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**Original Research Article**

# Cardiac changes in asphyxiated neonates-need for early detection to improve long-term outcome

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## ABSTRACT

**Background:** Cardiac changes are common in Perinatal asphyxia. Detecting them early will help in specific management and good long-term outcome. Cardiac monitoring is recommended in every resuscitation as per NRP 7. Objective of present study was to evaluate myocardial dysfunction in neonates having Hypoxic Ischemic Encephalopathy by clinical, electrocardiographic and echocardiographic examination.
**Methods:** Observational analytical study of 79 neonates who suffered from asphyxia as per selection criteria were done at medical college hospital. Myocardial dysfunction was evaluated by clinical features assessed on first 2 days and electrocardiographic and echocardiographic examination was done after 24 hours. Data were collected and analysed by SPSS 21 software.
**Results:** Respiratory distress was found in 67.08% of the subjects. Prolonged capillary refill time was found in 39.24% of the subjects. Mean QTc (ms) interval was prolonged in non-survivors (496.6±20.8) when compared with survivors (418.2±33.7) (p - 0.000). T wave changes were present in non-survivors (94.7%) when compared to survivors (41.7%) (LR- 19.558). ST wave changes were present in non-survivors (73.7 %) compared with survivors (10%) (LR- 28.483). Mean Ejection Fraction (%) was shortened in non-survivors (53.79±7.53) compared with survivors (71.48±10.28) (p-0.000). Significant tricuspid regurgitation (78.94%) was present among non-survivors compared to survivors (10%) (LR- 17.859). Evidences of significant pulmonary artery hypertension were found in most of the (78.94%) non-survivors compared to survivors (10%) (LR-22.4). Similarly, severe grades of asphyxia had significantly high findings of increased CRT, reduced EF, LVES, ST and T wave changes etc.
**Conclusions:** Cardiac changes in severe asphyxia are high and can be identified early by clinical, electroctrocardiographic and echocardiographic examination.

**Keywords:** Birth asphyxia, Echocardiography, Electrocardiography, Myocardial changes, Neonate

## INTRODUCTION

The Under-Five Mortality Rate (U5MR) and Infant Mortality Rate (IMR) of a nation are widely accepted long standing indicators of well-being of Children. A high IMR is an indicator of risk of death during the first year of life and is indicative of unmet health needs and unfavorable environmental factors.[1] U5MR in MP is 65 and IMR is 51 (NFHS-4 report).[2] In Madhya Pradesh, Neonatal Mortality Rate(NMR) was 39 (29 Early NMR and 10 Late NMR).[1]

Perinatal asphyxia has an incidence of 1 to 6 per 1,000 live full-term births, and represents the third most common cause of global neonatal death (23%) after preterm birth (28%) and severe infections (26%).[3,5] In our institution, last year in 2015, it accounts for about 26.02% of total admissions and 29.4% of all neonatal deaths.

Birth asphyxia is remaining as a major cause of Under 5 Morbidity and mortality in our institution.

Birth asphyxia leads to multi-organ damage involving every system in the body. Cardiac changes occur in 25-60% in neonates with asphyxia.[6] Myocardial contractility and papillary muscle dysfunction with tricuspid regurgitation resulting in significant reduction in cardiac output leading to hypotension and Cardiogenic shock.[6,8]

When severity progresses, child will become hypotensive manifested by low blood pressure that can be measured by either automated/invasive Blood pressure monitoring or clinically prolongation of Capillary Refill time. Neonate will have signs of respiratory distress, cyanosis initially peripheral progressing to central cyanosis at later time period. Clinically a systolic murmur due to Tricuspid Regurgitation may be evident.

