

Case Report

Acute fulminating viral myocarditis: clinically mimicking ARDS

Sit NK¹, Saha K², Bandyopadhyay A³, Sarkar S⁴

Abstract

Viral myocarditis usually presented with influenza like manifestations, cardiac symptoms and signs related to myocarditis. We are presenting a case which was clinically mimicking acute respiratory distress syndrome but careful history taking with chest x-ray examination saves life of the patient.

Key words: viral myocarditis, ARDS

Introduction

ARDS (acute respiratory distress syndrome) is a life threatening condition of the respiratory system. Main causes of ARDS are severe infections (pulmonary and extra-pulmonary); trauma (pulmonary and extra-pulmonary); and others (inhalation of toxins, aspiration of gastric content, near drowning, pancreatitis, drugs etc.).¹ The diagnosis of ARDS is based on acute onset of respiratory distress, bilateral infiltrates on chest radiology, severe hypoxia with PaO₂/FiO₂ ratio = 200 (PaO₂ = partial pressure of arterial oxygen; FiO₂ = percentage of inspired oxygen), identification of etiological factor and exclusion of cardiogenic pulmonary edema by pulmonary artery wedge pressure = 18 mm Hg or absence of clinical evidence of left atrial hypertension.² As measurement of pulmonary capillary wedge pressure is invasive and has many fallacies, in usual clinical practice left heart disease is excluded by clinical assessment as well as absence of cardiomegaly. Differential diagnosis of ARDS includes left ventricular failure, acute interstitial pneumonia, diffuse alveolar haemorrhage, acute eosinophilic pneumonia, hypersensitivity pneumonitis, post obstructive pulmonary edema and bilateral pneumonia.

We are presenting an unusual case of acute fulminating viral myocarditis presenting as ARDS where diagnosis was suspected only after careful examination of chest X-ray (CXR).

Case report

An 18 year female was admitted to an infectious disease hospital in Kolkata with low grade fever and loose motion for 3 days. She developed increasing shortness of breath with hypoxemia and was transferred to the intensive respiratory care unit of our medical college. On admission, she was cyanosed with respiratory rate 42/min, pulse rate 140/min, oxygen saturation (SpO₂) 74% at room air, temperature 102°F and blood pressure 110/70 mm of Hg. Examination of respiratory system revealed few bilateral basal crepitation in the lungs. Cardiovascular system examination revealed tachycardia but there was no gallop or murmur. Examination of other systems was essentially normal. Peripheral blood examination showed hemoglobin 9.4gm/dl, white blood cell count 10,700/mm³ (neutrophil 74%, lymphocyte-20%, monocyte-5%, eosinophil-1%), erythrocyte sedimentation rate 38 mm/h and platelet count 80,000/mm³. Blood urea nitrogen, creatinine, sodium, potassium levels and liver function tests were normal. C - reactive protein and procalcitonin values 48.8 mg/l and 0.619 ng/ml respectively. Her arterial blood gas (ABG) showed PH 7.52, PaCO₂ (partial pressure of arterial carbon di-oxide) 32mm of Hg, bicarbonate 28.6 meq/l and PaO₂ 58 mm of Hg with FiO₂ 0.6 (calculated PaO₂ and FiO₂ ratio was 96.67). Her CXR, done in the morning of the day of transfer showed bilateral lower zone infiltrates with normal cardiac size (Figure I).

1. Niranjana Kumar Sit, Assistant Professor, Department of Respiratory Medicine, NRS Medical College and Hospital, Kolkata, email - niranjansit2010@gmail.com
2. Kaushik Saha, RMO cum Clinical Tutor, Department of Respiratory Medicine, NRS Medical College and Hospital, Kolkata, email - doctorkaushiksaha@gmail.com
3. Ankan Bandyopadhyay, RMO cum Clinical Tutor, Department of Respiratory Medicine, NRS Medical College and Hospital, Kolkata, email - drankan11@gmail.com
4. Supriya Sarkar, Professor, Department of Respiratory Medicine, NRS Medical College and Hospital, Kolkata, email - drssupriyo@yahoo.co.in

Corresponds to: Dr. Kaushik Saha, Rabindra Pally, 1st Lane. P.O. - Nimta. Kolkata - 700049, email - doctorkaushiksaha@gmail.com

