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Infant acute myocarditis mimicking acute myocardial infarction

Maher Jedidi^a, Samia Tilouche^b, Tasnim Masmoudi^a, Maha Sahnoun^a, Youssef Chkirbène^a, Sarra Mestiri^c, Lamia Boughamoura^b, Mohamed Ben Dhiab^a, Mohamed Kamel Souguir^a

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ABSTRACT

Myocarditis is an inflammatory disease of the myocardium with heterogeneous clinical manifestations and progression. In clinical practice, although there are many methods of diagnosis of acute myocarditis, the diagnosis remains an embarrassing dilemma for clinicians. The authors report the case of 9-month-old infant who was brought to the Pediatric Emergency Department with sudden onset dyspnea. Examination disclosed heart failure and resuscitation was undertaken. The electrocardiogram showed an ST segment elevation in the anterolateral leads with a mirror image. Cardiac enzyme tests revealed a significant elevation of troponin and creatine phosphokinase levels. A diagnosis of acute myocardial infarction was made, and heparin therapy was prescribed. The infant died on the third day after admission with cardiogenic shock. The autopsy showed dilatation of the ventricles and massive edema of the lungs. Histological examinations of myocardium samples revealed the presence of a marked lymphocytic infiltrate dissociating myocardiocytes. Death was attributed to acute myocarditis. The authors call attention to the difficulties of differential diagnosis between acute myocarditis and acute myocardial infarction especially in children, and to the important therapeutic implications of a correct diagnosis.

Keywords

Myocarditis; Myocardial Infarction; Diagnosis, Differential; Infant Death

CASE REPORT

A 9-month-old girl, with no documented medical history, was brought to the Pediatric Emergency Department for sudden onset dyspnea. She had a recent history of rhinorrhea and cough. On examination, she had a pulse of 180/min (normal range: 80-160/min), a blood pressure of 70/40 mmHg, and a respiratory rate of 48/min with chest indrawing (normal value: 30-40/min). Her temperature was 38.9 °C. The electrocardiogram (ECG) showed ventricular

tachycardia, which spontaneously returned to a regular rhythm with extrasystoles (Figure 1).

Acute heart failure was diagnosed. Orotracheal intubation and mechanical ventilatory assistance were immediately started, and the patient was transferred to the pediatric intensive care unit. A diagnostic work-up was subsequently performed. The chest x-ray showed cardiomegaly with bilateral pulmonary congestion (Figure 2).

^c Department of Pathology - Farhat Hached University Hospital, Sousse – Tunisia.



^a Department of Legal Medicine - Farhat Hached University Hospital, Sousse - Tunisia.

^b Department of Pediatrics - Farhat Hached University Hospital, Sousse – Tunisia.

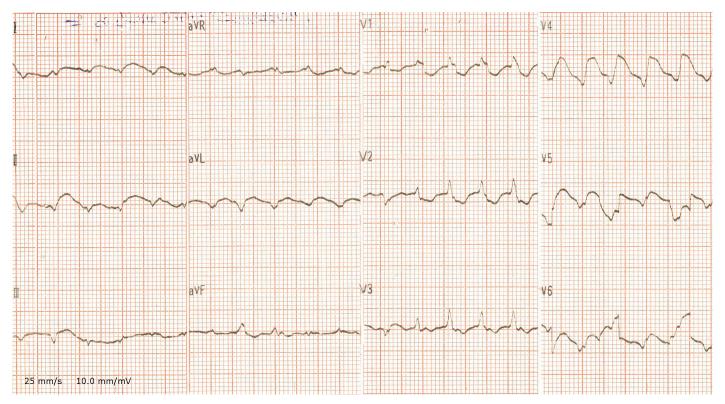


Figure 1. Twelve-lead electrocardiogram (ECG) showing ventricular tachycardia (ECG on admission).

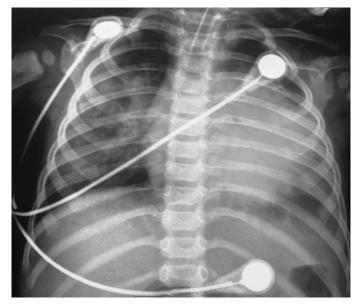


Figure 2. Chest x-ray showing cardiomegaly with bilateral pulmonary congestion.