Subclinical Myocardial Ischaemia in asphyxiated stressed infants is common. T- wave inversion and ST depression and in the left chest leads is a common finding. Other changes include deep and wide Q waves and initial elevation of ST segment, followed by T wave inversion. Q waves persist for long-time but ST segment changes resolve in a week or so and T waves return to normal over a much longer timescale.[9]

Typical Echocardiographic findings include diminished ventricular function (including wall motion abnormalities), atrioventricular valve regurgitation (most commonly Tricuspid Regurgitation) and evidences of pulmonary hypertension can be present.[6-8,10]

Hypotension following HIE is common due to myocardial dysfunction rather than hypovolaemia. Fluids should be given cautiously and if not respond, early use of inotropes is recommended.[10,11] Infant with PPHN should be identified as it is a medical emergency and appropriate intervention should be given. Cardiac changes are common in Perinatal asphyxia.[6] Detecting them early will help in specific management and good long-term outcome. Cardiac monitoring is must in every resuscitation as per NRP 7.[12]

Present study was undertaken to evaluate the myocardial dysfunction in neonates having HIE by clinical features, electrocardiographic (ECG) and echocardiographic (ECHO) examination and correlate with outcome.

## METHODS

Setting: Labour wards, operation theatres, Special Newborn Care Unit, Department of Paediatrics in tertiary care teaching institute hospital.

Study design: Hospital based prospective analytical study. Sample size: 79. Duration: One year (JULY 2015-JULY 2016)

***Inclusion criteria***

The cases included term babies with evidence of asphyxia indicated by any three of the following[4]:

- Gestational age> 37 weeks
- The neonates who were identified to have experienced perinatal asphyxia by following criteria.[5]

i. APGAR< 7 at 1 minute of life.
ii. Requirement of positive pressure ventilation at 1 min
iii. Features of Mild, Moderate or Severe hypoxic ischemic encephalopathy, as defined by Levene classification.

***Exclusion criteria***

All preterm neonates, congenital malformations, Neonates born to mothers who would have received any drugs causing depression, Patient leaving treatment against medical advice before the stabilization phase, informed written consent was taken from parents/close relatives

The cases included 79 term babies fulfilling inclusion and exclusion criteria. All births with probable fetal distress were attended in labour rooms and operation theatre. Informed consent was taken from the parents or close relatives of all babies included in the study. Apgar score of newborns was evaluated at 1and 5 mins. Maternal risk factors, child general condition, systemic examination, Levene grading was done at 1st day. Twelve lead electrocardiography was conducted at end of first 24 hours. Echocardiography (M-mode, Two-dimensional as well as Doppler study) conducted after 1st day. The cases who had cardiac involvement were advised on follow up at 6 weeks for the presence of any murmur, respiratory distress or cardiac failure.

The incidence of cardiac dysfunction using clinical, electrocardiographic and echocardiographic criteria will be calculated. Correlation of electrocardiographic (ECG) and echocardiographic (ECHO) with outcome of asphyxiated neonates will be made. Also, correlation between Clinical features with electrocardiographic (ECG) and echocardiographic (ECHO) findings will be made. Following statistics applications were done: For difference in mean student's t test was applied. For difference in proportions chi square test or Fischers exact test was applied depending on the counts in each cell of the contingency table. A P value of less than 0.05 was considered statistically significant. Descriptive analysis was carried out to describe categorical variables in terms of percentages and continuous variables in terms of mean and standard deviation. All analysis was carried out in SPSS version 21.

## RESULTS

Basic Characteristics among the study neonates (Table 1) shows that antenatal complications were found in all

grades of asphyxia but they were significant in severe grade of asphyxia (71.42%) more in non-survivors (82%). APGAR scores were significant at 1 min and 5 min. Value of APGAR score at 5 min was significantly low in non-survivors. (p-0.000). There was no significant difference in sex, gestational weeks, colour of meconium, maternal age, gravida and type of presentation in severe grades of asphyxia. Likelihood ratio for non-survival is marginally significant in caesarean section (1.864) and significantly in primigravida (7.269).

