Zagazig University Medical Journal
www.zumj.journals.ekb.eg
https://doi.org/10.21608/zumj.2025.375724.3908 Volume 31, Issue 6, June. 2025
Manuscript ID:ZUMJ-2504-3908
DOI: 10.21608/zumj.2025.375724.3908

ORIGINAL ARTICLE

Evaluation of Cardiac Functions in Children with Lower Respiratory Tract Infections

Khaled Mohamed Salah[1], Dina Tawfeek Sarhan[1], Hanan Samir Ahmed[2], Asmaa Mohamed Mohamed Mohamed[1*], Marwa Lotfy Mohammed Rashad [1]

1 Pediatrics Department, Faculty of Medicine, Zagazig University, Zagazig.
2 Clinical Pathology Department, Faculty of Medicine, Zagazig University.

Corresponding author: Asmaa Mohamed Mohamed Mohamed

Email: badawyasmaa19@gmail.com

Submit Date: 15-04-2025
Accept Date: 01-05-2025

ABSTRACT:

Background: Lower respiratory tract infections (LRTIs) are among the major causes of morbidity and mortality in children under five years. While primarily affecting the lungs, LRTIs can also impair cardiac functions, though more wide cardiac affection, which remain underexplored. This study aimed to assess myocardial involvement in pediatric LRTI cases by evaluating cardiac functions using echocardiography and measuring high-sensitive cardiac troponin T levels.

Methods: This case-control study included 52 children aged 2 months to 5 years. The patient group (n=39) had confirmed LRTIs and was subdivided into 3 groups (n=13 in each): pneumonia (Group I), bronchiolitis (Group II), and pleural effusion (Group III). Group IV included 13 age- and sex-matched healthy children serving as controls. All were tested for hs-Troponin T and C reactive protein, with detailed echocardiographic evaluation of both left and right ventricular functions.

Results: Children with LRTIs had significantly elevated high-sensitive troponin T and CRP levels. Echocardiography revealed reduced left ventricular ejection fraction in bronchiolitis and pleural effusion groups ($p < 0.001$), but with preserver systolic functions. LV diastolic dysfunction affected by decreased A-wave velocity ($p < 0.001$). Right ventricular systolic function, measured by TAPSE, was significantly lower in-patient groups ($p < 0.001$), with reduced TAPSE/RVSP ratios ($p < 0.001$) and elevated systolic pulmonary pressures ($p = 0.001$). TDI of mitral valve showed significant rise of S` and significant decline regarding E/e` among the cases compared to the control ($p < 0.001$). while TDI of tricuspid valve revealed significant decline regarding S` among the cases compared to the control ($p < 0.001$).

Conclusion: LRTIs in young children are associated with subclinical and overt cardiac involvement, affecting both ventricles. Echocardiographic assessment and cardiac biomarkers, especially high-sensitive troponin, can provide valuable insights for early detection of potential cardiac complications in this population.

Keywords: Children; Lower Respiratory Tract Infections; Serum Troponin, Cardiac affection; echocardiographic changes.

INTRODUCTION

Lower respiratory tract infections (LRTIs) refer to infections and inflammatory processes involving the trachea, major bronchi, and lung bronchi, essentially affecting areas below the vocal cords. Common examples include acute bronchitis, bronchiolitis, bronchiectasis, and pneumonia [1–3].

Globally, LRTIs remain one of the most common causes of illness and death, especially in young children. They are responsible for approximately 4 million deaths out of 15

million globally among children under five, as previously reported in earlier epidemiological studies [4,5].

The respiratory and cardiovascular systems are closely linked, functioning as a unified system. Any disturbance in their interaction -such as in the case of LRTIs- can significantly impact heart function. Although LRTIs are primarily pulmonary, evidence shows they can also adversely affect other organs, including the heart. Pulmonary infections may lead to changes in right ventricular function and structure, especially when associated with pulmonary hypertension. One of the earliest cardiac changes seen in these patients is right ventricular diastolic dysfunction, often due to increased afterload. In fact, both the right and left ventricles can be impacted by respiratory illness, altering their size and performance. Pneumonia, in particular, has been linked to later cardiovascular complications such as heart failure, arrhythmias, and even myocardial infarction [6-8].

Accurate assessment of cardiac function requires high-quality imaging and multiple parameters. Point-of-care cardiac ultrasound is especially valuable in critical care settings, representing real-time insights that guide timely clinical decisions. When used by skilled clinicians, echocardiography is an essential tool to detect early cardiac involvement in patients with LRTIs, aiding in diagnosis and modifying appropriate management. It provides crucial data not only for assessing disease severity and treatment strategies but also for predicting prognosis and monitoring treatment response. Tissue Doppler echocardiography (TDE) can be applied to measure myocardial rather than blood velocities. The difference resides in filtering: imaging myocardial velocities requires filtering structures that are moving at high velocity with low scattering power (i.e., the blood) while imaging blood velocity requires filtering out slowly moving and strongly reflecting structures (i.e., the myocardium). Also, it is helpful for assessing systolic and diastolic myocardial function among children [9-10].

In addition to imaging, cardiac biomarkers have become key components of cardiovascular care. Over the past decades, they've transformed how we approach prevention, diagnosis, and management of conditions like acute myocardial infarction and heart failure. More recently, they are helping guide personalized treatment strategies [11,12]. This study aimed to assess myocardial involvement in pediatric LRTI cases by evaluating cardiac function using echocardiography and measuring high-sensitive cardiac troponin T (hs-Troponin T) levels.

METHODS

We performed this case-control study in the Pulmonology and Cardiology Units of the Pediatric Department at Zagazig University Children's Hospital over a period from January 2023 to April 2024 after obtaining approval from the Institutional Review Board (IRB#10768-7-5-2023) and written informed consent from all cases' legal guardians. The research was conducted under the World Medical Association's Code of Ethics (Helsinki Declaration) for human research.

A total of 52 children were enrolled, with their ages ranging from 2 months to 5 years, patients were included who had confirmed lower respiratory tract infection (bronchiolitis, pneumonia, and pleural effusion). LRTI diagnosis was confirmed based on clinical presentation (e.g., cough, tachypnea, wheezing), physical examination, and supportive chest X-ray findings. The exclusion criteria involved children who had pre-existing congenital or having acquired heart disease, as well as those with chronic conditions affecting the lungs, kidneys, liver, or blood. Additionally, children with genetic syndromes or evidence of malnutrition were excluded from the study.

