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ALCOHOLIC CARDIOMYOPATHY: A CASE REPORT

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ABSTRACT

Introducion: Alcoholic cardiomyopathy is a type of acquired dilated cardiomyopathy associated with excessive and prolonged alcohol consumption. There are two phases: the preclinical phase and the symptomatic phase (characterized by signs and symptoms of heart failure). The diagnosis is often made at a later stage, with significant impairment of overall systolic function. **Objective:** To report the clinical case of a patient with alcoholic cardiomyopathy. **Case Report:** A 62-year-old male patient, hypertensive, with a history of hospitalization for upper gastrointestinal bleeding, presented for consultation to adjust antihypertensive medication. No significant changes were observed in the semiological examination. Upon physical examination, left heart cavity dilation and an ejection fraction of 34% were detected. Following this, a cardiac magnetic resonance imaging was requested, which confirmed the diagnosis of dilated cardiomyopathy, likely of alcoholic etiology, and an implantable cardioverter-defibrillator (ICD) was recommended. **Final considerations:** Dilated cardiomyopathy is a disease with a significant impact on morbidity, mortality, and patients' quality of life, and its early diagnosis is essential for preventing or slowing the progression of the disease. Patient education on the adverse effects associated with excessive alcohol consumption is crucial for preventing the development of this pathology and/or the progression of heart failure.

Keywords: Alcohol; Alcoholic cardiomyopathy; Heart failure.

INTRODUCTION

Alcoholic cardiomyopathy, formerly known as alcoholic heart muscle disease, is a type of acquired dilated cardiomyopathy that occurs in two distinct phases: an initial preclinical phase and a symptomatic phase, characterized by excessive and prolonged alcohol consumption.[1]

During the progression of cardiac dysfunction, certain signs of abnormality can be identified before it becomes clinically evident. Detecting these signs is crucial because, in this preclinical/asymptomatic phase, early cessation of alcohol consumption can reverse left ventricular (LV) dysfunction.[2] In asymptomatic patients, based on the duration of alcohol use, the following echocardiographic findings may be observed: after 5 to 9 years of consumption, an increase in LV volume and prolonged isovolumic relaxation time; after 10 to 15 years, an increase in LV mass and deceleration time; and after 16 to 28 years, an increase in the peak A-wave velocity and a decreased E/A wave peak ratio.[3] Other studies also commonly show echocardiographic findings of diastolic dysfunction in asymptomatic patients.[4]

The exact prevalence of alcoholic cardiomyopathy remains to be determined.[5] However, alcoholic cardiomyopathy accounts for 21 to 36% of non-ischemic dilated cardiomyopathy (DCM) cases in Western societies, and without alcohol abstinence, it has an approximate 4-year mortality rate of 50%6. In the United States, alcoholic cardiomyopathy is the most common cause of non-ischemic dilated cardiomyopathy, representing 3.8% of all cardiomyopathies.[7]

In alcoholic cardiomyopathy, two distinct phases are recognized in the disease's natural progression: an initial preclinical/asymptomatic phase, characterized by left ventricular (LV) dilation, with or without diastolic dysfunction, and a second clinical phase, presenting classic symptoms of heart failure (HF), such as dyspnea, orthopnea, edema, nocturia, and tachycardia. HF symptoms may result from initial diastolic dysfunction or later systolic dysfunction. In advanced stages, when the risk of developing atrial fibrillation (AF) increases, there is a possibility of thrombus formation in the dilated atria.[8]

A key factor in identifying alcoholic cardiomyopathy is a history of chronic excessive alcohol consumption, along with a range of cellular, histological, and structural changes in the myocardium that may be present in these individuals.[9]

In clinical practice, echocardiography is the primary imaging test used to monitor cardiac function, while other tests, such as electrocardiography and magnetic resonance imaging, may also be utilized. In patients with alcoholic cardiomyopathy, the echocardiogram may show a dilated left ventricle, with increased diastolic and systolic dimensions and a reduced ejection fraction. Early detection of these echocardiographic signs, which indicate cardiac abnormality, can lead to earlier treatment and, consequently, a better prognosis.[10]

