**Case Report**

# ALCOHOLIC CARDIOMYOPATHY: A CASE REPORT PRESENTATION

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## ABSTRACT

**Introduction.** Alcoholic cardiomyopathy is a form of acquired cardiomyopathy associated with daily alcohol consumption of more than 80 g/day for a period of over 5 years. According to studies, its pathophysiology includes direct oxidative damage from ethanol and its metabolites, primarily acetaldehyde, as well as activation of the renin-angiotensin-aldosterone system.

**Methods.** We describe the case of a 35-year-old male patient with a 20-year history of alcohol consumption (AUDIT > 25), who presented to the emergency department with difficulty breathing, oedema, and numbness of the extremities, thoracic and abdominal discomfort, jaundice, and physical weakness.

**Discussion:** Alcoholic cardiomyopathy is a variant of dilated cardiomyopathy, caused by excessive alcohol use, accompanied by left ventricular enlargement and systolic dysfunction.

**Result & Conclusion.** This case study clearly shows the correlation between high levels of alcohol use and the development of dilated cardiomyopathy at a young age with multiorgan involvement. This case highlights the importance of a comprehensive evaluation of heart function. Imaging, particularly echocardiography, and the exclusion of alternative structural and ischaemic aetiologies reveal alcohol as the primary cause of this form of cardiomyopathy.

**Keywords:** Alcoholic cardiomyopathy, alcohol consumption, heart failure, ethanol

# KARDIOMIOPATIA ALKOOLIKE: PREZANTIM RASTI KLINIK

## ABSTRAKT

**Hyrje.** Kardiomiopatia alkoolike është një formë e kardiomiopatisë së fituar, e lidhur me konsumin e përditshëm të alkoolit më shumë se 80 g/ditë për një periudhë mbi 5 vjet. Studimet kanë treguar që fiziopatologjia e saj përfshin aktivizimin e sistemit renine-angitensine-aldosteron, si dhe dëmtimin oksidativ drejtpërdrejt nga etanoli dhe metabolitëve të tij kryesisht acetaldehidit.

**Metodologji.** Prezantim rastit të një pacienti, mashkull 35 vjeç, me një histori 20 vjeçare të konsumit të alkoolit (AUDIT > 25), i cili paraqitet në urgjencë me vështirësi në frymëmarrje, edemë dhe mpirje të anësive, dhimbje torakale dhe abdominale, ikter dhe dobësi trupore.

**Diskutime:** Kardiomiopatia alkoolike është një variant i kardiomiopatisë dilatuar, i shkaktuar nga përdorimi i ekzagjeruar i alkoolit, shoqëruar me zgjerimi të ventrikulit te majtë dhe mosfunksionimi sistolik.

**Rezultate & Përfundime.** Ky studim rasti tregon qartë korrelacionin midis niveleve të larta të përdorimit të alkoolit dhe zhvillimit të kardiomiopatisë së dilatuar në një moshë të re me përfshirje të shumë organeve. Ky rast nënvizon nevojën për të kryer një ekzaminim të plotë të funksionit të zemrës. Imazheria, veçanërisht ekokardiografia, dhe përjashtimi i etiologjive alternative strukturore dhe iskemike zbulojnë alkoolin si shkakun kryesor të kësaj forme të veçantë të kardiomiopatisë.

**Fjalë kyçe:** kardiomiopatia alkoolike, konsumi i alkoolit, insuficenca kardiake, ethanol

## INTRODUCTION

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

WHO's Global Status Report on alcohol and health, based on 2019 data on the public health impact of alcohol worldwide, shows an estimated 400 million people live with alcohol use disorders globally, and 209 million people live with alcohol dependence. The new report from the WHO highlights that 2.6 million deaths per year were attributable to alcohol consumption, accounting for 4.7% of all deaths. [ii]

From a health perspective, the complications of excessive alcohol use are both mental & physical. They range from psychiatric, gastrointestinal, neurological, and cardiological disturbances. In Western countries, up to 10% of the adult population suffers from an alcoholism-related disease.[iii]

Alcohol is one of the toxic substances frequently consumed globally. Although daily intake of low to moderate amounts of alcohol improves the cardiovascular health of ischemic and non-ischemic patients, chronic and excessive consumption of alcohol could result in progressive cardiac dysfunction and heart failure (HF). Chronic alcohol consumption or abuse is defined as the daily intake of more than 80 g of alcohol for a period of at least five years. [iv]

Alcoholic cardiomyopathy (ACM) is a heart disease caused by long-term alcohol use. It is marked by ventricular dilation and reduced heart function. ACM is a major cause of non-ischemic dilated cardiomyopathy.[v]

