Clinical Case Report

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Cardiac tamponade as the initial symptom due to systemic lupus erythematosus in a young man

A case report

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Abstract

Rationale: Systemic lupus erythematosus (SLE) is a connective tissue disease that has many clinical manifestations. However, cardiac tamponade has been rarely reported especially as an initial presenting feature of systemic lupus erythematosus. Herein, we describe a case of cardiac tamponade as the first presentation of systemic lupus erythematosus in a male and presented the course of diagnosis and treatment of this patient.

Patient concerns: A 32-year-old male patient developed a rapid progression of pericardial effusion and he was almost healthy in the past. Vital signs were significantly marked by high fever, tachycardia, and accelerated breathing rate of 37 times per minute. The ANA titer was 1:320 and anti-dsDNA was positive during his hospitalization. The complement levels were decreased but the ESR and the CRP level were increased obviously. Soon after, he appeared anemic and thrombocytopenic.

Diagnoses: The diagnosis of SLE was made based on the clinical and biochemical findings according to 2012 SLICC SLE Criteria.

Interventions: The interventions included use CT-guided pericardial puncture to relieve symptoms in time; utilize high-dose glucocorticoids and immunosuppressants to therapy SLE; closely monitor the vital signs, blood routine, blood biochemical indicators, and volume of pericardial effusion.

Outcomes: After 2 months, the symptoms were disappeared almost completely and TTE showed his pericardial effusion had decreased significantly.

Lessons: We should also keep SLE in mind when assessing male patients with pericardial effusions. Early examinations of sero-immunological markers and closely monitoring the performances are important for the diagnosis of the disease. Early pericardial puncture can quickly relieve symptoms and improve prognosis.

Abbreviations: ADA = adenosine deaminase, ALB = albumin, ANA = antinuclear antibody, C3 = complement C3, C4 = complement C4, CEA = carcino-embryonic antigen, sCl = chlorine, CRP = C-reactive protein, CT = computed tomography, CXR = chest x-ray, EKG = electrocardiogram, ESR = erythrocyte sedimentation rate, GLU = glucose, LDH = lactate dehydrogenase, RBC = red blood cell, SLE = systemic lupus erythematosus, SLICC = Systemic Lupus International Collaborating Clinics, TP = total protein, T-SPOT.TB = T cell spot detection of tuberculosis, TTE = transthoracic Doppler echocardiography, WBC = white blood cell.

Keywords: cardiac effusion, tamponade, systemic lupus erythematosus

1. Introduction

Systemic lupus erythematosus (SLE) is a connective tissue disease that can involve every organ and frequently affects the cardiac system. The common cardiac manifestations of the disease include pericarditis, pericardial effusions, pulmonary hypertension, myocardial lesions, arrhythmias, and valve disease.[1] However, cardiac tamponade is rarely the initial symptom of the disease.[2] We describe the case of a 32-year-old Chinese man in whom cardiac tamponade was the initial manifestation of SLE.

2. Case presentation

A 32-year-old male presented to an outpatient clinic with a 10-day history of fever and progressive dyspnea. The highest temperature was 38.1 °C. He also reported having an intermittent cough and cough-related chest pain. An initial chest x-ray (CXR) showed small effusion and blunting of the right costo-diaphragmatic angle (Fig. 1). The patient's dyspnea gradually became more and more serious. Transthoracic Doppler echocardiography (TTE), which was performed just before the patient was hospitalized, showed a large pericardial effusion and pulmonary artery hypertension (46 mm Hg) (Fig. 2). Many flocculi were present. Further history-taking revealed that the patient had experienced knee pain while climbing stairs for 2 years. He also had a history of allergic dermatitis and had previously undergone a facial hemangioma excision. He reported consuming 250 g of white wine per day for 7 years. He denied smoking or using drugs. The remainder of his medical and family history was unremarkable.

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Statement: The patient has provided informed consent for publication of the case.

The authors have no conflicts of interest to disclose.

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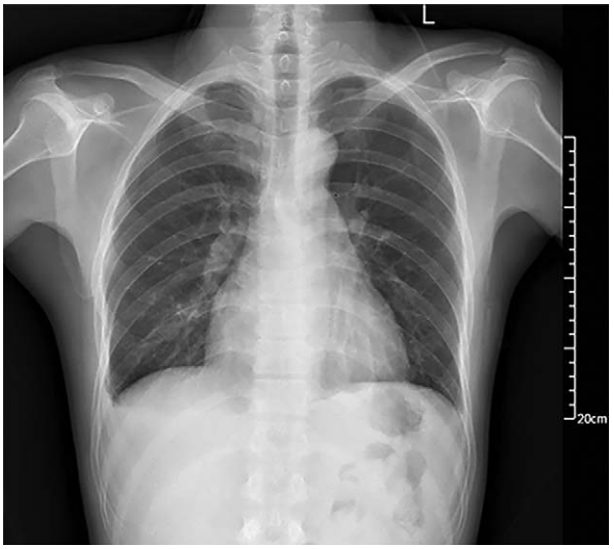

Figure 1. Chest x-ray on admission showing small effusion and blunting of the right costo-diaphragmatic angle.

