

Diagnostic Utility of Troponin I and Troponin T in Pediatric Myopericarditis: Comparison with Cardiac MRI Findings

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Abstract

Background

Cardiac troponins and cardiac magnetic resonance imaging (CMR) are increasingly used as non-invasive diagnostic tools in the diagnosis of myopericarditis. This study aimed to compare cardiac troponin I (cTnI) and troponin T (cTnT) levels in children diagnosed with myopericarditis and to evaluate their association with cardiac MRI findings.

Methods

This prospective study included 75 pediatric patients diagnosed with myopericarditis. High-sensitivity cTnI and cTnT levels were measured simultaneously within the first three days of admission. Clinical findings, electrocardiography, echocardiography, microbiological investigations, and CMR findings were evaluated.

Results

The mean age of the patients was 12.77 ± 4.6 years (74.6% male). A significant positive correlation was found between cTnI and cTnT levels. Cardiac Troponin T demonstrated 97.2% sensitivity compared to cTnI but lower specificity. MRI was performed in 29 patients and 82.7% had late gadolinium enhancement. No statistically significant correlation was found between troponin levels and CMR findings.

Conclusion

Both cTnI and cTnT are highly sensitive biomarkers for detecting myocardial injury in pediatric myopericarditis. Troponin T showed almost the same sensitivity to troponin I but lower specificity. CMR remains an important confirmatory imaging modality, particularly in severe cases. Combining cardiac biomarkers with cardiac imaging may improve diagnostic accuracy in pediatric myopericarditis.

Introduction

Myocarditis/myopericarditis are inflammatory diseases of the heart muscle with a wide range of clinical presentations in children, ranging from mild chest pain to severe acute heart failure or life-threatening arrhythmias.¹ Viral infections remain the most common etiologic factor. However, the initial symptoms are often non-specific such as fever, fatigue, or respiratory and gastrointestinal complaints, frequently leading to delay in diagnosis.¹

Diagnosing myocarditis in children remains challenging. Definitive diagnosis by endomyocardial biopsy is limited by its invasive nature and low sensitivity. Therefore, clinical practice relies on a combination of clinical findings, cardiac biomarkers, and imaging modalities.¹ Cardiac magnetic resonance (CMR) imaging has become a key non-invasive diagnostic tool by detecting myocardial edema and fibrosis through late gadolinium enhancement (LGE), however a negative CMR does not exclude myocarditis.¹

Cardiac troponins, particularly troponin I (cTnI) and troponin T (cTnT), are widely used biomarkers of myocardial injury and are typically elevated in acute myocarditis and myopericarditis due to cardiomyocyte damage.¹ The cardiac troponin complex in myocytes consists of three subunits, troponin T (cTnT), troponin I (cTnI), and troponin C which regulate cardiac muscle contraction.^{2,3} Of these, cTnI and cTnT are the isoforms measured in clinical practice. While both are highly sensitive, their release kinetics differ, and elevation may occur even in reversible myocardial injury. Studies suggest that cTnI levels rise and peak more rapidly than cTnT after acute myocardial damage, often reaching higher peak concentrations and returning to normal sooner, whereas cTnT may increase more gradually as a larger fraction remains bound within the myocyte during early injury.^{2,3} Notably, troponin release may occur even with reversible cell injury or apoptosis, so an elevated troponin does not always indicate extensive irreversible necrosis. Therefore, cardiac troponin measurement is a convenient and sensitive screening test for myocardial injury.

Despite its value, troponin alone is not specific enough to distinguish myocarditis from other causes of myocardial damage. Elevated troponin may be seen in conditions such as sepsis, acute pericarditis, or acute coronary syndromes, so clinical context is essential. Moreover, a normal troponin level does not completely rule out myocarditis, since some biopsy-confirmed cases have had negative troponin tests. Thus, cardiac troponins are an important clue but insufficient for a definitive diagnosis on their own.¹ In practice, troponin I is often regarded as a gold standard cardiac injury marker, but both subtypes are used.

In this article, based on the findings of a prospective study conducted at the Department of Pediatric Cardiology, Marmara University, the aim is to compare troponin I and T levels in children diagnosed with myopericarditis or myopericarditis, and to evaluate the value of these biomarkers in predicting cardiac MR findings.

