ORIGINAL RESEARCH

**DOI:** 10.4274/ejgg.galenos.2025.2025-6-7
Eur J Geriatr Gerontol

# Sarcopenia and Arrhythmia Risk: An Electrocardiographic Analysis in Older Adults

**Onur Erdoğan[1], Tuğba Erdoğan[2], Serdar Özkök[2], Zeynep Fetullahoğlu[2], Duygu Erbaş Saçar[3], Deniz Seyithanoğlu[2], Ömer Kümet[4], Mehmet Akif Karan[2], Gülistan Bahat[2]**

*[1]University of Health Sciences Türkiye, Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital, Clinic of Cardiology, İstanbul, Türkiye*
*[2]İstanbul University, İstanbul Faculty of Medicine, Department of Internal Medicine, Division of Geriatrics, İstanbul, Türkiye*
*[3]Kartal Koşuyolu High Specialty Education and Research Hospital, Clinic of Internal Medicine, Division of Geriatrics, İstanbul, Türkiye*
*[4]University of Health Sciences Türkiye, Van Education and Research Hospital, Clinic of Cardiology, Van, Türkiye*

## Abstract

**Objective:** Sarcopenia and cardiac arrhythmias are common in older adults and may have similar processes such as inflammation, fibrosis, mitochondrial dysfunction, and oxidative stress. These processes can cause myocardial remodeling and electrical instability. This study was designed to examine the link between sarcopenia and electrocardiographic (ECG) markers of arrhythmia.

**Materials and Methods:** In this cross-sectional, retrospective study, 283 older adults living in the community aged 60 years or older who underwent comprehensive geriatric assessment were included. Sarcopenia was defined per the European Working Group on Sarcopenia in Older People 2 criteria, using handgrip strength with population-specific thresholds (men <35 kg, women <20 kg), and Skeletal Muscle Mass Index (SMMI) [SMMI = SMM/BMI (body mass index)]. Standard 12-lead ECGs were analyzed for arrhythmic patterns and conduction abnormalities, including P-wave dispersion (PWD), corrected QT interval (QTc), Tp-e/QTc ratio, Tp-e interval, fragmented QRS, frontal QRST angle, first-degree atrioventricular block, bundle branch block, premature ventricular and atrial contractions, and atrial fibrillation (AF). Multivariate logistic regression identified independent associations.

**Results:** Sarcopenia was present in 35.7% of participants and was associated with older age, female sex, obesity, frailty, and functional impairment. ECG abnormalities were more frequent in individuals with sarcopenia, including AF (p=0.038), fragmented QRS (p=0.032), and increased PWD (p=0.010). In multivariate analysis, fragmented QRS [Odds Ratio (OR): 2.464, 95% confidence interval (CI): 1.068-5.683, p=0.035], obesity (OR: 2.030), frailty (OR: 1.970), and age (OR: 1.104) were independently associated with sarcopenia. When PWD replaced fragmented QRS, it also showed a significant association (OR: 1.018; 95% CI: 1.001-1.037; p=0.042).

**Conclusion:** Sarcopenia is independently associated with ECG abnormalities suggestive of atrial and ventricular electrical remodeling, especially fragmented QRS and PWD.

**Keywords:** Sarcopenia, arrythmia, electrocardiography, atrial fibrillation

## Introduction

Cardiac arrhythmia prevalence rises significantly with ageing, contributing to elevated morbidity and mortality in older adults and placing a growing burden on healthcare systems (1-3). Arrhythmias can range from benign to life-threatening, with atrial fibrillation (AF) being the most commonly observed in the older population. The presence of arrhythmias is linked to increased incidence of heart failure, cerebrovascular events, and sudden cardiac mortality, all of which result in notable strain on healthcare resources (4). Before clinical symptoms of arrhythmia appear, the heart often undergoes subtle structural and electrical remodeling, which can disrupt its normal electrical conduction

**Address for Correspondence:** Tuğba Erdoğan, Assoc, Prof, İstanbul University, İstanbul Faculty of Medicine, Department of Internal Medicine, Division of Geriatrics, İstanbul, Türkiye
**E-mail:** tubaobekli@hotmail.com **ORCID:** orcid.org/0000-0001-8690-0189
**Received:** 21.06.2025 **Accepted:** 17.07.2025 **Epub:** 29.09.2025 **Publication Date:** xxxxxxxxxxxxxxx

