Cureus
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Open Access Case Report

Surgical and Clinical Insights Into Libman-Sacks Endocarditis of the Mitral Valve: A Case Report and Literature Review

Review began 09/27/2026
Review ended 11/02/2026
Published 11/14/2026

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DOI: 10.7759/cureus.119675

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Abstract

Libman-Sacks, a relatively rare cause of endocarditis, is usually associated with underlying systemic lupus erythematosus and/or antiphospholipid syndrome (APS). In cases of clinical or severe valve regurgitation or stenosis, the treatment approach is to perform a valve replacement or valvuloplasty.

We report two cases of young adult male patients with APS who developed Libman-Sacks endocarditis. The first patient is a 38-year-old man, and the second is a 33-year-old man. Both patients had a documented history of thromboembolic events and were receiving anticoagulant therapy. They presented to the emergency department with acute stroke symptoms. The first patient underwent mechanical valve replacement, whereas the second was managed with mitral valvuloplasty. We did a literature review of 10 case reports involving mitral valve replacement and four case reports of mitral valve repair. This literature search extracted demographic, clinical, surgical, and outcome data of 14 patients.

Libman-Sacks is an important differential diagnosis when encountering a case of endocarditis with vegetations. We should especially suspect this entity when a patient has an underlying rheumatologic disease. More studies are needed to better comprehend the pathogenesis and treatment of Libman-Sacks.

Categories: Rheumatology, Cardiac/Thoracic/Vascular Surgery
Keywords: antiphospholipid syndrome (apls), libman-sacks endocarditis (lse), mitral valve regurgitation, systemic lupus erythematosus, valve surgery

Introduction

Libman-Sacks endocarditis is a disease characterized by the presence of vegetations involving the heart valves [1-3]. This pathology is a noninfectious form of endocarditis and is strongly associated with systemic lupus erythematosus (SLE) and antiphospholipid syndrome (APS). In the past, its prevalence was underestimated due to asymptomatic cases; however, in recent years, advances in imaging diagnostics have increased awareness of this disease, with current diagnostic rates ranging from 13% to 50% depending on the literature [1-3].

The pathogenesis of this disease is not yet clear, but it is believed that immune complex activation and deposition at the valvular level precipitates the formation of vegetations [1-3]. The clinical presentation of this disease often mimics that of infectious endocarditis, causing fever, arthritis, new-onset murmurs, splinter hemorrhages, valvulopathies with their complications depending on the affected valve, and thromboembolic events [1-3].

A definitive diagnosis is achieved through imaging studies, with transesophageal echocardiography offering the best specificity and sensitivity. Finally, management focuses on controlling the underlying disease with immunosuppressants and anticoagulants. Surgery is reserved for patients with complications such as embolic and thrombotic events, especially ischemic stroke, transient ischemic attack, and systemic emboli (renal, splenic, mesenteric), in addition to valvular insufficiency, most frequently affecting the mitral valve, which can progress to heart failure. There is also a risk of arterial and venous thrombosis, especially in patients with APS, and although rare, it can present with an enhanced clinical state of infective endocarditis [1-3].

In this paper, we present two cases worth documenting. Both were male patients, a notable contribution given that this disease is predominantly observed in women. Also, both of our patients had cerebrovascular accidents; despite being on anticoagulation, this shows the importance of considering Libman-Sacks as an embolic source, even in patients who are already on anticoagulation. Finally, these cases offer a direct side-by-side comparison of two surgical approaches in a disease where there is no formal consensus. In addition,

we conducted a literature review with the objective of presenting the most common clinical features and the management associated with Libman-Sacks endocarditis.

Case Presentation

The two cases presented are retrospective in nature, as we have reviewed two clinical cases that were operated on in the past and observed their evolution until the last known records, taking place at the Hospital Metropolitano of Quito, Ecuador; the study period for case 1 was from November 23, 2023, to April 1, 2024, and for case 2 from August 2, 2025, to November 19, 2025. The cases were included consecutively.

Case 1

The patient is a 38-year-old man with a history of APS, treated with Rivaroxaban 10 mg, following a pulmonary thromboembolic episode. He presented with weakness in the right lower and upper limbs, accompanied by a nine-day history of dizziness, nausea, and headache. He also had Broca's aphasia. He was diagnosed with an ischemic stroke. On clinical evaluation, physical findings were negative for paresis and aphasia.

A magnetic resonance imaging of the brain was performed, showing findings suggestive of ischemia involving the left frontoparietal region and the right cerebral hemisphere (Figure *1*). A transesophageal echocardiogram (TEE) revealed symmetrical mass-like structures on the mitral valve leaflets, preserved left ventricular ejection fraction, and normal volume and size of the cardiac chambers (Figure *2*).

