neonate and high blood urea were more common in female than male. No significant associations were noticed between blood urea levels with gestational age, sex, and weight for age. In this study, sepsis, hypovolemic secondary to dehydration, congenital heart disease, asphyxia, were common predisposing factors for AKI in neonates.

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