A CASE REPORT OF 23-YEARS-OLD YOUNG WOMAN WITH PERICARDIAL EFFUSION IN SYSTEMIC LUPUS ERYTHEMATOSUS DIAGNOSED WITH ECHOCARDIOGRAPHY AND CT-SCAN

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ABSTRACT

Pericardial effusion (PE) is not a daily case in the emergency room (ER). It has a wide spectrum of etiologies, and one of them is autoimmune diseases like systemic lupus erythematosus. PE in SLE patients could increase morbidity and mortality rates. The clinical manifestation and laboratory findings might not be specific. Imaging modalities, such as emergency echocardiography and CT scan, are important for diagnosing PE and evaluation beyond to the adjacent structures. We presented a 23 years-old female diagnosed with systemic lupus erythematosus (SLE) and cardiac involvement of PE diagnosed with imaging modalities, echocardiography, and CT scan. In this case, echocardiography is the most available and reliable imaging modality for pericardial effusion, and CT usually obtains to clarify or to rule out the cause of an effusion rather than confirm the diagnosis.

Keyword: *Pericardial Effusion, Systemic Lupus Erythematosus" Echocardiography, CT-scan*

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INTRODUCTION

Pericardial effusion (PE) is not an every-day case found in the emergency room (ER). To date, the incidence of PE is wide-ranging between medical centers. However, a single-centered study report from 65,000 total patients in ER shows 1003 PE patients with a chief complaint of dyspnea when arrived at ER.[1] However, PE could be asymptomatic and, on the other hand, could be life-threatening, especially if it quickly progresses into cardiac tamponade.[2] Despite PE could be one of the concerning conditions at ER, both to diagnose and figure out the PE etiology could be challenging.[1,3] Clinical signs leading to the PE diagnosis (such as distended jugular veins, pulsus paradoxus, or electrical alternans) might not appear on every single PE case or could emerge late in the disease process.[1] Those situations could be worsening the condition of the ER

physician. However, to diagnose this condition, physicians need some imaging modalities.[3,4]

Sometimes PE could be related to a known underlying disease, like autoimmune diseases.[5-7] The systemic lupus erythematosus (SLE) is classified as a systemic autoimmune disease with unpredictable flare-up and remissions patterns.[5,6] The pathogenic immune-complex could deposit into several internal organs, like the pericardial.[8] Pericardial Effusion as a manifestation of the SLE is quite high, 40-50%.[8,9] We present the case of a 23-year-old female who undergone persistent dyspnea and was diagnosed with both pericardial effusion and SLE.

CLINICAL PRESENTATION

A-23 years old female came into the emergency room with persistent dyspnea in the past 4 hours before arrival to the hospital. She felt much better when sitting rather than lying down. There were no other exacerbating factors. Additionally, she also had chest discomfort that persisted for 4 hours before admission. She had a history of SLE and was routinely controlled by our internal medicine department. No alteration of the mental status during the admission. Her respiratory rate was 26 times per minute with stable peripheral oxygen saturation, 97% at room air. Her blood pressure was normal (107/83 mmHg), and her heart rate 127 bpm. Unfortunately, her temperature was rising 38.3 degrees Celsius. A physical examination revealed a faint sound on the right chest percussion at the 6th intercostal space and diminished vascularity sound at that level and below on auscultation. A muffled heart sound was revealed on heart examination. However, paradoxical pulse, pericardial friction rubs, and increasing jugular vein pressure were absent.

The patient underwent a rapid COVID-19 test (IgM and IgG), dengue NS1 antigen, and CRP check since it was our standard hospital procedure during the pandemic. The result for COVID-19 and dengue were negative; however, CPR quantitative remarkably high (63.2 mg/L). Other complete blood counts also showed abnormal levels; leucocyte 28.000/µL, hemoglobin 9.3 g/dL, hematocrit 27%, thrombocyte 83.000/µL, and high immature platelet fraction (8.9%). The neutrophil differential count was conducted, and the result revealed; high neutrophil and low lymphocyte and eosinophil. The N/L ratio was 14.15 (high), and the plasma albumin was 2 g/dL (low). The ALT and AST results were 9 U/L and 38 U/L, respectively. The ECG examination (figure 1) confirmed tachycardia and low voltage result without electrical alternans.

