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Troponin Evaluation in Hemodynamically Unstable Neonates and its Correlation with Echocardiographic Findings

Wafaa O Ahmed[1], Walid A. Afifi[2], Hisham A. Awad[3], Heba E. Radwan[4], Mohamed M. El-Sonbaty[5], Heba E. Hashem[6], Ahmad A. Obaid[7], Osama A. Obaid[8], Eman M. Elsayed[9]

[1,3,9]Pediatric Department, Faculty of medicine, Ain Shams University, Cairo , Egypt

[2]Department of acute medicine, London North West Trust University Hospital, London, UK.

[4]Pediatric resident, Ministry of Health, Egypt.

[5]Child health department, National Research Centre, Giza, Egypt.

[6]Clinical pathology department, Cairo, Egypt.

[7]Laboratory Medicine Department, Faculty of Applied Medical Sciences, Umm Al-Qura University, Makkah, Saudi Arabia.

[8]Department of Pediatrics, Maternity and Children Hospital, Makkah, Saudi Arabia.

Corresponding author (*): Wafaa O Ahmed
Email: wafaaosman83@med.asu.edu.eg

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Abstract

Objectives: An electrocardiogram can provide real-time information on the cardiovascular stability in conjunction with the clinical symptoms of the ill newborn. Cardiac troponin T (cTnT) is also a specific and sensitive biomarker for myocardial affection. **Theoretical frame work:** This controlled cross sectional study evaluated level of cTnT levels in hemodynamically sick newborns and correlate it with echocardiography. **Methods:** Sixty neonates were subdivided into 2 groups; 30 healthy controls and 30 hemodynamic unstable ventilated and non-ventilated cases, admitted to the tertiary care NICU. Clinical examination, laboratory investigations, such as complete blood count (CBC), liver function tests, kidney function tests, bleeding profile, serum electrolytes, capillary blood gas, and C-reactive protein (CRP), cTnT level were measured within 48 hours of diagnosis of neonatal hemodynamic instability. Conventional M mode and tissue Doppler imaging (TDI) were recorded. **Results: The** studied cases had higher cTnT levels compared to controls (p=0.000)]. Both ejection fraction% (EF) and fraction shortening (FS) were significantly decreased in cases(p<0.001), Mitral and tricuspid E/A ratios were significantly decreased in cases(p=0.041) (p=0.023) respectively, Tie index of left and right ventricles were significantly increased in cases versus controls (p=0.000) (p=0.002) respectively, Troponin T was significantly higher in hypotensive cases on inotropic support (p=0.001). **Conclusions:** increased cTnT in hemodynamically unstable neonates can be used as a marker for cardiac affection in sick babies. Hemodynamically unstable neonates revealed LV systolic and diastolic dysfunction

Key words: Cardiac troponin T; neonatal shock; Echocardiography, tissue doppler

Introduction

A collection of proteins called troponin is a part of the heart muscle's contractile system. Troponin (cTn) T and I, which are cardiac-specific, are regarded as the most reliable biochemical indicators of myocardial necrosis [1]. Many healthy newborns' blood samples have been discovered to contain cTnT Concentrations, which reflect oxygen consumption or inotropic support, are much more noticeable in ill babies experiencing shock. For neonates and those with cardiorespiratory derangement, cTnT may be a useful marker [2]. 2-4 hours after the insult, blood levels of cTn begin to rise, peak at around 12 hours, and then continue to rise for 7–10 days [3].

The key to effective treatment is early discovery and comprehension of the shock's underlying pathophysiology. Some of the first symptoms are pallor, poor appetite, tachycardia, tachypnea, and temperature instability. Hypotension is a late indicator of neonatal shock. Hypotension is the most common sign of decreased perfusion in newborn critical care units, despite being a late finding (NICUs).

Hypotension may be a clinical sign of a depleted cardiovascular reserve and is associated with a higher risk of death and less favourable neurological outcomes. Myocardial ischemia and necrosis have been proposed as the causes of this dysfunction or shocking [3].