**Table 1: Basic characteristics of HIE neonates based on LEVENE staging.**

| Parameters | Categories | HIE stage | | | P value |
|---|---|---|---|---|---|
| | | Mild (n=20) | Moderate (n=24) | Severe (n=35) | |
| Gender | Male | 12 | 14 | 25 | 0.411 |
| | Female | 8 | 10 | 10 | |
| Gestational age | Weeks | 39.40±1.50 | 39.50±1.47 | 39.86±1.44 | 0.470 |
| Antenatal complications | % | 65% | 58.33% | 71.42% | 0.001 |
| Birth weight (g) | In grams | 2797.5±459.25 | 2685.42±395.49 | 2778.57±410.93 | 0.615 |
| | <2500g | 5 | 6 | 8 | |
| | >2500g | 15 | 18 | 27 | |
| Maternal gravida | | 2±0.95 | 2.08±0.95 | 1.88±1.13 | 0.617 |
| Amniotic fluid | Clear | 50% | 41.70% | 40% | 0.761 |
| | Meconium stained | 50% | 58.30% | 60% | |
| Mode of delivery | NVD | 12 | 15 | 17 | 0.517 |
| | LSCS | 8 | 9 | 18 | |
| Apgar at 1 min | /10 | 4.0±1.03 | 4.29±0.62 | 3.4±1.01 | 0.001 |
| Apgar at 5 min | /10 | 7.1±0.97 | 6.88±0.90 | 5.17±1.15 | 0.000 |

**Table 2: Clinical features among HIE neonates between survivors and non-survivors.**

| Clinical features | Survivors (n=60) | | Non-survivors (n=19) | | Likelihood ratio | |
|---|---|---|---|---|---|---|
| | 1st day | 2nd day | 1st day | 2nd day | 1stday | 2nd day |
| Heart rate mean (/min) | 146.93±24.24 | 145.13±11.61 | 159.37±16.24 | 140.63±15.73 | | |
| Respiratory distress (%) | 56.7% | 28.3% | 100% | 100% | 23.38 | 49.8 |
| Inotropes support (%) | 18.3% | 16.7% | 78.90% | 100% | 17.99 | 37.37 |
| CPAP/Mechanical ventilation (% | 20% | 0.00% | 63.20% | 78.95% | 11.96 | 46.95 |

**Table 3: Electrocardiographic changes among HIE neonates between survivors and non-survivors.**

| ECG parameters | R/A | Expired | Likelihood ratio | P value |
|---|---|---|---|---|
| PR interval (ms) | 80.7±18.4 | 73.7±24.2 | | 0.435 |
| QRS interval (ms) | 42.7±6.9 | 37.9±09.2 | | 0.105 |
| QTc interval (ms) | 418.2±33.7 | 496.6±20.8 | | 0.000 |
| T wave changes (%) | 41.70 | 94.7 | 19.558 | 0.000 |
| ST changes (%) | 10 | 73.7 | 28.483 | 0.000 |
| Arrhythmias (%) | 0 | 16.7 | 9.053 | 0.002 |

Comparison of Clinical features (Table 2) among survivors and non-survivors reveals that there was no significant difference in heart rates between different grades of asphyxia although HIE 3 neonates had higher basal heart rate. All HIE 3 neonates, 2/3rd of HIE II, 15% of HIE 1 had respiratory distress on day 1, which reduced to 80%, 33.33% and 0% respectively on day 2. Among HIE 3 neonates 75%, HIE II 12.5%, none of HIE 1 had prolonged CRT on day 1, which reduced to 56.29%, 12.5% and 0% respectively on day 2. 2/3rd HIE 3 neonates, 4.17% of HIE II, none of HIE 1 had requirement of mechanical ventilation/ CPAP on day 1, which reduced to 37.14%, 0% and 0% respectively on day 2.

On study of electrocardiographic findings among the neonates (Table 3) we find that PR Interval became shorter as severity increases. But that is not statistically significant. For QRS interval too, there was no significant difference between different grades. QTc interval (Figure 1) was more prolonged in severe grade of asphyxia which is statistically significant. T wave changes including

either flattening or inversion is found in all grades of asphyxia but significantly more in severe asphyxia. ST changes including ST depression and ST elevation were seen in 16.67% of moderate and 45.71% of severe asphyxia. Arrhythmias was seen only in 3 neonates all with HIE 3.

