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FREQUENCY OF RENAL DERANGEMENT IN NEONATES WITH ASPHYXIA NEONATORUM

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ABSTRACT

Background: Asphyxia (insufficient oxygen supply) can lead to severe hypoxic ischemic organ damage in newborns followed by a fatal outcome or severe life-long pathologies like renal insufficiency. In some particular cases it is difficult to discriminate between mild and severe asphyxia. Advanced methodology to improved diagnosis of birth asphyxia and prediction of individual short and long term outcomes obligatory needs to be developed

Materials & Methods: A total of 264 patients were included in the study as per inclusion criteria were included in the study. Main outcome variable was renal derangement in asphyxia neonatorum. It was noted in the Performa as well.

Results: Total neonates included in this study were 264 (100%) having mean of weight was 2.54 kg with standard deviation was 0.50 and having mean of APGAR score was 4.43 with SD 1.66. Out of 264 neonates, it was noted that 189 (71.6%) neonates were suffered from **Renal derangement** in which 109 (57.7%) were males and 80 (42.3%) were females with mean of weight was 2.53kg and standard deviation was 0.5 and having mean of APGAR score was 4.44 with standard deviation was 1.667.

Conclusion: We conclude that renal failure is a significant problem in asphyxiated neonates with majority of babies having nonoliguric failure. Severity of renal function abnormality correlates well with degree of asphyxia.

Keywords: Asphyxia Neonatorum, Renal Derangement, Apgar score, Low Birth Weight.

INTRODUCTION:

Perinatal asphyxia is a global problem resulting in neonatal morbidity and mortality.[(1)] The incidence of perinatal asphyxia is 1.0 to 1.5% in most centers.[(2).] WHO reports that approximately 1 million children die worldwide every year from the diagnosis of birth asphyxia. In some particular cases it is difficult to discriminate between mild and severe asphyxia. Advanced methodology to improved diagnosis of birth asphyxia and prediction of individual short and long-term outcomes obligatory needs to be developed [3].

Most of time asphyxia damages the CNS. Complications associated with the central nervous system are attributed to oxidative stress, increased cerebral permeability, birth trauma and metabolic complications [4]. The oxidative stress markers as measured in blood are well recognized as the good predictors of poor outcomes in newborns with asphyxic deficits [(5, 6)]. Acute kidney injury is a common consequence of perinatal asphyxia, occurring in up to 56% of these infants. Therefore, the pathology specific biomarkers are of great clinical value being currently under extensive consideration by researchers [7]. Renal insufficiency may occur within 24 hours of a hypoxic ischemic episode, which if prolonged, may even lead to irreversible cortical necrosis [8]. Early

recognition of renal failure is important in babies with HIE to facilitate appropriate fluid and electrolyte management. Diagnosis of renal failure is difficult in neonates as many of the established clinical and biochemical parameters are unreliable in this age group [9]. S100B is considered as one of the most potent blood-markers, significantly increased in blood serum 24 h after severe birth asphyxia insult in newborns [10, 11].
Gupta BD conducted a study on renal failure in asphyxiated neonates, in his study majority of asphyxiated neonates had nonoliguric renal failure (26/33-78%) [12] while oliguric failure was seen in (7/33) 21% cases. The mean urine output being 1.494 ± 0.31mL/ kg/hour and 0.61± 0.28mL/kg/hr respectively in the 2 groups. Since oliguria was seen in only 6 babies the mean urine output in the study and control group.

MATERIAL AND METHODS:

Study Design:

This is a descriptive study conducted in department of pediatrics, Nishtar Hospital, Multan. This includes 264 patients with non probability consecutive sampling technique.

Inclusion Criteria:

1. Term Neonates delivered in Gynecology department of the hospital.
2. Neonates with asphyxia (as per defined in operational definition).
3. Both gender

Exclusion Criteria:

1. Renal insufficiency diagnosed by antenatal ultrasound.
2. Oligohydrominas diagnosed by antenatal ultrasound.
3. Babies with history of maternal addiction of analgesia and severe infection.

Data Collection Technique:

Informed consent was taken from patient's guardians before including patient's data in research and they were ensured about their confidentiality. Patient's telephonic contacts and addresses were taken. Blood sample for serum creatinin taken at 72 hours of life. Risks and benefits of treatment were discussed with patients/parents/Guardians. Weight of baby was noted at the time of birth by the researcher himself.

Data Analysis:

All the data entered and analyzed using computer software SPSS version 10. Mean and standard deviation was calculated for quantitative variables like APGAR score and birth weight. Frequency and percentage was calculated for qualitative variables like gender and renal derangement. Effect modifier like birth weight (≤ 2.5 kg and >2.5 kg), APGAR score and gender was controlled by stratification of data. Post stratification chi square test was applied.

A p value ≤ 0.05 was considered statistically significant.

RESULTS:

Total neonates included in this study were 264 (100%) having mean of weight was 2.54 kg with standard deviation was 0.50 and having mean of APGAR score was 4.43 with SD 1.66. Out of 264 neonates it was observed that 147(55.7%) were males with mean weight was 2.59 kg and standard deviation was 0.493 And 117(44.3%) were females with mean weight was 2.47 kg and standard deviation was 0.501.

Out of 264 neonates, it was noted that 189 (71.6%) neonates suffered from **Renal derangement** in which 109 (57.7%) were males and 80 (42.3%) were females with mean of weight was 2.53kg and standard deviation was 0.5 and having mean of APGAR score was 4.44 with standard deviation was 1.667. It was also observed that 75 (28.4%) neonates were not suffered from **Renal derangement** in which 38 (50.7%) were males and 37 (49.3%) were females with mean of weight 2.56 kg and standard deviation was 0.5 and having mean of APGAR score was 4.40 with standard deviation was 1.652.