Other conventionally used clinical signs that serve as indirect markers of cardiovascular condition include clinical evaluation, heart rate (HR), capillary refill time (CRT), urine output, and serum lactate. Instead of excluding congenital heart problems, early bedside functional echocardiography can aid in the early detection of underlying pathology by obtaining physiological data that can be used to deliver goal-oriented, time-specific therapy. Additionally, it offers reliable organ perfusion indicators and monitors changes following the intervention [4].

Measurements of the left ventricular (LV) cavity's shortening fraction (FS) and ejection fraction (EF), which are impacted by after load and preload, are not sensitive to subclinical functional changes in the infant heart. Neonatal heart function has recently been assessed using quantitative echocardiographic methods such Tissue Doppler Imaging (TDI), two-dimensional speckle tracking echocardiography, and others (2D, 3D-STE). These methods have a higher level of sensitivity [5].

The myocardial performance index, often known as the Tie index, is a powerful measure that efficiently assesses both systolic and diastolic functioning. In individuals with myocardial dysfunction, DTI is a sensitive heart failure indicator and provides prognostic information [6].

29 asphyxiated newborns and 30 control-term infants show a link between cTnT and echocardiographic myocardial damage and perinatal asphyxia in the first 24 h of life. Comparing the asphyxiated group to controls, cTnT was greater [7].

Materials and Methods

This controlled cross sectional study recruited 60 full-term neonates; 30 cases with hemodynamic instability consisted of 15 (50%) males and 15 (50%) females with a mean of 5.8 days and 30 healthy controls consisted of 14 (46.7%) males and 16 (53.3%) females with a mean age of 5.5 days, all were admitted to the NICU of Children's Hospital, Faculty of Medicine, Ain Shams University in the period from September 2021 to August 2022. Full term infants who were hemodynamically unstable, hypotensive and on cardiac support neonates were included as cases. Preterm infants, neonates with congenital heart diseases, neonates of parents that refused to give consent, asphyxiated neonates, neonates on first day of life, neonates delivered by vaginal delivery, cases of placental insufficiency and respiratory distress syndrome were all excluded from the study. Consent was taken from the care givers of the recruited neonates.

All studied newborns had thorough history, clinical examination including anthropometric measurements and the Ballard score for estimating gestational age[8]. Routine investigations in the form of complete blood count (CBC), liver function tests, kidney function tests, bleeding profile, serum electrolytes, capillary blood gas, and C-reactive protein (CRP) were done for the

studied groups. Specific investigations included cTnT using Human Troponin T enzyme-linked immune sorbent assay (ELISA) Kit for cell culture supernatants, plasma, and serum samples, with a detection range:0.35 ng/ml - 25 ng/ml. complete echocardiographic evaluation of cardiac function was performed within 48 hours of the occurrence of hemodynamic instability using M-Mode and 2-Dimensional echocardiography that helped in measuring LV internal diameter end diastole (LVIDD), LV posterior wall diameter (LVPWD), diastolic pulmonary artery pressure, PA acceleration time (PAAT), and ejection fraction that was calculated as follows; EF = $\frac{EDV - ESV}{EDV} \times 100\%$ and fraction shortening as calculated by; FS = $\frac{LVEDD - LVESD}{LVEDD} \times 100\%$ [8]. TDI was used to assess Mitral and tricuspid inflow velocities, E/A ratios, and Tie index (myocardial performance index in both ventricles by the formula IVCT+IVRT/ET) [9].

Statistical methods:

Statistical package for Social Science was used to review, code, tabulate, and introduce the acquired data to a computer (SPSS 20 for windows). Data cleaning, data quality testing, and data entry were done. Data was presented and suitable analysis was done according to the type of data obtained for each parameter. Descriptive statistics: Mean ± Standard deviation (± SD) for parametric numerical data. Frequency and percentage of qualitative data.

The statistical significance of the difference between the means of the two study groups was determined using the student t test. To explore the association between two qualitative variables, the Chi-Square test was performed. Fisher's exact test: was used to examine the relationship between two qualitative variables when the expected count is less than 5 in more than 20% of cells.

