Original Article

# Frequency of Renal Derangement in Patients of Asphyxia Neonatorum



**Waseem Asghar, Rana Mubarik Ali, Uzma Latif and Farman Ali**

## ABSTRACT

**Objectives:**To find the incidence of Renal Derangement in Neonates with Asphyxia Neonatorum**.**
**Study Design:**Observational / descriptive study.
**Place and Duration of Study:**This study was conducted at the Department of Pediatric Medicine, Nishtar Hospital, Multan from January 2016 to January 2017.
**Materials and Methods:**A total number of 100% (n=264) neonates were included in this study, both genders. SPSS version 23 was used to analyze data, mean ± SD deviation was calculated for numerical variables and frequency (percentages) was calculated for categorical variables. Chi square test was applied to see the effect modification. P value ≤ 0.05 considered as significant.
**Results:** A total number of 100% (n=264) neonates were included in this study, both gender. Out of these 100% (n=264) patients, 71.6% (n=189) patients having Renal Derangement (creatinine > 0.8 mg/dl). When chi-square was applied to check the effect modification it was observed that gender, stratified Apgar score and stratified birth weight (≤2.5 kg and >2.5 kg) were significantly associated with Renal Derangement with P-values 0.000, 0.05 and 0.000 respectively.
**Conclusions:**Asphyxia is a major cause of Acute Renal Failure in Neonates, its outcome can be improved with early monitoring of patients renal parameters e.g urea and serum creatinine.
**Key Words:**Renal derangement, Asphyxia, Neonates, Creatinine.

**Citation of article:Asghar W, Ali RM, Latif U, AliF.Frequency of Renal Derangement in Patients of Asphyxia Neonatorum.Med Forum 2017;28(5):81-84.**



## INTRODUCTION

Asphyxia (inadequate supply of oxygen) can give rise to hypoxic ischaemic organ damage of variable severity in newborns resulting in severe life-long fatal outcome including renal insufficiency. We are in dire need of highly advanced techniques and methods in order to diagnose birth asphyxia, determine its severity and anticipate its outcomes destined to develop in future[1].

CNS is the most vulnerable system prone to develop hypoxic injury. Birth trauma, oxidative stress, metabolic complications and increased cerebral permeability are the different mechanisms contributing to complications resulting from asphyxial damage to central nervous system[2]. It is a renowned fact that poor outcome of newborn suffering from asphyxia deficits can be predicted to great extent by measuring oxidative stress marker level in the blood.[3,4]About 56% of these infants suffer from acute kidney injury making it a leading outcome of perinatal asphyxia.

Therefore, researchers have paid a great deal of attention to pathology specific biomarkers due to their clinical worth and applicability[5]. Renal deficiency can appear within a day of hypoxic ischaemic injury. If this change persists, it can induce irreversible kidney damage i.e., cortical necrosis[6].

Early suspicion and timely diagnosis of renal failure is of supreme importance in order to maintain fluid and electrolyte balance which in turn helps to keep a stable biochemical milieu. However, unreliability of routinely accepted clinical and established biochemical parameters add difficulty to the process of diagnosis making of renal failure in this age group[7]. Out of many blood markers, S100B is the most potent blood-markers. Its concentration increases significantly after 24 hour of severe birth asphyxia.[8,9]

A study conducted by Gupta B, focusing on the development of renal failure in asphyxiated neonates showed that majority (26 out of 33, approximating to 78%) of asphyxiated neonates had nonoliguric renal failure in contrast to oliguric failure which was reported in only 7 out of 33 patients mounting only to 21%[10].

## MATERIALS AND METHODS

After approval from ethical committee of Nishtar Hospital, Multan, 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. Serum creatinine was investigated by blood samples. Risks and benefits of treatment was discussed with patients/ parents/Guardians. Weight of baby was noted at the

Departmentof Cardiology, Ch. PervaizEllahi Institute of Cardiology, Multan.