**Table 4: Echocardiographic changes among HIE neonates among survivors and non-survivors.**

| ECHO parameters | Survivors | Non survivors | Likelihood ratio | P value |
|---|---|---|---|---|
| Ejection fraction (%) | 71.48±10.28 | 53.79±7.53 | | 0.000 |
| Fractional shortening (%) | 38.2±8.22 | 25.42±4.83 | | 0.000 |
| LVED (in mm) | 14.35±2.97 | 15.32±2.89 | | 0.218 |
| LVES (in mm) | 8.58±2.71 | 11.47±1.74 | | 0.000 |
| LVPWD (in mm) | 4.22±0.88 | 4.79±0.98 | | 0.019 |
| **TR** | | | | |
| Mild | 15 | 2 | 17.859 | 0.000 |
| Moderate | 6 | 4 | | |
| Severe | 1 | 11 | | |
| **Pulmonary hypertension** | | | | |
| Mild | 11 | 2 | 22.399 | 0.000 |
| Moderate | 6 | 4 | | |
| Severe | 1 | 11 | | |
| Others (no. of neonates) | PDA – 6 | PDA – 1 | 2.302 | 0.167 |
| | PFO – 4 | | | |
| | ASD – 1 | | | |

LVED- left ventricular end diastolic diameter, LVES- left ventricular end systolic diameter, LVPWD- left ventricular Posterior wall diameter, TR- tricuspid regurgitation, PDA- patent ductus arteriosus, PFO- Patent Foramen Ovale, ASD- Atrial Septal Defect.

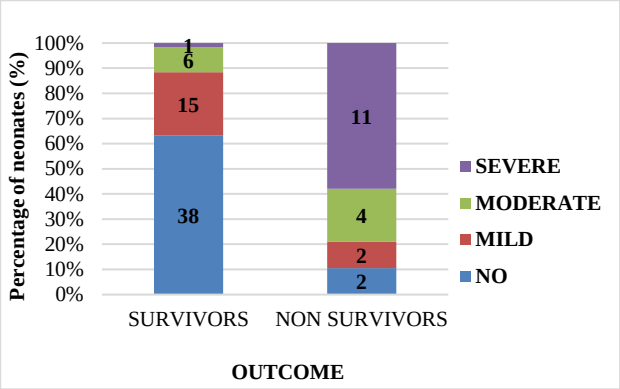


**Figure 1: Evidence of TR among survivors and non-survivors.**

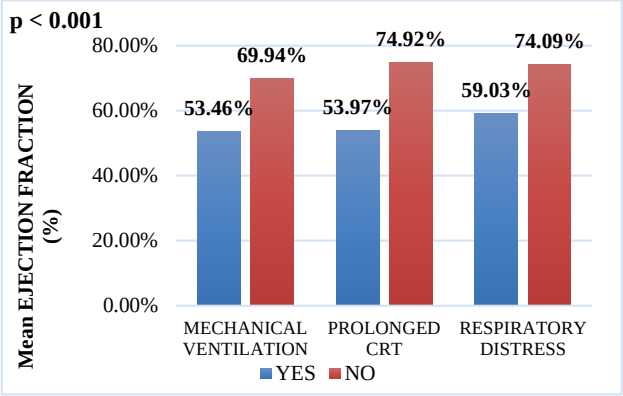


**Figure 2: Correlation of clinical features with ejection fraction.**

Analysis of Echocardiographic Changes (Table 4) in neonatal Asphyxia finds that Ejection fraction and Fractional shortening were less among HIE III neonates which was statistically significant. LVED increased as HIE grade increased but was not significant. LVES and LVPWD values increased with increasing grade of HIE which was statistically significant. TR (Figure 1) and Pulmonary hypertension were recorded in more neonates as HIE severity increased and were statistically significant. PDA was present in 2 HIE I neonates, 3 HIE II neonates and 2 HIE III neonates. ASD was found in 1 HIE I neonate. PFO was found in 2 neonates each in HIE II and III stages.

