Case Report

Ghost on the Valve: Lupus Valvulopathy Without Libman-Sacks Endocarditis

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Systemic lupus erythematosus (SLE) is a relapsing, remitting autoimmune disease characterized by heterogeneous multisystem involvement. It most commonly presents with constitutional symptoms, nonerosive symmetric polyarthritis, mucocutaneous manifestations, and variable renal and hematological abnormalities. Each presentation carries a different diagnostic weight and is clinically important. We report a case of a young female with inflammatory polyarthritis, significant non-vegetative mitral regurgitation suggestive of lupus valvulopathy, and recurrent pregnancy loss due to secondary antiphospholipid syndrome. Further investigations and systemic evaluations confirmed a diagnosis of SLE.

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Introduction

Systemic lupus erythematosus is a multisystem autoimmune condition characterised by sustained immune system activation, resulting in the formation of autoantibodies and protein products that cause immune complex-mediated damage to tissues and organs.[1,2]

Hippocrates is credited with the earliest observation of skin ulcers linked to systemic lupus erythematosus (SLE). Later, in 1833, Laurent-Théodore Biett gave the first formal clinical description of the condition, referring to it as erythema centrifugum.[3]

WHO mortality database shows the international ASMR for SLE105 was 2.7 deaths per million inhabitants: 4.5 deaths per million inhabitants for women and 0.8 deaths per million inhabitants for men.[4] Mortality rates were strikingly heterogeneous between countries, with a several hundred-fold variation between countries, from 0.1 deaths per million inhabitants in Morocco to 27.1 deaths per million inhabitants in Saint Lucia. In 2014, the highest overall ASMR was in Latin America, and the lowest was in Europe.[4]

In India, the prevalence has been reported as 3.2 per 100,000 individuals.[5] Survival data indicate that nearly 85% of patients live at least 10 years after diagnosis, while about 75% survive for 20 years. Mortality rates are higher among younger patients, with SLE-related deaths occurring in 78.9% of individuals under 20 years of age compared to 18.7% among those aged 70 to 79 years.[5]

Here, we discuss a case of systemic lupus erythematosus presenting with classical, textbook-like multisystem involvement. The patient, referred from a peripheral health centre to Rajiv Gandhi Government General Hospital for evaluation of polyarticular pain, was found to have inflammatory polyarthritis, lupus nephritis, non-vegetative mitral regurgitation, and recurrent pregnancy loss due to secondary antiphospholipid syndrome, making it a clinically comprehensive and diagnostically relevant presentation.

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Case Presentation

A 25-year-old female presented with joint swelling and fever for 2 days, along with breathlessness for 1-month. The patient had been apparently asymptomatic until 2 years prior, after which she experienced two recurrent abortions, the first in the third trimester at 7 months and the second in the first trimester at 3 months. Subsequently, she developed bilateral knee pain and involvement of multiple small joints, more pronounced in the early morning. These symptoms were associated with fever, chills and rigours, lower backache, breathlessness on exertion, and increased frequency of urination. She also noticed bilateral lower-limb swelling over the preceding 10 days. There was no history of decreased urine output, facial puffiness, jaundice, photosensitivity, oral ulcers, or other comorbid illnesses.

The patient had received a blood transfusion 3 months prior. The exact indication for transfusion could not be established from the available records; however, severe anaemia secondary to gynaecological causes was considered the most likely indication. There was no significant family history.

On examination, the patient was tachycardic with a heart rate of 110 beats/minute and blood pressure of 140/80 mmHg. She had mMRC grade III dyspnea with bilateral pitting pedal oedema. Cardiovascular examination revealed a high-volume pulse with wide pulse pressure and a pansystolic murmur best heard over the mitral area, radiating to the axilla. Respiratory examination showed decreased air entry in the bilateral basal regions. Inflammatory polyarthritis involving both small and large joints was present. Abdominal examination was unremarkable, and no malar or discoid rash was noted.

Ultrasonography of the abdomen revealed bilateral grade I renal parenchymal disease and an isoechoic thrombus measuring 1.7 × 0.6 cm in the hepatic segment of the inferior vena cava. Electrocardiography demonstrated atrial fibrillation with an irregularly irregular rhythm and a ventricular rate of approximately 110 beats/minute. P waves were absent, the cardiac axis was normal, QRS complexes were of normal duration, and no significant ST-T changes were observed.

