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ORIGINAL ARTICLE

Effect of insomnia and excessive daytime sleepiness on cardiac functions in older adults

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Key words: *biventricular systolic and diastolic functions, cardiac function, excessive daytime sleepiness, insomnia.*

Abstract

Background: We investigated the potential harmful effect in older adults of insomnia and excessive daytime sleepiness (EDS) on myocardial functions and electrophysiologic changes of the heart in terms of heart rate and QT intervals corrected for heart rate (QTc).

Methods: The study included 32 insomnia patients and 30 control subjects. An Insomnia Severity Index score of ≥15 indicated insomnia, while a score of <8 was accepted as the control group. The Epworth Sleepiness Scale was used to assess EDS, with a score of ≥11/24 points indicating EDS. Diastolic and systolic functions were evaluated in each patient by transthoracic two-dimensional, conventional and tissue Doppler echocardiography. Heart rate and QTc were calculated for electrophysiologic changes.

Results: The mean age was 73.2 ± 7.9 years, with 59.7% being female. Biventricular systolic and diastolic functions were impaired in the insomnia patients. The E' value for diastolic function was lower in the patients with insomnia than the controls (5.99 ± 1.59 vs. 6.88 ± 0.97, $P = 0.053$). Furthermore, values for the systolic function parameters Lateral-S (7.41 ± 1.92 vs. 9.37 ± 1.83, $P < 0.001$), Septal-S (6.69 ± 1.40 vs. 8.10 ± 1.30, $P = 0.001$), and Tricuspid-S (12.25 ± 2.00 vs. 14.37 ± 3.13, $P = 0.004$) were lower for insomnia patients than for controls. In the case of EDS coexistence, the heart rate and QTc values were higher than the controls (76.47 ± 7.18 vs. 71.03 ± 10.95, $P = 0.001$, and 413.72 ± 28.24 vs. 394.67 ± 24.47, $P = 0.015$, respectively).

Conclusion: Insomnia is associated with impaired systolic-diastolic functions, independent of EDS. The co-existence of insomnia and EDS may lead to electrophysiological changes in older adults, including increased heart rate and longer QTc.

INTRODUCTION

Sleep quality is a basic physiological process that protects our physical and mental health. Insomnia, one of the most common sleep disorders in the elderly (65.6%), is defined as difficulty in initiating and maintaining continuous sleep or waking up very early in the morning and not being able to go back to sleep.[1] Experimental and observational studies have shown that insomnia may be associated with diabetes mellitus, obesity, dementia, malnutrition, cardiovascular events, and all-cause mortality.[2–4] On the other hand, excessive daytime sleepiness (EDS), defined as the state of sleepiness during the day when the individual should be active and awake, is the second most common sleep disorder among the sleep disorders of increasing importance in recent years. EDS is associated with malnutrition, dysphagia, vitamin D deficiency, sarcopenia, falls, and anaemia.[5,6] Therefore, both insomnia and EDS are very important in geriatric practice.

Although the underlying mechanisms are unknown, a few studies have shown an association between impaired sleep quality and heart failure.[7,8]

Some experimental studies have reported that both poor sleep quality and short sleep duration may lead to the development of weight gain by affecting the activation of the sympathetic nervous system and the hypothalamus-pituitary-adrenal axis,[9,10] and increasing the levels of hunger-inducing hormones.[11,12] All these may impair the structural content of the left ventricle, which plays a critical role in the development of diastolic and systolic failure in the elderly population.[13] In addition, impaired sleep quality is associated with affecting the balance between muscle protein synthesis and degradation through increased proteolysis[14] leading to loss of muscle mass. This point of view is supported by the observation of higher urinary protein secretion after 72 h of sleep deprivation, a feature of increased proteolytic processing.[15] The way myocardial muscle cells are affected by this process is not known exactly. On the other hand, EDS is also common in the elderly with insomnia, and EDS itself can double the risk of cardiovascular events and mortality.[16]

Therefore, the effect of EDS should also be considered in studies investigating the cardiac effects of insomnia. Given all of the aforementioned factors, we hypothesised that insomnia and EDS can have a cumulative effect on cardiac function. The aim of this study is to investigate whether insomnia has a negative effect on cardiac functions in older patients with and without EDS.

