CASE REPORT

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Fatal Embolization of Ball Portion of Starr-Edwards Aortic Valve Prosthesis

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ABSTRACT: We report an unusual case of a Starr-Edwards aortic valve malfunction with fatal dislodgment and embolization of its ball portion to the distal abdominal aorta, just above the bifurcation, leading to sudden death. This case is unusual in that it is, to date, the longest time elapsed between valve placement and ball dislodgment.

KEYWORDS: forensic science, forensic pathology, heart valves, prosthesis, Starr-Edwards ball valve

The first Starr-Edwards ball valve was developed in 1960 (1). Since its introduction, thousands of Starr-Edwards valves have been implanted, and various models have been developed, the model used in our case is Model 1260. The complication of ball embolization from valve malfunction has been described before (2–4). However, according to our review of the literature, this case manifests the longest interval between valve placement and ball dislodgment, this case being seven years as opposed to four years previously reported (2). Most probably the dislodgment which occurred in our case was due to ball deformation (flattening in several areas); which appears to have facilitated passage through the cage. There was saccular dilatation of the root of the aorta seen at autopsy, which also could be a factor.

Case History

This 60-year-old Caucasian male had a history of aortic insufficiency, ascending aortic aneurysm, hypertension, obesity, and Parkinson's disease. On March 23, 1988, the decedent had a Starr-Edwards Ball-valve prosthesis (#26), Model number 1260, placed in his aortic ring to correct his aortic insufficiency. In 1991 he developed a 7 cm diameter ascending aortic aneurysm which was resected and repaired. The decedent had been placed on Coumadin (warfarin) since the valve surgery in 1988 and was treated with Sinemet (carbidopa/levodopa) for Parkinson's disease. In 1994 the decedent began complaining of burning right leg pain, radiating down to the right ankle; but a definitive diagnosis was never established. He reported feeling poorly just before his death. He collapsed at home on August 1, 1995 and death was pronounced at a nearby emergency room.

A private autopsy on August 5, 1995 revealed the wire basket of the Starr-Edwards ball valve to be empty. Its white silicone ball was lodged just proximal to the bifurcation of the abdominal aorta (Figs. 1, 2). The prosthesis itself appeared to be intact and firmly sutured in place, with no evidence of suture failure or rupture. There was a saccular dilation of the root of the aorta around the lower portion of the prostheses. The ball on exam showed deformation (flattening in several areas), which appears to have facilitated passage through the cage.

Other pertinent findings at autopsy included: an enlarged heart (750 gm), no evidence of pulmonary emboli or coronary artery emboli, some hepatosplenomegaly, and evidence of aspirated food in the tracheo-bronchial tree. The lower extremities were pale and showed loss of hair.

The findings at autopsy brought the case under the jurisdiction of the Los Angeles County Medical Examiner-Coroner. The cause of death was aortic insufficiency and congestive heart failure due to malfunction of prosthetic valve placed for aortic insufficiency with dislodgment of the ball valve. Other significant factors contributing to death included: cardiac hypertrophy, hypertension, obesity, and Parkinson's disease.

Discussion

The usual clinical presentation of ball embolization has been reported to consist of: "(1) sudden onset of severe dyspnea or chest pain; (2) shock; (3) block of the arterial circulation to the legs leading to severe cramping pains in the lower extremities; and (4) absence of the opening and closing clicks of the ball valve prosthesis on physical examination (2)."

It has been reported that prosthesis associated complications are a more important cause of late death than cardiovascular abnormalities and operative complications (5,6). Specifically, cardiovascular and operative causes include congestive heart failure, myocardial infarction, arrythmias, sepsis, and other surgically related causes. Prosthesis associated causes are: thrombosis and thromboembolism (5), valve dehiscence, hemorrhage during anticoagulation

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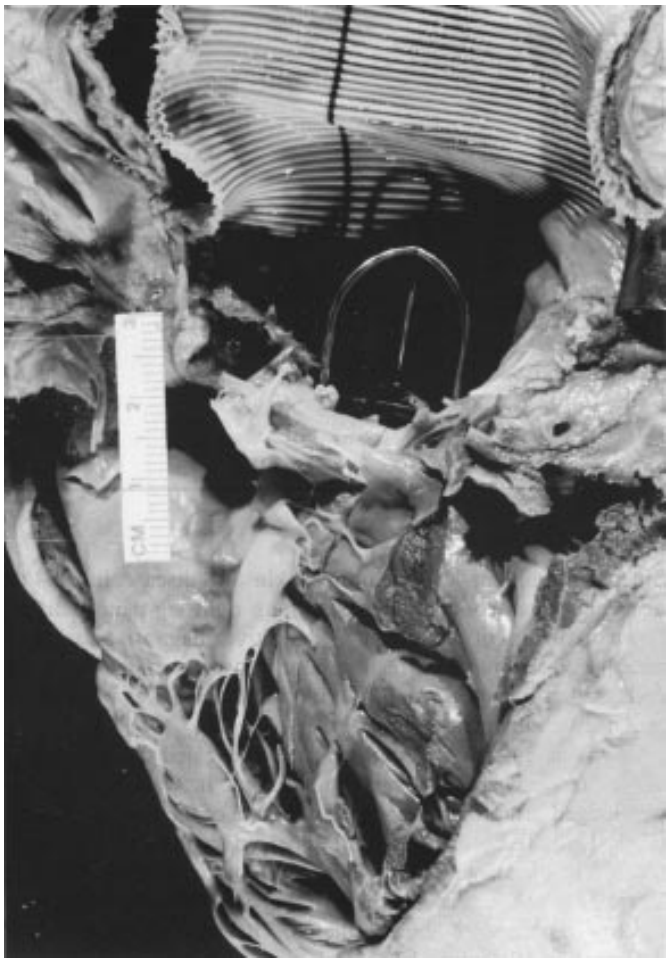