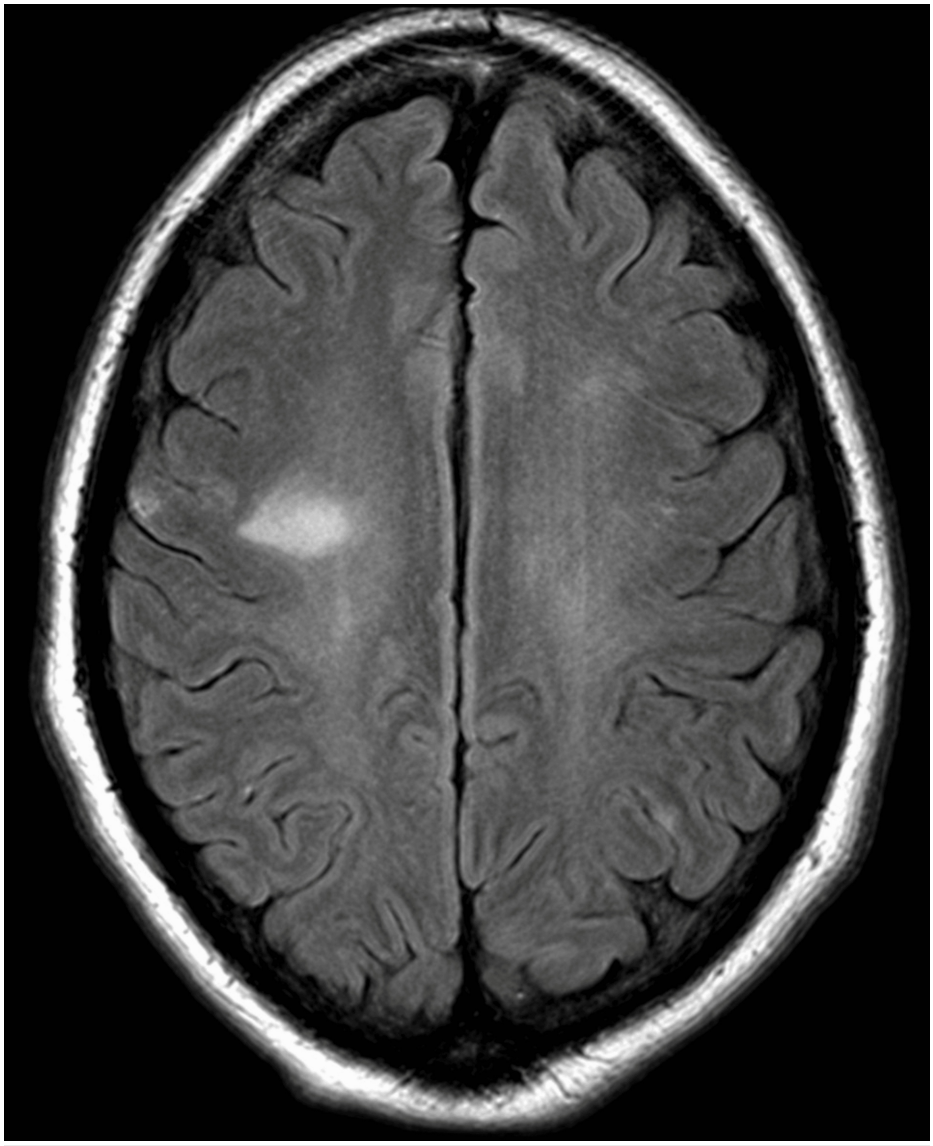


FIGURE 1: Magnetic resonance imaging: FLAIR sequence. Hyperintense nodular foci in the right middle cerebral artery territory

FLAIR: fluid-attenuated inversion recovery

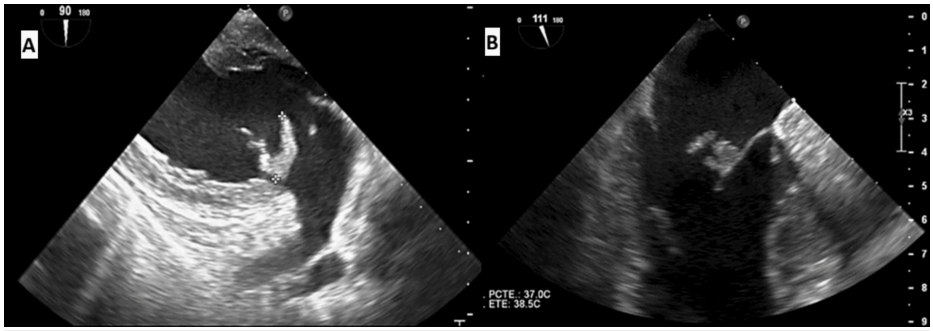


FIGURE 2: Transesophageal echocardiogram showing multiple mass-like structures in the anterior and posterior leaflets of the mitral valve (approximately 16 mm). There is no thrombus in any chamber of the heart. (A) 90° and (B) 111°

