Case Report ISSN 2771-5906

Trends in Internal Medicine

Alcoholic Cardiomyopathy, The Silent Enemy of the Heart: A Case Report

David Fernando Ortiz-Pérez[1,2*], Camilo Lopera-Martínez[3], Daniela López-Quintero[3], Camila Andrea Veloza-Valcárcel[3], Jhonatan Rodríguez-Vásquez[4], Robin Luis Petro-Noriega[4], María Isabel Arias-Bello[5], Marianella Molina-Angarita[6], Jhon Alexander Perez-Sanmiguel[7] and Carlos Mauricio De La Peña Pérez[1,2]

[1]*Department of Internal Medicine, Cartagena del Mar Medical Center, Cartagena, Colombia.*

[2]*Internal Medicine Program, Universidad del Sinú, Cartagena, Colombia.*

[3]*General Physician, University of Cartagena, Cartagena, Colombia.*

[4]*General Physician, University of Sinú, Cartagena, Colombia.*

[5]*General Physician, Simón Bolívar University, Barranquilla, Colombia.*

[6]*General Physician, University of the North, Barranquilla, Colombia.*

[7]*General Physician, Rafael Nuñez University, Cartagena, Colombia.*

***Correspondence:**
David Fernando Ortiz-Pérez, Department of Internal Medicine, Cartagena del Mar Medical Center, Cartagena, Colombia.

Received: 20 Feb 2025; **Accepted:** 22 Mar 2025; **Published:** 07 April 2025

Citation: David Fernando Ortiz-Pérez, Camilo Lopera-Martínez, Daniela López-Quintero, et al. Alcoholic Cardiomyopathy, The Silent Enemy of the Heart: A Case Report. Trends Int Med. 2025; 5(1): 1-4.

ABSTRACT

Alcoholic cardiomyopathy is a form of acquired dilated cardiomyopathy associated with chronic, excessive alcohol consumption, defined as more than 80 g/day for a minimum of five years. Its pathophysiology involves activation of the renin–angiotensin–aldosterone system (RAAS) and direct oxidative damage from ethanol, primarily via its main metabolite, acetaldehyde. This metabolite impairs mitochondrial function, promotes fibrosis and apoptosis, and affects structural myocardial proteins such as actin and myosin. Clinically, it presents as heart failure, showing no specific features that distinguish it from other dilated cardiomyopathies. Diagnosis is based on a history of chronic alcohol use, the presence of withdrawal symptoms, and imaging findings particularly on echocardiography. We describe the case of a 55-year-old man with a 25-year history of heavy alcohol intake (AUDIT score of 34), who presented with resting dyspnea, intolerance to lying flat, and mild oppressive chest pain. Physical examination revealed a displaced point of maximal impulse and lower-limb edema. Echocardiography confirmed a dilated cardiomyopathy with a left ventricular ejection fraction of 17%. Other etiologies were excluded through additional tests, including low vitamin B12 levels, negative serology for Chagas disease, and normal coronary angiography, supporting the diagnosis of alcoholic cardiomyopathy. This case underscores the importance of a detailed clinical history and the use of imaging techniques for prompt diagnosis, as well as the toxic impact of chronic alcohol use on the myocardium. Furthermore, ruling out other structural and ischemic causes confirms alcohol as the main etiological factor in the development of this cardiomyopathy.

Keywords

Alcoholic Cardiomyopathy, Heart Failure, Ethanol, Left Ventricular Dysfunction, Alcohol-Induced Disorders.

Introduction

Excessive alcohol consumption is a substantial public health concern worldwide, with far-reaching social, economic, and healthcare implications [1]. In addition to its social and behavioral consequences, alcohol has been linked to multiple medical conditions, including hepatic, neurological, and cardiovascular disorders.

Estimates suggest that a significant portion of the global population has consumed alcohol at least once since adolescence, highlighting the early and widespread exposure to this substance [1,2]. Among the medical complications associated with alcohol abuse, cardiovascular manifestations are particularly notable. Within this spectrum, alcohol-induced cardiomyopathy is one of the primary forms of myocardial damage [2,3]. It is a type of dilated cardiomyopathy that develops after several years of heavy alcohol intake, generally defined as a daily consumption exceeding 80 grams for at least five years [1]. Before establishing this diagnosis, it is crucial to rule out other potential causes of dilated cardiomyopathy, such as valvular disease, ischemic heart disease, hypertension, or systemic disorders that affect the cardiac muscle [1,2,4].

