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Research Article

Cardiac Troponin T-Based Assessment of Myocardial Dysfunction in Critically Ill Children Admitted to a Pediatric Intensive Care Unit: A Prospective Observational Study

Dr. V Sharmila[1], Dr. N. Ragavendran[2*], Dr. K. Jayaprakash[3]
[1,2*,3]Assistant Professor Department Of Pediatrics Government Dindigul Medical College Dindigul, Tamil Nadu, India.
Corresponding Author: Dr. N. Ragavendran
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ABSTRACT
Background: Myocardial injury in critically ill children is often clinically silent at admission but may become important when shock, hypoxia, sepsis, respiratory failure, or multiorgan dysfunction increases myocardial oxygen demand while reducing effective oxygen delivery.
Aim: To assess myocardial status in critically ill children admitted to a pediatric intensive care unit using cardiac troponin T, bedside echocardiography, electrocardiography, and chest radiography, and to examine its association with selected clinical outcomes.
Materials and Methods: This hospital-based prospective observational study was conducted in the Pediatric Intensive Care Unit, Institute of Social Pediatrics, Government Stanley Medical College and Hospital, Chennai, from 1 April 2017 to 30 September 2017. Children aged 1 month to 12 years admitted to PICU without pre-existing congenital or acquired cardiac disease were included. Cardiac troponin T was measured at admission by electrochemiluminescence immunoassay; values >0.1 ng/mL were considered positive. Bedside two-dimensional/M-mode echocardiography, 12-lead ECG, and chest X-ray were recorded. Associations were tested using chi-square/Fisher exact tests and independent-samples t-test, with $p<0.05$ considered statistically significant.
Results: Among 112 children, 33 (29.5%) were troponin T positive. Troponin positivity was not significantly associated with sex (p=0.428) or age group (p=0.422). System-wise distribution among 87 children with a predominant major-system diagnosis showed significant variation in troponin positivity (p=0.036), with the highest proportions in cardiovascular disease (3/3, 100.0%), inborn errors of metabolism (2/3, 66.7%), and sepsis (3/7, 42.9%). Echocardiographic abnormality was present in 13/112 children and all 13 were troponin positive (Fisher exact p=0.0005). ECG abnormality (20/33 vs 23/79, p=0.002) and chest X-ray abnormality (20/33 vs 28/79, p=0.014) were also significantly more frequent in troponin-positive children. Mortality was higher in the troponin-positive group (26/33, 78.8%) than in the troponin-negative group (12/79, 15.2%; p=0.0005). Troponin positivity was also associated with need for mechanical ventilation (31/33, 93.9%; p=0.0005) and inotropic support (32/33, 97.0%; p=0.0005). Mean recorded stay was longer in the troponin-positive group, but the difference was not statistically significant (9.82±11.19 vs 6.75±7.68 days; p=0.156).
Conclusion: Admission troponin T positivity identified a clinically vulnerable subgroup of critically ill children with higher echocardiographic, ECG, and radiographic abnormalities, greater need for organ support, and markedly higher mortality. A bedside cardiac assessment pathway combining troponin T with echocardiography and ECG may help earlier recognition of myocardial involvement in PICU, although serial biomarker and imaging studies with adjustment for illness severity are needed before routine prognostic use can be generalized.

Keywords: Cardiac Troponin T, Critically Ill Children, Myocardial Dysfunction, Pediatric Intensive Care Unit, Echocardiography, Mortality.

INTRODUCTION

Cardiovascular dysfunction in a child admitted to the pediatric intensive care unit rarely presents as an isolated cardiac problem. It is more often hidden inside sepsis, hypoxia, shock, acute respiratory failure, severe neurological illness, renal dysfunction, metabolic disturbance, poisoning, or postoperative physiological stress. This makes myocardial injury easy to miss at the bedside. The child may look primarily septic or ventilatory, yet the heart is already working against fever, acidosis, catecholamine

exposure, fluid shifts, and altered coronary perfusion. In critically ill patients, biochemical evidence of myocardial injury has long been recognized as a frequent and clinically meaningful event, even when acute coronary syndrome is not the diagnosis [1].

