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CLINICAL RESEARCH

Semaglutide as adjunctive therapy to catheter ablation in obesity-related paroxysmal atrial fibrillation

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Aims Obesity adversely affects atrial fibrillation (AF) outcomes and is associated with higher recurrence after catheter ablation. Glucagon-like peptide-1 receptor agonists (GLP-1RAs) promote weight loss and improve metabolic inflammation, but their role as adjuncts to ablation has not been completely defined. This study investigated the impact of semaglutide on post-ablation rhythm outcomes in obese patients with AF.

Methods and results This single-centre, propensity-matched study included obese patients [body mass index (BMI) ≥ 30 kg/m²] undergoing first-time catheter ablation for paroxysmal AF (2019–2024). Patients who initiated semaglutide within 3 months before or 1 month after ablation were compared with matched controls who did not receive GLP-1RA therapy. All patients underwent continuous rhythm monitoring using implantable cardiac monitors. The primary endpoint was any atrial tachyarrhythmia recurrence beyond a 2-month blanking period. The final cohort included 181 semaglutide-treated patients and 181 controls with matched clinical and procedural characteristics. At 18-month follow-up, freedom from recurrence was 80.2% vs. 65.2%; semaglutide was associated with a significantly lower risk of recurrence (hazard ratio 0.52; 95% confidence interval 0.34–0.78; $P = 0.002$). Weight and BMI decreased significantly in the semaglutide group (−11.8 ± 3.8 kg; −4.0 ± 1.4 kg/m²) compared with controls (−1.9 ± 1.2 kg; −0.3 ± 0.8 kg/m²; both $P < 0.001$). A substantial proportion of treated patients achieved ≥10% weight reduction.

Conclusion Glucagon-like peptide-1 receptor agonist therapy using semaglutide is associated with a reduced risk of AF recurrence in obese patients undergoing AF catheter ablation, indicating its potential as an adjunctive treatment. Further studies are needed to confirm these findings and elucidate the effects of GLP-1RA on AF recurrence.

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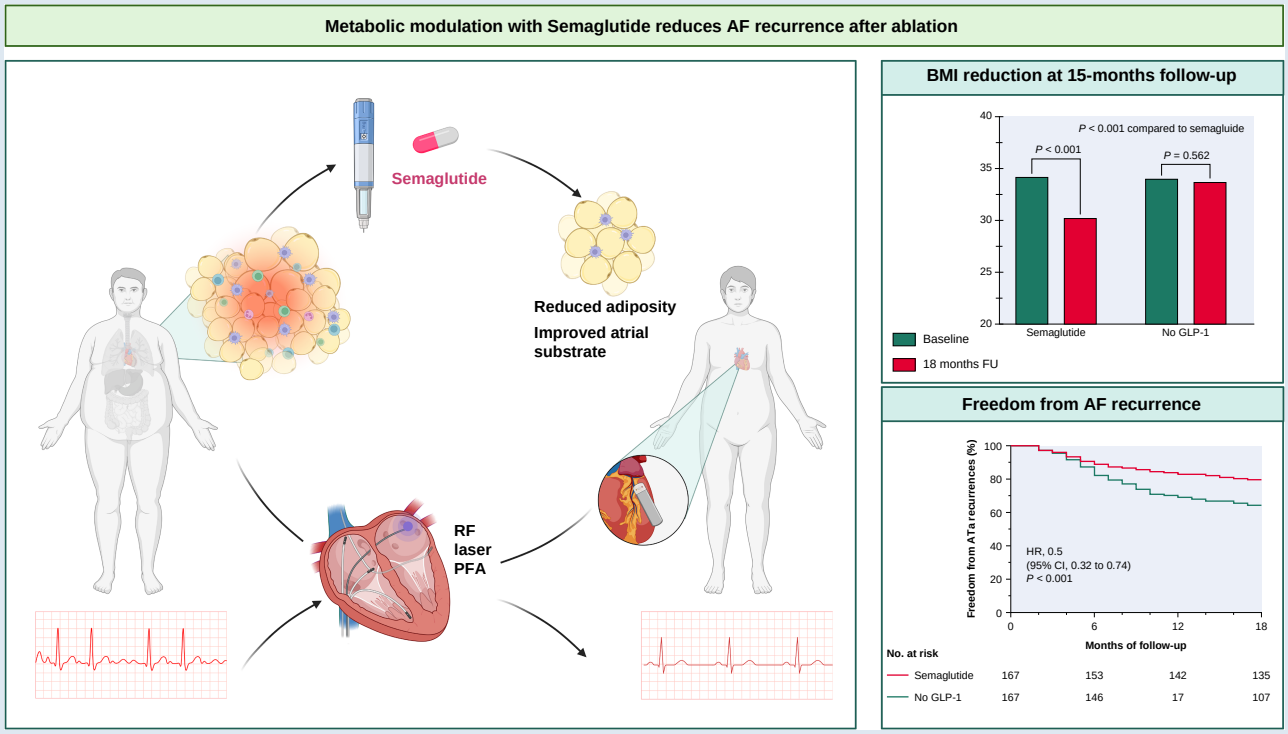
Graphical Abstract



Keywords Atrial fibrillation • Catheter ablation • Obesity • Implantable cardiac monitors • Semaglutide • GLP-1 receptor agonist

What's new?

