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Review

Predictors of symptomatic atrial fibrillation recurrence and all-cause mortality at 12 months in obese and non-obese patients

Predictores de recurrencia sintomática de fibrilación auricular y mortalidad de cualquier causa a 12 meses en pacientes obesos y no obesos

Shyla Gupta[a,*], Rohan Kalasipudi[b], Praveen Tharusha Gurusinghe[c], Ayush Gautam[c], Ken Huang[d], Edward Dababneh[c], Prithviraj Dhonde[d], María Gabriela Matta[d]

[a] *Faculty of Medicine, University of Ottawa, Ottawa, Canada*
[b] *Faculty of Medicine, St. George's University, Grenada*
[c] *Department of Cardiology, Gold Coast University Hospital, Gold Coast, Australia*
[d] *Department of Cardiology, Townsville University Hospital, Townsville, Australia*

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ABSTRACT

Background and objective: Obesity is a modifiable risk factor for atrial fibrillation (AF); however, its influence on recurrence and mortality remains controversial. This study aimed to compare clinical characteristics and 12-month outcomes between obese and non-obese patients with AF and to identify predictors of recurrence and mortality.

Materials and methods: A retrospective analysis was conducted on 225 patients hospitalized for AF, classified according to obesity (BMI ≥30 kg/m^2). The primary outcome was AF-related rehospitalization within 12 months, and the secondary outcome was all-cause mortality. Clinical, biochemical, and sociodemographic data were collected.

Results: Obesity was present in 43% of patients and was associated with younger age, a higher prevalence of sleep apnea, and hypertriglyceridemia. At 12 months, 24.9% of patients were rehospitalized and 14.0% experienced AF recurrence. Obesity (OR 2.84; 95% CI 1.17–6.90; $p = 0.021$) and excessive alcohol consumption (OR 3.49; 95% CI 1.07–11.41; $p = 0.039$) independently predicted AF recurrence. Advanced age, low socioeconomic status, and hypokalaemia were associated with higher mortality, whereas obesity was linked to a lower risk (OR 0.38; 95% CI 0.16–0.95; $p = 0.038$).

Conclusions: In this cohort, obesity was associated with increased AF recurrence but reduced mortality, supporting the "obesity paradox." Excessive alcohol intake and low socioeconomic status also influenced outcomes, underscoring the need for individualized management strategies.

Palabras clave:
Fibrilación auricular
Australia
Obesidad
Mortalidad por recurrencia

RESUMEN

Antecedentes y objetivo: La obesidad es un factor de riesgo modificable para la fibrilación auricular (FA); sin embargo, su influencia sobre la recurrencia y la mortalidad sigue siendo controvertida. Este estudio tuvo como objetivo comparar las características clínicas y los desenlaces a 12 meses entre pacientes obesos y no obesos con FA, e identificar predictores de recurrencia y mortalidad.

Materiales y métodos: Análisis retrospectivo de 225 pacientes hospitalizados por FA, clasificados según obesidad (IMC ≥30 kg/m^2). El desenlace primario fue la rehospitalización por FA en 12 meses y el secundario, la mortalidad por todas las causas. Se recopilaron datos clínicos, bioquímicos y sociodemográficos.

* Corresponding author.
E-mail address: shyla.gupta@uottawa.ca (S. Gupta).

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Resultados: La obesidad estuvo presente en el 43% de los pacientes y se asoció con menor edad, mayor prevalencia de apnea del sueño e hipertrigliceridemia. A los 12 meses, el 24,9% fue rehospitalizado y el 14,0% presentó recurrencia de FA. La obesidad (OR 2,84; IC95% 1,17–6,90; p = 0,021) y el consumo excesivo de alcohol (OR 3,49; IC95% 1,07–11,41; p = 0,039) predijeron recurrencia de FA. La edad avanzada, el nivel socioeconómico bajo y la hipocalemia se asociaron con mayor mortalidad, mientras que la obesidad se relacionó con menor riesgo (OR 0,38; IC95% 0,16–0,95; p = 0,038).
Conclusiones: En esta cohorte, la obesidad se asoció con mayor recurrencia de FA pero menor mortalidad, apoyando la «paradoja de la obesidad». El consumo excesivo de alcohol y el bajo nivel socioeconómico también afectaron los desenlaces, destacando la necesidad de un manejo individualizado.

