

A Life-Threatening Cardiac Arrhythmia: Pediatric Myocarditis

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Abstract

Cardiac arrhythmias results from disturbance of electrical impulse formation and conduction. Supraventricular tachycardia is the most common emergent paediatric arrhythmias, with an incidence of 1 in 250 to 1000 children. Symptoms may vary from mild chest pain, to congestive cardiac failure. A 7-year-old male with no underlying comorbidities presented to the emergency department with sudden onset dyspnoea and chest pain. Upon examination he was tachypnoeic, normotensive and afebrile. The electrocardiogram revealed a narrow complex regular tachycardia with a rate of 250 bpm. The child was in cardiac failure. He did not respond to pharmacological cardioversion. Due to respiratory distress and haemodynamic instability, he was electrically cardioverted. However, his condition deteriorated and he developed pulseless electrical activity. Following two cycles of cardiopulmonary resuscitation, return of spontaneous circulation was achieved, and he was admitted to the paediatric intensive care unit. Blood serology was tested positive for Mycoplasma Pneumoniae, and he was treated as Mycoplasma myocarditis. He went on to make an uneventful recovery and was discharged six days later.

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INTRODUCTION

The diagnosis of myocarditis in pediatric populations is challenging due to a broad spectrum of clinical presentations, which include chest pain, shortness of breath, fever, tachycardia, heart failure, systolic hypotension and arrhythmias.¹

Myocarditis is an important cause of morbidity and mortality in children, with a high percentage of patients requiring hospitalization, intensive care admission, mechanical ventilation, inotropes and mechanical circulatory support.² This is a case of a previously healthy child who presented with an

unstable supraventricular tachycardia (SVT) to the emergency department (ED). He was subsequently diagnosed with *Mycoplasma mycocarditis*.

CASE REPORT

A 7-years-old male with no underlying comorbidities presented to the ED with a sudden onset of chest pain and breathlessness since midnight. Three weeks prior to presentation, he was treated by a general practitioner (GP) for a gum infection, however was not compliant with the antibiotics prescribed. Two weeks prior to presentation, the child was found to be more lethargic but was still attending school. One week before presentation, he had reduced exercise tolerance, abdominal distension and bilateral periorbital oedema. He was treated by the same GP for a chest infection with oral cefuroxime. However, his condition deteriorated with sudden onset of chest pain and shortness of breath. He was brought to a private hospital and was subsequently referred to the ED of a public hospital for 'extreme tachycardia'.

On arrival, the child was alert, cooperative with increased work of breathing and facial oedema. Initial vital signs showed a heart rate of 250 beats per minute (bpm), he was normotensive with a blood pressure of 114/70 mmHg, a respiratory rate of 30

breaths per minute, a temperature of 36.5°C and oxygen saturations of 96% on room air. Peripheries were cold with a delayed capillary refill time. Heart sounds were normal, however, there were bilateral basal lung crepitation, ascites, hepatomegaly and pedal oedema.

The electrocardiogram (ECG) revealed a narrow complex regular tachycardia with a rate of 250 bpm, with absent P waves, suggestive of supraventricular tachycardia (SVT) (Figure 1). The initial diagnosis was SVT with acute heart failure. Supplementary oxygen was administered and an early referral to the paediatric team was made. Vagal manoeuvres were unsuccessful, as were two consecutive doses of intravenous (IV) adenosine at 0.1mg/kg and 0.2mg/kg respectively. Subsequently he was started on an IV amiodarone infusion 5mg/kg. The child became restless and hypotensive with respiratory distress. Three consecutive synchronized cardioversions were performed at 0.5 J/kg, 1 J/kg and 2 J/kg respectively. Repeated ECGs following cardioversion showed sinus rhythm with heart rate of 120 bpm (Figure 2). However, the child became drowsy and was intubated for airway protection. He developed pulseless electrical activity (PEA) post intubation. Return of spontaneous circulation was achieved following two cycles of cardiopulmonary resuscitation.