**Cite this article as:** Erdoğan O, Erdoğan T, Özkök S, Fetullahoğlu Z, Erbaş Saçar D, Seyithanoğlu D, Kümet Ö, Karan MA, Bahat G. Sarcopenia and Arrhythmia Risk: An Electrocardiographic Analysis in Older Adults. Eur J Geriatr Gerontol. [Epub Ahead of Print]

system. This subclinical electrical dysfunction may remain undetected for years before manifesting as an arrhythmia. Timely identification of patients at high-risk is critical for early intervention and improved outcomes. Several clinical risk factors predisposing for cardiac arrhythmias include hypertension (5), diabetes mellitus (6), coronary artery disease (7), valvular heart disorders (8), and chronic kidney disease (9). However, the underlying molecular mechanisms predisposing older adults to arrhythmogenesis remain incompletely understood.

Sarcopenia, a progressive and generalised skeletal muscle disorder, has been increasingly recognized as a potential contributor to adverse outcomes in older individuals, including falls, disability, and mortality (10). Multiple interconnected biological mechanisms have been implicated in sarcopenia, such as chronic systemic inflammation, mitochondrial dysfunction, oxidative damage, impaired regenerative capacity of satellite cells, increased apoptotic activity, endocrine alterations, and physical inactivity (11,12). These processes can also adversely affect cardiac structure and function. Notably, mitochondrial dysfunction and chronic inflammation are implicated in cardiac fibrosis and electrophysiological remodeling-both recognized substrates for arrhythmogenesis (13,14).

Given the shared biological mechanisms that underlie both sarcopenia and cardiac electrical remodeling, it is plausible that sarcopenia may contribute to the development of arrhythmogenic substrates. We hypothesized that this constellation of factors may be associated with specific electrocardiographic (ECG) abnormalities reflecting atrial and ventricular conduction disturbances. This study aimed to investigate whether sarcopenia serves as an independent factor associated with arrhythmias and arrhythmia-related ECG parameters.

## Materials and Methods

### Population and Setting

A retrospective, cross-sectional design was adopted for the study, which included community dwelling older adults aged 60 and over, who were evaluated at the geriatrics outpatient clinic at our university hospital and underwent a comprehensive geriatric assessment between January 2022-April 2024. Only those patients who demonstrated adequate cognitive function to provide accurate information and who voluntarily agreed to participate via informed consent were enrolled in the study. Demographic information and clinical data were obtained via in-person interviews. We excluded patients who had conditions that could interfere with accurate assessments using bioelectrical impedance analysis (BIA), such as the presence of edema or an implantable pacemaker, as well as conditions affecting handgrip strength (HGS) assessments, such as osteoarthritis. In addition, patients with known heart failure, ischemic ST-T changes on ECG, or significant valvular heart disease were excluded. These conditions were excluded as they could lead to ECG alterations and potentially confound the interpretation of ECG findings. Each participant gave written informed consent for the research. The study was approved by the İstanbul University, İstanbul Faculty of Medicine Ethics Committee (protocol number: 2024/2147, date: 15.11.2024) and was done in accordance with the principles outlined in the Helsinki Declaration.

### Assessment of Sarcopenia

The presence of sarcopenia was diagnosed per the guidelines published by the European Working Group on Sarcopenia in Older People 2 (EWGSOP2). Confirmed sarcopenia was diagnosed with concurrent presence of low muscle mass (LMM) and low muscle strength in the absence of secondary causes (e.g., stroke, vascular diseases, or osteoarthritis) (10).

Muscle strength was assessed through HGS test using a hydraulic hand dynamometer (Jamar®), following an established protocol by a geriatric physiotherapist from the same healthcare profession had previously been certified in these assessments (15). Participants were requested to perform the HGS test 3 times for each hand by squeezing the hand dynamometer with maximum strength. The measurements were performed while the patients were seated, with the elbow flexed at 90 degrees and the wrist positioned neutrally. The highest grip strength recorded was considered the final measurement. We applied population-specific cut-off values for HGS, which were set at 35 kg for males and 20 kg for females (16).

BIA with the Tanita BC-532 model was employed to assess body composition, following an 8-hour fasting period. Fat-free mass (FFM) was derived from BIA, and skeletal muscle mass (SMM) was subsequently calculated using the formula: SMM (kg) = FFM × 0.566 (17).

