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ABSTRACT

Background: Acute Kidney Injury (AKI) is an independent predictor of morbidity and mortality for critically ill neonates at Neonatal Intensive Care Unit (NICU).

Aim of work: The objective of this study was to evaluate the risk factors for AKI in neonates with hypoxic ischemic encephalopathy (HIE) admitted to NICU of Tanta University hospital (TUH).

Subjects and Methods: This cross-sectional study was carried out on 190 neonatal patients incubated at NICUs of TUH with HIE diagnosed by World Health Organization (WHO) definition of HIE. 84 age and sex matched neonates with HIE served as controls (group1) and 106 neonates with HIE meeting neonatal AKI diagnostic criteria of Kidney Disease Improving Global Outcome (KDIGO) served as group2. The two groups were analyzed for risk factors of AKI during the study duration. This study reported that AKI was reported in 84 (44.2 %) of the studied neonates with HIE. There was no statistically significant difference in incidence of AKI between the studied two groups (cases and controls) as regard gestational age (full term versus preterm neonates), or as regard different stages of HIE. 80(95.24%) of group 2 had non oliguric renal failure. 78(92.86 %) of them recovered before discharge and the remaining 6(7.14%) recovered at 1 month follow-up. Difficult or instrumental delivery was reported to be the main predisposing factors for AKI 16 (19.1 %). Patients with shock had more advanced stages of AKI compared to those without shock. Conclusion .It was recommended to suspect all neonates who were diagnosed as HIE for AKI: Based on clinical features of HIE, difficult delivery was the main predisposing factors for AKI .Shock was associated with advanced stages of AKI. Neither oliganuria nor APGAR scoring pointed to AKI in neonates with HIE.

Keywords: Neonates, Acute kidney injury, Hypoxic ischemic encephalopathy.

INTRODUCTION

HIE is a common disease in neonatal intensive care units (NICU) and constitutes a major aetiology of morbidity and mortality in NICU. Overall, the incidence of HIE was mentioned to be about 1 to 10 per 1000 neonates^{.[1,2]} HIE redistributes cardiac Output (CO) to variable organs compromising tissue perfusion, thus results in to multiorgan dysfunction namely the kidney. Because glomerular and renal tubular tissues are highly sensitive to hypoxemia, disturbed renal functions can occur within one day of the insult of HIE. If hypoxia is prolonged in time, irreversible. Intrinsic AKI can occurs resulting in disturbed fluid, electrolyte, and acid-base balance^{.[3]}

Neonatal AKI as a common practical disorder faced in neonates with HIE worsen the overall outcome of HIE. AKI can affects 8 % -24% of critically ill neonates and lead to a mortality rate of 10% - 60%.^[4]

Early identification of neonatal AKI in HIE patients is very important for planning of different lines of treatment of ill neonates. Early management of neonatal AKI can prevent dangerous complications. In most of NICU, elevated single measurement of serum creatinine is used for diagnosing AKI which is not ideal as it may has false positive results so serial multiple measures should be done for adequate diagnosis of neonatal AKI.^[5,6]

As HIE is considered to be one of the commonest aetiologies of AKI in the age of

newly born infants, the accurate prevalence of AKI in neonates with HIE is not yet known. This may be attributed to the concept of general pediatrician about suspecting acute kidney injury when the neonate become presented by reduced urine output. Non oliguric AKI represent about one third of neonates with HIE^[7]

Previous publications suggested that acute renal failure occurs commonly in association with multisystemic affections more than primary kidney disease. It has been previously published that about 35 % of critically ill pediatric patients acquire AKI, which is 4 times higher incidence than non critically ill pediatric patients.^[8]

Acute renal failure has been reported to be associated with prolonged duration of stay in hospital, pediatric intensive care unit (PICU) or NICU.^[9] AKI was reported to be an independent predictor for unfavorable prognosis in critically ill neonates.

