



## CASE REPORT

### Impending Cardiac Tamponade as the Initial Manifestation of SLE

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#### Abstract

Systemic lupus erythematosus (SLE) is an autoimmune disorder with protean manifestations. most commonly affecting women Cardiovascular manifestations are common but rare at initial presentation. Our patient presented with impending cardiac tamponade and was subsequently diagnosed with SLE. A 31-year-old female presented with fever, rash, and progressive exertional dyspnea. On examination, she had an elevated JVP and muffled heart sounds and features of pulmonary hypertension her baseline blood investigations showed acute kidney injury, albuminuria, and grossly elevated BNP. Chest radiography showed an enlarged cardiac silhouette and echocardiography showed large pericardial effusion with impending cardiac tamponade for which the patient underwent emergency pericardiocentesis. ANA profile confirmed our diagnosis of SLE with positive anti-dsDNA, anti-nucleosomes, anti-histones, anti-SSA, and anti-Jo 1. Being positive she was treated with pulse steroids and hydroxychloroquine. Renal biopsy was also done which showed class IV lupus nephritis. She improved clinically and was discharged with oral steroids, mycophenolate mofetil, and hydroxychloroquine. Cardiac tamponade is a life-threatening condition and SLE is an important differential to be considered during evaluation.

**Keywords:** SLE, cardiac tamponade, pericardiocentesis

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### Abbreviations

AFB: Acid fast bacilli  
ANA: Antinuclear antibodies  
BNP: Brain natriuretic peptide  
CBNAAT: Cartridge-based nucleic acid amplification test  
CK-MB: Creatine kinase-myoglobin binding  
ECG: Electrocardiogram  
ESR: Erythrocyte sedimentation rate  
JVP: Jugular venous pressure  
KOH: Potassium hydroxide  
LDH: Lactate dehydrogenase  
NSAIDs: Non-steroidal anti-inflammatory drugs  
SLE: Systemic lupus erythematosus  
SLEDAI: Systemic Lupus Erythematosus Disease Activity Index  
TSH: Thyroid stimulating hormone  
WBC: White blood cell

### Introduction

Systemic lupus erythematosus (SLE) is a disease characterized by chronic, autoimmune, multisystem involvement with female preponderance (F: M = 10:1) [1]. The manifestations are protean with half of SLE patients having cardiovascular involvement [2]. However, cardiac involvement as an initial presentation is rare. We present a 31-year-old female with an initial presentation of pericardial tamponade who was subsequently diagnosed with SLE.

### Case Report

A 31-year-old female came with complaints of intermittent, low-grade fever for the past ten days with rapidly

progressive breathlessness for one month associated with orthopnea. She also had bilateral pedal edema without any chest pain, palpitations, decreased urine output, or wheezing. She noticed a non-pruritic, maculopapular rash beginning over her palms and soles. There was no history of hair loss or oral ulcers, headache, altered behavior, or involuntary movements. Family history was noncontributory.

She had a second-trimester pregnancy loss 3 weeks prior to presentation. She had high blood pressure and hypothyroidism during her pregnancy for which she was taking labetalol and levothyroxine supplementation.

On arrival, she was tachypneic, tachycardic, and febrile. Blood pressure was 118/88 mmHg, elevated jugular venous pressure (5 cm above sternal angle) with prominent v wave, and muffled heart sounds. Breath sounds were decreased in bilateral basal regions.

Laboratory tests are outlined in Table 1. ANA profile was done which was positive for anti-dsDNA, anti-nucleosomes, anti-histones, anti-SSA, and anti-Jo 1. Complement levels were low.

The electrocardiogram showed low voltage complexes and lateral lead T wave inversions (Figure 1A). Cardiac silhouette was enlarged on the roentgenogram. Echocardiography was evident for a swinging heart with paradoxical septal motion and large pericardial effusion (21 mm anteriorly and 29 mm posteriorly) with a normal ejection fraction of 55% (Figure 1B).