The studied population was categorized based on clinical presentation and chest X-ray findings into four distinct groups. Group I included 13 patients diagnosed with severe pneumonia, presenting with cough, dyspnea, fever, rigors, and malaise, and showing lobar pneumonia or bronchopneumonia on plain radiographs. Group II comprised 13 patients

with bronchiolitis, typically presenting with low-grade or absent fever, a preceding runny nose, progressing to cough, tachypnea, hyperinflation, chest indrawing, and characteristic wheezing. Group III included 13 patients with pleural effusion, who presented with dyspnea, dry cough, and pleuritic chest pain, and demonstrated diagnostic X-ray findings of homogenous opacity obliterating the costophrenic angle and extending to the axilla, or mild pleural opacity. Group IV served as the healthy control group selected from relatives of the patient group and matched according to age and sex.

All children underwent thorough clinical evaluation, including history taking (age, sex, prior chest infections, congenital heart disease, history of previous surgeries), and symptoms such as tachypnea, tachycardia, dyspnea, cough, or feeding difficulties. Physical examination included general assessment and measurement of anthropometric parameters—weight, height, and body mass index (BMI) in addition to the vital signs such as heart rate (HR), respiratory rate (RR), as well as the temperature. Laboratory investigations were performed, including CRP and serum cardiac troponin.

To evaluate biomarkers, a 2 mL sample of venous blood was drawn from each participant after completing the echocardiography. The blood was collected into pro-coagulant tubes and left to clot at room temperature for about 10 to 20 minutes. After clotting, the samples were centrifuged at a speed of 2000 to 3000 rpm for 20 minutes to separate the serum. The serum was then stored at −20°C until it was time for testing. High-sensitivity troponin levels in the serum were assessed utilizing a commercially available Enzyme-Linked Immunosorbent Assay (ELISA) kit, following the instructions provided by the manufacturer. The limit of detection was considered between 1.3 and 3.2 ng/L, while the limit of quantitation fell within the range of 2.9 to 4.9 ng/L. The normal reference range was considered to be less than 0.01 ng/mL, according to the recommended guidelines [13].

Measurement of C-reactive Protein (CRP): Serum C-reactive protein (CRP) levels were measured using the Cobas 6000 c501 modular analyzer (Roche Diagnostics, Germany). Blood samples were collected into plain tubes, allowed to clot at room temperature, and then centrifuged at 3000 rpm for 10 minutes to separate the serum. The analysis was performed using an immunoturbidimetric assay, where CRP in the sample reacts with specific anti-CRP antibodies, forming immune complexes. The resulting turbidity was measured photometrically, and CRP concentrations were automatically calculated by the analyzer based on a calibration curve. All measurements were carried out according to the manufacturer's instructions, with strict adherence to quality control procedures.

All echocardiographic evaluations were performed by a pediatric cardiologist who was blinded to the clinical grouping and laboratory results to minimize interpretation bias. Echocardiographic evaluations were conducted for all participants while they were in the supine position, using the Philips EPIQ CVX system (USA) with transducers of 2–3 MHz and 8 MHz and the patients were connected to the ECG tracing of the echocardiographic machine. All measurements adhered to the standardized guidelines set by the American Society of Echocardiography [14].

Transthoracic echocardiography (TTE): initially conventional routine echocardiography was performed to exclude missed undiagnosed congenital or acquired heart disease. the standard TTE was done, included motion mode (M mode), two-dimensional (2D), pulsed wave (PW), continuous wave(CW) as well as color flow Doppler studies in the, subcostal, apical 4 chamber, apical 5 chambers, parasternal long axis and short axis parasternal views [15].

Assessment of ventricular dimensions and functions were assessed: left ventricular end diastolic diameter [left ventricular internal dimension during diastole (LVIDD)], ejection fraction (EF), and shortening fraction (FS) of left ventricle, right ventricular diameter (RVD). Assessment of ventricular diastolic function:

pulsed wave Doppler of mitral and tricuspid inflow velocities by the measurement of peak early filling (E wave) velocity, peak atrial filling (A wave) velocity, and E/A ratio [15].

Left ventricular (LV) systolic function was assessed using M-model, as LV systolic performance was determined using fractional shortening (FS) and ejection fraction (EF). The FS, which reflected circumferential LV contraction, was considered reduced if <25% and hyperdynamic if >45%. EF, calculated as EF = (SV/EDV) × 100, indicated the proportion of blood ejected per beat, with normal values typically ≥55% [15].

Right Ventricular (RV) Function included RV dimensions & longitudinal right ventricular systolic function was determined utilizing tricuspid annular plane systolic excursion (TAPSE). TAPSE was measured by two-D guided M-mode from the apical four-chamber view with the cursor placed at the free wall of the tricuspid annulus. TAPSE reflects longitudinal myocardial shortening, the main component reflecting the RV contraction in normal hearts. Measurement of TAPSE has provided a chance to assess RV function in a simple, and highly reproducible way. Reduced values indicated systolic dysfunction [15].

CW Doppler echocardiography of the tricuspid flow provides the estimated systolic pulmonary artery pressures (sPAP) [9,10]. Using tricuspid regurgitation jet velocity (V) and simplified Bernoulli equation, the sPAP is best derived from RV systolic pressure (RVSP): RVSP = 4(V)2 + derived RA pressure. The normal estimated systolic pulmonary artery pressure (SPAP) is ≤35 mmHg [17].

Assessment of diastolic functions of both ventricles, using pulsed Doppler recordings were used to detect trans mitral and tricuspid inflow velocities through measuring of Peak early filling velocity (E wave), peak atrial filling velocity (A wave), and E/A ratio, decreased E/A ratio suggested impaired relaxation and decreased diastolic filling and it is a hall mark of diastolic dysfunction [18].

Tissue Doppler imaging (TDI) was assessed the recordings of myocardial velocities in the apical four chamber view with the pulse-wave Doppler sample volume placed at the level of RV lateral tricuspid, and LV lateral mitral annuli after activating the TDI function in the same machine to calculate the peak systolic velocity (S′) which measures the systolic longitudinal function of the RV or the LV, the early diastolic velocity (e′) and late diastolic velocity (a′) both denote the diastolic function of the ventricles. [19]. (E'/A' ratio) is the ratio of mitral early to late annular diastolic velocity was also measured by tissue Doppler imaging. (E/E' ratio): by dividing the peak E wave diastolic velocity by the peak E' velocity, the early E wave diastolic velocity which is influenced by atrial pressure, ventricular relaxation and ventricular systolic pressure [20-22] , the tissue Doppler-derived peak early diastolic E' velocity at mitral or tricuspid annulus is considered as a noninvasive tool for LV relaxation, although its preload dependence [23-25]. So, E/E' ratio served as a non-invasive estimate of LV filling pressures, assisting in the identification of diastolic dysfunction [26].