The following diagnostic criteria have been proposed to confirm the alcoholic etiology of cardiomyopathy:

1. **History of excessive alcohol consumption:** More than 80 grams per day for at least five years.
2. **Echocardiographic findings:** An increase in the left ventricular end-diastolic diameter exceeding two standard deviations from the normal value and an ejection fraction below 50%.
3. **Exclusion of other etiologies:** Essential to ensure that myocardial dysfunction is not attributable to valvular, ischemic, hypertensive, or systemic causes.

The prevalence of alcoholic cardiomyopathy among dilated cardiomyopathies varies depending on the populations studied and the diagnostic criteria used [5]. It occurs more frequently in men, although underreporting of excessive alcohol consumption in women due to cultural factors, as well as potential differences in alcohol metabolism, could contribute to a lower recorded incidence [1,5,6].

In this context, we present the case of a patient with a 25-year history of chronic alcohol consumption who was admitted with clinical features of heart failure and chest pain. Following an exhaustive evaluation that excluded other etiologies of dilated cardiomyopathy, a diagnosis of alcohol-induced cardiomyopathy was established. This case highlights the relevance of chronic alcohol use as a contributing factor to myocardial dysfunction and underscores the importance of a comprehensive clinical assessment to ensure accurate diagnosis and timely treatment.

Case Presentation

A 55-year-old male patient with no known medical history was admitted due to a rapid decline in functional capacity over the course of one week. He reported progressive dyspnea reaching New York Heart Association (NYHA) Class IV, as well as non-radiating, oppressive chest pain rated 6/10 on the visual analog scale in the preceding 24 hours.

On initial physical examination, his heart rate was 94 beats per minute, blood pressure 104/59 mmHg, and respiratory rate 23 breaths per minute. Significant findings included displacement of the point of maximal impulse toward the axillary line and 2+ pitting edema in the lower limbs. An electrocardiogram (ECG) revealed a complete left bundle branch block and left ventricular hypertrophy as per the Peguero-Lo Presti criteria. Laboratory tests, including two measurements of high-sensitivity troponin I, were negative. The lipid profile showed elevated LDL levels, while complete blood count, renal function, and uric acid were within normal limits. Given these initial clinical and laboratory findings, an echocardiogram was performed, confirming dilated cardiomyopathy with a severely reduced left ventricular ejection fraction (17%) (Figure 1). In order to exclude coronary artery disease, a percutaneous coronary angiography was carried out, revealing no significant angiographic lesions.

Once ischemic heart disease was ruled out, further investigations for metabolic or infectious causes were conducted. Serologic tests for Chagas disease were negative, but a notable vitamin B12 deficiency was identified (90 pg/mL; reference range 160–950 pg/mL). This finding prompted a more in-depth exploration of his lifestyle habits, revealing 25 years of excessive alcohol consumption with an AUDIT score of 34, corresponding to a daily intake of over seven standard drinks. Based on these results, a diagnosis of alcohol-induced dilated cardiomyopathy was established. Upon discharge, standard therapy for heart failure with reduced ejection fraction was initiated, featuring spironolactone, empagliflozin, metoprolol succinate, and sacubitril/valsartan. In addition, the patient was scheduled for close cardiology follow-up and referred to social services and psychology for cognitive-behavioral therapy, aiming to address alcohol dependence and promote long-term abstinence.

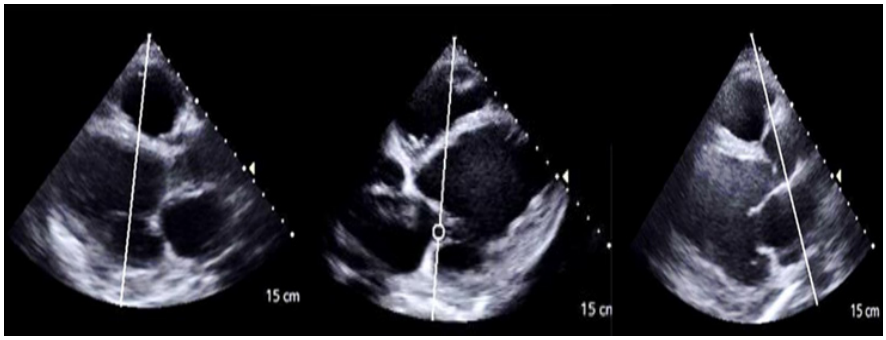

Figure 1: Transthoracic echocardiogram: Demonstrates marked dilation of the right heart chambers, global left ventricular hypokinesia, and an ejection fraction of 17%.