- Glucagon-like peptide-1 receptor agonist therapy using semaglutide is associated with a significant reduction in post-ablation AF recurrence in predominantly non-diabetic obese patients.
- Neoadjuvant and adjuvant semaglutide therapy may provide a synergistic therapeutic window potentially improving long-term atrial fibrillation (AF) rhythm-control management.
- The observed antiarrhythmic benefit was paralleled by substantial weight loss supporting metabolic modulation as an adjunct to catheter ablation in obesity-related AF.

Introduction

Obesity represents a major modifiable risk factor for the development and progression of atrial fibrillation (AF).[1,2] The relationship between excess adiposity and AF extends beyond epidemiological association, encompassing structural, electrical, and metabolic alterations that promote atrial remodelling and arrhythmia persistence.[3–5] Increased visceral and epicardial fat volumes contribute to local inflammation, oxidative stress, and interstitial fibrosis, ultimately facilitating AF initiation and its progression from paroxysmal to persistent forms.[6,7]

Despite continuous technological advances in catheter ablation, including refined mapping systems and improved energy delivery strategies, obese patients remain a challenging population.[8,9] They consistently exhibit higher rates of arrhythmia recurrence, procedural complications, and less durable rhythm control compared with non-obese individuals.[10] These findings emphasize the intrinsic limitations of ablation when performed in the context of obesity-related atrial disease and underscore the need for comprehensive strategies aimed at modifying the underlying metabolic substrate. Metabolic optimization and weight reduction have been shown to improve long-term ablation outcomes, highlighting the importance of addressing obesity as a therapeutic target in rhythm control. Interventions capable of reducing visceral adiposity and attenuating atrial inflammation may therefore represent a pivotal adjunct to ablation therapy in this high-risk population.

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) have emerged as highly effective therapies for weight reduction and cardiometabolic risk improvement in both diabetic and non-diabetic individuals.[11,12] Beyond their systemic metabolic effects, GLP-1RA therapy has been associated with reductions in epicardial fat thickness, inflammatory activation, and oxidative stress, mechanisms that are mechanistically linked to atrial remodelling and AF progression.[13] Recent observational studies have hinted at a possible reduction in incident AF among patients treated with GLP-1RA,[14,15] but their role in the setting of catheter ablation remains insufficiently investigated. Importantly, no prior study has specifically evaluated semaglutide, the most potent and widely adopted GLP-1RA, in a well-defined obese cohort undergoing first-time AF ablation with continuous rhythm monitoring.[16,17] Therefore, the present study aimed to evaluate the role of semaglutide therapy in a cohort of obese patients undergoing AF catheter ablation.

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Methods

Study design and patient population

This single-centre study was conducted at a high-volume tertiary Italian centre specialized in AF ablation. This analysis used data from a prospective institutional registry of patients undergoing catheter ablation for paroxysmal AF at the same institution. The institutional AF ablation registry is approved by the local ethics committee. All consecutive obese patients [body mass index (BMI) ≥ 30 kg/m²] who underwent first-time AF ablation between January 2019 and December 2024 were screened for inclusion. Among them, patients who had initiated therapy with semaglutide within 3 months before or within 1 month after the ablation procedure constituted the treatment cohort. From the same registry, a control cohort was derived among obese patients who underwent ablation during the same period but had never received GLP-1RA therapy. Enrolment was continuous across the study period, with overlapping inclusion dates in both groups. Patients who did not meet either exposure definition were not included in the analytical cohort. To ensure a balanced comparison between groups, propensity score matching (PSM) was performed, as detailed in the Statistical Analysis section. Exclusion criteria included age <18 years, persistent AF, previous AF ablation, hyperthyroidism, severe left atrial enlargement, intracardiac thrombus, significant valvular disease, uncontrolled heart failure, contraindications to oral anticoagulation, recent myocardial infarction or unstable angina, coronary artery bypass grafting within 6 months, pregnancy, active infection, or major comorbidities, such as malignancy, end-stage renal disease requiring dialysis, severe chronic obstructive pulmonary disease, or cirrhosis. The study protocol complied with the principles of the Declaration of Helsinki.

Ablation procedure

All catheter ablation procedures were performed according to current international guidelines for AF management.[18] The choice of energy source was at the discretion of the operator and included radiofrequency ablation using the QDOT Micro™ catheter (Biosense Webster, Irvine, CA, USA),[19] third-generation endoscopic laser balloon ablation with the HeartLight X3™ system (Boston Scientific, Marlborough, MA, USA),[20] and pulsed field ablation (PFA) using either the Farawave™ catheter (Farapulse™, a Boston Scientific company, Menlo Park, CA, USA) or the Varipulse™ catheter (Biosense Webster, Irvine, CA, USA).[21–23] Pulmonary vein ablation aiming at their electrical isolation was the primary procedural endpoint in all patients. All procedures were performed under fluoroscopic tridimensional mapping system guidance when indicated. Heparin was administered as a bolus followed by continuous infusion to maintain an activated clotting time above 300 s throughout the procedure. Patients remained under continuous telemetry monitoring during hospitalization and underwent routine electrocardiographic assessments before discharge.

Glucagon-like peptide-1 receptor agonist therapy

As part of routine clinical management at our centre, all obese patients receive standardized counselling on lifestyle modification and weight reduction according to contemporary recommendations for AF risk factor management. Nutritional advice, physical activity encouragement, and weight control strategies are always uniformly provided. All patients in the treatment group received semaglutide, with the formulation and dosage adjusted based on individual patient characteristics. It was administered either subcutaneously at weekly doses ranging from 0.25 to 1 mg or orally at daily doses between 3 and 14 mg, according to standard clinical indications and tolerability. In the control group, weight loss was recommended as part of routine clinical management, given its role as a modifiable risk factor for AF recurrence. Any dose adjustments, treatment discontinuation, and adverse events were systematically documented during routine follow-up visits as part of standard clinical care.

