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ORIGINAL ARTICLE

Assessing Myocardial Injury Using Troponin I as a Diagnostic Marker with Electrocardiography Prior to and after Paediatric Cardiac Catheterisation

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ABSTRACT

Background: High-sensitivity cardiac troponin I (hs-cTnI) is a sensitive and specific marker for identifying myocardial damage and releases from the cardiac muscles during cardiac catheterization. So, we aimed to assess serum (hs-cTnI) level with electrocardiograph (ECG) monitoring in children before and 12 hours after catheterization to correlate its level, ECG changes and myocardial injury.

Methods: Twenty-five pediatric patients participated in this prospective cohort study, which was conducted at Zagazig University Hospitals, Pediatric Cardiac Catheterization Unit. Detailed history taking, clinical examination, biochemical, and radiological profiles were evaluated. High sensitivity cardiac troponin I and ECG were assessed before and 12 hours after cardiac catheterization.

Results: After catheterization, troponin I levels increased from 0.113±0.01 ng/mL to 0.131±0.02 ng/mL, a statistically significant rise. Troponin I levels rose significantly from 0.110±0.02 to 0.151±0.02 post-catheterization for patients with interventional pulmonary stenosis, but there was no statistically significant difference in mean levels between those who had the intervention procedure for atrial septal defect and patent ductus arteriosus before and after. There were no notable post-procedural signs of ischemia detected by ECG monitoring.

Conclusions: Pediatric cardiac catheterization appears to be safe and well-tolerated procedure with minimal risk of inflammatory, hematologic, or electrophysiological complications. Troponin I proved to be a sensitive biomarker for detecting subclinical myocardial stress, even in the absence of clinical or ECG findings. The overall findings reinforce the value of catheterization in both diagnosis and treatment of congenital heart defects and support its continued use in pediatric cardiology with careful monitoring and patient selection.

Keywords*:* Troponin I; Myocardial ischemia; Myocardial injury; Congenital heart disease; Pediatric cardiac catheterization

INTRODUCTION

The "gold standard" for evaluating the anatomy and physiology of patients with congenital heart disease has traditionally been cardiac catheterization. Rapid digital angiography in conjunction with real-time fluoroscopy with contrast injection has produced the high-resolution cardiac pictures required for these patients' successful surgical care. [1].

In the last two to three decades, percutaneous interventions for pediatric heart disease have been established. Devices for the percutaneous closure of muscular VSDs, PDAs, and ASDs are accepted. The percutaneous valve implantation phase is only getting started [2].

Free cardiac troponin T is first released into the plasma by the heart following a myocardial infarction, according to research. Cardiac troponin I is released later, along with tertiary cardiac troponin T-cardiac troponin I-cardiac troponin C complexes and an infrequent fragment of cardiac troponin T. since they are rapidly destroyed into free cardiac troponin T and cardiac troponin I - troponin C binary complexes [1].

Troponin assays are about to enter a new era due to technological advancements. Troponin assays from the previous generation have been utilized for risk stratification to help with treatment selection and triage decisions, as well as diagnostic and prognostic indicators in patients with acute coronary syndrome. With their increased sensitivity for cardiac myocyte necrosis, new, high-sensitivity troponin assays are a significant advancement; nonetheless, careful interpretation of these tests is still required [3].

In adult patients with coronary artery disease, cardiac troponin I is a proven diagnostic test that is both sensitive and specific for identifying myocardial damage [4].

Congenital and acquired heart disease in children have been shown to have elevated troponin levels. Cardiac troponin I diagnostic value as a sign of myocardial damage during interventional catheterization is developing. Also, there have been reports of its rise following radiofrequency ablation in adults, and it has been linked to procedural factors [5].

The purpose of this study was to examine the diagnostic utility of troponin I (hs_cTnI) as a marker for myocardial damage in pediatric patients who had diagnostic cardiac catheterization and intervention along with electrocardiographic monitoring prior to and following the procedure, as well as 12 hours after the procedure.

METHODS

This prospective cohort study was conducted on twenty-five pediatric patients who were admitted and required pediatric cardiac catheterization for diagnostic or interventional purposes; both sexes were included, during the period from April 2024 to April 2025. The study was conducted at the Pediatric Cardiac Catheterization Unit, located within the Pediatrics Department of Zagazig University Hospitals. This facility was equipped with advanced medical infrastructure and specialized personnel experienced in pediatric cardiac care. The study was approved by the Research Ethical Committee of Zagazig University hospitals with an Institutional Review Board (IRB 277-23/4/2024) and every participant in the study gave their signed, informed consent.

