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Case Report

COVID-19 related myopericarditis in a 29-year-old patient: A case report

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ABSTRACT

Introduction: The manifestations of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), also known as COVID-19, are mainly characterized by respiratory symptoms. However, cardiac manifestations such as acute myopericarditis have been reported to be associated with COVID-19 infection.

Case Description: A 29-year-old female patient presented with a 2-day history of fever, cough, runny nose, and myalgia, and tested COVID-19 positive at Penang General Hospital, Pulau Pinang, Malaysia. On day 2 of admission, the patient complained of acute onset central chest pain, radiating to her back, associated with cold sweat, shortness of breath and generalised body ache. On examination, tachycardia and tachypnoea were elicited. The serial electrocardiography (ECG) showed persistent non-specific sinus tachycardia. Troponin T level was elevated at 99 ng/L (normal <15 ng/L) and creatine kinase (CK) was at 10990 U//L (normal: <190 U/L). Her chest radiograph revealed cardiomegaly and otherwise clear lung field. CT pulmonary angiogram demonstrated evidence of bilateral pleural effusion and pericardial effusion and ruled out pulmonary embolism. The diagnosis of myopericarditis was established based on clinical, electrocardiographic, radiological, and biochemical findings. She was treated successfully with IV morphine, oral colchicine, ibuprofen, and oxygen therapy. A follow-up echocardiogram 10 weeks post-COVID demonstrated complete resolution of pericardial effusion, with an election fraction of >70%.

Conclusion: COVID-19 patients may develop severe cardiac complications such as myopericarditis. Clinicians should have a high index of suspicion of COVID-related myopericarditis in COVID-19 management. Further study should be implemented to investigate the association between COVID-19 and myopericarditis.

1. Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), also known as COVID-19, was declared a pandemic by the World Health Organization in March 2020.[1] The manifestations of COVID-19 are mainly characterized by respiratory symptoms such as cough and sore throat. Nonetheless, COVID-19 can also affect the cardiac system. Cardiovascular manifestations such as acute myopericarditis have been reported to be associated with COVID-19 infection.[2] In our case report, we describe a young 29-year-old female with COVID-19 infection who presented with acute myopericarditis in Penang General Hospital, Malaysia.

2. Case Presentation

A 29-year-old female patient presented with a 2-day history of fever, cough, runny nose, and myalgia at Penang General Hospital, Pulau Pinang, Malaysia. She tested positive for COVID-19 by a rapid test kit (RTK), followed by a positive COVID-19 Gene Xpert with a CT value of 35.8. On day 2 of admission, she complained of acute chest pain. Further history revealed that the

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pain was over the central and left side of her chest with radiation to her back and was associated with cold sweat, shortness of breath, and generalised body aches. Her chest pain was worsened by physical exertion and deep breathing. She has no underlying cardiovascular disease and denied a family history of cardiovascular disease. On examination, she was tachypnoeic and tachycardic (respiratory rate: 24 breaths/min, heart rate: 123 beats/min). Other vital signs were within the normal range (blood pressure: 111/80 mmHg, temperature: 36.7-degree Celsius, oxygen saturation: 98% under room air). Her cardiovascular examination was unremarkable with no audible murmur. Her lungs were clear on auscultation.

Urgent electrocardiography (ECG) showed non-specific sinus tachycardia, which was persistent with no dynamic changes in her serial ECGs (Figure 1). Her cardiac biomarkers were significantly raised; troponin T level was elevated at 99 ng/L (normal: <15 ng/L), and creatine kinase (CK) was at 10990 U/L (normal: <190 U/L). Inflammatory markers such as C reactive proteins (CRP) were raised, while the erythrocyte sedimentation rate (ESR) was normal (CRP: 24; ESR: 2). The autoimmune screen was negative (anti-nuclear antibodies, rheumatoid factor, thyroglobulin antibody and thyroid peroxidase antibody). Other blood investigations, including full blood counts, renal profile, liver function test, thyroid function test, coagulation profile, D-dimer, and biohazard screen were unremarkable. Her chest X-ray (CXR) revealed cardiomegaly and otherwise clear lung fields (Figure 2). CT pulmonary angiogram (CTPA) was done to rule out pulmonary embolism. While it demonstrated no evidence of pulmonary embolism, it revealed pericardial effusion measuring up to 0.9cm in thickness in the right ventricle region. Bilateral pleural effusions, consolidations, and ground glass opacities at the left lung upper and lower lobes were also demonstrated, suggesting left lung infective changes (Figure 3). The diagnosis of myopericarditis was established based on clinical features, electrocardiographic findings, radiological findings (pericardial effusion), and raised cardiac biomarkers. Her myopericarditis was attributed to being COVID-related after ruling out other potential causes of myocarditis and pericarditis.

