

Letter to the Editor

Myocarditis as a Complication of Influenza A (H1N1): Evaluation Using Cardiovascular Magnetic Resonance Imaging

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Although the clinical presentation of patients with H1N1 influenza has already been described in detail, there are few references to sporadic cardiac involvement during the course of the disease.¹ In particular, there are no data available concerning the prevalence of heart involvement during infection with H1N1, although there have been some references to myocarditis published in this Journal.²⁻⁵ In this report, we describe the cardiac involvement of patients with documented infection due to H1N1 influenza.

Of the 100 patients studied, 20/100 presented with mild chest discomfort, 50/100 with sinus tachycardia, 25/100 with dyspnoea and 5/100 with severe chest pain radiating to both arms. An abnormal ECG, with ST elevation equal to 1 mm in II, III and aVF, was found in 2/5 patients with severe chest pain and a normal echocardiogram (without wall motion abnormalities, pericardial effusion or impaired ejection fraction). Myocardial phosphocreatine (CK-MB) was 62 ng/ml in one and 82 ng/ml in the other (normal values <3.6 ng/ml), while troponin (Tn) was 6.9 ng/ml and 10 ng/ml (normal values <0.10 ng/ml), respectively. All patients with abnormal cardiac findings (mild chest discomfort, sinus tachycardia, dyspnoea and severe chest pain radiating to both arms) were young, with concurrent pneumonia and no history of systemic disease.

Cardiovascular magnetic resonance imaging (CMR) was performed in all 5 patients who presented with severe chest pain. Evaluation of myocardial inflammation was performed in a 1.5 T system using T2-weighted (T2-W), T1-weighted (T1-W) before and after contrast media injection and late enhanced images. Evaluation of images was performed according to previously described protocols.²⁻⁴

A small pericardial effusion was documented in 1/5 by both echo and CMR, but without evidence of myocardial inflammation. However, myocardial inflammation was documented in the two patients with abnormal cardiac enzymes and an abnormal ECG. In these two patients with abnormal cardiac enzymes, in T2-W images the ratio of heart to skeletal muscle, measured as already described in other publications,^{6,7} was 2.24 and 2.2 (normal values in our hospital 1.7 ± 0.05). Focal areas of high T2-W were not identified, because the T2 signal increase affected the myocardium globally, without much regional variation in signal.

In early T1-W images, the relative myocardial enhancement was 10.23 and 12.5 (normal values in our hospital 3.5 ± 0.62), respectively. Intramyocardial late gadolinium enhanced areas were identified in the interventricular septum and the inferior wall of the left ventricle in both of these

two patients, described as typical in previous reports⁸ about myocarditis (Figure 1). The left ventricular ejection fraction and the pericardium were normal in both patients.

In conclusion, mild cardiac symptoms were detected in a considerable number of patients with H1N1 influenza. However, we also documented the presence of pericarditis and myocarditis in a minority of them. These findings should make clinicians aware of pos-

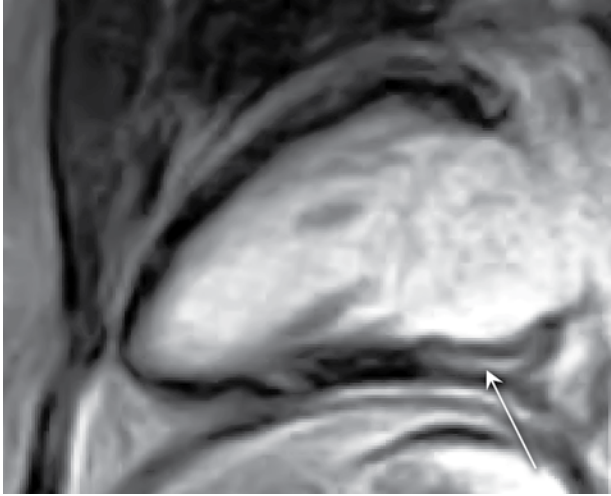


Figure 1. Late gadolinium enhanced areas (arrow) in the inferior wall of a patient with myocarditis due to H1N1.

sible pericardial/myocardial involvement during H1N1 infection and support the application of CMR as a tool for cardiac assessment in these patients, especially if the echocardiographic evaluation is negative.

References

1. Weiss TW, Stensaeth KH, Eritsland J. Myocarditis in a juvenile patient with influenza. A virus infection. *Eur Heart J.* 2010; 31: 277.
2. Yilmaz A, Klingel K, Kandolf R, Sechtem U. Imaging in inflammatory heart disease: from the past to current clinical practice. *Hellenic J Cardiol.* 2009; 50: 449-460.
3. Chrysohoou C, Tsiamis E, Brili S, Barbetseas J, Stefanadis C. Acute myocarditis from Cocksackie infection, mimicking sub-endocardial ischaemia. *Hellenic J Cardiol.* 2009; 50: 147-150.
4. Xanthos T, Lelovas P, Kantos H, Dontas I, Perrea D, Kouskouni E. Lyme carditis: complete atrioventricular dissociation with need for temporary pacing. *Hellenic J Cardiol.* 2006; 47: 313-316.
5. Karatolios K, Pankuweit S, Kisselbach C, Maisch B. Inflammatory cardiomyopathy. *Hellenic J Cardiol.* 2006; 47: 54-65.
6. Abdel-Aty H, Boyé P, Zagrosek A, et al. Diagnostic performance of cardiovascular magnetic resonance in patients with suspected acute myocarditis: comparison of different approaches. *J Am Coll Cardiol.* 2005; 45: 1815-1822.
7. Mavrogeni S, Spargias K, Markussis V, et al. Myocardial inflammation in autoimmune diseases: investigation by cardiovascular magnetic resonance and endomyocardial biopsy. *Inflamm Allergy Drug Targets.* 2009; 8: 390-397.
8. Mahrholdt H, Wagner A, Deluigi CC, et al. Presentation, patterns of myocardial damage, and clinical course of viral myocarditis. *Circulation.* 2006; 114: 1581-1590.