International Journal of Research in Medical Sciences
Kothia D et al. Int J Res Med Sci. 2024 Nov;12(11):4330-4334
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20243397

Case Report

Systemic lupus erythromatosus presenting as cardiac tamponade: a rare and life-threatening presentation

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Received: 22 August 2024
Accepted: 14 October 2024

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ABSTRACT

We present a case of a 35-year-old female with 3-4 months history of left sided chest pain, low grade fever, dyspnea on exertion, dry cough, fatigue and myalgia. Initial investigations including pleural fluid CBNAAT was negative. Empiric anti- tuberculosis therapy was initiated based on clinical and radiological findings. Despite treatment, symptoms persisted. Subsequent evaluation including echocardiography which showed moderate pericardial effusion along with special investigations leading to a diagnosis of systemic lupus erythematosus. Treatment with corticosteroid resulted in significant symptom improvement. This case highlights the importance of considering systemic lupus erythematosus in patients with atypical presentations, even in absence of classic features.

Keywords: Cardiac tamponade, Case Report, SLE

INTRODUCTION

Cardiac tamponade is a life-threatening condition that occurs when fluid accumulates in the pericardial sac, leading to increased pressure on the heart and subsequent hemodynamic instability. This condition can result from a variety of causes, including malignancy, trauma, pericarditis, and autoimmune diseases such as systemic lupus erythematosus (SLE).[1] SLE is a chronic autoimmune disorder characterized by the production of autoantibodies and widespread inflammation that can affect multiple organ systems.

Among its many manifestations, cardiac involvement is relatively common and can present as pericarditis, myocarditis, endocarditis, or coronary artery disease. However, cardiac tamponade as a complication of SLE, though well-documented, is considered rare but potentially fatal.[2,3] This case report describes a patient with SLE who developed cardiac tamponade as a complication of her disease. It highlights the importance of early recognition and intervention in patients with SLE presenting with symptoms indicative of pericardial effusion, as delayed treatment can lead to severe outcomes. The case underscores the need for vigilance in monitoring cardiac involvement in patients with SLE, given the potentially rapid progression to life-threatening conditions like cardiac tamponade.

CASE REPORT

Presentation

A 35-year-old female presented with Generalized swelling, jaundice, 4 episodes of vomiting, bilateral wrist and metacarpophalangeal joint arthralgia, dyspnea on exertion for 6 days. A month ago, the patient visited a community health care center with complaints of left

sided chest pain, low grade fever, dyspnea on exertion, dry coughing, easy fatigability for 2 to 3 months. X-ray chest was suggestive left-sided pleural effusion. (Figure 1) Pleural fluid ADA and CBNAAT was negative, but based on the clinical and radiological findings the patient was empirically started on anti-tuberculosis treatment (AKT) with presumptive pleural kochs.

Patient also complained of one episode of generalized tonic- clonic seizure, with loss of consciousness for 30 minutes post convulsion, before 4 days. X-ray chest done during this time was suggestive of enlarged cardiac shadow with clinical correlation of pericardial effusion (Figure 2).

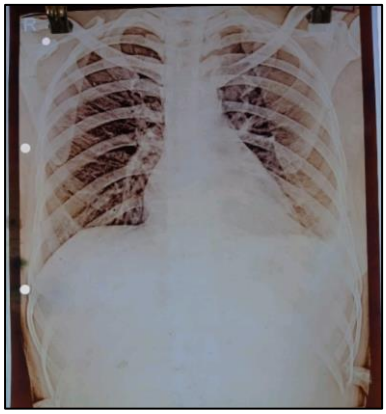

Figure 1: Left sided pleural effusion 1 month before presentation, empirical AKT was started.

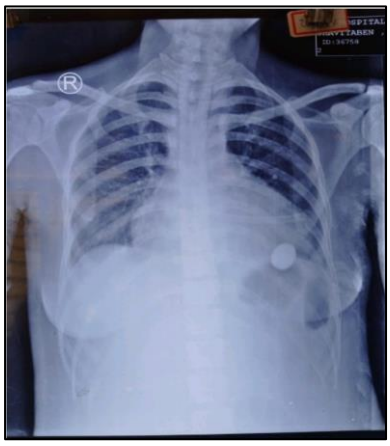

Figure 2: Enlarged cardiac silhouette, water bottle appearance.

