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CASE REPORT : MYOPERICARDITIS IN 19 YEARS OLD MALE

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ABSTRACT
Inflammation of the pericardium due to either an infectious or non-infectious etiology, which is known as pericarditis. About 1% of hospitalized patients had this illness, and 5% of those patients complained of chest discomfort. About one-third of individuals with acute pericarditis had cardiac involvement, suggesting that the two diseases may have similar etiologies. We describe a 19-year-old man who experienced chest discomfort and was later diagnosed with myopericarditis. Empirical treatment utilizing NSAIDs and colchicine produced excellent outcomes in terms of symptom relief, ECG improvement, and CRP decrease. To reduce morbidity and mortality from sudden cardiac death, it is critical to recognize the signs and symptoms that fit the criteria for myopericarditis..
Keywords: *chest pain; myocarditis; myopericarditis; pericarditis*

INTRODUCTION

Inflammation of the pericardium due to either an infectious or non-infectious etiology, which is known as pericarditis.[1] About 5% of individuals came with chest pain and 0.1% case hospitalized for this condition.[2] Myocarditis is an inflammatory disorder that affects the heart muscle and is caused by infections, exposure to harmful drugs, and a triggered immunological response.[3] About one-third of patients with acute pericarditis have myocardial involvement, which suggests that the two diseases may have similar etiologies. In addition, overlapping forms may occur in clinical practice.[1,4] Here we present a case of 19 years old male with chest pain diagnosed as myopericarditis.

CASE REPORT

A 19 years-old male patient came to the emergency unit with complaints of chest pain that had been come and gone since 2 days before he was admitted to the hospital. According to the patient, chest pain was like being stabbed, radiated to the jaw and left arm, felt worse when lying down and improved slightly when sitting. The patient also complained of weakness and nausea. The patient had complained of sore throat 1 week before but was said to be improving on its own. No complaints of vomiting, fever, cold sweat. History of diabetes, hypertension, autoimmune, and previous similar complaints in the patient and family was denied. The patient admitted that he smoked but never drank alcohol. The patient's occupation is a wood factory worker. On physical examination found patient was compos mentis, blood pressure was 105/66 mmHg, the heart rate was 75 bpm regular, respiration frequency was 20 times per minute, temperature 36.5°C, and O2 saturation was 100% with free air. Auscultation examination of the heart revealed no pericardial friction rub, murmur, or gallop with normal heart sound. On the first day of examination, the ECG revealed a regular sinus rhythm with diffuse ST-segment elevation in leads I, II, III, aVF, V2, V3, V4, V5, V6.

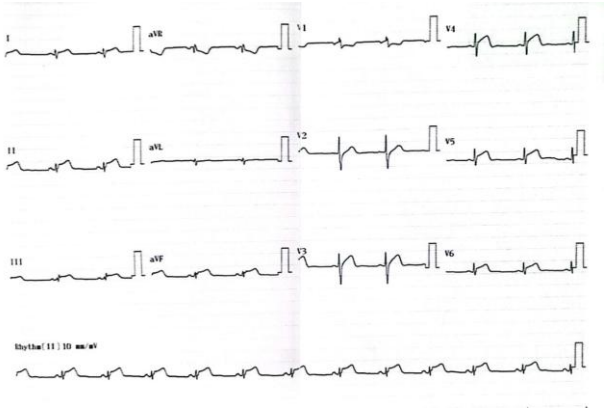

Figure 1.ECG result at emergency room

On laboratory examination, there were founded elevation on white blood cell count (11,31x103/mm3), SGOT (215 U/L), CK-MB (228 U/L), LDH (523 U/L), and CRP (11,98 mg/L), other laboratory examination was found to be in normal limit. Chest x-ray examination did not reveal any significant abnormalities with CTR <50%. Echocardiographic examination did not reveal any

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pericardial effusion or impaired cardiac function with ejection fraction of 70.6%, but pericardial thickening was found.

