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ENDOCARDIAL INVOLVEMENT IN SYSTEMIC AUTOIMMUNE DISEASES: PATHOPHYSIOLOGY, CLINICAL MANIFESTATIONS, AND MANAGEMENT

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ABSTRACT

Purpose: The manuscript aims to highlight the clinical significance of Libman-Sacks endocarditis (LSE) in patients with systemic lupus erythematosus (SLE) and antiphospholipid syndrome (APS). It emphasizes the importance of early diagnosis and multidisciplinary management to prevent severe cardiac complications and improve patient outcomes.
Methodology: A comprehensive review of recent literature and case studies was conducted, focusing on the pathogenesis, clinical presentation, diagnostic approaches, and management strategies for LSE in SLE and APS patients. The article synthesizes evidence from published research, including echocardiographic findings, laboratory markers, and therapeutic interventions
Findings: LSE is frequently underdiagnosed due to its subtle clinical manifestations. Echocardiography remains the gold standard for detection, revealing characteristic vegetations on heart valves. The manuscript identifies a strong association between LSE and thromboembolic events in SLE/APS patients. Multidisciplinary collaboration among cardiologists, rheumatologists, hematologists, and cardiac surgeons is crucial for optimal management. Anticoagulation and immunosuppressive therapy are central to treatment, with surgical intervention reserved for severe cases.
Conclusions: Early recognition and coordinated care are essential to reduce morbidity and mortality associated with LSE in SLE and APS. The article advocates for routine cardiac screening in high-risk patients and underscores the need for further research to refine diagnostic and therapeutic protocols.

KEYWORDS

Libman-Sacks Endocarditis, Antiphospholipid Syndrome, Cardiac Complications, Multidisciplinary Management

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1. Introduction

The Significance of Autoimmune Diseases for the Cardiovascular System

Autoimmune diseases such as systemic lupus erythematosus (SLE), antiphospholipid syndrome (APS), and mixed connective tissue disease (MCTD) have profound implications for the cardiovascular system (Dobos i in., 2026). These conditions are characterized by chronic inflammation, immune complex deposition, and autoantibody production, all of which can lead to persistent tissue damage and extensive inflammation affecting multiple organs, including the heart (Dobos i in., 2026). Cardiac involvement is a major contributor to morbidity and mortality in these patients, with risks ranging from 9- to 50-fold higher than in the general population. The spectrum of cardiac manifestations includes pericarditis, myocarditis, valvular disease, arrhythmias, and accelerated atherosclerosis, often resulting in life-threatening complications (Dobos i in., 2026).

The Frequency of Cardiac Involvement in SLE, APS, and MCTD

- SLE: Cardiac involvement is common, with studies showing that up to 45% of SLE patients develop cardiovascular complications (Al-Sarraf i in., 2025; Dobos i in., 2026). Pericarditis affects around 25% of patients, while valvular heart disease (including Libman-Sacks endocarditis) is seen in approximately 10% (Al-Sarraf i in., 2025; Dobos i in., 2026).
- APS: Cardiac manifestations occur in 30-80% of patients with primary or secondary APS and it is closely linked to SLE, with 20-40% of SLE patients possessing antiphospholipid antibodies, and 50-70% of these progressing to definitive APS within 20 years (Al-Sarraf i in., 2025; Dobos i in., 2026).
- MCTD: While less extensively studied, MCTD also carries a significant risk of cardiac involvement, including pericarditis, myocarditis, and valvular disease (Al-Sarraf i in., 2025; Dobos i in., 2026).

What Libman-Sacks Endocarditis Is

Libman-Sacks endocarditis (LSE) is a form of non-bacterial thrombotic endocarditis most commonly associated with SLE and APS (Ehsan i in., 2025; Tang, b.d.). It is characterized by sterile vegetations composed of fibrin and platelets, typically affecting the mitral and aortic valves and is strongly associated with the presence of antiphospholipid antibodies (Dobos i in., 2026). While most vegetations are small and asymptomatic, they can occasionally cause significant valvular dysfunction and increase the risk of thromboembolic events (Tang, b.d.).