Cardiac troponins are structural regulatory proteins of the contractile apparatus and become detectable in blood when cardiomyocyte integrity is lost. Their diagnostic value lies in the tissue specificity of cardiac isoforms and their relatively prolonged circulation after injury [2]. In pediatrics, interpretation is more delicate than in adults because age, underlying illness, renal handling, congenital or acquired cardiac disease, and assay characteristics may influence observed values. Even so, troponin measurement remains one of the most accessible biochemical windows into myocardial injury in children [3].

Echocardiography provides the complementary anatomic and functional view. While troponin signals cell injury, bedside echocardiography can show reduced systolic performance, ventricular dilatation, valvular regurgitation, pulmonary hypertension, or pericardial effusion. Standardized M-mode and pediatric echocardiographic measurement principles have therefore remained important in converting a bedside impression into reproducible cardiac information [4,5]. ECG and chest radiography add a different kind of support: rhythm disturbance, ischemic ST-T change, low-voltage complexes, cardiomegaly, and lung pathology may all modify the way myocardial dysfunction is understood in the PICU.

The issue is especially relevant for tertiary-care pediatric units in India, where late presentation, infection-related shock, severe pneumonia, encephalitis, undernutrition, and referral delay may converge in the same child. Pediatric sepsis guidelines emphasize early recognition of cardiovascular compromise, timely vasoactive support, and continuous reassessment of perfusion and organ dysfunction [6]. Yet, the practical question remains: can a simple admission troponin T value, read together with bedside echocardiography and ECG, identify children at greater risk of adverse outcomes? Previous pediatric studies have linked troponin elevation with disease severity, ventricular dysfunction, respiratory illness, septic shock, and mortality, but local PICU-level data remain limited [7,8].

The present study was therefore designed to assess myocardial status in critically ill children admitted to a pediatric intensive care unit, using quantitative cardiac troponin T, bedside echocardiography, ECG, and chest X-ray. The analysis also examined whether troponin T positivity was associated with mortality, mechanical ventilation, inotropic support, shock correction, and duration of stay.

MATERIALS AND METHODS

Study Design and Setting: A hospital-based prospective observational study was conducted in the Pediatric Intensive Care Unit, Institute of Social Pediatrics, Government Stanley Medical College and Hospital, Chennai.

Study Period: The study was carried out from 1 April 2017 to 30 September 2017.

Study Population: Critically ill children admitted to the PICU during the study period were screened for eligibility.

Sample Size: A total of 112 children were included.

Inclusion Criteria: Children aged 1 month to 12 years admitted to the PICU, with no pre-existing congenital or acquired cardiac illness, were included.

Exclusion Criteria: Neonates, children with pre-existing congenital or acquired cardiac disease, children after post-cardiac-arrest resuscitation, and children whose parents did not provide consent were excluded.

Ethical Considerations: Institutional Ethics Committee clearance was obtained. Written informed consent was obtained from parents or caregivers before enrolment. The study did not add any therapeutic risk to the child because blood sampling was performed during routine venous access and bedside investigations were non-invasive.

Clinical Assessment and Investigations: A detailed history and clinical examination were performed for all eligible children. Cardiac troponin T was measured at admission using an electrochemiluminescence immunoassay method with a Roche kit. A value >0.1 ng/mL was considered positive. A 12-lead ECG was recorded on the day of admission. Bedside two-dimensional/M-mode echocardiography was performed by a pediatric cardiologist using a GE 2012 bedside echocardiography machine. Echocardiographic assessment included left and right ventricular hypokinesia, ejection fraction, pulmonary hypertension, right atrial/right ventricular dilatation, and other structural or functional abnormalities. Normal ejection fraction was considered 55-

75%. Bedside chest X-ray findings were recorded where available.

Outcome Measures: The primary assessment focused on myocardial injury identified by troponin T positivity and its association with echocardiographic cardiac dysfunction. Secondary outcome measures included need for mechanical ventilation, need for inotropic support, number of inotropes, shock correction status, duration of stay, and mortality.

Statistical Analysis: Data were analysed using IBM SPSS Statistics version 23.0. Categorical variables were summarized as frequencies and percentages. Continuous variables were summarized as mean and standard deviation. Chi-square test was used for categorical associations, and Fisher exact test was used where expected cell counts were small. Independent-samples t-test was used for comparison of mean duration of stay between troponin groups. A p-value <0.05 was considered statistically significant.