Correspondence:Farman Ali, ICU Technologist, Ch. PervaizEllahi Institute of Cardiology, Multan.
Contact No: 0300 3658675
Email: Chfarmanali358@gmail.com

Received: March 11, 2017; Accepted: April 18, 2017

time of birth by the researcher himself. Neonates suffering renal insufficiency diagnosed by antenatal ultrasound, oligohydrominas diagnosed by antenatal ultrasound, babies with history of maternal addiction of analgesia and sever infection were excluded from our study. Renal function tests were done twice, first within 24 hours of birth and then again on 3rd day of life in the form of serum creatinine. Only those babies who had deranged renal parameter on 3rd day of life were undergone serum creatinine and other required tests on every other day until the recovery of the baby. The standard hospital protocol of conservative management was followed for neonates with renal failure. Other tests like ABGs (arterial blood gases), FENa+ (fractional excretion of sodium) and ECG were recommended only on the basis of individual requirement of patients. Serum creatinine level of > 0.8mg/dl was the criteria opted to label renal failure in asphyxiated newborn. Similarly newborns with APGAR score of ≤ 7 at five minutes according to APGAR scale (Annexure II) asphyxia were labelled as asphyxiated newborns. All the data entered and analyzed using computer software SPSS version 23. Mean and standard deviation was calculated for quantitative variables like APGAR score and birth weight (≤ 2.5 kg and >2.5 kg). 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

A total number of 100% (n=264) neonates were included in this study, both genders. Gender distribution showed that there were more males i.e., 55.7% (n=147) and 44.3% (n=117) were females. The mean APGAR score of the patients was 4.43±1.66. The mean birth weight of the patients was 2.54±0.50 kg. It was observed that out of these 100% (n=264) patients, 71.6% (n=189) patients having Renal Derangement (creatinine > 0.8 mg/dl) and 28.4% (n=75) have normal creatinine values. When patients were grouped in different categories with respect to Apgar score and birth weight it was seen that 23.9% (n=63) patients having APGAR score from 1 to 3 and a big majority of the patients 76.1% (n=201) having APGAR score from 4 to 7. It was also observed that 46.2% (n=122) patients were weighted of ≤2.5 kg and 53.8% (n=142) were weighted of >2.5 kg.

When chi-square was applied to check the effect modification it was observed that gender, stratified APGAR score and stratified birth weight (≤2.5 kg and >2.5 kg) were significantly associated with Renal Derangement with P-values 0.000, 0.05 and 0.000 respectively.

**TableNo.1:Demographic Variables**

| Characteristics | Frequency n=264 | Percentage (%) |
|---|---|---|
| **Gender** | | |
| Male | 147 | 55.7 |
| Female | 117 | 44.3 |
| **Renal Derangement** | | |
| Present | 189 | 71.6 |
| Absent | 75 | 28.4 |
| **Apgar Score** | | |
| 1-3 Apgar Score | 63 | 23.9 |
| 4-7 Apgar Score | 201 | 76.1 |
| **Stratified Duration of Perforated Ulcer** | | |
| **≤2.5 kg** | 122 | 46.2 |
| **>2.5 kg** | 142 | 53.8 |

**Table No.2: Inferential Results (n = 264)**

| Renal Derangement | Gender | | P – value |
|---|---|---|---|
| | Male (n = 147) | Female (n = 117) | |
| Yes (n = 189) | 121 | 68 | 0.000 |
| No (n = 75) | 26 | 49 | |

**Table No.3: Inferential Results**

| Apgar Score | Renal Derangement | | P Value |
|---|---|---|---|
| | No | Yes | |
| 1-3 Apgar Score | 12 | 51 | 0.05 |
| 4-7 Apgar Score | 63 | 138 | |
| Total | 75 | 189 | |

**Table No.4: Inferential Results**

| Birth Weight | Renal Derangement | | Total |
|---|---|---|---|
| | No | Yes | |
| 2 kg | 20 | 102 | 0.000 |
| 3 kg | 55 | 87 | |
| Total | 75 | 189 | |