Data collection and study outcomes

Baseline characteristics, including demographic data, comorbidities, medication use, and echocardiographic parameters, were recorded at the time of the ablation procedure. The primary outcome of the study was the recurrence of any atrial tachyarrhythmia (ATa), including AF, atrial flutter, or atrial tachycardia, lasting at least 30 s after a 2-month blanking period. Continuous rhythm monitoring was achieved using implantable cardiac monitors (ICMs) in all patients. Those without a pre-existing device received an ICM in the peri-ablation window (within 1–2 months before or after the procedure) according to institutional protocol. This strategy ensured homogeneous rhythm surveillance, including asymptomatic events, and reduced detection bias between groups. Secondary outcomes included weight reduction and BMI reduction, given their potential role in modulating AF recurrence, as well as ATa burden and the separate evaluation of atrial flutter and atrial tachycardia episodes during follow-up. Atrial tachyarrhythmia burden was defined according to the EHRA consensus statement[24] as the proportion of time spent in ATa expressed as a percentage of the ICM recording time during specified follow-up windows. Burden was calculated cumulatively from the end of the 2-month blanking period to 6, 12, and 18 months post-ablation, using continuous ICM data in all patients. Distribution of burden at 18 months was categorized as 0%, <0.1%, 0.1–1%, 1–5%, 5–10%, and >10% among the entire matched cohort. Safety outcomes included the incidence of major cardiovascular events, hospitalizations for HF, and adverse effects potentially related to semaglutide use. As per standard practice at our centre, follow-up visits were routinely scheduled at 3, 6, and 12 months after ablation, with additional visits performed thereafter based on clinical indications. During these visits, clinical assessments, electrocardiographic evaluations, and transthoracic echocardiography were routinely performed to monitor arrhythmia recurrence and assess overall patient status. Antiarrhythmic drugs were routinely continued throughout the blanking period and, in the absence of documented recurrence, discontinued thereafter. Patients were followed with clinical visits and a 12-lead ECG at 3, 6, and 12 months post-procedure and subsequently at the discretion of the treating cardiologist. Automatic transmissions from the ICM were obtained periodically or in the event of an arrhythmic event recording. Implantable cardiac monitor-detected ATa episodes were reviewed and adjudicated by a cardiologist–electrophysiologist to confirm arrhythmia diagnosis and exclude artefacts.

Statistical analysis

Continuous variables were expressed as mean ± standard deviation (SD) and compared using the Student's *t*-test or the Mann–Whitney *U* test, as appropriate. Categorical variables were presented as absolute numbers and percentages and compared using the χ^2 test or Fisher's exact test, as applicable. To improve comparability between groups, a PSM approach was applied. The propensity score was estimated using a logistic regression model including clinically relevant baseline variables potentially associated with treatment allocation (see Supplementary material for the full covariate list). Patients treated with semaglutide were matched 1:1 to control patients using nearest-neighbour matching without replacement within a caliper of 0.2 SDs of the logit of the propensity score. Covariate balance before and after matching was evaluated using standardized mean differences (SMDs) and visually summarized using a Love plot (see Supplementary material online, *Table S1* and *Figure S1*). All eligible patients were included in the analysis. Treatment group assignment was defined at the time of PSM and remained fixed during follow-up. Follow-up started on the ablation date. We used follow-up through the earlier of 18 months or the last available follow-up assessment. Discontinuation of semaglutide did not trigger censoring. Survival analysis for arrhythmia recurrence was conducted using Kaplan–Meier curves, with differences between groups assessed by the log-rank test. Cox proportional hazards regression was used to estimate hazard ratios (HRs) with 95% confidence intervals (CIs) for the association between treatment group and arrhythmia recurrence. The proportional hazards assumption was tested and met. Models accounted for the

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Table 1 Baseline and clinical characteristics of the study population

Variable	Semaglutide (*n* = 181)	No GLP-1RA (*n* = 181)	SMD
Age, years	56.8 ± 10.1	58.3 ± 9.1	0.16
Male, *n* (%)	132 (73)	128 (71)	0.05
Body weight, kg	101.0 ± 11.8	101.9 ± 10.5	0.08
BMI, kg/m²	34.2 ± 4.4	34.0 ± 3.8	0.05
Comorbidities, *n* (%)			
Heart failure	12 (7)	16 (9)	0.07
CAD	17 (9)	24 (13)	0.13
Previous stroke or TIA	14 (8)	18 (10)	0.08
Hypertension	129 (71)	107 (59)	0.24
Dyslipidaemia	73 (40)	76 (42)	0.04
COPD	23 (13)	20 (11)	0.06
T2DM	65 (36)	63 (35)	0.02
OSA	41 (23)	45 (25)	0.05
CKD	16 (9)	20 (11)	0.07
Echo findings			
LVEF, %	58.0 ± 7.4	57.3 ± 6.7	0.10
LA volume/BSA, mL/m²	34.7 ± 7.2	33.8 ± 5.6	0.14
Moderate LA enlargement, *n* (%)	50 (28)	47 (26)	0.04
Moderate MR, *n* (%)	33 (18)	29 (16)	0.05
Drug therapy, *n* (%)			
Oral anticoagulation	146 (81)	154 (85)	0.11
SGLT2i	39 (22)	44 (24)	0.05
Statins	65 (36)	62 (34)	0.04
Any AAD at baseline	112 (62)	109 (60)	0.02
Class Ic	94 (52)	92 (51)	0.02
Amiodarone	12 (7)	13 (7)	0.02
Sotalol	6 (3)	4 (2)	0.06
Time from first AF episode, months	38.3 ± 26.8	37.6 ± 26.1	0.03
AF-related ED or clinic visits, no.	2.8 ± 1.4	2.9 ± 1.8	0.06

