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Case Report

Antiphospholipid Syndrome Patient with Libman-Sacks Endocarditis

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ABSTRACT

Antiphospholipid syndrome (APS) is a systemic autoimmune disorder characterized by recurrent arterial and venous thrombosis, often associated with complications such as pregnancy morbidity. Libman-Sacks Endocarditis (LSE), a form of nonbacterial thrombotic endocarditis, is commonly observed in APS patients. This case report presents a 29-year-old Javanese woman with APS and Libman-Sacks Endocarditis. The patient, previously diagnosed with a transient ischemic attack, was admitted to Dr. Soetomo General Hospital with progressive shortness of breath, leg swelling, and abdominal distension. Initial investigations revealed thrombocytopenia, anemia, pleural effusion, cardiomegaly, and valve abnormalities. Despite anticoagulant therapy and supportive measures, the patient's condition worsened, and she developed signs of heart failure and neurological deficits. Blood cultures remained negative, ruling out bacterial endocarditis. Imaging studies confirmed the presence of sterile vegetations on the mitral valve, a hallmark of LSE. The patient was diagnosed with primary APS based on clinical and laboratory findings, including positive lupus anticoagulant. After 28 days of hospitalization, she was discharged in stable condition but was re-admitted two days later with neurological decline. Despite aggressive management, including corticosteroids, diuretics, and anticoagulants, the patient suffered multiple complications, including seizures and possible thrombotic stroke and eventually passed away. This case underscores the diagnostic challenges and complexity in managing APS with Libman-Sacks Endocarditis, highlighting the need for early diagnosis and comprehensive treatment. The objective of this case report is to highlight the diagnostic challenges and management complexities of APS with Libman-Sacks Endocarditis, emphasizing the importance of early recognition and comprehensive treatment to mitigate associated morbidity and mortality. LSE remains a rare but severe manifestation of APS, with potential for thromboembolic events and significant morbidity.

Keywords: Antiphospholipid syndrome, Libman Sacks disease, Libman Sacks endocarditis, Lupus anticoagulant.

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INTRODUCTION

Antiphospholipid syndrome (APS) is a systemic autoimmune disease characterized by recurrent arterial and venous thrombosis and/or pregnancy morbidity. It can also affect heart valves, leading to valve thickening and sterile vegetations known as Libman-Sacks Endocarditis. Most APS events are directly related to thrombotic incidents that can affect small, medium, or large blood vessels. Other clinical features such as thrombocytopenia, nephropathy, heart valve disease, cognitive dysfunction, and skin ulcers contribute significantly to the morbidity of this syndrome and represent challenging clinical situations. APS was initially described in patients with systemic lupus erythematosus (SLE) but can also occur in patients without autoimmune disease. Despite the autoimmune nature of this syndrome, APS management remains based on anticoagulant and antiplatelet therapies.[1]

Libman-Sacks Endocarditis, also referred to as nonbacterial thrombotic endocarditis (NTBE), was first described in 1924 by Libman and Sacks.[2] APS is a rare but clinically significant condition, with a global prevalence estimated to range between 40 to 50 cases per 100,000 people, predominantly affecting women, particularly during their reproductive years.[3] The condition poses serious health risks due to its association with both arterial and venous thrombotic events. Regarding cardiac involvement, LSE is present in approximately 33% of APS patients both primary or secondary (SLE).[4] Libman-Sacks Endocarditis is characterized by sterile verrucous lesions that predominantly affect the aortic and mitral valves. In most cases, patients do not experience significant valvular dysfunction. However, those with substantial valve dysfunction may present with serious complications such as heart failure, arrhythmias, and thromboembolic events. More recently, an association between Libman-Sacks Endocarditis and antiphospholipid antibody syndrome (APS) has been identified. APS is most commonly defined by venous and arterial thrombosis, recurrent miscarriages, and thrombocytopenia. Murtaza et al. (2017) explained that APS can either be a primary syndrome or secondary to SLE.[5] Environmental factors like industrial materials, ultraviolet B exposure, smoking, oral contraceptives, infections, and toxins can trigger the pathogenesis of SLE.[6]

Sacks Endocarditis being the most common valvular abnormality. The prevalence of valvular abnormalities reaches 35% in SLE patients and up to 48% in those with concurrent APS.[7]

In this case report, we discuss a rare case of a young female patient with Antiphospholipid Syndrome and Libman-Sacks Endocarditis.

