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Predicting Cardiac Disease Based on Troponin Levels in Children and Adolescents

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Abstract

Objectives

Cardiac troponin (cTn) is frequently used to evaluate pediatric patients with suspected cardiac disease, yet reference thresholds remain poorly established. This study evaluated associations between troponin levels and cardiac pathology identified through diagnostic testing and sought a threshold to identify patients at low risk for cardiac magnetic resonance (CMR)-confirmed myocarditis.

Study Design:

This single-center retrospective study included patients aged 1–18 years with at least one elevated troponin measurement defined as > 0.029ng/mL at Nationwide Children's Hospital between January 2014 and December 2024. Medical records were reviewed for cardiac testing, inpatient events, arrhythmias, and outpatient outcomes. Cut point analysis was performed to maximize sensitivity and specificity, with additional analysis targeting a high-sensitivity threshold for CMR-confirmed myocarditis.

Results

Among 352 patients, troponin demonstrated limited predictive value for abnormalities on echocardiography, electrocardiography, Holter monitoring, and exercise stress testing. Among 149 patients undergoing acute CMR evaluation, a peak troponin threshold of 0.2 ng/mL yielded 95% sensitivity and an 86% negative predictive value for CMR-confirmed myocarditis. Of the patients with a peak troponin below 0.2 ng/mL with an initial normal echocardiogram and electrocardiogram, three patients had recorded inpatient arrhythmia with no sudden cardiac arrests, readmissions, or deaths.

Conclusions

Troponin elevation demonstrated limited predictive utility across most cardiac diagnostic modalities in children. A peak troponin threshold of 0.2 ng/mL may help guide CMR evaluation and risk stratification. In selected patients, this threshold may inform decisions regarding further evaluation and disposition. Prospective multicenter studies are needed to validate these findings and establish evidence-based clinical recommendations.

Introduction

Cardiac troponins (cTn) are commonly obtained in the evaluation of patients presenting with symptoms of cardiac disease. Although elevated troponin levels are often attributed to myocardial injury, numerous non-cardiac conditions including pulmonary embolism, end-stage renal disease, sepsis, medication, systemic inflammatory states, and even strenuous physical activity can also result in increased serum

concentrations. In pediatric populations, reported etiologies of elevated cTn are diverse, with myopericarditis representing only a subset of cases [1]. Although adult population-based studies have published abnormal cutoff values for high sensitivity cardiac troponin T (hs-cTnT) assays, most pediatric institutions do not employ high sensitivity troponin testing, and it is unclear whether the adult recommendations can be extrapolated to the pediatric population [2].

While myocarditis is a common cardiac etiology for elevated cTn levels, the diagnosis and management of suspected myocarditis remains a clinical challenge in pediatrics. In the past, biopsy proven pathologic evidence of inflammatory cardiomyopathy was required for diagnoses, but more recent guidelines have broadened the diagnostic criteria to include “biopsy proven,” “cardiac magnetic resonance (CMR) confirmed,” “clinically suspected,” and “possible myocarditis” [3]. Updated in 2018, the Lake Louise Criteria requires at least one T2-based criterion (global or regional increase of myocardial T2 relaxation time or an increased signal intensity in T2 weighted CMR images) with at least one T1 based criterion (increased myocardial T1, extracellular volume, or late gadolinium enhancement) for diagnosis of myocarditis. While the presence of both a T2 and T1 based marker will increase specificity for diagnosing acute myocardial inflammation, the presence of one marker may still support a diagnosis of acute myocardial inflammation in an appropriate clinical scenario [4]. While prior studies have proposed cTn cutoff values for the diagnosis of cardiac disease and myocarditis, these investigations have been limited either by small sample sizes or by the identification of low troponin thresholds that may be difficult to apply in routine clinical practice [5, 6]. Although associations between troponin levels and myocarditis have been explored, comparisons of admission versus peak troponin values in relation to a definitive myocarditis diagnosis have not been well characterized. In addition, long-term outcomes among patients with elevated troponin levels, with or without a diagnosis of myocarditis, remain poorly described in the literature. Given these gaps, this study sought to evaluate the relationship between troponin levels at admission and during hospitalization with cardiac pathology identified on echocardiography, electrocardiography, Holter monitoring, and CMR imaging. Additionally, we aimed to determine if a troponin threshold exists to rule out CMR confirmed myocarditis.

Methods

This single-center retrospective chart review included children aged 1–18 years in whom at least one troponin level was obtained during inpatient evaluation between January 1st, 2014 and December 30th, 2024. Patients who presented secondary to trauma, pulmonary embolism, a previous diagnosis of congenital heart disease or arrhythmia, post-arrest, chemotherapy-induced troponin elevation, drug ingestion, or patients that lacked an echocardiogram at the time of their initial troponin were excluded from the study. This study was approved by the Nationwide Children’s Hospital Institutional Review Board. No informed consent was required.