Methods

Our study is a prospective study that included 75 pediatric patients who were diagnosed with myocarditis/myopericarditis after presenting to the Marmara University School of Medicine, Department of Pediatric Cardiology with complaints of chest pain and/or elevated troponin levels between the years 2021–2025. The study was approved by the Marmara University Ethics Committee (approval no.: 09.2023.787). All procedures involving human participants were performed in accordance with the relevant institutional and national guidelines and regulations and with the ethical principles of the Declaration of Helsinki. Written informed consent to participate in the study was obtained from all

participants and/or their parents or legal guardians, as appropriate, prior to enrollment. The diagnosis of all patients included in the study was established by using patient history, physical examination, diagnostic tests (laboratory, electrocardiography (ECG), and echocardiography (ECHO) findings) evaluated by two different pediatric cardiology specialists before MRI or histologic evaluation if needed as a previously decided methodologic acceptance.

The demographic information and presenting complaints of the patients were recorded. Within the first three days following the presentation, blood samples of 5 ml were collected from the patients simultaneously to determine both serum Troponin I and serum Troponin T levels. The collected blood samples were centrifuged at 4000 rpm for 10 minutes by the biochemistry specialist responsible for the study in the institution's biochemistry laboratory, and the serum samples were separated. The separated sera were stored in an Eppendorf tube at -80°C until analysis. High-sensitivity cardiac troponin T (hs-cTnT) levels were measured using the Roche Cobas e602 autoanalyzer with the Troponin T hs kit (Ref No: 05092744 190, Roche Diagnostics GmbH, Mannheim, Germany) based on the electrochemiluminescence immunoassay (ECLIA) method. The limit of detection (LoD) and limit of quantification (LoQ) for hs-cTnT were 5.48 ng/L (10%CV) and 13 ng/L, respectively, and values ≥ 14 ng/L were accepted as positive. High-sensitivity cardiac troponin I (hs-cTnI) measurements were performed using the Beckman Coulter UniCel Dxl-600 immunoassay analyzer with the Access hsTnI kit (Ref No: B52699, Beckman Coulter Inc., Brea, CA, USA). The assay is a two-step chemiluminescent immunoassay employing paramagnetic particles. The assay had a limit of detection (LoD) of 2.3 ng/L and a 10% coefficient of variation (CV) limit of quantification (LoQ) of 5.6 ng/L and hs-cTnI values ≥ 11 ng/L were considered positive.

Patients diagnosed with myocarditis/myopericarditis were hospitalized in the clinic, where routine monitoring and treatment were carried out. During the hospitalization, daily clinical evaluations were performed; vital signs, cardiac biomarker levels (troponin levels), and other biochemical and inflammatory parameters were regularly monitored. For patients showing signs of infection, broad nasopharyngeal swabs and polymerase chain reaction (PCR) tests for microbiological agents were conducted for pathogen isolation. ECG checks were conducted daily and at discharge during the hospitalization. Echocardiographic evaluations were performed at admission and then at 7 days, 1 month, and 3 months post-discharge. Routine follow-ups with ECG, ECHO, and laboratory tests were conducted during discharge control of the patients. For patients showing significant impairment in cardiac functions via echocardiography (Philips Affiniti CVx, Philips Healthcare, Netherlands®), progressing through a more severe clinical course, and/or displaying ECG abnormalities, early-stage contrast-enhanced cardiac magnetic resonance imaging (MRI) was conducted within the first 3 months of diagnosis to scan for myocardial damage signs compatible with myopericarditis in terms of late gadolinium enhancement (LGE). Cardiac MRI imaging was performed using protocols appropriate to the children's age and with sedation if necessary.

The cardiac magnetic resonance imaging was performed on a 1.5 T MR scanner (Philips Ingenia, Philips Healthcare, Best, Netherlands®). The software was IntelliSpacePortal image and information

management software version 10.1. Axial TSE images with black blood technique was used to obtain information on the morphology of the right ventricle and left ventricle. For ventricular function, conventional steady state free precession (bSSFP) cine images were obtained in ventricular short and long axis plane. Myocardial edema was defined as increased signal intensity on T2-weighted imaging. A segmented phase sensitive inversion recovery (PSIR) spoiled gradient echo (GRE) sequence was used for contrast dynamics. For evaluation of delayed enhancement, late contrast-enhanced imaging at 10 minutes after intravenous administration was obtained in short and long axis.