Results and Discussion

The results showed a significant decrease in hemoglobin (HG), hematocrit (HCT), platelets (PLT), sodium (Na), and potassium (K) in cases. Also there was significant increase in creatinine (Cr), AST, prothrombin (PT), partial thromboplastin (PTT), International normalized ratio (INR), and CRP in cases; and no significant difference between cases and controls as regard WBCS, ALT, PCO2, and HCO3.There was high significant increase in cases as regard heart rate (beat per minute) & perfusion (sec) but there is high significant decrease in cases as regard mean blood pressure. There was significant decrease in LVIDD, fraction shortening & ejection fraction, and a highly significant increase in PA acceleration time & diastolic PA pressure. There was a significant increase in diastolic PA pressure in cases & there is no significant difference between cases and controls as regards LVPWD. There is high significant increase in cases as regard troponin T level.

There was a significant decrease in cases as regards the mitral E/A ratio, a highly significant increase in cases as regards mitral A velocity, and the Tie Index of LV There was no significant difference between cases and controls as regards mitral E velocity.

Also a significant decrease in cases as regards tricuspid E/A ratio, a significant increase in cases as regard Tie Index RV & no significant difference between cases and controls as regard tricuspid E velocity and tricuspid A velocity.

Tricuspid A and E velocities were not linked with cTnT in the current investigation.

Blood pressure and cTnT showed a significant Pearson association.

Pearson's correlation of the lab results showed a strong association between cTnT and both creatinine and INR.

A highly substantial Pearson connection between cTnT and the tie index of the LV, tricuspid E velocity, LVIDD, and tricuspid E/A ratio was found.

Additionally, a strong Pearson association between cTnT and the tie index of RV and LVPWD was discovered.

Table (1): Comparison between studied groups as regard sex, Order of birth & consanguinity.

			Patient group		X2/Exact Fischer	P value	Sig.
			Controls	Cases			

Sex	Male	N.	14	15	0.067	0.796	NS
		%	46.70%	50.00%			
	Female	N.	16	15			
		%	53.30%	50.00%			
Order of birth	1	N.	9	10	2.307	0.679	NS
		%	30.00%	33.30%			
	2	N.	4	5			
		%	13.30%	16.70%			
	3	N.	9	10			
		%	30.00%	33.30%			
	4	N.	6	5			
		%	20.00%	16.70%			
	5	N.	2	0			
		%	6.70%	0.00%			
Consanguinity	Negative	N.	26	22	1.667	0.197	NS
		%	86.70%	73.30%			
	Positive	N.	4	8			
		%	13.30%	26.70%			

Table (2): Comparison between the two studied groups as regard vital signs.

	The studied group	N	Mean	SD	t	P value	Sig.
Heart Rate (beat per minute)	Controls	30	135.0	10.7	6.838	<0.001	HS
	Cases	30	160.5	17.4			
Perfusion (sec)	Controls	30	1.7	0.7	58.000	<0.001	HS
	Cases	30	3.7	0.8			
Mean blood pressure (mm hg)	Controls	30	54.3	8.2	-11.523	<0.001	HS
	Cases	30	29.3	8.6			

Table (3): Distribution of signs of heart failure among cases.

		N.	Percent
Palpable liver	Negative	9	30%
	Positive	21	70%
Lower limb edema	Negative	9	30%
	Positive	21	70%
Murmur	Negative	22	73.3%
	Positive	8	26.7%

Table (4): The need for oxygen support among cases.

	N.	Percent (%)
Nasal cannula	10	33.3
Mechanical ventilator	20	66.7

Table (5): Comparison between studied groups as regard laboratory findings.

	The studied group	N	Mean	SD	t	P value	Sig.
HG	Controls	30	14.3	2.0	3.268	0.002	HS
	Cases	30	12.5	2.4			
HCT	Controls	30	42.5	6.1	3.260	0.002	HS
	Cases	30	36.9	7.2			

