

ISSN Print: 1813-176x
ISSN Online: 1994-4624

International Journal of Molecular Medicine and Advance Sciences
https://ijmmas.com/

Research Article

Evaluation Of Association Of Troponin I With The Outcome In Children With Shock Aged 1 Month To 18 Years At Tertiary Care Hospital.

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Received: 18-09-2026
Revised: 02-10-2026
Accepted: 18-10-2026
Published: 09-11-2026

DOI: 10.61336/ijmmas.22-04-26

Abstract: Objectives: To evaluate the association of serum Troponin I levels with clinical outcomes in children aged 1 month to 18 years presenting with shock, and to assess its relationship with mortality. ***Background***: Shock in children is a life-threatening condition associated with high morbidity and mortality. Early identification of myocardial involvement is crucial for prognostication. Cardiac Troponin I, a sensitive biomarker of myocardial injury, may serve as a useful predictor of outcome in pediatric shock. ***Methods***: This hospital-based prospective observational study was conducted in the PICU of a tertiary care center over 18 months. A total of 85 children with shock were enrolled using consecutive sampling. Clinical evaluation and laboratory investigations, including Troponin I estimation using chemiluminescent microparticle immunoassay, were performed at admission. Patients were followed until discharge or death. Data were analyzed using SPSS, and statistical significance was set at $p \leq 0.05$. ***Results***: Out of 85 patients, 79 (92.9%) survived and 6 (7.1%) died. Elevated Troponin I levels were significantly associated with mortality, with mean levels of 1910.00 ± 1212.44 in non-survivors compared to 512.99 ± 662.31 in survivors ($p < 0.001$). A strong positive correlation ($r = 0.910$, $p < 0.001$) was observed between Troponin I levels. All patients with normal Troponin I survived, whereas mortality occurred only in those with elevated levels. ***Conclusion***: Serum Troponin I is a valuable prognostic biomarker in pediatric shock. Elevated levels are strongly associated with increased mortality. Routine assessment of Troponin I may aid in early risk stratification and guide clinical management in critically ill children.

Keywords: Troponin I, Pediatric Shock, Mortality, Prognosis, PICU.

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INTRODUCTION

Shock in children remains one of the most critical and life-threatening conditions encountered in emergency and intensive care settings. It is characterized by inadequate tissue perfusion, resulting in cellular hypoxia and organ dysfunction. Shock may arise from various etiologies, including septic, cardiogenic, hypovolemic, and distributive causes [1]. Despite advances in pediatric critical care, it continues to be associated with considerable morbidity and mortality, particularly among children aged one month to 18 years. Early recognition, timely intervention, and accurate risk stratification are essential for improving outcomes. In this context, identifying reliable biomarkers that predict disease severity, guide treatment, and assess prognosis is an important aspect of pediatric intensive care. Cardiac biomarkers have gained attention because of their potential to identify myocardial involvement and systemic stress in children experiencing shock [2].

Troponin I (TnI), a regulatory protein involved in myocardial contraction, is a sensitive and specific marker of myocardial injury. In adults, elevated troponin levels are well-established indicators of myocardial infarction, ischemia, and cardiac stress, with considerable prognostic significance in critical illness. However, its role as a prognostic marker in children remains less clearly defined because of age-related variations in baseline levels, differences in the underlying causes of shock, and limited pediatric-specific evidence. Emerging research suggests that elevated troponin I in critically ill children may indicate myocardial dysfunction, correlate with disease severity, and predict adverse outcomes, multiple organ dysfunction, and mortality. Myocardial injury may result from ischemia, inflammatory mediators, or catecholamine excess and may remain clinically unrecognized. Consequently, relying exclusively on clinical signs and hemodynamic monitoring may delay the identification of subclinical cardiac dysfunction [3].

