STUDY OF RENAL FUNCTION IN NEONATAL ASPHYXIA

Meena Varma¹, Prachi Paliwal², M.K.S. Shaikh³, Swati Mulye⁴, Manoj Narayan Paliwal⁵

HOW TO CITE THIS ARTICLE:

Meena Varma, Prachi Paliwal, MKS Shaikh³, Swati Mulye, Manoj Narayan Paliwal. "Study of renal function in neonatal asphyxia". Journal of Evolution of Medical and Dental Sciences 2013; Vol2, Issue 38, September 23; Page: 7361-7365.

ABSTRACT: BACKGROUND/AIMS: Birth asphyxia occurs when a baby does not receive enough oxygen before, during or after birth. It is an insult to the fetus or newborn due to lack of oxygen (hypoxia) and /or a lack of perfusion (ischemia) to various organs. Fetus totally depends for its oxygen supply and other nutrients on the blood supplied through placenta. In any case, if blood supplied through placenta is hampered, it leads to asphyxial injury. We performed this study to determine the incidence of renal failure in birth asphyxia by estimating serum creatinine, blood urea and blood urea nitrogen / creatinine ratio on I & III day of life. **MATERIAL AND METHODS:** The study included 50 asphyxiated neonates and 25 healthy neonates as controls. Blood urea and serum creatinine were estimated by Vitros 950 dry chemistry autoanalyzer. **RESULT:** Blood urea and serum creatinine were significantly higher in asphyxiated babies compared to control groups ($p < 0.001$). Blood Urea nitrogen / Creatinine ratio was significantly higher in asphyxiated babies compared to control groups ($p < 0.001$) on day 3 but insignificant on day 1. We conclude that renal failure is a significant problem in asphyxiated neonates.

KEY WORDS: Birth asphyxia, Renal failure, Serum creatinine, Blood urea nitrogen, Blood urea.

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.

Birth asphyxia occurs when a baby does not receive enough oxygen before, during or after birth. It is an insult to the fetus or new born due to lack of oxygen and or lack of perfusion to various organs.⁽³⁾ Fetus totally depends for its oxygen supply and other nutrients on the blood supplied through placenta. In any case, if blood supplied through placenta is hampered, it leads to asphyxial injury.

Prolonged hypoxia in perinatal asphyxia causes decreased cardiac output, cerebral blood flow is compromised and combined hypoxic ischemic insult produces failure of ATP production with accumulation of ADP and AMP. Catabolism of these products leads to increased uric acid production with increased urinary excretion. Hypoxia and ischemia produced can cause damage to almost every tissue and organ of the body with common involvement of kidneys, brain, heart and lungs.⁽⁴⁾

Target organs of perinatal asphyxia are the brain, heart, lungs, kidneys, gut and bone marrow. The most frequent abnormalities involving kidneys (50%) followed by CNS (28%), cardiovascular (25%) and pulmonary system (23%)⁽⁵⁾. Thus there is evidence of multiorgan system dysfunction in the immediate neonatal period.⁽⁶⁾

The kidneys are very sensitive and immature at birth and slowly mature as the age increases. Kidneys are very sensitive to oxygen deprivation; prolonged hypoxic ischemic episode can lead to irreversible cortical necrosis with onset of acute renal failure (ARF).

Renal failure refers to temporary or permanent damage to the kidneys that result in loss of normal kidney function. It may be acute or chronic.⁽⁷⁾

Acute renal failure in the newborn has been defined as urine output less than 1 ml/kg/hour and blood urea nitrogen more than 20 mg % and serum Creatinine of more than 1 mg %.⁽⁸⁻⁹⁾

Diagnosis of renal failure is difficult in neonates as many of the established clinical and biochemical parameters are unreliable in this age group.

We performed this study to determine the incidence of renal failure in Birth asphyxia and to correlate renal failure with severity of asphyxia.

MATERIAL AND METHODS: The present study was carried out in department of Biochemistry in active collaboration with Neonatal intensive care unit of department of pediatrics, S.A.I.M.S Medical College and PG institute, Indore.

- Consent from the institutional Ethical Committee was also taken to carry out the above research.

Inclusion Criteria: 75 newborns admitted to department of pediatrics and its neonatal unit was enrolled for the present study. The enrolled babies were further divided into study group and control group.

Control group --- The control group had 25 healthy neonates who were free from any systemic disease.

Study group --- The study group comprised of 50 asphyxiated neonates.

Exclusion Criteria: Predefined exclusion criteria for both the groups were congenital anomalies, tumors, maternal drug addiction, severe infections and congenital mental disorders.

Clinical Examination: A detailed clinical examination was carried out as soon as the neonates were admitted. Gestational age⁽¹⁰⁾, birth weight, relevant perinatal history; findings on physical examination were recorded on a pre-designed proforma.