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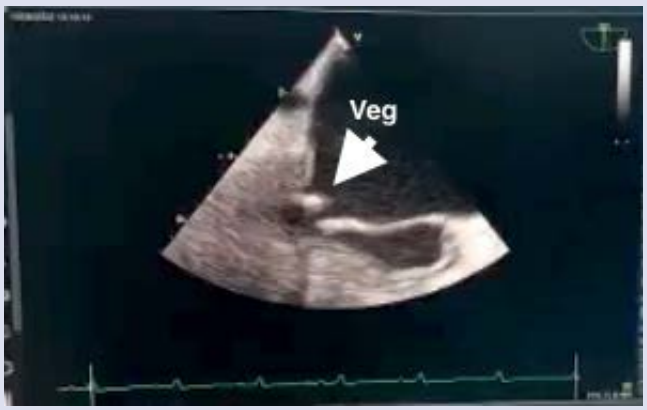

Figure 1. The image shows cardiac mitral valve vegetation observed during TEE.

CASE REPORT

A 29-year-old Javanese woman, a Muslim factory worker living in Surabaya, was admitted to Dr. Soetomo General Hospital (RSDS) on August 17, 2022.

History of Present Illness

The patient presented at the emergency room (ER) of RSDS with complaints of shortness of breath, which had persisted for a week before admission. Initially, she was not severely affected, but her breathing difficulties worsened with light activities, such as walking. The symptoms were accompanied by swelling in both legs and increasing abdominal distention. Initially, only the left leg was swollen, but over the past week, both legs had become more swollen. The patient had previously experienced a transient ischemic attack (TIA) stroke in February 2022, resulting in weakness in the left side of her body. She was treated at the Army Hospital, receiving blood-thinning medications and undergoing physical therapy twice a week, which improved her muscle strength, though numbness persisted. The patient also experienced continuous menstruation and was referred to the gynecology department, where she was prescribed hormonal medication. After taking the medication, the patient felt weak, shaky, and cold, leading to a return visit to the Army Hospital's emergency room. She received a transfusion of 2 units of packed red blood cells (PRC) due to a hemoglobin level of 7 g/dL. After being discharged, the swelling in her legs worsened, and she experienced increased shortness of breath. She was prescribed furosemide but saw no significant improvement. In July 2022, she sought treatment at the William Booth Hospital emergency room and was hospitalized for three days. A referral to the cardiology clinic at RSDS was given due to valve abnormalities discovered during an examination, which indicated the potential need for surgery. She visited the cardiology clinic at RSDS three times before her symptoms worsened. On August 17, 2022, due to increasing shortness of breath and swelling, the patient was brought to the RSDS emergency room by her family. She was admitted to the cardiology department, and after a suspicion of autoimmune disease, she was referred to the internal medicine department on the fourth day of hospitalization.

Past Medical History

The patient denied experiencing similar conditions previously, as well as a history of miscarriage or autoimmune disease. She also denied having hypertension or diabetes mellitus.

Family History

The patient reported no family history of similar illnesses, autoimmune diseases, diabetes, hypertension, or miscarriage.

Psychosocial History

The patient had stopped working after suffering from a stroke in February 2022. Before that, she worked in a speaker factory for 11 years, in a closed, air-conditioned environment. She reported a metallic odor after returning home from work. The patient has a 6-year-old child and is insured under the BPJS healthcare system. She denied any history of drug use, smoking, or alcohol consumption.

Physical Examination

The general condition of the patient was fair, with a Glasgow Coma Scale (GCS) score of E4M6V5. Blood pressure was 102/60 mmHg, pulse rate was 92 beats per minute with a regular rhythm and normal amplitude. Respiratory rate was 24 breaths per minute, axillary temperature was 36.6°C, and oxygen saturation was 98% without supplemental oxygen. The pain scale was 2/10. The patient weighed 58 kg, height was 160 cm, and her BMI was 22.7, indicating normal nutritional status.

