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Case Report **Medicine**

Acute Myocarditis Mimicking ST-Elevation Myocardial Infarction in 19 Years Old Male with Pneumonia

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Abstract

Background: Acute myocarditis is an inflammatory disease of the heart muscle that manifests clinically with a spectrum of symptoms, ranging from mild chest pain to myocardial infarction. It can also be linked to viral or bacterial illnesses like pneumonia. ***Case summary:*** A 19-year-old male with no notable medical history reported to the Emergency Department with substernal chest pain radiating to the left arm, accompanied by nausea and profuse perspiration. His vital signs were stable and his physical examination was normal, with the exception of left lower chest crackles. The electrocardiogram revealed ST segment increase in inferior and lateral leads, along with ST depression in leads V1 through V3. A portable chest x-ray revealed a patchy opacity in the lower lobe of the left lung. Invasive coronary angiography revealed normal coronary arteries. Further evaluation with echocardiography and cardiac MRI revealed characteristics of acute myocarditis, including a minimally dilated LV and subtle hypokinesia in the midventricular inferior and lateral segments, as well as mild global hypokinesia in the LV and RV. Perimyocarditis was detected in the patient, and antiplatelet medication were terminated. Due to a low ejection fraction, Bisoprolol, Ramipril, and spironolactone were administered, and he was hospitalised until his intravenous treatments for pneumonia were completed. Further laboratory tests revealed a decrease in troponin T, white blood cell count, and C-reactive protein. He was discharged with medications including spironolactone, metoprolol, Ramipril, and colchicine. Unfortunately, he failed to comply with the directive. On the eighth day of hospitalisation, a discharge ECG revealed sinus rhythm with no ST-T alterations. ***Conclusion:*** When evaluating an acute cardiac event, healthcare practitioners should examine the possibility of uncommon bacterial myocarditis and keep in mind that Streptococcus pneumoniae infection might imitate acute MI in otherwise healthy young males. The timely diagnosis and administration of appropriate antibiotics are critical in the treatment of such conditions.

Keywords: myocarditis, crackles, perimyocarditis, colchicine, Streptococcus pneumoniae.

INTRODUCTION

Myocarditis is an inflammatory disease of the heart that may occur because of infections, immune system activation, or exposure to drugs [1]. Clinically, acute myocarditis (AM) implies a short time elapsed from the onset of symptoms and diagnosis (generally <1 month) [2]. In contrast, chronic inflammatory cardiomyopathy indicates myocardial inflammation with established dilated cardiomyopathy or hypokinetic nondilated phenotype, which in the advanced stages evolves into fibrosis without detectable inflammation [3]. Patients with suspected AM are generally evaluated in the emergency room due to chest pain, dyspnea, fatigue, palpitations, or syncope [4]. The ECG is abnormal in about 85% of cases, ST-segment elevation mimicking acute myocardial infarctions is the most frequent abnormality; inferior and lateral leads are commonly involved [5].

Recommended laboratory tests for identification of patients with suspected AM are myocardial necrosis biomarkers (high-sensitivity troponins, creatinine kinase-MB), markers of inflammation such as C-reactive protein, and differential white blood count can show eosinophilia, suggesting the presence of eosinophilic myocarditis (EM) [6]. Finally, peripheral blood serological and virological tests are rarely informative, with some exceptions (eg, HIV and Borrelia burgdorferi

antibodies) [7]. A search for viral genomes with polymerase chain reaction in aerial tract fluids and pharyngeal swabs can identify viruses of the respiratory tract, such as influenza, and severe acute respiratory syndrome coronavirus-2, which can trigger an AM [8]. Autoantibodies (eg, antinuclear antibody test) and other tests may be indicated in patients with known or possible history of autoimmune disorders [9].

Echocardiography may show a broad spectrum of findings. Even when LVEF is normal, the presence of increased wall thickness, mild segmental hypokinesia, in particular, in the inferior and inferolateral walls, diastolic dysfunction, abnormal tissue Doppler imaging, mild right ventricular dysfunction, pericardial effusion, and abnormal myocardial echogenicity may suggest AM [10].

Cardiac magnetic resonance imaging (CMRI) has emerged as a powerful noninvasive diagnostic tool for tissue characterization [11]. CMRI is recommended in patients with clinically suspected AM or in patients with chest pain, normal coronaries, and raised troponin, for the differential diagnosis of ischemic versus nonischemic origin [12].

