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Mitral Valve Aneurysm associated with Aortic Valve Endocarditis and Regurgitation

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ABSTRACT

Mitral valve aneurysms are rare complications occurring most commonly in association with aortic valve infective endocarditis. [Decroly 1989, Chua 1990, Northridge 1991, Karalis 1992, Roguin 1996, Mollod 1997, Vilacosta 1997, Cai 1999, Vilacosta 1999, Teskey 1999, Chan 2000, Goh 2000, Marcos-Alberca 2000] While the mechanism of the development of this lesion is unclear, complications such as perforation can occur and lead to significant mitral regurgitation. [Decroly 1989, Karalis 1992, Teskey 1999, Vilacosta 1999] The case of a 69-year-old male with Streptococcus Sanguis aortic valve endocarditis and associated anterior mitral leaflet aneurysm is presented. Following surgery, tissue pathology of the excised lesion revealed myxomatous degeneration and no active endocarditis or inflammatory cells. This may add support to the hypothesis that physical stress due to severe aortic insufficiency and structural weakening, without infection of the anterior mitral leaflet, can lead to the development of this lesion.

INTRODUCTION

Mitral valve aneurysms are rare complications occurring most commonly in association with aortic valve infective endocarditis. [Decroly 1989, Chua 1990, Northridge 1991, Karalis 1992, Roguin 1996, Mollod 1997, Vilacosta 1997, Cai 1999, Vilacosta 1999, Teskey 1999, Chan 2000, Goh 2000, Marcos-Alberca 2000] While the mechanism of the development of this lesion is unclear, complications can occur such as recurrent infection, embolization and perforation leading to significant mitral regurgitation. [Decroly 1989, Karalis 1992, Teskey 1999, Vilacosta 1999] We report a case of a mitral valve aneurysm diagnosed by transesophageal echocardiogram due to aortic valve endocarditis and severe aortic insufficiency.

CASE REPORT

A 69 year old male presented to his family physician with an acute left homonymous hemianopsia and a 3 month history of intermittent fever, chills anorexia and weight loss. A head CT scan was performed and revealed an acute right occipital infarction. Blood cultures were drawn and grew Streptococcus Sanguis that was penicillin sensitive and a transthoracic echocardiogram was performed which confirmed aortic valve vegetations and moderate aortic insufficiency. Following the investigations in hospital, the patient was discharged home with outpatient intravenous antibiotic therapy.

At 5 weeks from his initial presentation, the patient developed gradually progressive dyspnea and orthopnea. A chest x-ray revealed bilateral pleural effusions and pulmonary edema and the patient was subsequently hospitalized. Diuretics were administered. A repeat transthoracic echocardiogram revealed "shaggy" vegetations on the aortic valve with severe aortic regurgitation and moderately severe mitral regurgitation. A 1.0 cm echogenic mass resembling a "cyst" was evident on the atrial side of the anterior mitral leaflet. A transesophageal echocardiogram was performed confirming a mitral valve aneurysm. (see Figures 1, 2, and 3, ◎)

The patient was transferred to our tertiary care hospital for consideration of surgical intervention. A cardiac catheterization was performed and the coronary arteries were normal.

Nineteen days after repeat hospitalization and 2 months after initial presentation, a midline sternotomy was carried out under general anesthesia with subsequent cardiopulmonary bypass. The aortic valve was grossly distorted with multiple friable vegetations seen particularly on the left and right coronary cusps on the ventricular side. The valve was excised and the surrounding tissue debrided and a #23 Freestyle Porcine aortic valve was inserted. The mitral valve was approached from a right atrial transeptal incision and the cystic lesion was examined (Figure 4, ◎). A 3mm hole was visualized entering the cystic cavity from the ventricular side. The lesion was excised and the pathology of this mitral valve tissue revealed an expanded myxoid spongiosa suggestive of myxomatous degeneration. There were areas of excess collagenous fibrosis and and focal calcification, however, no evidence of inflammatory infiltrate or overt endocarditis was present. The patient was discharged in excellent condition.