The patient was treated with perioperative antibiotic prophylaxis (cefuroxime 1.5 g intravenous every 12 hours for three doses), antihypertensives (perindopril + amlodipine 5/5 mg oral tablet every 12 hours), postoperative anticoagulants (warfarin 5 mg oral tablet every 24 hours), diuretics (furosemide 5 mg orally every 24 hours and spironolactone 25 mg orally every 24 hours), and beta-blockers (bisoprolol 2.5 mg orally every 12 hours), along with respiratory therapy using an incentive spirometer.

Based on these findings, the therapeutic plan was to perform either a valve repair or a valve replacement with temporary epicardial pacemaker implantation, assisted with extracorporeal circulation. During surgery, a TEE was performed, and a patent foramen ovale was discovered. A transseptal approach to the left atrium was performed, and upon exploration of the mitral valve, several vegetations were seen on the anterior leaflet measuring 0.5 cm and on the posterior leaflet measuring approximately 2 cm (Figure *3*). Additionally, the mitral ring had a diameter of 29 mm.

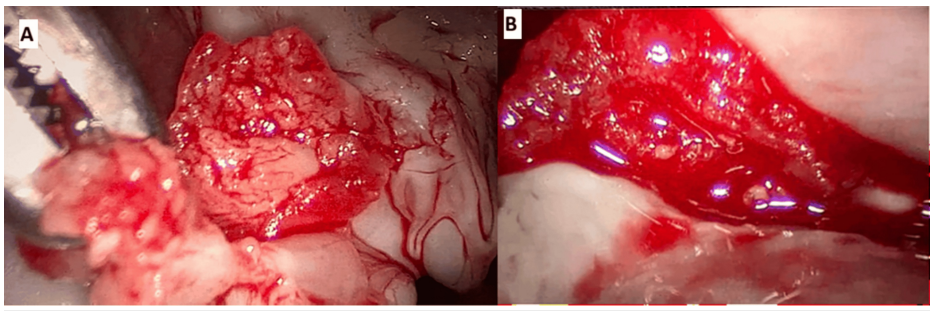


FIGURE 3: Vegetations at the level of the (A) posterior (4 cm) and (B) anterior (0.5 cm) mitral valve

The mitral leaflets with the vegetations were excised, and a mechanical prosthesis was implanted using a 29 mm S.J.M. Regent (Abbott, North Chicago, IL) valve. Intraoperative TEE confirmed that the mechanical valve was correctly positioned, with a maximum valvular gradient of 4 mmHg and a mean gradient of 1 mmHg.

Histopathological examination of the excised mitral valve revealed dense connective tissue and the presence of vegetations composed of fibrinoid material with polymorphonuclear cells, neutrophils, mineralization foci, and no microorganisms.

The patient remained clinically stable during the postoperative course and was discharged home after nine days of hospitalization. He was prescribed warfarin for anticoagulation. There were no complications from the surgery or from the medications the patient received. The patient has remained stable since discharge, without recurrence of any stroke episodes. Up to his last follow-up, the patient is in good condition and without symptoms.

Case 2

A 33-year-old male patient with a previous diagnosis of APS, on treatment with Warfarin 2.5 mg/day, and with a history of an ischemic cerebrovascular event. Before this hospitalization, he experienced dizziness for six days with impaired mobility, accompanied by nystagmus, asthenia, discomfort, and two episodes of nausea with vomiting. He presented to the emergency room, where diagnostic evaluation revealed a mass on the mitral valve. He was admitted to our hospital for surgery. He denied hemoptysis, fever, recent travel, or family illnesses. Physical examination showed no significant findings. Laboratory tests revealed an inflammatory process with leukocytosis, neutrophilia, and apparent acute renal failure. A TEE revealed lesions attached to the atrial surface of the anterior and posterior mitral valve leaflets, measuring approximately 0.2 x 0.1 cm and 1.3 × 0.9 cm (Figure *4*).

