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Original Research Article

Evaluation of cardiac dysfunction in newborns with HIE with special reference to cardiac troponin I (hsTrop I)

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ABSTRACT

Background: Perinatal asphyxia is a major cause of neonatal and under 5 mortalities particularly in developing countries. Multiorgan dysfunction is common in newborns with perinatal asphyxia and cardiovascular involvement is associated with poor outcome.
Methods: This prospective cohort study conducted at neonatal section of Jawaharlal Nehru Medical College and Hospital (JNMCH), AMU, Aligarh, included 126 newborns with hypoxic-ischemic encephalopathy (HIE) as case study group fulfilling inclusion criteria and 126 normal newborns as control group. All the newborns were then followed with high-sensitivity troponin I (hs-TropI) done in all newborns at 12 hours of life and echocardiography within 72 hours of life to evaluate cardiac function.
Results: In our study cardiac dysfunction was present in 51.56% of newborn with HIE. Systolic dysfunction was present in only 9.4% case study group whereas diastolic dysfunction was present in 60 (46.9%) of case study group which was significantly higher than the control group (n=6;4.7%) (p value <0.001). In our study pulmonary arterial hypertension (PAH), mitral regurgitation (MR), tricuspid regurgitation (TR), and patent ductus arteriosus (PDA) were present in 38.3%, 2.3%, 46.1% and 4.7% in case study group. ROC curve analyses showed that a cut off value of hs-Trop I 64.8 ng/l can predict cardiac dysfunction in HIE newborn with sensitivity, specificity and accuracy of 78.89%, 58.06%, 68.75% respectively.
Conclusions: Cardiac dysfunction was prevalent in children with HIE especially associated with diastolic dysfunction along with PAH. Cardiac dysfunction signifies poor outcome among HIE cases. Hs-Trop I showed positive association with cardiac dysfunction and can early predict the myocardial injury at 12 hours of life.

Keywords: HIE, HsTrop I, Cardiac dysfunction, PAH

INTRODUCTION

Perinatal asphyxia is a major cause of neonatal and under 5 mortalities particularly in developing countries.[1] Hypoxic-ischemic encephalopathy (HIE) occur in 1-3/1000 live births in high income countries and up to 20 per 1000 live births in low and middle income countries.[2] According to National Neonatal-Perinatal Database (NNPD) report from India 2003, 28.8% of total neonatal death occur due to perinatal asphyxia and incidence of HIE is 1.4%.[3] According to United Nations International Children's Emergency Fund (UNICEF) birth asphyxia account for 20% of all neonatal death in India.[4] World Health Organization (WHO) defined perinatal asphyxia not able to begin and sustain breathing at birth. About 19.8% of newborn admitted to neonatal intensive care unit (NICU) have birth asphyxia.[5]

In perinatal asphyxia multiorgan dysfunction is common and cardiovascular involvement is associated with poor outcome. As per study in term infants with asphyxia, renal, CNS, cardiac and lung dysfunction occur in 50%, 28%, 25%, and 25% cases respectively.[6]

28% to 73% of neonatal asphyxia cases can have myocardial damage.[7] Hypoxic injury resulting in cardiac dysfunction leads to subendocardial tissue scarring, papillary muscle damage and myocardial ischemia, poor contractility, cardiac stunning, tricuspid insufficiency, hypotension and pulmonary arterial hypertension (PAH).[8] Abnormalities in cardiac function in birth asphyxia by includes transient ischemia of myocardium, transient tricuspid insufficiency, mitral incompetence, persistent pulmonary hypertension of newborn, dilated cardiomyopathy, cardiac dysrhythmia, congestive cardiac failure.[9]

Cardiac Trop I is known to be a gold standard biomarker for myocardial injury.

It was observed that cases experienced birth asphyxia has significant rise in levels of Trop I making this cardiac biomarker good indicator for predicting myocardial injury.[10] Troponin I has high sensitivity and specificity for depicting microscopic cardiac injury that is released in response to myocardial ischemia. Troponin appear in blood in 4-6 hour after perinatal asphyxia with cardiac compromise and peaks between 18 to 24 hour and remain elevated for 21 days.[11] So early cardiac Trop I concentration can aid diagnosis of myocardial dysfunction in perinatal asphyxia and can be used as a proxy marker for anticipated severity of myocardial dysfunction.[12]

Due to scarce health care facilities, a large proportion of mother deliver the babies in suboptimal conditions. This in turn leads to increased risk of birth asphyxia. The early identification of dysfunction is of vital importance to aid timely management. Therefore, the present study is done to explore the role of high-sensitivity troponin I (hs-Trop I) in early identification of myocardial dysfunction.

