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Chronic heavy alcohol consumption and asymptomatic cardiovascular effects: An observational study

Chronic heavy alcohol consumption, cardiovascular effects

Ertan Aydın[1], Murat Akcay[2]
[1]Clinic of Cardiology, Giresun University, Prof. Dr. A. Ilhan Ozdemir Training and Research Hospital, Giresun
[2]Department of Cardiology, Faculty of Medicine, Ondokuz Mayis University, Samsun, Turkey

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Abstract

Aim: The association between chronic heavy alcohol consumption and a number of adverse cardiovascular consequences such as hypertension, dyslipidemia, dysrhythmia, coronary artery disease, sudden cardiac death, and particularly dilated cardiomyopathy is becoming increasingly more evident.
In this study, our objective was to determine the preclinical cardiac effects of chronic heavy alcohol consumption in young and middle-aged asymptomatic healthy individuals.
Material and Methods: The study is planned as cross-sectional and observational. A total of 40 men between 25 and 55 years of age with weekly alcohol consumption of ≥ 850 g for a minimum duration of 8 years (chronic heavy alcohol group) and 40 men with no alcohol use (control group) were included. The demographic characteristics, results of echocardiographic and electrocardiographic assessments and epicardial adipose tissue thickness were recorded.
Results: Individuals with alcohol use had significantly higher systolic and diastolic blood pressures, increased left atrial antero-posterior diameter, increased interventricular septum and posterior wall thickness, and higher incidence of stage 1 diastolic dysfunction ($p< 0.05$). Epicardial adipose tissue thickness was higher in alcohol users group compared to the control group (5.46±1.65 vs 3.20±1.03, $p = 0.0001$). Also, the incidence of atrial fibrillation and right bundle branch block were increased compared to the control group.
Discussion: Our results have shown that chronic heavy alcohol consumption is associated with diastolic dysfunction, increased epicardial adipose tissue thickness, electrocardiographic disturbances, and atrial fibrillation. These findings show that alcohol use leads to a variety of asymptomatic changes in cardiac functions.

Keywords
Chronic alcohol; Cardiovascular effects; Epicardial adipose

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Corresponding Author: Murat Akcay, Department of Cardiology, Faculty of Medicine, Ondokuz Mayis University, Samsun, Turkey.
E-mail: drmuratakcay@hotmail.com GSM: +90 506 779 57 60
Corresponding Author ORCID ID: https://orcid.org/0000-0002-4610-8514

Introduction

Alcohol is one of the most widely abused drinks worldwide and influences many organ systems, covering the cardiovascular system [1, 2]. Alcohol consumption in light and moderate doses has a beneficial and protective effect against cardiovascular events; however chronic heavy alcohol intake causes different cardiovascular diseases [3, 4]. In these individuals, the mechanisms of alcohol toxicity and potential cardio-protective effects continue to be investigated due to different outcomes in various studies [5, 6, 7].

Chronic heavy alcohol drinking may lead to cardiovascular, metabolic, and toxic disorders, abnormalities in ventricular diastolic and/or systolic dysfunction, elevated arterial blood pressure, angina pectoris, coronary artery disease, arrhythmia, and even sudden cardiac death, increased risk of stroke, obesity, cancer, liver diseases, etc [6-13]. Therefore, early detection of the sub-clinical cardiac abnormalities is essential to chronic heavy alcohol drinkers which can be detected with electrocardiographic and echocardiographic evaluation and, benefit from early treatment.

In this study, we aimed to detect the pre-clinical cardiac effects of chronic heavy alcohol consumption in asymptomatic young and middle-aged subjects using echocardiographic and electrocardiographic alterations. Also, we examined the association between chronic heavy alcohol consumption and epicardial adipose tissue thickness, which is a marker of visceral fat tissue, is being increasingly investigated, associated with a variety of untoward atherosclerotic cardiac diseases [14].

Material and Methods

Study Design

The present study is cross-sectional and observational.

