

Acute myocarditis mimicking acute myocardial infarction associated with pandemic 2009 (H1N1) influenza A virus

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Abstract

The prevalence of myocardial involvement in influenza infection ranges from 0% to 11% depending on the diagnostic criteria used to define myocarditis. Whether such an association holds for the novel influenza A strain, pandemic-2009-H1N1, remains unknown. The clinical presentation of myocarditis varies and often mimics myocardial infarction. Although history, physical examination, laboratory data points, and electrocardiogram are helpful in distinguishing myocarditis from myocardial infarction, differential diagnosis can sometimes be difficult. Here, we present the first known report of acute myocarditis mimicking acute myocardial infarction associated with the pandemic influenza A virus (H1N1) infection. (Cardiol J 2011; 18, 5: 552–555)

Key words: pandemic-2009-H1N1, myocarditis, mimics myocardial infarction

Introduction

Acute myocarditis is a well-recognized, albeit rare, manifestation of numerous viral infections with a broad spectrum of symptoms and clinical features [1]. The prevalence of myocardial involvement in influenza infection ranges from 0% to 11%, depending on the diagnostic criteria used to define myocarditis [2, 3]. Whether such an association holds for the novel influenza A strain, pandemic-2009-H1N1, remains unknown.

A few case reports and series [4, 5] represent the incidental diagnoses of influenza-associated acute myocarditis with the pandemic 2009-H1N1 influenza A virus infection, but the true prevalence remains unknown.

Although, there are many case reports of acute myocarditis mimicking [6–9] acute myocardial infarction, here we present the first known report of acute myocarditis mimicking acute myocardial in-

fraction associated with the pandemic H1N1 influenza A virus infection.

Case report

A 34 year-old woman was admitted to our hospital with moderate to severe retrosternal chest pain, emerging six hours before, radiating to back. The pain had an acute onset, progressive course and a compressing nature. She perceived no changes in the quality of pain with position or on inspiration. Within the past week, she had experienced fever, cough, sore throat, nausea and shortness of breath. There was no history of smoking, diabetes, hypertension, dyslipidemia, family history of coronary artery disease or cocaine abuse. Her initial heart rate was 92 beats/min; temperature 37.6°C, blood pressure 112/72 mm Hg, and respiratory rate 24 breaths/min with an oxygen saturation of 94% on room air. Cardiac examination revealed normal S1,

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S2, with no additional sounds, murmurs or pericardial rub. The rest of the physical examination was normal. Electrocardiogram (ECG) on admission revealed sinus rhythm, normal QRS axis, a Q-wave at leads II, III, aVF and ST-segment elevation at leads II, III, aVF with reciprocal ST segment depression at leads I, aVL (Fig. 1). On chest X-ray the heart size was normal and there were no signs of pulmonary pathology. Results of initial cardiac enzyme tests revealed serum troponin I of 8.5 ng/mL (normal < 1 ng/mL), creatine kinase (CK) of 272 U/L (normal 20–150 U/L), and CK-MB of 32.8 IU/L (normal < 25 IU/L). The laboratory investigations were otherwise normal except for elevated WBC (17200/mL) and ESR (68 mm/h). Echocardiography, which was done in the emergency department, showed a left ventricle with normal internal dimensions, an apparent regional hypokinesia of the posterior, middle and basal segments of the inferior wall with an ejection fraction of 45%. There was no evidence of pericardial thickening or effusion. The patient was started on aspirin, intravenous metoprolol, heparin and morphine therapy. The chest pain and ST elevation persisted despite sublingual and intravenous nitroglycerin administration, and she was taken as an emergency to the cardiac catheterization laboratory 30–35 min after admission. Coronary angiography showed normal epicardial coronary arteries (Figs. 2, 3). The left ventricular end diastolic pressure was 17 mm Hg. Her chest pain resolved spontaneously within a few hours.

As the symptoms and clinical findings of the patient fitted the criteria defined by World Health Organization for the influenza (H1N1) pandemic 2009, we collected nasal wash specimens and these were sent to the University of Istanbul Virology Laboratory working as the National Influenza Reference Laboratory (NIRL) in Turkey. Without waiting for the laboratory test results, the patient was treated with oseltamivir (Tamiflu, Roche, Basle, Switzerland) 75 mg orally twice a day for five days. Over the following days, a reverse-transcriptase polymerase chain reaction test of NIRL showed a positive result. CK and CK-MB levels peaked on the 2nd day of hospitalization at 835 IU/L and 189 IU/L respectively. Over the following days, ST-segment elevations gradually decreased and negative T-waves appeared at the same leads and precordial leads (Fig. 4).

