

Assessment Of Renal Functions In Neonates Of Perinatal Asphyxia

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ABSTRACT

BACKGROUND: prospective case controlled study was conducted in the NICU of a tertiary level teaching hospital for assessment of renal functions in asphyxiated neonates and to correlate severity and type of renal failure with hypoxic ischemic encephalopathy (HIE) grading of the neonates. 64 neonates for perinatal asphyxia and 64 babies selected randomly from non asphyxiated babies for the control group. Blood samples were taken for measurement of serum urea, creatinine, sodium and potassium levels on 1st and 3rd day of life. If any abnormality detected, values were repeated every alternate days till it become normal. Blood urea and serum creatinine were significantly higher in asphyxiated babies compared to the control group (P<0.001). Biochemical derangements correlated well with HIE staging. Among 64 cases 28 (43.75%) had elevated levels of urea and creatinine on day 1 [Mean urea (43.21± 23.08), creatinine (1.14 ± 0.57)], 36 (56.25%) had elevated levels of urea and creatinine on day 3. Mean urea (58.06 ± 28.52) and creatinine (1.24±0.5) were significantly higher on day 3 (p value<0.05) in study group as compared to control. Mean urea and creatinine levels showed increasing trend with degree of severity of hypoxic ischaemic encephalopathy. Eighteen babies with perinatal asphyxia developed renal failure (56.25%). 18 had Hyponatremia on day 1 (56.25%), 3 of them had value < 125 meq/l. Though, mean sodium and potassium level was within the normal limit, the value of potassium was higher among cases than controls.

Key words: Perinatal asphyxia, Hypoxic Ischaemic Encephalopathy, Renal failure.

INTRODUCTION

Perinatal asphyxia will occur due to gas exchange and oxygen transportation disorders at birth that result lack of oxygen supply and difficulties in carbon – di-oxide expiration. This lack of oxygen supply in brain gives rise to hypoxic ischemic injury to central nervous system; the clinical manifestation of this injury is termed as hypoxic ischemic encephalopathy. Almost any organ can be affected, but the brain, myocardium, kidneys and bowel appears to be most sensitive to severe damage. Renal system is involved in 50% of the cases. Conversely, perinatal asphyxia producing ischemia is the commonest cause of renal failure in neonates. The kidneys of neonates are particularly susceptible to hypoperfusion, because of the physiologic characteristics of neonatal kidneys; high renal vascular resistance,

high plasma renin activity, low glomerular filtration rate, decreased intercortical perfusion, and decreased reabsorption of sodium in the proximal tubules are the susceptibilities of the kidneys in the first days of a neonate. Thus, newborn infants are vulnerable to acute tubular necrosis or cortical necrosis. In term babies, the concentration of serum creatinine normally rises somewhat in the first 24 to 36 hours after birth, subsequently decreasing and stabilizing at about 0.4 mg/dL (35.4 μmol/L) by 5 days of age¹⁰. A clearly elevated value beyond the normal range indicates decreased glomerular function. Urine output is another key indicator of renal function. Commonly, ARF is suspected when oliguria is present, defined as a period during which urine output is less than 0.5 mL/kg per hour.

MATERIALS AND METHODS

The study was undertaken in tertiary level care hospital over a period of 24 months. All neonates were resuscitated as per Neonatal Resuscitation Program (NRP) guidelines. Inclusion criteria were apgar score ≤ 6 at 1 minute and gestational age of ≥37 weeks. Exclusion criteria were

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babies with congenital malformation, gestational age < 37 wks, babies who developed early or late onset sepsis, babies who developed neonatal hyperbilirubinemia and babies born with meconium stained liquor. Babies with perinatal asphyxia are staged by Sarnat and Sarnat Scoring System. After admission appropriate measures were taken for initial stabilization for asphyxiated babies. Detailed history and systemic examinations were recorded in a pre-designed proforma. Severely affected babies were given IV fluids, oxygen initially. Patients who were stable clinically and lab values are normal, gavage or katori spoon feeding started with expressed breast milk and as the condition improves shifted to breast feeding. Control group was allowed breast feeding. Disposable plastic urine bag was used to collect urine sample for different tests & for measurement of 24hrs urine output. Blood samples were taken from peripheral veins for measurement of serum urea, creatinine, and sodium, potassium levels on 1st & 3rd day. If any abnormality detected, values were repeated every alternate day till it became normal. In some of the cases who developed renal failure Fractional Excretion of Sodium (FENa) % were estimated to determine intrinsic type of renal failure. $FENa = 100 \times (U Na \times P Cr) / (P Na \times U Cr)$. Criteria for renal failure was defined as urine

