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Case Report

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When Pneumonia is Not Pneumonia: Acute Myocarditis Encountered in a Pediatric Emergency Service

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ABSTRACT

Pediatric acute myocarditis often presents with dominant respiratory symptoms, creating a significant diagnostic overlap with common lower respiratory tract infections. This clinical mimicry frequently results in misdiagnosis, such as pneumonia, and potentially fatal delays in specialized cardiac care. We report three consecutive male patients (aged 6 months to 3.5 years) initially referred to a Pediatric Pulmonology Service for presumed consolidative pneumonia. The diagnosis was shifted to acute myocarditis only after identifying cardiac "red flags," including tachycardia disproportionate to fever, cardiomegaly on imaging, and elevated cardiac biomarkers or pericardial effusions. Clinical management included intravenous immunoglobulin, pulse steroids, and supportive therapy. One patient required electrical cardioversion for refractory ventricular tachycardia. All three patients achieved clinical recovery or significant improvement, with normalization of cardiac function or resolution of effusions.

Emergency medicine specialists and pediatricians must maintain a high index of suspicion for acute myocarditis in children presenting with respiratory distress. Recognizing clinical findings that are disproportionate to the degree of parenchymal lung involvement is essential to prevent diagnostic delays and ensure the timely initiation of life-saving interventions.

Introduction

Acute myocarditis (AM) in children is an inflammatory myocardial disease associated with severe outcomes, including heart failure and cardiac transplantation [1-5]. Although rare—with an annual incidence of 1–2 per 100,000 children—AM follows an unpredictable course that can lead to sudden hemodynamic collapse [1-6]. Since many deaths occur within the first 72 hours of admission, prompt recognition is vital to improving survival [2, 5, 7, 8].

Diagnosis is challenging due to a clinical spectrum that overlaps with common childhood viral illnesses [1, 3, 8]. Respiratory manifestations dominate the initial presentation; shortness of breath (99% of cases) and

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tachypnea (up to 80%) frequently result in nearly 62% of AM cases being initially mismanaged as pneumonia, bronchiolitis, or asthma [1, 4, 7, 9-11]. This ambiguity leads to 84% of patients requiring multiple physician visits before a correct diagnosis, allowing for the rapid progression of myocardial inflammation toward cardiogenic shock or arrhythmia [1, 7, 8, 10].

A significant knowledge gap persists within specialized pediatric emergency services, where baseline diagnostic error rates for consolidative pneumonia can reach 45% [1, 7, 11-13]. Clinicians often focus on primary pulmonary pathology while overlooking subtle signs of myocardial injury [3]. Highlighting specific respiratory "red flags"—such as tachycardia disproportionate to fever or cardiomegaly on initial radiographs—is a clinical imperative [5, 8, 10]. We describe a consecutive series of three children referred to a Pediatric Pulmonology Service with suspected pneumonia who were ultimately diagnosed with AM, to establish a practical framework for early recognition [4].

Case presentation

Case 1

A 2-year-8-month-old male (13 kg) presented with a one-week history of rhinorrhea and cough progressing to vomiting, diarrhea, lethargy, and cyanosis. Examination revealed tachycardia, tachypnea, and 83% oxygen saturation. Initially managed for suspected pneumonia, AM was suspected after identifying tachycardia disproportionate to the febrile state and cardiomegaly. Laboratory results confirmed myocardial injury with a troponin I of 1.34 ng/mL and an NT-proBNP of 2182 pg/mL. Echocardiography showed reduced cardiac function (53%) and bilateral pleural effusions. Chest CT demonstrated collapse-consolidation, and pleural tap revealed transudative fluid. Treatment included IVIG (2 g/kg), broad-spectrum antibiotics, and sacubitril/valsartan. Following an immunology consultation, combined immunodeficiency was suspected. He stabilized and remained asymptomatic at two-week follow-up.