The patient was discharged on angiotensin-converting-enzyme inhibitor and beta-blocker therapy in good condition without chest pain. Two weeks later, she remained asymptomatic and echocardiographic assessment showed that the regional wall motion abnormality was greatly improved.

Discussion

Influenza A virus-associated myocarditis is rare, with only a few cases reported in the literature [2]. Here, we present the first known report of acute myocarditis mimicking acute myocardial infarction associated with the 2009 pandemic influenza A virus (H1N1) infection.

The prevalence of influenza-associated myocarditis is not known because of a lack of comprehensive screening, with only a handful of clinical cases and autopsy findings reported in the literature [2, 3]. Whether such an association holds for the novel influenza A strain, pandemic-2009-H1N1, remains unknown.

Martin et al. [4] reviewed 123 sequential cases of patients hospitalized with pandemic-2009-H1N1 influenza A at a single academic medical center in the United States between April 1 and October 31, 2009. They identified that 4.9% (6/123) of patients had either new or worsened left ventricular dysfunction. Two thirds (4/6) of the cases had follow-up echocardiograms, and in all four left ventricular function improved. Therefore, they found that potentially reversible cardiac dysfunction is a relatively common complication associated with hospitalized pandemic-2009-H1N1 influenza. In our case, we also found that left ventricular function was highly improved in follow-up.

Bratincsak et al. [5] documented four cases within a 30-day period in 2010. Comparing them to their previous experience, they declared that it was possible that H1N1 influenza A virus was more commonly associated with a severe form of myocarditis than previously encountered influenza strains, and that those observations warranted a high index of suspicion for myocarditis in children with H1N1 influenza A infection.

However, there are many case reports of acute myocarditis mimicking acute myocardial infarction [6–9]. A thorough and detailed history, examination and ECG interpretation by an expert cardiologist is mandatory in this situation to avoid complications that may arise from inaccurate diagnosis. Despite a low cardiac risk score supporting a diagnosis of myocarditis in our patient, the localized dramatic inferior ST-segment elevation and segmental left ventricular dysfunction raised the possibility of an acute coronary syndrome. Differential diagnosis often requires cardiac catheterization. Diagnosis in these kinds of patients is difficult, as they may not have an acute coronary syndrome. A diagnosis of myocarditis needs to be kept in mind. Unfortunately, the clinical diagnosis of myocarditis remains a challenge