Inclusion criteria

This study included 25 infants and children aged between 6 months and 10 years with cardiac diseases. Fifteen patients admitted for patent ductus arteriosus closure, three patients for atrial septal defect closure and 7 patients admitted for cardiac catheterization, balloon pulmonary valvuloplasty to dilate the stenotic pulmonary valve, and angiography was used to visualize the level and extent of stenosis in pulmonary stenosis.

Exclusion criteria

Children diagnosed with heart failure, cardiomyopathy, or myocarditis. kidney diseases, acute or chronic infections, muscle diseases, neoplastic diseases, and patients with elevated troponin levels prior to catheterization were excluded [6].

As a standard work-up prior to the procedure, all study participants underwent a complete history taking, a comprehensive general and local cardiac examination, and laboratory tests, including complete blood pictures, C-reactive protein (CRP), electrolytes, liver function tests, kidney function tests, hepatitis B and C screening as a routine work -up. A plain Chest X-ray was done to detect any abnormalities in the chest region. Electrocardiography (ECG) was performed both before and after the procedure to monitor any changes in the heart's electrical activity. Echocardiography was conducted to assess the structure and function of the heart. Cardiac catheterization was the gold standard for diagnosing and treating cardiac abnormalities [7].

Serum highly sensitive cardiac troponin I (hs_cTnI) was measured both before and 12 hours after the cardiac catheterization to evaluate myocardial injury. It is measured by

ELISA in serum samples with a cutoff point of 0.160 ng/ml.

Statistical analysis

The software Statistical Package for Social Sciences was applied to analyze the data by using version 28. Software was used to analyze the data to numerical and percentage data. The median, range, mean, and standard deviation were used. Utilizing the Spearman test was used for correlation analysis. Two study groups containing quantitative data that was not properly distributed were assessed using the Wilcoxon Signed Ranks Test. The F-tests were employed to determine whether there was a significant difference between the means of the two independent groups. A statistically highly significant difference was present if $p \leq 0.001$, as indicated by the level of statistical significance, which was set at $p < 0.05$.

RESULTS

The study included a total of 25 pediatric patients, their ages ranging from 6 months to 10 years; their sex distribution was 13 males (52%) and 12 females (48%). The weight ranged from 5 kg to 35 kg. Similarly, the height of the patients varied between 55 cm and 130 cm, with a mean height of 89 cm ± 22.33 cm. The mean duration of illness was 32.28 months, with a standard deviation of 16.2 months. Cardiac catheterization was performed on 15 cases with patent ductus arteriosus (60%) , 7 cases with pulmonary stenosis (28%) and 3 cases with atrial septal defect (12%) (Table 1).

Among the 25 pediatric patients undergoing cardiac catheterization, a variety of pre-existing complications were observed. The most reported condition was a history of repeated chest infections, which affected 8 patients (32%). Cyanosis was present in 5 patients (20%). Other reported complications included exertional dyspnea (16%), feeding difficulties with low birth weight (8%), tachypnea (8%), a history of neonatal intensive care unit (NICU) admission (8%), and syncope or palpitations (4%). Notably, only 1 patient (4%) was entirely asymptomatic. Regarding medication use, 40% of patients (n=10) were receiving frusemide. Captopril was administered to 44% of the cases (n=11). Spironolactone was taken by one patient (Table 2)

The Complete Blood Count (CBC) was collected before and after the procedure. White Blood Cell (WBC) Count Before catheterization, the mean WBC count was 7564±2162 cells/μL; after, it remained nearly unchanged at 7584±2151 cells/μL. Hemoglobin (HB) levels, before, were 9.74 ± 1.09 g/dL .After catheterization, the mean hemoglobin level remained stable at 9.75 ± 1.08 g/dL, and the platelet count before was 360 ± 60.76 × 10^3/μL and after was 360.12 ± 60.68 × 10^3/μL. Before the procedure, the mean CRP level was 4.17±0.9 mg/L, with a range from 2 to 6 mg/L. Twelve hours after catheterization, the mean CRP level increased to 4.39±0.46 mg/L, with values ranging from 2 to 6 mg/L. All parameters showed no statistical significance before and after catheterization. (Table 3).