Positive correlation obtained when asphyxiated neonate having respiratory distress, prolonged CRT and requirement of mechanical ventilation is observed for electrocardiographic and echocardiographic findings. (Figure 2, 3, 4) Likelihood for non-survival was more associated with significant TR in presence of respiratory distress (22.46), significant PAH in presence of respiratory distress (17.53), significant TR in presence of prolonged CRT (17.25), significant PAH in presence of prolonged CRT (15.03), significant TR in requirement of mechanical ventilation (10.8), significant TR in presence of mechanical ventilation (8.3) in that order.

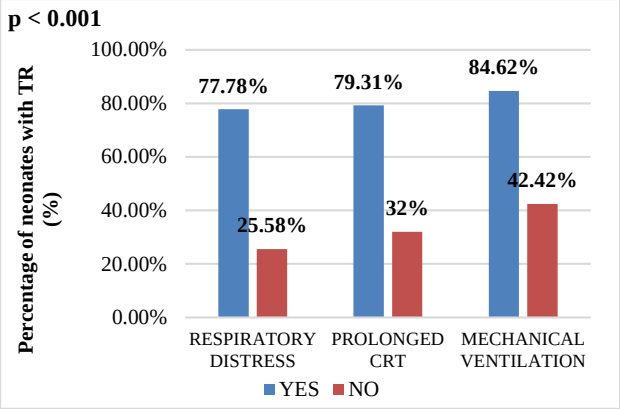


**Figure 3: Correlation of clinical features with TR.**

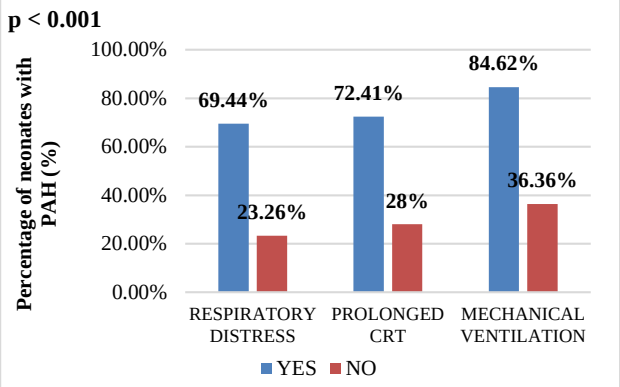


**Figure 4: Correlation of clinical features with PAH.**

## DISCUSSION

Perinatal asphyxia is one of the preventable common causes of neonatal mortality. Among the study neonates, antenatal complications were found in all grades of asphyxia but significant in severe grade of asphyxia (71.42%) more in non-survivors (82%). APGAR scores were significant at 1 min and 5 min. Value of APGAR score at 5 min was significantly low in non-survivors (p-0.000). There was no significant difference in sex, gestational weeks, colour of meconium, maternal age, gravida and type of presentation in severe grades of asphyxia. Likelihood ratio for non-survival is marginally significant in caesarean section (1.864) and significantly in primigravida (7.269). similar results obtained by Agrawal et al.[15]

Analysis of clinical feature reveals that there was no significant difference in heart rates between different grades of asphyxia and related to outcome. All non-survivors had respiratory distress on both day 1 and 2 whereas 56.7% of survivors had respiratory distress on day 1 which reduced to 28.3% on day 2. Likelihood ratio was highly significant (49.8) on day 2. Overall incidence was 67.08%. Rajakumar et al reported similar incidence of respiratory distress in 66.7%.[13] Goel et al reported respiratory distress only in 70% of severe grade and 25% of moderate asphyxia.[14] Agrawal et al reported 53.3%.[15]

Prolongation of CRT was seen in 75.71% HIE III neonates, 12.5% HIE II %, none of HIE 1 had prolonged CRT on day 1, which reduced to 56.29%, 12.5% and 0% respectively on day 2. Overall incidence was 39.24%. Rajakumar et al reported overall incidence of prolonged CRTs in 16.7% of overall Cases.[13] It was because there was only very few HIE III neonates in that study. Goel et al reported incidence of prolonged CRT was 75% in severe and 15% of moderate grade whereas Agrawal et al reported around 48.3%.[14,15] Among non-survivors 78.9% had prolonged CRT on day 1 whereas all of them had prolonged CRT on day 2. 18.3% of survivors had prolonged CRT on day 1 which reduced to 16.7% on day 2. The difference between two groups was highly statistically significant. Likelihood ratio 37.37 was more significant on day 2. Aleksandra et al reported prolonged CRT in 92.3% in non-survivors and 26.2% in non-survivors.[18]

Requirement of mechanical ventilation shows that Among non-survivors 63.2% had requirement of mechanical ventilation/ CPAP on day 1 which increased to 78.9% on day 2. The difference between two groups was highly statistically significant. Likelihood ratio 46.951 was more significant on day 2.