Elevated troponin levels were defined as an initial Troponin I level ≥ 0.029 ng/mL, which is the established clinical laboratory reference value at our institution, deemed to be consistent with myocardial injury. Troponin levels and symptoms at presentation along with peak troponins for each

admission were collected, as well as the primary diagnosis upon admission. A confirmed myocarditis diagnosis was based on positive CMR findings. A suspected myocarditis diagnosis was given if there was a high clinical suspicion for myocarditis during admission without confirmatory testing. An elevated troponin diagnosis was given to patients if there were elevated troponin levels without other evidence of myocarditis or other myocardial injury. An inpatient diagnosis of Multisystem Inflammatory Syndrome in Children (MIS-C) was given to those patients who met CDC diagnostic criteria [7]. A diagnosis of "Other, non-cardiac" was given for patients with an elevated troponin secondary to non-cardiac etiology. Echocardiograms were defined as abnormal in the presence of any of the following: depressed ejection fraction (EF < 50%) or fractional shortening (< 29%), any mild or more severe chamber dilation, greater than physiologic tricuspid or mitral regurgitation, and the presence of a pericardial effusion. ECGs were defined as abnormal in the presence of T wave inversion, ST segment changes, left ventricular hypertrophy, strain pattern, prolonged QT, or clinically significant arrhythmias (atrial fibrillation, non-sustained ventricular tachycardia, etc.). Holter monitors were defined as abnormal by the presence of more than rare atrial or ventricular ectopy, couplets, non-sustained or sustained arrhythmias, or the presence of atrioventricular block. Exercise stress tests (EST) were defined as abnormal in the setting of ST segment changes or arrhythmias. CMR findings were considered abnormal with either T2-based criterion (global or regional increase of myocardial T2 relaxation time or an increased signal intensity in T2 weighted CMR images) or T1 based criterion (increased myocardial T1, extracellular volume, or late gadolinium enhancement) for diagnosis of myocarditis. Arrhythmias were defined as abnormal in the presence of non-sustained or sustained atrial or ventricular arrhythmias, heart block, or junctional rhythm.

For each patient that met inclusion criteria, a complete chart review was completed, including evaluation of all pertinent cardiac studies (echocardiogram, ECG, EST, Holters, CMR) as well as all serial cardiology visits and subsequent admissions in the electronic medical record system (EMR). Each cardiac study was examined in chronological order until an abnormality was identified. Once an abnormality was identified, future cardiac studies of that type were not further reviewed. If no abnormality was seen, all further studies of that type were reviewed in the EMR. For example, if a patient was admitted with an abnormal echocardiogram, no further future echocardiograms for that patient were reviewed. If no abnormalities were seen on the admission echocardiogram, all further echocardiograms were reviewed to ensure no abnormalities were identified in the future. All data was entered and managed via RedCap.

Data were summarized using count and percentage for categorical variables and median/interquartile range (IQR, 25th and 75th percentile) for continuous variables. To assess the ability of troponin to rule out the presence of underlying cardiac abnormalities, cut point analysis maximizing sensitivity plus specificity was performed to determine optimum cut points and predictive value for troponin at admission and maximum troponin during admission for predicting an abnormal result on each modality separately, as well as on any modality. For each troponin-modality pair, troponin levels for patients with and without abnormal studies were visualized using side-by-side boxplots and ROC curves were created with the optimal cut point as determined by the sum of sensitivity and specificity and resulting metrics were presented in the figure. Additional cut point analysis was done to identify a cutoff with sufficiently

high sensitivity and negative predictive value (NPV) to “rule-out” CMR confirmed myocarditis. All analyses were performed using R version 4.3.0 [8–12].

Results

Admission & Demographics

After exclusion of 371 patients (Fig. 1), a total of 352 patients met study inclusion criteria and were included in the final cohort, with a mean age of 14.2 years (IQR 8.7–16.5 years). In the cohort, 224 (64%) of the patients were male, 68% were white, 27% black, 6% other, and 2% Asian, with some patients having identified under multiple races. Patients most commonly presented with chest pain (44%), fever (48%), other symptoms (45%), and shortness of breath (29%). Other symptoms included: headache, lightheadedness, dizziness, etc. (Table 1, Supplementary Table). In addition, the cohort’s inpatient primary diagnoses were 30% MIS-C, 21% with myocarditis, 19% with other diagnoses (including sepsis, infection, autoimmune conditions, etc.), 18% with elevated troponins not meeting criteria for myocarditis, and 8.2% with suspected myocarditis.

