Case Report

Journal of Surgery and Surgical Care

Incidental Finding of Subacute Infective Endocarditis with Bioprosthetic Mitral Valve Failure in a Patient Presenting with Lethargy

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Received Date: 25 Apr 2026;
Accepted Date: 06 Jun 2026;
Published Date: 26 Jun 2026.

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Citation: Akbar S, Trap P, Goldstein LB. Incidental Finding of Subacute Infective Endocarditis with Bioprosthetic Mitral Valve Failure in a Patient Presenting with Lethargy. J Surg Surg Care. 2026; 2(1): 1-4.

ABSTRACT

A prosthetic mitral valve replacement is a major surgical procedure that patients may undergo when experiencing severe refractory symptoms. When a patient presents with progressive lethargy, the differential diagnosis should be broadened, and appropriate imaging should be performed, particularly in the context of a significant surgical history. This may reveal severe underlying pathology, such as a chronically infected valve with a ruptured bioprosthetic mitral valve left wide open. We present the case of a 78-year-old female with a history of prior mitral valve repair followed by bioprosthetic mitral valve replacement and a remote episode of septic bacteremia requiring intensive care unit admission, who subsequently presented to her cardiologist with a 3-month history of worsening lethargy and declining functional capacity. A transesophageal echocardiogram ordered by her cardiologist revealed severe bioprosthetic mitral valve leaflet dehiscence with 4+ mitral regurgitation and mobile vegetations consistent with subacute infective endocarditis. Blood cultures obtained at the time of presentation grew Streptococcus viridans, which was considered the likely causative organism. The patient was initiated on broad-spectrum intravenous antibiotics and subsequently underwent successful bioprosthetic mitral valve re-replacement at a tertiary referral center, with an uncomplicated postoperative course and excellent functional recovery. This case exemplifies the importance of avoiding early closure in clinical decision-making and remaining open-minded when pursuing diagnostic testing for conditions for which a patient has risk factors, even in the absence of characteristic symptoms.

***Keywords:** Prosthetic Heart Valve; Mitral Valve Replacement; Subacute Endocarditis; Valve Rupture; Lethargy; Infective Endocarditis.*

Introduction

Mitral valve repair is the ideal intervention for many forms of mitral valve disease and may provide excellent long-term survival comparable to that of the age-matched general population [1]. When the mitral valve is not repairable, valve replacement may be required, and mechanical prostheses may be associated with improved durability and survival in selected younger patients compared with biological prostheses [1]. After valve replacement, ongoing surveillance is important because prosthetic valves may develop structural valve degeneration, thrombosis, paravalvular leak, pannus formation, or infective complications. Current valvular heart disease guidelines recommend post-procedure transthoracic echocardiography followed by surveillance imaging at 5 and 10 years and then annually, with additional imaging such as transesophageal echocardiography, fluoroscopy, or cardiac computed tomography when symptoms or signs suggest prosthetic valve dysfunction [2].

Prosthetic valve endocarditis (PVE) is a microbial infection involving a prosthetic heart valve or reconstructed native valve tissue [3]. PVE accounts for approximately 20% of infective endocarditis cases and is considered one of the most severe forms of infective endocarditis because it is associated with high morbidity,

high mortality, abscess formation, dehiscence, heart failure, embolic complications, and frequent need for surgery [3,4]. Based on the timing of disease acquisition, PVE is commonly classified as early or late, with early PVE occurring within 1 year of valve surgery and late PVE occurring after 1 year [3]. Infective endocarditis can present with fever, chills, malaise, fatigue, dyspnea, embolic phenomena, new murmur, or heart failure, but symptoms may be subtle or nonspecific, particularly in older adults or patients with prosthetic valves [5].

