

Recurrent Prosthetic Mitral Valve Dehiscence due to Infective Endocarditis: Discussion of Possible Causes

Suleyman Ercan, M.D.¹, Gokhan Altunbas, M.D.², Hayati Deniz, M.D.³, Gokhan Gokaslan, M.D.³,
Vuslat Bosnak, M.D.⁴, Mehmet Kaplan, M.D.¹, Vedat Davutoglu, M.D.¹

Prosthetic valves are being widely used in the treatment of heart valve disease. Prosthetic valve endocarditis (PVE) is one of the most catastrophic complications seen in these patients. In particular, prosthetic valve dehiscence can lead to acute decompensation, pulmonary edema, and cardiogenic shock. Here, we discuss the medical management of late PVE in a patient with a prior history of late and redo early PVE and recurrent dehiscence. According to the present case, we can summarize the learning points as follows. A prior history of infective endocarditis increases the risk of relapse or recurrence, and these patients should be evaluated very cautiously to prevent late complications. Adequate debridement of infected material is of paramount importance to prevent relapse. A history of dehiscence is associated with increased risk of relapse and recurrent dehiscence.

Key words: 1. Prosthetic mitral valve
2. Endocarditis
3. Dehiscence

CASE REPORT

A 30-year-old male patient had undergone mitral valve replacement with a No. 27 St. Jude metallic valve six years previously. He also underwent mitral valve replacement with a bioprosthetic valve one year previously due to dehiscence of the metallic valve and cardiogenic shock caused by PVE (Figs. 1, 2). Six weeks after this second operation, the patient again was evaluated for cardiogenic shock and prosthetic valve dehiscence was noted. He underwent a third operation, and the mitral valve was replaced with a No. 27 St. Jude metallic valve. Vancomycin (2 g/day), rifampicin (600 mg/day), and gentamicin (80 mg/day) were started immediately. After

the third operation, the patient received six weeks of antibiotic therapy and was discharged without sequelae. Several blood cultures were drawn, and all were negative for a causative agent. He was scheduled for outpatient follow-up monthly to adjust the warfarin dose. Fourteen months after the last operation, he was admitted to our clinic with fever for nearly two weeks. Transthoracic echocardiography showed a mobile mass on the mitral valve prosthesis. Transesophageal echocardiography (TEE) was performed, and a partially mobile mass on the prosthetic valve was revealed. The mass was on the atrial side of the medial valve within the suture line and sized 8×5 mm (Fig. 3A). Laboratory evaluation showed an increased white blood count (13,500 mm/L), erythrocyte sedimentation

Department of Cardiology, ¹Gaziantep University School of Medicine, ²Kilis State Hospital, Departments of ³Cardiovascular Surgery and ⁴Infectious Diseases, Gaziantep University School of Medicine

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Corresponding author: Suleyman Ercan, Department of Cardiology, Gaziantep University School of Medicine, TR-27310 Gaziantep, Turkey
(Tel) 90-505-231-5501 (Fax) 90-342-360-3928 (E-mail) sleymanercan@yahoo.com

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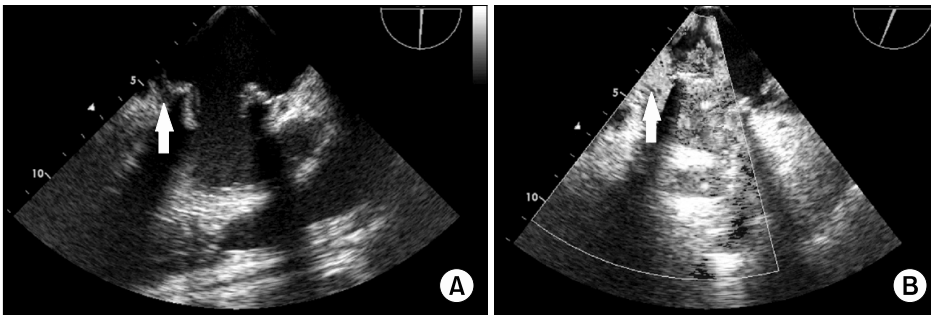


Fig. 1. Transesophageal echocardiography shows prosthetic valve dehiscence. (A) 2-D image, (B) Color Doppler image. Arrow shows prosthetic valve dehiscence.

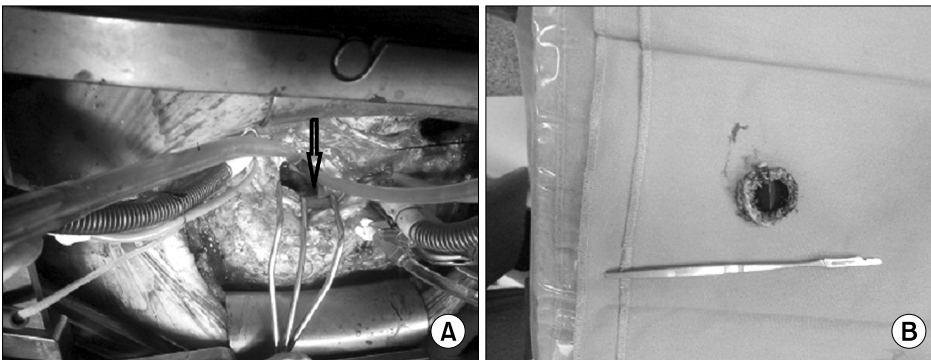


Fig. 2. Photos of second operation field. (A) Arrow shows prosthetic valve dehiscence, (B) prosthetic valve.