In pediatric patients who survive after experiencing HIE complicated by AKI, residual renal abnormalities are likely to be experienced.^[10]

The initial objective in management of AKI in pediatric age is to prevent and to adequately treat AKI by early prediction of neonates, infants, children or adolescents who have high risk factors thus detecting AKI early so that further damage can be prevented.

The previously known classic definitions of acute renal failure were based on level of blood urea nitrogen (BUN) which was influenced by various factors including dehydration, corticosteroid therapy, and acid base balance.

Variable next definitions had been published which result in confusion in literatures and difficulty in comparison among different publications.^[11]

Later on, a multiple levels classification system known as the Risk, injury, failure, loss, end-stage kidney disease (RIFLE) criteria which was established for accurate stratification of AKI. ^[12] It was based on estimated creatinine clearance (eCrCl), serum creatinine, and oliguria. The latest definition is based on AKI network (AKIN) criteria ^[13].

The previously published researches have reported that AKI constitute from one third to one half of full term neonates who diagnosed as HIE^{.[14-17]}. The objective of the present study

was to assess the incidence and risk factors of AKI in neonates with HIE in a sample of Egyptian neonates.

PATIENTS AND METHODS

Design of the Study and Setting

This Case Controlled cross sectional study was conducted after approval from the Ethical Research Committee of the Faculty of Medicine of Tanta University and informed parental written or verbal consents from all subjects involved in the study, upon 190 neonatal patients who were hospitalized at NICUs of Tanta Universality hospital in the period from December 2015 to December 2017. 84 neonatal patients were diagnosed as HIE as per the World Health Organization (WHO) definition ^[18] with AKI according to diagnostic criteria of Neonatal Kidney Disease Improving Global Outcome (KDIGO)^[19]. Their mean of gestational ages was 37.19+ 3.2 weeks They were 59 males and 25 females, as group(2).108 neonates with diagnosis of HIE of comparable age and sex during their neonatal ICU admission were served as a controls (group 1). Their gestational ages their mean of gestational ages was 37.16+ 2.9 weeks. They were 58 males & 48 females.

Inclusion Criteria

Neonatal patients with AKI .AKI was diagnosed when serum creatinine ≥ 0.3 mg/dl within 48 hours or serum creatinine ≥ 1.5 -1.9 X reference range of serum creatinine (meaning the lowest previous value)within 7 days or urine output from 0.5 to 1 ml/kg/hr for 1st 24 hours of life according to proposed KDIGO definition for neonatal AKI.^[19]

Exclusion Criteria

Neonates with perinatal history of maternal azotemia or kidey disorders ,congenital anomalies of kidney or urinary tract (as detected by antenatal or postnatal ultrasonography) and neonates with other factor which may change kidney function tests such as septicemia, respiratory distress syndrome(RDS), necrotizing enterocolitis(NEC), major congenital anomalies and those who died during the research period.

The Sample Size

The size of the sample was calculated using the formula n = 4P (100-P/d2)

Where, 'n' is sample size,

"P" is anticipated incidence of AKI in birth asphyxia HIE

'd' is absolute precision which was taken as 10%.

The sample size was found to be 85 on the basis of incidence of AKI in neonates with HIE from previous studies.

Study Protocol

All patients and controls were subjected to:

- Through history taking focusing on
- Baseline maternal data (including maternal age ,any medical illnesses associated with or complicating pregnancy, Hemoglobin during gestation, number of antenatal obstetric follow up ,type of delivery, prolonged labour(total duration of labor > 20 hours), instrumental labour, meconium aspiration and purberal sepsis,antibiotic medications).
- Baseline neonatal history in details including gestational age ,APGAR scoring at one and five minutes, infant maturity, birth weight, and resuscitation details, onset of AKI, or other critical illness, and disease outcome ,gender, ,NICU stay, precipitating factors for AKI including sepsis and shock, time of start of entral feeding, antibiotic usage for neonates.
- Full clinical examination :including
- Vital signs.
- Anthropometric measurements: Weight and height for age percentiles according to Egyptian growth curves.^[20]
- Assessment for staged for hypoxic-ischemic encephalopathy (HIE) using Sarnat and Sarnat Clinical staging of HIE^[18].
- The laboratory investigations: included
- Complete blood count (CBC) by ERMA BC 210
- C-Reactive protein (CRP) by Adiva Centaur CP/ Immunoassay
- Prothrombin time (PT) and activity by Sysmex CA 1500
- On the first 3 consecutive days of illness Serum creatinine by kinetic assay kinetic assay fully automated clinical biochemistry analyzer (HITACHI 912) which useed JAFFE's method, using commercially