METHODS

The current study complies with the Declaration of Helsinki and was approved by our local ethics committee. The study was approved by the Bezmialem Vakif University ethics committee (E-70946001-050.99-12748/2021). It includes 32 insomnia patients and 30 control subjects who are older than 65 years of age. We paid attention to the fact that the insomnia patients must have experienced this problem for at least 6 months. Patients with any of the following conditions or histories were excluded from this study: moderate or severe heart valve insufficiency and/or stenosis; congenital heart disease; atrial fibrillation; left bundle branch block; established coronary artery disease; myocardial infarction; unstable angina pectoris; angiographically proven significant coronary stenosis (more than 50% lumen stenosis in at least one coronary artery in coronary angiography); revascularization; abnormal exercise test; left ventricular (LV) ejection fraction of <50%; symptomatic heart failure; poor echocardiographic image quality; stroke; chronic obstructive pulmonary disease; and sleep apnea. Patients with dementia or delirium were also excluded because self-reports based on their memory may be unreliable for questions about insomnia and EDS. Informed consent was obtained from all subjects.

Assessment of insomnia

The Insomnia Severity Index (ISI) includes seven questionnaire items that capture self-reported symptoms and daytime outcomes of insomnia. ISI scores range from 0 to 28, with higher scores indicating more severe insomnia. ISI scores of eight or higher indicate insomnia, and severity is classified according to the ISI score as subthreshold.[1] In our study, those with a score of ≥15 were included in the insomnia group, and those with a score of <8 were included in the control group.

Assessment of excessive daytime sleepiness

EDS was assessed using the Epworth Sleepiness Scale (ESS). The ESS is a 4-point Likert-type questionnaire consisting of eight items in which the patient marks the possibility of taking a nap while watching TV, travelling in a vehicle, and lying down to rest, etc. Scoring for each item is on a scale from 0 (no chance to take a nap) to 3 (high probability of napping). The total score ranges from 0 to 24. A score of ≥11 indicates EDS.[1]

All of the participants underwent a complete clinical, anthropometric, and laboratory investigation. Laboratory tests included haematocrit, total cholesterol, low-density lipoprotein (LDL)-cholesterol, high-density lipoprotein (HDL)-cholesterol, triglycerides, alanine transaminase (ALT), aspartate aminotransferase (AST), fasting plasma glucose, serum creatinine, serum albumin, serum uric acid, serum sodium, and potassium levels. Height and weight of all subjects were measured and the body mass index (BMI) was calculated as weight (kg) divided by height^2 (m).

Cardiologic evaluation

During the data collection phase, 12-lead electrocardiography was performed using a standard paper speed of 25 mm/s with standard lead positions. QT intervals were corrected for heart rate (QTc) using the

computer-generated value (i.e., not overread by a cardiologist) from Bazett's correction equation (QT/√RR). Long QTc was defined using standard criteria: QTc ≥470 ms in females and ≥ 460 ms in males.[17] Heart rate (HR) was calculated via 12-lead electrocardiography.

Echocardiographic measurements

All echocardiographic assessments were performed by a single operator, unaware of the clinical and laboratory variables of patients and controls, using the Vivid 7 Dimension echocardiography device with 2.5 MHz phased array converter (GE, Vingmed, Horten, Norway) in the left decubitus position. Left ventricular size and wall thickness were measured from two-dimensional guided M-mode echocardiographic traces at the mid-chordal level in parasternal long-axis view. The M-mode traces were recorded at a speed of 50 mm/s. Ejection fraction was calculated using the Teicholz formula.

Pulse wave Doppler recordings of mitral flow were obtained from the apical four-chamber view with the sample volume placed at the tips of the mitral valve leaflets. The following parameters were measured by pulse-wave Doppler: peak velocities of early (E) and late diastolic filling (A). The ratio of early diastolic to late diastolic mitral inflow velocities was calculated (E/A). All Doppler signals were recorded with a chart recorder set at 100 mm/s. The averages of three cycles were used.

