Cardiac benefits after alcohol withdrawal

Matteo Cameli1§, Matteo Lisi1, Benedetta Maria Natali1, Ester Nucci1, Stefano Lunghetti1, Marta Focardi1 and Sergio Mondillo1

1 Department of Cardiovascular Diseases, University of Siena, Siena, Italy
§ Corresponding author

Key words: Cardiac toxicity; Alcohol withdrawal; Cardiomyopathy; Echocardiography; Left ventricular function; Speckle tracking.

Introduction
Numerous studies demonstrated that a chronic consumption of alcohol is directly related to alcoholic cardiomyopathy (ACM), which is a specific dilated cardiomyopathy.

At an early stage of heart disease, before a clinical evidence of cardiac abnormality appears, the repeated ingestion of ethanol in intoxicating doses causes an asymptomatic diminished left ventricle (LV) function1,2,3. In asymptomatic alcoholic patients the most frequent echocardiographic findings are LV dilation, an increase of LV mass, diastolic and/or systolic dysfunction and sometimes a modest degree of wall thickening, which, coupled with LV dilation, would serve to offset wall tension and lead to a compensated and asymptomatic form of ACM. Manifested alcoholic cardiomyopathy is characterized by an increased LV mass, cavity dilatation, wall thinning and LV ventricular dysfunction; this pattern is seen in the absence of coronary artery disease and nutritional deficiencies.

These patients are clinically similar to people affected by idiopathic dilated cardiomyopathy (IDCM). An important difference between ACM and IDCM, is that the former has a good prognosis if abstinence of alcohol is kept, while the latter has a worse survival outcome4; in fact many studies, concerning the prognosis of these patients, confirmed the reversibility or the partial recovery of cardiac function after abstinence from alcohol intake5. At the same time, for alcoholic patients that refuse abstinence, the prognosis becomes worse than for IDCM patients, with a very short survival outcome3.

Case history
A 47-year-old man was admitted to our hospital because of severe dyspnoea. The patient reported that in the last month he presented a progressive fatigue and less tolerability to efforts. The rest of potential risks for ventricular dysfunction were absent and the patient confirmed his previous wellness. Nevertheless, he admitted a history of heavy alcohol intake in the last few years. His daily consumption consisted of an average of 7-8 standard alcoholic drinks equivalent to ethanol 93.5 g/day6 of red wine per day. An urgent chest radiography demonstrated cardiomegaly and interstitial pulmonary edema. An echocardiographic examination revealed the presence of a

Figure 1: Echocardiographic measurement of left ventricular ejection fraction during the first exam (left) and 15 months after alcohol withdrawal (right). Note the significative difference between ventricular volumes, that allowed an increase of left ventricular ejection fraction from 34% to 52%.
dilated LV (end diastolic diameter, EDD: 60 mm and end systolic diameter, ESD: 51 mm) with a depressed systolic function (ejection fraction, EF of 34%) (Figure 1). This moderate-severe systolic dysfunction, extended mainly to the antero-lateral segments, was confirmed by a more in-depth and topographic analysis of myocardial deformation by speckle tracking echocardiography (STE) (Figure 2); the global longitudinal strain (GLS), derived by the analysis of LV myocardial deformation in four-, two-chamber and apical long-axis view, was depressed (12.2%). There was no significant structural valve disease.

After clinical stabilization with loop diuretics, intravenous nitrates and ACE inhibitor, the patient was discharged with a standard therapy for the heart failure and with the recommendation of cutting down alcohol intake to the minimum or complete abstinence. Subsequently, after 10 months of abstinence, the patient came back to hospital for a visit. He was well, reported good exercise tolerance and no new episodes of dyspnea. The echocardiographic examination showed a significant recovery of LV systolic function (EF: 43%). In addition LV dimensions decreased (EDD: 55 mm and ESD: 40 mm). STE analysis demonstrated a moderate recovery of GLS (16.5%), with evidence of an improvement of LV myocardial deformation in antero-lateral segments (Figure 2). Five months later, the echocardiographic images showed a good left ventricular systolic function, with a further improvement with normalization of the LV dimensions (EDD: 50 mm and ESD: 32 mm) and of systolic function, (EF: 52%; GLS: 20.6%) (Figure 1). The bull eye, topographic representation of global segmental myocardial deformation, showed complete normalization in all LV segments (Figure 2).

Discussion

It has been demonstrated that excessive long-term alcohol consumption produces a number of histological and cellular changes like myocyte loss, intracellular dysfunction, as alterations in mitochondrial ultrastructure and sarcoplasmic reticulum, alterations of contractile proteins and calcium homeostasis and increase of myocardial lipid peroxidation by oxygen radicals production. These may represent the initial myocardial alteration that could finally lead to ACM. In fact, one of the direct consequences of these alterations is the activation of sympathetic nervous and of renin-angiotensin systems through this way high level of norepinephrine conduces to myocyte hypertrophy, toxicity, apoptosis and LV remodeling causing a progressive fall in LV contractility.

In fact, it is well known that LV function begins deteriorating before the patient becomes symptomatic, and that an early withdrawal of alcohol consumption may improve significantly the prognosis. However, although it is demonstrated that alcohol is related to DCM, the real correlation between specific doses of ethanol, genetic susceptibility and cardiomyopathy development still remains unclear. This is the first image study that well reports improvement of LV myocardial shape and function in a patient with ACM after alcohol withdrawal. The visual impact of images is strongly suggestive of the clear cardiac benefits derived by the termination of the abuse of alcohol. The execution of serial echocardiograms allowed us to follow various stages of patient’s medical history, documenting a progressive reduction of LV volumes and a progressive improvement of global and segmental LV performance.
Disclosure:
The authors have no conflicts of interest

Correspondence to:
Dr. Matteo Cameli, MD
U.O. Cardiologia Universitaria, Policlinico “Le Scotte”, viale Bracci 1, Siena, Italy.
Tel./Fax: +390577585377.
E-mail: cameli@cheapnet.it

References