She was treated with intravenous morphine, oral colchicine (0.5mg once daily), and oral ibuprofen (600mg 3 times daily). A good response was observed with the resolution of her chest pain within 4 hours of treatment commencement. Both her serial CK and CRP were in reducing trends. She was hemodynamically stable throughout this admission and complications of myopericarditis such as arrhythmias, syncope, and acute heart failure were not observed. She was discharged well on day 8 of admission. A follow-up at the cardiology clinic with a transthoracic echocardiogram 10 weeks post-COVID demonstrated complete resolution of pericardial effusion, with an election fraction of >70% and no regional wall motion abnormalities (Figure 4).

3. Discussion

This case report aims to describe the symptomatology, clinical findings, and management of COVID-related myopericarditis observed in a young 29-year-old lady with no previous known cardiac-related medical illness. In previous reported cases, most patients are found to have underlying cardiovascular morbidities, which prone them to be at higher risk of developing COVID-related cardiovascular disease.[3–5] Typically, the clinical manifestation of myopericarditis tend to overlap with COVID-19 symptomatology, making the diagnosis challenging.

Although the spectrum of clinical presentations of COVID-19 is largely associated with the respiratory system,[6] cardiovascular manifestations of COVID-19 have been reported such as acute coronary syndrome (ACS), heart failure and myocarditis.[3,4] Acute myopericarditis refers to the inflammation of the myocardium and pericardium. Acute pericarditis is diagnosed in the presence of two or more of the following diagnostic criteria: (i) characteristic chest pain, (ii) pericardial friction rub, (iii) electrocardiograph (ECG) changes of widespread concave ST segment elevation with PR depression, (iv) new onset or worsening pericardial effusion.[7] Myopericarditis is diagnosed with the presence of one additional feature: elevated cardiac biomarkers, presumed new left ventricular systolic dysfunction based on echocardiography or cardiac magnetic resonance imaging (cMRI), and evidence of myocardial inflammation by cMRI.[7]

In this case, the clinical features of acute chest pain and shortness of breath are consistent with the previous cases reported.[4] Typical ECG findings of pericarditis such as diffuse concave ST-segment elevation and PR depression were not observed. Myocardial involvement in our case was supported by markedly raised troponin, a sensitive cardiac biomarker of myocardial inflammation. A negative autoimmune screen effectively ruled out autoimmune causes. Her chest X-ray revealed cardiomegaly, which is consistent with the commonest radiographic finding in previous COVID-19 myocarditis cases reported.[4] The diagnosis of myopericarditis was established based on clinical features (acute chest pain), radiological findings (new onset pericardial effusion), and raised cardiac biomarkers. Her myopericarditis was attributed to being COVID-related after ruling out other potential causes of myocarditis and pericarditis.

A transthoracic electrocardiogram (TTE) is recommended for patients who are suspected of having acute myopericarditis. Cardiac magnetic resonance imaging (cMRI) is a useful non-invasive diagnostic test to assess the degree of myocardial and pericardial involvement.[3,5,7]

Endomyocardial biopsy (EMB) is often considered the criterion standard for the diagnosis of myocarditis as it could identify the nature of inflammation and its relationship with COVID-19 via immunohistology and genome analysis. However, routine EMB in establishing the diagnosis of COVID-related myopericarditis is rarely done due to its risk of infection and limited availability.[3] In our case, both cMRI and EMB were not conducted due to limited availability for patients with COVID-19 infection in the local hospital.