Endomyocardial biopsy remains the gold standard for diagnosing many cardiac conditions, both primary and secondary. However, while there are indeed pathologies such as amyloidosis and cardiac sarcoidosis that can be definitively diagnosed by cardiac biopsy, other etiologies display less specific histopathological features, so their definitive diagnosis is not histological.[11]

In treating individuals with alcoholic cardiomyopathy (ACM), two main objectives should be considered: preventing further damage to the heart muscle by stopping alcohol consumption and reducing cardiac dilation.[12] Total alcohol abstinence is necessary, and additional measures include promoting proper nutritional habits, smoking cessation, and other healthy practices. Thus, ACM treatment follows the standardized heart failure (HF) treatment regimen, including ACE inhibitors, beta-blockers, diuretics, and digitalis, along with anticoagulants when appropriate.[13]

In light of this, the overall objective of this study is to analyze the national and international scientific production indexed in the databases LILACS, SciELO, and PubMed, to report the clinical case of a patient with alcoholic cardiomyopathy.

CASE REPORT

A 62-year-old male patient from Nova Veneza, GO, attended a private consultation for medication adjustment for hypertension on June 5, 2024.

He had a history of hospitalization 7 years before due to hematemesis associated with abdominal pain. However, there was no history of the use of potentially ulcerogenic medications, gastritis, H. pylori infection, peptic ulcer disease, malignancy, angiodysplasia, aortoenteric fistula, or gastroenteric anastomosis. An important factor was his alcohol consumption since his youth.

Upon physical examination, the patient weighed 83 kg; his height was 1.70 m, resulting in a BMI of 28.7 kg/m². Respiratory rate was normal, heart rate was 70 bpm, and blood pressure was 110/80 mmHg. In the cardiovascular examination, the heart rhythm was regular, with normal heart sounds present in two beats, and no murmurs; the abdomen was flat, with present bowel sounds, tympanic, non-tender, and without signs of portal hypertension. No edema was observed in the lower limbs, and the calves were free. He reported alcohol consumption of 200 ml of distilled spirits daily from ages 30 to 50, after which his intake increased to 2 liters of beer per day. He has a

history of systemic arterial hypertension and is taking captopril 25 mg once daily. He denied smoking, illicit drug use, and engaging in physical activity.

Laboratory tests conducted on May 6, 2024, revealed the following results: hemoglobin 13 g/dL, hematocrit 40.3%, leukocytes 11 k/uL (56.2% neutrophils, 1% basophils, 29% lymphocytes, 12.7% monocytes), glucose 85 mg/dL; hemoglobin A1c of 5.7%; creatinine 1.1 mg/dL; potassium 3.6 mEq/L; urinalysis: specific gravity 1.025, proteinuria 15 mg/L, sediment: leukocytes 3,000/mL, erythrocytes 1,000, hyaline casts 0/mL. TSH was 0.7 U/L; free T4 was 6 µg/L; NT-pro BNP was 759 pg/mL; total cholesterol was 148 mg/dL; HDL was 35 mg/dL; LDL was 101 mg/dL; and triglycerides were 62 mg/dL. The serology for Chagas disease was negative.

The transthoracic echocardiogram conducted on May 6, 2024, revealed an aortic diameter of 37 mm, a left atrium measuring 42 mm, a left ventricle diastolic diameter of 62 mm, and a systolic diameter of 53 mm, with a significantly reduced left ventricular ejection fraction of 30%. There were no valvular abnormalities. Thus, a moderately enlarged left atrium was observed, with the left ventricle showing mild eccentric myocardial hypertrophy and significant impairment of systolic function.

The 24-hour Holter monitor conducted on May 6, 2024, revealed a regular rhythm, with a PR interval within normal limits, interventricular conduction disturbance with a QRS complex duration of 170 ms, and the presence of isolated supraventricular ectopic beats. No pauses were observed.

Based on this, the presence of heart failure with reduced ejection fraction (HFrEF) was evidenced. To complement the diagnosis, a cardiac magnetic resonance imaging (MRI) was requested to assess for fibrosis and myocardial viability to elucidate the etiology of the heart failure, and the patient was advised to cease alcohol consumption. Captopril was also discontinued, and the following medications were initiated: Forxiga 10 mg, losartan 50 mg, spironolactone 25 mg, Concardio 1.25 mg, and amiodarone 200 mg.

