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Pericardial Tamponade in COVID-19: A Case Report

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Abstract

Background: Tuberculous pericarditis is a rare manifestation of tuberculosis infection. COVID-19 pandemic poses a challenge in detecting uncommon disease. Pericardial effusion with tamponade has been described with COVID-19 but the association with tuberculosis is not yet known.

Case summary: A 47-year-old man was admitted with symptoms of COVID-19 infection. Rapid progression of cardiomegaly on radiograph with clinical deterioration were suggestive of pericardial tamponade. Urgent pericardiocentesis revealed hemoserous fluid, elevated adenosine deaminase and positive TB PCR. He was started on anti-tuberculous therapy and Remdesivir with marked improvement of symptoms. Repeat echocardiogram and CT Thorax showed resolution of pericardial fluid and patient was discharged well.

Discussion: This case highlights the difficulty in detecting a concomitant rare but important disease. The development of massive pericardial tamponade acutely is not pathognomonic for COVID-19, and a careful diagnostic process involving multi-modality imaging, occurred to arrive at a diagnosis of tuberculosis.

Keywords: COVID-19; Pericardial effusion; Tamponade; Tuberculosis; Pericarditis; Case report

Introduction

Tuberculous pericarditis is rare and associated with significant morbidity and mortality [1]. We present the case of a patient admitted with symptoms of COVID-19 infection that developed pericardial tamponade subsequently. Urgent pericardiocentesis revealed evidence of tuberculous pericarditis and he was appropriately managed.

Case Presentation

A 47 year old gentleman presented with productive cough, pleuritic chest pain and fever for two days. Physical examination revealed a febrile, generally ill appearing gentleman. He had a regular pulse, S1/S2 were normal without murmurs or rub. Lung examinations revealed left basal crepitations. Vital signs were blood pressure 130/83 mmHg, heart rate 104 beats/min, oxygen saturation 97% on room air, respiratory rate 16/min, and temperature 38oC. Chest radiograph showed left lower zone retrocardiac opacities and he was transferred to the isolation ward. SARS-CoV-2 PCR came back positive from his nasopharyngeal swab. The patient did not have any significant medical history. He denied travel but he was in close contact with a colleague with COVID-19. He came from a TB endemic area (Figure 1).

On day 3 of hospitalization he deteriorated requiring 4L nasal cannula to achieve SpO2 94%. His BP was 125/80mmHg, his rate rate 110 beats/min and he had tachypnea 20/min. There was no evidence of heart failure or tamponade. Electrocardiogram (ECG) showed sinus tachycardia with normal QRS complexes. High sensitive troponin I was 4 ng/ml (normal values: <14 ng/ml). There was absolute monocytosis (0.92 x 109/L) and elevated C-reactive protein (CRP) at 134.7 mg/L (normal values < 5 mg/L). A repeat chest radiograph showed marked increased in heart size. He was started on active drug remdesivir as a part of an ongoing trial (Figures 2 and 3).

Subsequent ECG revealed persistent sinus tachycardia and no evolution of ST-T wave changes. Labs were remarkable for

monocytosis (1.02 x 109/L). Liver function tests and coagulation panel were normal. Arterial blood gas showed acute respiratory alkalosis with pH 7.48, pCO_2 39, pO_2 68, Bicarbonate of 29 on 3L nasal cannula. Lactate was raised at 2.7 mmol/L (normal value < 2 mmol/L).

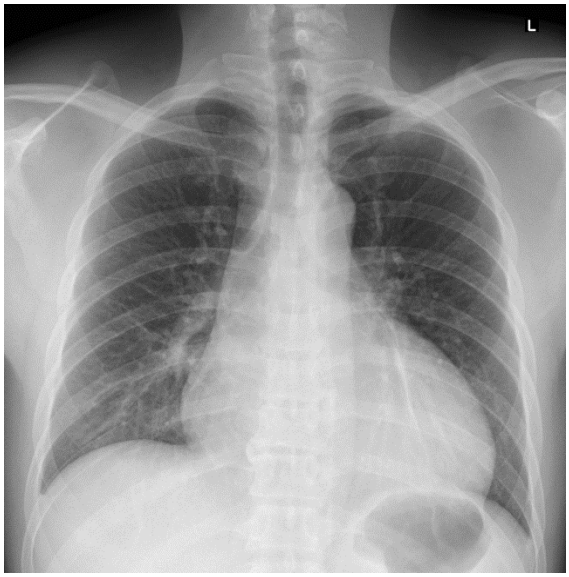

Figure 1: *Chest radiograph showed left retrocardiac opacities. Cardiac silhouette appears normal.*

