Infective Endocarditis With Extensive Calcified Granulation of the Mitral Annulus and Valve
— A Case Report —

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Mitral annular calcification, a degenerative process usually seen in the elderly or in chronic renal failure, is rarely seen in an extensive form. A 69-year-old man with no history of renal failure, rheumatic fever, or heart disease had mitral valve vegetation and regurgitation, together with extensive mitral annulus and valve calcification, which may or may not have been secondary to the infective endocarditis. (Jpn Circ J 2000; 64: 990–992)

Key Words: Calcification; Granulation; Mitral valve

Mitral annular calcification is a degenerative process related to aging! and it occurs quite frequently in patients with chronic renal failure receiving long-term hemodialysis.\(^2\) Extensive calcification of the mitral anulus is, however, rare. We present a case of infective endocarditis with severe mitral regurgitation caused by extensive calcification of the mitral annulus and valve.

Case Report

A 69-year-old man was referred to hospital in April 1999 because of nocturnal dyspnea (New York Heart Association functional class II) and stroke. In early April, he had had a high grade fever and was treated with antibiotics for several days. Although he had a history of total gastrectomy for gastric cancer at the age of 62 and chronic type C hepatitis, he did not have a history of rheumatic fever, heart disease or renal disease. His body temperature was 36.5°C, and blood pressure was 148/92 mmHg with a regular pulse rate of 72 beats/min. A pansystolic murmur of grade 3/6 was audible at the apex and moist rales were heard in both lower lung fields.

Routine laboratory tests showed a normochromic normocytic anemia with hemoglobin 10.0 g/dl and hematocrit 38%. The number of platelets was reduced, at 9.9 × 10^4/μl. The serum C-reactive protein was elevated, at 1.03 mg/dl, and the proteinemia with a serum albumin level of 2.4 g/dl was attributed to his poor nutritional state. A chest X-ray revealed a cardiothoracic ratio of 57% with severe pulmonary congestion. An ECG showed normal sinus rhythm and a mitral P wave in lead V1.

An M-mode echocardiogram showed a left ventricular diastolic dimension of 49 mm, a systolic dimension of 29 mm, and an ejection fraction of 71%. Two-dimensional

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Echocardiography showed marked thickening of the posterior mitral leaflet (PML) with vegetation; the PML mass was 19×17 mm and had a heterogeneous echo density with a partial high-echo density because of the calcification (Fig 1). Color Doppler echocardiography showed both severe mitral regurgitation and severe tricuspid regurgitation. Transesophageal echocardiography confirmed the marked thickening of the PML. The mass appeared to have a stalk originating from the right atrium, suggesting tumor involvement from the right atrium to the calcified PML (Fig 2). Right heart catheterization showed pulmonary hypertension with a systolic pulmonary arterial pressure of 42 mmHg, a mean pulmonary capillary wedge pressure of 21 mmHg, and a mean right atrial pressure of 6 mmHg.

The preoperative diagnosis was mitral regurgitation with a mitral valve tumor or vegetation, and tricuspid regurgitation. Mitral valve replacement (MVR) using a 27-mm Carpentier-Edwards pericardial mitral valve (Baxter, Santa Ana, CA, USA) and tricuspid annuloplasty by the De Vega procedure were performed under cardiopulmonary bypass and intermittent tepid blood cardioplegia. Although the anterior leaflet of the mitral valve was less involved and pliable, the annulus of the PML had a small amount of vegetative growth and thickened calcific deposits extending into the ventricular muscle (Fig 3). In this region, the subvalvular structures were also calcified. To prevent atrioventricular groove disruption, several Teflon felt pledget-reinforced sutures were placed on the atrioventricular groove between the left atrium and ventricle. Thereafter, standard MVR was performed.

Histopathologically, extensive mitral valve calcification and formation of granulation tissue with cellular infiltration were observed (Fig 4).

Postoperative echocardiography revealed mild mitral perivalvular leakage, but no tricuspid regurgitation. Two months after the operation, an AAI pacemaker was implanted because of sick sinus syndrome (Rubenstein type II). The patient was well at his most recent follow-up examination.

Discussion

Massive calcification of the mitral annulus and valve is
rare. In rheumatic valvular diseases, the calcification usually involves the commissures and the leaflet tissue, with only a late extension to the annulus. Extensive calcification of the mitral valve annulus is encountered either in elderly persons^1 or in patients with chronic renal failure; in fact, it occurs in 9–36% of patients receiving long-term hemodialysis^2,^3 Although the present patient did not have a history of rheumatic valvular disease, heart disease or chronic renal failure, there was massive calcification of the mitral annulus and valve accompanied by partial calcification of the left atrium. In addition to the marked calcification of the mitral annulus and valve, vegetation on the posterior valvular leaflet, together with chronic infection, resulted in calcified granulation. Although the precise etiology of the massive calcified granulation of mitral annulus and valve in the present case is difficult to ascertain, we suppose that the extensive mitral annular and valve calcification was accompanied by bacterial endocarditis, not a result of recurrent endocarditis.

In summary, this patient did not have a history of renal failure, rheumatic fever, or heart disease, yet exhibited mitral valve vegetation and regurgitation, together, unusually, with extensive mitral annulus and valve calcification, which may or may not have been caused by infective endocarditis.

References