Subacute endocarditis has a slower disease process and may be present for weeks to months with gradual progression unless complicated by a major embolic event, acute valvular dysfunction, abscess, fistula, or ruptured cardiac structure [5]. Common causative organisms include viridans streptococci, Streptococcus gallolyticus, Staphylococcus aureus, coagulase-negative staphylococci, HACEK organisms, and enterococci [5]. Endothelial injury, turbulent blood flow, prosthetic material, and transient bacteremia may permit microbial adherence to platelet-fibrin thrombus, followed by vegetation formation and persistent bloodstream infection [5]. Because clinical findings may be nonspecific, echocardiography is central to diagnosis. Transthoracic echocardiography is typically used as the first-line imaging modality in suspected infective endocarditis, while transesophageal echocardiography is recommended when there is a prosthetic valve, suspected complications, or persistent concern despite negative or inconclusive transthoracic imaging [6].

The management of endocarditis requires an interprofessional approach involving infectious disease, cardiology, cardiac imaging, microbiology, and cardiac surgery [7]. Infectious disease consultation is important because antimicrobial selection depends on organism identification, prosthetic material, timing of infection, susceptibility testing, renal function, and the presence of complications [7]. Early cardiothoracic surgery consultation is recommended when complications are present, including heart failure, uncontrolled infection, abscess, prosthetic valve dehiscence, recurrent embolization, or severe acute valvular regurgitation [7,8]. Current endocarditis guidance identifies heart failure, uncontrolled infection, and prevention of embolic events as major indications for surgery, with emergency surgery recommended for aortic or mitral native or prosthetic valve endocarditis complicated by severe acute regurgitation, obstruction, or fistula causing refractory pulmonary edema or cardiogenic shock [8].

This case report presents a patient with a bioprosthetic mitral valve who was found to have subacute infective endocarditis and severe prosthetic mitral valve failure on transesophageal echocardiography after presenting primarily with progressive lethargy.

Case Presentation

The patient was a 78-year-old female with a complex cardiac surgical history. Approximately 12 years prior to the current presentation, she underwent surgical mitral valve repair for degenerative mitral regurgitation. Due to progressive repair failure and recurrent severe regurgitation, she subsequently required bioprosthetic mitral valve replacement approximately 7 years prior to the current presentation. Her postoperative course following valve replacement was uncomplicated, and she remained under routine cardiology surveillance. Approximately 2 years prior to the current presentation, she experienced an episode of septic bacteremia, presumed to be of urinary source based on concurrent urinalysis findings and the absence of an identified alternative source. She was hospitalized with an extensive intensive care unit stay and recovered fully with antibiotic therapy. At that time, echocardiographic evaluation did not demonstrate clear valvular involvement, and the bacteremia was treated as an isolated event unrelated to her prosthetic valve; however, in retrospect, the possibility that subclinical seeding of the bioprosthetic valve occurred during this bacteremia episode cannot be excluded. In the 3 months preceding the current presentation, the patient noted a gradual decline in functional capacity and worsening lethargy. She denied cardiovascular symptoms including syncope, palpitations, dizziness, dyspnea, or presyncope, and presented for what she believed to be a routine surveillance transesophageal echocardiogram with her cardiologist.

The echocardiogram showed highly mobile vegetations at the left/noncoronary commissure area of the aortic valve (Figure 1). The bioprosthetic mitral valve replacement was noted to have a torn leaflet in the anterior (Figure 2). There was a 4+ wide-open mitral valve regurgitation with excessive leaflet mobility (Figure 2). The mean gradient was 7 mmHg indicating severe mitral regurgitation. There was also significant pulmonary systolic flow reversal in both left and right upper pulmonary veins. The tricuspid valve had normal-appearing leaflet anatomy without signs of vegetation. There was moderate tricuspid regurgitation and a dilated tricuspid annulus.

