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Case report

# Systemic lupus erythematosus presenting as cardiac tamponade—a case report

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KEYWORDS

Cardiac tamponade
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ABSTRACT

Although pericarditis and pericardial effusion (PE) are some of the common manifestations of systemic lupus erythematosus (SLE), the occurrence of cardiac tamponade is quite rare. We present herewith a young girl with cardiac tamponade presenting as initial manifestation of SLE.



## Introduction

Although pericarditis is one of the most common manifestations of systemic lupus erythematosus (SLE) with cardiac involvement, there have been very few case reports of cardiac tamponade as an initial presenting feature of SLE. Here, we present a previously well 17-year-old girl from a West African nation presenting with cardiac tamponade as the initial manifestation of SLE.

A 17-year-old female of Nigerian National, currently studying at a university in the US presented with the primary complaint of the New York Heart Association (NYHA) functional class II dyspnoea for 2 years, which had gradually progressed to a class III dyspnoea on arrival here. She also experienced pleuritic type of retrosternal chest pain, generalised fatigue, and chronic low-grade fever off and on for several months.

She had been fully immunised as a child and did not have a previous history of rheumatic fever or exposure to an active case of tuberculosis. She had been treated for "intercostal muscle strain" with Naproxen for several weeks prior to the visit to our hospital.

On Examination, she was febrile, tachycardia with a pulse rate of 120/min and a blood pressure of 100/80 mmHg. There was pulsus paradoxus, jugular venous distension, impalpable apical impulse, muffled heart sounds, a doubtful gallop, hepatomegaly, and normal respiratory system findings.

An electrocardiogram (ECG) showed PR segment depression as well as diffuse ST segment elevation in all the leads. A chest radiograph showed a "bottle shaped heart" with massive cardiomegaly. The echocardiogram showed a large pericardial effusion (PE) with diastolic collapse of both right atrium and right ventricle suggestive of cardiac tamponade, thickened pericardium, and adhesions.

The blood reports showed anaemia, elevated white blood cell count with predominant neutrophilia, elevated erythrocyte sedimentation rate (ESR), and increased activated partial thromboplastin time (aPTT). The patient was taken up for emergency pericardiocentesis, which was however, aborted due to the inability to introduce and negotiate the guide wire into the pericardial cavity because of thickened pericardium with significant adhesions. Emergency surgical pericardiectomy with window procedure was performed and 1000 mL of sanguino-purulent pericardial fluid was drained. The pericardial fluid contained 1.5 million red blood cells and 4000 white blood cells/mm$^3$ (56% polymorphs, 44% lymphocytes). The pericardial fluid sugar content was 12 mg%, protein was 6.9 g%, and lactate dehydrogenase (LDH) was 21,400. Gram-stain and acid-fast stain revealed no organisms. There was no evidence of malignancy by cytology.

After surgical pericardiectomy, she was started on treatment with non-steroidal anti-inflammatory drugs, and broad spectrum antibiotics for presumed bacterial pericarditis. Although the patient improved symptomatically, she continued to be febrile. Pericardial fluid culture was sterile.

Further investigations were carried out to determine the aetiology. Mantoux testing showed no reaction to tuberculin.

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C-reactive protein was raised, anti-nuclear antibody (ANA) was 4+ positive in 1:40 dilution. Rheumatoid factor was negative. The pericardial biopsy revealed features of chronic non-specific inflammation, with no evidence of any granulomas and negative for acid-fast bacilli. Pericardial fluid polymerase chain reaction (PCR) for tuberculosis and blood cultures was negative. Anti-double stranded deoxyribonucleic acid (ds-DNA) antibodies was positive in 1:10 dilution. Antiphospholipid antibody (APLA) and lupus anticoagulant were positive. The patient had C4 hypocomplementemia, raised aPTT, raised reticulocyte count, positive direct coomb's test, and positive anti-cardiolipin antibody.

The patient's ANA and anti-ds-DNA positivity together with serositis and features of immune haemolytic anaemia satisfied 4 out of the 11 American Rheumatism Association criteria for diagnosing SLE. In this patient, there was also an associated secondary APLA syndrome.

The antibiotics were stopped and the patient was started on intravenous methyl prednisolone 15 mg/kg over 8 hours for 3 days and later changed over to oral prednisolone 90 mg/day (tapering dose) along with non-steroidal anti-inflammatory drugs (NSAID) and hydroxychloroquine.

The patient became afebril and a repeat echocardiogram prior to discharge showed only trace PE with pericardial thickness of only 2–3 mm.

## Discussion

Pericarditis is one of the most common manifestations of SLE accounting for 60% with cardiac involvement. But cardiac tamponade is very rare with only 12 cases reported until 1987 and a report of four cases in 2000 from Brazil.[1] The incidence of cardiac tamponade as an initial presenting feature is extremely rare and has been reported only in four adult cases and two children so far.[1–6] Drug-induced lupus syndromes with hydralazine[7] and procainamide were sometimes reported to present initially as cardiac tamponade. Patients with lupus-induced PE require high doses of prednisolone therapy after pericardiocentesis with concomitant use of hydroxychloroquine to reduce recurrences of serositis in SLE.

This case is reported for the rarity of cardiac tamponade as an initial manifestation of SLE and for the fact that effusion significant enough to warrant a pericardial window, which is extremely rare in SLE.

## Conclusion

The possibility of SLE presenting with tamponade should be considered.

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