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Influenza H1N1 Infection Leading To Cardiac Tamponade in a Previously Healthy Patient: A Case Report

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Abstract

Introduction: The cardiac manifestations of influenza A are broad, ranging from self-limited pericarditis to fatal cardiomyopathy. The 2009 H1N1 influenza A (H1N1) strain is a rare cause of pericarditis, and its role in developing a pericardial effusion leading to tamponade has infrequently been reported.
Case Presentation: We describe a case of a young female with no prior cardiovascular history who presents with a pericardial effusion and shock secondary to cardiac tamponade from pericarditis due to H1N1 influenza A.
Conclusions: This case highlights the potential severity of H1N1 infections and the utility of considering cardiac tamponade in patients presenting with influenza symptoms and circulatory collapse.

Keywords: H1N1, Influenza, Cardiac Tamponade, Myopericarditis

1. Introduction

H1N1 influenza A (H1N1) was initially identified in Mexico, with subsequent cases reported worldwide resulting in a pandemic alert being raised to the highest level by the World Health Organization in 2009. While the pandemic was declared over, H1N1 has resulted in significant morbidity and mortality, with estimates of 201,200 respiratory deaths and 83,300 cardiovascular deaths worldwide. A significant 80% of these deaths occurred in patients less than 65 years of age (1).

The cardiac manifestations of H1N1 are not well defined. However, case reports indicate similar presentations as other strains of influenza A (2). Here, we present a case of H1N1 infection leading to pericarditis and subsequent pericardial effusion with cardiac tamponade. Influenza causing a pericardial effusion is rare, and to our knowledge there is only one other case report of an adult patient presenting with cardiac tamponade due to an H1N1 infection (3). This case report highlights a rare but life threatening complication of H1N1 infection.

2. Case Presentation

A 22-year-old female with a distant history of alcohol and cocaine use presented to the emergency room with a one week history of significant nausea, vomiting, subjective fevers, myalgias, and fatigue. She denied chest pain, dyspnea, or cough. There was no recent illicit substance use. Her initial vital signs showed sinus tachycardia of 130 beats/min, a temperature of 36.9 degrees Celsius, a blood pressure of 75/40 mmHg and oxygen saturations of 90% on room air. Physical examination demonstrated an elevated jugular venous pressure of 9 cm. Heart sounds were difficult to hear on auscultation. The remainder of her examination was unremarkable. Initial blood work showed an elevated white blood cell count of 14.4 $\times$ 10^9/L with a neutrophil count of 13.0 $\times$ 10^9/L. Cardiac troponin I, creatinine, electrolytes, and beta-HCG were normal. Urine toxicology was negative for illicit drugs. Her initial electrocardiogram (ECG) showed sinus tachycardia with small voltages in the limb leads (Figure 1). Chest X-ray showed a generous sized cardiac silhouette and clear lung fields. She was admitted into the intensive care unit and was given fluids and ionotropic support with norepherine. Pipericillin-tazobactam and vancomycin were started empirically after blood cultures were drawn. A nasopharyngeal swab (NPS) was conducted and returned positive for Influenza A H1N1. Oseltamivir was initiated following the positive result. However, she remained hypotensive despite starting these aggressive therapies for presumed sepsis. Subsequently, an echocardiogram was performed which demonstrated a moderate sized pericardial effusion with maximal diameter of 1.5 cm in maximal width (Figure 2). There were echocardiographic features of cardiac tam-

ponade including right ventricular diastolic collapse with significant respiratory variation seen on mitral valve annular doppler velocity. An urgent pericardiocentesis was conducted and resulted in an immediate improvement of her hemodynamic status and withdrawl of all ionotropic support. A pericardial drain was inserted and a repeat echocardiogram conducted two days later demonstrated a resolution of the pericardial effusion and absence of echocardiographic findings of tamponade. The pericardial effusion was negative for bacterial cultures, acid fast bacilli, and malignancy. Serologic testing for HIV, Hepatitis B, and C were negative. High dose ibuprofen (800 mg po tid) and colchcine (0.6 mg po bid) were initiated. The pericardial drain was removed and a repeat echocardiogram was conducted within two weeks which did not show any return of the pericardial effusion. The patient was eventually discharged from the hospital in stable condition.

3. Discussion

Influenza is a severe respiratory illness which accounts for up to 300,000 deaths annually worldwide. Influenza pandemics are rare and historically 3 major pandemics have occurred: 1918, 1957, 1968, with the most severe being in 1918 (4). Influenza A and B are enveloped viruses with a segmented genome made up of eight single-stranded RNA segments which can be further subdivided on the basis of the antigenicity of the surface proteins hemagglutinin and neuraminidase. Influenza A can be further divided into 16 hemagglutinin (H1 to H16) and nine neuraminidase (N1 to N9) subtypes (5). The 2009 H1N1 influenza A strain resulted in a global pandemic from 2009-2010 resulting in significant morbidity and mortality (1). While respiratory failure remains the major cause of morbidity and mortality, emerging reports suggest cardiovascular manifestations contribute significantly to poor clinical outcomes.

