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Case Report

Fulminant myocarditis in an adult with 2009 pandemic influenza A (H1N1 influenza) infection

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Abstract

The worldwide outbreak of pandemic influenza A (H1N1 influenza) infection in 2009 caused numerous hospitalizations and deaths resulting from severe complications such as pneumonia, sepsis, and acute respiratory distress syndrome. Fulminant myocarditis caused by H1N1 infection has been reported in children but rarely in adults. We present an adult who contracted H1N1 infection followed by fulminant myocarditis. Early implementation of extra-corporeal membrane oxygenation support in conjunction with a specific anti-influenza viral medication (Oseltamivir) led to the patient's complete recovery from cardiogenic shock in 2 weeks.

Keywords: Fulminant myocarditis; H1N1 infection; Pandemic influenza A

1. Introduction

Fulminant myocarditis may present as fatal arrhythmias, atrioventricular (AV) block, various degrees of heart failure, and cardiogenic shock, leading to a high mortality rate in acute stage.[1] Although various types of virus have been documented to cause fulminant myocarditis, fulminant myocarditis caused by pandemic influenza A (H1N1 influenza) infection has rarely been reported, especially in adults. Herein, we report an adult who had H1N1 infection complicated with acute fulminant myocarditis and cardiogenic shock. By prompt implementation of extra-corporeal membrane oxygenation (ECMO) in conjunction with a specific anti-influenza medication (Oseltamivir), this patient completely recovered in 2 weeks.

2. Case report

In December 2009, a 44-year-old man was transferred to our hospital due to fever and cough, following worsening dyspnea for 5 days. He denied a history of cardiovascular disease and any related risk factor. At the time of admission, his blood pressure was low (85/50 mmHg) and the respiratory rate was relatively high (22/min). Physical examination revealed diffuse wheezing with crackle in breath sound, S3 gallop at the cardiac apex, jugular vein engorged, and bilateral leg edema, suggestive of heart failure and cardiogenic shock. Pulmonary edema with pleural effusion was evident on chest film (Fig. 1A). Electrocardiograph (ECG) revealed sinus or atrial rhythm with variable PP intervals and fixed RR intervals, suggesting AV dissociation with left bundle branch block-like junctional or ventricular rhythm (Fig. 2A). Laboratory data showed elevated cardiac enzymes [creatine kinase 387 U/L (normal range <160 U/L), creatine kinase MB 16 U/L (normal range <16 U/L), cardiac troponin-I 12.0 ng/mL (normal range <0.034 ng/mL)], liver enzymes [aspartate aminotransferase

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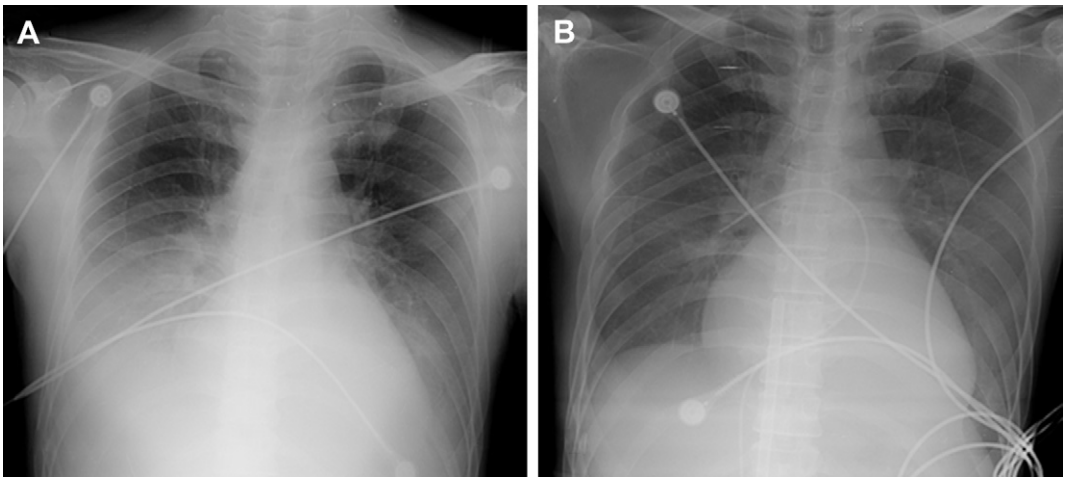

Fig. 1. Chest films taken on (A) Day 1 and (B) Day 7 of hospitalization. Note that in A, pulmonary vasculature showed marked cephalization and interstitial edema. Bilateral ground-glass pattern was compatible with the presence of pleural effusion. In B, severity of pulmonary edema and amount of pleural effusion were significantly subsided.