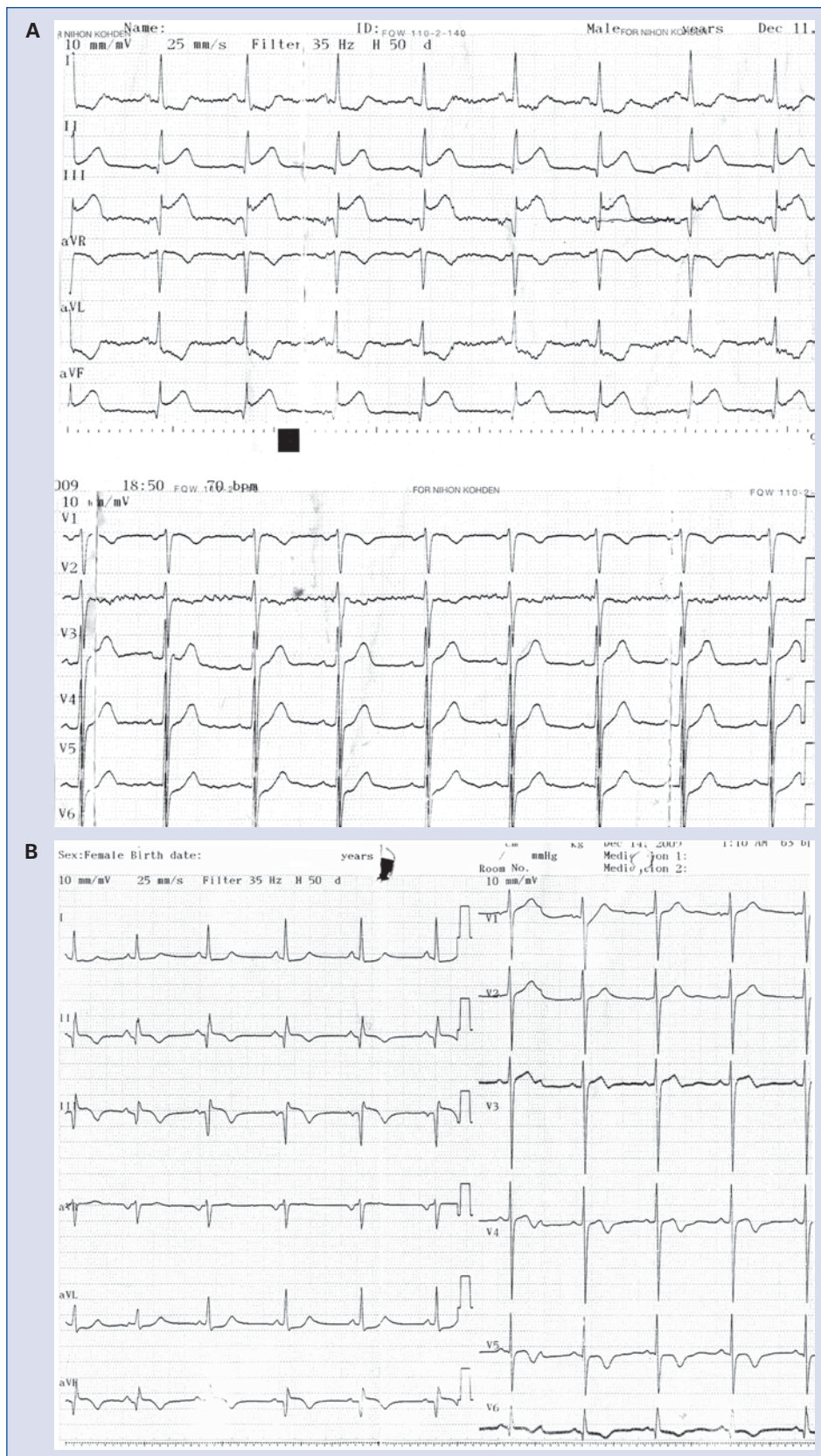


Figure 1. A. Electrocardiogram on admission revealed Q-wave at leads II, III, aVF and ST-segment elevation at leads II, III, aVF with reciprocal ST segment depression at leads I, aVL; **B.** Electrocardiogram on discharge revealed ST-segment elevations gradually decreased and negative T-waves appeared at the same leads and precordial leads.

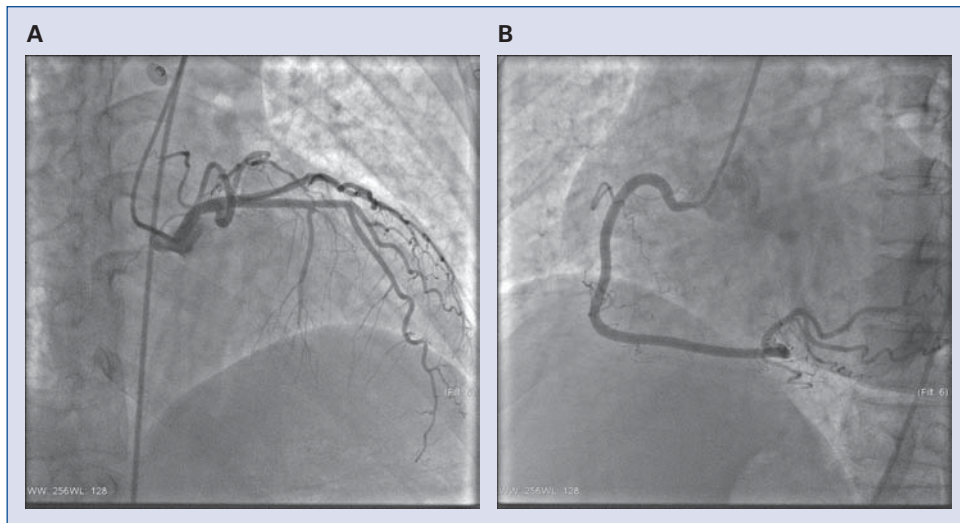


Figure 2. A. Left coronary angiogram in a right anterior oblique-cranial projection shows no significant coronary artery disease; **B.** Right coronary angiogram shows no significant coronary artery disease.

owing to the non-specific pattern of the clinical presentation and the lack of universally accepted and standardized diagnostic criteria. Primary percutaneous coronary intervention should always be the treatment of choice, since all of the preliminary signs and test results may be confused with acute myocardial infarction.

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