Statistical analyses were performed using MedCalc Statistical Software (Version 23.5.2, MedCalc Software Ltd.). Descriptive statistics were expressed as mean $\pm$ standard deviation (SD), median (Q1-Q3), and percentages, as appropriate. Since the continuous variables were non-parametrically distributed, comparisons between two independent groups were performed using the Mann–Whitney U test. Correlations between troponin I and troponin T levels were assessed using Spearman's rank correlation analysis. Receiver operating characteristic (ROC) curve analysis was performed, and sensitivity and specificity values were calculated to evaluate diagnostic performance. Categorical variables were compared using Pearson's chi-square test or Fisher's exact test, as appropriate. A p value of < 0.05 was considered statistically significant.

Results

The average age of the 75 patients diagnosed with myopericarditis included in the study was 12.77 ± 4.6 years, and 74.6% were male. The most common presenting complaint was chest pain, with 84% (63 patients) presenting with chest pain. Three patients without chest pain at presentation were admitted with respiratory distress or respiratory pathology, four patients with abdominal pain and gastrointestinal symptoms, three with general malaise, one with intoxication, and one with widespread muscle pain. Fever was relatively rare at the time of presentation; only 12% of patients reported a fever above 38°C . None of the patients had any previously known illnesses, except for two patients with already diagnosed heart disease, one being followed-up for past rheumatic heart disease, and the other for mitral valve prolapse. Only one patient had a history of substance abuse. Six patients (8%) had a history of previous myopericarditis. In only 13 out of 75 patients (17%), a specific microbiologic agent related to myopericarditis etiology was identified; while viral etiology was mostly considered, microbiologic confirmation was not possible in the remaining patients. (Table 1) The average hospital stay was 5.9 ± 5.4 days, indicating that most patients became clinically stable within approximately a week. Ibuprofen was administered as treatment to all patients, and heart failure treatment was given to those with left ventricular systolic dysfunction. Treatment targeted to the detected bacterial-viral agents was administered if needed. IVIG treatment was given to 9 clinically severe patients.

Table 1
Microbiologic agents isolated from patients with myopericarditis

Isolated microbiological agents from all patients		Microbiological agents isolated from patients with LGE uptake on Cardiac MRI	
Agents	Number	Agents	number
Rhinovirus	2	Rhinovirus	2
Rhinovirus + Enterovirus	2	Rhinovirus + Enterovirus	1
Sars-Cov-2 (COVID-19)	1	Sars-Cov-2 (COVID-19)	1
Parainfluenza-4	1	Influenza B	1
Influenza A	1		
Influenza B	3		
RSV	1		
CMV	1		
Bocavirus	2		
Mycoplasma pneumoniae	1		

Cardiac troponin levels in the first blood samples taken during admission were elevated in all patients. The mean cTnT level was 219 ± 299 ng/L, and the cTnI level was 1480 ± 2612 ng/L. When simultaneously measured troponin T and I values were compared, a statistically significant positive correlation was found between them (Spearman's $\rho = 0.891$, $p < 0.001$) (Fig. 1). A very strong positive correlation was observed between hs-cTnI and hs-cTnT levels indicating a close concordance between the two biomarkers in detecting myocardial injury. When troponin I measurement was accepted as the “gold standard” in confirming the diagnosis of myopericarditis, and Troponin T levels were evaluated with ROC analysis; sensitivity of cTnT was 97.2%; however, specificity of cTnT was low. Receiver operating characteristic (ROC) analysis was performed using hs-cTnI as the reference standard. An hs-cTnT cutoff value of 20.15 ng/L provided a sensitivity of 90.3% and a specificity of 33.3%. Increasing the cutoff to 51.23 ng/L resulted in a sensitivity of 68.1% and a specificity of 66.7%. A cutoff value of 113 ng/L yielded a specificity of 100%, with a corresponding sensitivity of 41.7%.