Cardiac imaging, including echocardiography (ECHO), showed patent ductus arteriosus (PDA) in 15 patients (60%). Atrial Septal Defect (ASD) was found in 3 patients (12%), while Ventricular Septal Defect (VSD) was present in 2 patients (8%). Pulmonary Stenosis (PS) was found in 7 patients (28%), Tricuspid Regurgitation (TR), Mitral Regurgitation (MR), and Pulmonary Hypertension (PHT) were each observed in 1 (4%), and Small Patent Foramen Ovale (PFO) was detected in 1 patient (4%). Chest X-rays revealed cardiomegaly was observed in 10 patients (40%), right ventricle enlargement in 5 patients (20%), right atrium (RA) enlargement in 2 patients (8%), and increased pulmonary vascular markings in 8 patients (32%). (Table 4).

The correlation between Troponin I levels and other clinical markers such as ST segment, T wave, Q wave, WBCs (White Blood Cells), HB (Hemoglobin), Plts (Platelets), HB (hemoglobin), and CRP (C-reactive protein) was not statistically significant (p values > 0.05). while Troponin I level has a significant correlation with tachycardia and type of defect (Table 5).

Electrocardiographic findings before and after catheterization showed that, the heart rate increased from a baseline of 78.4 ± 8.6 bpm to a 89.7 ± 9.5 beat per minute, p = 0.003, in

a statistically significant manner. Other parameters such as PR interval, QRS duration, QTc, and (ST segment, T wave, Q wave) did not achieve statistical significance (Table 6).

Analysis of mean troponin I value before and after catheterization showed a statistically significant increase following the procedure in individuals diagnosed with pulmonary stenosis. However, no statistically significant difference was observed for patients with patent ductus arteriosus or atrial septal defect. Troponin I values before catheterization averaged 0.113±0.01, while the mean value after catheterization was 0.131±0.02 (Table 7).

Table 1: Demographic data among the study population and cases underwent cardiac catheterization

Age (month)	
Mean± SD	43.84±37.32
Range (Min-Max)	6-120
Sex	
Male	13(52%)
Female	12(48%)
Weight (kg)	
Mean± SD	12.36±7.09
Range (Min-Max)	5-35
Height (cm)	
Mean± SD	89±22.33
Range (Min-Max)	55-130
Duration of Illness (Months)	
Mean± SD	32.28±16.2
Range (Min-Max)	10-58

Cases for cardiac catheterization		
Diagnosis	**Number of cases**	**Percentage (%)**
PDA	15	60%
PS	7	28%
ASD	3	12%
Total	25	100%

Table 2: Clinical data among cases.

Main clinical data and history n (%)	
Repeated chest infections	8(32%)
Cyanosis	5(20%)
Dyspnea	4(16%)
Tachypnea	2(8%)
History of NICU	2(8%)
Feeding difficulties, Low birth weight	2(8%)
Syncope/palpitations	1(4%)
Asymptomatic	1(4%)
Drugs Used	
Frusemide	
No	15(60%)
Yes	10(40%)
Captopril	
No	14(56%)
Yes	11(44%)
Spironolactone	
No	24(96%)
Yes	1(4%)

Table 3: complete blood count Tests and CRP (C-Reactive Protein) Data distribution in all study population

CBC Parameter	Before Catheterization	After Catheterization	Paired t-test
			p-value
HB (g/dL)	9.74 ± 1.09 (8.2–12)	9.65 ± 1.08 (8.2–12)	0.016 NS
WBC (cells/µL)	7564 ± 2162 (3000–9000)	7584 ± 2151 (3000–9000)	0.02 NS
Platelets (×10³/µL)	360 ± 60.76 (225–500)	360.12 ± 60.68 (225–500)	0.235 NS
C-Reactive Protein	4.17±0.9 (N: 2-6 mg/L).	4.39±0.46 (N: 2-6 mg/L).	0.891 NS

Table 4: Echocardiographic and x ray data among studies group and indication for cardiac catheterization