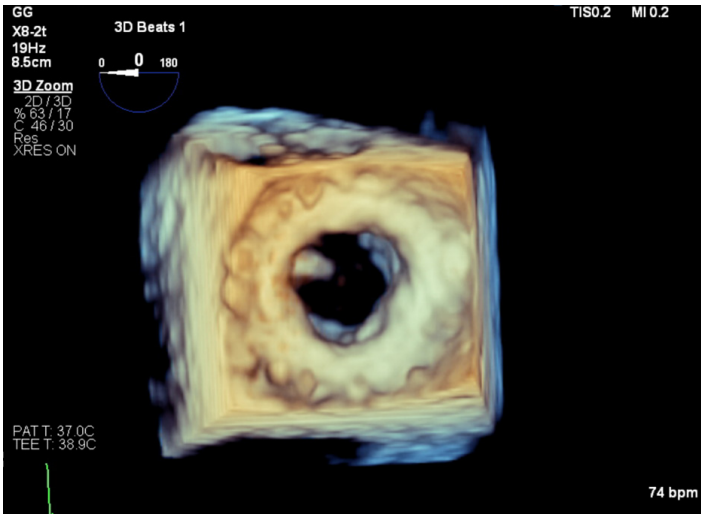

Figure 1: *3D transesophageal echocardiography (TEE) rendering of the aortic valve demonstrating vegetations.*

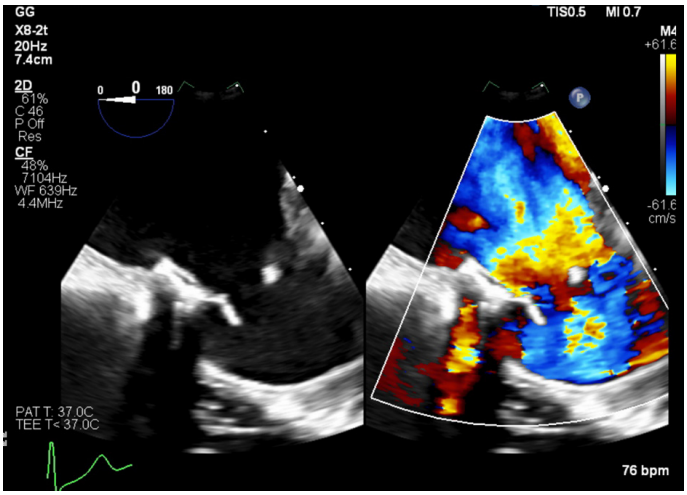

Figure 2: *The above Figure 2 depicts the TEE that the patient had, which revealed wide-open mitral regurgitation secondary to bioprosthetic mitral valve leaflet dehiscence.*

The patient was urgently advised to present to the emergency department given the echocardiographic evidence of active endocarditis with severe bioprosthetic mitral valve leaflet dehiscence and wide-open mitral regurgitation. Blood cultures were obtained in duplicate on arrival and subsequently grew Streptococcus viridans in both sets, with sensitivities demonstrating susceptibility to penicillin and ceftriaxone. Given the clinical picture and culture results, the findings were interpreted as late prosthetic valve endocarditis due to viridans group streptococcus, with the prior episode of septic bacteremia considered a plausible, though unconfirmed, predisposing event. The patient was initiated on intravenous cefepime and vancomycin empirically pending final culture susceptibilities. She developed acute kidney injury during the hospitalization, which was attributed to nephrotoxic antibiotic exposure and resolved promptly after the offending agents were held and antibiotic therapy was narrowed. The patient was stabilized medically and subsequently transferred to a tertiary referral center, where she underwent successful bioprosthetic mitral valve re-replacement with a 27 mm tissue prosthesis. The intraoperative course was uncomplicated. Postoperatively, she was extubated within 12 hours and transferred out of the cardiac intensive care unit on postoperative day 2. She was discharged home on postoperative day 7 with a 6-week course of intravenous ceftriaxone per infectious disease recommendations. At her 6-week follow-up visit, she reported marked improvement in energy levels and functional capacity, with no recurrence of symptoms. Transthoracic echocardiography at follow-up demonstrated a well-seated prosthesis with normal gradient and no evidence of residual infection or perivalvular leak.