Electrocardiography showed pathologic STsegment and T wave changes in 26 of the patients at presentation or during follow-up. Ventricular extra beats were observed on the ECG of one patient. Echocardiographic examination revealed pathological findings in 36% of the patients (26 patients). Patients with ECG abnormalities had significantly higher hs-cTnT levels than patients without ECG abnormalities (Mann–Whitney U test, $p = 0.007$). Patients with ECG abnormalities had higher hs-cTnI levels than those without ECG abnormalities; however, there was no statistical significance (Mann–Whitney U test, $p = 0.088$). Left ventricular ejection fraction has decreased in 5; and in two of them dilated cardiomyopathy (DCMP) has developed. during follow-up. In the patients with LV dysfunction,

mean cTnT level was 166.3 ± 242.9 ng/L, and the cTnI level was 573 ± 1036.5 ng/L. There is no statistical significance differences in terms of hs-cTnI or hs-cTnT levels between patients with and without left ventricular dysfunction.

Coronary imaging was performed in some patients for differential diagnosis; coronary CT angiography was applied to 18 patients, and coronary artery pathology was found in none of them. Emergency conventional coronary angiography was performed in two patients, all were normal. The performed imaging modalities and their results are presented in Table 2.

Table 2
The results of cardiac imaging in patients with myopericarditis

N:75	Rate of patients with pathologic findings	Pathologic findings	Number of patients with pathologic findings
	Number (percentage)		
Echocardiography (N:75)	26 (36%)	Aort valve insufficiency	3
		Mitral valve insufficiency	19
		LV systolic dysfunction	5
		LV hypertrophy	5
		Pericardial effusion	2
Cardiac MRI (N:29)	25 (86,2%)	Positive Late gadolinium enhacment	23
		Pericardial effusion	6

During the study, cardiac MR imaging (CMR) was administered to 29 patients (38%). This selected subgroup consisted of cases with significant dysfunction detected via echocardiography or with a more severe or prolonged clinical course. Pathologic late gadolinium enhancement (LGE increase) indicating myopericarditis was observed in 24 out of 29 patients (82%) who underwent CMR. (Fig. 2) When cardiac biomarker levels of the 23 patients with detected LGE were examined, the mean cTnT level was found to be 350.7 ± 377 ng/L and the mean cTnI level was 2946.9 ± 3723.2 ng/L. When only patients with documented LGE-positive and LGE-negative CMR findings were compared, hs-cTnT and hs-cTnI levels were numerically higher in the LGE-positive group; however, the differences did not reach statistical significance (hs-cTnT: $p = 0.054$; hs-cTnI: $p = 0.102$; Mann–Whitney U test). In contrast, patients with LGE-positive findings had significantly higher hs-cTnT and hs-cTnI levels than the remaining study cohort ($p = 0.001$ and $p = 0.001$, respectively; Mann–Whitney U test).

Discussion

In our study, we found that both cardiac troponin I (cTnI) and troponin T (cTnT) were elevated in all patients at presentation. This confirms the high sensitivity of troponin as a marker of myocardial injury in children with myocarditis. The levels of cTnI and cTnT rose in parallel and were strongly correlated ($p < 0.001$), indicating that both biomarkers reflect the same underlying cardiomyocyte damage. Notably, cTnT was as sensitive as cTnI; since cTnT was also positive in 97.2% of the cases with an elevated cTnI. However, cTnT was less specific because Troponin I was negative in some patients with elevated Troponin T levels. Lower cutoff values, such as 20.15 ng/L, achieved high sensitivity (90.3%) but poor specificity (33.3%), making them suitable for screening purposes. In contrast, a higher cutoff value of 113 ng/L provided perfect specificity (100%) at the expense of substantially lower sensitivity (41.7%). This suggests that troponin T is a sensitive indicator in the presence of myocarditis, but not specific. Thus, while cTnT is an excellent screening tool for myocardial injury, an isolated troponin T elevation should be interpreted with caution in the absence of other findings, since it may not always indicate definite myocarditis. Lower hs-cTnT thresholds may be useful when the primary goal is to avoid missing cases of myocardial injury, whereas higher thresholds may be more appropriate when diagnostic certainty is required. Therefore, the optimal cutoff value should be selected according to the intended clinical use. This interpretation is consistent with the American Heart Association statement that troponin I and troponin T may be elevated in acute pediatric myocarditis, but these biomarkers do not differentiate myocarditis from other causes of myocardial injury.¹ The lower specificity of cTnT may, in part, be explained by differences in the release kinetics of cTnI and cTnT, as the two biomarkers are known to reach peak concentrations at different time points following myocardial injury.