Introduction

Atrial fibrillation (AF) is the most common sustained arrhythmia worldwide, affecting over 60 million people and significantly increasing the risk of heart failure, stroke, and mortality – particularly in older adults. Its prevalence is expected to rise by over 60% by 2050 due to population aging and increasing cardiovascular risk factors.[1,2]

Multiple risk factors contribute to AF development, including age, hypertension, diabetes, obstructive sleep apnea, and notably, obesity.[3] Obesity is one of the strongest modifiable predictors of AF, second only to hypertension. A 5-unit increase in body mass index (BMI) raises AF incidence by 30%, and obese individuals have a 50% higher risk than those of normal weight.[4,5] The link between obesity and AF involves structural and electrical atrial remodeling, systemic inflammation, atrial fibrosis, and the paracrine and mechanical effects of epicardial fat on atrial electrophysiology.[6] These changes may also impact treatment response and recurrence after interventions like catheter ablation or drug therapy.[6]

Despite growing interest in the obesity–AF relationship, data specific to Australian populations – especially in regions like North Queensland – are limited.[7,8] Understanding these local trends is essential, as demographic, lifestyle, and healthcare access factors may differ from metropolitan or international cohorts. For example, a study from 2019 showed that 60% of Australian adults were overweight or obese, with 34% overweight and 26% obese.[9] The prevalence varied by geographic remoteness, with adults in regional and rural areas significantly more likely to be affected than those in major cities. These findings underscore notable regional disparities and highlight the limited availability of local evidence on obesity patterns, and hence the obesity–AF relationships in particular regions of Australia.

This study compares the clinical, biochemical, and echocardiographic characteristics of obese versus non-obese patients admitted with AF to a tertiary hospital in North Queensland and examines 12-month outcomes, including AF-related readmission and all-cause mortality. We hypothesized that obesity would be associated with higher AF recurrence but lower short-term mortality in a regional cohort.

Material and methods

Study population and data collection

The study was conducted at Townsville University Hospital, a leading tertiary referral center in North Queensland serving 244,781 persons (2022). The hospital's catchment area encompasses a vast and geographically diverse region, extending from the Cape York Peninsula and Torres Strait Islands in the north to Mount Isa and the Gulf of Carpentaria in the west. Contemporary health surveillance data suggesting that 32% of adults within this region are living with obesity and estimates suggest a 1.6–1.8% prevalence of AF.[10]

This retrospective cohort study employed a retrospective design, utilizing the hospital's patient database for admissions to the coronary care unit and cardiology ward between 2021 and 2022. To assess comorbidity profiles, a five-year look-back period was applied to evaluate the prior medical histories, hospital admissions, and prescription records of first-time AF patients.

All adult patients (aged ≥18 years) admitted to the cardiology department (including the cardiac ward and coronary care unit) with a diagnosis of AF, either as the primary reason for admission or as a secondary diagnosis, were included. Exclusion criteria encompassed patients admitted electively for electrical cardioversion or percutaneous coronary intervention, individuals with atrial flutter, patients requiring inotropic or vasopressor support during hospitalization, and cases of postoperative AF following cardiovascular surgery.

Demographic data collected included age, sex, Indigenous status (Aboriginal and/or Torres Strait Islander), and socioeconomic categorization according to the Socio-Economic Indexes for Areas (SEIFA) index.[11] Clinical data included anthropometric measures such as BMI, cardiovascular risk factors, and prior history of cardiovascular disease. Obesity was defined as a BMI ≥30 kg/m^2.

Alcohol consumption was categorized according to the National Institute on Alcohol Abuse and Alcoholism definitions.[12] Admission laboratory investigations included electrolytes, lipid profile (including LDL cholesterol), renal function, natriuretic peptides (B-type natriuretic peptide, BNP), glycaemic control (hemoglobin A1c), and iron studies (ferritin).

