CASE REPORT

Systemic Lupus Erythematosus with Lupus Nephritis and Pericarditis in an Adolescent

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Background:

Systemic lupus erythematosus (SLE) in adolescents often presents more severely than in adults, with common complications including lupus nephritis (LN) and less frequently cardiac involvement such as pericarditis. Early recognition and treatment of these manifestations are critical, especially in resource limited settings where diagnostic and therapeutic delays can worsen the outcomes.

We report the case of a 16 year old Bangladeshi girl with previously diagnosed SLE who presented with highgrade fever, fatigue, generalised edema, and pleuritic chest pain, and was diagnosed with SLE complicated by severe LN and a large lupus pericardial effusion. One year earlier, she had manifested with fever, rash, arthritis, oral ulcers, and renal involvement with serological confirmation of the diagnosis and responded well to corticosteroids and immunosuppressive therapy. On current presentation, examination revealed hypertension with bilateral pitting edema and signs of pericardial effusion (raised jugular venous pressure, distant heart sounds, and a pericardial friction rub), alongside anaemia but no active skin rash or arthritis. Echocardiography demonstrated a large pericardial effusion (~28 mm) with early features of cardiac tamponade. Due to financial constraints and urgent clinical need, invasive procedures were deferred. The patient was managed for a lupus flare with high dose corticosteroids, mycophenolate mofetil, and hydroxychloroquine, along with supportive care for cardiorenal syndrome resulting in marked clinical improvement and complete resolution of the effusion without invasive intervention. She was discharged on maintenance immunosuppression after showing marked improvement for a week. However, socioeconomic factors have impeded regular followup; at last telephone contact, she remains clinically stable on treatment.

This case highlights the rare concurrent presentation of severe lupus nephritis and significant pericardial effusion in adolescent SLE and underscores the importance of early recognition, comprehensive multisystem evaluation, and timely immunosuppressive therapy, particularly in resource limited settings, to prevent life-threatening complications and improve patient outcomes.

Introduction:

Systemic lupus erythematosus is a chronic autoimmune disease characterized by autoantibody production and immune complex deposition affecting multiple organs. Approximately 10–20% of SLE cases begin in childhood or adolescence1. Pediatriconset SLE (pSLE) generally exhibits a more severe phenotype than adultonset disease, with more aggressive flares and faster cumulative organ damage [1,2]. These patients experience higher rates of major organ involvement, especially renal and hematologic, leading to greater longterm morbidity. Lupus nephritis (LN) is one of the most serious manifestations of pSLE, occurring in a large proportion of patients (~60–80% of children with

SLE)[2-4]. If not treated aggressively, LN can cause irreversible kidney damage and progression to end-stage renal disease (ESRD)[3,4]. Meanwhile, cardiac involvement in SLE, particularly pericarditis, is less common by comparison. Pericarditis reported in a significant subset of SLE patients (studies in adults and children quote ranges from ~12% up to 40%)[7-9], but symptomatic large effusions with cardiac tamponade are exceedingly rare (on the order of only a few percent of cases. Notably, recent studies indicate that children with lupus nephritis are at markedly increased risk of cardiac complications; for instance, the presence of nephritis in pSLE has been associated with a four-to-sevenfold higher likelihood of developing acute pericarditis or myocarditis[5].

In low income countries like Bangladesh, managing SLE poses additional challenges. Limited financial and medical resources often lead to delays in diagnosis, suboptimal monitoring, and interruptions in therapy[6]. Expensive diagnostic tests (such as kidney biopsy or advanced imaging) may be forgone due to cost, and patients can be lost to followup because of travel and treatment expenses. These factors contribute to worse outcomes in resourceconstrained settings. We present an adolescent SLE case from Bangladesh that exemplifies severe renal and cardiac involvement at disease onset, managed with constrained resources. This case highlights the importance of early recognition of multi organ lupus flares and the need to adapt standard care guidelines to a resource-limited context.

