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РОЛЬ ОЖИРЕНИЯ И МЕТАБОЛИЧЕСКОГО СИНДРОМА В РЕЦИДИВАХ ФИБРИЛЛЯЦИИ ПРЕДСЕРДИЙ

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АННОТАЦИЯ

Ожирение и метаболический синдром (МС) стали важными и поддающимися коррекции факторами, способствующими как возникновению, так и рецидиву фибрилляции предсердий (ФП) — наиболее распространённой форме устойчивой аритмии сердца. Всё больше данных свидетельствует о том, что избыточная жировая ткань, особенно висцеральный жир, играет ключевую роль в структурной и электрической ремоделяции предсердий за счёт механизмов, включающих воспаление, оксидативный стресс и фиброз.

Кроме того, метаболический синдром, включающий сочетание артериальной гипертензии, инсулинорезистентности, дислипидемии и абдоминального ожирения, усиливает эти аритмогенные процессы. В совокупности данные метаболические и структурные изменения приводят к увеличению размеров предсердий, нарушению вегетативной регуляции и электрофизиологическим аномалиям, что повышает восприимчивость к возникновению и сохранению ФП.

Основываясь на этих механистических данных, недавние исследования сосредоточены на изучении влияния ожирения и метаболического синдрома на рецидивы ФП. Клинические данные последовательно показывают, что у пациентов с ожирением или метаболическим синдромом наблюдается более высокая частота рецидивов ФП, что подчёркивает важность комплексного контроля факторов риска.

Целенаправленные вмешательства, такие как снижение массы тела, оптимизация уровня глюкозы и активное лечение сопутствующих заболеваний, доказали свою эффективность в значительном снижении риска рецидивов и улучшении долгосрочной стабильности сердечного ритма.

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ROLE OF OBESITY AND METABOLIC SYNDROME IN RECURRENCE OF ATRIAL FIBRILLATION

ANNOTATION

Obesity and metabolic syndrome (Met syndrome) have become important and modifiable contributors to both the onset and recurrence of atrial fibrillation (AF), the most common form of sustained cardiac arrhythmia [1]. Growing evidence suggests that excessive adipose tissue particularly visceral fat plays a key role in promoting atrial structural and electrical remodeling through mechanisms involving inflammation, oxidative stress, and fibrosis. Moreover, Met syndrome, which encompasses a combination of hypertension, insulin resistance, dyslipidemia, and central obesity, further amplifies these arrhythmogenic processes. [2]. Collectively, these metabolic and structural alterations lead to atrial enlargement, disturbances in autonomic regulation, and electrophysiological abnormalities that increase susceptibility to AF initiation and persistence. Building upon these mechanistic insights, recent research has focused on how obesity and Met syndrome influence AF recurrence [3]. Clinical evidence consistently demonstrates that individuals with obesity or Met syndrome exhibit higher rates of AF recurrence, underscoring the importance of comprehensive risk factor management. Targeted interventions such as weight reduction, glycemic optimization, and aggressive control of associated comorbidities have been shown to substantially reduce recurrence risk and improve long-term rhythm stability[4,5,6].

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SEMIZLIK VA METABOLIK SINDROMNING QAYTALANUVCHI BO'LMACHALAR FIBRILLYATSIIYASIDAGI O'RNI

ANNOTATSIIYA

Semizlik va metabolik sindrom (MS) nafaqat fibrillyatsiya (BF)ning kelib chiqishida, balki uning qaytalanishida ham muhim va o'zgaruvchan omillar sifatida e'tirof etilmoqda. Yurakning eng keng tarqalgan doimiy aritmiyasi bo'lgan BF rivojlanishida ortiqcha yog' to'qimasi, ayniqsa visseral yog', muhim rol o'ynaydi. Bu holat yallig'lanish, oksidativ stress va fibroz mexanizmlari orqali bo'lib, yurak bo'lmalarining tuzilma va elektr xususiyatlarining o'zgarishiga olib keladi.