Table 1
Rates of abnormal findings across cardiac modalities

Study	Normal	Abnormal
Echocardiogram (n = 352)	219 (62%)	133 (38%)
Electrocardiogram (n = 348)	251 (72%)	97 (28%)
Holter (n = 129)	102 (79%)	27 (21%)
Exercise Stress Test (n = 126)	124 (98%)	2 (2%)
CMR (n = 179)	78 (44%)	101 (56%)

Cardiac Testing

Rates of abnormal findings on cardiac testing are shown in Table 1. Detailed abnormalities within each modality are shown in Tables 2 and 3 in the supplementary data. Of the 179 CMRs obtained, 101 were abnormal, with 84 obtained during admission and 17 during follow up. Out of the 101 abnormal CMRs, 7 (6.9%) had only edema, 25 (25%) had only fibrosis, and 69 (68%) had both fibrosis and edema.

Table 2
Cutoff Point Analysis Correlation Peak Troponin Levels to Myocarditis Diagnosis by CMR. Negative Predictive Value (NPV). Positive Predictive Value (PPV)

Cut Point	Sensitivity	Specificity	NPV	PPV
0.05	0.97	0.08	0.67	0.58
0.10	0.96	0.19	0.79	0.61
0.15	0.95	0.29	0.82	0.64
0.20	**0.95**	**0.41**	**0.86**	**0.68**
0.25	0.93	0.46	0.84	0.69
0.30	0.92	0.50	0.83	0.70
0.35	0.90	0.55	0.81	0.72
0.40	0.88	0.55	0.78	0.72
0.45	0.88	0.58	0.79	0.73
0.50	0.88	0.62	0.80	0.75
0.55	0.88	0.65	0.81	0.77
0.60	0.85	0.67	0.78	0.77
0.70	0.83	0.68	0.76	0.77
0.80	0.80	0.69	0.73	0.77
0.90	0.78	0.69	0.71	0.77
1.00	0.78	0.72	0.72	0.78

Table 3
Comparison of Abnormal Findings Seen by Cardiac Modalities Compared to CMR Proven Myocarditis. Calculated percentages are based on each individual cardiac modality.

Cardiac Study	Abnormal CMR N = 101	Normal CMR N = 78	No CMR N = 173
Echo			
Abnormal	45/134 (34%)	32/134 (24%)	57/134 (43%)
Normal	56/218 (26%)	46/218 (21%)	116/218 (53%)
ECG			
Abnormal	61/158 (39%)	27/158 (17%)	70/158 (44%)
Normal	40/190 (21%)	51/190 (27%)	99/190 (52%)
Unknown	0	0	4
Holter			
Abnormal	13/27 (48%)	9/27 (33%)	5/27 (19%)
Normal	53/102 (52%)	26/102 (25%)	23/102 (23%)
Unknown	35	43	145
EST			
Abnormal	2/2 (100%)	0/2 (0%)	0/2 (0%)
Normal	70/124 (56%)	39/124 (31%)	15/124 (12%)
Unknown	29	39	158

Of all 352 patients, 92% of patients did not have significant arrhythmias during their hospitalization. Three percent of patients had non-sustained or sustained ventricular tachycardia or ventricular fibrillation, 2.8% had non-sustained or sustained atrial arrhythmias, 1.4% had heart block, and 1.4% had junctional rhythm noted on telemetry while admitted. ROC curves for admission troponin and cardiac testing abnormalities are shown in Fig. 2. A similar analysis was conducted for peak troponins (Supplementary Fig. 1).

CMR Findings

Cut point analysis evaluating optimal troponin cut-off for peak troponin as a screening marker in CMR proven myocarditis is shown in Table 2. A cutoff point of 0.2 ng/mL was found to have the highest sensitivity of 95% with the highest NPV level of 86% for CMR-proven myocarditis.

Admission and peak troponins stratified by CMR findings for those obtained in the acute setting (defined as within 30 days of the initial drawn troponin) were used for this analysis (n = 149) are shown in Fig. 3. Under an admission troponin level cutoff point of 0.2 ng/mL there were nine positive CMRs (four with positive fibrosis, one with positive edema, four with both positive fibrosis and edema). Under a peak troponin cutoff point of 0.2 ng/mL there were five positive CMRs (two with positive fibrosis, one with positive edema, 2 with positive fibrosis and edema). The two patients with fibrosis alone had admission troponins of 0.034 ng/mL and 0.043 ng/mL and peak troponins of 0.034 ng/mL and 0.049 ng/mL, respectively. Both patients had repeat CMRs 3–4 months later that showed no LGE or edema, otherwise negative cardiac testing, and no negative clinical outcomes. Both patient presentations were thought to be non-acute myocarditis and likely indicators of chronic myocardial inflammation that was not clinically detected in the outpatient setting with subsequent fibrosis by CMR. A third patient had an admission and peak troponin of 0.129 ng/mL in the emergency department but had a troponin level from an outside hospital of 0.674 ng/mL. Patient four had an admission and peak troponin of 0.048 ng/mL at our institution with an outside hospital peak troponin of 0.119ng/mL. CMR showed both LGE and edema, however the patient was lost to follow up after their initial presentation, and a subsequent CMR was never obtained. Patient five had an admission and peak troponin of 0.062 ng/mL with CMR showing edema with no LGE, however the patient was lost to follow up and a repeat CMR was not obtained. Neither patient four nor five had any further hospital admissions for cardiac causes in their EMR charts.