Case 2

A 3-year-5-month-old boy (13.5 kg, 25th–50th percentile) presented with three days of fever and seizures followed by respiratory distress and cyanosis. On admission, he was hemodynamically unstable (HR 280 bpm, BP 82/41 mmHg). ECG demonstrated regular wide-complex tachycardia without visible P waves, raising the differential diagnosis of supraventricular tachycardia with aberrancy versus ventricular tachycardia (Figure 1). Three bolus doses of adenosine and synchronized cardioversion (1 J/kg) failed to restore sinus rhythm but unmasked premature ventricular complexes, supporting a diagnosis of ventricular tachyarrhythmia (Figure 2). Intravenous lidocaine successfully restored sinus rhythm. Echocardiography showed preserved left ventricular systolic function (ejection fraction 55%) with significant bilateral pleural effusions.

AM was confirmed via elevated biomarkers (troponin I 0.08 ng/mL, NT-proBNP 1698 pg/mL), cardiomegaly, and pleural effusions. Management included IVIG,

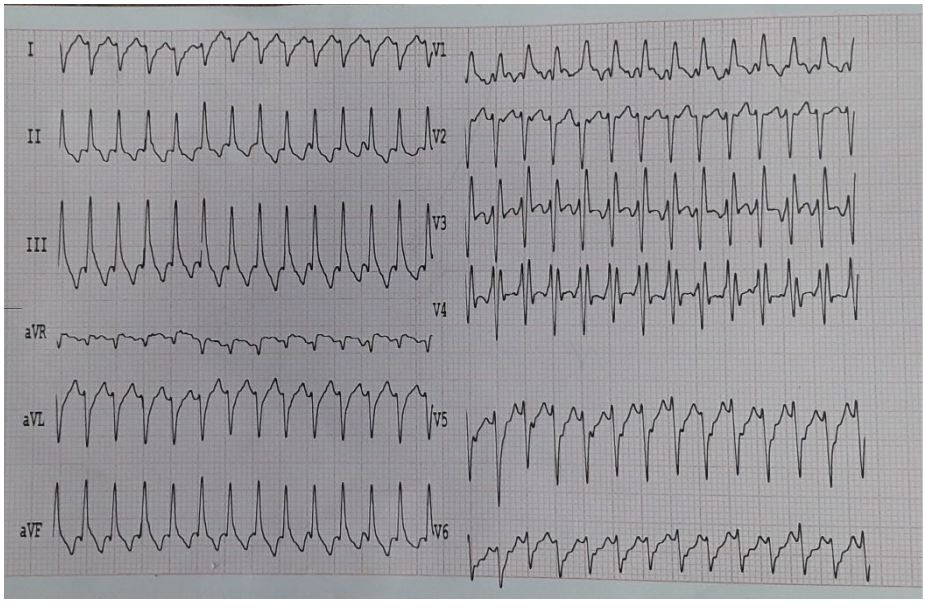


Fig. 1. First electrocardiogram. Supraventricular tachycardia with aberrancy versus ventricular tachycardia

