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"Incidence of Acute Kidney Injury in Neonates with Perinatal Asphyxia"

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Abstract
Introduction: *Perinatal asphyxia is an essential reason for neonatal mortality and neurological morbidity. Birth asphyxia occurs when a baby does not receive enough oxygen before, during or after birth. Perinatal asphyxia remains a common problem in the neonatal nursery and is a significant cause of morbidity and death in the term and preterm neonate.*
Aim of the study: *The aim of this study was to determine the incidence of acute kidney injury (AKI) in neonates with perinatal asphyxia.*
Methods: *This was a cross sectional observational study was conducted in NICU of Dr. MR Khan Shishu Hospital & Institute of Child Health, Dhaka, Bangladesh during the period from January 2018 to June 18. In total 125 neonates with perinatal asphyxia was selected as the study population. The cases were categorized according to Sarnat and Sarnat staging of HIE without EEG. Acute kidney injury (AKI) was considered in neonates who had a serum creatinine concentration >125 µmol/l or a serum creatinine concentration>100 µmol/l and oliguria (urine output <1ml/Kg/hour) for 24 hours or more on third day of neonatal age. All data during hospital stay including cases with AKI were taken and snalyzed using SPSS version 20.*
Result: *In our study we found among 30 HIE-I stage patients only 1 had AKI which was 3.33%. Besides this, among 69 HIE stage II patients 13 had AKI which was 18.84% and among the rest 26 HIE-III patients 12 had AKI which was 46.15%. This ratio of HIE-III stage patients was the highest ratio of neonates with AKI. On the other hand in our study in analyzing the mean urine output and their biochemical values we found the mean Urine output (ml/kg/hr) was 1.21±0.17, the mean Blood urea (mg/dl) was 71.80±28.79, the mean Serum creatinine (mg/dl) was 2.01±0.11, the mean was Serum sodium (meq/L) , the mean Serum potassium (meq/L) was 4.97±0.51, the mean Serum chloride (meq/L) was 101.21±5.01, and the mean Creatinine clearance was 16.08±5.23.*
Conclusion*: Birth asphyxia is an important cause of neonatal AKI in developing country especially like in Bangladesh. Majority of the neonates had non-oliguric AKI and responded well to fluid challenge. Abnormalities in the renal function correlates well with the severity of HIE staging. Intrinsic AKI, oliguria, hyponatremia, reduced creatinine clearance and abnormal sonographic scan suggest bad prognosis in neonatal AKI secondary to perinatal asphyxia.*
Key words*: Acute kidney injury, Perinatal asphyxia, Neonates, Oliguria.*

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I. Introduction

Perinatal asphyxia is defined by the World Health Organization (WHO) as "Failure to initiate and sustain breathing at birth."1. There is a high incidence of AKI among the asphyxiated term infants, (50 – 72%).2 Asphyxia is an important cause of AKI and transient kidney impairment with adverse effects, especially in the five days of birth3,4. The kidney is the most damaged organ in asphyxiated full- term infants.4 Perinatal asphyxia is an imperative reason for neonatal mortality and long haul neurological morbidity and late sequelae especially in a developing country like Bangladesh5. The general rate of this condition is assessed to be between 1 to 10 for every 1000 live births and is affected by the birth weight and gestational age of the infant furthermore by the neighborhood accessibility of therapeutic resources6. Hypoxic harm can influence all organs

of the neonate, with kidneys being the commonest (half) influenced. Different organs are influenced in the accompanying request CNS (28%), CVS (25%) and respiratory system (23%)7. Renal inadequacy may happen as right on time as 24 h from the season of affront, which if delayed may even prompt irreversible cortical necrosis 8. Asphyxia can lead to multi-organ dysfunction and a redistribution of cardiac output to maintain cerebral, cardiac, and adrenal perfusion while potentially compromising renal, gastrointestinal, and skin perfusion 9,10,11,12. Hypoxia and ischemia can cause damage to almost every tissue and organ in the body 1. It is therefore not surprising that acute kidney injury (AKI) is common in asphyxiated neonates, occurring in up to 56% of these cases13. Subsequently, convenient ID of renal disappointment is essential in neonates with hypoxic ischemic encephalopathy (HIE) to keep up a stable biochemical milieu by starting fitting liquid and electrolyte management8. A major difficulty in diagnosing neonatal AKI is the lack of a consensus definition, largely because of dearth of specific measurable variables and biochemical markers. This study was performed to determine the incidence of acute kidney injury (AKI) in asphyxiated neonates and also to correlate the severity and type of AKI with Apgar score and HIE.

