Original paper

Body mass index and adverse cardiovascular events in individuals with atrial fibrillation taking oral anticoagulants: a prospective, observational, long-term follow-up cohort study

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Key words: anticoagulation, atrial fibrillation, body mass index, cardiovascular outcomes, left atrial appendage thrombus.

Abstract

Introduction: Obesity is a known variable associated with increased risk of atrial fibrillation (AF); however, the impact of body mass index (BMI) on adverse cardiovascular outcomes in individuals with AF taking anticoagulants is not well established.
Aim of the research: This study aimed to evaluate the association between BMI categories (normal weight, overweight, and obese) and adverse cardiovascular outcomes in individuals with AF taking anticoagulants.
Material and methods: This prospective, observational, long-term follow-up cohort study included 500 individuals with AF who underwent transoesophageal echocardiography before electrical cardioversion. Individuals were stratified by BMI and followed up for a median of 1927.5 days. Endpoints included major cardiovascular events (stroke, myocardial infarction, transient ischaemic attack, systemic thromboembolism, hospitalisation for heart failure, and cardiovascular death) and left atrial appendage thrombus (LAAT).
Results: Adverse cardiovascular event rates were similar across BMI categories. Individuals with normal weight presented with fewer comorbidities, such as diabetes mellitus and arterial hypertension, and demonstrated more favourable echocardiographic measurements, including smaller left atrial diameters. Despite these differences, no significant variations in LAAT prevalence or other adverse cardiovascular outcomes were observed between the BMI groups. BMI did not significantly influence long-term adverse cardiovascular outcomes or LAAT in individuals with AF taking anticoagulants. The low proportion of normal-weight individuals in the cohort suggests a potential protective role of normal weight against AF.
Conclusions: Further research is required to understand the complex interplay among BMI, comorbidities, and cardiovascular risk. These findings question the utility of BMI as a standalone risk predictor in this population.

Introduction

Atrial fibrillation (AF) is the most common persistent cardiac arrhythmia worldwide, affecting approximately 44 million people [1, 2], and its prevalence is expected to double in the coming decades [3]. This arrhythmia leads to adverse cardiovascular outcomes [4]. Numerous studies have linked obesity to an enhanced risk of AF [5, 6]; however, the influence of body mass index (BMI) on clinical events in individuals with established AF taking oral anticoagulants (OACs) remains unclear [4, 7].

The "obesity paradox" describes an intriguing phenomenon wherein overweight or obese individuals may experience better cardiovascular outcomes than those with normal BMI [8]. Although some studies support this in individuals with AF [9–13], other studies suggest no protective effect or even an enhanced risk of complications associated with a higher BMI [8, 14–16]. Additionally, evidence regarding the impact of BMI on cardiac thrombogenesis [2, 17, 18] and long-term cardiovascular events in individuals with AF taking OACs remains sparse and inconsistent.

Aim of the research

Given the importance of identifying factors influencing AF prognosis, we aimed to explore the association between BMI categories (normal weight, overweight, and obesity) and adverse cardiovascular outcomes, including left atrial appendage thrombus (LAAT) and other major events. By addressing these knowledge gaps, we hope to enhance our understanding of the complex relationship between BMI and cardiovascular risk in this high-risk population.

Material and methods

Study group

This prospective observational cohort study was conducted using data collected from the electronic medical database of the Provincial Combined Hospital

in Kielce, Poland. Eligible participants were individuals with AF admitted to the Cardiology Department for electrical cardioversion guided by transoesophageal echocardiography (TEE) between December 2010 and March 2023. The inclusion and exclusion criteria are presented in Table 1. Consecutive recruitment ensured a representative cohort of individuals with AF.

This study adhered to the ethical principles of the Declaration of Helsinki from 1989. Ethical approval was granted by the Committee on Ethics of the Jan Kochanowski University of Humanities and Sciences in Kielce, Poland (ratification document code: 21/2010; date of agreement: 28 January 2010). Informed consent was obtained from all study participants. To ensure high-quality reporting, our study followed the Strengthening of Reporting Observational Studies in Epidemiology (STROBE) recommendations.

