

Alcoholic Cardiomyopathy In A 39 Year Old Female: A Case Report

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Citation

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Abstract

Background: Alcoholic cardiomyopathy is a dilated cardiomyopathy, caused by long standing chronic ingestion of alcohol. It is very similar to idiopathic dilated cardiomyopathy (DCM). However, total cessation of alcohol is strongly associated with improvement of symptoms and even reversal of the DCM. Method: Case Report Result: A 39 year old school teacher, presented on account of progressive dyspnoea associated with orthopnea, palpitations and bilateral leg swelling. There was no previous remarkable illness or hospital admission. She had a history of daily ingestion of alcohol based fertility potions for 8 years. On examination she was in respiratory distress, had bilateral basal crepitations, an irregular pulse, elevated jugular venous pulse, a displaced non heaving apex with left parasternal heave, and a non radiating apical pansystolic murmur. She also had a tender hepatomegaly, and bilateral pitting pedal oedema. A chest radiograph showed upperlobe diversion, bilateral hilar opacities and a multichamber cardiomegaly. A 12 lead surface electrocardiogram (ECG) showed atrial supraventricular and ventricular ectopics, and echocardiography showed, four chamber dilatation with poor systolic function and absent a waves. Conclusion: Congestive cardiac failure (CCF) secondary to alcoholic cardiomyopathy, precipitated by arrhythmias.

INTRODUCTION

Alcoholic cardiomyopathy is a common cause of dilated cardiomyopathy and the most common secondary cardiomyopathy. Phenotypically and clinically, alcoholic cardiomyopathy closely resembles idiopathic DCM. It is linked to ongoing excessive alcohol consumption and appears to be both dose related and responsive to cessation of alcohol exposure. Alcohol exposure also increases risks for comorbidities that can contribute to cardiovascular disease such as hypertension, arrhythmias and sudden death¹.

We report a case of alcoholic cardiomyopathy in a 39 year old female school teacher.

CASE REPORT

A 39 year old female school teacher, presented with a 2 month history of breathlessness, palpitations and leg swelling. Breathlessness was initially on moderate exertion, then progressed to breathlessness at rest, associated with orthopnoea and some episodes of paroxysmal nocturnal dyspnoea. There was cough, productive of a small amount of sputum but no hemoptysis. There was no chest pain or fever. Palpitations were abrupt in onset, and also stopped abruptly. There was no syncope or dizziness, no heat intolerance or tremors. Leg swelling was bilateral and progressive. There

was no facial or abdominal swelling. She admitted to some reduction in urine volume. There was no hematuria, dysuria or frothiness of urine. There was also no significant weight loss, joint pains, rashes or sore throat. The patient was not a known hypertensive or diabetic. She had a history of childhood asthma. Her menstrual cycle was regular.

She had been married for 8yrs without children and admitted to ingestion of alcohol based fertility potions for 8yrs, approximately 6units (48gms) of alcohol per day. She had never smoked.

Physical examination revealed an acutely ill looking young lady in respiratory distress. She was afebrile (36.8C), anicteric, mildly pale, with bilateral pedal oedema. Cardiovascular system findings were; irregular pulse of 120 b/m, Blood pressure 110/70mmHg, elevated JVP, a non heaving apex beat in the 6th left intercostal space, lateral to the mid clavicular line, with a left parasternal heave. 1st, 2nd and 3rd heart sounds with a gallop, and a non radiating apical pansystolic murmur were present.

Bibasal crepitations were present in the chest. Abdominal examination revealed a tender hepatomegaly, and a non tender suprapubic mass of about 18wks size.

CXR showed upper lobe diversion, pulmonary congestion and a globular cardiomegaly with mitralization of the left heart border. (Fig 1)

ECG showed a heart rate of 110 b/m, supraventricular and polymorphic ventricular ectopics, normal QRS axis and broad bifid p waves suggesting left atrial enlargement.

Echocardiography showed; multichamber dilatation, global hypokinesia, severe systolic dysfunction (EF 27.2%, FS 12.9%), absent A waves on Pulse wave doppler, transmitral flow regurgitation (fig 2), with normal mitral leaflets.

