ISSN: 3029-0732

Case Report

Journal of Cardiovascular and Cardiology

Embolic Stroke Caused by Non-Bacterial Thrombotic Endocarditis

Vinh Sieu Lam[1] and Pham Thao Vy Le[2*]

[1]*Cardiovascular Research, Methodist Hospital, Merrillville, Indiana, USA*

[2]*Ascension St. Joseph Hospital, Chicago, USA*

[*]**Corresponding author**

Pham Thao Vy Le, Ascension St. Joseph Hospital, Chicago, USA.

Received: September 05, 2025; **Accepted:** September 12, 2025; **Published:** September 19, 2025

ABSTRACT

Libman-Sacks endocarditis (LSE) is a form of nonbacterial thrombotic endocarditis (NBTE) observed in patients with malignancies, systemic lupus erythematosus (SLE), and antiphospholipid syndrome (APS), resulting from endothelial injury in a hypercoagulable state. The mitral and aortic valves are most commonly affected. Complications can include valvular injury, heart failure (HF), and widespread embolic events. We present a rare case of embolic stroke caused by NBTE in a 51-year-old man with no significant medical history. The patient presented to the emergency department with the sudden onset of slurred speech, numbness, and weakness in his right hand. Physical examination revealed Janeway lesions, splinter hemorrhages, and widespread livedo reticularis. Laboratory tests showed elevated anticardiolipin antibody (aCL-IgM), increased BNP and high-sensitivity troponin, thrombocytopenia (68,000/μL), and elevated homocysteine. Repeated blood cultures were negative. Computed tomography (CT) of the brain revealed chronic right posterior temporal, occipital, and left occipital infarctions. Magnetic resonance imaging (MRI) of the brain demonstrated numerous small foci of early subacute ischemia in both cerebral hemispheres and the left cerebellar hemisphere. A transthoracic echocardiogram (TTE) identified a 0.93 x 0.51 cm mobile echodensity near the right coronary cusp. Transesophageal echocardiography (TEE) confirmed verrucous and nodular vegetations with heterogeneous echodensity at the commissural border of both aortic valve leaflets, severe aortic regurgitation (AR), and reduced left ventricular ejection fraction (LVEF = 45–50%). The patient was diagnosed with an embolic stroke caused by LSE in the context of underlying APS. He was treated with low molecular weight heparin (LMWH), vancomycin, and piperacillin-tazobactam (Zosyn). Subsequently, he underwent mechanical aortic valve replacement followed by warfarin therapy (target INR 3-4). Intraoperative findings confirmed thickening and numerous tiny nodular vegetations on the aortic valve. This case highlights the importance of early diagnosis of embolic stroke caused by NBTE in young patients without significant medical history. Prompt and appropriate treatment, including antibiotics, anticoagulants, and surgery, is crucial to prevent serious complications.

Keywords: Libman-Sacks endocarditis, nonbacterial thrombotic endocarditis, primary antiphospholipid syndrome, embolic stroke, mechanical aortic valve replacement, anticoagulation

Introduction

Libman-Sacks endocarditis (LSE) is a form of nonbacterial thrombotic endocarditis that primarily affects the mitral and aortic valves. The most prevalent causes of LSE are primary antiphospholipid syndrome (APS), malignancies, and systemic lupus erythematosus (SLE). Diagnosis of LSE is challenging due to consistently negative blood cultures. Early detection of the disease and timely valve replacement surgery are crucial to prevent severe systemic embolism complications in the future [1-8]. This report presents a case of an embolic stroke caused by LSE in a patient with underlying APS. The patient underwent mechanical aortic valve replacement followed by anticoagulation therapy.

Case Presentation

A 51-year-old man with no significant medical history, except for a 30 pack-year smoking history and alcoholism, presented to the emergency room. He reported sudden onset of slurred speech, numbness, and weakness in his right hand. The patient was subsequently admitted to our hospital. Upon admission, an emergency brain computed tomography (CT) revealed chronic right posterior temporal, occipital, and left occipital infarctions. Physical examination was notable for the presence of Janeway lesions, splinter hemorrhages, widespread livedo reticularis, and cold, cyanotic extremities with diminished pulses (Figure 1).