Transthoracic echocardiography demonstrated thickening of both anterior and posterior mitral valve leaflets, restricted mobility of the posterior mitral leaflet, and doming of the anterior mitral leaflet. No rheumatic morphological features or valvular vegetations were identified. Severe mitral regurgitation with a prominent regurgitant jet was noted. The left atrium was dilated, measuring 48 × 53 mm, while left ventricular dimensions measured 60 mm in diastole and 36 mm in systole. Mitral

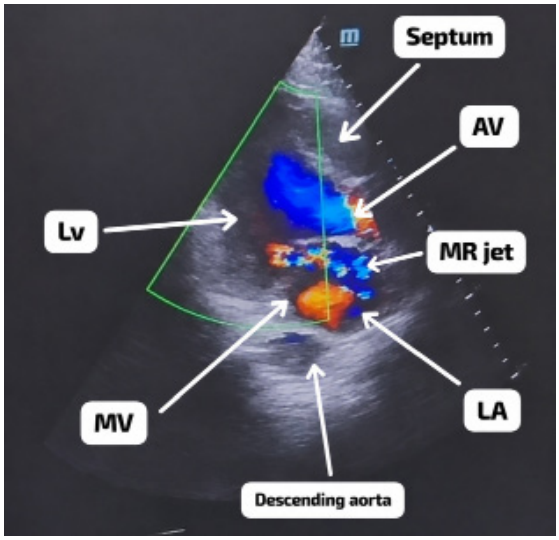

Figure 1: Color Doppler echocardiographic image demonstrating severe mitral regurgitation jet across the mitral valve in the absence of valvular vegetations.

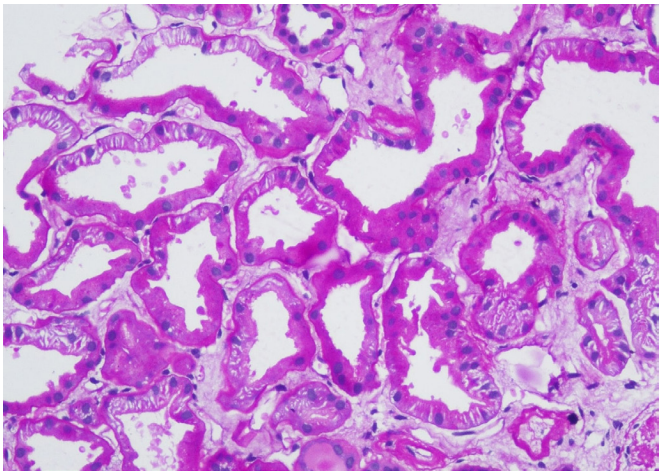

Figure 2: Light microscopy showing renal tubular changes in lupus nephritis

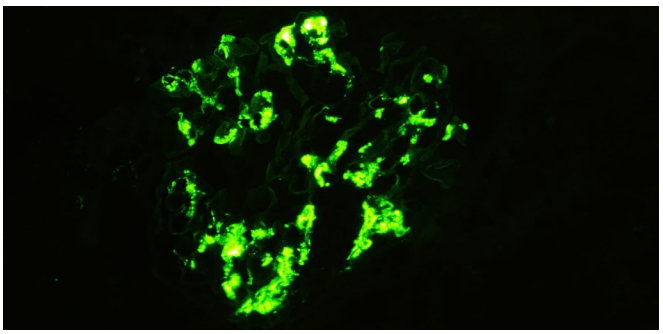

Figure 3: Immunofluorescence showing granular immune complex deposition in glomeruli

Table 1: Echocardiographic parameters of mitral valve function

Parameter	*Value*	*Reference range*
Mitral valve thickness	14 mm	<5 mm
Mitral valve area*	3.1 cm^2	4–6 cm^2
Peak transmitral gradient	10 mmHg	<5 mmHg
Mean transmitral gradient	6 mmHg	<2 mmHg
Mitral valve maximum velocity	1.64 m/s	<1.3 m/s
Left atrial dimension	48 × 53 mm	<40 mm
Pulmonary artery systolic pressure (PASP)	42 mmHg	<35 mmHg

Nephrology investigations suggested IgA nephropathy. Renal biopsy confirmed grade II lupus nephritis.

