

CASE REPORT

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Coronary Artery Compression by Teflon Pledget Granuloma Following Aortic Valve Replacement*

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ABSTRACT: We describe a massive fatal posterior myocardial infarct resulting from compression of the right coronary artery by a Teflon pledget granuloma in a patient who had undergone aortic valve replacement six months before death. Other iatrogenic causes of coronary artery occlusion or compression are discussed in this paper. To our knowledge this is the first report of this surgical complication.

KEYWORDS: forensic science, forensic pathology, thoracic surgery adverse effects, postoperative complications, coronary vessels injury, heart valve prosthesis, aortic valve

History

A 47-year-old white male was found unresponsive in a horse stall by his wife. He was found to be in asystole by paramedics and pronounced dead a short time later in a hospital emergency department.

At age 12 he had acute rheumatic fever with subsequent development of aortic stenosis and mitral regurgitation. Six months before death he underwent cardiac catheterization which revealed widely patent coronary arteries. Several days later aortic valve replacement with a 25-mm homograft and insertion of a 34-mm Carpentier-Edwards annuloplasty ring in the mitral annulus were performed. Teflon pledgets were used to buttress the aortotomy incision. Postoperative laboratory and electrocardiograms showed no evidence of myocardial infarct. For several months following discharge, he had mild congestive heart failure, but this began to resolve six months before death.

Autopsy Findings

Significant autopsy findings included an 860-g heart with compression of the proximal right coronary artery by a 0.7 cm epicardial mass that histologically was found to be extensive granulomatous inflammation around and within a Teflon pledget. There was mild,

nonocclusive intimal hyperplasia in the compressed segment of the right coronary artery (Fig. 1). There was an extensive old infarct of the posterior (inferior) wall of the left ventricle, the posterior portion of the interventricular septum, and the posterior wall of the right ventricle.

Discussion

In summary, we describe a 47-year-old man who had a massive myocardial infarct in the distribution of the right coronary artery, which was compressed by a Teflon pledget granuloma. The intimal hyperplasia probably resulted from turbulent blood flow in the region of the inwardly compressed coronary artery causing endothelial injury. It is possible that narrowing caused by the compression and intimal hyperplasia was increased further by coronary spasm.

Causes of coronary obstruction or compression complicating aortic valve replacement are listed in Table 1. In most of the cases there was preoperative coronary angiography demonstrating widely patent arteries. All patients had documented ischemia or infarct in the distribution of the affected artery.

The most commonly and recently reported cause of coronary narrowing resulting from mitral or aortic valve replacement is coronary cannulation injury. In most cases the result of this injury is intimal hyperplasia. The mechanisms of injury include endothelial avulsion or disruption by a balloon cannula, especially if a large catheter is forced or wedged into the vessel, and endothelial damage from a jet lesion from a high pressure catheter tip (1-3). One patient (1) had re-implantation of the coronary arteries into an aortic prosthesis after aortic valve replacement, whereas in the others the coronary arteries remained in aorta.

Intimal hyperplasia is a common (30-40% incidence) complication of balloon angioplasty, but is unusual (1 to 3% incidence) following aortic valve replacement (2). Disruption of the internal elastic lamina is especially likely to result in intimal hyperplasia as is the ϵ -4 allele of the apolipoprotein E phenotype, which is associated with a high incidence of coronary atherosclerosis (2). Intimal hyperplasia presents clinically between 1 $\frac{1}{2}$ months and one year after surgery. Coronary artery dissection is another complication of cannulation (4).

There have been three cases of right coronary occlusion by sutures closing the aortotomy incision (5). The left anterior descending coronary artery was occluded by suture during treatment of postoperative hemorrhage (6) in one patient.