Case Presentation:

Ms. Aklima Akhtar" (pseudonym), a 16-year-old female from a rural area of Bangladesh, presented to our tertiary care hospital in March 2025 with a one month history of intermittent high grade fevers (peaking at 40 °C), progressive swelling of the face and legs, and a 3-day history of sharp pleuritic chest pain accompanied by shortness of breath. She had no known chronic illnesses prior to 2024. Notably, one year earlier (January 2024, at age 15), the patient had been admitted to our facility with a two-week history of fever, a malar rash, facial puffiness, bilateral lower limb edema, polyarthralgia with joint swelling, and painful oral ulcers. That initial episode in 2024 was treated empirically with corticosteroids and hydroxychloroquine, with an initial impression of nephrotic syndrome (given the edema and heavy proteinuria) versus an evolving connective tissue disease. She improved markedly on this therapy and was discharged, but a complete diagnostic workup was pending at that time. Apart from that episode, there was no prior medical or family history of note. The patient's socioeconomic background is poor, and her access to regular medical care has been inconsistent due to financial constraints.

On presentation, the patient was tachypneic (22 breaths/ minute), tachycardic (120 beats/min), febrile (38.2 °C), hypertensive (150/95 mmHg), and body mass index~17 kg/m². She was markedly pale with periorbital and pretibial edema (pitting +2). JVP was elevated. On precordial examination, heart sounds were distant with an audible pericardial friction rub; no gallop rhythm or murmurs were noted. (Pulsus paradoxus was difficult to assess due to tachycardia). Lung fields were clear on auscultation except for slightly reduced breath sounds at the left base. There was no active skin rash, no synovitis in the joints, no oral ulcers, and no lymphadenopathy at this time. In summary, the clinical picture on this admission suggested a recurrence of active SLE with significant pericardial effusion (signs of impending cardiac tamponade) accompanied by a relapse of nephrotic-range renal involvement.

Given her history and examination findings, a thorough series of investigations was reviewed and repeated to confirm the extent of disease activity. Table 1 summarizes the key laboratory and imaging results from the initial presentation (2024) and the current flare (2025).

Table 1: Key Laboratory and Imaging Investigation Results

Investigation	Result (Jan–Feb 2024)	Result (Mar 2025)	Reference Range
24-h urine protein	19.84 g/24 h	>12 g/24 h	<0.15 g/24 h
Urinalysis	RBC casts present	RBC casts present; urine protein 4+	–
Serum albumin	2.3 g/dL	2.1 g/dL	3.5–5.0 g/dL
ANA (Antinuclear Ab)	Positive, titer 1:640 (homogeneous pattern)	Positive, titer 1:1280 (homogeneous pattern)	<1:80 (negative)
ENA panel	Positive for Histone, SSA/Ro-60, and SSA/Ro-52; all others negative	(Not repeated in 2025 flare)	–
Anti-dsDNA	Negative (Crithidia assay, Feb 2024)	Strongly positive, titer 1:160 (Crithidia assay)	<1:10 (negative)
Complement C3	Low (below normal range)	Low	90–180 mg/dL
Complement C4	Low (below normal range)	Low	10–40 mg/dL
Complete blood count	Hemoglobin 10.8 g/dL; WBC 4.2×10^9/L; Platelets 280×10^9/L (Feb 2024)	Hemoglobin 9.1 g/dL; WBC 3.5×10^9/L (leukopenia with lymphopenia); Platelets 310×10^9/L	Hb: 12–16 g/dL; WBC: 4–11×10^9/L
ESR (Erythrocyte sedimentation rate)	48 mm/1st hour	65 mm/1st hour	<20 mm/1st hour
CRP (C-reactive protein)	30 mg/L	48 mg/L	<5 mg/L
Serum creatinine	0.8 mg/dL	1.3 mg/dL	0.5–1.1 mg/dL
Blood urea	28 mg/dL	46 mg/dL	10–50 mg/dL
Chest imaging	HRCT (4 Feb 2024): bilateral interlobular septal thickening and ground-glass opacities in lungs; mild right pleural effusion; moderate pericardial effusion with enlarged cardiac silhouette.	Chest X-ray (Mar 2025): enlarged cardiac silhouette (suggestive of pericardial effusion); clear lung fields.	–
Abdominal ultrasound	(1 Feb 2024): Hepatomegaly (liver span 15.6 cm) with homogeneous texture; gallbladder wall edema with ~8 mm pericholecystic fluid; no biliary dilatation; normal spleen and pancreas; minimal free fluid in pelvis.	(Mar 2025): Mild hepatomegaly; no ascites; bilateral minimal pleural effusions at bases.	–

