

Mitral valve endocarditis in a patient with pre-existing severe primary mitral regurgitation

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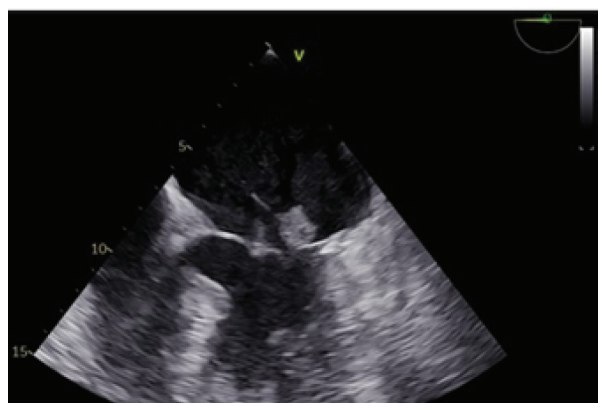


FIGURE 1. Large vegetation on the posterior mitral leaflet and smaller vegetation on the anterior mitral leaflet.

Introduction: Severe primary mitral regurgitation predisposes patients to serious complications, including infective endocarditis, which remains a life-threatening disease with high morbidity and mortality, particularly when affecting previously damaged valves^{1,2}. The mitral valve is among the most involved sites³. We present a case of mitral valve endocarditis in a patient previously followed for severe primary mitral regurgitation, highlighting diagnostic and therapeutic challenges.

Case report: 50-years-old male patient with known severe asymptomatic primary mitral regurgitation (P2/P3 prolapse) was admitted due to persistent fever. There were no signs or symptoms of heart failure. Blood cultures came positive with coagulase-negative Staphylococcus and suspicion of endocarditis was raised. Transesophageal echocardiography revealed large vegetations on both mitral leaflets, with larger one on the posterior mitral leaflet (15x16 mm), suspected chordal rupture and severe regurgitation (**Figure 1**). The patient was started on standard antibiotic treatment according to ESC guidelines. No signs of septic embolization were found. Given the size and mobility of the vegetation, early surgery was considered. Nine days later, the patient was transferred to Cardiac Surgery Department where he underwent mitral valve replacement (Epic Mitral 33 mm). Pseudomonas spp. was isolated from the vegetation and targeted antibiotic treatment with meropenem was administered for a total duration of four weeks with repeated negative blood cultures. Afterwards, the patient was discharged and remained asymptomatic on follow-up.

Conclusion: This case highlights severe primary mitral regurgitation as a predisposing factor for infective endocarditis of the mitral valve. However, the pre-existing regurgitation may have played a protective role by preventing acute cardiac decompensation during infection.

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