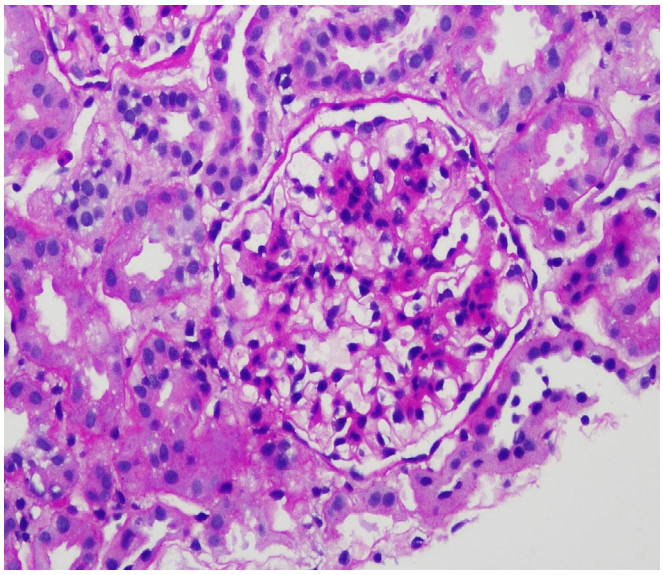

Figure 4: Light microscopy showing mesangial proliferation in glomerulus consistent with class II lupus nephritis

Table 2: Basic Laboratory Investigations were as Follows

Parameters	*Value*	*Reference Range*
Hemoglobin	6.2 g/dL	12–16 g/dL
Hematocrit	20.2%	36–46%
WBC	4900/μL	4000–11000 μL
Platelets	2.5 lakh/μL	1.5–4 Lakh/ μL
INR *	1.03	0.8–1.2
Serum Urea	74 mg/dL	15–40 mg/dL
Creatine	2.3 mg/dL	0.6–1.2 mg/dL
SGOT †	25 U/L	5 U/L - 40 U/L
SGPT ‡	16 U/L	7 U/L – 40 U/L

(* INR = International Normalized Ratio, † SGOT = Serum Glutamic Oxaloacetic Transaminase, ‡ SGPT = Serum Glutamic Pyruvic Transaminase)
Urinalysis showed :
Albumin +1, Trace of Blood in urine, suggesting Acute Kidney Injury

Table 3: Biomarker investigations showed positivity for

Anti DS DNA	β2 Glycoprotein 1 immunoglobulin G	Anti-Cardiolipin Antibody immunoglobulin G	Anti-cardiolipin Antibody Iimmunoglobulin M

valve area assessed by two-dimensional planimetry was 3.1 cm^2, with a peak transmitral gradient of 10 mmHg and a mean gradient of 6 mmHg. Pulmonary artery systolic pressure was estimated at 42 mmHg. Left ventricular systolic function was preserved with an ejection fraction of 60%. No tricuspid regurgitation, right ventricular dilatation, pericardial effusion, left atrial appendage clot, or atrial septal defect was observed. Representative colour Doppler echocardiographic imaging demonstrating severe mitral regurgitation is shown in Figure 1.

Table 4: Tabular comparison of the literature specific to the present case

Characteristics	*Present case*	*Case 1*	*Case 2*
Age	25 years old female	35 years old male	33 years old female
Chief complaints	Joint swelling, fever, and breathlessness	Palpitations, dizziness, and shortness of breath. Repeated readmissions	Progressive shortness of breath, chest pain on exertion, bilateral pitting oedema, and ascites
Cardiac manifestations	Non-vegetative lupus valvulopathy, mitral regurgitation, pansystolic murmur in the mitral area radiating to the axilla, high-volume pulse, and wide pulse pressure	Extensive myocardiolysis and interstitial fibrosis, biventricular scarring, and impairment.	Aortic stenosis, mixed mitral valve disease, and severe tricuspid regurgitation. TEE* confirmed vegetation on all mechanical valves
Management	hydroxychloroquine and mycophenolate mofetil with supportive therapy, iron supplementation, proton pump inhibitors, and antihypertensive agents	6 cycles of outpatient cyclophosphamide before being put on maintenance Azathioprine 100 m/day.	Triple mechanical prosthetic valve replacement surgery, but the patient underwent a massive stroke and subsequently died
Reference	-	[13]	[14]

(* TEE – Transesophageal echocardiography)

Table 5: Biomarkers for SLE in the defined criteria of ACR-1997 *, SLICC-2012 †, and EULAR/ACR-2019 ‡.[18]