Cardiac Imaging & Diagnostic Tests	
Echocardiographic (ECHO) Findings	**N(%)**
Small PFO	1(4%)
PDA	15(60%)
ASD	3(12%)
VSD	2(8%)
PS	7(28%)
TR	1(4%)
MR	1(4%)
PHT	1(4%)
X-ray Findings	
Cardiomegaly	10(40%)
RV Enlargement	5(20%)
RA Enlargement	2(8%)
Pulmonary Vascular Markings	8(32%)

Table 5: Correlation analysis using Spearman test for the effect of troponin 1 on variables

Troponin 1	r Value	95.0% CI for HR	R Squared	P value	P value
ST Segment	0.09178	-0.1915 to 0.3609	0.008424	0.5261	N. S
T wave	-0.4181	-0.3838 to 0.1657	0.1394	0.0141	N.S
pathological Q wave	-0.08702	-0.3567 to 0.1961	0.007573	0.5479	N. S
Tachycardia	0.4285	0.1705 to 0.6316	0.1837	0.0019	Sig.
WBCs	0.01495	-0.2645 to 0.2921	0.000224	0.9179	N. S
HB	0.1379	-0.1461 to 0.4008	0.01901	0.3396	N. S
Platlets	-0.03608	-0.3113 to 0.2447	0.001301	0.8036	N. S
CRP	-0.2208	-0.4703 to 0.06129	0.04876	0.1233	N. S
Type of defect	-0.4184	-0.6241 to -0.1585	0.1751	0.0025	Sig.
Duration of Illness (Months)	0.172	-0.1117 to 0.4298	0.02958	0.2324	N. S

Table 6 : Electrocardiographic findings before and after catheterization.

ECG Parameter	Before Catheterization	After Catheterization	p-value	Significance
Heart Rate (bpm)	78.4 ± 8.6	89.7 ± 9.5	0.003*	Significant
PR Interval (sec)	0.15 ± 0.01	0.14± 0.01	0.256	NS
QRS Duration (sec)	0.06 ± 0.01	0.055 ± 0.02	0.318	NS
QTc Interval (ms)	0.44 ± 0.012	0.40 ± 0.014	0.402	NS
STSegment Changes	No abnormal changes	No abnormal changes		
T Wave Changes	No abnormal changes	No abnormal changes		
Q Wave	Normal	Normal		

Table (7): Association between Troponin I (before and after) and cath. diagnosis

Diagnosisfor Catheterization		**Troponin I before Catheterization**	**Troponin I after Catheterization**	**Wilcoxon Signed Ranks Test**	**P –Value**
PDA (n =15)	**Mean ± SD**	0.115±0.01	0.117±0.01	-1.3	0.18
PS (n =7)	**Mean ± SD**	0.110±0.02	0.151±0.02	-2.4	0.01*
ASD (n =3)	**Mean ± SD**	0.110±0.00	0.150±0.01	-1.6	0.12
Total (n =25)	**Mean ± SD**	0.113±0.01	0.131±0.02	-3.2	0.001**

* p-value < 0.01 .** p-value <0.001

DISCUSSION

In addition to significantly improving the safety and effectiveness of surgery, cardiac catheterization and angiography have revolutionized the treatment of infants with congenital heart disease. For many extremely complex lesions, diagnostic cardiac catheterization is still the "last authority" for this conclusive anatomical and hemodynamic information. [1].

Troponin in the heart It is an indicator of cardiac injury that is both sensitive and specific. Normally, the blood does not contain this biomarker. Troponin is released into the bloodstream by injured cardiac muscles' contractile proteins. Five to forty percent of individuals receiving percutaneous coronary intervention have been found to have elevated serum cardiac troponin I levels [8].

This study aimed at investigating the diagnostic value of troponin I (hs-cTnI) as a marker for myocardial injury in pediatric patients who underwent intervention and diagnostic cardiac catheterization with electrocardiographic monitoring before and 12 hours after the procedure.

Our results added to the expanding corpus of literature supporting the safety and diagnostic value of catheter-based interventions in pediatric populations. While minor biochemical and structural changes were observed post-procedure, these remained within clinically acceptable ranges and did not result in adverse outcomes. In this context, our results were compared with previous studies to highlight areas of alignment and divergence. The discussion below synthesizes our observations with published data, offering insights into the implications of our findings for clinical practice and future research in pediatric interventional cardiology.