FIG. 1—*Prosthetic Starr-Edwards valve cage, without ball, is seen at the aortic root. A Dacron graft is in place at the site of repair of an ascending aortic aneurysm.*

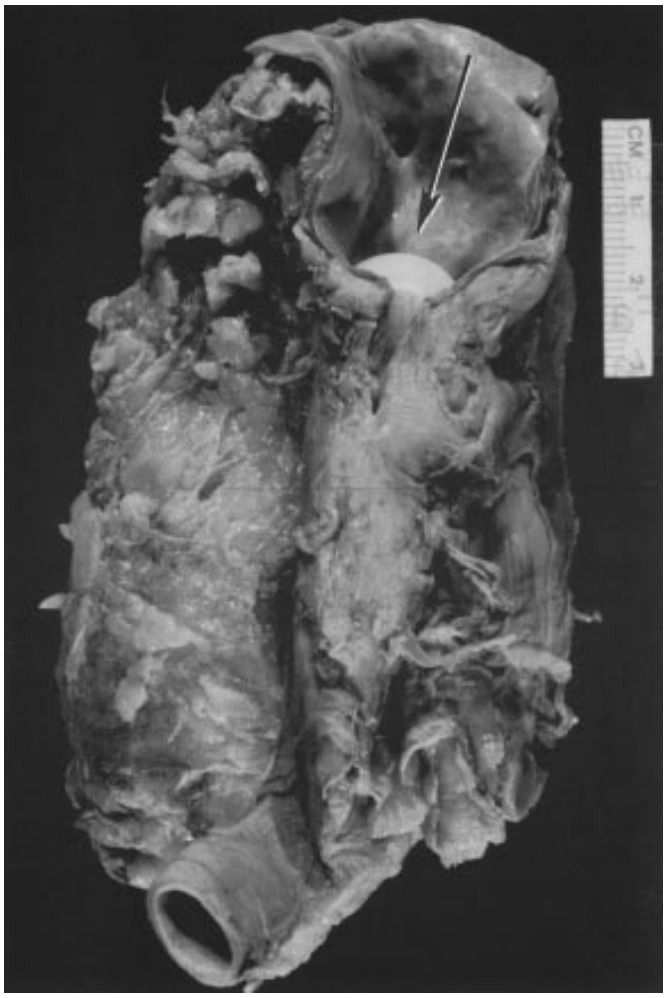

FIG. 2—*Distal aorta show embolized shiny white ball (see arrow) from Starr-Edwards prosthesis firmly wedged into its lumen just above the bifurcation.*

therapy, endocarditis, and mechanical degeneration/dysfunction (6). Overall ball valve dislodgment is a rare but universally lethal complication.

The Starr-Edwards ball valve prosthesis first became commercially available in April 1961 (7). The composition of the ball-valve prosthesis is as follows: (1) the cage is highly polished, uncoated casting of stellite 21, cobalt (61–63%), chromium (25.5–29.0%), molybdenum (5.0–6.0%) and nickel (1.75–3.75%) alloy used extensively in orthopedic appliances; (2) the sewing margin is knitted Teflon cloth similar to intracardiac patch-graft material. The cloth is bound to the metallic cage by means of a Teflon ring and etched Teflon thread windings bonded in silicone adhesive; and (3) the poppet is a ball of "medical grade" silicone rubber, this is a polysiloxane polymer plus a vulcanizing agent, dichlorobenzoylperoxide (7).

Recovered Starr-Edwards aortic prostheses have been studied. Ball variances observed include increases and decreases in size, deformation or lobulation, changes in color, and considerable softening in some (while others retain their original hardness) (7).

As noted by Starr et al. (7), it is difficult to ignore the abnormalities of valve implantation, in most of the cases of ball damage: leak, tight fit with impingement of aortic wall or cloth margin on ball pathway, misalignment of the prosthesis with respect to the aortic axis, and thrombotic stenosis of the prosthesis. All of these abnormalities increase the impact of the ball against the cage. The loss of random motion of the ball due to pathway impingement leads to more injury. The velocity of blood flow in the aortic root is increased by leak or obstruction around the ball. This in turn may drive lipids and other materials into the ball at a greater rate, leading to ball swelling and thus further impingement or obstruction, resulting in a vicious cycle with ball destruction (4,7,8). The most frequent abnormality observed in these aortic balls is surface dullness, with loss of glistening white appearance of the ball (which was not true for our case reported here), an early indication of ball degeneration. Discoloration appears to be of no functional significance (8).

Overall mechanical cardiac valvular prostheses are more popular than bioprostheses for heart valve replacement operations. Five different brands of mechanical heart valves are currently approved for implantation in the United States: Starr-Edwards models 1260 and 6120, Medtronic-Hall, St. Jude Medical, Omniscience, and CarboMedics (9). A review of long term results with these valves shows that the Medtronic-Hall and St. Jude Medical valves are favored (9,10).

In conclusion, dislodgment and embolization of the ball from a Starr-Edwards ball valve to the bifurcation of the abdominal aorta is a rare but lethal complication. Most commonly this event is due

to ball variance, sometimes with loss of structural support at the outflow tract. Therefore it is important to note any abnormalities of the ball and the cage and their implantation site at autopsy.

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