Does the Influenza Vaccine Prevent Sequelae Such as Myocarditis from Developing?

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ABSTRACT

Vaccination continues to be a valuable and simple procedure to guard patients from an illness that may prevent them from completing their normal everyday tasks, missing days of work, and even lead to unnecessary sequelae. The following case describes one of the many complications that are seen on a regular basis in any community hospital in different regions of the world. The objective of this publication is to remind the public and practitioner of the urgency to vaccinate each season; thereby, curbing the virus's ability to mutate and preventing unwanted consequences such as bacterial super infection or myocarditis.

Key words: Complication, Influenza, Length of stay, Vaccination

INTRODUCTION

Infection due to influenza virus is a major burden on the healthcare system and in many cases is associated with severe complications. Nearly 60% of the flu-associated hospitalizations reported to CDC's influenza surveillance system belonged to the age group of 18-64 year.[1] In the United States alone, the population-based risk for hospitalization due to influenza infection complications is as high as 150 per 100,000 annually.[2] Severe complications have been well-documented and mostly involve the development of pneumonia. Other more rare cases involve neurological complications, myositis, rhabdomyolysis, and cardiac complications; including myocarditis and in some cases myocardial infarctions.[3]

Myocarditis is an inflammatory disease of cardiac muscle, in developed countries; viral infections most commonly cause this potentially devastating condition. It can cause acute heart failure, chronic dilated cardiomyopathy, sudden death, and is underdiagnosed in the United States. One article reports that the involvement of the myocardium due to influenza has been reported to be 0-11%, depending on the standards used to define it.[4] Another paper reports that the frequency of myocarditis in severely-ill patients due to previous seasonal influenza virus infections is 0.4-13%,[5] incidence of pandemic influenza associated myocarditis is reported to be even higher. We report a case of influenza A in a 35-year-old male complicated by acute myocarditis and a superinfection of community-acquired pneumonia (CAP).

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CASE REPORT

The patient is a 35-year-old male, with a past medical history of nephrolithiasis and idiopathic vertigo. He had no prior influenza virus vaccination. He was discharged from another hospital the day prior with a diagnosis of CAP and was given doxycycline.

One day following discharge, the patient reported to the emergency room (ER) with syncope after waking up with shortness of breath. He also reported having fever, chills, nausea, vomiting, and palpitation. He has no family history of sudden cardiac death with syncope.

Physical examination revealed that the patient had a temperature of 101.6 F, heart rate of 98 beats/min in sinus rhythm, blood pressure of 102/66 mmHg, with respirations of 24 breaths/min. Patient was alert and oriented times three, and pupils equal, round, and reactive

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to light. No signs of dehydration present in mucous membranes. Jugular venous distention was 12 cmH_2O and no carotid bruits present. Cardiovascular examination was performed which was evaluated to be in regular rate and rhythm with normal S1 and S2 sounds, with a S3 and S4 auscultated. Electrocardiograph was done and showed a sinus rhythm of 119 with multifocal preventricular contractions, and nonspecific ST and T wave changes followed by ventricular tachycardia (VT). Chest radiograph revealed left atrial enlargement, with no acute infiltrates. Bilateral crackles were auscultated on lung fields, all else was noncontributory. Chest computed tomography (CT scan) showed small left lower lobe consolidation. While in the ER he developed VT patient was defibrillated twice and went back into sinus rhythm.

The patients had creatine kinase-MB 11.5 ng/mL and a troponin of 0.12 ng/mL. Comprehensive metabolic panel revealed elevated serum glucose at 142 mg/dL and transaminasemia with an aspartate aminotransferase (AST) of 255 IU/L and alanine transaminase (ALT) of 105 IU/L. B-type natriuretic peptide was 185 ng/L. Patient was transferred to the coronary care unit and a cardiac catheterization demonstrated non-obstructive coronary artery disease with a low ejection fraction of approximately 30%. Cardiac magnetic resonance imaging was done and showed four-chamber enlargement with diffuse generalized dysfunction. No focal areas of enhancement were seen to suggest sites of inflammation, infection, or scarring. Legionella and *Streptococcus pneumoniae* testing was found to be negative. Mycoplasma IgM was <770. Blood cultures were all negative. Cytomegalovirus was also negative. Serology for influenza virus was found to be positive for influenza A.

Patient was started on furosemide 80mg intravenously (IV) and then 12 h later was changed to 40 mg IV every 12 h for acute biventricular systolic heart failure. Amiodarone was started at 900/500 mg in D5W 1mL/min IV for 6 h, then 0.5 mL/minute for the VT. Vancomycin and cefepime per IV were also started for the CAP and fever.

DISCUSSION

There are many severe complications which can develop with influenza A infection, included in these are pulmonary manifestations, musculoskeletal,[6] neurologic, and cardiac. The chance of developing a severe complication dramatically increases depending on the patient's age, and associated comorbidities or other risk factors such as age, obesity, and asthma.[7] The majority of these hospitalizations could be eliminated with a yearly influenza vaccination. Particular emphasis on the importance of vaccination should be given to those with the abovementioned risk factors. In a moderately, well-vaccinated population of older adults, laboratory-confirmed influenza virus accounted for 9.3% of all respiratory hospitalizations, and influenza vaccination prevented 61.2% of such hospitalizations.[8] In healthy individuals, influenza vaccine prevents 50-80% of infections with influenza depending on how well the match of the specific subtype has been predicted for that specific season and virus.[9]

The fact that the "gold standard" for diagnosis is endomyocardial biopsy, which is very invasive and is prone to sampling errors and an undefined sensitivity, is another explanation for underreporting of myocarditis in influenza infections.[10] Hence, other methods of detection are used including echocardiography, MRI, and coronary angiography. The male presented in this case was diagnosed with acute myocarditis based on the instrumentation, along with clinical manifestations, including the acute onset of heart failure, elevated cardiac enzymes, and transient cardiac global dysfunction. The influenza A virus was detected in the patient using serology and ideally a convalescent titer 4 weeks later. The patient's clinical course coupled with the detection of influenza A suggests the viral infection as the primary cause of the myocarditis.

CONCLUSION

It should be recognized that influenza A is a major reason for serious hospitalizations and increased length of stay for reasons including myocarditis, bacterial pneumonia, asthma, or bronchitis exacerbations. The majority of these hospitalizations and long duration length of stays could be prevented with the seasonal vaccination offered in a number of locations.

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