Data are reported as mean ± standard deviation for continuous variables or number (percentage) for categorical variables.
AAD, antiarrhythmic drug; AF, atrial fibrillation; BMI, body mass index; BSA, body surface area; CAD, coronary artery disease; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; ED, emergency department; GLP-1RA, glucagon-like peptide-1 receptor agonist; LA, left atrium; LVEF, left ventricular ejection fraction; MR, mitral regurgitation; OSA, obstructive sleep apnoea; SGLT2i, sodium–glucose cotransporter 2 inhibitors; SMD, standardized mean difference; TIA, transient ischaemic attack; T2DM, Type 2 diabetes mellitus.

matched design using robust standard errors clustered by matched pair identifier. Due to a 0.24 SMD in hypertension after matching, we included hypertension as an additional covariate in the Cox model. Sensitivity analyses without hypertension adjustment and with further adjustment for all propensity score covariates are reported in the Supplementary material. All statistical analyses were conducted using R software (R Core Team, 2023, R Foundation for Statistical Computing, Vienna, Austria) and GraphPad Prism (GraphPad Software, San Diego, CA). A two-tailed *P*-value < 0.05 was considered statistically significant.

Results

Baseline characteristics

Between March 2019 and December 2024, we screened 587 consecutive patients with obesity (BMI ≥ 30 kg/m²) who underwent first-time catheter ablation for paroxysmal AF at our centre. All 181 patients receiving semaglutide were included and comprised the treatment group. The remaining 406 patients had no GLP-1RA exposure and constituted the control pool. Pre-matching analyses showed baseline imbalances between groups (see Supplementary material online, *Table S1* and *Figure S1*). We performed 1:1 PSM, yielding 181 matched pairs and a final matched cohort of 362 patients. The year-by-year distribution of matched patients is shown in Supplementary material online, *Figure S2*. Baseline characteristics after matching are reported in *Table 1*. Most covariates were well balanced after matching; only hypertension remained more frequent in the semaglutide group (71% vs. 59%; SMD 0.24). In the matched cohort, the mean age was 56.8 ± 10.1 years in the semaglutide group and 58.3 ± 9.1 years in controls, and 73% and 71% were male, respectively. BMI was similar between groups (34.2 ± 4.4 vs. 34.0 ± 3.8 kg/m²). Baseline antiarrhythmic drug use was comparable (62% vs. 60%).

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Table 2 Procedural characteristics

Variable	Semaglutide (*n* = 181)	No GLP-1RA (*n* = 181)	SMD
Energy source, *n* (%)			
Radiofrequency	139 (77)	141 (78)	0.03
Third-generation laser balloon	27 (15)	25 (14)	0.03
Pulsed field ablation	15 (8)	15 (8)	0.00
Procedural timings, minutes			
Procedure duration	90.7 ± 21.0	91.8 ± 24.2	0.05
Fluoroscopy time	10.4 ± 5.1	9.9 ± 6.1	0.09
Left atrial time	36.5 ± 9.1	36.9 ± 4.6	0.06
Complications, *n* (%)			
Deaths	0 (0)	0 (0)	NA
Periprocedural stroke or TIA	0 (0)	0 (0)	NA
Pericardial effusion	2 (1)	1 (1)	0.06
Transient phrenic nerve palsy	0 (0)	1 (1)	0.11
Persistent phrenic nerve palsy	0 (0)	0 (0)	NA
Vascular access site complications	7 (4)	4 (2)	0.10
Anaesthesia type, *n* (%)			
General anaesthesia	92 (51)	96 (53)	0.04
Deep sedation	89 (49)	85 (47)	0.04

Data are reported as mean ± standard deviation for continuous variables or number (percentage) for categorical variables. NA indicates that the SMD is not estimable when both groups have zero events.
GLP-1RA, glucagon-like peptide-1 receptor agonist; SMD, standardized mean difference; TIA, transient ischaemic attack.

Procedural characteristics

Procedural characteristics were similar between groups (*Table 2*). Energy source distribution was comparable, with radiofrequency used in 77% vs. 78%, third-generation laser balloon in 15% vs. 14%, and pulsed field ablation in 8% vs. 8% (all SMD ≤ 0.03). Procedural timings were similar, including procedure duration (90.7 ± 21.0 vs. 91.8 ± 24.2 min, SMD 0.05), fluoroscopy time (10.4 ± 5.1 vs. 9.9 ± 6.1 min, SMD 0.09), and left atrial time (36.5 ± 9.1 vs. 36.9 ± 4.6 min, SMD 0.06). Two patients (1%) in the semaglutide group and one patient (1%) in the control group experienced pericardial effusion, while one patient (1%) in the control group developed transient phrenic nerve palsy. Vascular access site complications occurred in 4% and 2% of patients, respectively. No deaths, cerebrovascular events, or major adverse events were reported. Anaesthesia type was balanced, with general anaesthesia in 51% vs. 53% (SMD 0.04) and deep sedation in 49% vs. 47% (SMD 0.04).

