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(CASE REPORT)

From alcohol consumption to heart failure: Severe myocarditis with biventricular involvement, Case report

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Abstract

Background: Alcoholic cardiomyopathy (ACM) is a form of acquired dilated cardiomyopathy secondary to chronic ethanol consumption. Its association with acute inflammatory events can precipitate catastrophic heart failure. It is important to note that it is a diagnosis of exclusion.

Case presentation: A 39-year-old male with a 20-year history of chronic alcoholism presented with progressive dyspnea, palpitations, and edema following a bout of influenza. Atrial fibrillation with a rapid ventricular response and a proBNP level of 14,678 pg/mL were documented. Cardiac magnetic resonance imaging (CMR) revealed a severely dilated left ventricle with an ejection fraction (EF) of 15%, accompanied by myocardial edema (RAT 2.9) and late gadolinium enhancement, consistent with acute myocarditis progressing from a background of acute cardiomyopathy. In this case, other possible causes were first ruled out, and when none were found, this diagnosis was established.

Conclusion: This case highlights the vulnerability of myocardium with prior toxic damage to secondary inflammatory insults. Accurate diagnosis using cardiac magnetic resonance imaging (CMR) is essential to differentiate chronic structural damage from the recoverable acute process.

Keywords: Heart Failure; Cardiomyopathy; Pro BNP; Troponin; Magnetic Resonance Imaging

1. Introduction

Alcoholic cardiomyopathy (ACM) is one of the leading causes of non-ischemic heart failure in the Western world. It is defined as systolic dysfunction and ventricular dilation directly proportional to chronic and excessive alcohol consumption (more than 80 g/day for >5 years), in the absence of other heart diseases (1,2). Pathophysiologically, ethanol and acetaldehyde induce oxidative stress, mitochondrial dysfunction, and myocyte apoptosis (4). Although the diagnosis is one of exclusion, the coexistence of acute inflammatory processes, such as myocarditis, can exacerbate the clinical phenotype, leading to critical states of hypoperfusion and complex arrhythmias (3).

2. Clinical case presentation

This is a 39-year-old male patient with a 20-year history of chronic alcoholism, during which he consumed alcohol weekly until intoxication. He presented with a one-month history of palpitations, progressive dyspnea, orthopnea, and lower extremity edema, following a flu-like respiratory illness. Atrial fibrillation with rapid ventricular response, dyspnea, and grade II lower extremity edema were identified distal hypoperfusion Tests show an elevated PRO-BNP

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(14,678 pg/mL), consistent with acute heart failure, and a slightly elevated troponin T (0.016 ng/mL) suggests mild myocardial injury. The chest X-ray reveals an enlarged cardiac silhouette and mild pulmonary congestion.

Cardiac magnetic resonance imaging confirms a markedly dilated left ventricle, with severely increased end-diastolic and end-systolic volumes and an ejection fraction of 15%, severe diffuse hypokinesis, eccentric hypertrophy, and left atrial dilation. The right ventricle is of normal size but exhibits moderate systolic dysfunction (RVE 42%) and epicardial late gadolinium enhancement in the free wall, indicating biventricular involvement. Tissue findings show increased T1, T2, and extracellular volume, along with patchy intramyocardial late gadolinium enhancement in multiple segments of the left ventricle and edema with a 2.9 ratio, consistent with acute myocarditis. Mild pericardial effusion and bilateral pleural effusion are also present. Overall, this is a case of severe acute myocarditis progressing to dilated cardiomyopathy, heart failure, and high-response atrial fibrillation. All of the above is based on the exclusion of infectious causes such as Chagas disease with negative *Trypanosome IgG e IgM*, and virus etiologies with negative *Coxsackie* serology, for which serologies were negative, as well as viral etiologies, along with dismissing a possible ischemic cause, for which there is coronary arteriography which shows healthy epicardial arteries.

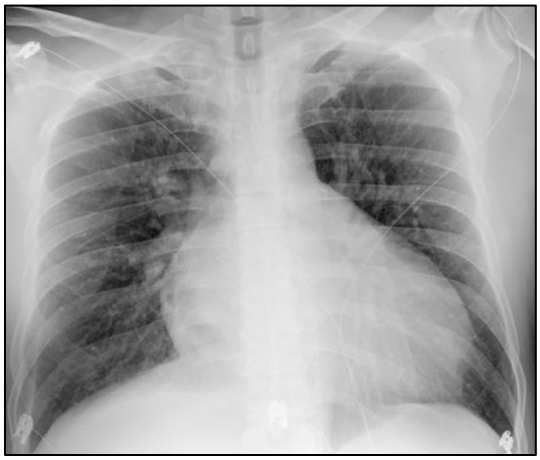

Figure 1 Chest X-ray: Patent trachea. Enlarged cardiac silhouette. Normal aorta. Prominent hila. Opacity with tendency towards consolidation at the right base. Bone structures without alterations

