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Case report

A Challenging Course of Systemic Lupus Erythematosus with Valvular Heart Disease and Multisystem Involvement

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Abstract
We present a complex case of Systemic Lupus Erythematosus (SLE) with secondary antiphospholipid syndrome (APS). Diagnosed initially with discoid lupus (2019) and progressed to severe systemic flare (Nephritis, hypocomplementemia, arthritis) by 2021, resulting in hypertension and cerebrovascular events. The clinical course was dominated by widespread encephalomalacia and new-onset epilepsy. Alongside progressive congenital aortic valve stenosis. Despite concern over non-sustained ventricular tachycardia (NSVT), it was successfully suppressed by beta-blockers. However, the patient developed overt heart failure in early 2024, due to advanced left ventricular hypertrophy (graded 54mmHg). Ultimately, aortic valve replacement (AVR) was performed. This case highlights the compounded thrombotic and inflammatory risk in SLE/APS and the importance of coordinated multidisciplinary management.
Keywords. Systemic Lupus Erythematosus, Antiphospholipid Syndrome, Aortic Valve Stenosis, Lupus Nephritis.

Introduction

Systemic lupus erythematosus (SLE) is a chronic systemic autoimmune disease characterized by heterogeneous clinical manifestations involving the skin, joints, kidneys, nervous system, hematological system, and cardiovascular system [1–3]. Cardiovascular involvement contributes substantially to morbidity and mortality in SLE and may result from accelerated atherosclerosis, myocarditis, pericardial disease, pulmonary hypertension, coronary disease, or valvular abnormalities [2,3]. Valvular heart disease is a well-recognized manifestation of SLE. Echocardiographic studies have demonstrated that valvular abnormalities may be considerably more frequent than clinically apparent disease. In a prospective echocardiographic study of 69 patients with SLE, Roldan et al. identified valvular abnormalities in 61% of patients at baseline, with valvular thickening and vegetations being the most frequent findings. Although stenosis was uncommon, valvular disease was associated with clinically important outcomes including stroke, peripheral embolism, heart failure, infective endocarditis, and valve replacement [4].

Libman–Sacks Endocarditis (LSE), a form of non-infective valvular involvement characterized by sterile verrucous lesions, is particularly associated with SLE and antiphospholipid antibodies. In a cohort of 342 patients with SLE, Moyssakis et al. identified LSE in approximately 11% of patients and demonstrated associations with disease duration, disease activity, thrombosis, stroke, thrombocytopenia, anticardiolipin antibodies, and APS [5]. The presence of APS may further complicate the clinical course because thrombotic events can occur in multiple vascular territories, including the cerebral and mesenteric circulations. Severe valvular disease may ultimately require surgical intervention, and patients with SLE and APS may face additional perioperative challenges related to inflammation, thrombosis, anticoagulation, infection, and organ dysfunction [5–8]. We present a complex case of SLE with secondary APS, multisystem involvement, cerebrovascular disease, chronic kidney disease, severe aortic valve disease, and subsequent mechanical aortic valve replacement. The case is accompanied by a focused systematic review of the available literature concerning SLE-associated valvular heart disease.