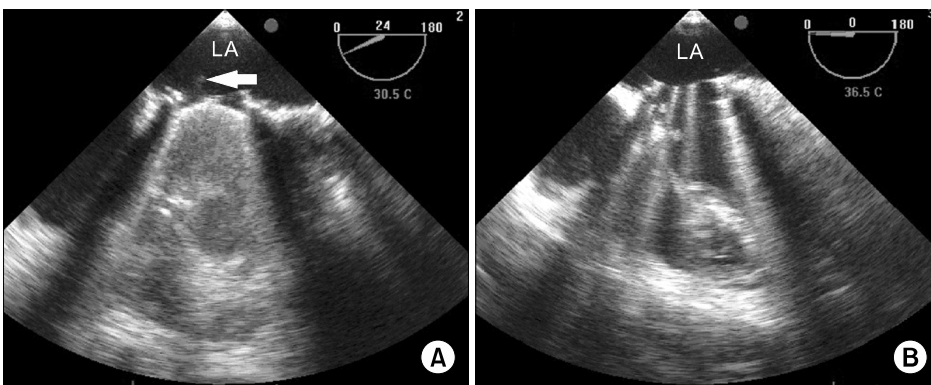


Fig. 3. Transesophageal echocardiography shows (A) a mass on the prosthetic valve, (B) regression of the mass. Arrow shows mass on the prosthetic valve. LA, left atrium.

rate (80 mm/hr) and C-reactive protein (161 mg/L). The international normalized ratio (INR) was 2.4. The patient was then hospitalized for PVE, and three sets of blood cultures were drawn. Vancomycin (2 g/day) and rifampicin (600 mg/day) were started immediately. Blood cultures were negative for a causative agent. On follow-up, clinical and laboratory values were improved. After six weeks of treatment, the mass was shown to regress on TEE (Fig. 3B). By adjusting the dose of warfarin, a steady INR level between 3 and 3.5 was achieved and the patient was discharged. There was no relapse during the first six months of close follow-up.

DISCUSSION

Due to the increased number of patients with prosthetic heart valves, PVE is now more frequently encountered. PVE carries the worst prognosis among patients with infective endocarditis and is seen in 1% to 6% of patients with prosthetic heart valves [1]. Early PVE occurs within the first year of surgery, and the risk is 1% to 3%. The difference in the causative microorganism plays a key role both in the diagnosis and management. Early PVE is frequently caused by *Staphylococcus epidermidis*. Infective endocarditis caused by

staphylococci has a higher rate of morbidity and mortality than that caused by other microorganisms, and surgery is usually required. Late PVE shares a common microbiological profile with native valve endocarditis [1].

The diagnosis of endocarditis is challenging in patients with prosthetic heart valves. Although less specific than native valve endocarditis, the Duke criteria are used in the diagnosis of PVE. Blood culture positivity reaches 85% in these patients. We could not isolate a causative microorganism in our patient. Transthoracic echocardiography has low sensitivity and specificity for certain complications, such as perivalvular abscess formation, fistula, and dehiscence. Thus, TEE is recommended for evaluation of patients with PVE.

In approximately half of the patients with infective endocarditis, surgical treatment is needed. The timing of surgery is of utmost importance. The main indications for early surgery are hemodynamic instability; infective endocarditis caused by staphylococci, fungi, *Coxiella*, and *Chlamydia*; septic embolism; and signs of uncontrolled infection (persistent fever, paravalvular abscess, and vegetations greater than 1 to 2 cm in size) [2].

Habib et al. [3] reported that valvular insufficiency, heart failure, uncontrolled fever, and new onset conduction defects are poor prognostic factors for patients with PVE. The mortality rate for these patients reaches 23%.

Patients with dehiscence can have a stable hemodynamic profile but they can present with cardiogenic shock just as our patient did. Even if hemodynamically stable at presentation, patients with valvular insufficiency due to dehiscence eventually develop circulatory collapse. Rostagno et al. [4] reported that appropriate antibiotic treatment and timely surgery resulted in lower rates of recurrence and a nearly 90% chance of survival without complications. However, advanced age, a history of PVE, and recurrent endocarditis are reported to be poor prognostic factors [5].

Another challenge in patients undergoing surgery is choosing the type of prosthesis. Newton and Hunter [6] reported that in patients with PVE undergoing surgery, the choice of the prosthesis, either a bioprosthesis or mechanical valve, did not affect the rates of recurrence. However, mechanical valves resulted in better survival in patients younger than 60 to 65 years of age, and this difference was not seen in pa-

tients older than age 65.

Recurrences within the first six months of treatment are usually the relapse of the same microorganism [7]. The main reasons for relapse endocarditis are inappropriate antibiotic treatment both in terms of duration and the choice of antibiotic without an antibiogram in medically managed patients. In surgically managed patients, the presence of deep infections and inadequate debridement of infected material are the main reasons for relapse. The second dehiscence seen in our patient seems to be a consequence of insufficient debridement.

In conclusion, the learning points from this case report are as follows. 1) A prior history of infective endocarditis increases the risk of relapse or recurrence and these patients should be evaluated very cautiously to prevent late complications. 2) Adequate debridement of infected material is of paramount importance to prevent relapse. 3) A history of dehiscence is associated with increased risk of relapse and recurrent dehiscence.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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