

ACUTE MYOCARDITIS IN H1N1 INFLUENZA A VIRUS INFECTION

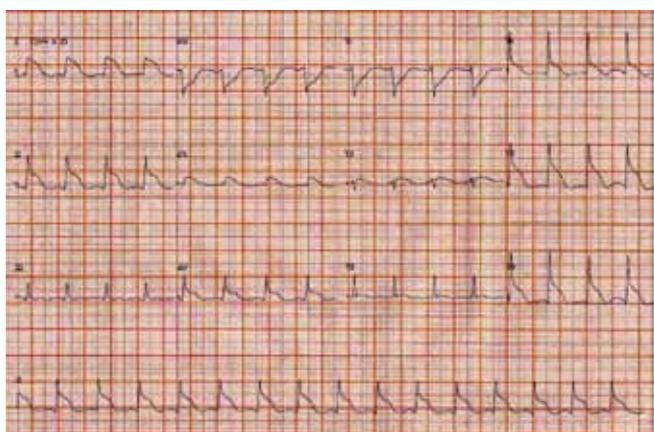
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A 28 year-old man, previously healthy, was admitted to the emergency room with nine days of abdominal pain, myalgia, and headache. Two days after these initial symptoms, he developed high fever, cough and dyspnea. On physical examination he was tachycardic, tachypneic, hypoxemic (HbO₂ saturation breathing room air: 85%), and hypotensive. Subsequently, he was transferred to the intensive care unit for hemodynamic control with vasopressors, invasive mechanical ventilation, and dialysis. It should be emphasized that the patient was admitted to the hospital in July 2009, during the initial phase of H1N1 Influenza A pandemic in Brazil and empirical oseltamivir was promptly initiated. H1N1 influenza A virus was detected on a nasopharyngeal swab using RT-PCR. On the 4th ICU day, after significant reduction of vasopressors, the patient had paroxysms of acute atrial fibrillation and hemodynamic deterioration. Soon after, the electrocardiogram (EKG) showed diffuse ST-segment elevation (Figure 1) and the echocardiography (ECHO) disclosed mild left ventricular enlargement and hypocontractility of apical and medial segments in the septal, inferior, posterior and anterior wall. Left ventricular ejection fraction was 48% (normal range above 55%). Pericardium was normal. Troponin analysis showed marked elevation without a rise and fall pattern: 0.06 → 2.02 → 1.04 → 1.64 → 1.36 (ng/mL; normal range: < 0.03 ng/mL). Hemodynamic support for distributive shock was maintained and continuous intravenous amiodarone infusion was initiated. During the following 12 hours the diffuse ST elevation in the EKG progressively resolved, without new Q waves. A new ECHO was repeated after a week and left ventricular contractility and function had completely recovered. The patient's condition slowly improved, renal function recovered and he was successfully weaned from mechanical ventilation. He was discharged from hospital without cardiac sequelae.

Myocarditis has been associated with several viral infections, including influenza. Although the vast majority of patients present subclinical symptoms¹, massive and fatal myocarditis has been described in previous influenza A outbreaks^{2,3}. In this year, the first report of acute myocarditis associated with H1N1 influenza A infection in a pediatric population was published⁴, and to our knowledge, this is the first case of myocarditis in an adult related to 2009 pandemic influenza A H1N1 infection. We did

Figura 1- Electrocardiogram with diffuse ST segment elevation



not have an histological sample to confirm our diagnosis, but the EKG and ECHO findings, added to the troponin behaviour and clinical evolution, strongly support the diagnosis of myocarditis. Although there were no cardiac sequelae in this case, we stress the importance of cardiac evaluation for patients with severe influenza A H1N1 infection.

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