## DISCUSSION

Acute renal failure in Perinatal asphyxia take place by two pathways i.e., reduced circulating blood volume and prolonged ischemia leading to pre renal impairment and acute tubular necrosis respectively[11]. Just like a double edge sword, renal injury and its complications including renal failure, renal vein thrombosis and acute tubular necrosis are the well-known end-results of compensative adaptive mechanisms triggered by birth asphyxia. Out of all the complications of renal damage, ARF is most common and severe one with poor prognosis. Possibility of irreversible renal damage is the destination of up to 40% of survivors of ARF[12].

In our study, 264 neonates of both genders were included. Gender distribution showed that there were

more males (55.7%) than female (44.3%). Their mean Apgar score was 4.43±1.66 and mean birth weight was 2.54±0.50 kg. 71.6% (189 out of 264) were suffering from Renal Derangement. Additionally, our study showed that gender, stratified Apgar score and stratified birth weight (≤2.5 kg and >2.5 kg) were significantly associated with Renal Derangement.

In present study, the incidence of ARF is 71.6% which is comparable with the study done by Pammi et al[13], Gopal et al[14] and Karlowicz et al[15]. Only few studies conducted by Gopal G et. Al[14] and Jayashree G et. Al[16] contradict our finding by reporting lower incidence. Only one study done by Girish et al. had narrated a little higher incidence of ARF than that of our study. Although data is not shown but non oliguric ARF is the predominant type of ARF encountered in asphyxiated newborn in our study similar to most of the studies including the findings presented by Gupta et al.[10] but it was not comparable with the findings of Jayashree et al. in which oligouric ARF was the predominant type. Biochemical parameters used to define renal failure in our study were similar to the parameters used in a research conducted by Jayashree et al. in the present study. In addition to these studies our findings are supported by previously conducted studies. In our study, birth weight, Apgar score and stages of HIE were found to be related with higher incidence of ARF similar to the findings of Gopal et al.

Our criteria for diagnosis enabled us with early recognition of AKI and timely intervention. Thus preventing the conversion of pre-renal AKI to intrinsic AKI. This also signifies the importance of fluid bolus and fluid electrolyte balance in preventing permanent renal damage. Though kidney occupies the title of one of the best oxygenated organ but redistribution of blood flow to other more vital organs makes it susceptible to hypoxicischemic injury and in turn lead to temporary loss of renal concentrating ability.

Asphyxiated neonates have reduced creatinine clearance compared to healthy neonates. The reduced creatinine clearance is in direct relation with the severity of HIE. Only one study[17] in present literature contradicts this general concept by reporting increase creatinine clearance in asphyxiated newborns. This contradiction might be attributed to the fact that newborns with severe asphyxia and those dying within a week of their birth were not included in that study. The degree of deterioration or improvement of renal functions is found to give a better view of the prognosis of the patient.

A study conducted by Brochieback[18] narrated the fact that deranged renal functions like concentrating defect, renal tubular acidosis or reduced creatinine clearance can develop up to 40% of survivors. Ominous signs that can predict mortality are oliguria, hyponatermia and abnormal renal ultra sound. Our study was limited due to our inability to monitor BP, residual renal tubular dysfunction, RTA, urinary concentrating ability, and renal imaging.[19]

In our study, raised level of plasma creatinine concentration for at least two days was used as a single criteria for labeling an asphyxiated child with acute renal failure. Oliguria was not included in the criteria for the fear of neglecting non-oliguric acute renal failure in asphyxiated infants. In order to overcome the error produced by chromogens present in plasma in modified Jaffe technique, high performance liquid chromatography was used to determine plasma creatinine concentrations[20].

## CONCLUSION

Asphyxia is a major cause of acute renal failure in neonates, its outcome can be improved with early monitoring of patients renal parameters e.g urea and serum creatinine.