Comorbidities were assessed using standard clinical criteria, including the CHA_2DS_2-VASc and HAS-BLED scores. Echocardiographic data included left ventricular size and mass, left atrial volume, ejection fraction, and the presence of moderate-to-severe valvular disease. In-hospital AF management was documented, including anticoagulation (vitamin K antagonists or NOACs), rate and rhythm control therapies, and cardioversion (pharmacologic or electrical, including TOE-guided procedures).

In addition, in Queensland, patient data are captured through a fully integrated electronic health system that links hospital, outpatient, pathology, pharmacy, and national health records (*ieMR* and *My Health Record*). This system is linked to *The Viewer*, which a secure interface that aggregates data from over 65 Queensland Health applications and provides access to validated information such as demographics, admission and discharge history, medications, radiology and pathology results, and clinical reports. This allows comprehensive longitudinal tracking of admissions, investigations, and outcomes across the public health network, minimizing loss to follow-up and ensuring complete ascertainment of clinical events.

In our study, readmissions and deaths were identified through this integrated electronic system. Deaths occurring in or outside hospital were notified through *My Health Record* and recorded within ieMR. Patients without events were traced through these systems until the pre-specified study cutoff date.

Outcomes

The primary outcome of this study was AF recurrence, defined as hospital readmission within 12 months of the index admission for symptoms attributable to AF, including but not limited to tachyarrhythmia-related palpitations, symptomatic tachycardia, or heart failure exacerbation secondary to AF. Asymptomatic AF recurrences identified solely through ambulatory or device-based monitoring were not assessed in this analysis.

The secondary outcome was all-cause mortality occurring within 12 months following the index hospitalization.

Ethical considerations

The research adhered to the guidelines for medical investigations outlined in the Declaration of Helsinki, the Good Clinical Practice Guidelines, and the current ethical regulations and was approved by TUH AQUIRE.

Statistical analysis

The frequency and distribution of variables were initially examined overall and then stratified by obesity status. For continuous variables with a normal distribution, the standard deviation was used to assess dispersion. For non-normally distributed continuous variables, the median represented central tendency, and percentiles were used to describe dispersion.

To compare groups based on obesity status, categorical variables were analyzed using the chi-square test, with Fisher's exact test applied when appropriate due to small sample sizes. For continuous variables, Student's *t*-test was used for normally distributed data, and the Wilcoxon rank-sum test was applied for non-normally distributed data.

To identify independent predictors of adverse outcomes – including 12-month readmission and all-cause mortality – a multivariable logistic regression model was constructed using the entire study cohort. Variables that showed a significant association in the bivariate analysis were included in the model. This allowed us to evaluate whether obesity, among other clinical and biochemical parameters, was an independent predictor of these outcomes. The model was adjusted for potential confounders such as age at admission, socioeconomic status and comorbidities. Results were reported as odds ratios (ORs) with 95% confidence intervals (CIs) for both recurrence and mortality.

All statistical analyses were two-tailed, and a p-value of <0.05 was considered statistically significant. Analyses were conducted using Stata Statistical Software, version 14.0 (StataCorp. 2015. Stata Statistical Software: Release 14. College Station, TX: StataCorp LP).

Results

Of the 225 patients admitted with AF, 43% were classified as obese (BMI $\geq$30 kg/m^2). Baseline clinical, biochemical, and echocardiographic characteristics of obese and non-obese patients are summarised in Table 1 .

Obese patients were significantly younger than their non-obese counterparts (66.2 $\pm$ 12.0 vs. 71.6 $\pm$ 12.6 years, $p = 0.0017$). Obstructive sleep apnoea was markedly more prevalent in the obese group (34.0% vs. 4.7%, $p < 0.001$), and heart failure was more commonly the primary admission diagnosis among obese patients (59.8% vs. 39.8%, $p = 0.020$). This group also had higher bleeding risk (HAS-BLED $\geq$3: 40.2% vs. 26.6%, $p = 0.030$), elevated HbA1c (6.84 $\pm$ 1.53% vs. 6.18 $\pm$ 1.03%, $p = 0.0259$), and higher triglyceride levels (1.50 $\pm$ 0.84 vs. 1.10 $\pm$ 0.57 mmol/L, $p = 0.0177$).