Oral anticoagulants

Patients receiving OACs were eligible for inclusion if they fulfilled the following criteria: vitamin K antagonist therapy with dose adjustment and international normalised ratio monitoring, maintaining a therapeutic range (international normalised ratio ≥ 2.0), or uninterrupted anticoagulation with apixaban, rivaroxaban, or dabigatran for at least 3 weeks before enrolment [19–22]. For dabigatran therapy, the standard full dose was 150 mg twice daily; however, a reduced dose of 110 mg twice daily was also permitted if any of the following applied: concomitant use of verapamil, age ≥ 80 years, "hypertension, abnormal renal/liver function, stroke, bleeding history or predisposition, labile international normalised ratio, elderly (> 65 years), drugs/alcohol concomitantly" (HAS-BLED) score ≥ 3, or estimated glomerular filtration rate (eGFR) 30–49 ml/min/1.73 m^2 [20]. For rivaroxaban therapy, the standard full dose was 20 mg once daily; however, a reduced dose of 15 mg once daily was also permitted if any of the following applied: HAS-BLED score ≥ 3 or eGFR 15–49 ml/min/1.73 m^2. For apixaban therapy, the standard full dose was 5 mg twice daily; however, a reduced dose of 2.5 mg twice daily was also permitted if two out of three of the following applied: serum creatinine level ≥ 1.5 mg/dl, body weight ≤ 60 kg, or age ≥ 80 years. For individuals on vitamin K antagonist therapy, the international normalised ratio was checked weekly for 3 weeks before enrolment, with all values required to be within the therapeutic range. eGFR was evaluated using the Modification of Diet in Renal Disease method [23].

The inclusion and exclusion criteria for the study were clear and unambiguous and were adhered to throughout the recruitment period. However, the quality of anticoagulant regimens in relation to the current guidelines for AF was always monitored and, if necessary, adjusted at discharge during the index hospitalisation.

Table 1. Inclusion and exclusion criteria

Inclusion criteria
• Patients aged 18 years or older who are taking oral anticoagulants.
• Symptomatic atrial fibrillation episode lasting 48 hours or more.
Exclusion criteria
• Heart rate lower than 60 bpm.
• Systolic blood pressure below 90 mm Hg.
• Exacerbation of heart failure.
• Abnormal peripheral perfusion.
• Prior intracardiac thrombus.
• History of ablation for atrial flutter or atrial fibrillation.
• Previous electrical cardioversion.
• Presence of prosthetic heart valve.
• Mitral stenosis characterised by a mean mitral valve gradient of ≥ 5 mm Hg and a mitral valve area of ≤ 1.5 cm^2.

Echocardiographic examination

Cardiac ultrasound measurements were performed by certified cardiac sonographers using the GE Vivid E95 and GE Vivid E9 ultrasound systems (GE Vingmed Ultrasound AS, Horten, Norway). The examinations followed established protocols and current guidelines [24]. The left atrial appendage (LAA) was imaged using a multiplanar ultrasound transducer from the mid-oesophageal view on TEE. The LA diameter was assessed at the end of ventricular systole using transthoracic echocardiography in the parasternal long-axis view, measured perpendicular to the long axis of the aortic root. The left ventricular ejection fraction was evaluated using the biplane Simpson method as per echocardiographic guidelines [25]. Cardiac blood clots (thrombi) were defined as echodense intracardiac masses with well-defined margins, clearly separated from the endocardial border, visualised in at least two imaging planes throughout the cardiac cycle, and distinguishable from the pectinate muscles on echocardiographic examination [26]. All measurements were documented and archived for further analysis to ensure reproducibility and compliance with clinical standards.

Statistical analysis

Categorical variables are summarised as frequencies and percentages. Continuous variables are summarised as means, SDs, medians, and interquartile ranges. The normality of continuous variables was evaluated using the Shapiro–Wilk test. For all contin-

uous variables, the Kruskal–Wallis test was performed because of their non-normal distribution. For categorical variables, the Fisher exact test (when the expected frequency was < 5) or the χ^2 test was applied. To evaluate the differences between the subgroups presented in Tables 2 and 3, the Fisher exact test was performed for variables such as prior transient ischaemic attack, stroke, systemic thromboembolism, chronic obstructive pulmonary disease, previous myocardial infarction, and peripheral artery disease, and χ^2 tests were applied to other categorical variables. Kaplan–Meier survival curves for cardiovascular death and hospitalisation for heart failure were created according to BMI categories (normal weight, overweight, and obese). For the multiple-sample survival analysis, the Mantel procedure was used to assign a score to each survival time. Then, a χ^2 value was computed on the basis of the sums of these scores for each group.

