ORIGINAL RESEARCH

DOI: 10.17944/interdiscip.1467199
Interdiscip Med J 2025;16(54):18-24

Adiponectin and Lone atrial fibrillation

Özden Kutlay[1], Arzu Keskin Aktan[1], Zafer Yalım[2]

[1]Afyonkarahisar Health Sciences University, Faculty of Medicine, Department of Physiology, Afyonkarahisar, Türkiye.
[2]Afyonkarahisar Health Sciences University, Faculty of Medicine, Department of Cardiology, Afyonkarahisar, Türkiye.

Abstract

Objective: Lone atrial fibrillation is an idiopathic arrhythmia seen in younger individuals without any secondary disease. Adiponectin is an endogenous adipocytokine that increases insulin sensitivity with anti-inflammatory and anti-proliferative effects. Although the relationship between circulating adiponectin and atrial fibrillation has been suggested, it is questionable whether this relationship is arrhythmia-related. Therefore, the focus of this study is to investigate the relationship between adiponectin and Lone atrial fibrillation.
Methods: In this prospective study, 26 healthy individuals in sinus rhythm, 34 patients with Lone Atrial Fibrillation, and 38 patients diagnosed with Atrial Fibrillation were included by questioning their cardiovascular histories and risk factors upon their arrival at the hospital. Echocardiography was performed to evaluate the left ventricular ejection fraction. Plasma adiponectin levels were studied with Enzyme-Linked ImmunoSorbent Assay (ELISA).
Results: Plasma adiponectin levels were significantly lower in the Atrial Fibrillation and Lone Atrial Fibrillation groups compared to the control group ($p<0.001$, $p<0.001$). Adiponectin levels did not differ significantly between Atrial Fibrillation and Lone Atrial Fibrillation groups ($p=0.191$). Furthermore, adiponectin was positively correlated with left ventricular ejection fraction ($r=0.208$, $p=0.04$).
Conclusion: This study reveals, for the first time, the relationship between plasma adiponectin levels and Lone Atrial Fibrillation. Our results indicated that low adiponectin levels are associated with Lone Atrial Fibrillation and that this relationship persists in patients with secondary Atrial Fibrillation. Therefore, we predict that adiponectin decreases in Atrial Fibrillation due to arrhythmia independent of secondary diseases.
Keywords: Atrial fibrillation, Lone atrial fibrillation, adiponectin, arrhythmia, left ventricular ejection fraction

INTRODUCTION

Atrial fibrillation (AF) emerges as one of the most prevalent arrhythmias, affecting 3-4% of the population and leading to increased morbidity and mortality, along with a heightened risk of stroke (1, 2). Well-defined major risk factors for AF include advanced age, hypertension, congestive heart disease (CHD), diabetes mellitus (DM), and thyroid disease (TD) (3). AF frequently arises as a secondary manifestation, either linked to a cardiac-origin condition like acute coronary syndrome and heart failure or associated with extracardiac comorbidities such as infectious or chronic lung disease (4). However, AF, usually observed in a proportion of young individuals, develops as an unexplained or idiopathic primary disorder called “lone AF” without an identifiable trigger (2). By general convention, the subtype of AF identified in individuals under the age of 60, where clinical assessment and imaging methods exclude structural cardiopulmonary diseases like coronary artery disease (CAD), hypertension, and heart valve disease, is termed lone AF. The typical age of individuals undergoing treatment for Lone AF within the young and middle-aged demographic hovers around 44 (5). Adiponectin is an endogenous adipocytokine with positive effects on cardiovascular diseases, and it stands out as a potential risk marker for AF (6,7). Adiponectin levels in plasma are reduced in obese individuals, patients with type 2 DM, CAD, and hypertension (8). Nevertheless, certain studies have linked elevated circulating adiponectin levels

Cite this article: Kutlay Ö, Keskin-Aktan A, Yalım Z. Adiponectin and Lone atrial fibrillation. Interdiscip Med J. 2025;16(54):18-24. https://10.17944/interdiscip.1467199

Corresponding Author: Dr. Özden Kutlay, Afyonkarahisar Health Sciences University Faculty of Medicine, Department of Physiology, Afyonkarahisar, Türkiye
Email: ozden.kutlay @afsu.edu.tr
ORCID iD: 0000-0001-5509-6650

Received: Apr 9, 2024
Accepted: Feb 1, 2025

with an unfavorable prognosis in conditions like heart failure, coronary heart disease, and cardiovascular disease (6,7). Furthermore, certain researchers have underscored a notable association between adiponectin and AF, suggesting that elevated adiponectin levels might pose a risk factor for AF (6, 8-10). The findings from these studies prompt us to hypothesize that the levels of adiponectin play a role in shaping the development and prevalence of AF.