**Conflict of Interest:** The study has no conflict of interest to declare by any author.

## REFERENCES


1. Golubnitschaja O, Yeghiazaryan K, Cebioglu M, Morelli M, Herrera-Marschitz M. Birth asphyxia as the major complication in newborns: moving towards improved individual outcomes by prediction, targeted prevention and tailored medical care. EPMA J 2011;2(2):197-210.
2. Shah S, Goel A, Padhy M, Bhoi S. Correlation of oxidative stress biomarker and serum marker of brain injury in hypoxic ischemic encephalopathy. Int J Med Appl Sci 2014;3(1):106-15.
3. Mondal N, Bhat BV, Banupriya C, Koner BC. Oxidative stress in perinatal asphyxia in relation to outcome. Ind J Pediatr 2010;77(5):515-7.
4. Coulibaly G, Ouédraogo-Yugbaré S, Kouéta F, Yao L, Savadogo H, Dao L, et al. Perinatal asphyxia and acute renal insufficiency in Ouagadougou. Arch de Pediatr2016;23(3): 249-54.
5. Durkan AM, Alexander RT. Acute kidney injury post neonatal asphyxia. The J Pediatr2011;158(2):e29-e33.
6. Walker RG, Hewitson TD, Becker GJ. Chronic Interstitial Nephritis 24. Core Concepts in Parenchymal Kidney Disease 2013;341.
7. Aridas JD, Yawno T, Sutherland AE, Nitsos I, Ditchfield M, Wong FY, et al. Detecting brain injury in neonatal hypoxic ischemic encephalopathy: Closing the gap between experimental and clinical research. Experiment Neurol2014;261:281-90.
8. Zhou W, Li W, Qu L-H, Tang J, Chen S, Rong X. Relationship of plasma S100B and MBP with brain damage in preterm infants. IntJ Clin Experiment Med 2015;8(9):16445.

9. Florio P, Abella R, Marinoni E, Di Iorio R, Volti GL, Galvano F, et al. [Frontiers in Biosci S2, 47-72, January 1, 2010] Biochemical markers of perinatal brain damage. Frontiers in Biosci2010;2:47-72.
10. Gupta B, Sharma P, Bagla J, Parakh M, Soni J. Renal failure in asphyxiated neonates. IndPediatr 2005;42(9):928.
11. Jetton JG, Askenazi D. Acute Kidney Injury in the Newborn. Kidney and Urinary Tract Diseases in the Newborn: Springer 2014;287-306.
12. Mao H, Katz N, Kim JC, Day S, Ronco C. Implantable left ventricular assist devices and the kidney. Blood purification 2014;37(1):57-66.
13. mohanan P, Pai PM. Renal insult in asphyxia.Neonatorum2000.
14. Gopal G. Acute Kidney Injury (AKI) in perinatal asphyxia. 2014.
15. Karlowicz MG, Adelman RD. Nonoliguric and oliguric Acute Renal Failure in asphyxiated term neonates. PediatrNephrol 1995;9(6):718-22.
16. Jayashree G, Dutta A, Sarna M, Saili A. Acute renal failure in asphyxiated newborns. IndPediatr 1991;28(1):19-23.
17. Dickinson H, Ellery S, Ireland Z, LaRosa D, Snow R, Walker DW. Creatine supplementation during pregnancy: summary of experimental studies suggesting a treatment to improve fetal and neonatal morbidity and reduce mortality in high-risk human pregnancy. BMC pregnancy and childbirth 2014;14(1):150.
18. Brocklebank J. Renal failure in the newly born. Arch Dis Childhood 1988;63(8):991.
19. Kramer W, Wizemann V, Mandelbaum A, Ritz E. Cardiologic problems in uraemic patients. In "Oxford Textbook of Clinical Nephrology" ed Cameron S, Davison AM, p 1264-1278. Oxford Uni versity Press, Oxford; 1992.
20. Peake M, Whiting M. Measurement of serum creatinine-current status and future goals. ClinBiochem Rev 2006;27(4):173.