Table 1. Summary of laboratory parameters

Laboratory test	Value	Normal range
Hemoglobin	10 g/dl	12-15.5 g/dl
WBC	3450 cells/mm <sup>3</sup>	4000-10000 cells/mm <sup>3</sup>
Platelet count	1.98 lakh/mm <sup>3</sup>	1.5-4.5 lakh/mm <sup>3</sup>
ESR	110 mm/hour	0-10 mm/hr
Urea	37 mg/dl	12.6-42.6 mg/dl
Creatinine	1.32 mg/dl	0.7-1.2 mg/dl
Sodium/Potassium	136/4.8 mEq/L	136-145 mEq/L, 3.5-5.1 mEq/L
TSH	4.65 µIU/ml	0.27-4.2 µIU/ml
Urine analysis	2+ albumin, 4-6 RBCs	
Troponin T	0.024 ng/ml	0.0127-0.0249 ng/ml
CK-MB	0.81 ng/ml	0-4.94 ng/ml
Brain natriuretic peptide (BNP)	9594 pg/ml	0 – 125 pg/ml
ECG	Low voltage complexes, lateral lead T wave inversions	
Chest radiography	Enlarged cardiac silhouette with well-defined borders.	
Echocardiography	Swinging heart with paradoxical septal motion and a large pericardial effusion which was 21 mm anteriorly and 29 mm posteriorly (Fig 1). Her left ventricular systolic function was normal with an ejection fraction of 55% and there were no clots or vegetations.	
ANA profile	anti-dsDNA, anti-nucleosomes, anti-histones, anti-SSA, anti-Jo 1 positive	
Pericardial fluid: - Cell count - Protein - Glucose - Chloride - LDH - AFB - KOH - Culture & sensitivity - CBNAAT	Exudative 40 (mesothelial cells) 45 mg/dl 106 mg/dl 107.4 mEq/L 137 U/L Negative Negative No growth Negative	
C3	21.4 mg/dl	75-135 mg/dl
C4	5.8 mg/dl	9-36 mg/dl