When comparing the correlation between abnormalities seen on cardiac studies in CMR confirmed patients with myocarditis (Table 3), there were associated abnormalities seen 34% of the time by echocardiograms, 39% by ECGs, and 48% by Holter monitor. Out of the 101 studies with CMR proven myocarditis, 70 had normal ESTs and 29 did not have ESTs done in follow up.

Inpatient arrhythmias, death, transplant, and re-admission rates were evaluated using a troponin cutoff of 0.2 ng/mL (both admission and peak) as seen in Table 4. In the cohort of 161 patients with a peak troponin level below 0.2 ng/mL there were three patients that experienced inpatient arrhythmias, one patient death, one patient that required a transplant, and no patient readmissions. Of the three patients with arrhythmias, one patient had a peak of 0.041 ng/mL with mildly depressed LV function, a normal ECG, and non-sustained ventricular tachycardia (NSVT) at a rate of 100 bpm. This patient was discharged with a Holter that showed no further arrhythmias was ultimately diagnosed with MIS-C with a CMR negative for fibrosis or edema.The other two patients had LV dysfunction by echocardiogram and were ultimately diagnosed with pericarditis and likely cardiomyopathy. When evaluating long term outcomes, the only reported death in a patient with a troponin level below 0.2 ng/mL was a 16-month-old born prematurely at 27 weeks with severe bronchopulmonary dysplasia who remained intubated and admitted since birth and passed away months following when the initial troponin level was obtained. One patient ultimately required a transplant with a peak troponin level of 0.182 ng/mL,however, this patient's initial presentation showed severely diminished biventricular dysfunction, and the patient was ultimately admitted to the cardiac intensive care unit for inotropic and mechanical support. There were no sudden cardiac arrests or re-admissions in patients with a peak troponin less than 0.2 ng/mL. Ultimately, aside from the previously described patients, there were no long-term negative outcomes

seen in patients with a peak troponin less than 0.2 ng/mL, with the rest of the patients showing complete resolution of their symptoms at their last cardiology appointment.

Table 4
Troponin cut point analysis with negative outcomes. Both admission and peak troponin was analyzed in relationship to inpatient arrhythmias, death, transplant, and readmissions for cardiac etiology.

Characteristic	Admit Troponin		Max Troponin	
	< 0.2 N = 184	>=0.2 N = 168	< 0.2 N = 161	>=0.2 N = 191
Inpatient Arrhythmias	6 (3.3%)	22 (13%)	3 (1.9%)	25 (13%)
Patient Outcomes				
Death	1 (0.5%)	3 (1.8%)	1 (0.6%)	3 (1.6%)
Transplant	2 (1.1%)	1 (0.6%)	1 (0.6%)	2 (1.0%)
Readmission	0 (0%)	8 (4.8%)	0 (0%)	8 (4.2%)
Sudden Cardiac Arrest	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Complete resolution	170 (92%)	149 (89%)	148 (92%)	171 (90%)

Discussion

Cardiac troponin has become an increasingly utilized biomarker in the evaluation of pediatric patients presenting with possible cardiac pathology. Despite its widespread clinical use, the interpretation of elevated troponin levels in children remains challenging due to the broad range of potential etiologies and the lack of well-established pediatric reference thresholds. In this single center retrospective study, no clinically meaningful correlations were found between troponin and cardiac abnormalities via any modality except for CMR proven myocarditis. Patients with a peak troponin below 0.2 ng/mL had a high sensitivity and NPV for myocarditis with no significant negative outcomes in patients that initially presented with a normal cardiac workup. Previous studies have shown that elevated cardiac serologic biomarkers such as troponin I and troponin T are elevated in children with myocarditis [5, 6, 13–18].Overall mortality has been estimated at 1–7%, with higher troponin levels associated with electrocardiographic (ECG) abnormalities, extracorporeal membrane oxygenation (ECMO) utilization, and mortality, but inconsistently associated with arrhythmias and cardiac dysfunction [14, 17, 19–24].Multiple studies have shown that non-cardiac etiology and physiologic exercise induced troponin elevations complicate the diagnosis of suspected myocarditis as they may also exceed the upper reference limit of normal in numerous individuals [25–28].Patients presenting with elevated troponin levels and suspected myocarditis are frequently admitted for monitoring and diagnostic evaluation. Given the risk of exercise-associated sudden cardiac death, these patients are typically restricted from physical activity for three to six months, with serial assessment of myocardial edema, fibrosis, and ventricular function, as well as exercise stress testing prior to clearance for return to sports.While these

