Case report

A 42-year-old patient with alcoholic cardiomyopathy

Robert Irzmański[1], Ewa Serwa-Stępień[1], Marcin Barylski[1], Maciej Banach[2], Jacek Rysz[3], Jan Kowalski[1], Lucjan Pawlicki[1]

[1]Department of Internal Medicine and Cardiological Rehabilitation, University Hospital No. 5 in Lodz, Medical University of Lodz, Poland
[2]Department of Cardiac Surgery, University Hospital No. 3 in Lodz, Medical University of Lodz, Poland
[3]2nd Department of Family Medicine, University Hospital No 2 in Lodz, Medical University of Lodz, Poland

Submitted: 18 November 2005
Accepted: 15 December 2005

Arch Med Sci 2005; 1, 4: 249-253

Corresponding author:
Robert Irzmański, MD, PhD
Department of Internal Medicine and Cardiological Rehabilitation
University Hospital No. 5 in Lodz
Medical University of Lodz
Hallera Sq. 1
90-647 Lodz, Poland
E-mail: robik03@mp.pl

Abstract

Introduction: Alcoholic cardiomyopathy represents about 3.8% of all cardiomyopathy cases and it is a result of long-term (>5 years) alcohol consumption (>90 g of alcohol per day). It occurs twice as frequently in men as in women.
Case report: We present the case of a 42-year-old patient, not treated for chronic diseases so far, with positive incriminating familiar anamnesis for ischemic heart disease, who was hospitalized in our Department because of progressive reduction of effort tolerance for 2 weeks with squeezing in the chest and dyspnea ("asthma cardiale" type). Basing on the clinical course, anamnesis from family members and additional tests performed, alcoholic cardiomyopathy was diagnosed.
Conclusions: Alcoholism is one of the main causes of myocardial damage. Treatment of patients with alcoholic cardiomyopathy should include alcohol abstinence and recommended heart failure pharmacotherapy.

Key words: alcoholic cardiomyopathy, echocardiography, heart failure.

Case report

A 42-year-old patient, not treated for chronic diseases so far, with positive incriminating familiar anamnesis for ischaemic heart disease (his mother died of myocardial infarction at the age of 52, his brother and sister are treated for ischaemic heart disease) was admitted to the Clinic because of retrosternal pain, squeezing in the chest, dyspnea ("asthma cardiale" type) and considerable reduction of effort tolerance. These affections lasted for 2 weeks before admission.

In the ECG record we stated: sinistogram, regular sinus rhythm with a rate of 90 beats/min, poor R wave progression with 2 mm J-point elevation in V1-V3, 1 mm ST segment depression in V5-V6, biphasic negative-positive T-wave in V6 and negative T-wave in I, aVL (Figures 1, 2).

In biochemical tests we observed: raised levels of CK – 450 IU/l, CK-MB – 22 IU/l, AST – 107 IU/l, ALT – 133 IU/l.

In the RTG examination, stagnation in the small circulation and enlarged heart figure were described. In the Echo test (TTE): enlargement of all heart chambers: LA (52), LV (61/70), RV (34) and generalized hypokinesis with EF – 28%. Insufficient mitral valve (++/+++), without organic changes on the flaps and trace of tricuspid regurgitation. The other valves had a correct

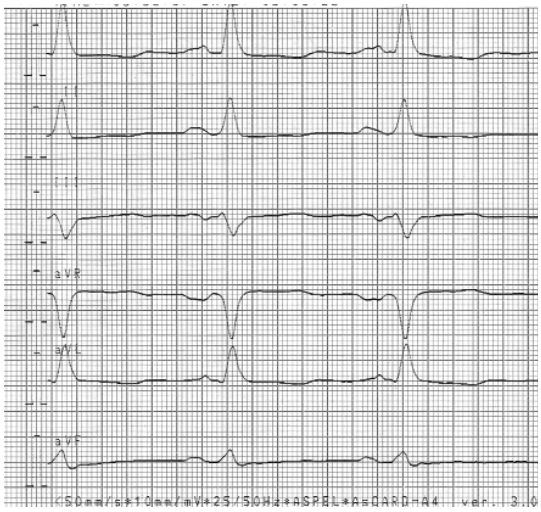

Figure 1. Sinistogram, regular sinus rhythm with heart rate [HR]=90 bpm, 2 mm negative T-wave in I, aVL