DISCUSSION

A mitral valve aneurym is a localized, thin-walled saccular bulge of the mitral leaflet toward the left atrium associated with systolic expansion and diastolic collapse. [Karalis 1992, Vilacosta 1999] There is also discontinuity at the base of the aneurysm resulting in direct communication between the

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Table 1. Conditions Predisposing to Mitral Valve Aneurysms

Aortic Valve Endocarditis (1–13)	Mitral valve prolapse (8)
Osteogenesis Imperfecta (8, 12)	Marfan's syndrome (8, 12)
Ehlers Danlos syndrome (8, 12)	Syphilis (5)
Rheumatic Fever (5)	Iridocyclitis (5)
Hypertrophic Obstructive Cardiomyopathy (5)	

aneurysm and the left ventricle. [Vilacosta 1999] This may be detected by colour doppler as an abnormal regurgitant flow pattern within the aneurysm. [Decroly 1989, Karalis 1992, Vilacosta 1999] There is a predilection for these aneurysms to form on the anterior mitral leaflet, however isolated posterior leaflet and bileaflet involvement have been reported. [Northridge 1991, Roguin 1996, Teskey 1999, Vilacosta 1999] The size of these lesions can vary from a few millimeters to as large as 51mm in diameter [Mollod 1997] and certain predisposing conditions have been reported as indicated in Table 1 (◎).

The differential diagnosis of these lesions includes mitral valve abscess, atypical mitral valve prolapse, cystic atrial myxoma, flail leaflet, a blood cyst of the papillary muscle, and a non-endothelialized cyst of the mitral valve. [Mollod 1997, Northridge 1991]

The strongest predisposing factor for the formation of mitral valve aneurysm is aortic valve endocarditis, usually in the presence of significant aortic regurgitation. [Decroly 1989, Roguin 1996, Mollod 1997, Teskey 1999, Vilacosta 1999, Marcos-Alberca 2000] Other factors have been reported and are listed in Table 1. Proposed mechanisms for the formation of mitral valve aneurysm in the setting of aortic valve endocarditis include either direct extension of the aortic valve endocarditis to the anterior mitral leaflet, abscess formation and subsequent drainage, or seeding of the anterior mitral leaflet by the regurgitant jet with inflammation, local weakening, dissection and later expansion of the valvular tissue. [Decroly 1989, Chua 1990, Chan 2000, Marcos-Alberca 2000, Roguin 1996] In previously reported cases where mitral aneurysm tissue pathology was mentioned, the presence of either active endocarditis or chronic inflammatory changes were observed. [Cai 1999, Decroly 1989, Karalis 1992] Goh et al. described a case of mitral valve aneurysm with a quadricuspid aortic valve and aortic regurgitation. [Goh 2000] Interestingly, no pathologic evidence of endocarditis of the aortic valve could be identified and the pathology of the mitral valve aneurysm was not mentioned. Cai et al. reported a case of previously treated aortic valve endocarditis with subsequent mitral aneurysm formation, yet no identifiable active endocarditis was seen. [Teskey 1999] The authors imply that the aneurysm formation was secondary to severe aortic regurgitation instead of active endocarditis, yet inflammatory cells were seen in the tissue specimen.

To our knowledge, the patient described here represents the only reported case of mitral valve aneurysm associated with aortic valve endocarditis without any pathological evidence of infection or inflammation of the lesion. This observation supports the hypothesis that the presence of significant chronic aortic regurgitation might mechanically weaken the ventricular side of the anterior mitral leaflet due to excessive shear stress and result in secondary myxomatous degeneration without active infection or inflammation. An alternative hypothesis in this patient may be that the aortic valve endocarditis was unrelated to the formation of the mitral valve aneurysm. In other words, the aneurysm was simply an incidental congenital anomaly. A third possibility, although less likely, is that while the aneurysm initially formed with active endocarditis, several weeks of antibiotics sterilized it, and no residual infection could be detected. However, one would expect to see residual inflammatory cells that were clearly not present in this patient's mitral valve aneurysm at pathology. Regardless of the mechanism of the formation of the mitral valve aneurym, the high risk of serious complications such as valvular perforation, emboli or recurrent infection mandate surgical removal of these aneurysms with subsequent repair of the mitral valve.

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