available kits for the same and blood urea were measured also .

• Urine output was evaluated using diaper weight method.

The diagnosis and staging of severity of AKI were determined on the basis of definition given by diagnostic criteria of Neonatal Kidney Disease Improving Global Outcome (KDIGO) [19]

• Pelviabdominal ultasonography: for all neonates who had AKI (group 2 only) to rule out gross congenital renal anomalies.

Sampling

Samples withdrawn at first and 3rd day of admission. Seven milliliters of venous blood was drawn under aseptic conditions and distributed as follows:

- One milliliter of whole blood was taken in an EDTA vacutainer (violet cap) and mixed gently. This sample was used to measure complete blood count (CBC) which was done for all samples by sysmex KX-21N.
- Three milliliters of blood were taken in citrated test tubes (blue cap), the samples were centrifuged at 1500 rpm for 15 min. The separated plasma was used for the assay of PT, INR and PTT and serum Cystatin C and Beta 2-Microglobulin.which were measured using human enzyme-linked immuno-sorbent assay (ELISA) (sandwich technique) kits.
- Three milliliters of blood were taken in plain test tubes without anticoagulant (red cap) and left until coagulation. After coagulation, the samples were centrifuged at 1500 rpm for 15 min. The separated serum was used for the assay of ALT&AST and seum creatinine (Cr)], Serum Creatinine was evaluated with the BioSystems reagent kit provided by BioSystems S.A.(Barcelona, Spain) bv modified Jaffee reaction. Serum urea was determined by the enzymatic colorimetric test, using a Diamond kit, (Diamond Diagnostics, Holliston, USA) and C-reactive protein (CRP), Quantitative C- Reactive protein CRP: serum was separated and analyzed using Turbox plus. Results were considered positive above 6 mg/l.

Statistical Analysis

Analysis of results was done using SPSS 11.2 software. Mean \pm standard deviation/median

(minimum-maximum) was calculated for continuous variables and frequency for categorical variables. The prevalence and 95% confidence intervals were reported. Categorical variables were compared using Chi-Square/ Fisher's exact test .Continuous variables following the normal distribution between the two groups were compared by independent t-test (2 groups) or by one way ANOVA test (>2group) . (Data not following normal distribution were compared using Kruskal-Wallis/ Wilcoxon rank sum test among the groups as applicable. P < 0.05 was considered statistically significant ^[21]

RESULTS

Out of 190 neonates with HIE who were included in this study and who were evaluated for AKI. AKI was diagnosed in 84 newborns (44.21%) who were enrolled in this work as Group II (AKI group). and the remaining neonates were enrolled as controls (Group I) (non-AKI group).

Different demographic ,clinical and laboratory variables including maternal age, gestational age, mode of delivery, prolonged or instrumental labour, meconium aspiration , infant maturity, birth weight ,gender, APGAR scores at 1 and 5 min, and duration of resuscitation ,NICU stay, precipitating factors for AKI including sepsis and shock, time of start of entral feeding, antibiotic usage for mothers or neonates, mortslity rate , and serum creatinine were compared between the two studied groups as summarized in Table1.