Laboratory Investigation: The blood collected was allowed to clot and then centrifuged to obtain serum for estimation of biochemical parameters.

1. **The investigations included:** Blood urea and Serum Creatinine estimated by Vitros 950 dry chemistry autoanalyzer
2. **BUN** – Calculated parameter (BUN= blood urea * 2.14)

Renal Function Parameters: Blood urea and serum Creatinine were monitored initially within 24 hrs of birth and then on 3rd day of life. Criteria adopted for labeling as asphyxiated neonate as having renal failure were urine output < 0.5ml/kg/hr., blood urea > 40 mg/dl and serum Creatinine > 1 mg/dl.

These criteria were applied on day 3 of life and the criteria when fulfilled were considered as indication of renal failure.

Statistical Analysis: Values have been expressed as Mean $\pm$ SD. The data were compiled and analyzed using descriptive statistics using student t test. P<0.05 was considered as significant.

RESULTS: Total 50 asphyxiated babies and 25 healthy non- asphyxiated babies were included in the study. In this study out of 75 neonates, 45 were males and 30 females. Average gestational age of the study group was 36.92 weeks and that of the control group was 37.25 weeks. Mean birth weight of

the study group was 2.45 Kg and that of the control group was 2.18 Kg. Number of vaginal deliveries in study group were 20(40%) and LSCS were 30 (60%) while in the control group number of vaginal deliveries were 16 (64%) and LSCS were 9 (36%).

Blood urea and serum Creatinine were significantly higher in asphyxiated babies compared to control group ($p<0.001$). (Table 1)

Blood urea and serum Creatinine were significantly higher in asphyxiated babies on day 3 of life as compared to day 1. ($p<0.001$) (Table 3)

Blood urea nitrogen / Creatinine ratio was also significantly higher in asphyxiated babies compared to control group ($p<0.001$) on day 3, but was found insignificant on day 1 ($p<0.10$) (Table 2). Of the 50 asphyxiated babies in our study group, 27(54%) babies had ARF and 23 (46%) babies had normal renal functions.

DISCUSSION: Renal injury in Birth asphyxia is a potential consequence of adaptive mechanism. Amongst the recognized complications, ARF is commonest and carries a poor prognosis and even may result in permanent renal damage in upto 40% of survivors ⁽¹¹⁾.

Jayashree et al (1991) in his study on 30 newborns with severe birth asphyxia and 30 normal newborns observed that 43% of asphyxiated babies developed ARF.

M. Gary Karłowicz et al (2004) studied 66 neonates and found ARF occurred in 61% of infants with asphyxia.

In the present study, out of 50 asphyxiated babies, 54% of infants had ARF. ⁽¹²⁾

Oliguria is seen in only 3 neonates in our study but it has been reported in higher numbers of neonates by other authors with figures ranging from 25% to 69.2% babies.

Jayashree et al (1991) in their study on 30 newborns with severe birth asphyxia observed that 62.2% babies had oliguric renal failure.

M. Gary Karłowicz et al (2004) studied 66 neonates and found that ARF associated with severe asphyxia was predominantly non-oliguric.

Gupta B.D. et al (2005) in his study on 70 asphyxiated babies and 28 healthy controls found that blood urea and serum creatinine were significantly higher in asphyxiated babies compared to control group. ⁽¹³⁾

In the present study, blood urea and serum creatinine were significantly higher in asphyxiated babies on day 3 and as compared to control group ($p<0.001$). This can be explained on the fact that in the first 48 hours of life these levels are reflection of maternal renal functions. In normal babies there is subsequent fall in blood urea and creatinine, whereas in cases with renal damage these levels rise above normal. ⁽⁴⁾ Our observation is in close approximation to those reported by Aggarwal et al (2005) and Gupta B.D. et al (2005).

CONCLUSION: Thus it can be concluded that birth asphyxia is a significant cause of ARF in asphyxiated neonates. The renal function tests can be useful to establish ARF diagnosis and predict outcome especially on day 3 of life. Despite tremendous advancements in medicine and current supportive techniques of the patient with ARF, death occurs from collapse of other body systems. Birth asphyxia is still common, more so in developing countries where obstetric and newborn resuscitation facilities are not universally available yet. Combination of dehydration, sepsis, shock

ORIGINAL ARTICLE

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.

	Study gr	Control gr	p Value
Blood Urea(mg/dl)	(n=50)48.4 ± 15.23	(n=25)34.2 ± 4.16	<0.001
Serum creatinine(mg/dl)	(n=50)1.6 ± 0.83	(n=25)0.54 ± 0.14	<0.001

TABLE 1 – Urea and Creatinine Levels (mean ± SD) on Day 3 in Study and Control Group

There is significant difference in biochemical parameters of the two groups. p value <0.001 is statistically highly significant in study group as compared to controls.