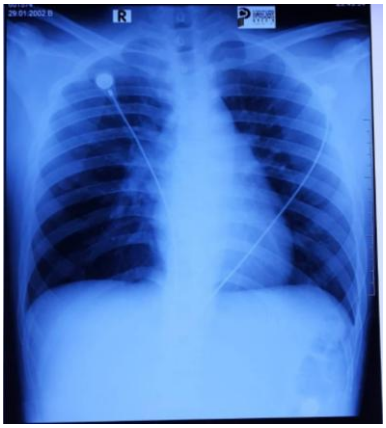

Figure 2. Chest Xray result

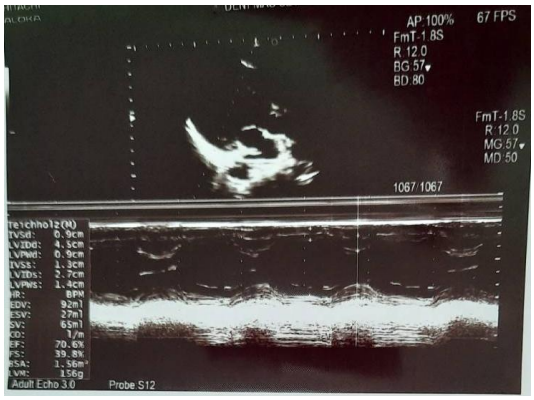

Figure 2. Echocardiography result

Patient was then hospitalized with a diagnosis of acute myopericarditis. The therapy given to this patient was aspirin tablets 100 mg/24 hours, ibuprofen tablet 600 mg/8 hours, cochicine tablet 0.5 mg/24 hours, and gastroprotector using pantoprazole iv 40 mg/12 hours, sucralfate syrup 1 table spoon/8 hours. During treatment for 8 days, chest pain symptomps were monitored reduced to complete resolution. Re-examination of CRP on day 7th found a significant reduction from 11,98 to 4.65 mg/L. The ECG on the last day of treatment showed improvement and evolution in the form of T inversion in leads V3, V4, and V5.

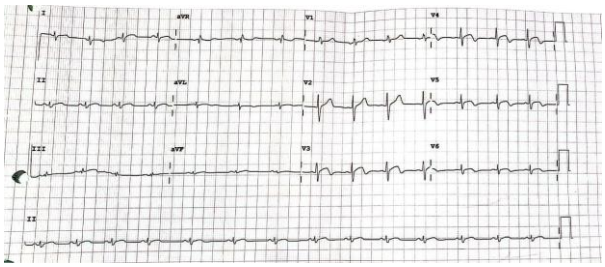

Figure 2. ECG result (last day care)

DISCUSSION

Infection or non-infectious agent may became the etiology of acute myopericarditis. Infection from virus are the most common causes of pericarditis in developed countries, whereas *Mycobacterium tuberculosis* is the most frequent cause of pericarditis in underdeveloped countries. The most frequent noninfectious causes include postcardiac injury syndromes, metastatic malignancies, and autoimmune illnesses.[5] However, a routine assessment failed to identify a specific etiology in roughly 80–90% of cases with acute myopericarditis.[6] Cardiotropic viruses may directly damaged the pericardium or myocardium or may have cytotoxic effects. Injuries to virally-induced myocytes may liberate locked-up intracellular proteins that, in the presence of a genetic predisposition, set off an innate response. Even in the absence of a residual infective agent order, molecular mimicry and epitope spreading may result in postviral myopericardial harm in process. When the pericardium is injured, mediators are produced that boost the transcription of inflammatory molecule precursors and related cytokines needed for the NLRP3 inflammasome to polymerize. These cytokines include large amounts of IL-1b and IL-18. Prostaglandins and thromboxanes are then produced as a result of cascading the arachidonic acid pathway, which is stimulated by another mediator .[7]

The electrocardiogram (ECG) may indicate ischemic alterations or even imitate an ST-segment elevation myocardial infarction in cases of myopericarditis, which can resemble AMI and present promptly with typical ischemic symptoms (STEMI).[8] Acute pericarditis was discovered to be a substantial cause of cardiovascular hospitalizations in 1.7% of patients with ST-segment elevation in those in whom myocardial infarction had been ruled out.[4,9] In general, males had a higher chance of