There was no statistically significant difference between studied cases and controls as regard maternal age, sex, birth weight ,APGAR scoring at 1 and 5 minutes ,duration of resuscitation, meconium aspiration (p > 0.05). While there was no significant difference in Antibiotic usage either in the mother during the before completion of study in cases when compared to controls. (P > 0.05)

As regard modes of the labour, there was no significant differences between the two studied groups (p > 0.05). However, the AKI group had prolonged difficult labor (may be instrumental) more frequent than non AKI group, and the difference was statistically significant (P < 0.05) [Table 1].

In addition, the two groups did not differ significantly in terms of gestational age, low birth weight, and appropriate for gestational age (AGA)/small for gestational age (SGA)/large for gestational age . (P>0.05)

On comparing the delivery modes variables, there was no statistically significant differences between the two studied groups. (P>0.05). However the AKI group had prolonged labor more often than the non-AKI group, and the difference was statistically significant (P < 0.05).

Antibiotic usage either in the mother during last week of pregnancy or in the neonate was also not reported to be statistically significantly different between the two studied groups in this study. Shock as a predisposing factor for AKI was reported in this work to be more frequently (26.2 %) in AKI group than in non AKI group (11.32%) however the difference was not statistically significant (P = 0.06) .Moreover, among second group (HIE+AKI group) ,ill neonates complicated by shock had more severe staging of AKI if compared to those who were not complicated by shock, the difference was statistically significant (P = 0.04) [Tables 1].

As regard renal function tests, the neonates in the two studied groups had no significant difference in initial blood urea and serum creatinine values at the time of admission. Contrary to the expected values, blood urea and serum creatinine values were lower in patients with AKI when compared to patients with no AKI and the difference being statistically significant (P < 0.05).

This observation clarified that an initial normal blood urea and or serum creatinine levels cannot exclude AKI and also this observation emphasized the significance of serial follow up measurements of blood urea and or serum creatinine levels aiming accurate diagnosis of AKI.

On comparison between the studied two groups as regard morbidity duration of PICU stay, time of starting of central feeds and presence and degree of HIE, there were no statistically significant difference in any of these parameters between the studied cases and controls. Moreover, no significant relation was found between the severity of AKI and severity of HIE.

			Group 1(Controls) (HIE without AKI) (n = 106)	Group (HIE) (n	0 2(Cases) with AKI) . = 84)	Statistical test
Maternal age(Years)		Mean \pm SD	24.1 <u>+</u> 3.4	23.8+ 3.3		0.78
Gestational a	age (in weeks)	$Mean \pm SD$	37.16 <u>+</u> 2.9	37.19+ 3.2		0.97
Gender	Male:	No(%)	58 (54.7)	59 (70.2)		0.69
	Female:	No(%)	48(45.3)	25	25 (29.8)	
Mode of delivery	NVD	No(%)	56(52.8%)	48(57.1%)	
	CS	No(%)	46(43.4%)	26	(31%)	0.21
	Instrumental delivery	No(%)	4(3.8%)	10(11.9%)		
Birth weight (Kg)		Mean ± SD	2.48 <u>+</u> 0.61	2.5	2 <u>+</u> 0.68	0.74
APGAR	at 1 min	Mean \pm SD	2.2 ± 0.75	2.	1 +0.8	0.64
score	at 5 min	Mean \pm SD	4.6+1.5	4.	4.5 <u>+</u> 1.7	
Prolonged (diff	icult) labour (%)	No (%)	6 (5.7%)	16(19.1%)		0.04*
Meconium	n aspiration	No(%)	32 (30.2%)	30(35.7%)		0.57
Infant maturity	Preterm:	No(%)	58 (54.7)	59(70.2 %)	0.69
	Full term	No(%)	48(45.3 5)	25 (29.8 %)	
Types of AKI	Non oliguric	No(%)	-	80(95.24%)		-
	Oliguric	No(%)		4(4.76%)		
Resuscitation Du	ration (minutes)	Mean \pm SD	7.8 <u>+</u> 5.3	8.8 <u>+</u> 5.5		0.44
Staging of AKI	Stage 1	No (%)	-	24(28.6 %)		
	Stage 2	No (%)	-	18(21.4%)		-
	Stage 3	No (%)	-	42(50 %)		
Risk factor for sepsis		No (%)	16(15.1 %)	16 ((19.1%)	0.61
Shock		No (%)	12(11.3%)	Stag1	2 (2.4 %)	P1=0.06
				Stag2	4(4.8 %)	*
				Stage AKI	16(19 %)	-
Recovery of AKI	Beforedischage:	No(%)	-	78(92.86 %) 6(7.14 %)		-
	Afterdischarge:	No(%)	-			
Serum Creatinine (mg/dl)		Mean ± SD	1.5 ± 0.6	0.5 ± 0.2		0.048*
Staging of HIE	Stage 1	No (%)	56(52.8 %)	40(47.6 %)	>0.05
	Stage 2	No(%)	30(28.3 %)	23(27.4 %)		
	Stage 3	No(%)	20(18.9 %)	21(25 %)		1
Duration of N	ICU stay(days)	Mean \pm SD	8 <u>+</u> 5	9 <u>+</u> 6		0.443
Time of starting of entral feeds(days)		Mean \pm SD	9.3 <u>+</u> 0.7	9.3 <u>+</u> 0.8		0.97
Mortality rate		No (%)	3 (2.9%)	8(9.6%)		0.04*
Classification	low birth weight/	No (%)	58(54.4 %)	59(70.3 %)		0.7
according to	small for gestational					
gestational age:	age (SGA),					
No (%)	appropriate for gestational age (AGA)	No(%)	24(22.6 %)	16 (19 %)		
	large for gestational age	No(%)	24(22.6 %)	9(10.7 %)		
Antibiotic usage	In the mother during the last week of pregnancy	No (%)	18 (30 %)	27	(45 %)	0.7
	in the neonate.	No (%)	36 (60 %)	54	(90 %)	> 0.05

