Association between post-pericardiotomy syndrome and coronary occlusion after aortic valve replacement

IVAN DE SCHEERDER, MARC DE BUYZERE, DENIS CLEMENT

From the Department of Cardiology, Akademisch Ziekenhuis, State University Ghent, Belgium

SUMMARY Fever, leucocytosis, and pericardial and pleural effusions developed after the first postoperative week in a 56 year old man who had undergone aortic valve replacement. Four months later, coronary angiography showed bilateral proximal stenosis of the coronary arteries. In this patient post-pericardiotomy syndrome and subsequent coronary artery stenosis were thought to be associated and an immunological mechanism was suspected.

The post-pericardiotomy syndrome is a common complication after cardiac surgery.1-4 It is characterised by fever, leucocytosis, and signs of pericardial and often pleural reaction, frequently with effusion and pneumonitis, occurring or persisting after the first postoperative week. Although this syndrome has been well defined clinically, its aetiology remains unclear. The fact that this syndrome appears after the first postoperative week and is sensitive to corticosteroid therapy suggests that immunological factors are involved in its pathogenesis. The presence of antiheart antibodies and circulating immune complexes in sera of most cases supports this view.24-6

Previous studies have suggested that saphenous vein graft occlusion is more common in patients who develop the post-pericardiotomy syndrome after coronary artery bypass grafting.7 Pericardial adhesions involving the wall of the vein graft are thought to be responsible for the higher occlusion rate.8

We describe a patient in whom post-pericardiotomy syndrome developed after aortic valve replacement and in whom concurrent coronary artery occlusions required grafting.

Case report

A 56 year old man was admitted for cardiac catheterisation. He was known to have had a heart murmur for 38 years. He complained of progressive exertional dyspnoea for about six months and for six weeks he had had angina. Cardiac catheterisation showed a resting peak systolic aortic valve gradient of 60 mm Hg and an aortic valve area of 0.62 cm², but no important abnormalities were demonstrated at coronary arteriography.

He was transferred to the surgical department for insertion of a Xenomedica A28 aortic prosthesis. The immediate postoperative course was uncomplicated.

Eight days after operation, however, the patient complained of progressive dyspnoea and retrosternal pain, and he had fever of 39°C. Auscultation showed reduced heart sounds and decreased breath sounds at the left base. Chest x ray findings showed an obviously enlarged cardiac silhouette, pulmonary congestion, a large left pleural effusion, and a small right pleural effusion. Echocardiography showed a pericardial effusion. The mitral and tricuspid valves were normal and no vegetations were seen on the prosthetic aortic valve. A ventilation/perfusion lung scintigram did not suggest a pulmonary embolism. Sputum, urine, and serial blood samples were culture negative. Serum enzymes (creatine kinase, creatine kinase MB, lactate dehydrogenase, and transaminases) remained normal.

Fig. 1 shows the white blood cell count, temperature, anti-heart antibodies (determined by an indirect immunofluorescence method), and circulating immune complexes (determined by polyethylene glycol precipitation and the C₁₉₁₄ method). Because post-pericardiotomy syndrome was suspected salicylates (2 g a day) were started and continued for three weeks with excellent clinical improvement.

Four months after discharge from hospital the patient complained of severe substernal pain radiating to the left shoulder and arm. This responded well to nitrate treatment. Thallium scintigraphy
indicated pronounced reversible ischaemia in the apical, midseptal, and posterobasal area of the heart. Coronary angiography showed 80% stenosis of the left main coronary artery and 70% proximal stenosis of the right coronary artery. The patient was transferred to the department of cardiovascular surgery for a coronary artery bypass operation. During operation a myocardial biopsy specimen was taken from the left ventricle. Despite perioperative treatment with salicylates the patient again had fever between the seventh and twelfth postoperative day which was associated with a slight leucocytosis.

Histological examination of the biopsy specimen showed pericardial thickening. The vascular walls were also thickened with an excess of mononuclear cells. Direct immunofluorescence staining showed small granular deposits in the interstitium, mainly along blood vessels, which could be stained for IgG, IgM, and the C3 component of complement (Fig. 2a and b).

Discussion

In this patient aortic valve replacement for aortic stenosis was complicated by post-pericardiotomy syndrome and concurrent proximal coronary artery stenoses. Aortic valve replacement can be complicated by acute obstruction of the coronary ostium by the ring of stent on the aortic prosthesis. Moreover cannulation induced injury was reported to cause operative deaths by coronary artery dissection and by compression due to ecchymosis or obstruction. These complications increase immediate postoperative morbidity.

Late postoperative obstructive coronary disease has been reported after aortic valve replacement. Lesage et al described six patients with coronary disease after heart valve replacement. In one patient they found circumferential narrowing of both coronary vessels caused by internal proliferation in absence of any coronary atherosclerosis. Internal thickening in the aortic root was also reported. The intimal surfaces of the aorta and the coronary arteries may be damaged during operation or cannulation for installation of perfusion catheters. Removal of the endothelial cells exposes the underlying media. Healing usually occurs by proliferation of new endothelial cells, but occasionally severe
intimal thickening can narrow the coronary arteries and lead to myocardial ischaemia. In animal models removal of the endothelium causes smooth muscle cell proliferation and their migration from the arterial media into the intima. These processes are essential to the development of the atheromatous plaque.12–14

The association of coronary stenoses with the post-pericardiotomy syndrome in the present case is especially interesting. Saphenous vein graft occlusion was reported to be more common in patients who developed post-pericardiotomy syndrome after coronary artery bypass grafting.7 Pericardial adhesions involving the wall of the vein grafts were thought to be responsible for the higher occlusion rate.8 De Scheerder et al8 demonstrated a close correlation between anti-heart antibodies and circulating immune complexes in post-pericardiotomy syndrome which suggested that precipitation of these circulating immune complexes in the pericardium may be involved in the pathogenesis of this syndrome. Deposition of circulating immune complexes on the vascular wall may be part of the pathogenesis of vascular disease.15,16 The demonstration of IgG, IgM, and C3 in the vascular wall by direct fluorescence in the biopsy specimen taken from our patient during coronary bypass operation supports this hypothesis.

References