Positron emission tomography (PET) can be considered as an alternative noninvasive diagnostic tool in stable patients with contraindication to CMRI or in patients with suspected systemic autoimmune disease where other organs could be involved by the inflammatory process [13]. PET is especially useful for the diagnosis and monitoring of CS [12].

EMB is considered the reference standard for the diagnosis of myocarditis [14, 15]; however, it is an invasive procedure that portends some risks. Despite relatively low sensitivity, it is indicated in specific clinical scenarios both in AM and chronic infl-CMP:

1. AM presenting with severe heart failure (HF) or cardiogenic shock (ie, FM)
2. AM complicated by severe myocardial dysfunction, acute HF, ventricular arrhythmias, or high-degree atrioventricular block;
3. AM or suspected chronic infl-CMP associated with peripheral eosinophilia;
4. AM or chronic infl-CMP with persistent or relapsing release of biomarkers of myocardial necrosis, particularly if associated to a suspected/known autoimmune disorder or ventricular arrhythmias or high-degree atrioventricular block; and
5. Myocarditis in the setting of ICI, where appropriate diagnosis has implications for patients receiving additional cancer therapy.

Invasive coronary arteriography or computed tomography angiography are often necessary to rule out an acute coronary syndrome. Furthermore, patients with AM and acute pericarditis can complain of similar symptoms [4]. Management includes:

1) Hemodynamic support for unstable patient.
2) Heart failure management for stable patient.
3) Cardiac transplantation for refractory cases, usually after acute phase if possible.
4) Treatment of arrhythmia.
5) Activity restriction during acute phase for at least 6 months.
6) Consideration of immunosuppression (after ruling out active infection on EMB by PCR) in proven autoimmune (e.g. infection negative) forms of myocarditis, with no contraindications to immunosuppression, including giant cell myocarditis, cardiac sarcoidosis, and myocarditis associated with known extra-cardiac autoimmune disease.
7) All patients with myocarditis should be followed, with clinical assessment, ECG, and echocardiography [15].

CASE SUMMARY

A 19 years old male patient presented to Emergency Department with chest pain. Pain was substernal, moderate, described as aching, and radiating to left arm. Pain occurred at rest, started 1 hour before presentation, and was associated with nausea and diaphoresis. Patient denied fever on presentation, denied headache, denied abdominal pain, denied diarrhea, and denied vomiting.

However, later during admission, he mentioned symptoms of upper respiratory infection started 2 days before presentation including fever, headache, running nose, and productive cough. Patient was not known to have any significant medical history (history was negative for allergies, diabetes, hypertension, dyslipidaemia, procedures, chronic medications, or recreational drug use). Of note, he mentioned that his brother was diagnosed with coronary artery disease. Patient denies any alcohol use but he was smoker, smoking 1 packet of cigarettes daily for the last 2 years.

By examination

Tympanic temperature was 36.7° C. Respiratory rate was 17 breathe better minute. Heart rate was 83 beats per minute. Blood pressure was 109/65 mm Hg. SaO2 was 98% on room air.

Physical examination was unremarkable except for left lower chest crackles by auscultation. Otherwise, physical examination was normal. Including normal heart sounds, and normal cardiovascular, neurological, musculoskeletal, gastrointestinal, and integumentary examination

Electrocardiogram showed ST segment elevation in inferior and lateral leads with reciprocal ST depression in leads V1 to V3.