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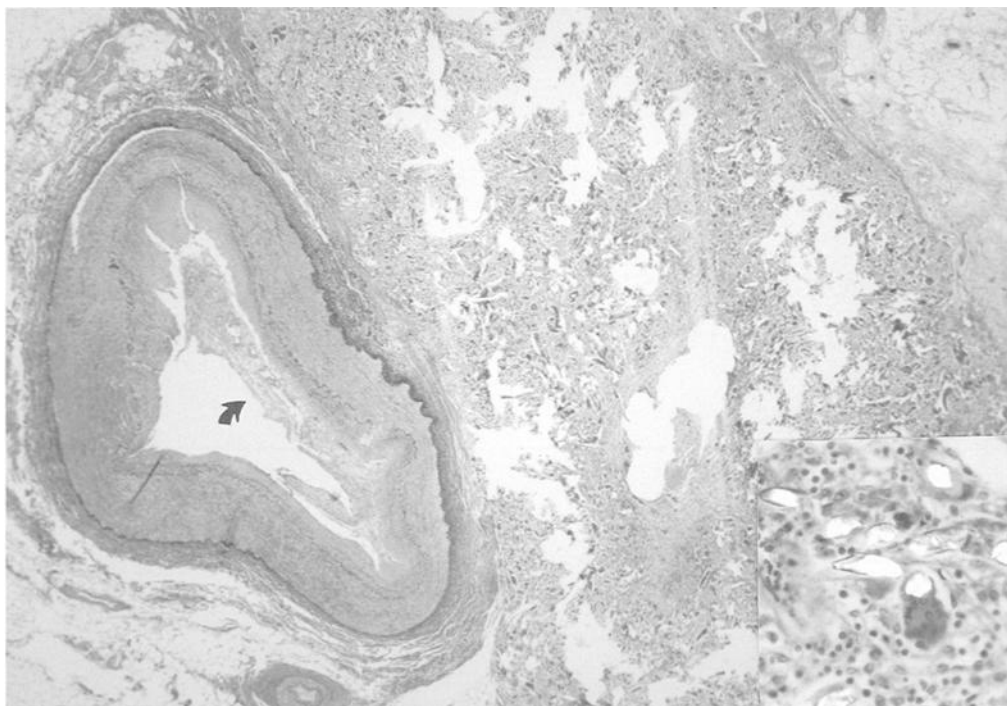


FIG. 1—Right coronary artery compressed by large Teflon granuloma (right). There is intimal hyperplasia (arrow) within the lumen. (Elastic trichrome; original magnification $\times 40$). Inset (in right lower corner): Birefringent strands of Teflon. (Elastic trichrome, polarized light. Original magnification $\times 200$).

Thrombi extending directly from the aortic valve prosthesis (6) and emboli arising from prosthetic valves (3,6) were reported in the 1970s. Embolization occurred from immediately postoperatively to one year after surgery, whereas the thrombi presented 2 weeks postoperatively.

In two cases a coronary artery ostium was obstructed following aortic valve replacement. In the first reported case (6) the ostium was occluded by a ball valve and in the other case the prosthesis was too large for the annulus and was placed 2 cm below the left coronary ostium. The resultant ostial intimal hyperplasia was thought to be caused by turbulence from the disc occluder moving in front of the coronary ostium (7).

Two cases of iatrogenic aortic trauma resulting in coronary ostial narrowing have been reported. In the first case (4) aortic dissection occurred during surgery and extended to the left coronary artery. In the other patient, rough handling of the aortic retractor caused thrombotic occlusion of the right coronary artery 20 h after surgery (5).

TABLE 1—Coronary artery compression/occlusion complicating aortic valve replacement.

Cannulation
intimal hyperplasia (months)*
dissection (immediately to one day postop)
thrombosis (immediately)
Suture occlusion (minutes)
Embolization from prosthetic valve (immediately postop to 2½ years)
Thrombi from prosthetic valve (2 weeks)
Aortic injury with associated coronary occlusion (during surgery, up to 20 h)
Valve-mediated coronary stenosis (one day and 6 months)
Coronary compression by pledget granuloma (6 months)

*Usual time from surgery to clinical presentation.

Teflon is widely known to produce granulomatous inflammation. Foreign body granulomas have been observed in aortic aneurysms which have occurred following Teflon patch aortoplasty for coarctation (8).

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