According to the information provided by studies so far, the uncertainty persists regarding whether the elevated adiponectin levels observed in patients with secondary AF accompanied by secondary diseases are a consequence of AF itself or a result of hypertension, diabetes, or cardiac diseases associated with AF. Consequently, the goal of this study is to investigate the plasma adiponectin levels in patients with Lone AF who do not have any accompanying diseases. The increasing interest in the role of biomarkers in the pathogenesis and prognosis of AF is notable. However, there is still limited data on biomarkers in Lone AF (11). Accurately identifying patients with true Lone AF, meaning those at genuinely low risk of complications, holds significant prognostic and therapeutic implications. This study will be the first to demonstrate a possible relationship between adiponectin and Lone AF.

METHOD

Patients who previously applied to the Afyonkarahisar Medical Faculty Cardiology Department with complaints of palpitations and were diagnosed with Lone AF and AF underwent routine follow-ups. Thirty-four participants with Lone AF, 38 with AF, and 26 entirely healthy individuals in sinus rhythm were enrolled in the control group, sorted based on their arrival sequence. The patients' cardiovascular histories and risk factors were thoroughly examined, and detailed physical examinations were performed. Baseline electrocardiograms (ECGs) were taken for all patients and controls involved in the evaluation. Transthoracic echocardiography was performed in all study participants to assess the left ventricular ejection fraction and exclude heart valve disease.

Exclusion criteria for the Lone AF group included systolic heart failure ejection fraction (EF)<45%), diastolic heart failure, dilated and hypertrophic cardiomyopathy, congenital heart disease, restrictive cardiomyopathy, AF associated with valvular diseases, patients with mechanical valves, post-op AF, chronic liver disease, chronic kidney insufficiency, malignancy, arthritis, infection, CAD, hypertension, diabetes, thyroid disease, and individuals with inflammatory and autoimmune diseases.

Twenty-six completely healthy volunteers, meticulously selected for gender balance, constituted the control group. Exclusion criteria for the control group involved the detection of organic heart disease, suspected inflammation, infection, or chronic lung disease through routine echocardiography and physical examination. The execution of this study adhered to the principles of the Declaration of Helsinki, following the approval of the institutional ethics committee (Ethics committee date/number: Date:05.08.2022/ Number:2022/428).

Echocardiography

Two-dimensional echocardiography and Doppler echocardiography were conducted by an experienced echocardiographer using the Vivid 3 device (GE Healthcare Systems, Piscataway, New Jersey, USA) in all patients and the control group. Measurements included left ventricular systolic and diastolic diameters, left atrial dimensions, volumes, and the left ventricular ejection fraction.

Plasma adiponectin measurement

A peripheral blood sample was obtained from both the patient and control groups during the morning hours of 9:00-10:00, following an overnight fast of 8-12 hours. The samples underwent centrifugation at 4000xg for 10 minutes, leading to the separation of plasma. The plasma samples were subsequently preserved at -80 °C until the moment of biochemical analysis, aiming to ascertain the adiponectin levels. Furthermore, blood samples were gathered from each patient for routine biochemistry analysis. The amount of plasma adiponectin was measured using the human adiponectin ELISA kit from Bioassay Technology Laboratory, Shanghai, China, following the kit's instruction manual. Absorbance readings were conducted on a Chromate 4300 brand ELISA reader device (Awareness Technology, Inc. Martin Hwy. Palm City, USA) at 450 nm, and results were calculated using linear regression.

Statistical analysis

The data acquired from the study underwent analysis using SPSS 22 Software for Windows. The normal distribution of the data was assessed through the Kolmogorov-Smirnov and Shapiro-Wilk tests. Categorical variables were represented as numbers (percentages), while continuous variables were expressed as "mean ± standard deviation (SD)." Intergroup comparisons for categorical variables were conducted using the Chi-square test, and for continuous variables, one-way ANOVA (post-hoc LSD) was applied. Pearson's r was computed to illustrate the correlation between plasma adiponectin level and other continuous variables.