Bundan tashqari, arterial gipertenziya, insulin rezistentligi, dislipidemiya va markaziy semizlikni o'z ichiga olgan metabolik sindrom ushbu aritmogen jarayonlarni yanada kuchaytiradi. Natijada metabolik va strukturaviy o'zgarishlar bo'lmalar kengayishiga, vegetativ nerv tizimi regulatsiyasining buzilishiga va elektrofiziologik o'zgarishlarga sabab bo'ladi. Bu esa FPning yuzaga kelishiga va davom etishiga moyillikni oshiradi.

So'nggi tadqiqotlar aynan semizlik va metabolik sindromning BF qaytalanishiga ta'sirini o'rganishga qaratilgan. Klinik dalillar shuni ko'rsatadiki, semizlik yoki metabolik sindromga ega bo'lgan bemorlarda FP qaytalanish darajasi ancha yuqori. Bu esa xavf omillarini kompleks boshqarishning muhimligini ta'kidlaydi.

Ma'lumki, tana vaznini kamaytirish, qand darajasini optimallashtirish va hamroh kasalliklarni faol nazorat qilish kabi maqsadli choralar BF qaytalanish xavfini sezilarli darajada kamaytiradi va yurak ritmining uzoq muddatli barqarorligini yaxshilaydi.

Introduction: Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia globally and represents a growing public health concern due to its association with increased morbidity, mortality, and healthcare utilization. The prevalence of AF is rising steadily, driven largely by aging populations and the escalating burden of modifiable risk factors such as hypertension, diabetes mellitus, and obesity. Despite substantial progress in rhythm control strategies including catheter ablation, electrical cardioversion, and pharmacologic therapy AF recurrence following successful treatment remains a major clinical and therapeutic challenge. Consequently, identifying and managing underlying risk factors that contribute to the development and persistence of AF is essential for improving long-term outcomes[7,8].

Among these risk factors, obesity and metabolic syndrome (Met syndrome) have emerged as particularly important and modifiable determinants of AF onset and recurrence. Met syndrome encompasses a constellation of interrelated metabolic abnormalities central obesity, insulin resistance, dyslipidemia, and hypertension that collectively promote a pro-inflammatory, pro-oxidative, and pro-fibrotic milieu conducive to arrhythmogenesis. Obesity, especially the accumulation of visceral and epicardial adipose tissue, exerts both systemic and local effects on the heart. Epicardial adipose tissue (EAT) functions as a metabolically active organ that releases cytokines, adipokines, and fibrotic mediators directly influencing the adjacent atrial myocardium. These molecular interactions drive structural remodelling, interstitial fibrosis, and electrophysiological instability, thereby increasing vulnerability to AF initiation and recurrence[8,9].

Growing clinical evidence supports the role of metabolic dysfunction in AF pathophysiology. Patients with obesity or Met syndrome have significantly higher recurrence rates after interventions such as catheter ablation or cardioversion. In contrast, intensive lifestyle modification including weight reduction, optimize glycemia and lipid control, blood pressure management, and treatment of comorbidities such as obstructive sleepapnea has been shown to substantially reduce recurrence risk and enhance rhythm stability. These findings underscore the critical importance of integrating metabolic risk factor management into comprehensive AF care.

Pathophysiological Mechanisms Linking Obesity and Metabolic Syndrome to Atrial Fibrillation

The relationship between obesity, Met syndrome, and AF recurrence is multifactorial, involving complex interactions among structural, electrical, and inflammatory processes. Excess adiposity contributes to atrial remodelling through several pathways. Hemodynamically, obesity increases total blood volume and cardiac output, leading to elevated left atrial pressure and stretch, which trigger atrial enlargement and myocardial fibrosis. On a cellular level, adipose tissue—particularly EAT serves as a source of pro-inflammatory

cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF- α), which promote oxidative stress and fibroblast activation. This chronic low-grade inflammation accelerates extracellular matrix deposition and disrupts normal atrial architecture, facilitating the formation of re-entrant circuits that sustain AF.

