# Transient AV Block as a Hemodynamic Complication of the Influenza A Virus: A Case Report

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**Influenza virus causes annual epidemics of respiratory illness characterized by sudden onset of fever, malaise, myalgias, headache, cough, and other respiratory complains. Each year in the United States, it is estimated that this debilitating respiratory illness accounts for 294,000 excess hospitalizations and 36,000 attributable deaths. Epidemiological studies describe increased cardiovascular mortality during influenza seasons. Cardiovascular involvement in acute influenza infection can occur through direct effects of the virus on the myocardium or through exacerbation of existing cardiovascular disease. The purpose of this report is to document a transient atrioventricular (AV) block with hemodynamic compromise after infection with the influenza virus in a patient with underlying cardiac disease without myocarditis. [*P R Health Sci J 2016;35:173-175*]**

*Key words: AV block, Influenza A, ECG*

Influenza virus causes annual epidemics of respiratory illness characterized by sudden onset of fever, malaise, myalgias, headache, cough, and other respiratory complains (1). Influenza A and B viruses are spherical or long shaped enveloped viruses with a segmented genome made of eight single stranded RNA segments of 890 to 2341 nucleotides each. Influenza A is further subdivided into 16 hemagglutinin (H1 to H16) and nine neuraminidase (N1 to N9) subtypes on the basis of the antigenicity of the surface proteins hemagglutinin and neuraminidase (2,3). Transmission of human influenza virus occurs by inhalation of infectious droplets of airborne droplet nuclei and, perhaps, by indirect contact followed by self-inoculation of the upper respiratory tract or conjunctival mucosa (4). Epidemiological studies describe increased cardiovascular mortality during influenza seasons (5). Each year in the United States, it is estimated that this debilitating respiratory illness accounts for 294,000 excess hospitalizations and 36,000 attributable deaths (1,3). The annual rate of influenza-associated deaths in the United States overall between 1976-2007 ranged from 1.4 to 16.7 deaths per 100,000 persons (6). In Puerto Rico by the end of 2015 there were 20,147 cases reported constituting 579 cases per 100,000 persons and mortality rate of 0.23 deaths per 1000,000 persons (7,8). The most common complication of influenza virus infection relates to extension of the viral infection distally to the lung, resulting in pneumonia (4). Cardiac involvement is variable and mostly seems to be related to myocarditis with a variety of fatal and nonfatal arrhythmias (1,9,10). The purpose of this report is to document a transient AV block with hemodynamic compromise after infection with the influenza virus in a patient with underlying cardiac disease without myocarditis.

## Case Report

An 85 year-old man presented to the emergency room complaining of chest discomfort, shortness of breath, unquantified fever, arthralgias, cough and poor oral intake for the past five days; his son and wife also had similar symptoms two weeks prior. Past medical history included heart failure with diastolic dysfunction, arterial hypertension and diabetes mellitus type II. Physical exam showed a heart rate of 50 beats/min, temperature 98°F, blood pressure 146/61mmHg, and respiratory rate 18 breaths/min. Cardiac examination was remarkable for a normal S1, S2, with no additional sounds, murmurs or pericardial rub. The rest of the physical examination was unremarkable. On chest radiograph, no cardiopulmonary process was noted and cardiac markers returned negative. A rapid test came back positive for Influenza A virus and oseltamivir 75mg orally was immediately started. He was admitted to the Internal Medicine ward for intravenous hydration in view of poor oral intake. On his 2nd day of hospitalization he developed worsening cough, severe hypoxemia, fever and respiratory failure requiring endotracheal intubation and transfer to the intensive care unit (ICU). In lieu of deterioration, broad spectrum antibiotics were started. Ventilation/Perfusion Lung Scan showed no evidence of pulmonary emboli. On the ICU, he presented with hypotension and bradycardia with heart rate of

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40 beats-per-minute. An electrocardiogram revealed a new 2:1 AV conduction (figure 1A) that showed evidence of dissociation while the patient was being bathed.

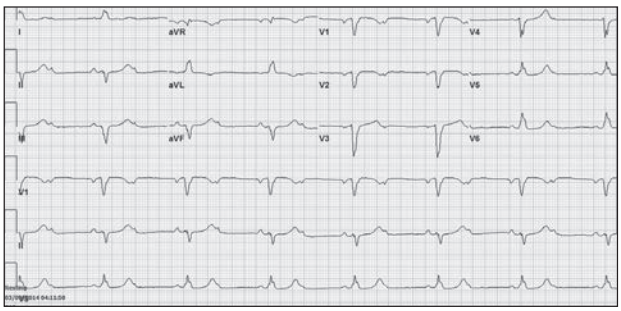

**Figure 1A**. Electrocardiogram on Intensive Care Unit demonstrating 2:1 AV conduction requiring temporary transvenous pacemaker.