precautions are clinically appropriate, experience suggests that many patients are admitted with elevated troponin levels despite a low clinical suspicion for myocarditis. This practice may lead to unnecessary hospitalizations, increased utilization of limited healthcare resources, prolonged exercise restriction, and an added emotional burden for patients and families.

Existing literature has attempted to establish clinically useful troponin thresholds in pediatric populations. Dionne et al. evaluated troponin values in children undergoing evaluation for cardiac disease and reported that a troponin cutoff of 0.005 ng/mL in children older than three months demonstrated high sensitivity for identifying cardiac diagnoses. However, this threshold is significantly lower than what is typically used in routine clinical practice and may result in unnecessary testing and hospitalizations.[6] Similarly, earlier investigations evaluating troponin levels in biopsy-confirmed myocarditis demonstrated elevated troponin concentrations in affected patients but were limited by relatively small sample sizes, and were published prior to updated clinical guidelines that now include CMR as an diagnostic modality for myocarditis. Our study expands on this prior work by examining troponin correlations with multiple cardiac diagnostic modalities within a larger pediatric cohort with additional analysis comparing both admission and peak troponin measurements including reporting on patient outcomes years after troponin levels were obtained.

In the present study, troponin values demonstrated limited predictive value for abnormalities on echocardiography, electrocardiography, ambulatory rhythm monitoring, and exercise stress testing. For example, an admission troponin cutoff of 0.11 ng/mL yielded only moderate sensitivity (60%) and specificity (45%) for echocardiographic abnormalities, while substantially higher troponin thresholds were associated with arrhythmia abnormalities and abnormalities on electrocardiography and holter monitoring, although with still low sensitivity and NPV. These findings highlight the difficulty in establishing a clinically meaningful troponin cutoff capable of reliably identifying patients with underlying cardiac pathology and are in line with previously published literature. Our results suggest that troponin levels alone are insufficient to reliably predict abnormalities across commonly used cardiac diagnostic tests.

Importantly, when evaluating the relationship between troponin levels and CMR findings, a more clinically meaningful signal emerged. Increasing troponin concentrations were associated with a greater likelihood of CMR-confirmed myocarditis. Cut point analysis demonstrated that a peak troponin threshold of approximately 0.2 ng/mL provided the highest sensitivity for identifying CMR abnormalities consistent with myocarditis. At this threshold, sensitivity approached 95% with a NPV of 86%, positive predictive value of 68%. While this cutoff would not be suitable for definitively ruling out myocarditis, it may have potential utility as a screening threshold to identify patients who would benefit from further evaluation with cardiac CMR. In our cohort two patients with peak troponin levels below 0.2 ng/mL were diagnosed with acute myocarditis with peak troponin levels of 0.119 ng/mL and 0.064 ng/mL. It is important to note that the 86% NPV accounts for a total of five abnormal CMRs under a cutoff of 0.2 ng/mL and would be higher if optimized for the two true cases of acute myocarditis in our cohort. These findings demonstrate that troponin levels cannot exclude a myocarditis diagnosis, although can help

clinicians risk stratify patients. When evaluating for arrhythmias, only three patients in our cohort of 161 patients with a peak troponin less than 0.2 ng/mL had clinically significant arrhythmias during inpatient monitoring, consisting of short runs of NSVT at a rate of 100 beats per minute. Ultimately all three patients were activity restricted and discharged with Holter monitors which did not show arrhythmia recurrence. In addition, there were no long term sudden cardiac deaths, or readmissions within our cohort in patients with a peak troponin under 0.2 ng/mL who were not already admitted to the hospital and had normal echocardiograms and electrocardiograms.