Case Presentation

Patient Information and Initial Diagnosis

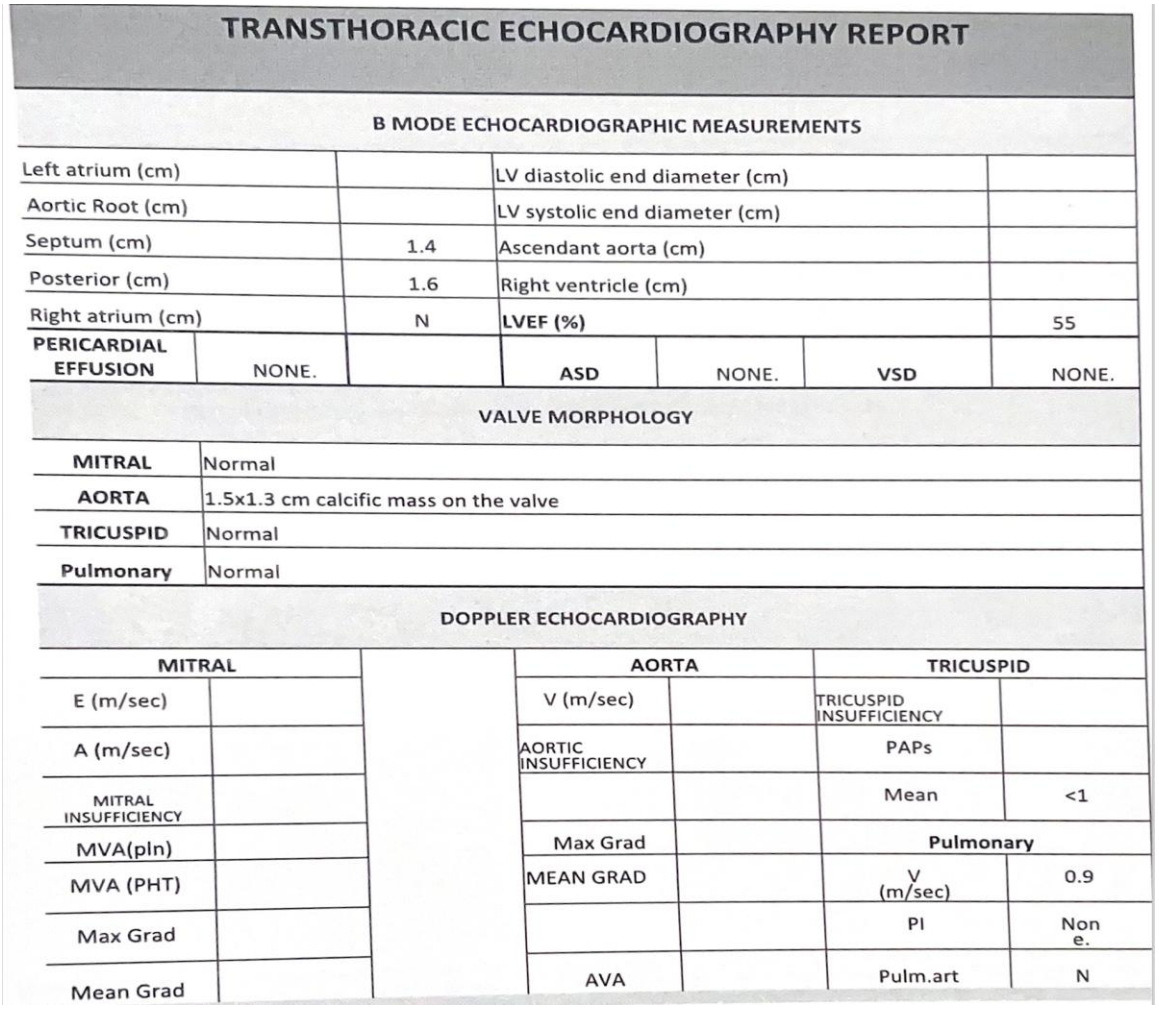
A 38-year-old man was diagnosed with SLE in 2019 following the development of facial cutaneous lesions. Skin biopsy demonstrated findings compatible with discoid lupus erythematosus, and antinuclear antibody testing was positive. The patient was subsequently followed as a case of SLE. During the subsequent disease course, he developed a severe systemic flare characterized by generalized skin manifestations and articular symptoms. Follow-up during this period was inconsistent, and corticosteroid therapy was reportedly intermittent. Laboratory reassessment demonstrated a high antinuclear antibody titer, reduced complement levels, and urinary abnormalities suggestive of active renal involvement. The overall clinical picture was considered compatible with lupus nephritis; however, renal biopsy was not performed because of limited available resources. Treatment included corticosteroid therapy at an initial dose of approximately 1 mg/kg/day followed by tapering, hydroxychloroquine 200 mg daily, an angiotensin-converting enzyme inhibitor, calcium and vitamin D supplementation, and mycophenolate mofetil 1 g twice daily. The patient subsequently achieved a period of relative clinical stability, although follow-up remained intermittent.

Cardiovascular Evaluation and Progression of Aortic Valve Disease

During follow-up, cardiac evaluation was requested after a clinical finding suggestive of aortic valve disease. Echocardiography demonstrated aortic valve stenosis. At that stage, no immediate surgical intervention was undertaken,

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and the patient continued under medical surveillance. After approximately 2 years of inconsistent follow-up, the patient returned with symptoms and clinical signs of heart failure. Echocardiography demonstrated advanced aortic stenosis with a reported mean transvalvular gradient ranging from 36 to 54 mmHg and marked left ventricular hypertrophy. The description of the valve as "congenital" in the original clinical documentation could not be independently verified from the available information. Therefore, the present report refers to the lesion as aortic valve stenosis rather than congenital aortic stenosis unless the original echocardiographic or operative documentation confirms a congenital morphology such as a bicuspid aortic valve. The patient was considered to be at increased operative risk according to the documented EuroSCORE assessment.



