



## RENAL AND CARDIAC PARAMETERS IN BIRTH ASPHYXIATED BABIES

## Paediatrics

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## ABSTRACT

**BACKGROUND:** Objective of the current study was to study the incidence of renal and cardiac involvement in birth asphyxia babies.

**METHOD:** A Prospective study was conducted among 50 asphyxiated term neonates with APGAR score less than or equal to 7 at 5 Minute of birth, admitted in NICU of Maharishi Markandeshwar Medical college during the period of January 2020 to July 2021 were included in this study. A detailed antenatal history and thorough examination was done using pre designed and pre structured proforma. Necessary investigations were done as indicated to identify organ dysfunction.

**RESULTS:** Among 60 asphyxiated neonates, 21 babies had Moderate asphyxia (APGAR score 4-6 at 1 Minute ) and 29 babies had Severe asphyxia ( APGAR score <4 at 1 Minute). Moderate asphyxia babies had 23.8% renal involvement and 14.3% CVS involvement. Neonates with Severe Asphyxia had 100% renal and cardiac involvement. Troponin T was positive in the neonates who expired. Among total asphyxiated neonates 68% had renal involvement and 64% had CVS involvement.

**CONCLUSION:** Multiorgan dysfunction is more common in birth asphyxia. Severe asphyxiated babies are significantly more prone to multiorgan dysfunction. Troponin T correlates with the severity and the outcome of the neonate.

## KEYWORDS

## INTRODUCTION

**Birth Asphyxia:** According to WHO birth asphyxia is defined as failure of initiation and maintenance of breathing at the time of delivery.(1) In India according to the National Neonatology forum birth asphyxia should be diagnosed when the baby has gasping and inadequate breathing or no breathing after one minute of birth.(2)

The AAP have established some diagnostic criteria for birth asphyxia as follows:

- 1) Cord umbilical artery pH < 7.0
- 2) Multisystem organ dysfunction (MODS)
- 3) Evidence of hypoxic- ischemic encephalopathy (HIE). (3)

The most acceptable definition for the purpose of estimating the incidence of birth asphyxia is APGAR score less than 4 at 1 minute. Birth asphyxia is the 3<sup>rd</sup> most important cause of neonatal death after Prematurity and Sepsis.(4)

Birth asphyxia is responsible for 9% of total death in neonatal. In which 27 lakhs stillbirths occur in the world, approximately 12 lakhs occurring during intra partum period, mainly due to asphyxia.(4)

NNPD states that birth asphyxia is responsible for 20% of newborn deaths that occur in India.

Birth asphyxia remains an important cause of neonatal mortality, morbidity and late sequelae are especially in developing countries like India. Hypoxia damages almost every organ and tissue.

During hypoxia there are a series of protective mechanisms collectively called the "diving sea reflex" which try to redistribute available blood flow from lesser to more vital organs. The blood flow to the brain, heart, adrenal gland of the newborn is preserved at the expense of reduction of perfusion to kidney, lung, gastrointestinal tract, liver, spleen and skeletal muscles.(5) The most commonly involved organ following asphyxia is kidney followed by CNS and

CVS with least involvement of the pulmonary system . Thus the development of evidence of multisystem organ failure in the immediate neonatal period is high . Renal systems are very sensitive and underdeveloped at birth and attain maturity as the age advances. Prolonged hypoxia ischemic episode and hypoxia can lead to irreversible cortical necrosis with onset of acute renal failure (ARF).(6)

Cardiac output is normally maintained early in asphyxia by selective regional Vasoconstriction which reduces blood flow to the less vital organs but as asphyxia increases to the severe stage, oxygen delivery to the heart and brain decreases. The myocardium starts utilising the stored glycogen reserve for energy. As the asphyxia progresses the glycogen reserve is utilised and the myocardium is exposed to progressively lower PO<sub>2</sub> and pH. This causes hypoxia and acidosis

and leads to depressed myocardial function and decreased blood flow to vital organs.(7)

Assessment of serum creatinine, urea, CPK-MB and Trop-T is easy, rapid and easily accessible at all centers which can be used as a marker for assessment of birth asphyxia and assessing the multi organ involvement in the birth asphyxia babies.

