Aortic Valve Replacement after Previous Coronary Artery Bypass Grafting in a Patient with Antiphospholipid Syndrome

We report a 55-year-old female patient with antiphospholipid syndrome secondary to systemic lupus erythematosus. The patient had undergone coronary artery bypass grafting for myocardial infarction due to left main trunk stenosis at the age of 52. Subsequently, she developed aortic insufficiency and underwent aortic valve replacement without any hemodynamic or hemostatic problems. Both coronary and valve disease should be considered in patients with antiphospholipid syndrome secondary to systemic lupus erythematosus. (JJTVCVS 1998; 46: 257-259)

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Genichi Sakaguchi, MD, Kazuaki Minami, MD, Shogo Nakayama, MD, and Hiroshi Tsuneyoshi, MD.

The antiphospholipid syndrome (APS) has been defined by the presence of antiphospholipid antibodies and venous or arterial thrombosis, recurrent abortion, or thrombocytopenia. Recently, the association of cardiac (valvular disease, myocardial infarction, and left ventricular dysfunction) and cerebral diseases with APS has also been reported. In particular, systemic lupus erythematosus (SLE) patients with APS have a higher prevalence of valvular involvement than those without APS. Aortic valvular lesions are relatively rare compared with those of mitral valve. Most of the cases are not of hemodynamic significance and rarely require valve replacement. Very few cases of re-do cardiac operation in APS patients have been reported in the literature. Here we report a successful aortic replacement after previous coronary artery bypass grafting in a APS patient secondary to SLE.

A 55-year-old woman began prednisolone therapy in 1973 following the diagnosis of SLE by serology and renal biopsy. Subsequently, she developed thrombocytopenia and systemic hypertension, and in 1990, she suffered from cerebral infarction with transient paralysis of the right extremities. In 1993, she underwent coronary artery bypass grafting for acute myocardial infarction due to left main trunk stenosis (left internal thoracic artery to left anterior descending artery, great saphenous vein to diagonal and obtuse marginal branch sequentially). At the first operation, mild aortic regurgitation was noted by echocardiography, and it gradually became worse. In 1996, she developed severe dyspnea with acute pulmonary edema necessitating emergent admission to our hospital. Chest X-ray film showed pulmonary edema (Fig. 1). Echocardiography showed severe aortic regurgitation and diffuse hypokinesis of the left ventricle. Catheter investigation revealed aortic dilatation with severe aortic regurgitation. LVEF was 41%. Coronary angiography demonstrated 90% stenosis in the left main trunk, the left anterior de-
Fig. 1. Chest X-ray film at admission shows lung edema.

Fig. 2. Microscopic examination of the excised valve shows hyalinous thickening and deposition of acid mucopolysaccharide. There was no finding of Libman-Sacks endocarditis. (H.E stain, ×25 above, ×50 below)

ascending artery and the circumflex artery, while the internal thoracic artery and the saphenous vein graft were patent. The left internal carotid artery, right common iliac artery, and left external iliac artery were totally occluded. In laboratory tests, lupus anticoagulant and false positive VDRL were detected, but anticardiolipin antibodies were negative. She underwent aortic valve replacement on April 16, 1996. The left axillary artery was exposed prior to the sternotomy for emergent cannulation in case of hemorrhage from the splitting of the sternum. Cardiopulmonary bypass was instituted through aortic and bicaval cannulation. Pulsatile systemic perfusion was used for brain and renal protection. After clamping the aorta and the patent left internal thoracic artery, retrograde intermittent cold cardioplegia was used for myocardial protection. The aortic root was dilated to a diameter of 45mm. The aortic valve showed diffuse thickening but no verrucous lesion. The aortic valve was excised and replaced with a 21mm St. Jude Medical valve. No hemostatic problems were encountered and post-operative recovery was uneventful.

Microscopic examination of the excised valve showed hyalinous thickening and deposition of acid mucopolysaccharide. There was no finding of Libman-Sacks endocarditis (Fig. 2).

Comment

In most patients with APS, although valvular lesions are of minor hemodynamic significance, severe cases require surgical replacement. Our patient had mild aortic regurgitation at the first operation and this gradually became worse. It is controversial whether the patient should have had the aortic valve replaced at the first CABG. Considering the tendency toward progression of the valvular lesion because of her systemic disease and the risk of re-operation for AVR, AVR at the time of CABG might have been a better choice. Pericardial adhesion was not dense probably because of long-term steroid therapy, and we could easily dissect and clamp the patent left internal thoracic artery. Since the left ventricular function was impaired and there were some stenotic lesions in the left coronary artery, appropriate cardioplegic perfusion was required for optimal cardiac protection. Retrograde cardioplegic perfusion seemed very beneficial in this regard. We chose a mechanical valve because of its greater durability compared with a bioprosthesis. However, a bioprosthetic valve may be