TLC	Controls	30	10.0	4.2	-0.637	0.527	NS
	Cases	30	10.9	6.8			
PLT	Controls	30	275.8	86.9	3.140	0.003	HS
	Cases	30	210.2	74.5			
NA	Controls	30	139.8	6.7	2.767	0.008	HS
	Cases	30	135.4	5.7			
K	Controls	30	4.5	0.6	2.179	0.033	S
	Cases	30	4.2	0.5			
CREAT	Controls	30	0.7	0.1	12.247	0.000	HS
	Cases	30	1.2	0.2			
AST	Controls	30	34.8	21.8	-4.377	0.000	HS
	Cases	30	55.4	13.8			
ALT	Controls	29	24.9	8.5	-1.953	0.056	NS
	Cases	30	33.2	21.1			
PT	Controls	30	14.3	2.9	-5.914	0.000	HS
	Cases	30	22.3	6.9			
PTT	Controls	30	35.5	13.5	-4.610	0.000	HS
	Cases	30	53.2	16.2			
INR	Controls	30	0.9	0.3	-5.088	0.000	HS
	Cases	30	1.7	0.8			
PH	Controls	30	7.4	0.0	-2.520	0.015	S
	Cases	30	7.4	0.1			
PCO2	Controls	30	39.3	4.1	-0.067	0.947	NS
	Cases	30	39.5	13.6			
HCO3	Controls	30	21.1	2.7	-0.537	0.593	NS
	Cases	30	21.6	4.5			
CRP	Controls	30	0.0	0.0	-7.127	0.000	HS
	Cases	30	36.9	28.3			

Hemoglobin (HG), hematocrit (HCT), platelets (PLT), sodium (Na), and potassium (K), creatinine (Cr), AST, prothrombin (PT), partial thromboplastin (PTT)

Table (6): Comparison between studied groups as regard Troponin T level.

	The studied group	N	Mean	SD	t	P value	Sig.
Troponin	Controls	30	21.5	13.1	-8.096	0.000	HS
	Cases	30	116.3	62.8			

Table (7): Troponin T level among cases.

		N.	Percent (%)
Troponin	Negative	3	10.0
	Equivocal	9	30.0
	Positive	18	60.0

Table (8): Cardiac support medication used in cases.

	N.	Percent (%)
Dopamine 10 mic, dobutrex 20 mic	8	26.6
Dopamine 5 mic	10	33.3
Dopamine 5 mic, dobutrex 10 mic	12	40.0
Adrenaline 0.1- 0.5 mic	8	26.6
Primacore 0.5-0.75 mic	8	26.6

Table (9): Comparison between the two studied groups as regard M mode & 2D Echo parameters.

	The studied group	N	Mean	SD	t	P value	Sig.
LVIDD (CM)	Controls	30	2.9	0.6	6.294	0.000	HS
	Cases	30	1.8	0.7			
LVPWD (CM)	Controls	30	0.6	0.3	-1.385	0.171	NS
	Cases	30	0.7	0.4			
FS%	Controls	30	39.7	6.7	-4.413	<0.001	HS
	Cases	30	29.8	10.3			
EF%	Controls	30	72.5	7.2	-6.990	<0.001	HS
	Cases	30	55.4	11.3			
PA acceleration time	Controls	30	121.2	28.7	-3.516	0.001	HS
	Cases	30	190.6	104.3			
Diastolic PA pressure	Controls	30	3.7	1.9	8.388	<0.001	HS
	Cases	30	21.4	11.4			

Table (10): Comparison of ECHO findings between the two studied groups as regards tissue Doppler image (left ventricular function).

	The studied group	N	Mean	SD	T	P value	Sig.
Mitral E velocity	Controls	30	0.65	0.4	-1.470	0.147	NS
	Cases	30	0.53	0.2			
Mitral A velocity	Controls	30	0.56	0.2	2.711	0.009	HS
	Cases	30	0.70	0.2			
Mitral E/A ratio	Controls	30	1.16	0.8	-2.090	0.041	S
	Cases	30	0.80	0.5			
Tie Index LV	Controls	30	0.5	0.2	-4.729	0.000	HS
	Cases	30	1.1	0.6			

Table (11): Comparison of ECHO findings between the two studied groups as regards Tissue Doppler Image (right ventricular function).

