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Study of troponin I levels in evaluation of cardiovascular status of full-term birth asphyxiated neonates

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Abstract

Introduction: Birth asphyxia is a critical condition in newborns resulting from a severe imbalance between oxygen supply and demand, significantly contributing to neonatal mortality worldwide. Troponin I, a biomarker for myocardial injury, has emerged as a valuable tool in assessing the cardiovascular status of asphyxiated neonates. This study explores the levels of Troponin I in full-term birth asphyxiated neonates and its relationship with cardiovascular outcomes.

Objectives:

1. To study the Troponin I level in birth asphyxiated full term neonates and full-term healthy controls.
2. Study of Troponin I level in evaluation of Cardiovascular status of birth Asphyxiated full-term Neonates.
3. To determine relationships between Troponin I and different gradings of Hypoxic-Ischemic Encephalopathy (HIE) and Mortality
4. Correlation of Troponin I level with systemic neurological involvement

Methods: This hospital-based cross-sectional observational study was conducted at Krishna Institute of Medical Sciences, Karad, Maharashtra. The study included 120 full-term neonates: 60 with birth asphyxia (Apgar score < 7 at five minutes) and 60 healthy controls. Blood samples were collected at 24-48 hours and at 5 days of life to measure Troponin I levels. Clinical data, including Apgar scores, HIE staging, and cardiovascular status, were recorded

Results: Troponin I levels were significantly higher in asphyxiated neonates compared to controls at 24-48 hours post-birth (mean 1.453 ng/ml vs. 0.0315 ng/ml, $p < 0.0001$). Troponin I level correlated with the severity of birth asphyxia, with higher levels indicating greater myocardial injury. Elevated Troponin I levels were associated with increased mortality and higher incidences of neurological and cardiovascular involvement.

Conclusion: Troponin I is a reliable marker for myocardial injury in birth asphyxiated neonates, with higher levels correlating with more severe outcomes. Routine screening of Troponin I in high-risk neonates could guide early interventions and improve neonatal care outcomes.

Keywords: Birth Asphyxia, Troponin I, Myocardial Injury, Neonates, Cardiovascular Status, Hypoxic-Ischemic Encephalopathy.

Introduction

Birth asphyxia is a critical condition in newborns that results from a prolonged or severe imbalance between oxygen supply and demand [1]. It significantly contributes to neonatal mortality worldwide and can lead to severe long-term health issues. Troponin I, a biomarker for myocardial injury, has emerged as a valuable tool in assessing the cardiovascular status of asphyxiated neonates. This study aims to explore the levels of Troponin I in full-term birth asphyxiated neonates and its relationship with cardiovascular outcomes.

Birth asphyxia occurs due to several factors that can be categorized as antepartum, intrapartum, and postpartum. Antepartum factors include maternal age, medical conditions like eclampsia, and multiple pregnancies. Intrapartum factors involve prolonged labor, foetal distress, and presence of meconium-stained liquor. Postpartum factors are less common but can include issues like respiratory distress syndrome.[2]

The effects of birth asphyxia on newborn health are profound. It can lead to hypoxic-ischemic encephalopathy (HIE), a condition characterized by brain dysfunction due to lack of oxygen. Other complications include cardiac issues, metabolic acidosis, and multiorgan failure. The severity of these outcomes largely depends on the duration and extent of oxygen deprivation.[3]

According to the World Health Organization (WHO), birth asphyxia accounts for approximately 24% of all neonatal deaths globally. The majority of these deaths occur in developing countries, where access to timely and adequate medical care is limited.[4]

Troponin I is a regulatory protein found in cardiac muscle that helps regulate the contraction of the heart. It is released into the bloodstream during myocardial injury, making it a sensitive and specific marker for cardiac damage.[5]

In the context of birth asphyxia, Troponin I level rise as a response to hypoxic-ischemic injury to the heart. Elevated levels indicate myocardial damage, which can be used to assess the severity of asphyxia and guide treatment decisions.

While other biomarkers like creatine kinase-MB (CK-MB) and lactate dehydrogenase (LDH) can also indicate cardiac injury, Troponin I is preferred due to its higher specificity and sensitivity in detecting myocardial damage.[6]

Methodology

Study Design: This is a hospital-based cross-sectional observational study conducted in Krishna Institute of Medical Sciences, Karad, Maharashtra. The study includes full-term neonates diagnosed with birth asphyxia and a control group of healthy full-term neonates.

Patients Included: The cases included term babies delivered in the hospital with evidence of asphyxia indicated by any of the following:

1. Failure to initiate and sustain breathing at birth (WHO).

2. Moderate asphyxia as slow gasping breathing or Apgar of 4-6 and severe asphyxia as no breathing or Apgar of 0-3 at 1 minute of life (NNPD).

3. Cord blood pH < 7 (AAP).

4.Clinical neurologic manifestation and any Multisystemic involvement (AAP).