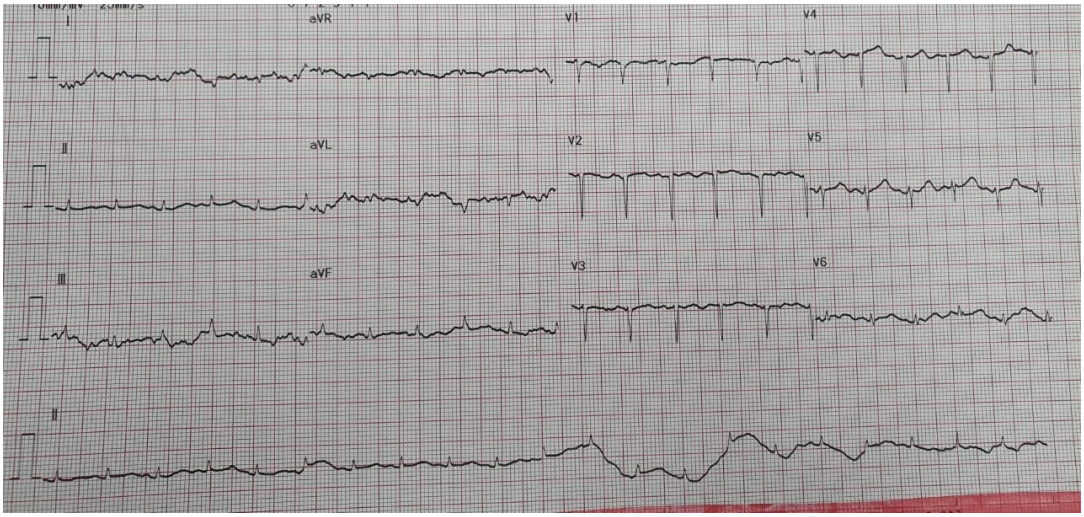

Figure 1. ECG result

The chemistry panel result like natrium, potassium, and chloride within normal values. On the other hand, urea and creatine significantly rose; 6.5mg/dL and 203 mg/dL, respectively. The emergency echocardiography exhibited good right ventricular and left ventricular function with an ejection fraction of 58% with global normo-kinetic. Mild hypertrophy with the intraventricular septal end-diastole (IVSd) value was 1.3 cm, and left ventricular posterior wall end-diastole (LVPWd) value was 1.4 cm. Pericardial effusion was found in all parts of the heart (figure 2A and figure 2B).

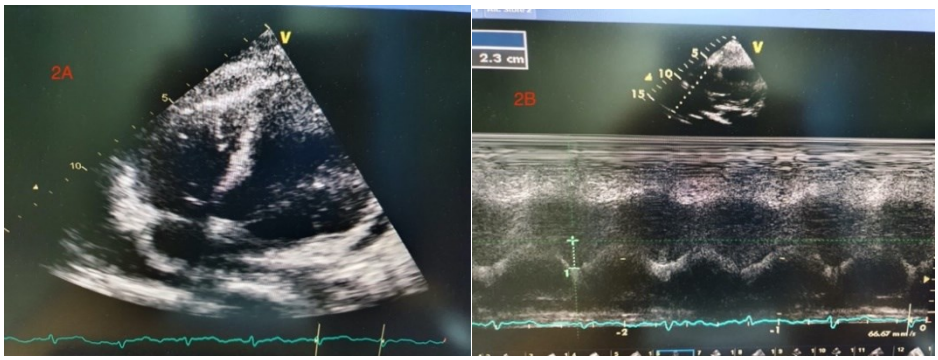

Figure 2A and 2B Echocardiography Result

The Thorax CT displayed right pleura effusion with fibro-infiltrate and fibrosis on the base of the right lung. Another old fibrosis was found on the antero-basal superior lobe, the antero-basal medial lobe of the right lung, and at the base of the inferior lobe of the left lung. The heart imaging revealed cardiomegaly, elongated of the aorta's arch, and pericardial effusion. There was also soft-tissue ~~o~~edema of the right chest. (figure 3)