Gaps in knowledge

Gaps include kagnitude of cardiac dysfunction in newborns with HIE, outcome in newborn with cardiac dysfunction in HIE, role of cardiac biomarker like hs-Trop I in predicting cardiac dysfunction.

Aim

The aim of the study was to evaluate cardiac function in newborns with HIE, and to evaluate hs-Trop I as early marker of myocardial dysfunction in HIE patient.

METHODS

Study design, location and duration

This was a hospital based prospective cohort study conducted in neonatology division (NICU) in collaboration with interdisciplinary pediatric cardiac center (IPCC), Department of Pediatrics, Jawaharlal Nehru Medical College and Hospital, Aligarh Muslim University, Aligarh during the period from July 2022 to July 2024.

Inclusion criteria

Newborn with birth asphyxia according to NNPD criteria, term babies and late preterm >34 weeks, and participants with APGAR<7 were included.

Exclusion criteria

Participants with antenatal CHD, sepsis, and chorioamnitis were excluded.

Sample size

The sample size in study was calculated by using formula given, where p is the proportion that is estimated prevalence of cardiac involvement among newborn with HIE.

$$n = 4p(100 - p)/d^2$$

The proportion of cardiac involvement in asphyxia not more than 25% according to data available. Hence by using the above formula estimated sample size was 115 at 95% confidence limit and 5% relative allowed error. After adding 10% attrition rate sample size came out to be 128. A total of 256 neonates (128 cases and 128 controls) were enrolled in the study.

Methodology

Neonate with perinatal asphyxia admitted within 12 hours of delivery into neonatology division, department of pediatrics, satisfying inclusion criteria of study were enrolled after having written informed consent, obtained from either parent along with clear explanation of the purpose of study, all expected benefits and potential harms of study. Detailed maternal and neonatal information was collected and recorded in predesigned performa. All enrolled subjects in the study were graded for HIE according to Sarnat and Sarnat classification. All relevant investigations were sent. The baby is then followed by echocardiography at 72 hours of life done at IPCC.

Systolic dysfunction

Based upon ejection fraction: normal >55%, mildly reduced - 41-55%, moderately reduced 31-40%, and severely reduced <30%.[13,14]

Diastolic dysfunction

On pulsed wave Doppler: grade 1 - E/A ratio 2 and E/e'>14, grade 2 - E/A ratio 0.8-2 and E/e' 10-14, and grade 3 - E/A >2 and E/e'>14.[13,14]

Then the baby is followed till discharge, all relevant investigations were collected, inotropic support, antiseizure medication, need for CPAP or ventilator support, duration of hospital stay and outcome.

Statistical analysis

SPSS-PC-25 version is used for data entry and analysis. Shapiro-Wilk normality test is used for normal distribution of different parameters for all qualitative variables Pearson Chi square test is used. Unpaired t test or Mann Whitney U test was used to compare mean of two groups. Kruskal Wallis H test was used to analyse if there were more than two groups. ROC curve was utilised to find correlation and cutoff values were calculated by Youden index. P value <0.05 was considered significant.

RESULTS

Two hundred fifty-six (256) babies were enrolled in study which include 128 controls and 128 cases with perinatal asphyxia. These 128 cases were further classified for HIE staging according to Sarnat and Sarnat staging system. Out of 128 cases, 88 (68.8%) were stage I HIE, 18 (14.1%) where stage II HIE and 22 (17.2%) where stage III HIE.

There was higher proportion of male population in case study group than control group. In case study group males (n=74; 57.8%) were more than females (n=54; 42.2%) with male: female 1.37:1, while in control group females (n=67; 52.3%) were similar to male (n=61; 47.7%) with male: female 0.91:1. There were more preterm population in study group (n=44; 34.4%) than control group (n=24;18.8%). There were more newborns delivered via LSCS in study group (n=65;50.8%) than control group (n=53; 41.4%) (p=0.16).