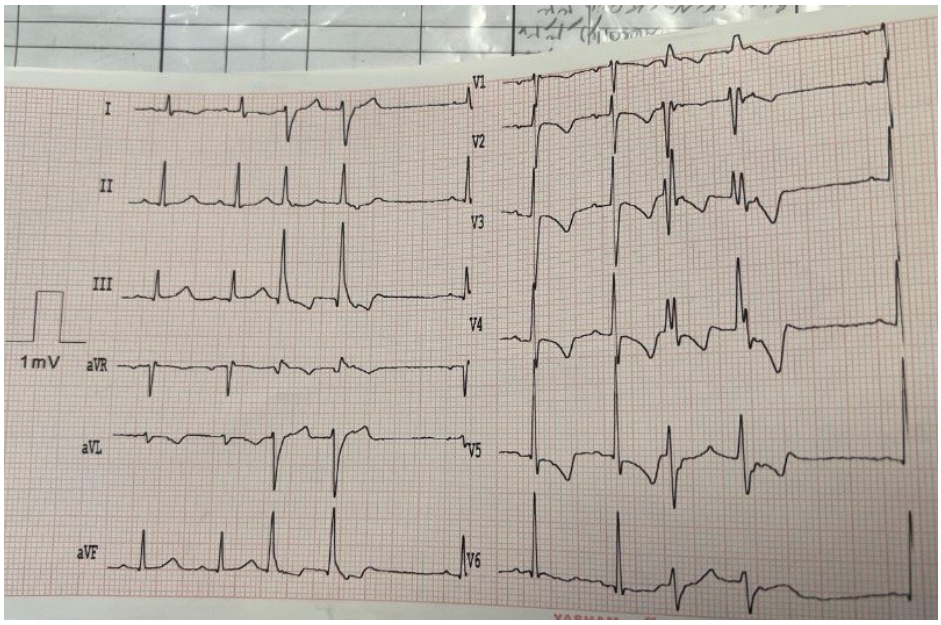


Fig. 2. Second electrocardiogram. After three doses of adenosine and one synchronized shock at 1 j/kg. Premature ventricular contractions (PVCs)

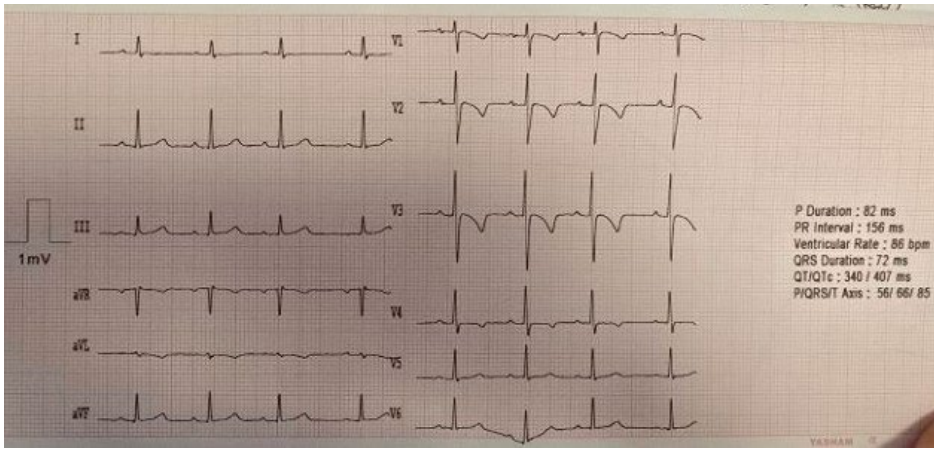


Fig. 3. Fourth electrocardiogram. After the Tablet Flecainide and propranolol initiation. Normal sinuous rhythm

pulse methylprednisolone, continuous lidocaine infusion, and broad-spectrum antibiotics. Despite initial stabilization, intermittent premature ventricular complexes and heart rate fluctuations recurred 10 hours later, prompting transfer to a tertiary pediatric electrophysiology center for flecainide and propranolol therapy, with consideration of catheter ablation if refractory arrhythmia persisted (Figure 3).

Case 3

A 6-month-old male (5 kg) presented with a one-week history of coryza and cough progressing to fever, weakness, and cyanosis. Referred for pneumonia with severe desaturation and wheezing, AM was suspected after recognizing tachycardia disproportionate to lung findings and significant cardiomegaly with a silhouette sign. Investigations revealed elevated liver transaminases and severe pericardial effusion on echocardiography (Figures 4, 5, 6). Congenital hypothyroidism was confirmed; the introduction of levothyroxine alongside IVIG therapy led to rapid resolution of the pericardial fluid within one week. Follow-up showed complete recovery.

Discussion

Nonspecific respiratory symptoms may appear and may be mistakenly diagnosed as pneumonia or bronchiolitis. This “respiratory mask” causes a significant diagnostic delay. For example, the present series showed that all three children were initially referred as having “consolidated pneumonia,” but subsequently, with the observation of disproportionate tachycardia and abnormal imaging