RESULTS

Of 112 critically ill children included in the study, 33 (29.5%) were troponin T positive and 79 (70.5%) were troponin T negative (Figure 1).

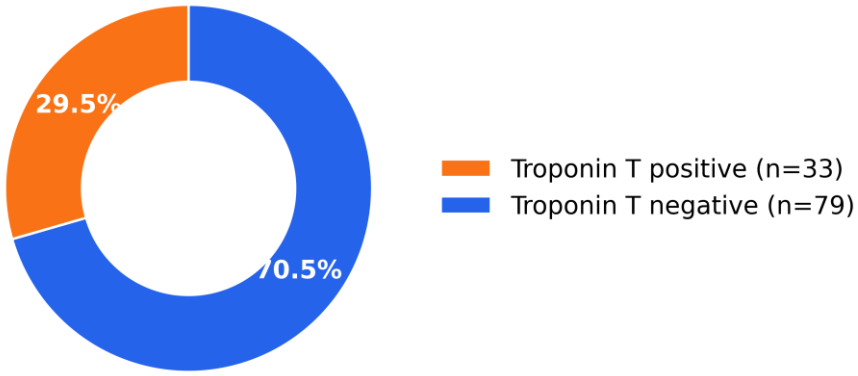

Figure 1. Distribution of Admission Cardiac Troponin T Status among Critically Ill Children

Donut chart shows the proportion and count of children with troponin T positivity using the study cut-off of >0.1 ng/mL.

There was no statistically significant association between troponin T status and sex. Males constituted 58/112 (51.8%) of the study population and females 54/112 (48.2%). Among troponin-positive children, 19/33 (57.6%) were male and 14/33 (42.4%) were female (p=0.428). Age distribution also did not differ significantly between troponin groups (p=0.422). The largest age category in the full cohort was >1-5 years (33/112, 29.5%), followed by up to 6 months (29/112, 25.9%) (Table 1 and Figure 2).

Table 1. Demographic Distribution According to Admission Troponin T Status (N=112).

Variable	Troponin T negative (n=79)	Troponin T positive (n=33)	Total (n=112)	p-value
Sex				0.428
Female	40 (50.6)	14 (42.4)	54 (48.2)	
Male	39 (49.4)	19 (57.6)	58 (51.8)	
Age group				0.422
Up to 6 months	18 (22.8)	11 (33.3)	29 (25.9)	
>6 months-1 year	11 (13.9)	4 (12.1)	15 (13.4)	
>1-5 years	22 (27.8)	11 (33.3)	33 (29.5)	
6-10 years	13 (16.5)	5 (15.2)	18 (16.1)	
>10 years	15 (19.0)	2 (6.1)	17 (15.2)	

Values are shown as n (% within troponin group), except total column where percentages are within the full cohort. Chi-square test was used.

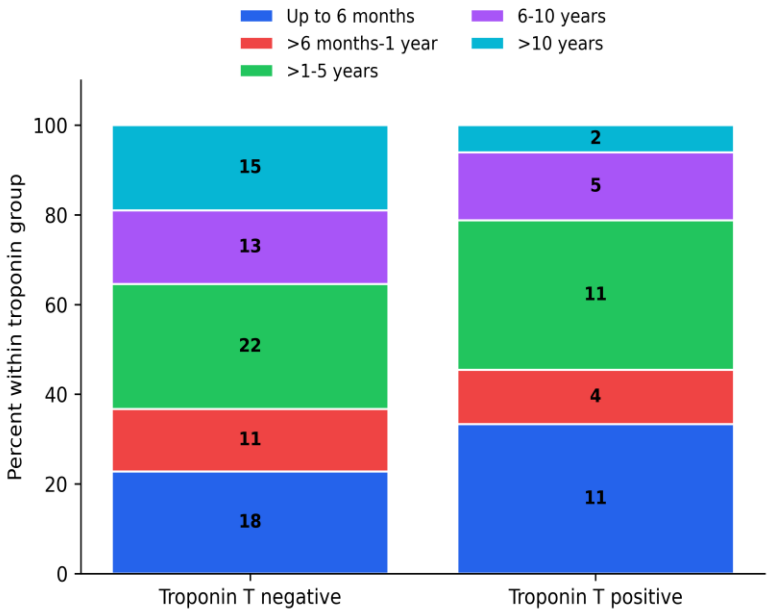

Figure 2. Age Distribution within Troponin T-Negative and Troponin T-Positive Groups

Stacked columns show percentage composition within each troponin group; segment labels display child counts.