Study Population

The study included 40 subjects who were followed up at the department of Psychiatry with a diagnosis of alcohol dependence or alcohol use disorder and 40 control subjects from the department of Cardiology matched for demographic and clinical characteristics. Asymptomatic male individuals with no history of systemic or cardiac disease and no substance abuse other than smoking who met the diagnostic criteria for alcohol abuse established by the American Psychiatric Association were included if they had a weekly consumption of ≥ 850 g of alcohol and ≥ 4 days/week, for a minimum duration of 8 years (Group 1). The patients drank alcohol one week before the examination at the earliest and blood alcohol levels were not evaluated. The control group (Group 2) consisted of healthy age-matched men who had never used alcohol. Since all subjects in Group 1 were smokers, control subjects were also chosen among smokers.

For the measuring of the amount of ethanol in grams, the mean alcohol percentage in the drinks was recorded. Then, the amount of pure alcohol intake in ml/week was measured according to the alcoholic concentration of each drink. The total duration of alcohol use, the weekly amount of unit consumption and alcohol drinks species such as beer, wine, whiskey, etc were recorded. A 70 cl Raki (a local drink) contains 248 g of ethanol and most subjects reported regular use of Raki. Ethanol content of Raki is 45%. A 70 cl Raki is 700 cm3 and the density of ethyl alcohol is 0.79 g / cm3 [15]. If the mean weekly consumption of ethanol was below 850 g when all beverages were considered, the subject was excluded from the study. Again, a minimum duration of 8 years of alcohol use was a prerequisite for study participation. Those with light alcohol consumption or those without psychiatric abuse were excluded. In both groups, subjects were > 25 and < 55 years of age, had no additional substance abuse problem and no history of hypertension, diabetes, systemic or cardiac disease history. Also, poor echocardiographic image quality and unwillingness for the study were reasons for exclusion from the study. Female subjects were not included in the study due to the extreme rarity of chronic heavy alcohol use among women and also social factors.

Clinical and Laboratory Assessments

A complete physical examination was performed in all subjects. Age, anthropometric assessments of height and weight were recorded, and body mass index (BMI, kg/m2) was calculated by dividing body weight in kilograms by the square of the height in meters. Systolic and diastolic blood pressures and heart rate were calculated after 10 min of rest in a quiet room. Routine biochemistry and lipid parameters could not be assessed, as most of the laboratory results consisted of hepatic and renal function test that were performed in other health clinics on an irregular basis, and were detected at normal limits and as most of the subjects experienced the compliance problems.

The study protocol was confirmed by the local ethics committee. Patients provided written informed consent after oral and written information was given.

Transthoracic Echocardiographic Evaluation

All echocardiographic measurements were performed with a Vivid 7 device (GE Vingmed Ultrasound, Horten, Norway) with a 2.5 MHz probe. The following were measured using parasternal long axis two-dimensional transthoracic echocardiography: left atrium (anterior-posterior) diameter, right ventricle diastolic diameter, the thickness of interventricular septum and posterior wall, and the systolic and diastolic diameters of the left ventricle. Using two-dimensional images, septum and posterior wall movements, valvular morphology, and left atrial and left and right ventricular chambers were evaluated. Apical four and two-chamber views were used to calculate the left ventricular ejection fraction using the modified Simpson's technique with left ventricular end-systolic and end-diastolic volume measurements.

Using the apical 4-chamber views, transmitral flow rates (E, A, E/A) were assessed and time-analyses (IVRT, DT) were performed. Also, tissue Doppler examination was performed on the mitral annulus, including the mitral septal and lateral annulus tissue (E', A', S'). The mean of the three consecutive mitral annulus pulsed wave tissue Doppler measurements was obtained. In apical 4-chamber views, a continuous wave Doppler cursor was placed on the trans-tricuspid flow, and the estimated right atrial pressure value was added to this flow to calculate the pulmonary artery pressure. The right atrial pressure was calculated using the subcostal images based on the diameter of the inferior vena cava and its variability during respiration.

Epicardial Adipose Tissue Measurement

For epicardial adipose tissue measurements, the aortic annulus

was taken as an anatomic reference in the parasternal long and short axes, and the area between pericardial layers with the lowest echo-density perpendicular to the RV free wall was measured two-dimensionally and with the M-mode (Figure 1). The average end-diastolic value measured in three cardiac cycles was recorded.

Electrocardiography (ECG)

Twelve-lead ECGs were obtained after a 10-minute rest, with 10 mm/mV amplitude and 25 mm/s rate with standard lead positions an ECG machine (Nihon Kohden, Tokyo, Japan). ECGs were manually measured by the use of a magnifying lens and subsequently, ECG recordings were examined with respect to rate, rhythm, axes, and time intervals (PR, QRS, QTc) as well as pathological findings.