TRANSTHORACIC ECHOCARDIOGRAPHY REPORT

B MODE ECHOCARDIOGRAPHIC MEASUREMENTS

Left atrium (cm)		LV diastolic end diameter (cm)	
Aortic Root (cm)		LV systolic end diameter (cm)	
Septum (cm)	1.4	Ascendant aorta (cm)	
Posterior (cm)	1.6	Right ventricle (cm)	
Right atrium (cm)	N	LVEF (%)	55

PERICARDIAL EFFUSION	NONE.		ASD	NONE.	VSD	NONE.

VALVE MORPHOLOGY

MITRAL	Normal
AORTA	1.5x1.3 cm calcific mass on the valve
TRICUSPID	Normal
Pulmonary	Normal

DOPPLER ECHOCARDIOGRAPHY

MITRAL		AORTA		TRICUSPID	
E (m/sec)		V (m/sec)		TRICUSPID INSUFFICIENCY	
A (m/sec)		AORTIC INSUFFICIENCY		PAPs	
MITRAL INSUFFICIENCY				Mean	<1
MVA(pln)		Max Grad		Pulmonary	
MVA (PHT)		MEAN GRAD		V (m/sec)	0.9
Max Grad				PI	None.
Mean Grad		AVA		Pulm.art	N

Figure 2. Transthoracic echocardiography report demonstrating a calcific mass on the aortic valve and preserved left ventricular ejection fraction.

Neurological and Thrombotic Manifestations

The neurological history was notable for previous cerebrovascular events. Brain magnetic resonance imaging demonstrated widespread areas of encephalomalacia involving different vascular territories, consistent with sequelae of previous ischemic cerebrovascular insults. The patient subsequently developed epilepsy, which was controlled with levetiracetam. There was no evidence that the epilepsy represented an isolated primary neurological disorder; rather, the clinical context suggested a relationship to previous cerebrovascular injury. Further rheumatological evaluation demonstrated positive anti-double-stranded DNA antibodies, low complement levels, and a high-titer lupus anticoagulant. Given the patient's thrombotic history and antiphospholipid antibody findings, he was considered to have SLE complicated by secondary APS. For publication, the original laboratory report should be reviewed to document the exact antiphospholipid antibody profile, including whether lupus anticoagulant and/or anticardiolipin or anti-β2-glycoprotein I antibodies were persistently positive according to the required testing interval.

Renal Involvement

Renal disease remained clinically relevant throughout the patient's course. He was considered to have chronic kidney disease stage 2, with a reported baseline serum creatinine of approximately 1.4 mg/dL during the year preceding valve surgery. Low-grade proteinuria was also reported in the clinical documentation. The original record described this as "30 mg"; however, the unit and method of measurement are not available in the present dataset and should be verified before publication. The absence of renal biopsy prevented histopathological classification of lupus nephritis.

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Arrhythmia and Cardiac Decompensation

The patient experienced episodes of nonsustained ventricular tachycardia (NSVT) in the setting of advanced aortic valve disease and marked left ventricular hypertrophy. Beta-blocker therapy was initiated, with subsequent suppression of clinically documented NSVT. No recurrent ventricular tachyarrhythmia was reported during the immediate perioperative period. The decision regarding electrophysiological testing or implantable cardioverter-defibrillator therapy was made clinically and should not be interpreted as being solely determined by the suppression of NSVT. The patient's eventual presentation with overt heart failure was considered predominantly related to advanced valvular disease and the resulting ventricular remodeling, although his systemic autoimmune and renal disease represented important comorbid factors.

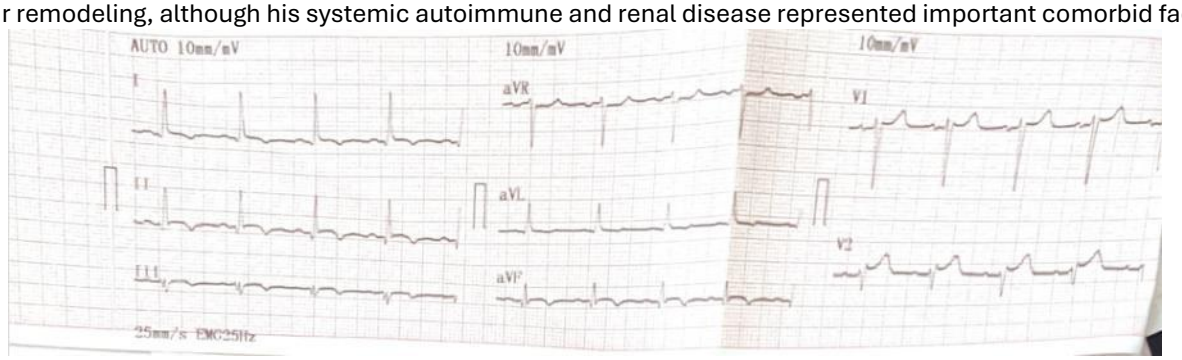


Figure 1. Preoperative electrocardiogram demonstrating the patient's cardiac rhythm and repolarization abnormalities