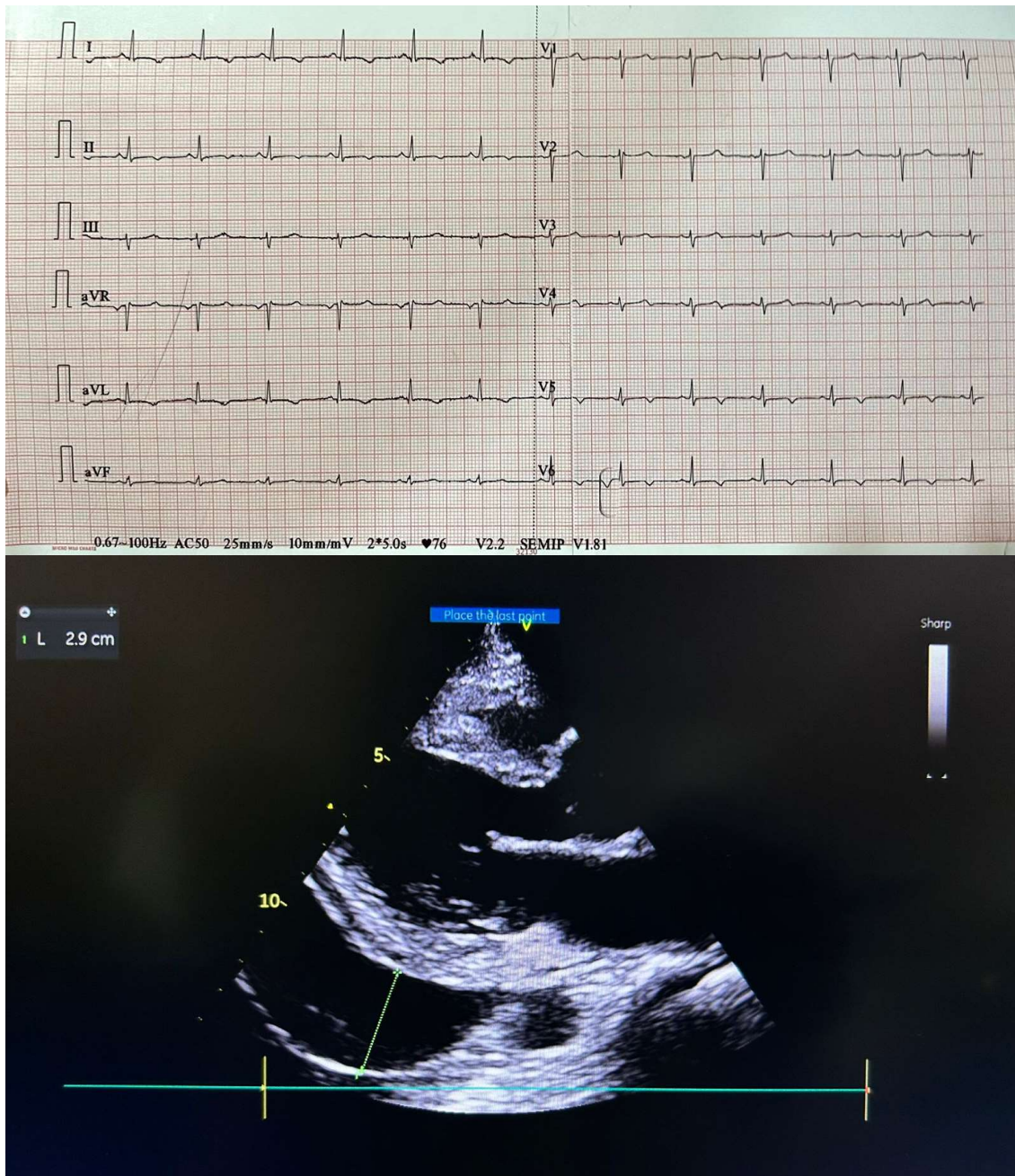


Figure 1A: Electrocardiogram showing low voltage complexes and T wave inversions in lateral leads; Figure 1B: Echocardiographic image of large pericardial effusion with impending cardiac tamponade.

Emergency pericardiocentesis was done and 800 ml of exudative fluid was drained followed by placement of a pigtail catheter.

Intravenous pulse methylprednisolone at a dose of 750 mg per day for 3 days was instituted following

which there was resolution of pericardial effusion. Her 24-hour urine protein was 0.945 gm/day hence renal biopsy was done which showed class IV lupus nephritis. She had a Systemic Lupus Erythematosus Disease Activity Index (SLEDAI) score of 18. The patient was given treatment

options of cyclophosphamide and mycophenolate mofetil (MMF) and she opted for MMF as she planned to start a family. She was discharged on mycophenolate mofetil, hydroxychloroquine, and steroids. She is on regular follow-up and is doing well.

### **Discussion**

Cardiovascular manifestations in SLE may affect the pericardium, myocardium, endocardium, heart valves, conduction system, or coronary vasculature [3]. Pericarditis is most common among these and often occurs in conjunction with serositis elsewhere. Occasionally, pericardial effusion is detected incidentally on routine echocardiography [4]. Cardiac tamponade is a rare manifestation of SLE and is seen during flares of lupus activity as was the case in our patient [5].

Cardiac tamponade is a life-threatening condition and prompt diagnosis is crucial to reduce mortality. Clinically, elevated jugular venous pressure, muffled heart sounds, hypotension, and tachycardia may be evident. Electrocardiogram may show low voltage complexes with electrical alternans, caused by swinging of the heart within the pericardial fluid. Chest radiography may reveal an enlarged cardiac silhouette once effusions are moderate in size. Echocardiography remains the standard method to establish the diagnosis and can reveal compression abnormalities of atria and ventricles throughout the cardiac cycle. Treatment necessitates emergency needle paracentesis under echocardiographic or fluoroscopic guidance. Open surgical drainage is reserved for cases where needle pericardiocentesis poses a challenge. Cardiac tamponade in SLE

generally requires high-dose steroids and immunomodulators such as hydroxychloroquine, mycophenolate mofetil, and azathioprine [6]. Non-steroidal anti-inflammatory drugs (NSAIDs) and colchicine help reduce serositis and size of the effusion and hence can be used in the absence of significant hemodynamic compromise [7]. Timely intervention can be lifesaving, as demonstrated in this case where urgent pericardiocentesis was performed when echocardiography revealed signs of impending cardiac tamponade.

Connective tissue disorders such as SLE, rheumatoid arthritis, and scleroderma are important differentials to be kept in mind when evaluating pericardial effusion. Other causes include tuberculosis, post-cardiac surgery, trauma, malignancy, bacterial infection, aortic dissection, post-myocardial infarction, radiation-induced, uremia, and thyroid disorders [8]. A retrospective study done by Goswami et al concluded that out of 409 SLE patients, pericarditis was seen in 25.4% and cardiac tamponade in 5.9% of patients of which, tamponade was the presenting feature in only half the above cases. They also found that the presence of pleuritis, anti-nucleosome antibody positivity, and larger size of pericardial effusion were strong predictors of the development of cardiac tamponade [9].

In conclusion, a point of interest in our case was the strikingly rare occurrence of cardiac tamponade as the initial manifestation of SLE. Clinical suspicion and timely intervention are crucial in such cases.

### **Author Contributions**

NS and MM drafted the article. DS, MM, and SR were involved in the concept and

design of the article. Every author is solely accountable for the accuracy and integrity of every part of this work.

#### **Declaration of Patient Consent**

Written informed consent was obtained from the patient for publication.

#### **Conflicts of interest**

The authors declares that they do not have conflict of interest.

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