The significant relationship between hs-cTnT levels and ECG abnormalities observed in our study may reflect the different temporal release patterns of cardiac troponins. Troponin I generally increases and normalizes earlier during the acute phase of myocardial injury, whereas troponin T tends to remain elevated for a longer period. Consequently, ECG abnormalities may not yet be apparent at the time of peak troponin I elevation, while significant hs-cTnT elevations may coincide with a stage at which electrocardiographic changes have become clinically evident.

In patients with late gadolinium enhancement on cardiac MRI, both troponin T and troponin I demonstrated 100% sensitivity and 100% specificity. Our findings align with prior pediatric studies showing that troponin testing is highly sensitive in suspected myocarditis. Eisenberg *et al.* reported that using a low cutoff of 0.01 ng/mL for cTnT in children yielded a sensitivity of 100% (negative predictive value 100%) for diagnosing myocarditis, with a specificity of about 85%.⁴ Similarly, a recent study by Degen *et al.* has showed that in all children with acute myocarditis had significantly elevated high-sensitivity cTnT levels at presentation; the authors determined an optimal hs-cTnT threshold of 90 ng/L, which provided 100% sensitivity and 95% specificity for distinguishing myocarditis from non-cardiac causes of troponin elevation in their cohort.⁵ These data underscore that a normal troponin makes myocarditis unlikely in a symptomatic child, whereas an elevated troponin should prompt careful cardiac evaluation. However, troponin elevation by itself is not diagnostic of myocarditis, since troponin can also

rise in other conditions (sepsis, pericarditis, myocardial strain, ischemia etc.), necessitating integration of clinical picture and further testing for confirmation.¹

Cardiac MRI (CMR) was performed in 38% of our patients; especially in those with more severe clinical presentations or ventricular dysfunction, which confirmed myocarditis in majority. Late gadolinium enhancement (LGE) indicating myocardial inflammation was present in 24 of 29 children (82%) who underwent CMR within 3 months following diagnosis. Therefore, we may conclude that CMR is a valuable confirmatory tool in high-risk pediatric cases. Recent pediatric imaging guidance describes CMR as a valuable non-invasive diagnostic alternative to endomyocardial biopsy and the key imaging modality for capturing the presence and distribution of myocardial inflammation.⁶ This positive yield is consistent with other pediatric series: for example, a multicenter study reported CMR findings consistent with myocarditis in about 80–82% of children imaged for suspected acute myocarditis.⁷ A more recent pediatric CMR series reported late gadolinium enhancement in all 28 children at baseline imaging and in 85.7% at 6-month follow-up.⁸

In our CMR subgroup, Although hs-cTnI and hs-cTnT levels were numerically higher in patients with LGE-positive CMR findings, the differences did not reach statistical significance when compared with the LGE-negative group. This finding should be interpreted with caution, as the number of patients with LGE-negative CMR findings was very limited. The small size of the LGE-negative subgroup may have reduced the statistical power to detect significant differences despite the substantial numerical separation between the groups. Therefore, the absence of statistical significance does not necessarily exclude a clinically meaningful association between troponin elevation and myocardial injury detected by CMR. However, the significant differences observed between the LGE-positive group and the remainder of the study cohort. This result may be due to selection of clinically severe cases for MRI study. This observation reinforces that troponin release correlates with the presence of myocardial injury that CMR can visualize. In other words, children with true myocardial inflammation (as evidenced by LGE on MRI) almost invariably showed high troponin levels in our study. Conversely, it is recognized that a negative CMR does not completely exclude myocarditis if clinical suspicion remains high, and cases of “troponin-positive, MRI-negative” myocarditis have been described.^{1,9} Recent ACC guidance likewise states that normal testing does not rule out myocarditis when the index of suspicion remains high.¹⁰ Each diagnostic modality has limitations when used in isolation: troponin-T is highly sensitive but not specific, whereas CMR is specific and provides anatomic detail but can miss very early or mild myocarditis and may not be readily available for all patients. A recent international survey found that only 35% of pediatric centers reported routine CMR use in all patients with suspected myocarditis, underscoring a continuing implementation gap.¹¹ Encouragingly, our data suggest that when troponin and CMR are used in combination, they provide complementary strengths; troponin as an early warning of myocardial injury, and CMR as confirmation of tissue inflammation. This multimodality approach accords with recent pediatric imaging guidance that integrates laboratory testing, cardiac biomarkers, and CMR to characterize disease severity and guide management.⁶

