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| RESEARCH ARTICLE

Myopericarditis: A Rare Presentation of Rheumatological Illness

Dr. Naser Mohamad Mansoor[1], Dr. Layla Al-Mutawa[2] Dr. Hasanin Ali Khudair[3], Dr. Ali Haider Ali[4]✉, Dr. Mahmood B. Dhaif[5], Dr. Mustafa Mahmood Alshbrawi[6], Dr. Hameed Mahdi Ali[7], and Dr. Mahmood Jasim Alaiwi[8]

[1]Consultant Emergency Medicine, Salmaniya Medical Complex, Kingdom of Bahrain
[2]Chief Resident, Cardiology Department, Salmaniya Medical Complex, Kingdom of Bahrain
[3]Senior Resident, Cardiology Department, Salmaniya Medical Complex, Kingdom of Bahrain
[4]Emergency Medicine Resident, Salmaniya Medical Complex, Kingdom of Bahrain
[5]Medical Intern, Salmaniya Medical Complex, Kingdom of Bahrain
[6]Senior Emergency Medicine Resident, Salmaniya Medical Complex, Kingdom of Bahrain
[7]Emergency Medicine Resident, Salmaniya Medical Complex, Kingdom of Bahrain
[8]Emergency Medicine Resident, Salmaniya Medical Complex, Kingdom of Bahrain
Corresponding Author: Dr. Ali Haider Ali, **E-mail**: Alihaiderali97@outlook.com

| ABSTRACT
Rheumatoid arthritis is a common rheumatological disease, which is seen more commonly in females, and usually affects the joints. It can affect other connective tissue, yet rarely seen to have cardiovascular features as the initial presentation. This paper presents a case of a young male with the first presentation of rheumatoid arthritis as myopericarditis. The patient is deemed to be unique as such rheumatological illness is not commonly seen in males, and such presentation aids in the understanding of differential diagnosis when dealing with such patients.

| KEYWORDS
Cardiology, Pericarditis, Rheumatoid Arthritis, Serositis

| ARTICLE INFORMATION
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1. Introduction

Rheumatoid arthritis (RA) is a common rheumatological disorder characterized by an autoimmune inflammatory disorder that primarily affects the joints. [1] In addition to the articular features of this rheumatological disease, it is also characterized by extra-articular features, some of which overlap with other connective tissue diseases. [2] Cardiovascular disease is a leading cause of mortality and poor prognostic factor in rheumatoid arthritis. [3] There are various cardiac manifestations and non-cardiac vascular complications of rheumatoid arthritis, including pericarditis, myocarditis, coronary artery disease, pericardial effusion, cardiac amyloidosis, atrial fibrillation, stroke, and vasculitis. [4] In this article, we explore an interesting case of a young patient who was presented with features of myopericarditis, which led to the diagnosis of rheumatoid arthritis.

2. Clinical Presentation and History:

A previously healthy 25-year-old Pakistani male presented to the emergency department in October 2024 with left sided pleuritic chest pain and left-sided lower extremity pain localized at the calves, which then migrated to the right lower extremity and a documented fever measuring 38°C. His heart rate was 124 beats/minute, blood pressure measured 144/80 mmHg, pulse oximetry revealed an oxygen saturation of 97% on room air, and core body temperature at the time of assessment was 36.9°C. His clinical examination was remarkable for bilateral knee and ankle joint effusions; otherwise, there were no unusual findings on the physical examination.

3. Initial differential diagnoses and additional investigation findings

His workup showed neutrophilic leukocytosis, with a white blood cell count of 17.09 $x10^9$/L (reference range: 3.4-9.6), neutrophil percentage of 78.5 and absolute neutrophil count of 13.3 $x10^9$/L, elevated inflammatory markers where CRP measured 385 mg/L, and ESR measured 120 mm/hr, and a high procalcitonin 2.09 ng/mL. His initial ECG revealed ST-segment elevation of the inferolateral leads without reciprocal ST-segment depression or T-wave inversion. The first set of cardiac enzymes revealed a Troponin-I level of 1.300 (reference range <0.05) and an elevated D-Dimer 2.70 (reference range: 0.09-0.33).

In the emergency department, the initial clinical examination, laboratory results, and ECG findings raised the suspicion of pulmonary embolism, which deemed an urgent CT Pulmonary Angiogram (CTPA) necessary. However, a CT Angiogram of the chest did not reveal any evidence of pulmonary embolism, the pulmonary trunk appeared mildly dilated, measuring 3.1 cm at its bifurcations, suggestive of pulmonary hypertension.