Citation: Vinh Sieu Lam, Pham Thao Vy Le. Embolic Stroke Caused by Non-Bacterial Thrombotic Endocarditis . J Cardiovas Cardiol. 2025. 3(3): 1-4.
DOI: doi.org/10.61440/JCC.2025.v3.46

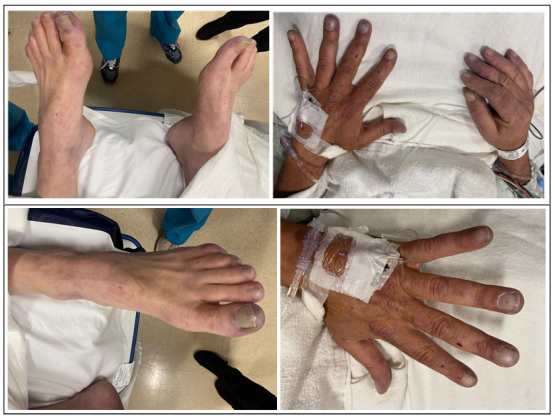

Figure 1: Skin examination showing Janeway lesions, splinter hemorrhages, widespread livedo reticularis, and cyanotic extremities

The electrocardiogram (ECG) showed a sinus rhythm at 78 beats per minute. Laboratory investigations revealed a normal level of anticardiolipin antibody aCL-IgG <10 CU (0.0–20.0 CU), and an elevated level of anticardiolipin antibody aCL-IgM at 18 CU (<12.5 CU). Platelet count was decreased at 63,000/mm³ (150,000-400,000/mm³). High-sensitivity troponin at 1 hour and 3 hour rest were elevated at 522 ng/L and 559 ng/L, respectively (<14 ng/L). Brain natriuretic peptide (BNP) was increased at 305 pg/mL (<100 pg/mL), and homocysteine levels were elevated at 15.9 µmol/L (5-15 µmol/L). The levels of red cell count, white cell count, prothrombin time (PT), partial thromboplastin time (PTT), international normalized ratio (INR), C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), creatinine, urea, and liver function tests were within normal limits.

Magnetic resonance imaging (MRI) of the brain revealed numerous small foci of early subacute brain ischemia in both cerebral hemispheres and the left cerebellar hemisphere, suggestive of an embolic source. Computed tomography angiography (CTA) of the head with and without contrast showed diminished perfusion involving the left posterior parietal lobe, while CTA of the neck with and without contrast was unremarkable.

Transthoracic echocardiogram (TTE) demonstrated a small mobile echodensity near the right coronary cusp, measuring 0.93 x 0.51 cm. Transesophageal echocardiography (TEE) further confirmed the presence of verrucous and nodular vegetations with heterogeneous echodensity at the commissural border of both aortic valve leaflets (Figure 2). The vegetation was tightly adhered to the valve's surface. Severe aortic regurgitation was detected at the central commissure during valve closure with a vena contracta of 0.45 cm, an effective regurgitant orifice area (EROA) of 0.32 cm², and a regurgitant volume of 85 mL. Holodiastolic reversal was observed in the descending thoracic aorta, with a velocity-time integral (VTI) of 14.3 cm. Mild mitral regurgitation and moderate left atrial dilatation were also present. The remaining valves were unremarkable. Left ventricular ejection fraction (LVEF) was reduced to 45-50%.

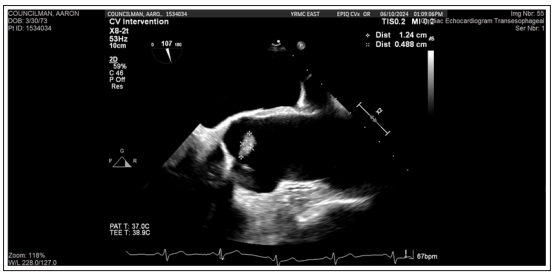

Figure 2: TEE demonstrating thickening and hyperechoic lesions of aortic valve leaflets, suggesting valvular vegetation.

Chest X-ray and chest CT were unremarkable. Repeated blood cultures were negative. Serological tests for HIV and hepatitis C were negative. The patient's family history was significant for his mother having hemolytic anemia and a history of splenectomy. Considering these results, the patient was diagnosed with primary antiphospholipid syndrome (APS), Libman-Sacks endocarditis (LSE), and non-infectious embolic stroke. Low molecular weight heparin was administered upon admission to address the manifesting symptoms. The patient was also started on vancomycin and piperacillin-tazobactam (Zosyn) to treat a potential infective source. The patient then underwent mechanical aortic valve replacement, which revealed thickening and numerous tiny nodular vegetations on the aortic valve. No penetration or damage of the aortic valve was detected (Figure 3).