RESULTS

Table 1 presents the clinical characteristics of participants in the control, AF, and lone AF groups. The average age in both the AF and Lone AF groups was significantly higher compared to the control group (p<0.001 for both). The mean age of the Lone AF group was lower compared to the AF group (p<0.001). Among the participants, 53.8% in the control group, 44.7% in the AF group, and 58.8% in the Lone AF group were male, with no significant difference in gender distribution between the groups (p=0.479). Both the AF and Lone AF groups exhibited higher body mass index (BMI) compared to the control group (p<0.001 for both), with no significant difference in BMI between the AF and Lone AF groups (p=0.108).

In the control group, only 7.7% of participants had hypertension and hypercholesterolemia, and they did not have other comorbidities. For patients in the AF group, AF was accompanied by at least one and at most five more diseases.

Table 1. Clinical characteristics of control, atrial fibrillation, and lone atrial fibrillation groups

	Control (n=26)	AF (n=38)	Lone AF (n=34)	P
Age (years)	44.81 ± 3.11	77.05 ± 7.15 *	51.53 ± 5.08 *#	<0.001
Men (n/%)	14 (53.8)	17 (44.7)	20 (58.8)	0.479
Body mass index (kg/m²)	24.31 ± 3.2	30.84 ± 2.88 *	29.66 ± 3.16 *	<0.001
Smoking (n/%)	14 (53.8)	12 (31.6)	16 (47.1)	0.174
Accompanying diseases				
Hypertension (n/%)	2 (7.7)	38 (100)	-	-
Diabetes mellitus (n/%)	-	32 (84.2)	-	-
Heart failure (n/%)	-	4 (10.5)	-	-
Prior stroke/TIA (n/%)	-	2 (5.3)	-	-
Coronary artery disease (n/%)	-	8 (21.1)	-	-
Hypercholesterolemia (n/%)	2 (7.7)	6 (15.8)	2 (5.9)	-
Ischemic heart disease (n/%)	-	4 (10.5)	-	-
Medications				
Antiarrhythmic agents (n/%)	-	32 (84.2)	22 (64.7)	-
Anticoagulant (n/%)	-	30 (78.9)	8 (23.5)	-
Echocardiography				
Left atrium diameter (mm)	32.46 ± 5.74	43 ± 5.88 *	35.21 ± 3.19 *#	<0.001
LVEF (%)	63.92 ± 3.45	57 ± 9.15 *	60.71 ± 3.81 #	<0.001
LVDS (mm)	31 ± 3.82	32.05 ± 3.77	30.18 ± 4.34	0.141
LVEDD (mm)	46.85 ± 4.12	47.95 ± 3.18	46.47 ± 2.85	0.158
LVPWth (mm)	10.46 ± 0.86	11.68 ± 0.66 *	10.88 ±0.77 *#	<0.001
IVST (mm)	10.62 ± 1.02	12.32 ± 0.93 *	11.12 ± 0.98 *#	<0.001
Biochemistry				
Fasting glucose (mg/dL)	96.77 ± 9.99	137.03 ± 48.84 *	100.29 ± 9.69 #	<0.001
Creatinin (mg/dL)	0.84 ± 0.21	0.99 ± 0.4 *	0.79 ± 0.16 #	0.012
LDL (mg/dL)	103.72 ± 30.47	106.06 ± 28.48	107.55 ± 26.66	0.874
HDL (mg/dL)	40.61 ± 9.18	42.31 ± 10.86	43.65 ± 8.12	0.476
Triglyceride (mg/dL)	173.89 ± 84.56	177.73 ± 110.34	144.41 ± 81.01	0.285
Total cholesterol (mg/dl)	171.21 ± 32.43	168.38 ± 32.49	167.75 ± 30.84	0.909

Continuous variables are presented as mean±SD and analyzed using one-way ANOVA. Categorical variables are presented as numbers (percentages) and analyzed using the Chi-square test. P values <0.05 are indicated in bold. *p<0.05 vs Control. #p<0.05 vs AF. AF: atrial fibrillation, TIA: transient ischemic attack, LVEF (%): left ventricle ejection fraction, LVDS (mm): left ventricular sistolic dimension, LVEDD (mm): left ventricular end-diastolic dimension, LVPWth(mm): left ventricle posterior wall thickness, IVST (mm): interventricular septum thickness, LDL: low density lipoprotein, HDL: high density lipoprotein

All participants in the AF group had hypertension, 84.2% had DM, 10.5% had heart failure, 5.3% had a prior stroke/TIA, 21.1% had CAD, 15.8% had hypercholesterolemia, and 10.5% had ischemic heart disease. In the Lone AF group, no other diseases accompanied AF except hypercholesterolemia (5.9%).