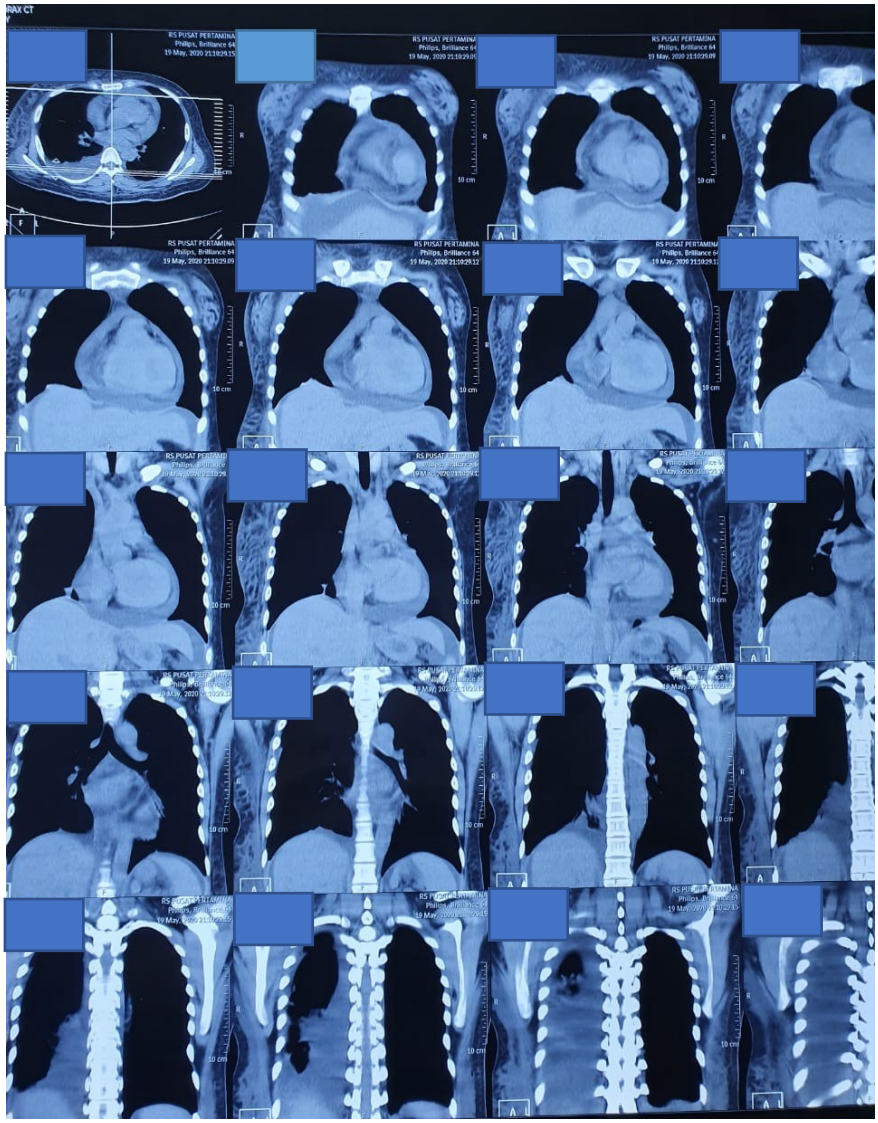

Figure 3 *Thorax CT result*

DISCUSSION

The SLE is characterized by abnormal autoantibodies that had lost their ability to self-tolerance and caused multiorgan inflammation.[10] This could be happening because of the interaction of both genetic factors and environmental triggers. Cardiac involvement is one of the common organs complicated by SLE.[8,11] The morbidity and mortality increases with cardiac involvement.[12] The cardiac complications encompass pericardium, myocardium, endocardium, valvular apparatus, conducting system, and coronary vessels. The dysfunctional innate immune is responsible for organ damage via the release of inflammatory cytokine and the activation of the autoreactive T and B cells.[10] Woman more commonly diagnosed by SLE than man and the contribution of sex hormones might cause it.[13] The Autopsy series shows pericardial involvement in 43-83% inpatient with SLE.[11] Pericardial effusion is observed

during the autopsy, and fluid analysis has tended to show a leucocytosis with increased neutrophils. In this case, our patient is a young female with SLE and diagnosed with pericardial effusion. The blood analysis also found leucocytosis, high neutrophil, and high N/L ratio. The high inflammation process is also visualized by high CRP. During COVID-19 pandemic era, we need to screen the patient with rapid test since it has mimicking presentation.[14]

Echocardiography is the most available and reliable imaging modality for pericardial effusion. Echocardiography should be the initial modality if the pericardial effusion is suspected.[7] Moreover, it provides valuable data correlate with hemodynamic status.[3] Pericardial effusion looks as an echo-free space between the two layers of the pericardium using standard view with two-dimensional (2D) echocardiographic. Four standard views that should be used; subcostal, four-chamber, and parasternal long and short axis.[16] Those views help to determine whether the effusion is global or localized. In our case, we found global effusion but the hemodynamic relatively stable. Sometimes echo finding could be mistaken with pericardial fat, and computed tomography (CT) is a more reliable imaging modality.[15] Nevertheless, CT usually obtains to try and clarify or rule out the cause of an effusion rather than just confirm the diagnosis. Our CT finding shows pericardial effusion and other lung pathological conditions that might be caused by SLE.

CONCLUSION

Pericardial effusion is one of the complications of SLE. Clinical finding sometimes inconsistent with pericardial effusion or appears in the late stage, when hemodynamically unstable. This makes ER doctors more difficult in diagnosing PE. Imaging modalities are needed for diagnosed PE and try to find or rule out the possible aetiologies. A combination of the fast echocardiography modality and CT-SCAN that helps us provide a more holistic picture of the disease is the best combination.

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