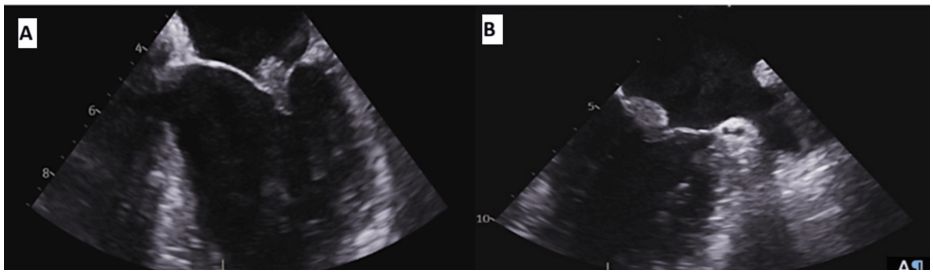


FIGURE 4: Transesophageal echocardiogram showing a rounded, moderately mobile mass attached to the posterior atrial leaflet, measuring 13 x 9 mm. Mild Carpentier type II mitral regurgitation. (A) 60° and (B) 10°

The patient was treated with perioperative antibiotic prophylaxis (cefuroxime 1.5 g intravenous every 12 hours for three doses), postoperative anticoagulants (warfarin 7.5 mg oral tablet every 24 hours), diuretics (furosemide 10 mg orally every eight hours), benzodiazepines (ketazolam 15 mg orally A.M. and 30 mg P.M.), along with respiratory therapy using an incentive spirometer.

The patient underwent a surgical sternotomy and intracardiac evaluation, resulting in resection of the mitral valve lesions (anterior and posterior leaflets) and repair of the mitral valve, temporary pacemaker placement, without complications from using extracorporeal circulation. Intraoperatively, we found two masses: one on the anterior mitral valve leaflet and another on the posterior leaflet (Figure *5*). The biopsy results are shown in Figure *6*.

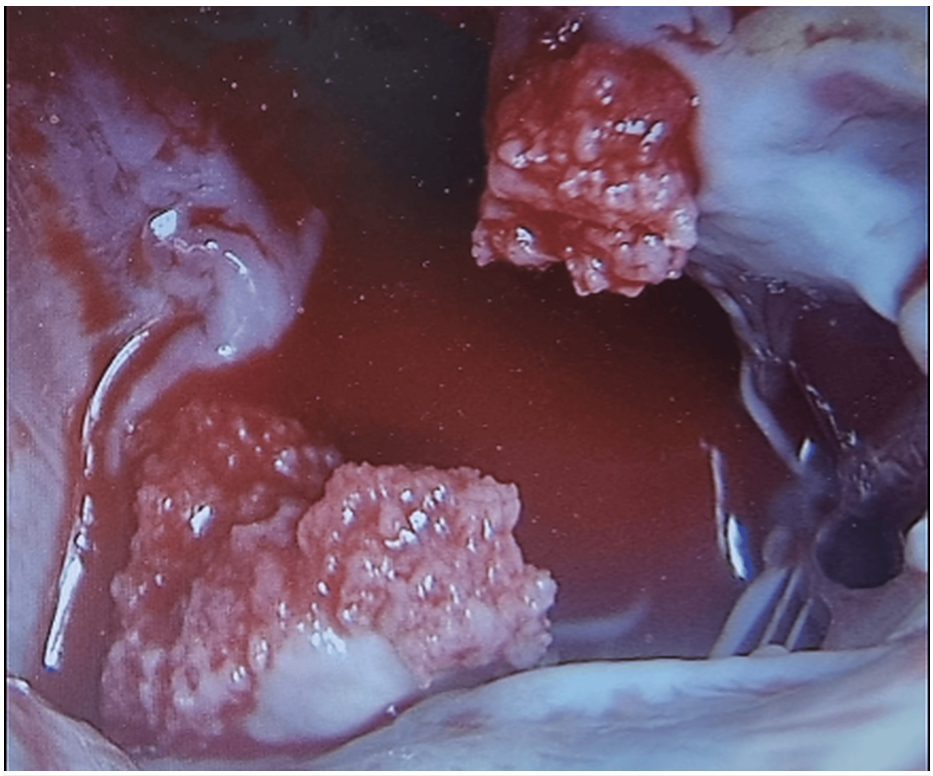


FIGURE 5: Lesions: one on the anterior mitral valve leaflet and another on the posterior leaflet

