



The obstruction and the repercussion

<https://doi.org/10.56238/homeIIsevenhealth-018>

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1 INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is a primary heart disease characterized by hypertrophy of the left ventricular muscle in the absence of cardiac or systemic disease that alone justifies the severity of this hypertrophy (hypertension or valvular heart disease). The obstructive form has a worse prognosis, involves more associated symptoms, leads to a worse quality of life, and increases the risk of sudden cardiac death.

Aortic stenosis, in turn, causes the functional area of the valve to decrease, causing obstruction to the flow. When left untreated it becomes symptomatic, with syncope, angina, and exertional dyspnea being the most frequent symptoms.

2 CLINICAL CASE

Case report of an 84-year-old female patient, Caucasian, admitted due to a consumptive condition for study and probable hematological neoplasm. Her personal history included type 2 diabetes mellitus, chronic pain, severe mitral valve insufficiency (valvular dysfunction) and pulmonary hypertension.

On admission to the emergency department, he presented with chest discomfort and dyspnea. On objective examination, cardiac auscultation was rhythmic and the mean heart rate (HR) was 95 bpm, revealing a holosystolic murmur at the mitral focus (grade 3/6). There was no edema in the lower limbs.

A transthoracic echocardiogram (TTE) was ordered and revealed a greatly dilated left auricle (31cm²), slightly dilated left ventricle with marked hypertrophy of the interventricular septum (17mm), preserving good overall systolic performance (LVEF 59%), with no changes in regional contractility and altered relaxation. The right chambers were of normal dimensions and the right ventricle had preserved function (TAPSE 24mm). The aortic valve showed fibrocalcification with



decreased opening, generating a mean gradient of 32mmHg and maximum velocity of 4m/s. The mitral valve was thickened by fibrosis, with good mobility, observing anterior systolic movement of the anterior leaflet of the mitral valve (SAM), conditioning a maximum intraventricular gradient of 166mmHg and severe regurgitation (IV/IV). Mild tricuspid regurgitation allowed estimating the PSAP at 38mmHg. The conclusion of the echocardiographic study suggests Hypertrophic Obstructive Myocardopathy.

Given the functional state, the therapeutic proposal excludes any other intervention than pharmacological therapy, in this case using Bisoprolol.

3 DISCUSSION

Taking into account the pathophysiology of MH, in its obstructive form (found in about 30% of these patients), there is an increased risk of progression to ventricular dilatation when compared to patients without obstruction, as we found in this case. Mitral regurgitation, almost always present in these patients, also contributes to the progressive enlargement of the left chambers. Narrowing in the left ventricular outflow chamber (LVESC) associated with mitral valve abnormalities are apparently at the origin of intraventricular obstruction and mitral regurgitation.

One of the complaints reported by the patient was precordial pain. Myocardial ischemia in MH is common, with explanation related to several pathophysiological mechanisms, among them, excessive muscle mass increase, accompanied by increased oxygen demand, insufficient coronary microvascular structure, microvascular compression and decreased aortic diastolic pressure.

The patient also reported dyspnea. This is the main clinical manifestation of this pathology (it occurs in about 90% of patients). Diastolic dysfunction, LVOT obstruction and mitral regurgitation are the main culprits. Impaired diastolic filling of the left ventricle results from decreased ventricular compliance, which in turn is due to myocyte derangement, poor ventricular geometry, and myocardial ischemia. CSVE obstruction and mitral regurgitation contribute to a decrease in aortic flow, while promoting an increase in pulmonary pressures, impacting the symptom in question.

The pharmacological option based on the use of β -blockers (Bisoprolol), recommended in patients with angina and dyspnea, is related to the need to decrease HR with consequent increase in diastole time, improvement in ventricular relaxation and decrease in excitability by its negative inotropic and antiarrhythmic effect.

4 CONCLUDING REMARKS

The literature review carried out to answer the questions imposed by our clinical case allowed us to conclude that patients with the obstructive form of HC have on auscultation a systolic murmur of variable degree, according to the severity of the obstruction and mitral regurgitation. Regarding



symptomatology, dyspnea and angina are the most recurrent possible symptoms. TTE reveals changes compatible with the pathology in question, namely changes in the mitral valve apparatus and hypertrophy of the wall(s), leading to hemodynamic repercussions (LVOT obstruction and mitral regurgitation) that may be significant. Diastolic dysfunction, present in the vast majority of cases, contributes substantially to the magnitude of the symptoms. The therapeutic decision can range from purely pharmacological to surgical intervention, and is conditioned by several factors, including functional capacity, age, and symptoms.



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