Head and neck examination revealed slight conjunctival pallor and no jaundice. Jugular venous pressure was elevated. The thoracic examination showed symmetric movement without intercostal or supraclavicular retractions. The liver-diaphragmatic border was at the 6th intercostal space, midclavicular line on the right. Cardiac examination revealed a single S1 and S2 heart sound, with a grade II/IV systolic murmur at the 5th intercostal space, midclavicular line. Lung examination revealed vesicular breath sounds in both hemithoraxes, without wheezing or rales. Abdominal examination showed distention and ascites. Extremities were warm and dry, with pitting edema in both lower extremities. A central venous catheter was placed in the right femoral vein.

Investigations

During the initial examination at the emergency room of Dr. Soetomo General Hospital on August 17, 2022, the following tests were conducted: COVID-19 Rapid Test: Non-reactive, Random Blood Sugar: 98 mg/dL, HbsAg: Non-reactive, Anti-HCV: Non-reactive, White Blood Cell Count (WBC): 8.78 x 10^3/µL, Hemoglobin (Hb): 12.5 g/dL, Platelets (PLT): 114 x 10^3/µL, Arterial Blood Gas: pH 7.47, pCO_2 26.0 mmHg, pO_2 90.0 mmHg, HCO_3 18.9 mmol/L, TCO_2 19.7 mmol/L, BEecf -4.8 mmol/L, SO_2 96%.

Further tests on August 20, 2022, showed: Potassium: 4.3 mg/dL, Sodium: 127.0 mg/dL, Chloride: 92.0 mg/dL, Blood Urea Nitrogen (BUN): 24.1 mg/dL, Serum Creatinine: 1.152 mg/dL, SGOT: 43.7 U/L, SGPT: 33.7 U/L, Direct Bilirubin: 4 mg/dL, Total Bilirubin: 7.7 mg/dL, ANA Test (ELISA): Negative, 18.19 AU/mL, C4: 7.38 mg/dL, C3: 53.5 mg/dL. On August 22, 2022: Albumin: 3.39 mg/dL, Procalcitonin: 0.15 ng/mL.

Chest X-ray (August 17, 2022): Adequate inspiration, right heart border obscured by pleural effusion, bilateral pleural effusion (right side more prominent), and cardiomegaly.

ECG (August 17, 2022): Sinus tachycardia at 103 beats per minute, *right axis deviation, counterclockwise rotation, and right ventricular hypertrophy.*

CT Angiography (Thorax, August 18, 2022): *Axial, sagittal, and coronal* reformatted images with and without contrast showed cardiomegaly with right atrial and ventricular hypertrophy, dilation of the superior and inferior vena cava, right pleural effusion with *compressive atelectasis* of the laterobasal segment of the right lower lung, and ascites. No thrombus, *aneurysm,* or *vascular malformation* was detected.

CT Pulmonary Angiography (August 18, 2022): Right ventricular hypertrophy, right atrial dilation, dilation of the superior and inferior vena cava, right pleural effusion with compressive atelectasis in the laterobasal segment of the right lower lung, and ascites.

Table 1. Comparison of Libman-Sacks Endocarditis and Bacterial Endocarditis.

Aspect	Libman-Sacks Endocarditis	Bacterial Endocarditis
Clinical Presentation	– Other active SLE symptoms such as joint pain, hair loss, alopecia, oral lesions, and photosensitive rash. – Clinical features of APS, including recurrent miscarriages or other thrombotic events. – May have fever in active SLE cases, but Libman-Sacks Endocarditis is often asymptomatic; valve dysfunction, such as mitral regurgitation, may occur, leading to heart failure symptoms.	– Fever – Weakness – Acute onset – Symptoms of acute heart failure, such as pulmonary edema – Immunological and septic embolic phenomena like Roth's spots, Osler's nodes, and Janeway lesions.
Diagnostic Approach	– Supported by the presence of antiphospholipid antibodies in SLE patients. – Valve mass or thickening on echocardiogram – Negative blood cultures, indicating sterile vegetations.	– Based on modified Duke's criteria – Positive blood cultures from at least two separate samples – Elevated infection markers like leukocytosis, CRP, and procalcitonin. – Echocardiography findings: The presence of vegetations on echocardiogram, along with clinical signs and blood test results indicating infection, supports the diagnosis
Management	– Lifelong anticoagulation with warfarin, with bridging therapy using low-molecular-weight heparin. – Surgical intervention may be required in cases of acute heart failure, severe valve dysfunction, or large vegetations.	– Antibiotic therapy is essential in bacterial endocarditis cases – Surgery may be needed for valve dysfunction leading to heart failure, uncontrolled endocardial infection, or to prevent systemic embolization, especially to the brain.