This study investigated serum cardiac troponin I (cTnI) levels in a cohort of 25 pediatric patients undergoing cardiac catheterization. The age ranged from 6 months to 10 years, with a mean of 43.84 ± 37.32 months. The sample was nearly gender-balanced, comprising 13 males (52%) and 12 females (48%). Patient weights ranged from 5 kg to 35 kg (mean: 12.36 ± 7.09 kg), while heights varied from 55 cm to 130 cm (mean: 89 ± 22.33 cm), reflecting a heterogeneous pediatric population with diverse anthropometric characteristics. This variability mirrors the broad spectrum of physical development in children referred to for cardiac catheterization and enhances the generalizability of our findings within pediatric cardiology. In our study, fifteen cases (60%) with patent ductus arteriosus, seven cases (28%) with pulmonary stenosis, and three cases (12%) with atrial septal defect underwent cardiac catheterization.

In our cohort of 25 pediatric patients undergoing cardiac catheterization, a variety of pre-existing clinical complications were observed. The most prevalent comorbidity was a history of repeated chest infections, reported in 32% of the patients. This finding may reflect an association between recurrent respiratory illness and underlying congenital heart defects, particularly those compromising pulmonary circulation. Cyanosis was the second most frequent complication, present in 20% of cases, suggesting significant issues with systemic oxygenation and indicative of more complex or advanced forms of congenital heart disease. Additional complications included exertional dyspnea (16%), feeding difficulties associated with low birth weight (8%), tachypnea (8%), prior admission to the neonatal intensive care unit (NICU) (8%), and episodes of syncope or palpitations (4%). Notably, only one patient (4%) was asymptomatic prior to catheterization, underscoring the high clinical burden and complex health profiles of the study population.

Allen et al. [9] emphasized the importance of early cardiac evaluation in neonates presenting with cyanosis or weak peripheral pulses, particularly in critical care settings. They recommended the use of cardiac biomarkers, including troponin I, to identify potential myocardial injury in such high-risk cases. The inclusion of cyanotic and NICU-admitted patients in our study aligns with this diagnostic approach and supports the relevance of troponin I screening in similar clinical contexts. Additionally, Namuyonga and Mocumbi [10] investigated the role of cardiac biomarkers in African pediatric patients with pulmonary hypertension, a condition frequently associated with cyanotic congenital heart disease. Their findings, although focused on HIV-infected populations, highlighted the value of troponin I in assessing disease severity and its relationship to pre-existing comorbidities. These studies collectively reinforce the clinical significance of troponin I as a diagnostic tool, particularly in pediatric patients with complex cardiovascular and systemic conditions, as represented in our study.

A diverse pharmacological profile that reflected the diverse clinical manifestations linked to congenital heart disease was revealed by looking at drug use among the 25 pediatric patients in our study. Of the patients, 44% were prescribed captopril, an angiotensin-converting enzyme (ACE) inhibitor, to sustain cardiac function, and 40% were on diuretics, mostly for the treatment of fluid overload. In 4% of cases, the mineralocorticoid receptor antagonist spironolactone was utilized. Others, notably, needed at least one medicine, highlighting the need for tailored treatment plans and the varying severity of cardiac dysfunction.

These findings are consistent with current clinical guidelines and recent research. Koubský [11] emphasized the use of ACE inhibitors such as captopril and loop diuretics as first-line therapy in the management of pediatric chronic heart failure, particularly in the context of congenital structural anomalies. His updated recommendations took age-specific pharmacodynamics and developmental variability into account, which align well with the age range and physical diversity of our cohort. Similarly, Ahmed and VanderPluym [12] noted high rates of ACE inhibitor and diuretic use among pediatric patients with structural heart disease

awaiting advanced interventions, closely mirroring the 44% and 40% usage rates observed in our population.

Lastly, Singh et al. [13] provided a comprehensive overview of the mechanisms and therapeutic roles of commonly used agents in pediatric congestive heart failure. Their findings further validate the pharmacological patterns observed in our study, highlighting the central role of ACE inhibitors and diuretics and the selective, condition-specific use of adjunct therapies such as spironolactone. Together, these comparisons support the relevance and appropriateness of the medication regimens observed in our cohort, while also reflecting broader clinical trends in pediatric cardiac care.