Statistical analysis

Statistical significance was defined as a two-tailed p-value < 0.05. Survival data analyses were performed using Statistica (data analysis software system, version 13; TIBCO Software Inc.), where the described survival analysis method is the default test in multiple-sample survival analysis. Other statistical analyses were conducted using R (version 4.0.3; R Foundation for Statistical Computing, Vienna, Austria).

Results

In total, 500 consecutive individuals with AF, who were taking OACs, were included in this study. Follow-up data were collected for a median duration of 1927.5 days (interquartile range: 1004–2643 days), beginning on the day of the TEE procedure. Follow-up was completed for all participants. Endpoint information was collected from the hospital database and directly from participants or their relatives during the first evaluation and 12 months after study enrolment and was reassessed annually. The proportion of women was significantly lower in the overweight group than in the normal-weight and obese groups ($p = 0.035$). Diastolic blood pressure was significantly lower in the normal-weight group than in the other groups ($p = 0.006$). The LA diameter was smaller in the normal-weight group than in the other groups ($p < 0.001$). Arterial hypertension and diabetes mellitus were less prevalent in the normal-weight group than in the other groups ($p < 0.001$ and < 0.001, respectively). Angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers were prescribed less frequently in the normal-weight group ($p < 0.001$) (Table 2).

During the index hospitalisation, a rate control strategy was applied without electrical cardioversion in 82 individuals (16.4%) due to either LAAT (65 [13%]) or recurrent arrhythmia (17 [3.4%]) in patients unlikely to maintain sinus rhythm. Among the 418 (83.6%) individuals who underwent electrical cardioversion, 365 (87.3%) achieved sinus rhythm. No periprocedural complications were observed. LAAT rates were similar across different BMI categories ($p = 0.25$), and no significant differences were observed in the rates of hospitalisation for heart failure, myocardial infarction, transient ischaemic attack, stroke, systemic thromboembolism, cardiovascular death, or all-cause mortality among the BMI groups (Table 3).

Kaplan–Meier survival curves for cardiovascular death and hospitalisation for HF in the normal, overweight, and obese groups are presented in Figures 1 and 2. Survival distributions showed no significant differences in cardiovascular death and hospitalisation for HF among the BMI groups ($p = 0.06$ for both analyses).

In summary, although there were notable differences in baseline characteristics, such as lower comorbidity rates and more favourable echocardiographic measurements in the normal-weight group, no significant differences were found in long-term cardiovascular outcomes or LAAT across the BMI categories.

Discussion

This study aimed to evaluate the effects of BMI on adverse cardiovascular outcomes in individuals with AF taking OACs. Our findings revealed that BMI status, stratified into normal-weight, overweight, and obese categories, did not significantly influence cardiovascular outcomes. Normal-weight individuals exhibited fewer comorbidities and had more favourable echocardiographic measurements; however, these differences in baseline characteristics did not translate into improved long-term outcomes or LAAT formation. Our findings suggest that BMI is not a predictor of adverse outcomes in individuals with AF taking OACs.

Previous studies have shown inconsistent associations between BMI and cardiovascular outcomes in individuals with AF. Some studies suggest that obesity negatively impacts prognosis and increases cardiovascular mortality and morbidity [8, 14–16], whereas others indicate the opposite [9–13]. This inverse relationship between obesity and improved long-term cardiovascular outcomes has been described as the "obesity paradox". Previous trials addressing this issue have shown varying representations of individuals receiving OAC therapy.