Investigation	Result (Jan–Feb 2024)	Result (Mar 2025)	Reference Range
Echocardiography	(Not done in Jan 2024) – pericardial effusion was noted on HRCT chest.	(5 Mar 2025): Large circumferential pericardial effusion (~25–28 mm in diastole) with early signs of tamponade (right atrial and RV diastolic collapse); moderate tricuspid regurgitation; mild pulmonary hypertension (estimated PASP ~40 mmHg); preserved LV systolic function (EF 68%). No valvular vegetations or wall motion abnormalities.	–

(Key: ANA = antinuclear antibody; ENA = extractable nuclear antigen; RBC = red blood cell; HRCT = high-resolution computed tomography; PASP = pulmonary artery systolic pressure; LV = left ventricle; EF = ejection fraction.)

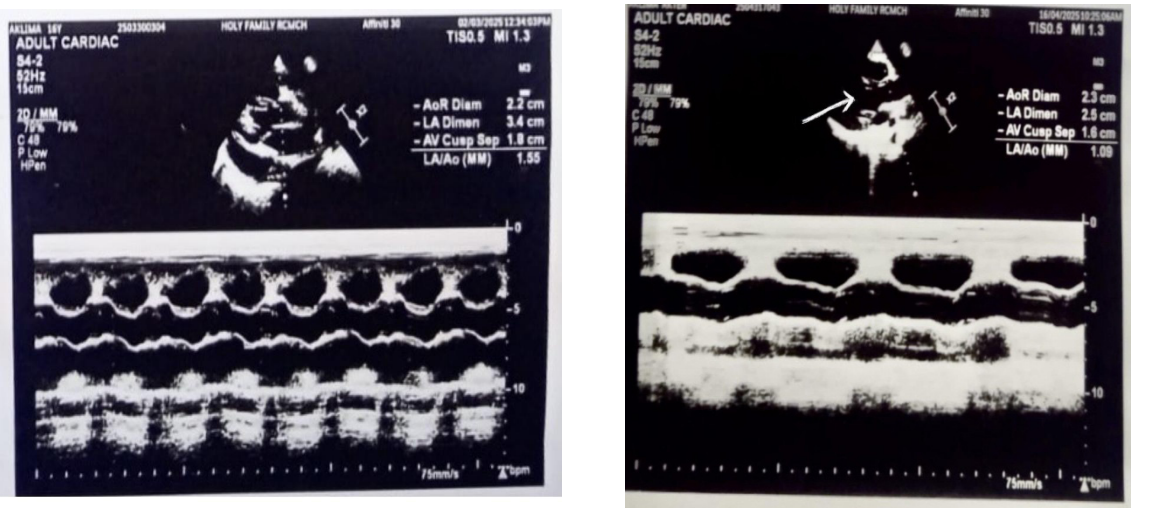

Figure 1a and 1b showing Pericardial Effusion on Echocardiogram, 2D view.

These investigations confirmed high disease activity consistent with an SLE flare involving both renal and cardiac systems. Notably, the 24-hour urine protein excretion was in the nephrotic range during both episodes, indicating severe lupus nephritis. The autoimmune serologies were strongly positive supporting active SLE. The echocardiogram in 2025 demonstrated a large pericardial effusion with signs of impending cardiac tamponade. Given the impending tamponade physiology, our team considered immediate pericardiocentesis; however, this invasive procedure was not readily available at our facility without significant upfront payment for surgical supplies, which the patient's family could not afford. We therefore elected to manage the effusion conservatively with medical therapy and close monitoring in an intensive care setting.