The relationship between elevated troponin I and adverse outcomes in pediatric shock involves several interconnected pathophysiological mechanisms. Systemic hypoperfusion and metabolic stress can produce myocardial ischemia and cellular injury, irrespective of the underlying etiology. In septic shock, inflammatory cytokines, nitric oxide, and endotoxins may contribute to myocardial depression, impaired contractility, and cardiomyocyte injury, resulting in troponin release into the circulation. In cardiogenic shock, reduced cardiac output further aggravates tissue hypoxia and myocardial damage. Therefore, the magnitude of troponin elevation may provide an indirect indication of myocardial stress and overall disease severity. Pediatric studies have also demonstrated associations between elevated troponin I and echocardiographic evidence of impaired ventricular function, supporting its potential role as an accessible biomarker for risk stratification in children with shock [4].

Despite its potential, the prognostic utility of troponin I in pediatric shock requires further investigation. Important questions remain regarding the optimal timing of measurement, age-specific reference ranges, and the influence of underlying comorbidities. Troponin release may vary among infants, younger children, and adolescents because of differences in physiological characteristics and compensatory mechanisms. Furthermore, transient elevations may reflect reversible myocardial stress and do not necessarily indicate an adverse outcome. Troponin I should therefore be interpreted alongside clinical findings and other biomarkers, such as B-type natriuretic peptide and lactate. Prospective investigations incorporating serial troponin measurements and clinical scoring systems, including the Pediatric Risk of Mortality (PRISM) and Pediatric Logistic Organ Dysfunction (PELOD) scores, have provided preliminary evidence of its prognostic relevance. However, larger multicenter studies are necessary to validate these findings and establish standardized pediatric-specific thresholds [5].

Troponin I assessment may also have important implications for therapeutic decision-making. Early identification of children at increased risk of adverse outcomes could support closer monitoring, timely escalation of hemodynamic support, and appropriate consideration of inotropic therapy. Information regarding myocardial injury may also assist clinicians in evaluating disease progression and counseling families about the anticipated clinical course. Furthermore, understanding the association between troponin elevation and outcomes may help identify patients who could benefit from interventions aimed at minimizing myocardial injury. These strategies may include optimizing fluid resuscitation, avoiding unnecessary catecholamine exposure, and evaluating potential cardioprotective approaches, while ensuring that treatment decisions remain guided by the overall clinical condition [6].

Beyond its prognostic applications, troponin I is a valuable research tool for investigating myocardial dysfunction in pediatric shock. Correlating troponin concentrations with echocardiographic findings, hemodynamic parameters, and clinical outcomes can improve understanding of the progression of myocardial injury and its response to treatment. Serial measurements may help characterize the timing and persistence of cardiac involvement and clarify the relationship between myocardial injury and systemic illness. Research in this area may also contribute to the development of integrated biomarker panels combining indicators of inflammation, tissue hypoxia, and myocardial damage. Such approaches could improve predictive accuracy and support more individualized management of critically ill children [7].

Several knowledge gaps continue to limit the routine use of troponin I for prognostication in pediatric shock. Its predictive performance may differ across septic, cardiogenic, hypovolemic, and distributive shock, while underlying comorbidities may further influence troponin concentrations and clinical outcomes. Differences in assay methodology, sample collection timing, and pediatric reference ranges also complicate the establishment of universally applicable thresholds. Well-designed prospective studies are therefore required to determine the independent prognostic value of troponin I, assess its incremental benefit over established clinical indicators, and develop standardized protocols for its interpretation in different pediatric populations [8].

Shock in children aged one month to 18 years remains a major clinical challenge associated with substantial morbidity and mortality. Troponin I offers considerable potential as a biomarker for assessing myocardial injury, evaluating disease severity, and predicting adverse outcomes. Its elevation may reflect myocardial stress, ischemia, and inflammation associated with critical illness. Although available evidence suggests a relationship between increased troponin I levels and poor outcomes, further research is necessary to standardize measurement protocols, establish age-specific reference ranges, and validate its prognostic utility across different shock etiologies. Integrating troponin I assessment with clinical examination, hemodynamic monitoring, echocardiography, and established severity scores may improve the identification of high-risk children and support more informed treatment decisions. Ultimately, its appropriate use may strengthen risk stratification and contribute to evidence-based care in pediatric critical illness [9].

