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Case Report

Myopericarditis in 19-year-old Male: A Case Report

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ABSTRACT

Pericarditis is an inflammation of the pericardium which may be caused by infectious or noninfectious etiology. This disease recorded about 0,1% of hospitalized patient and 5% patients came with chest pain. Involvement of myocardial in acute pericarditis recorded in about one-third patients due to both diseases might share common etiologies. **Case Summary.** We present a case of 19 years old male with chest pain diagnosed as myopericarditis. **Discussion.** Treatment using empirical therapy with NSAIDs and colchicine gave satisfactory result in resolution of symptoms, ECG, and reduction of CRP. 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.

Highlights:

1. A classic, yet, interesting case of myopericarditis.
2. Pericarditis with involvement of myocardial inflammation often share etiological agents, but both are different from each other.

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Introduction

Pericarditis is an inflammation of the pericardium which may be caused by infectious or noninfectious etiology[1]. This disease recorded about 0,1% of hospitalized patient and 5% patients came with chest pain[2]. Involvement of myocardial in acute pericarditis recorded in about one-third patients due to both disease might share common etiologies, and overlapping forms may be encountered in clinical practice[1,3]. Here we present a case of 19 years old male with chest pain diagnosed as myopericarditis.

Case Presentation

A 19 years-old male patient came to the emergency unit with complaints of chest pain that had been come and gone for 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.

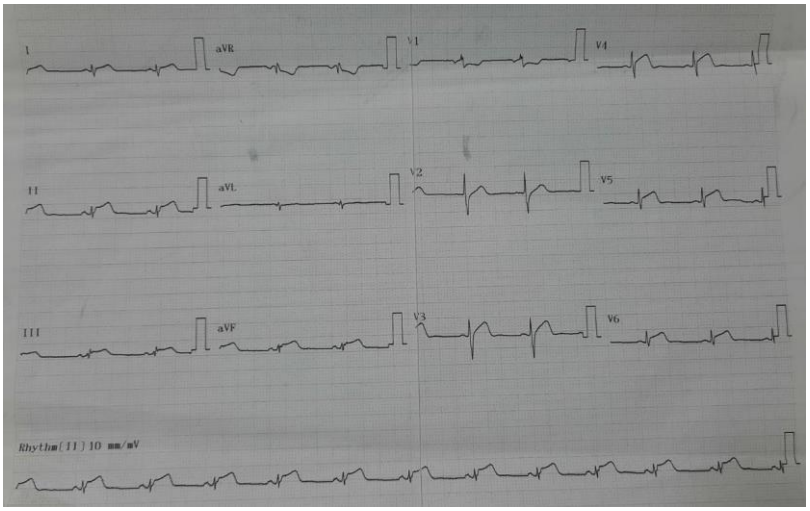

Figure 1. The ECG at emergency room

On laboratory examination, there were found elevation on white blood cell count (11,31x10^3/mm^3), 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 pericardial effusion or impaired cardiac function with ejection fraction of 70.6%, but pericardial thickening was found.