Overall, BMI has increased significantly in recent decades in adults, children, and adolescents [6], and obesity is a major risk factor for cardiovascular morbidity and mortality [27, 28]. It is well established that obesity increases AF susceptibility [29]. In a meta-analysis by Wanahita *et al.* [30], obesity was associated with a 49% increased risk of developing AF in the general population, and the risk increased in parallel with greater BMI. Similarly, a meta-analysis by Asad *et al.* [31] found that obesity was associated with

Table 2. Baseline characteristics of individuals with atrial fibrillation based on body mass index categories

Parameter	Normal weight (BMI < 25 kg/m²) (*N* = 63)	Overweight (BMI 25.0–29.9 kg/m²) (*N* = 224)	Obese (BMI ≥ 30 kg/m²) (*N* = 213)	*P*-value
BMI [kg/m²]				
Mean (SD)	23.1 (1.7)	27.8 (1.4)	34.2 (4.1)	
Median (Q1, Q3)	23.6 (21.9, 24.5)	28.0 (26.8, 29.0)	33.1 (31.9, 35.3)	
Sex				0.035*
Female	26 (41.3%)	75 (33.5%)	97 (45.5%)	
Male	37 (58.7%)	149 (66.5%)	116 (54.5%)	
Age [years]				0.24
Mean (SD)	65.3 (12.2)	65.2 (9.3)	63.7 (9.6)	
Median (Q1, Q3)	66.0 (60.0, 74.5)	65.5 (60.0, 72.0)	65.0 (58.0, 71.0)	
CHA2-DS2-VASc score				0.09
Mean (SD)	2.8 (1.8)	2.8 (1.5)	3.1 (1.5)	
Median (Q1, Q3)	3.0 (1.0, 4.0)	3.0 (2.0, 4.0)	3.0 (2.0, 4.0)	
Heart rate [bpm]				0.15
Mean (SD)	100.5 (18.2)	95.6 (18.6)	96.0 (20.2)	
Median (Q1, Q3)	100.0 (90.0, 110.0)	90.0 (80.0, 110.0)	90.0 (80.0, 110.0)	
Systolic blood pressure [mm Hg]				0.052
Mean (SD)	121.4 (10.6)	123.4 (10.7)	125.3 (11.0)	
Median (Q1, Q3)	120.0 (115.0, 130.0)	125.0 (120.0, 130.0)	130.0 (120.0, 130.0)	
Diastolic blood pressure [mm Hg]				0.006*
Mean (SD)	74.6 (7.5)	77.5 (7.4)	77.8 (7.2)	
Median (Q1, Q3)	75.0 (70.0, 80.0)	80.0 (70.0, 80.0)	80.0 (70.0, 80.0)	
Estimated glomerular filtration rate by Modification of Diet in Renal Disease formula, [ml/min/1.73 m²]				0.64
Mean (SD)	68.8 (15.4)	66.5 (14.8)	67.0 (15.9)	
Median (Q1, Q3)	66.8 (58.9, 76.7)	66.2 (56.8, 74.9)	67.2 (56.1, 77.6)	
Left atrial diameter [mm]				< 0.001*
Mean (SD)	42.9 (6.0)	45.0 (4.9)	45.8 (4.8)	
Median (Q1, Q3)	43.0 (40.0, 46.0)	44.0 (42.0, 48.0)	46.0 (42.0, 48.0)	
Left ventricular ejection fraction, %				0.15
Mean (SD)	50.0 (14.2)	52.9 (12.3)	54.4 (10.5)	
Median (Q1, Q3)	55.0 (43.0, 60.0)	56.5 (48.0, 60.0)	58.0 (50.0, 60.0)	
Previous stroke/transient ischaemic attack/systemic thromboembolism	5 (7.9%)	17 (7.6%)	16 (7.5%)	> 0.99
Arterial hypertension	34 (54.0%)	170 (75.9%)	184 (86.4%)	< 0.001*
Heart failure	36 (57.1%)	114 (50.9%)	122 (57.3%)	0.36
Diabetes mellitus	7 (11.1%)	35 (15.6%)	64 (30.0%)	< 0.001*
Chronic obstructive pulmonary disease	3 (4.8%)	11 (4.9%)	10 (4.7%)	> 0.99
Previous myocardial infarction	4 (6.3%)	20 (8.9%)	14 (6.6%)	0.61
Aortic plaque or peripheral artery disease	9 (14.3%)	14 (6.2%)	16 (7.5%)	0.13

Table 2. Cont.