II. Objectives

a) General objective:

- To determine the incidence of acute kidney injury (AKI) in neonates with perinatal asphyxia.

b) Specific Objectives:

- To assess the HIE stages of acute kidney injury (AKI) in neonates with perinatal asphyxia.

III. Methodology & Materials

This was a cross sectional observational study was conducted in NICU of Dr. MR Khan Shishu Hospital & Institute of Child Health, Dhaka, Bangladesh during the period from January 2018 to June 18. In total 125 neonates with perinatal asphyxia was selected as the study population. The cases were categorized according to Sarnat and Sarnat staging of HIE without EEG. Acute kidney injury (AKI) was considered in neonates who had a serum creatinine concentration >125 µmol/l or a serum creatinine concentration>100 µmol/l and oliguria (urine output <1ml/Kg/hour) for 24 hours or more on third day of neonatal age. Newborns with malformation syndromes and those who died within 3 days of admission were excluded from the study. Maternal and neonatal data was captured using pre-tested questionnaires and forms. Perinatal asphyxia was defined as failure to initiate and sustain breathing at birth[1], clinical evidence of HIE and Apgar score less than 7 at 5 minutes. Gestational age was determined using the Finnström score. A predesigned and pretested preform was used to collect data such as gestational age, birth weight and relevant perinatal history. On the basis of Apgar score at 5 minutes, asphyxiated neonates were further grouped into mild (score of 6 or 7), moderate (score of 4 or 5), and severe asphyxia (score of 3 or less). All neonates with clinical features of hypoxic ischemic encephalopathy (HIE) were staged by Sarnat and Sarnat scoring system. All the enrolled babies were subjected to ultrasonography within 24 hours of birth to rule out any congenital malformations of the urinary tract. Immediate clinical assessment was made by recording respiratory rate (RR), heart rate (HR), capillary filling time (CFT), blood pressure, temperature and spo_2. These neonates were monitored daily to detect derangements in clinical, metabolic and hemodynamic milieu so as to ensure prompt management. Renal function parameters–blood urea, serum creatinine, serum electrolytes, urinalysis including urinary sodium and creatinine were monitored initially within the first 24 hours of birth and then on day 3 of life (between 72-96 hrs of life). Urine output was monitored by applying plastic collection bag (minicom) or by catheterization if required. Criteria adopted for defining AKI was oliguria (urine output <0.5ml/kg/hr) and/or serum creatinine level more than 2 SD above the mean value for that particular gestational age or rising creatinine levels (0.3mg/dl/day)[14.]

IV. Result

In this observational study there were 125 asphyxiated neonates as study population. The age range of the patients was 0 to 72 hours whose gestational ages were between 35 to 42 weeks. The mean gestational age was 37.37±1.32 weeks. The mean birth weight was 2.79±0.48 Kg. According to severity of hypoxic ischemic encephalopathy by sarnat and sarnat stages without EEG the cases were divided into three groups. Among all cases 81 (64.80%) babies were full term and 44 (35.20%) were preterm. In this study male were 59.20% and female were 40.80%. In our study we found 24 (30%) newborns were in HIE stage-I, 69 (55.20%) in HIE stage-II and rest 26 (20.80%) in HIE-III. The most frequent clinical feature was convulsion and it was found in 57 (45.60%) followed by respiratory distress 43 (34.40%), oliguria 19 (15.20%) and comatose/stuporous 17 (13.60%). Among 125 prenatal asphyxiated neonates we found 26 with AKI which was 20.80%. There was significant difference of mean S. creatinine level between present of AKI and absent of AKI in perinatal asphyxia ($p<0.05$). In our study we found among 30 HIE-I stage patients only 1 had AKI which was 3.33%.

Besides this, among 69 HIE stage II patients 13 had AKI which was 18.84% and among the rest 26 HIE-III patients 12 had AKI which was 46.15%. This ratio of HIE-III stage patients was the highest ratio of neonates with AKI. On the other hand in our study in analyzing the mean urine output and their biochemical values we found the mean Urine output (ml/kg/hr) was 1.21±0.17, the mean Blood urea (mg/dl) was 71.80±28.79, the mean Serum creatinine (mg/dl) was 2.01±0.11, the mean was Serum sodium (meq/L) , the mean Serum potassium (meq/L) was 4.97±0.51, the mean Serum chloride (meq/L) was 101.21±5.01.