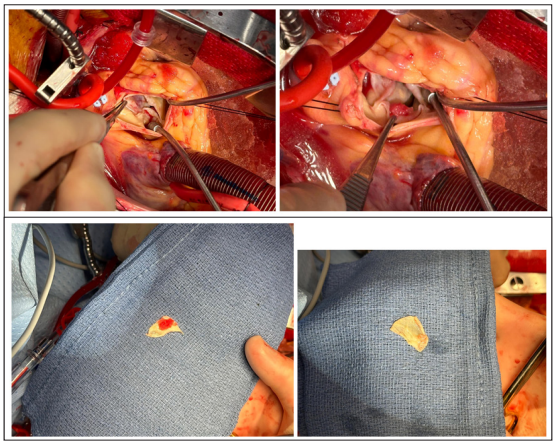

Figure 3: Intraoperative showing thickening and numerous tiny nodular vegetations on the aortic valve. No penetration or damage of the aortic valve was detected

Histopathology examination of the resected valve demonstrated fibrous tissue hyperplasia with hyaline degeneration and no inflammatory cell infiltration. After the surgery, oral warfarin was given with a targeted international normalized ratio (INR) of 3.0–4.0.

Discussion

Endocarditis is a condition characterized by endocardial inflammation, most commonly due to bacterial or fungal infection. Non-infectious causes, known as Libman-Sacks endocarditis (LSE), are rare and were first described as sterile vegetations on heart valves by Ziegler in 1888. Following this, Libman and Sacks presented a case series of four patients in 1924 in New York [9]. The mechanism of LSE development stems from endothelial injury in the setting of hypercoagulability, which is mainly observed in patients with malignancies,

systemic lupus erythematosus (SLE), and antiphospholipid syndrome (APS). The association between LSE and APS was first documented in 1985 in a young woman with SLE and positive lupus anticoagulant [10].

Diagnosing LSE is challenging due to its clinical presentation and imaging findings resembling those of bacterial endocarditis. In this case, LSE was diagnosed based on the vegetation discovered in the aortic valve via transesophageal echocardiography (TEE) and repeated blood cultures that yielded negative results. The patient had positive anticardiolipin IgM along with a family history of hemolytic anemia in the patient's mother, which led to the diagnosis of LSE caused by APS. Additionally, this patient exhibited other manifestations of APS, notably thrombocytopenia and elevated homocysteine levels. The causes of thrombocytopenia in patients with APS could be explained by depletion of platelets at the site of embolism; increasing activation and aggregation of platelets; increasing platelet pool caused by splenomegaly; and decreasing platelet production due to hemophagocytic syndrome [11]. Regarding hyperhomocysteinemia (HHS), the causes in patients with APS are not fully understood but have been documented to be associated with venous thromboembolism, cardiovascular, and cerebrovascular disease; and can worsen thrombotic tendency in patients with APS [12].

LSE is a pathogenic factor that contributes to the development of cerebrovascular disease (CVD) [14], and is associated with the presence of "white thrombi" on the cardiac valves [15]. These thrombi are characterized by blood clots mixed with fibrin fibers, antibodies, and mononuclear cells. The restricted inflammation that occurs where these white clots adhere makes them vulnerable to disassociation, potentially causing arterial embolism, which accounts for 59.5% of incidents [16]. In this case, brain MRI revealed small foci of early subacute brain ischemia in both cerebral hemispheres and the left cerebellar hemisphere, suggestive of embolic origins.
These emboli likely originated from the aortic valve vegetation confirmed by TEE.

In LSE, the mitral valve and aortic valve are the most frequently affected [13]. Prompt and appropriate treatment, including antibiotics, anticoagulants, and surgery, is crucial to prevent serious complications. Surgical valve replacement is preferred in young patients with LSE who have significant valvular injury, diminished LVEF, and cerebral embolism to prevent decompensation progression and lower the risk of future embolic events. Furthermore, anticoagulation plays a critical role in the management of LSE [17], although the risk of hemorrhagic transformation should be taken into account [18]. Brain CT scans ruling out cerebral bleeding should be performed prior to initiating anticoagulation. Regarding choices for anticoagulation, warfarin is preferable to direct oral anticoagulants (DOACs) for maintenance in cases of patients undergoing mechanical valve replacement. Additionally, DOACs should be avoided if APS is suspected [19]. Anticoagulation should be continued permanently unless life-threatening hemorrhages occur [20]. Underlying illnesses (cancer, lupus, APS) also require proper diagnosis and management.

Conclusions

In conclusion, our case highlighted an uncommon cause of embolic stroke in a young patient with no major past medical history caused by non-bacterial thrombotic endocarditis. The patient's significant complications and poor outcomes were caused by severe valve injury, diminished heart function, widespread dermatologic manifestations, and acute cerebrovascular disease. Causes for non-infectious endocarditis should be considered, especially if patients have an underlying autoimmune disorder or hypercoagulable states such as cancer, SLE, or APS. Because of the difficulty in recognizing and managing the disease's uncommon etiology, as well as the requirement of valve surgery, a multidisciplinary approach is required for optimal patient outcomes.