Follow-up and clinical outcomes

After a median follow-up of 18 months, 80.2% of patients in the semaglutide group remained free from ATa recurrence compared with 65.2% in the no GLP-1RA group (*Figure 1*). Semaglutide was initiated within 3 months before ablation in 81 patients (45%) and within 1 month after ablation in 100 patients (55%) (see Supplementary material online, *Figure S3*). In a Cox model accounting for matched pairs and adjusted for the small residual imbalance in hypertension, semaglutide was associated with a lower risk of ATa recurrence (HR 0.52; 95% CI 0.34–0.78; $P = 0.002$). At 90 days, the proportion of patients still receiving antiarrhythmic drugs was similar between groups. The mean ATa burden at 6, 12, and at 18 months or the last available assessment was 0.18, 0.22, and 0.28% in the semaglutide group and 0.90, 1.18, and 1.46% in the control group, respectively (*Figure 2A*). The distribution of ATa burden categories at the end of follow-up is shown in *Figure 2B*. Progression to persistent atrial fibrillation occurred in two patients (1.1%) in the semaglutide group and three patients (1.7%) in the control group. Atrial flutter or atrial tachycardia recurrences occurred in six patients (3.3%) and 11 patients (6.1%), respectively. At a median follow-up of 18 months, patients treated with semaglutide achieved a substantially greater reduction in body weight compared with controls. The mean body weight decreased from baseline by 11.8 ± 3.8 kg in the semaglutide group vs. 1.9 ± 1.2 kg in the control group (95% CI 9.3–10.4 kg; $P < 0.0001$). Correspondingly, the mean BMI declined from baseline by 4.0 ± 1.4 kg/m² in the semaglutide group, reaching 30.2 ± 1.4 kg/m² at follow-up ($P < 0.001$), whereas BMI remained largely unchanged in the control group (–0.3 ± 0.8 kg/m², $P = 0.450$ vs. baseline) (*Figure 3*). Regarding weight loss distribution in the semaglutide group, 18 patients (9.9%) lost <5%, 35 (19.3%) lost 5–10%, 71 (39.2%) lost 10–15%, and 57 (31.5%) lost >15%. Overall, 163 patients (90.1%) achieved at least 5% weight loss, 128 (70.7%) lost ≥10%, and 57 (31.5%) lost ≥15% a median follow-up of 18 months (*Figure 4*). The most common adverse events were gastrointestinal, with nausea in 70 patients (38.7%), diarrhoea in 34 (18.8%), and constipation in 27 (14.9%) among those treated with semaglutide. No severe adverse events occurred, and 14 patients (7.7%) discontinued therapy due to gastrointestinal intolerance.

Discussion

The present study shows that among obese patients undergoing AF catheter ablation, metabolic treatment using semaglutide was associated with a significantly lower risk of ATa recurrence

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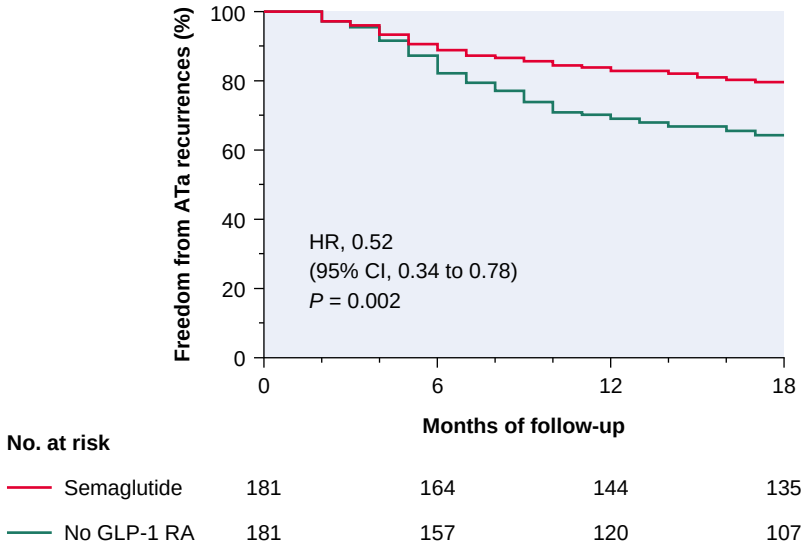

Figure 1 Freedom from atrial tachyarrhythmias recurrence after catheter ablation. Kaplan–Meier curve for recurrence of atrial tachyarrhythmias after catheter ablation in patients treated with semaglutide or never exposed to glucagon-like peptide-1 receptor agonists. ATas, atrial tachyarrhythmias; GLP-1RAs, glucagon-like peptide-1 receptor agonists.