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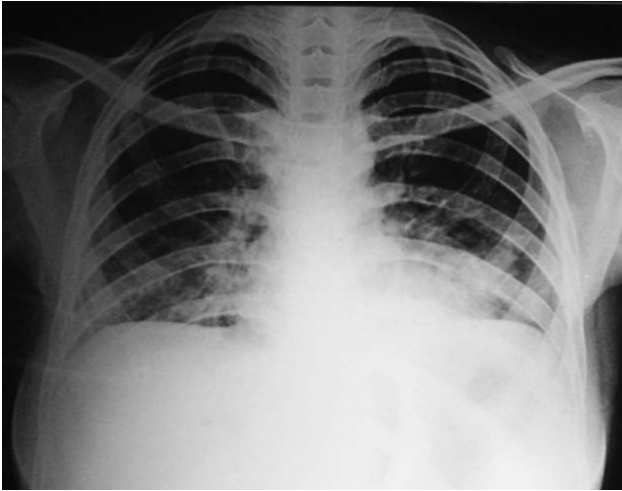


Figure I: Chest X-ray showing bilateral parenchymal infiltrations with bilateral pleural effusion. So, the diagnosis of ARDS was made and she was put on mechanical ventilation with positive end expiratory pressure (PEEP) 10 cm of H₂O along with antibiotics and other supportive treatment. A second careful look at that CXR (Fig I) showed that along with bilateral infiltrates there was suspicion of bilateral pleural effusion. The highest point of right dome of diaphragm was shifted laterally and the distance between fundic air and left dome of diaphragm was increased (more than normal). Portable ultrasonography (USG) of thorax confirmed bilateral pleural effusion and examination of pleural fluid revealed transudative nature of fluid. Her ECG showed (Figure II) sinus tachycardia (heart rate 144/min), ST-segment depression and T-wave inversion in all chest leads. Trop-T test was positive and her cardiac enzyme levels were elevated significantly (CPK-13,630 U/L and CPKMB-253U/L).

Echocardiography showed global left ventricular hypokinesia with left ventricular ejection fraction 42%. She was tested negative for HIV 1 & 2 and malaria dual antigen. Both anti-nuclear antibody (ANA) and rheumatoid arthritis factor (RA factor) were negative.

On the 4th day her sensorium deteriorated despite improving ventilator and ABG parameters. She was only grimacing with painful stimuli and her muscle powers of all limbs were 0/5 and all reflexes were lost. Cerebrospinal fluid study showed normal pressure, cell count 15/high power field (all mononuclear cells), protein 28mg/dl, glucose 149mg/dl, chloride 135mg/dl, and Indian ink preparation, Gram stain smear and Z-N smear were negative. Repeated blood biochemistry showed fasting glucose 86 mg/dl, urea 46 mg/dl, creatinine 0.8 mg/dl, sodium 133.98 mEq/l, potassium 3.4 meq/l, thyroid stimulating hormone 1.25 uIU/ml, serum bilirubin 1.4 mg/dl, SGPT 140 U/dl, SGOT 150 U/dl, total protein 5 gm% and albumin 2.7 gm/dl and C-reactive protein 7.0 mg/dl. Viral encephalitis with viral myocarditis was diagnosed clinically and intravenous acyclovir was started at a dose of 10 mg/kg 8 hourly and given for 14 days. Patient gradually improved conscious level, muscle power and jerks. On the 10th day patient was extubated from mechanical ventilation and repeat CXR (Figure III) showed improvement of parenchymal infiltrates with resolved pleural effusion on portable USG thorax. On the 20th day she was able to walk with support and was discharged.