While no antiarrhythmic or anticoagulant drugs were used in the control group, 84.2% of the AF group were using antiarrhythmic drugs, and 78.9% were using anticoagulant drugs. In the Lone AF group, 64.7% of the patients were using antiarrhythmic drugs, and 23.5% were using anticoagulant drugs. (Table 1).

Echocardiography findings indicated significant differences only in left atrium diameter, left ventricular ejection fraction (LVEF),(%), left ventricular posterior wall thickness (LVPWth), and interventricular septal thickness (IVST) among the groups.

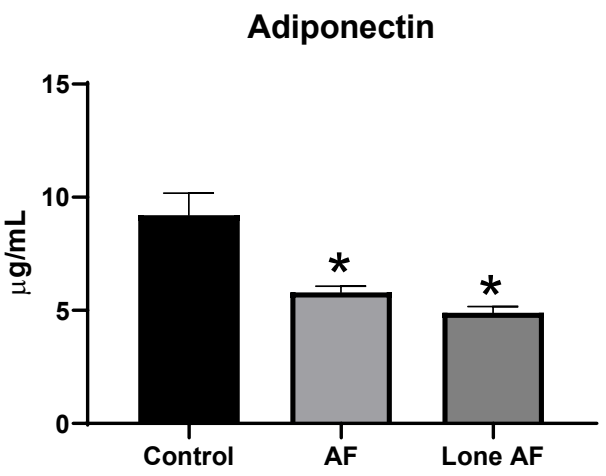

Figure 1. Plasma adiponectin level (µg/mL) of Control, AF and Lone AF groups. Data were presented as mean ± standard deviation (SD). One-way ANOVA (post-hoc LSD) was used for comparisons between groups. *p<0.001 vs control group. AF: atrial fibrillation.

The left atrium diameter increased in both the AF and Lone AF groups compared to the control group (p<0.001, p=0.04, respectively), with a further increase in the AF group compared to Lone AF (p<0.001). LVEF (%) decreased in the AF group compared to both the control and Lone AF groups (p<0.001, p=0.016, respectively). LVEF (%) also showed a lower value in the Lone AF group compared to the control group, though this difference approached borderline significance (p=0.056). Both LVPWth and IVST showed a significant increase in both the AF and Lone AF groups compared to the control group (p<0.001, p=0.035 for LVPWth; p<0.001, p=0.05 for IVST, respectively). Moreover, both LVPWth and IVST showed higher values in the AF group compared to the Lone AF group (p<0.001, p<0.001, respectively), (Table 1).

Biochemical variables, including HDL, LDL, triglycerides, and total cholesterol levels, showed no significant differences across the groups. However, fasting glucose and creatinine levels were higher in the AF group compared to the control group (p<0.001, p=0.048, respectively). Similarly, fasting glucose and creatinine levels were elevated in the AF group compared to the Lone AF group (p<0.001, p=0.004, respectively).

There was no significant difference in adiponectin levels between the AF and Lone AF groups (p=0.191) (Figure 1). The correlation analysis revealed a negative association between adiponectin levels and BMI (r=-0.241, p=0.017) (Figure 2). Additionally, adiponectin showed a positive correlation with LVEF (r=0.208, p=0.04) and a negative correlation with LVPWth and IVST based on echocardiographic data (r=-0.439, p<0.001; r=-0.416, p<0.001, respectively).

DISCUSSION

There remains uncertainty regarding whether the changing plasma adiponectin levels observed in AF patients is a consequence of AF itself or a result of hypertension, diabetes, or cardiac diseases associated with AF. Therefore, the aim of this study was to investigate plasma adiponectin levels in Lone AF patients who do not have any accompanying diseases.

