Published in final edited form as:

Can J Addict. 2017 December ; 8(2): 23–25.

DILATED CARDIOMYOPATHY IN A YOUNG MAN WITH POLYSUBSTANCE USE

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Abstract

Chronic excessive alcohol consumption is the leading cause of an acquired dilated cardiomyopathy. Here we report a case of a 25-year-old male with a severe alcohol use disorder, who presents with a viral prodrome and is found to be in cardiogenic shock secondary to a non-ischemic dilated cardiomyopathy. The case highlights the pathophysiology of chronic ethanol exposure on cardiac function and the importance of early screening for risky drinking.

Keywords

alcohol use disorder; alcoholic cardiomyopathy; dilated cardiomyopathy; heart failure

INTRODUCTION

Excessive alcohol consumption has been identified as a leading risk factor for morbidity and mortality among individuals between the ages of 15 and 49 years¹ and is globally ranked third among the modifiable risk factors for disease (behind hypertension and cigarette smoking).² Furthermore, a myriad of medical comorbidities are partially or wholly attributable to excessive alcohol use. Alcoholic cardiomyopathy is one such condition and is associated with long-term heavy alcohol use. Although the literature commonly cites a threshold required intake of greater than 80 g of alcohol per day for a period of at least 5 years,³ a recent review concluded the exact time requirement is uncertain because the consumption of large amounts of alcohol over a short duration can cause an acute toxic myocardial dystrophy. In addition, irregular heavy drinking is thought to have a role in the etiology as well.⁴ We report a case of a young male patient with a significant alcohol use disorder who presents with a new diagnosis of a dilated cardiomyopathy (DCM). Our case illustrates the importance of taking a thorough substance use history, and early screening for risky drinking among young adults.

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The authors report no conflicts of interest.

CASE REPORT

A 25-year-old white male presented to an emergency department in British Columbia, Canada, with a 2-day history of worsening shortness of breath and bilateral leg edema. These symptoms occurred on a background of severe nausea, vomiting, diarrhea, and rhinorrhea for the preceding 2 weeks. The patient denied subjective fevers and reported similar symptoms had recently been experienced by several friends and family members.

The patient's medical history was significant for a gunshot wound to the right leg, and a right hand fracture 6 years prior. He took no medications and had an anaphylactic allergy to penicillin. His substance use history was significant for heavy alcohol use. He described a 10-year history of binge drinking, which escalated to daily use over the preceding 3 years, consuming one to two 40 oz bottles of vodka daily and up to three 40 oz bottles on heavy drinking days. Despite this heavy intake, he reported that he found it difficult to drink fast enough to produce a state of intoxication. Beyond alcohol use the patient also reported intermittent intranasal crystal methamphetamine and cocaine use, approximately 1 to 2 times per month, typically in social situations while intoxicated with alcohol. He had quit smoking marijuana 1-year prior and did not smoke tobacco. He denied both intravenous drug use and use of other illicit drugs. The patient's family history was significant for an alcohol use disorder in both his mother and maternal grandmother. There was no family history of cardiomyopathy.

Physical examination revealed a temperature of 36.6°C, heart rate of 125 beats/min (bpm), blood pressure of 122/91 mmHg, oxygen saturation of 97% on room air, and respiratory rate of 20 to 24 breaths/min. He appeared fatigued. Cardiovascular examination revealed elevation of the jugular venous pulse at 4 cm, and the presence of hepatojugular reflux. Heart sounds were normal with no murmurs or extra sounds. Respiratory examination revealed decreased air entry at the bases, bilaterally. His abdomen was soft with slight tenderness to palpation in the right upper quadrant. There was no hepatosplenomegaly or ascites. He had mild pitting edema of his bilaterally lower extremities to the mid-calf. Complete blood count normal aside from a slightly monocytosis. Creatinine was 130 µmol/L and lactate 3.1 mmol/L. He had a significant transaminitis (aspartate aminotransferase 3607 U/L and alanine aminotransferase 2854 U/L), mildly low albumin (31 g/L) and increased international normalized ratio (2.4). Serial troponins were elevated but stable at 23 µg/L. Both creatine kinase and β-natriuretic peptide were significantly increased at 1082 U/L and 7407 nl/L, respectively. A urine drug screen was positive for cocaine and amphetamines. Urinalysis showed a urine protein of 0.15 g/L with no hematuria. Chest radiograph showed an enlarged globular heart but was otherwise normal. Electrocardiogram was significant for sinus tachycardia at 112 bpm, with inverted T waves in V4–V6. An echocardiogram revealed a severely dilated left ventricle with severe systolic dysfunction and an ejection fraction of 10% to 15%. There was severe generalized cardiac hypokinesia, mild left ventricular hypertrophy, moderate mitral regurgitation, and mild tricuspid regurgitation. Additionally, the right ventricle was dilated and showed depressed systolic function. A cardiac magnetic resonance imaging revealed a significantly dilated left ventricle with severely impaired systolic function (ejection fraction 13.8%), a normal-sized right ventricle with severely impaired function, and no late gadolinium enhancement to

suggest infiltration or scarring. Cardiac catheterization revealed elevated pulmonary artery and right heart pressures, but no evidence of coronary atherosclerotic disease. Cardiac output was low at 2.4 L/min. Viral and immune serologies and blood cultures were all normal.