Proteinuria	*Persistent proteinuria > 0.5 g/24 h or >3+, if quantitation not performed*	*Proteinuria > 0.5 g/24 h by 24-h urine or equivalent spot urine protein to creatinine ratio*
Urinary casts	Cellular casts may be red cell, haemoglobin, granular, tubular, or mixed.	—
Hemolytic anemia	Hemolytic anaemia with reticulocytosis	Evidence of hemolysis, such as reticulocytosis, low haptoglobin, elevated indirect bilirubin, elevated Lactate Dehydrogenase, and positive Coombs' (direct antiglobulin) test
White blood cell count	White blood cell count < 4000/mm 3 on 2 or more occasions; OR Lymphocyte count < 1500/mm3on 2 or more occasions	White blood cell count < 4000/mm^3
Antinuclear antibody levels	An abnormal titer of antinuclear antibody by immunofluorescence or an equivalent assay at any point in time and in the absence of drugs known to be associated with "drug-induced lupus" syndrome	ANA § at a titer of ≥1:80 on HEp-2 cells or an equivalent positive test at least once; testing by immunofluorescence on HEp-2 cells or a solid-phase ANA screening immunoassay with at least equivalent performance is highly recommended
Complement 3	Complement 3	Low complement 3
Complement 4	Complement 4	Low complement 4
Antiphospholipid antibody	Antiphospholipid antibody positivity	Anticardiolipin antibodies (IgA ‖, IgG ¶, or IgM **) at medium or high titer (>40 APL, GPL, or MPL, or >the 99th percentile) or positive anti-β2GPI antibodies †† (IgA, IgG, or IgM) or positive lupus anticoagulant

(* ACR: American College of Rheumatology; † SLICC: Systemic Lupus International Collaborating Clinics; ‡ EULAR: the European Alliance of Associations for Rheumatology. § ANA: antinuclear antibody; ‖ IgA: immunoglobulin A; ¶ IgG: immunoglobulin G; ** IgM: immunoglobulin M; †† anti-β2GPI: anti-β2-glycoprotein I;)

The patient was confirmed to be a case of Low C3, C4 Anti-DS DNA positive systemic lupus erythematosus with non-vegetative valvulopathy and grade II nephritis, presenting with secondary APLA positivity.

The patient is currently being managed with hydroxychloroquine and mycophenolate mofetil for systemic lupus erythematosus with renal involvement, along with supportive therapy including iron supplementation, proton pump inhibitors, and antihypertensive agents. She is under regular follow-up with serial monitoring of renal parameters, complement levels, and hematological indices.

Discussion

Systemic lupus erythematosus is a chronic, multisystem autoimmune disorder characterized by the production of autoantibodies and immune complex deposition, leading to inflammation and damage across multiple organ systems. It predominantly affects women of reproductive age and follows a relapsing, remitting course.[1,2] The pathogenesis of SLE involves loss of immune tolerance, formation of autoantibodies, particularly against nuclear antigens, and deposition of immune complexes in tissues.[6] This results in complement activation, inflammation, and tissue injury, often reflected by decreased complement levels (C3, C4) during active disease.[6]

Musculoskeletal Features

Musculoskeletal involvement is one of the most common manifestations of SLE,[7] occurring in the majority of patients. It typically presents as a non-erosive, symmetrical inflammatory polyarthritis involving small joints, although large joints may also be affected.[6]

Renal Involvement

Renal involvement, most commonly presenting as lupus nephritis, is a frequent and clinically important feature of SLE, affecting a significant proportion of patients.[7] The disease exhibits a broad histopathological spectrum, ranging from minimal mesangial involvement (Class I–II) to more aggressive proliferative forms of glomerulonephritis.[6] Early stages, particularly Class II lupus nephritis, may manifest with relatively mild findings such as low-grade proteinuria and microscopic hematuria, yet still reflect underlying systemic disease activity. Histological classification plays a key role in prognostication, even when clinical renal manifestations are subtle or absent.[8] Importantly, no single modality, whether light microscopy, electron microscopy, or immunofluorescence, provides a complete assessment in isolation, making classification essential. Lower classes, including Class IIA and IIB, are generally associated with a more favorable prognosis, and excessive

immunosuppressive therapy should be avoided, given the increased risk of infections.[8]

Cardiovascular Involvement

Cardiac valvular involvement in systemic lupus erythematosus[7] is well recognized and is not limited to overt vegetations. Echocardiographic series have shown that valvular abnormalities are common in SLE, with lesions detected in 61% of patients at baseline and 53% on follow-up, and transesophageal echocardiography being the most sensitive method for identifying them.[9] Classic clinicopathologic work also described severe mitral regurgitation in SLE as the consequence of "healing" of Libman–Sacks lesions through scarring and calcification, supporting the concept that significant mitral regurgitation may arise from chronic valvular damage rather than obvious vegetations alone.[10] In addition, valvular disease has been linked to antiphospholipid antibodies, suggesting an immune-mediated contribution to lupus valvulopathy.[11] Taken together, the literature supports that SLE can present with non-vegetative mitral regurgitation as part of the broader valvular spectrum of the disease, making echocardiographic evaluation important even when classical vegetations are not seen. Studies show that valvular regurgitation was more frequent and mitral or aortic regurgitation was more common in patients with active SLE, but was not related to the duration of SLE.[12]