Apart from being one of the most abundant endogenous adipocytokines in the body, adiponectin is recommended as one of the biomarker proteins for prognosis in cardiovascular diseases (12). Plasma levels of adiponectin decrease in diseases such as obesity, type 2 DM, CAD, and hypertension (8). The decrease in adiponectin levels in diseases such as obesity, type 2 DM, CAD, and hypertension, which are included in the etiology of AF and pave the way for the formation of AF, creates an expectation that the level of adiponectin will also decrease in AF. The observation of reduced adiponectin levels in patients with Lone AF and AF, a central finding in our study, is consistent with this anticipation. The study of Assar et al., who pointed out that low adiponectin levels may contribute to postoperative AF, also supports our findings (13). Nevertheless, some studies highlight a substantial association between adiponectin and AF; conversely, they propose that elevated adiponectin levels might pose a risk factor for AF (6, 8-10). To clarify the inconsistency among various studies, it is essential to assess the subtypes of AF.

Following a similar line of thought (8) hoped that plasma adiponectin levels in patients with AF would be lower compared to control patients, but they found high adiponectin levels in patients with persistent AF. On the

other hand, despite high adiponectin in the persistent AF group, they found lower adiponectin levels in paroxysmal AF compared to the control group.

Another group of researchers corroborated the earlier study by demonstrating that serum adiponectin levels were notably elevated in patients with persistent AF compared to those with sinus rhythm, yet significantly lower in patients with paroxysmal AF (14). Perhaps the discrepancy between these two cross-sectional studies (8, 14) and, other studies (6, 8-10) that suggests that high levels of adiponectin in AF may be attributed to the fact that AF was not divided into subtypes such as paroxysmal, permanent, and persistent.

Unfortunately, the AF group could not be subdivided into subtypes due to an insufficient number of patients for statistical analysis. Although the AF group could not be categorized further, the majority (42.1%) of the AF group in our study consisted of patients with paroxysmal AF. Out of the remaining patients, 31.6% had persistent AF, and 26.3% had permanent AF. In this study, it was observed that plasma adiponectin levels were lower in both the AF and Lone AF groups when compared to the control group. There was no significant difference between the AF and Lone AF groups in terms of adiponectin levels. In fact, our results contribute to the literature on the role of adiponectin as follows: Previous studies have demonstrated the relationship between adiponectin levels and AF, but factors predisposing to AF (such as diabetes, hypertension, acute coronary syndrome) could not be excluded. However, the observation that the adiponectin level was lower in the Lone AF group, which was completely free of these variables, compared to the control group, supports the idea that adiponectin decreases due to arrhythmia independently of secondary factors.

In the present study, left atrium size, LVEF, LVPWth, and IVST, which are echocardiographic findings, were found to differ significantly between the groups. The left atrium size, elevated in both the AF and Lone AF groups in comparison to the control group, exhibited a further increase in the AF group when compared to the Lone AF group. Hypervolemia, which increases left ventricular filling pressures in obesity, which is one of the risk factors for AF, leads to left atrial enlargement, which is one of the early signs of left ventricular dysfunction (15). To our knowledge, the relationship between adiponectin and left atrial size in obese patients has not been resolved to date. While one study indicated that adiponectin was negatively correlated with left atrium size (15) another study found a positive correlation (9). However, in this study, no correlation was found between adiponectin level and left atrium dimensions in patients with Lone AF and AF. Low LEVF in atrial fibrillation is considered a left ventricular dysfunction (16).

In this study, LVEF was lower in the AF group compared to the control and Lone AF groups. It was also lower in the Lone AF group compared to the control, but this difference was at a level that could be considered borderline significant. These results align with our rationale for selecting patients with Lone AF. Thamilarasen et al. observed an increase in left atrium size and a decrease in LVEF in patients with Lone AF (17). The increased left atrium dimensions in the Lone AF and AF groups of our study compared to the control group, and especially the decreased LVEF in AF, align with this study. The positive correlation of plasma adiponectin, released from adipose tissue with a favorable cardiovascular profile, with LVEF, and the negative correlation with LVPWth and IVST in our study further supports its characterization as a beneficial adipocytokine. Despite its release from adipose tissue, increased adiposity, especially visceral fat, reduces adiponectin secretion (18).