On physical examination on admission, he was tachycardic (108 bmp) and tachypneic (37/min). His blood pressure was 127/97 mm Hg, and his jugular venous pressure was elevated. On cardiac examination, the patient exhibited diminished heart sounds. On abdominal examination, the lower margin of the liver was below the costal margin. An initial EKG showed atrial flutter, and a repeat CXR showed that the effusion had been larger and resembled a wide-bottom flask (Fig. 3). A chest CT confirmed that a large pericardial effusion was present (Fig. 4).

The patient underwent an emergency CT-guided pericardial puncture, during which 240 ml of hemorrhagic viscous fluid was drained from his chest. Routine laboratory examination of the effusion showed that the RBC was $2.145 \times 10^6/mm^3$, the WBC was $3417/mm^3$, the multinucleated cell percentage was 79.7%, and the specific gravity was 1.023. The fluid was Rivatta positive. Further laboratory examination showed that the TP was 56 g/L, the ALB was 27 g/L, the GLU was 0.5 mmol/L, the LDH was 499 U/L, the Cl was 93.8 mmol/L, the ADA was 17 U/L, and the CEA was < 0.5 ng/mL. The effusion was X-pert negative, and the pathology revealed that large number of RBCs were present. Sero-immunological marker testing showed that the ANA titer was 1:320 and that the patient was anti-dsDNA (luminescence immunoassay) positive. The C3 and C4 levels were decreased, while the ESR and the CRP level were increased much more. Routine analyses performed during the hospitalization indicated that the patient was anemic and thrombocytopenic. The results of other in-hospital serological analyses, including T-SPOT.TB, thyroid hormone, and tumor marker analyses, were normal.

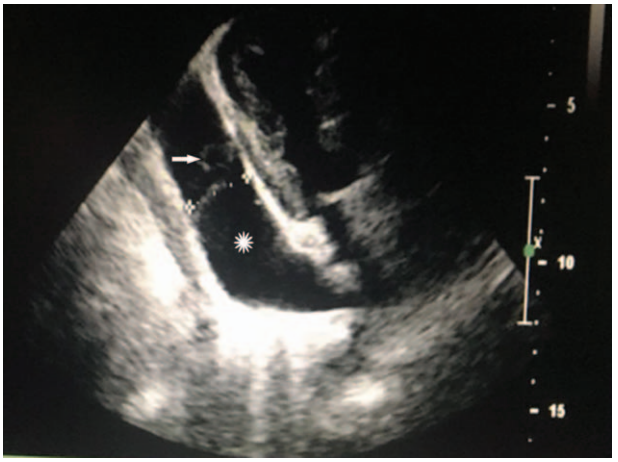

Figure 2. Transthoracic Doppler echocardiography showing a large pericardial effusion and pulmonary artery hypertension. The star marker showing pericardial effusion and the arrow showing fibrous cord shadow.

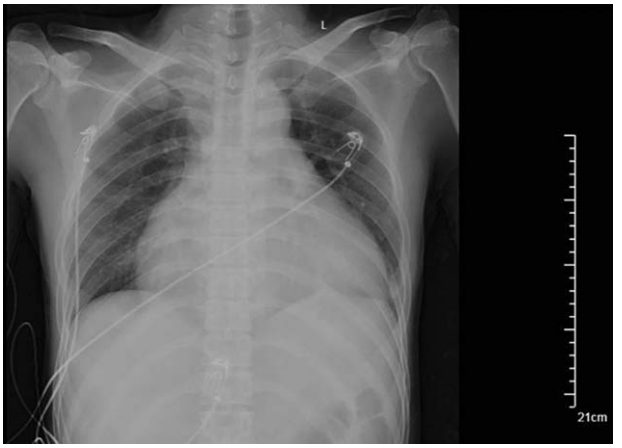

Figure 3. Repeated chest x-ray showing a large pericardial effusion.

The diagnosis of SLE was established according to the 2012 Systemic Lupus International Collaborating Clinics (SLICC) SLE Criteria. The patient's condition improved following treatment with prednisone and cyclophosphamide. Approximately 2 months after his initial presentation, TTE showed that the size of the pericardial effusion had decreased significantly (Fig. 5).