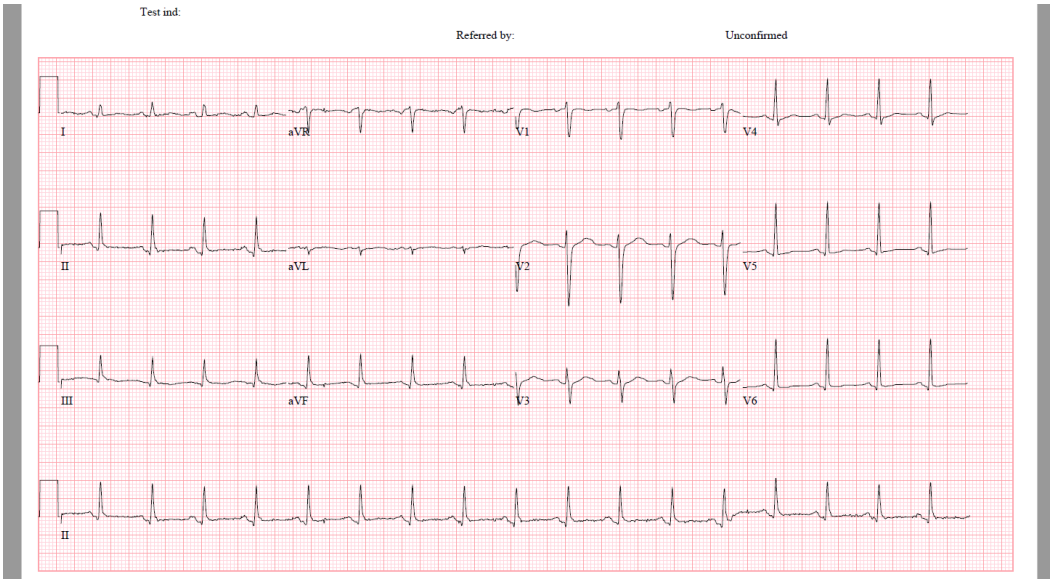

Figure 2: *ECG: sinus tachycardia with normal QRS complexes.*

Transthoracic echocardiogram demonstrated hyperdynamic left ventricle with LVEF of 65%. There was right atrial collapse, diastolic collapse of right ventricle, 3.5 cm of pericardial effusion and plethoric inferior vfiena cava of 2.2 cm with < 50% variation.The effusion was noted to be complex with fibrin deposits adhering to the myocardium. The transmitral flow variation was 30% and transtricuspid variation was 50%. The patient was transferred to the intensive care unit. The patient developed sinus tachycardia (range up to 130 beats per minute) with concomitant febrile episodes of 39^{oC}. Pericardiocentesis was performed in view of persistent tachycardia and rapid accumulation of pericardial effusion. The procedure was done under echocardiographic guidance. Pericardiocentesis yielded 900 mL of hemoserous fluid [fluid lactate dehydrogenase (LDH) 2,253 IU/L, fluid/serum LDH > 0.6]. Cytology was negative for malignancy. Adenovirus PCR, Enterovirus PCR and SARS-CoV-2 PCR were negative. Acid fast bacilli was detected and TB PCR was positive. Fluid microscopy revealed predominantly nucleated cells (8,513 cells/uL) with 91% lymphocytes. Adenosine deaminase for pericardial fluid was significantly elevated at

44U/L (normal value < 20U/L). Retroviral screen was negative. The immediate resolution of tachycardia (heart rate reduced to 80-90 beats per minute) signifies the hemodynamic improvement gained from relieving the tamponade. The pericardial effusion was highly diagnostic of tuberculous pericarditis in the absence of coagulopathy, malignancy and autoimmune etiologies. He was commenced on rifampicin, isoniazid, ethambutol and pyrazinamide. Subsequent echocardiogram showed resolution of effusion with marked improvement of symptoms. A follow up CT Thorax revealed left lung lower lobe collapse-consolidation, small pleural effusion with marked reduction in pericardial effusion (Figure 4).