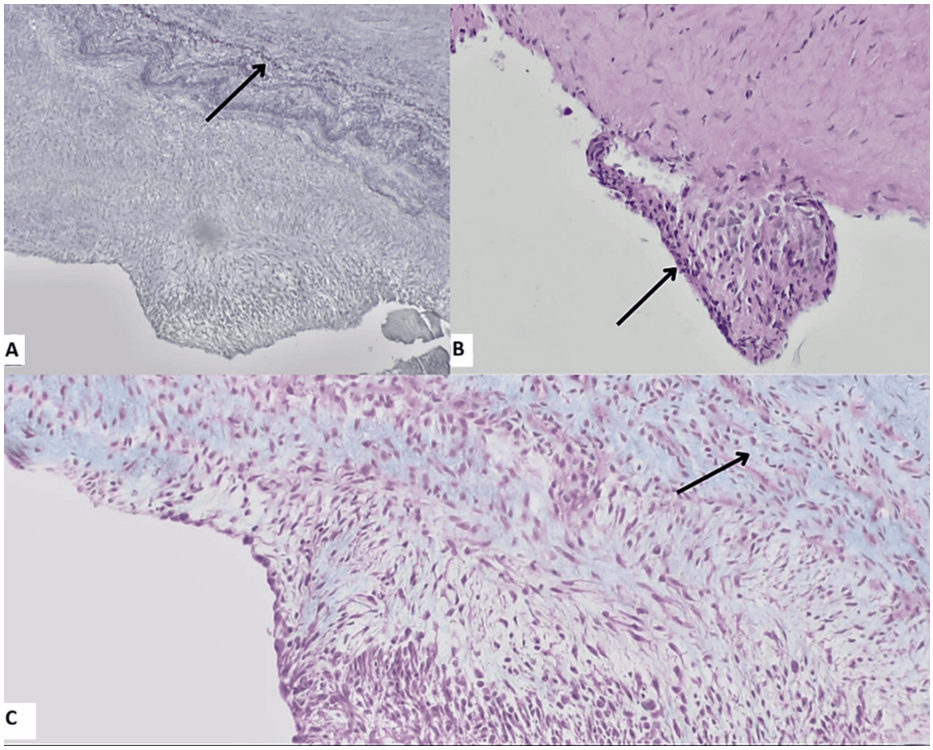


FIGURE 6: Pathology report: (A) elastic fiber technique: mitral valve areas with dense, fibrous-appearing tissue. (B) H&E technique: vegetation with surface deposits of fibrinoid. (C) Trichrome technique: fibrinoid tissue

H&E: hematoxylin and eosin

Postoperatively, the patient was admitted to the intensive care unit for three days. On postoperative day 2, the pericardial drains and pacemaker leads were removed. He then remained hospitalized for two additional days in the general ward before being discharged. There were no complications from the surgery or from the medications the patient received. At his most recent follow-up, the patient was in good condition and without symptoms.

Discussion

Our literature review and our presented case reports of Libman-Sacks endocarditis highlight important clinical patterns, surgical approaches, and outcomes. We searched the literature centering on surgical approaches: mitral valve replacement and mitral valve repair, with one of each used in our patients. The literature review summary, as shown in Table *1*, includes 10 cases of valve replacement and four cases of valve repair. Our cases presented in this report involve the etiology, the mitral valve disease, the presence of heart failure at the time of surgery, the imaging, the histopathology, the outcome, and the follow-up for each case. For the literature review, the following databases were searched: PubMed, Scopus, and Biblioteca Virtual en Salud. The time period of the literature search was from March 2010 to March 2025.

Reference	Gender /age (years)	SLE/APS/both	MR/MS/both	HF preop	Image (TEE)	Histopathology	Surgical procedure	Outcome	Follow-up
Senesael et al. [4]	F/30	SLE	Both	Yes	Severe MS (mean gradient of 6.1 mmHg, MVA 1.0 cm2), moderate MR, severe left atrial dilatation (48 mm, 59 mL/m^2), moderate systolic PHT (40 mmHg)	Fibrosis, angiogenesis, inflammatory cell infiltration, and calcification	Mechanical valve replacement	Stable	1 year
Al-Jehani et al. [5]	M/39	SLE	Both	Yes	Mild-to-moderate tricuspid regurgitation, severe MS with moderate-to-severe MR, thickened mitral valve leaflets with symmetrical mass-like structure involving the	Degenerative changes, myxoid areas with hemorrhage, acute and chronic inflammatory	Mechanical valve replacement	Stable, an episode of hyphema and	1 year