This study has several limitations. A retrospective single-center study may not be generalizable to other institutions or patient populations. Not all patients underwent the full range of cardiac diagnostic testing, introducing potential selection bias. Since patients who presented following an arrest, sudden cardiac death as an outcome may be underestimated. Finally, CMR acquisition and reporting practices evolved over the study period. Several of the older studies did not specifically comment on myocardial edema, and when edema was assessed, it was often based on subjective interpretation of T2-weighted imaging rather than quantitative T2 mapping, which has become increasingly used in recent years. Notably, the studies categorized as having LGE without edema tended to be older overall (average CMR date October 2019 versus January 2021 for the rest of the cohort). This raises the possibility that some patients categorized as having LGE without edema may have had under-recognized myocardial edema.With more robust longitudinal data and outcome measures, it may be reasonable to consider that patients with troponin levels below 0.2 ng/mL undergo serial troponin testing in the emergency department, along with echocardiography, electrocardiography, and telemetry monitoring. If this evaluation is reassuring—demonstrating down trending troponin levels, a normal cardiac workup, and no arrhythmias on telemetry after a short period of observation—shared decision-making could support activity restriction, close outpatient follow-up, and CMR in the outpatient setting as opposed to routine inpatient admission. However, before formal clinical recommendations or management guidelines can be established, prospective multicenter studies are needed to better define the role of troponin testing in pediatric cardiac evaluation and to determine clinically meaningful thresholds to guide imaging and hospitalization decisions.

In conclusion, troponin elevation in pediatric patients demonstrates limited predictive value for general cardiac abnormalities detected on echocardiography, electrocardiography, Holter monitoring, or exercise stress testing. However, higher troponin levels appear to correlate more strongly with myocarditis diagnosed by CMR. A peak troponin threshold of approximately 0.2 ng/mL demonstrated high sensitivity for CMR abnormalities, suggesting a potential role as a screening threshold to guide further evaluation. Nevertheless, troponin should not be used in isolation to diagnose or exclude myocarditis, and clinical decision-making should incorporate the full clinical context and available diagnostic data.

Abbreviations

Cardiac troponin (cTn), Cardiac magnetic resonance (CMR), High-sensitivity cardiac troponin T (hs-cTnT), T1 relaxation time (T1), T2 relaxation time (T2), Ejection fraction (EF), Electrocardiogram (ECG), Exercise

stress test (EST), Electronic medical record (EMR), Interquartile range (IQR), Receiver operating characteristic (ROC), Negative predictive value (NPV), Positive predictive value (PPV), Multisystem inflammatory syndrome in children (MIS-C), Centers for Disease Control and Prevention (CDC), Late gadolinium enhancement (LGE), Left ventricular (LV), Nonsustained ventricular tachycardia (NSVT), Extracorporeal membrane oxygenation (ECMO), Pediatric Health Information System Plus (PHIS+), R statistical software (R).