Majority of mothers of study population in our study were multiparous in both case study group (n=73; 57%) and control group (n=75; 58.6%). There were significantly higher neonates with meconium-stained amniotic fluid cases in study group population (n=53; 41.4%) in comparison to control group (n=26; 20.3%).

Maternal risk factors like premature rupture of membrane (PROM) was present in 28 (21.9%) newborns in HIE case study group which is only 12 (9.4%) in control group. It is observed that PIH was present in 29 (22.7%) newborns in HIE case study group whereas this proportion is only 12 (9.4%) in control group. Other maternal risk factors like GDM, oligohydramnios and polyhydramnios were present in 7.8%, 16.4%, and 7.8% of HIE case study group.

In our study 11 (8.5%) require nasal prongs only, 52 (40.6%) require CPAP oxygen support, whereas 23 (17.9%) require ventilator support in HIE case study group. Majority of study population in HIE stage I (n=41; 46.6%) and HIE stage II (n=11; 61.1%) require CPAP for respiratory support while, most of neonates in stage III HIE (n=21; 95.5%) require mechanical ventilation for respiratory support.

The mean value of hs-Trop I at 12 hours of life in newborns with HIE was 83.85±48.98 ng/l which was significantly higher than control group 28.14±20.64 ng/l (p value <0.001) (Table 1).

Table 1: Hs-Trop I levels between cases and controls.

Parameter	Cases (n=128)	Control (n=128)	P value
Hs-Trop I	83.85±48.98	28.14±20.64	<0.001

The high levels of hs-Trop I correlate with the stages of HIE. The mean value of hs-Trop I in HIE stage I, II, III were 67.23±33.63 ng/l, 100.10±56.79 ng/l, 137.04±53.53 ng/l respectively (Table 2).

Table 2: Hs-Trop I level between different stages of HIE.

Parameter	HIE I (n=88)	HIE II (n=18)	HIE III (n=22)	P value
Hs trop I	67.23± 33.63	100.10± 56.79	137.04± 53.53	<0.001

As shown in Table 3, M mode parameters in echo were compared between cases and controls. LV diameter was significantly increased in cases as compared to controls in both systole and diastole. IVS thickness during diastole was significantly increased in cases suggestive of septal hypertrophy when compared to controls. LVEF was significantly increased in cases when compared to controls indicating systolic functions were not much compromised in initial first 72 hours of life in newborns with HIE.

Table 3: M mode echo parameters in between cases and controls.

Echo parameter	Cases (n=128)	Control (n=128)	P value
IVSs	0.39±0.07	0.38±0.01	0.41
IVSd	0.42±0.05	0.38±0.02	<0.001
LVDd	2.48±0.34	2.36±0.38	0.01
LVDs	1.60±0.18	1.54±0.28	0.03
LVPWd	0.35±0.05	0.40±0.02	<0.001
LVPWs	0.41±0.05	0.39±0.01	<0.001
EDV	12.81±1.11	12.95±0.86	0.25
ESV	04.94±0.45	05.11±0.45	<0.01
LVEF	62.11±4.85	60.77±4.40	0.02
%FS	34.91±4.42	34.67±5.33	0.69

Parameters measured in cm; *statistically significant, LVDd: left ventricle dimension during diastole; LVDs: left ventricle dimension during systole; IVSs: inter ventricular septal thickness during systole; IVSd: inter ventricular septal thickness during diastole; LVPWd: left ventricle posterior wall thickness during diastole; LVPWs: left ventricle posterior wall dimension during systole; LVEF: left ventricle ejection fraction

On comparing pulsed wave Doppler parameters, early diastolic velocities were decreased as compared to late diastolic velocities across mitral and tricuspid valves in cases when compared to controls. M-E/A and T-E/A ratios

were significantly reduced in cases when compared to controls which shows diastolic dysfunction in newborns with HIE.

Table 4 shows comparison in tissue Doppler parameters in between cases and controls. LV late diastolic (A') reduced in cases when compared to controls. E/e' velocity was significantly increased in cases when compared to controls signifying increased LV end diastolic pressure and thus LV diastolic dysfunction.

Table 4: Pulsed Doppler and tissue Doppler echo parameters in between cases and controls (n=128).