The myocardial performance index (MPI) is a non-invasive measurement of global ventricular function (systolic and diastolic) independent of geometric assumptions. It can be applied to the RV and the LV. It is defined as the ratio of isovolumic time divided by the ET. MPI = (IVCT + IVRT) / ET [27].

Statistical analysis

Statistical analysis was conducted using IBM SPSS Statistics version 28 (IBM Corp., Armonk, NY, USA). Categorical data were expressed as frequencies and compared using the chi-square test. For continuous variables, normality was assessed using the Shapiro-Wilk test. Data were presented as mean ± standard deviation for normally distributed variables, or as median with interquartile range (IQR) when the distribution was non-normal. Comparisons between groups were performed using one-way ANOVA for normally distributed data and the Kruskal-Wallis test for non-parametric data. When a significant difference was identified, post hoc analysis was carried out using Tukey's HSD test. Correlations between variables were

analyzed using Spearman's rank correlation coefficient. A p-value of less than 0.05 was considered statistically significant, with values below 0.001 indicating a highly significant result.

RESULTS

The children's age ranged from two months to five years. Table 1 shows that the study groups were well-matched as regards the age (p = 0.83) and sex distribution (p = 0.418), without any statistically significant differences.

Serum troponin levels were significantly higher among all patients groups (Groups I, II and III) than the control group (Group IV) (p < 0.001). Additionally, a significant higher in Group I than Group III (Table 2). Similarly, highly significant decline in Group IV than all the patient groups.

There is statistically significant increase regarding CRP among the cases compared to the control. On doing pairwise comparison, difference is significant lower in Group IV (healthy control group) than each other group.

The best cut-off of serum troponin in prediction of cardiac affection in LRTI is ≥10.55 with area under curve 0.879, sensitivity 82.1%, specificity 84.6%, positive, negative predictive value and overall accuracy are 94.1%, 61.1% and 82.7% respectively (p<0.001) (Supplementary Table 1, Supplementary Figure 1).

1. Left Ventricular Function

1.1. Systolic Function (FS and EF)

The LV fractional shortening (FS) was significantly higher among the pneumonia group than both the effusion and control groups (p = 0.015). Post hoc analysis revealed significant differences between Group I and both Group III (p = 0.013) and Group IV (p = 0.03). In contrast, LV ejection fraction (EF) showed a significant reduction among the patient groups, especially in Groups II and III, when compared to the control group (p < 0.001). Pairwise comparisons also revealed significant differences between Group III and Group IV (p < 0.001), as well as between Group II and Group IV (p = 0.02) (Table 3).

There are no statistically significant differences between the studied groups regarding occurrence of MR. While there is statistically significant decline regarding occurrence of AR, or change in LA, and AO diameter among the cases compared to the control.

1.2. Diastolic Function – Trans-Mitral Inflow Profile

No significant variations were revealed among the groups in terms of LV E velocity or the LV E/A ratio. However, LV A-wave velocity was significantly lower among the patient groups than the controls (p < 0.001). Post hoc comparisons showed significant reductions in Group III vs Group IV (p < 0.001) and in Group II vs Group IV (p < 0.001) (Table 4). Tissue Doppler of the Mitral Valve, There is statistically significant rise regarding S` among the cases compared to the control (p < 0.001),with no statistically significant differences between the studied groups regarding E`.

2. Right Ventricular Function

2.1. Systolic Parameters – TAPSE and Pulmonary Arterial Pressures

The TAPSE and TAPSE/RVSP ratios were significantly lower among the 3 patient groups than the control group (p < 0.001 for both). Post hoc analysis confirmed significantly lower TAPSE and TAPSE/RVSP values in each patient group than the controls. Specifically, TAPSE was significantly lower in Group III versus Group IV (p < 0.001) and in Group II versus Group IV (p = 0.001). Similarly, TAPSE/RVSP ratios were significantly decreased in both Group III and Group II compared to Group IV (p < 0.001 for both comparisons) (Table 3).

Right ventricular diameter (RVD) was significantly higher in the patient groups compared to the control group (p = 0.002). Post hoc comparisons showed a statistically significant difference between Group III and Group IV (p = 0.007). Furthermore, pulmonary regurgitation pressure (PR) showed a significant rise among the patient groups (p = 0.001), especially between Group III and IV (p = 0.001).

2.2. Diastolic Function – Tricuspid Valve Inflow

No significant differences were detected in tricuspid E-wave velocities across groups. However, significant decreases were found in tricuspid A-wave (p = 0.009) and RV E/A ratio (p = 0.002), regarding TDE, there is statistically significant decline regarding S` among the cases compared to the control. with significant reductions in Group III compared to the control group (Table 6).

Among pneumonia group, there is statistically significant positive correlation between troponin and LV MPI, also There is statistically significant negative correlation between TAPSE and troponin. Among bronchiolitis group, there is statistically significant positive correlation between troponin, and T e`. Within pleural effusion group, there is significant positive correlation between troponin and all of e`, E/e`, E`/A` and T E/e`(Table 6).

3. Combined Ventricular Function – Myocardial Performance Index (MPI)

No statistically significant differences were observed in either left or right ventricular MPI among the studied groups (p = 0.984 and p = 0.605, respectively), as presented in Table 7.

Table (1): Demographic data of the studied groups.