Parameter	Normal weight (BMI < 25 kg/m²) (*N* = 63)	Overweight (BMI 25.0–29.9 kg/m²) (*N* = 224)	Obese (BMI ≥ 30 kg/m²) (*N* = 213)	*P*-value
Anticoagulation on admission				
Vitamin K antagonist	9 (14.3%)	29 (12.9%)	26 (12.2%)	0.91
Rivaroxaban	14 (22.2%)	68 (30.4%)	63 (29.6%)	0.44
Apixaban	15 (23.8%)	28 (12.5%)	30 (14.1%)	0.08
Dabigatran	25 (39.7%)	99 (44.2%)	94 (44.1%)	0.8
Medication at discharge				
Vitamin K antagonist	8 (12.7%)	31 (13.8%)	24 (11.3%)	0.72
Rivaroxaban	13 (20.6%)	63 (28.1%)	63 (29.6%)	0.37
Apixaban	16 (25.4%)	31 (13.8%)	35 (16.4%)	0.09
Dabigatran	26 (41.3%)	99 (44.2%)	91 (42.7%)	0.9
ACEI or ARB	36 (57.1%)	157 (70.1%)	172 (80.8%)	< 0.001*
Beta-blocker	56 (88.9%)	196 (87.5%)	196 (92.0%)	0.3
Statin	35 (55.6%)	135 (60.3%)	145 (68.1%)	0.1

**Significant P-value. Data are presented as median (interquartile range), mean (standard deviation), or number (percentage). ACEI – angiotensin-converting enzyme inhibitor, ARB – angiotensin II receptor blocker, BMI – body mass index, CHA2DS2-VASc – risk scale to predict stroke and thromboembolism in atrial fibrillation.*

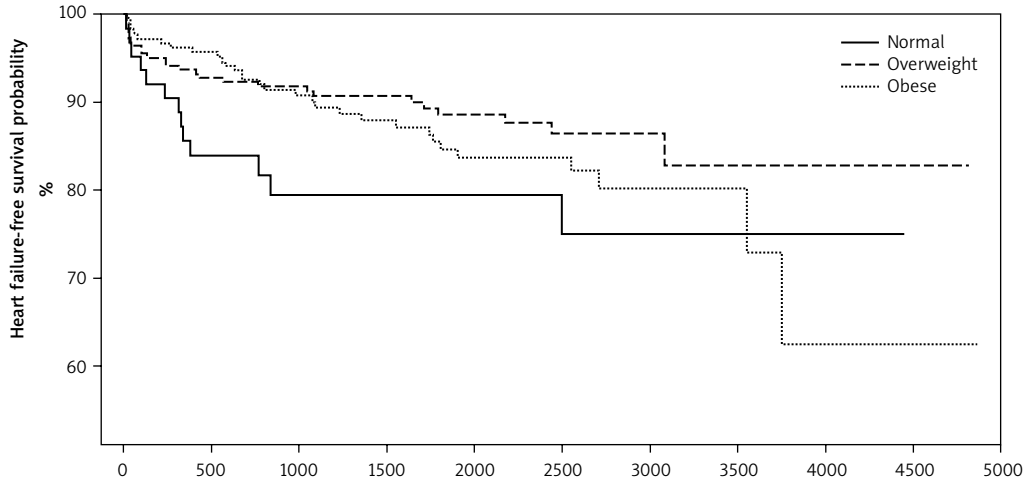
Table 3. Body mass index categories and adverse cardiovascular events in individuals with atrial fibrillation taking anticoagulants