Echo parameter	Cases (n=128)	Control (n=128)	P value
T- E	0.54±0.05	0.52±0.03	0.001
T- A	0.58±0.07	0.49±0.03	<0.001
T- E/a	0.93±0.13	1.06±0.10	<0.001
M- E	0.506±0.087	0.501±0.033	0.54
M- A	0.557±0.045	0.499±0.028	<0.001
M- E/A	0.915±0.195	1.07±0.71	0.01
RV- e'	0.058±0.007	0.057±0.006	0.16
RV- a'	0.062±0.010	0.056±0.006	<0.001
LV- e'	0.059±0.041	0.058±.007	0.86
LV- a'	0.053±0.010	0.055±0.006	0.06
LV- s'	0.055±0.009	0.056±0.006	0.18
E/e'	9.15±1.75	8.63±0.96	<0.001

*Statistically significant, velocity in m/s; M-E: early diastolic velocity across mitral valve; M-A: late diastolic velocity across mitral valve; T-E: early diastolic velocity across tricuspid valve; T-A: late diastolic velocity across tricuspid valve; LV-E': annular velocity of LV during early diastole; LV-A': annular velocity of LV during late diastole; LV-S': annular velocity of LV during systole; RV-E': annular velocity of RV during early diastole; RV-A': annular velocity of RV during late diastole

Systolic dysfunction was present in only 9.4% case study group which is comparable to 7% in control group (p value 0.48).

Diastolic dysfunction was present in 60 (46.9%) of HIE case study group with respect to 6 (4.7%) in control group with statistically significant p value <0.001.

In our study high grades of diastolic dysfunction were associated with higher stages of HIE. Grade III diastolic dysfunction was only present in stage III HIE whereas grade II diastolic dysfunction was also present in high proportion in stage III HIE (45.45%) than stage II (33.33%) and stage I HIE (2.27%). Severity of diastolic dysfunction can be correlated with stages of HIE.

Cardiac dysfunction was present in 51.56% of newborn with HIE. In our study cardiac dysfunction in HIE stage I, II and III was 42%, 72.2% and 72.7% respectively.

ROC curve for predicting cardiac dysfunction in HIE showed that in comparison of echocardiography which is gold standard for evaluating cardiac dysfunction, a cut off value of hs-trop I 64.8 ng/l can predict cardiac dysfunction with sensitivity of 78.8%, specificity of 58.1%, accuracy of 68.8% with high NPV of 72% and PPV of 66.7%.

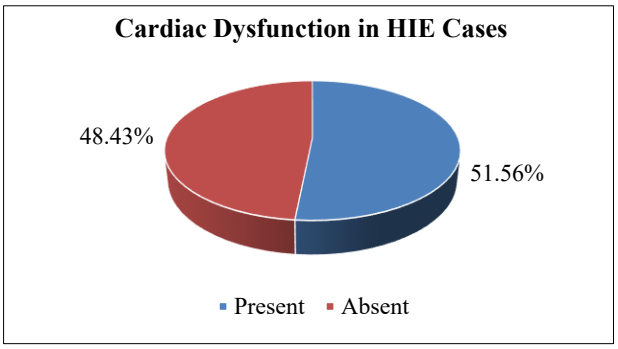

Figure 1: Cardiac dysfunction in hie cases.

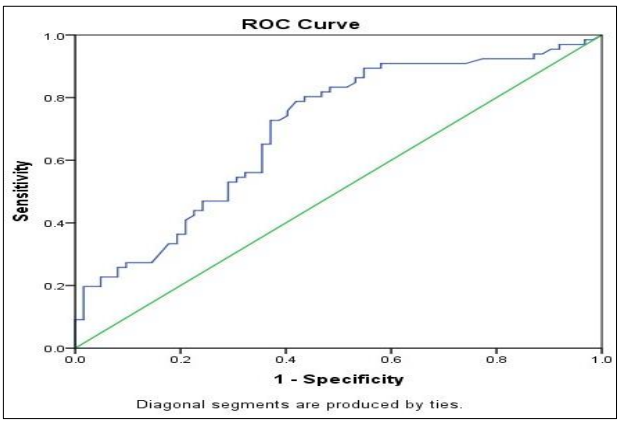

Figure 2: ROC curve using Hs trop I to predict cardiac dysfunction.