Laboratory exams showed hyperkalemia at 5.6 mmol/L with normal renal function, increased serum lactate dehydrogenase (1234 UI/L; reference value [RV]: 230-4600 UI/L), normal creatine phosphokinase (CPK), and a slight increase of troponin I level (0.81 ng/mL; RV: < 0.25 ng/mL). Initial treatment consisted of cardiac drugs, alkalinization, and antibiotic therapy.

A control ECG achieved after 3 hours showed ST segment elevation in the anterolateral leads (V3-V6) and ST depression on V1-V2 producing a "mirror image" (Figure 3). Myocardial necrosis markers disclosed an increased CPK at 2066 UI/L (RV: 95-195 UI/L) and a significant elevation of troponin I level (1.92 ng/mL). A diagnosis of acute myocardial infarction was established, and heparin therapy was started.

An echocardiogram showed a diffuse hypocontractility and dilation of the left ventricle without any other abnormality. The infant died on the third day after admission due to cardiogenic shock, and a forensic autopsy was performed because of the unexplained cause of death.

AUTOPSY FINDINGS

The deceased was 78 cm in height and weighed 10 kg. The external examination showed an apparently healthy infant presenting cyanosis of the lips. Internal examination disclosed a normal appearance of the scalp, skull, and brain. The heart weighed 70 g (RV: 40 g). Coronary arteries were normal and had a normal origin. Both ventricles were dilated, and the heart muscle was homogeneous (Figure 4A and 4B).

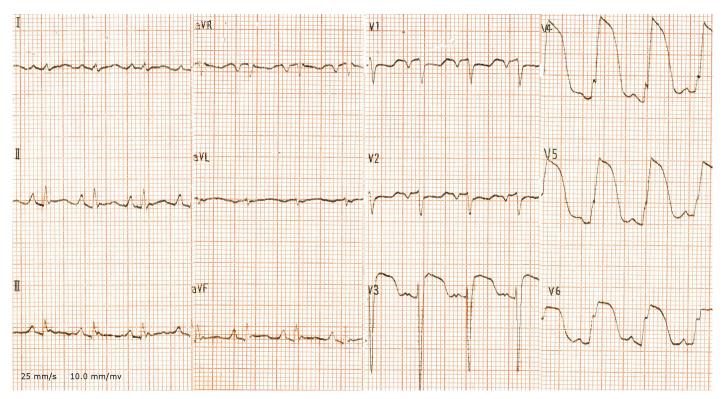


Figure 3. Twelve-lead electrocardiogram (ECG) showing ST segment elevation on the anterolateral leads (V3–V6) and ST depression on V1–V2 (ECG 3 hours after admission).

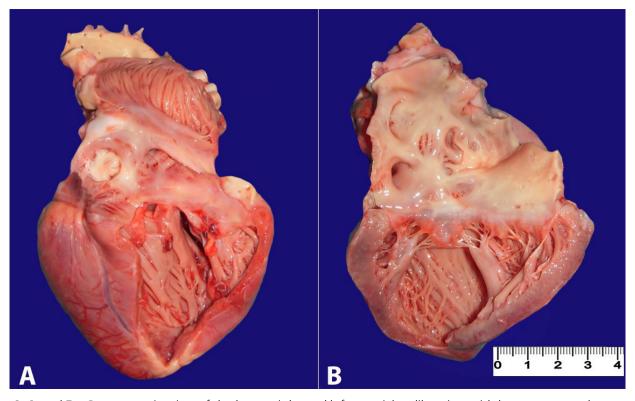


Figure 4. A and **B** - Gross examination of the heart: right and left ventricles dilatation with homogeneous heart muscle.

The lungs were markedly congested and massively edematous. Histological examinations of the heart revealed the presence of an important lymphocytic infiltrate dissociating myocardiocytes with scattered ischemic or necrotic focal points (Figure 5A and 5B).