	Group I	Group II	Group III	Group IV	$\chi 2$	P
	N=13 (%)	N=13 (%)	N=13 (%)	N=13 (%)		
Gender Female Male	7 (53.8%) 6 (46.2%)	4 (30.8%) 9 (69.2%)	4 (30.8%) 9 (69.2%)	7 (53.8%) 6 (46.2%)	2.836	0.418
	Median (IQR)	Median (IQR)	Median (IQR)	Median (IQR)	KW	P
Age (years)	3(2 – 4.5)	3.2(1 – 4)	3.9(2.05 – 4.2)	3.5(1.7 – 4.2)	0.88	0.83

χ^2: Chi-square test; KW: Kruskal-Wallis test; IQR: Interquartile range; N: Number of patients

Table (2): Comparison between the studied groups regarding laboratory data (troponin and CRP):

	Group I	Group II	Group III	Group IV	KW	P
	Median (IQR)	Median (IQR)	Median (IQR)	Median (IQR)		
Troponin (ng/mL)	30 (16 – 68.5)	40 (9.17–59.1)	20.33 (11.3 –51)	8 (5 – 10)	17.135	<0.001**
Pairwise	P1 0.622	P2 0.751	P3 0.003*	P4 <0.001**	P5 <0.001**	P6 0.001**
CRP (mg/L)	150 (71 – 410)	122 (32.5 – 395)	50 (16 – 225)	5 (4 – 6)	27.681	<0.001**
Pairwise	P2 0.641	P1 0.288	P3 0.001**	P4 0.126	P5 <0.001**	P6 <0.001**

KW: Kruskal-Wallis test; IQR: Interquartile range; CRP: C-reactive protein; P1: Group I vs II; P2: Group I vs III; P3: Group I vs IV; P4: Group II vs III; P5: Group II vs IV; P6: Group III vs IV.

Table (3): Comparison between the studied groups regarding conventional 2D echoparameters of both ventricles:

	Parameter	Group I	Group II	Group III	Group IV	F	P
LV		Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD		
	MR (mmHg)	23.23 ± 8.5	23.69 ± 5.78	19.92 ± 4.23	23.38 ± 7.18	0.927	0.435
	AR (mmHg)	15.77 ± 4.73	17.31 ± 4.46	13.92 ± 3.62	23.08 ± 4.19	11.156	<0.001**
	LV. FS (%)	38.23 ± 3.31	36.0 ± 2.21	35.46 ± 1.56	35.75 ± 1.16	4.259	0.015*
	Post hoc	P1 0.062	P2 0.925	P3 0.988	P4 0.013*	P5 0.991	P6 0.03*
	LV. EF (%)	69.78 ± 4.06	66.95 ± 2.23	66.33 ± 1.58	71.26 ± 3.09	8.413	<0.001**
	Post hoc	P1 0.075	P2 0.949	P3 <0.001**	P4 0.02*	P5 0.002*	P6 0.565
RV	MPA (mm)	15.39 ± 2.23	16.25 ± 1.35	16.42 ± 1.48	18.19 ± 1.48	3.458	0.021*
	Post hoc	P1 0.762	P2 0.998	P3 0.198	P4 0.653	P5 0.139	P6 0.014*
	TR (mmHg)	27.77 ± 6.86	30.69 ± 3.12	28.77 ± 2.83	23.46 ± 6.57	4.509	0.007*
	Post hoc	P1 0.484	P2 0.781	P3 0.057	P4 0.961	P5 0.005*	P6 0.163
	PASP(mmHg)	37.77 ± 6.86	40.69 ± 3.12	38.77 ± 2.83	34.62 ± 7.19	2.877	0.046*
	Post hoc	P1 0.517	P2 0.8	P3 0.217	P4 0.965	P5 0.03*	P6 0.452
	PR(mmHg)	13.69 ± 2.56	14.23 ± 2.01	14.92 ± 2.43	11.08 ± 2.78	6.053	0.001**
	Post hoc	P1 0.944	P2 0.89	P3 0.001**	P4 0.583	P5 0.011*	P6 0.045*
	RVD (mm)	18.23 ± 3.14	18.39 ± 2.63	17.5 ± 3.66	23.08 ± 4.77	5.938	0.002*
	Post hoc	P1 >0.999	P2 0.948	P3 0.007*	P4 0.97	P5 0.01*	P6 0.008*
	TAPSE (mm)	16.67 ± 1.67	16.49 ± 1.79	16.4 ± 1.54	18.52 ± 2.56	7.896	<0.001**
	Post hoc	P1 0.995	P2 0.999	P3 <0.001**	P4 0.984	P5 0.001**	P6 0.002*
	TAPSE/RVSP	0.45 ± 0.1	0.41 ± 0.07	0.42 ± 0.07	0.59 ± 0.15	8.955	<0.001**
	Post hoc	P1 0.648	P2 0.993	P3 <0.001**	P4 0.807	P5 <0.001**	P6 0.007*

F: One way ANOVA test; SD: Standard deviation; LV FS: Left ventricular fractional shortening; LV EF: Left ventricular ejection fraction;
MPA: Main pulmonary artery diameter; TR: Tricuspid regurgitation pressure gradient; PASP: Pulmonary artery systolic pressure; PR: Pulmonary regurgitation pressure gradient; RVD: Right ventricular diameter; TAPSE: Tricuspid annular plane systolic excursion; RVSP: Right ventricular systolic pressure; P1: Group I vs II; P2: Group I vs III; P3: Group I vs IV; P4: Group II vs III; P5: Group II vs IV; P6: Group III vs IV.

Table (4): Comparison between the studied groups regarding diastolic functions of both ventricles (Trans mitral and Tricuspid Inflow Profile)

		Group I	Group II	Group III	Group IV	F	P
		Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD		
MV	LV. A (m/s)	62.05 ± 7.55	57.74 ± 8.15	60.52 ± 7.78	63.19 ± 5.73	19.089	<0.001**
	LV. E (m/s)	83.2 ± 8.47	88.02 ± 3.22	88.99 ± 1.92	101.69 ± 9.16	1.334	0.274
	Post hoc	P1 0.247	P2 0.982	P3 <0.001**	P4 0.121	P5 <0.001**	P6 <0.001**
	LV. E/A	1.38 ± 0.28	1.54 ± 0.29	1.47 ± 0.23	1.61 ± 0.19	1.998	0.128
	Post hoc	P1 0.778	P2 0.469	P3 <0.001**	P4 0.089	P5 0.055	P6 0.352
T. V	RV. A (m/ sec)	79.41 ± 12.94	82.05 ± 19.8	69.25 ± 14.06	84.8 ± 17.22	4.29	0.009*
	RV. E (m/ sec)	54.46 ± 19.05	66.52 ± 11.94	67.17 ± 11.51	52.85 ± 8.42	2.272	0.092
	RV. E/A	1.4 ± 0.32	1.28 ± 0.36	1.1 ± 0.35	1.62 ± 0.25	5.854	0.002*
	Post hoc	P_1 0.778	P_2 0.469	P_3 <0.001**	P_4 0.089	P_5 0.055	P_6 0.352

F: One way ANOVA test; SD: Standard deviation; LV E: Left ventricular early diastolic filling velocity; LV A: Left ventricular late diastolic filling velocity; LV E/A: Ratio of early to late diastolic filling velocities;
TV A: Tricuspid valve late diastolic (A-wave) velocity; TV E: Tricuspid valve early diastolic (E-wave) velocity; TV E/A: Ratio of early to late diastolic filling velocities; P1: Group I vs II; P2: Group I vs III; P3: Group I vs IV; P4: Group II vs III; P5: Group II vs IV; P6: Group III vs IV.

