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Correspondence

A Case of Acute Myocarditis Caused by Pandemic (H1N1) 2009 Influenza Virus

Antonios N Pavlidis,[1] Antreas K Giannakopoulos,[1] Peter G Danias,[2,3] Athanasios J Manolis[1,4]*

The new strain of swine-origin H1N1 influenza virus was initially described in April 2009, and the first cases of infection were reported in the Mexican state of Veracruz. As the disease spread rapidly to other countries, the World Health Organization declared the new strain of influenza virus as pandemic on June 11, 2009.[1] Infection with this virus predominantly affects young patients in relatively good health, without documented underlying illnesses. Although the majority of them experience mild symptoms, severe complications associated with high mortality can occur.

Acute myocarditis can cause substantial myocardial injury and lead to arrhythmia, atrioventricular block, cardiogenic shock and sudden cardiac death. The majority of myocarditis cases are of viral etiology and presentation can often mimic myocardial infarction, pulmonary embolism or acute heart failure. Although endomyocardial biopsy remains the gold standard, cardiac magnetic resonance imaging (CMRI) has recently been shown to be a powerful tool in the diagnosis of acute myocarditis. We present a rare case of acute myocarditis caused by H1N1 influenza virus in a young patient.

An 18-year-old man of Greek origin was admitted with a 3-day history of fever, malaise, sore throat and dry cough, and a 5-hour history of central chest pain of sudden onset. The pain radiated to the left shoulder and was relieved when the patient leaned forward. The pain did not respond to sublingual glyceryl trinitrate. He was a smoker and had a history of spontaneous ventricular septal defect closure at the age of 3 years. On admission, his heart rate was 66 beats/min, oxygen saturation in room air was 98%, blood pressure was 115/75 mmHg, and axillary temperature was 37.1 °C. Heart auscultation revealed normal heart sounds, a pericardial friction rub and no murmurs. Electrocardiography showed sinus rhythm, with incomplete right bundle branch block, ST segment elevation with biphasic T-waves in leads V3 and V4, and ST segment depression with biphasic T-waves in leads I, II and aVL. Chest radiography was normal. Echocardiography depicted normal left ventricular ejection fraction, with no regional wall motion abnormalities or pericardial effusion. White blood cell count was 12.5×10^9/L, C-reactive protein was 17.1 mg/L, and erythrocyte sedimentation rate was 17 mm/hr.

[1]Department of Cardiology, Asklepeion General Hospital, [2]Cardiac MR Center, Hygeia Hospital, Athens, Greece; [3]Tufts University School of Medicine, Boston, and [4]Emory University School of Medicine, Atlanta, USA.

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***Correspondence to:** Dr Antonios N Pavlidis, Department of Cardiology, Asklepeion General Hospital, V. Pavlou 1 Street, 16673, Athens, Greece.
E-mail: antonispav@yahoo.com

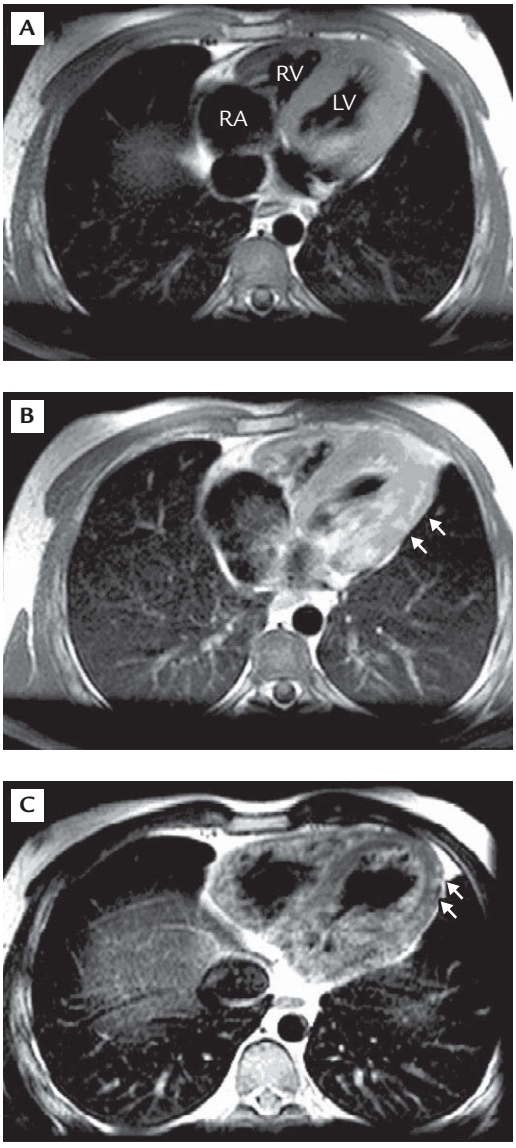

Figure. (A) T1-weighted sequence prior to contrast administration, and (B) post-contrast demonstrating patchy signal enhancement (arrows) at the lateral and inferolateral walls. (C) T2-weighted transverse image demonstrating patchy signal enhancement at the same regions, which suggested focal edema and inflammation.

Troponin I peaked at 23.2 ng/dL at 24 hours after admission. Blood cultures were negative. Antibodies to coxsackievirus, echovirus, parainfluenza virus, respiratory syncytial virus and Epstein–Barr virus (Roche Diagnostics, Germany) were not detectable. A nasopharyngeal smear, tested with real-time polymerase chain reaction (Roche Diagnostics, Germany), was positive for the pandemic (H1N1) 2009 strain of human influenza virus. CMRI demonstrated increased enhancement on the T1-weighted images and patches of edema on the T2-weighted sequence (Figure). The left ventricle was mildly dilated and left ventricular ejection fraction was 73%. The above findings were consistent with the diagnosis of acute myocarditis.

The patient was treated with high doses of ibuprofen and ramipril and his symptoms resolved within 24 hours. As the patient was not hemodynamically compromised and recovered rapidly, an endomyocardial biopsy was not performed. On discharge, he was asymptomatic and electrocardiography demonstrated T-wave inversion in most leads. On follow-up, 1 month later, he was still asymptomatic, with normal electrocardiographic and echocardiographic findings. No clinical and laboratory signs of ongoing cardiac inflammation were present, therefore, repeat CMRI was not performed.

Pandemic (H1N1) 2009 influenza virus continues to cause significant illness and death worldwide. Although influenza A virus is a well-known cause of acute myocarditis,[2] only a few cases of myocarditis due to pandemic (H1N1) 2009 virus have been reported,[3–5] and data regarding the course of the disease are still sparse. In view of the high mortality rate associated with acute myocarditis, this case emphasizes the importance of a high index of clinical suspicion in healthcare providers, to diagnose the disease early and treat it appropriately.

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