MATERIALS AND METHODS

A hospital-based prospective observational study was conducted in the Pediatric Intensive Care Unit (PICU) of Hassan institute of Medical Sciences (HIMS), which is a tertiary-care teaching hospital to observe, record, and analyze clinical and biochemical

parameters in real time among children diagnosed with various types of shock, without intervening in their management protocols. The study was carried out over a period of two years. The study was conducted after obtaining ethical clearance from the Institutional Ethics Committee of HIMS. All eligible patients who met the inclusion criteria and none of the exclusion criteria were enrolled consecutively. A consecutive sampling method was employed to select the participants. The calculated sample size was 83.66, which was rounded off to 85 patients for convenience.

Inclusion Criteria: Children aged 1 month to 18 years who were admitted to the PICU with a diagnosis of shock of any etiology, only those patients whose parents or legal guardians provided written informed consent were included in the study.

Exclusion Criteria: Neonates younger than 1 month and individuals older than 18 years, Children with pre-existing congenital or acquired cardiac illnesses, children with shock after cardiac arrest and revival.

Study Groups

Outcome-based groups: **Survivors and Non-survivors at the time of discharge**.
This classification facilitated comparative analysis of troponin I trends and clinical parameters across varying clinical courses and outcomes. Grouping was purely analytical and did not influence patient management.

STUDY PROCEDURE:
Each patient admitted with suspected shock underwent a detailed clinical evaluation at the time of admission. Shock was diagnosed on the basis of physical findings such as tachycardia, weak or thready pulse, cold extremities and hypotension for age. Once diagnosed, the attending physician assessed eligibility against inclusion and exclusion criteria.
After obtaining written consent, demographic and baseline clinical information was recorded in a pre-designed proforma. A comprehensive systemic examination was conducted, and relevant laboratory investigations were ordered.
Blood Sampling and Laboratory Protocol
Sample will be collected at admission.
Venous samples were processed in the hospital's central biochemistry laboratory.

Estimation of Serum Troponin I:
Analysis was performed using the Abbott Architect system, which utilizes the Chemiluminescent Microparticle Immunoassay (CMIA) technique. This method offers high sensitivity and specificity for detecting minimal myocardial injury. Additional investigations such as complete blood count and CRP were conducted following standard institutional protocols to rule out infectious etiologies. All patients were followed up daily until discharge or death. Clinical progress, requirement of mechanical ventilation, inotrope use, and organ dysfunction were meticulously recorded. Data from all patients were compiled systematically for subsequent statistical analysis.

RESULTS

Table 1: Clinical Outcome of Children with Shock.

Outcome	Frequency	Percentage
Died	6	7.10%
Survived	79	92.90%
Total	**85**	**100.00%**

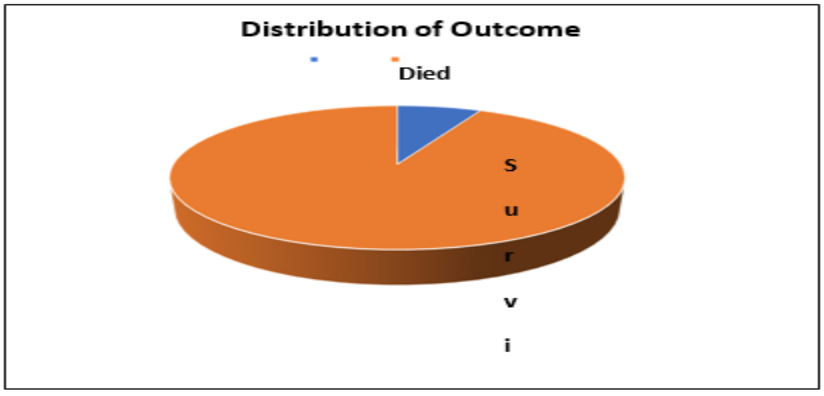


Figure 1 shows

Clinical Outcome

The survival rate was high at 92.9% (n=79), with a mortality rate of 7.1% (n=6). This indicates favourable overall outcomes in the study population despite the severity of shock. The relatively low mortality suggests effective management and timely intervention. However, the presence of mortality highlights the critical nature of shock in children. These findings emphasize the importance of early diagnosis and aggressive treatment to improve survival rates in critically ill children.