Body mass index (BMI) was calculated as weight (in kg) divided by height squared (in $m^2$). SMM Index (SMMI) was derived from the total SMM divided by BMI. Although EWGSOP2 advises employing standard cut-off values for appendicular SMM, it does not define specific limits for total SMMI. Instead, they suggest employing population-specific cut-offs when available (18). Following this guidance, the LMM thresholds were determined using mean values minus two standard deviations from a reference group of younger individuals, adhering to EWGSOP recommendations. The resulting thresholds for SMMI were found to be 0.823 kg/BMI and 1.049 kg/BMI for men and women, respectively (19).

### Clinical Data

Frailty was determined with the Frail scale, comprising five items evaluating Fatigue, Resistance, Ambulation, Illness, and Loss of Weight (20). The total score ranges from 0 to 5, with scores 3 to 5

designating frailty, scores 1 to 2 designating pre-frailty, and a score of 0 representing a robust condition.

The six-item Katz Activities of Daily Living (ADL) scale (score range: 0-6) (21) and the eight-item Lawton Instrumental ADL (IADL) scale (score range: 0-8) (22) were used for assessment of fuctionality. For each item, 1 point was assigned for independence and 0 for dependence. Participants scoring 6 on the ADL and 8 on the IADL scales were considered fully independent; those with scores less than 6 on ADL and/or less than 8 on IADL were classified as having impaired functioning (23).

Nutritional evaluation was done with the Short Form of Mini Nutritional Assessment (MNA-SF), which categorizes scores as follows: 0-7 reflects malnutrition, 8-11 signifies a risk of malnutrition, while score greatrer than 11 denotes normal nutritional status. A score of ≤11 on the MNA-SF was used as the criterion to define undernutrition (24,25).

### Electrocardiogram

A 12-lead surface ECG (Schiller Cardiovit AT-102) with a recording speed of 25 mm/s and a voltage amplitude of 10 mm/mV was obtained while the patient was lying supine. All measurements were digitally scanned and magnified 400 times using Adobe Photoshop to reduce potential errors. Two seperate cardiologists who were blinded to the patients' clinical information performed the measurements. Standard 12-lead electrocardiogram was obtained to evaluate QT, corrected QT intervals (QTc), fragmented QRS, P-Wave dispersion (PWD), Tp-Tend interval, Tp-Tend/QTC ratio, and the frontal QRS-T angle. Complete bundle branch block (BBB) patterns, first-degree atrioventricular (AV) block, premature ventricular (PVC) and atrial contractions (PAC), and AF were also evaluated and documented as part of the ECG analysis. The QT interval was measured as the distance from the start of the QRS complex to the end of the T wave from leads of V1-V6, DII, DIII, and aVF with an electronic caliper. Additionally, Bazett's formula ($QTc = QT/\sqrt{RR}$) was applied to correct the QT interval for heart rate (26).

Fragmented QRS refers to the appearance of an R' wave or the R or S wave notching in the absence of a typical BBB (27). PWD refers to the difference between the longest (P max) and shortest (P min) P-wave durations observed on an ECG (28). In patients with AF, P wave parameters, including PWD, were not assessed due to the absence of consistent atrial activity.The Tp-e interval is defined as the duration from the peak of the T wave to its endpoint (29). This measurement was taken from the precordial leads, with an average calculated from at least three readings for each lead and measurement. The ratio of Tp-Tend to QTc was calculated (30).

The frontal QRST angle was determined by taking the absolute difference between the QRS and T axes in the frontal plane. If this difference exceeded 180°, the QRST angle was adjusted to reflect the smallest angle, calculated as the absolute difference subtracted by 360° (31).

Interobserver variability was analyzed to determine the reliability of ECG measurements.

