

Restenosis 3 months after successful percutaneous aortic valvoplasty. A clinicopathological report

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A 76-year-oldman with severe, calcific aortic stenosis experienced recurrence of symptoms 3 months after a successful percutaneous aortic valvoplasty. Echo Doppler revealed a marked increase of peak aortic flow velocity as compared with the immediate post-valvoplasty value. The patient underwent an uncomplicated aortic valve replacement. Adjacent to fragmented calcification, histology demonstrated a scarring reaction which might well be a major factor in the restenosis process.

Key words: Balloon valvoplasty; Aortic valve stenosis

Introduction

Percutaneous balloon aortic valvoplasty can achieve significant immediate hemodynamic and symptomatic improvement in elderly patients with severe aortic stenosis [1]. Up to now, however, few data are available concerning the long-term follow-up after valvoplasty. We report a case of restenosis after a successful valvoplasty which required valvar replacement.

Case Report

A 76-year-old man, with a 6-year history of insulin-dependent diabetes mellitus, was admitted to our hospital with chest pain and dyspnea on minimal effort and a history of a syncopal episode during walking. Physical examination and non-invasive assessment indicated a severe valvar aortic stenosis. In particular, continuous wave Doppler showed a peak aortic flow velocity of 5 m/sec. Cardiac catheterization confirmed a severe, calcific aortic stenosis and showed normal left ventricular function (ejection fraction: 65%), marked left

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ventricular hypertrophy (mass/volume ratio: 1.7), minimal aortic regurgitation and multiple coronary stenoses, involving the distal vessels. It was felt that percutaneous valvoplasty was justified because of the advanced age and the associated disease. Dilations of the valve with balloons of increasing diameter (from 15 to 20 mm) resulted in a reduction of the mean systolic transaortic gradient from 57 to 39 mm Hg and of the peak systolic gradient from 56 to 28 mm Hg. Cardiac index increased from 2.1 to 2.5 l/min/m², aortic flow from 179 to 245 ml/sec and aortic valve area from 0.50 to 0.87 cm². After the procedure, supra-avalvular aortography showed no increase in aortic regurgitation. After valvoplasty, peak aortic flow velocity showed a decrease to 2.5 m/sec. A significant improvement in effort tolerance persisted for 3 months. Thereafter, because of recurrence of previous symptoms including a syncopal episode, an echo Doppler was repeated. This showed an increase in the peak aortic flow velocity to 4.5 m/sec. Further intervention was felt necessary and the patient underwent an uncomplicated replacement of the aortic valve 5 months after valvoplasty. Intraoperatively, the aortic valve was found to be bicuspid, without commissural fusion but with massive nodular calcifications and severe impairment of cusp motility. No leaflet tearing nor other damage which might be related to the previous valvoplasty were recognized macroscopically. Histology of the valve showed hyaline degeneration of the connective tissue with severe calcification and degenerative bone formation (Fig. 1). Subendocardial particles of calcium were embedded in organizing connective tissue consisting of young fibroblasts with lymphocytes and plasma cells (Fig. 2).



Fig. 1. Hyaline valve stroma (S) with degenerative bone formation, in which the trabecula consist of lamellated bone and osteocytes in lacuna. Fatty bone marrow (★) exists between the trabeculations (hematoxylin and eosin, $\times 40$).