Alcohol induces several changes in the myocardial structure (myocyte loss, intracellular organelle dysfunction, and contractile protein alterations). However, the exact pathogenesis of ACM is still unclear. Total alcohol abstinence, together with heart failure treatment drugs,

results in at least partial recovery of the myocyte damage, with a consequent improvement in cardiac function.[vi]

Epidemiological studies investigating the nexus between chronic alcohol abuse and the development of ACM report a reciprocal relationship between chronic alcohol abuse and ACM. In a case-controlled study evaluating the relationship between alcohol consumption and idiopathic dilated cardiomyopathy (DCM), a correlation was demonstrated between patients diagnosed with DCM and a history of alcohol consumption. The incidence of excessive alcohol abuse among the 100 DCM patients was 40% compared to 23% in a control group of 211 healthy individuals.[4]

ACM is a leading cause of non-ischemic dilated cardiomyopathy in the United States. The incidence of alcoholic cardiomyopathy ranges from 1-2% of all heavy alcohol users. It is estimated that approximately 21-36% of all non-ischemic cardiomyopathies are attributed to alcohol. The prevalence of alcoholic cardiomyopathy in addiction units is estimated to be around 21-32 %. Overall data with regards to alcohol induced cardiomyopathy is insufficient and does not illustrate significant available data. The most common age population for ACM is males from age 30-55 with a significant history of alcohol use for more than 10 years. Females constitute roughly 14 % of cases of alcohol induced cardiomyopathy; however, the lifetime exposure required for women to develop alcohol induced cardiomyopathy is less compared to men.[5]

Early reports of clinical manifestations of ACM established by Otto von Bollinger, a German pathologist, indicated structural abnormalities such as left ventricular dilation and hypertrophy, and comorbidities such as delirium tremens and cirrhosis of the liver. Other clinical manifestations reported by William McKenzie include accelerated heart pulse, swelling and enlargement of the veins, poor prognosis with progressive HF, dilated cavities of the heart, and fatty degeneration of the ventricular walls. Having coined the expression ***"Munich beer heart"*** to demonstrate the effect of chronic alcohol abuse on the human heart in the mid-19th century, Bollinger became the first to highlight the clinical manifestations of ACM.[4]

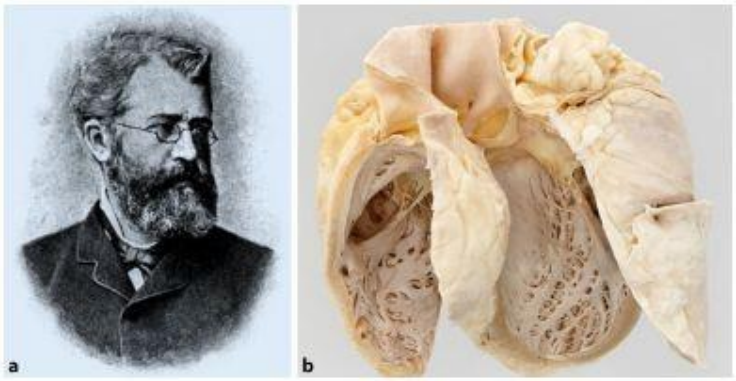


**Figure 1:** **a** Otto von Bollinger. (© de.wikipedia.org). **b** Munich beer heart.(© Philipp Mansmann in http://www.bayerische-staatszeitung.de/staatszeitung/kultur/detailansicht-kultur/artikel/ bierherz.html)[vii]

The pathogenesis of ACM also involves interaction between genetically related factors, such as HLA subtypes or the alcohol dehydrogenase enzyme allele, as well as non-genetic factors, including thiamine deficiency and exposure to various substances that are directly toxic to

cardiac cells. These structural and intracellular alterations cause activation in compensatory mechanisms in response to cardiac dysfunction, such as the renin-angiotensin-aldosterone system, increased sympathetic signal firing, and an increase in brain natriuretic peptide release.[5]

Chronic heavy alcohol abuse will also increase blood pressure and cause a downregulation of the immune system that could lead to increased susceptibility to infections, which in turn could add to the development of heart failure. Myocardial tissue analysis resembles idiopathic cardiomyopathy or chronic myocarditis. [7]