Table 2: Comparison of Mean Troponin I Levels with Outcome.

	Outcome	Mean	Std. Deviation	p-value
Troponin I	Died	1910.00	1212.442	<0.001*
	Survived	512.99	662.31	

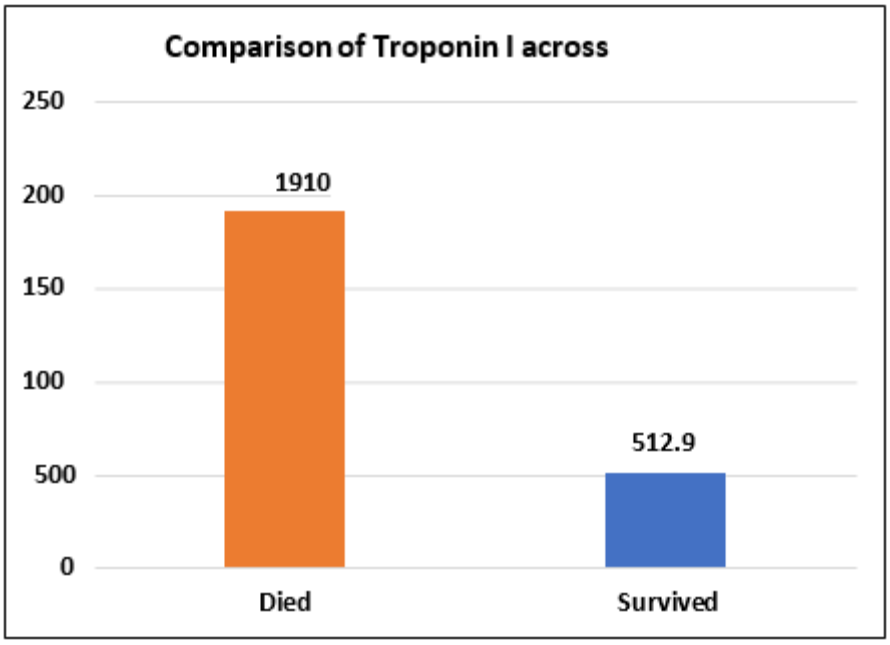


Figure 2 shows

Troponin I vs Outcome

Mean troponin I levels were significantly higher in patients who died (1910.00) compared to survivors (512.99), with a statistically significant p-value (<0.001). This indicates a strong association between elevated troponin levels and mortality. Higher troponin levels likely reflect severe myocardial injury and worse prognosis. These findings suggest that troponin I can serve as an important prognostic biomarker in shock in children, aiding in risk stratification and early identification of high-risk patients.

Table 3: Association Between Troponin I categories and Clinical Outcome (Mortality) Among Study Participants

Troponin categories	Outcome		Total	p-value
	Died	Survived		
<1500	2	74	**76**	<0.001*
>1500	4	5	**9**	
Total	**6**	**79**	**85**	