Although rare at a young age, ST segment changes suggestive of cardiac ischemia alongside elevated cardiac markers, in this presence of chest pain, raised suspicion for acute coronary syndrome. A transthoracic echocardiogram was done for this patient and demonstrated global hypokinesia, which was most severe in the inferioseptal region with a mildly reduced left ventricular ejection fraction, which measured 45%. The patient was planned to undergo primary percutaneous coronary intervention (PCI) at our national cardiac center. However, the patient did not consent to be transferred to the procedure and was subsequently admitted to the coronary care unit (CCU) for close monitoring. Serial cardiac enzymes to measure troponin-I levels, the levels started to rise and reached up to 8.750 ng/mL. Subsequent ECGs performed at 4-hour intervals in the first 24 hours of admission revealed diffuse ST segment elevation in all leads and PR depression. Diffuse ST elevation is more likely to be caused by pericarditis or myocarditis rather than myocardial ischemia. Additionally, PR segment depression is a highly specific ECG feature for myopericarditis. This patient's chest pain resolved within the first few hours in the emergency department and did not reoccur. Furthermore, etiological investigation with viral serologies yielded normal results.

Subsequent clinical examinations with the patient revealed bilateral knee and ankle swelling, which the patient then revealed experiencing joint stiffness typically in the morning as well as bilateral knee and ankle pain for 4 days. This raised the suspicion of underlying connective tissue diseases such as rheumatoid arthritis and systemic lupus erythematosus, which are associated with myocarditis and pericarditis. In light of the joint involvement, there was, although low, a narrow index of suspicion for septic arthritis. Nevertheless, a septic workup involving blood cultures was sent, and they were found to be sterile.

Given the suspicion of an underlying rheumatological disorder, the rheumatology team advised investigating for rheumatological conditions, including rheumatoid arthritis, systemic lupus erythematosus (SLE), and vasculitis. These results were followed by the rheumatology team on an outpatient basis. Rheumatoid factor and antinuclear antibodies test yielded positive results. Subsequently, anti-cyclic citrullinated peptides (anti-CCP) autoantibodies were found to be positive. There were no significant results that yielded from complement levels, c-ANCA levels, or anti-ds DNA in our laboratory. These results allowed us to label this patient with a diagnosis of Rheumatoid Arthritis and attribute the episode of peri myocarditis to this entity of connective tissue diseases.

4. Treatment:

The management of this patient involved a multidisciplinary team, which primarily consisted of cardiology and rheumatology. As mentioned earlier, this patient was admitted to the cardiac care unit (CCU) at our center. The patient was started on a dual antiplatelet therapy regimen consisting of aspirin 81 mg and clopidogrel 75 mg, in addition to bisoprolol and atorvastatin, which was for the initial suspicion of coronary artery disease. Meanwhile, the rheumatology team at our hospital recommended commencing oral prednisolone 20 mg whilst monitoring clinical improvement and inflammatory marker trends. The trend of inflammatory markers was plotted in Figure 1, which shows that there was a rapid downward trend in ESR and CRP with treatment. In Figure 2, we plotted the trend in cardiac enzymes in the first 48 hours, which were done every 6 hours and included – Troponin-I, Lactate Dehydrogenase (LDH), and Creatinine Kinase (CK). In Figure 2, we noticed an initial rise in cardiac enzymes before commencing antiplatelet therapy and a further decrease after commencing oral corticosteroids. Furthermore, a prophylactic proton pump inhibitor (pantoprazole) was added to this patient's treatment regimen. This patient was admitted for

a total of three days; he was discharged after significant improvement of his clinical condition and significant reduction of inflammatory markers ESR and CRP, with a close follow-up after 1 week in the rheumatology outpatient clinic to review the result of the rheumatology workup and further management by the rheumatology team. From the cardiology team, this patient was followed up after 6 weeks in the outpatient clinic. This patient's cardiac medications were not discontinued despite the likely diagnosis until coronary artery disease was ruled out by coronary angiography, which did not reveal any occlusive coronary disease.

5. Discussion:

We present a case of myopericarditis in a young gentleman who presented acutely with fever and pleuritic chest pain, which led to a diagnosis of rheumatoid arthritis as an underlying cause. Initially, this patient was investigated for pulmonary embolism, acute coronary syndrome, and septic arthritis, which were all ruled out before the diagnosis of myopericarditis. This patient's hospital course took a convoluted route before establishing the diagnosis due to dynamic changes in the patient's clinical status and the results of both laboratory and radiology investigations. Eventually, both articular and nonarticular features resolved and responded to the evidence-based medical treatment of myopericarditis, which consisted of oral corticosteroids. In addition to his clinical status, the clinical biomarkers of cardiac injury and inflammatory markers both improved significantly as an inpatient as well as an outpatient, which enabled the rheumatology team to taper down the doses of steroids until discontinuation.