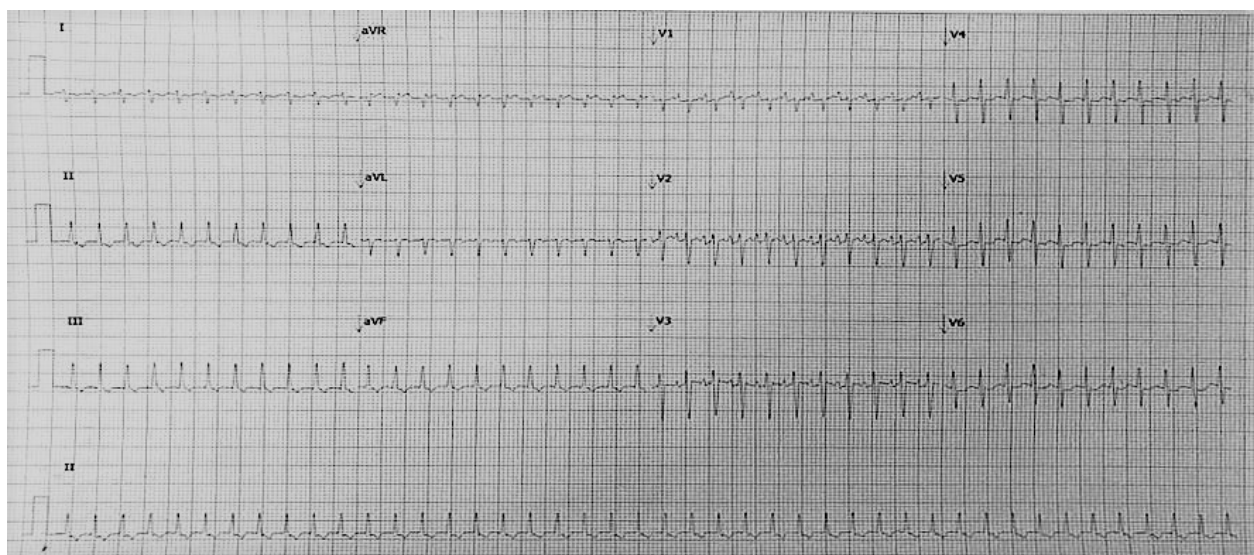


Figure 1: Electrocardiogram at initial presentation showing absence of P wave, regular narrow QRS complex with heart rate of 250 beats per minute, suggestive of supraventricular tachycardia.

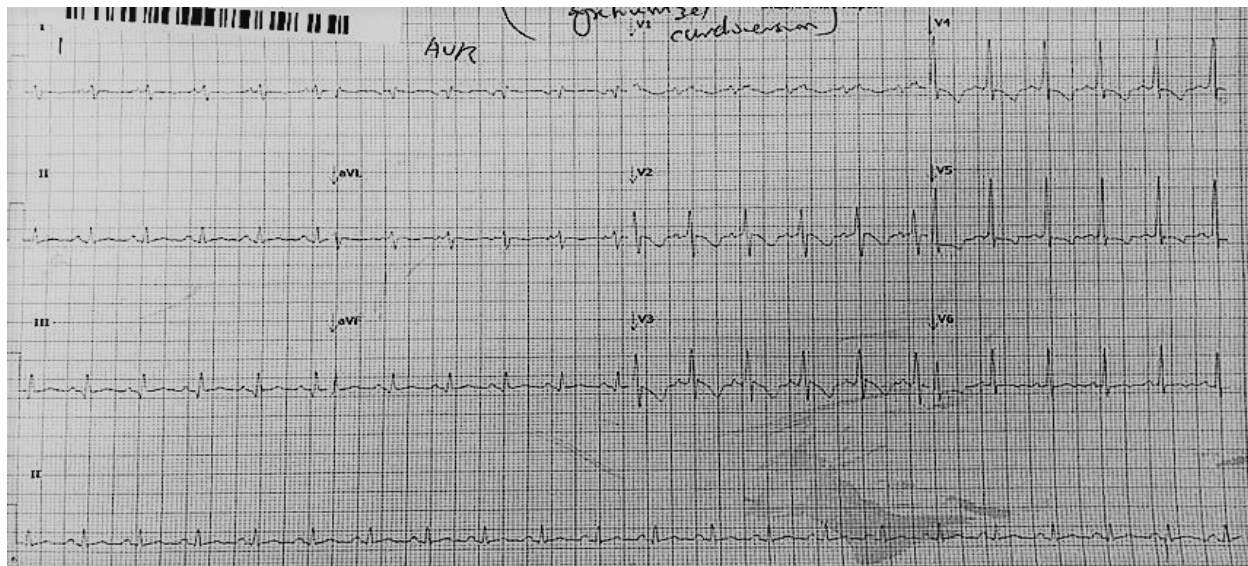


Figure 2: Electrocardiogram after electrical cardioversion, sinus rhythm with heart rate of 120 beats per minute

Inotropic support with a dobutamine infusion at 10 mcg/kg/min was initiated and the child was admitted to the paediatric intensive care unit (PICU).

Laboratory findings showed a leucocytosis with a white cell count of $12.3 \times 10^9/L$, neutrophils 46%,

lymphocytes 46%, haemoglobin 14.8 g/dL, and a platelet count of $340 \times 10^9/L$. Renal profile, blood electrolytes and capillary blood glucose were within normal range. Creatine kinase (CK) was elevated with a reading of 350 U/L (normal range 170 U/L).

