International Journal of Recent Advances in Multidisciplinary Topics
Volume 3, Issue 4, April 2022
https://www.ijramt.com | ISSN (Online): 2582-7839

Myocarditis Associated with H1N1 Influenza

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***Abstract*: Influenza is an acute respiratory illness caused by influenza viruses that occur in outbreaks and epidemics worldwide. Signs and symptoms of upper and/or lower respiratory tract involvement are present, along with indications of systemic illness such as fever, headache, myalgia, and weakness. Influenza is usually a self-limited disease; however, in certain individuals, there can be complications like myocarditis, ARDS, etc. Influenza A virus, subtype H1N1 is known to cause severe illness. We report an unusual case of H1N1 Influenza, in an elderly female presenting with fever and syncope. She was diagnosed case of hypertension and diabetes on treatment. On evaluation, it was found that she had bradycardia, due to myocarditis, which attributed to her symptom of syncope. This kind of presentation of H1N1 Influenza infection is rare, complete examination and high degrees of suspicion are necessary. The rapid progression in lack of active management and treatment can be fatal.**

***Keywords*: Influenza A, H1N1 virus, seasonal flu, syncope, blackout, fever, bradycardia, myocarditis.**

1. Introduction

Myocarditis is inflammation of the myocardium which is caused by many bacterial and viral infections. Viral Myocarditis is caused by more than 20 different types of viruses namely Coxsackie B virus, adenovirus, echovirus, influenza virus, Human herpesvirus 6, and parvovirus B19. Acute myocarditis is a well-known complication of influenza infection. The clinical expression varies from asymptomatic to fulminant myocarditis, which can result in severe hemodynamic dysfunction, necessitating high-dose catecholamines and mechanical circulatory support [1].

The clinical features of H1N1 are similar to those of seasonal influenza. Although myocardial involvement in seasonal influenza infection has been reported in up to 10% of cases, the frequency of myocarditis in H1N1 influenza remains unclear. The presentation of influenza myocarditis varies from asymptomatic infection to early fulminant myocarditis, cardiogenic shock, and death. Patients typically present within 4 to 7 days of their illness with shortness of breath, pleuritic chest pain, and rarely, fulminant heart failure. Supportive care remains the mainstay of treatment. Mild cases rarely require vasopressor support, whereas fulminant myocarditis may necessitate supportive measures such as intra-aortic balloon pumps and extracorporeal membrane oxygenation therapy to achieve hemodynamic stability [2].

We report a case of fulminant myocarditis associated with the H1N1 influenza A virus to emphasize the importance of this virus as an etiological agent, describe the clinical course, emphasize the importance of some basic investigations, and discuss aspects relevant to the treatment and prognosis of myocarditis.

2. Case Report

73-year-old female known case of Hypertension, Diabetes mellitus for 10 years came to the emergency department with chief complaints of fever and blackouts since last 1 day. The patient was also having body aches and generalized body weakness. She was taking medications for hypertension and diabetes. Blackouts lasted for 2-3 seconds with the frequency of 3-4 episodes per day for 1 day. She had no chest pain, palpitations, cough, breathlessness, or history of swelling of feet.

She also had no history of seizures, weakness in any part of the body. She is not a known case of Tuberculosis, Chronic obstructive pulmonary disease. She has no known allergy. There is a history of contact with the person coming from abroad. She was taking Furosemide 40mg OD for hypertension and, Metformin, Rosuvastatin, and aspirin for Diabetes Mellitus.

On examination: The patient was alert, conscious, and febrile with no cyanosis, pallor, or icterus. Vitals were, Pulse – 64/min, BP -140/80 mm of Hg, RR – 24/min, SpO2 – 94% on room air and 100% at 2L/min O2, Temp – 100.3 F, Random Blood glucose – 124mg/dl. On respiratory examination, there was equal bilateral air entry with minimal bilateral wheeze. Neurological examination was well within the normal limit. Cardiovascular examination was normal, S1S2 heard, no murmur present, JVP was under normal range. The abdomen was soft and non-tender with no significant findings. The patient was admitted to Medicine ICU and her blood was sent for routine investigations which initially were as s/o mild anemia, but subsequent CBC showed lymphocytosis with low-normal TLC (Table 1).

Her Liver and Renal function tests were well within normal limits with serum sodium being on a lower borderline value (Table 2).