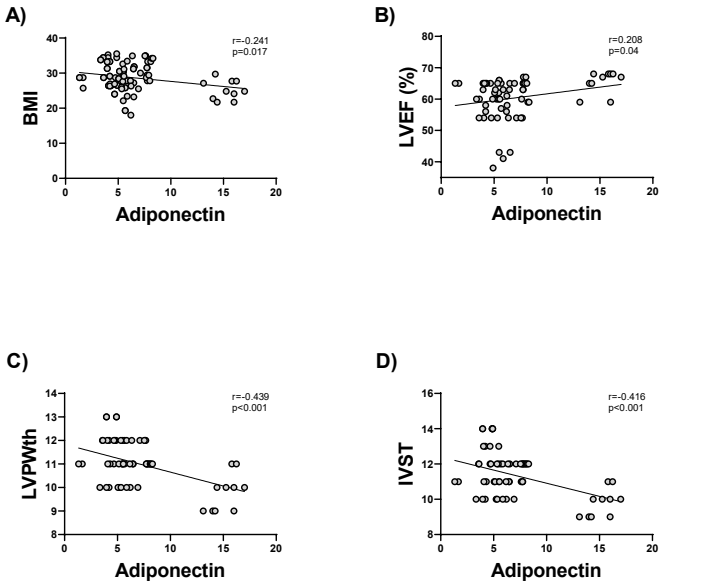

Figure 2. Scatter plots showing the relationship between plasma adiponectin level and body mass index:BMI (A), left ventricular ejection fraction:LEVF (%) (B), left ventricular posterior wall thickness:LVPWth (C), and interventricular septal thickness:IVST (D).

Therefore, circulating adiponectin levels also decrease in obesity due to abdominal adiposity. Studies examining the relationship between BMI and AF suggest that obesity may partially mediate the increased risk of AF but it does not solely contribute to it (6, 9, 19). Moreover, there is a suggestion that obesity could act as a risk factor for Lone AF, underscoring the connection between individuals with Lone AF and elevated BMI (20). Consistent with this study, our research revealed a negative correlation between BMI and adiponectin.

In summary, the current study is the first one to establish the connection between adiponectin and Lone AF. Our results indicate that low adiponectin is independently associated with Lone AF, and this association persists in patients with secondary AF. Consequently, we propose that adiponectin decreases in Lone AF and AF due to arrhythmia independently of secondary diseases.

Limitations of the study

Due to the fact that this study was conducted at a single center and the number of patients was small, patients with AF could not be divided into subgroups.

CONCLUSION

These findings imply that adiponectin levels might contribute to the pathophysiology of arrhythmia. Currently, it remains unclear whether low adiponectin is merely a marker in Lone AF or an active participant in pathogenesis. We anticipate that our findings will guide future studies in unraveling the mechanisms underlying the pathogenesis associated with the relationship between low adiponectin levels and Lone AF.

ACKNOWLEDGEMENTS

Peer-Review: Both externally and internally peer reviewed.

Conflict of Interest: The authors declare that they have no conflict of interests regarding content of this article.

Financial Support: The Authors report no financial support regarding content of this article.

Ethical Declaration: All procedures were approved by the Afyonkarahisar Health Sciences University Non-interventional Clinical Studies by the Ethics Committee (Approval number: 2022/428).

Athorship Contributions: Concept: ÖK, AKK, ZY, Design: ÖK, AKK, ZY, Supervision: ÖK, AKK, ZY, Financing and equipment: ÖK, AKK, ZY, Materials: ÖK, AKK, ZY, Data collection and entry: ÖK, AKK, ZY, Analysis and Interpretation: ÖK, AKK, ZY, Literature Search: ÖK, AKK, ZY, Writing: ÖK, AKK, ZY, Critical Review: ÖK, AKK, ZY.