### Statistics

For all statistical analyses, IBM SPSS Statistics (version 21.0) was used. P-values under 0.05 were accepted as statistically significant. All variable entries were cross-checked by a second researcher for accuracy. Both the Kolmogorov-Smirnov test and visual examination of histograms were done to evaluate the normality for continuous variables. Normally distributed continuous variables were reported in terms of mean and standard deviation, while skew- distributed continuous variables were documented as median (interquartile range). Categorical variables were reported in counts and percentages. Group comparisons between patients with and without sarcopenia were conducted using the independent samples t-test for normally distributed continuous variables, and the Mann-Whitney U test for non-normally distributed variables. For the analysis of categorical data, either chi-square or Fisher's exact test was used depending on the expected cell frequencies. To identify independent predictors of sarcopenia, multivariate logistic regression analyses were performed. The presence of multicollinearity among the independent variables was evaluated through correlation analyses, including Pearson, Spearman, and Kendall's tau-b. Independent variables with correlation coefficients greater than 0.7, indicating multicollinearity, were not included in the same regression analysis. Findings were expressed in terms of odds ratios (ORs) along with their 95% confidence intervals (CIs). The intraclass correlation coefficient (ICC) was used to assess interobserver reliability, based on a two-way mixed-effects model with absolute agreement. ECG measurements from a randomly selected subgroup of 30 participants were independently evaluated by two blinded cardiologists. An ICC ≥0.75 was considered to indicate good agreement.

## Results

A total of 283 older adults living in the community (mean age: 73.55±6.68 years; 67.5% female) were included in the study. Sarcopenia was diagnosed in 101 participants (35.7%). Patients with sarcopenia were significantly older (75.93±6.30 vs. 72.23±6.40 years,p<0.001) and had a higher prevalence of frailty (41.4% vs. 16.2%, p < 0.001) and obesity (47.5% vs. 34.6%, p=0.042). Patients with sarcopenia were more likely to be female (79.2% vs. 61.0%, p=0.002), while current smoking was

more prevalent among individuals without sarcopenia (40.1% vs. 24.2%, p=0.009). Patient diagnosed with sarcopenia showed significantly low HGS and SMMI (both p<0.001) (Table1).

In terms of ECG parameters, the sarcopenia group had a significantly higher prevalence of AF (p=0.038) and fragmented QRS complexes (p=0.032). PWD, an established marker of atrial electrical remodeling, was also significantly increased in participants with sarcopenia (p=0.010). There were no statistically significant differences between groups in terms of QTc interval, frontal QRS-T angle, Tp-e interval, Tp-Te /QT, or Tp-e/QTc ratio. Conduction abnormalities were also assessed. BBB was detected in 16 participants (4 with left BBB and 12 with right BBB), and first-degree AV block was present in 33 participants. Additionally, extrasystoles were identified in 18 individuals (8 atrial and 10 ventricular). Although conduction abnormalities-including BBB, first-degree AV block, and PVC-were more frequent in patients with sarcopenia, they were not statistically associated with sarcopenia (Table 2).

In all multivariate logistic regression models, sarcopenia served as the dependent variable, while age, gender, obesity, frailty, and impaired instrumental IADL were considered as independent variables. In the first model, fragmented QRS was also included and was independently associated with sarcopenia (OR: 2.464; 95% CI: 1.068-5.683; p=0.035), along with age (OR: 1.104; 95% CI: 1.055-1.156; p<0.001), obesity (OR: 2.030; 95% CI: 1.125-3.661; p=0.019), and frailty (OR: 2.464; 95% CI: 1.068-5.683; p=0.035) (Table 3). In the second model, fragmented QRS was replaced by PWD, and PWD was associated with sarcopenia (OR: 1.018; 95% CI: 1.001-1.037; p=0.042), while age, obesity, and frailty remained significant (Table 3). In the third model, fragmented QRS was replaced by AF, but AF was not associated with sarcopenia (OR: 3.736; 95% CI: 0.823-16.967; p=0.088) (Table 3).

Interobserver analysis demonstrated high consistency between the two independent cardiologists. The ICCs for all ECG parameters ranged from 0.86 to 0.94, reflecting good to excellent interobserver agreement.