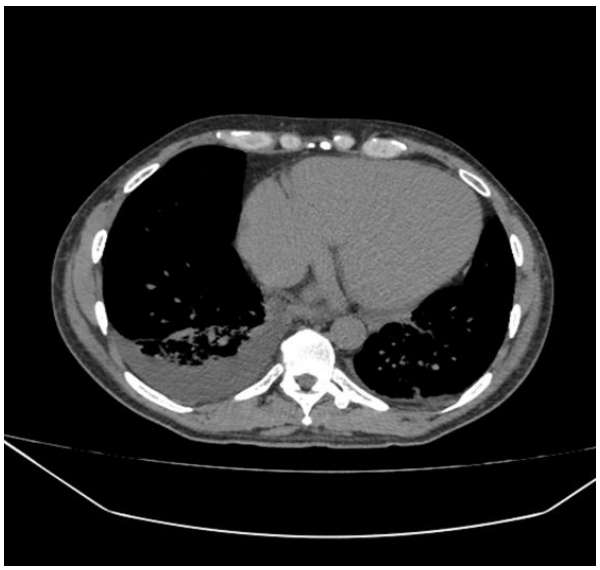

Figure 2 Chest CT scan: Marked increase in cardiothoracic ratio, indicating pericardial effusion in its posteroinferior recess measuring 31 mm. No lymphadenopathy within the pathological range. The esophageal tract and azygos arch are normal. Bone structures and soft tissues have typical CT characteristics

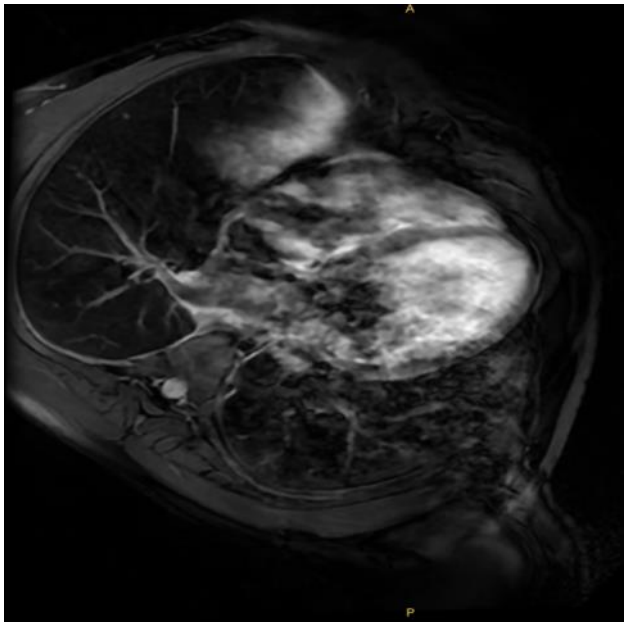

Figure 3 Cardiac magnetic resonance imaging: Dilated cardiomyopathy with severe systolic dysfunction (LVEF 15%). Normal-sized right ventricle with systolic dysfunction (RVEF 42%). Severe diffuse hypokinesis. Eccentric hypertrophy. Diffuse right ventricular hypokinesis. Left atrial dilation. Non-ischemic late gadolinium enhancement was observed in the anterior, anterolateral, and inferior walls of the left ventricle with edema on STIR sequences, with an ER (edema ratio) greater than 2. Tissue characterization by T1, T2, and ECF mapping showed increased T1, T2, and ECF values in areas of edema. Epicardial late gadolinium enhancement was also observed in the right ventricular free wall, findings consistent with acute myocarditis. Mild global pericardial effusion without hemodynamic compromise. Mild bilateral pleural effusion. The study was performed in the presence of atrial fibrillation