Table (5): Comparison between the studied groups regarding tissue Doppler of both mitral and tricuspid valve and ventricular MPI

		Group I	Group II	Group III	Group IV	F	P
MV		Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD		
	S` (M/ sec)	10.28 ± 3.0	8.56 ± 2.31	7.29 ± 1.61	7.86 ± 1.5	4.359	0.007*
	Posthoc	P1 0.201	P2 0.848	P3 0.913	P4 0.465	P5 0.006*	P6 0.034*
	e` (M/ sec)	14.02 ± 5.18	14.62 ± 3.58	12.13 ± 2.79	14.78 ± 2.66	1.406	0.252
	a` (M/ sec)	7.8(5.55 – 8.3)	7.8(4.79 – 14.4)	4.79(4 – 4.79)	7.3(6.8 – 9.68)	8.67	0.034*
	Pairwise	P1 0.819	P2 0.033*	P3 0.009*	P4 0.018*	P5 0.624	P6 0.794
	E/e`	6.0 ± 1.55	6.79 ± 1.62	7.75 ± 1.4	7.04 ± 1.21	3.213	0.031*
	Posthoc	P1 0.522	P2 0.336	P3 0.597	P4 0.018*	P5 0.97	P6 0.277
	E`/A`	2.19 ± 0.6	2.38 ± 0.38	2.47 ± 0.51	1.84 ± 0.46	4.415	0.011*
	Posthoc	P1 0.754	P2 0.964	P3 0.011**	P4 0.464	P5 0.038*	P6 0.292
		Median (IQR)	Median (IQR)	Median (IQR)	Median (IQR)	KW	P
TV	S (M/ sec)	9.79 ± 1.8	9.11 ± 1.89	8.43 ± 1.24	11.54 ± 2.72	5.848	0.002*
	Posthoc	P1 0.816	P2 0.826	P3 0.001**	P4 0.316	P5 0.016*	P6 0.127
	e` (M/ sec)	12.01 ± 1.38	11.91 ± 1.16	11.65 ± 1.12	18.74 ± 5.19	19.636	<0.001**
	Posthoc	P1 >0.999	P2 0.996	P3 <0.001**	P4 0.988	P5 <0.001**	P6 <0.001**
	a` (M/ sec)	7.91 ± 1.51	7.43 ± 1.12	6.63 ± 0.92	10.92 ± 3.35	11.705	<0.001**
	Posthoc	P1 0.924	P2 0.735	P3 <0.001**	P4 0.361	P5 <0.001**	P6 <0.001**
	E/e`	6.39 ± 1.14	6.92 ± 1.51	6.01 ± 1.4	4.73 ± 1.15	6.577	<0.001**
	Posthoc	P1 0.74	P2 0.301	P3 <0.001**	P4 0.361	P5 <0.001**	P6 0.002*
	E`/A`	1.53 ± 0.31	1.63 ± 0.28	1.79 ± 0.2	1.75 ± 0.28	2.571	0.067
	LV-MPI	0.44 ± 0.07	0.45 ± 0.09	0.44 ± 0.09	0.45 ± 0.14	0.051	0.984
	RV-MPI	0.49 ± 0.09	0.45 ± 0.11	0.44 ± 0.08	0.44 ± 0.1	0.62	0.605

S`: Peak velocity of systolic mitral by doppler A`: late diastolic wave by tissue Doppler, E: early diastolic wave by Doppler, e`: early diastolic wave by tissue doppler a`: late diastolic wave M/sec: meter per second performance index, *:KW Kruskal Wallis test F One way ANOVA test LV-MPI: Left ventricular myocardial performance index; RV-MPI: Right ventricular myocardial performance index.

P1: Group I vs II; P2: Group I vs III; P3: Group I vs IV; P4: Group II vs III; P5: Group II vs IV; P6: Group III vs IV.

Table (6): Correlation between serum troponin and the Echo parameters among the studied groups

	Troponin							
	Group I		Group II		Group III		Group IV	
	R	p	R	P	R	P	R	P
MR(mmHg)	-0.336	0.219	-0.34	0.256	-0.378	0.203	0.311	0.302
AR(mmHg)	-0.215	0.48	-0.279	0.355	-0.164	0.592	0.412	0.162
LA (mm)	-0.011	0.971	-0.048	0.877	0.577	0.039*	-0.079	0.796
AO (mm)	-0.249	0.413	-0.154	0.615	-0.095	0.758	0.518	0.07
MPA	-0.219	0.473	-0.029	0.925	0.746	0.003*	-0.046	0.883
TR (mmHg)	0.421	0.152	0.032	0.917	-0.051	0.867	0.053	0.864
PASP(mmHg)	0.421	0.152	0.032	0.917	-0.051	0.867	0.084	0.786
PR(mmHg)	0.215	0.482	-0.221	0.496	-0.298	0.322	-0.061	0.842
RVD (mm)	-0.309	0.304	-0.723	0.005*	0.566	0.143	-0.205	0.627
TAPSE (mm)	-0.627	0.022*	-0.277	0.36	-0.581	0.037*	0.028	0.928
TAPSE/RVSP	-0.705	0.007*	0.042	0.891	-0.142	0.642	0.105	0.733
LV. FS (%)	-0.598	0.031*	-0.067	0.828	0.762	0.002*	0.118	0.7
LV. EF (%)	-0.598	0.031*	0.332	0.268	0.762	0.002*	0.118	0.7
LV. A	-0.457	0.116	-0.465	0.109	-0.667	0.013*	-0.191	0.531
LV. E	0.711	0.006*	0.238	0.434	-0.762	0.002*	-0.118	0.7
LV. E/A	0.67	0.012*	0.038	0.903	0.54	0.057	0.048	0.877
RV. A	0.27	0.372	0.155	0.612	-0.835	<0.001**	-0.282	0.35
RV. E	0.276	0.361	-0.055	0.859	0.683	0.01*	0.355	0.234
RV. E/A	-0.257	0.397	-0.34	0.256	0.789	<0.001**	0.334	0.265

r Spearman rank correlation coefficient *p<0.05 is statistically significant **p≤0.001 is statistically highly significant

Table (7): Performance of troponin in prediction of abnormal Echo in children with LRTI (TAPSE<18)

Cutoff	AUC	Sensitivity	Specificity	PPV	NPV	Accuracy	p
≥12.425	0.833	82.4%	88.9%	93.3%	72.7%	84.6%	<0.001**

AUC area under curve PPV positive predictive value NPV negative predictive value **p≤0.001 is statistically highly significant

DISCUSSION

The LRTI is considered the most frequently encountered infectious disease in children and remains a major global health burden. They are consistently reported as leading infectious causes of pediatric mortality and continue to significantly affect the health of children under five years of age worldwide [6].