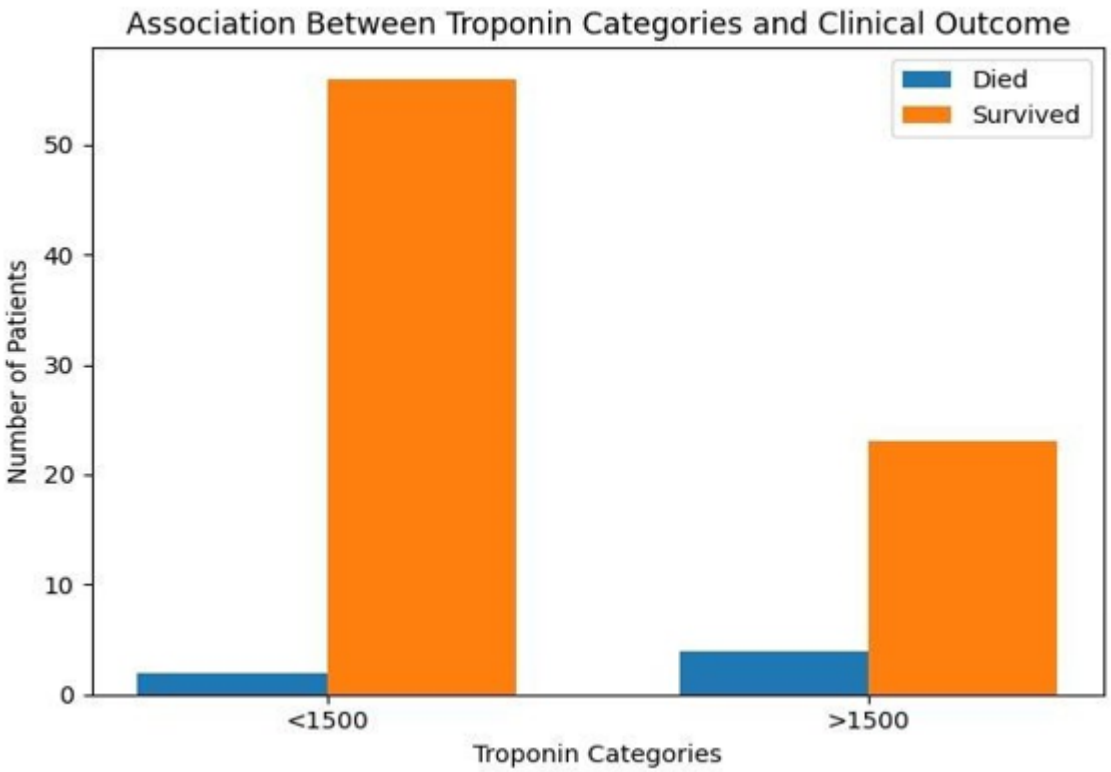


Figure 3 shows

Troponin Categories vs Outcome

Most patients (89.4%) had troponin levels below 1500, with a high survival rate (93.6%) and few deaths (33.3% of total deaths). In contrast, patients with troponin levels above 1500 (10.6%) had a much higher proportion of deaths (66.7%) and lower survival (6.4%). Overall, 6 patients died and 79 survived out of 85 participants. The statistically significant p-value (<0.001) indicates a strong association between elevated troponin levels and increased mortality, suggesting that higher troponin is an important predictor of poor clinical outcome in this cohort.

DISCUSSION

Aim and Significance of the Study

The present study aimed to evaluate the clinical profile, laboratory parameters, and outcomes of children presenting with shock, with particular emphasis on the prognostic significance of serum troponin I. The study assessed the association of troponin I levels with mortality, alongside demographic characteristics, clinical presentation, physiological parameters, and requirements for advanced life support.

The findings highlight the potential role of troponin I as a biomarker of myocardial injury and an indicator of adverse outcomes in pediatric shock. The significantly higher troponin I levels among non-survivors suggest that troponin I may assist in early risk stratification and clinical decision-making. The study also provides insights into the clinical severity and presenting characteristics of children with shock at a tertiary care hospital.

Symptom Distribution

The most frequently reported symptoms were cough/cold (71.8%, n=61) and fever (63.5%, n=54). Convulsions occurred in 29.4% (n=25), decreased appetite in 25.9% (n=22), and vomiting or loose stools in 8.2% (n=7). This symptom profile suggests that respiratory and infectious presentations were common among children with shock, although the underlying etiology cannot be confirmed from symptoms alone. The inflammatory findings reported in comparative studies are broadly relevant to the clinical presentation observed in this cohort [10,12,14]. The coexistence of fever, respiratory symptoms, and neurological manifestations emphasizes the importance of identifying and managing underlying infections and associated organ dysfunction. Further etiological classification would be required to establish the specific causes of shock in this population.

