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Research Paper

H1N1 Positive Case Represent As Cardiogenic Syncope In Cardiac Emergency Department.

Medical Science

KEYWORDS : swine flu, sinus bradycardia, syncope.

Dr. Naresh Sen — Ex- Cardiologist- Narayana Hrudayalaya, Bangalore & Jaipur Consultant Cardiologist -Anand Multispeciality Hospital,Beawar (Rajasthan)India-305901

Dr. Sonal Tanwar — Consultant -Anand Multispeciality Hospital,Beawar (Rajasthan)India-305901

ABSTRACT

36 Year old man was reffered to our cardiac center with severe breathlessness. But on the time of admission he had an episode of syncope in the emergency department. He is a known case of H1N1 Positive (swine flu) presenting as bilateral pneumonitis with abnormal cardiac conduction in the form of prolonged PR interval and sinus bradycardia during the initial course of disease process. 2D Echocardiography revealed poor LV Systolic function which became normal after 8 days of intensive management with mechanical ventilator support. Clinically and electrocardiographically he was improved without support of pacing and discharged safely on optimized treatment.

Introduction

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. 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. Epidemiological studies have demonstrated an association between influenza epidemics and cardiovascular mortality.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 coxasackie virus, EBV virus, adenovirus, hepatitis C, HIV etc. Influenza A infection is a debilitating respiratory illness rarely affecting the Cardiovascular system. 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.

Case Report

A 36 years old male was reffered from periphery at our cardiac centre with complain of severe breathlessness and low grade fever for three days. Patient had no past history of hypertension , diabetes, tuberculosis, thyroid disorders, copd or asthma .Also he had no history of stroke, trauma, tumor and surgery. At the time of admission, patient was unconscious due to syncope. His vital signs revealed pulse = 52/min regular, blood pressure = 98/60 mmHg, respiratory rate = 38/min with mild cyanosis. Immediately ECG was done which revealed proloned PR interval with sinus bradycardia. Jugular venous pressure was subnormal. 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=13.1 gm%, TLC=11260 cells/mm3, polymorphs were 79%, ESR=46mm of 1st Hour , Blood sugar = 98.0 mg/dl, blood urea=112.0 mg/dl, S.creatinine=1.4 mg/dl, CPK-MB=22.3 IU/L, Troponin-I value was normal, S.Sodium = 135.2 meq/dl, S.Potassium=4.9 meq/dl, S.calcium=9.1 meq/dl, SPO2=72% ,LFT was normal, pH=7.12 with respiratory and metabolic acidosis in arterial blood gas analysis. Initially (at previous centre) patient was treated as a case of left ventricular failure but patient did not respond to decongestive therapy and had persistent low arterial oxygen saturation. On the basis of disproportionate and persistent signs and symptoms, diagnosis of bilateral bronchopneumonia had been considered. Chest X-ray PA view showed bilateral heterogenous infiltration in lower zones with cardiothoracic ratio of 0.6(Fig-1).

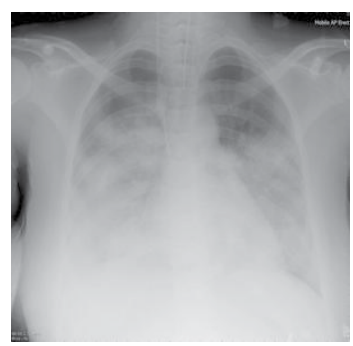

Figure-1

(Chest X-ray PA View showed bilateral heterogenous infiltration in lower zones with cardiothoracic ratio of 0.6)

Echocardiography showed mild global hypokinesia without LV wall thining and LVEF=45% (Fig-4A). and normal valvular structures. Patient was intubated and shifted to cardiac ICU and was kept on mechanical ventilator. On 2nd day of admission, repeat investigations showed S.Sodium =141.0 meq/l, S.Potassium =4.7 meq /l, SPO2= 94%, pH=7.30, blood urea=25.0 mg/dl, S. creatinine= 1.1 mg/dl, CPK-MB=4.8. Electrolytes were within normal limits during the further course of the disease . Electrocardiogram on day 1 showed sinus bradycardia HR=52/min, regular, normal axis and prolonged PR interval 280 msec (Fig-2) ,on day 3rd electrocardiograph showed HR=66/min, regular and PR interval 210 msec. on day 8th electrocardiograph showed heart rate of 89/min, PR interval was 132 msec. We started Oseltamivir(Tamiflu) from Day 1 and other medical management with support of mechanical ventilator and proper periodic vital monitoring. We planned to place temporary pacemaker to the patient to resolve the symptoms but after intensive cardiac care of 8 days patient improved clinically. Also at the time of discharge ECG and 2D Echo revealed normal sinus rhythm and good LV systolic function- LVEF=68% (Fig-4B).

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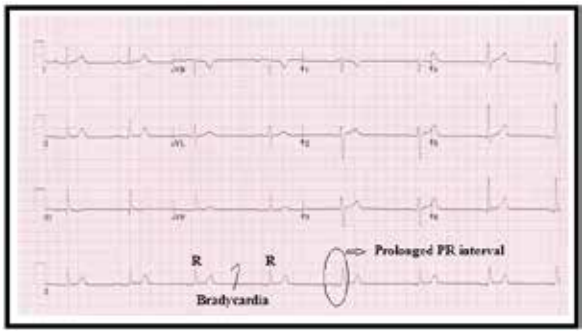

Figure-2

(ECG showed sinus bradycardia HR=52/min, regular, normal axis and prolonged PR interval 280 msec.)

Figure-3

(ECG showed normal sinus rate rhythm and normal PR interval .)

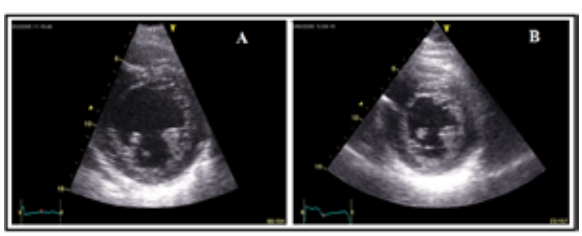

Figure 4 A and 4B (2D Echo short axis view)
Echocardiography showed mild global hypokinesia without LV wall thining and LVEF=45% (Fig-4A).
2D Echo revealed normal sinus rhythm and good LV systolic function- LVEF=68% (Fig-4B).

Discussion.

We have reported H1N1 positive case presented as cardiogenic syncope in Cardiac Emergency department. He was a known case of swine flu presenting as bilaterial lobar pneumonitis with abnormal cardiac conduction system. Electrocardiographically ,we found an increase in PR interval (278 msec) with deterioration in patient's clinical status with refractory hypotension not responding to vasopressors. Initially patient had hypoxia, increased blood urea, normal electrolytes with respiratory and metabolic acidosis, which improved with treatment. The temporal sequence of increase in PR interval and sinus bradycardia after onset of symptoms is suggestive of progressive increase in involvement of conducting tissue and 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. We started Oseltamivir(Tamiflu) and other medical management with support of mechanical ventilator and proper periodic vital monitoring. In initial 2-3 days, patient had episodes of sinus bradycardia due to conduction defect and poor LV Systolic function due to viral myocarditis. We planned to place temporary pacemaker to the patient to resolve the symptoms but after intensive cardiac care of 8 days patient improved clinically with resolved ECG Conduction defect. Also 2D Echocardiography of the patient revealed improved LV systolic function.

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

Conclusion

H1N1 positive cases may cause fulminant myocarditis and cardiac conduction defect which may further lead to fatal conditions. So awareness for the same and its timely aggressive management is mandatory to decrease the morbidity and mortality.

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