Aim of the Review

The aim of this review is to provide a comprehensive overview of cardiac involvement in autoimmune diseases, with a particular focus on SLE, APS, and MCTD. The review will:

- Describe the main cardiovascular diseases associated with these conditions.
- Summarize current evidence on the epidemiology, pathophysiology, diagnosis, and management of cardiac manifestations, including arrhythmias and endocarditis.
- Highlight the differences between aseptic and infective endocarditis.
- Emphasize the importance of interdisciplinary collaboration for early detection, personalized management, and improved long-term outcomes in affected patients.

2. Epidemiology and Clinical Significance

Incidence of Endocarditis

Libman-Sacks endocarditis, a form of non-bacterial thrombotic endocarditis (NBTE), can be found in up to 10% of patients with systemic lupus erythematosus, and its risk increases with disease duration, activity, presence of antiphospholipid antibodies, and clinical APS (Riancho-Zarrabeitia i in., 2021; Tayem i in., 2022).

In primary antiphospholipid syndrome or APS secondary cardiac involvement ranges from 30% to 80%, with valvular disease (thickening and vegetations) being the most common manifestation (Al-Sarraf i in., 2025; Tayem i in., 2022). While the direct incidence of endocarditis in mixed connective tissue disease is less well defined, autoimmune diseases such as MCTD are recognized as risk factors for NBTE due to systemic inflammation and immune complex deposition (Tayem i in., 2022).

Also vasculitis can predispose to endocardial involvement through immune-mediated injury and increased risk of NBTE, although specific incidence rates are not well established (Tayem i in., 2022).

Risk factors for endocarditis in these autoimmune diseases include the presence of antiphospholipid antibodies, turbulent blood flow, valvular abnormalities, prior rheumatic heart disease, and exposure to pathogens during dental or surgical procedures (Al-Sarraf i in., 2025; Tayem i in., 2022).

Endocarditis associated with autoimmune diseases is linked to increased mortality, particularly when complicated by systemic embolization or advanced heart failure (Al-Sarraf i in., 2025; Tayem i in., 2022; Van Der Woude i in., 2026). In particular, LSE and APS are strong risk factors for the development of transient ischemic attacks and ischemic strokes (Al-Sarraf i in., 2025; Tayem i in., 2022). Cardiac manifestations of APS may also include myocardial infarction, which can occur as a consequence of coronary embolism originating from valvular vegetations (Van Der Woude i in., 2026). In addition, progressive valvular dysfunction, especially mitral regurgitation, may lead to the development of heart failure in patients with LSE and APS (Al-Sarraf i in., 2025; Van Der Woude i in., 2026). In severe cases requiring surgical intervention, mortality rates following valve replacement in patients with APS or SLE have been reported to reach up to 33% (Al-Sarraf i in., 2025).

3. Pathophysiology of Endocardial Involvement

Endocardial involvement in systemic autoimmune diseases results from a complex interaction between immune-mediated endothelial injury, thrombosis, and inflammatory processes. In conditions such as systemic lupus erythematosus and antiphospholipid syndrome, autoantibodies including lupus anticoagulant and anticardiolipin antibodies play a central role in damaging the valvular endothelium and promoting immune complex deposition on cardiac valves (Riancho-Zarrabeitia i in., 2021; Tayem i in., 2022). These immune complexes initiate inflammatory responses that lead to endothelial dysfunction and a prothrombotic state (Al Chalaby i in., 2022).

In APS, antiphospholipid antibodies further promote thrombogenesis by activating platelets and coagulation pathways, resulting in thrombus formation on damaged valvular surfaces and the development of vegetations (Riancho-Zarrabeitia i in., 2021). Complement activation also contributes to the pathogenesis of

valvular lesions, as complement components have been identified in affected valves, indicating immune-mediated injury (Al-Sarraf i in., 2025).