A tissue Doppler imaging (TDI) program was used, set to pulse-wave Doppler mode. Filters were set to exclude high frequency signals. Gains were minimised to allow a clear tissue signal with minimal background noise. The TDI of the diastolic and systolic velocities was obtained from the apical 4-chamber view. A 1.5-mm sample volume was placed at the lateral and septal corner of mitral annulus and lateral tricuspid annulus. Analysis was performed for early diastolic velocity for septal and lateral mitral annulus. The E' parameter was calculated average of these two data.

Left ventricular systolic function was evaluated by myocardial systolic velocity for mitral lateral (Lateral-S) and septal (Septal-S), and the diastolic function by mitral inflow E/A ratio and E' parameter. In addition, E/E' was calculated as an indicator of left ventricle filling pressure. Right ventricular systolic function was assessed by tricuspid myocardial systolic velocity (Tricuspid-S).

Statistical analysis

All of the statistical analysis was performed by SPSS for Windows, version 26 (SPSS Inc., Chicago, Illinois, USA). The numeric variables were summarized using descriptive statistics: mean, median, minimum, maximum, and so forth. Numbers and percentages were used for categorical variables. The suitability of numerical variables as normal distribution was examined using visual (histogram and probability graphs) and analytical methods (Kolmogorov–Smirnov/Shapiro–Wilk tests). The chi-squared test was performed for categorical variables when the condition was met in pairwise comparisons (expected value of >5); if not, Fisher's exact test was used for ordinal variables. The Mann–Whitney *U*-test was used when the normal distribution condition was not met in pairwise group comparisons for numerical variables. Correlation coefficient and *P*-value were given using Spearman's correlation analysis to show the relationship with the variables. Analysis of covariance (ANCOVA) was used for multivariate tests. Statistical significance level was accepted as $P < 0.05$.

RESULTS

Thirty-two insomnia patients (mean age 73.7 ± 8.6 years) and 30 controls (mean age 72.7 ± 7.1 years) were included in our study. The ISI and ESS scores of the insomnia group were higher than the control group ($P < 0.001$). In the insomnia group, 12 (37.5%) patients had moderate insomnia, while 20 (62.5%) patients were severe. Also, while the number of patients with EDS in the insomnia group was 12 (37.5%), there were none in the control group. There was one patient with a long QTc value in the insomnia group, but none in the control group.

The clinical and biochemical parameters of the study population are given In Table 1. Both groups were similar in terms of age, gender, prevalence of diabetes mellitus, hypertension and smoking, BMI, haematocrit, fasting glucose, total cholesterol, LDL-cholesterol, HDL- cholesterol, serum creatinine, ALT, AST, serum sodium, and serum uric acid levels. Serum triglycerides and potassium values were higher in the insomnia group, while serum albumin

Table 1 Baseline clinical and biochemical parameters of all patients

Variables	Insomnia patients ($n = 32$)		Controls ($n = 30$)		P
	Mean ± Std.	Median (min–max)	Mean ± Std.	Median (min–max)	
Age (years)	73.69 ± 8.57	71.0 (65–93)	72.73 ± 7.13	71.5 (64–92)	0.805[†]
Sex (Female) *n* (%)	19 (59.4%)		18 (60%)		0.960[‡]
ISI, *n*	22.56 ± 5.05	23.5 (15–28)	1.93 ± 2.33	1 (0–7)	<0.001
ESS, *n*	7.84 ± 4.11	8 (0–14)	2.43 ± 2.67	2 (0–9)	<0.001
BMI (kg/m^2)	28.40 ± 4.19	28.76 (21–35)	26.78 ± 3.40	26.89 (20–35)	0.111
Diabetes mellitus, *n* (%)	6 (18.8%)		5 (16.7%)		0.830[‡]
Hypertension, *n* (%)	15 (46.9%)		14 (46.7%)		0.987[‡]
Smoking, *n* (%)	4 (12.5%)		4 (13.3%)		1.000[§]
Haemoglobin (g/dl)	13.42 ± 2.55	13.0.5 (10.2–15.1)	13.31 ± 1.85	13.6 (11.1–15.6)	0.872
Fasting glucose (mg/dl)	107.59 ± 20.58	99 (82–148)	100.93 ± 16.36	98.5 (75–169)	0.472
Total cholesterol (mg/dl)	237.28 ± 33.63	241.5 (161–303)	227.33 ± 37.95	227.5 (128–320)	0.251
LDL-cholesterol (mg/dl)	141.38 ± 35.53	138.5 (73–221)	134.80 ± 29.51	131.5 (53–188)	0.477
HDL-cholesterol (mg/dl)	49.66 ± 12.42	49 (32–90)	54.10 ± 12.70	51.5 (33–97)	0.114
Triglycerides (mg/dl)	212.94 ± 103.08	197.5 (52–390)	151.83 ± 63.43	147.5 (48–290)	0.021
AST (U/l)	20.00 ± 6.90	20 (10–51)	20.13 ± 5.68	19 (14–41)	0.871
ALT (U/l)	17.38 ± 4.38	17 (6–31)	18.73 ± 7.38	17.5 (9–46)	0.498
Serum creatinine (mg/dl)	0.84 ± 0.19	0.79 (0.40–1.30)	0.86 ± 0.22	0.84 (057–1.50)	0.983
Sodium (mmol/l)	140.00 ± 3.08	140 (130–145)	140.27 ± 1.80	140 (135–145)	0.903
Potassium (mmol/l)	4.51 ± 0.38	4.5 (3.9–5.6)	4.26 ± 0.43	4.2 (3–5.2)	0.023
Albumin (g/dl)	4.18 ± 0.34	4.2 (3.4–5)	4.33 ± 0.27	4.4 (3.5–5)	0.020
Uric acid (mg/dl)	4.74 ± 1.14	5 (2.76–7.70)	5.14 ± 1.22	4.85 (2.7–8.1)	0.507

