Cardiac Conduction System Affection in a Case of Swine Flu

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Abstract

We present a case of swine flu presenting as bilateral pneumonia with involvement of cardiac conduction system in the form of increased PR interval and sinus bradycardia during the initial course of disease process. To the best of our knowledge, affection of conducting system in a case of swine flu has not been reported in the literature so far.

Introduction

Cardiac conduction system involving SA node and AV node has been found to be affected by various systemic disorders including infections (Viral myocarditis, endocarditis, Lyme disease, Chagas disease, Diphtheria, tuberculosis, syphilis, rheumatic fever etc.), infiltrative (Amyloidosis, haemochromatosis, sarcoidosis), collagen vascular disorders, drug toxicity and post cardiac surgery.

Affection of conducting system and myocarditis by various viral disorders in not a rare entity. This has been reported in coxsackie virus, EBV virus, adenovirus, hepatitis C, HIV etc. Influenza A infection is a debilitating respiratory illness rarely affecting the Cardiovascular system.

Influenza A and B viruses are enveloped viruses with a segmented genome made up of eight single-standard 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. Cardiac involvement has been reported in cases of swine flu.2,3 Epidemiological studies have demonstrated an association between influenza epidemics and cardiovascular mortality.

Case Report

A patient, 42 years old male, presented in the department of cardiology as a case of accelerated hypertension with shortness of breath and low grade fever for three days. Patient had past history of hypertension with no documents available. There was no history of tuberculosis, diabetes or any other significant illness. At the time of admission, patient was conscious, oriented. His vital signs revealed pulse = 102/min regular, blood pressure = 220/120 mmHg, respiratory rate = 34/min with mild cyanosis. Jugular venous pressure was normal. Respiratory system examination revealed bilateral coarse crepts and rhonchi. Other system was normal except the presence of soft left ventricular third heart sound on cardiac auscultation. On investigations, sputum was positive for H1N1 virus. At the time of admission, Hb=12.3g%, TLC=4300 cells/mm3, polymorphs were 83%, ESR=50mm of 1st Hour, Blood sugar=61.0 mg/dl, blood urea=182.0 mg/dl, S.creatinine=1.6mg/dl, CPK-MB=27.7 IU/L, S.Sodium=138.8 meq/dl, S.Potassium=5.1 meq/dl, S.calcium=8.9 meq/dl, S.PH=7.30, blood urea=15.0 mg/dl, S.creatinine=1.0 mg/dl, CPK-MB=3.0. Electrolytes were within normal limits during the further course of the disease. Electrocardiogram (Fig.1a) on day 1 showed HR=88/min, regular, normal axis, LVH, T in lead I and aVL, on day 3rd electrocardiograph (Fig.1b) showed HR=90/min, regular and PR interval=0.28 sec. on day 4th electrocardiograph (Fig.1c) showed severe sinus bradycardia with heart rate of 36/min, PR interval was 0.28 sec, QTC=49 sec, and QRS duration was 0.12 sec. Thus, patient had involvement of cardiac conduction tissue involvement on the 3rd, 4th and subsequent days of disease course. Patient died on 9th day.

Discussion

We have reported a case of swine flu presenting as bilateral lobar pneumonia with involvement of cardiac conduction system. In our case, PR interval was normal during first three days of onset of symptoms. On 4th day, we found an increase in PR interval (0.28 s) with deterioration in patient’s clinical status with refractory hypotension not responding to vasopressors. On 7th day, patient developed sinus bradycardia with heart rate of 36/min, PR interval of 0.28 sec. Initially patient had hypoxia, increased blood urea, normal electrolytes and respiratory and metabolic acidosis, which improved with treatment. The temporal sequence of increase in PR interval and sinus bradycardia after 3- days of onset of symptoms is suggestive of progressive increase in involvement of conducting tissue and

9. Centers for Disease Control and Prevention. Interim guidance for infection control for care patients with confirmed or suspected novel influenza A (H1N1) virus infection in a health-care setting. Available at: www.cdc.gov/h1n1flu/guidelines_infection_control.htm.
severity of disease, culminating into a fatal outcome.

It suggests that in the terminal phase, SA node and AV node affection was preferentially more than the inter nodal connecting pathways, bundle branch or Purkinje fibres. It may be a rare manifestation. In our experience, at our institute till now, we had 27 confirmed cases of swine flu, and among those, only one patient had conducting system involvement.

A total of 9 cases of influenza myocarditis were diagnosed during the winter epidemic of influenza 1998-1999. But exact incidence and predisposition of influenza virus to cardiac conducting system without myocarditis has not been reported in the literature so far.

Bratiniscak. A et al has raised the possibility of associated severe form of myocarditis than previously encountered influenza strains.5

Fulminant myocarditis caused by a viral infection in uncommon and influenza A virus associated fulminant myocarditis is “extremely rare with only a few cases reported in the literature”. Patients with fulminant myocarditis can present with fatal arrhythmias, atrioventricular blocks and or varying degree of cardiogenic shock. The exact potential mechanism for conduction tissue abnormalities remains to be postulated. Whether it is due to inflammatory edema pressing the AV node and other conduction tissue or primarily directly affecting this conduction tissue/AV nodal pacemaker cells resulting into resting membrane potential changes to explain conduction abnormality remains to be verified.5,7

Relative bradycardia has been reported in various viral hemorrhagic fevers but affection of conduction tissue involvement has not been demonstrated, through postulated to be caused as a result of either a part of myocarditis or immune related.5-8

References


Influenza A (H3N2) Associated Acute Necrotising Encephalopathy

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Abstract

We present a case of 16 year old female admitted with complaints of influenza like symptoms followed by convulsions and sudden impairment of consciousness. Magnetic resonance imaging abnormalities were found in bilateral thalami including cerebellum. Diagnosis of influenza associated acute necrotizing encephalopathy was made on the basis of clinical features, neuroimaging findings and isolation of influenza A(H3N2) virus from throat swab. This is probably first case of Influenza associated acute necrotizing encephalopathy reported in India in 2009.

Introduction

Influenza virus associated encephalopathy is a disease of young children. It has mostly been reported from Japan and Taiwan, but cases have been reported from Europe and North