The cardiopulmonary systems work in close synchrony, and disruptions in their interaction can significantly impact cardiac function. It is now well recognized that cardiovascular complications are not uncommon in LRTIs, particularly in community-acquired pneumonia (CAP), with up to 30% of hospitalized cases showing some form of cardiac involvement [7]. Although the lungs are typically the primary focus in such conditions, attention to cardiac effects especially right ventricular (RV) involvement and pulmonary hypertension (PH) is vital for appropriate risk stratification and management [8].

In our study, which involved 52 children under the age of five, we aimed to assess the cardiovascular implications of LRTIs using high-sensitive troponin levels and echocardiography. Our findings demonstrated a significant rise in serum troponin among LRTI cases compared to controls, particularly in pneumonia and bronchiolitis subgroups. This aligns with prior findings by Hegazy et al. [13], who observed elevated troponin T levels in all studied children with acute chest infections.

We found the best cut-off of serum troponin in prediction of cardiac affection in LRTI is ≥10.55 with area under curve 0.879, sensitivity 82.1%, specificity 84.6%, positive, negative predictive value and overall accuracy are 94.1%, 61.1% and 82.7% respectively ($p<0.001$) this level was notably higher than documented in children with septic shock [3.1 ± 2.6 ng/ml (0.01–9.80 ng/ml) reported by Gürkan et al. [28].

.The discrepancy in cTnI levels between the two studies may be attributed to differences in the underlying pathophysiology of myocardial involvement in LRTIs versus septic shock. While septic shock often leads to myocardial depression through inflammatory mediators and circulatory compromise, LRTIs may cause cardiac strain due to hypoxia and increased pulmonary pressures, potentially resulting in more pronounced myocardial injury and higher cTnI release. Additionally, variations in study design, patient populations, and timing of biomarker measurement could contribute to the observed differences. Further research is warranted to elucidate the mechanisms underlying elevated cTnI levels in LRTIs and to validate its utility as a marker for cardiac involvement in this context.

CRP, an acute-phase reactant, was also significantly elevated in our LRTI groups compared to controls. These findings are supported by studies such as Gupta et al. [29], who reported higher CRP in cases of RSV-related bronchiolitis and pneumonia, and Williams et al. [30], who showed similar trends in children with severe pneumonia. The rise in troponin may reflect not only myocardial strain but also systemic inflammation and hypoxia, consistent with known mechanisms in acute cardiac injury during pneumonia [31].

Echocardiographic assessment revealed a significant reduction in left ventricular ejection fraction (EF) in some patient subgroups, particularly those with pleural effusion, despite preserved systolic function. Our data is supported by Massolo et al. [32], who found reduced LV function in infants hospitalized for bronchiolitis. However, contrary findings were reported by Jarallah et al. [31], who found no significant LV changes in children with various LRTI forms.

Regarding diastolic function, we noted a significant decrease in LV A-wave velocities, suggesting impaired myocardial relaxation. This is consistent with Formenti et al. [33], who reported left ventricular diastolic dysfunction in a third of patients with ARDS. TDI of mitral valve provided further insights, revealing increased S′ wave velocities and elevated E′/A′ ratios, indicative of myocardial stiffness and restrictive filling. TDI of tricuspid valve

revealed significant decline regarding S` among the cases compared to the control. Similar findings have been described in bronchiolitis-associated cardiac involvement by Rossi et al. [34].

For right ventricular function, TAPSE, a key parameter of RV systolic performance—was significantly reduced in LRTI cases compared to controls. This supports the observations of Hameed et al. [35], who emphasized TAPSE's utility in assessing RV dysfunction in pulmonary hypertension and related pathologies. Our study confirms its value in pediatric LRTI as well, echoing findings by Biteker et al. [36], who showed TAPSE was strongly associated with outcomes in patients hospitalized for CAP. Although cardiac complications in adult CAP patients are well-documented, caution is warranted in directly extrapolating these findings to pediatric cases. Nonetheless, they provide important pathophysiological insights that may be relevant in severe pediatric LRTIs.

RV pressures (e.g., PASP, TR) were elevated in patient groups, especially in bronchiolitis cases, indicating pulmonary hypertension. We also observed significantly lower MPA values in patients versus controls, which may reflect increased pulmonary vascular resistance. Similar observations were made by Lazzeri et al. [37] in COVID-19-related respiratory compromise.

Concerning RV diastolic function, the tricuspid inflow pattern showed significant reductions in A-wave velocity and E/A ratio, especially in the pleural effusion group. These results are in agreement with those of Chotalia et al. [38], who linked RV dysfunction with poor outcomes in pediatric ARDS. In contrast, Messina et al. [39] reported relatively preserved RV function in mild respiratory infections.

Our TDI assessment of the tricuspid valve further revealed significant declines in S′, E′, and A′ waves in LRTI groups, confirming early and subclinical changes in RV performance. These changes were more pronounced in bronchiolitis and effusion subgroups, suggesting that even non-severe LRTIs can lead to detectable cardiac effects.

The cardiac involvement observed in children with LRTIs can be attributed to several intertwined pathophysiological mechanisms. First, systemic inflammation and the cytokine storm triggered by severe infections result in elevated circulating levels of pro-inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-1β (IL-1β). These mediators contribute to myocardial depression, impair calcium handling, and promote oxidative stress, leading to subclinical or overt myocardial dysfunction. Additionally, cytokine-induced endothelial activation and increased vascular permeability can exacerbate myocardial strain through volume shifts and pulmonary hypertension.

Second, certain viral etiologies of LRTIs, such as respiratory syncytial virus (RSV), adenovirus, and influenza, have been shown to cause direct myocardial invasion, leading to viral myocarditis. This direct cytopathic effect is accompanied by local inflammatory infiltration and myocyte necrosis, further impairing myocardial contractility and electrical stability. In these cases, elevated cardiac biomarkers like troponin may reflect true myocardial injury rather than mere strain, especially in the absence of hypoxia or shock [31,33, 40].