1587 U/L (normal range 8–38 U/L), alanine aminotransferase 1497 U/L (normal range 4–44 U/L)], and serum creatinine 2.4 mg/dL (normal range 0.7–1.4 mg/dL), indicating a shock status with multiple organ involvement. Coronary angiography demonstrated normal coronary trees. However, the left ventriculogram disclosed global hypokinesis with an ejection fraction of 27%.

Under the impression of fulminant myocarditis with cardiogenic shock and probable AV block, ECMO and transvenous temporary pacing were immediately implemented. Since rapid influenza A antigen test showed a positive result, empirical anti-influenza medication with Oseltamivir (75 mg twice daily) was concomitantly administrated for 5 days. A positive H1N1 influenza polymerase chain reaction was subsequently confirmed during the admission. On the second day of hospitalization, sinus rhythm was rapidly restored with continuous left bundle branch block but a narrower QRS complex (Fig. 2B). There was no further evidence of AV dissociation or AV block. Chest film on Day 7 showed diminished pulmonary edema and less pleural effusion (Fig. 1B). All blood biochemical parameters were progressively improved during hospital course. Seven days after implementation of ECMO, transthoracic echocardiogram (TTE) showed complete recovery of left ventricular function except for the presence of a small amount of pericardial effusion. The patient was successfully weaned from ECMO without any complication. The patient was discharged 2 weeks after admission.

3. Discussion

Viral infection remains the major cause of acute and fulminant myocarditis. Among viruses, influenza A is an extremely rare etiology.[2,3] The Chinese cohort of 426 patients who were hospitalized because of the 2009 pandemic influenza A (H1N1 influenza) infection, elevated serum creatine kinase and creatine kinase MB levels, which suggested cardiac involvement, were reported in 13% of patients.[4] However, cardiac involvement in this study was not routinely confirmed by ECG, left ventriculogram, or endomyocardial biopsy. Therefore, currently, the frequency of myocardial involvement in patients with H1N1 infection remains unclear. Information about the prevalence of H1N1 infection in Taiwan general population was also limited.[5,6] To date, only two adult patients and seven children have been reported in the literature as having acute fulminant myocarditis because of H1N1 infection.[7–9]

Although influenza-associated myocarditis carries a high mortality rate in acute stage, the survivors have excellent outcome when compared to individuals who contract other types of acute viral myocarditis.[3,10] However, during acute stage, it is still unclear which medication is appropriate for H1N1 infection complicated with myocarditis. In a series of three patients with acute influenza-associated myocarditis treated by ribavirin, two of them died shortly afterward, while the other one died 8 months later despite rapidly decline of viral titers.[11] Intravenous immune globulin was reported not to be superior to placebo in treating acute fulminant myocarditis.[12] In addition, steroid only showed marginal benefit in improving the outcomes of acute fulminant myocarditis.[13] In our patient, we used oseltamivir, a neuraminidase inhibitor, as the empirical treatment for H1N1 infection. Early administration of oral oseltamivir increases the benefits of influenza treatment.[14] However, its effects on H1N1 myocarditis and outcomes deserve further investigation.[3]

A previous study showed that ECMO was indicated in patients with profound acute lung injury with hypoxia that were refractory to mechanical ventilation support during H1N1 infection. In the above study,[15] acute lung injury secondary to either viral pneumonia or secondary bacterial infection universally prevailed in patients. In contrast, our patient was free of acute lung injury. The purpose for using ECMO in this particular case was circulatory/hemodynamic