[†] Mann–Whitney *U*-test. [‡] Pearson's chi-squared test. [§] Fisher's exact test. Abbreviations: ALT, alanine transaminase; AST, aspartate aminotransferase; BMI, body mass index; ESS, Epworth Sleepiness Scale; HDL, high-density lipoprotein; ISI, Insomnia Severity Index; LDL, low-density lipoprotein.

levels were lower than in the control group. The major structural echocardiographic parameters of all the patients are presented in Table 2. There was no difference between the insomnia and control groups in terms of left ventricular wall thickness, left ventricular diameters, and left atrium diameters ($P > 0.05$). The left ventricular ejection fraction was similar in both groups ($P = 0.617$).

The diastolic function, tissue Doppler, and electrocardiographic parameters are listed in Table 3. There was no difference between the insomnia and control groups in terms of mitral inflow parameters such as E, A, and the E/A ratio. E' on the TDI values was lower in the insomnia patients than in the controls (5.99 ± 1.59 vs. 6.88 ± 0.97, $P = 0.053$). The indicator of LV filling pressure (E/E') was higher in insomnia patients than in the controls (11.97 ± 4.46 vs. 8.91 ± 1.36, $P = 0.002$). Among the systolic function parameters, Lateral-S (7.41 ± 1.92 vs. 9.37 ± 1.83, $P < 0.001$), Septal-S (6.69 ± 1.40 vs. 8.10 ± 1.30, $P = 0.001$), and Tricuspid-S (12.25 ± 2.00 vs. 14.37 ± 3.13, $P = 0.004$) values were lower in the insomnia group compared to the control group. When the effect of ANCOVA and ESS was neutralised between the groups, the significant difference in Lateral-S disappeared, while those of the E', E/E', Septal-S, and Tricuspid-S values continued. Heart rate and QTc values were higher in the insomnia group than in the control group (76.47 ± 7.18 vs. 71.03 ± 10.95, $P = 0.001$, and 413.72 ± 28.24 vs. 394.67 ± 24.47, $P = 0.015$, respectively). When the effect of ANCOVA and ESS was neutralized between the groups, the significant differences in HR and QTc disappeared ($P = 0.313$, $P = 0.173$).

Spearman's correlation analysis including ISI, ESS, E', E/E', Septal-S, Tricuspid-S, HR, QTc, serum albumin, triglycerides, and potassium levels was performed in insomnia patients, which showed a moderately strong negative correlation between ISI and Lateral-S ($r = -0.64$, $P < 0.001$), Septal-S ($r = -0.66$, $P < 0.001$), and E' ($r = -0.43$, $P = 0.014$) Fig. 1. Furthermore, there was a mild negative corelation between ESS and Lateral-S ($r = -0.38$, $P = 0.034$).