at 18 months compared with standard care alone in a matched control group. This benefit emerged in the context of comparable baseline clinical and echocardiographic characteristics and was evaluated through continuous rhythm monitoring using ICM, supporting the hypothesis that this therapy may enhance rhythm-control outcomes in this population. In addition to the arrhythmic benefit, patients receiving semaglutide experienced substantially greater weight reduction compared with controls under standard lifestyle counselling alone. Semaglutide treatment was also associated with a lower cumulative ATa burden during follow-up and a shift towards absent or minimal burden categories at the last assessment. This signal complements time-to-first recurrence and supports a reduction in overall arrhythmia exposure captured by continuous ICM monitoring. Burden-based endpoints account for recurrent and asymptomatic episodes and add context to event-based analyses after ablation. These findings support an association between peri-ablation semaglutide exposure and improved rhythm outcomes; causal mechanisms, including the contribution of weight loss, remain to be confirmed. Nonetheless, these findings indicate that adjunctive therapy with semaglutide may enhance long-term rhythm outcomes after catheter ablation in obese patients, a population known to exhibit a higher arrhythmia recurrence rate, reduced ablation durability, and an increased need for repeat ablation compared with non-obese individuals.[25,26] Catheter ablation is a well-established rhythm-control strategy in the management of AF, but its efficacy is significantly reduced in patients with obesity.[27] Procedural success declines progressively with increasing BMI.[28] This reduced efficacy is attributed to obesity-related atrial remodelling, characterized by increased left atrial volume, conduction heterogeneity, and fibrosis, which contribute to persistent arrhythmogenic substrate despite pulmonary vein ablation.[3,29] Excess visceral and epicardial adipose tissue (EAT) promotes local inflammation and oxidative stress, further impairing lesion durability and predisposing to late reconnection.[4,30,31] Notably, the proximity of EAT to the myocardium facilitates direct infiltration of adipocytes into the atrial myocardium, potentially disrupting the depolarization wavefront and favouring micro re-entry circuits and local conduction block.[32] Additionally, EAT is a source of various pro-inflammatory cytokines and adipokines, which can determine both a structural and an electrical remodelling on the atrial myocardium.[3,33] Moreover, obese patients are exposed to greater procedural complexity, including longer procedure duration, increased fluoroscopy time, and a higher risk of complications, such as vascular access injury and pericardial effusion.[34,35] These observations underscore that, in this population, catheter ablation alone may be insufficient without concomitant management of modifiable upstream risk factors. Growing evidence supports the concept that targeted control of cardiometabolic risk can improve rhythm outcomes in AF.[18,36] Therefore, integrating rhythm control with metabolic strategies is increasingly recognized as a necessary evolution in the management of obesity-related AF.[37–39]

Obesity-driven atrial remodelling is sustained by excess visceral and epicardial adiposity, systemic inflammation, oxidative stress, and fibrotic signalling, all of which promote electrical and structural abnormalities that perpetuate AF beyond pulmonary vein triggers.[6,40] Glucagon-like peptide-1 receptor agonists exert potent metabolic effects—including clinically meaningful weight reduction, improved insulin sensitivity, and decreases in visceral adiposity—that directly counter these upstream arrhythmogenic mechanisms.[41] Beyond their metabolic impact, GLP-1RAs have demonstrated cardiovascular benefits, such as reductions in inflammatory activation and improvements in functional status in heart failure with preserved ejection fraction, supporting a pathophysiological link relevant to atrial remodelling.[42,43] Observational data also suggest a lower incidence and progression of AF among GLP-1RA users, indicating a potential protective electrophysiological role.[14,15]

In this context, the present study provides new clinical evidence supporting the potential role of metabolic modulation in rhythm-control strategies. In a strictly obese cohort undergoing AF ablation, semaglutide therapy was associated with a

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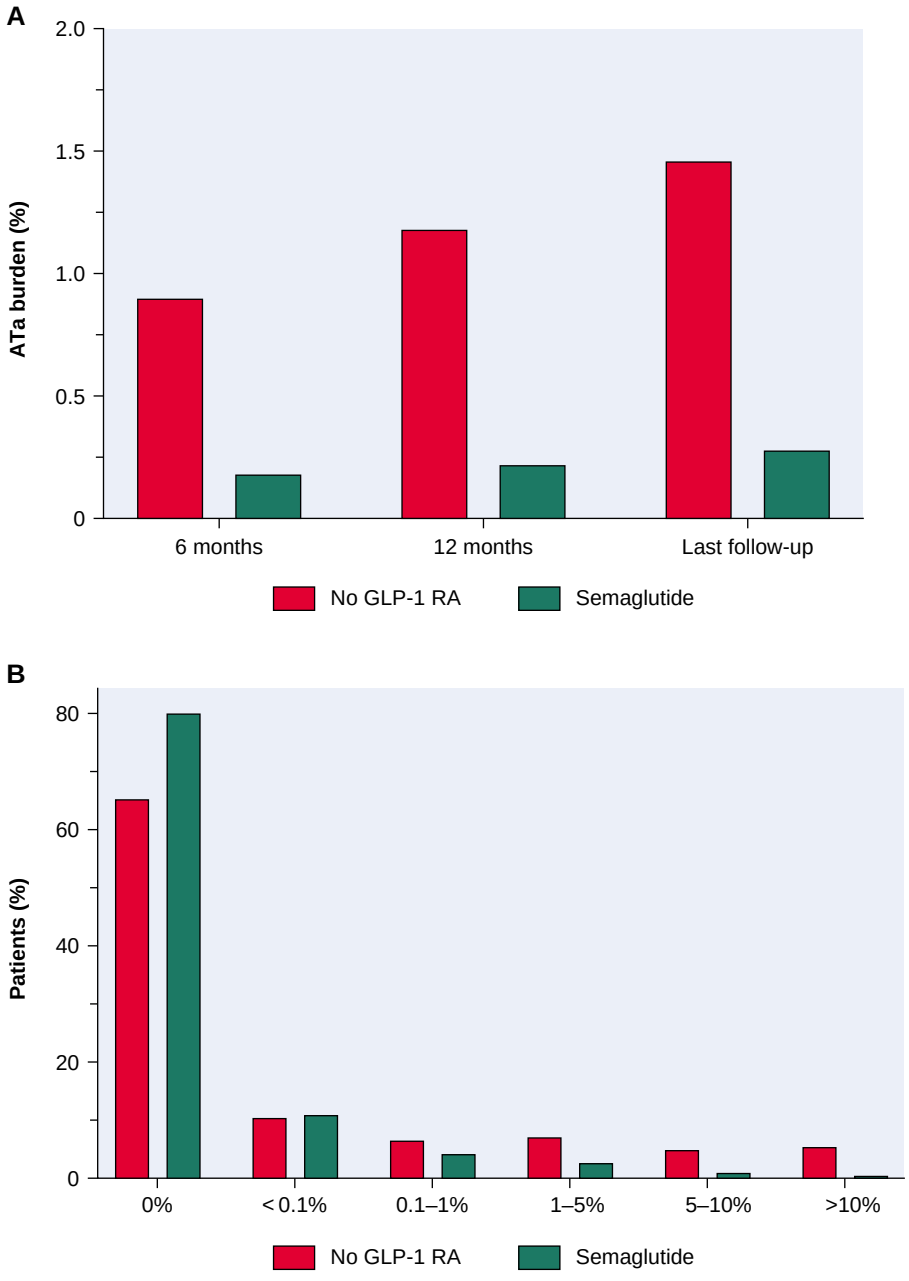