Echocardiographic assessment revealed that obese patients had larger left ventricular end-diastolic diameters (51.9 $\pm$ 7.6 vs. 49.3 $\pm$ 8.3 mm, $p = 0.038$), thicker interventricular septum (9.86 $\pm$ 2.47 vs. 9.14 $\pm$ 2.00 mm, $p = 0.039$), and smaller indexed left atrial volumes (43.4 $\pm$ 14.8 vs. 49.7 $\pm$ 20.7 mL/m^2, $p = 0.032$). No significant differences were observed in LVEF or in-hospital management strategies (Supplementary Table 1).

Table 1
Baseline clinical, biochemical, and echocardiographic characteristics of obese and non-obese patients with atrial fibrillation.

Variable	Obese (n = 97)	Non-obese (n = 128)	p-Value
Demographics			
Age, years	66.2 $\pm$ 12.0	71.6 $\pm$ 12.6	0.0017*
Male sex, n (%)	54 (55.7%)	78 (60.9%)	0.63
Low IRSAD, n (%)	29 (29.9%)	56 (43.7%)	0.003
Risk factors			
Hypertension, n (%)	71 (73.2%)	84 (65.6%)	0.22
Diabetes, n (%)	33 (34.0%)	31 (24.2%)	0.11
Current smoker, n (%)	11 (11.3%)	20 (15.6%)	0.58
Alcohol use, n (%)			0.67
Never	60 (61.9%)	73 (57.0%)	
Ex-drinker	8 (8.2%)	12 (9.4%)	
Moderate	21 (21.6%)	25 (19.5%)	
Excessive/binge	8 (8.2%)	18 (14.0%)	
Obstructive sleep apnoea, n (%)	33 (34.0%)	6 (4.7%)	<0.001*
CAD, n (%)	33 (34.0%)	47 (36.7%)	0.89
Heart failure, n (%)	21 (21.6%)	28 (21.9%)	–
CKD, n (%)	24 (24.7%)	40 (31.3%)	0.28
CHA_2DS_2-VASc ≥2, n (%)	65 (67.0%)	91 (71.1%)	0.51
HAS-BLED ≥3, n (%)	39 (40.2%)	34 (26.6%)	0.03*
In-hospital characteristics			
Severe electrolyte imbalance, n (%)	10 (10.4%)	8 (6.3%)	0.53
AF on discharge, n (%)	38 (40.0%)	52 (41.3%)	0.85
Length of stay, days (median, IQR)	4 (1–7)	3 (2–6)	0.70
Biochemical characteristics			
Triglycerides (mmol/L)	1.50 $\pm$ 0.84	1.10 $\pm$ 0.57	0.018*
LDL cholesterol (mmol/L)	2.25 $\pm$ 1.01	2.34 $\pm$ 1.06	0.71
eGFR (mL/min/1.73 m^2)	67.27 $\pm$ 19.54	62.85 $\pm$ 21.49	0.12
HbA1c (%)	6.84 $\pm$ 1.53	6.18 $\pm$ 1.03	0.026*
BNP (pg/mL)	411 $\pm$ 441	1844 $\pm$ 2692	0.095
Ferritin (μg/L)	149.41 $\pm$ 117.43	249.85 $\pm$ 277.93	0.072
Echocardiographic parameters			
LVEF (%)	44.0 $\pm$ 15.1	42.7 $\pm$ 15.3	0.54
LV end-diastolic diameter (mm)	51.9 $\pm$ 7.6	49.3 $\pm$ 8.3	0.038*
LV posterior wall thickness (mm)	9.63 $\pm$ 1.90	9.25 $\pm$ 1.71	0.19
IV septum thickness (mm)	9.86 $\pm$ 2.47	9.14 $\pm$ 2.00	0.039*
Left atrial indexed volume (mL/m^2)	43.4 $\pm$ 14.8	49.7 $\pm$ 20.7	0.032*