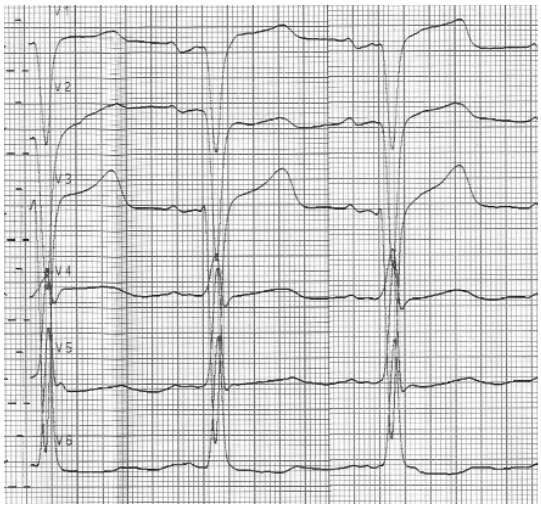

Figure 2. 2 mm J-point elevation in V1-V3, 1 mm ST segment depression in V5-V6, biphasic negative-positive T-wave in V6

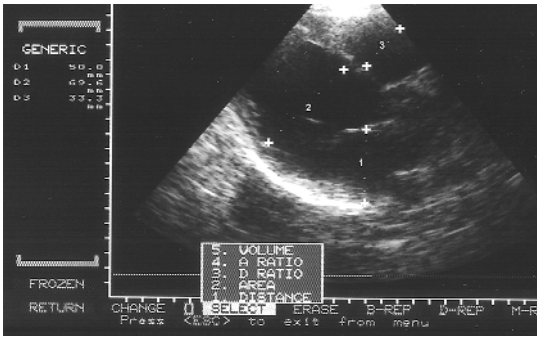

Figure 3. Enlargement of the left atrium and left ventricle. The Echo test made at the beginning of the treatment (left parasternal long axis view)

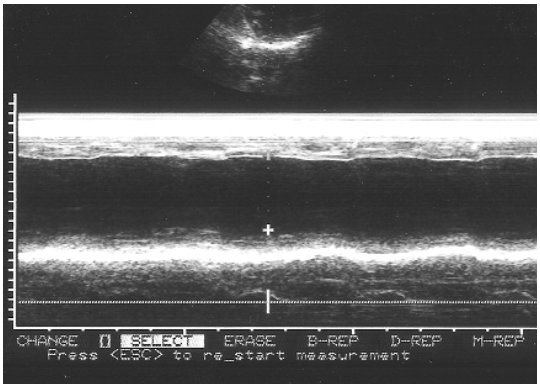

Figure 4. Large, hypokinetic left ventricle (M-mode)

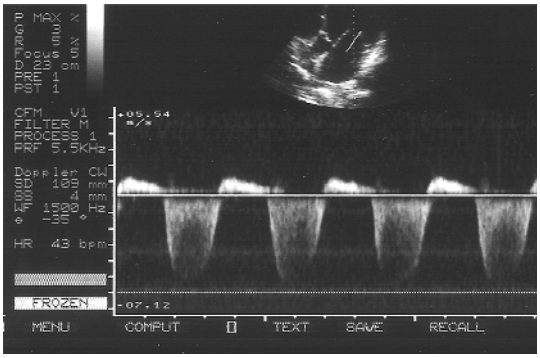

Figure 5. Mitral valve insufficiency (++/+++)

structure. No thrombi were found. Pericardium had no changes (Figures 3, 4, 5).

After precise anamnesis from the family, it turned out that the patient is dependent on alcohol and during the previous year he consumed 500 mg of alcohol per day.

After 7 days of heart failure treatment (Metoprolol, Metildigoxin, Trandolapril, Acidum acetylsalicylicum, Trimetazidine, Isosorbide-5-mononitrate, Timonacic, Furosemidum and then Chlortalidon) there was a reduction of the left ventricular volume (55/62), right ventricular volume (30) and improvement of the left ventricular contractibility, especially of the lateral wall and apex, with EF – 34% (Figure 6).