Variable	Normal weight (BMI < 25 kg/m²) (*N* = 63)	Overweight (BMI 25.0–29.9 kg/m²) (*N* = 224)	Obese (BMI ≥ 30 kg/m²) (*N* = 213)	*P*-value
Left atrial appendage thrombus	11 (17.5%)	32 (14.3%)	22 (10.3%)	0.25
Heart failure hospitalization	13 (20.6%)	26 (11.6%)	33 (15.5%)	0.16
Myocardial infarction	1 (1.6%)	6 (2.7%)	4 (1.9%)	0.91
Systemic thromboembolism	1 (1.6%)	1 (0.4%)	1 (0.5%)	0.5
Transient ischaemic attack	2 (3.2%)	1 (0.4%)	2 (0.9%)	0.16
Stroke	0 (0.0%)	7 (3.1%)	6 (2.8%)	0.49
Cardiovascular death	11 (17.5%)	30 (13.4%)	22 (10.3%)	0.29
All-cause death	12 (19.0%)	35 (15.6%)	24 (11.3%)	0.21

BMI – body mass index.

an enhanced risk of new-onset AF in susceptible patients, which appeared consistent in men and women.

The association between BMI categories and adverse cardiovascular outcomes has been studied previously. Grymonprez *et al.* [32] conducted a meta-analysis of observational and randomised studies and found that the overweight and obese groups with AF taking anticoagulation therapy had significantly lower risks of systemic embolism, stroke, and all-cause mortality than the normal-weight group. Similarly, Zhou *et al.* [33] demonstrated a reduced risk of systemic embolism, stroke, and all-cause mortality in overweight or obese individuals with AF who received OAC therapy. Proietti *et al.* [34] conducted a meta-analysis of the ARISTOTLE, RE-LY, and ROCKET AF trials that revealed an obesity paradox in individuals with AF; the overweight and obese groups had a significantly lower risk of stroke/systemic embolic events than those with a normal BMI. Ardestani *et al.* [12] analysed individuals with AF, of whom 87%



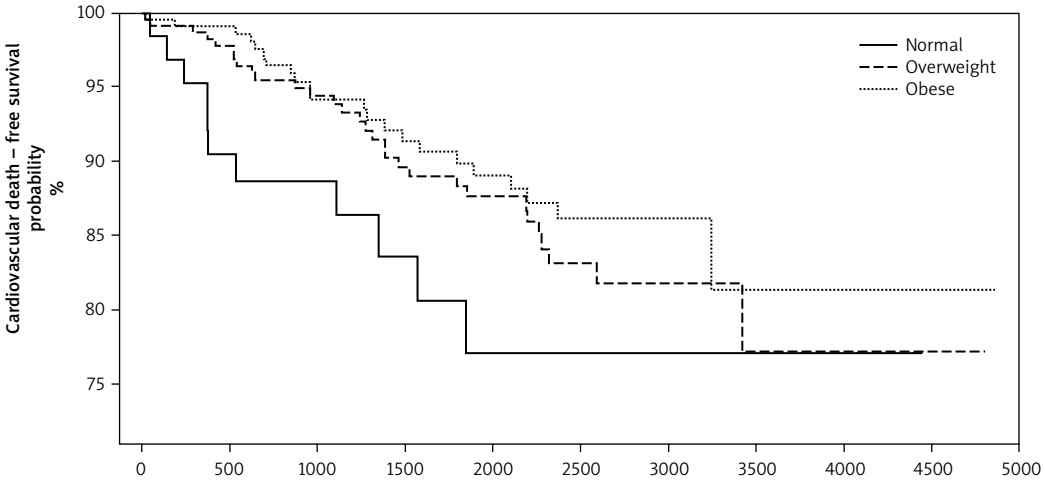
Number at risk

63	46	34	25	21	17	7	4	2	0	0
224	202	167	135	109	63	27	16	4	2	0
213	194	142	114	87	61	21	13	5	3	0

Cumulative number of heart failure events

0	10	12	12	12	13	13	13	13	13	13
0	16	18	20	23	25	25	26	26	26	26
0	10	20	24	29	29	31	31	33	33	33

Figure 1. Kaplan–Meier analysis for heart failure hospitalisation according to body mass index categories (normal, overweight, and obese). The 0-time point on the x-axis indicates the day transoesophageal echocardiography (TEE) was performed



Number at risk

63	52	40	29	22	18	8	4	2	0	0
224	215	177	142	118	70	28	17	5	3	0
213	203	158	128	101	68	25	16	7	4	0

Cumulative number of cardiovascular death

0	6	7	9	11	11	11	11	11	11	11
0	5	12	20	23	28	29	30	30	30	30
0	2	11	15	18	21	21	22	22	22	22