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Figures

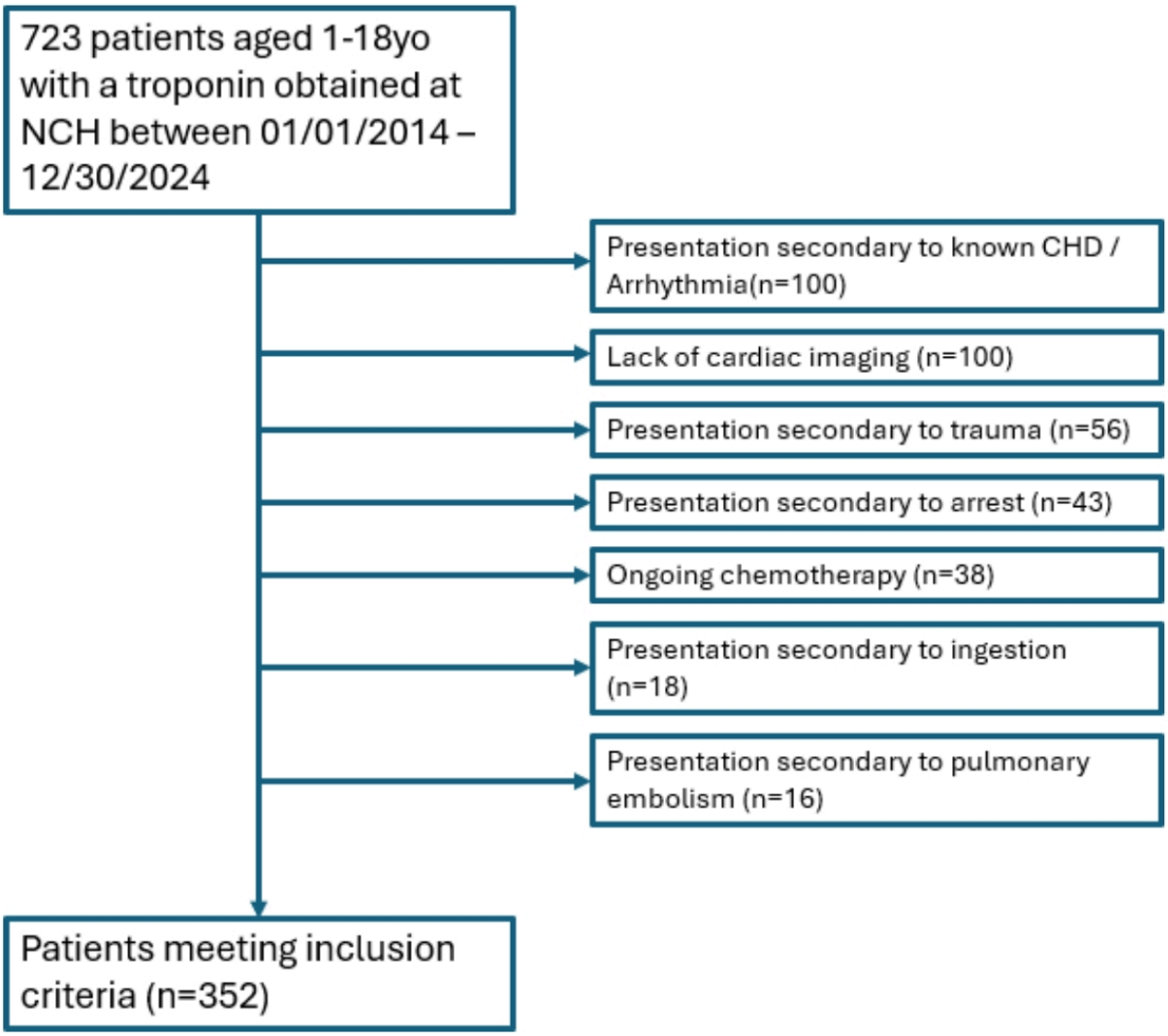


Figure 1

Inclusion and exclusion criteria. 723 patients had a troponin obtained between 1/1/2014-12/30/2024. 100 patients were excluded due to known CHD or arrhythmia. 100 were excluded due to lack of an echocardiogram during the time of the troponin drawn. 56 patients presented secondary to trauma and excluded. 43 patients presented secondary to an arrest and were excluded. 38 patients had a troponin obtained during inpatient chemotherapy and excluded. 18 patients had a troponin drawn following and ingestion and 16 had an elevated troponin in the setting of a pulmonary embolism and ultimately excluded.