A predominant major-system diagnosis was analysed in 87 children. Troponin T positivity varied significantly across diagnostic systems (Pearson chi-square=11.897, p=0.036). Cardiovascular presentations had the highest observed positivity (3/3, 100.0%), followed by inborn errors of metabolism (2/3, 66.7%), sepsis (3/7, 42.9%), respiratory system illness (12/36, 33.3%), gastrointestinal illness (2/9, 22.2%), and central nervous system illness (5/29, 17.2%) (Table 2 and Figure 5).

Table 2. System-Wise Distribution of Troponin T Positivity among Children with A Predominant Major-System Diagnosis (N=87).

Predominant system	Troponin T negative n (%)	Troponin T positive n (%)	Total	Positive (%)
CNS	24 (82.8)	5 (17.2)	29	17.2
CVS	0 (0.0)	3 (100.0)	3	100.0
GIT	7 (77.8)	2 (22.2)	9	22.2
IEM	1 (33.3)	2 (66.7)	3	66.7
Respiratory system	24 (66.7)	12 (33.3)	36	33.3
Sepsis	4 (57.1)	3 (42.9)	7	42.9

Percentages in troponin columns are row percentages. Pearson chi-square=11.897, df=5, p=0.036.

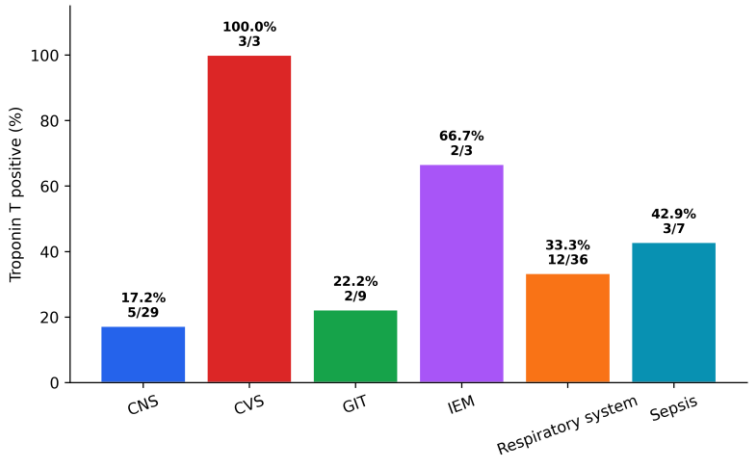

Figure 3. Troponin T Positivity across Predominant Major-System Diagnostic Categories

Bar labels display percentage positivity and corresponding positive/total counts. CNS: central nervous system; CVS: cardiovascular system; GIT: gastrointestinal system; IEM: inborn errors of metabolism.

Cardiac evaluation findings showed a clear association with troponin T positivity. Echocardiographic abnormality was found in 13/112 (11.6%) children, and all 13 were in the troponin-positive group (Fisher exact p=0.0005). ECG abnormality was recorded in 43/112 (38.4%) children and was more frequent among troponin-positive children than troponin-negative children (20/33, 60.6% vs 23/79, 29.1%; p=0.002). Chest X-ray abnormality was present in 48/112 (42.9%) and again occurred more often in the troponin-positive group (20/33, 60.6% vs 28/79, 35.4%; p=0.014) (Table 3 and Figure 4).

Table 3. Cardiac Investigation Abnormalities According to Admission Troponin T Status (N=112).

Investigation abnormality	Troponin T negative (n=79)	Troponin T positive (n=33)	Total (n=112)	Chi-square	z	p-value
Echocardiogram abnormal	0 (0.0)	13 (39.4)	13 (11.6)	35.208		0.0005
ECG abnormal	23 (29.1)	20 (60.6)	43 (38.4)	9.760		0.002
Chest X-ray abnormal	28 (35.4)	20 (60.6)	48 (42.9)	6.018		0.014

Values are n (% within troponin group). Fisher exact p-value is reported for echocardiographic abnormality due to a zero cell.

