Coronary ostial stenosis after aortic valve replacement

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Key words:
Angina; Aortic stenosis; Aortic valve replacement; Left main stem stenosis.

Coronary ostial stenosis is a life-threatening complication of aortic valve replacement. We describe the case of a patient who developed symptoms and signs related to coronary insufficiency 4 months after aortic valve replacement.

In view of a coronary angiogram revealing a 99% left main stem stenosis, an urgent revascularization procedure was performed.

Intraoperative inspection revealed a dense fibrous reaction of the aortic root involving the left coronary ostium.

The postoperative course was uneventful and since the operation the patient remained asymptomatic.

The possible mechanisms involved in the pathogenesis of coronary ostial stenosis after aortic valve replacement and the surgical strategy to be employed for the treatment of this complication are discussed.

(Ital Heart J 2002; 3 (2): 133-136)

Received July 27, 2001; revision received December 11, 2001; accepted January 7, 2002.

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Introduction

Coronary ostial stenosis following aortic valve replacement is a clinically recognized entity with a reported incidence varying between 1 and 3.5%[1,2].

Symptoms, usually including a rapidly progressive anginal syndrome, develop within the first months of surgery[3].

Two pathogenetic mechanisms have been proposed to explain iatrogenic coronary ostial stenosis: traumatic intimal damage of the coronary artery due to coronary perfusion[2,4-7] and fibrous proliferation in the aortic root secondary to turbulent flow around a prosthetic valve[8-11].

Whether coronary ostial stenosis is a complication of aortic valve replacement or a complication of coronary artery perfusion is still a controversial topic[12].

We have recently observed a case of left coronary ostial stenosis developing after aortic valve replacement. The most plausible explanation of this complication was the intimal proliferation of fibrous tissue consequent to the turbulent flow around the prosthetic valve.

Case report

A 52-year-old man was referred to our department for aortic valve replacement because of severe aortic stenosis.

Rest ECG showed sinus rhythm and signs of left ventricular hypertrophy.

Echocardiography revealed a normal left ventricular function and a Doppler peak gradient across the aortic valve of 88 mmHg.

Coronary angiography confirmed a normal coronary anatomy (Fig. 1).

The patient was submitted to a minimally invasive aortic valve replacement through a ministernotomy. The operation was performed with the patient in moderate systemic hypothermia; both coronary ostia were cannulated using cannulae with a soft silicone tip (Medtronic, Minneapolis, MN, USA) for the intracoronary injection of crystalloid cardioplegia. A 25 mmHg St. Jude Medical Hemodynamic Plus aortic valve (St. Jude Medical, St. Paul, MN, USA) was implanted using single sutures of ethibond 2-0 (Ethicon, Somerville, NJ, USA).

The postoperative course was uneventful and the patient was discharged 7 days after surgery.

Four months following the operation the patient developed exertional angina pectoris and a cycloergometric test revealed ischemic signs in the antero-lateral region.

Echocardiographic Doppler examination showed a normal function of the mechanical valve and of the left ventricle.

Coronary angiography revealed a 99% stenosis of the left main stem (Fig. 2); for

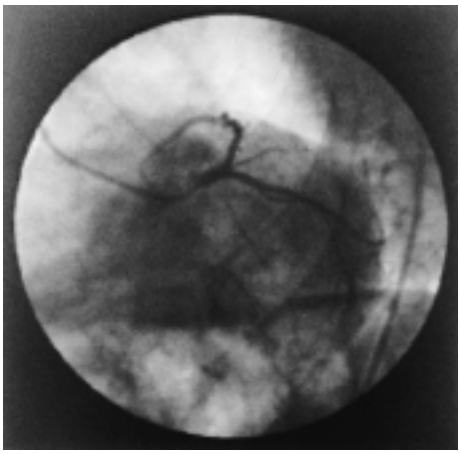

Figure 1. *Preoperative left coronary angiogram showing a normal left main coronary artery.*

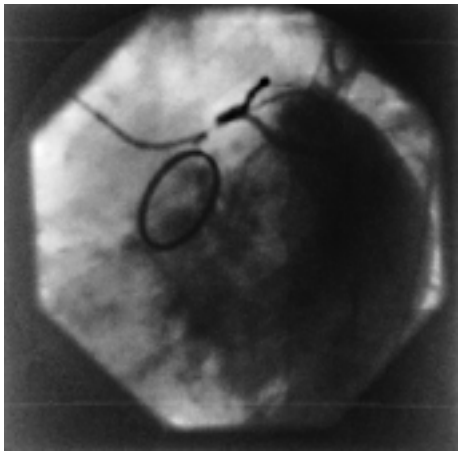

Figure 2. *Postoperative left coronary angiogram showing a 99% left main stem stenosis.*

this reason, the patient was scheduled for an urgent surgical myocardial revascularization procedure.

