Detection of Mitral Ring Abscess by Two-dimensional Echocardiography

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SUMMARY M-mode and two-dimensional echocardiographic features of mitral ring abscess are described. A round, dense echo mass between the posterior mitral leaflet and left ventricular posterior wall was demonstrated in long- and short-axis views of the left ventricle. The diagnosis of mitral ring abscess was confirmed at surgery. The superiority of two-dimensional echocardiography over M-mode echocardiography for evaluating patients with mitral ring abscess is also discussed.

M-MODE and two-dimensional echocardiography can be used to identify valvular vegetations and flail valve in patients with infective endocarditis.1,2 Certain patients with active infective endocarditis require cardiac valve replacement before completion of adequate antibiotic therapy.3,4 A major determinant of the outcome of cardiac valve replacement during active infective endocarditis is the presence or absence of a valve ring abscess. If a valve ring abscess is present, the infection may persist behind the site of attachment of the prosthesis, or significant paravalvular leakage may follow valve replacement.5,6

In this report, we describe the use of two-dimensional echocardiography in detecting the valve ring abscess in a patient with infective endocarditis.

Case Report

A 39-year-old man was transferred to the Heart Institute of Japan for cardiac valve replacement after treatment of infective endocarditis. Eight months before, he had been admitted to another hospital because of fatigue and fever of 2 weeks’ duration. On his initial admission, the patient appeared chronically ill, blood pressure was 150/80 mmHg and heart rate was 88 beats/min and regular.

A grade 4/6 diastolic regurgitant murmur was heard at Erb’s area radiating to the apex. Laboratory data disclosed a normocytic, normochromic anemia with a hemoglobin of 10 g/dl and a white blood cell count of 16,000 with a shift to the left. Three blood cultures grew Streptococcus viridans, sensitive to penicillin. Antibiotic therapy was initiated with i.v. penicillin and intramuscular streptomycin and was continued for 4 months.

On admission to the Heart Institute, the carotid pulse was brisk and the apical impulse was dynamic. A grade 3/6 diastolic regurgitant murmur was heard at the left sternal border and a grade 2/6 holosystolic murmur was also present at the apex.

Findings from the remainder of the physical examination were normal. The chest x-ray showed no cardiac enlargement and the ECG revealed a sinus rhythm with a normal ST-T wave. M-mode echocardiography taken on admission showed the characteristic coarse, irregular echoes of a vegetation attached to the aortic valve (fig. 1), mitral stenosis and a dense, band-like echo between the posterior mitral leaflet and left ventricular posterior wall (fig. 2).

Two-dimensional echocardiographic examination with a real-time, phased-array scanner was performed in multiple views. The long-axis view of the aorta and left ventricle showed a round, dense echo mass between the posterior mitral leaflet and the left ventricular posterior wall (fig. 3). In the short-axis view of the left ventricle, this abnormal, round echo extended from the level of the chordae tendineae to the level of the mitral ring, and a dense, crescent-shaped band of echoes was not observed at the juncture of the posterior mitral leaflet and the left ventricular posterior wall (fig. 4).

At cardiac catheterization, the right atrial, ventricular and pulmonary arterial pressures were all normal. The pressure in the ascending aorta was 150/40 mm Hg (mean 80 mm Hg). Because of the suspicion of vegetations on the aortic valve, we decided not to catheterize the left ventricle. Cineangiography showed massive aortic regurgitation and no abnormal shadow in the mitral ring.

At surgery, massive vegetations were found on the right and noncoronary cusps, and behind the posterior mitral leaflet a huge abscess with dense contents was separated from the left ventricular cavity by a thin membrane (fig. 5).

The aortic and mitral valves were replaced with Björk-Shiley prostheses. Cultures of resected valvular material and the contents of the abscess were sterile. Postoperatively, the patient was treated for 6 weeks with i.v. penicillin and remained asymptomatic for 8 months of observation.

Discussion

Although the problem of cardiac valve replacement in patients with active infective endocarditis has been described, the frequency and location of valve ring abscesses have seldom been reported. If valve ring abscess is present, the infection can persist behind the site of attachment of the prosthesis, and significant paraavalvular leak can follow valve replacement.

Of 74 necropsy patients with infective endocarditis studied by Arnett and Roberts,7 22 had aortic ring...
abscess and only two had mitral ring abscess. One of these two patients had an isolated mitral valve infection with a ring abscess and the other had aortic valve vegetations and infection of a heavily calcified mitral valve annulus. A similar observation was made by Burnside and DeSanctis. Arnett and Roberts also reported that among 22 necropsy patients with prosthetic valve endocarditis, 15 had aortic and seven had mitral ring abscesses.