					tips of both leaflets, causing a restriction in the valve function	cells, and fibroid necrosis		choroidal hemorrhage	
Ichikawa et al. [6]	F/59	Both	MS	Yes	Left atrial volume index of 42 mL/m². MPG 18.6 mmHg, confirming severe MS. The mitral valve area was calculated as 1.02 cm². PHT (30 mmHg)	Not reported	Bioprosthetic valve replacement	Left middle cerebral artery infarction, due to APS, and died	6 months
Kotkar and Said [7]	F/61	APS	Both	Yes	Moderate-to-severe MR, severe MS; mean diastolic gradient of 12 mm Hg, and thickened, mobile vegetations on both anterior and posterior mitral valve leaflets	Nonbacterial thrombotic endocarditis with no evidence of microorganisms	Mechanical valve replacement	Stable	5 days
Bani Hani et al. [8]	F/37	APS	MR	Yes	1.9 × 2 cm mass on the posterior mitral leaflet, causing severe MR and EF of 65% with normal left ventricular size	Organized nodules of fibrin thrombus and granulation tissue	Mechanical valve replacement	Stable	Unknown
Nakasu et al. [9]	F/46	Both	None	Yes	Preserved EF of the left ventricle and a mobile vegetation measuring 9 × 6 mm^2 at the mitral valve	Fibrin thrombus without evidence of microorganisms	Mechanical valve replacement	Stable	6 months
Yamashita et al. [10]	F/62	Both	MR	Yes	Left ventricular EF of 72.1% and severe MR mainly due to systolic anterior motion of the mitral valve	Necrosis, fibrin, hyalinized collagen, and edematous/myxoid granulation	Mechanical valve replacement	Stable	17 days
Mizrahi et al. [11]	F/36	Both	MR	Yes	Severe MR, regurgitation fraction of 69%; the left ventricle was dilated and had an EF of 55%. The right ventricle and left atrium were dilated. PHT (51 mmHg)	Aseptic fibrinous-thrombotic vegetations	Mechanical valve replacement	Stable	10 months
Abouelela et al. [12]	M/32	Both	MR	Yes	Severe MR due to retracted leaflets with vegetation	Fibrinous exudate with neutrophils, lymphocytes and histiocytes, but no bacterial or fungal colonies	Mechanical valve replacement	Stable	1 year
Sladek and Accola [13]	M/36	APS	Both	Yes	Severely thickened bioprosthetic mitral valve with echogenic density attached to anterior leaflets. The pressure gradient was 45 mmHg with a valve area of 1.0 by pressure half-time, revealing severe MS and MR	Fibrinous exudate attached to the prosthetic valvular leaflets. Fungal, bacterial, and acid-fast bacilli staining negative	Mechanical valve replacement	Stable	Unknown
Samura et al. [14]	F/58	Both	MR	Yes	Thickening and poor coaptation of both mitral valve leaflets	Deposition of fibrin and small collections of neutrophils and monocytes, but no bacterial colonies	Valve repair	Stable, but 1 year later needed a reoperation for severe MR and severe MS	1 year
Bouma et al. [15]	M/49	SLE	MR	No	Severe MR with thickened mitral valve leaflets and a small vegetation on the posterior mitral valve leaflet	No reported	Valve repair	Stable	11 years
Gur et al. [16]	F/20	SLE	MS	No	A mobile mass on both the anterior and posterior leaflets of the tricuspid valve	Consistent with Libman-Sacks endocarditis	Valve repair	Stable	6 months
Perier et al. [17]	F/25	Both	MR	No	Small nodules on both cusps, as well as a thickening and shortening of the valves	Lesion homogeneously eosinophilic, absence of neutrophils	Valve repair	Stable	11 months

TABLE 1: Literature review of Libman-Sacks cases

F: female; M: male; SLE: systemic lupus erythematous; APS: antiphospholipidic syndrome; MR: mitral valve regurgitation; MS: mitral valve stenosis; HF: heart failure; EF: ejection fraction; Preop: preoperative; TEE: transesophageal echocardiogram; PHT: pulmonary hypertension; MPG: mean pressure gradient; MVA: mitral valve area

The pathogenesis of this disease is believed to be related to inflammatory and thrombotic mechanisms in the presence of antiphospholipid antibodies. Ziporen et al. showed deposits of antiphospholipid antibodies and complement components on deformed heart valves in patients with APS, isolated or associated with SLE. Another study detected the presence of anti-β2-glycoprotein I antibodies on deformed valves from a patient with APS; this could support a hypothesis of molecular mimicry as part of the valve disease in Libman-Sacks. Finally, it is well known of the association of antiphospholipid antibodies, especially Lupus Anticoagulant with hypercoagulability, which supports the thrombotic manifestations observed on heart valves. The pathogenesis of Libman-Sacks is believed to be caused by the secondary deposition of fibrin-platelet thrombi [1-3].