	The studied group	N	Mean	SD	t	P value	Sig.
Tricuspid E velocity	Controls	30	0.65	0.3	-1.095	0.278	NS
	Cases	30	0.55	0.4			
Tricuspid A velocity	Controls	30	0.54	0.2	1.063	0.292	NS

	Cases	30	0.61	0.3			
Tricuspid E/A Ratio	Controls	30	1.2	0.5	-2.324	0.023	S
	Cases	30	0.90	0.5			
Tie Index RV	Controls	30	0.5	0.2	-3.190	0.002	S
	Cases	30	3.9	5.9			

Discussion:

A total of 60 full-term newborns were used in the study, and they were split into two groups: 30 were designated as cases, and the remaining 30 as controls. The mean age was 5.5±2.1 days for controls and 5.8±2.2 days for cases, with 14 (46.7%) males and 16 (53.3%) females in the control group compared to 15 (50%) males and 15 (50%) females in the cases group. Comparatively, there are two studies by Clark et al. and Awany et al. that included younger and older age groups as compared to the present study, respectively. In Clark et al study's there were two groups: 49 sick infants as cases and 113 healthy newborns as controls. Of the controls, 57% of the males had a median age of 68 hours and a gestational age of 38 weeks. With a median age of 26 hours and a gestational age of 29 weeks, the 49 unwell infant cases had 67% males. Awany et al study's however, included two groups: 40 full-term healthy neonates who were cases, with a median age of 11.5 days and a gestational age of 38 weeks (males, 52.5%), and 40 full-term healthy neonates who were controls, with a median age of 13.8 days and a gestational age of 37.9 weeks, and 55% males[11, 12].

In the present study, 13.3% and 26.7% of the studied controls and cases group, respectively, had positive consanguineous parents.

In the current study, prenatal history-taking was strengthened; rheumatic fever, antenatal history of medication intake, and hypertension were found in 13.3%, 3.3%, and 3.3% of the participants, respectively. This is consistent with the findings of Awada et al., who looked at the risk variables for pre-eclampsia, chorioamnionitis, smoking, maternal age, parity, prenatal antibiotic use, antenatal corticoids, and tocolysis in a group of women [13].

According to clinical assessments of the study groups, patients' capillary refill times (CRT) were longer than controls' (p 0.001). Similar findings were made by Fahmey et al. who found that healthy babies (n = 25) had a shorter capillary refill time than sick newborns (n = 50) [15]. Additionally, the study group's heart rate was higher than that of the controls (p 0.001). Of note, The Nem-Yun boo et al. study found that 26% of asphyxiated newborns had hypotension, while the Fahmey et al. study found that cases had lower mean blood pressure than controls (p = 0.025) [14,15]. Additionally, the current studied cases had lower mean blood pressure than controls (p 0.001), which is consistent with those studies.

Further clinical evaluation revealed that the investigated individuals had functional murmurs, palpable livers, and edoema in the lower limbs in percentages of 70, 70, and 26.7, respectively, all of which were signs of heart failure. Similar findings were made in the study by Nem-Yun Kim et al., which discovered that 11 (or 22%) of the newborns who had been asphyxiated showed symptoms of cardiac failure [15].

In the current study, 66.7% of patients required mechanical breathing, whereas 33.3% of patients required nasal oxygen assistance. Comparatively, 42.3% of patients in Joseph et al study's required ventilation, while 22 ill neonates (46%) received ventilatory support in Awada et al study's [16,13].

In line with the El Mesiry et al. study, which discovered that haemoglobin (p0.001) and platelet (p=0.002) levels were noticeably lower in the cases group than in the controls group [5], laboratory evaluation revealed a significant decrease in haemoglobin (HG) (p = 0.002) and platelet (plt) (p = 0.003) levels in the current studied cases compared to controls. Additionally, the cases group's hematocrit (HCT) levels were considerably lower than those of the controls group (p=0.002). Between the examined patients and controls in the present study, there was no statistically significant difference in the total leucocytic count (TLC) (p=0.527). Similar results were obtained by El Mesiry et al., who discovered that the TLC between patients and controls was the same (p=0.099). The results of Awany et al., on the other hand, revealed a significant rise in the TLC in ill newborns (p0.0001) [5, 12].

In contrast to El Mesiry et al investigation's which found no difference in sodium (p=0.585) or potassium (p=968) levels between cases and controls [5], the current study showed a significant drop in sodium (p=0.008) and potassium (p=0.033) levels between cases and controls.