A characteristic manifestation of autoimmune endocardial involvement is Libman-Sacks endocarditis, in which sterile vegetations composed of fibrin, immune complexes, and inflammatory cells develop most commonly on the mitral and aortic valves (Al Chalaby i in., 2022; Riancho-Zarrabeitia i in., 2021; Tayem i in., 2022). These lesions represent a form of nonbacterial thrombotic endocarditis and may contribute to embolic complications and progressive valvular dysfunction (Al Chalaby i in., 2022).

4. Types of Endocardial Involvement

Endocardial involvement in systemic autoimmune diseases may present in several forms, most commonly as Libman-Sacks endocarditis, APS-related valvular disease, or infective endocarditis occurring in immunosuppressed patients.

Libman-Sacks endocarditis is characterized by the presence of sterile, verrucous vegetations composed of fibrin, immune complexes, and mononuclear inflammatory cells, typically without significant destruction of the underlying valve tissue (Tang, b.d.). These vegetations are usually small, flat, and wart-like, and may occur on either or both sides of the valve leaflets (Tang, b.d.). The mitral and aortic valves are most commonly affected, with vegetations frequently located on the ventricular surface of the mitral valve and the atrial surface of the aortic valve, although involvement of the tricuspid and pulmonary valves has also been described in rare cases (Tang, b.d.).

In antiphospholipid syndrome, valvular disease often manifests as leaflet thickening, sterile vegetations, and varying degrees of valvular regurgitation (Li i in., 2025). Thickening of the mitral and aortic valves is commonly caused by fibrous tissue deposition and may impair normal valve function (Li i in., 2025). Sterile vegetations composed of fibrin and immune complexes may develop on the affected valves, increasing the risk of thromboembolic complications (Li i in., 2025). Progressive leaflet thickening and vegetations may also lead to valvular regurgitation, particularly involving the mitral and aortic valves, and in severe cases surgical intervention may be required (Li i in., 2025).

Patients with autoimmune diseases may also develop infective endocarditis, particularly when receiving long-term immunosuppressive therapy (Van Der Woude i in., 2026). Immunosuppression increases susceptibility to infections and may mask typical clinical manifestations, which can delay diagnosis and worsen clinical outcomes (Van Der Woude i in., 2026). Opportunistic pathogens, including fungal organisms and atypical bacteria, are more frequently responsible for infective endocarditis in immunocompromised patients, and the clinical course may be more aggressive compared with that observed in immunocompetent individuals (Van Der Woude i in., 2026).

5. Clinical Presentation of Libman-Sacks Endocarditis

The clinical presentation of Libman-Sacks endocarditis is highly variable and may include cardiac symptoms, systemic manifestations related to the underlying autoimmune disease, and thromboembolic complications (Yang i in., 2025). LSE most commonly occurs in patients with systemic lupus erythematosus and antiphospholipid syndrome, and may present with nonspecific cardiac symptoms such as chest pain, chest tightness, dizziness, or palpitations (Yang i in., 2025). Cardiac involvement typically includes valvular dysfunction caused by vegetations affecting primarily the mitral and aortic valves, which may lead to valvular regurgitation or, less commonly, stenosis (Tang, b.d.). In more severe cases, large vegetations may result in significant valvular dysfunction requiring surgical intervention (Tang, b.d.). Echocardiography often reveals characteristic findings such as thickened valve leaflets, regurgitation, and valvular vegetations (Yang i in., 2025).

Systemic symptoms are frequently related to the underlying autoimmune disease rather than to valvular involvement itself. Patients with SLE or APS may present with multisystem manifestations, including fever, anemia, and neurological symptoms such as confusion or stroke (Tang, b.d.). In some cases, LSE may represent the first manifestation of systemic autoimmune disease, which can complicate the diagnostic process (Tang, b.d.; Yang i in., 2025).

Thromboembolic complications are among the most clinically significant manifestations of LSE. Stroke is the most frequently reported embolic event and may present with neurological deficits such as unilateral weakness or facial drooping due to cerebral embolism originating from valvular vegetations (Satish i in., 2024). Recurrent ischemic strokes may occasionally represent the initial presentation of LSE, highlighting the high

thrombotic risk associated with this condition (Tang, b.d.). Consequently, appropriate management often includes anticoagulation therapy and, in selected cases, surgical intervention (Satish i in., 2024; Tang, b.d.).