An important question is whether the magnitude of troponin elevation in myocarditis correlates with disease severity or outcomes. The prognostic significance of troponin elevation levels in childhood myocarditis is limited. The degree of troponin rise does not consistently correlate with the clinical severity of myocarditis. In our cohort, clinical outcomes were generally favorable: most patients stabilized within a week of therapy (mean hospital stay ~ 6 days), and only a small subset developed persistent ventricular dysfunction (two cases evolved to dilated cardiomyopathy during follow-up). We did not find a clear relationship between peak troponin level and the clinical course or degree of cardiac dysfunction on echocardiography. This is in agreement with several pediatric studies that have noted absence of correlation between troponin levels and short-term prognosis.

Kobayashi *et al.* described a series of adolescent myopericarditis patients in which median cTnI was 21 ng/mL, yet all patients had preserved ventricular function and benign short-term outcomes the elevated troponin I did not predict adverse events or myocardial dysfunction.¹² Likewise, Brosilow *et al.* recently reported that higher peak troponin I in pediatric myocarditis was associated with slightly longer hospital stay, but the correlation was weak, and there was no significant association with need for intensive care or inotropic support.¹³ Intriguingly, in that study some of the patients who required inotropic or mechanical cardiac support actually had lower peak troponin levels than others who did not; which was an unexpected finding that underscores the complex biology of troponin release in myocarditis.¹³ Our results and these studies collectively suggest that troponin magnitude alone is an unreliable predictor of clinical severity in myocarditis. This contrasts with acute myocardial infarction, where troponin elevations correlate with infarct size and outcomes; in myocarditis, troponin may be released due to diffuse inflammatory cell injury or reversible membrane leakage rather than large areas of irreversible necrosis, which may explain the discrepancy between troponin levels and functional outcomes.⁹ Janardhanan has noted that extremely high troponin levels in myocarditis often reflect increased cell membrane permeability during acute inflammation, and thus troponin can surge without causing (or predicting) severe left ventricular dysfunction.⁹ Consistent with this, the American Heart Association scientific statement on myocarditis observes that troponin levels do not reliably correlate with ejection fraction or arrhythmia risk in children, and that even a normal troponin does not exclude myocarditis if clinical suspicion is strong.¹ Therefore, clinicians should use troponin as a sensitive diagnostic clue, but avoid over-interpreting the absolute troponin value as a predictor of prognosis in pediatric myocarditis. Other factors such as clinical presentation, hemodynamic status, and imaging findings (echocardiography or CMR) are more informative for risk stratification. Recent ACC guidance identifies biventricular dysfunction, Stage D presentation, and late gadolinium enhancement on CMR as predictors of poor outcome.¹⁰

It is worth noting that troponin levels may have some prognostic implications over a longer term when combined with other data. Recent pediatric evidence suggests that the degree of initial myocardial injury (reflected by troponin and inflammatory markers) could influence the persistence of myocardial fibrosis. For example, a CMR follow-up study by Yükcü *et al.* found that children who had persistent LGE (scar tissue) on repeated CMR at 6 months had significantly higher admission troponin T and C-reactive

protein levels compared to those whose LGE resolved.⁸ This implies that a more intense initial injury, as indicated by higher troponin, might reflect more prolonged myocardial inflammation or scarring. Still, such findings need further confirmation, and they do not contradict the observation that the acute phase troponin elevation is not a strict predictor of short-term clinical deterioration. Overall, our study and the literature suggest that while troponin I and T are indispensable for recognizing pediatric myocarditis, they should be interpreted in context. Using troponins alongside cardiac imaging and clinical assessment provides the best insight into both the presence and implications of myocarditis in a child.