REFERENCES

1. Sawano M. Lone Atrial Fibrillation Reconfirmed as Unfavorable Arrhythmia. J Atheroscler Thromb. 2021;28(3):225-6. doi: 10.5551/jat.ED137. https://doi.org/10.5551/jat.ED137
2. Roston TM, Islam S, Hawkins NM, et al. A Population-Based Study of Unexplained/Lone Atrial Fibrillation: Temporal Trends, Management, and Outcomes. CJC Open. 2021;4(1):65-74. https://doi.org/10.1016/j.cjco.2021.09.006
3. Potpara TS, Lip GHY. Lone atrial fibrillation: what is known and what is to come. Int J Clin Pract. 2011;65(4):446-57. https://doi.org/10.1111/j.1742-1241.2010.02618.x
4. Zoni-Berisso M, Lercari F, Carazza T, et al. Epidemiology of atrial fibrillation: European perspective. Clin Epidemiol. 2014;6:213-20. doi: 10.2147/CLEP.S47385. Available from: https://doi.org/10.2147/CLEP.S47385
5. Frost L, Anderson LV, Godtfredsen J, et al. Age and risk of stroke in atrial fibrillation. Neuroepidemiology. 2007;28(2):109-15. https://doi.org/10.1159/000101509
6. Yamaguchi N, Okumura Y, Watanabe I, et al. Clinical implications of serum adiponectin on progression of atrial fibrillation. JArrhythm. 2017;33(6):608-12. https://doi.org/10.1016/j.joa.2017.07.009
7. Barnett AS, Piccini JP. Adiponectin: an accurate biomarker for patients at risk for atrial fibrillation? Heart. 2015;101(17):1351-2. https://doi.org/10.1136/heartjnl-2015-307816
8. Shimano M, Shibata R, Tsuji Y, et al. Circulating Adiponectin Levels in Patients With Atrial Fibrillation. Circ J. 2008;72(7):1120-4. https://doi.org/10.1253/circj.72.1120
9. Zhu T, Wang Z, Wang S, et al. Association between Serum Adiponectin and Atrial Fibrillation: A Case-Control Study Stratified by Age and Gender. Cardiol Res Pract. 2021;2021:6633948. https://doi.org/10.1155/2021/6633948
10. Guo Y, Liu L, Wang J. Adiponectin and the risk of new-onset atrial fibrillation: a meta-analysis of prospective cohort studies. Biosci Rep. 2019;39(6):BSR20182284. https://doi.org/10.1042/BSR20182284
11. Lin K, Marine JE, Calkins H, et al. Should we abandon the term "lone atrial fibrillation"? Hellenic J Cardiol. 2019;60(4):216-23. https://doi.org/10.1016/j.hjc.2019.04.005
12. Hernández-Romero D, Jover E, Marín F, et al. The prognostic role of adiponectin levels in atrial fibrillation. Eur J Clin Invest. 2013;43(2):168-73. https://doi.org/10.1111/eci.12028
13. Assar O. Low adiponectin level may contribute to higher incidence of post-cardiac surgery atrial fibrillation in obese patients. Ann Thorac Surg. 2012;93(5):1762-3. https://doi.org/10.1016/j.athoracsur.2011.09.025
14. Choi BJ, Heo JH, Choi IS, et al. Hypoadiponectinemia

in patients with paroxysmal atrial fibrillation. Korean Circ J. 2012;42(10):668-73. https://doi.org/10.4070/kcj.2012.42.10.668

15. Ybarra J, Resmini E, Planas F, et al. Relationship between adiponectin and left atrium size in uncomplicated obese patients: adiponectin, a link between fat and heart. Obes Surg. 2009;19(9):1324-32. https://doi.org/10.1007/s11695-009-9924-5
16. Wijesurendra RS, Liu A, Eichhorn C, et al. Lone Atrial Fibrillation Is Associated With Impaired Left Ventricular Energetics That Persists Despite Successful Catheter Ablation. Circulation.2016;134(15):1068-81. https://doi.org/10.1161/CIRCULATIONAHA.116.024679
17. Thamilarasan M, Grimm RA, Rodriguez LL, et al. Left ventricular diastolic dysfunction in lone atrial fibrillation determined by Doppler tissue imaging of mitral annular motion. Am J Cardiol. 2000;86(9):1026-9, A10. https://doi.org/10.1016/s0002-9149(00)01144-9
18. Pahlavani HA. Exercise Therapy for People With Sarcopenic Obesity: Myokines and Adipokines as Effective Actors. Front Endocrinol (Lausanne). 2022;13:811751. https://doi.org/10.3389/fendo.2022.811751
19. Ermakov S, Azarbal F, Stefanick ML, et al. The associations of leptin, adiponectin, and resistin with incident atrial fibrillation in women. Heart. 2016;102(17):1354-62. https://doi.org/10.1136/heartjnl-2015-308927
20. El-Assaad I, Al-Kindi SG, Saarel EV, et al. Lone Pediatric Atrial Fibrillation in the United States: Analysis of Over 1500 Cases. Pediatr Cardiol. 2017;38(5):1004-9. https://doi.org/10.1007/s00246-017-1608-7