Doppler Ultrasound of the Lower Extremities (August 18, 2022): No deep vein thrombosis was detected in the deep veins of the right and left lower extremities.

Abdominal Ultrasound (August 23, 2022): Ascites detected, with no abnormalities in the liver, gallbladder, spleen, pancreas, kidneys, bladder, uterus, or adnexa.

Initial Diagnosis

The patient was initially diagnosed with suspected Antiphospholipid Syndrome (APS) and suspected Libman-Sacks Endocarditis, with a differential diagnosis of bacterial endocarditis and hyperbilirubinemia for further evaluation.

Treatment Plan

Diagnostic Plan: Further testing was planned, including IgM ACA, LAI/LA2, IgM anti-beta 2 Glycoprotein, and blood cultures.

Therapeutic Plan: High-protein, high-calorie diet (2100 kcal/day), Fluid restriction: 500 mL/day, Intravenous furosemide 40 mg once daily, Spironolactone 50 mg once daily by mouth, Ursodeoxycholic acid (UDCA) 1 tablet, three times daily by mouth, Intravenous methylprednisolone 1 mg/kg body weight after laboratory samples were taken

Monitoring Plan: The patient was monitored for diagnosis confirmation of APS and the management of endocarditis.

Course of Illness

On the sixth day of hospitalization (August 23, 2022), the patient felt weak. Laboratory tests showed the following results: Serum creatinine: 1.04 mg/dL, WBC: 9.21 x 10^3/µL, RBC: 4.96 x 10^6/µL, Hemoglobin: 11.9 g/dL, Platelets: 165 x 10^3/µL, Basophils: 0.5%, Neutrophils: 71.3%, Lymphocytes: 19.5%, Eosinophils: 0.8%, Monocytes: 7.9%. Urinalysis revealed: Glucose: Negative, Bilirubin: Negative, Ketones: Negative, Specific gravity: 1.017, Blood: 3+, pH: 5.5, Protein: 1+, Urobilinogen: Normal, Nitrite: Negative, Leukocytes: Negative, Color: Red, Clarity: Clear. Albumin-Creatinine ratio: >300 mg/g Cr, Protein-Creatinine ratio: >0.50 g/g Cr. The patient had undergone an abdominal ultrasound and awaited the results.

On the seventh day of hospitalization (August 24, 2022), the patient continued to suffer from shortness of breath. Treatment involved oxygen via nasal cannula at 3 L/min, fluid intake restricted to 1000 mL per day, and intravenous administration of ampicillin 3 grams four times daily (Day 5), gentamicin 160 mg once daily (Day 5), and furosemide 40 mg three times daily. Additionally, the patient was given spironolactone 50 mg daily, rivaroxaban 15 mg twice daily (Day 5), bisoprolol 1.25 mg daily (postponed), and oral medications such as ursodeoxycholic acid (UDCA), N-acetylcysteine (NAC), and Vipalbumin, alongside daily intravenous methylprednisolone 62.5 mg.

On the eighth day (August 25, 2022), treatment was intensified with the addition of a Lasix pump at 10 mg/hour and a Dobutamine pump at 10 mcg/kg/min. Oral hydrochlorothiazide (HCT) 50 mg was also introduced once daily.

On the ninth day (August 26, 2022), the patient reported feeling physically lighter. The Dobutamine pump dose was reduced to 5 mcg/kg/min, while the Lasix pump continued.

On the tenth day (August 27, 2022), despite reduced numbness, the patient still felt weak. Fluid balance showed a 24-hour input of 1000 mL and an output of -7200 mL, resulting in a negative balance of -6200 mL. Blood tests for ACA IgM, Anti-Beta 2 Glycoprotein 1 IgG, and IgM were negative. Lupus anticoagulant tests indicated elevated DRVV screen times and positive results for lupus anticoagulant. Electrolyte results showed low potassium (2.2 mg/dL), sodium (128.0 mg/dL), chloride (71.0 mg/dL), and BUN (24.3 mg/dL). The patient's blood counts were notable for a WBC of 11.24 x 10^3/µL and hemoglobin of 10.8 g/dL. Antiphospholipid Syndrome was diagnosed, and rivaroxaban therapy was suspended.