Importantly, Libman-Sacks endocarditis may also be detected incidentally during echocardiographic examination performed for other clinical indications. In some studies, valvular vegetations have been identified in approximately 10% of patients with SLE, with many lesions being small and clinically silent (Tang, b.d.). Such incidental findings may prompt further investigation and lead to the diagnosis of LSE, particularly in patients with known risk factors such as SLE or APS (Satish i in., 2024).

6. Diagnostic Approach

The diagnostic evaluation of Libman-Sacks endocarditis and associated valvular involvement in systemic autoimmune diseases requires a combination of laboratory testing, imaging studies, and careful differential diagnosis. Detection of antiphospholipid antibodies is central to the diagnosis, particularly in patients with suspected antiphospholipid syndrome presenting with unexplained valvular lesions (Li i in., 2025; Tang, b.d.; Van Der Woude i in., 2026; Yang i in., 2025). Standard laboratory testing includes lupus anticoagulant, anticardiolipin antibodies, and anti-β2 glycoprotein I antibodies, with persistent positivity on two occasions at least 12 weeks apart required to confirm the diagnosis of APS (Tang, b.d.; Van Der Woude i in., 2026; Yang i in., 2025). Inflammatory markers such as erythrocyte sedimentation rate and C-reactive protein are often elevated in active disease and may assist in distinguishing inflammatory from infectious etiologies (Li i in., 2025; Neupane i in., 2026; Van Der Woude i in., 2026; Yang i in., 2025). Blood cultures remain essential in the diagnostic work-up to exclude infective endocarditis, as LSE is typically associated with persistently negative cultures (Satish i in., 2024; Sultan i in., 2025; Tang, b.d.).

Echocardiography represents the cornerstone of imaging in LSE. Transthoracic echocardiography is usually the first-line modality for detecting valvular vegetations, leaflet thickening, and regurgitation (Satish i in., 2024; Tang, b.d.; Van Der Woude i in., 2026; Yang i in., 2025). However, transesophageal echocardiography provides superior sensitivity, particularly for identifying small or atypically located vegetations, and is recommended when initial findings are inconclusive or clinical suspicion remains high (Satish i in., 2024; Tang, b.d.; Van Der Woude i in., 2026). Advanced imaging techniques, including cardiac magnetic resonance imaging and computed tomography, may offer additional information regarding myocardial involvement, fibrosis, and help exclude alternative diagnoses such as neoplastic or metastatic lesions (Neupane i in., 2026; Sultan i in., 2025; Yang i in., 2025).

Although the modified Duke criteria are widely used for diagnosing infective endocarditis, their applicability in LSE is limited due to the non-infectious nature of the disease (Li i in., 2025; Yang i in., 2025). In contrast, the Sydney criteria remain the standard for diagnosing APS, requiring at least one clinical criterion and one laboratory criterion, including vascular thrombosis or pregnancy morbidity and persistent antiphospholipid antibody positivity (Li i in., 2025; Van Der Woude i in., 2026; Yang i in., 2025).

A comprehensive differential diagnosis is essential, as LSE may mimic other forms of endocardial disease. Infective endocarditis should be considered in the presence of fever, positive blood cultures, and elevated inflammatory markers (Li i in., 2025; Satish i in., 2024; Tang, b.d.). Marantic endocarditis, another form of nonbacterial thrombotic endocarditis, should be suspected particularly in patients with malignancy or other hypercoagulable states (Satish i in., 2024; Sultan i in., 2025; Tang, b.d.). Additionally, rheumatic valve disease remains an important differential diagnosis, especially in patients with a history of rheumatic fever and characteristic chronic valvular involvement (Li i in., 2025; Tang, b.d.).