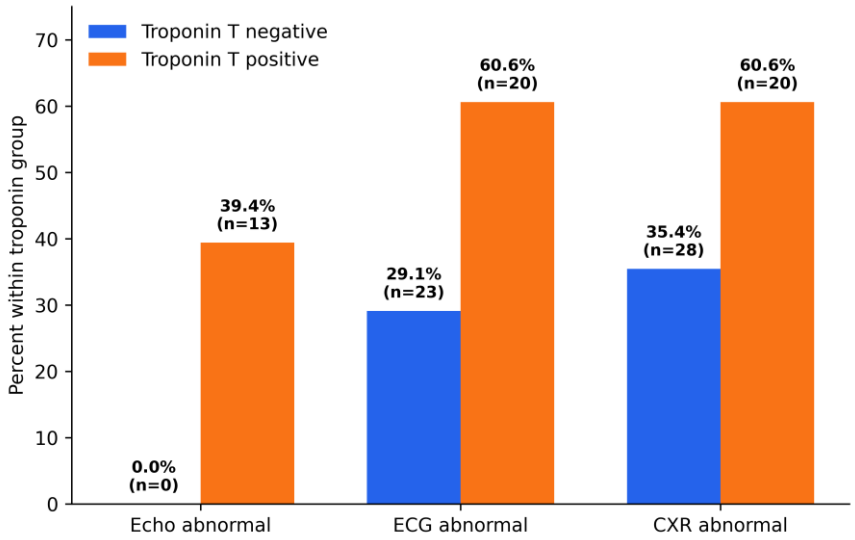

Figure 4. Cardiac Investigation Abnormalities in Troponin T-Negative and Troponin T-Positive Children.

Grouped bars show percentage abnormality within each troponin group; labels display percentage and count.

Clinical outcomes were substantially worse in troponin T-positive children. Overall mortality was 38/112 (33.9%). Mortality was 26/33 (78.8%) in the troponin-positive group compared with 12/79 (15.2%) in the troponin-negative group (Pearson chi-square=41.998, p=0.0005). Need for mechanical ventilation and inotropic support was also significantly higher among troponin-positive children. Ventilation was required in 31/33 (93.9%) troponin-positive children compared with 39/79 (49.4%) troponin-negative children (p=0.0005). Inotropic support was required in 32/33 (97.0%) troponin-positive children and 45/79 (57.0%) troponin-negative children (p=0.0005) (Table 4 and Figure 5).

Table 4. Clinical Outcomes and Organ-Support Requirements According to Admission Troponin T Status

Outcome / clinical variable	Troponin T negative (n=79)	Troponin T positive (n=33)	Test statistic	p-value
Non-survivor	12 (15.2)	26 (78.8)	41.998	0.0005

Mechanical ventilation required	39 (49.4)	31 (93.9)	19.731	0.0005
Inotropic support required	45 (57.0)	32 (97.0)	17.342	0.0005
No inotrope	34 (43.0)	2 (6.1)	29.698	0.0005
1 inotrope	34 (43.0)	12 (36.4)		
2 inotropes	11 (13.9)	15 (45.5)		
>2 inotropes	0 (0.0)	4 (12.1)		
Shock corrected: yes (subset n=82)	32/49 (65.3)	20/33 (60.6)	0.188	0.665
Recorded stay, days, mean ± SD	6.75 ± 7.68	9.82 ± 11.19	t=-1.441	0.156

Values are n (% within troponin group), except where stated. Shock-correction analysis included 82 children with available shock-correction status. Independent-samples t-test was used for duration of stay.