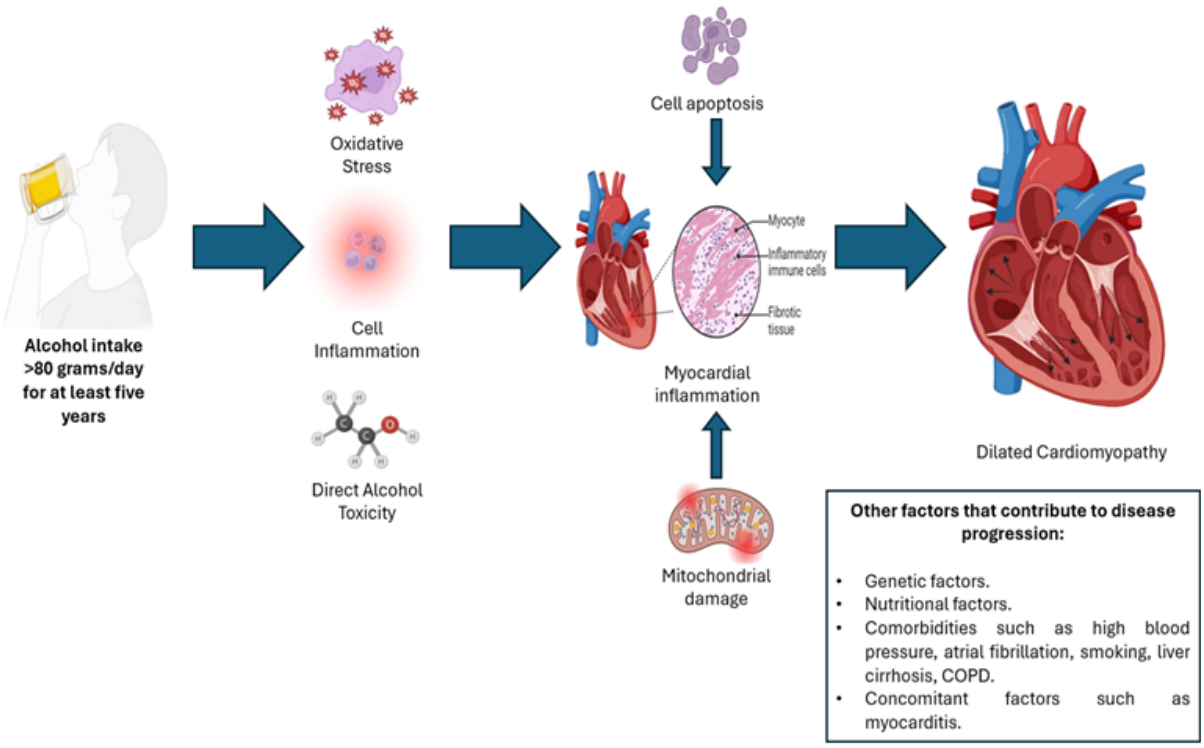

Figure 2: Pathogenic mechanisms of alcohol-induced cardiomyopathy.
Source: Figure created by the author, using, BioRender.com.

Discussion

Alcoholic cardiomyopathy is a variant of dilated cardiomyopathy triggered by prolonged, excessive alcohol use, characterized by left ventricular enlargement and systolic dysfunction [1,4]. This deterioration usually manifests as heart failure, sharing similar clinical features with other forms of dilated cardiomyopathy, which can make differential diagnosis challenging [1,2,5].

From a pathophysiological perspective (Figure 1), chronic alcohol consumption initiates multiple processes that compromise myocardial function. Both ethanol and its primary metabolite, acetaldehyde, generate significant oxidative stress by promoting the accumulation of reactive oxygen species, disrupting mitochondrial function, and creating an energy imbalance in cardiac cells [7-10]. These events foster apoptosis and fibrosis, ultimately causing irreversible myocardial structural changes. Furthermore, the persistent activation of the renin–angiotensin–aldosterone system (RAAS) contributes to adverse ventricular remodeling, exacerbating systolic dysfunction. Nutritional deficiencies particularly vitamin B12 and folate may aggravate these effects by interfering with cellular maintenance and repair mechanisms [1,2,4,8,9].

