Renal Status (BU, UO) in Birth Asphyxia

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ABSTRACT

Introduction: Perinatal asphyxia is one of the common causes of neonatal mortality and morbidity and it affects various organs depending upon severity of asphyxia. This study was undertaken to evaluate and monitor the renal functions (urinary output and blood urea) in birth asphyxia and to correlate renal function with severity of birth asphyxia.
Material and methods: A prospective cross sectional study conducted on newborns delivered in Obstetrics and Gynaecology and admitted to neonatology section of Department of Pediatrics, Govt Medical College, Rajindra Hospital, Patiala. 100 asphyxiated newborns were taken as cases and 35non-asphyxiated newborns were taken as control group.
Results: Among the 100 asphyxiated newborns, 40% were having mild birth asphyxia, 36 % moderate and 24% were having severe birth asphyxia. Out of 100 newborns 45 were preterms and 55 were term newborns with weight of <1000gms in 6 babies, 60 babies with weight of 1000-2500 and > 2500 gms were 34 babies. In present study, mean BUN level of severely asphyxiated babies on day 1 and 3 was significantly higher than that of mildly and moderately asphyxiated babies ($p<0.01$). Mean urinary output in severely asphyxiated babies on day 1 was significantly lower as compared to mean UOP in mildly and moderately asphyxiated babies ($p<0.01$).
Conclusion: Perinatal asphyxia is important cause of affecting renal functions. BUN on day 1st and 3rd and UOP day 1, day 2 and day 3 were significantly different among mildly, moderately and severly asphyxiated babies, concluding that mean levels of BUN and UOP are related to grade of asphyxia.

Keywords: perinatal asphyxia,renal failure, Blood urea nitrogen, hypoxic ischemic encephalopathy, neonate.

INTRODUCTION

World Health Organization (WHO) has defined Birth asphyxia as "Failure to initiate and sustain breathing at Birth."

Perinatal asphyxia is about 1 to 1.5% of live births in most centres and is inversely related to gestational age and birth weight. It occurs in 0.5% of live born infants more than 36 weeks gestational age and accounts for 20 % of perinatal deaths.[1]

Asphyxia occurs when the organ of gas exchange fails. When this occurs, arterial carbon dioxide partial pressure ($PaCO_2$) rises, and arterial oxygen partial pressure (PaO_2) and pH falls.[2]

In the presence of a hypoxic –ischemic challenge, reflexes are initiated, causing shunting of blood to the brain, heart and adrenals, and away from the lungs, gut, liver,kidneys,spleen,bones,skeletal muscle and skin ("diving reflex). In a study of asphyxiated newborn, 34% had no evidence of organ injury, 23% had an abnormality confined to one organ, 34% involved two organs and 9% had three affected organs. The most frequent abnormalities involved the kidneys (50%), followed by CNS (28%) cardiovascular system 25%, and pulmonary (23%) system.[2]

Whenever a neonate develops severe hypoxia or hypotension kidney damage may result. The presentation and course of renal damage depend on the severity and duration of the insult. Mild ischemia results in transient loss of renal concentrating capacity, owing to extreme sensitivity of the medullary thick ascending limb to tissue hypoxia. More prolonged injury produces widespread tubular dysfunction, with significant impairment in sodium and water reabsorption and decreases in GFR.[3]

Seeing the gravity of involvement of kidneys in birth asphyxia, this study was designed. Renal status was evaluated on the basis of estimation and monitoring of urinary output and blood urea nitrogen.

MATERIAL AND METHODS

The study group comprised of 100 newborns delivered in Department of Obstertrics and Gynaecology, admitted to Neonatology section of Department of Pediatrics, Govt Medical College, Rajindra Hospital, and Patiala.

100 asphyxiated newborns served as cases and 35 normal neonates served as control group. Newborns with congental anomalies of kidneys and urinary tract, respiratory distress syndrome, DIC, septicaemia were excluded. The study was approved by the Institutional Ethical Committee, and informed consent was obtained from the parents of each subject. In this study, 100 asphyxiated neonates (Apgar score at one minute 7 or less) were taken as cases of study. 35 normal neonates (Apgar score at 1 minute more than 7) were taken as control. The renal functions were evaluated in the form of urinary output and blood urea. Blood urea was estimated at 24 hours because the renal damage due to birth asphyxia occurs within 24 hours. The second estimation of blood urea was done at 72 hours to monitor the renal functions. Likewise the urinary output was measured till 72 hours.

Value of blood urea was converted into BUN by using the following:

Blood urea × 0.467 = BUN (Newman and Price, 2001)[4]

Urine was collected by using urinary bags, both in male and female neonates. Urine output was monitored daily till 72

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hours. Blood urea was estimated by diacetyl monooxime method using commercially available kit (J. Mitra and co. Ltd). Urea reacts with diacetyl monoxime in acidic medium at 95^0-100^0 to give pink coloured complex. Ferric ions are used to oxidise hydroxylamine formed in the reaction. Absorbance of pink coloured end product is measured at 520 nm. ARF will be considered in a newborn when urine output is < 0.5 ml/kg/hr and BUN> 20 mg/dl.

STATISTICAL ANALYSIS

Statistical comparison of measured values between two groups was performed by the unpaired' test of the means and chi square test.