Aortic Valve Replacement

Given severe symptomatic aortic stenosis and clinical deterioration, the patient underwent surgical aortic valve replacement through median sternotomy. A mechanical St. Jude Regent valve was implanted. Intraoperatively, the native aortic valve was described as markedly calcified. A vegetation-like lesion was also identified and submitted for microbiological evaluation. Because SLE and APS can be associated with non-infective valvular lesions, particularly Libman–Sacks endocarditis, the differential diagnosis included both infective and non-infective valvular disease. The available clinical information did not establish Libman–Sacks endocarditis histologically; therefore, the lesion should be described as a vegetation-like valvular lesion rather than definitively classified as LSE. Multiple blood cultures were obtained, and no bacterial growth was reported. The patient received empiric antimicrobial treatment while infective endocarditis was being evaluated.

Early Postoperative Course

The initial postoperative course was favorable. The patient was extubated on postoperative day 2 and did not develop a new neurological deficit or documented acute perioperative stroke. No recurrence of NSVT was reported during the early postoperative period. Epilepsy remained controlled with levetiracetam. Renal function remained relatively stable at the patient's baseline CKD stage during the initial postoperative period. Because of the combination of a mechanical valve and APS, anticoagulation represented a major component of long-term management. This required close coordination between cardiology, cardiac surgery, rheumatology, and anticoagulation services because both thrombotic and bleeding risks were substantial.

Mesenteric Infarction

Approximately 1 month after valve replacement, the patient was admitted to the intensive care unit with acute abdominal pain. Imaging demonstrated mesenteric infarction, prompting emergency laparotomy and localized bowel resection. The patient subsequently deteriorated and required transfer to a tertiary-care facility. His clinical course was complicated by septic shock and prolonged intensive care treatment. Thoracic and abdominal imaging demonstrated minimal bilateral pleural effusions and a pericardial effusion considered likely reactive in the postoperative setting. Postoperative pneumonia was also reported. Management included vasopressor support, antimicrobial therapy, corticosteroid replacement/therapy, and adjustment of anticoagulation according to the clinical circumstances. The occurrence of mesenteric infarction in this setting was clinically concerning for a thrombotic complication, particularly given the patient's established thrombotic phenotype and APS. However, the exact mechanism should be stated as presumed unless operative pathology or vascular imaging definitively established arterial thrombosis.

Long-Term Outcome

After the prolonged postoperative illness, the patient initially stabilized and was discharged. During the following year, however, he experienced multiple hospital admissions because of recurrent medical complications. Renal function progressively deteriorated, ultimately requiring dialysis. Difficulties with coagulation control were also reported, accompanied by worsening electrolyte and nutritional abnormalities. Despite repeated medical interventions and multidisciplinary management, the patient's condition progressively deteriorated, and he ultimately died from multiorgan failure.

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Timeline of the Clinical Course

Year/Period| Major clinical event

- 2019| Facial cutaneous lesions; skin biopsy compatible with discoid lupus; ANA positive; diagnosis of SLE.
- 2020–2021| Systemic flare with cutaneous and articular manifestations, hypocomplementemia, and urinary abnormalities suggestive of lupus nephritis.
- 2021 onward| Immunosuppressive therapy including corticosteroids, hydroxychloroquine, and mycophenolate mofetil
- Subsequent follow-up| Aortic valve stenosis identified by echocardiography.
- Later period| Cerebrovascular events with subsequent MRI evidence of widespread encephalomalacia; epilepsy developed
- Preoperative period| Positive dsDNA, low C3, and high-titer lupus anticoagulant; secondary APS considered.
- 2024| Symptomatic severe aortic stenosis, marked LV hypertrophy, and heart failure; NSVT controlled medically.
- 2024| Surgical mechanical aortic valve replacement.
- Early postoperative period| Initial recovery without new neurological deficit or recurrent NSVT.
- Approximately 1 month after AVR| Mesenteric infarction requiring emergency laparotomy and bowel resection.
- Subsequent period| Septic shock, pneumonia, and prolonged critical illness.
- Following year| Recurrent admissions, progressive renal failure requiring dialysis, and coagulation instability.
- Outcome | Multiorgan failure and death.