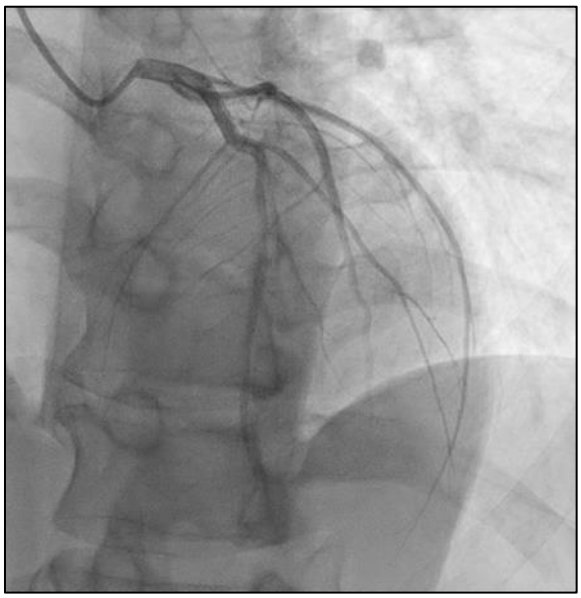

Figure 4 Coronary arteriography: no angiographically significant lesions

3. Discussion

Alcoholic cardiomyopathy (ACM) is defined as a disease of the heart muscle characterized by dilation and impaired systolic function of one or both ventricles, secondary to chronic and excessive alcohol consumption in the absence of other pre-existing heart conditions. Epidemiologically, it is responsible for up to one-third of cases of non-ischemic

dilated cardiomyopathy in Western countries, predominantly affecting men between the ages of 40 and 50, as in the 39-year-old patient reported here. A risk threshold has been established with an intake of more than 80 g/day of ethanol for at least five years, although genetic susceptibility, particularly polymorphisms in the alcohol dehydrogenase enzyme and the titin gene (TTN), can accelerate the clinical phenotype even with lower consumption levels (1–3). In this case, the 20-year history of weekly drinking leading to intoxication constitutes a cumulative toxic load that explains the severe structural remodeling observed.

The pathophysiology of acute cardiomyopathy (ACM) is multifactorial and centers on the direct toxicity of ethanol and its primary metabolite, acetaldehyde. These compounds induce massive oxidative stress by increasing the production of reactive oxygen species (ROS) and uncoupling the mitochondrial electron transport chain, resulting in impaired cellular bioenergetics. Simultaneously, there is an alteration in intracellular calcium homeostasis and in the synthesis of contractile proteins, promoting myocyte apoptosis and fibroblast activation, which generate diffuse interstitial fibrosis (4–9). A critical aspect in this patient is the transition from chronic toxic injury to acute heart failure; it is postulated that alcohol sensitizes the myocardium ("second hit"), such that a subsequent insult, like the reported flu-like respiratory symptoms, triggers a disproportionate inflammatory response that explains the myocardial edema and the drastic drop in left ventricular ejection fraction (LVEF) to 15%.

When comparing this case with the literature, cardiac magnetic resonance imaging (CMR) emerges as the gold standard for differential diagnosis. While classic acute cardiomyopathy (ACM) presents with ventricular dilation and absent or minimal late gadolinium enhancement (LGE), the presence in this patient of a patchy intramyocardial LGE pattern and an edema ratio of 2.9 confirms superimposed acute myocarditis (10,12). This etiological collision is crucial, since myocarditis itself can mimic ACM, but the degree of dilation and pre-existing fibrosis suggest that alcohol had already compromised the cardiac architecture. The extreme elevation of pro-BNP (14,678 pg/mL) reflects not only volume overload but also the critical wall stress of a ventricle that has lost its contractile reserve after decades of ethanol exposure.

The importance of timely diagnosis lies in the potential for partial or complete reversibility of acute cardiomyopathy (ACM) through absolute abstinence, especially in phases where fibrosis is not transmural. Unlike other dilated cardiomyopathies, early cessation of alcohol consumption can normalize the ejection fraction and reduce end-systolic volume (1,3). However, delays in identifying this condition, often due to underreporting of alcohol consumption in the patient's history, lead to irreversible complications such as persistent atrial fibrillation, malignant ventricular arrhythmias, and sudden death. In conclusion, this case underscores that in cases of acute heart failure with a history of alcoholism, multimodal evaluation with cardiac magnetic resonance imaging (CMR) is imperative to distinguish between chronic toxic damage and acute inflammatory processes, thus enabling a targeted therapeutic strategy that maximizes functional recovery (1, 10–12).

4. Conclusions

Alcoholic cardiomyopathy is a challenging condition requiring a holistic approach. Current scientific evidence documents that patients with this condition have a high probability of developing arrhythmic, structural, and functional complications, as well as recurrent acute decompensations with a variety of presenting phenotypes. Therefore, early diagnosis is crucial to initiate targeted treatment, beginning with the exclusion of other possible causes of heart failure.

Compliance with ethical standards

Disclosure of conflict of interest

The authors have no statement to make.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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