

Alcohol-Induced Cardiomyopathy Presenting with Left Ventricular Apical Thrombus

Farid Khan¹, Casey Manzanero², Amit Bansal¹, Sreekanth Kondareddy^{1*}

¹UHS Heart & Vascular Institute, Wilson Medical Center, Johnson City, NY, USA

²Upstate Clinical Campus, Binghamton, NY, USA

Email: drfaridkhan@hotmail.com, ManzaneC@upstate.edu, *Sreekanth.Kondareddy@nyuhs.org

How to cite this paper: Khan, F., Manzanero, C., Bansal, A. and Kondareddy, S. (2022) Alcohol-Induced Cardiomyopathy Presenting with Left Ventricular Apical Thrombus. *World Journal of Cardiovascular Diseases*, 12, 353-359.
<https://doi.org/10.4236/wjcd.2022.127034>

Received: April 29, 2022

Accepted: July 11, 2022

Published: July 14, 2022

Copyright © 2022 by author(s) and Scientific Research Publishing Inc. This work is licensed under the Creative Commons Attribution International License (CC BY 4.0).

<http://creativecommons.org/licenses/by/4.0/>



Open Access

Abstract

Background: Chronic excessive alcohol consumption has been strongly associated with alcohol-induced cardiomyopathy (AC) in patients with no evidence of coronary artery disease (CAD). AC may cause cardiovascular complications and significant impact on the quality of life. We discuss an interesting case of dilated cardiomyopathy, associated complication, diagnostic work-up and management. **Case Report:** A young male presented to our service with worsening dyspnea, orthopnea, and scrotal and lower extremity edema. On average, he consumed a pack of 12 beers every day and had a 30-pack-years smoking history. He was found to be in acute heart failure with evidence of pulmonary edema and cardiomegaly on chest imaging. He had biventricular dilatation and severely reduced left ventricular ejection fraction (LVEF) < 15% in addition to a thrombus in the LV apex. The cardiac catheterization was unremarkable for CAD. He was diuresed appropriately resulting in significant weight loss and resolution of symptoms. LV thrombus was treated with unfractionated heparin infusion that was transitioned to warfarin. He was maintained on guidelines-directed medical therapy for heart failure. Extensive counseling was provided regarding alcohol and tobacco cessation. On follow-up echocardiogram, his LVEF improved and there was no evidence of LV thrombus. We think, the readership will benefit from our experience of treating a case of AC, and the importance of clinical history. **Conclusion:** Chronic excessive alcohol use is detrimental to cardiac function leading to alcohol-induced cardiomyopathy. A careful approach to clinical history of alcohol consumption and prompt diagnostic workup negative for ischemic causes may confirm the diagnosis. Cardiac function improves with guidelines-directed medical therapy for heart failure and abstinence from alcohol.

Keywords

Alcohol-Induced Cardiomyopathy, Heart Failure, Echocardiography,

1. Introduction

Chronic excessive alcohol consumption has been strongly associated with alcohol-induced cardiomyopathy (AC). AC comes under a large group of dilated cardiomyopathies [1]. This diagnosis becomes important when patients present with heart failure symptoms and have negative work-up for coronary artery disease (CAD). Among heart failure patients, the prevalence of alcoholic cardiomyopathy varies from 4% to 40% with the age group of 40 to 60 appears to be the most affected [2]. Higher disease burden has been documented in men; however, women develop AC at a lower total dose of ethanol [2] [3].

Similar to other cardiomyopathies and heart failure, AC is also associated with complications and significant impact on the quality of life. We report a case of dilated cardiomyopathy with associated complications, necessary work-up and management.