DISCUSSION

In our study, we demonstrated that the biventricular systolic and diastolic functions were impaired in the insomnia patients. When insomnia and EDS coexist, we also determined detrimental electrophysiological changes such as increased heart rate and prolonged QTc.

In our study, age, BMI, smoking, prevalence of hypertension, and diabetes mellitus, which are the

Table 2 Structural echocardiographic parameters of all patients

Variables	Insomnia patients ($n = 32$)		Controls ($n = 30$)		
	Mean ± Std.	Median (min–max)	Mean ± Std.	Median (min–max)	$P^{\dagger}$
IVS (mm)	10.75 ± 1.85	10 (9–18)	10.23 ± 0.86	10 (8–13)	0.567
PW (mm)	9.09 ± 1.80	9 (7–16)	8.93 ± 0.78	9 (7–10)	0,506
LVESD (mm)	26.66 ± 2.50	27 (22–33)	27.00 ± 3.80	27 (21–35)	0.859
LVEDD (mm)	43.81 ± 2.49	44 (38–48)	44.23 ± 3.69	45 (39–52)	0.681
EF (%)	65.38 ± 2.31	65 (60–70)	65.70 ± 1.49	66 (63–72)	0.617
LA (mm)	33.50 ± 3.11	33 (29–44)	32.10 ± 3.17	31.5 (26–38)	0.145

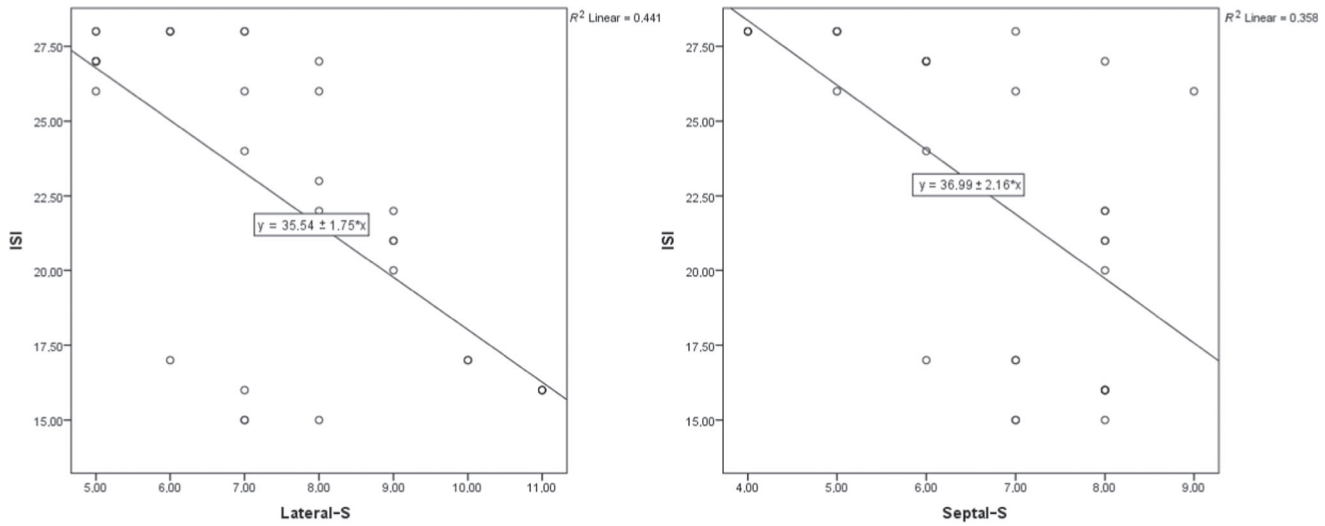
† Mann–Whitney *U*-test. Abbreviations: EF, ejection fraction; IVS, interventricular septum diastolic thickness; LVESD, left ventricular end-systolic diameter; LVEDD, left ventricular end-diastolic diameter; LA, left atrial systolic diameter; PW, posterior wall diastolic thickness.