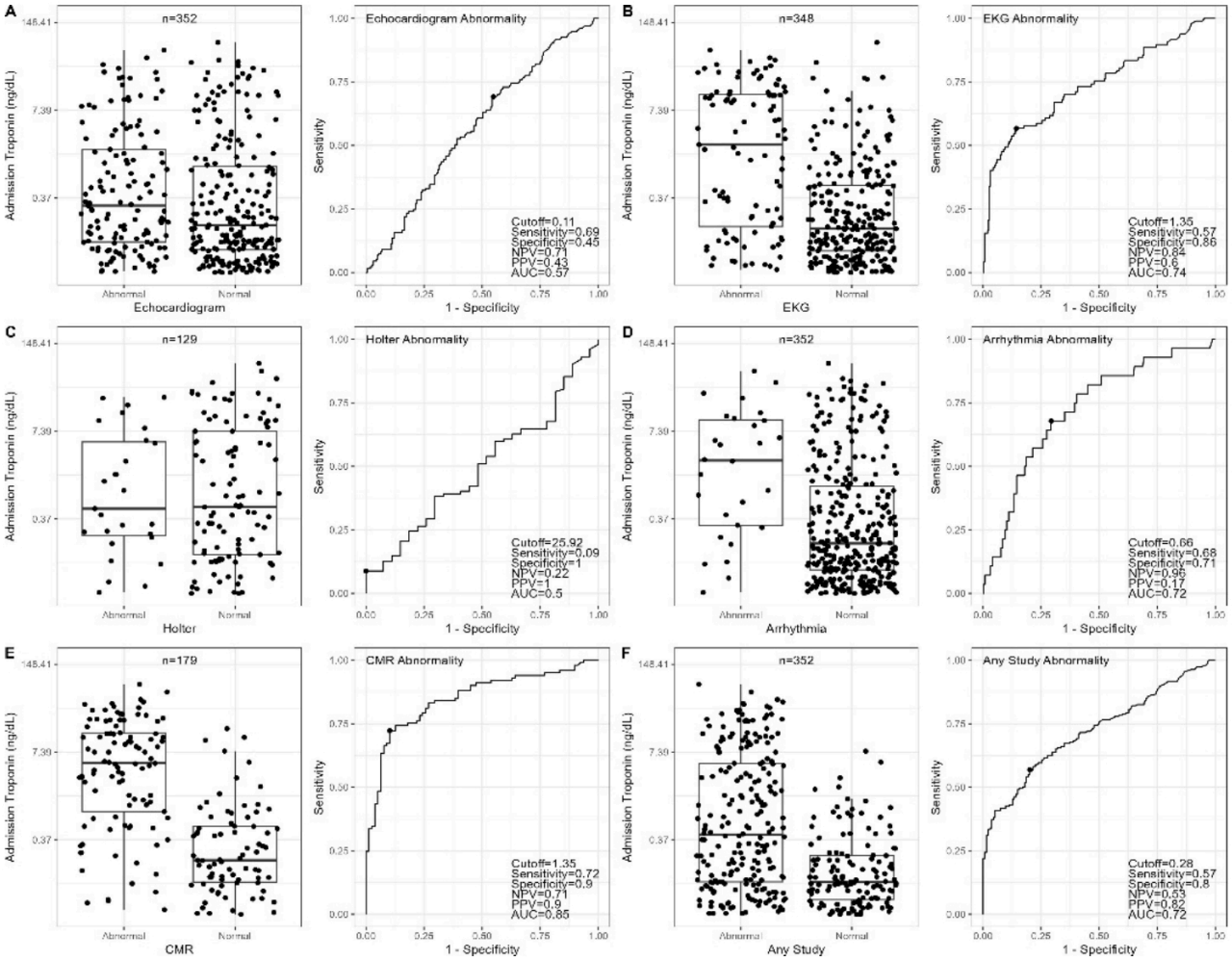


Figure 2

For each troponin-modality pair, admission troponin levels for patients with and without abnormal studies were visualized using side-by-side boxplots and ROC curves were created with the optimal cutpoint and resulting metrics. y-axis is log-scaled for visualization purposes.

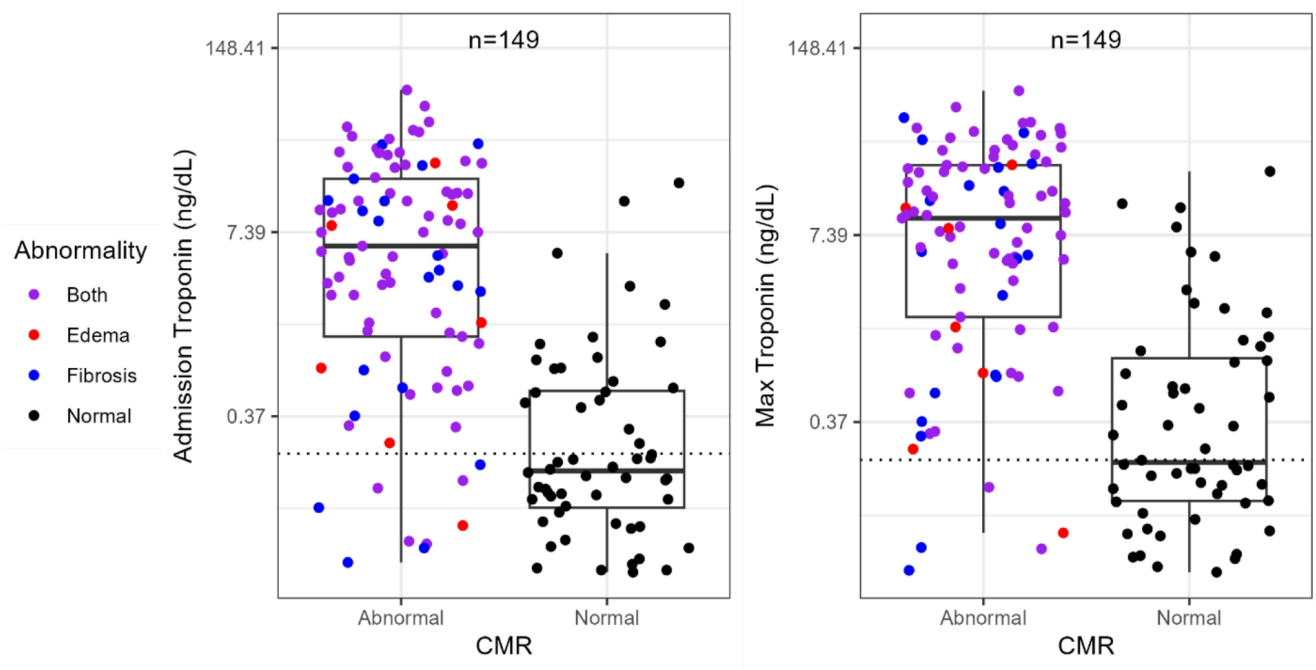


Figure 3

Admission (left) and max (right) troponin in relation to CMR findings. The dotted line represents a cutoff point of 0.2 ng/mL. Black dots represent normal CMRs. Red dots represent the presence of only edema on CMR. Blue dots represent the presence of only fibrosis on CMR. Purple dots represent the presence of both edema and fibrosis. y-axis is log-scaled for visualization purposes.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- SupplementalTablesFinal.docx