2. Case Presentation

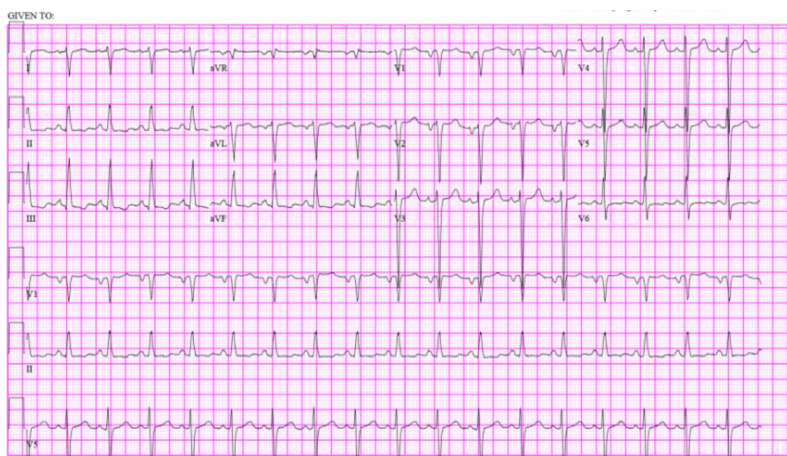
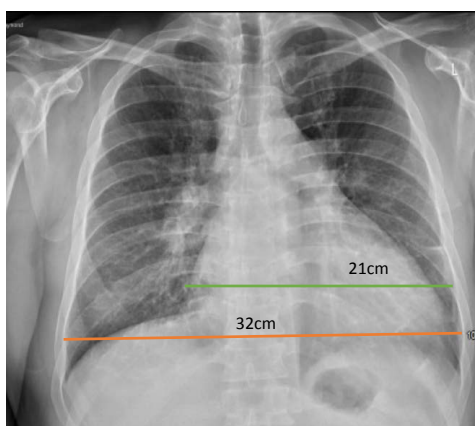
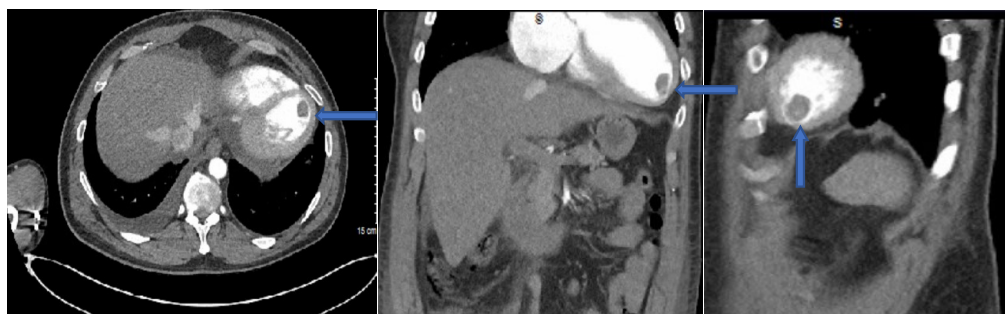
A 44-year-old male was admitted to our hospital with progressively worsening exertional dyspnea, orthopnea, scrotal swelling and lower extremity edema started 3 - 4 weeks prior to presentation. He reported no chest pain, palpitations, dizziness or syncopal episodes. His medical history was unremarkable, and he was not taking any medications. Family history was significant for CAD in his mother. Social history was significant for consuming at least a pack of 12 beers every day and 30-pack-years smoking.

On presentation, his vitals were significant for a pulse of 109/minute and blood pressure of 174/106 mmHg with systolic more than his baseline systolic range of 110 - 125 mmHg. He was tachypneic and had diffuse crackles over all lung fields. Heart sounds were normal with no murmur or gallop. There was no jugular venous distension; however, he had an edematous abdominal wall, scrotal swelling and 3+ pitting edema of the legs.

Pertinent blood work showed a troponin of 0.038 ng/mL and elevated BNP of 12,700 pg/mL with normal chemistries, electrolytes and hemogram. **Table 1** Electrocardiogram showed normal sinus rhythm, rate 110/minute and no ischemic changes (**Figure 1**). Chest x-ray revealed cardiomegaly and pulmonary edema (**Figure 2**). A chest computed tomography (CT) with intravenous (IV) contrast showed cardiomegaly with a filling defect in the left ventricle (LV) apical region suspicious for a thrombus (**Figure 3**). Transthoracic echocardiogram (TTE) revealed biatrial enlargement and dilated bilateral ventricles with severely reduced LV ejection fraction (EF) < 15%, moderate tricuspid regurgitation, and a filling defect (1.8 × 1.3 cm) at the LV apex consistent with a thrombus (**Figure 4**). A cardiac catheterization was unremarkable for CAD.

Table 1. Pertinent laboratory results.

Lab	Results	Ref. Range/unit
Troponin I	0.05	<0.03 ng/mL
proBNP	12,000	pg/mL
Hemoglobin	14.0	mg/dL

**Figure 1.** Electrocardiogram showed normal sinus rhythm, no significant ischemic changes.**Figure 2.** Chest x-ray posterior-anterior view revealed cardiomegaly and pulmonary edema. Cardiothoracic ratio (CTR) 0.65.**Figure 3.** A chest CT with IV contrast showed cardiomegaly with a filling defect (arrows) in the left ventricle apical region suspicious for a thrombus.