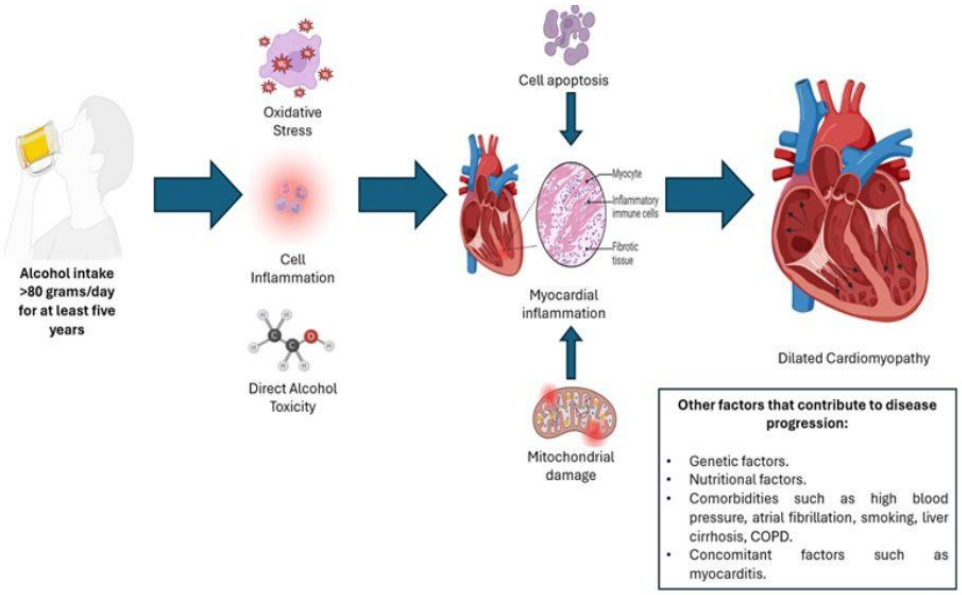


Figure 2: Pathogenic mechanisms of alcohol-induced cardiomyopathy.

Source: Figure created by the author David Fernando Ortiz-Perez, using BioRender.com[1]

Before making this diagnosis, it is essential to exclude other possible causes of dilated cardiomyopathy, such as valvular disease, ischemic heart disease, hypertension, or systemic disorders affecting the cardiac muscle, and to determine the average daily dose consumed by the patient.

Drinks as measures of alcohol are often given in ounces (oz), whereby 1 oz equals 28.35 g or 29.57 ml. Examples for 100 % alcohol in ml of one standard drink in consumed beverages are between 17.6 to 17.76 ml:

- Beer: 12 fluid ounces of 5 % beer = 355 ml fluid = 17.5 ml 100 % alcohol.
- Wine: 5 fluid ounces of 12 % wine = 148 ml fluid = 17.76 ml of 100 % alcohol.
- Distilled spirits: 1.5 fluid ounces of ~40 % liquor = 44 ml = 17.6 ml of 100 % alcohol.[7]

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.[1]

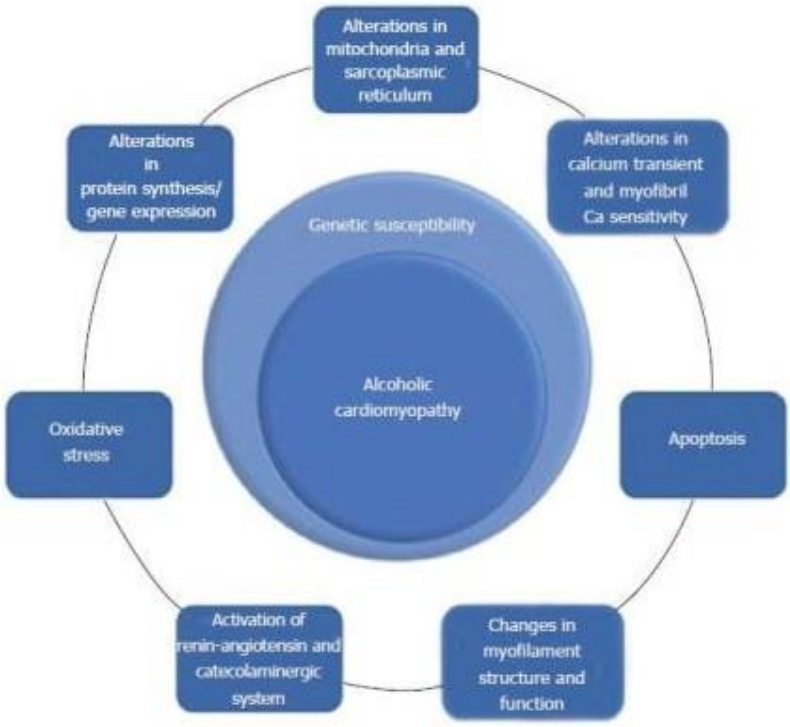


Figure 3: The Pathophysiology of Alcoholic Cardiomyopathy
Common pathophysiologic mechanisms for ACM are oxidative stress, apoptosis, alteration in calcium hemostasis, impaired mitochondrial stress, altered protein synthesis, and changes in myofilament structure and function. Adapted from Guzzo-Merello et al. 2014[viii]

In this context, we report a rare case of early-onset alcoholic cardiomyopathy with multiorgan involvement. A 35-year-old patient with a 20-year history of heavy alcohol use was diagnosed with icterus and admitted to the hospital for further testing.