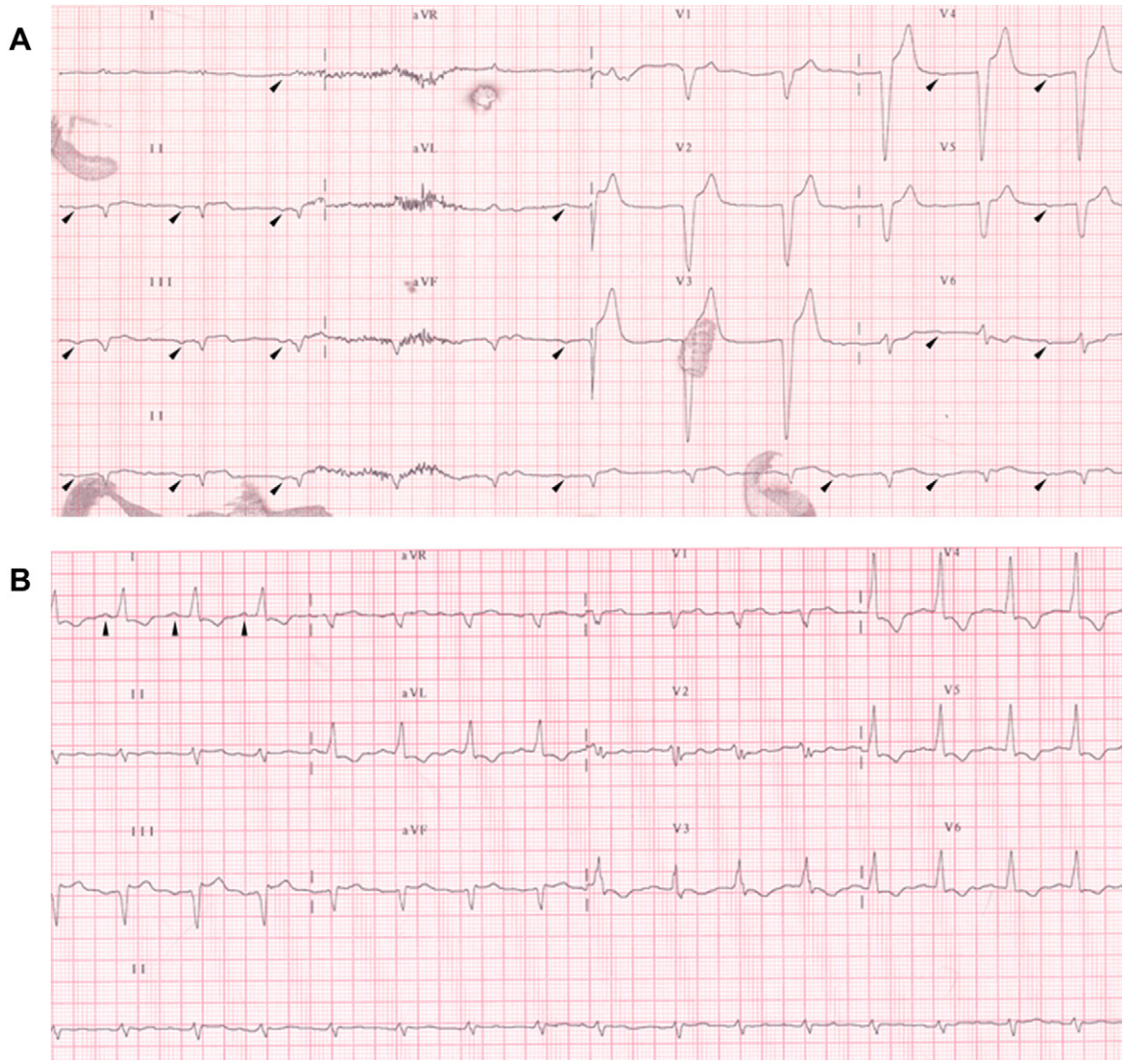

Fig. 2. ECGs performed on (A) Day 1 and (B) Day 2 of hospitalization. Note that in A, ECG revealed sinus or atrial rhythm with variable PP intervals and fixed RR intervals, suggesting AV dissociation with junctional or ventricular rhythm. Arrow heads indicate the identifiable P waves. In B, sinus rhythm with left bundle branch block was observed. Arrow heads in lead I indicate the P waves. AV = atrioventricular; ECG = electrocardiogram.

support. Al-Amoodi et al.[7] and Bratincsak et al.[8] have reported limited experiences in the use of ECMO in H1N1 infection with fulminant myocarditis. In our case, we implemented ECMO at a very early stage, resulting in a favorable outcome.

In conclusion, H1N1 infection can cause fulminant myocarditis not only in children but also in adults, as in this patient. In adult patients with acute fulminant myocarditis, H1N1 influenza should be routinely screened as a potential etiology. Aggressive cardiac support and the use of oseltamivir are essential and may improve the outcome in such patients.

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