At the follow-up on June 18, 2024, the MRI revealed a left ventricle with significantly increased dimensions and important global systolic dysfunction, with the presence of septal dyssynchrony, as well as faint linear basal septal mesocardial fibrosis (non-ischemic pattern), which is frequently found in dilated cardiomyopathy. A small late enhancement in the inferior junction was also noted, which is often seen in right chamber overload. Therefore, an implantable cardioverter-defibrillator (ICD) implantation was requested to prevent sudden death in patients with heart failure and reduced ejection fraction of non-ischemic etiology.

DISCUSSION

According to epidemiological data, alcoholic cardiomyopathy is one of the main non-ischemic etiologies of heart failure in the Western world[14]. The development of alcoholic cardiomyopathy appears to be related to the amount of alcohol consumed daily and the duration of the period of alcohol abuse. Although the exact quantity and duration of abuse are not well defined, a consumption exceeding 80 g/day for at least 5 years is associated with an increased risk of developing cardiomyopathy.[15]

The prevalence of alcoholic cardiomyopathy is higher in men due to the greater prevalence of alcoholism in the male sex[16], as observed in the above report. However, women reach a higher blood alcohol concentration than men for the same amount of alcohol consumed.[17]

A history of chronic alcohol abuse in the absence of other etiologies of dilated cardiomyopathy suggests a diagnosis of alcoholic cardiomyopathy. The findings on chest radiography are similar to those seen in other causes of cardiomyopathies, such as cardiomegaly, pulmonary congestion, and pleural effusion.[18] The electrocardiogram (ECG) is also non-specific and may show ST segment and T wave changes, low voltage in the presence of significant fibrosis, bundle branch blocks, and cardiac arrhythmias. Biomarkers, such as natriuretic peptides and high-sensitivity troponins, may be elevated and should be interpreted similarly to other etiologies of heart failure. The

echocardiogram can help exclude other causes of heart failure and define the phenotypic pattern of hypertrophy, dilation, diastolic dysfunction, or left ventricular systolic dysfunction, which may precede the onset of symptoms. Cardiac magnetic resonance imaging may reveal areas of late enhancement, indicating myocardial fibrosis.[19]
Abstinence from alcohol is fundamental in the treatment of alcoholic cardiomyopathy. Heart failure syndrome should be managed similarly to other etiologies. Therefore, beta-blockers, angiotensin-converting enzyme inhibitors or angiotensin receptor blockers, spironolactone, and diuretics for cases of congestion are indicated.[20]
In certain groups with heart failure, such as our patient, it has been shown that an implantable cardioverter-defibrillator (ICD) can prolong patient survival. It may be indicated to prevent sudden death in patients with heart failure and reduced ejection fraction (HFrEF) who have left ventricular dysfunction due to anterior myocardial infarction (MI) with an ejection fraction (EF) of 30-40% or less; those with sustained ventricular tachycardia (VT) that is hemodynamically unstable; those with cardiac arrest due to VT/fibrillation ventricular (VF) from a non-reversible cause and EF ≤ 35%; and those with spontaneous VT with hemodynamic compromise or syncope, also from a non-reversible cause and EF ≤ 35%.[21] Important to note that an ICD should not be indicated for patients with a life expectancy of less than one year.[22]

FINAL CONSIDERATIONS

Alcoholic dilated cardiomyopathy is a myocardial dysfunction that causes heart failure, characterized by predominant ventricular dilation and systolic dysfunction. The symptoms include dyspnea, fatigue, and peripheral edema. The diagnosis is clinical and is supplemented by tests such as transthoracic echocardiography and cardiac MRI. The treatment is directed at the cause of heart failure (HF). Among the pharmacological therapies, we can mention angiotensin-converting enzyme (ACE) inhibitors, beta-blockers, aldosterone receptor blockers, angiotensin II receptor blockers, neprilysin inhibitors, sodium-glucose cotransporter 2 (SGLT2) inhibitors, hydralazine/nitrates, as well as diuretics. Furthermore, when ventricular dysfunction is significant, cardiac resynchronization therapy and implantable cardioverter-defibrillator (ICD) are indicated.

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