Pure Cusp Tear of Trifecta Bioprosthesis
2 Years after Aortic Valve Replacement

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Trifecta is a stented bioprosthetic heart valve with a bovine pericardial sheet externally mounted on a titanium stent. This valve is applied only for aortic valve replacement (AVR), providing excellent hemodynamics and extremely low incidence of structural valve deterioration (SVD). A 76-year-old woman presented with dyspnea on effort 24 months after AVR with a 21-mm Trifecta valve. Echocardiography revealed severe aortic regurgitation with prolapse of a cusp of Trifecta valve, which suggested that she developed acute heart failure due to early SVD. In the operation, Trifecta valve had a cusp tear near the commissure with circumferential fibrous pannus ingrowth only at the inflow side. There was neither calcification nor infection. The Trifecta valve was successfully replaced with a new porcine bioprosthesis.

Keywords: pericardial bioprosthesis, structural valve deterioration, aortic valve replacement, Trifecta

Introduction

Trifecta (St Jude Medical, Inc., St Paul, MN, USA) is a third-generation, stented bioprosthetic heart valve for the aortic valve replacement (AVR). This valve is made from one bovine pericardial sheet, which is externally mounted on a titanium stent with an anti-calcification treatment, providing excellent hemodynamics. The medium- and long-term outcomes were favorable including low incidence of early structural valve deterioration (SVD) including cusp tear, calcification, pannus formation, and thrombosis. Modi et al.1) reported a single-center experience of 400 patients showing no SVD during 1 ± 0.6 year follow-up. Permanyen et al.2) also revealed no SVD for 200 AVR patients 2 years postimplantation. Barvaria et al.3) demonstrated only one SVD of 1022 cases of AVR during 2-year follow-up from the multicenter, prospective study.

In Japan, Trifecta valve has been implanted since 2012. To date, there are few published case reports regarding early SVD of Trifecta valve. Herein, we report a rare case of cusp tear of Trifecta bioprosthesis 2 years after the operation and review the articles related to early SVD requiring reoperation.

Case Report

A 76-year-old woman with severe aortic regurgitation (AR), tricuspid regurgitation, and ascending aortic dilatation with a diameter of 51 mm with no concomitant dilatation of Valsalva sinus had undergone AVR with a 21-mm Trifecta valve, tricuspid valve repair using an Edwards MC3 ring, and graft replacement of the ascending aorta. In the AVR, the Trifecta was selected one size smaller and implanted with 12 pairs of everting mattress sutures. The sutures were tied without removing the valve holder to not injure the pericardial leaflets. After the operation, there were no abnormal echocardiographic
findings including paravalvular leakage and significant interprosthetic pressure gradient of the Trifecta valve. She was doing well for 24 months without any signs of heart failure. However, she presented with dyspnea on effort at regular outpatient follow-up. On physical examination, a grade 3 diastolic murmur was heard along the upper left sternal border. Transthoracic echocardiography (TTE) revealed newly development of mild AR with normal dimension of the left ventricle. The patient was followed medically with oral diuretics. After 2 months, however, she suddenly developed acute heart failure (Fig. 1A). TTE revealed severe AR with prolapse of a cusp and significant mitral regurgitation caused by mitral annular dilatation due to acute volume overload of the left ventricle (Fig. 1B). Because optimal medications and respiratory support were unable to eliminate pulmonary edema, re-do AVR with a porcine bioprosthesis and mitral annuloplasty with a rigid band were performed under second sternotomy. During the operation, we found that the Trifecta valve had a cusp tear close to the commissure at the right coronary cusp without any calcification (Figs. 2A and 2B). Circumferential fibrous pannus ingrowth was also observed on the inflow side of the valve within the annular suture line extending 2–4 mm onto the cusp base. There was no pannus ingrowth on the outflow surface including the cusps and the stent posts (Fig. 2C). All three cusps were of normal thickness. Microscopic examination revealed loss of collagen fibers at the tear site. There was neither infection nor acute inflammatory reactions of all three cusps. Postoperative course was uneventful with rapid improvement of lung congestion.

**Discussion**

Recently, the articles describing early SVD of Trifecta valve have been published as a case report in western countries (Table 1). In these articles, the early SVD occurred at 3 months to 4 years (mean: 26 months) after the operation including our case. All the patients’ ages at the time of AVR were over 65 years old, which were
appropriate indication for bioprosthetic valves. The mode of SVD was cusp tear in 3 (9, 27, and 34 months after implantation), calcification in 1 (4 years after implantation), and adhesion of a cusp to the Valsalva sinus in 1 (3 months after implantation). The authors described the causes of early SVD as follows. Cusp tear could be caused by mechanical stress on the parastent sutures which promotes abrasion and rupture of the cusps, and by peculiar pericardial fragility with no visible calcification. Early calcification could be caused by an immunologic reaction. Cusp adhesion to the aortic wall could be caused by a foreign body reaction to polypropylene sutures used for aortotomy closure. The Trifecta valve failure seems to be complex and multifactorial, as just described above.

Cusp tear is the main cause of early SVD in pericardial valves. Pericardial valves are subjected to the several stresses during valve opening and closing, such as tensile stress during valve closing, bending stress throughout opening and closing, and abrasion stress during valve opening. Tensile stress is intense at the free edge of the cusp near the commissure. Bending stress produced by repeated hinging action of the cusps accelerates the disruption of the glutaraldehyde-fixed cusps, especially near the commissure. Abrasion stress in pericardial cusps mounted outside the stent post provides mechanical injury by frequent attachment of the cusps to the aortic wall during valve opening, especially in a case with small and calcified aortic root. Of these stresses, tensile and bending stresses are crucial for the development of cusp tear, which is commonly observed close to the commissures independent of cusp calcification. Actually, the Trifecta SVD articles showed that cusp tears were recognized close to the commissures.

In our case, a 9-mm cusp tear existed along the stent post near the commissure. Inflammation, infection, and calcification were excluded as the cause of the tear. The pannus ingrowth was so localized at the inflow side and circumferential that it was unlikely to cause the tear only at the right coronary cusp. The aortic root was large enough not to contact the cusps to the aortic wall, showing low possibility of abrasion stress on the cusps. Overall, tensile and bending stresses seem to cause the cusp tear in the following mechanisms. Tensile and bending stresses would cause a minor tear to the free edge of a cusp at the commissure. The cusp with minor tear would prolapse gradually and result in the imbalance of the structural characteristics of a pericardial valve as leaning against each other among three cusps. The structure imbalance would bring uneven mechanical loads, providing stronger stress on a prolapsed cusp leading to further progression of the tear toward the cusp belly.

**Conclusion**

Although Trifecta valve has extremely lower possibility of early SVD, cusp tear close to the commissure should be considered when diastolic cardiac murmur was heard and new development of AR would be detected by echocardiography.

**Disclosure Statement**

We have no financial or other interest in the manufacture or distribution of the device.
References