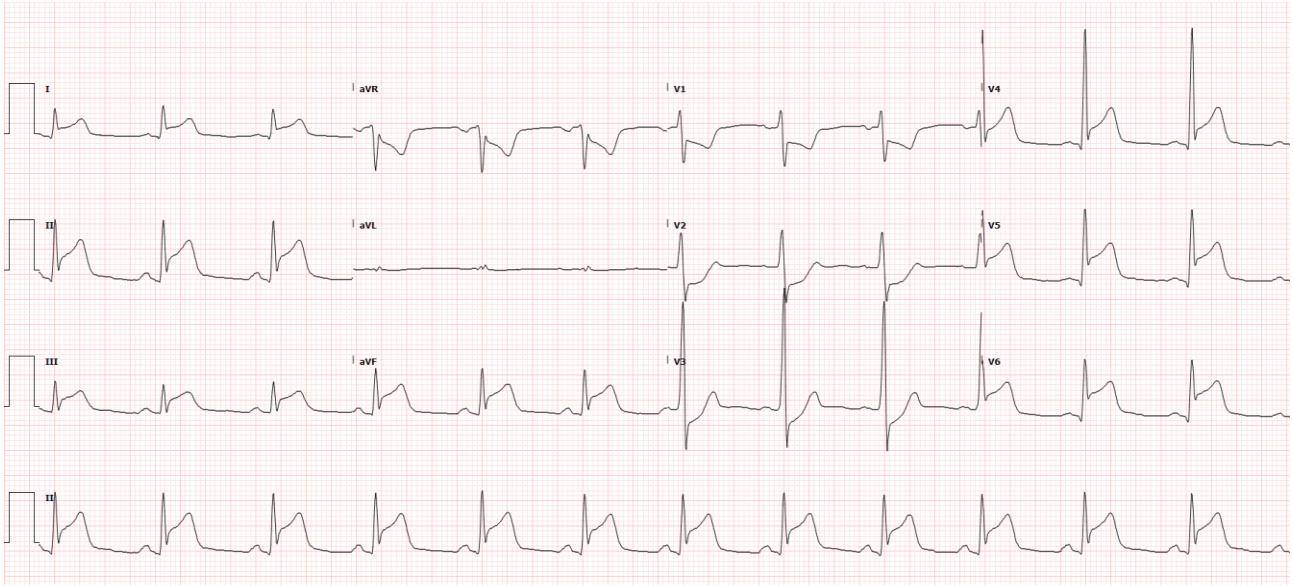

Figure 1: ECG on presentation showing ST segment elevation in inferior and lateral leads with reciprocal ST depression in leads V1 to V3

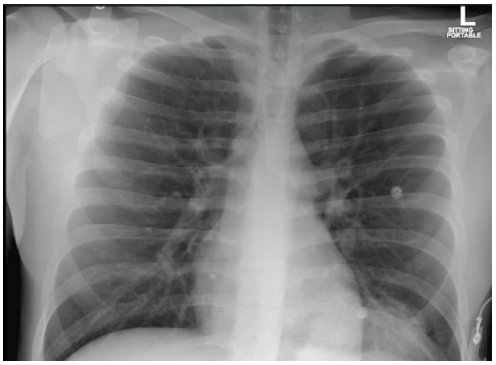

Figure 2: portable chest x-ray showing left lower lung lobe patchy opacity

Patient was taken to Cath Lab with diagnosis of inferior ST-elevation myocardial infarction due to chest pain, and significant electrocardiogram findings.

Invasive coronary angiography showed normal coronary arteries with no obstructive lesions.

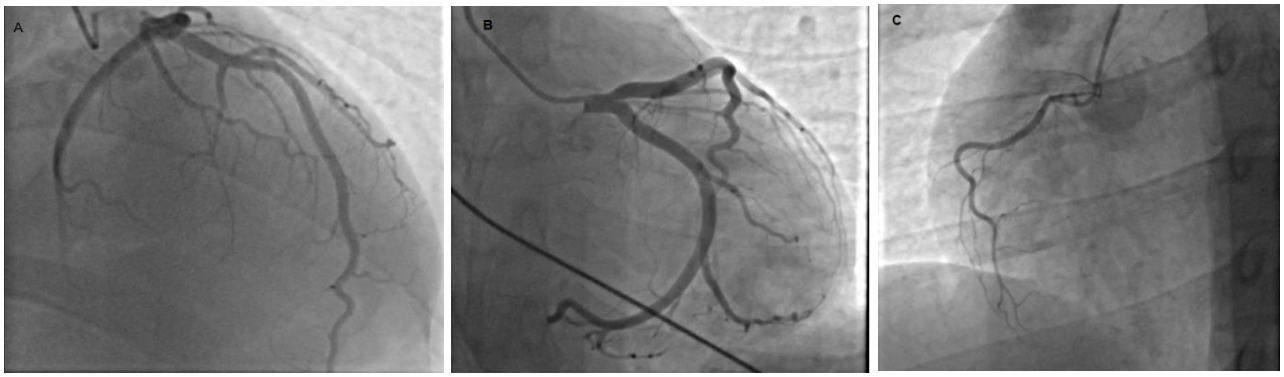

Figure 3 (A-B-C): Invasive coronary angiogram showing normal coronary arteries

Patient was admitted in coronary intensive care unit, differential diagnosis was myocardial infarction with no obstructive coronary arteries, coronary spasm, and perimyocarditis. His chest pain resolved and electrocardiogram showed resolving of ST-T changes.