Patients Excluded: Cases with:

1. Congenital anomalies

2. Congenital heart disease

Sample Size: A sample size of 120 was calculated (60 each for subjects and controls) using the formula for comparison of two means. Hence, a total of 120 newborn babies consisting of 60 birth asphyxiated babies with HIE (subjects) and an equal number of healthy non-asphyxiated babies (controls) were recruited over a period of 18 months**.** Blood samples were collected from the neonates at 24-48 hours and at 5 days of life to measure Troponin I levels. Clinical data, including Apgar scores, HIE staging, and cardiovascular status, were recorded.

Recruitment procedure: The study subjects were recruited from the NICU, while the controls were selected from the hospital's postnatal wards. Consecutive term neonates with HIE, admitted to the NICU within 24 hours of birth and meeting the study inclusion criteria, were enrolled after obtaining written informed consent from their parents or caregivers. Each baby was assessed using Sarnat and Sarnat staging at admission and then daily, with the highest stage assigned to the subjects. Serum Troponin I was done. Two milliliters of blood was collected and transferred into a plain bottle. The blood sample collected was allowed to clot and fully retract for 1 h at room temperature and then centrifuged at 3000 rpm for 10 min; serum was harvested. Serum troponin I was subsequently analyzed using AIA-360 Automated Immunoassay Analyzer. The ELISA kit is an in vitro ELISA for the quantitative measurement of human troponin I in serum.

Results

Baseline characteristics such as birth weight, gestational age, and maternal health factors were compared between the asphyxiated and control groups as shown in **Table 1** to ensure that any observed differences in Troponin I levels and outcomes could be attributed to birth asphyxia rather than other variables.

Table 1: Comparison of baseline characteristics of asphyxiated group and controls.

Characteristic	Cases (N=60)		Controls (N=60)		Unpaired t Value	p Value
	Mean	SD	Mean	SD		
GA (Week)	38.685	0.99	38.6	1.01	0.4655	0.6424
Birth Weight (Kg)	2.867	0.38	3	0.349	1.9967	0.0482
APGAR (1 Min)	2.56	0.738	6.8	0.4	39.1252	<0.0001
APGAR (5 Min)	4.66	0.723	8.81	0.387	63.4433	<0.0001

Mean value of Troponin I is 1.128, 1.254 and 2.286 across HIE Stage 1 ,2 and 3 (**Table 2**) respectively, which shows an increasing trend in value of Troponin I as the birth asphyxia gets severe.

Table 2: Serum Troponin I values across stages of disease:

Variable	HIE 1	HIE 2	HIE 3
Troponin I (ng/ml)	1.128	1.254	2.286

The study found significantly higher Troponin I level in asphyxiated neonates compared to the control group at 24-48 hours post-birth. Mean Troponin I Value of

Asphyxiated group is 1.453 and Control group was 0.0315 as shown in **Table 3**.

Table 3: Value of Troponin I at 24-48 hours

Troponin I	Cases (N=60)	Controls (N=60)	Unpaired t-Value	p Value
Mean	1.453	0.0315	16.3574	< 0.0001
SD	0.673	0.014		

Number of subjects having Neurological Involvement who have Troponin i value between 0–1 ng/ml are 5 with no one having Cardiovascular Involvement, in subjects having Troponin i Value Between 1-2 ng/ml Neurological Involvement is in 6 subjects and lastly in subjects having Troponin i value > 2 ng/ml Neurological Involvement is in 7 subjects while 6 subjects are having Cardiovascular Involvement as shown in Table 4.

Table 4: Correlation of Troponin I value with Neurological and Cardiovascular Involvement in Asphyxiated subjects

Systemic Involvement	Troponin I (0-1 ng/ml)	Troponin I (1-2 ng/ml)	Troponin I (>2 ng/ml)
Neurological Involvement	5 (8.33%)	6 (10%)	7 (11.66%)
Cardiovascular Involvement	0	0	6 (10%)

Number of deaths in subjects having Troponin I value between 0–1 ng/ml are 0, in subjects having Troponin I Value Between 1-2 ng/ml number of deaths are 3 and 28 subjects survived and lastly in subjects having Troponin I value > 2 ng/ml number of deaths are 11 with 0 survivors as shown in Table 5.

Table 5: Correlation of Troponin I value with Outcomes in Asphyxiated subjects

Outcome	Troponin I (0-1 ng/ml)	Troponin I (1-2 ng/ml)	Troponin I (>2 ng/ml)
Survived (46)	18 (39%)	28 (61%)	0
Death (14)	0	3 (21.4%)	11 (78.6%)

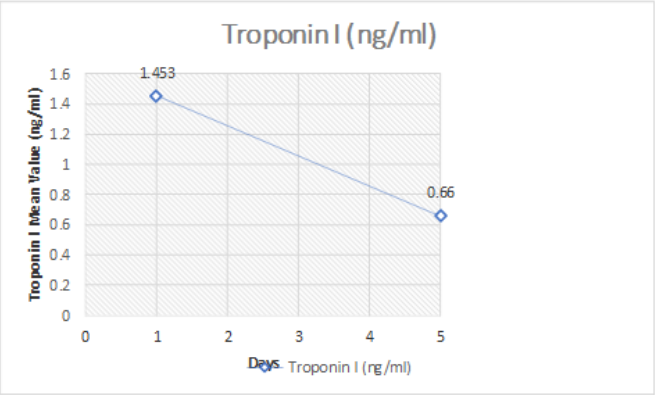
The difference in Troponin I levels between the asphyxiated and control groups was statistically significant ($p < 0.0001$). This indicates that Troponin I is a reliable marker for myocardial injury in birth asphyxiated neonates.