On the eleventh day of hospitalization (August 28, 2022), laboratory tests revealed potassium levels at 2.9 mg/dL, sodium at 124.0 mg/dL, chloride at 66.0 mg/dL, procalcitonin at 0.07 ng/mL, and C-reactive protein (CRP) at 0.78 mg/dL. The patient's fluid balance showed an intake of 1300 mL and an output of -4000 mL, resulting in a negative balance of -2700 mL. Blood cultures were negative for aerobic, anaerobic, or fungal organisms. Additional treatment included a KCl premix (50 mEq in 100 mL NaCl 0.9%), while Dobutamine and Lasix pumps were discontinued and intravenous furosemide (3x20 mg) was administered. The patient was scheduled for an echocardiogram.

On the fifteenth day (September 1, 2022), the patient's shortness of breath improved, allowing her to sleep while lying down. Fluid balance indicated an intake of 800 mL and an output of 3150 mL, with a negative balance of -2350 mL. Laboratory results showed electrolyte imbalances, with elevated potassium (5.5 mg/dL) and sodium (116.0 mg/dL) levels, as well as an increased BUN (36.6 mg/dL). Methylprednisolone was reduced to 32 mg daily, and antibiotics were stopped due to negative culture results.

On the sixteenth day (September 2, 2022), the patient experienced a brief seizure (<5 minutes). A CT scan with contrast and an EEG were planned. Neurology prescribed 3% NaCl, phenytoin, folic acid, and vitamin B6.

On the seventeenth day (September 3, 2022), an MSCT head scan revealed a subacute to chronic infarction in the right thalamus, with no signs of hemorrhage, infection, or masses..

On the nineteenth day of hospitalization (September 5, 2022), the patient's shortness of breath improved, but she felt discomfort with the oxygen mask. Fluid balance was neutral, with both input and output at 1500 mL. The patient's INR was 4.65, prompting an adjustment in warfarin therapy. Gentamicin was discontinued due to concerns about nephrotoxicity and negative blood cultures.

On the twentieth day (September 6, 2022), the patient's shortness of breath returned, requiring oxygen at 12 L/min via a non-rebreather mask (NRM).

On the twenty-eighth day (September 14, 2022), the patient's clinical condition had improved, and she was discharged for outpatient care, scheduled to follow up with internal medicine and cardiology after three days. However, two days after being home, she became incoherent, agitated, and developed a fever. Her family brought her back to the emergency room, where it was reported that she had no shortness of breath or seizures, though her appetite had decreased. Her physical exam showed no chest pain or swelling in the legs. The patient's condition deteriorated rapidly, leading to a suspected thrombotic stroke or autoimmune encephalitis. An MRI with contrast was planned, but her condition worsened, leading to loss of consciousness and respiratory failure. The patient was intubated, sedated, and managed with a ventilator under anesthesia care. After four days without improvement, the patient's condition further deteriorated, and she passed away. An MRI with contrast was not performed due to the patient's instability, and no autopsy was conducted due to cultural and religious reasons.

DISCUSSION

The diagnosis of Antiphospholipid Syndrome (APS) requires both clinical and laboratory criteria, based on the International Consensus in Sapporo, Japan, on October 10, 1998[8] Clinically, vascular thrombosis and pregnancy history are key indicators, as demonstrated by Dwi Aninnaimah and Awalia (2022).[9] Laboratory criteria include the presence of one or more antiphospholipid antibodies (anticardiolipin, lupus anticoagulant, or anti-beta2 glycoprotein) measured at least 12 weeks apart. These findings, combined with clinical signs of vascular thrombosis or fetal death, form the basis for diagnosing APS.[10]

The patient in this case was diagnosed with primary APS based on anamnesis, physical examination, and diagnostic tests. A history of stroke in February indicated thrombosis, but there was no history of recurrent miscarriages, and the criteria for systemic lupus erythematosus (SLE) were not met. The laboratory findings of lupus anticoagulant detection supported the diagnosis of primary APS. Although certain studies (Gunawan, Awalia & Soeroso, 2018) suggest that C3 and C4 may not always be elevated in non-flaring SLE cases, this patient's laboratory results indicated primary APS.[11]