Table 5: Echo finding between cases and controls (n=128).

Echo parameters	Cases, N (%)	Control N (%)	P value
Diastolic dysfunction	60 (46.9)	6 (4.7)	<0.001
Systolic dysfunction	12 (9.4)	9 (7.0)	0.48
PAH	49 (38.3)	10 (7.8)	<0.001
PDA	10 (7.8)	6 (4.7)	0.43
TR	59 (46.1)	12 (3.9)	<0.001
MR	3 (2.3)	2 (1.6)	1.0

PAH was present statistically significant in case study group (n=49; 38.3%) as compared to control group (n=10, 7.8%) with p<0.001.

Out of 128 neonates with HIE 59 (46.1%) had TR which was statistically significant when compared to control group (n=12, 3.9%) with p value <0.001.

Mortality in neonates with HIE was 20.31% which is quite significant when compared to control group (p value

<0.001). Mortality occurred in stage III HIE was 86.4% (n=19) which is very high mortality when compared to stage I HIE 6.8% (n=6) and stage II HIE 5.6% (n=1).

Table 6: Values of hs-Trop I in terms of outcome of study.

Outcome	Recovered (n=102)	Not recovered (n=26)	P value
Hs-Trop I (ng/l)	72.20±41.03	127.44±52.38	<0.001

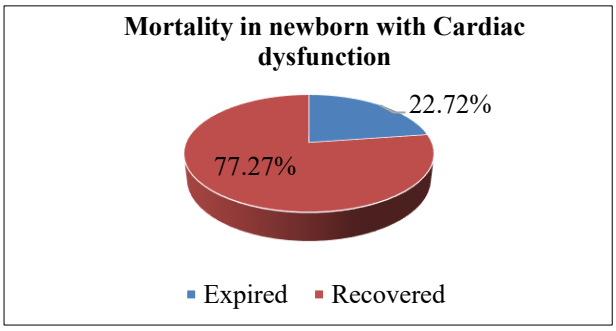

Figure 3: Mortality in newborn with cardiac dysfunction.

In our study out of 66 (51.56%) newborns with cardiac dysfunction 15 (22.72%) were not recovered.

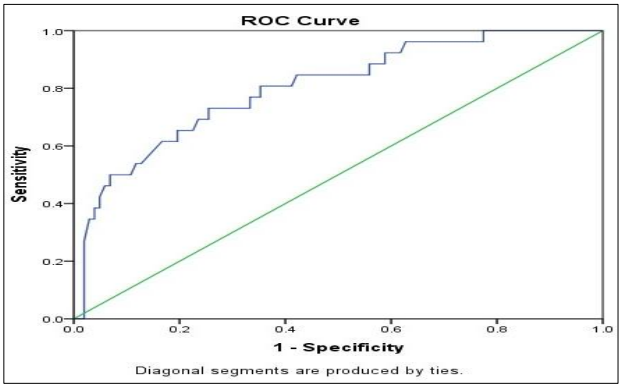

Figure 4: ROC curve using Hs trop I to predict outcome in HIE subjects.

On plotting ROC curve for predicting outcome in HIE, it is observed that a cut off value of hs-trop I 92.1 ng/l can predict outcome (mortality) with sensitivity of 73.1%, specificity 74.5%, accuracy of 74.2% with high NPV of 91.6% and a PPV of 42.2%.

DISCUSSION

Overall cardiac dysfunction present in our study in HIE case study group was 66 (51.56%). Diastolic dysfunction (46.9%) was present more than the systolic dysfunction (9.4%). Singh et al reported 32% incidence of cardiac dysfunction in their study with 27% having abnormal echo.[15] In other study Armstrong et al cardiac dysfunction was present in one-third of cases.[16] Divya et al also reported cardiac dysfunction in 30% cases.[17] Cardiac dysfunction was present more in higher stages of HIE. In our study cardiac dysfunction was 72.2% in HIE stage II and 72.7% in HIE stage III whereas it was only 37 (42%) in HIE stage 1. Jain et al also reported more cardiac dysfunction in higher stages of HIE with increase proportion of MR, TR, RV dilatation, LV dilatation with reduced ejection fraction in higher stages of HIE.[18] As the high levels of hs-Trop I are related with stages of HIE and cardiac dysfunction was also high in higher stages of HIE.