Troponin I level remained elevated at 5 days in the asphyxiated group but showed a slight decrease compared to the levels at 24-48 hours. 4

The mean level at 24-48 Hours Of life was 1.453 ng/ml and at Day of Life 5 was 0.0315 ng/ml in the asphyxiated group which is showing a drop in the values.

This has been depicted in Figure 1.

Figure 1: Troponin I Mean Values of Cases over Days following birth



Discussion

The elevated Troponin I levels in asphyxiated neonates highlight the presence of myocardial injury due to birth asphyxia. The decrease in levels at 5 days suggests some recovery but still indicates ongoing cardiac stress.

Our findings are consistent with previous studies that have reported elevated Troponin I levels in neonates with birth asphyxia.

The study presents data on the outcomes of infants with different stages of HIE, highlighting the correlation between the severity of HIE and mortality rates. In HIE Stage 1 all 28 cases (100%) survived, In HIE Stage 2: 15 cases (88.2%) survived while 2 died and lastly in HIE Stage 3 only 3 cases (20%) survived while there were 12 deaths (80%). In Issa, et al [7] HIE Stage I: 23 (100.0%) survived while 0 (0.0%) died, in HIE Stage II: 51 (98.1%) survived and 1 (1.9%) died and in HIE Stage III: 6 (60.0%) survived ,4 (40.0%) died, this is not similar to our study.

The mean Troponin I value in the asphyxiated group was 1.453 ng/ml with a standard deviation (SD) of 0. 673. The mean Troponin I value in the control group was 0.0315 ng/ml with an SD of 0.014. The unpaired t-value was 16.3574, and the p-value was less than 0.0001, indicating a highly significant difference can be seen between the two groups. Troponin I value in Asphyxiated subjects in Issa, et al [7] was 1.26 which is similar to our present study, while in Trevisanuto, et al [9] it was 0.36 in cases lower than the present study while in control the mean value is 0.04 similar to findings of present study. The levels observed in our study were similar to those reported by Issa et al. (2018) [7] and Shastri et al. [8] (2019), further validating the use of Troponin I as a biomarker.

Number of subjects having Neurological Involvement who have Troponin I value between 0–1 ng/ml are 5 with no one having Cardiovascular Involvement, in subjects having Troponin I Value. Between 1-2 ng/ml Neurological Involvement is in 6 subjects and lastly in subjects having Troponin I value > 2 ng/ml Neurological Involvement is in 7 subjects while 6 subjects are having Cardiovascular Involvement. Cardiovascular involvement, specifically hypotension and cardiogenic shock, was noted in subjects with Troponin I levels >2 ng/ml. This correlation further supports the role of Troponin I as the biomarker for assessing the extent of systemic involvement in BA. In Shastri AT, et al., [8] duration of ionotropic use is increasing (which is suggestive of Cardiovascular Involvement) with increasing levels of Troponin and Stage of HIE similar to our study. Similar to Issa, et al [7] there is increasing severity of HIE with increasing levels of Troponin I in our study.

The study underscores the importance of measuring Troponin I levels in neonates with suspected birth asphyxia. Early identification of myocardial injury can help guide interventions to improve outcomes. Routine screening of Troponin I in high-risk neonates could be beneficial in neonatal intensive care units.

Conclusion

Cardiac troponin I was significantly higher in Birth asphyxiated subjects compared with controls. Elevated troponin I was associated with increased mortality, Cardiovascular and Neurological Involvement. Higher values are correlating with higher mortality, Cardiovascular and Neurological Involvement and higher values >2ng/ml can be considered as additional biomarker for the above parameters.

Limitations

1. The follow-up period of the study is relatively short, focusing on immediate outcomes rather than long-term effects. Longer follow-up periods are necessary to understand the lasting impacts of elevated Troponin I levels and birth asphyxia on neonatal health.

2. The study may have unaccounted factors that influenced the results. Factors such as maternal health, prenatal care, and socio-economic status can significantly impact neonatal outcomes and need to be controlled for.

3. While Troponin I is a crucial biomarker, relying solely on it might not provide a complete picture of cardiac health. Including other cardiac biomarkers such as Troponin T, CK-MB, and myoglobin could offer a more comprehensive assessment of myocardial injury.

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