A diagnosis of an SLE flare was established, with Class III/IV lupus nephritis (presumed clinically, without biopsy confirmation) and lupus pericarditis causing a large effusion. The patient was started on high-dose immunosuppressive therapy. This included pulse intravenous methylprednisolone (500 mg daily for 3 days), followed by high-dose oral prednisolone (1 mg/kg/day). Mycophenolate

mofetil (MMF) was initiated at 1 g twice daily as induction therapy for lupus nephritis (in place of cyclophosphamide, given the patient's young age and the desire to avoid gonadotoxic effects, as well as limited resources for cyclophosphamide monitoring). Hydroxychloroquine 300 mg daily was continued for its disease-modulating benefits. Supportive care measures were provided: an ACE inhibitor (enalapril) was started for blood pressure control and renal protection, and diuretics (furosemide 20–40 mg as needed) were used to manage fluid overload and help alleviate the pericardial effusion. The patient was closely observed in a highdependency unit with serial clinical examinations and point-of-care ultrasound to monitor the effusion.

Over the course of one week, the patient's condition improved significantly. Her fever subsided, the chest pain resolved, and the pericardial friction rub disappeared. A repeat echocardiogram after one week of therapy showed that the pericardial effusion had decreased from "large" to mild (≤10 mm) with no signs of tamponade. Her urine output increased and the edema regressed; follow-up urinalysis showed a reduction in proteinuria. Given these improvements and the family's financial constraints, pericardiocentesis was ultimately not performed at all – fortunately, the effusion responded to anti-inflammatory and immunosuppressive treatment alone.

The patient was discharged after 10 days of hospitalization on a tapering course of oral prednisolone (initially 60 mg/day, to be gradually reduced), along with maintenance doses of MMF (1 g twice daily), hydroxychloroquine, and other supportive medications (calcium and vitamin D supplementation, and a proton pump inhibitor for gastroprotection). We emphasized the importance of regular followup visits to adjust therapy and monitor for any recurrence. However, the patient's family has struggled with follow-up due to cost and travel distance. At the last telephone contact (approximately 3 months postdischarge), the patient reported adherence to medications and no recurrence of major symptoms (no fever, chest pain, or significant swelling). We are coordinating with local healthcare workers to help the family access ongoing care and to find ways to subsidize her treatment.

Discussion:

This case documents an unusual concurrent presentation of severe lupus nephritis and a significant pericardial effusion with tamponade physiology at the initial onset of SLE in an adolescent. Such a combination of critical organ manifestations is rarely reported, especially in resource-limited settings where milder lupus flares might be under-diagnosed until multiple organs are involved. Several important learning points emerge from this case:

High frequency of renal involvement in pSLE: Renal disease is one of the most common severe manifestations in pediatric SLE, reported in about 60–80% of cases[2-4]. Renal involvement not only often dictates the initial presentation (e.g. nephrotic syndrome, as seen in our patient) but also is the strongest predictor of long-term morbidity and mortality in lupus3. Uncontrolled lupus nephritis can rapidly lead to chronic kidney disease and eventually ESRD, underscoring the need for prompt, aggressive therapy to induce remission [3,4].

Cardiac involvement is less frequent but can be life-threatening: Among cardiac manifestations of SLE, pericarditis is the most prevalent, occurring in a significant subset of patients (estimates ranging roughly from 12% to 44% across studies). However, symptomatic pericardial effusions with cardiac tamponade are exceedingly rare, especially as an initial presentation of SLE[7-9]. In our patient, the presence of a large effusion with early tamponade signs signified a severe disease flare. The literature indicates that only a small percentage (~1–2%) of pediatric SLE cases have cardiac tamponade as an initial manifestation, with few pediatric cases reported in detail[8,9]. This rarity makes it a diagnostic and management challenge – clinicians must distinguish a lupus-related effusion

from other causes (e.g. tuberculosis pericarditis in endemic regions, malignancy, etc.) and intervene urgently to prevent hemodynamic collapse.