output: <0.5ml/kg /hr (Oliguria), blood urea: >40mg/dl, serum creatinine: >1mg/dl and presence of significant hematuria or proteinuria. Any three of these criteria on day 3 of life when fulfilled, is to be considered as indication of renal failure.

Z test for mean is applied for comparison between cases and controls. We have applied t-test of Mean to compare between above groups. A p value of < 0.05 was considered as statistically significant.

RESULTS: We had a total of 64 study subjects and 64 in control groups. Among cases 59.37% were males and 40.63% were females. There were 62.5% males and 37.5% were females in the control group. Mean birth weight in study group was 2.62 ± 0.4 kg and in control group

2.56 ± 0.35 kg. In our study, 18 patients were HIE 3, 26 in HIE 2 and 20 in HIE 1 category. 14 patients needed resuscitation beyond 5 minutes. 30 patients had Apgar ≤ 4 at 5 minutes. The mean urine volume gradually decreased as HIE grading increased. Mean 24 hours urine volume showed negative correlation with HIE stages which was statistically significant ($r = -0.4661, p < 0.05$). The values of mean 24 hours urine volume decreased when HIE grade increased and negative correlation noted which was significant ($r = -0.4437, p < 0.05$).

	Urine Vol in 24 hrs (ml) Mean \pm SD
Study Group D 1	38.59 \pm 11.37
HIE 1	46.66 \pm 10
HIE 2	37.30 \pm 8.56
HIE 3	33 \pm 12.51
Control group D1	78.12 \pm 19.03
Study group D3	52.18 \pm 18.79
HIE1	62.77 \pm 11.75
HIE 2	53.07 \pm 17.74
HIE 3	41.5 \pm 20.82
Control group D3	82.34 \pm 13.19

Mean urea and creatinine values were higher in study group as compared to control. Statistical analysis showed this to be significant ($p < 0.05$) for both urea and creatinine. The values were on higher side on 3rd day as compared to day 1. Mean urea level on day 1 increased when hypoxic ischaemic encephalopathy staging was increased, though this correlation was not statistically significant (r value = 0.3086, $p > 0.05$). Day 3 urea level rose significantly when HIE staging increased ($r = 0.5334, p < 0.05$). Mean creatinine level on day 1 was found to be increased with higher HIE grading and correlation coefficient value was significant ($r = 0.4471, p < 0.05$). Day 3 creatinine level rose significantly with increase in HIE stage ($r = 0.6033, p < 0.05$).

Table 1: Blood Urea and Creatinine levels on day 1 and 3 in study and control Groups

	Study Group N=64 Mean \pm SD	Control Group N=64 Mean \pm SD	z test for mean (p value)
Blood Urea Day 1 (mg/dl)	43.21 \pm 23.08	28.06 \pm 4.08	3.659 (<0.05)
Serum Creatinine Day 1 (mg/dl)	1.14 \pm 0.57	0.73 \pm 0.11	3.9001 (<0.05)
Blood Urea Day 3 (mg/dl)	58.06 \pm 28.52	28.46 \pm 3.58	5.825 (<0.05)
Creatinine Day 3 (mg/dl)	1.24 \pm 0.5	0.73 \pm 0.09	5.679 (<0.05)

Table 2: Urea and Creatinine levels in relation with HIE Staging

	Blood Urea (mg/dl)		Serum Creatinine (mg/dl)	
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
	Day 1	Day 3	Day 1	Day 3
Study Group	43.21 ± 23.08	58.06 ± 28.52	1.14 ± 0.57	1.24 ± 0.5
HIE 1 (n=18)	37.11 ± 23.24	40.22 ± 25.23	0.86 ± 0.2	0.83 ± 0.33
HIE 2 (n=26)	38.38 ± 15.48	54.38 ± 20.11	1.05 ± 0.51	1.23 ± 0.40
HIE 3 (n=20)	55 ± 28.46	78.9 ± 29.59	1.52 ± 0.70	1.61 ± 0.49
Control Group	28.06 ± 4.08	28.46 ± 3.58	0.73 ± 0.11	0.73 ± 0.09