Statistical Analyses

Data obtained from study participants were entered into the database of SPSS v.15.0 software for Windows (SPSS Inc. Chicago, Illinois, USA). Normal distribution of the data was tested using the Kolmogorov-Smirnov test. For data without normal distribution, the Kruskal- Wallis analysis of variance and the Mann-Whitney U tests were used. The data were presented as number, percentage, and arithmetic mean ± standard deviation. Groups were compared using the Student's t-test and the Chi-square test, and the correlations between variables were evaluated by means of the Pearson's correlation analysis. The grade of importance was set at a P- value of less than 0.05.

Results

When comparing alcohol users (Group 1) and non-users (Group 2) a trend toward higher bodyweight and BMI in Group 1 was shown, but there was no statically significant difference ($p > 0.05$). There were no significant differences in terms of age, height, body weight, and baseline demographic characteristics ($p > 0.05$). However, alcohol users had significantly higher heart rate and systolic and diastolic blood pressure ($p = 0.001$; $p = 0.005$; $p = 0.034$, respectively) (Table 1).

Individuals in Group 1 had significant increase in the left atrial anterior-posterior diameter, septal and posterior thickness, and systolic pulmonary artery pressure (sPAB) ($p = 0.003$; $p = 0.001$; $p = 0.016$ and $p = 0.001$ respectively). There were no important variations in terms of left ventricular end-diastolic diameter, end-systolic diameter, and right ventricular diameter between the two groups ($p > 0.05$). Also, epicardial adipose tissue thickness (EAT) was significantly higher in Group 1 (5.46±1.65 vs 3.20±1.03, $p = 0.0001$) (Table 1). When comparing the transmitral flow (E, A), time parameters (IVRT, DT), and mitral annulus tissue Doppler parameters (S', E', A'), there were no significant differences between two groups ($p > 0.05$). There was only statically significant higher E/A ratio and grade 1 diastolic dysfunction in Group 1 ($p = 0.011$; $p = 0.034$, respectively) (Table 2).

Electrocardiographic analysis showed significant elevation of heart rate in alcohol users (77 bpm/min vs 68 bpm/min $p = 0.001$). Atrial fibrillation and right bundle branch block were identified in each of the two patients, while there were no such cases in alcohol non-users (Table 3).

Table 1. Basic demographic, echocardiographic characteristics and epicardial adipose tissue thickness parameters of patients with chronic heavy alcohol (+) and control groups

	Chronic heavy alcohol group (n = 40)	Control group (n = 40)	P- value
Age (year)	40.650	40.925	NS
Height (cm)	174.525	174.350	NS
Weight (kg)	82.425	79.375	NS
BMI (kg/m2)	27.2	26.3	NS
Systolic BP(mmHg)	129.250	119.000	0.005
Diastolic BP(mmHg)	83.500	78.625	0.034
LVEDD (mm)	44.500±3.41	46.25±3.62	NS
LVESD(mm)	29.575±3.85	28.825±3.46	NS
IVS (mm)	12.075±1.81	10.887±1.19	0.003
PW (mm)	10.600±1.27	9.712±1.04	0.001
LA diamater (mm)	34.375±2.92	32.775±2.86	0.016
RV (mm)	27.750±3.32	26.775±2.45	NS
sPAB (mmHg)	28.800±4.15	25.425±3.88	0.001
LVEDV (cm^3)	65.1±23.4	66.1±15.8	NS
LVESV(cm^3)	20±7.1	20.6±5.3	NS
LVEF (%)	61.4±3.22	61.6±2.4	NS
EAT thickness (mm)	5.46±1.65	3.20±1.03	0.0001

BP = Blood pressure; EAT = Epicardial adipose tissue; LVEDD = left ventricular end-diastolic diameter; LVESD = left ventricular end-systolic diameter; IVS = Interventricular septum; PW = Posterior wall; LA = Left atrium-parasternal long axis; RV = Right ventricle; sPAB = Systolic pulmonary artery pressure; LVEDV = left ventricular end-diastolic volume; LVESV = left ventricular end-systolic volume; LVEF = left ventricular ejection fraction; P > 0.05; Non-significant (NS), P < 0.05; Significant