Mucocutaneous Involvement

Mucocutaneous manifestations are among the most common clinical features of SLE, occurring in a majority of patients and often serving as early diagnostic clues. These include malar rash, photosensitivity, oral and nasal ulcers, and alopecia.[6,15] However, their absence does not exclude the diagnosis.[7] In the present case, notably, there were no mucocutaneous manifestations, which may have contributed to the initial diagnostic challenge, as the patient lacked the more classical and recognizable cutaneous features of SLE.

Antiphospholipid Syndrome & Obstetric Manifestations

A subset of patients with systemic lupus erythematosus develops antiphospholipid antibodies, leading to secondary antiphospholipid syndrome (APS), a condition marked by a prothrombotic tendency.[6,7] This state increases the risk of both arterial and venous thrombotic events, in addition to adverse pregnancy outcomes such as recurrent fetal loss. The diagnosis is supported by the detection of antibodies, including anti–β2 glycoprotein I (IgG) and anticardiolipin antibodies (IgG and IgM), which are closely linked to both thrombotic complications and obstetric morbidity. Evidence indicates that women with SLE experience higher rates of pregnancy-related complications compared to the general population, including miscarriage, preeclampsia, preterm birth, and low birth weight infants.[16] Among these, preterm delivery before 37 weeks is particularly common, occurring in approximately 30 to 40% of cases, while preeclampsia has been reported in about 9 to 23% of SLE pregnancies.[16,17]

Immunological Markers and Biomarkers

Immunological abnormalities are central to the diagnosis and assessment of disease activity in SLE.[7] The presence of antinuclear antibodies and anti–double-stranded DNA antibodies, along with hypocomplementemia (reduced C3 and C4 levels), reflects ongoing immune complex–mediated inflammation.[6] Low complement levels, in particular, are associated with active disease and are commonly seen in patients with renal involvement. About 40% of patients with SLE have anti-Ro/SSA and/or anti-La/SS-B autoantibodies, which can cross the placenta and cause neonatal lupus (NL).[16,17]

Future Directions

Future efforts should aim to establish well-defined, consensus-driven guidelines across key domains. One priority is to develop a structured strategy for cardiovascular surveillance in patients with SLE, outlining when to initiate monitoring, how frequently to continue it, and the role of baseline tools such as ECG and echocardiography, along with criteria for escalation to advanced imaging when initial assessments are inconclusive. Another important area is the incorporation of biomarker evaluation into routine care to enable earlier detection of cardiovascular risk; relevant markers may include troponins, inflammatory indices such as CRP and ESR, autoantibody profiles, and other emerging indicators. The creation of validated risk stratification models could further assist in identifying patients who would derive the greatest benefit from early intervention. In addition, longitudinal cohort studies and registry-based data are needed to better understand the long-term clinical impact of imaging modalities and biomarker-guided strategies. Novel therapeutic approaches, particularly CAR-T cell therapy, also hold significant promise in SLE management, and future research should

focus on improving target specificity, ensuring safety, and confirming sustained efficacy through large-scale clinical trials.[18,19]

Conclusion

Systemic lupus erythematosus typically manifests in females of reproductive age with multisystem involvement. Cardiac manifestations most commonly include Libman–Sacks endocarditis affecting the mitral valve. Non-vegetative mitral regurgitation represents an under-recognised form of lupus valvulopathy. Antiphospholipid antibodies, particularly anti-β2 glycoprotein I IgG and anticardiolipin antibodies (IgG and IgM), are associated with valvular involvement and recurrent pregnancy loss, while low C3 and C4 indicate active disease.

In this patient, non-vegetative mitral regurgitation with inflammatory polyarthritis, class II lupus nephritis, recurrent pregnancy loss due to secondary antiphospholipid syndrome, and hypocomplementemia led to a complex presentation. The diagnosis of SLE with secondary antiphospholipid syndrome was established after evaluation. The patient is currently on hydroxychloroquine and mycophenolate mofetil and is under regular follow-up, showing stabilization of disease activity.

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