The cardiac manifestations of influenza A are broad and typically occur 4 to 9 days after symptom onset. They range from self-limited pericarditis to fatal cardiomyopathy (2). However, influenza as a cause of a cardiomypathy (infectious myocarditis) is rare. In the largest case report in children, an infectious myocarditis was implicated in 0.4% of all confirmed influenza cases (6), and 0.97% of adult cases (7). Typically, clinical manifestations are mild and result in uncomplicated cases, but there have been reports of fatal cases from cardiac involvement (8). ECG abnormalities can be seen in up to 45% of individual infected with influenza, including ST deviation, T wave flattening, Q wave formation, and atrial fibrillation (9). Observational studies have suggested that influenza viruses can trigger cardiovascular death and that vaccines against influenza may decrease the risk of cardiovascular events in susceptible individuals (10). The mechanism whereby these cardiovascular complications occur is unclear. It is believed that coagulopathy; up regulation of inflammatory cytokines (11), and transient endothelial dysfunction are contributing factors (12).

Case reports of H1N1 indicate that cardiac manifestations are similar to other strains of influenza. A predominance of cases is seen in the pediatric population, reflecting the overall population group largely affected by H1N1. However, as case reports emerge, there is a strong suggestion that H1N1 can result in an infectious cardiomyopathy particularly in the pediatric population (13). In adults, myocardial dysfunction is also common with severe H1N1 infections with reports of up to 72% patients having left ventricular and/or right ventricular dysfunction (i.e infectious cardiomyopathy) (14).

Viral etiologies of pericardial effusion have been described with the most common organisms being coxsackievirus (types A and B) and echovirus in children, and cytomegalovirus, herpes viruses, and HIV in adults (15). H1N1 as a cause of pericardial effusion and cardiac tamponade is extremely rare. In our case, the patient had preceding symptoms of an influenza-like illness for seven days and presented with cardiogenic shock secondary to tamponade. We did find another reported adult case of cardiac tamponade in a 62 year old male who presented after six weeks of symptoms with a pericardial effusion requiring pericardiocentesis. The case was complicated by a chronic pericardial effusion requiring pericardiectomy (3). The only other adult case report was a 50 year old female with a pericardial effusion due to H1N1, without tamponade, of which the patient responded to medical management with high dose ibuprofen and Oseltamivir (16). The only pediatric case report was the fatal case of an 11 year old girl who developed cardiac tamponade in the context of H1N1 infection (infectious myocarditis). These cases indicate the variability in presentation with age, and potentially indicating an increased propensity to developing hemodynamic compromise with a pericardial effusion. The pathogenesis of pericardial effusion in the setting of myocardial viral infections is unclear. One study suggests viral infections can lead to the activation of inflammatory cytokines such as vascular endothelial growth factor (VEGF) and basic fibroblast growth factor (bFGF) (17), while another has shown elevation in levels of tumor necrosis factor (TNF-alpha) and (Interferon) IFN-gamma in viral pericardial disease (18). Further research is needed to identify the pathways of inflammation leading to pericardial effusions.

The optimal management of H1N1 induced pericardial effusion is unclear. Typical treatment would be supportive, with drainage of pericardial effusion in the setting of