The pathophysiology of myopericarditis in connective tissue diseases such as rheumatoid arthritis is not fully understood. The typical clinical features include pleuritic chest pain, low-grade fever, and pericardial friction rub on examination. Patients with myopericarditis are typically evaluated with routine laboratory investigations, cardiac enzymes, electrocardiogram, and echocardiography. Enhanced imaging techniques such as Cardiac MRI can aid diagnosis in cases of diagnostic uncertainty as it allows for an imaging assessment for myocardial inflammation. However, it requires specialized experience for accurate interpretation to provide a diagnostic utility. [5] Endomyocardial biopsy is an invasive diagnostic test that can confirm the underlying cause of myocarditis in most cases, although it is typically performed in patients with fulminant myocarditis. [6] As for therapeutic management, there is a lack of treatment guidelines that specifically address myopericarditis in connective tissue diseases. [7]

According to the European Society of Cardiology (ESC), aspirin or NSAIDs are the cornerstone of medical therapy and symptomatic treatment for myopericarditis. Based on the medical history, aspirin is favored by other NSAIDs, such as ibuprofen, if there is an indication for antiplatelet therapy or based on the patient's medical history. [8] In this case, there was still a suspicion of coronary artery disease, given the initial ECG changes despite the final diagnosis. Therefore, aspirin was kept as an antiplatelet and beneficial in the treatment of myopericarditis. Gastroprotection is also advised by the ESC, which was present in this case. [9] There is strong evidence to support the benefit of adding colchicine to aspirin or NSAID therapy in cases of myopericarditis. [10] In this case, the suspicion of autoimmune disease has deemed the use of corticosteroids necessary. [11] However, in cases where an infectious cause has been ruled out, and there is no reason to suspect an autoimmune cause, corticosteroids are reserved for failure of first-line therapy as there is evidence to support the risk of encouraging recurrence and dependence. [12] This case reaffirms the importance of interdisciplinary collaboration and the multidisciplinary team (MDT) approach in the field of medicine, which aids in appropriate diagnosis and developing an effective treatment plan.

6. Conclusion:

Myopericarditis is a rare disease with multiple etiologies. We present a case with a convoluted yet short course, which helped aid in the diagnosis of rheumatoid arthritis. Treatment of myopericarditis shares some similarities across multiple etiologies. In cases where autoimmune disorders are suspected as an etiology, corticosteroids. Corticosteroids are indicated in select cases of myopericarditis.

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Appendix 1: Figures

Figure 1: Trend of Cardiac Enzymes across eight sets during admission

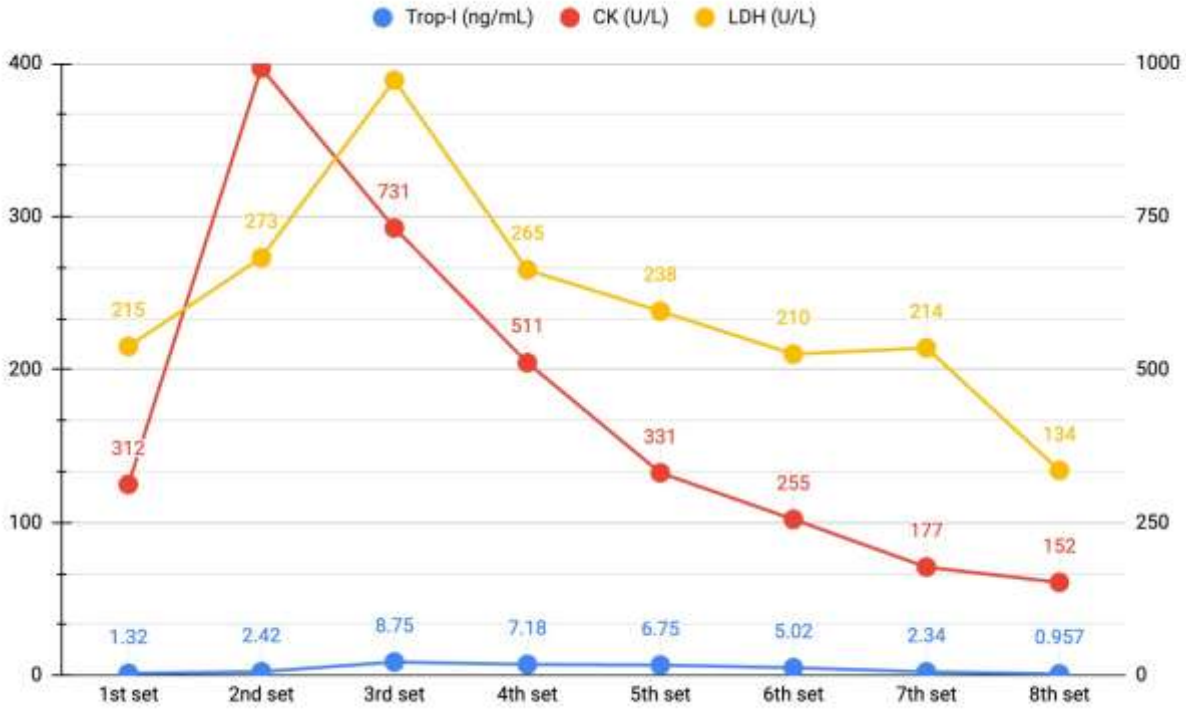

Figure 2: Trend of inflammatory markers and white blood cells during admission