## MATERIALS AND METHOD

A Prospective study was conducted among 50 asphyxiated term neonates with APGAR score less than or equal to 7 at 5 Minute of birth, admitted in NICU of Maharishi Markandeshwar Medical college during the period of January 2020 to July 2021 were included in this study. A detailed antenatal history and thorough examination was done using pre designed and pre structured proforma. Necessary investigations were done as indicated to identify organ dysfunction.

## AIMS AND OBJECTIVES:

To study the acute kidney injury (serum creatinine & urea) and cardiac ischemia ( enzyme-Troponin T and cpk-MB) in birth asphyxiated babies at 8 hour and 48 hour of postnatal life

**Inclusion Criteria:**

- TERM BABY WITH APGAR SCORE <7 AT 5 MINUTE OF LIFE

**Exclusion Criteria:**

1. Preterm and postterm neonates.
  2. Neonates with gross congenital malformation
  3. Birth trauma
  4. Maternal Drug Addiction
  5. Mother received MgSo4 within a period of 4 hrs prior to delivery
  6. parents who are not willing to give consent
- **Study method:** Serum creatinine is the best parameter to comment on acute kidney injury. Creatinine is the only product that is completely excreted by the kidney, if creatinine is not excreted then this indicates that there is some insult to the kidney Troponins are group of protein found in skeletal and heart muscle fiber. There are 3 types of troponin protein-C, I and T. Troponin C initiates contraction by binding calcium and moves troponin I so that the two proteins that pull the muscle fiber shorter can interact. Troponin T anchor the troponin complex to the muscle fiber structure. Troponin test measure the level of cardiac-specific troponin I or T in the blood to detect heart injury. Troponin test is most specific and sensitive for cardiac damage.

Creatine kinase and its isoenzymes values increases in ischemia, induces leakage of enzymes from injured tissues. MB iso enzymes is highly specific to cardiac. Hence significant increase of CK-MB may be a sensitive indicator of myocardial damage.

The MB isoenzyme of CK is better to TOTAL CK which is not present more in extra cardiac tissues hence considered more specific

- **Method of sample collection:** Pretest counseling will be given to patients. After taking written informed consent from the patient, case will be enrolled, data will be collected in a predesigned semi-structured questionnaire. After proper management of airway, breathing, circulation as per standard guidelines .under all aseptic precaution 2ml of venous blood sample will be collected from patients for kidney function, troponin and CPK-MB at 8th and 48th Hr.

**Statistical analysis:**

The analysis included profiling of patients on different demographic, clinical, diagnosis, laboratory parameters. Descriptive analysis of quantitative parameters were expressed as means and standard deviation. Categorical data were expressed as absolute number and percentage. Data not following Normal Distribution was compared using Mann-Whitney U test and Wilcoxon Signed Rank Test among the group as applicable. Cross tables were generated and Chi square test was used for testing of associations.  $p < 0.05$  were considered statistically significant.

All analysis was done using SPSS software, version 24.0.

**RESULTS:**

Among total 50 asphyxiated term neonates, males 41 (82%) were more affected than the females 9 (18%). There was no significant difference between primigravida mothers 26 (52%) and multigravida mothers 24 (48%). Babies born out of LSCS 29 (58%) were more as compared to babies born out of NVD 21 (42%). The mean birth weight of the neonates in our study was 2734 grams. Asphyxiated neonates in our study were resuscitated according to NALS Guidelines, which in our study was Bag and Tube ventilation 50 (100%). Soon after the birth, all the babies in our study had Respiratory Distress and needed respiratory support by means of Oxygen by nasal prongs 10 (20%), CPAP 14 (28%) and Mechanical Ventilation 26 (52%). The neonates in our study had various complications which included Respiratory Distress 50 (100%), Shock 13 (26%), seizures 9 (18%), hypoglycemia 4 (8%) and Necrotising Enterocolitis 2 (4%). 45 (90%) of the asphyxiated neonates of our study were discharged and 5 (10%) of them expired. Neonates having Moderate asphyxia were 21 (42%) whereas neonates having Severe asphyxia in our study were 29 (58%).