At surgery, the ascending aorta was opened and inspection confirmed a widespread thickening of the aortic wall while the left coronary ostium appeared to be pinpoint in size and enshrouded by a dense fibrous reaction.

Surgical angioplasty of the left main coronary artery (LMCA) was performed using the posterior approach: an incision was made in the right-posterior aortic wall, close to the left coronary ostium; the aortic root was then unrolled towards the left and finally the incision was extended towards the posterior aspect of the LMCA.

A venous patch was used to enlarge the LMCA and the adjacent aortic incision.

Two adjunctive procedures were performed: the left internal mammary artery (LIMA) was anastomosed to the left anterior descending coronary artery and the aortic prosthesis was rotated by 90° with respect to the original orientation with the two leaflets opening towards the coronary ostia.

Histologic examination revealed intimal proliferation and fibrosis of the aortic wall. The patient had a normal postoperative course and since surgery he has not presented with symptoms. He was not submitted to angiographic evaluation following the surgical procedure.

Discussion

Iatrogenic coronary ostial stenosis is a life-threatening complication of aortic valve replacement.

Symptoms are rapidly progressive and include angina pectoris and sudden-onset left ventricular failure or acute pulmonary edema that usually develop within 6 months of surgery[3].

The ostial lesion is most frequently confined to the left coronary artery although it can involve the right or even both coronary arteries[9,13].

Many clinical reports support the hypothesis that this complication is most probably secondary to the local pressure necrosis produced by perfusion catheters. This, in turn, leads to scar formation and subsequent obstruction of the coronary arteries. In our opinion, even the hypothermic cardioplegic solution may contribute to the damage of the intima with subsequent fibrous proliferation.

Winkelmann et al.[14] showed that this complication can be avoided by resorting, for myocardial protection, to retrograde delivery instead of direct antegrade cardioplegia.

In the same paper, the authors suggested that there might be a genetic predisposition for the development of such a lesion. This, in view of the fact that 70% of affected subjects in their series had an epsilon 4 allele apolipoprotein E genotype compared to 10-15% in a control group. This could perhaps explain the low incidence of coronary ostial stenosis in spite of the extensive use of direct coronary artery perfusion.

Hazan et al.[5] described a post-perfusion stenosis of the common left coronary artery in a patient submitted to replacement of the ascending aorta but not of the aortic valve. This renders the hypothesis of the involvement of the coronary ostia in the fibrous reaction secondary to turbulent flow unlikely.

In our opinion, although the mechanism of intimal injury at the coronary ostia by perfusion cannulae or by hypothermic cardioplegia cannot be completely excluded, the present case report is a reasonable confirmation of what extensive pathological data and clinical reports have already described.

In a study of 224 necropsy patients, Roberts et al.[9] described extensive aortic root fibrosis in 66 patients submitted to aortic valve replacement. In 37 of these patients the fibrous reaction also involved the coronary ostia.

The finding of intimal proliferation and fibrosis with involvement of the coronary arteries by

nonatheromatous fibrous tissue in patients with coronary ostial stenosis following aortic valve replacement has been confirmed by Yates et al.[4] and by Uys[15].

Moreover, this complication has been reported to occur even in patients in whom either continuous coronary perfusion was not used[10] or in whom at no time during surgery were the coronary arteries cannulated[11].

Assuming that turbulent flow across the aortic prosthetic valve may play a role in determining fibrous proliferation in the aortic root, one wonders about the true importance of a specific orientation of the prosthetic leaflets in the aortic position.

As stated by Baudet et al.[16], the St. Jude Medical bileaflet prosthesis requires specific orientation to provide optimal hemodynamics and subsequently to reduce turbulence in the downstream areas: one of the pivot guards has to be placed in the center of the right coronary cusp, that is, with the long axis of the prosthesis perpendicular to the septum. This orientation permits optimal hemodynamic utilization of both valve areas and subsequent prevention of turbulence.