International normalized ratio (INR) was 1.3, urinalysis was normal, abdomino-pelvic ultrasonography(USS) revealed essentially normal internal organs except for hepatic congestion and uterine leiomyomata. She was seronegative for HIV 1 and 11, HBsAg and HCV Ab.

A diagnosis of congestive cardiac failure secondary to alcoholic cardiomyopathy, precipitated by arrhythmias was made.

The patient was admitted, nursed in cardiac position, and given intermittent oxygen, had a start dose of 60 mg of iv frusemide and maintained on iv 40mg bd, lisinopril 5mg dly, aldactone 12.5mg bd, digoxin 0.25mg dly and thiamine 10mg bd . She was counseled on the need for total abstinence from all forms of alcohol, and was placed on a low salt diet.

The patient did very well on the above regimen, and echocardiography done prior to discharge showed improved systolic function (EF 44%, FS 22.4%). She was discharged after 3 weeks in hospital, and continued follow up on an out patient basis. She remained stable and out of failure.

ECG done 3 months post discharge showed sinus rhythm and a left atrial enlargement (fig 3).

Figure 1

Fig 1: CXR showing globular cardiomegaly, pulmonary congestion, and upper lobe diversion.



Figure 2

Fig 2: flow across the mitral valve in the apical four chamber view, showing mitral regurgitation. LA left atrium, LV left ventricle, RJ regurgitant jet.

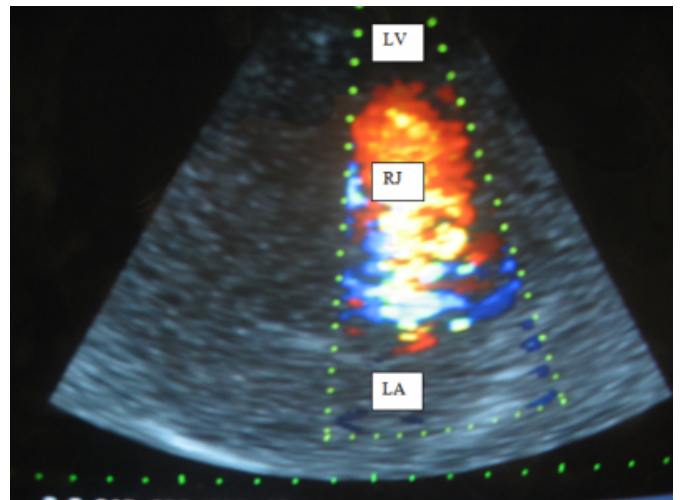
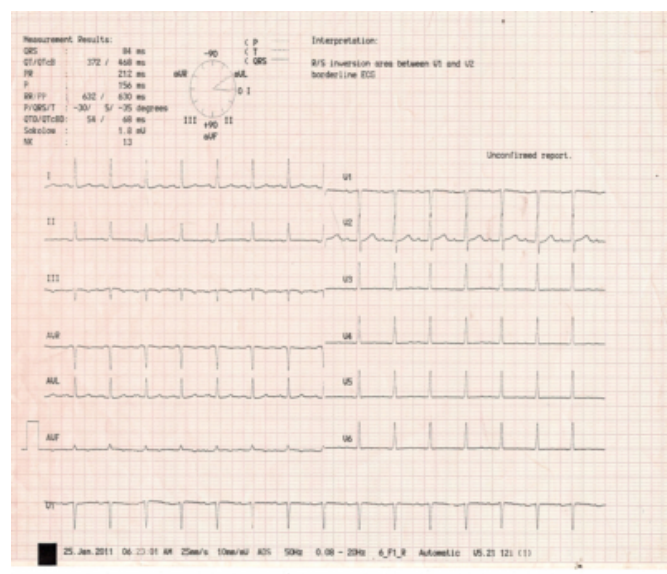


Figure 3

Fig 3: ECG done 3 months after discharge showing sinus rhythm and broad bifid P waves.



