Fulminant Myocarditis Due to H1N1 Influenza

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We report 2 cases of fulminant myocarditis caused by the H1N1 strain of influenza. As the incidence of H1N1 influenza infection continues to rise, physicians should be aware of this rare and potentially fatal complication because early diagnosis and aggressive supportive measures are imperative.

Case 1

A 52-year-old woman presented with a 3-day history of chest pain, dyspnea, diffuse myalgias, and fever. The ECG demonstrated low voltage with diffuse ST segment elevation (Figure 1). The troponin I was 5 ng/mL (0 to 0.75 ng/mL), and B-type natriuretic peptide was 1629 pg/mL (0 to 100 pg/mL). The patient was transferred to our facility for urgent left heart catheterization. On presentation, her blood pressure was 87/50 mm Hg. Coronary angiography demonstrated normal coronary arteries. Dopamine was started for blood pressure support. The patient was treated with oseltamivir 150 mg twice a day and IV ceftriaxone for suspected viral and/or bacterial myocarditis and pneumonitis. A transthoracic echocardiogram demonstrated global left ventricular systolic dysfunction with an ejection fraction of 29% and a trivial pericardial effusion. Blood cultures and rapid influenza A and B antigen tests were negative. On day 3 of hospitalization, the patient had worsening respiratory distress requiring mechanical ventilation. A repeat transthoracic echocardiogram demonstrated an ejection fraction of 10%. A milrinone infusion was started at that time. Right heart catheterization demonstrated a pulmonary artery pressure of 31/20 mm Hg and cardiac index of 1.33 L/(min·m²) by the Fick method. An

Figure 1. ECG demonstrating sinus tachycardia and low voltage with diffuse ST segment elevation in leads I, II, III, avF, and V3–V6.
intra-aortic balloon pump was placed for hemodynamic support. A norepinephrine infusion was started for persistent hypotension, and extracorporeal membrane oxygenation was performed due to persistent hypoxia, hypotension, and oliguria. On day 6 of hospitalization, a positive H1N1 influenza polymerase chain reaction was reported. After a 3-week hospital course complicated by a cardio-embolic stroke and compartment syndrome, the patient was discharged home. Echocardiography at the time of discharge demonstrated normal left ventricular function.

Case 2
A 34-year-old man presented with a 1-week history of sore throat, cough, fever, and shortness of breath. Transthoracic echocardiogram showed global hypokinesis, moderate pericardial effusion, and an ejection fraction of 15%. The patient was transferred to our hospital for further management. On admission, his pulse was 110 bpm, blood pressure was 70/40 mm Hg, temperature was 101°F, and oxygen saturation was 85% on 100% supplemental oxygen. ECG revealed sinus tachycardia with low voltage (Figure 2). The leukocyte count was 23 000 per mm³, and serum troponin I was 3.68 ng/mL. Rapid influenza A and B antigen tests were negative. The patient was resuscitated with intravenous fluid, dopamine, and epinephrine infusion and later placed on mechanical ventilation. He underwent left and right heart catheterization, which demonstrated normal coronary arteries. The pulmonary artery pressure was 47/28 mm Hg, and the cardiac index was 1.47 L/(min · m²) by Fick method. An intra-aortic balloon pump was placed for hemodynamic support, and the patient underwent surgical drainage of the pericardial effusion, with hemodynamic improvement. The patient was treated with oseltamivir 75 mg twice daily and IV vancomycin and piperacillin/tazobactam. Nasopharyngeal swab for H1N1 polymerase chain reaction was positive. A repeat transthoracic echocardiogram showed normal systolic function with a left ventricular ejection fraction of 65% on day 7 of hospitalization, and the patient was discharged home.

Discussion
In these 2 cases, the acute onset of heart failure, abnormal cardiac biomarkers, and ECG changes suggesting diffuse myocardial injury in the setting of normal coronary perfusion are suggestive of myocarditis. The prodromal symptoms coupled with positive laboratory findings are highly suggestive of H1N1 influenza as the primary cause for both the cases.

In March 2009, a novel H1N1 strain of the influenza A virus was detected. As of October 30, 2009, there were >440 000 laboratory-confirmed cases and >5700 deaths due to H1N1 infection reported to the World Health Organization in 76 countries. Estimates indicate that H1N1 may affect one half of the US population, requiring 1.8 million hospitalizations, and could result in 30 000 to 90 000 deaths.

The clinical features of H1N1 are similar to those of seasonal influenza. Although myocardial involvement in seasonal influenza infection has been reported in up to 10% of cases, the frequency of myocarditis in H1N1 influenza remains unclear. The presentation of influenza myocarditis varies from asymptomatic infection to early fulminant myocarditis, cardiogenic shock, and death. Patients typically present within 4 to 7 days of their illness with shortness of breath, pleuritic chest pain, and rarely, fulminant heart failure. Supportive care remains the mainstay of treatment. Mild cases rarely require vasopressor support, whereas fulminant myocarditis may necessitate supportive measures such as intra-aortic balloon pump and extracorporeal membrane oxygenation therapy to achieve hemodynamic stability.

In summary, we report 2 cases of fulminant myocarditis as a complication of H1N1 influenza infection. Early supportive measures including inotropic support, intra-aortic balloon pump, and extracorporeal membrane oxygenation played an
important role in patient management. At this time, the frequency of myocardial involvement with H1N1 infection is unclear. Early recognition and initiation of aggressive cardiac support is essential as full recovery may be possible in patients who survive the acute illness.

**Disclosures**

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**References**


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