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MITRAL VALVE PROLAPSE, NOT ALWAYS AN EASY DIAGNOSIS

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Abstract

Prezentăm cazul unui pacient de sex masculin în vârstă de 61 de ani, fără antecedente medicale patologice, care se prezintă pentru o durere toracică anterioară, apărută brusc după efort fizic, însoțită de palpitații, oboseală și dispnee. Examenul fizic a evidențiat prezența unui suflu sistolic grad 4/6, cu auscultație maximă la apex și iradiere axilară stângă. Evaluarea ecocardiografică a evidențiat elemente diagnostice sugestive pentru prolaps sever de valvă mitrală posterioară, insuficiență mitrală severă, prolaps de valvă tricuspidă, insuficiență tricuspidiană de gradul III și hipertensiune pulmonară. Pentru a exclude o ruptură de mușchi papilar a fost efectuată ecocardiografie transesofagiană, care nu a obiectivat ruptura. În contextul durerii toracice anterioare, s-a impus efectuarea angiografiei coronariane care a evidențiat artere coronare fără leziuni semnificative hemodinamic. În acest context, s-a stabilit diagnosticul de prolaps de valvă mitrală cu regurgitare mitrală severă, fără elemente sugestive de sindrom coronarian acut complicat, în pofida simptomatologiei acute. Am interpretat durerea toracică secundară: anginei microvasculare, tracțiunii la nivelul cordajelor sau hipertensiunii pulmonare. Pacientul a avut indicație de intervenție chirurgicală pentru înlocuirea sau reconstrucția valvei mitrale. În cadrul departamentului de chirurgie cardiovasculară s-a efectuat înlocuirea valvei mitrale cu o proteză mecanică, cu evoluție favorabilă.

Cuvinte cheie: *prolaps valvă mitrală, ecocardiografie, durere toracică, insuficienţă cardiacă.*

Abstract

We present the case of a 61-year-old male patient, with no medical history, who was presenting for an anterior chest pain, which appeared suddenly during an effort, accompanied by palpitations, fatigue and dyspnea Physical examination revealed the presence of a 4/6 systolic murmur with maximal auscultation at the apex and radiation in the left axilla.



Internal Medicine

Clinical Cases

Echocardiographic evaluation revealed diagnostically suggestive elements for severe posterior mitral valve prolapse without any rupture, color Doppler showing severe mitral regurgitation, a tricuspid valve prolapse, tricuspid regurgitation grade III and pulmonary hypertension. Coronary angiography revealed coronary arteries without hemodynamically significant lesions, and Holter ECG/24-hour monitoring detected atrial fibrillation in 44% of the monitoring time. In this context, the possible causes of the anterior chest pain were: microvascular angina, traction on the chordae or pulmonary hypertension. The patient had a surgical indication for mitral valve replacement or reconstruction. The department of cardiac surgery undergoes mitral valve replacement with a mechanical prosthesis with a favorable outcome.

Keywords: *mitral valve prolapse, chest pain, heart failure, echocardiography.*

Introduction

Mitral valve prolapse (MVP) is a prevalent cardiac condition characterized by the improper closure of the mitral valve. The prevalence varies depending on the population studied and the diagnostic criteria used. In general, the reported prevalence ranges from 2% to 3% of the general population. It appears to be more common in women than in men.

Mitral valve prolapse can occur at any age, but it is most commonly diagnosed in individuals between the ages of 20 and 60. It is less frequently seen in children and the elderly. While MVP is generally considered a benign condition, it can lead to various symptoms and, in some cases, complications[1].

The exact cause of mitral valve prolapse is not always clear, but it is believed to have a multifactorial etiology. Genetics may play a significant role, as MVP often runs in families. Structural abnormalities in the connective tissue of the valve, which affects its flexibility and durability, have also been associated with the condition. Additionally, certain factors like age, gender, and other heart-related issues may contribute to the development of MVP[1].

One of the hallmarks of MVP is its asymptomatic nature in many cases. However, when symptoms do manifest, they can vary widely among individuals. Common symptoms include palpitations (feeling of

irregular heartbeats), chest pain, fatigue, shortness of breath, and lightheadedness. In severe cases or when complications arise, individuals may experience arrhythmias, infective endocarditis (infection of the heart valve), or even mitral regurgitation (severe backflow of blood), which can lead to heart failure[2,3].

Treatment for MVP is usually unnecessary unless symptoms are bothersome or complications are evident. In cases where symptoms are severe or complications occur, medical intervention may be necessary. Beta-blockers and other medications can help manage symptoms like palpitations and chest pain. Rarely, surgery may be recommended to repair or replace the affected mitral valve, especially if significant mitral regurgitation is present[2,3]. Though often benign and asymptomatic, it warrants attention and monitoring by healthcare professionals, especially in cases where symptoms are present or complications arise.