Data are presented as mean $\pm$ standard deviation or number (%). Statistical comparisons were performed using chi-square or Fisher's exact test for categorical variables, and Student's *t*-test or Wilcoxon rank-sum test for continuous variables, as appropriate. *Abbreviations*: IRSAD: Index of Relative Socio-economic Advantage and Disadvantage; OSA: obstructive sleep apnoea; CAD: coronary artery disease; HF: heart failure; CKD: chronic kidney disease; AMI: acute myocardial infarction; AF: atrial fibrillation; BNP: B-type natriuretic peptide; eGFR: estimated glomerular filtration rate; LVEF: left ventricular ejection fraction; LV: left ventricle; IV: interventricular.

* $p < 0.05$.

Twelve-month outcomes: predictors of AF recurrence and all-cause mortality

Within the 12-month follow-up, 56 patients (24.9%) were readmitted: 31 (14.0%) for symptomatic AF recurrence and 25 (11.1%) for either coronary artery disease or heart failure exacerbation. Stroke was infrequent (1.35%).

Table 2
Multivariable logistic regression analysis of predictors for atrial fibrillation recurrence at 12 months.

Variable	Odds ratio (95% CI)	*p*-Value
Age at admission (years)	1.01 (0.98–1.05)	0.455
Male sex	0.73 (0.30–1.75)	0.476
Obstructive sleep apnoea	1.08 (0.38–3.04)	0.882
Obesity	2.84 (1.17–6.90)	0.021
Diabetes mellitus	0.72 (0.27–1.89)	0.499
Hypertension	0.63 (0.26–1.53)	0.311
Moderate left atrial dilation	1.34 (0.52–3.50)	0.546
Excessive alcohol use or binge drinker	3.49 (1.07–11.41)	0.039
Chronic kidney disease (CKD)	0.61 (0.22–1.68)	0.339
Severe left ventricular dysfunction	0.80 (0.31–2.03)	0.632
Moderate to severe valvular disease	0.81 (0.26–2.54)	0.719

Obesity: body mass index (BMI) ≥30 kg/m^2; moderate LA dilation: left atrial volume index >41 mL/m^2; excessive alcohol use: includes binge drinking or heavy alcohol consumption; chronic kidney disease: estimated glomerular filtration rate (eGFR) <30 mL/min/1.73 m^2; severe LV dysfunction: severe reduction in left ventricular ejection fraction.

Table 3
Logistic regression analysis of factors predicting mortality: odds ratios, confidence intervals, and *p*-values.

Variable	Odds ratio (OR)	95% confidence interval (CI)	*p*-Value
Age at admission	1.055	1.012–1.099	0.011
Male sex	1.658	0.696–3.950	0.254
Obesity	0.384	0.155–0.950	0.038
Diabetes	1.492	0.628–3.545	0.365
Hypertension	1.859	0.601–5.747	0.282
Low IRSAD	2.648	1.155–6.073	0.021
Severe LV dysfunction	2.185	0.938–5.090	0.070
Electrolyte imbalance	2.241	1.257–3.995	0.006
CKD	1.571	0.686–3.596	0.285

Low IRSAD quintiles 4 and 5 (most disadvantaged), severe LV dysfunction: severe reduction in left ventricular ejection fraction, electrolyte imbalance (hypokalemia or hypomagnesemia on index admission), CKD: chronic kidney disease: estimated glomerular filtration rate (eGFR) <30 mL/min/1.73 m^2.

Overall, 40 patients (17.8%) died during follow-up. Causes of death included malignancies (e.g., metastatic lung, pancreatic, or bladder cancer), cardiovascular events (advanced heart failure, malignant arrhythmias), sepsis, and cerebrovascular events.

Multivariable logistic regression analysis (Table 2) identified obesity (OR 2.84, 95% CI 1.17–6.90, $p = 0.021$) and excessive alcohol use or binge drinking (OR 3.49, 95% CI 1.07–11.41, $p = 0.039$) as independent predictors of AF recurrence after adjustment for age, sex, diabetes, hypertension, moderate left atrial enlargement (>41 mL/m^2), obstructive sleep apnoea, chronic kidney disease, severe LV dysfunction, and valvular disease.