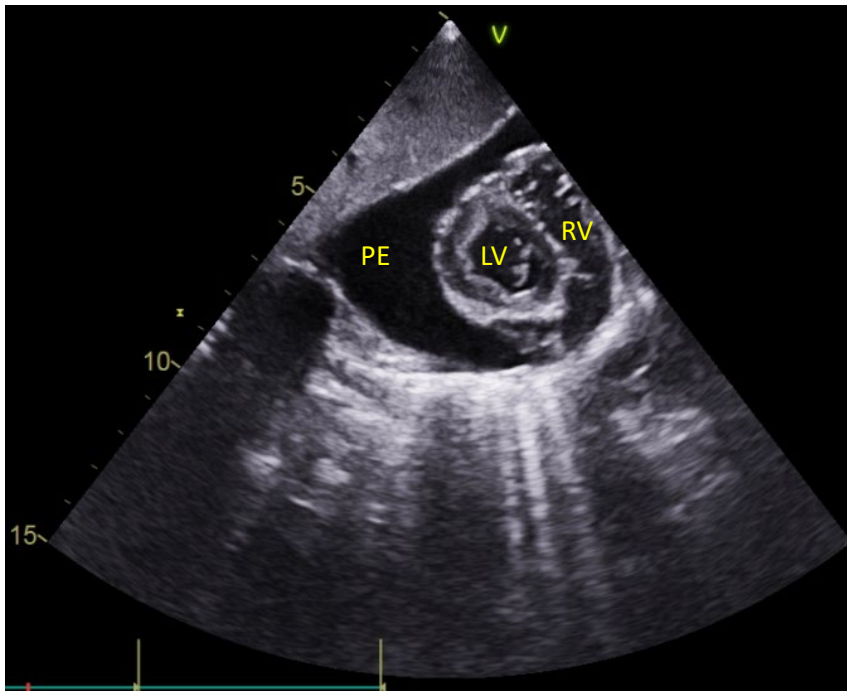


Fig. 4. Severe Pericardial effusion in subcostal RVOT view of Transthoracic echocardiography. RV, Right ventricle; LV, Left ventricle; PE, Pericardial effusion

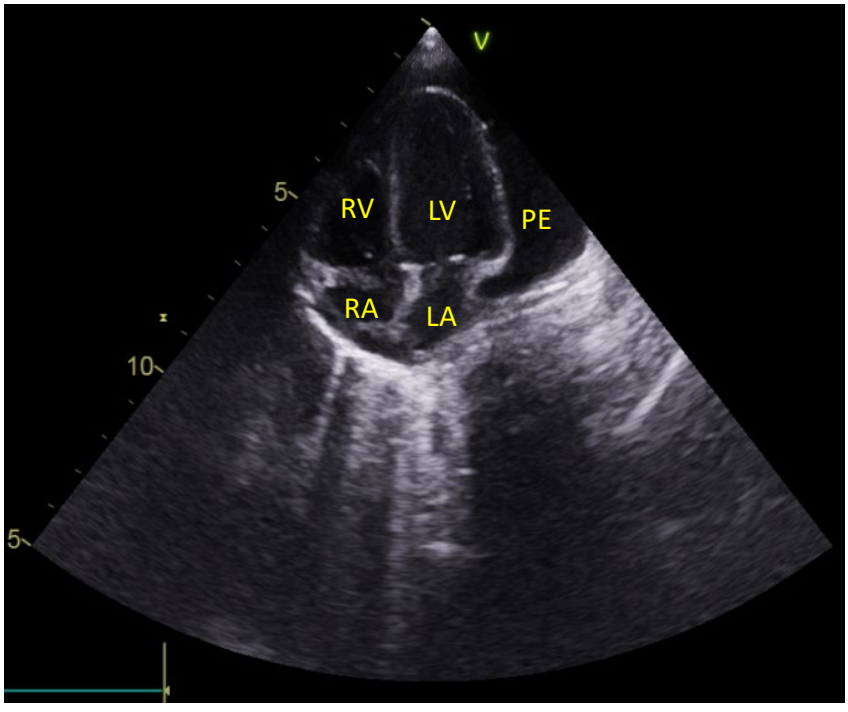


Fig. 5. Severe Pericardial effusion in the 4-chamber view of Transthoracic echocardiography. RA, Right atrial; LA, Left Atrial; RV, Right ventricle; LV, Left ventricle; PE, Pericardial effusion.

