

Reversible Alcoholic Cardiomyopathy: Letter to Editor*Düzelebilen Alkolik Kardiyomiyopati: Editöre Mektup***Dursun Çayan Akkoyun, Aydın Akyüz, Seref Alpsoy, Kubilay Erselcan, Hasan Değirmenci, Niyazi Güler**

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Dear Editor;

Severe alcohol consumption is one of the important causes of dilated cardiomyopathy (DCMP). Although beneficial effects of moderate alcohol intake on cardiovascular system has been shown, long term and severe alcohol intake may cause clinical cases from asymptomatic left ventricle diastolic dysfunction to DCMP due to alcohol. Pathophysiology of DCMP dependent on alcohol is not known certainly but many mechanisms are condemned. Heavy alcohol consumption has been associated with ventricular myocyte loss in some animal models and heavy drinking (by abnormalities in calcium balance) can lead to myocyte dysfunction (1,2).

If the patient who developed alcoholic DCMP goes on alcohol intake or doesn't decrease the amount of intake, the disease progresses gradually and may occur severe complications such as death. But if patients who developed alcoholic DCMP leave alcohol and take proper heart failure treatment, certain clinical improvement may occur.

In this report we'll present a rare case who has come with acute pulmonary edema due to alcoholic DCMP and after leaving alcohol has occurred certain improvement on left ventricle systolic functions by heart failure treatment.

A 53 year-old male patient who had a story of exercise dyspnea for last 4 months, came to the emergency room with resting dyspnea became stronger for 2 days. In physical examination, general condition was poor, he was cold, pale, cyanosed, dyspneic and orthopneic. In auscultation, there were widespread rales extending to apex in bilateral lungs. The patient with blood pressure 80/40

mmHg, pulse 110 bpm, arterial blood gas sO_2 %60, intubated and admitted to the coronary intensive care unit because of respiratory failure due to acute pulmonary edema. There were sinus rhythm and incomplete left bundle branch block (LBBB) on ECG. There were global widespread severe hypokinesia of the left ventricle and left ventricular systolic dysfunction due to dilatation on echocardiography. Left ventricular end-diastolic diameter (LVDD) was 61mm, left ventricular systolic diameter (LVSD) was 55mm, EF was %21, mitral regurgitation was moderate (Figure 1-2).

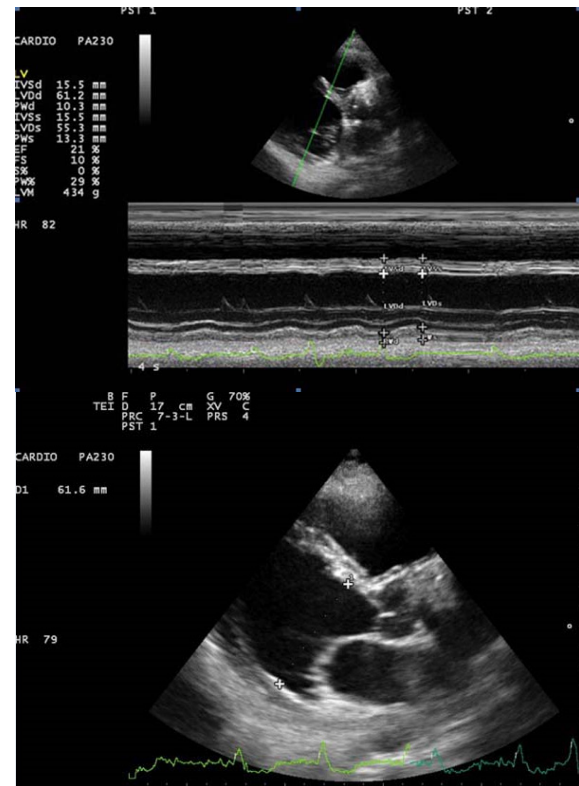


Figure 1. Parasternal long axis M-mode echocardiographic measurement before treatment. **Figure 2.** Parasternal long axis echocardiographic view before treatment.