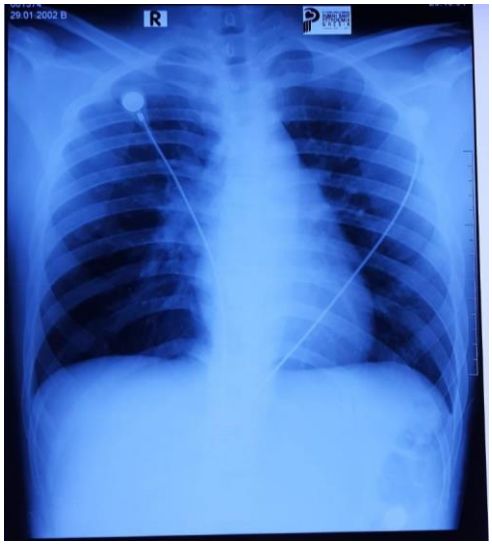

Figure 2. Chest X-ray

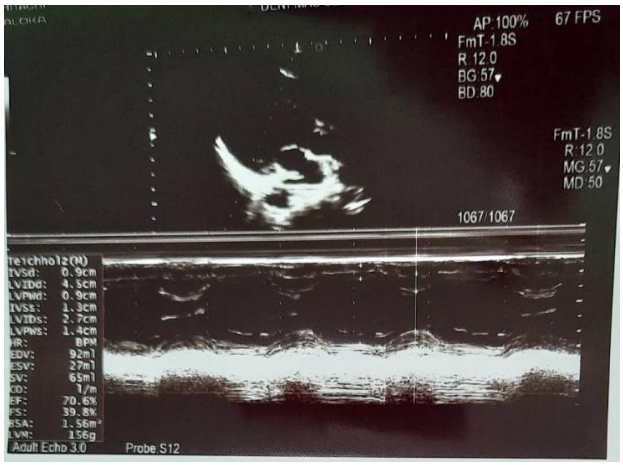

Figure 3. Echocardiography Examination

The 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.

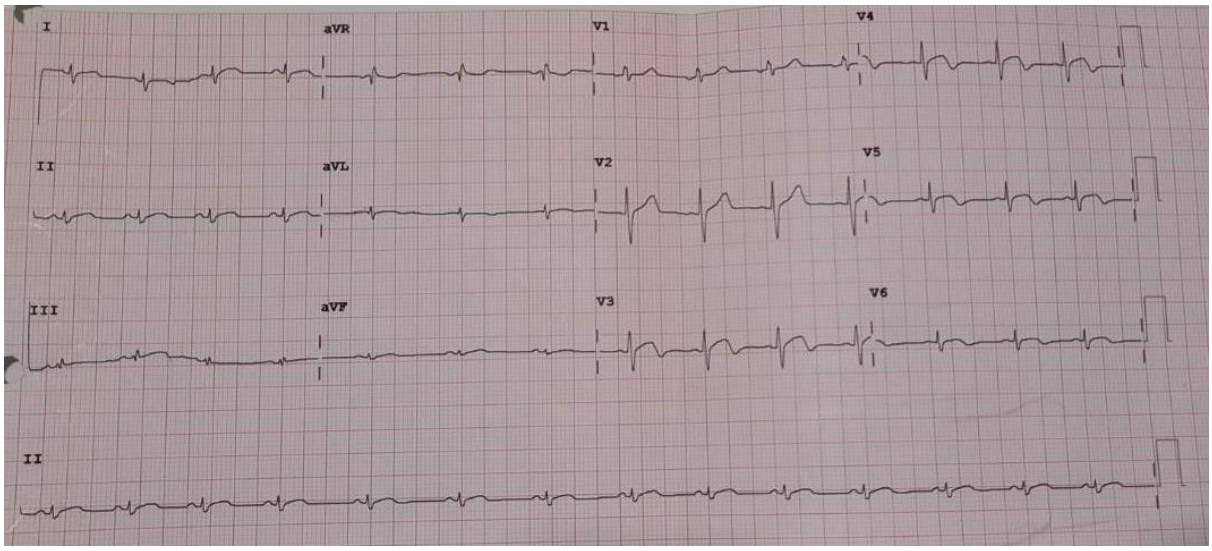

Figure 4. ECG in last day care

Discussion

Acute pericarditis could be caused by infectious or noninfectious agent, viral infections are the most common etiologies for pericarditis in the developed countries while Mycobacterium Tuberculosis is the most frequent cause of pericarditis in developing countries. The most prevalent noninfectious causes are secondary to autoimmune diseases or metastatic tumors, as well as postcardiac injury syndromes[4]. Nevertheless, about 80-90% the cause of acute pericarditis was idiopathic which there was no definite cause found after a routine evaluation[5]. Cardiotropic viruses can cause damage to pericardium and myocardium via direct cytolysis or cytotoxic effects. Viral-induced myocyte injury might cause released of sequestered intracellular proteins that trigger an innate reaction within the presence of a predisposing genetic background. Molecular mimicry and epitope spreading may cause in progress postviral myopericardial damage even in the absence of residual infective agent order[3,6]. Injury to the pericardium leads to release mediators which increases the transcription of precursors of inflammatory molecules and associated cytokines required for the polymerization of the NLRP3 inflammasome, released massive IL-1b and IL-18. Other mediator stimulates synthesis of phospholipase-A2 requiring for cascading the arachidonic acid pathway and the subsequent synthesis of prostaglandins and thromboxanes.[7]