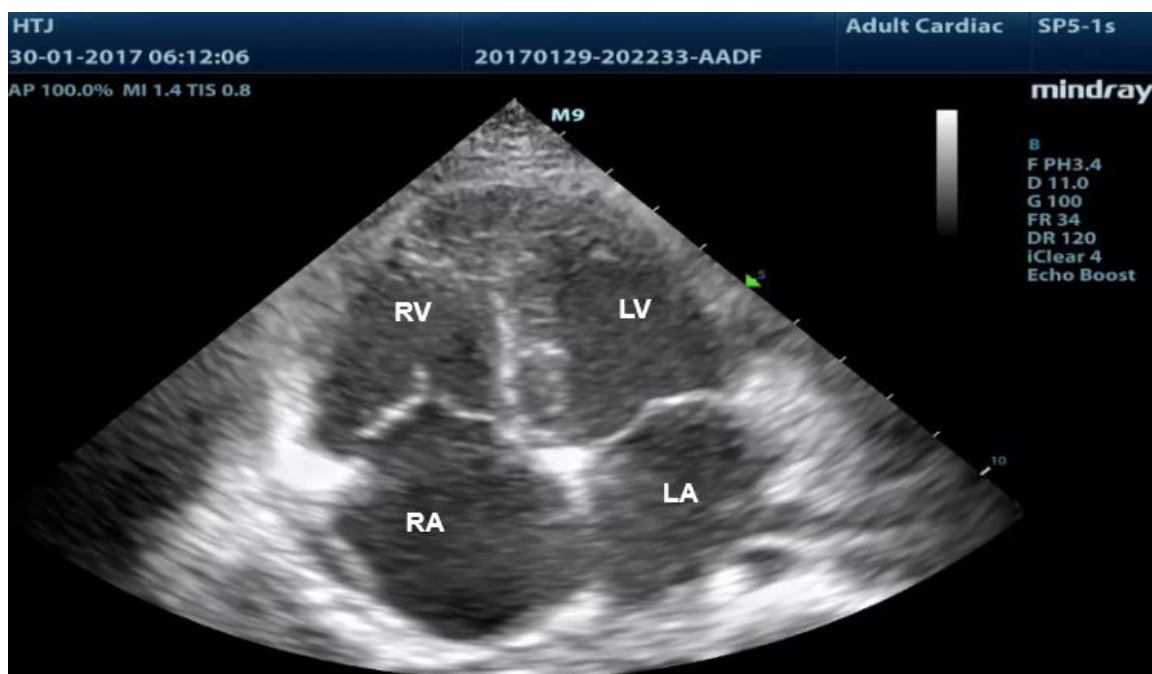


Figure 3: Transthoracic echocardiogram shows all four cardiac chambers to be dilated on the apical 4 chamber (A4C) view. RA, right atrium; RV, right ventricle; LA, left atrium; LV, left ventricle.

A chest radiograph showed cardiomegaly, a left pleural effusion and pulmonary oedema. Transthoracic echocardiogram (TTE) revealed dilatation of all heart chambers, with global hypokinesia, poor cardiac contractility and ejection fraction of 30% with minimal pericardial effusion (<0.5cm), with no structural abnormality (Figure 3). *Mycoplasma pneumoniae* serology was positive. He was diagnosed with *Mycoplasma myocarditis* complicated with unstable SVT in cardiogenic shock and congestive cardiac failure.

He was treated with IV ceftriaxone and extubated two days later. No further arrhythmias were observed in PICU. He was transferred to the general ward and repeated transthoracic echocardiograms were normal. He was discharged after 6 days of hospitalization without neurological sequelae.

DISCUSSION

According to the World Health Organization (WHO) in 1995, inflammatory cardiomyopathy is defined as myocarditis in association with cardiac dysfunction. Myocarditis is an inflammatory disease of the myocardium and is diagnosed by established histological and immunological criteria, with the absence of ischaemia.³ The true incidence of myocarditis in the paediatric population is difficult to measure due to a wide variety of presentations and the lack of diagnostic protocols. Patients of all ages may be affected but the majority of cases are seen in the first year of life.^{1,3} There are multiple aetiologies of myocarditis. The commonest cause is via infectious exposure, especially viruses such as Adenovirus, Enterovirus and Coxsackievirus. Other causes include autoimmune diseases, genetic malformations, metabolic abnormalities, hypersensitivity reactions and toxic reaction to drugs.^{1,4}