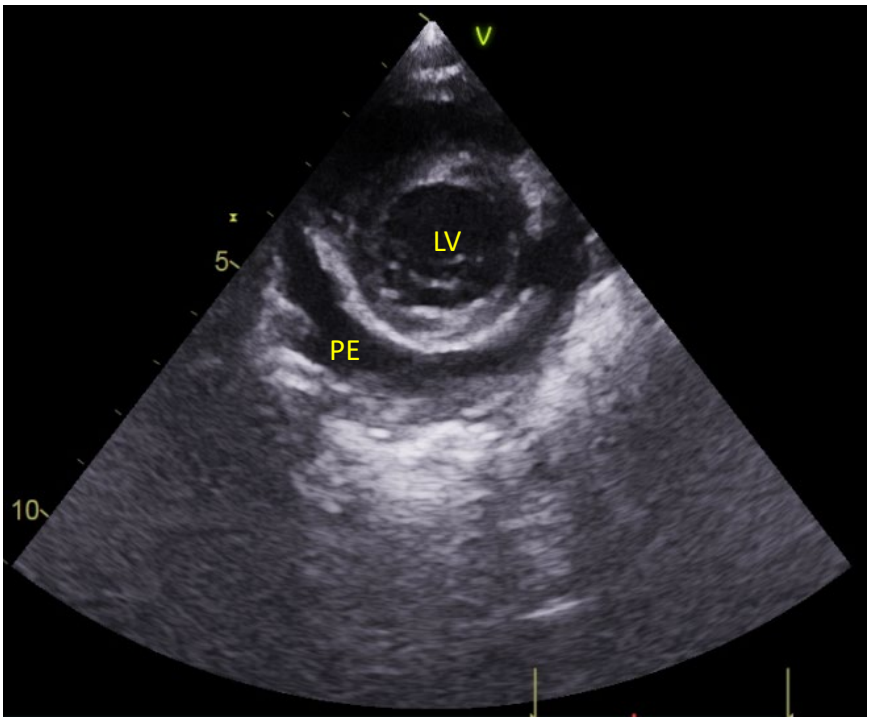


Fig. 6. Severe Pericardial effusion in the short axis view of Transthoracic echocardiography. LV, Left ventricle; PE, Pericardial effusion.

findings (meniscus and silhouette signs), the correct diagnosis of myocarditis was established. The findings of this series are consistent with previous reports: studies similar to this in Spain and Iran have shown that more than half of children with myocarditis present to emergency departments with tachycardia (50–57%) and tachypnea [1-4]. At the same time, unusual cardiac warning signs such as peripheral hypoperfusion or hepatomegaly (signs of cardiac congestion) are helpful; our study of the child who developed a seizure during hospitalization showed that systemic manifestations (such as epilepsy and liver injury) may indicate extension of cardiovascular inflammation.

Respiratory imaging: Chest radiography in more than half of cases may show cardiomegaly or pleural effusion. The “meniscus” or “silhouette” sign, which was evident in this series, classically distinguishes pleural effusion secondary to CHF from pneumonia [5-8]. It also notes that cardiac effusions are often transudative and bilateral, in contrast to pneumonic effusions, which are usually unilateral and exudative [9, 10]. In our series, transudative pleural fluid was solely the prominent cardiac finding, because pleural sampling in the first child showed clear, low-protein fluid consistent with CHF (with no purulent or viral findings observed on culture).

Biomarkers and history: Recent studies indicate that troponin and NT-proBNP are often elevated in AM, but one-third of cases based on recent tests may be normal [11, 12]. In our study, two cases had elevated troponin, whereas the third case had only a borderline increase in NT-proBNP and a normal troponin level. In one study, a troponin threshold of approximately 0.05 ng/mL was considered suitable for diagnosis; however, its sensitivity was 71%, and it may be associated with false-positive results [12]. Therefore, a normal troponin level, particularly in the early stages of the disease, does not rule out myocarditis [13]. In any case, measurement of these biomarkers (troponin and NT-proBNP) in the presence of such diagnostic uncertainty helps with clinical decision-making (Table 1).