In patients whom myocardial infarction has been ruled out, 1.7% of patients with ST-segment elevation were found to be acute pericarditis as a significant cause of cardiovascular admissions among young adults[3,8]. Men in younger ages have higher risk for pericarditis rather than women in general and the incidence of admission due to

pericarditis declined by an estimated 51% per 10-year increase in age.[8]

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)[3]. The other main criteria were pericardial friction rub which only found in ≤33% of cases and might be missed because it can only be heard in the first several hours[3,5]. Echocardiography examination was also recommend and became one of the main criteria to diagnose acute pericarditis if it was found pericardial effusion which found in >60% cases and usually mild effusion[3]. Symptoms that arised in acute pericarditis often preceded by a gastrointestinal or a flu-like syndrome[7]. C-reactive protein (CRP), LED, and white blood cell commonly found arise in pericarditis as there were inflammation process. CRP could be used as a marker of inflammation also became the guide to evaluate therapy progress[3]. Increased of cardiac marker mark out damage in cardiac muscle as an involvement myocardium inflammation which in also shown in this case such as creatine kinase (CK) and troponin[3,7,9]. Thorax xray usually found to be normal unless pericardial effusion exceeds 300 ml causing an increase in the cardiothoracic ratio[3,5]. coronary angiography and cardiac MRI are recommended to rule out acute coronary syndromes and confirm myocardial involvement.[4]

Pericarditis with involvement of myocardial inflammation recorded in about one-third patients, the main reason for these findings is that pericarditis and myocarditis may share common etiological agents[6]. The diagnosis of myopericarditis is usually based on the combination of chest pain, pericardial rubs, signs of inflammation, electrocardiographic and echocardiographic examination which shown normal ejection fraction and wall motion, associated with troponin release suggesting myocardial damage.[2,9]

Patients who exhibit at least one high-risk prognostic factor are recommended to be hospitalized Risk factors include high fever (>38 °C/100.4 °F), subacute onset, large pericardial effusion (more than 20 mm on echocardiography examination), cardiac tamponade, failure to respond within 7–10 days to nonsteroidal anti-inflammatory drugs (NSAIDs), myopericarditis, immunosuppression, trauma, and oral anticoagulant therapy.[3,4]

The recommended non-pharmacological treatment options are limiting physical activity other than daily activities until symptoms resolve and normalization of CRP. It is recommended that athletes return to competitive sports only after symptoms resolve and diagnostic tests, namely CRP, ECG, and echocardiogram, have been normal for at least 3 months for patients who are not involved in competitive sports and 6 months in athletes after the initial attack.[3,10]

Aspirin or NSAIDs are the treatment of choice in acute pericarditis and must combined with colchicine to prevent treatment failures and recurrences[3]. In conditions where symptom resolution and CRP normalization have occurred (<3.0 mg/l), the dose can be gradually reduced[7]. Duration is symptoms and CRP guided but generally 1–2 weeks for uncomplicated cases and gastroprotection should be provided to protect mucosal damage in gastrointestinal tract as an effect from long term NSAIDs and colchicine. Corticosteroids are only considered as a second choice in patients with contraindications and failure of aspirin or NSAIDs, unsatisfactory response, or in special patient conditions such as patients with autoimmune pericarditis.[3,7]

Conclusion

Here we reported a 19 years old male with myopericarditis. Myopericarditis is a rare causes of chest pain but can occur especially 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 gave satisfactory result.

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There is no conflict of interest.

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