As this was exactly the orientation of the implanted valve, the present case probably suggests that even a reduced turbulence of the flow across the prosthesis can produce an extensive fibrous reaction. This hypothesis is supported by the reported case of aortic root fibrosis in a patient submitted to aortic valve replacement with a porcine xenograft[11].

Although critical isolated stenosis of the LMCA is usually treated by conventional bypass surgery, it seems clear that direct surgical angioplasty provides some advantages: it reduces the probability of definitive occlusion of the LMCA[17,18] and avoids, according to the prizometer principle[19], the drop in the head of pressure caused by retrograde perfusion.

This technique deserves an important place in the array of surgical strategies, provided that well-defined contraindications (involvement of the distal bifurcation, heavy calcifications) are respected.

Currently, LMCA surgical angioplasty can be expected to be just as safe and reproducible. Increasingly good results have been reported since the introduction of retrograde cardioplegia and with the expanding experience with surgical techniques such as the use of the saphenous vein patch instead of the pericardial one and with the anterior approach to the LMCA. The saphenous vein onlay patch should be preferred in view of the lack of fibrinolytic activity of the pericardium. The posterior approach to the LMCA should be abandoned because the exposure of the distal part of the LMCA is less than optimal and because incising the posterior wall is more hazardous than incising the anterior wall which is protected from the head of pressure by the acute angle that it makes with the aortic wall[20].

While in their first series of 21 patients Dion et al.[21] reported that only 78.3% of all surgical LMCA angioplasties could be regarded as successful, more recently[20] the same authors documented better immediate and midterm results with LMCA patency demonstrated in 87% of patients at an average of 38 months of follow-up; the anterior approach to the LMCA was the only one used and a saphenous vein onlay patch was preferred to a fresh autologous pericardial one. It is noteworthy that this last series included 2 patients previously submitted to an aortic valve replacement (in whom the LMCA stenosis consisted of fibrous scar tissue) and 18 patients in whom the etiology was probably non-atherosclerotic but "idiopathic" or "fibro-muscular dysplasia".

Moreover, recently, Meseguer et al.[22] have described their experience with 7 cases of LMCA angioplasty. These authors reported no perioperative morbidity and mortality and good midterm results in 5 of the 7 patients, all of them remaining symptom-free after a mean period of 51 months. Early restenosis occurred in 2 patients in whom a pericardial patch was used. The anterior approach was preferred, being adopted in 6 patients.

Prete and Turina[23] have applied this procedure to 5 patients with non-atherosclerotic disease of the coronary arteries reporting very good results.

Excellent results have also been reported by Moro et al.[24]: at a mean follow-up of 62 ± 21 months the patients were free from angina and did not necessitate a second surgical procedure because of the recurrence of stenosis. In all cases, an anterior approach to the LMCA was employed.

Even though the anterior approach seems to be the preferred one, some surgeons[25] have advocated the incision of the coronary ostium at its obtuse, rather than its acute angle of insertion into the aortic wall. This, in order to avoid the development of kinks in the onlay patch as described for the anterior approach.

We believe that, despite the increased technical difficulties and the words of caution expressed by some surgeons[26], LMCA angioplasty should be offered to patients, such as those with LMCA stenosis following aortic valve replacement, who would particularly benefit from the ideal hemodynamic characteristics restored.

One must consider however that, in such patients, turbulent flow in the aortic root and the subsequent possibility of delayed fibrosis and of restenosis of the coronary ostia cannot be completely abolished. Therefore, as in the present case, we think that it is advisable to anastomose even a LIMA to the left descending coronary artery in order to prevent late myocardial ischemia and eventually a new surgical procedure in patients already submitted to a first reoperation.

In conclusion, coronary ostial stenosis must be suspected in patients who develop ischemic cardiac pain after aortic valve replacement.

Although it is unlikely that soft rubber catheters intermittently used can be sufficiently traumatic to produce intimal damage, this possibility cannot be exclud-

ed and alternative methods of myocardial protection must be considered whenever possible.

Optimal orientation of the St. Jude Medical bileaflets prosthesis, with subsequent reduction of turbulence, reduces the risk of fibrous proliferation but does not seem to totally prevent it.

For this reason, correction of this complication should be accomplished through a surgical angioplasty of the left main stem eventually guarded by a "stand-by" LIMA directed to the left coronary system.

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