Figure 2 Atrial tachyarrhythmia burden during follow-up. Bar charts showing atrial tachyarrhythmia burden outcomes derived from continuous implantable cardiac monitor recordings. (*A*) Mean atrial tachyarrhythmia burden at 6, 12, and last available follow-up (up to 18 months) in the semaglutide and never exposed to glucagon-like peptide-1 receptor agonists groups. (*B*) Distribution of atrial tachyarrhythmia burden categories at last available follow-up (up to 18 months). ATa, atrial tachyarrhythmia; GLP-1RAs, glucagon-like peptide-1 receptor agonists.

significantly lower risk of arrhythmic recurrences. The substantial reductions in body weight and BMI seen in the semaglutide group reinforce the hypothesis that targeting obesity as a disease modifier may enhance the durability of ablation by improving atrial substrate stability. These findings are consistent with previous data demonstrating that structured weight loss improves post-ablation outcomes[44] and support the concept that adjunctive pharmacological strategies acting on the metabolic substrate may complement catheter ablation in patients with obesity-related AF.

The present findings align with emerging evidence suggesting that GLP-1RA therapy may enhance rhythm control after AF ablation.[16,45] However, prior studies were limited by heterogeneous populations, variable BMI profiles, mixed GLP-1RA agents, and intermittent rhythm surveillance. In contrast, this study focused on a strictly obese population, evaluated a single

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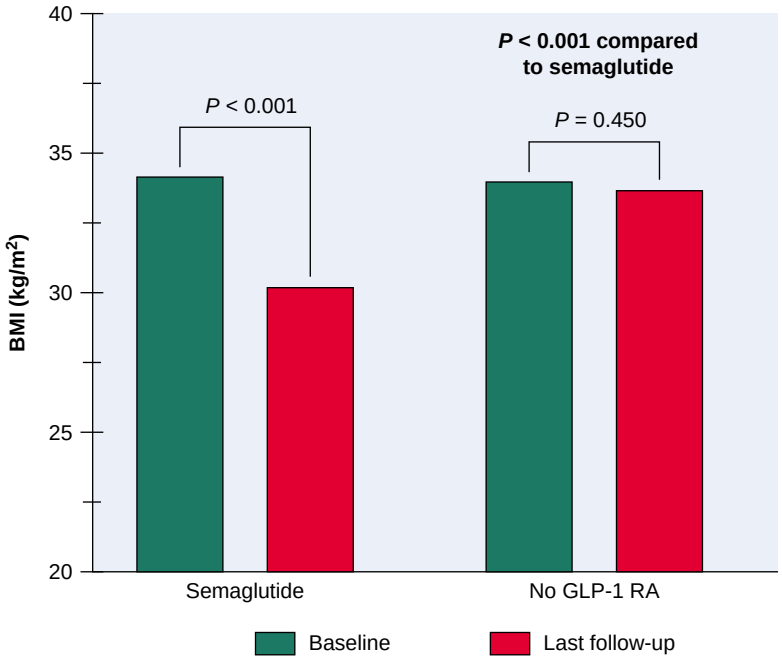

Figure 3 Change in body mass index by treatment group. Bar chart showing body mass index at baseline and at the last available follow-up assessment (up to 18 months) in patients treated with semaglutide or never exposed to glucagon-like peptide-1 receptor agonists. BMI, body mass index; GLP-1RAs, glucagon-like peptide-1 receptor agonists.