DISCUSSION

Alcoholic cardiomyopathy is a common cause of dilated cardiomyopathy and the most common secondary cardiomyopathy¹. An extensive review article by Silwa et al found alcohol to be a contributory factor in 45% of patients with DCM in Africa^{2,3}.

The mechanism for the cardiac damage produced by alcohol remains unclear. Several theories have arisen over many years. Original theories regarding the mechanism focused on nutritional deficiencies (eg thiamine deficiency), secondary exposures (eg tobacco, cobalt, arsenic) and other comorbidities (eg hypertension). Although these mechanisms continue to play a role in selected patients, most evidence in the literature indicates that the effects of alcohol on the myocardium are independent of these factors and that the effect is a direct toxic result of ethanol or its metabolites⁴. Experimental studies show that alcohol and its metabolite acetaldehyde can disrupt cardiac calcium cycling, mitochondrial respiration, myocardial synthesis of proteins and lipid signal transduction, and myocardial redox state⁵⁻⁷. There is also evidence to support a direct toxic effect of alcohol on both cardiac and skeletal myocytes which may in turn increase the rate of cellular apoptosis⁸. Genetic factors play a role as evidenced by studies showing that individuals with the angiotensin – converting enzyme DD genotype have an increased risk of developing alcoholic cardiomyopathy⁹. Several authors have reported that though alcoholic cardiomyopathy is a disease that affects males more often,

typically in the 4th to 6th decade, females may be more sensitive to cardiotoxic effects of alcohol¹⁰⁻¹². Most men who develop alcohol cardiomyopathy have consumed 80g of ethanol per day for at least 5 years. Women develop cardiomyopathy following the consumption of a smaller amount of ethanol per day and per lifetime¹³. The patient in this report is a female, and consumed less than 80g of alcohol daily. A careful alcohol history is warranted in all subjects presenting with cardiomyopathy¹, with attention to daily maximal, lifetime, and duration of intake⁴. In this environment, a careful alcohol history includes a detailed history of ingestion of alcohol based herbs/concoctions, which is very prevalent, and effort should be made to quantify the alcohol content.

Mild reductions in cardiac performance manifest in chronic alcoholics before symptoms appear. In the symptomatic phase, abnormalities in both systolic and diastolic function occur. The onset of symptoms ranges from progressive exercise limitation to acute fulminant heart failure, in the setting of biventricular dilation and hypokinesia. Not infrequently, paroxysmal atrial fibrillation is the initial finding. Palpitations accompany SVTs, especially atrial fibrillation¹. Atrial fibrillation in the reported case was paroxysmal and was manifested in the echocardiography done at presentation. When the electrocardiogram was done, there was no atrial fibrillation, but supraventricular and ventricular ectopics were present. Syncope can result from ventricular and possibly supraventricular arrhythmias. It is unusual for cardiomyopathy and cirrhosis to coexist although cirrhotics often have asymptomatic ventricular dysfunction¹.

With abstinence from alcohol, left ventricular systolic and diastolic function often improves. The earlier in the course of ethanol consumption that abstinence is initiated, the more pronounced the benefit. Even subjects with markedly symptomatic ethanol induced dilated cardiomyopathy, may manifest a substantial improvement in left ventricular systolic function and symptoms of heart failure with complete abstinence or a dramatic reduction in ethanol consumption. Although most of this improvement occurs in the first 6 months of abstinence, it often continues for as long as 2 years of observation¹³.

This case report really demonstrates the remarkable improvement in cardiac function that can occur with cessation of alcohol consumption as evidenced by improvement in systolic function prior to discharge, return

of the patient to sinus rhythm, and sustained absence of symptoms as she continued to abstain from alcohol.

CONCLUSION

The recognition of alcoholic cardiomyopathy is important because of the remarkable cardiac improvement associated with timely abstinence from alcohol, leading to reduction in morbidity and mortality. It is also very important to realize that in this environment many herbal/native remedies are alcohol based, and the alcohol content could be substantial and sometimes the ultimate source of alcohol significant enough to cause dilated cardiomyopathy as demonstrated in this case report.

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