The patient was treated in the cardiac intensive care unit for cardiogenic shock. The cardiac transplant team evaluated the patient and recommended a 3-month period of abstinence from alcohol and illicit drugs before consideration for orthotopic heart transplant. The patient's clinical status improved and he was subsequently discharged from hospital with outpatient cardiology follow-up. Unfortunately, despite attempts to contact the patient, he was lost to follow-up.

Consent

Written and verbal consent for the preparation and publication of this articles was obtained from the patient.

DISCUSSION

This report describes a previously healthy young male who presents with cardiogenic shock secondary to a severely DCM. Our case demonstrates a potential severe health outcome of heavy alcohol intake, irrespective of age, and highlights the importance of early screening for risky drinking.

DCM is characterized by left ventricular dilatation and systolic dysfunction, with or without similar changes in the right ventricle.⁵ The differential diagnosis for DCM is broad and includes ischemia, genetic causes, viral infection, hypertension, autoimmune or connective tissue disorders, infiltrative diseases, or toxins.⁶ Chronic, excess alcohol consumption is a leading cause of acquired DCM, and accounts for 21% to 32% of cases.^{7,8} A recent review of DCM by Weintraub et al.⁹ highlights the fact genetic causes are also important and account for transmission in 20% to 35% of cases.

Alcoholic cardiomyopathy is thought to occur as a result of several pathogenetic mechanisms. Chronic exposure to ethanol (and its metabolites) can result in increased free radical production and myocardial oxidative stress.¹⁰ This process may ultimately result in increased rates of myocardial apoptosis, reduced myocardial protein synthesis, and accelerated protein catabolism,^{11,12} which may consequently lead to a series of stages of cardiac remodeling including inotropic incompetence, left ventricular hypertrophy, and dilatation.¹³ Impaired fatty acid metabolism and mitochondrial ineffectiveness are also proposed mechanisms for myocardial dysfunction associated with alcoholic cardiomyopathy¹² as is overactivation of the renin–angiotensin system resulting in ethanol-induced hypertension.^{14,15} Genetics is also thought to play a role with 1 study demonstrating an increased risk for alcoholic cardiomyopathy among individuals with a particular polymorphism of the angiotensin-converting enzyme gene.¹³

Clinically, despite the development of left ventricular dysfunction, individuals may remain asymptomatic for a prolonged period or symptoms may have an insidious onset.¹⁶ For example, it has been postulated that consuming 90 g of pure alcohol per day for 5 years can

cause clinically asymptomatic DCM, and another 15 years of continued drinking to manifest symptoms.⁴ Furthermore, no histochemical or immunological markers exist to aid in the diagnosis of alcoholic cardiomyopathy. As such, regular screening for risky drinking and its potential adverse consequences is crucial, particularly as, in the context of alcoholic cardiomyopathy, early detection and a reduction or cessation of alcohol intake has been associated with an improvement in cardiac function. A review of the literature found a variable (<1%–40%) rate of diagnosed alcoholic cardiomyopathy in patients with alcohol use disorder and concluded that especially in treatment populations, alcohol use disorder is associated with a high rate of cardiovascular disease, particularly an increased heart size and mass.⁴

In this case, the patient was diagnosed with alcoholic cardiomyopathy. Although the history of the preceding viral symptoms raises the possibility of an acute fulminant viral myocarditis, his alcoholic intake is likely the major contributor, with a calculated dose of ethanol in between 373 and 747 g/day in the 3 years prior. His occasional cocaine and crystal methamphetamine use may also have contributed to his clinical presentation given the known association between stimulant use and the development of DCM.¹⁷ Methamphetamines and cocaine are both inotropic and chronotropic and are thought to induce cardiac ischemia in myriad ways, including increased oxygen demand, vasospasm, as well as prothrombotic effects.^{9,16,17} Direct toxicity and production of reactive oxygen species may also play a role.^{16,17} Among young adult patients diagnosed with cardiomyopathy, there is a high prevalence of methamphetamine abuse.⁹

In summary, this case highlights the importance of regular screening for risky drinking patterns, particularly among young adults who are disproportionately affected by such behavior and, given the absence of comorbidities, tend to have fewer interactions with healthcare providers. Future research should focus on the development of formal guidelines to help identify patients who engage in heavy drinking and would benefit from routine screening for early onset, asymptomatic left ventricular dysfunction. Such knowledge could assist healthcare providers in motivating patients early to reduce or cease their alcohol consumption, resulting in an improvement in cardiac function and overall health.

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