In our study, Table I shows that the mean blood urea, creatinine and CPK-MB values were significantly higher in severely asphyxiated neonates at 8th Hour as compared to moderately asphyxiated neonates. These values tended to normalise gradually over 48 Hours. Table II shows that Troponin T was positive only for the asphyxiated neonates who died (10%). In Moderate asphyxia, CVS involvement was in

14.3% of the babies and renal involvement was present in 23.8% babies. In severely asphyxiated neonates, there was 100% renal and cardiac involvement.

Among 50 asphyxiated neonates, renal involvement was 68% and CVS involvement was in 64% of the neonates.

**DISCUSSION:**

Multiorgan dysfunction is part of perinatal asphyxia which occurs due to diving reflex wherein there is redistribution of blood flow to vital organs like brain, heart and kidneys. With continued asphyxia there is hypotension, loss of autoregulation of blood flow to the brain and loss of compensatory mechanisms resulting in severe hypotension, hypoxic and ischemic injury even to the vital organs. Organs suffering hypoxic and ischemic injury develop dysfunction and failure if this hypoxic ischemia is not corrected. Hence degree of multiorgan dysfunction would directly correlate with severity of asphyxia. Multiorgan dysfunction increases the risk of morbidity and mortality. In our study, APGAR score of 4-6 at 1 min, 21 (42%) neonates were under the category of Moderately Asphyxiated neonates and APGAR score of <4 at 1 min, 29 (58%) neonates were under the category of Severely asphyxiated neonates.

In a study conducted by **Seema Rai, Narinder Singh(8)**, 40% were having Mild asphyxia, 36% of asphyxiated neonates were having Moderate Asphyxia, while 24% of asphyxiated neonates were having Severe Asphyxia. This is different from the study conducted by us as the inclusion criteria of us was the APGAR score at 5 Minutes whereas the inclusion criteria of their study was APGAR score at 1 Minute.

**CPK-MB and Troponin T levels in the study group****CPK-MB Levels**

In our study, mean CPK-MB levels of Moderate Asphyxia were 22 U/L and 18 U/L at 8th and 48th Hour respectively. Mean CPK-MB levels of Severe Asphyxia were 110 U/L and 44 U/L at 8th and 48th Hour respectively. In our study, 64% of the neonates had CNS involvement. In our study we have seen that the CPK-MB levels of Severe Asphyxiated neonates were significantly raised with a p-value of <0.001.

This was similar to a study which was conducted by **Rajkumar PS et al(9)**, as shown in Table II where the CPK-MB values were 121 U/L and according to **Sanath et al (10)** study, it was 176.1.

In our study, it has been seen that the mean CPK-MB value of Severe Asphyxiated babies was increased at 8th Hour of postnatal life and gradually it tended to normalise at 48th Hour of postnatal life. This is similar to a study conducted by **Vivek Singh et al(11)**, in which the CPK-MB values were significantly raised at the 12th Hour of life and gradually came to Normal at 72 Hours.

**Troponin T**

In our study, Troponin T was done by a qualitative test. Troponin T was positive among 5 (10%) babies. Troponin T was positive only for the babies who expired. Troponin T was positive for Severely Asphyxiated babies and it correlates with the severity of the outcome of the asphyxiated babies. This was similar to a study conducted by **P.S. Rajkumar et al(9)**, in which normal level of Troponin T was considered 0.2 ng/mL. Mortality was found in 5 out of 7 cases with levels >0.2 ng/mL and thus, he has shown that Troponin T correlates with the outcome of the neonates. In a similar study conducted by **Pal P, Goel M(12)**, the Troponin T levels in Severely Asphyxiated neonates were significantly higher (4.6 g/mL) than the Moderately Asphyxiated babies. Thus, similar to our study, they also concluded that the Troponin T levels may be useful in predicting the mortality and outcome in perinatal asphyxia. Also in a similar study conducted by **W E**

**Sadoh(13)**, he also showed that Troponin T levels were higher in the neonates who died, which correlates with our study.