Microscopic sections of the remaining internal organs did not show any abnormality, except for massive lung edema. The toxicology screening, which included conventional medicines (salicylates, tricyclics, benzodiazepines, phenothiazines, and paracetamol),

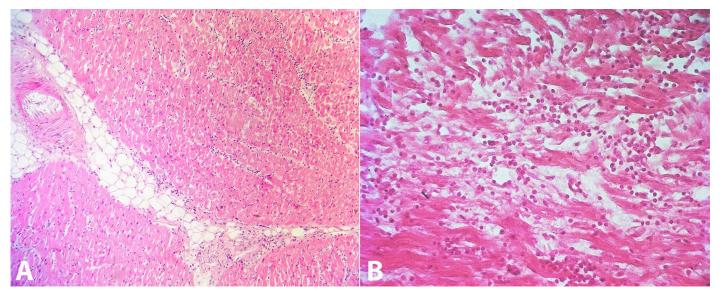


Figure 5. Photomicrography of the heart. **A** - Myocardiocytes dissociated by interstitial edema and inflammatory infiltrate (H&E, 40X); **B** - High magnification view shows the inflammatory infiltrate consisting predominantly by lymphocytes (H&E, 400X).

pesticides (organophosphates, carbamate, chloralose), and alcohol, was negative.

Death was attributed to acute heart failure due to acute myocarditis. In addition to the toxicological study, no other cause of myocarditis was further investigated.

DISCUSSION

Myocarditis is an inflammatory disease of the myocardium with heterogeneous clinical manifestations and progression.^{1,2} Infectious myocarditis is the most common; it is often caused by a virus and is occasionally associated with bacterial infections.^{2,3} The clinical features of acute myocarditis vary from case to case, ranging from oligosymptomatic cases to heart failure and sudden unexpected death.^{1,4,5}

In clinical practice, although there are many methods of diagnosis of acute myocarditis, including clinical symptoms, laboratory exams, ECG, echocardiogram, magnetic resonance imaging (MRI) findings, and cardiac endo-myocardial biopsy,¹ the diagnosis of myocarditis remains an embarrassing dilemma for clinicians, especially in children where the symptoms may vary substantially.^{2,6,7}

Acute myocarditis may be associated with heart tissue necrosis in some cases. The exact pathogenesis is unclear; hypotheses state that ischemia is a consequence of local endothelial dysfunction, coronary

spasms, and in situ thrombi formation.^{1,8,9} Also, it may clinically resemble myocardial infarction.^{1,2,10} The differential diagnosis between acute myocarditis and acute myocardial infarction can be very challenging and sometimes indistinguishable in children.¹¹

In this case, ECG repolarization abnormalities in a single coronary distribution (ST segment elevation in anterolateral leads), plus serum elevation of CPK and troponin, entirely mimic an acute myocardial infarction presentation, which was firstly suspected by the pediatricians. Cardiomyopathy was also raised before the echocardiogram signs.

Usually, in adult care, ST segment abnormality associated with an elevation of specific biomarkers of cardiac injury—especially troponin—confirm myocardial infarction and allow interventional catheterization.^{7,12} This is largely because there is a high prevalence of atherosclerotic coronary artery disease in the adult population. In children, the causes of elevated troponin differ from that of adults. The most common causes are myocarditis and cardiomyopathy; however, coronary-related ischemia is rare.^{7,13}

Clinicians should consider the diagnosis of acute myocarditis in children who present troponin elevation and acute heart failure. Endomyocardial biopsy remains the gold standard exam for diagnosing myocarditis. However, due to its invasiveness with a high risk of complications and the possibility of false negative results, it is not often performed in common practice. 10,14 MRI is a good diagnostic tool and when

the "Lake Louise Criteria" ¹⁴ are applied, the sensitivity and specificity of MRI prove to be high compared to endomyocardial biopsy. ¹⁵ A correct diagnosis of myocarditis has significant therapeutic implications for patients, and non-realization of MRI can be criticized in this case. An MRI could have guided the diagnosis of myocarditis in this infant.

In this instance, we confirmed the important role of forensic autopsy in explaining the cause of death in such a challenging case.

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Correspondence

Maher Jedidi

Department of Legal Medicine - Farhat Hached University Hospital Sousse

Tunisia

Phone: +216 (98) 536-440/Fax: + 216 (73) 226-702

jedidimaher@yahoo.fr