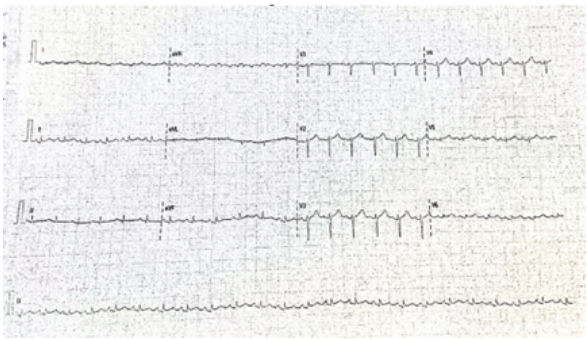

Fig. 1: ECG showing non-specific sinus tachycardia

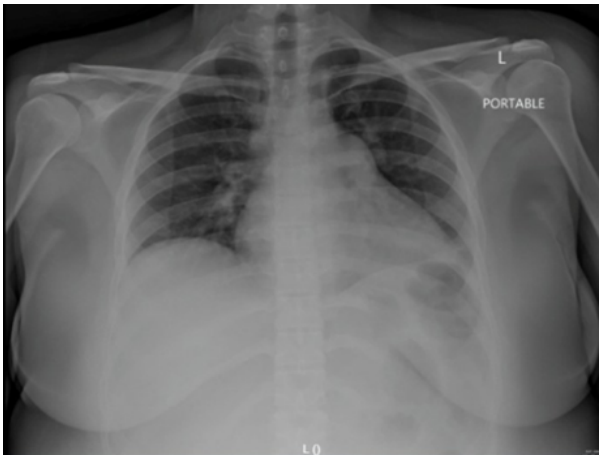

Fig. 2: Chest X-ray showing cardiomegaly and clear lung fields

The overall prognosis of COVID-related myopericarditis is good with no long-term sequelae in most patients. The clinical presentation of COVID-related myopericarditis varies from a subtle, self-limiting illness to severe cardiogenic shock. The treatment is largely supportive and should always be tailored to the individual clinical presentation, however there is limited data are available to guide the treatment of COVID-related myopericarditis. In this case, combination therapy of non-steroidal anti-inflammatory drugs (NSAIDs) and colchicine was used as the first line of management, which led to good clinical and biochemical response. Antivirals such as ritonavir and inflammation-modulating agents like tocilizumab and

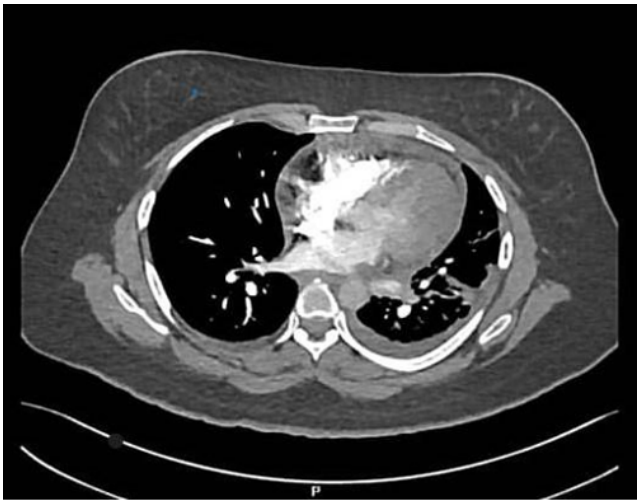

Fig. 3: CTPA demonstrating pericardial effusion measuring up to 0.9cm in thickness in the right ventricle region, bilateralpleural effusions, consolidations, and ground glass opacities at the left lung field.

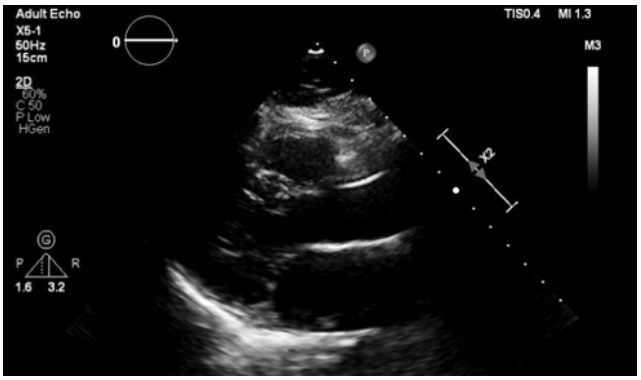

Fig. 4: Transthoracic echocardiogram at 10 weeks post-COVID demonstrating complete resolution of pericardial effusion

baricitinib were not administered in her care. Other treatment modalities to manage COVID myocarditis include glucocorticoids (58%) and immunoglobulin therapy (25%).[4]

4. Conclusions

Our case demonstrates that COVID-19 could be complicated by cardiac manifestations such as acute myopericarditis even in a young patient with no known cardiovascular risk factor. Hence, clinicians should have high clinical suspicion for early recognition and prompt treatment commencement for COVID-related acute myopericarditis. Large multicentre studies are recommended to elucidate further on the cause-effect relationship between COVID-19 infection and myopericarditis, as well as to establish clinical practice guidelines for COVID-19 myopericarditis.

5. Consent Statement

Informed consent was obtained from the patient.

6. Source of Funding

None.

7. Conflict of Interest

None.

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