When stratified analysis was done to see the effect modification it was noted that there was no significant effect of renal derangement on birth weight (≤2.5 kg and >2.5 kg) and gender having p values 0.650 and 0.301 respectively. But significant association of APGAR score with renal derangement was seen with p value 0.0001

Table- 1

Frequency distribution of Renal Derangement

(n=264)

Gender	Frequency	Percentage (%)
Male	**147**	**55.7**
Female	**117**	**44.3**
Total	**264**	**100.0**

Table-2

Frequency distribution of Renal Derangement

Renal Derangement	Frequancy	%
Yes	**189**	**71.6**
No	**75**	**28.4**
Total	**160**	**100.0**

Table- 3

Mean Birth weight and Apgar Score

(n=264)

Characteristics	Mean	SD
Birth weight	**2.54**	**0.50**
Apgar Score	**4.43**	**1.660**

Table 4:

Stratification of renal derangement and gender

Renal Derangement	Gender		Total
	Male	Female	
Yes	109	80	189
No	38	37	75
Total	147	117	264
p-value = 0.301			

Table 5:

Stratification of Renal derangement and Birth weight

Renal Derangement	Birth weight		Total
	2kg	3kg	
Yes	89	100	189
No	33	42	75
Total	122	142	264
p-value = 0.650			

Table 6:

Stratification of renal derangement and Apgar score

Renal Derangement	Apgar Score		Total
	1-4	5-7	
Yes	110	79	189
No	14	61	75
Total	124	140	264
p-value = 0.0001			

Table 7:

Stratification of renal derangement with respect to gender

Renal Derangement	Gender	Frequency	Percent
Yes	Male	109	57.7
	Female	80	42.3
	Total	189	100.0
No	Male	38	50.7
	Female	37	49.3
	Total	75	100.0

DISCUSSION:

Renal injury in birth asphyxia is a potential consequence of adaptive mechanism. Amongst the recognized complications i.e., acute tubular necrosis, renal vein thrombosis and renal failure, ARF is the commonest and carries a poor prognosis and may even result in permanent renal damage in up to 40% of survivors [8]. Urinary output was slightly less in neonates with severe birth asphyxia but it was statistically insignificant when compared with cases of mild and moderate asphyxia. But oliguria has been reported in higher number of neonates by other authors with figures ranging from 25% to 69.2% babies [9, 10].

Non-oliguric renal failure is a recognized entity secondary to perinatal asphyxia. Renal parenchymal injury in non oliguric as well as 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. But if damage to tubular epithelium is less severe there occurs decrease in fractional reabsorption which exceeds the decrease in single nephron GFR leading to polyurea in non-oliguric renal failure [11].

11.4% of our asphyxiated neonates showed both proteinuria and hematuria and babies with HIE II or III had more urinary anomalies. Proteinuria is disproportionately higher in the neonatal period than it is later in life with levels as high as 240 mg/m2/d in 1st 48 hours of life. In addition about 10% of neonates will have hematuria detected by dipstick and may have urinary red cell count up to 10 cells/mL [12]. Impaired tubular function after asphyxia leads to occurrence of significant tubular proteinuria and qualitative assessment of proteinuria by measuring p2-M-a low molecular weight protein to detect tubular injury has been proposed by various authors [13, 14]. Obstruction of tubular lumen and back leak mechanism contributed to increase in urea and creatinin levels in asphyxiated neonates and other authors too noted great correlation between severity of HIE and ischemic damage to the kidneys manifesting as ARF [15].

The capacity of sodium reabsorption is limited and if the load of sodium reaching the DCT increases significantly, reabsorption does not occur proportionately and the sodium load is excreted in the urine. Other contributing factors to hyponatermia may be occurrence of SIADH secondary to perinatal asphyxia and partial resistance to aldosterone [16]. Hyponatremia per se may lead to contraction of intravascular volume further reducing the renal functions [17]. The incidence of renal involvement observed in our study (47.14%) compared well with figures of other author [16, 17].

Sonographic abnormalities were seen in babies with biochemical evidence of renal dysfunction and 3 of them were oliguric. Four out of 5 babies with abnormal sonography expired. In our study no neonate remained oliguric by day 4-6 of life, which compares well with observations of Pertman, et al [8] who reported transient oliguria on

1st day in 23% of newborns and urine output increased to normal values on 3rd day of life. A reduction in number of functional nephrons caused by asphyxia and leading to ARF evokes compensatory hypertrophy of the residual nephrons thus leading to improved renal functions in early months of life. But whether subtle defects may persist, can be said only after long term follow-up and one must be cautious in prognosticating these neonates.

Brochieback reported that up to 40% of survivors may have decreased creatinin clearance, renal tubular acidosis or concentrating defect [18]. Oliguria, abnormal renal sonographic scan and hyponatermia were noted to be the ominous signs predicting mortality in our study. The limitation of our study has been our inability to check for residual renal tubular dysfunction, BP monitoring, evidence of RTA, urinary concentrating ability, and renal imaging which can provide information on residual subtle defect.

CONCLUSION:

Thus it can be concluded that birth asphyxia is a significant cause of ARF in asphyxiated neonates mostly having nonoliguric failure. Severity of renal function abnormality correlates well with degree of asphyxia. Oliguria, hyponatremia and abnormal sonographic scan are bad prognostic signs in renal failure secondary to birth asphyxia. Combination of dehydration, sepsis, shock and nephrotoxic drugs is not an uncommon situation in NICU. These lead to high incidences of neonatal failure. They are often reversible if identified and managed in time.

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