	Study gr	Control gr	p Value
BUN/Crt.(mg/dl) (Day 1)	(n=50)17.4 ± 5.35	(n=25)17.4 ± 3.6	<0.10
BUN/Crt.(mg/dl) (Day 3)	(n=50)30.8± 8.61	(n=25)16.7 ± 6.10	<0.001

TABLE 2 – BUN/ Crt. ratio (mean ± SD) on Day 1 and day 3 in Study and Control Group

There is significant difference (p<0.001) on day 3 but is insignificant on day 1(p<0.10)

	Day I	Day III	p value
Blood Urea (mg/dl)	(n=50) 42.0 ± 14.31	48.4 ± 15.23	< 0.001
Creatinine (mg/dl)	(n=50) 1.2 ± 0.64	1.6 ± 0.83	< 0.001

TABLE 3 - Urea and creatinine levels (mean± SD) on day 1 and 3 in study

There is significant difference in biochemical parameters of the two groups. p value <0.001 is statistically highly significant in study group on day 3 as compared to day 1.

FIGURE - Urea and creatinine levels (mean) on day 1 and 3 in study:

REFERENCES:

1. Volpe JJ. Hypoxic ischemic encephalopathy. In: Volpe JJ. Ed. Neurology of newborn. 4th edition Philadelphia: WB Saunders; 2001:217-394.
2. Aurora S, Synder E Y, Perinatal asphyxia. In Cloherty J P. Manual of neonatal care. 4th edn. New York: Lippincott; 1997, pp 536-555.

3. Stapleton FB, Jones DF, Green R S: Acute renal failure in neonates. *Pediatr Nephrol* 1987; 1:314-320.
4. Varma Vandana, Varma Amit, Varma Meena, Bharosay Anuradha, Bharosay Vivek, Varma Deepak. Study of basic biochemical and haematological parameters in perinatal asphyxia and its correlation with Hypoxic ischemic encephalopathy staging. *Journal of advance researches in biological sciences* 2011; 3(2):79-85.
5. Birth Asphyxia University of California. San Francisco, Reviewed by health care specialist at USCF Children Hospital. Last updated Sept. 13, 2006.
6. 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.
7. Norman Jones AS, James E, Bland H. Groshong T. Renal failure in the newborn. *Clin Ped* 1979; 18: 286-290.
8. Anand SK, Northway, JD, Cruss, FG: Acute renal failure in newborn, infants. *J. Pediatr*, 1978; 92:985-8.
9. Rahman N, Boineau FG, Lewy JE: Renal failure in perinatal period. *Clinics in Perinatology* 1981; 8: 241-249.
10. F. Bruder Stapleton, Deborah P. Jones, and Robert S. green. (1) Pediatric Research Laboratory, University of Tennessee, 956 Court Avenue, 38163 Memphis, TN, USA. (2) Lebonheur Children's Medical Centre, University of Tennessee, 956 court avenue, 38163 Memphis, TN, USA. *Pediatr Nephrol* 1987; 1(3): 314-320.
11. Singh M. Care of Newborn. 5th Ed. New Delhi: Sagar Publication; 1999: pp 113-114.
12. M Gary Karlowicz, Raymond D Adelman. Non oliguric and oliguric renal failure in asphyxiated term neonates. *Pediatric Nephrology* 2004:718-722.
13. Gupta BD, Sharma P, Bagla J, Parakh M, Soni JP. Renal failure in asphyxiated neonates. *Indian paediatr* 2005; 42; 42:928-34.

AUTHORS:

1. Meena Varma
2. Prachi Paliwal
3. M.K.S. Shaikh
4. Swati Mulye
5. Manoj Narayan Paliwal

PARTICULARS OF CONTRIBUTORS:

1. Professor, Department of Biochemistry, SAIMS Medical College & PG Institute, Indore.
2. PhD Scholar, Department of Biochemistry, SAIMS Medical College & PG Institute, Indore.
3. Professor and H.O.D, Department of Biochemistry, SAIMS Medical College & PG Institute, Indore.
4. Professor and H.O.D, Department of Pediatrics, SAIMS Medical College & PG Institute, Indore.

5. Assistant Professor, Department of Biochemistry, RD Gardi Medical College, Surasa, Ujjain.

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Mrs. Prachi Paliwal,
C/O Sonu Saluja,
35, Goldan Palace Colony,
Near Basantpuri, AB road,
Indore, M.P. 452012.
Email- prachi.paliwal@rediffmail.com
Semu_jayu@yahoo.co.in

Date of Submission: 11/09/2013.
Date of Peer Review: 12/09/2013.
Date of Acceptance: 14/09/2013.
Date of Publishing: 23/09/2013