Acute Myocardial Infarction Secondary to
Thromboembolism from a Fractured
Prosthetic Aortic Valve

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SUMMARY
A case of coronary artery embolism from thrombus in a fracture in an aortic Starr-
Edwards prosthesis was documented at necropsy. This 40-year-old white man had
replacement of the aortic and mitral valves by ball-valve prostheses 5 years prior to his
death. Following surgery, he was able to return to full-time work. He did not return for
follow-up for 2 years, and when next seen he had an acute myocardial infarction and
left ventricular failure. An embolus to the left anterior descending coronary artery was
demonstrated at the postmortem examination. The mitral ball was intact, but the aortic
ball had two large fractures which contained thrombi.

Additional Indexing Words:
Ball-valve prostheses Coronary artery emboli Ball valve variance

With increasing use of prosthetic heart valves, the early detection and treatment of complications from such valves has become of paramount importance. The present case is unique in that a thrombus in a fractured ball of a Starr-Edwards prosthesis resulted in a fatal embolism to a coronary artery 5 years after operation.

Report of Case
The patient when first seen in April 1963 at the age of 35 years, had a 2-year history of intermittent dyspnea on exertion, peripheral edema, and hemoptysis. He related a history of rheumatic fever but denied syncope and angina. Physical examination revealed blood pressure of 130/80 with irregular pulse, cardiomegaly, and murmurs of mitral stenosis, aortic stenosis, and aortic insufficiency. Chest roentgenograms revealed an enlarged left atrium and a prominent pulmonary artery segment. An electrocardiogram indicated right axis deviation, right ventricular hypertrophy, and left atrial enlargement.

Cardiac catheterization demonstrated an end-diastolic gradient across the mitral valve of 8 mm Hg. The left atrial mean pressure was 14 mm Hg, going to 22 mm Hg with moderate exercise. The cardiac output increased from 5.0 to 7.0 L/min during exercise. There was no peak systolic gradient across the aortic valve. Mild mitral insufficiency and severe aortic insufficiency were demonstrated angiographically. The mitral valve was heavily calcified.

In June 1963 the aortic and mitral valves were replaced by Starr-Edwards prostheses by Dr. A. Starr of Portland, Oregon. The patient's postoperative course was uneventful; he was discharged from the hospital and continued to take penicillin as prophylaxis, an anticoagulant, and digitalis.

On March 3, 1964, postoperative heart catheterization demonstrated mean pulmonary artery wedge pressure of 8 mm Hg, increasing to 18 mm Hg during supine leg exercise. There was an end-diastolic gradient of 3 mm Hg across the prosthetic mitral valve at rest and a systolic gradient of 10 mm Hg across the prosthetic aortic valve at rest. The cardiac output was 6.2 L/min at rest and 8.9 L/min during exercise. Angiography demonstrated an apparently intact prosthetic valve with no aortic insufficiency and a normalized left ventricle and left atrium.

For the next 2 years the patient was followed in the outpatient clinic. Since there was no
Figure 1
An electrocardiogram recorded on 1-10-64 (upper) is suggestive of biventricular hypertrophy. (Lower) The one recorded several hours prior to death shows an acute anterolateral myocardial infarction.
Figure 2

(Left) A normal-sized heart on 3-4-64. The aortic prosthesis is not visible in this view. (Right) A portable chest roentgenogram taken the day of death shows massive cardiomegaly.

Figure 3

A sketch (left) of the photograph (right) demonstrates two large fractures in the aortic ball. A thrombus, present in each fracture, has been largely removed.
ventricular failure and he was in sinus rhythm, treatment with digitalis was stopped, but the use of an anticoagulant and prophylactic penicillin was continued. An electrocardiogram (Fig. 1) and vectorcardiogram, taken in January 1964 were suggestive of biventricular hypertrophy. In regular follow-up on May 25, 1966, he was asymptomatic and worked full time as a carpenter. His blood pressure was 120/80, and there was a systolic ejection murmur at the base. The sounds of the prosthetic valves were loud and distinct. Subsequently, he failed to return for any clinic appointments.