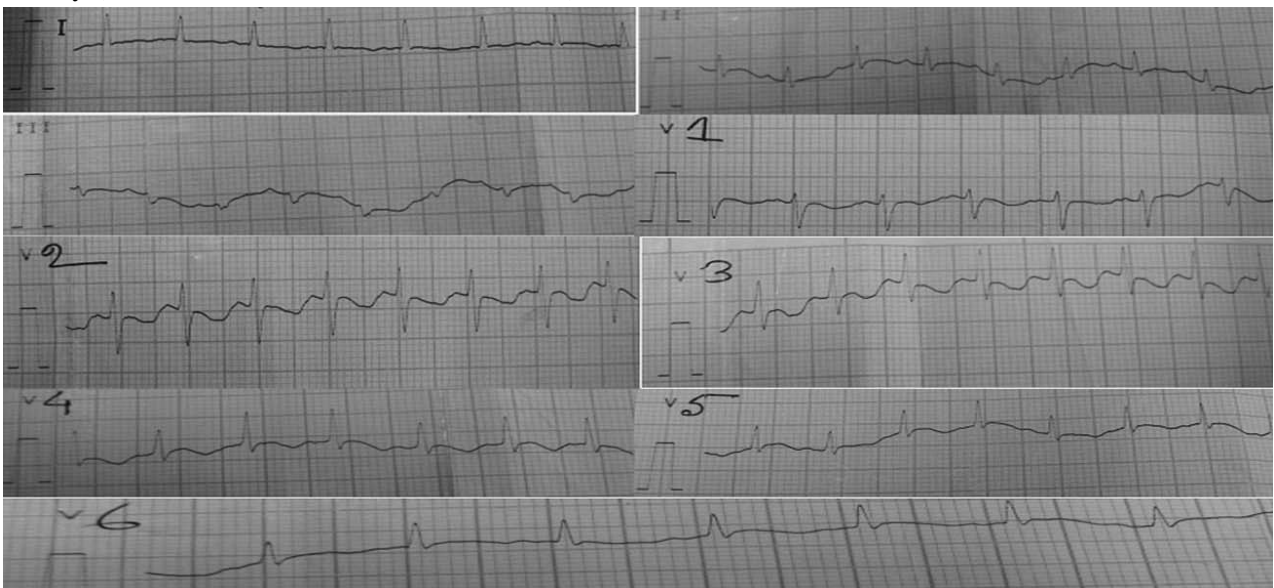


Figure II: Electrocardiography showing ST-T changes suggestive of myocarditis in all chest and limb leads.



Figure III: Follow up chest x-ray after 10 days of treatment showing improvement of alveolar opacities with resolved pleural effusion.

She recovered gradually but completely. Follow up investigations after one month including routine blood, biochemistry, echocardiography and computed tomography of brain were normal.

Discussion

The term myocarditis was coined in the early 19th century by Corvisart. There are three phases in the pathogenesis of myocarditis, i) direct virus-mediated destruction of cardiac myocytes, ii) immune mediated destruction and iii) development of dilated cardiomyopathy (DCM). Two distinct clinical presentations of myocarditis have been observed: acute fulminant myocarditis with haemodynamic instability that recover rapidly within days or weeks with better long term prognosis and subacute myocarditis that leads to the development of DCM with poor long term prognosis. Patients with acute fulminating myocarditis who survived the initial insult had 93% survival at 11 years compared with 45% for subacute myocarditis.³ It is postulated that fulminant inflammatory reaction clears the virus load and the disease does not enter the 2nd and 3rd phases of reaction.³ Symptoms of acute myocarditis are usually preced-

ed by upper respiratory tract or gastrointestinal tract manifestation. Cardiac symptoms are usually non-specific like fatigue, dyspnea, palpitation, malaise, chest discomfort etc. Signs vary from sinus tachycardia, a diminished first heart sound, gallops, regurgitation murmur and pericardial friction rub.⁴ Myocarditis should be considered in cases of rapidly progressive DCM, idiopathic ventricular arrhythmias, cardiovascular collapse and ECG mimicking acute myocardial infarction (AMI) with normal coronary arteries. Acute myocarditis is always a diagnostic challenge. The gold standard is the endomyocardial biopsy (EMB) with immunohistochemistry and viral polymerase chain reaction. That was not possible in our setting. So we diagnose the case on the basis of clinical evaluation, ECG, CXR, blood cardiac enzyme studies and non-invasive imaging. Traditional viral serology and culture from blood lack sensitivity and specificity. In general cardiac enzymes are elevated but cardiac troponins are more sensitive. ECG may look like AMI, acute pericarditis, or it may show PQ-segment depression, ST-T segment elevation followed by depression and arrhythmia. Echocardiography in fulminant myocarditis show non-dilated, thickened and hypocontractile left ventricle resulting from interstitial oedema.⁵ Patients with viral myocarditis should be treated with limited physical activity and standard treatment of heart failure with diuretics, beta-blockers, angiotensin-converting enzyme inhibitors and receptor blockers. Short term use of prednisolone and azathioprine improves long term left ventricular function.⁶ Antiviral therapy prevents direct viral damage and immune signal amplification system.

In our case the diagnosis of ARDS was made on the basis of clinical, radiological and ABG parameter and by cardiac disease clinically and the absence of cardiomegaly in CXR. Subsequently cardiac disease was suspected by the discovery of bilateral pleural effusion and the diagnosis of acute viral myocarditis was established by ECG, echocardiography and elevated cardiac enzyme levels. Viral etiology was suspected by typical clinical picture, starting with gastrointestinal manifestation then myocarditis and encephalitis; by excluding immunological disease.

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