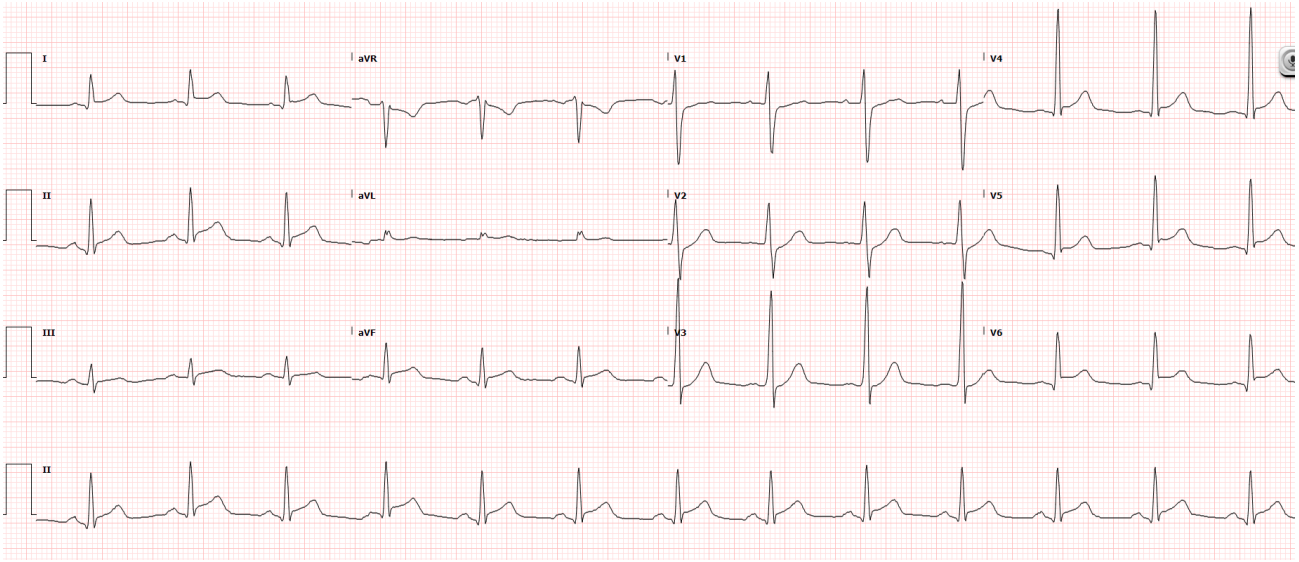

Figure 4: Electrocardiogram in coronary care Unit showing resolving of ST-T changes

Blood test revealed mild hyponatremia 134 millimole per L, mild hypokalemia 2.98 millimole per mL, mild hypochloremia 96 millimole per L, elevated NT proBNP 288 picograms/liter. Leukocytosis white blood count count 19.23* 10^9 per L with 74.8% neutrophils. Troponin T after 5 hours was very high 1947 ng/L, procalcitonin 3.99 ng/mL C-reactive protein 287 milligram/liter, COVID test polymerase chain reaction was negative.

Patient was started on medications with dual antiplatelet therapy, high-dose statin, potassium supplements IV, and antibiotic started empirically with piperacillin/tazobactam for pneumonia. Detail echocardiography reported as normal LV size and wall motion, normal LV systolic function EF 55-60%., and no hemodynamically significant valve disease.

Cardiac MRI was done and reported: Mildly dilated LV. RV at upper limits of normal. Subtle hypokinesia in the midventricular inferior and lateral segments and mild global hypokinesia of the LV and RV, LVEF 39%, RVEF: 44%. Epicardial/subepicardial late gadolinium enhancement in the lateral wall from base to apex and in the basal and apical inferior segments. Subtle subepicardial myocardial edema in the apical lateral segment. SAX edema images show artefacts. Large left lower lobe pulmonary consolidation. Observations are in keeping with acute myocarditis.