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CBC and biochemical parameters was normal. After one-day mechanic ventilation patient was extubated as his own spontaneous respiration was enough and after five-day treatment coronary angiography was performed and normal coronary arteriogram was detected. There was alcohol consumption as 50 gr per day in patient medical history. There wasn't any known cardiovascular disease. The patient diagnosed as alcoholic DCMP because of he had non-ischemic cardiomyopathy (CMP), chronic alcohol intake, normal thyroid function, no story of myocarditis and drug usage. A treatment of ASA 100 mg 1x1, carvedilol 6,25 mg 2x1, spironolactone-hydrochlorothiazide 25 mg 1x1, trandolapril 2 mg 1x1, furosemide 40 mg 1x1 (once in two days) was ordered to the patient and suggested him to leave the alcohol and he discharged from the hospital. In 2 year-follow, an improvement was determined on left ventricle diameters and systolic function of the patient who accomodate himself attentively to drugs and suggestions. On the last control LVDD was 51mm, LVSD was 37mm, EF was %54 and mitral regurgitation was mild (Figure 3-4).

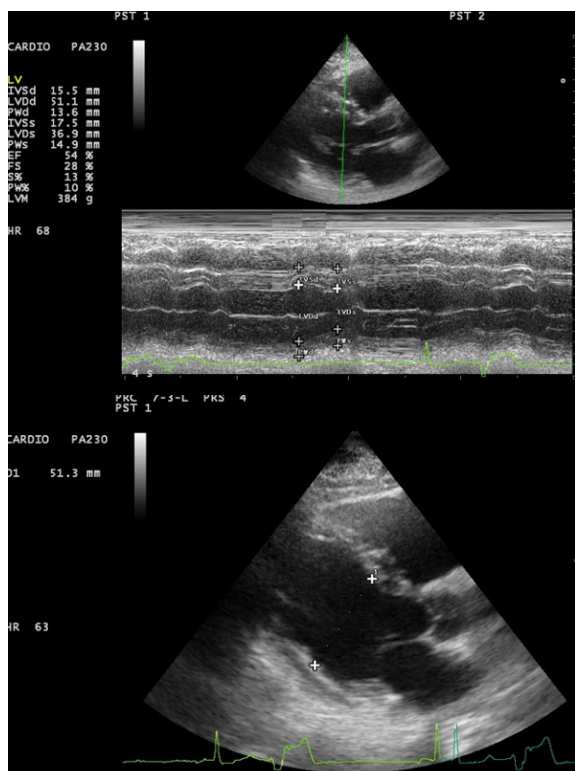


Figure 3. Parasternal long axis M-mode echocardiographic measurement after alcohol left and heart failure treatment.

Figure 4. Parasternal long axis echocardiographic view after alcohol left and heart failure treatment.

Although moderate alcohol consumption has protective effects on cardiovascular events (3), its potentially harmful effects appear with increasing amount and time of alcohol consumption. Daily consumption of alcohol less than 25 grams is described as mild consumption and as severe consumption when it's greater than 90 grams (4). Alterations in cardiac structure and functions may be seen in those who've consumed alcohol for more than 5 years (5).

Alcoholic cardiomyopathy is diagnosed in DCMP patients with chronic alcohol consumption in the absence of ischemia, cardiotoxic drug intake and myocarditis. Ischemic heart disease which may cause DCMP, myocarditis history and intake of cardiotoxic drugs were not found in our case. Alcohol is considered as the reason of dilated cardiomyopathy due to the history of daily alcohol consumption of 50 grams for 30 years in our case.

It is reported that approximately 20-50% of all dilated CMP's are related to alcohol (6,7) the reason of these different rates may be related to the prevalence of alcoholism in a population. Prognosis of patients with alcohol related cardiomyopathy is poor in case they continue to consume alcohol, 3-year mortality rates are reported nearly 42% (8). There are studies indicating that the effects of alcohol on myocarditis may be reversible. Additionally, significant improvements are determined clinically and echocardiografically in systolic heart functions at least 6 months after the alcohol is given up (9, 10, 11). Although this situation is presumed to be related to giving up alcohol before the development of fibrosis, it is still in question.

Our patient gave up alcohol after he had been discharged and fully recovered by a regular treatment. Although alcohol related DCMP has high morbidity and mortality, our case is important when the reported cases are also considered, since it shows that the situation may be reversed if the alcohol is given up and a good heart failure treatment is carried out.



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