## CASE PRESENTATION

A 35-year-old male patient with no known medical history was admitted to a university hospital for difficulty breathing, edema, and numbness in his limbs, thoracic and abdominal pain, jaundice, and physical weakness. The patient described beginning to consume alcohol at age fifteen and increasing his intake over the past five years. On initial physical examination,

he had severe jaundice of the skin, a heart rate of 90 beats per minute, a blood pressure of 105/60 mmHg, a respiratory rate of 25 breaths per minute, and an oxygen saturation of 96%.

Laboratory examinations showed Azotaemia 66 mg/dl, Creatinine 0.52 mg/dl, Total bilirubin 7.62 mg/dl, AST 178 U/l, GGT 2132 U/l, Amylase 12 U/l, Lipase 112 U/l, LDH 585 U/l, Potassium 2.7 mmol/l, PT 66%, INR 1.33, RBC 3.22*106, Hgb 12 g/dl, Hct 34.6%, Total Protein 5.5, N-proBNP/BNP 4370 pg/ml.

Abdominal ultrasound showed fatty liver with an enlarged size up to 24 cm, with minimal fluid around the liver, hilum, and ansa.

Angio-CT of the abdomen revealed minimal bilateral pleural fluid, with a thickness of 26 mm dexter and 15 mm sinister. Bilateral postero-basal fibrotic bands are applied. Liver with increased steatosis dimensions. Contracted cholecyst with a round lesion 22-18 mm, probable calculus. Bile ducts not distended. Homogeneous pancreas. Both kidneys are without calculus without stasis. Urinary bladder with regular contours. Intestinal loops are not visible. Generalized desiccation of mesenchymal and subcutaneous adipose tissue. Minimal fluid in the pelvis.

ECG: Sinus rhythm with a frequency of 100/min, low QRS voltage in the precordial and extremity leads, and nonspecific repolarization changes are observed.

The patient experienced acute alcohol withdrawal symptoms, including tremor, disorientation, and psychomotor agitation, which peaked 48 hours after being admitted to the hospital. The patient was sent to the Clinical Toxicology Service for alcohol withdrawal management. Reduced BP, reduced $O_2$sat, and elevated N-proBNP/BNP complicated the patient's overall health.

In echocardiography was observed diffuse hypokinesis of the left ventricle. Dilated left ventricle with a severe decrease in EF = 0.25. Restrictive pattern of left ventricular filling. Mitral regurgitation to advanced. Dilatation of the left and right atrium. Dilated right ventricle. Tricuspid regurgitation to moderate. PsAP 47mmHg. Pericardial fluid in a small circumferential amount.

Because alcohol withdrawal and cardiomyopathy are both life-threatening conditions that require immediate medical attention, the patient's treatment was challenging. Antiepileptic medications, vitamin therapy, and benzodiazepines form the cornerstone of withdrawal management treatment. To treat cardiomyopathy, beta-blockers and diuretics were added to the medication.

## DISCUSSION

Alcoholic cardiomyopathy is a variant of dilated cardiomyopathy, caused by excessive alcohol use, accompanied by left ventricular enlargement and systolic dysfunction.[ix]

Current research suggests that alcoholic cardiomyopathy is detected as a result of a deterioration in the patient's overall state and is a co-occurring symptom of alcohol withdrawal syndrome beyond the young age of the patient. The initial clinical therapy of patients identified with ACM should be based on aetiology,[x] and it should be suspected in all patients who exhibit hazardous or harmful alcohol

consumption, and a score of 15 or more (AUDIT Test) indicates the likelihood of alcohol dependence (moderate-severe alcohol use disorder).[xi]

Since the primary etiology of ACM is chronic alcohol intake, the cornerstone of treatment should be total abstinence from alcohol. Abstinence from alcohol improves LV function and relieves pulmonary artery and pulmonary capillary wedge pressure associated with ACM.[4]

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]

## CONCLUSION

This case study clearly shows the correlation between high levels of alcohol use and the development of dilated cardiomyopathy at a young age with multiorgan involvement. This case highlights the importance of conducting a thorough examination of heart function. Imaging, particularly echocardiography, and the exclusion of alternate structural and ischaemic aetiologies reveal alcohol as the primary cause of this form of cardiomyopathy. Early identification and thorough care, including alcohol withdrawal management and a multidisciplinary approach, are critical for improving the prognosis and quality of life for affected people.

**Conflicts of interest:** The authors declare that they have no conflicts of interest.

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