**Table 1. Demographic and clinical characteristics of the patients**

| Variable | All patients (n=283) | No sarcopenia (n=182) | Sarcopenia (n=101) | p-value |
|---|---|---|---|---|
| **Sex (n, %)** | | | | **0.002** |
| Male | 92 (32.5%) | 71 (39.0%) | 21 (20.8%) | |
| Female | 191 (67.5%) | 111 (61.0%) | 80 (79.2%) | |
| Age (mean ± SD) | 73.55±6.68 | 72.23±6.40 | 75.93±6.30 | <0.001 |
| Weight (mean ± SD) | 72.63±13.52 | 72.84±14.58 | 72.26±11.44 | 0.665 |
| BMI (mean ± SD) | 29.17±5.23 | 28.22±5.39 | 30.87±4.46 | <0.001 |
| Obesity (n, %) | 111 (39.2%) | 63 (34.6%) | 48 (47.5%) | 0.042 |
| Atrial fibrillation (n, %) | 10 (3.5%) | 3 (1.6%) | 7 (6.9%) | 0.038 |
| Ischemic heart disease (n, %) | 61 (21.6%) | 41 (23.6%) | 20 (21.3%) | 0.670 |
| Diabetes mellitus (n, %) | 91 (32.2%) | 58 (33.3%) | 33 (35.1%) | 0.770 |
| Hypertension (n, %) | 194 (68.6%) | 126 (72.4%) | 68 (72.3%) | 0.990 |
| Impaired ADL (n, %) | 46 (16.3%) | 23 (12.6%) | 23 (22.8%) | 0.027 |
| Impaired IADL (n, %) | 87 (30.7%) | 43 (23.6%) | 44 (43.6%) | <0.001 |
| **Frailty (n, %)** | | | | <0.001 |
| Frail | 70 (25.2%) | 29 (16.2%) | 41 (41.4%) | |
| Prefrail | 130 (46.8%) | 87 (48.6%) | 43 (43.4%) | |
| Robust | 78 (28.1%) | 63 (35.2%) | 15 (15.2%) | |
| Undernutrition (n, %) | 94 (33.5%) | 61 (33.7%) | 33 (33.0%) | 0.905 |
| Smoking (n, %) | 92 (32.5%) | 69 (40.1%) | 23 (24.2%) | 0.009 |
| Alcohol status (n, %) | 20 (7.1%) | 16 (9.5%) | 4 (4.2%) | 0.121 |
| HGS (mean ± SD) | 23.66±8.25 | 26.55±8.00 | 18.44±5.77 | <0.001 |
| Low HGS (n, %) | 173 (61.1%) | 72 (39.6%) | 101 (100.0%) | <0.001 |
| SMMI (BMI) (mean ± SD) | 0.88±0.18 | 0.93±0.19 | 0.77±0.12 | <0.001 |
| Low SMMI (BMI) (n, %) | 163 | 62 (34.1%) | 101 (100.0%) | <0.001 |

*5 missing variable
SD: Standard deviation, BMI: Body mass index, HGS: Hand grip strength, SMMI: Skeletal muscle mass index; ADL: Activities of daily living, IADL: Instrumental activities of daily living

**Table 2. ECG parameters according to sarcopenia**

| Variable | All patients | No sarcopenia | Sarcopenia | p-value |
|---|---|---|---|---|
| AF (n, %) | 10 (3.5%) | 3 (1.6%) | 7 (6.9%) | 0.038 |
| 1° AV block (n, %) | 33 (11.7%) | 18 (9.9%) | 15 (14.9%) | 0.213 |
| Complete bundle branch block (n, %) | 16 (5.7%) | 9 (4.9%) | 7 (6.9%) | 0.488 |
| PVC (n, %) | 10 (3.5%) | 5 (2.7%) | 5 (5.0%) | 0.336 |
| PAC (n, %) | 8 (2.8%) | 6 (3.3%) | 2 (2.0%) | 0.716 |
| Pmax (ms) (mean ± SD) | 104.88±18.81 | 103.35 (±19.04) | 107.78 (±18.13) | 0.062 |
| Pmin (ms) (mean ± SD) | 56.26±14.79 | 56.98±14.14 | 54.97 (±15.96) | 0.306 |
| P dispersion (ms) (mean ± SD) | 48.72±16.63 | 46.51±16.52 | 51.92±16.11 | 0.010 |
| QTc (ms) (mean ± SD) | 417.02±24.63 | 416.54±25.04 | 417.91 (±23.97) | 0.655 |
| Frontal QRS–T angle [median (IQR)] | 31 (14-60) | 30 (13-62) | 33 (14-59) | 0.736 |
| Fragmented QRS (n, %) | 32 (11.3%) | 15 (8.2%) | 17 (17.0%) | 0.032 |
| Tp–Te interval (ms) (mean ± SD) | 79.42±18.99 | 79.93±19.24 | 78.52 (±18.62) | 0.558 |
| Tp–Te/QT (mean ± SD) | 0.20±0.04 | 0.2 (±0.05) | 0.2 (±0.05) | 0.673 |
| Tp–Te/QTc (mean ± SD) | 0.19±0.04 | 0.19 (±0.45) | 0.19 (±0.44) | 0.461 |

