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Case report - Valves

Extended (31 years) durability of a Starr-Edwards prosthesis in mitral position

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Abstract

A 62-year-old man underwent mitral valve replacement with a Starr-Edwards caged-ball prosthesis in 1974. He was asymptomatic until February 2005 when he underwent a new cardiac evaluation because of increasing dyspnea and peripheral edema. The echocardiogram showed a severe aortic regurgitation and a mitral valve prosthesis well functioning. At reoperation, the mitral prosthesis and the aortic valve were replaced with St. Jude Medical® bileaflet mechanical prostheses. At macroscopic and radiographic inspection the Starr-Edwards was free from signs of structural valve degeneration. This case demonstrates the impressive durability of a Starr-Edwards prosthesis in mitral position.

Keywords: Retrieval analysis; Starr-Edwards prosthesis; Prophylactic replacement

1. Introduction

The Starr-Edwards valve prosthesis was first successfully implanted in mitral position in September 21, 1960. The Starr is a caged-ball prosthesis manufactured and commercialized by Edwards Lifesciences in several models. The housing system is constructed in Stellite alloy and is composed of three or four monocast struts which, joining at the apex, cage the occluder represented by a silicone rubber ball. The sewing ring material is composed of porous Teflon and polypropylene cloth. The Starr-Edwards valve prosthesis was durable over a prolonged follow-up period, but systemic embolization, ball variance and chronic hemolysis remained a persistent problem. This case demonstrates the impressive durability of a Starr-Edwards (model 6120) implanted in mitral position over a period of 31 years.

2. Clinical summary

A 62-year-old man with a previous history of atrial fibrillation underwent mitral valve replacement with a Starr-Edwards caged-ball prosthesis in 1974 for a mixed mitral valve disease due to rheumatic fever. He was in good health until February 2005 and at echocardiographic follow-up the caged-ball prosthesis never showed signs of dysfunction. Very recently he underwent a new cardiac evaluation because of increasing dyspnea and peripheral edema. Chest X-ray showed a cardiomegaly with pulmonary congestion. The echocardiogram showed a severe aortic regurgitation and a well functioning mitral valve prosthesis. The mean trans-prosthetic gradient was 5 mmHg, the left ventricle ejection fraction was depressed and the patient was suffering from pulmonary hypertension. The cardiac catheterization confirmed the echo-diagnosis. At reoperation the patient underwent double valve replacement through a standard median sternotomy, cardiopulmonary bypass at a flow rate of ≥ 2.4 l/m^2 and mild hypothermia. The aortic valve was replaced with a bileaflet prosthesis, St. Jude Medical Regent® 21 mm, whereas the Starr-Edwards (caged-ball prosthesis) was prophylactically replaced with a St. Jude Medical® 29 mm bileaflet valve prosthesis. The postoperative course was uneventful. Echocardiogram before discharge confirmed that the prosthetic valves were functioning normally. At macroscopic and radiographic inspection the Starr-Edwards was free from evident signs of structural valve deterioration. The yellow appearance of the explanted valve suggested a lipid insudation of the ball occluder. Furthermore, we observed signs of imprinting due to the contact of the occluder with the struts of the housing. However, we did not observe any impairment to ball excursion (Fig. 1).

3. Comment

The Starr-Edwards is among the mechanical heart valve prostheses and the one with the longest long-term follow-up. Nevertheless, potential complications remained a persistent problem. According to literature and our experience, the silastic ball due to lipids insudation might

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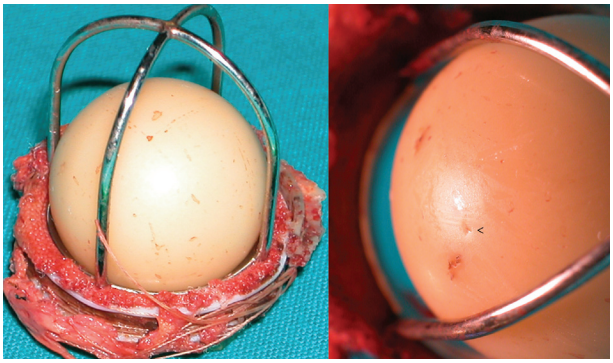

Fig. 1. Starr-Edwards model 6120 implanted in mitral position 31 years earlier. Time of functioning: 367 months. Note the yellow appearance of the poppet due to lipid insudation. Furthermore, the ball shows signs of imprinting (arrow) due to strut interaction and a small erosion.

change in color and sometimes in dimension leading to a decreasing poppet excursion and consequent valve incompetence and/or thrombosis. Additionally, ball damage with fracture and systemic embolization have also been reported. Thrombosis and pannus tissue overgrowth often occurred at the base of the valve or at the apex of the cage leading to either stenosis and block of the valve. Due to the high profile, this prosthesis sometimes injured, according to the implant position, the aortic wall and/or the left ventricle posterior wall. The design allowing only a lateral flow guaranteed high transvalvular gradients with important hemolysis and related complications. Therefore, the combination of stasis (i.e. higher gradients) and non-physiological surfaces yielded a tendency toward thrombus formation and fibrous pannus tissue overgrowth. We did not observe any of these complications, nor did we observe left ventricular posterior wall injury. The replacement of this mitral Starr-Edwards valve prosthesis was performed prophylactically to obtain a better hemodynamic performance because of pulmonary hypertension and a lower INR ratio because of a concomitant intestinal bleeding for polyps.

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