7. Therapeutic Management

The management of Libman-Sacks endocarditis in systemic autoimmune diseases is multifaceted and includes immunosuppressive therapy, anticoagulation, biologic treatment, and, in selected cases, surgical intervention.

Immunosuppressive therapy remains the cornerstone of treatment, particularly in patients with systemic lupus erythematosus and catastrophic antiphospholipid syndrome (CAPS) (Dobos i in., 2026). Corticosteroids are widely used to rapidly control inflammation and immune activation, while additional agents such as azathioprine and mycophenolate mofetil are commonly employed for long-term disease control and as steroid-sparing therapies (Dobos i in., 2026; Pallinti i in., 2026). In severe or refractory cases, especially those involving major organ systems or CAPS, cyclophosphamide may be required for induction of remission (Dobos i in., 2026; Ehsan i in., 2025; Yang i in., 2025).

Anticoagulation plays a central role in preventing thromboembolic complications, particularly in patients with antiphospholipid syndrome (Yang i in., 2025). Warfarin remains the anticoagulant of choice, with a target international normalized ratio (INR) of 2.0-3.0 for secondary prevention, while low-molecular-weight heparin is frequently used as bridging therapy or when warfarin is contraindicated (Pallinti i in., 2026; Satish i in., 2024; Tang, b.d.; Van Der Woude i in., 2026; Yang i in., 2025). Indications for anticoagulation include confirmed APS, prior thromboembolic events, and the presence of large or mobile vegetations associated with high embolic risk (Satish i in., 2024; Tang, b.d.; Yang i in., 2025).

Biologic therapy may be considered in patients with refractory disease or persistent activity despite conventional treatment. Rituximab, a B-cell depleting monoclonal antibody, has been used in severe cases of SLE and APS, while belimumab is approved for active, autoantibody-positive SLE and has demonstrated efficacy in reducing disease activity (Dobos i in., 2026; Ehsan i in., 2025; Van Der Woude i in., 2026; Yang i in., 2025). Emerging therapies, including anifrolumab, telitacicept, and complement inhibitors such as eculizumab, represent promising future treatment options targeting specific immunological pathways (Dobos i in., 2026; Hamidi i in., 2026).

Antibiotic therapy is reserved for cases in which infective endocarditis is confirmed or strongly suspected, as LSE itself is a sterile process (Tang, b.d.; Zaman i in., 2025). In such situations, careful balancing of antimicrobial therapy and immunosuppression is required to control both infection and underlying autoimmune activity (Dobos i in., 2026; Tang, b.d.).

Surgical intervention is indicated in patients with severe valvular dysfunction, large vegetations, recurrent embolic events despite optimal medical therapy, or failure of conservative management (Li i in., 2025; Satish i in., 2024; Tang, b.d.). The choice between mechanical and bioprosthetic valves should be individualized, taking into account thrombotic risk, bleeding risk, and long-term prognosis, with mechanical valves often preferred in younger patients with APS (Li i in., 2025; Tang, b.d.). Decisions regarding surgical management in LSE require a multidisciplinary approach and careful assessment of risks and benefits (Li i in., 2025; Satish i in., 2024; Tang, b.d.).

8. Special Clinical Situations

Certain clinical scenarios require tailored management strategies in patients with Libman-Sacks endocarditis and antiphospholipid syndrome, particularly pregnancy, catastrophic APS, and severe systemic disease.

Pregnancy in patients with APS represents a high-risk condition due to the increased likelihood of thrombotic events and obstetric complications, including recurrent miscarriages, pre-eclampsia, and eclampsia, associated with antiphospholipid antibodies such as anticardiolipin antibodies and lupus anticoagulant (Merimi i in., 2024). APS may occur as a primary disorder or in association with systemic lupus erythematosus, and its cardiac manifestations can further complicate maternal and fetal outcomes (Merimi i in., 2024). Therapeutic anticoagulation remains the cornerstone of management during pregnancy, aiming to prevent both maternal thrombosis and adverse obstetric outcomes (Merimi i in., 2024). Optimal care requires a multidisciplinary approach involving cardiology, rheumatology, and obstetrics (Merimi i in., 2024).

