Introduction

It is difficult to determine whether regurgitation is due to a leaflet tear after mitral valve replacement (MVR) following repair when the regurgitation appears soon after the valve replacement because the valve is implanted in the fragile mitral annulus during repeated surgery.

Structural Valve Deterioration of Porcine Bioprosthesis Soon after Mitral Valve Repair and Replacement

An 81-year-old woman, who had undergone mitral valve replacement (MVR) with a porcine bioprosthesis after mitral valve repair, presented with hemolysis 4 years and 6 months after MVR. Transthoracic echocardiography (TTE) revealed trivial mitral regurgitation, which was diagnosed based on the observed perivalvular leakage. Hemolysis gradually increased, and she developed dyspnea and edema 2 years after the appearance of mitral regurgitation. We performed a reoperation. Intraoperative transesophageal echocardiography (TEE) after intubation showed no perivalvular leakage of the mitral prosthesis, but transvalvular leakage through a leaflet perforation was present. The leaflets of the bioprosthesis had slit-shaped perforations at their hinges. There was no sign of infection on the leaflet or annulus. We implanted a new bioprosthesis after removal of the deteriorated valve. The postoperative course was uneventful. Microscopic examination verified collagen degeneration, histiocyte infiltration, and hyalinization. It is important to perform TEE to rule out structural valve deterioration (SVD) even when regurgitation occurs soon after valve replacement.

Keywords: prosthesis, biomaterials, mitral valve repair, mitral valve replacement

Case Report

An 81-year-old woman was referred to our hospital for surgery. She underwent MVR with a 27-mm Mosaic Mitral Heart Valve (model 310; Medtronic, Minneapolis, Minnesota, USA) for residual mitral regurgitation 20 days after mitral valve repair with a 29-mm Duran AnCore Ring (Medtronic) at the age of 74 years. Hemolysis appeared 4 years and 6 months after the MVR, and transthoracic echocardiography (TTE) revealed trivial
mitral regurgitation, which was diagnosed due to the observed perivalvular leakage. She did not present with any signs of heart failure and received medical treatment because the regurgitation was considered to have resulted from perivalvular leakage. Hemolysis gradually increased, and she developed dyspnea and edema 2 years after the diagnosis of mitral regurgitation. Her blood cell count showed mild anemia characterized by a hemoglobin level of 9.6 mg/dl, and her blood chemistry showed a lactic dehydrogenase level of 1070 U/dl. TTE revealed a mild degree of mitral regurgitation due to perivalvular leakage and slightly reduced left ventricular function with a 62.3% ejection fraction. We performed a re-MVR. Transesophageal echocardiography (TEE) revealed no perivalvular leakage of the mitral prosthesis, but transvalvular leakage through a leaflet perforation was revealed after intubation prior to the surgery. After cardiopulmonary bypass (CPB) had been initiated with ascending aorta and bicaval cannulation, the ascending aorta was cross-clamped, and a left-sided left atriotomy was performed. All leaflets of the Mosaic prosthesis were fragile, and scaphoid-shaped perforations were found at the hinge of the two leaflets (Fig. 1). There was no aperture between the prosthetic valve and mitral annulus. Neither vegetation nor abscesses were found on the leaflet or annulus. We removed the Mosaic prosthesis, and a new 25-mm Epic bioprosthesis (St. Jude Medical, Inc., Saint Paul, Minnesota, USA) was implanted using 14 mattress sutures. The patient was weaned from CPB without difficulty under continuous administration of a low-dose catecholamine. The postoperative course was uneventful. Ultrasound cardiography showed no mitral regurgitation with a mean pressure gradient of 7 mmHg, and mitral valve area was 1.51 mm².

Macroscopic examination of the explanted valve demonstrated tears at the hinge of the leaflet and intracuspal hematomas in the two cusps, and a small amount of pannus was noted on the inflow margin (Fig. 2). Microscopic evaluation verified hyalinization and collagen degeneration within the leaflets. Histiocyte infiltration was observed although neutrophil migration was not apparent (Fig. 3). There were no signs of infection, and radiography showed no mineralization in the prosthetic valve and patient’s tissue.

Discussion

Durability of bioprosthetic valves is improving because of advancement in tissue treatment methods. The actuarial freedom from explantation due to SVD was 83.4% at 14 years with the mitral Carpentier-Edwards Perimount¹ and 95.3% at 10 years with the mitral Mosaic bioprosthesis² when the valves were implanted in patients older than 60 and 70 years, respectively. The most frequent cause of regurgitation after re-MVR is perivalvular leakage.³,⁴ In the present case, it was difficult to determine whether the regurgitation was due to SVD because the regurgitation was noted 4 years and 6 months after the initial MVR that followed valve repair. The patient has been on only medical treatment for 2 years since the diagnosis of mitral regurgitation was made. She might

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Fig. 1 Mitral bioprosthesis from surgeon’s view. All leaflets are fragile. Scaphoid-shaped tear is shown at the leaflet edge (white arrow).

Fig. 2 Explanted Mosaic valve. The noncoronary cusp is removed during surgery. Tears are apparent at the leaflet edge (white arrow), and intracuspal hematomas are observed (black arrows).
have undergone the re-MVR earlier if the diagnosis of SVD had been correctly made after the first TTE. Further examination should be performed by TEE with consideration of SVD even when hemolysis and regurgitation appear soon after MVR.

Cuspal tears were apparently caused by degeneration of the porcine tissue in the present case. The tears were positioned along the porcine aortic annulus, and the cusps were extremely fragile and easy to tear by explant manipulation during the surgery. Histopathological examination revealed collagen degeneration with hemorrhage of the porcine valve leaflets, and histiocyte infiltration and hyalinization were observed, suggesting an immune response and chronic inflammation. Early collagen degeneration is considered to be related to inflammatory cell reaction. Khan, et al. reported morphological changes in porcine bioprosthetic valves of left ventricular devices. They found cusp tears and hemorrhage of the bioprosthesis and a massive influx of mononuclear cells on the cusp. An immune response may have caused the early cuspal tear in the present case.

**Conclusion**

We experienced a case of mitral valve regurgitation due to SVD after mitral valve repair and replacement. Further examination should be performed by TEE with consideration of SVD even when hemolysis and regurgitation appear soon after valve replacement.

**Disclosure Statement**

All of the authors have read the manuscript and have approved of its submission. The authors report no conflicts of interest.

**References**