An Unusual Lethal Complication Associated with Starr-Edwards Prosthetic Aortic Valve Holder*

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This is the first report of an unusual fatal complication associated with the Starr-Edwards prosthetic aortic valve holder. The patient died 51 days after replacement of his aortic valve with a Starr-Edwards prosthetic aortic valve. The cause of death was coronary arterial embolus caused by a fragment broken off of the prosthetic aortic valve holder.

The common complications associated with prosthetic aortic valves are thromboembolism, hemolytic anemia, and mechanical malfunction due to ball variance. So far, there has been no report of any complication associated with the prosthetic aortic valve holder. Recently, we have observed such a complication. Fifty-one days after replacement of his aortic valve with a Starr-Edwards aortic valvular prosthesis, a patient died from coronary arterial embolus caused by a fragment broken off the prosthetic aortic valve holder. We report this case in order to bring the possibility of this intraoperative hazard to the attention of the many cardiac surgeons who regularly use this device.

CASE REPORT

A 56-year-old man was admitted to Queen Mary Hospital, Hong Kong, in February 1975 with fever, pain in the chest, and symptoms of heart failure for two days prior to admission. His heart was enlarged, with aortic systolic and diastolic murmurs. The VDRL test and six cultures of blood were negative. An x-ray film of the chest showed cardiomegaly and calcification of the aortic valve and the aortic knuckle. An electrocardiogram revealed left ventricular hypertrophy and episodic atrial fibrillation. Cardiac catheterization and cardiac angiographic studies were not performed. The patient responded to therapy with digoxin, furosemide, and a prolonged course of antibiotics.

The patient was readmitted in November 1975 with left ventricular failure. Replacement of the aortic valve was undertaken on Nov 26, 1975. Moderate hypothermia and coronary arterial perfusion was employed during the procedure for replacement of the valve. The surgical findings were:

1. an aneurysm (measuring 1.5 cm in diameter) of the right coronary sinus of Valsalva, which had not ruptured into the right ventricle, and
2. a tricuspid aortic valve that was grossly calcified and predominantly stenotic. The aortic valve was excised, and the neck of the aneurysm of the right coronary sinus of Valsalva was repaired using interrupted sutures with backing of felt pledges. A size-11 Starr-Edwards aortic prosthesis (model 1260) was inserted.

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The pathologic examination showed a myocardium consistent with myocardial infarction. The patient was repeatedly noted to have an acute cardiac infarct, with electrocardiographic changes indicative of extensive myocardial infarction. Immediate intensive care was instituted, including intravenous support with catecholamines and infusion of glucagon. A diagnosis of coronary arterial embolization was made. While arrangements were being made for emergency surgery under cardiopulmonary bypass, the patient went into irreversible ventricular fibrillation and died shortly afterwards.

Postmortem Findings

At necropsy the heart weighed 620 gm, with hypertrophy of both ventricles. There was an area of discoloration in the myocardium of the anterior and lateral walls of the left ventricle and in the anterior part of the ventricular septum, indicating a recent infarct. The tip of a blue plastic foreign body could be seen at the left coronary ostium. Figure 1 shows this foreign body after the left coronary artery had been exposed. At its site of impaction, and for a short distance distal to this, a small amount of fresh thrombus adhering to the coronary endothelial lining was detected, thus confirming its lodgment during life. The rest of the left coronary artery and its main branches showed no significant pathologic findings. The aortic valve prosthesis was in place; a few small pale thrombi were attached to its rim. The other cardiac valves were free from gross abnormality.

Microscopic examination confirmed the presence of recent and old myocardial infarcts. In addition, birefringent material engulfed by foreign-body type giant cells was seen in the lumen of a small branch of the left coronary artery.

Discussion

This is a case of a tragic accident that occurred during the course of a straightforward procedure for replacement of an aortic valve. The valve holders are being used regularly every day all over the world. To our knowledge, this is the first report of a lethal complication associated with the Starr-Edwards prothetic aortic valveholder. We are certain that the valve holder was intact while the aortic prosthesis was fitted into it. The offending strut-holding hook must have broken off (Fig 2) at some stage during the insertion of the prosthesis into the aortic root. This went unrecognized at that particular moment, and when the accident was discovered, all search to find the loose hook proved unsuccessful. Considerable time was spent looking for the broken fragment of the holder. The aorta was cross-clamped for a total of 79 minutes (the usual time taken in our center to insert an aortic prosthesis is around 40 minutes), and the duration of the bypass was 102 minutes. X-ray films were taken immediately, but no radiopaque foreign body was seen in the heart. The plastic component of the valve holder is not radiopaque. This was subsequently double-checked with separate x-ray films of the whole plastic component of the valve holder and the loose fragment which was recovered from the orifice of the left coronary artery at postmortem examination (Fig 1); however, underpene-
trated x-ray films showed the body of the plastic component to be opaque because it contained some radiopaque material in the thread that accommodates the screwable piston of the valve holder, although this does not show up on x-ray films using normal penetration. The strut-holding hooks of the holder have no such radiopaque material in them. We also tested the plastic component for its buoyancy; the plastic part as a whole sank in water and blood, as did the loose fragment.

It would appear that the loose fragment was lodged in a crevice among the papillary muscles in the left ventricular cavity. This fragment subsequently dislodged and was ejected into the aorta; in the process, it hit either the poppet or the cage of the valve, bounced back, and finally lodged in the orifice of the left coronary artery.

REFERENCES

Echocardiographic Recognition of Silent Aortic Root Dilatation in Marfan’s Syndrome

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Echocardiography has proven a useful and sensitive means to determine noninvasively the cardiac dimensions. This report describes the echocardiographic detection of progressive dilatation of the aortic root over an 18-month period, which led to death in a 33-year-old man with Marfan’s syndrome; however, at no time was the aortic dilatation evident on chest x-ray films. This patient illustrates the sensitivity of the echocardiogram in detecting and assessing the severity of disease of the aortic root not readily assessed by other noninvasive techniques and the value of serial measurements of aortic dimensions in patients with proven or suspected dilatation of the aortic root.

Dilatation of the aortic root, leading to aortic incompetence, dissection of the aortic wall, and rupture, is a major cause of death in patients with Marfan’s syndrome. Aortic disease associated with this syndrome maximally involves, and may be limited to, that proximal portion of the thoracic ascending aorta containing the annulus of the aortic valve and the sinuses of Valsalva. Since that region of the ascending aorta lies within the cardiac silhouette, conventional chest x-ray films may fail to reveal evidence of dilatation of the aortic root, necessitating the use of contrast angiographic studies of the aortic root in the radiographic evaluation of these patients.

Recently, the use of the noninvasive technique of echocardiography to measure the diameter of the aortic root in patients with Marfan’s syndrome has been described; however, whether echocardiographic studies can detect aortic root dilatation not evident on chest x-ray films has not been clarified. We studied a 33-year-old man with Marfan’s syndrome who died of aortic dissection, in whom serial echocardiographic examinations performed over an 18-month period prior to his death revealed massive dilatation of the aortic root, which was at no time apparent on posteroanterior and lateral chest x-ray films.

CASE REPORT

This 33-year-old man was recognized as having Marfan’s syndrome for eight years prior to his death. From the time of initial examination, he was tall (185.4 cm; 6 ft 1 in) and had an arm span exceeding his height by 3 inches, cutaneous...