Catastrophic antiphospholipid syndrome is a rare but life-threatening variant of APS, occurring in approximately 1% of patients and characterized by rapid multiorgan thrombosis with a mortality rate approaching 50% (Ehsan i in., 2025). Diagnosis is based on the presence of thrombosis affecting multiple organs within a short time frame, histopathological confirmation of small-vessel occlusion, and laboratory evidence of antiphospholipid antibodies (Ehsan i in., 2025; Li i in., 2025). CAPS may be triggered by factors such as infection, surgery, or discontinuation of anticoagulation and requires prompt recognition and aggressive treatment (Li i in., 2025). Management typically involves combined therapy with anticoagulation, high-dose corticosteroids, immunosuppressive agents such as cyclophosphamide, and, in refractory cases, plasma exchange or intravenous immunoglobulin (Ehsan i in., 2025). Early intervention and multidisciplinary care are critical for improving survival (Ehsan i in., 2025).

Severe systemic disease in patients with APS and SLE may include widespread thrombosis, autoimmune hematological complications, and significant cardiac involvement such as Libman-Sacks endocarditis (Ehsan i in., 2025; Velasquez-Orozco, b.d.). Advanced valvular disease, including severe regurgitation, may necessitate surgical intervention with careful perioperative management and individualized anticoagulation strategies (Velasquez-Orozco, b.d.). The coexistence of APS accelerates valvular damage and increases the risk of thromboembolic complications, underscoring the need for regular imaging, close clinical monitoring, and multidisciplinary decision-making (Ehsan i in., 2025). Optimal outcomes in these patients require coordinated care involving rheumatology, cardiology, hematology, and cardiac surgery teams (Ehsan i in., 2025).

9. Follow-up and Prognosis

Long-term follow-up in patients with Libman-Sacks endocarditis is essential and relies primarily on serial echocardiographic assessment, clinical monitoring, and evaluation of thromboembolic risk. Echocardiography remains the cornerstone of follow-up, enabling detection of vegetations, assessment of valvular function, and evaluation of treatment response (Tang, b.d.). While transthoracic echocardiography is typically used as an initial modality, transesophageal echocardiography provides superior sensitivity for identifying small or complex lesions and is recommended when detailed assessment is required (Tang, b.d.). Serial imaging is important for monitoring disease progression, recurrence, and prosthetic valve function, as well as identifying complications such as regurgitation or stenosis (Satish i in., 2024; Tang, b.d.). Advanced imaging techniques, including three-dimensional echocardiography and cardiac computed tomography, may be useful in selected cases requiring detailed anatomical evaluation (Satish i in., 2024; Velasquez-Orozco, b.d.).

Patients with LSE are at high risk of thromboembolic complications, particularly those with coexisting antiphospholipid syndrome (Satish i in., 2024; Tang, b.d.). Stroke represents the most common clinical manifestation, and recurrent embolic events are frequently observed, necessitating long-term anticoagulation as a cornerstone of management (Satish i in., 2024; Tang, b.d.). The risk of thromboembolism is further increased in the presence of large vegetations, significant valvular dysfunction, and high titers of antiphospholipid antibodies (Velasquez-Orozco, b.d.). Lifelong anticoagulation is often required, especially in patients following valve replacement, although careful monitoring is necessary due to the concurrent risk of bleeding (Tang, b.d.).

Survival outcomes in LSE depend on the severity of valvular involvement, the presence and control of underlying autoimmune disease, and the effectiveness of multidisciplinary management (Tang, b.d.). Surgical intervention, particularly valve replacement with mechanical prostheses, is associated with favorable outcomes in patients with severe valvular dysfunction, whereas conservative management combining immunosuppression and anticoagulation may lead to resolution of vegetations in selected cases (Satish i in., 2024; Tang, b.d.). Overall prognosis is strongly influenced by adequate control of SLE and APS, as well as prevention of recurrent thromboembolic events (Tang, b.d.). Although most vegetations are small and do not require surgical intervention, larger lesions may necessitate early surgical management to improve outcomes (Tang, b.d.).