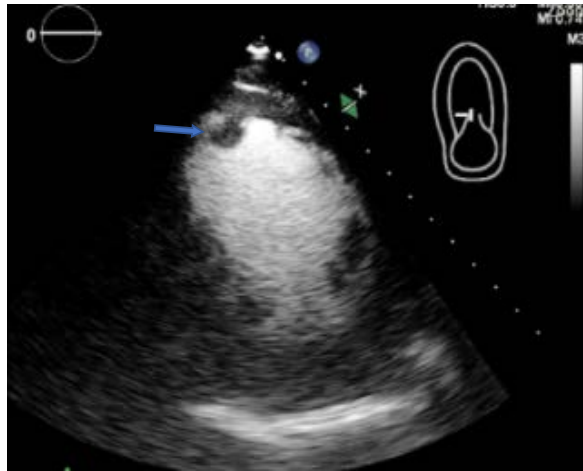


Figure 4. Transthoracic echocardiogram with contrast Definity revealed dilated LV with severely reduced LV ejection fraction (EF) < 15%, and a filling defect (1.8 × 1.3 cm) at the LV apex consistent with a thrombus.

Patient was initially treated with an IV diuretic including furosemide. For LV thrombus, unfractionated heparin (UFH) infusion was initiated. During the course of hospitalization, he had appropriate diuresis and lost significant weight. Subsequently, he was able to ambulate without dyspnea. He was started on guidelines-directed medical therapy (GDMT) for heart failure with reduced ejection fraction (HFrEF) including diuretic, beta-blocker and angiotensin receptor-neprilysin inhibitor. UFH was bridged with warfarin on which he was discharged. Detailed alcohol and tobacco cessation counseling was provided. On repeat TTE within 4 weeks of discharge, his EF improved and there was no evidence of LV thrombus, **Figure 5**.

3. Discussion

Excessive ethanol use has been established as one of the leading causes of secondary dilated cardiomyopathy contributing to about 10% of all cardiomyopathies [4] [5]. Subclinical changes in cardiac function may begin after at least 5 years of high alcohol consumption (defined as >80 g/day) [6]. Acetaldehyde, a toxic metabolite, from ethanol is thought to cause oxidative stress within myocardium and impairment of myocyte protein synthesis. Simultaneously, there is activation of renin-angiotensin system and arterial hypertension that may contribute to or worsen heart failure. Symptoms appear when there is ventricular dilation and decreased ejection function. Echocardiography is the key diagnostic test for chambers quantification.

Several prospective studies have reported J-shaped relationship between alcohol and heart failure. Increased risk was noted at higher levels of alcohol intake while reduced risk was associated with low to moderate levels of alcohol consumption [7] [8] [9] [10].

Diagnosis of Alcohol-induced cardiomyopathy requires all three of the following:

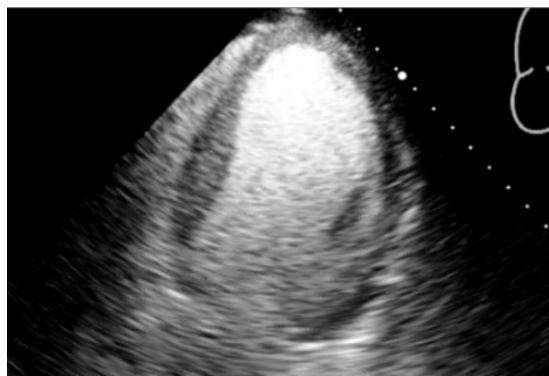


Figure 5. Transthoracic echocardiogram with contrast Definity revealed dilated LV with the absence of a filling defect noted in **Figure 4**.

1) Long-term heavy alcohol consumption (defined as >80 g/day over a period of at least five years).

2) LV dilation defined as LV end-diastolic volume or LV diastolic dimension > 2 standard deviations above normal (by echocardiography, an LVDD > 58.4 mm in men and >52.2 mm in women). [11] & LVEF below normal (<50% an accepted threshold for dilated cardiomyopathy) [12] [13].

3) Absence of hypertensive disease, valvular, and CAD.

Our patient fulfills the criteria for AC that includes long-term heavy alcohol consumption, reduced EF, dilated LV as evidenced on TTE and absence of CAD based on cardiac catheterization.

In our patient, AC was complicated by LV apical thrombus. This was observed both on CT angiogram and TTE, and was treated with anticoagulation initially with heparin and subsequently bridged with warfarin. LV thrombus was not observed on repeat TTE 4 weeks later suggesting resolution of the thrombus (**Figure 5**). Patients with AC and LV thrombus not only need GDMT for HFrEF but also require anticoagulation for 3 - 6 months [3]. Warfarin has been a well-studied agent for this purpose [14]. Intracardiac thrombus such as LV thrombus is a complication of blood stasis in a dilated ventricle. In similar fashion, alcohol-related dilated cardiomyopathy may predispose these patients to intracardiac thrombus necessitating anticoagulation.