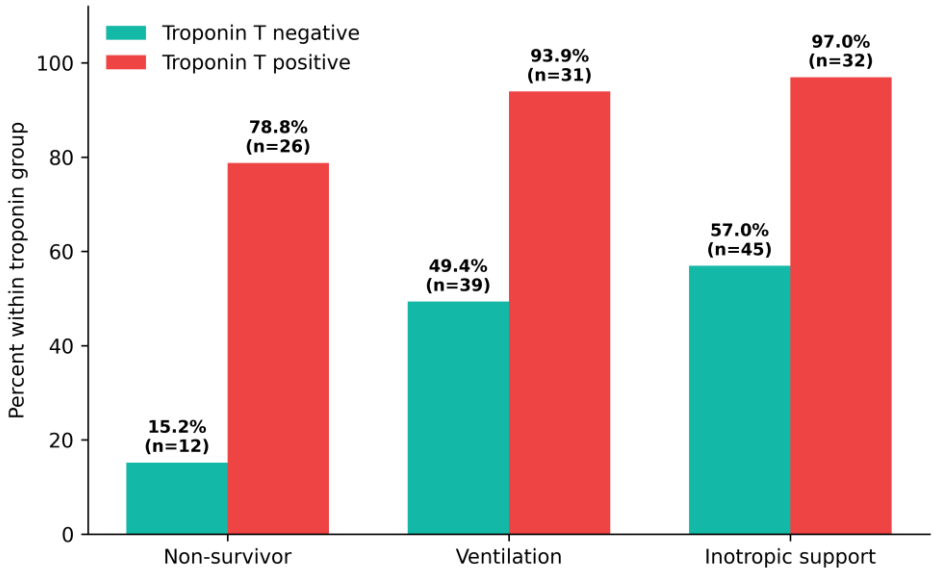

Figure 5. Mortality and Organ-Support Requirements According to Troponin T Status

Grouped bars show percentage within each troponin group; labels display percentage and count.

Although the mean recorded duration of stay was longer in troponin-positive children (9.82±11.19 days) than in troponin-negative children (6.75±7.68 days), this difference did not reach statistical significance (p=0.156). Shock correction, among 82 children with recorded shock status, also did not differ significantly between troponin groups (p=0.665).

DISCUSSION

The central observation in this PICU cohort was simple but clinically important: nearly one-third of critically ill children had biochemical evidence of myocardial injury at admission. Troponin T positivity was not explained by age or sex distribution. It was instead clustered with illness phenotype, abnormal cardiac investigations, and adverse clinical course. This pattern suggests that admission troponin T may act less like a standalone diagnosis and more like an early warning marker, pointing to children whose myocardium is being stressed or injured during critical illness.

The finding that all children with abnormal echocardiography were troponin positive deserves attention. Echocardiography in this study was used to document functional or structural cardiac abnormalities such as reduced ejection fraction, ventricular hypokinesia, chamber dilatation, pulmonary hypertension, valvular regurgitation, and pericardial effusion. Troponin T, on the other hand, indicated myocardial cell injury. Their convergence is clinically useful. In a busy PICU, where shock may be attributed entirely to infection, dehydration, respiratory failure, or neurological catastrophe, a positive troponin

result may prompt a more deliberate cardiac examination rather than assuming the myocardium is only a passive bystander.

These results are broadly consistent with earlier pediatric observations. Clark et al. showed that cardiac troponin T levels were elevated among ventilated infants in PICU and correlated with severity of illness, reinforcing the idea that myocardial injury can occur in critically ill children without congenital heart disease [7]. Fenton et al. reported that troponin I was increased in more than half of children with septic shock and was associated with echocardiographic systolic dysfunction and disease severity [8]. Hassan et al. similarly found higher troponin T concentrations in critically ill children than controls, with positive correlations with ventilation duration and illness severity and a negative correlation with left ventricular fractional shortening [9]. The present findings sit within that same clinical frame, though the current analysis used troponin T positivity as a categorical admission marker rather than serial quantitative biomarker trends.

Sepsis and severe systemic inflammation offer one plausible mechanism. In septic shock, myocardial injury may arise from oxygen supply-demand mismatch, inflammatory cytokine-mediated myocardial depression, microcirculatory dysfunction, mitochondrial injury, catecholamine exposure, and altered calcium handling. Thiru et al. demonstrated that troponin I elevation in meningococcal septicemia correlated with disease severity, echocardiographic myocardial depression, and peak inotrope requirement [10]. Severe respiratory infections can create a similar load through hypoxia, pulmonary hypertension, increased right ventricular work, and ventilator-associated cardiopulmonary interactions. Eisenhut et al. described troponin T elevation and myocardial involvement in children with severe respiratory syncytial virus lung disease [11]. In the present cohort, respiratory illness contributed the largest number of system-classified cases and accounted for 12 troponin-positive children among 36 respiratory cases.

The association between troponin positivity and mortality was strong. More than three-fourths of troponin-positive children were non-survivors, compared with a much smaller proportion of troponin-negative children. Adult critical-care studies have repeatedly shown similar directionality. Spies et al. reported serum cardiac troponin T as a prognostic marker in early sepsis [12], Ammann et al. found troponin to be a mortality risk factor in critically ill patients without acute coronary syndromes and linked it with lower left ventricular function and higher inflammatory mediators [13], and Quenot et al. showed an association between increased cardiac troponin I and hospital mortality in critically ill patients [15]. Pediatric generalization must still be careful, but the biological signal is coherent: troponin elevation in critical illness often marks a heart under serious stress in a body already moving toward organ failure.