main confounding factors for left ventricular systolic and diastolic functions, were similar between patients with and without insomnia. However, there was no difference between the groups in terms of left ventricular wall thicknesses and diameters, suggesting that insomnia in older patients impairs myocardial functions by accelerating catabolic processes. There is compelling evidence that even short-term reductions in sleep duration or quality may have detrimental effects on glucose regulation, inhibiting the secretion of anabolic hormones (growth hormone,[18] prolactin,[19] testosterone[20]); changing the amount and timing of catabolic hormones glucocorticoids,[6] catecholamines[21]; changing food regulation (food preferences,[22] timing of food consumption,[23] quantity of food); and having effects on the immune system. Zhang *et al*. reported that impaired sleep quality was correlated with higher 24 h urinary catecholamine levels in healthy subjects.[24] Several experimental studies demonstrated that sleep fragmentation increases sympathetic nervous activity, adrenocortical activity, and blood pressure.[25–27] Another study showed that sleep restriction increased insulin resistance.[25] An animal study indicated that the presence of insulin resistance altered the cardiac structure and contractile function at the level of the myocyte.[28] In our study, the high heart rate and triglyceride values in the insomnia group suggested that the toxic effects of increased sympathetic activation and increased insulin resistance may contribute to the deterioration of myocardial functions. Although Lee *et al*. reported increased left ventricular hypertrophy in connection with impaired sleep quality population in their study, they could not find any difference in terms of diastolic functions.[29] The most important difference between the aforementioned study and ours was that our average age of the patients participating in the study was much higher. Insomnia may cause left ventricular hypertrophy by mostly increasing blood pressure at young ages, even though it may impair systolic and diastolic functions by accelerating catabolic processes in geriatric populations. In addition, the fact that the left ventricular wall thicknesses were within normal reference values in both groups and there was no difference between the groups in our study suggested that

Table 3 Diastolic function, tissue Doppler and electrocardiographic parameters

Variables	Insomnia patients ($n = 32$)		Controls ($n = 30$)			
	Mean ± Std.	Median (min–max)	Mean ± Std.	Median (min–max)	$P*$	$P**$
E (cm/s)	66.69 ± 14.32	65 (48–116)	61.37 ± 13.33	57.5 (40–92)	0.095	
A (cm/s)	85.41 ± 15.62	81.5 (70–130)	81.07 ± 19.96	81.5 (40–118)	0.553	
E/A ratio	0.79 ± 0.16	0.79 (0.56–1.38)	0.81 ± 0.29	0.75 (0.45–1.63)	0.337	
E' (cm/s)	5.99 ± 1.59	6 (3–8)	6.88 ± 0.97	7 (4.5–9)	0.053	0.007
E/E' ratio	11.97 ± 4.46	9.85 (8.13–25.30)	8.91 ± 1.36	9.05 (6.66–11.66)	0.002	<0.001
Lateral-S (cm/s)	7.41 ± 1.92	7 (5–11)	9.37 ± 1.83	9.5 (7–15)	<0.001	0.061
Septal-S (cm/s)	6.69 ± 1.40	7 (4–9)	8.10 ± 1.30	8 (6–11)	0.001	0.003
Tricuspid-S (cm/s)	12.25 ± 2.00	12 (8–17)	14.37 ± 3.13	14 (10–24)	0.004	0.011
Heartrate beats/min	76.47 ± 7.18	76 (62–97)	71.03 ± 10.95	69.5 (55–100)	0.001	0.313
QTc (ms)	413.72 ± 28.24	412.5 (376–496)	394.67 ± 24.47	395 (341–447)	0.015	0.173

* Mann–Whitney *U*-test. ** Analysis of covariance (ANCOVA; covariate variable: ESS). Abbreviations: A, peak velocity of late diastolic filling; E, peak velocity of early diastolic filling; E', average of early diastolic velocity for septal and lateral mitral annulus; Lateral-S, myocardial systolic velocity of the mitral lateral anulus; QTc, QT interval corrected for heart rate; Septal-S, myocardial systolic velocity of the mitral septal anulus; Tricuspid-S, tricuspid myocardial systolic velocity.