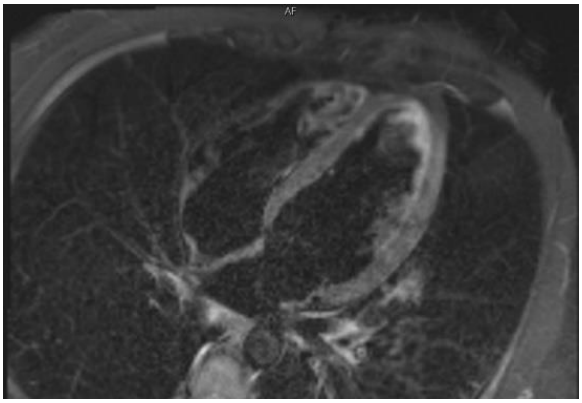

Figure 5: Cardiac MRI showing increased T2 signal intensity in apical and mid Infero-lateral segments

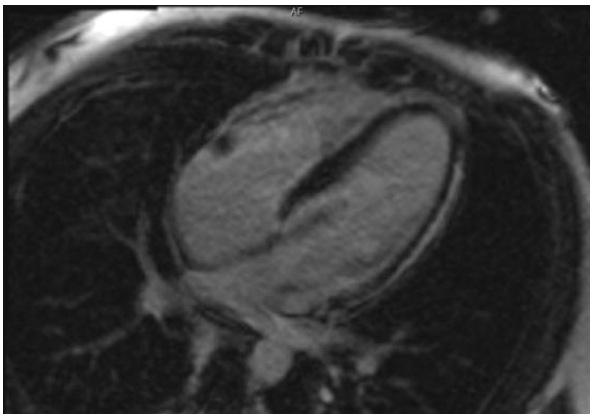

Figure 6: Cardia MRI showing sub-epicardial late gadolinium enhancement involving apical and mid and basal Infero-lateral segments

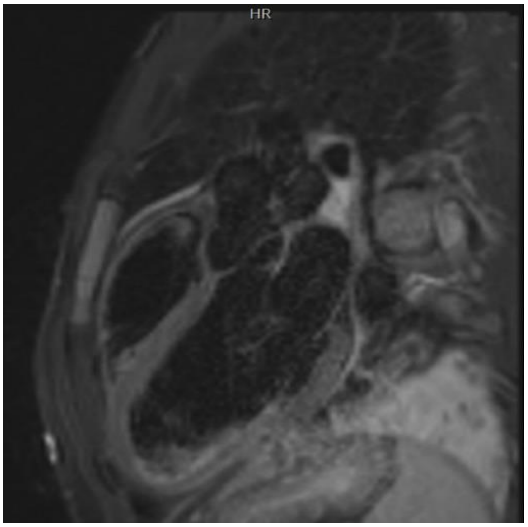

Figure 7: Cardiac MRI showing increased T2 signal intensity involving Infero-lateral wall

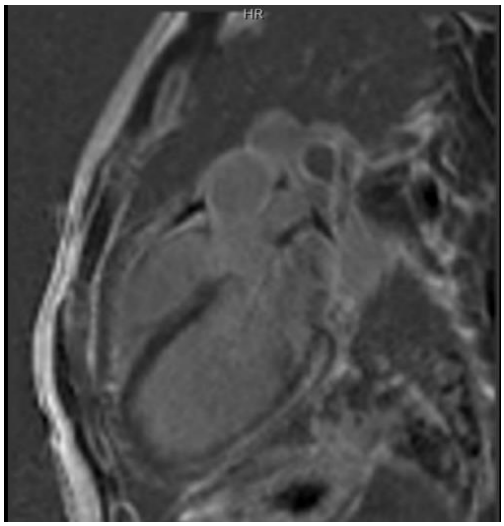

Figure 8: Cardiac MRI showing sub-epicardial late gadolinium enhancement involving the Infero-lateral wall

Patient was diagnosed as perimyocarditis and antiplatelet therapy was discontinued. Bisoprolol, Ramipril, and spironolactone were added due to low ejection fraction. He was kept in hospital until completing his intravenous antibiotic for pneumonia. Next day ECG:

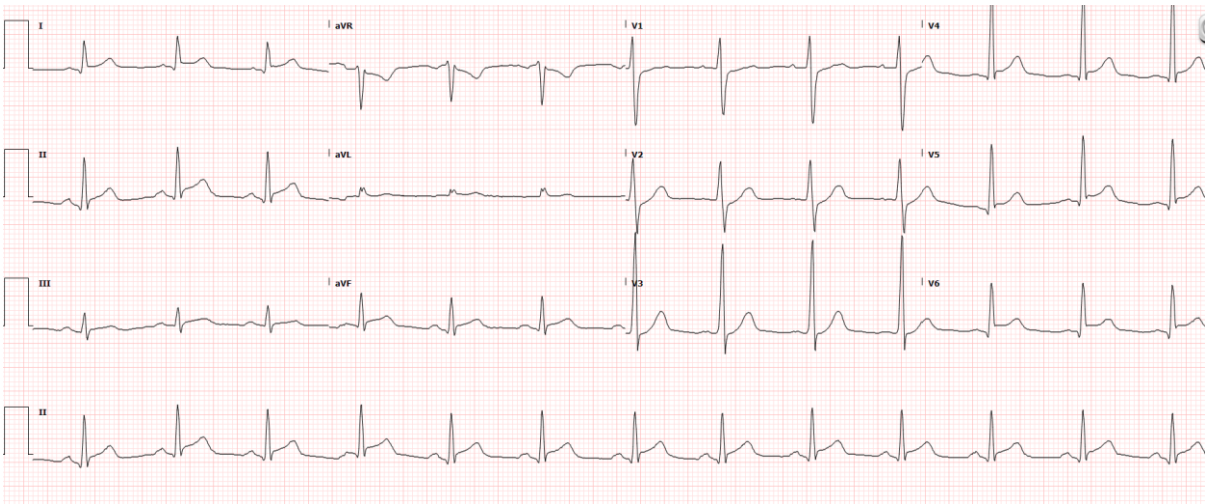

Figure 9: Next day electrocardiogram

Sputum culture showed light growth of normal upper respiratory flora and blood culture showed no growth. Subsequent labs showed dropping in troponin T, white blood count and C-reactive protein. He was discharged on spironolactone, metoprolol, Ramipril, and colchicine. He did not follow-up as directed. Discharge ECG on day 8 of admission:

