a calcified tear in the noncoronary leaflet with perforation was observed. It is difficult to determine whether the tear was primary or calcium related.[4] Probably, the presence of these calcium depositions encouraged tearing. We speculate that the postoperative finding of elevated peak transvalvular gradient was associated with a dynamic obstruction in the left ventricular outlet, and this situation led to mechanical stress during the systolic phase. The right coronary leaflet had intrinsic and vegetating calcifications with stiffness but without tearing. In addition, the commissural areas were severely calcified. These areas are designed to absorb and distribute the mechanical stress during the cardiac cycle. Thus when the leaflets are opened, they are completely free of stresses. Although a stent distortion is extremely rare and unusual, we can not reject this possibility completely.

The Sorin Biomedica Cardio technical report concludes that in presence of severe aortic stenosis and massive calcification before the implant, calcifications may have been triggered by the presence of such risk factors as hyperlipidemia and hypercholesterolemia.

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Complete destruction of a tissue-engineered porcine xenograft in pulmonary valve position after the Ross procedure

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The Ross procedure is an attractive technique for aortic valve replacement using a viable pulmonary autograft. Current data from the German Ross Registry show favorable clinical and hemodynamic results.[1] Pilot work on the use of a tissue-engineered decellularized porcine xenograft for right ventricular outflow tract reconstruction has reported excellent early and midterm results.[2,3] We report a case of recurrent right-sided heart failure after the Ross procedure caused by complete destruction of the pulmonary xenograft.

CLINICAL SUMMARY

A 61-year-old male patient underwent the Ross procedure for calcified aortic valve stenosis and received a tissue-engineered porcine xenograft (Matrix P, diameter 28 mm; AutoTissue GmbH, Berlin, Germany) for reconstruction of the right ventricular outflow tract. Four weeks after surgery, severe dyspnea developed in the patient, and the chest x-ray indicated abundant right-sided pleural effusion without evidence of pulmonary infection in computed tomography, bronchoscopy, or serology. Because of refractory pleural transudation, the patient underwent right-sided partial pleurectomy. Ten months later, he presented with severe dyspnea and progressive weight loss (35 kg since Ross operation). Outpatient echocardiography showed a pulmonary peak velocity of 1.5 m/s without any signs of regurgitation, preserved left ventricular ejection fraction, and minimal aortic autograft regurgitation. Because objective physical fitness was good (100W in ergometry), the patient was scheduled for ambulatory follow-up 2 weeks later. Echocardiography revealed enlargement of the right ventricle and moderate hypertrophy. Morphologic evaluation of the pulmonary xenograft was scarcely feasible. Mean and peak pulmonary pressure gradients were 10 and 22 mm Hg, respectively; peak velocity was 1.9 m/s, and there was mild early diastolic regurgitation. Systolic pulmonary pressure was estimated at 55 mm Hg, indicating moderate to severe pulmonary hypertension. Two weeks later the patient was hospitalized again for severe dyspnea and newly developed left-sided pleural effusion. Transthoracic echocardiography demonstrated progressive

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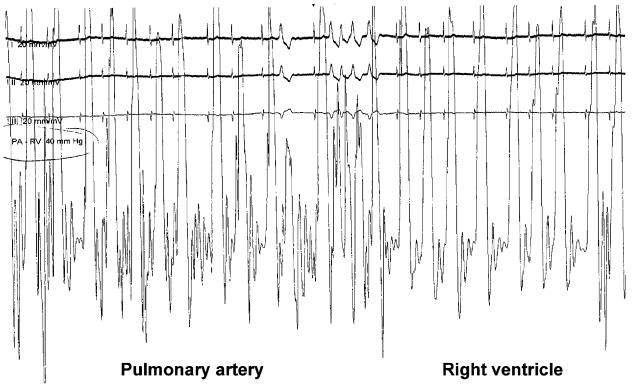

FIGURE 1. Right-sided heart catheterization showing pressure equilibration at 40 mm Hg between the pulmonary artery and the right ventricle as the result of free regurgitation into the right ventricular outflow tract.

right ventricular and right atrial dilation. Color Doppler echocardiography of the xenograft showed a slight, short subvalvular regurgitation jet in diastole. Artefacts caused by the prosthesis made exact quantification by color Doppler uncertain, but we observed a short, early diastolic, triangular, continuous-wave insufficiency signal and a short pressure half-time with a long no-flow time in the late part of diastole. There was no evidence of infection in serology or pleural aspiration. The patient underwent medical treatment for heart failure and was discharged. Seven months later, the patient presented again with progressive right-sided heart failure. In right-sided heart catheterization, a pressure equilibration at 40 mm Hg was found between the pulmonary artery and the right ventricle caused by free regurgitation into the right ventricular outflow tract (Figure 1). The patient underwent urgent pulmonary valve re-replacement using a biological prosthesis. Intraoperatively, the leaflets of the xenograft were found to have undergone complete destruction. Histologic examination of the xenograft showed remnants of a vascular wall of elastic type with focal fibrosis and scars. The media presented with reticular fibrosis and degeneration of elastic lamellae, but areas of fibrotic scars also were found. The adventitia consisted of normal adipose and connective tissue; however, there was evidence of moderate infiltration of mononuclear cells and formation of lymphatic follicles. Postoperative pericardial and pleural effusions and peripheral edema were treated successfully with diuretics, and the patient was discharged 20 days postoperatively in good condition.

DISCUSSION

Early failure of tissue-engineered decellularized matrices used for pulmonary valve replacement has been reported in 1 case with suture incompetence (xenograft Matrix P),[2] in 1 case with inflammation leading to narrowing at the distal anastomosis (not declared: xenograft Matrix P or cryopreserved pulmonary allograft),[3] and in 4 cases with degradation causing 3 lethal courses (xenograft Synergraft, Cryolife Inc., Kennesaw, GA).[4] Our patient had complete destruction of valve leaflets and fibrotic degradation of xenograft tissue. Because the presence of infection or endocarditis was not demonstrated, we suggest our finding to be a foreign body-type reaction.[4] Diagnosis of pulmonary valve insufficiency after prosthetic valve implantation may be difficult even if it is severe. Evaluation of subvalvular regurgitation by color-wave Doppler analysis of continuous-wave flow pattern, pressure half-time, and duration of no-flow time and its derivates is important for noninvasive assessment of pulmonary valve insufficiency[5] and should be part of routine follow-up after prosthetic valve implantation in the pulmonary position.

CONCLUSIONS

Any sign of right-sided heart failure after the Ross procedure with a tissue-engineered pulmonary graft is an alert signal, and right-sided heart catheterization may be necessary for the early diagnosis of pulmonary graft dysfunction and the prevention of right-sided heart deterioration.

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