		Lateral-S	Septal-S
ISI	***r***	–0.636	–0.662
	p	<0.001	<0.001
	N	32	32

Figure 1 Correlations between the Insomnia Severity Index (ISI) and myocardial systolic velocity of the lateral (Lateral-S) and septal (Septal-S) mitral anulus in insomnia patients.

the effect of blood pressure on myocardial functions was not important. Another important finding was that the E/E' value was higher in the insomnia group, although it was within normal ranges. Increased left ventricular filling pressures may cause hypertrophy and enlargement in the left atrium, making patients prone to rhythm disorders such as atrial fibrillation over time.[30] Christensen *et al*. reported that sleep disorder was consistently associated with increases in the incidence of atrial fibrillation, which resulted from impaired autonomic functions due to poor sleep quality.[31] The fact that the high E/E' values in insomnia patients were revealed by our study will contribute to the pathophysiological explanation of the increase in the incidence of atrial fibrillation in the insomnia patients.

The QT interval represents the time for both ventricular myocardial depolarization and repolarization.[32] Abnormal ventricular repolarization is the usual cause of QT interval prolongation.[33] In patients with myocardial infarction, malignant arrhythmias[34] and sudden cardiac death[35] are frequently observed by prolongation of the heart rate's corrected QT (QTc) interval. Furthermore, QTc prolongation was shown to be an independent risk factor for cardiovascular disease and mortality in the general population.[36] Another important finding in our study is that the QTc value is higher in the presence of insomnia and EDS, although it is within normal limits. Shi *et al*. have shown in a recent study that sleep disturbance contributes synergistically to an increase in QT duration.[37] In a recent study, Lee *et al*. have revealed a significant relationship between high triglyceride levels and QTc.[38] Furthermore, the previous studies demonstrated that arrhythmias are related to proinflammatory cytokines such as interleukin 6 and C-reactive protein by the modulation of ion-channel function[39,40] and aggravation of the sympathetic system.[39] High triglyceride and heart rate values in the insomnia group suggested that the detrimental effect on myocardial ion channels caused by increased sympathetic activation and insulin resistance may cause the prolongation. Also, the disappearance of statistical significance in the heart rate and QTc values when ESS adjustment is performed by ANCOVA supports the view that sympathetic activation may be more increased in patients in whom insomnia and EDS coexist. Another important finding of our study is that serum triglyceride levels were higher in the insomnia group. In their study, Bos *et al*. found that serum and hepatic triglyceride levels were

higher in patients with short sleep duration, but the significance disappeared after adjusting for BMI and sleep apnea.[38,41] Although there was no difference between the groups in the BMI values, and patients with sleep apnea were not included in our study, serum triglyceride levels were higher in the insomnia group. The possible mechanism, as shown in a previous study, may be due to the fact that shorter sleep times alter serum leptin and ghrelin levels, and to patients' excessive tendency to consume carbohydrate-rich foods and increased insulin resistance.[11] For the same reason, it has been shown that the nutritional status of older patients with insomnia is deteriorated and that malnutrition is higher in these patients, which may explain why the serum albumin levels of those in the insomnia group were lower than those without insomnia.[42]

Our study's strengths include the evaluation of both insomnia and EDS together, and the simultaneous performance of a detailed cardiological evaluation. However, our study also has some limitations. First, self-report scales were used for assessing insomnia and EDS; hence, future studies should consider using objective measures of sleep to diagnose insomnia, such as actigraphy. Second, if the number of our patients had been sufficient and if we could have created three groups as insomnia, insomnia and EDS, and control, we could have more clearly investigated the effect of EDS on the parameters we examined. Third, if we could have quantified the insulin resistance status of the patients by looking at the Homeostatic Model Assessment (HOMA) values, we could have more clearly revealed the effect of insulin resistance in the deterioration of myocardial functions.

In conclusion, insomnia may result in the deterioration of biventricular systolic and diastolic functions in elderly patients. When EDS is added to insomnia, harmful electrophysiological changes such as increased heart rate and prolonged QTc may also occur. Accordingly, an elderly individual with insomnia and EDS may be predisposed to cardiac events. Therefore, sleep disorders in the elderly should be questioned and treated not only by geriatricians but also by clinicians, especially cardiologists. Thus, future cardiac problems may be prevented. Future studies are needed to investigate the role of insomnia and EDS more clearly, especially in older patients with underlying structural heart disease.

DISCLOSURE

The authors declare that they have no conflicts of interest in the research.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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