Discussion

This case illustrates the complex interaction between systemic lupus erythematosus (SLE), antiphospholipid syndrome (APS), valvular heart disease, cerebrovascular injury, renal dysfunction, and thromboembolic complications. Although valvular abnormalities are relatively common in SLE, severe stenotic lesions are less frequent than valvular thickening, vegetations, and regurgitation [4,5]. In this patient, severe aortic stenosis with marked left ventricular hypertrophy and heart failure represented an unusual and clinically significant manifestation. The marked calcification suggests that multiple mechanisms may have contributed to the valve pathology rather than SLE alone. The vegetation-like lesion found on the native aortic valve raised the possibility of Libman–Sacks endocarditis (LSE), particularly given the coexistence of SLE, APS, previous cerebrovascular events, and negative blood cultures. However, the absence of documented valve histopathology prevents a definitive diagnosis of LSE. Therefore, the lesion is best described as vegetation-like with possible non-infective valvular involvement. This distinction is important because infective and non-infective endocarditis require different management strategies [5–8]. The patient's high-titer lupus anticoagulant and previous thrombotic events supported consideration of secondary APS.

MRI demonstrated extensive encephalomalacia involving multiple vascular territories, consistent with previous ischemic injury. These findings highlight the potential contribution of APS-associated thrombosis and, possibly, valvular embolization to the patient's neurological disease. The subsequent mesenteric infarction further demonstrated his thrombotic vulnerability, although its exact mechanism could not be established. Severe symptomatic aortic stenosis required surgical valve replacement. Mechanical valve implantation initially resulted in favorable recovery, without new neurological deficits or recurrent NSVT. However, the need for lifelong anticoagulation created additional challenges in the setting of APS. Approximately one month later, mesenteric infarction led to emergency bowel resection, septic shock, and prolonged critical illness, followed by progressive renal failure requiring dialysis and recurrent hospitalizations. Overall, the case emphasizes that severe valvular disease in SLE and APS requires careful differentiation between infective and non-infective lesions, close echocardiographic and thrombotic surveillance, and individualized anticoagulation. Although valve replacement can correct severe structural disease, the underlying systemic autoimmune and thrombotic risks may continue to determine long-term outcomes.

Strengths and Limitations

The major strength of this report is the longitudinal description of a patient with SLE, APS, multisystem disease, severe aortic valve pathology, cerebrovascular complications, mechanical valve replacement, and subsequent systemic complications. The case also provides an opportunity to compare an individual complex clinical trajectory with published cohorts of SLE-associated valvular disease and severe SLE requiring surgery. Several limitations should be acknowledged. First, renal biopsy was not performed, preventing histopathological classification of lupus nephritis. Second, the available information does not include the complete antiphospholipid antibody profile and documentation of persistence required for rigorous APS classification. Third, the histopathological examination of the excised valve has not been provided, preventing definitive classification of the vegetation-like lesion as Libman–Sacks endocarditis. Fourth, some quantitative clinical data, including the precise proteinuria measurement and complete echocardiographic parameters, require verification from the original medical record. Finally, the systematic literature search was initially conducted using Google Scholar and subsequently supplemented by targeted database searching; therefore, exact PRISMA flow numbers should not be reported until a reproducible final database search has been completed.

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Conclusion

This case illustrates the complex clinical interaction between systemic lupus erythematosus, antiphospholipid syndrome, valvular heart disease, cerebrovascular injury, renal dysfunction, and thromboembolic complications. Although valvular involvement is a recognized manifestation of SLE, severe stenotic disease requiring surgical replacement remains relatively uncommon. The coexistence of a valvular vegetation-like lesion, APS, and previous cerebrovascular events further complicates the diagnostic and therapeutic pathway. The literature demonstrates that SLE-associated valvular disease, particularly Libman–Sacks endocarditis, is associated with antiphospholipid antibodies, thrombotic events, cerebrovascular disease, and, in severe cases, the need for valve surgery [4–8]. In patients with SLE and APS who develop significant valvular disease, careful longitudinal echocardiographic surveillance, assessment of thrombotic risk, differentiation between infective and non-infective endocardial lesions, and individualized anticoagulation are essential. The present case also emphasizes that successful valve replacement represents only one component of management, as the underlying systemic autoimmune and thrombotic disorders may continue to produce life-threatening complications.

Informed Consent

Written informed consent for publication of the case report and accompanying clinical information was obtained from the patient/authorized representative, according to the documentation available to the authors.

Ethics Approval

Ethics committee/institutional review board requirements should be reported according to the policy of the institution and the target journal.

Conflict of Interest

The authors declare no conflicts of interest related to this work.

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