Fox et al. reported that ring abscess of the aortic valve could be diagnosed echocardiographically by the presence of double echoes in the aortic wall and aortic-mitral discontinuity. These M-mode echocardiographic findings are, however, nonspecific and indistinguishable from echocardiographic appearance of aortic dissection. Mardelli et al. emphasized the radiolucent cavity confined to the aortic valve ring in the two-dimensional echocardiogram; but in their cases, necropsy confirmed the presence of abscess with a large pocket of edematous fluid.

Welton et al. reported angiographic findings in patients with mitral ring abscess, but to our knowledge, we are the first to describe the echocardiographic features of mitral ring abscess.

**FIGURE 1.** M-mode echocardiogram of the aortic valve showing coarse, irregular echoes in systole and diastole with normal opening motion. AO = aorta; LA = left atrium.

**FIGURE 2.** M-mode echocardiogram showing a dense, band-like echo (arrow) between the posterior mitral leaflet (PML) and left ventricular posterior wall (LVPW). When a scan is made from the left atrium (LA) to the left ventricle (LV), this band-like echo ends abruptly, contiguous with the LVPW, as seen in mitral annular calcification. AO = aorta; AML = anterior mitral leaflet.

**FIGURE 3.** Two-dimensional echocardiograms in the long-axis view of the left ventricle showing a round, dense echo mass (arrow) between the posterior mitral leaflet and left ventricular posterior wall. (A) Systole. (B) Diastole. AO = Aorta; LA = Left atrium.
In the present case, the diagnosis of mitral ring abscess was never suspected from the M-mode study, because a dense, band-like mass of echoes between the posterior mitral leaflet and left ventricular posterior wall was always seen in patients with mitral annulus calcification. Kotler et al.\textsuperscript{13} described the two-dimensional echocardiographic visualization of mitral annulus calcification as a dense, crescent-shaped band of echoes between the posterior mitral leaflet and left ventricular posterior wall. D’Cruz reported the M-mode and two-dimensional echocardiographic features of submitral calcification and classified it into anterior and posterior submitral calcification, and also emphasized the shape and location of the calcification echo when a transverse scan was performed from the left ventricle to the left atrium.\textsuperscript{14}

In our patient, short-axis, two-dimensional echocardiography was useful for estimating the shape and location of the abnormal, dense echo mass and was extremely helpful in differentiating mitral annulus calcification. The characteristic echo mass of the mitral ring, as well as the vegetation of the aortic valve, suggested the presence of a ring abscess with dense contents.

In conclusion, two-dimensional echocardiography, in combination with the clinical condition of the patient, was useful in the diagnosis and management of a patient with mitral ring abscess secondary to infective endocarditis.

References

Pneumonitis and Pulmonary Fibrosis Associated with Amiodarone Treatment: A Possible Complication of a New Antiarrhythmic Drug

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SUMMARY Six patients are presented who developed pulmonary infiltrates of undetermined origin while being treated for severe ventricular arrhythmias with amiodarone hydrochloride. Biopsy material was available in four patients and revealed interstitial or alveolar fibrosis and pneumonitis. Four patients recovered and two died of severe cardiopulmonary decompensation; all of the patients who recovered received corticosteroid therapy. Pulmonary fibrosis is a previously unreported complication of amiodarone therapy.

AMIODARONE hydrochloride is a benzofuran derivative, introduced in Europe in 1961 as an antianginal agent and subsequently found to have important antiarrhythmic properties. It is widely used as an antiarrhythmic agent in Europe and in South America. Experience with large numbers of patients in the past decade has shown that the drug may be associated with side effects, of which the most common is corneal microdeposits, appearing in 90-100% of patients who receive chronic treatment. These are rarely symptomatic, and resolve when the drug is discontinued. Less frequent adverse effects include constipation, photosensitivity dermatitis, headache, nausea and hyperthyroidism. These symptoms are usually mild and only occasionally necessitate discontinuing the drug. Uncommon toxic effects have included hypothyroidism, gray-blue skin discoloration and peripheral neuropathy. Until 1980, there were no reports in the world literature of pulmonary toxicity due to amiodarone. However, Rotmensch and associates suggested that this drug may have been responsible for the appearance of pulmonary infiltrates in one of their patients.

In the United States, amiodarone is available in only a few centers as an investigational agent, and has been used on a limited number of patients. As this report was being prepared, 432 patients in this country were being treated with the drug; all had life-threatening or severely symptomatic arrhythmias and were either resistant or intolerant to treatment with standard antiarrhythmic drugs. Despite the small number of patients exposed, we know of six cases of pulmonary disease occurring unexpectedly in patients treated with amiodarone.

Case Reports

Patient 1

A 67-year-old man with severe mitral valve prolapse and recurring atrial flutter and ventricular ectopy was begun on amiodarone in December 1977. Doses were increased weekly from 200 to 800 mg/day; 800 mg/day was the maintenance dose. He did well until May 1978, when transient fever occurred with mild dyspnea, weight loss and weakness. In late June, bilateral upper lobe infiltrates were noted on chest
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