RESULTS

In this study, 100 asphyxiated neonates (Apgar score at 1 minute less than 7) were taken as study group and 35 normal neonates (Apgar score at one minute more than 7) were taken as control. Blood urea levels were determined at 24 hours and at 72 hours of life in all neonates. Blood urea nitrogen values were calculated from blood urea levels. Urinary output was measured in all neonates for first three days of life. Statistical comparison of measured values between two groups were performed by the unpaired' test of the means and chi square test. 40 cases were mild birth asphyxia, 36 cases were moderate birth asphyxia and 24 were severe birth asphyxia. Blood urea nitrogen and urinary output in study group according to grades of birth asphyxia and control group is shown in table -1. In present study, mean BUN level of severely asphyxiated babies on day 1 and 3 was significantly higher than that of moderately and mildly asphyxiated babies ($p<0.01$) (Table-2) The mean BUN levels of moderately asphyxiated babies were also significantly higher than that of mildly asphyxiated babies at day 3 of life, whereas at day 1 the difference between them was nonsignificant. The mean urinary output in severely asphyxiated babies on day 1, 2 and of life was significantly lower as compared to mean UOP in mildly and moderately asphyxiated babies ($p<0.01$) (Table-3)

In terms of UOP, difference between study and control group was statistically highly significant on day 2 and day 3 but was not significant on day1 and for BUN levels difference between study and control group was statistically highly significant on day 1 and day3 as shown in Table 2 and 3.

On day 3, 43% of asphyxiated babies developed renal failure (BUN level > 20 mg/dl). 41.86% (18/43) developed oliguric renal failure (UOP <0.5 ml/kg/hr) and 58.14% developed non-oliguric renal failure (UOP>0.5 ml/kg/hr) as shown in Table-4.In severely asphyxiated babies, on day 2 and day 3, percentage of oliguria increased to 33.3% and 50 % respectively. Corresponding values in moderately asphyxiated babies were 5.56% and 16.6% on day 2 and day 3 of life respectively. The difference between moderately and severly asphyxiated babies were also significant statistically significant ($p<0.01$) on day 2 and 3as shown in Table-5

DISCUSSION

Perinatal asphyxia is a common neonatal problem and contributes significantly to neonatal mortality and morbidity. During Hypoxic ischemia,blood flow is redistributed in order to preserve circulation to most vital organs- brain, heart and adrenals. This is at the expense of the kidneys, liver and gastrointestinal tract, which are therefore vulnerable to hypoxic-ischemic damage.[5]

Out of 100 cases, 40 babies had mild asphyxia, 36 had moderate birth asphyxia and 24 had severe birth asphyxia, 55 were males and 45 were females. Babies with Apgar score less than or equal to 7 were defined as asphyxiated and babies with Apgar score more than 7 constituted the control group. Blood urea was determined and BUN values calculated of the entire neonate (both study group and control group) on 1st and 3rd day of life. In the present study, mean BUN level of severely asphyxiated babies on day 1 and day 3 was significantly higher than that of moderately and mildly asphyxiated babies ($p<0.01$) table-1. The mean BUN levels of moderately asphyxiated babies were significantly higher than that of mildly asphyxiated babies on day 3 of life whereas on day 1 the difference between them was non-significant. This shows that mean BUN are related to severity of asphyxia. This is in accordance with the study of Gupta BD et al that high values of blood urea were seen with lower apgar scores.[6-8]

In present study, mean urinary output in severely asphyxiated babies on day 1 of life was significantly lower as compared to mean UOP in mildly and moderately asphyxiated babies ($p<0.01$) table-1. Similarly mean UOP in severely asphyxiated babies was significantly lower than in mildly and moderately asphyxiated babies on day2 and day 3 of life. The mean UOP of moderately asphyxiated babies were also significantly lower as compared to mildly asphyxiated babies on day 2 and 3 of life ($p<0.01$) table-1. This shows that UOP was inversely related to severity of asphyxia which in accordance with the study of Pejovic B et al (2002), who concluded that there is a good prediction of severity of oliguric renal failure according to the degree of perinatal asphyxia determined by Apgar score at one minute.[9,10]

In present study, 4.17% of severely asphyxiated babies on day 1 was having decreased UOP (<0.05ml/kg/hr) as against 2.78% in moderately asphyxiated babies and 0% of control babies. Difference between severely asphyxiated babies and moderately asphyxiated babies were not significant statistically ($p<0.01$) as shown in table-2.

In severely asphyxiated babies, on day 2 and day3, percentage of oliguria increased to 33.3% and 50% respectively. Corresponding values in moderately asphyxiated babies were 5.56% and 16.6% on day 2 and day 3 of life respectively. The difference between moderately and severely asphyxiated babies was also statistically significant on day 2 and 3. This shows that UOP is inversely related to degree of asphyxia which is co-relating well with the study of Pejovic B et al (2002), who found highly positive linear correlations between Apgar score and urinary output in their study on acute oliguric renal failure in hypoxic neonates born at full term. The difference between UOP on day 1 and day 3 was also significantly different in moderately and severely asphyxiated babies

Out of 43 asphyxiated babies who developed renal failure (i.e. BUN >20 mg/dl), 18 (41.86%) had oliguria and rest 25 (58.14%) were having normal urinary output. None of the

babies had anuria in the present study. Incidence of non-oliguric renal failure in the present study was similar with study done by Karlowicz MG and Adelman RD (1996), who had observed non-oliguric renal failure in 60% of asphyxiated term neonates. However in their study number of oliguric cases was 25%, and of anuric cases was 15%. This in contrast to the present study where number of oliguric cases were 41.86%. This difference could be due to the fact that they included the babies in their study with higher degree of asphyxia (5 min. Apgar Score ≤ 6) as compared to the present study.[11-13]

CONCLUSION

Perinatal asphyxia is important cause of affecting renal functions. BUN on day 1 and 3 and UOP day 1, day 2 and day 3 were significantly different among mildly, moderately and severely asphyxiated babies, concluding that mean levels of BUN and UOP are related with grade of asphyxia.

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