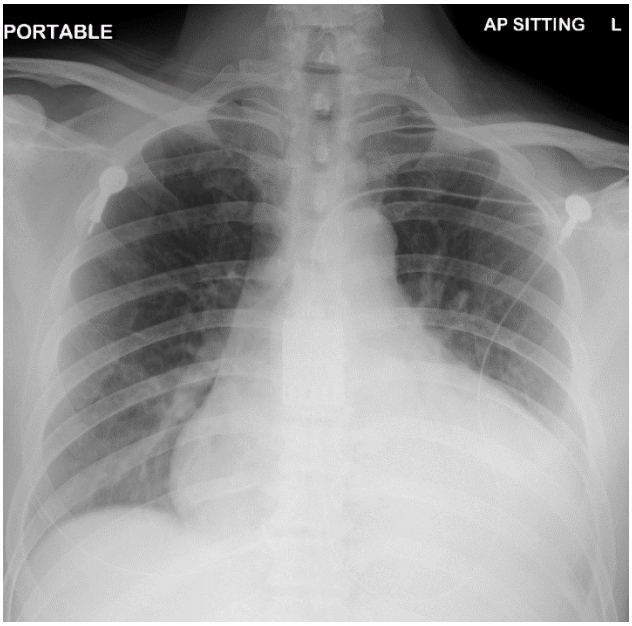

Figure 3: *Chest radiograph showed persistent opacities over left retrocardiac region. Interval increased in cardiomegaly.*

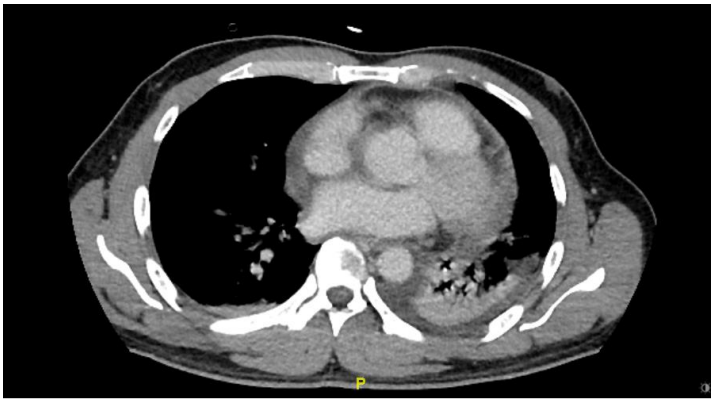

Figure 4: *CT Thorax showed left lower lobe collapse-consolidation with small pleural effusion. Minimal pericardial effusion.*

He was discharged after 2 weeks into anti-tuberculous therapy. Subsequently, he was reviewed during follow up (4 weeks post discharge) with resolution of pericardial effusion and residual left retrocardiac consolidation on chest X-ray.

Discussion

Ever since the first cases of pneumonia of unknown origin were described in Wuhan, China in January 2020, COVID-19 has rapidly spread worldwide resulting in a public health emergency. Complications described in the Intensive Care Unit (ICU) include shock, Acute Respiratory Distress Syndrome (ARDS), arrhythmias and acute cardiac injury.2 Case reports of cardiac involvement including Acute ST-Elevation Myocardial infarction, myocarditis, stress cardiomyopathy and arrhythmias have also been reported [2-5].

While viral infections such as Epstein-Barr virus, Parvovirus B19 and Coxsackievirus are known to cause pericarditis and pericardial effusion, little is known about the pericardial complications of COVID-19 and their pathophysiology [6]. The fibrinoid appearance of pericardial effusion has been strongly associated with pericardial inflammation, as in the case of tuberculoid, bacterial or malignant pericardial effusion [7,8]. This could also be postulated to be due to increased viral expression in the heart via angiotensin-converting enzyme 2 (ACE2) as the entry receptor, resulting in an inflammatory response, although more studies are required to substantiate this [9]. The appearance of fibrin, lymphocyte rich, elevated adenosine deaminase level with detection of acid fast bacilli and positive TB PCR in the pericardial fluid is pathognomonic of tuberculous involvement [1,11]. There is a possibility that COVID-19 infection induced an inflammatory response that serves as a nidus for TB reactivation in this patient. In addition, this may explain the rapid progression of pericardial tamponade as TB normally runs an indolent course. TB pericarditis is closely linked to constrictive pericarditis with significant morbidity and mortality.1 Follow-up is required to detect the development of constrictive pericarditis. Treatment with steroids may shorten the time to resolution of symptoms, such as tachycardia and restriction of activity. However, this was not shown to reduce mortality or retard the progression to irreversible constrictive pericarditis [12].

Case series from Italy reported 20 patients with active TB who developed COVID-19 infection subsequently, but none was associated with pericarditis or tamponade [13].

Conclusion

In conclusion, TB pericarditis is a rare manifestation of rapid development of massive pericardial effusion. The presence of TB pericarditis, and consequently its risk, may not be easily identified in the face of COVID-19 pandemic. Thus, a low threshold to use serial echocardiography and dedicated imaging modalities, including CT may be appropriate, particularly in young patient

who deteriorate at an alarming speed. Noteworthy, to the best of our knowledge, the current case comprises the first case of concurrent tuberculous pericarditis with tamponade in COVID-19.

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