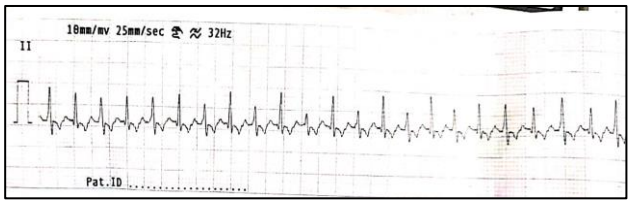

Figure 3: ECG on admission Day1, suggestive of electrical alternans.

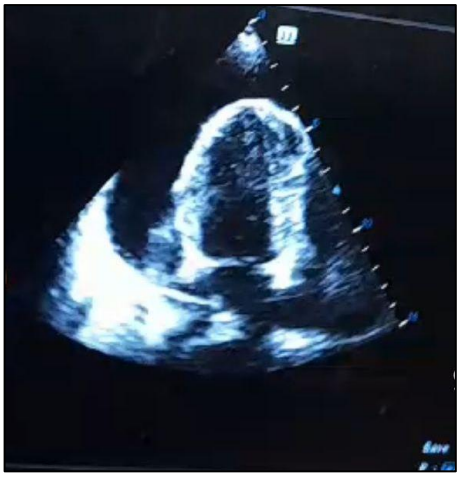

Figure 4: 2D Echo showing moderate pericardial effusion.

Investigation

The presentation of arthralgia and thrombocytopenia was thought to be due to the side-effects of AKT drugs and pericardial effusion was thought to be due to possible pericardial tuberculosis. The moderate pericardial effusion showed clear fluid and normal ADA and CBNAAT results.

RBC cast and proteinuria were present in urine. Along with these convulsions, pancytopenia, and arthralgia lead to suspicion of autoimmune cause. High levels of antinuclear antibodies and anti dsDNA revealed an underlying SLE.

Treatment and follow up

Urgent pericardiocentesis was done on presentation for cardiac tamponade, and 1000ml of pericardial fluid was drained, which led to immediate hemodynamic stability and continuous drain for next 48 hours. Extensive workup led to diagnosis of SLE causing serositis and pericardial effusion with possible SLE nephritis.

Patient was started on pulse dose steroid-intravenous Methylprednisolone 1gm/day for 3 days followed by tapering dose over next 2 months. Patient was started on high dose intravenous Cyclophasphamide 1 gm IV on day 3.

Patient was discharged on day 7 with 60 mg/day oral prednisolone once a day on tapering dose and referred to higher centre for renal biopsy which revealed Stage 3 Lupus Nephritis and, then cyclophosphamide was given every month for next 6 doses.[4]

At the six-month follow-up, the patient showed significant improvement, with no signs of pericardial effusion, a trace amount of urine albumin, and no arthralgia, indicating disease remission. The patient

tolerated the treatment well, with no notable side effects from either the steroids or cyclophosphamide. The maintenance therapy included a low-dose oral prednisolone (5 mg/day) and mycophenolate mofetil (1 gram twice daily) to manage ongoing SLE activity.

Table 1: Vitals.

Vitals	Day 1 admission	Day 1- 4-hour post pericardiocentesis	Day 3
Temperature (°C)	99.9	99.6	98.6
Pulse rate (beats/minute)	126, pulsus paradoxus	98, No pulsus paradoxus	132
Blood pressure (mmHg)	90/68 mmHg (expiration), 74/60 mmHg (inspiration)	104/76 mmHg	112/82 mmHg
Respiratory auscultation	No abnormalities		
Cardiac auscultation	Muffled and distant heart sound	Normal S1, S2	Normal S1, S2
Jugular venous pulse	9 cm above sternal angle (45° bed)	7cm above sternal angle	4 cm above sternal angle
Spo2	95% 2literl/minute nasal O2	97% with 2 liter/minute nasal O2	98% with room air
Sensorium	Conscious, oriented to time, place and person, drowsy	Conscious, oriented to time, place and person, drowsy	Fully conscious, oriented to time, place and person

Table 2: Investigations.