SD: Standart deviation, AF: Atrial fibrillation, AV: Atrioventricular, PVC: Premature venticular contractions, PAC: Premature Atrial contractions, IQR: Interquartile range, ECG: Electrocardiographic, max: Maximum, min: Minimum

**Table 3. Results of multivariate logistic regression analyses for sarcopenia**

| Variable | Model 1 OR (95% CI) | p-value | Model 2 OR (95% CI) | p-value | Model 3 OR (95% CI) | p-value |
|---|---|---|---|---|---|---|
| Age | 1.104 (1.055-1.156) | **<0.001** | 1.092 (1.042-1.144) | **<0.001** | 1.097 (1.049-1.148) | **<0.001** |
| Sex | 1.719 (0.908-3.252) | 0.096 | 1.507 (0.767-2.961) | 0.234 | 1.751 (0.927-3.305) | 0.084 |
| Obesity | 2.030 (1.125-3.661) | **0.019** | 2.347 (1.265-4.354) | **0.007** | 1.978 (1.103-3.548) | **0.022** |
| Frailty | 1.970 (1.286-3.019) | **0.002** | 1.921 (1.236-2.986) | **0.004** | 2.124 (1.385-3.259) | **0.001** |
| Impaired IADL | 1.267 (0.667-2.408) | 0.470 | 1.331 (0.678-2.612) | 0.406 | 1.100 (0.577-2.099) | 0.771 |
| Fragmented QRS | 2.464 (1.068-5.683) | **0.035** | - | - | - | - |
| P dispersion | - | - | 1.018 (1.001–1.037) | **0.042** | - | - |
| AF | - | - | - | - | 3.736 (0.823–16.967) | 0.088 |

Dependent variable: sarcopenia,
Independent variables: age, gender, obesity, frailty, impaired IADL, Fragnented QRS/P dispersion/AF.
OR: Odds ratio, IADL: Instrumental activities of daily living, AF: Atrial fibrillation, CI: Confidence interval

## Discussion

A significant correlation was observed in this research between sarcopenia and ECG-based markers reflecting arrhythmogenic potential in geriatric patients. Specifically, individuals with sarcopenia exhibited higher rates of fragmented QRS complexes, AF, and increased PWD-thereby indicating subclinical electrical remodeling in both atrial and ventricular myocardium. In addition, fragmented QRS and PWD were independently associated with sarcopenia.

The gradual deterioration of both muscle mass and muscle strength in sarcopenia is increasingly viewed as part of a broader systemic condition, rather than solely a musculoskeletal issue. Growing evidence links sarcopenia to increased cardiovascular risk, such as myocardial infarction, AF, heart failure, and peripheral artery disease (32-35). However, studies on its association with cardiac arrhythmias-particularly atrial and ventricular conduction abnormalities-are still limited. To address this gap, we evaluated several ECG parameters reflecting arrhythmogenic risk in older adults with sarcopenia, including QT/QTc intervals, fragmented QRS complexes, PWD, Tp-e interval, Tp-e/QTc ratio, and frontal QRS-T angle. Among these, fragmented QRS, PWD, and AF were associated with sarcopenia, suggesting myocardial electrical remodeling. Similarly, findings from the Bushehr Elderly Health Program support our results, reporting associations between sarcopenia and major ECG aberrations, like Q-QS waves, left ventricular hypertrophy, BBB, AF/flutter, and major ST-T changes. These findings collectively suggest that sarcopenia may contribute to electrical and structural remodeling of the aging heart, predisposing to arrhythmias (36).

Fragmented QRS indicates heterogeneous depolarization of the ventricular myocardium, which may arise from conditions such as ischemia, fibrosis, or scarring. The presence of fQRS suggests structural abnormalities in the myocardium, often associated with fibrosis, which can disrupt normal electrical conduction and predispose individuals to various arrhythmogenic events (37,38). In our cohort, fQRS was significantly more prevalent among patients with sarcopenia (17.0% vs. 8.2%) and independently associated with sarcopenia (OR: 2.464), supporting the hypothesis that sarcopenia and myocardial remodeling may share common pathophysiological mechanisms. These include low-grade prolonged inflammation, oxidative stress, endocrine alterations and mitochondrial dysfunction, all well-recognized features of sarcopenia that may also contribute to myocardial fibrosis and conduction abnormalities (3,39,40.)