In the current study, the levels of the enzymes creatinine (Cr) and aspartate transaminase (AST) were significantly higher in cases than in controls (p0.000). Contrarily, El Mesiry et al investigation's discovered no difference in Cr levels between patients and controls (p=0.858) [5].

In the present study, there was a highly significant increase in C reactive protein (CRP) in cases compared to controls (p=0.000). Which is consistent with the findings of Awany et al study, which found an increase in CRP levels in 85% of cases (p<0.0001) [12].

Although the cases in the current study had somewhat higher HCO3 levels than the controls, there was no discernible statistical difference between the two groups (p=0.593). However, El Mesiry et al investigation 's discovered that HCO3 was significantly lower in the cases group compared to the controls group (p=0.001) [5]. Between patients and controls, there was no discernible statistical change in PCO2 levels in the current study (p=0.947). El Mesiry et al study's which reported no statistically significant difference in PCO2 between patients and controls (p=0.131), and this are in agreement. [5].

The median cTnT level in the current study ranged from 21.5 pg/ml in the controls to 116.3 pg/ml in the patients. Similar to this, Clark et al study's revealed that 113 patients had median cTnT levels of 25 pg/ml in 113 healthy newborns versus 159 pg/ml in 49 sick neonates. In addition, Matter et al study 's revealed cTnT levels with a median of 30 pg/ml in controls compared to 170 pg/ml in cases, while Ismail et al study 's reported cTnT levels(pg/ml) with a mean of 88.8 $\pm$4.92 in controls compared to 184.87± 4.31 in cases[11,6,3].

In the current investigation, the cTnT level was negative in 10% of cases, equivocal or positive (high) in 30% and 60% of cases, respectively. p=0.0001), and it was significantly higher in cases than in controls (p=0.0001), which is in line with Clark et al findings 's (p=0.0001), Awada et al(p=0.0001), Awany et al (p=0.0001) and El Mesiry et al which also found higher cTnT levels in cases than controls (p=0.001)[11, 13, 12, 5].

In the current study, initial diagnoses for NICU admission were pneumonia, jaundice, sepsis, and surgical causes in proportions of 30%, 16.7%, 43.3%, and 10%, respectively. Similarly, 41 cases of neonatal sepsis, 16 cases of hypoxic-ischemic encephalopathy (HIE), 15 cases of transient tachypnea of the newborn (TTN), 14 cases of neonatal jaundice, 4 cases of newborn hemorrhagic illness, and 10 cases of post-surgical causes were found in the study by El Mesiry et al. [5].

Dopamine, dobutrex, adrenaline, and primacor were all employed as forms of inotropic support in the current study, whereas just six ill infants (12.5 percent) in Awada et al study 's and twenty two in Clark et al study 's received inotropic treatment for hypotension [13,11].

LVIDD was considerably reduced in cases compared to controls (P = 0.000) for the M mode echocardiogram evaluation of the study group, but LVPWD did not change in cases compared to controls (P = 0.171). This is consistent with the findings of the study by Tomark et al., which indicated that LVIDD was considerably reduced in patients compared to controls (P = 0.001) and LVPWD did not differ between cases and controls (P = 0.715). However, this study by Ismail et al., which discovered a significant difference in the parameters LVEDD (P = 0.021) & LVPWT (P = 0.024) in the instances [17, 3], is in opposition to the current study.

In our study, the LV's EF% and FS%, which measure its systolic function, were significantly lower in cases than in controls. This is consistent with the Alazharani study, which indicated that sick neonates had significantly lower EF (p0.001) and fraction shortening (p0.001) than nonseptic neonates. [18] On the other hand, the results of Awany et al., who found no significant differences in EF% between cases and controls (p = 0.09), and those of Tomerk et al., who found no significant differences in EF (0.032) and FS (p = 0.183) between the full-term and preterm neonates with sepsis and their control groups [12,17], were the exact opposite. Additionally, cases significantly outperformed controls in terms of PA acceleration time (ms) (p 0.001).