Table 3: Correlation of levels of urea and creatinine with HIE staging

	Day 1	p- value	Day 3	p-value
S. Urea	0.3086	>0.05	0.5334	<0.05
S. Creatinine	0.4471	<0.05	0.6033	<0.05

On day 1 and 3 mean sodium level increased in control group than study group, whereas mean potassium value was higher in the study group. These changes were statistically significant (p<0.05). Variation in serum sodium levels between HIE 1 and HIE 2 was not significant, but noticeable change of mean sodium level was observed in HIE 3 in comparison to stage 1 and 2. No noticeable change of potassium level was seen on day 1 with different stages of HIE. But the mean value of potassium on day 3 increased proportionately with increase in grade of HIE. Day 3 mean sodium value showed significant negative correlation (r = -0.5227, p<0.05) with degree of HIE. Day 3 mean potassium levels showed positive correlation with increase in degree of asphyxia but was not statistically significant (r = 0.3350, p >0.05).

Table4: Comparison of Serum Sodium and Potassium values on day1 and day 3

	Study Group Mean ± SD	Control Group Mean ± SD	z test for mean (p-value)
Serum Sodium (meq /L) Day 1	132.78 ± 6.9	136.65 ± 2.08	3.037 (<0.05)
Serum Potassium Day 1 (meq/L)	5.14 ± 1.45	3.99 ± 0.36	4.354 (<0.05)
Serum Sodium Day 3 (meq /L)	132.15 ± 4.74	136.43 ± 2.44	4.541 (<0.05)
Serum Potassium Day 3 (meq/L)	5.24 ± 1.39	4.009 ± 0.38	4.832 (<0.05)

Table 5: Serum Sodium and Potassium values in relation with HIE staging

	Serum Sodium		Serum Potassium	
	Day 1 Mean ± SD	Day 3 Mean ± SD	Day 1 Mean± SD	Day 3 Mean ± SD
Study Group	132.78 ± 6.9	132.15 ± 4.74	5.14 ± 1.45	5.24 ± 1.39

HIE 1	134.11 ± 6.91	135.22 ± 3.66	5.22 ± 1.44	4.37 ± 0.79
HIE 2	134.4 ± 5.97	132.53 ± 5.50	4.97 ± 1.10	5.57±1.83
HIE 3	129.5 ± 7.53	128.9 ± 1.96	5.28 ± 1.94	5.60 ± 0.77
Control Group	136.65 ± 2.08	136.43 ± 2.44	3.99 ± 0.36	4.0 ± 0.38

DISCUSSION: In this study blood urea and creatinine values were significantly higher in asphyxiated babies in comparison to controls both in day 1 and day 3. In our study significant positive correlation found on day 3 urea, creatinine values with increase severity of birth asphyxia. We found 56.25% babies developed elevated urea & creatinine levels on day 3. In our study, Day 1 and Day 3 mean 24 hours urine volume was significantly lower than the control group. Also the mean urine volume was gradually decreasing with increased severity of birth asphyxia and it was statistically significant. Among these 21.87% had oliguria. In the oliguric group 4 cases (57.15%) belonged to hypoxic ischaemic encephalopathy (HIE) stage 3, 3 cases (42.85%) belonged to HIE stage 2 and none of HIE stage 1. So here overall prognosis of patients with oliguric renal failure is bad, also oliguric type of renal failure mainly associated with severe birth asphyxia; though statistical analysis is not significant. Hyponatremia is one of the complications of birth asphyxia which may or may not be associated with renal failure. Serum sodium level decreased as the severity of asphyxia increased. There was no significant difference in serum potassium level among the cases of birth asphyxia without renal failure and controls. So prompt detection and early supportive management of renal failure in asphyxiated babies is important.

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