Similarly, increased PWD observed in individuals with sarcopenia may reflect atrial conduction heterogeneity and structural remodeling, which are known precursors of AF (28). Although we observed a higher prevalence of AF in patients with sarcopenia, it was not independently associated with sarcopenia in our analysis-potentially due to the limited number of AF cases in our cohort. Nonetheless, our findings are aligned with those of Tang et al. (40), who reported that both probable and confirmed sarcopenia were associated with a significantly elevated long-term risk of AF, independent of genetic predisposition and cardiovascular risk factors.

Sarcopenia and cardiac arrhythmias, though affecting different organ systems, share several pathophysiological mechanisms that may contribute to their co-occurrence, particularly in older adults. Chronic low-grade inflammation, a hallmark of sarcopenia, has also been implicated in myocardial remodeling and fibrosis (13), which in turn can alter electrical conduction and promote arrhythmogenesis.

Evidence suggests that Interleukin-6 and tumor necrosis factor-α as major inflammatory mediators, are actively involved in promoting apoptosis of cardiomyocytes and structural changes in the extracellular matrix. This inflammation-induced fibrotic process disrupts atrial conduction properties and helps maintain a substrate favorable for arrhythmia development (41).

Furthermore, mitochondrial dysfunction-an established factor in both sarcopenia and cardiac arrhythmias-also compromises myocardial energetics. Mitochondrial dysfunction is strongly linked to electrophysiological and structural abnormalities observed in cardiac arrhythmias. As the primary source of adenosine triphosphate (ATP) production, mitochondria serve a fundamental function in meeting the constant energy demands of cardiac electrical activity. In arrhythmias, the balance between myocardial energy supply and demand becomes disrupted, frequently in association with impaired mitochondrial function, resulting in diminished ATP synthesis and increased production of reactive-oxygen-species. One of the primary sources of excessive reactive oxygen species is mitochondrial oxidative stress. Elevated mitochondrial oxidative stress can interfere with the normal operation of key ion channels-such as potassium, calcium, and sodium channels-as well as ryanodine receptors, thereby increasing susceptibility to cardiac arrhythmias. In parallel, disruptions in ion homeostasis, cell membrane excitability, and myocardial architecture can arise due to pathological alterations in gap junctions and inflammatory signaling pathways. These changes collectively compromise the heart's electrical stability and contribute to arrhythmogenesis (42,43).

Endocrine alterations such as reduced anabolic hormone levels [e.g., testosterone, insulin-like growth factor (IGF-1)] and dysregulated renin-angiotensin-aldosterone signaling may also contribute to both sarcopenia progression and the development of myocardial fibrosis, thus linking muscle degradation to increased arrhythmic vulnerability. Taken together, these

overlapping mechanisms suggest that sarcopenia may not merely coexist with arrhythmias but may actively contribute to their pathophysiology.

Our previous study demonstrated an independent association between sarcopenia and left ventricular diastolic dysfunction (LVDD), suggesting that sarcopenia may contribute to myocardial structural and functional impairment beyond traditional risk factors (44). Given that LVDD is a well-known substrate for atrial remodeling and a significant risk factor for arrhythmias such as AF, it is plausible that the link between sarcopenia and arrhythmias identified in this study may be partially attributable to alterations in diastolic function. These findings collectively highlight the need to further investigate the interconnected pathways linking sarcopenia, myocardial remodeling, and arrhythmogenesis.

Although QTc interval, Tp-e interval, Frontal QRS-T angle, and Tp-e/QTc ratio are documented markers of ventricular repolarization and arrhythmia risks, we did not find significant associations with sarcopenia in our cohort. QTc prolongation and increased Tp-e durations have been linked to life-threatening arrhythmias and sudden cardiac death (45). The Tp-e/QTc ratio similarly indicates arrhythmia risk, especially in those with structural heart disease. The absence of a significant relationship in our study might be attributed to the multifactorial nature of ventricular repolarization, influenced by factors like myocardial integrity, electrolyte balance, autonomic tone, medication use, and subclinical ischemia, all of which were not fully controlled in our retrospective design. Moreover, our relatively stable, community-dwelling older population without overt structural heart disease may have limited variability in repolarization parameters. Detecting subtle changes in QTc or Tp-e may also require larger samples or high-resolution ECGs. While these markers remain clinically relevant, their association with sarcopenia warrants further prospective investigation.