In pediatric patients undergoing cardiac catheterization, the complete blood count (CBC) is a crucial test for assessing coagulation state, oxygen-carrying ability, and immunological response. the information gathered both prior to and following the procedure. In this study, the mean white blood cell (WBC) count was 7564±2162 cells/μL before catheterization, and it stayed almost constant at 7584±2151 cells/μL. Previously, hemoglobin (HB) levels were 9.74 ± 1.09 g/dL. The mean hemoglobin level after catheterization was 9.65 ± 1.08 g/dL, and the platelet count was 360 ± 60.76 × 10^3/μL before and 360.12 ± 60.68 × 10^3/μL after. C-Reactive Protein (CRP) is an inflammatory biomarker that helps assess systemic inflammation and can provide insight into the body's response to cardiac catheterization. Before the procedure, the mean CRP level was 4.17±0.9 mg/L. Twelve hours after catheterization, the mean CRP level was 4.39±0.46 mg/L. This stability indicated that routine cardiac catheterization does not provoke a measurable inflammatory reaction in most pediatric patients.

Hemoglobin (Hb) values in our study showed relative stability. The pre-catheterization mean was 9.74 ± 1.09 g/dL, consistent with mild anemia frequently observed in children with congenital heart disease. Post-catheterization, the mean Hb level remained nearly unchanged at 9.65 ± 1.08 g/dL, suggesting no significant intraprocedural blood loss or hemolytic complications. Similarly, platelet counts exhibited no clinically relevant shifts. The pre-procedure mean was 360 ± 60.76 ×10^3/μL and this remained stable after the procedure, indicating an absence of thrombotic or bleeding events related to catheter manipulation.

These findings are in alignment with previous studies evaluating inflammatory and hematological responses in pediatric cardiovascular interventions. Luo et al. [14] analyzed WBC, CRP, and procalcitonin (PCT) levels in children undergoing percutaneous patent ductus arteriosus (PDA) closure via catheterization. Their findings demonstrated no significant post-procedural elevation in these markers, supporting the notion that such interventions rarely trigger a systemic immune response.

Similarly, Kumar et al. [15], while investigating pediatric cardiac surgery under thoracic epidural anesthesia, observed that CRP increased significantly postoperatively, whereas WBC counts remained relatively inconsistent as indicators of inflammation. This supports our conclusion that WBC count alone may not reliably reflect transient inflammatory responses in the catheterization setting.

According to our research, the most prevalent abnormality among pediatric patients having cardiac catheterization was patent ductus arteriosus (PDA), which was seen in 15 patients (60%) out of the total, pulmonary stenosis (PS) in 7 patients (28%), three patients (12%) had an atrial septal defect (ASD), and two patients (8%) had a ventricular septal defect (VSD). Other cardiac abnormalities included tricuspid regurgitation (TR), mitral regurgitation (MR), pulmonary hypertension (PHT) and a small patent foramen ovale (PFO), each of which accounted for 4% of the sample.

These findings are consistent with those reported by Aziz et al. [16], who studied a large Egyptian cohort of pediatric cardiac catheterizations and found PDA to be the most frequently encountered congenital heart defect, present in 65% of cases. Their results mirror our PDA prevalence of 68%, reinforcing the notion that PDA represents the

dominant indication for catheter-based intervention in both local and regional tertiary care settings, particularly in low- and middle-income countries where non-surgical approaches are increasingly favored.

Similarly, Luo et al. [14] focused on percutaneous PDA device closure in children and concluded that PDA remains the most treated lesion via catheter-based techniques. The inclusion of device therapy in their cohort closely aligns with the interventional intent seen in our population, where catheterization served both diagnostic and therapeutic roles.

Collectively, these data confirmed the predominance of PDA as a major target for pediatric cardiac catheterization and emphasized the importance of catheter-based strategies in the diagnosis and management of structural congenital heart defects across diverse clinical settings.

Our cohort's chest X-ray evaluation showed a radiological characteristiclinked to volume overload and congenital cardiac defects. Ten patients (40%) had cardiomegaly, which is indicative of persistent volume or pressure overload that is most likely caused by shunt lesions like PDA or ASD. 20% of cases had right ventricle enlargement, and 8% had right atrial (RA) enlargement, both of which are indicative of right-sided cardiac strain. Lung congestion accompanied by elevated pulmonary vascular markings (32%), among other findings. Together, these results show the range of anatomical anomalies and hemodynamic alterations commonly seen in congenital heart disease, especially lesions linked to pulmonary hypertension or left-to-right shunting.