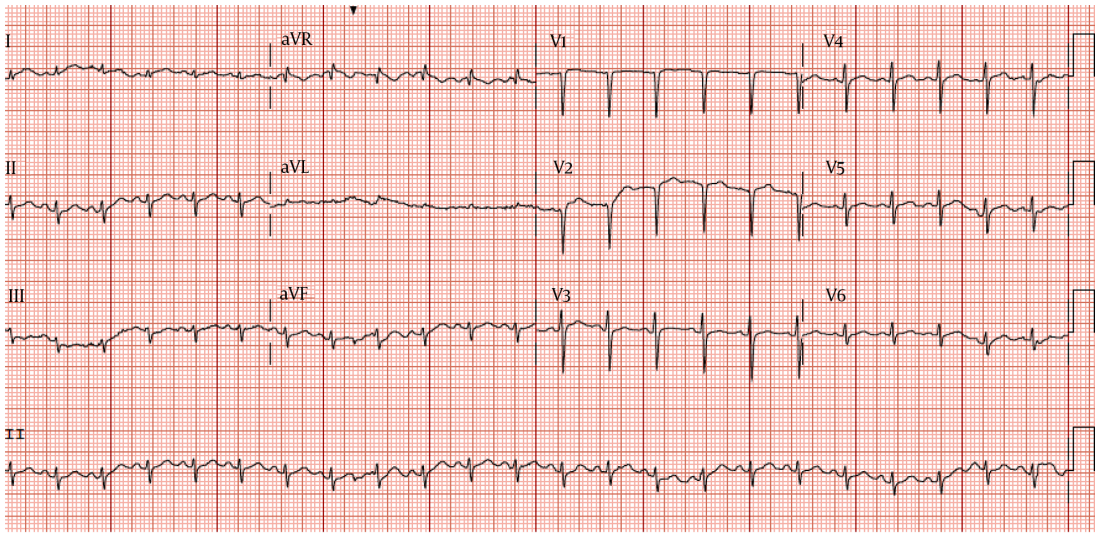

Figure 1. Electrocardiogram (ECG) of a 22-Year-Old Female, with no underlying cardiac disease, presenting with H1N1 influenza; demonstrating low voltages in the limb leads.

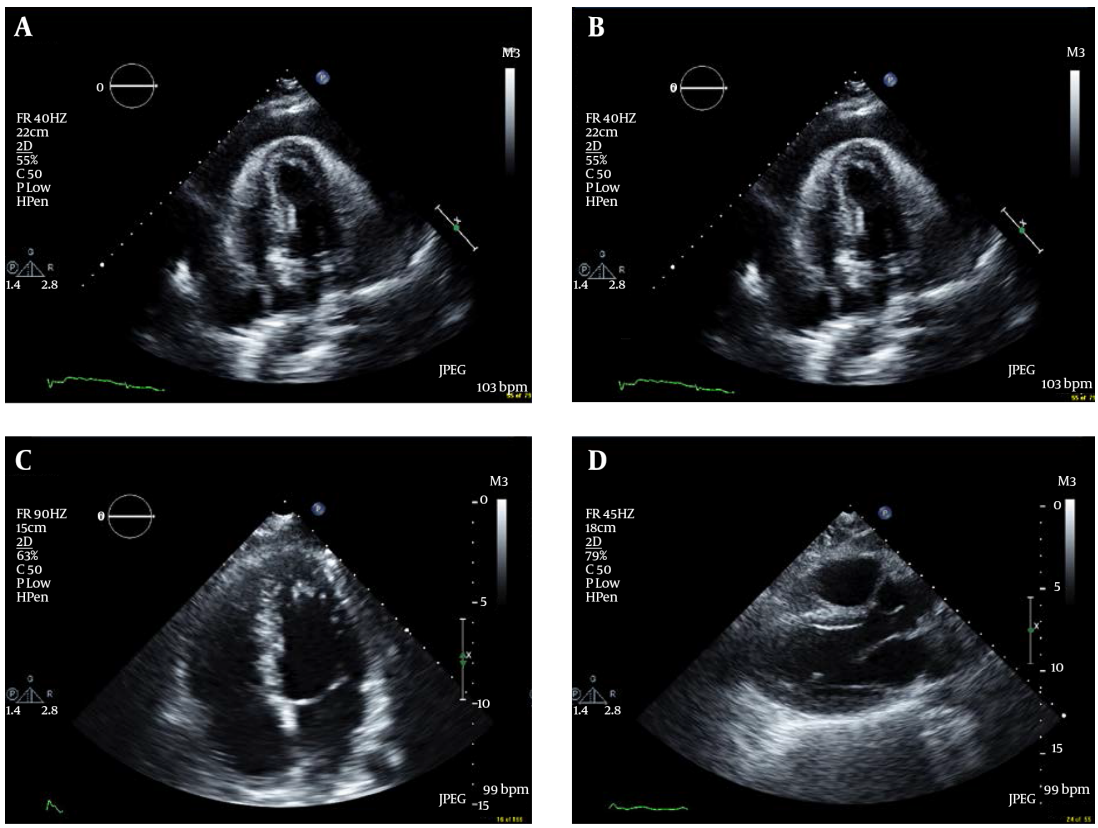

Figure 2. Transthoracic Echocardiogram Images Showing: A, four chamber view demonstrating large pericardial effusion; B, parasternal long axis view; C, four chamber view post pericardiocentesis and D, parasternal long axis post pericardiocentesis.

hemodynamic deterioration. Initiation of non-steroidal anti-inflammatory drugs and colchicine in our case was empiric, but did appear to prevent the short-term recurrence of pericardial effusion. Antiretroviral therapy with oseltamivir is unproven in this circumstance. However, as per CDC guidelines, it should be initiated in any adult with H1N1 illness requiring hospitalization, with progressive, severe, or complicated illness regardless of previous health status.

Our case report highlights an important, albeit rare complication of H1N1 infection. We present a case of cardiac tamponade in a previously healthy patient, with no known cardiac predisposition, highlighting the potential severity of influenza infections. Our case highlights an important clinical presentation of H1N1 infection that clinicians need to consider in critically ill patients.

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