Conclusion

Both hs-cTnI and hs-cTnT are highly sensitive and important biomarkers of myocardial injury in pediatric myopericarditis and show a strong correlation, reflecting the same underlying inflammatory process. Although hs-cTnT demonstrated sensitivity comparable to hs-cTnI, its lower specificity indicates that isolated hs-cTnT elevation should be interpreted cautiously and within the clinical context. However, elevated troponin levels alone do not reliably reflect the severity of the disease. Cardiac MRI confirm myocardial involvement in the majority of high-risk patients and remains an important complementary diagnostic tool. Taken together, our findings support a multimodal diagnostic approach in which troponins serve as an early biochemical signal of myocardial injury and cardiac MRI provides tissue-level confirmation. In pediatric myopericarditis, the most accurate diagnosis is achieved not by relying on a single test, but by integrating biomarkers, imaging findings, and clinical assessment into a unified diagnostic framework.

Limitations

This study has several limitations. The overall cohort size was sufficient to demonstrate a significant correlation between cTnI and cTnT; however, differences in the temporal release patterns of cTnI and cTnT may have contributed to the lower specificity observed for cTnT. Although the number of patients in whom troponin levels were measured was sufficient, the limited number of patients undergoing CMR (29 patients) reduced the statistical power for evaluating associations between troponin levels and cardiac MRI findings. In addition, CMR was mainly performed in patients with more severe clinical presentations, introducing potential selection bias. Finally, this was a single-center study, and larger multicenter studies are needed for generalization of our findings.

Declarations

Data Sharing Statement:

The data that support the findings of this study are not openly available due to reasons of sensitivity and are available from the corresponding author upon reasonable request.

Authorship and contributorship

SK: Conceptualization (supporting), methodology, investigation (lead), and writing original draft preparation (lead). **EU:** Investigation (supporting). **SA:** Investigation (supporting). **EE:** Investigation (supporting). **EGO:** Investigation (supporting), statistics. **GH:** Investigation (supporting). **FA:** Conceptualization (lead), supervision (lead) and writing review and editing (lead).

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Conflicts of interest

All authors declare that they have no conflict of interest.