It was connected with improvement in the patient's condition. After stabilization of the general condition, the patient was discharged from hospital with total prohibition of consuming alcohol.

Discussion

It is estimated that 10% of population in Poland indulge in alcohol which makes 4 million people. The number of people dependent on alcohol is estimated at 1 million (2.5% of population).

In the United States, the frequency of alcoholic cardiomyopathy occurrence is 23 to 40% and it occurs twice as frequently in men as in women [1]. Men also have greater mortality, which is greater in black-race

people, in general. Independently of the race, alcohol consumption for 5 years (>90 g of alcohol a day, 7-8 drinks) causes alcoholic cardiomyopathy, asymptomatic initially, which turns into manifest heart failure when the habit is continued [2].

Alcoholic cardiomyopathy represents about 3.8% of all cardiomyopathy cases, which may seem slightly important. But if we take into consideration the frequency of dilated cardiomyopathy occurrence, it appears that alcohol is one of the main causes.

What is more, if we compare the frequency of cardiomyopathy occurrence in general population and in alcoholics, it would turn out that cardiomyopathy occurs much more frequently in the latter group [3].

Alcoholism is one of the crucial health problems in the contemporary world. Longstanding dependence on alcohol causes many changes which influence worsening of function of the organism and social relations between alcoholics and other people. Alcohol's effects on the organism are presented in Table I.

Under the influence of its toxic activity, it causes damage of the central nervous system, alimentary tract, hematopoietic organs and circulatory system. The influence on that latter system manifests as increased occurrence of hypertension, brain strokes, rhythm disturbances and cardiomyopathy [4]. Most of the researches report that symptoms of cardiomyopathy appear after 15 years of alcohol consumption, more than 90 grams of alcohol per day (8 to 21 drinks – 12 grams of alcohol as one drink) [1, 5]. However, basing on the trials, there is no linear dependency between alcohol concentration and its toxic influence on myocardial cells [6, 7]. In trials carried out by Mathiews et al., alcoholics with heart failure symptoms consumed alcohol for over 10 years, while in asymptomatic alcoholics this period was over 6 years [8]. All of them consumed comparable amounts of alcohol.

It is known that alcoholic cardiomyopathy occurs in 2 phases: asymptomatic and symptomatic. The latter manifests itself with heart failure symptoms and appears in an advanced stadium of the illness. In the primary stage it gives very discrete clinical symptoms. Basing on many trials it is known that an early symptom of this illness is enlargement of heart chambers, especially in end-diastolic dimension (EDD) and also end-systolic dimension (ESD). Apart from it, there is also an increase in left ventricular mass and moderate hypertrophia of the interventricular septum and posterior wall. In a 4 year trial carried out by Fernandez-Sola et al., in which they estimated disturbances of diastolic function both in a group of patients with alcoholic cardiomyopathy (EF <50%) and in alcoholics without heart failure symptoms (EF>50%), it turned out that disturbance of the diastolic function was seen in 1/3 of patients without cardiomyopathy and in 2/3 of patients with heart failure. What is more, the rate of disturbances described, depended on the dose of alcohol consumed [9].

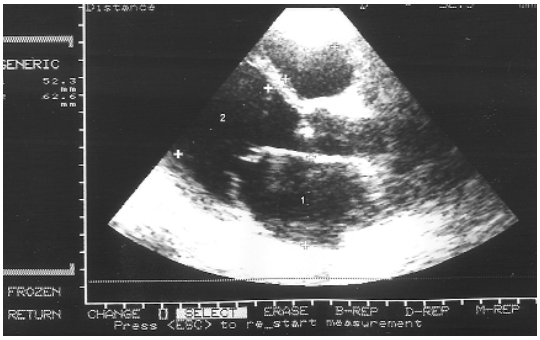

Figure 6. Reduction of the left ventricular volume after starting treatment

The toxic influence of alcohol, results from the harmful metabolite of ethanol-acetic aldehyde. After a great exposure of heart muscle to alcohol, disturbances in the mitochondrial structure and endoplasmatic reticulum were noticed. These changes were connected both with a decrease in mitochondrial function and dysfunction in protein synthesis and also with a tendency to arrhythmia. In biopsy taken from a muscle damaged by alcohol, in about 30% of cases, myocarditis with lymphocyte infiltration, myocyte degeneration and focal necrosis were identified.