Diastolic PA pressure (mmHg) in the current investigation was substantially higher in cases (21.4 mmHg) than in controls (3.7 mmHg) (p 0.001). This is consistent with research by Yousef et al., which found that diastolic PA pressure was 25.2 mmHg in cases versus 22.4 mmHg in controls (p = 0.005). Additionally, Fahmey et al investigation's which found that the cases group had significantly greater pulmonary artery pressure than the controls group (p0.001), validated our findings [19, 14].

According to tissue Doppler analysis, there was no discernible difference between the examined cases' tricuspid E velocity and that of the controls (p = 0.278). In contrast, Awany et al study .'s [12] found that patients had lower tricuspid E velocity than controls.

Regarding tricuspid A velocity, there was no discernible difference between patients and controls in the current investigation (p = 0292). This contradicts the findings of Yousef et al investigation 's which found that the tricuspid A velocity was significantly higher in cases compared to controls (p = 0.001) [19].

The right ventricle's (RV) diastolic function is represented by the tricuspid E/A ratio, which was found to be considerably poorer in the examined neonates with hemodynamic instability than in the control group in the current study (p = 0.023). This is consistent with the findings of the study by Yousef et al., which showed that the tricuspid E/A ratio was significantly lower in cases than in controls (p 0.001) [19].

In the current investigation, cases had a tie index RV that was considerably greater than controls (p = 0.002). The results of Awany et al., who discovered that patients had a longer tie index of the RV (p = 0.0001) [12], are consistent with this.

Regarding mitral E velocity, there was no discernible difference between patients and controls in the current study (p = 0.147). This goes against the findings of Yousef et al study's which showed that the mitral E velocity was considerably lower in the septic newborn group compared to the control group (p 0.001) [19].

In the current investigation, the cases group's mitral A velocity significantly increased (0.009), which is consistent with Yousef et al study 's the cases group's mitral A velocity significantly increased (p0.001).[19]

In the current study, the mitral E/A ratio significantly dropped in situations where (0.041), this is consistent with Yousef et al study's which found that the mitral E/A ratio in the septic newborns was considerably higher than in the control group (p 0.001) [19].

The tie index of the LV significantly increased in the current study's instances (p = 0.000). The investigations by Tomerk et al. (p = 0.001) and Awany et al., which discovered a significant rise in the LV tie index, are consistent with this. The Alazharani study, in contrast, found no discernible difference in the LV tie index between patients and controls (p = 0.001) [17,12,18].

Age, consanguinity, weight, and birth order were all demographic factors in the current study that had no impact on troponin levels. This is consistent with Clark et al study's results, which showed no relationship between cTnT concentration and gestational age or birth weight. Similar to the previous study, Clark et al investigation 's revealed no connection between the groups' cTnT levels and weight or gestational age. In contrast, Awada et al research 's discovered strong correlations between cTnT and birth weight, length, and age [20, 1, 13].

There was no distinction in cTnT levels between males and females in the current investigation. This is in contrast to a thorough study published by Baum et al. who evaluated cTnT concentrations in the cord blood of 869 healthy newborns and discovered statistically significant differences in cTnT concentrations between males and females [21]. Baum et al findings contradict those of Clark et al., who found no correlation between cTnT and gender.[1] Larger sample sizes may provide an explanation for this.

Maternal risk factors in the current study had no impact on cTnT levels. This is in agreement with Awada et al., who reported no differences between newborns with normal and high cTnT levels in terms of mother age, parity, smoking, preeclampsia, chorioamnionitis, prenatal antibiotics, antenatal corticoids, and tocolysis.[13]

In the current study, there was no correlation between clinical signs of heart failure, capillary perfusion and heart rate and cTnT levels. This may need a larger sample size.

Pneumonia, sepsis, direct hyperbilirubinemia, and surgical factors all significantly impacted cTnT in the current study. This is consistent with the findings of Clark et al study's which indicated that newborns with congenital pulmonary hypertension and a diaphragmatic hernia had significantly elevated cTnT levels. While there was a modest increase in the number of newborns with gastroschisis, exomphalos, and tracheoesophageal fistula [1].