Case presentation

A 61-year old male patient, with no medical history, is currently presenting with anterior chest pain after exertion, palpitations, fatigue and dyspnea. The patient had no significant family history, he was a non-smoker, denied chronic alcohol consumption and had no chronic treatment previously. Clinical examination on admission revealed a hemodynamically stable patient, with BP 130/90mmHg, and a systolic murmur graded 4/6, with maximum auscultation at the apex and radiation to the left axilla.

Subsequent paraclinical examinations were conducted in two main directions: the etiology of the anterior chest pain and dyspnea (which required chest X-Ray, electrocardiography, echocardiography, troponin and natriuretic peptides levels) and the etiology of the systolic murmur (echocardiography). The electrocardiography showed a sinus rhythm, with a heart rate of 60 bpm, atrial ectopic beats, without ST-T changes suggestive of ischemia (figure 1). Chest X-Ray showed normal cardio-thoracic index, accentuation of bronchovascular markings and Kerley-B lines (figure 2).

Laboratory blood tests revealed normal repeated hs-cardiac troponin I levels, but an increased NT-proBNP value of 2525 pg/mL. Echocardiography showed normal systolic and diastolic function of the left ventricle, with an ejection fraction of 60%, severe mitral valve prolapse of the posterior cusp, without signs of papillary muscle rupture, severe mitral valve regurgitation, tricuspid valve prolapse, severe tricuspid valve regurgitation and pulmonary hypertension (figure 3, figure 4). For better characterization of mitral valve, a transesophageal echocardiography was performed, which confirmed the mitral valve regurgitation, posterior leaflet mitral valve prolapse, without signs of vegetations or rupture of the mitral valve or mitral valve pillars. Coronary angiography was performed given the presence of chest pain, showing the coronary arteries without hemodynamically significant lesions. The presence of extrasystolic arrhythmia required Holter ECG/24 hours monitoring, which detected atrial fibrillation in 44% of the monitoring time.

By the correlation of clinical and paraclinical results we established the final diagnosis as: severe mitral valve prolapse, grade III mitral valve insufficiency, chronic heart failure NYHA III class with preserved ejection fraction, grade III tricuspid insufficiency, severe pulmonary hypertension, angina pectoris « de novo », and paroxysmal atrial fibrillation.



The treatment aimed to improve patient's symptoms (chest pain, dyspnea) and prognosis, following the ESC Guidelines for heart failure, pulmonary hypertension, atrial fibrillation and valvular disease[4-8]. Consequently, the patient was treated with diuretics, ACE inhibitor, beta-blocker, vasodilator, statin and anticoagulant. Given the severity of mitral regurgitation associated with mitral valve prolapse, the patient was referred to cardiac surgery for mitral valve replacement with a mechanical prosthesis and tricuspid valve annuloplasty.

Discussion

The particularity of the case consisted in the diagnosis of severe mitral valve prolapse, in a patient totally asymptomatic for a long time, which had an acute onset of the symptoms. The clinical presentation was highly suggestive for an acute etiology of mitral regurgitation, like rupture of chords or papillary muscles due to an acute coronary syndrome. In this regard, transesophageal echocardiography was mandatory for a better characterization of mitral valve apparatus. Also, even after excluding the diagnosis of acute coronary syndrome (negative troponin, ECG without ischaemic modifications), coronary angiography was indicated given the typical anginous chest pain. Thus, an apparently benign condition needed in our case complex paraclinical investigations for a complete diagnosis.

Mitral valve prolapse is two-times more common in women, but in can be also present in men. Our case involved a male patient with this annomaly which was another particularity. Also, mitral valve prolapse is more frequently associated with ventricular arrhythmias, which may have an impact on prognosis. In this context, Holter ECG monitoring is useful in detecting arrhytmic complication. For the case presented, Holter ECG monitoring showed episodes of atrial fibrillation, probably due to high atrial pressures in the context of severe mitral and tricuspid regurgitations. The presence of atrial fibrillation changes the therapeutic approach, with an indication for therapeutic anticoagulation.