Table I. The effects of alcohol on the organism

Units	Beer-mug	Glass of wine	Glass of vodka	Blood level mg/100 ml	Typical effects
2	1	2	2	30	good humour
3	1.5	3	3	50	relax
5	2.5	5	5	80	clumsiness
6	3	1 bottle	6	100	shaky step
10	5	1 litre	10	150	problems with speech
12	6	2 bottles	1/2 bottle	200	drowsiness and intoxication
18	9	3 bottles	3/4 bottle	300	drunken stupor
24	10	4 bottles	1 bottle	400	extreme stupor

Inflammatory process involved also microtubule filament system and increased the activity of fibroblasts, endothelial cells, granulocytes, macrophages and lymphocytes. Patients indulging in alcohol and having symptoms of myocarditis, have worse prognosis, because coexistence of these two factors intensifies the signs of left and right ventricular heart failure: tachycardia, supraventricular and ventricular arrhythmias, hepatomegaly, periferal edema, inclusively with pulmonary edema [10].

Acetic aldehyde mentioned above, directly decreases contractibility of the heart muscle and increases lipid oxidation, mainly LDL cholesterol, which is a crucial factor in atherogenesis and causes a greater risk of cardiovascular events in alcoholics. These processes result from oxidative stress caused by alcohol, which is closely connected with metabolism of ethanol.

So far, three metabolic pathways have been described, including numerous enzymes: dehydrogenase, microsomal ethanol-oxidizing system (MEOS) and catalase. In each of these pathways, free radicals are produced, influencing the antioxidative system, mainly by a decrease of superoxide dismutase and glutation peroxidase activity [11].

Alcohol also causes hypokalemia, which may predispose to paroxysmal atrial fibrillation (holiday heart syndrome). In trials carried out by Fauchier et al., it turned out that frequency of atrial arrhythmias and also ventricular tachycardias does not differ significantly both in patients with idiopathic and alcoholic cardiomyopathy. In the trial mentioned above, including 194 patients with dilated cardiomyopathy, without ischemic background, 119 had idiopathic cardiomyopathy and 75 alcoholic cardiomyopathy. Among patients with alcoholic cardiomyopathy, 47 were abstinents and 28 were still consuming alcohol. The period of follow-up was 42-51 months. During that time there were 14 sudden deaths, 10 sustained ventricular tachycardias and ventricular fibrillation in 3 groups of patients, but the risk of sudden death and arrhythmias was comparable to the group of patients with idiopathic and alcoholic cardiomyopathy, not keeping abstinence [12].

It is worth mentioning that alcohol abuse also leads to the change of cellular concentration of copper, phosphor and calcium. In alcoholics, the increase of intracellular concentration of calcium was observed and usage of verapamil prevented from contractibility and metabolism disturbances [13]. Other microelements which were examined in alcoholics were zinc and magnesium and their concentrations were considerably lower in the control group, correlating with the raised concentration of copper [14].

As it results from the studies of Patel at al. acetic aldehyde and acid ethyl esters may disturb mitochondrial oxidative phosphorylation processes. In severe alcohol intoxication, activations of xantin oxidase and beta-adrenergic stimulation are increased, which may provoke myocardial ischemia [15].

Because of myocyte dysfunction there is an activation of the sympathetic nervous system (norepinephrine) and neuropeptides. The higher level of norepinephrine is closely connected with myocardial remodelling through the influence on myocyte hypertrophy and apoptosis [16, 17]. Probably, the influence of theses hormones takes place through beta-1 receptors. The role of alfa-1 receptors is still controversial. In one of the trials, it turned out that 60 days alcohol consumption had no influence on alfa receptors stimulation [18]. In another trial, researchers observed that activation of alfa receptors causes myocardial hypertrophy through the activation of phosphatidyl inozitol and protein kinase C [19]. The diagram describing pathogenesis of alcoholic cardiomyopathy is presented below [20].