Table 2. Tissue Doppler and diastolic echocardiographic parameters of patients with chronic heavy alcohol (+) and control groups

	Chronic heavy alcohol group (n = 40)	Control group (n = 40)	P- value
Transmitral E (m/sec)	0.69±0.12	0.76±0.16	NS
Transmitral A (m/sec)	0.59±0.11	0.56±0.16	NS
E/A	1.20±0.28	1.37±0.25	0.011
Mitral DT (msn)	160.57±23.2	155.9±42.3	NS
IVRT (msn)	78.17±26.4	79.3±18.9	NS
Mitral lateral annulus S' (cm/sec)	11.5±2.4	11.6±3.0	NS
Mitral lateral annulus E' (cm/sec)	17.4±3,2	16±2.9	NS
Mitral lateral annulus A' (cm/sec)	9.8±2.0	10.1±2.7	NS
Mitral septal annulus S' (cm/sec)	10.2±2.1	9.7±3.5	NS
Mitral septal annulus E' (cm/sec)	14.76±2.5	14.±2.9	NS
Mitral septal annulus A' (cm/sec)	11.5±2.9	10.09±3.0	NS
Mitral annulus mean S' (cm/sec)	10.8±1.3	10.4±1.9	NS
Mitral annulus mean E' (cm/sec)	16±2.0	17.1±1.8	NS
Mitral annulus mean A' (cm/sec)	11.3±1.7	10.8±1.9	NS
Mitral annulus E'/A'	1.59±0.22	1.68±0.27	NS
Mitral E / mean E'	4.67±0.77	4.49±0.55	NS
Evre I diastolic dysfunction (n)	10	3	0.034

E wave velocity; Mean peak early filling wave velocity, A wave velocity; Mean peak late filling wave velocity due to atrial contraction, E'm wave velocity; Mean peak early diastolic annulus velocity, A'm velocity; Mean peak late diastolic annulus velocity, S'm wave velocity; Mean peak systolic annulus velocity, IVRT; Isovolumetric relaxation time, DT; E wave deceleration time

Table 3. Electrocardiographic parameters of patients with chronic heavy alcohol (+) and control groups

	Chronic heavy alcohol group (n = 40)	Control group (n = 40)	P- value
Sinus rhythm	38	40	NS
Atrial fibrillation	2	0	NS
Heart Rate (bpm/min)	77	68	0.001
PR interval (msn)	155	151	NS
QRS (msn)	87.65	88.45	NS
QTc (msn)	387.35	381.45	NS
Bundle block	2 (RBBB)	0	NS

P > 0.05; Non-significant (NS), P < 0.05; Significant, RBBB; Right bundle branch block

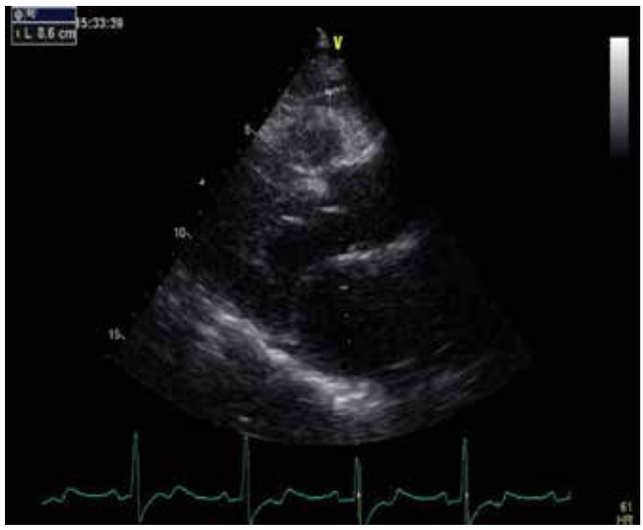

Figure 1. Two-dimensional echocardiographic epicardial fat measurement, aortic annulus was considered as an anatomical landmark, and low echogenic density area perpendicular to the lines drawn from parasternal long axis to the free wall of RV.