Peripheral Perfusion Status

Cold peripheries were observed in 97.6% (n=83) of children, while 2.4% (n=2) had warm peripheries. This finding indicates a predominance of cold peripheral perfusion at presentation, consistent with peripheral vasoconstriction and impaired tissue

perfusion in children with shock. Although the comparative studies did not provide directly comparable proportions of cold and warm shock, Dezfulian described myocardial dysfunction and hemodynamic instability as important contributors to adverse outcomes in critically ill patients [11]. Trembach et al. similarly emphasized the association between cardiovascular dysfunction, persistent hemodynamic instability, and mortality in pediatric septic shock [16]. The predominance of cold peripheries provides relevant clinical context for the assessment of myocardial injury using troponin I, although a direct association between peripheral perfusion status and troponin levels would require additional analysis.

Arterial Blood Gas Findings

Metabolic acidosis was present in 24.7% (n=21) of children, while 75.3% (n=64) had normal arterial blood gas (ABG) findings. Among children with metabolic acidosis, 6 died and 15 survived. In contrast, all 64 children with normal ABG findings survived. These observations suggest that metabolic acidosis identified a subgroup with greater mortality in the present cohort. Metabolic acidosis in shock may reflect impaired tissue perfusion, inadequate oxygen delivery, and increased anaerobic metabolism. Mickiewicz et al. described alterations in energy metabolism in pediatric septic shock [10]. Dezfulian and Li et al. also discussed the relevance of metabolic disturbances and myocardial dysfunction in critical illness [11,12]. The present findings support the clinical importance of ABG assessment in evaluating physiological derangement. Nevertheless, the association between acidosis and mortality should be interpreted cautiously because only six deaths occurred in the study.

Clinical and Laboratory Parameters at Admission

The admission profile indicated substantial physiological stress. Mean heart rate was 165.28 ± 22.82 beats/min, mean respiratory rate was 44.54 ± 14.58/min, and mean SpO_2 was 87.74 ± 10.10%. Mean temperature was 36.91 ± 0.61°C, while mean C-reactive protein (CRP) was 51.22 ± 49.15. The average fluid bolus administered was 19.88 ± 4.95 mL/kg. These findings indicate the presence of tachycardia, respiratory compromise, hypoxemia, and systemic inflammation in the study population. Trembach et al. reported that adverse hemodynamic parameters and greater vasoactive support requirements were associated with mortality in pediatric septic shock [16]. The admission findings in the present study provide a clinical context for evaluating myocardial injury and its relationship with outcomes. However, the independent prognostic contribution of these parameters relative to troponin I would require multivariable analysis.

Troponin I and Clinical Outcome

The most important finding was the significant difference in mean troponin I levels between children who died and those who survived. Non-survivors had a mean troponin I level of 1910.00 ± 1212.442, compared with 512.99 ± 662.31 among survivors.

CONCLUSION

The present study demonstrated a significant association between elevated troponin I levels and mortality among children with shock. The mean troponin I level was substantially higher in children who died (1910.00 ± 1212.442) than in survivors (512.99 ± 662.310), with a statistically significant difference ($p < 0.001$). Categorical analysis further showed that mortality was higher among children with troponin I levels above 1500, with 4 of 9 children (44.4%) dying, compared with 2 of 76 children (2.6%) whose levels were below 1500. These findings suggest that troponin I may be a useful prognostic biomarker for mortality risk stratification in children presenting with shock. However, given the small number of deaths (n = 6), the findings should be interpreted cautiously. Further studies with larger sample sizes and appropriate multivariable analysis are needed to establish the independent predictive value of troponin I for mortality.

Acknowledgement:
We sincerely thank the faculty of the institute for helping us in each aspect of the study.
Declarations Funding: None
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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