Discussion

The patient initially presented with a generalized complaint of progressive lethargy spanning approximately 3 months. Notably, the patient did not have any notable cardiovascular symptoms such as chest pain, syncope, presyncope, dizziness, or palpitations, symptoms that are commonly expected in patients with significant valvular pathology. The echocardiogram revealed bioprosthetic mitral valve leaflet dehiscence with wide-open mitral regurgitation and valvular vegetations consistent with subacute infective endocarditis, findings that would not have been apparent from the patient's laboratory workup alone. This clearly exemplifies the importance of obtaining transesophageal echocardiography to definitively diagnose infective endocarditis and assess prosthetic valve integrity, particularly when clinical suspicion exists based on surgical history. Had the transesophageal echocardiogram not been ordered, the bioprosthetic mitral valve leaflet dehiscence might not have been discovered, and the patient could have deteriorated fatally from unrecognized severe mitral regurgitation or septic embolization. The physician's expertise in ensuring the appropriate workup for this patient demonstrates the importance of recognizing specific risk factors a patient may carry for a given condition, even in the absence of its characteristic symptoms.

The presentation in this case is consistent with previously reported cases of late prosthetic valve endocarditis and bioprosthetic valve failure occurring with minimal or atypical symptoms. Several published case reports and series have documented patients with hemodynamically significant prosthetic valve dysfunction who presented without classic features of endocarditis such as fever, new murmur, or embolic phenomena [3,4,9]. Musci et al. reported that a substantial proportion of patients undergoing redo surgery for prosthetic valve endocarditis presented with non-specific constitutional complaints rather than overt cardiovascular decompensation, underscoring the diagnostic challenge in this population [9]. Similarly, Alonso-Valle et al. noted that atypical presentation of infective endocarditis is particularly common in elderly patients and those with bioprosthetic valves, where immune senescence and the dampening of the inflammatory response may blunt the classic febrile syndrome [10]. The viridans group streptococci, as identified in our patient, are well-recognized causative organisms in late prosthetic valve endocarditis and have been associated with indolent, slowly progressive disease courses that may evade detection for weeks to months [3,5]. Consistent with guideline recommendations, the identification of a vegetation on prosthetic valve material, combined with a positive blood culture growing a recognized endocarditis pathogen, was sufficient to fulfill the modified Duke criteria for definite infective endocarditis in this patient [6,8]. It is further notable that the prior episode of septic bacteremia in this patient represents a recognized risk factor for subsequent prosthetic valve seeding; current endocarditis guidelines emphasize the importance of echocardiographic evaluation in patients with a prosthetic valve who develop bacteremia, regardless of the presumed source [8].

A bioprosthetic mitral valve leaflet dehiscence complicated by infective endocarditis is a life-threatening condition, and the patient in this case was entirely free of cardiovascular symptoms at the time of diagnosis. The prior surgical history of valve replacement was appropriately weighted in the clinical decision-making process, and severe underlying pathology was discovered as a result. The successful surgical outcome in this case, including uncomplicated valve re-replacement, early extubation, and full functional

recovery at 6 weeks, reinforces that timely diagnosis and definitive surgical intervention can lead to excellent outcomes even in elderly patients with complex valvular histories. With a patient presenting only with lethargy, a physician practicing a minimal standard of care might have ordered only a complete blood count and thyroid stimulating hormone panel. The patient could have been discharged with normal results and no further workup, with potentially fatal consequences.

Conclusion

Lethargy is a common clinical complaint that could be due to a vast array of causes. When a patient presents with any given complaint, it is important for the physician to take into account the patient's entire medical history when assessing for a likely culprit. The incidental finding of subacute endocarditis and a ruptured prosthetic mitral valve saved this patient's life. Further research needs to be done into atypical presentations of common symptoms in patients with prosthetic valves. Additionally, more concrete follow-up echocardiography guidelines are necessary to provide adequate surveillance for these potentially severe complications of prosthetic valves. Doctors of all specialities must be kept well-informed of the potentially subtle symptoms a patient with a prosthetic valve may have that might correlate to very severe etiologies.

Statement of Informed Consent

Informed consent was obtained from the patient described in this case report.

Conflict of Interest and Funding

The author declares no conflict of interest and received no specific funding for this work.

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