Conclusion

Pediatric myocarditis often necessitates admission to the intensive care unit (ICU) and is associated with high mortality. Pediatricians must maintain a high level of vigilance regarding cardiac warning signs to avoid misdiagnosis and improve outcomes. When encountering a febrile child with respiratory symptoms who exhibits severe, “unexplained” tachycardia or signs of cardiac or pulmonary congestion, it is recommended that a cardiac evaluation

Table 1. Comparative Summary of Pediatric Myocarditis Cases

Variable	Case 1: Seyed Shahan Elmi	Case 2: Mohammad Sadra Zinati	Case 3: Mohsen Khavari
Age / Sex	2 years, 8 months / Male	3 years, 5 months / Male	6 months / Male
Weight (Percentile)	13 kg (25th–50th)	13.5 kg (25th–50th)	5 kg (<3rd percentile)
Underlying Disease	Skin sensitivity and eczema	Chronic seizures (on medication)	Congenital hypothyroidism; neonatal jaundice
Primary Symptoms	Respiratory + Gastrointestinal	Respiratory + Neurological (seizure)	Respiratory + Weakness
CXR Findings	Lung involvement + Meniscus sign	Cardiomegaly + Lung involvement	Cardiomegaly + Silhouette sign
Fibrinogen (mg/dl)	Not reported	120.0 (Low)	Not reported
D-dimer (ng/ml)	Not reported	1511 (High)	**1271 / 1147 (High)**
Ferritin	Not reported	Not reported	**1290.3**
Thyroid Tests	Not reported	Not reported	**T4: 12.8 (H) / TSH: 7.9 (H)**
Cardiac Enzymes	Troponin I: 1.34 (H)NT-proBNP: 2182 (H)	Troponin I: 0.08 (H)NT-proBNP: 1698 (H)	**Troponin I:** 0.01 (Normal)**NT-proBNP:** 326 (Borderline)
Other Labs	AST: 59 (H)Suspicion of CID	AST: 40–47 (H)LDH: 660–637 (H)	**AST:** 1146–231 (H)**ALT:** 1157–512 (H)
CT Imaging	Bilateral pleural effusion; consolidation	Pleural effusion; atelectasis bands	Pericardial effusion; consolidation
Echocardiography	EF 60% → 53%	EF 60% → 55%	EF 58% → 57%; pericardial effusion
Primary Treatments	IVIG (2g/kg); sacubitril/valsartan; Lasix, Antibiotics	IVIG; Pulse Steroids; Lidocaine, Antibiotics	IVIG; Methylprednisolone; Oseltamivir, Antibiotics
Final Outcome	Complete recovery; discharged	Recovery after arrhythmia control	Improved (No residual pericardial effusion)

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; BNP, B-type natriuretic peptide; CBC, complete blood count; CK-MB, creatine kinase-MB; CRP, C-reactive protein; CT, computed tomography; CXR, chest radiograph; ECG, electrocardiogram; EF, ejection fraction; ESR, erythrocyte sedimentation rate; IVIG, intravenous immunoglobulin; MRI, magnetic resonance imaging; NT-proBNP, N-terminal pro-B-type natriuretic peptide; PaO_2, arterial oxygen tension; PCR, polymerase chain reaction; PICU, pediatric intensive care unit; SpO_2, peripheral oxygen saturation; TAP, diagnostic thoracentesis; WBC, white blood cell count.

(echocardiography) and an electrocardiogram (ECG) be performed immediately, and that appropriate treatment be pursued in consultation with a cardiologist.

Ethical Considerations

Ethics approval and consent to participate:

For the preparation of this case report, written consent was obtained from the infant's parents to collect and use the necessary information, without disclosing the child's name.

Consent for publication

Written informed consent for the publication of the infant's clinical details and echocardiographic images was obtained from the parents.

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Conflict of Interests

The authors declare that they have no competing interests.

Authors' contributions

A.S. and M.T. proposed the idea for this report and supervised the clinical assessment. M.T. and A.S. collected clinical and echocardiographic data. M.T. and A.S. interpreted the imaging findings and drafted the manuscript. All authors reviewed and approved the final version of the manuscript.

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