In our dataset, women constituted most patients, which aligns with the well-documented female predominance of SLE and APS [4-17]. Libman-Sacks endocarditis has been reported in 6%-11% of patients with SLE, and valvular involvement is present in approximately 33% of APS cases [18]. Interestingly, APS, either alone or in combination with SLE, was more frequently observed in our literature review, which was also the diagnosis in our two case reports, supporting evidence that antiphospholipid antibodies exacerbate the risk of valvular pathology [4-18]. An important difference between our literature review and our case reports was that both were male patients.

Regurgitation of the mitral valve was more common than stenosis; both regurgitation and stenosis were involved in four cases [4-17]. There was a particularly interesting case where the recommendation for surgery did not involve valvular dysfunction, but rather recurrent thromboembolic events [9]. This observation is consistent with prior reviews reporting mitral regurgitation as the most common functional abnormality, while stenosis remains rare [1]. Moreover, left-sided valve involvement, particularly the mitral, is recognized as the hallmark of Libman-Sacks endocarditis. Our two cases involved the mitral valve, although advanced imaging modalities, per the literature, have revealed cases of multivalvular disease [1].

Per the literature, clinical complications were significant in some patients, which included cerebral infarction, multiorgan failure, and ocular hemorrhage [4-17]. Our two case reports had thromboembolic complications. These findings echo published reports that thromboembolic events such as stroke, transient ischemic attacks, and systemic embolization are among the most serious manifestations of Libman-Sacks endocarditis, particularly in patients with APS secondary to the hypercoagulable state [3]. Most of the patients we found in the literature review presented with heart failure before surgery [4-17]. Although most valvular lesions are subclinical, approximately 4%-6% of APS patients may progress to severe cardiac dysfunction requiring surgical intervention [19]. In our case reports and per our literature review, the majority of patients received surgical management, either by valve replacement or by valve repair.

Surgical management in our literature dataset assessment was dominated by mechanical prosthesis valve replacement (nine cases), with only one bioprosthetic replacement and four cases of valve repair [4-17]. Our case reports had, respectively, one mechanical valve replacement and one valve repair. This reflects the literature, where valve replacement is favored over repair due to leaflet distortion, fibrosis, and vegetations that often preclude durable repair [20]. The choice of prosthesis remains debated. Mechanical valves offer durability in prothrombotic settings but require lifelong anticoagulation, with bleeding risks. In contrast, bioprosthetic valves avoid anticoagulation dependence but may degenerate more rapidly, especially in autoimmune and prothrombotic states. The aim of this paper is descriptive in nature, intended to describe the findings rather than establish the superiority of one surgical approach over the other. Larger studies are needed to draw that conclusion.

Conclusions

Libman-Sacks endocarditis should be considered in the differential diagnosis of valvular vegetations in patients with APS and/or SLE. In our series, mechanical valve replacement was the most frequent surgical intervention, reflecting the structural severity of the valvular lesions. Postoperative outcomes were generally favorable, but thromboembolic complications remained a significant risk.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Acquisition, analysis, or interpretation of data: Paula Espinosa-Villagomez, Santiago A. Endara, Gerardo A. Davalos, Marlon P. Aguirre, Patricia Pontón-Villalba, Daniel A. Larrea

Drafting of the manuscript: Paula Espinosa-Villagomez, Santiago A. Endara, Gerardo A. Davalos, Marlon P. Aguirre, Patricia Pontón-Villalba, Daniel A. Larrea

Critical review of the manuscript for important intellectual content: Paula Espinosa-Villagomez, Santiago A. Endara, Gerardo A. Davalos, Marlon P. Aguirre, Patricia Pontón-Villalba, Daniel A. Larrea