4. Conclusion

Chronic excessive alcohol use is detrimental to cardiac function leading to alcohol-induced cardiomyopathy. A careful approach to clinical history of alcohol consumption and diagnostic workup negative for ischemic causes may confirm the diagnosis. A prompt diagnosis is recommended due to the risk of intracardiac thrombus. Cardiac function improves with GDMT for heart failure and abstinence from alcohol.

Informed Consent

The case was reviewed by the Institutional Review Board and informed consent

was obtained from the patient.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

- [1] Elliott, P., Andersson, B., Arbustini, E., *et al.* (2008) Classification of the Cardiomyopathies: A Position Statement from the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. *European Heart Journal*, **29**, 270-276. <https://doi.org/10.1093/eurheartj/ehm342>
- [2] Guzzo-Merello, G., Cobo-Marcos, M., Gallego-Delgado, M. and Garcia-Pavia, P. (2014) Alcoholic Cardiomyopathy. *World Journal of Cardiology*, **6**, 771-781. <https://doi.org/10.4330/wjc.v6.i8.771>
- [3] Ram, P., Lo, K.B., Shah, M., Patel, B., Rangaswami, J. and Figueredo, V.M. (2018) National Trends in Hospitalizations and Outcomes in Patients with Alcoholic Cardiomyopathy. *Clinical Cardiology*, **41**, 1423-1429. <https://doi.org/10.1002/clc.23067>
- [4] Piano, M.R. (2002) Alcoholic Cardiomyopathy: Incidence, Clinical Characteristics, and Pathophysiology. *Chest*, **121**, 1638-1650. <https://doi.org/10.1378/chest.121.5.1638>
- [5] Shaaban, A., Gangwani, M.K., Pendela, V.S., *et al.* (2022) Alcoholic Cardiomyopathy. StatPearls Publishing, Treasure Island, FL.
- [6] Fernández-Solà, J. (2020) The Effects of Ethanol on the Heart: Alcoholic Cardiomyopathy. *Nutrients*, **12**, 572. <https://doi.org/10.3390/nu12020572>
- [7] Walsh, C.R., Larson, M.G., Evans, J.C., *et al.* (2002) Alcohol Consumption and Risk for Congestive Heart Failure in the Framingham Heart Study. *Annals of Internal Medicine*, **136**, 181-191. <https://doi.org/10.7326/0003-4819-136-3-200202050-00005>
- [8] Xi, B., Veeranki, S.P., Zhao, M., Ma, C., Yan, Y. and Mi, J. (2017) Relationship of Alcohol Consumption to All-Cause, Cardiovascular, and Cancer-Related Mortality in U.S. Adults. *Cardiology*, **70**, 913-922. <https://doi.org/10.1016/j.jacc.2017.06.054>
- [9] Kajander, O.A., Kupari, M., Laippala, P., *et al.* (2001) Dose Dependent But Non-Linear Effects of Alcohol on the Left and Right Ventricle. *Heart*, **86**, 417-423. <https://doi.org/10.1136/heart.86.4.417>
- [10] Lazarević, A.M., Nakatani, S., Nesković, A.N., *et al.* (2000) Early Changes in Left Ventricular Function in Chronic Asymptomatic Alcoholics: Relation to the Duration of Heavy Drinking. *Journals of the American College of Cardiology*, **35**, 1599-1606. [https://doi.org/10.1016/S0735-1097\(00\)00565-9](https://doi.org/10.1016/S0735-1097(00)00565-9)
- [11] Lang, R.M., Badano, L.P., Mor-Avi, V., *et al.* (2015) Recommendations for Cardiac Chamber Quantification by Echocardiography in Adults: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *Journal of the American Society of Echocardiography*, **28**, 1-39.e14. <https://doi.org/10.1016/j.echo.2014.10.003>
- [12] Bozkurt, B., Colvin, M., Cook, J., *et al.* (2016) Current Diagnostic and Treatment Strategies for Specific Dilated Cardiomyopathies: A Scientific Statement from the American Heart Association. *Circulation*, **134**, e579-e646. <https://doi.org/10.1161/CIR.0000000000000474>
- [13] Pinto, Y.M., Elliott, P.M., Arbustini, E., *et al.* (2016) Proposal for a Revised Defini-

tion of Dilated Cardiomyopathy, Hypokinetic Non-Dilated Cardiomyopathy, and Its Implications for Clinical Practice: A Position Statement of the ESC Working Group on Myocardial and Pericardial Diseases. *European Heart Journal*, **37**, 1850-1858.
<https://doi.org/10.1093/eurheartj/ehv727>

- [14] Camaj, A., Fuster, V., Giustino, G., *et al.* (2022) Left Ventricular Thrombus Following Acute Myocardial Infarction: JACC State-of-the-Art Review. *Journals of the American College of Cardiology*, **79**, 1010-1022.
<https://doi.org/10.1016/j.jacc.2022.01.011>