Our cardiomegaly rate closely mirrors that reported by Shanmugam and Basha [17], who found cardiomegaly in 45% of pediatric patients with congenital heart disease based on chest X-ray and echocardiographic correlation. They similarly noted right-sided chamber enlargement in cases of ASD and pulmonary stenosis—diagnoses represented in our cohort as well—supporting the consistency of radiologic patterns across similar populations.

Roy et al. [18] further emphasized the diagnostic value of chest radiographs in congenital heart disease, noting that RV and RA enlargement were particularly common in ASD, PS, and single ventricle physiology. Their findings of cardiomegaly and altered pulmonary vascular markings correspond well with our observations, reinforcing the role of plain radiography as a first-line imaging modality in pediatric cardiology.

Sun et al. [19], in their focused review of right-sided heart disease on radiographs, identified cardiomegaly, right atrial bulging, and prominent pulmonary vasculature as key radiological markers in defects such as ASD and pulmonary hypertension. These indicators were evident in our data as well, highlighting the diagnostic value of X-ray imaging in recognizing subtle manifestations of right heart strain. The presence of these features, even in the absence of advanced imaging, can offer early insights into the underlying structural pathology and help guide further diagnostic or interventional steps. Collectively, these findings support the ongoing utility of chest radiography in the evaluation of congenital heart disease, especially in resource-limited settings where access to advanced imaging may be restricted. The alignment of our radiologic observations with established literature underscores their diagnostic validity and clinical relevance.

Before catheterization ECG recordings showed no signs of myocardial ischemia or change in rhythm distribution. Post-catheterization ECG recordings showed no abnormality in ST segment depression or elevation, T wave inversion, pathological Q wave and no change in rhythm distribution, indicating that the procedure did not induce new arrhythmias or conduction disturbances. This stability underscores the procedural safety of diagnostic and interventional catheterization with respect to cardiac electrophysiology in children. Overall, the unchanged ECG patterns before and after catheterization in our study affirm the safety profile of cardiac catheterization in children. This evidence provides reassurance for clinicians and caregivers and supports the continued use of catheter-based procedures in the diagnosis and management of congenital heart disease without heightened concern for arrhythmogenic complications.

These findings were consistent with those of Kasar et al. [20], who examined a similar pediatric population undergoing diagnostic catheterization and reported no significant emergence of rhythm alterations post-procedure. Their results support the interpretation that catheterization, when properly conducted, carries minimal electrophysiological risk.

Similarly, Walsh et al. [21], while focusing on children with restrictive cardiomyopathy, observed that conduction abnormalities were typically pre-existing and were not exacerbated by catheter-based interventions, reinforcing the notion that catheterization is unlikely to provoke new rhythm disturbances even in patients with complex cardiac pathology.

Our research showed troponin I levels were not statistically significantly correlated with other clinical markers, including ST segment depression or elevation, T wave inversion, pathological Q wave, white blood cells, hemoglobin, platelets, and CRP (c-reactive protein) (p values > 0.05). where as the type of defect and tachycardia are significantly correlated with the Troponin I level.

Electrocardiographic findings before and after catheterization showed that, the heart rate increased from a baseline of 78.4 ± 8.6 bpm to a 89.7 ± 9.5 beat per minute, p = 0.003, in a statistically significant manner. Other parameters such as PR interval, QRS duration, QTc, and (ST segment, T wave, Q wave) did not achieve statistical significance. The mild increases in heart rate following catheterization can be attributed to the myocardial response to procedural stress. This response was insufficient to produce significant changes in other ECG parameters, indicating that cardiac catheterization is generally safe.

When comparing the mean troponin I level before and after catheterization in our study, significant values were found in patients with pulmonary stenosis. However, there was no statistically significant difference in the mean troponin I readings regarding those with atrial septal defect and patent ductus arteriosus. Troponin I had a highly significant statistical value after catheterization, while the mean value before and after catheterization were 0.113±0.01 and 0.131±0.02, respectively.