Her Random blood sugar was a little raised. One notable finding was her elevated Creatine Phosphokinase which raised suspicion of muscle injury/inflammation (Table 3).

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Table 1
Complete blood count of first 4 days showing lymphocytosis

Test	**Results**			
Date:	Day 1	Day 2	Day 3	Day 4
Hemoglobin	10.8	11.9	12.9	12.9
Total Leukocyte count	4720	4600	4490	4480
Differential leukocyte ct				
Neutrophils	80	48	51	28
Lymphocytes	16	48	46	71
Eosinophils	01	01	00	00
Monocytes	01	01	01	01
Basophils	00	00	00	00
Band forms	02	02	02	00
Platelets	150	150	150	150
MCV	84.0	84.1	83.9	83.7

Table 2
Liver, Renal, and other metabolic panel tests

Test	**Result**
Calcium	8.9
RBS	**234**
LDH	187
CPK	**206**
Serum Bicarbonate	27
Free T3	2.73
Free T4	0.86
Total Cholesterol	92
HDL	43
LDL	**38***
TGL	53
VLDL	11

Table 3
Other metabolic panel tests

Test	**Result**			
	Day 1	Day 2	Day 3	Day 4
BUN	10.0	11.0		
Creatinine	0.8	0.8		
Sodium	**132**	135		136
Potassium	4.3	4.0		3.8
Chloride	98	101		98
Bilirubin (Total)	0.3	0.3		
Bilirubin (Direct)	0.1	0.1		
SGOT	51			
SGPT	50			
ALP	73			
Albumin	3.7			
Globulin	3.0			
Total Protein	6.7			

Chest X-Ray showed mild Peribranchial Cuffing in the bilateral perihilar region (figure1).

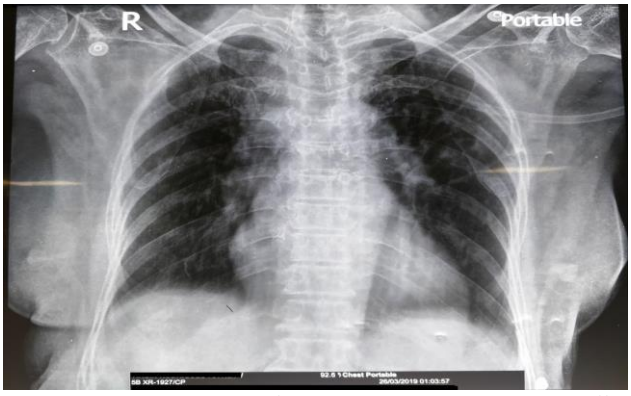

Fig. 1. Chest X-ray on 1st day of admission showing peribronchial cuffing

Her E.C.G. along with Cardiac Monitor showed sinus bradycardia so an E.C.G. was taken which showed sinus bradycardia (figure 2). Her heart rate went as low as 38 beats/min at night while sleeping.

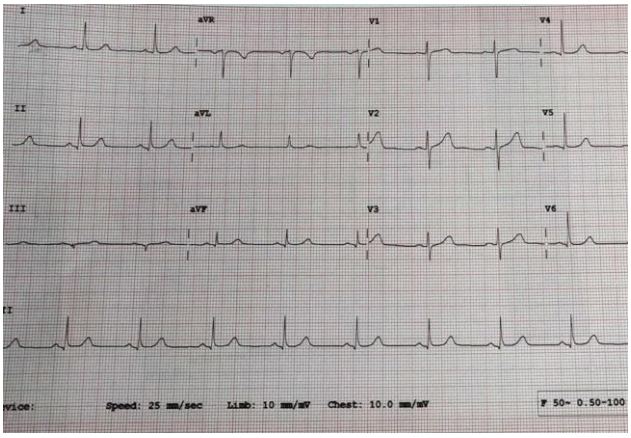

Fig. 2. E.C.G. showing sinus bradycardia

NTproBNP and hsTrop I were sent given elevated Creatine Phosphokinase and E.C.G. findings, both of which came high (Table 4).