Following Electrocardiographic changes were noticed in neonates with birth asphyxia.

PR interval was short in non-survivors (7.37±2.42) when compared to survivors (8.07±1.84). The difference was not significant (p value - 0.435). No significant difference in Mean QRS interval among various groups.

Mean QTc interval was prolonged in non-survivors (496.6±20.8) when compared to survivors (418.2±33.7). The difference was significant (p value - 0.000). Aleksandra et al had incidence of prolonged QTc among non-survivors (450±12ms)when compared to survivors (420±6ms)and controls(360±2 ms).[18] Fang Xiao-yi (2012) et al reported prolonged QTc in severe asphyxia but value was very much higher.[17]

T wave changes was present more significantly in non-survivors (94.7%) when compared to survivors (41.7%). The difference was significant (Likelihood ratio- 19.558). Aleksandra et al reported less incidence of ST-T changes around 76.9% and 52.4% in non-survivors and survivors respectively.[18]

ST wave changes was present more significantly in non-survivors (73.7 %) when compared to survivors (10%). The difference was significant (Likelihood ratio- 28.483). Goel et al reported ST changes in 40% and 35% in severe and moderate grade respectively.[14]

Overall ECG evidences based on jedeikin grading found in 54.43%(16). This is similar to Rajakumar et al report 56.7% of total cases. 94.7% non-survivors had ECG changes in the present study. Rajakumar reported 100%

ECG changes found in expired neonates.[13] Ventricular arrythmias was found in 3 non-survivors.

Following Echocardiogarphic changes were noticed - Mean ejection fraction (%) was less in non-survivors (53.79±7.53) when compared to survivors (71.48±10.28). The difference was significant (p value - 0.000). Still low values were reported by Aleksandra et al (43.9±16.8 and 56.8±14.6 in non-survivors and survivors respectively).[18]

Mean fractional shortening (%) was shortened in non-survivors (25.42±4.83) when compared to survivors (38.2±8.22). The difference was significant (p value - 0.000) Still low values were reported by Aleksandra et al (21.4±10.3 and 28.8±8.2 in non-survivors and survivors respectively).[18]

Mean LVED (in mm) was more in non-survivors (15.32±2.89) when compared to survivors (14.35±2.97). The difference was significant (p value - 0.000). Goel et al had higher mean LVED diameter compared to moderate and control groups.[14] Mean LVES (in mm) was increased in non-survivors (11.47±1.74) when compared to survivors (8.58±2.71). The difference was significant (p value - 0.000). Goel et al had higher mean LVES diameter compared to moderate and control groups.[14]

Mean LVPWD (in mm) was increased in non-survivors (4.79±0.98) when compared to survivors (4.22±0.88). The difference was significant (p value - 0.019)

Overall incidence of TR in present study was 27.84%. Rajakumar et al reported overall incidence of TR in 23.3%.[13] Incidence of significant TR was 78.94% among non-survivors compared to survivors (10%). The difference was significant (p value was 0.001) and likelihood ratio was 17.859. Similar observations were obtained by Aleksendra et al (76.9% and 11.9% in non-survivors and survivors respectively).[18]

Evidences of significant PAH were seen in 78.94% among non-survivors compared to 10% survivors. The difference was significant (p value was 0.001) and Likelihood ratio was 22.399. Overall incidence of PAH in present study was 27.84%. Rajakumar et al reported PAH in only 6.7%.[13] Goel et al reported around 50% in severe asphxiated and 10% in moderate asphyxiated group.[14] Abdel Hakim concluded that 43.75% of asphxiated neonates developed PPHN.[19]

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