Clinically, patients with alcoholic cardiomyopathy often present with nonspecific findings [1]. In this case, the patient's symptoms were classic for heart failure, including dyspnea progressing to NYHA Class IV, orthopnea, supine intolerance, and lower-limb edema. Physical examination revealed a displaced point of maximal impulse, while ECG findings included complete left bundle branch block and signs of left ventricular hypertrophy. These features, together with the severely reduced ejection fraction, aligned with marked systolic dysfunction. Normal troponin levels ruled out an acute coronary syndrome, and negative coronary angiography, coupled with negative serologies for infectious causes, allowed for exclusion of other structural or infectious etiologies. Diagnosis hinges on careful etiological investigation, with a thorough history of alcohol use serving as a cornerstone. The clinical presentation is often indistinguishable from other dilated cardiomyopathies, necessitating a high index of suspicion, particularly in patients with no known prior cardiac disease [1-3,5]. Therapeutic management primarily involves complete cessation of alcohol intake, a crucial measure to halt the progression of myocardial damage and potentially restore partial ventricular function in earlier stages [1,3,5,11,12]. Pharmacological treatment follows standard guidelines for heart failure with reduced ejection fraction, including beta-blockers, RAAS inhibitors, mineralocorticoid receptor antagonists, and sodium-glucose co-transporter 2 inhibitors [5,11]. A multidisciplinary strategy is vital, addressing not only heart failure management but also alcohol dependence through psychological support, nutritional counseling, and cardiac rehabilitation programs. Early detection and prompt intervention are critical because delays in treatment may reduce reversibility of myocardial injury and significantly worsen long-term outcomes [1-3,5,8].

Conclusion

This case clearly demonstrates the association between long-standing alcohol consumption and the development of

dilated cardiomyopathy. It also emphasizes the need for a detailed assessment of alcohol intake in patients with heart failure of unknown etiology. Imaging modalities, particularly echocardiography, and the exclusion of other structural and ischemic etiologies confirm alcohol as the primary causative factor in this form of cardiomyopathy. Early recognition and comprehensive management including abstinence and a multidisciplinary approach are crucial to improving prognosis and quality of life in affected patients.

Reference

1. Domínguez F, Adler E, García-Pavía P. Alcoholic cardiomyopathy: An update. Eur Heart J. 2024; 45: 2294-2305.
2. Guzzo-Merello G. Alcoholic cardiomyopathy. World J Cardiol. 2014; 6: 771-781.
3. https://www.wjgnet.com/1949-8462/full/v6/i8/771.htm
4. George A, Figueredo VM. Alcoholic cardiomyopathy: A review. J Card Fail. 2011; 17: 844-849.
5. Piano MR, Phillips SA. Alcoholic cardiomyopathy: Pathophysiologic insights. Cardiovasc Toxicol. 2014; 14: 291-308.
6. Arbelo E, Protonotarios A, Gimeno JR, et al. 2023 ESC Guidelines for the management of cardiomyopathies: Developed by the task force on the management of cardiomyopathies of the European Society of Cardiology (ESC). Eur Heart J. 2023; 44: 3503-3626.
7. Dunlay SM, Roger VL, Redfield MM. Epidemiology of heart failure with. Nature Publishing Group. 2017; http://dx.doi.org/10.1038/nrcardio.2017.65
8. Albakri A. Alcoholic cardiomyopathy: A review of literature on clinical status and meta-analysis of diagnostic and clinical management methods. Clin Med Invest. 2018; 3.
9. Fernández-Solà J. The effects of ethanol on the heart: Alcoholic cardiomyopathy. Nutrients. 2020; 12: 572.
10. Villacorta H, Avila MS, Souza GEC, et al. Toxic Cardiomyopathies: Alcohol, Amphetamines, and Anabolic Steroids. ABC Heart Fail Cardiomyop. 2023; 3.
11. Ihekire NL, Okobi OE, Adedoye EA, et al. Heartache in a Bottle: Understanding Alcoholic Cardiomyopathy. Cureus. 2023; 15.
12. Heidenreich PA, Bozkurt B, Aguilar D, et al. 2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2022; 79: 263-421.
13. Tumelty E, Chung I, Hussain S, et al. An Updated Review of the Management of Chronic Heart Failure in Patients with Chronic Kidney Disease. Rev Cardiovasc Med. 2024; 25: 144.