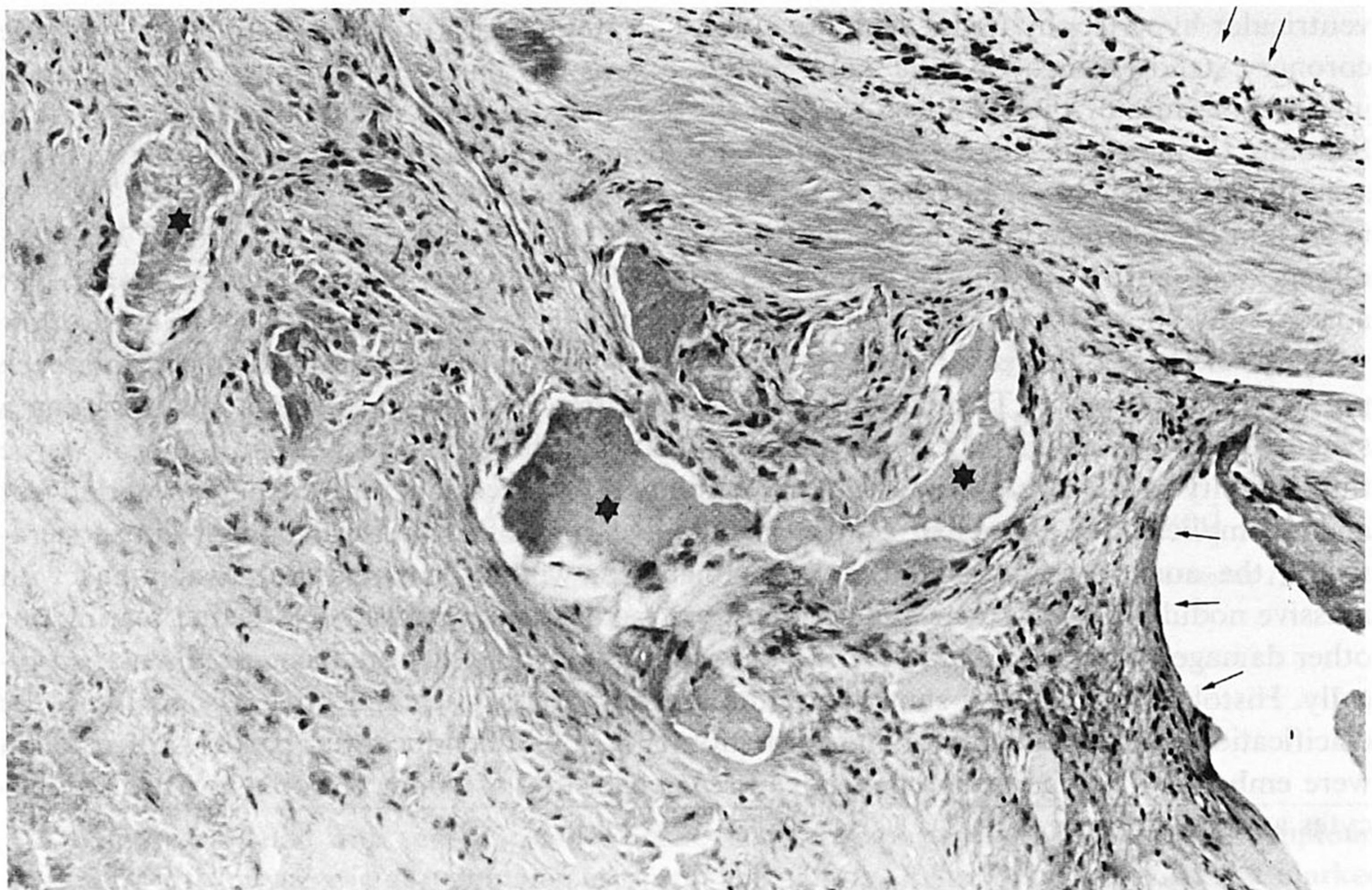


Fig. 2. Just beneath the endothelial covering of the valve (arrows), particles of calcium (★) are embedded in cellular scar tissue consisting of young fibroblasts and a lymphoplasmacellular inflammatory infiltrate (hematoxylin and eosin, $\times 100$).

Discussion

Percutaneous aortic valvoplasty is an alternative treatment to surgery in severe aortic stenosis. It is still uncertain whether this procedure has only palliative effect or if long-term benefits can be expected. In elderly patients with degenerative aortic stenosis, the tricuspid, or less frequently, bicuspid aortic valve is characterized by nodular calcific deposits on the aortic aspects of the cusps and by lack or near-lack of commissural fusion [3]. Our knowledge of the pathogenesis would suggest that the main operative mechanisms of aortic valvoplasty are both fracture of gross nodular calcifications and microfractures in the smallest calcific deposits, induced by the stretching of the leaflets and leading to an increased mobility of aortic leaflets [3,4].

In our patient, histology suggests that a scarring reaction may occur adjacent to the fragmented calcification and may result in increased cusp rigidity which leads to restenosis. The importance of this process on the long-term effect of aortic valvoplasty requires further evaluation. A strict follow-up program, including serial echo-Doppler assessment, is recommended at this stage.

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Myocardial function in alcoholic cardiac beriberi

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A case of alcoholic beriberi is described. Left ventricular systolic function as measured by the radionuclide ejection fraction showed an abnormal fall with exercise. Subsequent return to normal with the administration of thiamine suggests myocardial involvement in the condition.

Key words: Beriberi; Myocardial function; Ejection fraction; Thiamine

Introduction

In Western society cardiac beriberi is almost exclusively a disease of alcoholics. Vasodilation of muscle arterioles effectively produces an arteriovenous fistula, low systemic vascular resistance and consequent high output heart failure. The major unsettled question is whether there is a concomitant myocardial lesion [1].

Case Report

A 36-year-old man presented with 6 months of worsening breathlessness on exertion which had improved slightly with frusemide 40 mg daily from 2 months prior to admission. He had no chest pain, orthopnoea or nocturnal dyspnoea but admitted to drinking 5 litres of

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