This study is among the first to comprehensively evaluate multiple ECG parameters-including both atrial and ventricular markers of arrhythmic risk-in older adults with and without sarcopenia. The use of EWGSOP2 criteria for sarcopenia diagnosis and standardized ECG measurements performed by blinded cardiologists enhances the methodological rigor. Furthermore, the inclusion of community-dwelling older adults reflects a real-world clinical population, which strengthens the generalizability of our findings. A standardized and accurate evaluation of sarcopenia is fundamental for drawing meaningful conclusions in both observational and interventional studies. In our study, SMM was adjusted by BMI rather than height$^2$, as BMI-based adjustment is considered more appropriate in populations with high adiposity. Height$^2$-adjusted indices may underestimate sarcopenia prevalence in obese individuals by failing to account for body weight. Given the high proportion of obese participants in our cohort and the growing evidence linking BMI-based muscle indices to functional outcomes, we selected this method to better reflect clinically relevant muscle deficits (46).

### Study Limitations

However, certain limitations need to be noted. First, the retrospective and cross-sectional nature of the study limits the drawing of any causal relationship between sarcopenia and ECG abnormalities. Second, our sample size, although adequate for detecting differences in major ECG parameters, may have been underpowered to detect subtle alterations in repolarization indices such as QTc or Tp-e intervals. Third, we did not assess certain potential confounders, including medication use or electrolyte levels, which could influence ECG findings. Fourth, we assessed muscle mass using BIA, which is generally considered less accurate than dual-energy X-ray absorptiometry. However, BIA is accepted as a valid and practical method of SMM estimation due to its advantages: portability, wide availability, rapidity, non-invasiveness, cost-effectiveness, and ease of use. Moreover, previous studies have demonstrated that BIA shows a good correlation with magnetic resonance imaging for assessing muscle mass. Importantly, the threshold values used to define LMM in our study were derived from population-specific reference data and were established using the same BIA device model employed in our study, thereby enhancing internal consistency and applicability to our cohort (18).

## Conclusion

In conclusion, the present study demonstrates a significant association between sarcopenia and certain ECG markers of arrhythmogenic risk, including fragmented QRS complexes, increased PWD, and a higher prevalence of AF. These findings suggest that sarcopenia may contribute to subclinical myocardial electrical remodeling and highlight the need for increased arrhythmic surveillance in this vulnerable population. While no significant associations were observed with repolarization-related parameters, future research should aim to better elucidate the electrophysiological consequences of sarcopenia through prospective studies with larger and more heterogeneous populations, and to determine whether early identification of such ECG changes could guide preventive strategies for arrhythmia-related adverse outcomes. Given the observed association between sarcopenia and specific ECG abnormalities, regular ECG screening may be considered in sarcopenic older adults to enable timely identification and management of potential arrhythmias.

### Ethics

**Ethics Committee Approval:** All study procedures were done based on the ethical principles of the İstanbul University, İstanbul

Faculty of Medicine Ethics Committee (protocol number: 2024/2147, date: 15.11.2024) and was done in accordance with the principles outlined in the Helsinki Declaration.

**Informed Consent:** Informed consent was obtained from all participants who were prospectively enrolled in the study. For retrospectively included individuals, data were collected from medical records and handled anonymously. No procedures beyond standard clinical assessments or routine laboratory tests were performed as part of this study.

### Footnotes

### Authorship Contributions

Concept: O.E., T.E., M.A.K., G.B., Design: O.E., T.E., M.A.K., G.B., Data Collection or Processing: O.E., Z.F., D.E.S., D.S., Ö.K., Analysis or Interpretation: T.E., S.Ö., G.B., Literature Search: O.E., T.E., S.Ö., Writing: O.E., T.E., S.Ö., G.B.

**Conflict of Interest:** No conflict of interest was declared by the authors.

**Financial Disclosure:** The authors declared that this study received no financial support.

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