Supervision: Santiago A. Endara, Gerardo A. Davalos, Marlon P. Aguirre

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Zuily S, Huttin O, Mohamed S, Marie PY, Selton-Suty C, Wahl D: Valvular heart disease in antiphospholipid syndrome. Curr Rheumatol Rep. 2013, 15:320. 10.1007/s11926-013-0320-8
2. Ziporen L, Goldberg I, Arad M, et al.: Libman-Sacks endocarditis in the antiphospholipid syndrome: immunopathologic findings in deformed heart valves. Lupus. 1996, 5:196-205. 10.1177/096120339600500306
3. Lee JL, Naguwa SM, Cheema GS, Gershwin ME: Revisiting Libman-Sacks endocarditis: a historical review and update. Clin Rev Allergy Immunol. 2009, 36:126-30. 10.1007/s12016-008-8113-y
4. Senesael E, Plein D, La Meir M, Droogmans S: A case report of an unusual cause of mitral stenosis in a young woman. Eur Heart J Case Rep. 2018, 2:yty118. 10.1093/ehjcr/yty118
5. Al-Jehani M, Al-Husayni F, Almaqati A, Shahbaz J, Albugami S, Alameen W: A case of systemic lupus erythematosus in a patient presenting with Libman-Sacks endocarditis. Case Rep Cardiol. 2021, 2021:5573141. 10.1155/2021/5573141
6. Ichikawa-Ogura S, Mochizuki Y, Toyosaki E, Fukuoka H, Shinke T: Rapidly progressive mitral valve disease from non-bacterial thrombotic endocarditis to mitral stenosis in systemic lupus erythematosus: a case report. Eur Heart J Case Rep. 2025, 9:ytaf098. 10.1093/ehjcr/ytaf098
7. Kotkar KD, Said SM: Libman-Sacks endocarditis in a patient with antiphospholipid syndrome . Ann Thorac Surg. 2016, 102:e31-2. 10.1016/j.athoracsur.2015.11.004 External Link
8. Bani Hani A, Abu-Abeeleh M, Al Kharabsheh MM, Qabba'ah L: Libman-Sacks endocarditis with unusual large size vegetation involving the mitral valve. Heart Surg Forum. 2016, 19:E294-6. 10.1532/hsf.1612
9. Nakasu A, Ishimine T, Yasumoto H, Tengan T, Mototake H: Mitral valve replacement for Libman-Sacks endocarditis in a patient with antiphospholipid syndrome secondary to systemic lupus erythematosus. J Surg Case Rep. 2018, 2018:rjy069. 10.1093/jscr/rjy069
10. Yamashita G, Kanemitsu N, Nakashima Y, Matsuo T, Nakane T, Honda M, Okabayashi H: Hypertrophic obstructive cardiomyopathy and mitral regurgitation in Libman-Sacks endocarditis. Gen Thorac Cardiovasc Surg. 2020, 68:181-4. 10.1007/s11748-018-1042-7
11. Mizrahi M, Jimenez-Becerra S, Rivera-Navarrete E, Gomez-Sanchez M: Severe mitral valve regurgitation secondary to Libman-Sacks endocarditis. Am J Cardiovasc Dis. 2020, 10:382-5.
12. Abouelela Y, Gukop P, Livesey S: Surgical management of Libman-Sacks mitral valve endocarditis . Int J Surg Case Rep. 2021, 79:428-30. 10.1016/j.ijscr.2021.01.003
13. Sladek EH, Accola KD: Antiphospholipid syndrome and Libman-Sacks endocarditis in a bioprosthetic mitral valve. Ann Thorac Surg. 2016, 101:e29-31. 10.1016/j.athoracsur.2015.06.113
14. Samura T, Toda K, Yoshioka D, et al.: Libman-Sacks endocarditis due to systemic lupus erythematosus activation after mitral valve plasty. Ann Thorac Surg. 2017, 104:e109-11. 10.1016/j.athoracsur.2017.01.073
15. Bouma W, Klinkenberg TJ, van der Horst IC, et al.: Mitral valve surgery for mitral regurgitation caused by Libman-Sacks endocarditis: a report of four cases and a systematic review of the literature. J Cardiothorac Surg. 2010, 5:13. 10.1186/1749-8090-5-13
16. Gur AK, Odabasi D, Kunt AG, Kunt AS: Isolated tricuspid valve repair for Libman-Sacks endocarditis . Echocardiography. 2014, 31:E166-8. 10.1111/echo.12558
17. Perier P, Jeserich M, Vieth M, Pohle K, Hohenberger W, Diegeler A: Mitral valve reconstruction in a patient with Libman-Sacks endocarditis: a case report. J Heart Valve Dis. 2011, 20:103-6.
18. Yoo BW, Lee SW, Song JJ, Park YB, Jung SM: Clinical characteristics and long-term outcomes of Libman-Sacks endocarditis in patients with systemic lupus erythematosus. Lupus. 2020, 29:1115-20. 10.1177/0961203320930097
19. Eviatar T, Niznik S, Elkayam O, et al.: Heart valve surgery in antiphospholipid syndrome patients-morbidity and mortality. Life (Basel). 2023, 13:891. 10.3390/life13040891
20. Van Dyck M, Glineur D, de Kerchove L, El Khoury G: Complications after aortic valve repair and valve-sparing procedures. Ann Cardiothorac Surg. 2013, 2:130-9. 10.3978/j.issn.2225-319X.2012.12.03