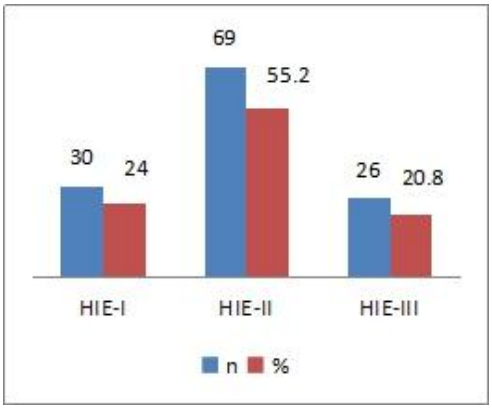

Figure I: Distribution of neonates according to HIE stage (n=125)

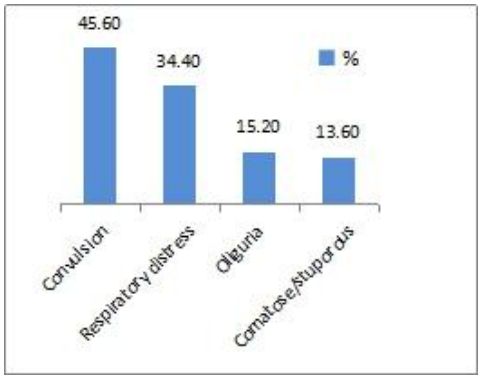

Figure II: Distribution features among participants (n=125)

Table I: HIE stage distribution among total participants (n=125)

HIE stage	n=125	With AKI	%
HIE-I	30	1	3.33
HIE-II	69	13	18.84
HIE-III	26	12	46.15

Table II: Mean urine output and biochemical value of participants (n=125)

Mean urine output/ biochemical value	Cases (n=125)
Urine output (ml/kg/hr)	1.21±0.17
Blood urea (mg/dl)	71.80±28.79
Serum creatinine (mg/dl)	2.01±0.11
Serum sodium (meq/L)	141±4.89
Serum potassium (meq/L)	4.97±0.51
Serum chloride (meq/L)	101.21±5.01

V. Discussion

Perinatal asphyxia is an affront amid the intrauterine or prompt extrauterine period to the embryo or the infant due to hypoxic and/or ischemic harm to different organs of adequate greatness which prompts temporary practical and biochemical changes[6]. As kidneys are very sensitive to oxygen deprivation, renal insufficiency may occur within 24 hours of a hypoxic ischemic episode which if prolonged, may even lead to irreversible cortical necrosis. This observational was conduct on total 125 asphyxiated neonates. The study was done to determine the incidence of acute kidney injury in term neonates with perinatal asphyxia in the NICU of Dr. MR Khan Shishu Hospital & Institute of Child Health, Dhaka, Bangladesh during the period from January 2018 to June 18. In the present study, enrolled 125 neonates the mean birth weight was 2.79±0.48 Kg. This study as can

be expected, 26 (20.80%) of the asphyxiated neonates had AKI. In the present study, the criteria which was used to diagnose AKI was oliguria (urine output < 0.5ml/kg/hr) or serum creatinine level of >2SD above the mean for the gestational age or rising creatinine values. This enabled early diagnosis of AKI, and timely intervention with fluid boluses in neonates with pre-renal AKI helped them to recover fast and prevented them from developing intrinsic AKI. In this study we found that, 19 (15.20%) were oliguric neonates. In the study of Girish, et al. examined 50 cases out of which 64% had AKI and amongst them 37.5% had oliguric AKI and 62.5% had non-oliguric AKI[15] which was very high than that of us. Renal parenchymal injury in oliguric as well as non-oliguric renal failure is essentially similar but heterogenous response of individual nephron and variable damage to tubular epithelium results in anatomical damage in majority of nephrons leading to reduction in single nephron GFR and decreased tubular fluid flow. Occurrence of AKI is most normal in stage III of HIE nearly took after by HIE stage-II. The after-effects of our study are equivalent to Girish, et al. study[15]. In our study 18.84% of stage II HIE neonates had AKI and 46.15% of stage III HIE had AKI. Levels of urea and creatinine were significantly higher as the severity of HIE progressed. The consequences of our study are practically identical to investigations of Gupta, et al., et al., and Girish, et al.,[8,15]. The mean serum creatinine levels among the cases in our study was 2.01±0.11 while Gupta, et al. contemplated 70 suffocated neonates of them 32 cases had no HIE highlights so the mean quality was just 1.08 ± 0.49 among the cases. Aggarwal, et al. concentrated on 25 cases and 25 controls demonstrated the mean worth was 1.0 ± 0.5 among the cases and no notice about the biochemical technique for estimation of creatinine was made. The consequences of our study are equivalent to investigations of Gupta, et al. [8,15]. Sonographic abnormalities were found in all 12 (23.08%) neonates with intrinsic renal failure and all of them succumbed to the illness. Similar observation was found in the study conducted by Gupta et al[8]. Thus, sonographic abnormalities in renal size/echotexture indicates poor prognosis in asphyxiated neonates with AKI. In the present study, levels of serum creatinine used to diagnose AKI was lower than those used in previous studies [16,17], which enabled early intervention and lower mortality rates. Most of the neonates who succumbed had intrinsic AKI. Thus, early recognition of AKI and intervening when neonates have pre-renal AKI can help to decrease the mortality rates.