Investigation	Day 1	Day 2	Day 5
Haemoglobin (12.0- 15.0 g/dl)	8	7.8	10.6
White blood cell (4000-1000 cells/cumm)	5,500	7800	5,800
Platelet (150,000-450,000/cumm)	125,000	126000	260,000
Serum sodium (135-145 m/liter)	134.5		138.6
Serum potassium (3.5-5.0 meq/liter)	4.2		3.6
Serum calcium (8.5-10.5 mg/dl)	8.7		
Serum creatinine (0.7-1.3 mg/dl)	1.2	1.6	1.1
Blood urea (8-24 mg/dl)			20
Serum albumin (3.5- 5.5 gm/l)	2.9	2.4	3.2
SGPT (<40 IU/l)	177	137	76
ESR	120	122	
Bilirubin total		14.8 (AKT induced)	
Bilirubin direct		5.8	
Bilirubin indirect		2.4	
HIV/HBV/HCV Ag		Negative	
Serum trop-I			
Urine r/m			
Protein	+2	+2	
Pus cell	5-7/HPF		
RBC	25-30/HPF		
Cast	occasional RBC cast		
Pericardial tapping	30 ml clear fluid		
Protein	2.7 g/dl		
Glucose	g/dl		
Total count	560/cumm		
Neutrophil/lymphocyte	10%/90%		
Ada (u/liter)	26		
Fluid AFB stain & CBNAAT	MTB not detected		
ECG (Figure 3)	Electrical Alternans Sinus tachycardia Rate related St depression in lead II,	Normal sinus rhythm	

Continued.

Investigation	Day 1	Day 2	Day 5
	III, aVF		
2D Echo (Figure 4)	Moderate pericardial effusion (maximum inferior band of 18mm) Right atrium and ventricular collapse in diastole. Lv function. Normal IVC size 2.2 cm, with <30% collapse in inspiration	Minimal pericardial fluid rim. Normal LV and RV function. IVC 1.6 cm >50 % collapse with inspiration.	
ANA by Immunofluorescence (Normal <1:160 liter)		1: 320 Positive	
DsDNA (0-15 IU/ml)		29.3	
ANA profile by immunoblot		positive for nucleosome, dsDNA, histone, SSA/RO, m2 native	
Urine ACR (mg/g)		128 mg/g	
ABGA	Metabolic Acidosis PH:7.28 Bicarb: 15 meq/l PCO2: 38 mmhg Pao2: 68 mmhg Lactate: 4.2 mmol/l	Lactate: 1.8mmol/l	
Malaria smear/antigen Dengue Ns1/IgM	Negative		

IVC= Inferior vana cava

DISCUSSION

This case underscores the complexity of diagnosing SLE, particularly when it presents with atypical symptoms such as cardiac tamponade. In regions with a high prevalence of tuberculosis like India, patients presenting with pleural effusion might be prematurely treated for TB, as was initially done in this case.[5,6]

However, the worsening of symptoms and development of additional features like arthralgia, thrombocytopenia, and pericardial effusion should prompt clinicians to consider alternative diagnoses, including autoimmune diseases. The improvement seen with corticosteroid treatment in this patient highlights the effectiveness of early and aggressive immunosuppressive therapy in managing SLE-related complications.[7]

The case also emphasizes the importance of a thorough and systematic approach to diagnosis, particularly in patients who do not respond to initial treatments. Monitoring for cardiac involvement in SLE patients is crucial as it can rapidly progress to life-threatening conditions like cardiac tamponade.

This, case also illustrates the challenges of managing SLE in resource-limited settings where access to advanced diagnostic tools may be limited. It reinforces the need for clinicians to maintain a high index of suspicion for autoimmune conditions in patients with unexplained symptoms and to initiate prompt treatment to prevent severe complications.[8]

CONCLUSION

This case highlights the importance of considering SLE in the differential diagnosis of patients presenting with atypical symptoms, especially in regions where other diseases like tuberculosis are prevalent. Early recognition and appropriate treatment of SLE can prevent life-threatening complications such as cardiac tamponade.[9]

Long-term management with immunosuppressive therapy is essential to control disease activity, reduce the frequency of flare-ups, and prevent recurrence. Clinicians should remain vigilant for autoimmune causes in patients with persistent or unexplained symptoms, as early intervention is key to improving outcomes.[10]

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Kothia D, Nagar M, Nandania P, Chatterjee SS. Systemic lupus erythromatosus presenting as cardiac tamponade:a rare and life-threatening presentation. Int J Res Med Sci 2024;12:4330-4.