Figure 2. Kaplan–Meier analysis for cardiovascular death according to body mass index categories (normal, overweight, and obese). The 0-time point on the x-axis indicates the day on which transoesophageal echocardiography (TEE) was performed

were taking warfarin anticoagulant therapy across all BMI categories. Their findings indicated that overweight individuals had a significantly reduced risk of cardiovascular death (hazard ratio [HR]: 0.47, p = 0.002), whereas obesity did not negatively affect cardiovascular (p = 0.15) or overall survival (p = 0.3). Similarly, Badheka *et al.* [9] conducted a post hoc analysis of the AFFIRM trial, comprising individuals with AF (approximately 87% taking warfarin across BMI groups), and found that those who were overweight and obese had significantly lower risks of cardiovascular and all-cause mortality than normal-weight individuals. In a prospective study by Wang *et al.* [10] involving 2016 participants with AF or atrial flutter followed up for a mean of 12 months (with only 20% receiving anticoagulation therapy [warfarin]), those with normal and low weights were associated with higher risks of cardiovascular and all-cause mortality than those who were overweight. Agarwal *et al.* [35] analysed a cohort of individuals hospitalised for AF, of whom 15.3% were obese, finding that obesity was associated with significantly lower rates of in-hospital mortality and stroke events, although anticoagulation status was not explored. Zhu *et al.* [36] performed a meta-analysis of nine studies (participants were not fully receiving anticoagulation therapy), reporting that individuals who were underweight (BMI < 18.5 kg/m^2) faced higher risks of cardiovascular death, all-cause mortality, and systemic embolism/stroke than those who were overweight or obese.

Contrasting evidence exists regarding the positive effects of obesity and overweight status on cardiovascular outcomes. Overvad *et al.* [14] analysed a cohort of more than 3000 individuals with AF, of whom only 21.8% received anticoagulation therapy. They reported higher risks of composite endpoints in those who were overweight or obese. Nteli *et al.* [8] observed increased mortality and hospitalisation rates of AF, heart failure, or stroke in individuals across abnormal BMI categories, noting that anticoagulant use was consistent across groups but involved only 60% of the study population. In a retrospective study by Wang *et al.* [15] that included 1286 individuals with AF observed over a median of 2.1 years, 13.3% of the participants were anticoagulated with warfarin. The study found that being overweight was an independent risk factor for ischaemic stroke, obesity was an independent risk factor for thromboembolism, and being underweight was an independent risk factor for all-cause mortality. However, BMI did not significantly affect cardiovascular death, with normal weight serving as the reference category for all comparisons. Similarly, Patti *et al.* [16] examined the influence of BMI on clinical outcomes in individuals with established AF over a 1-year follow-up period, with the majority of participants receiving OACs. Without anticoagulant therapy, obesity was associated with a higher risk of thromboembolic events than a lower BMI; however, anticoagulant therapy effectively equalised the rates of thromboembolic events across all the BMI groups.

In our previous report [4], conducted on the same study population as the present study, we provided Cox proportional hazards regression analyses to correlate demographics and comorbidities with cardiovascular outcomes. They revealed that a lower left ventricular ejection fraction, lower eGFR, greater left atrial diameter, and the presence of LAAT, but not BMI, were associated with a poor prognosis in anticoagulated individuals with AF.

The present study evaluated the relationship between BMI and LAAT in individuals with AF treated with OACs. Findings from previous studies have been inconsistent, reflecting the complexity of this relationship. Uziębło-Życzkowska *et al.* [17] conducted a multicentre study involving 2816 individuals with AF or atrial flutter referred for TEE before cardioversion or ablation, most of whom were receiving anticoagulant therapy. LA/LAA thrombi were detected in 7.9% of the participants; abnormal BMI (overweight or obese) did not significantly increase the prevalence of thrombi compared to normal weight. Similarly, Turek *et al.* [2] analysed 296 individuals with AF taking anticoagulants and found that 14.5% had LAA thrombi, with no association between BMI and thrombus formation. In contrast, Tang *et al.* [18] examined 433 individuals with nonvalvular AF, 6% of whom had LA/LAA thrombi detected using TEE; the prevalence of thrombi was significantly higher in individuals with BMI ≥ 27.0 kg/m^2 (10.6%) than in those with BMI < 27.0 kg/m^2 (3.0%; p = 0.001), with BMI ≥ 27.0 kg/m^2 identified as an independent predictor of LA/LAA thrombus (odds ratio: 4.02, p = 0.025). In addition, 12 (2.8%) individuals received anticoagulant therapy. These findings underscore the need for further research to clarify the impact of BMI on thromboembolic risk in individuals with AF, particularly in those taking anticoagulants.