Anterior chest pain was the main symptom that guided the diagnostic strategy. An incidental discovery of a mitral valve prolapse differs significantly from the situation in which the diagnosis is established in the context of the acute onset of symptoms. After ruling out an acute coronary syndrome complicated by papillary muscle rupture, we still needed to understand the cause of the patient's chest pain for an appropriate therapeutic target. In our case, the possible causes for chest pain were: microvascular angina, traction on the chordae, pulmonary hypertension and severe

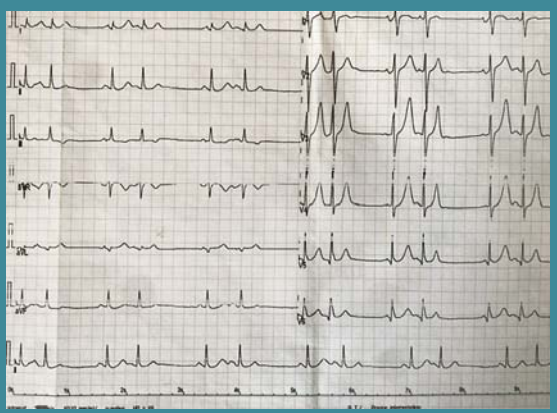


Figure 1. *ECG tracing*

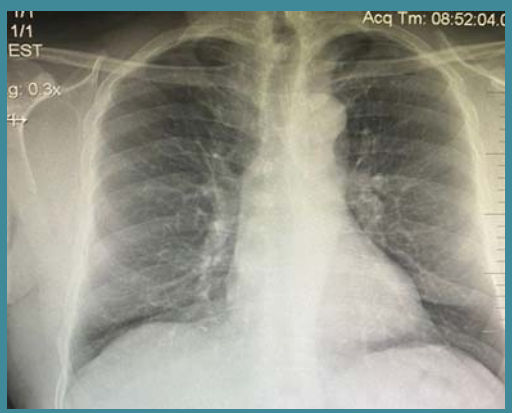


Figure 2. *Chest X-Ray*

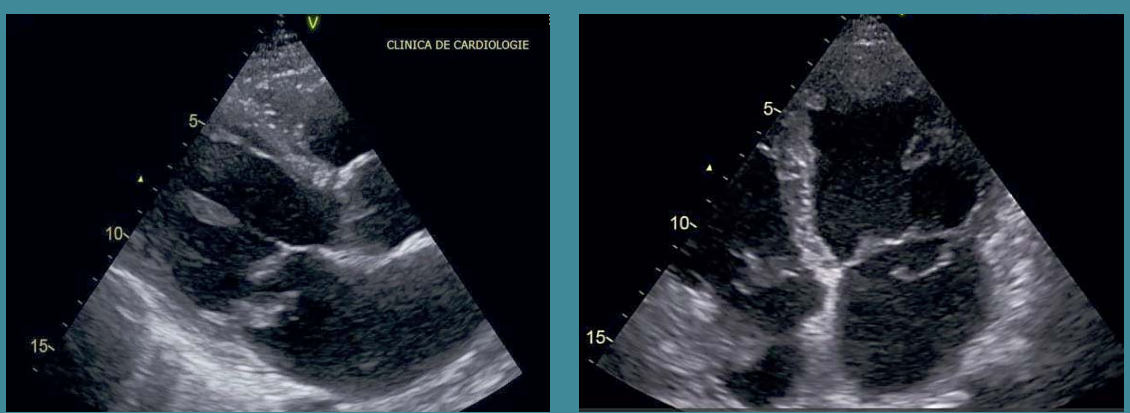


Figure 3. *Echocardiography - 2D imaging, PLAX and Apical 4 chamber view*

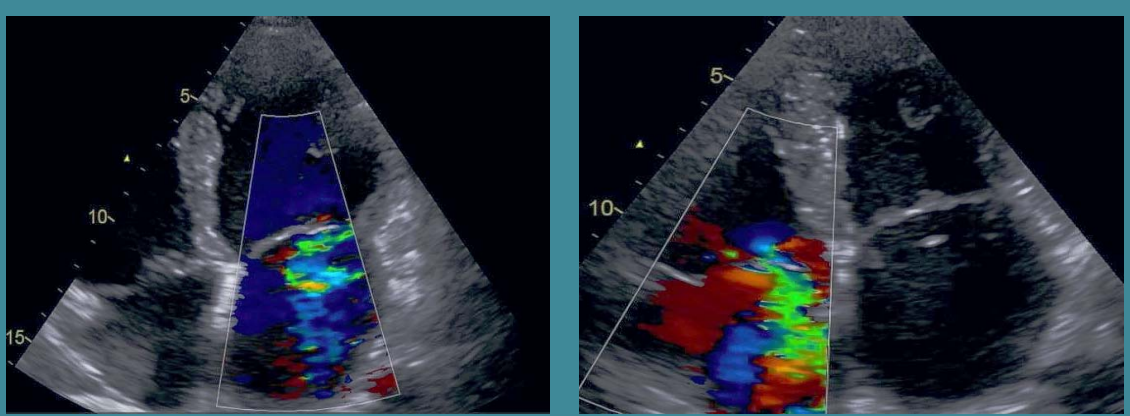


Figure 4. *Echocardiography - Color Dopper, Apical 4 Chamber view, Severe Mitral and Tricuspid regurgitations*



mitral regurgitation, reversible after surgical intervention on the mitral valve.

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