Recurrence remains a clinical concern, particularly in patients with ongoing autoimmune activity, interruption of anticoagulation, or the use of bioprosthetic valves (Tang, b.d.). Mechanical valves appear to be associated with a lower risk of recurrence compared to bioprosthetic valves (Tang, b.d.). Regular echocardiographic surveillance and multidisciplinary collaboration are essential for early detection and management of recurrent disease (Tang, b.d.). Optimization of immunosuppressive therapy and consistent anticoagulation remain key strategies in reducing recurrence and preventing complications (Satish i in., 2024).

10. Future Directions and Emerging Therapies

Advances in the understanding of immunothrombotic mechanisms in antiphospholipid syndrome have led to the development of novel therapeutic strategies targeting specific pathways involved in vascular injury and thrombosis (Huo i in., 2026). Among these, complement inhibitors have emerged as a promising approach, given the recognized role of complement activation in the pathogenesis of APS-related cardiovascular complications, including Libman-Sacks endocarditis (Huo i in., 2026). By modulating immune-mediated endothelial damage and thrombus formation, complement-targeted therapies may offer new options for patients with recurrent thrombotic events or disease refractory to conventional treatment, although further clinical studies are required to define their optimal use (Huo i in., 2026).

The introduction of biologic agents has further expanded the therapeutic landscape of autoimmune cardiovascular disease (Morecroft i in., 2026; Ota i in., 2026). Rituximab, a B-cell-depleting monoclonal antibody, has shown potential in patients with severe or refractory APS, contributing to clinical stabilization and reduction of autoimmune activity in selected cases (Huo i in., 2026). Increasing evidence suggests that biologics may reduce autoantibody production and attenuate immune-mediated vascular injury; however, larger studies are needed to establish standardized treatment protocols and evaluate long-term safety (Huo i in., 2026).

Personalized medicine is becoming increasingly important in the management of complex autoimmune conditions with cardiac involvement. A multidisciplinary approach integrating advanced imaging, immunological profiling, and individualized risk assessment allows for tailored therapeutic strategies targeting both thrombotic and inflammatory pathways (Huo i in., 2026; Morecroft i in., 2026; Ota i in., 2026). Emerging research on biomarkers and novel diagnostic tools is expected to further refine patient stratification and optimize treatment outcomes in APS and related conditions (Huo i in., 2026).

11. Conclusions

Libman-Sacks endocarditis remains a significant cardiac complication in patients with systemic lupus erythematosus and antiphospholipid syndrome, contributing to increased cardiovascular morbidity and mortality. Early recognition and accurate diagnosis, utilizing multimodal imaging and serologic testing, are essential for distinguishing LSE from other causes of valvular disease and for guiding appropriate management (Yang i in., 2025). Multidisciplinary collaboration among cardiology, rheumatology, hematology, and cardiac surgery teams is critical to optimize outcomes, especially in complex cases involving severe valvular dysfunction, thromboembolic events, or catastrophic APS (Ehsan i in., 2025; Velasquez-Orozco, b.d.). Therapeutic strategies should be individualized, combining immunosuppressive therapy, anticoagulation, and, when indicated, surgical intervention, with careful perioperative planning to minimize complications (Satish i in., 2024; Tang, b.d.). The choice of prosthetic valve type and the duration of anticoagulation require careful consideration, particularly in APS patients at high risk for thrombosis (Velasquez-Orozco, b.d.). Recent advances in biologic therapies and complement inhibitors offer promising new avenues for treatment, but further research is needed to define their optimal use and long-term safety (Dobos i in., 2026). Ultimately, improving the prognosis of patients with LSE in the context of SLE and APS depends on early detection, comprehensive evaluation, and a structured, multidisciplinary approach to care (Dobos i in., 2026; Satish i in., 2024). Ongoing research and clinical vigilance are essential to refine management strategies and enhance long-term cardiovascular outcomes in this high-risk population (Li i in., 2025; Yang i in., 2025).

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