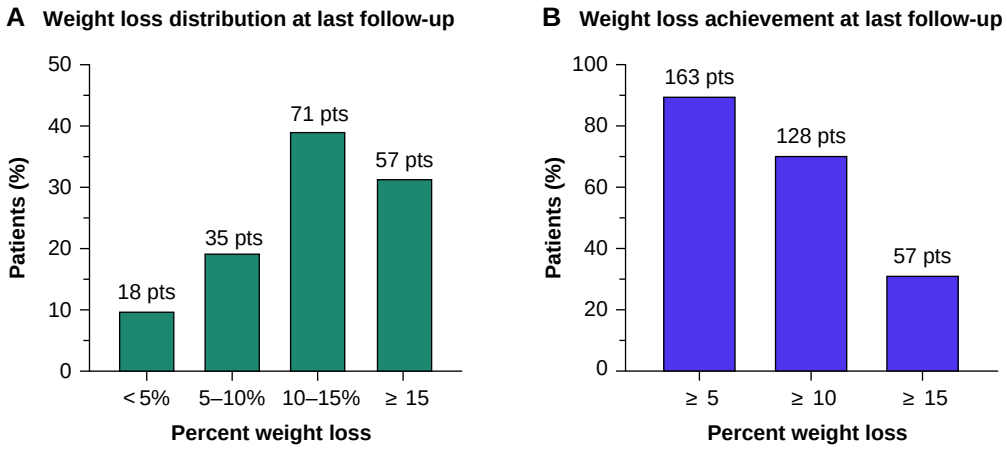

Figure 4 Weight loss distribution in patients treated with semaglutide. Bar charts showing weight loss outcomes in patients treated with semaglutide at the last available follow-up assessment (up to 18 months). (*A*) Number of patients stratified by weight loss intervals. (*B*) Proportion of patients achieving predefined thresholds of body weight reduction.

agent (semaglutide), and employed ICM, thereby improving the precision of outcome assessment. Moreover, by initiating treatment within a peri-ablation therapeutic window and quantifying weight response, this analysis provides novel insights into the importance of timing and metabolic efficacy in determining therapeutic benefit. Discrepancies with neutral studies may reflect differences in treatment exposure, metabolic response, and confounder control, highlighting the added value of the present findings. Moreover, semaglutide was well tolerated, with few gastrointestinal side effects and a low discontinuation rate, supporting its feasibility as an adjunct in rhythm-control strategies for obese patients undergoing AF ablation.

Although the mechanisms underlying the observed reduction in arrhythmic recurrences among semaglutide-treated patients remain to be fully elucidated, the present findings are biologically plausible and allow speculation on several pathophysiological

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pathways that may mediate this effect. First, semaglutide induces meaningful reductions in body weight and visceral fat, including EAT,[46] whose pro-arrhythmic effects on atrial electrophysiology and lesion durability are well established.[4] Secondly, GLP-1RAs exert anti-inflammatory effects, attenuating profibrotic and oxidative pathways implicated in atrial remodelling.[47] Thirdly, improvements in insulin resistance and autonomic balance,[48,49] together with favourable haemodynamic effects, such as reduced left atrial pressure and diastolic load,[50] may further stabilize the atrial substrate. Collectively, these mechanisms suggest that semaglutide may exert disease-modifying effects beyond weight loss, potentially explaining the lower arrhythmia recurrence observed in this study.

The present findings reinforce the concept that rhythm control in obesity-related AF should not rely solely on catheter ablation but requires concomitant metabolic intervention. Remarkably, this analysis introduces the concept of a peri-ablation therapeutic window, during which modulation of visceral adiposity, inflammatory signalling, and atrial substrate vulnerability may reduce arrhythmia recurrence. The initiation of semaglutide within 3 months before or after the procedure suggests potential clinical value for periprocedural metabolic conditioning, although the optimal timing and duration of therapy remain to be defined. While these data support a combined electrophysiological–metabolic strategy, definitive conclusions require higher-level evidence. A prospective randomized controlled trial evaluating semaglutide as an adjunct to AF ablation, particularly within a structured peri-ablation treatment window, will be essential to determine causality, identify responders, and define treatment protocols for clinical practice.

Limitations

This study presents some limitations, including the single-centre design. Although all patients were clinically eligible for weight management and received standardized lifestyle counselling, semaglutide was prescribed only in a subset based on real-world factors, such as progressive drug adoption, reimbursement and access limitations, patient preference, and contraindications. Semaglutide exposure was heterogeneous in terms of route, dose, and timing of initiation, reflecting real-world practice. Propensity score matching minimized treatment selection bias and semaglutide exposure spanned the entire study period, but residual confounding by indication and some degree of calendar time bias cannot be fully excluded; therefore, causal inference should be made with caution. We did not perform formal dose–response analyses linking the magnitude of weight loss to arrhythmic outcomes; dedicated prospective evaluations are needed to address this question. Therefore, multicentre prospective randomized trials are needed to confirm these findings and clarify the causal role of metabolic modulation in post-ablation rhythm control. Although prior evidence suggests comparable efficacy,[51–53] the use of three different ablation energy sources may represent a source of variability and could limit the uniformity of procedural effects, although their distribution was balanced between treatment groups. Metabolic laboratory indices were not systematically available in the registry, precluding analyses of biomarker changes and their relation to rhythm outcomes. Finally, given the small number of redo procedures, post-ablation electroanatomical mapping data were not available to assess substrate progression in cases and controls. Although PSM was used, alternative approaches, such as inverse probability of treatment weighting, may be more appropriate; however, residual confounding cannot be fully excluded, and a randomized study would be required to overcome this limitation.

Conclusions

In obese patients undergoing catheter ablation for paroxysmal atrial fibrillation, adjunctive pharmacological therapy with GLP-1RAs, such as semaglutide, might be beneficial in improving rhythm-control strategies. In this analysis, treatment with semaglutide was associated with a lower risk of arrhythmia recurrence, as assessed by continuous ICM monitoring. These findings suggest that semaglutide may represent a useful adjunct within rhythm-control strategies for patients with obesity. Future randomized trials are warranted to confirm these promising observations and to clarify causal effects.

Supplementary material

Supplementary material is available at *Europace* online.

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Data availability

The data underlying this article will be shared on reasonable request to the corresponding authors.

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