 Table1. Demographic, clinical, clinical and laboratory characteristics of the studied cases and the controls.

**P*-value <0.05 significant P1 comparison between group 1 & 2, P2 comparison between stages of AKI within group 2.

Out of the 84 cases of AKI, 26 neonates succumbed to their illness, and two neonates were taken against medical advice by parents.

Although death occurred in a higher percentage in the AKI group when compared to controls, the difference was not statistically significant (P > 0.05).

In this study, it was of value to report that only 4 (4.76%) of the 84 of the neonates diagnosed to have AKI in our study had oliguria.

Only 6 (7.14%) of the neonates had still impared renal function at the time of discharge These neonates had decreasing trend of serum Creatinine on follow up as they were followed up in the specialized Pediatric Nephrology outpatient clinic of TUH. 4 of them became having normal renal function tests after 1 month of follow-up visits while 2 patients were lost to follow-up.

Out of the 84 cases of AKI in our study ,24 cases (28.6%) were found to be in AKI Stage 1, 18 cases (21.4%) in AKI Stage 2, and 42 cases (50%) were reported to be in AKI Stage 3.

In our study, it was reported that emographic data included maternal age, gestational age, sex of the neonates , and birth weight do not differ significantly among different stages of AKI in group 2.

DISCUSSION

AKI is defined as a rapid reversible rise in serum creatinine and nitrogenous wastes levels, in addition failure of the renal glomeruli and tubules to maintain fluid and electrolyte and acid base balace.^[22]

Most of publications reported that single measurement of serum creatinine level was neither sensitive nor specific for rerflecting diagnosis or severity of AKI. It cannot distinguish between variable classes of AKI.^[21]

Previousouly published researches had studied the releationship, incidence and previlance of HIE and AKI al over the world ^[14-17]

Incidence of AKI in HIE had varied from one eighth to two third of patients^{.[14-17]}

In studies of Alaro D et al, Amardiyanto R et al, Bhantnagar A et al and Gopal G. et al, no clear uniform definition of AKI was identified, resulting in lack of uniform and comparable definition for AKI^{.[14-17]} Moreover, different risk factors for AKI were not reported in the previously published articles. In our stydy, basic demographic data was comparable between the two studied groups (non AKI and AKI groups). Univariate analysis of variables such as gender, mean birth weight, mean gestational age, mean maternal age, and resuscitation (mode and duration) a birth did not reveal any significant difference between the two groups (P > 0.05).