Table 4
hsTrop-I and NTproBNP values

Test	**26/03/2019**	**Normal Range**
hsTrop I	2473.3 pg/ml (by chemiluminescent immunoassay)	Up to 15.6 pg/ml
NTproBNP	1810 pg/ml (by fluorescence immunoassay)	Up to 900pg/ml (age 50-75yrs)

This led to the suspicion of myocardial damage and inflammation for which we ran 2D ECHOCARDIOGRAPHY which came out to be normal with an Ejection Fraction of 60% and no regional wall motion abnormality at rest. Trivial TR was found with no significant PH. No clots/pericardial effusion was seen.

The patient's nasopharyngeal swab was sent for H1N1 (Swine Flu 2009 influenza A) P.C.R. which came out to be POSITIVE. The patient has immediately shifted to the isolation room of Medicine ICU and Oseltamivir 150mg BD was started on the second day of admission. Her vitals remained stable at all times with minimal requirement of oxygen support via facemask/nasal cannula.

Her subsequent hsTrop I value showed an increasing trend followed by a declining trend (Table 5). Her bradycardia also got resolved though she had brief episodes of ventricular trigeminy (figure 3) which later also got resolved. There was no electrolyte imbalance during all this time. Subsequent chest X-rays showed improvement (figure 4)

Table 5
Trend of hsTrop I levels

Date	26/3/19	27/3/19	28/3/19
hsTrop I	2473.3 pg/ml	10360.7 pg/ml	3013.2 pg/ml

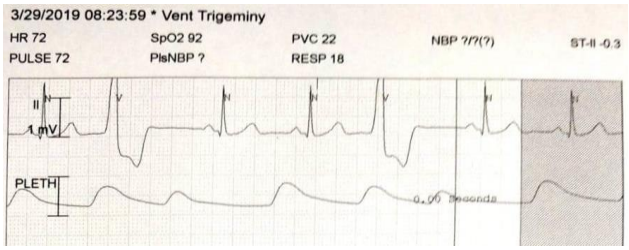

Fig. 3. E.C.G. showing ventricular Trigeminy

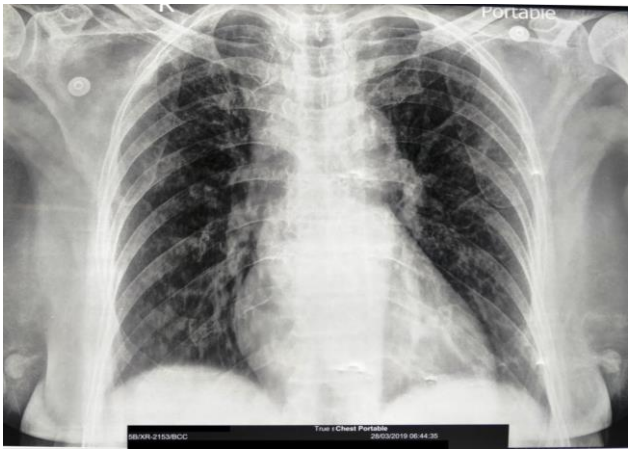

Fig. 4. Chest X-ray showing improvement

After 5 days of treatment with Oseltamivir, all her symptoms got resolved. She was no longer having any fever or blackouts. The patient was shifted out of Medicine ICU to the ward and later discharged with stable vitals.

3. Discussion and Conclusion

People frequently attribute blackouts to some neurological cause such as TIA, epilepsy, etc., and many of them are right as well. Other common causes are orthostatic hypotension, dehydration. They don't often think about having heart pathology when patients present with blackouts. And if there is fever accompanying bradycardia one should suspect having viral myocarditis as the cause and should go for investigations. Though tachycardia is the most finding in cases of myocarditis, this case presented with bradycardia and hence syncope as a consequence of that.

The patient didn't have classical symptoms of flu-like cough, coryza, cold, she only got fever followed by blackouts as the primary presentation of this infection. Some simple investigations such as CBC, ECG, CPK-MB, Trop-I are of great value in making differentials and coming more towards the diagnosis.

And after screening and appropriate evaluation, one should go for more complex investigations such as 2D-ECHO, Nasopharyngeal swab PCR for the virus, Coronary angiography, etc.

Though this patient didn't develop severe hemodynamic instability, one should always be vigilant of dreaded complications such as fulminant myocarditis that one may develop anytime which might require ionotropic support, IABP, etc.

We stress the need for increased awareness of influenza-associated myocarditis. Such knowledge will facilitate earlier diagnosis and treatment of this potentially fatal complication during future influenza pandemics.

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