Alcohol consumption >90 grams >5 years

⇓

• Apoptosis (programmed cell death caused by the direct influence via alcohol or indirect via ↑ norepinephrine)
• ↓ synthesis and/or accelerated degradation of contractile proteins
• ↓ myofilament Ca++ sensitivity
• Intrinsic myocyte dysfunction due to mitochondrial and sarcoplasmic dysfunction (due to Ca++ overload, fatty ethyl esters or norepinephrine)

⇓

Cell drop out and weakly contracting myocytes

⇓

Decreased cardiac output

⇓

• LV dilation to increase EDV (preload) to compensate for ↓ cardiac output however, this may be accompanied by wall thinning due to cell drop out
• Hypertrophy of normal myocytes to compensate for weakly contracting neighboring myocytes

⇓

Continued drinking >15 years
• Progressive LV dilation and wall thinning
• Activation of other neurohormonal systems
• Signs and symptoms of heart failure

Symptoms of cardiomyopathy in women occur between 45-50 years of age, which is comparable to men. Also symptoms of heart failure are similar [21]. However, women seem to be more susceptible to the toxic influence of alcohol and a shorter time of its consumption causes occurrence of clinical symptoms.

Treatment of cardiomyopathy is the same as in heart failure. In Fauchier et al. trial [5] it turned out that usage of drugs inhibiting left ventricular remodelling and decreasing symptoms of heart failure: β-blockers, angiotensin-converting enzyme inhibitors, diuretics and digitalis glycosides, increases left ventricular ejection fraction (LVEF). These results were observed both in the group of patients keeping

abstinence and in the group continuing drinking. Different results were published by Gavazzi who noticed improvement only in the group of abstinents. Probably it resulted from the fact that in the first trial more patients were treated with β-blockers. However, in both trials mortality in the group of patients still consuming alcohol is higher than in the group of abstinents. La Vecchia et al. evaluated male alcoholic subjects (n=19) with systolic dysfunction (EF <40%). Fifteen of these patients abstained, while 4 patients reduced their alcohol intake to <40 g/d (approximately three drinks). All of these patients received digitalis and diuretics. At 23 months of follow-up, approximately one half of the patients had a significant improvement in LV EF (baseline EF, 28.5±9%; follow-up EF, 53.3±10%), whereas the other one half of the patients did not exhibit an improvement in ventricular function (no change in EF). The difference between the improved and nonimproved groups were baseline (enrollment) PAP and PCWP values, in that baseline PAP and PCWP values were significantly higher in the nonimproved group (40.3±12.4 mm Hg and 26.5±7.7 mm Hg, respectively) compared to the improved group (27.8±13.3 mm Hg and 18.4±8.9 mm Hg, respectively). These large PAP and PCWP values may be indicative of very diseased hearts and heart failure, such that medical therapy is not very effective [22].

Conclusions

Alcoholism is one of the main causes of myocardial damage (apart from ischemic etiology). Long-term alcohol consumption causes alcoholic cardiomyopathy which represents about 3.8% of all cardiomyopathy cases. Pathophysiology and progression of alcoholic cardiomyopathy is complex and involves changes in many aspects of myocyte function.

It manifests as myocardial hypertrophy, steatosis and enlargement of heart chambers, which decreases its contractibility and cardiac output. Also the risk of hypertension and brain stroke is higher. There are multiple theories concerning the cause of cardiomyopathy: damage of oxidative processes, excessive cumulation of triglycerides, decreased myofilament Ca++ sensitivity and synthesis of proteins.

The symptomatic alcoholic cardiomyopathy stage is characterized by pronounced LV dilation, increased LV mass, wall thinning, systolic dysfunction, and signs and symptoms of heart failure. Treatment of these patients should include alcohol abstinence and recommended heart failure pharmacotherapy.