The mean blood pressure and cTnT had a strong negative connection in the current investigation. Similarly, Joseph et al. showed a statistically significant difference in mean cTnT values in neonates with shock versus those without shock [16]. Clark et al. also identified a substantial

increase in cTnT levels in hypotensive sick newborns on inotropic support compared to normotensive sick neonates.[11]

In the present study, cTnT levels was not correlated with the use of mechanical ventilation (MV). This comes against Clark et al.'s study, which found increased troponin levels in ventilated neonates [11].

No statistically significant correlation between cTnT and laboratory data was found in the current investigation (HG, HCT, PLT, TLC, Sodium, CREAT, AST, ALT, PT, PTT, INR, PCO2, HCO3, & CRP). The Tarkowaska and Wanda study, which found no correlation between cTnT and laboratory tests like TLC, platelet count, blood urea, ALT, AST, CRP, and serum sodium in both groups, supports this. In contrast, Ismail et al investigation 's discovered a statistically significant connection between serum cTnT and each of TLC, PLT, ALT, AST, CRP, serum sodium, and phosphorus. Our findings, which suggested a connection between Troponin T and TLC, PLT, ALT, AST, CRP, and serum sodium, were also in conflict with Awany et al study [22, 3, 12]

A statistically significant association between cTnT and potassium was found in the current investigation. This is consistent with research by Awany et al. and Ismail et al., which found a statistically significant relationship between serum cTnT and serum potassium that showed a substantial association. The work by Daly et al. suggests that the impact of hypo- or hyperkalemia on heart function may help to explain this. Contrarily, the Tarkowaska and Wanda investigation found no conclusive relationship between troponin T and serum potassium. [12, 3, 23, 22].

A substantial association between cTnT and PH was found in the current investigation.

As in previous research by Trevisanuto et al. and Awany et al., who found a correlation between cTnT levels and the use of vasopressors and MV [24,12], the current study found that cTnT levels were greater in infants who needed inotropic support (a statistically significant connection).

According to the results of the current investigation, cTnT has a negative relationship with LVIDD. LVIDD decreased in cases in our study, which was opposed by Tomark et al., El Azharani et al. and Matter et al. studies; that had a significant increase in LVIDD in cases than controls; also, Ismail et al study which found a positive correlation between cTnT and LVIDD [17, 18,6,3].

A strong positive connection between cTnT level and LVPWD was found in the current study. This is consistent with the findings of the study by Ismail et al., which discovered a positive connection between cTnT and LVPWD [3].

In the present study, cTnT levels were inversely linked with EF% and FS%. This contradicts the findings of research by Ismail et al. and Matter et al. Ismail et al. reported a positive correlation between cTnT and EF while, Matter et al showed no link between cTnT and FS [3, 6].

In the current investigation, cTnT had a negative correlation with diastolic PA pressure but no correlation with PA acceleration time.

The mitral E/A ratio and cTnT were shown to be adversely linked in the current study. This is consistent with the research by Awany et al., which discovered a negative correlation between troponin and the E/A ratio [12].

The tie index of the LV and cTnT had a favourable association in the current investigation. This supports the findings of Awany et al study which discovered an association between troponin and the LV's tie index [12].

In this investigation, there was no discernible relationship between cTnT and either the mitral E velocity or the mitral A velocity.

The tricuspid E/A ratio and the cTnT were not associated in the current investigation. The work by Awany et al., which discovered a favourable correlation between cTnT and tricuspid E/A ratio, contradicts this.[12]

The right ventricle's tie index and cTnT were not associated in the current investigation. In contrast, Awany et al study [12] discovered a positive correlation between cTnT and the right ventricle's tie index.

Abbreviations:

CTnT: Cardiac Troponin T, CBC: Complete blood count, CRP: C-reactive protein, TDI: Tissue Doppler imaging, EF: Ejection fraction, FS: Fraction shortening, LV: Left ventricle, NICU: Neonatal intensive care unit, HR: Heart rate, CRT: Capillary refill time, LVESD: LV end-systolic diameter,

LVIDD: LV internal diameter end-diastole, EDV: End diastolic volume, ESV: End systolic volume, HCT: Hematocrit, HG: Hemoglobin, PT: Prothrombin, PTT: Partial thromboplastin, PLT: Platelets, NA: Sodium, CR: Creatinine, INR: International normalized ratio.

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