Discussion

In this study, asymptomatic cardiac effects were compared between young and middle-aged, asymptomatic chronic heavy alcohol users and age-matched, healthy males with no known medical conditions. Alcohol users were found to have significantly elevated systolic and diastolic blood pressure, heart rate, left atrial dimensions, interventricular septal and posterior wall thickness, systolic pulmonary artery pressure, epicardial fat tissue thickness, and diastolic dysfunction (Grade 1) as compared to alcohol non-users. In electrocardiography, asymptomatic right bundle branch block and atrial fibrillation were detected in two patients each.

Alcohol is among the most widely abused drinks worldwide and affects many organ systems, including the heart and vessels [1]. Currently, disorders involving alcohol use are categorized with DSM-IV criteria, into two groups: alcohol use disorder and alcohol dependence [16]. These disorders are a significant cause of mortality, morbidity, and social problems in many countries. Some epidemiological studies have shown beneficial effect of low alcohol consumption [6-13]. Although low alcohol consumption has some beneficial effects, the number of investigations of side effects of alcohol consumption commonly increase, as in our study [6-13].

Alcoholic cardiomyopathy (CMP) is defined as enlarged cardiac chambers with largely hypertrophy and extracellular matrix (ECM) remodeling [1]. Major mechanisms implicated in the development of alcoholic cardiomyopathy include the direct toxic effect of ethanol and it metabolites (acetaldehyde and acetate) on the myocardium; vitamin (thiamine) and mineral (selenium) deficiencies as well as electrolyte (Mg, P, K) imbalance-deficits seen in heavy alcohol users; additional toxic effect of substances such as lead or cobalt found in alcoholic beverages; and genetic predisposition for ethanol toxicity (e.g. ACE gene) [17, 20]. Chronic and intense toxic effects lead to systolic dysfunction and dilated cardiomyopathy [17, 18, 20]. In the current study, alcohol use was found to be associated with significant diastolic dysfunction, although no subjects had systolic failure. These findings are in corroboration with the observation that diastolic heart failure represents an initial stage in the development of alcoholic CMP [17, 18]. Accordingly, in a study by Fernandez Sola J et al. [18], involving a total of 112 chronic heavy alcohol users, diastolic dysfunction was present in two-thirds of those with systolic HF and in one-third of individuals with an ejection fraction of higher than 50%, and these findings correlated with the amount of alcohol consumed irrespective of age. Similarly, in our study alcohol users had significantly higher incidence of diastolic dysfunction. In an echocardiographic study, involving 34 young (< 45 age) male alcoholic subjects with no cardiovascular symptoms, patients were found to have diastolic abnormality despite normal systolic functions, suggesting that this represents an early sign in the process of alcoholic CMP [19]. In another study by Kycina P et al. [20], a total of 100 individuals with mean daily alcohol consumption of ≥ 120 g and < 120 g alcohol were assessed clinically and echocardiographically during a 4-year follow up period. No significant differences between the two groups were observed with regard to LV ejection fraction, LA diameter, biochemical parameters, DM, and atrial fibrillation. Except for LA dimensions, these findings are consistent with our observations regarding the left ventricular ejection fraction. Therefore, diastolic dysfunction associated with alcohol use carries clinical significance as an initial sign of the CMP process [1, 9, 17, 20]. It should also be borne in mind that other factors may have a predictive role in the development of alcoholic cardiac disease and these include the duration, frequency, and amount of alcohol consumption, the type of alcohol consumed, presence of additional substance abuse (cigarette, marihuana), and family history of cardiomyopathy [3, 4, 12, 13].

Alcohol leads to an elevation of systemic arterial blood pressure, particularly when consumed in moderate or high quantities. Comparisons between age- and gender-matched individuals suggest that those with daily consumption of >2 units of alcohol (20-30 g ethanol) are 1.5 to 2-times more likely to experience hypertension [4, 8]. In the Polish Wobasz study [21] with the participation of 6912 males between 20 and 74 years of age, the cardiovascular risk was classified on the basis of daily alcohol consumption, and a positive correlation between systolic/diastolic blood pressure and alcohol use was found. Polish men with a daily > 30 g of alcohol consumption had a 52% increased risk of developing hypertension [21]. Also,

in our study alcohol users had significantly elevated systolic and diastolic blood pressure values than those without alcohol consumption. On average, systolic and diastolic blood pressures were 10.250 mmHg and 4.875 mmHg were higher in alcohol users. However, there were 10 individuals (25%) with systolic and diastolic hypertension in our study; these measurements did not have diagnostic value as they represent values measured in a single visit.

