Excellent Durability of Starr-Edwards Ball Valves Implanted in the Aortic and Mitral Positions for 27 Years: Report of a Rare Surgical Case

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A 59-year-old male who had undergone aortic and mitral valve replacement with Starr-Edwards ball valves 27 years ago was admitted to our hospital for hemolytic anemia and heart failure. Echocardiography revealed prosthetic valve failure with a high-pressure gradient and small effective orifice area. The Starr-Edwards ball valves were successfully replaced with bileaflet mechanical valves. The explanted valves revealed no structural abnormalities.

Keywords: Starr-Edwards ball valve, reoperation

Introduction

The Starr-Edwards ball valve has been widely used as a prosthetic replacement for aortic and mitral valves, since its introduction in 1960.1) Surgical treatment of heart valve disease has advanced dramatically with the development of new techniques in the past decade. Therefore, although caged-ball valves are no longer commonly used today, the Starr-Edwards ball valve had played a significant role in treating valve disease in past decades.2–6) The long-term results of using the Starr-Edwards ball valve were satisfactory, with reliable durability and a low rate of complications; this caged-ball valve was, therefore, frequently used until the recent development of novel, improved prostheses. Here, we report a rare case wherein a patient implanted with Starr-Edwards ball valves in the aortic and mitral positions underwent reoperation 27 years after the initial operation.

Case

A 59-year-old male was admitted to our hospital for prosthetic valve failure; he had a history of rheumatic fever at the age of 10 years and had undergone mitral and aortic valve replacement with Starr-Edwards ball valves (model 6120, 32 mm; model 2400, 24 mm) 27 years ago. Over 20 years postoperatively, he remained asymptomatic; however, in the last 5 years, heart failure and hemolytic anemia due to prosthetic valve dysfunction progressed. He had been presented dyspnea on effort, and cardiomegaly and hemolytic anemia (hemoglobin 8.3 g/dl, lactate dehydrogenase 1004 IU/l, brain natriuretic peptide 333 pg/ml) were noticed by the routine examination (Fig. 1). Echocardiography demonstrated stenosis in the aortic prosthesis with an effective orifice area of 0.5 cm², a peak pressure gradient through the valve of 90 mmHg, and trivial aortic insufficiency. Moderate mitral stenosis with large left atrium without thrombus and moderate tricuspid regurgitation were also evident. Therefore, reoperation was indicated for prosthetic valve failure.

Through re-median sternotomy, the chest was re-opened without major bleeding. Cardiopulmonary bypass was established with femoral and right axillary artery cannulation and bicaval drainage. Under cardiac arrest, the Starr-Edwards ball valves were excised, and new bileaflet mechanical prostheses were implanted.
The patient recovered uneventfully and was discharged at postoperative day 22 after reinitiation of anticoagulation therapy. Macroscopic examination of the explanted Starr-Edwards ball valves revealed no structural abnormalities (Fig. 2).

**Discussion**

The first successful mechanical prosthesis for mitral valve replacement in humans was achieved with a Starr-Edwards caged-ball valve in 1960. The Starr-Edwards ball valve has played an important role in cardiac surgery with reliable durability and satisfactory results, until the advent of recent bileaflet mechanical prostheses that demonstrated excellent long-term results. Although the time of implantation may affect the rate of embolization, survival, or reoperation, the survival rate of patients who underwent aortic valve replacement with Starr-Edwards valve was 53.0%–62.3%, 23.0%–39.4%, and 8.0%–19.9% over 10, 20, and 30 years, respectively. Further, the survival rate of patients after mitral valve replacement was reported as 51.0%–75.0%, 23.0%–61.0%, and 8.0%–33.0% over 10, 20, and 30 years, respectively. The proportion of patients not requiring reoperation after Starr-Edwards aortic valve replacement was reported as 75.0%–93.7%, 62.5%–86.1%, and 56.2%–62.3% at 10, 20, and 30 years, respectively, while that for mitral valve replacement was reported to be 94.3%–96.4%, 83.0%–87.0%, and 78.9%–87.0% at 10, 20, and 30 years, respectively.

Previous studies concerning the Starr-Edwards ball valve reported several complications such as thromboembolic events, hemolytic anemia, valve dysfunction, lipid infiltration of the silicon ball, tissue ingrowth around the perivalvular area, and cloth wear, although several modifications were made to these valves in order to reduce the incidence of thromboembolism and hemolytic anemia. Further, Starr-Edwards valves have been reported to cause injury to the aortic wall or the left ventricular posterior wall according to its implantation position.

In the present case, the Starr-Edwards valves endured without complications over 20 years; however, reoperation was required for treating progressive hemolytic anemia and heart failure. This patient was treated as a New York Heart Association Class I patient for more than 20 years after the initial valve implantation. While hemolytic anemia and thromboembolism are well-known complications that may have occurred due to cloth wear of the caged-ball valves, cloth wear is difficult to diagnose by echocardiography due to artifacts from the stainless cage. Although there were no findings of cloth tear or pannus formation in the explanted valves, grade 1 aortic valve insufficiency was detected by preoperative echocardiography. Intraoperative finding demonstrated that there was paravalvular leakage due to the small detachment of the aortic valve. I think that might be a reason for the hemolytic anemia.

The pressure gradients of the Starr-Edwards valves are
known to be relatively higher than those for bileaflet valves, ranging from 20 to 73 mmHg (mean, 36.2 mmHg) in aortic Starr-Edwards valves and from 9 to 30 mmHg (mean, 16.6 mmHg) in mitral valves. Further, grade 1 insufficiency has been reported in 33% of aortic and 20% of mitral valves. The Starr-Edwards ball valve implanted in the mitral position may have caused subvalvular aortic stenosis due to the presence of the cage and ball in the left ventricular outflow tract. In the present case, echocardiography could identify prosthetic valve failure with a small orifice area and a high-pressure gradient through the valves; however, it is often difficult to accurately measure the pressure gradient through Starr-Edwards ball valves since the presence of the ball causes strong artifacts, preventing the measurement of the true axial flow through the valve.

In conclusion, we report here, a rare case of Starr-Edwards ball valves that were implanted 27 years previously in the aortic and mitral positions that required replacement for valve failure leading to hemolytic anemia and cardiac failure. The explanted valves showed no functional damage and had remained durable for over 20 years. We suggest that close follow up with echocardiography should be scheduled in patients with Starr-Edwards valve implantation even in the absence of symptoms, in order to prevent complications. Irrespective of the occurrence of structural damage, we consider that Starr-Edwards ball valves should be replaced after 20 years of implantation for maintaining good hemodynamic performance and for fewer complications.

References