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having pericarditis than women do, and when age increased by a decade, the number of pericarditis-related hospital admissions fell by an estimated 51%..[9] In our case, the patient complaining pleuritic chest pain and diffuse ST elevation from the ECG which met 2 of 4 diagnostic criteria for acute pericarditis according to European Society of Cardiology (ESC).[4] Another important factor was pericardial friction rub, which was only identified in 33% of instances and could have gone unnoticed because it can only be heard in the first few hours.[4,6] Echocardiography evaluation was also advised and became one of the key criteria to identify acute pericarditis. If pericardial effusion was discovered, which was in >60% of patients and was typically a minor effusion.[4] The onset of acute pericarditis symptoms was frequently accompanied by gastrointestinal or flu-like symptoms.[7]

As a result of the inflammatory process, C-reactive protein (CRP), LED, and white blood cells are frequently observed in pericarditis. The ability of CRP to serve as an indicator of inflammation and as a benchmark for therapeutic effectiveness.[4] Increased cardiac markers, such as creatine kinase (CK) and troponin, indicate cardiac muscle injury and myocardial inflammation, both of which were present in this case.[4,7,10] Unless the amount of pericardial effusion is greater than 300 ml, a normal thorax x-ray is often detected.[4,6] Coronary angiography and cardiac MRI are recommended to rule out acute coronary syndromes and confirm myocardial involvement.[5] About one-third of patients had pericarditis with myocardial inflammation involved; this finding is mostly explained by the possibility that the two conditions may have similar etiological causes.[11] Despite being infrequently used, endomyocardial biopsy is the gold standard for diagnosis. It still has a limited role and is rarely used unless the patient is not responding adequately to medical treatment because to its poor sensitivity.[12] Myopericarditis is often diagnosed based on a combination of chest discomfort, pericardial rubs, indications of inflammation, electrocardiograms, and echocardiograms that reveal normal wall motion and ejection fraction along with troponin release that suggests myocardial injury.[2,10]

Patients who exhibit at least one high-risk prognostic indicator, such as a high temperature (above 38 °C/100.4 °F), subacute onset, a large pericardial effusion (more than 20 mm on an echocardiogram), cardiac tamponade, a failure to respond to nonsteroidal anti-inflammatory drugs (NSAIDs) within 7–10 days, myopericarditis, oral anticoagulant therapy, immunosuppression, and trauma are encouraged to stay in the hospital.[4,5] Limiting physical activity other than routine daily activities and returning CRP to normal are suggested non-pharmacological therapy methods. Only after symptoms subside and diagnostic exams, such as CRP, ECG, and echocardiogram, have been negative for at least 3 months in the case of patients who are not participating in competitive sports and 6 months in the case of athletes after the initial attack, are athletes advised to return to competitive sports.[4,13] The preferred course of treatment for acute pericarditis is aspirin or an NSAID, which must be paired with colchicine to avoid treatment failures and recurrences.[4] When symptoms have subsided and CRP has returned to normal (<3.0 mg/l), the dosage can be progressively decreased.[7] Duration is determined by symptoms and CRP, although in general, for simple patients, it lasts for 1-2 weeks. Gastroprotection should be given to prevent mucosal damage in the gastrointestinal tract brought on by long-term NSAID use and colchicine. Only patients with contraindications, failure of aspirin or NSAIDs, a poor response, or unique patient situations, such as those with autoimmune pericarditis, are given corticosteroids as a second option[4,7]

The long-term prognosis for most people with acute myopericarditis is good. The etiology has a significant impact on the myopericarditis prognosis[4,7,14] The 1.1% in-hospital death rate for acute pericarditis increases with age and serious coinfections (pneumonia or sepsis)[4] A dilated cardiomyopathy will eventually develop in 36% to 40% of myocarditis patients. About 9–50% of people with dilated cardiomyopathy experience myocardial inflammation.[15]

CONCLUSION

Here we reported a 19 years old male with myopericarditis. Myopericarditis is a rare causes of chest pain but can occur espescially in young adult. Recognize the sign and symptoms based on the criteria to diagnose myopericarditis is an important

thing to prevent morbidity and mortality due to sudden cardiac death. Empirical therapy for myopericarditis using NSAIDs and colchicine would gave satisfactory result.

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