To our knowledge, this is among the new studies to comprehensively evaluate both right and left ventricular function in children with LRTIs using echocardiography and serum troponin levels. Echocardiographic evaluation of both ventricles should be performed in all children with LRTIs to assess cardiac function and exclude undiagnosed congenital heart disease. High-sensitive troponin testing is recommended, especially in severe cases, to predict potential complications. Follow-up using tissue Doppler echocardiography is advised in patients with acute cardiac abnormalities to monitor recovery.

This study had some limitations that warrant consideration. First, the relatively small sample size may limit the broader applicability of the

results. Second, being a single-center study, there is a possibility of selection bias influencing the findings. Third, while echocardiography and serum biomarkers offered important diagnostic insights, cardiac MRI—which is considered the gold standard for detailed cardiac evaluation—was not utilized due to feasibility constraints. Additionally, the lack of longitudinal follow-up limited our ability to determine the reversibility or persistence of cardiac changes and outcome. Future studies with serial echocardiographic and troponin measurements are recommended to monitor cardiac recovery over time.

CONCLUSION

This study highlights that lower respiratory tract infections in children under five are associated with biventricular cardiac involvement. Right ventricular systolic dysfunction, shown by reduced TAPSE elevated sPAP and peak S wave significantly and RV diastolic dysfunction was detected by significantly reduced TV. E/A and E/e` ratio. The left ventricular changes, including decreased EF in patient groups with preserved systolic functions, LV diastolic dysfunction noticed in decreased MV E`/A` ratio and E/e` ratio significantly. High-sensitive cardiac troponin proved its value in detection of cardiac involvement in LRTI and its significant correlation to variant cardiac functions and its elevation together with elevated CRP levels reflected systemic inflammation.

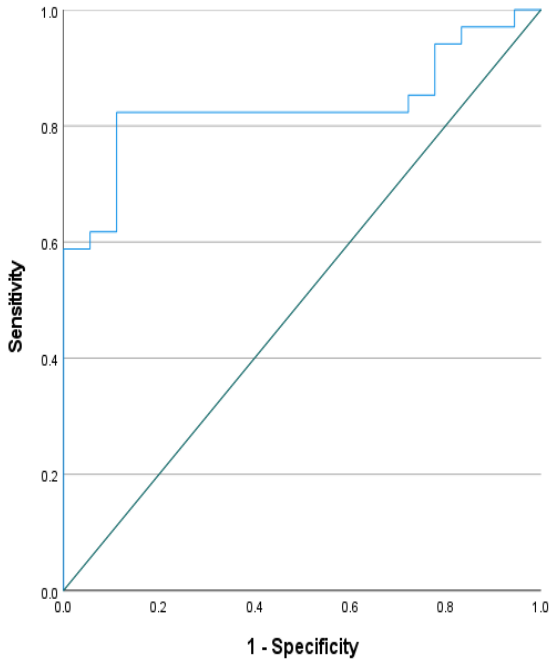
Conflict of interest: None.

Financial Disclosures: None.

REFERENCES

1. Zhong B, Sun SL, Du JT, Li SB, Xu Y, Tang JJ, et al. Risk factors for lower respiratory tract infection in children with tracheobronchial foreign body aspiration. *Medicine (Baltimore)* 2019;98(3):e14655.
2. Buchan BW, Windham S, Balada-Llasat JM, Leber A, Harrington A, Relich R, et al. Practical comparison of the BioFire FilmArray Pneumonia Panel to routine diagnostic methods and potential impact on antimicrobial stewardship. *J Clin Microbiol* 2020;58(7):e00135-20.
3. Ali Mahjoub Saeed E, Awadalla H. Knowledge, attitude and practice among mothers of under-five children about acute lower respiratory tract infections in Khartoum urban area, Sudan. *J Environ Sci Public Health* 2020;4:126–35.
4. Bradley BT, Bryan A. Emerging respiratory infections: the infectious disease pathology of SARS, MERS, pandemic influenza, and Legionella. *Semin Diagn Pathol* 2019;36(3):152–9.
5. Seidu AA, Dickson KS, Ahinkorah BO, Agbaglo E, Yaya S, Adu C, et al. Prevalence and determinants of acute lower respiratory infections among children under-five years in sub–Saharan Africa: evidence from demographic and health surveys. *SSM Popul Health* 2019;8:100443.
6. De-Paula CR, Lopes N, Barbosa E, Carmo JRM, Pereira FL, Silva KS, et al. Echocardiographic assessment of ventricular function in young patients with asthma. Arq Bras Cardiol 2018;110(3):225–32.
7. Allinson JP, Chaturvedi N, Wong A, Babu H, Hardy R, Kuh D, et al. Early childhood lower respiratory tract infection and premature adult death from respiratory disease in Great Britain: a national birth cohort study. Lancet 2023;401(10380):1183–93.
8. Fisher JP, Zera T, Paton JFR. Respiratory–cardiovascular interactions. In: Aminoff MJ, Boller F, Swaab DF, eds. Handbook of Clinical Neurology. Vol 190. Elsevier; 2022:279–308.
9. Counseller Q, Aboelkassem Y. Recent technologies in cardiac imaging. Front Med Technol 2023;4:984492.
10. Rudski LG, Lai WW, Afilalo J, Hua L, Handschumacher MD, Chandrasekaran K, et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. J Am Soc Echocardiogr 2010;23(7):685–713.
11. Mair J. Cardiac biomarkers. Dis Markers 2015;2015:1–3.
12. Jaffe AS, Wu AHB, Moe GW, Apple FS, Chew DP, Mills NL, et al. Single troponin measurement to rule out myocardial infarction. J Am Coll Cardiol 2023;82(1):60–9.
13. Hegazy LAG, Mossad H, Wahba N, Ishak S. Troponin T level in children with acute chest diseases and respiratory distress. Egypt J Chest Dis Tuberc 2020;69(4):585–91.
14. Zacchigna S, Paldino A, Falcão-Pires I, Puzzi L, Zentilin L, Kreutzer C, et al. Towards standardization of echocardiography for the evaluation of left ventricular function in adult rodents: a position paper of the ESC Working Group on Myocardial Function. Cardiovasc Res 2021;117(1):43–59.
15. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pelikka PA, et al. Recommendations for chamber quantifications: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the