Limitations of the study

This was a single centered study with a small sized sample. So the findings of this study may not reflect the exact scenario of the whole country. Hence, a study with even a larger sample size is necessary to know the complete clinical profile of AKI in birth asphyxia.

VI. Conclusion

Perinatal asphyxia is an important cause of neonatal renal failure. Monitoring of blood levels of blood urea and serum creatinine helps in the early diagnosis and management of renal failure. AKI in birth asphyxia is predominantly non oliguric so monitoring only urine output does not help in the diagnosis of AKI. In asphyxiated neonates, correlation of biochemical parameters with the urine output is necessary.

References

[1]. World Health Organization. Basic Newborn resuscitation. A practical guide. World Health Organization; Geneva, 1997.
[2]. Gupta B, Pramod S, et al. The incidence of renal failure in asphyxiated neonates and to correlate severity and type of renal failure with Apgar score and hypoxic ischemic encephalopathy (HIE) grading of the neonates. Indian Pediatrics 2005; 42:928-934.
[3]. Carter B, McNabb F. Prospective validation of a scoring system for predicting neonatal morbidity after acute perinatal asphyxia. J Pediatr.1998; 132: 619-23.
[4]. Adams-Chapman I, Stoll B. Nelson textbook of pediatrics. Philadelphia: WB Saunders; 2007. Nervous system disorders. p. 718.
[5]. Hansen, Anne R., and Soul, J. S. "Perinatal asphyxia and hypoxic-ischemic encephalopathy." Manual of Neonatal Care. 7th edition. Philadelphia: Lippincott Williams and Wilkins 71128 (2011).
[6]. McGuire, William. "Perinatal asphyxia." BMJ clinical evidence 2007 (2007).
[7]. Perlman, J. M., et al. "Acute systemic organ injury in term infants after asphyxia." American Journal of Diseases of Children 143.5 (1989): 617-620.
[8]. Gupta, B. D., et al. "Renal failure in asphyxiated neonates." Indian pediatrics 42.9 (2005): 928.
[9]. Perlman JM, Tack ED, Martin T, Shackelford G, Amon E. Acute systemic organ injury in term infants after asphyxia. Am J Dis Child 1989; 143: 617-620.
[10]. Behrman RE, Lees MH, Peterson EN, De Lannoy CW, Seeds AE. Distribution of the circulation in the normal and asphyxiated fetal primate. Am J Obstet Gynecol 1970; 108: 956-969.
[11]. Cohn HE, Sacks EJ, Heymann MA, Rudolph AM. Cardiovascular responses to hypoxemia and acidemia in fetal lambs. Am J Obstet Gynecol 1974; 120: 817-24.
[12]. Rudolph AM. The fetal circulation and its response to stress. J Dev Physiol 1984; 6: 11-9.
[13]. Anne M. Durkan, Todd Alexander R. Acute Kidney Injury Post Neonatal Asphyxia. J Pediatr 2011; 158(2): e29-e33.
[14]. David J. Askenazi, Stuart L. Goldstein. Renal Conditions. Chapter 28 in: John P. Cloherty, Eric C. Eichenwald, Ann R. Stark. Manual of Neonatal Care. 7th Edition. New York: Lippincott, Williams and Wilkins; 2011, pp 350-376.
[15]. Gopal, Girish. "Acute Kidney Injury (AKI) in perinatal asphyxia." (2014).
[16]. Jayashree .G, Dutta. A.K., Sarna M.S. Acute renal failure in asphyxiated newborns. Indian Pediatr 1991; 289 (1): 19-23.
[17]. Pammi V. Mohan, Pragnya M. Pai. Renal insult in asphyxia neonatorum. Indian Pediatr 2000; 37: 1102- 1106.