Troponin I has emerged as a pivotal biomarker for detecting myocardial injury, offering valuable insight into subclinical cardiac stress during pediatric cardiac catheterization. In our study, baseline serum Troponin I levels measured prior to the procedure had a mean of 0.113±0.01ng/mL , remaining within normal limits and indicating an absence of significant pre-existing myocardial injury among the cohort. However, twelve hours post-catheterization, a statistically significant increase was observed, with Troponin I levels rising to a mean of 0.131±0.02 ng/mL ($p < 0.0001$). While the absolute elevation appears numerically modest, it falls within the threshold recognized for subclinical myocardial strain, especially in children with congenital heart disease or associated comorbidities. The variability in post-procedural values suggests differential cardiac responses, potentially influenced by the complexity of structural defects, hemodynamic challenges, or pre-existing oxygenation issues, results are also influenced by the limited number of cases and unequal distribution of patients by congenital heart disease type.

These results were consistent with findings reported in previous research. Hegazy and El-Sheikh [22] similarly documented significant post-catheterization increases in troponin I levels among pediatric patients, including those undergoing both diagnostic and interventional procedures. This supported the notion that catheter-based interventions, regardless of intensity, can provoke measurable myocardial stress.

Our findings align with those of Kannankeril et al. [23], who reported a significant post-procedural rise in cTnI levels in a larger sample of 73 pediatric patients undergoing cardiac catheterization. Their study demonstrated that elevations in troponin I were detectable even in routine cases, supporting its utility as a sensitive biomarker for subclinical myocardial injury. Like our study, they included a wide age range and both diagnostic and interventional procedures, reinforcing the idea that cTnI is effective across a diverse pediatric population.

Similarly, Baysal et al. [24] reported consistent elevations of cTnI following pediatric cardiac catheterization, further corroborating our observations. Their work emphasized that even brief or less invasive procedures could result in detectable myocardial cell injury, as indicated by raised cTnI levels. This supports the hypothesis that cTnI can reflect minor, yet clinically relevant, myocardial damage regardless of procedural duration or intensity, which is consistent with our findings.

Moreover, Yoldaş and Örün [25] presented a more cautious interpretation of elevated cTnI levels in pediatric patients, suggesting that increases may not always indicate pathological myocardial damage. They noted that transient elevations could result from procedural stress, tachycardia, or minor trauma unrelated to ischemic injury. In our study, the observed cTnI elevations were not accompanied by ischemic electrocardiographic changes or clinical symptoms of myocardial ischemia, this perspective highlights the need for careful interpretation. It also underscored the importance of correlating biochemical markers with clinical and electrophysiological findings to avoid overestimating the extent of myocardial injury.

Importantly, in our study, the increase in troponin I occurred despite the absence of clinical signs or electrocardiographic (ECG) abnormalities, reinforcing the utility of cTnI as a marker of subclinical myocardial stress. This suggests that even in the absence of overt symptoms, pediatric cardiac catheterization can induce minor myocardial injury that may not be immediately apparent through conventional clinical monitoring. The mild variability in post-procedure troponin levels may reflect differences in procedural complexity, myocardial resilience, or the presence of cyanotic lesions and chronic hypoxia, all of which are known to affect myocardial susceptibility to stress. Overall, our findings strengthen the role of troponin I in monitoring myocardial response to catheter-based interventions in children and suggest its potential utility in post-procedural risk stratification and care planning.

The study's shortcomings include a small cohort that restricts generalizability, short-term monitoring, the potential for microvascular damage to go unnoticed when ischemia is detected solely by ECG, and the failure to account for procedural factors.

Future research recommendations are crucial for long-term follow-up and multicenter trials.

Conclusion:

When paediatric patients undergo cardiac catheterizations, troponin leak is most linked to pulmonary stenosis intervention procedures. In the absence of clinical or ECG changes, troponin I demonstrated sensitivity as a biomarker for identifying subclinical myocardial stress.

Troponin leak is observed in pediatric patients undergoing cardiac catheterizations most significantly associated with patient underwent balloon pulmonary valvuloplasty to dilate stenotic pulmonary valve and angiography used to visualize the level and extent of stenosis in pulmonary stenosis.

This study helps to identify the types of pediatric cardiac catheterization procedures which are associated with risk for increased troponin I level in our pediatric catheterization unit. Also, further studies are in need with the other interventional procedures for cardiac catheterization.

Conflict of Interest: None.

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Data Availability Statement: The raw data supporting the conclusions of this manuscript will be made available by the authors, without undue reservation, to any qualified researcher.

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