European Society of Cardiology. J Am Soc Echocardiogr 2005;18:1440–63.
16. Berger M, Haimowitz A, Van Tosh A, Berdoff RL, Goldberg E. Quantitative assessment of pulmonary hypertension in patients with tricuspid regurgitation using continuous wave Doppler ultrasound. J Am Coll Cardiol 1985;6(2):359–65.
17. Jaffe AS, Wu AHB, Moe GW, Apple FS, Chew DP, Mills NL, et al. Single troponin measurement to rule out myocardial infarction. J Am Coll Cardiol 2023;82(1):60–9.
18. Amer MZ, Abd-Alaziz AA. Tissue Doppler assessment of left ventricular diastolic function in rheumatic mitral stenosis. Egypt Heart J 2011;63(3–4):161–7.
19. Sanfilippo F, Scolletta S, Morelli A, Vieillard-Baron A. Practical approach to diastolic dysfunction in light of the new guidelines and clinical applications in the operating room and in the intensive care. Ann Intensive Care 2018;8(1):100.
20. Ishida Y, Meisner JS, Tsujioka K, Gallo JI, Yoran C, Frater RW, et al. Left ventricular filling dynamics: influence of left ventricular relaxation and left atrial pressure. Circulation 1986;74:187–96.
21. Choong CY, Abascal VM, Thomas JD, Guerrero JL, McGlew S, Weyman AE. Combined influence of ventricular loading and relaxation on the transmitral flow velocity profile in dogs measured by Doppler echocardiography. Circulation 1988;78:672–83.
22. Thomas JD, Choong CY, Flachskampf FA, Weyman AE. Analysis of the early transmitral Doppler velocity curve: effect of primary physiologic changes and compensatory preload adjustment. J Am Coll Cardiol 1990;16:644–55.
23. Sohn DW, Chai IH, Lee DJ, Kim HC, Kim HS, Oh BH, et al. Assessment of mitral annulus velocity by Doppler tissue imaging in the evaluation of left ventricular diastolic function. J Am Coll Cardiol 1997;30:474–80.
24. Ommen SR, Nishimura RA, Appleton CP, Miller FA, Oh JK, Redfield MM, et al. Clinical utility of Doppler echocardiography and tissue Doppler imaging in the estimation of left ventricular filling pressures: a comparative simultaneous Doppler-catheterization study. Circulation 2000;102:1788–94.
25. Nagueh SF, Sun H, Kopelen HA, Middleton KJ, Khoury DS. Hemodynamic determinants of the mitral annulus diastolic velocities by tissue Doppler. J Am Coll Cardiol 2001;37:278–85.
26. Arques S. Usefulness of combined Doppler indexes in predicting left ventricular filling pressures in patients with acute heart failure. Am J Cardiol 2003;92:649–50.
27. Tei C, Dujardin KS, Hodge DO, Bailey KR, McGoon MD, Tajik AJ, et al. Doppler echocardiographic index for assessment of global right ventricular function. J Am Soc Echocardiogr 1996;9(6):838–47.
28. Gurkan F, Alkaya A, Ece A, Haspolat K, Bosnak M, Bilici M, et al. Cardiac troponin-I as a marker of myocardial dysfunction in children with septic shock. Swiss Med Wkly 2004;134(39-40):593–9.
29. Gupta J, Kumar DS, Karthikeyan S, Arora N, Harshitha R, Sharma M, et al. A hospital-based analytical observational study to assess CRP levels in children with acute bronchiolitis. Int J Pediatr Neonatol 2023;5(1):37–41.
30. Williams DJ, Zhu Y, Grijalva CG, Self WH, Ampofo K, Pavia AT, et al. Predicting severe pneumonia outcomes in children. Pediatrics 2016;138(4):e20161019.
31. Khatib M, Elbaz-Greener G, Nitzan O, Gilon D, Shacham Y, Meitus A, et al. Unmasking myocardial dysfunction in patients hospitalized for community-acquired pneumonia using a 4-chamber 3-dimensional volume/strain analysis. J Digit Imaging 2022;35(6):1654–61.
32. Massolo AC, Cantone GV, Stronati M, Boccuzzi E, Pugnaloni F, Zallocco F, et al. Myocardial strain on admission predicts disease severity in infants hospitalized for bronchiolitis. Pediatr Pulmonol 2020;55(5):1217–22.
33. Formenti P, Coppola S, Massironi L, Castelli G, Gatti S, Chiurazzi C, et al. Left ventricular diastolic dysfunction in ARDS patients. J Clin Med 2022;11(20):5998.
34. Rossi ML, Hadley SM, Randanne PC, Escobar-Diaz MC. Cardiac function in bronchiolitis: not only a right ventricle matter. Pediatr Pulmonol 2023;58(1):288–96.
35. Hameed A, Condliffe R, Swift AJ, Wild JM, Garg P. Assessment of right ventricular function—a state of the art. Curr Heart Fail Rep 2023;20(3):194–207.
36. Biteker FS, Başaran Ö, Doğan V, Duman D, Bozkurt B, Biteker M, et al. Prognostic value of transthoracic echocardiography and biomarkers of cardiac dysfunction in community-acquired pneumonia. Clin Microbiol Infect 2016;22(11):1006.e1–6.
37. Lazzeri C, Bonizzoli M, Batacchi S, Peris A. Echocardiographic assessment of the right ventricle in COVID-related acute respiratory syndrome. Intern Emerg Med 2021;16(4):1051–6.
38. Chotalia M, Ali M, Alderman JE, Patel JM, Parekh D, Bangash MN, et al. Right ventricular dysfunction and its association with mortality in COVID-19 acute respiratory distress syndrome. Crit Care Med 2021;49(11):1757–68.
39. Messina A, Sanfilippo F, Milani A, Di Falco D, Bianchi FP, Di Masi D, et al. COVID-19-related echocardiographic patterns of cardiovascular dysfunction in critically ill patients: a systematic review of the current literature. J Crit Care 2021;65:26–35.
40. Okuyan O, Elgormus Y, Dumur S, Inan S, Senel HA, Sefil F, et al. New generation of systemic inflammatory markers for respiratory syncytial virus infection in children. Viruses 2023;15(5):1245.



Supplementary Figure (1): ROC curve showing Performance of troponin in prediction of abnormal Echo

Citation

Salah, K., Sarhan, D., Ahmed, H., Mohamed, A., Rashad, M. Evaluation of Cardiac Functions in Children with Lower Respiratory Tract Infections. *Zagazig University Medical Journal*, 2025; (2346-2360): -. doi: 10.21608/zumj.2025.375724.3908