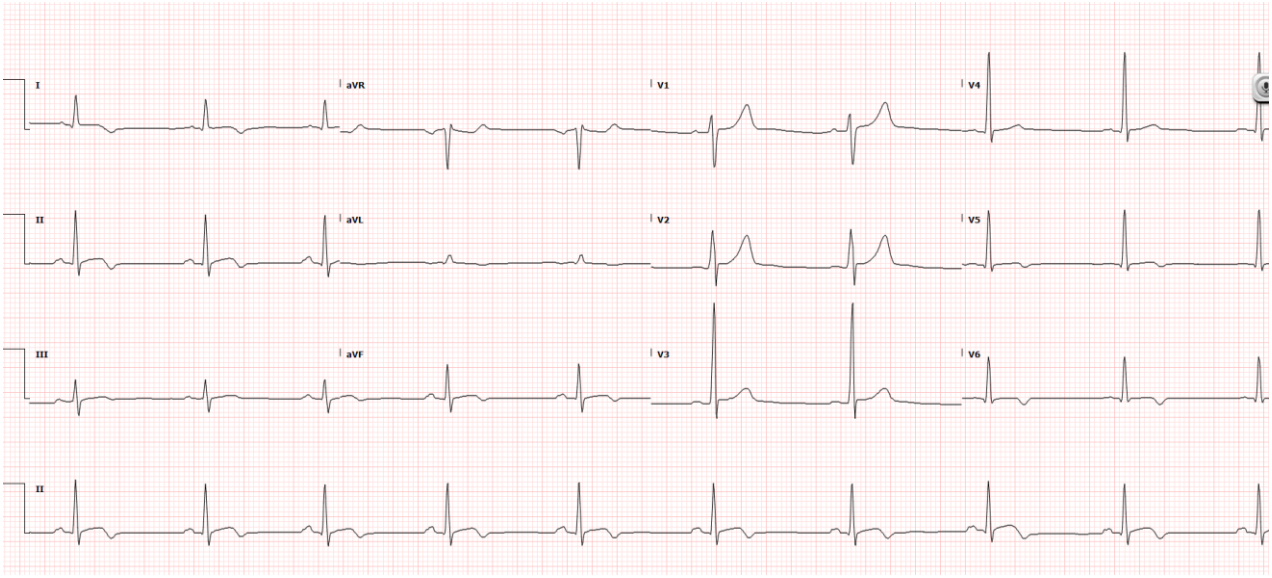

Figure 10: Discharge electrocardiogram

DISCUSSION

This case represents acute perimyocarditis mimicking ST-elevation myocardial infarction in the setting of community-acquired pneumonia in 19 years old male patient. Myocarditis is a condition that can be caused by "Campylobacter enteritis", "Corynebacterium diphtheria", Haemophilus influenzae, Mycoplasma, Staphylococci aureus, Borrelia burgdorferi, "Group A Streptococcus infection", Neisseria, Salmonella, typically in conjunction with enteritis or pneumonia [16]. It is widely recognized that pediatric myocarditis has a variety of clinical presentations; but adolescents are more prone to exhibit notable heart related symptoms and indications [17]. One such cardiovascular indication is Heart block, which is the most prevalent ECG abnormality, followed by sinus tachycardia, ST/T abnormalities and the majority of patients have elevated troponin levels [18]. Our patient also did face ST elevation and depression with higher troponin levels.

Furthermore, researchers believe that pneumococcal pneumonias in adults is connected with acute cardiac events that result in significant mortality. A researcher described patients with concomitant pneumococcal pneumonia and myocardial infarction [19]. All of these patients had at least two "risk factors" for "coronary artery disease", and eight of them developed these risk factors during their pneumonia stay. They suspected that pathophysiology of this syndrome is a mismatch between perfusion and ventilation resulting from pneumonia. Pneumonia is associated with inflammation and an increase in the release of cytokines, which leads to hypotension. In turn, hypotension increases the chance of myocardial stress and thrombogenesis making the patient more prone to a myocardial infarction. In this research, five of these twelve individuals passed away during their hospitalization. However, our patient is still alive and a discharge ECG revealed sinus rhythm with no ST-T alterations.

Case studies by Aguirre et al and O'Brien et al. demonstrate that myocarditis induced by non-rheumatic Group A Streptococcus has the potential to mimic STEMI [20, 21]. This is observed in young men without "coronary artery disease risk factors" and with "normal coronary artery anatomy". They also occur with tonsilitis or streptococcal pharyngitis. A complete recovery can be seen within weeks to months. Additionally, acute coronary events and myocarditis can cause ST segment elevation, chest pain and increased cardiac enzymes. All these indications were observed in our case. Therefore, we suggest that it is very important consider myocarditis in the setting of very young patient presenting with localized ST-elevation, chest pain, and troponin T elevation, even in the presents of reciprocal ST changes on electrocardiogram.

Regarding testing, Endomyocardial biopsy is the only conclusive procedure currently available for diagnosing myocarditis. However, the invasive nature of the process and the likelihood of sampling errors due to patchy inflammation limit its application but immunohistochemistry can improve diagnostic accuracy [22]. In addition to EMB, cardiac magnetic resonance imaging, echocardiography and ECG are

performed more frequently, with indicators of viral serology, inflammation and cardiac biomarkers, thereby assisting in diagnosis [23].

CMRI has emerged as a particularly helpful tool for non-invasively visualising myocardial inflammation and identifying sites of fibrosis, scarring, edoema and inflammation with a high degree of specifity and sensitivity [24]. Unfortunately, its expensive cost prevents some patients from using it. Heart computed tomography is an alternate imaging technology that provides diagnostic accuracy comparable to conventional approaches, as well as various extra advantages. They include a more accurate evaluation of myocardial inflammation, the ability to examine coronary artery structure, and reduced total costs [25]. Although echocardiography cannot reveal myocarditis-specific abnormalities, it can be used to rule out other probable causes of intracavitary thrombi and heart failure. Our patient also underwent CMRI, Chest X ray and ECG.

Besides these tests, taking careful history is very important to unmask any upper respiratory tract infection symptom. Treating such patients in a center with Cath Lab will facilitate diagnosis and management, with avoiding of unnecessary use of thrombolytic therapy and the possible complications Also, the availability of cardiac MRI is crucial to confirmed diagnosed and establish treatment plan.

CONCLUSION

The patient's presentation with acute myocarditis and pneumonia highlights the need for a comprehensive evaluation to determine the underlying cause of chest pain and direct appropriate treatment. Rare bacterial myocarditis should be considered while evaluating an acute cardiac event, and physicians should be aware that Streptococcus pneumoniae infection might mimic acute myocardial infarction in otherwise healthy young males. The timely diagnosis and administration of appropriate antibiotics are critical in the treatment of such conditions.

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