References

1. Gavazzi A, De Maria R, Parolini M, Porcu M. Alcohol abuse and dilated cardiomyopathy in men. Am J Cardiol 2000; 85: 1114-8.
2. McKenna CJ, Codd MB, McCann HA, Sugrue DD. Alcohol consumption and idiopathic dilated cardiomyopathy: a case control study. Am Heart J 1998; 135: 833-7.
3. Vital and Health Statistics. Hyattsville, MD: U.S. Department of Health and Human Services. Centers for Disease Control and Prevention. 1995; 13 (122).
4. Schoppet M, Maisch B. Alcohol and the heart. Herz 2001; 26: 345-52.
5. Fauchier L, Babuty D, Poret P, Casset-Senon D, Autret ML, et al. Comparison of long-term outcome of alcoholic and idiopathic dilated cardiomyopathy. Eur Heart J 2000; 21: 306-14.
6. Kupari M, Koskinen P, Suokas A, Ventila M. Left ventricular filling impairment in asymptomatic chronic alcoholics. Am J Cardiol 1990; 66: 1473-7.
7. Lazarevic AM, Nakatani S, Neskovic AN, Marinkovic J, Yasumura Y, et al. Early changes in left ventricular function in chronic asymptomatic alcoholics: relation to the duration of heavy drinking. J Am Coll Cardiol 2000; 35: 1599-606.
8. Urbano-Marquez A, Estruch R, Navarro-Lopez F, Grau JM, Mont L, et al. The effects of alcoholism on skeletal and cardiac muscle. N Engl J Med 1989; 320: 409-15.
9. Fernandez-Sola J, Nicolas JM, Pare JC, Sacanella E, Fatjo F, et al. Diastolic function impairment in alcoholics. Alcohol Clin Exp Res 2000; 24: 1830-5.
10. Wilke A, Kaiser A, Ferency I, Maisch B. Alcohol and myocarditis. Herz 1996; 21: 248-57.
11. Zima T, Fialova L, Mestek O, Janebova M, Crkovska J, et al. Oxidative stress, metabolism of ethanol and alcohol-related diseases. J Biomed Sci 2001; 8: 59-70.
12. Fauchier L. Alcoholic cardiomyopathy and ventricular arrhythmias. Chest 2003; 123: 1320.
13. Piano MR, Schwertz DW, Solaro RJ, et al. Effect of pimobenden and isoproterenol in the alcoholic myocardium. J Cardiovasc Pharmacol 1999; 33: 237-42.
14. Avsaroglu D, Inal TC, Demir M, Attila G, Acarturk E, et al. Biochemical indicators and cardiac function tests in chronic alcohol abusers. Croat Med J 2005; 46: 233-7.
15. Patel VB, Why HJ, Richardson PJ, Preedy VR. The effects of alcohol on the heart. Adverse Drug React Toxicol Rev 1997; 16: 15-43.
16. Adams MA, Hirst M. Lack of cardiac alpha 1-adrenoceptor involvement in ethanol-induced cardiac hypertrophy. Can J Physiol Pharmacol 1989; 67: 240-5.
17. Adams MA, Hirst M. Metoprolol suppresses the development of ethanol-induced cardiac hypertrophy in the rat. Can J Physiol Pharmacol 1990; 68: 562-7.
18. Piano MR, Schwertz DW. Effect of chronic ethanol exposure on myocardial phosphoinositide turnover. Alcohol Clin Exp Res 1997; 21: 721-7.
19. Molkentin JD, Dorn II GW 2nd. Cytoplasmic signaling pathways that regulate cardiac hypertrophy. Annu Rev Physiol 2001; 63: 391-426.
20. Piano MR. Alcoholic cardiomyopathy: incidence, clinical characteristics, and pathophysiology. Chest 2002; 121: 1638-50.
21. Fernandez-Sola J, Estruch R, Nicolas JM, Pare JC, Sacanella E, et al. Comparison of alcoholic cardiomyopathy in women versus men. Am J Cardiol 1997; 80: 481-5.
22. La Vecchia L, Bedogni F, Ometto R, Mosele GM, Bevilacqua P, et al. Prediction of recovery after abstinence in alcoholic cardiomyopathy: role of hemodynamic and morphometric parameters. Clin Cardiol 1996; 19: 45-50.