**UREA and CREATININE Levels in the study group**

In our study, mean Urea levels of Moderate Asphyxia were 22 mg/dL and 16mg/dL at 8th and 48th Hour respectively and the mean Urea levels of Severe Asphyxia were 30mg/dL and 22 mg/dL at 8th and 48th Hour respectively. The mean Creatinine levels of Moderate Asphyxia were 0.6 mg/dL and 0.5 mg/dL at 8th and 48th Hour respectively and

the mean Creatinine levels of Severe Asphyxia were 1.3 mg/dL and 0.6 mg/dL at 8th and 48th Hour respectively. In our study, it has been seen that the mean Urea and Creatinine values of Severe Asphyxiated babies was increased at 8th Hour of postnatal life and gradually it tended to normalise at 48th Hour of postnatal life. In our study, 68% of the neonates had Acute Kidney Injury, in which serum creatinine was increased in 68% and urea was increased in 26% among asphyxiated babies. This was similar to a study conducted by **Dr. D.Y. Shrikhande et al**(14), in which he also showed that creatinine was increased in 64% and urea was increased in 17% of the asphyxiated neonates. In another study conducted by **Aggarwal et al** and **Hankins Gary D. V. et al**, serum creatinine was raised in 56% and 61% asphyxiated neonates respectively. In our study, blood urea was raised in severe asphyxiated babies which was similar to the study conducted by **Seema Rai et al**(8), in which she shows that the mean level of blood urea nitrogen are related with grades of asphyxia.

**CONCLUSION**

Blood urea, serum creatinine, CPK-MB and Troponin T can be used as markers for perinatal asphyxia for screening in centers where other markers for assessing perinatal asphyxia are not available. Severely asphyxiated neonates are more prone to multiorgan failure as compared to moderately asphyxiated neonates. Troponin T levels correlates with the severity of the outcome.

**Limitations**

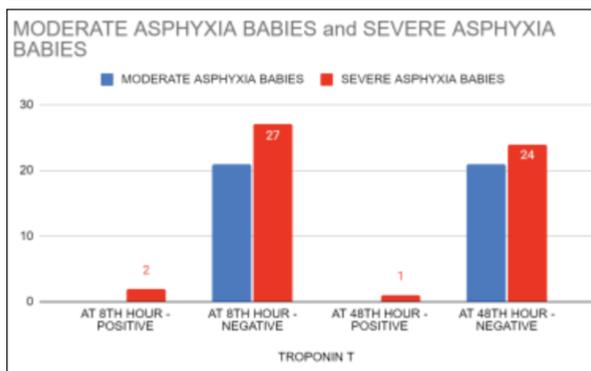
As it is a single centre study with a very small sample size, so therefore the result could not be generalized to the whole population. In our study, Troponin T was done as a qualitative study and not as a quantitative study. Urine output was not included to comment upon AKI.

**Table I Correlation Between Blood Urea, Creatinine And Cpk-mb Of Severely And Moderately Asphyxiated Neonates**

	MODERATE ASPHYXIA	SEVERE ASPHYXIA	P- VALUE
<b>BLOOD UREA AT 8th Hr</b>	22 (19,28) RANGE 14-35	30 (21,91) RANGE 18-118	0.003
<b>BLOOD UREA AT 48th Hr</b>	16 (14,21) RANGE 12-34	22 (15,36) RANGE 12,81	0.021
<b>SERUM CREATININE AT 8th Hr</b>	0.6 (0.4,1.0) RANGE 0.4-1.4	1.3 (1.2,1.4) RANGE 1.1-1.6	0.001
<b>SERUM CREATININE AT 48th Hr</b>	0.5 (0.3,0.6) RANGE 0.2-0.8	0.6 (0.6-0.8) RANGE 0.4-1.8	0.001
<b>CPK-MB AT 8th Hr</b>	22 (20-24) RANGE 14-82	110 (90,138) RANGE 66-333	0.001
<b>CPK-MB AT 48th Hr</b>	18 (16-22) RANGE 10-36	44 (38,47) RANGE 28-330	0.001

**Table II Comparison Of Cpk-mb Levels Of Different Studies With Our Study.**

CPK-MB in U/L	Rajkumar PS et al	Sanath et al	Masaraddi Sanjay K et al	Present study
<b>CASES</b>	121	176.1	101	110



**Graph I Troponin T Levels In Moderate Asphyxia Babies And Severe Asphyxia Babies At 8th Hour And 48th Hour Of Birth**

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