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Abstract:
Mitral valve aneurysm (MVA), is an alarming complication of infective endocarditis (IE), with worse outcomes seen among patients with preexisting valvular disease or intravenous drug use.¹

Other causes of this disorder are rheumatic heart disease, Marfan’s syndrome, aortic regurgitation and hypertrophic cardiomyopathy. The complications of MVA include expansion, perforation and severe valvular regurgitation. An early diagnosis followed by surgical treatment is of critical importance to reduce the rate of these complications.²

We present an aneurysm of the posterior mitral leaflet in a woman who presented with a severe mitral regurgitation (MR) in the context of infective endocarditis.

Introduction:
Mitral valve aneurysm (MVA) is uncommon and reported cases are rare and often the consequence of infective endocarditis³. Their prevalence is 0.2–0.29% of the transthoracic echocardiography studies,⁴,⁵ and they are most frequently seen in association with IE of the aortic valve⁶. An MVA is defined as a localized bulge of the mitral leaflet toward the left atrium with systolic expansion and diastolic collapse ⁷. The mechanism of the aneurysm evolvement is not clearly established. The diagnosis of MVA relies on a recognition of changes in clinical status and repeat echocardiography. Mitral valve repair or replacement is indicated when aneurysm is ruptured or when the unruptured aneurysm is large or accompanied by significant regurgitation.³ We present the case of a 60-year-old woman with diabetes mellitus, who was admitted for a heart failure attack, in whom we found a severe mitral insufficiency with infective endocarditis, complicated by an aneurysm of the posterior mitral leaflet.
Case report:
We report the case of a 60 year old patient, diabetic on insulin, with no history of rheumatic fever, dental care or recent transfusions or toxic habits, presenting to the cardiological consultation with a NYHA stage IV dyspnea. She reported a notion of heaviness of the left hemisphere and fever 3 months ago, treated with protected amoxicillin by a general physician. The evolution was marked by the occurrence of a dyspnea stage III-IV of the NYHA, with increase of the volume of the lower limbs. The clinical examination found a polypneic patient, with global cardiac failure, with a mitral insufficiency murmur on cardiac auscultation, Osler’s nodules on the fingers and toes, as well as a dry necrosis of the fifth finger of the left hand. The EKG showed sinus tachycardia, with left anterior hemiblock and the chest X-ray a V3 cardiomegaly. The transthoracic echocardiography was equivocal for valvular vegetation on the mitral valve with a mobile element measuring 8 mm at the level of the atrial side of the the posterior leaflet with severe mitral insufficiency by prolapse of P1-P2, and the presence of an aneurysmal sac at the level of the posterior mitral leaflet. We completed by a transesophageal echocardiography which confirmed the aneurysm of the posterior mitral leaflet (Figure 1A), and revealed the presence of a filiform element at the non-coronal cusp of the aortic valve and two filiform elements on the atrial side of the mitral valve, with a P2 prolapse (Figure 1B).

Figure 1A: Transesophageal echocardiography image showing the aneurysm of the posterior mitral leaflet

Figure 1B: Transesophageal echocardiography image showing the P2 prolapse of the mitral valve
The definite diagnosis of infective endocarditis was retained on the basis of 1 major and 3 minor criteria: predisposing cardiac damage, i.e. mitral insufficiency, vascular phenomena, i.e. a major arterial embolism, and immunological phenomena such as Osler nodules. The patient was put on antibiotic therapy for infective endocarditis and was referred to surgeons for mitral surgery. The patient underwent a mitral plasty.

Discussion:

MVA is a rare entity usually associated with aortic valve (AV) endocarditis. Among other complications, they bear a high risk of perforation with subsequent acute severe MR. The first case of MVA was reported by Morand in 1729. MVAs are rarely encountered in patients without endocarditis. Sporadic cases are reported in association with particular conditions, such as connective tissue diseases (e.g., Marfan syndrome, Ehlers-Danlos syndrome, pseudoxanthoma elasticum, and osteogenesis imperfecta), extreme forms of MV prolapse (e.g., MVP), congenital cardiac structural defects, or Libman-Sacks endocarditis. Untreated MVA can lead to severe mitral regurgitation and acute pulmonary edema through multiple mechanisms, including perforation of the aneurysm and rupture of the chordae tendineae. Moreover, MVA may cause a mass effect, leading to coaptation defect of the leaflets. Another serious complication of MVA is thrombus formation within the saccular bulge, with high risk of embolism and spreading of infection.

Diagnosis can be made by transthoracic echocardiography but transesophageal echocardiography is more sensitive and accurate. Echocardiography shows MVA as a localized saccular bulge of the leaflet toward the left atrium and communication between the aneurysm and the left ventricle during the cardiac cycle. Anterior MVAs are more common than posterior MVAs. The differential diagnosis of MVA includes mitral valve pseudo aneurysms (MVP), myxomatous degeneration of the MV, flail mitral leaflet, chordal rupture, papillary fibroelastoma, atrial myxoma involving the MV, blood cyst of the papillary muscle, and anterior MV diverticulum. MVP, can be distinguished from MVA by the diffuse leaflet thickening, the protrusion of the leaflet tip into the left atrium during systole, and the absence of a discrete neck.

No definite indications for treatment of MVAs exist. Surgery should definitely be considered in case of perforation or rupture, with severe MR and signs of heart failure. It might also be be considered if concomitant evidence of uncontrolled infection is present. Conservation may be possible for small ones, but close follow-up is needed. The decision of whether to perform surgery or to conservatively follow up a patient should rely on the dimension of the MVA and clinical status of the patient.
MV repair, primarily or using synthetic or pericardial patch, in the setting of IE has been shown to have better clinical in-hospital and long-term results as compared to MV replacement\textsuperscript{21,22}. Prosthetic valve replacement may be considered in patients unsuitable for repair or with persistent MR following repair. The best time for surgery is unclear. Early surgery for IE has been advocated in general. A period of antibiotic therapy before surgery can prevent possible extra-valvular involvement. Simplifying operations is an important benefit of presurgical antibiotic therapy\textsuperscript{13}.

**Conclusion:**

MVA is considered as a rare complication of IE that can lead to severe morbidity and mortality when not diagnosed early and managed quickly. Early intervention may prevent perforation of a MVA aneurysm and acute heart failure. Treatment is generally surgical; however, small and uncomplicated aneurysms can be managed with a conservative strategy with a close follow-up.

**References:**