Hypertension, which may occur in patients with chronic alcoholism, is also associated with increased left ventricular filling pressure, ventricular hypertrophy, diastolic dysfunction, and atrial remodeling and dilatation [22]. So, increased left atrial diameter is a risk factor for stroke and death [22]. In our study, alcohol users had higher left atrial diameter than those without alcohol use. Again, alcohol users with diastolic dysfunction and LA enlargement in our study had statistically significantly higher systolic pulmonary arterial pressure, suggesting that this process may be related to a backward effect of increased pressure secondary to left cardiac disease, i.e. diastolic dysfunction.

Alcohol use may be associated with the occurrence of a number of atrial or ventricular rhythm disorders. The most common dysrhythmia is atrial or ventricular premature beats, while other rhythm disturbances such as supraventricular tachycardia, atrial flutter, atrial fibrillation, ventricular tachycardia, and ventricular fibrillation may also occur. The most common chronic rhythm disorder due to ethanol is atrial fibrillation [23]. Although cardiac routine was not routinely monitored in our study, two individuals with heavy alcohol consumption were found to have atrial fibrillation. Also, the study by Thayer JH, et al. [24] from the US found reduced heart rate variability and increased heart rate among 542 male drinkers. Consistent with these reports, alcohol users in our study had a significantly higher heart rate.

Epicardial adipose tissue (EAT) is a structure of mesodermal origin that is located on the epicardium [14, 25] and that functions as a cushion against accumulation of toxic levels of fatty acids among the myocardium and local vascular layer and also a marker of increased visceral adiposity [25]. Recent studies have suggested a strong correlation between increased epicardial adipose tissue thickness and obesity, impaired glucose tolerance, metabolic syndrome, hypertension, diabetes, atherosclerosis [14, 25]. In our study, alcohol users had a statistically significant increase in epicardial adipose thickness. When one considers the causative role of alcohol in the above-mentioned conditions, a link between alcohol use and an increase in epicardial adipose thickness may exist. For instance, alcohol consumption may represent an independent precursor or even a prognostic marker for the development of alcoholic heart disease through an increase in epicardial adipose mass. At least, it can be assumed that alcohol may lead to increased epicardial fat mass, i.e. visceral adiposity.

Heavy alcohol use is related to an increased incidence of atherosclerotic coronary heart disease, hence the increased cardiovascular morbidity and mortality [5-7, 11]. This elevated risk is at least partially related to traditional risk factors commonly present in heavy drinkers such as systemic arterial hypertension, increased left ventricular muscle mass, and hypertriglyceridemia [11,12,13]. Furthermore, cigarette smoking is also a frequent habit among heavy drinkers. No further assessments for coronary artery disease were performed in our study.

Study Limitations

A major limitation of our study is the fact that the amount of alcohol consumption was estimated on the basis of self-reports of the participants (i.e. days without alcohol use, variability in the type of alcoholic beverages utilized, different amounts of alcohol consumption on different days, etc.). Another limitation of our study is not including female subjects due to the extreme rarity of chronic heavy alcohol use among women. Again, although our participants consisted of asymptomatic individuals, absence of objective assessments for the functional status and physical exercise capacity represent another limitation. Also, arterial blood pressure measurements were performed in a single visit without continuous blood pressure measurements, inadequate access to previous laboratory test results, operator-dependency of the echocardiographic assessments, and small sample size (light and moderate drinkers could also have been included for a more comprehensive comparison between the groups) should be mentioned in this respect.

Conclusion

As compared to a group of individuals with no use of alcohol and with similar baseline characteristics, from young to middle age and apparently healthy males with chronic heavy alcohol users were found to have a number of asymptomatic abnormalities including impaired diastolic function, increased systolic and diastolic blood pressure, LV hypertrophy, dilatation of LA, increased systolic pulmonary artery pressure, and elevated heart rate. Also, to the best of our knowledge, this study represents the first study to show an increased epicardial adipose thickness in chronic heavy alcohol users.

Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and human rights statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal or human studies were carried out by the authors for this article.

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Conflict of interest

None of the authors received any type of financial support that could be considered potential conflict of interest regarding the manuscript or its submission.

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