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Figures

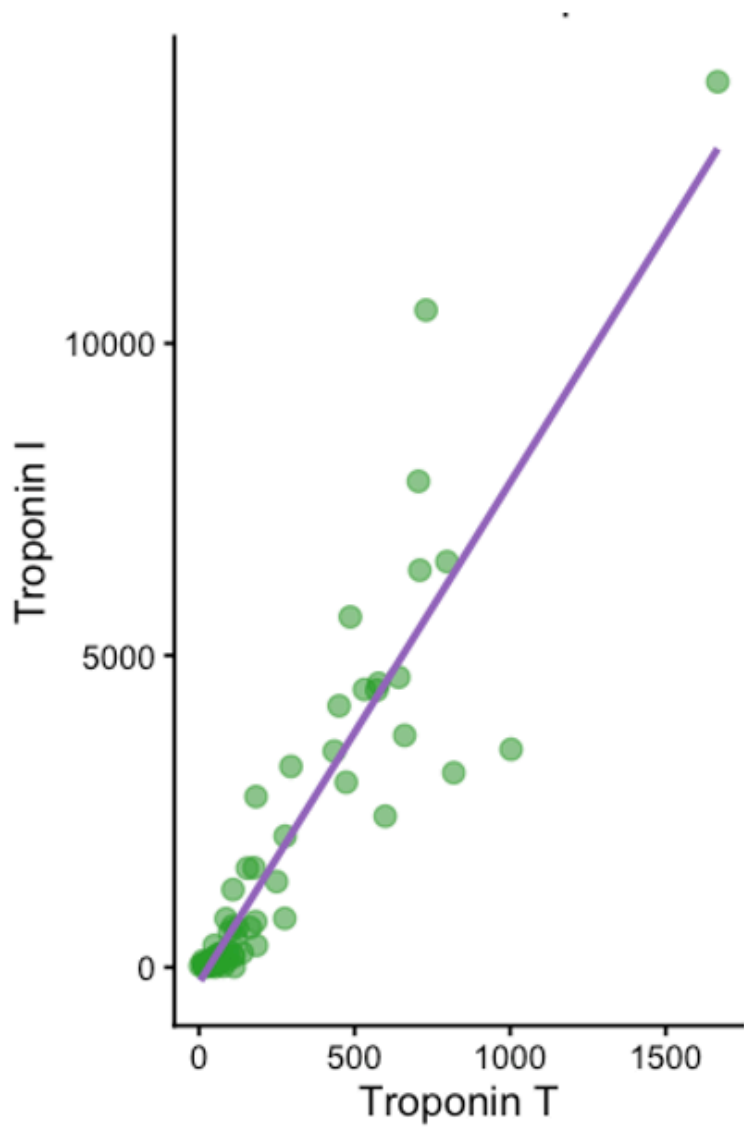


Figure 1

Spearman's Rho Rank Correlation of Troponin T and Troponin I levels (Spearman's rho = 0.891, $p < 0.001$);
purple line: Troponin I levels; green spots: Troponin T levels

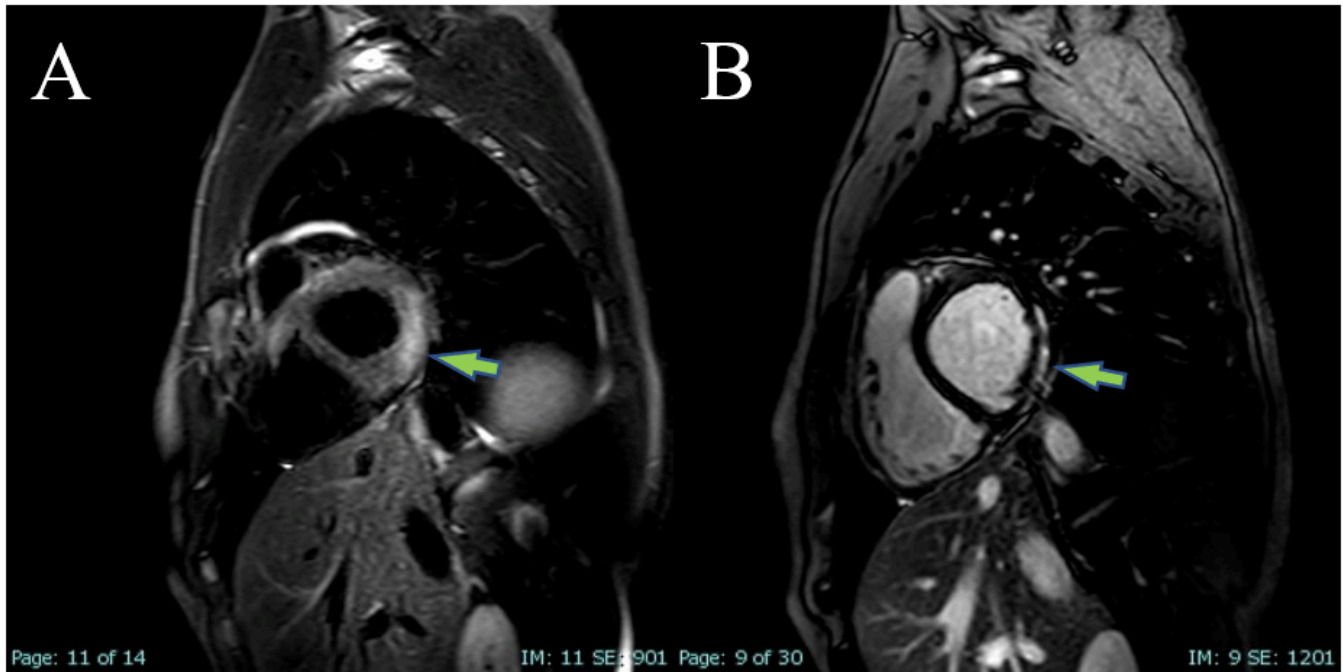


Figure 2

Late gadolinium enhancement, Cardiac MR images in Myopericarditis patients. **(A-B)** Late gadolinium enhancement involving the basal and midventricular segments of the left ventricular anterolateral and inferolateral walls, associated with hyperintensity on T2 mapping (green arrows)