References

1. Ibrahim AM, Siddique MS. Libman-Sacks Endocarditis. Treasure Island. (FL): StatPearls Publishing. 2024.
2. Malvar B, Almeida FM, Rebocho L, Moniz JC, Azevedo F. Cerebral embolism from Libman-Sacks endocarditis. BMJ Case Rep. 2011. 0420114071.
3. Scalia GM, Tandon AK, Robertson JA. Stroke, aortic vegetations and disseminated adenocarcinoma--a case of marantic endocarditis. Heart Lung Circ. 2012. 21: 234-236.
4. Liang H, Ma C, Chen X. Case report: Mitral valve replacement for Libman-Sacks endocarditis and cerebral embolism of primary antiphospholipid syndrome. Front Cardiovasc Med. 2022 9: 985111.
5. Yazidi MA, Merzouk FZ, Rabii H, Benyoussef H, Bensahi I, et al. Ischemic Stroke Revealing Libman-Sacks Endocarditis: A Case Report. J Saudi Heart Assoc. 2023. 35: 335-338.
6. Yazidi MA, Merzouk FZ, Rabii H, Benyoussef H, Bensahi I, et al. Ischemic Stroke Revealing Libman-Sacks Endocarditis: A Case Report. J Saudi Heart Assoc. 2023. 35: 335-338.
7. Hachiya K, Wakami K, Tani T, Yoshida A, Suzuki S, et al. Double-valve replacement for mitral and aortic regurgitation in a Patient with Libman-Sacks endocarditis. Intern Med. 2014. 53: 1769-73.
8. Erdozain JG, Ruiz-Irastorza G, Segura MI, Amigo MC, Espinosa G, et al. Cardiac valve replacement in patients with antiphospholipid syndrome. Arthritis Care Res (Hoboken). 2012. 64:1256-1260.
9. Hurrell H, Roberts-Thomson R, Prendergast BD. Non-infective endocarditis. Heart. 2020. 106:1023-1029.
10. D'Alton JG, Preston DN, Bormanis J, Green MS, Kraag GR. Multiple transient ischemic attacks, lupus anticoagulant and verrucous endocarditis. Stroke. 1985. 16: 512-514.
11. Tomasello R, Giordano G, Romano F, Vaccarino F, Siragusa S, et al. Immune Thrombocytopenia in Antiphospholipid Syndrome: Is It Primary or Secondary? Biomedicines. 2021. 9: 1170.
12. Richard M Lee, Melissa A Brown, Kenneth Ward, Lesa Nelson, Ware Branch D, et al. Homocysteine levels in women with antiphospholipid syndrome and normal fertile controls, Journal of Reproductive Immunology. 2004. 23-30.
13. Zuily S, Huttin O, Mohamed S, Marie PY, Selton-Suty C, et al. Valvular heart disease in antiphospholipid syndrome. Curr Rheumatol Rep. 2013. 15: 320.

14. Roldan CA, Sibbitt WL Jr, Qualls CR, Jung RE, Greene ER, et al. Libman-Sacks endocarditis and embolic cerebrovascular disease. JACC Cardiovasc Imaging. 2013. 6: 973-983.
15. Andrea Colli, Carlos A Mestres, Gerard Espinosa, Miguel A Plasín, Jose L Pomar, et al. Heart valve surgery in patients with the antiphospholipid syndrome: analysis of a series of nine cases, European Journal of Cardio-Thoracic Surgery. 2010. 37: 154-158.
16. Roldan CA, Sibbitt WL Jr, Qualls CR, Jung RE, Greene ER, et al. Libman-Sacks endocarditis and embolic cerebrovascular disease. JACC Cardiovasc Imaging. 2013. 6: 973-983.
17. el-Shami K, Griffiths E, Streiff M. Nonbacterial thrombotic endocarditis in cancer patients: pathogenesis, diagnosis, and treatment. Oncologist. 2007. 12: 518-523.
18. Andrea Colli, Carlos A Mestres, Gerard Espinosa, Miguel A Plasín, Jose L Pomar, et al. Heart valve surgery in patients with the antiphospholipid syndrome: analysis of a series of nine cases, European Journal of Cardio-Thoracic Surgery. 2010. 37: 154.
19. Capecchi M, Abbattista M, Ciavarella A, Uhr M, Novembrino C, et al. Anticoagulant Therapy in Patients with Antiphospholipid Syndrome. J Clin Med. 2022. 11: 6984.
20. Rogers LR, Cho ES, Kempin S, Posner JB. Cerebral infarction from non-bacterial thrombotic endocarditis. Clinical and pathological study including the effects of anticoagulation. Am J Med. 1987. 83: 746-756.