Several studies have introduced the concept of metabolic status to improve cardiovascular risk stratification. For example, the metabolically healthy obesity phenotype refers to obese individuals who do not exhibit features of metabolic syndrome. In contrast to metabolically healthy obesity, the remaining obese patients are classified as having metabolically unhealthy obesity. However, the concept of metabolically healthy obesity and its association with cardiovascular events remains controversial and has been insufficiently studied in patients with AF, particularly in those receiving anticoagulation therapy.

In a study by Caleyachetty *et al.* [37], obese patients without metabolic abnormalities had a higher risk of cerebrovascular disease, coronary heart disease, and HF than normal-weight patients with no metabolic abnormalities. Additionally, the risk of cerebrovascular disease, coronary heart disease, and HF increased with the number of metabolic abnormali-

ties, regardless of weight status (normal weight, overweight, or obese). In a retrospective study, Fauchier *et al*. [38] compared patients with and without obesity. During recruitment, patients with AF constituted a small percentage of the study participants (less than 10%). In age-, sex-, and smoking-adjusted analyses, obesity was associated with a higher risk of new-onset AF and HF in patients without metabolic abnormalities than in non-obese patients without metabolic abnormalities. Moreover, men with obesity but without metabolic abnormalities had a higher risk of clinical events than non-obese men without metabolic abnormalities, whereas women with obesity but without metabolic abnormalities had a lower risk for most events than non-obese women without metabolic abnormalities. Corica *et al*. [39] conducted a post hoc analysis of the GLORIA-AF registry to evaluate the impact of metabolic status on the risk of clinical events in individuals with established AF, categorised according to BMI (normal weight, overweight, and obese). A higher BMI was associated with a metabolically unhealthy status. Normal-weight metabolically unhealthy patients had a higher risk of the primary composite outcome as well as thromboembolism. Conversely, a lower risk of cardiovascular death and major adverse cardiovascular events was observed in metabolically healthy obese individuals.

Our findings indicate that BMI alone may not be a reliable indicator of adverse events in anticoagulated patients with AF. Despite variations in comorbidities, long-term outcomes were similar across BMI categories. Interestingly, patients with normal weight had a comparable risk of complications as those who were overweight or obese, suggesting that treatment effectiveness and other clinical factors may be more relevant. The small proportion of normal-weight patients in our cohort may reflect broader population trends, underscoring the need for further studies on the complex interaction between BMI, metabolic status, and cardiovascular risk in AF.

This study has several limitations. The sample size was relatively small, and all participants were referred for electrical cardioversion, introducing potential selection bias. BMI was assessed only at baseline, with no follow-up weight data. The use of standard imaging may have led to overdiagnosis of thrombi. Creatinine clearance was estimated using the MDRD formula, which may not fully reflect dabigatran or rivaroxaban adequacy. Adherence to non-vitamin K antagonist anticoagulants was not confirmed by diagnostic testing. We also did not assess LAA function or structure in detail.

Conclusions

In this study, BMI was not a significant predictor of adverse cardiovascular outcomes in patients with AF receiving OACs. Although normal-weight individuals had fewer comorbidities and better echocardiographic profiles, these did not translate into improved long-term outcomes.

Our findings challenge the role of BMI as a standalone risk marker and highlight the importance of additional clinical and echocardiographic factors. Further research should explore the combined impact of BMI, metabolic health, and anticoagulation quality on outcomes in AF.

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Ethical approval

Approval number: 21/2010.

Conflict of interest

The authors declare no conflict of interest.

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