Due to hemodynamic collapse a temporary transvenous pacemaker was inserted and dopamine infusion started. Cardiac markers remained negative. On the following days he remained dependent of the pacemaker due to persistent AV block. A transthoracic echocardiogram was performed but the patient had a poor echocardiographic window that precluded an adequate structural and functional cardiac assessment. Finally, on the 8th day dopamine infusion stopped and he was successfully extubated but remained dependent of the temporary transcutaneous pacemaker. He resumed 1:1 AV conduction (figure 1B) on the 10th day and the transvenous pacemaker was removed. The patient was transferred to the medicine ward where he remained stable and successfully discharged home after 16 days of hospitalization.

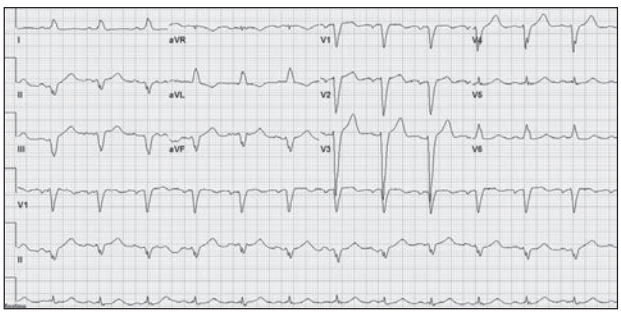

**Figure 1B**. Electrocardiogram upon resolution of infection and restored 1:1 AV conduction with old inferior myocardial infarction and left bundle branch block.

## Discussion

Viral influenza is a well established cause of seasonal illness, generally characterized by acute onset of fever, myalgias and respiratory symptoms. In healthy persons, disease usually resolves inconsequentially (11). During influenza season, complications of influenza may be difficult or impossible to distinguish from primary cardiac disease with incidental influenza (12). Cardiovascular involvement in acute influenza infection can occur through direct effects of the virus on the myocardium or through exacerbation of existing cardiovascular disease (10).

Acute influenza infection is an independent risk factor for fatal and nonfatal cardiovascular events, the mechanism underlying the risk is less clear but may relate to triggering the rupture of a vulnerable atherosclerotic plaque, fluid overload heart failure, myocarditis, arrhythmia, or the susceptibility of a frail and vulnerable patient (13,14). Also, adverse cardiovascular events may arise due to fever, vasodilation, hypovolemia, hypoxia, proinflammatory cytokine elaboration and procoagulant effects (1,15). Randomized controlled trials have demonstrated reduced risk of cardiovascular death and some coronary ischemic events with influenza vaccination in populations with pre-existing cardiovascular disease (3,14). A prospective study by Ison et al. found that previously healthy ambulatory young adults with acute uncomplicated influenza have frequent but clinically insignificant abnormal ECG findings during the early stage of illness which resolve promptly and are not associated with changes in markers of cardiac injury or echocardiogram findings (1). In murine model of acute influenza A virus infection by Kotaka et al. left ventricular dysfunction occurred in all the mice, irrespectively if they had myocarditis. In this experimental sturdy extrasystoles and conduction abnormalities were not documented in routinely recorded ECGs (16). Oseltamivir drug profile describes cardiac arrhythmias in less than 1% of the cases while nausea, vomiting, bronchitis, insomnia and vertigo where frequently seen during prophylaxis studies (17). Patients with influenza whose condition deteriorates or in which there is hemodynamic compromise, cardiac involvement should be considered and appropriate investigations should be initiated (10).

In our patient the diagnosis of myocarditis could not be made definitely as cardiac markers remained normal and there was no objective evidence that could suggest direct cardiac involvement. We consider that the presence of a new AV block could be an indirect result from the influenza infection by precipitating a critically ill phase, which imposed stress to an already diseased His-Purkinje network and led to a 2:1 AV conduction. To our knowledge this is the first case of influenza virus causing hemodynamic compromise due to a transient AV block by indirect cardiac involvement documented in Puerto Rico. This case should remind the clinicians the importance of yearly vaccination in patients with high risk for cardiac complications. Also to identify those with underlying cardiac disease and symptoms suggestive of influenza for early testing and installment of antiviral medications when appropriate. Failure to identify reversible cardiac involvement may result in avoidable complications including death.

## Resumen

El virus de la influenza causa epidemias anuales del tracto respiratorio caracterizadas por síntomas repentinos de fiebre,

malestar general, mialgias, dolor de cabeza, tos y otras quejas respiratorias. Cada año en los Estados Unidos se estima que esta enfermedad respiratoria debilitante resulta en un exceso de 294,000 hospitalizaciones y 36,000 muertes. Estudios epidemiológicos describen un aumento en la mortalidad cardiovascular durante las temporadas de influenza. El envolvimiento cardiovascular puede ocurrir através de sus efectos directos en el miocardio o por exacebación de enfermedad cardiovascular precedente. El propósito de este manuscrito es describir un bloqueo atrioventricular transitorio con compromiso hemodinámico como una complicación del virus de influenza en un paciente con enfermedad cardiaca conocida sin miocarditis.

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