In our study the mean value of hs-Trop I measured at 12 hours. of life in HIE case study group was 83.85±48.98 ng/l which in comparison to control group 28.14±20.64 ng/l was statistically significant with p value<0.001. So high levels of hs-Trop I are associated in newborn with HIE than in control group. Jiang et al also evaluated the level of hs-Trop I as a cut off value of 87 ng/l can predict myocardial injury in babies born with HIE.[7] Similarly another study Lee et al evaluated Trop I at 6 hours of life as a cut off value of 60 ng/l and above can be used to predict moderate to severe HIE stage and a cut off value of 180 ng/l can be used to predict need for resuscitation and inotropic support.[19] In our study the average value of high sensitivity troponin I came out in stage I HIE 67.23±33.63 ng/l, stage II HIE 100.10±56.79 ng/l, stage III HIE 137.04±53.53 ng/l, and these mean values were found to be statistically significant with p value <0.001. So, the levels of hs-Trop I corelates well with stages of HIE. Higher stages of HIE are associated with high value of hs-Top I. So, the levels of hs-Trop I correspond well with the stages of HIE. Jain et al also calculated Trop I levels in controls as 20 ng/l whereas levels of cardiac Trop I in stage I 90±40 ng/l, stage II 170±70 ng/l, and stage III 260±80 ng/l.[16] Shastri et al evaluated cardiac Trop I at 36 hour of life with median values as stage I HIE 40 ng/l, stage II HIE 120 ng/l, stage III HIE 670 ng/l.[15] Also in our study, the mean value of hs-Trop I in expired cases in HIE case study group was 127.44±52.38 ng/l which was statistically significant when compared to neonates who recovered 72.20±41.03 ng/l with p value <0.001. So, the expired cases of HIE has higher value of hs-Trop I than recovered cases. So hs-Trop I levels can correlate well in terms of outcome of HIE, as high levels of hs-Trop I can be associated with poor outcome of HIE.

A cut off value of hs-Trop I 64.8 ng/l can depict the cardiac dysfunction in HIE with sensitivity, specificity and accuracy of 78.79%, 58.06% and 68.75%. As the sensitivity and specificity are less than 80% but, it can be used to support the diagnosis of cardiac dysfunction with PPV of 66.7% and NPV of 72% where echocardiography is still not available in low- and middle-income countries.

In our study 26 (20.3%) cases were expired in which majority were HIE III neonates. Zainab et al showed high mortality in advance stages of HIE, 96.1% mortality in stage III HIE which is higher than our study (86.4%).[20] Cardiac dysfunction in our study was present in 51.56% of

case study population out of which 22.72% expired. Singh et al reported cardiovascular involvement in 32% of newborn having birth asphyxia out of which 42% newborn died.[13] Shah et al in his study showed 64% mortality in newborn with HIE having cardiovascular involvement which is quite high than our study.[6]

From the ROC curve the AUC is 0.80 with 95% CI and p value <0.001 shows hs-Trop I having positive correlation with mortality in HIE. Higher the value of hs-Trop I, higher will be chances of worst prognosis and mortality. A cut off value of hs-Trop I 92.1 ng/l can depict the worst outcome in HIE with sensitivity, specificity and accuracy of 73.08%, 74.51% and 74.22%.

Limitations

Sample size of our study is limited, LA volume index was not measured for diastolic dysfunction, and GLS was not measured for systolic dysfunction though it is most sensitive parameter for systolic dysfunction.

CONCLUSION

Cardiac dysfunction in our study was present in 51.56% of HIE cases especially in advance stages of HIE. Most of study population has diastolic dysfunction along with PAH. There was also high mortality associated with cardiac dysfunction in HIE. Cardiac dysfunction signifies poor outcome among HIE cases. Hs-Trop I at 12 hours of life showed significant rise in levels after myocardial injury depending on the stages of HIE. Hs-Trop I showed positive association with cardiac dysfunction and can early predict the myocardial injury at 12 hours of life. But it cannot be an ideal screening test in predicting myocardial dysfunction because of its low sensitivity and specificity. Though it can be used to depict cardiac dysfunction in resource limited setting where echocardiography is not available.

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