In our study, 44.21% of cases of HIE developed AKI. Our result was not in agreement with an Indian study which was conducted for assessment of AKI in asphyxiated newly born infants in North India by Bhantnagar A et al., it was found to be 61.7 % of their studied neonates. This difference might be due to the difference of methodology of definition for the diagnosis of AKI in their studied Indian patients which was based on a single blood urea and or serum creatinine level and not on the currently accepted neonatal KDIGO definition (based on rising values of serum creatinine and or decrease in urinary output).^[16]

We have reported in this study that the prevalence of AKI in HIE was not significantly different between term and preterm neonates, and also between various stages full of HIE (I, II &III).

Prolonged labor (total duration of labor > 20 hours) was found more frequently in AKI group compared to non-AKI group, the difference being statistically significant (P < 0.05). This indicates that prolonged labor predisposed to AKI in HIE.

This parameter had not been evaluated in previous studies.^[14-17]

Shock was reported in this study more frequently in HIE neonates with AKI than those without AKI. However, the difference was not statistically significant (P = 0.06). In addition, among AKI group, neonates with shock had more severe stages of AKI compared to those without shock, the difference being when statistically significant (P = 0.04).

An interisting and unexpected result in our study was that the mean initial blood urea and serum creatinine levels which were done within 24 hours of birth were lower in AKI group when compared to non-AKI group, the difference was statistically significant (P < 0.05). This result was not reported in previously published studies

- either because; they assessed only the difference between two serial values

or in studies which diagnosed AKI based on single blood urea and or serum creatinine values .so we can emphasize that a single normal value of blood urea and or serum creatinine which was obtained early in the neonatal period cannot rule out diagnosis of AKI, clarifying the significance of serial repeated measurements of serum creatinine (as in modified KDIGO definition of AKI). It also signified that the first blood urea and or serum creatinine values did not contribute significantly to the diagnosis of AKI.

In our study, AKI was not reported to be significantly different between full term and preterm neonates. There was no significant difference in the prevalence of AKI in preterm AGA, term AGA, and SGA in our study. Maternal factors such as maternal age, ante partum hemorrhage ,and meconium stained liquor did not differ significantly between the two groups. Maternal diseases such as hypertensive diseases of pregnancy and did not gestational diabetes also show significant difference.

The majority had nonoliguric renal failure. Only 4 (4.7%) of the neonates with AKI in our study had oliguric renal failure. Previous publications had also reported that non oliguric renal failure as being more common than oliguric type. ^[14-17]

In our study, 28.5% of cases were found to be in AKI Stage 1, 21.4% of cases in AKI Stage 2, and 50% of cases in AKI Stage 3. Only 6 of the patients (7.14%) were discharged with impaired renal function, and all of them had decreasing trend of serum creatinine. 4 of them had normal renal function at 1 month follow-up while 2 neonate were lost to follow-up. In our study, the mortality of neonates was found to be 31.7 % within the AKI group. It was found that demographic data such as maternal age, sex of the baby, gestational age, and birth weight do not differ significantly among the different stages of AKI. It was also found that the outcome of neonates with different stages of AKI did not differ significantly among each other. In addition, stage of AKI did not show any correlation with stage of HIE.

CONCLUSIONS

From the present work, we can concluded that it was a difficult task to predict AKI in neonates with AKI based on demographis and clinical criteria, oliguria, or APGAR score so it was recommended to screen all neonates with HIE for AKI so we could could detect them early thus managed them accordingly early leading to improvement in their outcomes.

Shock should be detected early in neonates with AKI and should be also treated adequetly as shock was associated with more advanced stages of AKI thus less favourable prognosis.

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