The inotropic and ventilatory findings add another layer. Troponin-positive children required mechanical ventilation and inotropic support far more often than troponin-negative children. This does not prove that myocardial injury caused the need for support. It may equally mean that the sickest children, already requiring ventilation and vasoactive drugs, were also the children in whom myocardial injury became biochemically detectable. Still, this is clinically meaningful. A child who is troponin positive at admission may need closer hemodynamic surveillance, repeat perfusion assessment, earlier bedside echocardiography, and cautious fluid-vasoactive titration rather than protocolized management alone.

The system-wise distribution also feels believable in real PICU practice. Cardiovascular presentations showed universal troponin positivity, which is expected, but the numerically small group prevents broad inference. Inborn errors of metabolism and sepsis also showed high proportions. Respiratory disease had a lower percentage than cardiovascular illness, but because it was the largest diagnostic group, it contributed a substantial number of positive cases. Central nervous system illness showed lower overall positivity, though acute encephalitis, status epilepticus, intracranial hemorrhage, autonomic surge, hypoxia, aspiration, and shock can all place secondary stress on the myocardium. Briassoulis et al. showed that pediatric purpura fulminans can involve myocardial injury with ECG ischemic changes and low ejection fraction, reminding us that cardiac involvement in severe infection may be both biochemical and functional [14].

ECG and chest X-ray abnormalities were significantly associated with troponin positivity, but they should not be interpreted as interchangeable with troponin or echocardiography. ECG abnormalities such as tachycardia, ST-T changes, low voltage, U

waves, and conduction changes may reflect myocardial injury, electrolyte disturbance, hypoxia, fever, catecholamine effect, or the underlying systemic illness. Chest X-ray abnormalities are even less specific because pneumonia, collapse, hyperinflation, pulmonary edema, cardiomegaly, and effusions can overlap. Their value in this study is therefore supportive, not confirmatory. Troponin and echocardiography together provide a tighter cardiac signal.

The practical implication for Indian pediatric intensive care units is not that every sick child should be labelled as having myocarditis when troponin is positive. That would be unsafe. A more balanced reading is that admission troponin T, when interpreted alongside hemodynamics, ECG, chest X-ray, and bedside echocardiography, can help identify a higher-risk group. In resource-constrained units, this may influence the timing of cardiology consultation, repeat bedside echo, vasoactive strategy, ventilation planning, and counselling of caregivers. It may also help avoid delayed recognition of myocardial dysfunction in children initially admitted for respiratory, infectious, neurological, or metabolic disease.

Several limitations temper the findings. There was no control group, so background troponin distribution in stable hospitalized or healthy children from the same setting could not be compared. Troponin T, echocardiography, and ECG were assessed at admission without serial follow-up, which limits understanding of whether myocardial injury was transient, progressive, or treatment responsive. Other biomarkers such as CK-MB, LDH, BNP/NT-proBNP, and troponin I were not compared. Illness severity scores were not available in the Results, and no multivariable model was performed; therefore, troponin positivity cannot be claimed as an independent predictor of mortality. Finally, the study was from a single tertiary-care PICU, and some subgroup counts were small, particularly cardiovascular disease and inborn errors of metabolism. These limitations do not weaken the observed associations, but they do restrict causal interpretation and generalizability.

CONCLUSION

Admission cardiac troponin T positivity was observed in 29.5% of critically ill children admitted to PICU and was significantly associated with echocardiographic abnormality, ECG abnormality, chest X-ray abnormality, mortality, mechanical ventilation, inotropic support, and requirement for multiple inotropes. Age and sex were not significantly associated with troponin status. The findings support the use of troponin T as an early biochemical marker that can strengthen bedside suspicion of myocardial injury when read together with echocardiography and ECG. Future studies should include serial troponin measurements, serial echocardiography, illness severity scoring, and multivariable analysis to clarify whether troponin T independently predicts outcome or mainly reflects the burden of critical illness.

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Conflict of Interest
Not declared.

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