Significant predictors of all-cause mortality included older age (OR 1.06 per year, 95% CI 1.01–1.10, $p = 0.011$), lower socioeconomic status (IRSAD category 4 and 5: OR 2.65, 95% CI 1.16–6.07, $p = 0.021$), and hypokalaemia (OR 2.24, 95% CI 1.26–4.00, $p = 0.006$). Severe LV dysfunction trended toward significance (OR 2.19, $p = 0.070$). In contrast, obesity was independently associated with a lower risk of all-cause mortality (OR 0.38, 95% CI 0.16–0.95, $p = 0.038$) (Table 3).

Discussion

This study provides novel insights into the complex relationship between obesity and AF in a regional Australian population. Among patients admitted with AF, obesity was highly prevalent (43%) and independently associated with a nearly threefold increase in the risk of symptomatic AF recurrence at 12 months. Paradoxically, it was also the only independent factor associated with reduced all-cause mortality in the cohort. Excessive alcohol use emerged as the strongest predictor of recurrence, while lower socioeconomic status was independently associated with higher mortality. Obese patients were younger, more likely to have obstructive sleep apnoea, and had elevated bleeding and metabolic risk profiles. Despite having larger absolute cardiac dimensions, they showed smaller indexed left atrial volumes.

The association between alcohol use and AF recurrence reinforces its role as a modifiable trigger of arrhythmia.[13–16] Prior studies have identified heavy alcohol consumption as a key driver of both new-onset and recurrent AF, mediated by autonomic dysregulation, atrial myocyte toxicity, and pro-inflammatory mechanisms.[13–16] In our cohort, excessive alcohol use conferred a more than threefold increased risk of AF recurrence, underscoring the importance of addressing alcohol consumption in both inpatient management and long-term risk reduction strategies.

Obesity contributes to AF recurrence through a constellation of interrelated pathophysiological mechanisms.[17] Excess adiposity results in hemodynamic changes, including increased blood volume and cardiac output, leading to elevated left atrial pressures and atrial stretch.[18–20] These changes promote atrial fibrosis and provide the substrate for reentrant arrhythmias. In parallel, systemic inflammation seen in obesity, with elevated circulating cytokines such as interleukin-6 and TNF-alpha, drives electrical remodelling via fibroblast activation and altered ion channel expression.[18–20] Autonomic imbalance and associated comorbidities, particularly obstructive sleep apnoea and insulin resistance, further contribute to an arrhythmogenic milieu.[18–20]

Epicardial adipose tissue (EAT) may play a central role in mediating these effects. As a metabolically active visceral fat depot in direct contact with the atrial myocardium, EAT exerts paracrine and vasocrine influence, secreting pro-inflammatory and profibrotic mediators that promote atrial remodelling.[19,20] Recent imaging studies have linked increased EAT volume with conduction slowing and recurrence after AF ablation, highlighting its relevance as a local risk marker independent of BMI.[21] These findings support a growing view that body fat distribution – rather than global adiposity alone – may better capture arrhythmia risk.

Interestingly, although left atrial volume index (LAVI) is a well-established predictor of AF recurrence, we did not observe this association in our cohort.[22–25] Obese patients demonstrated paradoxically lower LAVI values, despite larger absolute chamber sizes. This discrepancy likely reflects the limitations of BSA-based indexing in obesity, where standard methods may underestimate true atrial size. Alternative approaches, such as indexing to height or ideal body weight, may offer greater accuracy in assessing atrial remodeling and recurrence risk in this population.

