

**SWINE FLU
EMERGING THREAT****VIEW POINT****CVS Complications of Influenza A(H1N1)****Pawan Suri**

Influenza A infection is a debilitating respiratory illness rarely affecting the CVS. Influenza A and B viruses are enveloped viruses with a segmented genome made up of eight single-stranded RNA segments of 890 to 2341 nucleotides each (1). 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. It is estimated that 50% of those infected with influenza virus have no clinical symptoms, while in the remaining 50%, clinical presentation varies from afebrile respiratory symptoms (similar to the common cold) to febrile illnesses causing disorders affecting the lung, heart, brain, kidneys and liver (2). Every year, the global burden of influenza is believed to be three to five million cases of severe illness and up to 300,000 deaths annually. Myocardial involvement is a rare complication of influenza. Several studies using a molecular biological approach have reported that different cardiotropic viruses are implicated in most cases of human myocarditis. It is uncertain whether myocyte damage in the early phase of the disease is linked primarily to the viral presence or to immunomediated damage. However, it is now widely accepted that progression of the disease is mainly sustained by immunomechanisms. Proinflammatory cytokines, including tumour necrosis factor-alpha, have been recognized as important factors in the initiation and development of the pathophysiology of inflammatory cardiomyopathies. An association between depressed myocardial function and elevated TNF-alpha messenger RNA and protein levels in myocardium has been previously demonstrated in patients with myocarditis (3). TNF-alpha may depress myocardial contractility through a number of mechanisms, including NO-mediated mechanisms and direct actions on intracellular calcium handling in the myocardium. Spontaneous resolution in cardiac function has been reported in up to 40% of patients with viral myocarditis (4), although the role of immunomodulation remains uncertain in these cases. Cardiovascular involvement in acute influenza infection can occur through direct effects of the virus on the myocardium or through exacerbation of existing cardiovascular disease. Epidemiological studies have demonstrated an association between influenza epidemics

and cardiovascular mortality and a decrease in cardiovascular mortality in high risk patients has been demonstrated following vaccination with influenza vaccine. Influenza is a recognized cause of myocarditis which can lead to significant impairment of cardiac function and mortality (5). With recent concerns regarding another potential global pandemic of influenza the huge potential for cardiovascular morbidity and mortality is discussed. Myocardial involvement in influenza infection must be considered in patients presenting with raised cardiac markers or an abnormal ECG. An echocardiogram in these patients is warranted at an early stage to look for potentially fatal complications, such as cardiac tamponade, and to assess LV function to further guide treatment. Furthermore, cardiac involvement should be considered in any patient who becomes acutely more breathless or hypotensive during infection with influenza, and further cardiac investigations in these patients are warranted. In early 2009, emerging of swine flu brings attention to medical scientists around the world. Finally swine flu is classified as a new variant of H1N1 influenza virus infection. Since H1N1 influenza virus infection is already confirmed for cardiac involvement (6) the concern of swine flu infection is important in cardiology. Although there is no present specific report mentioning for cardiac manifestations in swine flu and it is needed to closely monitor all infected cases for cardiac involvement

References

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