On February 2, 1968, while working, he suddenly experienced diaphoresis and crushing substernal pain extending to both arms. The pain persisted for 1 day and was followed by dyspnea and right upper quadrant tenderness.

Physical examination on hospital admission 4 days later revealed a dyspneic, diaphoretic white man. The pulse was regular at 96 and the blood pressure was 108/80. No petechiae were visible. The neck veins were markedly distended. Massive cardiomegaly with bilateral inspiratory rales and rhonchi was present. A systolic ejection murmur at the left sternal border and an apical diastolic cage rattle were heard. A murmur of aortic insufficiency was not heard. A pulsatile,
tender liver was palpable 7 cm below the right costal margin. The spleen was not palpable. An electrocardiogram showed an acute anterior myocardial infarction (fig. 1). The chest x-rays revealed marked cardiomegaly with pulmonary edema and pleural effusion (fig. 2). The prosthetic mitral and aortic valves appeared in normal position.

The hemoglobin value was 13.2 g%; serum sodium, 127 mEq/L; potassium, 4.9 mEq/L; arterial pH, 7.49; and pO2, 56 mm Hg. The prothrombin time was 23 sec. The value of blood urea nitrogen was 61 mg%. Eighteen hours following admission the patient suddenly became apneic, cyanotic, and hypotensive, and valve sounds were inaudible. Attempts at resuscitation were unsuccessful.

Necropsy disclosed marked pulmonary edema with lobular atelectasis and hemosiderosis. There were multiple infarctions in both kidneys and the spleen. The liver was congested. Petechial hemorrhages were noted in the gastrointestinal tract. Examination of the mitral prosthesis revealed an intact ball with endothelialization of the metallic struts half way to the apex of the valve cage and organized adherent thrombi around the valve ring. Examination of the aortic valve revealed two fractures. Each of these two fractures covered one third of the circumference of the ball (fig. 3). A large thrombus was lodged in the center of each defect. The coronary arteries were patent except for an embolus occluding the anterior descending coronary artery (fig. 4). No appreciable coronary artery disease is present.

Discussion

This report illustrates two major complications of prosthetic heart valves—thromboembolism and fracture of the silicone rubber ball. To our knowledge this is the first case reported of thromboembolism to a coronary artery from a fractured ball. Thrombus formation appears to be common on prosthetic heart valves.1-4 The incidence of emboli from such a thrombus has been estimated to be as high as 40% in patients surviving 2 years or longer.3, 4 Thrombi form primarily at the junction of the metal ring and struts, where the endocardium is sutured to the Teflon fixation ring.2, 5 Fortunately, fatal emboli are much less common, and adequate, well-controlled anticoagulation appears to decrease the incidence of thromboembolism.1, 4, 5 The state of anticoagulation was not known over the preceding 18 months, but it is safe to assume that it was not optimal. The large thrombus which formed in the clefts in the ball was the most likely site for the origin of the embolus.

Coronary emboli are often suspected in patients with prosthetic valves, but they are not frequently demonstrated.2, 4 In addition, coronary atherosclerosis and postoperative intimal proliferation of the proximal coronary arteries may lead to myocardial ischemia.3 Teflon emboli to the coronary arteries have been seen from a Hufnagel prosthetic aortic cusp.6 The incidence of coronary embolism is, however, unknown.

Ball variance, alterations in the physical and chemical properties of the silicone rubber ball poppet, appears to be limited to aortic prostheses.3, 7 The ball has increased in size, with resultant fixation of the ball in the cage7-9 or has decreased in size with resultant expulsion of the ball from the cage10, 11 When recognized, this may be corrected surgically.9, 12 In addition, grooves in the ball as well
as fragmentation and fracture have been reported.5, 7, 11

Because ball variance is a frequent postoperative finding, follow-up is necessary. Change in the prosthetic valve sounds, such as a decrease or disappearance in the valve opening sounds and loss of cage rattle, will alert one to the possibility of ball variance.5, 11 This change is best documented phonocardiographically, even though a fractured ball has been reported to be accompanied by an unchanged phonocardiogram.13 The appearance of a diastolic insufficiency murmur may or may not be associated with ball variance.7

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References


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