Libman-Sacks Endocarditis, or nonbacterial thrombotic endocarditis (NBTE), involves sterile vegetations on heart valves, often associated with malignancy, SLE, or APS. It typically affects the mitral and aortic valves and is rare, occurring in 0.9% to 1.6% of postmortem cases. It mostly impacts individuals aged 40-80, though it can occur in other age groups. Gender distribution is equal, but SLE and APS are much more common in women of childbearing age, particularly among Black and Hispanic women.[12]

Before diagnosing Libman-Sacks Endocarditis, bacterial endocarditis must be ruled out using Duke's modified criteria, which include two major or one major and three minor, or five minor criteria.[13]

In this case, vegetation was found on the mitral valve, fulfilling one major criterion. However, blood cultures were negative, and no minor criteria were met. The diagnosis of APS and Libman-Sacks Endocarditis was confirmed after excluding bacterial endocarditis. This case emphasizes the difficulty of distinguishing between Libman-Sacks Endocarditis and bacterial endocarditis, as antibiotics are critical for bacterial endocarditis management. Early consultation with a cardiologist is crucial when suspicious lesions are detected via echocardiogram to ensure appropriate intervention.

Table 1 provides a comparison of the main features of Libman-Sacks Endocarditis and bacterial endocarditis in terms of clinical presentation, diagnostic approach, and management.

Management of Libman-Sacks Endocarditis lacks a definitive gold standard, primarily due to the rarity of untreated cases. However, treatment of underlying conditions (SLE or APS) is necessary. Anticoagulants are recommended for secondary prevention of thromboembolism in patients with prior thrombotic events. Warfarin, with a target INR of 2-3, is the standard for APS patients diagnosed after their first thrombotic event. For significant valve dysfunction, surgery should follow guidelines for valve disease. Patients must be closely monitored due to the risk of recurrent thromboembolism, even with anticoagulation therapy. Scheduled echocardiograms (every 3-6 months) are advised to track disease progression or resolution.[14]

In Libman-Sacks Endocarditis, lifelong anticoagulation with warfarin or low-molecular-weight heparin is standard, especially for APS patients with a history of thrombosis.[15]

Surgical intervention is similar to bacterial endocarditis, including valve replacement in cases of severe dysfunction, heart failure, or large vegetations. Unlike bacterial endocarditis, complete removal of infected tissue may not be necessary. Postoperative anticoagulation remains essential for preventing systemic embolization, particularly in APS patients.[16]

A comprehensive six-year study revealed several significant findings related to Libman-Sacks Endocarditis in patients with neuropsychiatric SLE (NPSLE), including an increased risk of cerebral microemboli, cognitive dysfunction, and stroke. Vegetations were identified as independent risk factors for these outcomes. Anti-inflammatory and antithrombotic therapies improved cognitive function and cerebral perfusion. Patients with vegetations had a poor prognosis, with shorter event-free survival for cerebrovascular incidents, cognitive decline, or death.[17] A study of 76 SLE patients further confirmed the correlation between Libman-Sacks Endocarditis and increased risk of embolic cerebrovascular disease.[12]

Libman-Sacks Endocarditis is often diagnosed postmortem due to its asymptomatic nature. Early clinical suspicion is essential for diagnosis, and advances in echocardiography allow for earlier detection.[18,19]

Thrombotic phenomena typically accompany the condition, leading to significant complications such as cerebrovascular embolism and systemic thromboembolism. Heart failure due to valve dysfunction is also common, with symptoms like fatigue, fever, night sweats, and weight loss.[5]

A prior ischemic stroke was confirmed by CT. During emergency care, the patient's condition worsened, and she was unable to undergo a contrast-enhanced brain MRI due to being on a ventilator. The exact cause of her decline, whether recurrent thrombotic stroke or autoimmune encephalitis, remains unclear as an autopsy was not performed due to cultural and religious considerations in Indonesia.

CONCLUSIONS

The patient, diagnosed with Antiphospholipid Syndrome (APS) and Libman-Sacks Endocarditis, experienced acute heart failure and vascular disease. Vegetations were observed during TEE, and a prior ischemic stroke was confirmed by CT. Her condition worsened in the emergency room, but she could not undergo further diagnostics, and the cause of her decline remains unclear due to the inability to perform an autopsy, influenced by cultural and religious reasons in Indonesia.

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CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest to disclose.

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