3. Discussion

Systemic lupus erythematosus is a chronic autoimmune disease that can affect virtually every organ system. Females are often affected by the disease. Cardiac involvement is common and more than 50% of patients having cardiac affected.[3] Cardiac manifestations of SLE include pericarditis, myocarditis, valvular disease, thrombosis, and cardiac conduction dysfunction.[4] Whereas pericarditis is most common in SLE patients and it has been associated with poor survival.[5] Almost 25% of patients with SLE develop symptomatic pericarditis at some stage of the disease, most often accompany with pleuritis.[6,7] More than 30% of patients are found to have pericardial involvement by imaging examinations.[8] However, cardiac tamponade is rarely the initial symptom of the disease. One large study of 1300 patients showed tamponade in less than 1% of patients.[9] SLE-related pericardial effusions are mostly yellow exudates and typically reveals inflammatory exudate with neutrophil predominance. Only a few hemorrhagic effusions have been reported. In the treatment of mild pericarditis can use nonsteroidal anti-inflammatory drugs, but medium to severe lupus pericarditis especially tamponade should require corticosteroids other than disease-modifying antirheumatic drug (DMARD) therapy.[10]

Our patient was not the classic female patient, and his initial presentation was cardiac tamponade. The pericardial effusion was hemorrhagic. TTE showed that many flocculi were present in the pericardium; thus, we surmised that the patient suffered from

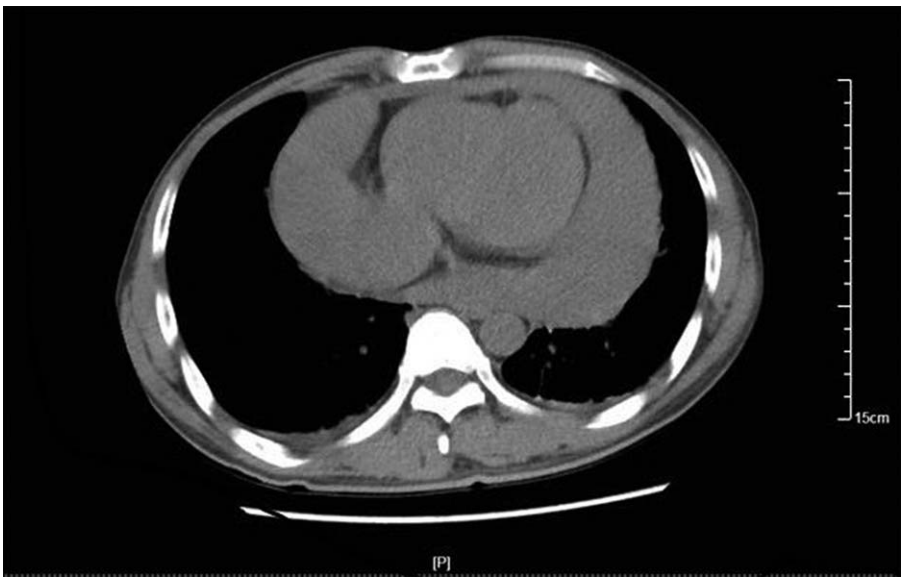

Figure 4. Chest CT showing pericardial effusion. CT = computed tomography.

fibrinous pericarditis with hemorrhage. During the hospitalization, the patient was found to be thrombocytopenic and anemic. No other systems were affected by the disease. Sero-immunological marker testing showed that the ANA titer was positive, while the C3 level was decreased. Our patient fulfilled 4 of the 17 2012 SLICC SLE Criteria.[11] The diagnosis of SLE was made after we determined that the patient's symptoms were not attributable to infection, tumors, hypothyroidism, drugs or other factors. An emergency pericardial puncture relieved the patient's symptoms, and the early application of high-dose glucocorticoids and immunosuppressants had a positive therapeutic effect.

4. Conclusion

Clinicians should keep SLE in mind when assessing male patients with pericardial effusions. Early examinations of sero-immunological markers are important for the diagnosis of the disease. We should closely monitor the performances of other systems in follow-up and watch out for pericardial effusion as the initial symptom of SLE. In the treatment, timely pericardial puncture can gain time for subsequent treatment, including corticosteroids and DMARD therapy.

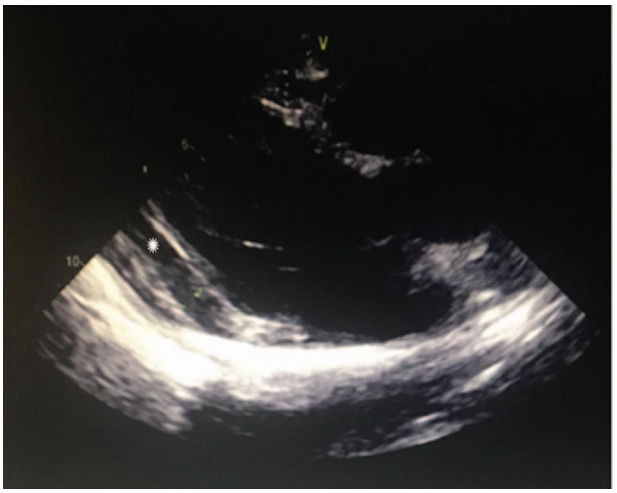

Figure 5. Repeated TTE showing smaller pericardial effusion. The star marker showing pericardial effusion. TTE = transthoracic Doppler echocardiography.

Author contributions

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Formal analysis: Xuan Zhang.
Investigation: Xuan Zhang.
Resources: Xuan Zhang.
Writing – original draft: Xuan Zhang.
Writing – review & editing: Wenbin Wu.

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