The mechanism of developing myocarditis following infection is mainly due to direct cellular damage of myocytes by virus or bacteria, causing activation of innate and adaptive immune responses. This excessive immune-mediated destruction of myocardium targets the infected cardiomyocytes,

producing chemicals that can damage the heart muscle, subsequently causing fibrosis and dilatation. As a result, all chambers of the heart can become dilated due to profound alteration in the extracellular matrix of cardiac muscle.^{1,3,4} Patients may develop varying degrees of ventricular dysfunction as damage to the myocardium continues, such as dilated cardiomyopathy characterized by a dilated left ventricle with systolic dysfunction and signs of congestive heart failure.²

The clinical presentation is extremely variable, ranging from asymptomatic to sudden unexpected death.^{1,2,5} The classical presentation of myocarditis is the development of symptoms of heart failure with dilated cardiomyopathy a few weeks after the onset of a viral illness, including fever, myalgia, respiratory and gastrointestinal symptoms. Chest pain, syncope and palpitations may also be seen at presentation. Clinical cardiac-specific signs, such as pallor, oedema, hypotension and hepatomegaly, are seen only in the minority of cases. Initial damage to myocardium causing reduced myocardial function may manifest as exercise intolerance. Meanwhile, dilated heart chambers with poor contractility result in pulmonary congestion. Rhythm disturbances occur due to atrioventricular nodal infiltration, while ventricular dysrhythmias can lead to seizures and sudden death.^{2,4,5,6} A study by Liberman et al. in 2014 reported the commonest significant tachyarrhythmias observed in paediatric myocarditis was ventricular tachycardia (11%), followed by atrial flutter (1.6%), atrial fibrillation (1%) and SVT (0.9%). The presence of significant tachyarrhythmias itself is a predictor of poor outcomes.⁷

According to American Heart Association (AHA) in 2015, supraventricular tachycardia (SVT) is an abnormally rapid rhythm originating from a conduction pathway prior to reaching the ventricles, and commonly caused by a reentry mechanism that involves an accessory pathway or the atrioventricular conduction system. It is the most common tachyarrhythmia that results in cardiovascular compromise during infancy and childhood. The diagnostic ECG criteria include; a heart rate greater than 220 bpm in infants and greater than 180 bpm in children, the

absence of or an abnormal P wave, and a regular narrow QRS complex (<0.09 sec).⁸ Common signs and symptoms of SVT in older children include chest pain, palpitation, shortness of breath, exercise intolerance, light-headedness and syncope. SVT is well-tolerated among infants and children, however it may lead to congestive cardiac failure and cardiovascular collapse if left untreated.^{4, 8}

There is no definitive diagnostic criteria for myocarditis or cardiomyopathy, instead the physician must have a high index of suspicion of myocarditis especially when dealing with a child with acute heart failure without congenital cardiac anomalies.^{1,5} Initial investigations should include cardiac enzymes, ECG, chest radiograph and echocardiogram. Cardiac troponin T may provide better sensitivity for detecting micronecrosis in myocarditis.^{1,5,6}

Cardiac biomarker elevation can help to determine the severity and prognosis. Sinus tachycardia is the commonest ECG finding followed by ST changes, heart block and low QRS voltage. Chest radiographs may reveal cardiomegaly, pulmonary oedema or pleural effusion.^{2,6} Transthoracic echocardiography remains a useful, non-invasive diagnostic tool when suspecting myocarditis. The most common findings are left ventricle (LV) dilatation and reduced global LV ejection fraction. Other abnormalities that may be seen are segmented wall motion abnormalities, mitral regurgitation and pericardial effusion. Cardiovascular Magnetic Resonance Imaging (cMRI) can detect acute inflammation within 14 days of symptoms including oedema, necrosis and fibrosis of the myocardium. However, cMRI is not readily available and is difficult to perform on unstable patients.^{1,2}

Treatment strategies for myocarditis are mainly supportive. This includes (1) stabilization of hemodynamic compromise, (2) treatment of congestive cardiac failure such as diuretics, afterload reduction and inotropic support, (3) management of arrhythmias and (4) treatment of underlying causes. It is recommended to involve the paediatric cardiologists early.^{1,4,5,8,9} Despite these treatment modalities, the mortality rate remains at 7-15% among the paediatric population.²

Mycoplasma associated myocarditis is rare, and incidence occur in 1-5% of paediatric myocarditis population.⁹ In this case, the patient presented with signs of heart failure supporting the clinical features of myocarditis, however, the manifestation of SVT among myocarditis is also rare (0.9%).⁷

CONCLUSION

The diagnosis of myocarditis in children is challenging due to its diverse clinical presentation. Myocarditis from various causes should be considered as a differential diagnosis in a child with prolonged illness unresponsive to treatment. Therefore, early diagnostic tests including an ECG in primary care and an early referral to a paediatrician should be considered.

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