Despite its association with recurrence, obesity was independently associated with reduced 12-month all-cause mortality, with an OR of 0.384. This supports the so-called “obesity paradox,” previously described in AF, coronary artery disease, and heart failure.[26–29] Several hypotheses have been proposed, including younger age at presentation, greater metabolic reserve, lower prevalence of cachexia or smoking, and more aggressive pharmacologic treatment due to higher baseline blood pressures.[30–32] Moreover, BMI itself has known limitations – it does not differentiate fat from muscle, nor does it account for regional adiposity or body composition.[33] As a result, BMI-defined obesity may reflect a heterogeneous group, some of whom possess protective physiologic characteristics.[33]

Socioeconomic disadvantage was independently associated with increased mortality in our cohort. Interestingly, it was more prevalent

among non-obese patients, suggesting that lower socioeconomic status alone – regardless of obesity – may drive poorer outcomes. Low socioeconomic status is associated with a higher burden of comorbidities, reduced access to care, and increased exposure to behavioural and environmental risk factors.[34,35] These structural inequities likely contribute to both the development of atrial fibrillation and its adverse outcomes, independent of obesity.[36,37]

Altogether, these findings highlight the need for a nuanced approach to obesity in AF care. Rather than viewing obesity solely as a risk factor, clinicians should consider the complex interplay between fat distribution, inflammatory burden, comorbidities, and socioeconomic context. Public health interventions must extend beyond individual lifestyle advice to address upstream drivers of obesity through environmental, structural, and policy-level strategies. Clinically, improved risk stratification tools – such as imaging of epicardial fat or alternative indexing methods for cardiac chamber size – may help guide more tailored management in obese patients with AF.

This study underscores the importance of personalized AF management in obese individuals, who face a heightened risk of symptomatic recurrence. Enhanced rhythm surveillance, ambulatory monitoring, and early rhythm control strategies may help prevent adverse atrial remodeling and improve outcomes in this population.

Although obesity was paradoxically associated with lower all-cause mortality, this should not be interpreted as protective per se. Instead, it highlights the importance of comprehensive cardiometabolic care that targets the broader systemic risks and comorbidities associated with excess adiposity.

Future research should aim to elucidate the biological mechanisms underlying the obesity paradox through prospective studies incorporating detailed body composition analysis, inflammatory biomarkers, and measures of cardiorespiratory fitness. Randomized trials evaluating the effect of structured weight loss and lifestyle interventions on both AF recurrence and survival are also warranted. Incorporating novel imaging-based metrics – such as epicardial adipose tissue volume and activity – into clinical risk models may further improve individualized care in this group.

This study has several limitations. Its retrospective design and modest sample size may limit generalisability and introduce selection or information bias. Although multivariable adjustment was applied, residual confounding is possible, particularly from unmeasured factors such as physical activity, dietary patterns, and health literacy. Socioeconomic status was assessed using the IRSAD index, but other social determinants of health were not comprehensively captured.

In addition, the primary endpoint – AF recurrence – was defined as hospital readmission for symptomatic AF. While this reflects clinically significant events, it does not capture asymptomatic or device-detected recurrences. As such, the findings pertain to predictors of symptomatic and healthcare-utilizing recurrence, rather than total arrhythmia burden.

Conclusion

In this North Queensland cohort, obesity was independently associated with an increased risk of symptomatic AF recurrence yet paradoxically conferred a survival advantage. Excessive alcohol consumption and socioeconomic disadvantage also independently impacted clinical outcomes. These findings underscore the need for individualized, risk-based AF management and further investigation into the biological and social drivers of the obesity paradox.

CRediT authorship contribution statement

Conception and design of the project: Maria Gabriela Matta MD, MSc.

Data collection: Praveen Tharusha Gurusinghe MBBS, Ayush Gautam MBBS, Ken Huang MD.

Data analysis and interpretation: Maria Gabriela Matta MD MSc.

Drafting the article: Maria Gabriela Matta MD, MSc., Shyla Gupta BHSc, Rohan Kalasipudi BSc, Edward Dababneh MD.

Drafting the tables/figures: Raj Dondhe, Rohan Kalasipudi BSc.

Critical revision of the article: Maria Gabriela Matta MD MSc, Shyla Gupta BHSc, Rohan Kalasipudi BSc, Edward Dababneh MD.

Ethical approval

Our research was based on existing data and received approved from the TUH AQUIRE.

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Conflict of interest

Nothing to declare.

Data availability

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

Appendix A. Supplementary data

Supplementary data associated with this article can be found in the online version available at https://doi.org/10.1016/j.medcle.2026.107290.

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