Nephritis portends a higher risk of cardiac complications: Systemic inflammation in SLE can simultaneously affect multiple organ systems. Notably, pediatric SLE patients with lupus nephritis are significantly more likely to develop cardiac manifestations compared to those without nephritis. For instance, the presence of active nephritis has been associated with approximately a four- to seven-fold increased risk of developing acute pericarditis or myocarditis[5]. Our case reflects this association – the patient's kidney flare and large pericardial effusion occurred together, suggesting a state of very high disease activity. The pathophysiology behind this may involve a more aggressive autoimmune process in patients with renal involvement, or overlapping immune complex deposition and inflammation affecting both glomerular and pericardial tissues.

The absence of a renal biopsy in this case does limit our ability to precisely classify the lupus nephritis (e.g. distinguishing between Class III vs Class IV, etc.) and to tailor the therapy accordingly. Ideally, histological confirmation via kidney biopsy guides treatment – for example, differentiating a proliferative LN (which mandates aggressive immunosuppression) from a purely membranous LN or other pathologies[10,11]. In our resource-limited context, we made a clinical diagnosis of lupus nephritis based on the combination of heavy proteinuria, active urine sediment (RBC casts), hypoalbuminemia, and positive lupus serologies, and we proceeded with standard therapy for a presumed Class III/IV LN without waiting for a biopsy. Despite the lack of histology, the patient's dramatic response to treatment supports the inference that her nephropathy was indeed lupus-related and proliferative in nature. Similarly, we managed the pericardial effusion without invasive drainage; while guidelines indicate that cardiac tamponade is an absolute indication for prompt pericardial drainage[7], we had to weigh this recommendation against real-world feasibility in our setting. Remarkably, the effusion in our patient resolved under medical therapy alone – likely because it was primarily due to active inflammation (lupus pericarditis) and responded to immunosuppression, rather than being due to an infection or mechanical cause that would necessitate drainage.

Our management was aligned as closely as possible with established SLE treatment guidelines, with necessary adaptations for the local setting. Current international guidelines for lupus nephritis recommend using high-dose corticosteroids together with either cyclophosphamide or mycophenolate mofetil as first-line induction therapy for severe LN[10,11]. In this patient, we chose MMF over cyclophosphamide for induction, given the considerations of age (to preserve ovarian function) and resource limitations. We also ensured the patient was on hydroxychloroquine, which is recommended for virtually all SLE patients as baseline therapy to reduce flare frequency and improve long-term outcomes[6]. Furthermore, the American College of Cardiology/American Heart Association (ACC/AHA) guidelines on pericardial disease emphasize that in hemodynamically significant effusions or tamponade, drainage is indicated[7] – a principle we acknowledge, even though we managed non-surgically due to the unique constraints. Throughout her care, we focused on the most critical treatments (corticosteroids, immunosuppressants, and supportive care) and used careful clinical monitoring in lieu of some advanced diagnostics, demonstrating that core lupus management principles can still be applied effectively in a constrained-resource environment.

In conclusion, this case illustrates that adolescent onset SLE can present with simultaneous severe organ manifestations, and that early diagnosis coupled with aggressive immunosuppressive therapy can be lifesaving. Clinicians should maintain a high

suspicion for multisystem involvement when a young patient presents with fever, edema, and chest pain, as these can herald lupus activity in various organs (renal, cardiac etc.). Our patient's favorable response to treatment highlights the importance of prompt recognition and intervention. However, this case also underscores the ongoing challenges of longterm management of chronic autoimmune diseases in developing countries. Socioeconomic hardships can impede regular followup and adherence, which are crucial for maintaining remission in SLE. Healthcare providers must therefore devise innovative strategies to ensure continuity of care, such as social support services, community health worker engagement, telemedicine follow ups, and patient educationto bridge gaps in the healthcare system. Ultimately, adapting standard treatment protocols to the local resource context while adhering to core principles of SLE management is essential for improving outcomes in patients with lupus in resource-poor settings.

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