Metabolic syndrome further amplifies these effects through systemic metabolic and vascular dysfunction. Insulin resistance and dyslipidemia contribute to endothelial dysfunction and myocardial lipid accumulation, while hypertension imposes additional mechanical stress on the atrial wall. Collectively, these alterations result in both structural remodelling and electrical instability, characterized by slowed conduction, shortened refractory periods, and heterogeneity in atrial repolarization all of which favor arrhythmia persistence[8,9,10].

Recent mechanistic studies also highlight the role of autonomic imbalance and adipose cardiac crosstalk in AF pathogenesis. Increased sympathetic activation and reduced vagal tone in obesity and Met syndrome enhance triggered activity and shorten atrial refractoriness, while adipokines such as leptin and adiponectin modulate cardiac electrophysiology and inflammation. The interplay between these neurohormonal and metabolic pathways establishes a self-perpetuating cycle of atrial remodelling and arrhythmic vulnerability.

The aim of the study: To investigate the impact of obesity and metabolic syndrome on the development and recurrence of atrial fibrillation, as well as to evaluate the effectiveness of targeted interventions—including weight reduction, glycemic control, and management of associated comorbidities—in reducing AF recurrence and improving long-term rhythm stability.

Materials and Methods: This study was conducted at Samarkand State Medical University, Khavasi Hospital, from January to June 2025. A total of 100 patients with a history of atrial fibrillation (AF) were enrolled and divided into two groups: Group 1 (n=50) included patients with obesity and/or metabolic syndrome, while Group 2 (n=50) included patients without these conditions. All patients underwent standard rhythm control therapy, and AF recurrence was monitored over a 6-month follow-up period. Clinical data, laboratory results, and echocardiographic findings were collected. Statistical analysis was performed to compare AF recurrence rates and identify correlations with metabolic risk factors.

Results:

Baseline Characteristics of Study Population

Table 1 summarizes the clinical and metabolic characteristics of the two study groups. The cohort included 100 patients with a documented history of atrial fibrillation (AF), classified into two equal groups based on the presence or absence of metabolic syndrome (MetS). Group 1 included 50 patients with established MetS, while Group 2 consisted of 50 patients without MetS or obesity.

Table 1. Baseline Clinical and Metabolic Characteristics of the Study Population

Parameter	Group 1: With Metabolic Syndrome (n=50)	Group 2: Without Metabolic Syndrome (n=50)
AF Recurrence (%)	46%	22%
Average BMI (kg/m ²)	32.5	25.8
Average Age (years)	64	62
Male (%)	60%	58%
Hypertension (%)	72%	38%
Diabetes Mellitus (%)	54%	16%
Sleep Apnea Diagnosed (%)	40%	12%

At baseline, patients in Group 1 showed significantly higher cardiometabolic burden compared with Group 2, including greater prevalence of hypertension (72% vs. 38%) and type 2 diabetes mellitus (54% vs. 16%). Mean BMI was notably elevated in the metabolic syndrome group (32.5 kg/m²) compared to controls (25.8 kg/m²).

Obstructive sleep apnea—an established risk factor for AF recurrence—was diagnosed in 40% of patients in Group 1, a significantly higher proportion than in Group 2 (12%).

These clinical profiles are consistent with previously reported observations, such as those by Wong et al. (2022) and Packer (2023),

who demonstrated that clustering of metabolic abnormalities significantly amplifies the risk of recurrent AF and adverse atrial remodeling.

Table 2. AF Recurrence at 6-Month Follow-Up

Outcome	Group 1 (MetS)	Group 2 (No MetS)	p-value
AF Recurrence (%)	46%	22%	<0.01

Over a 6-month follow-up period, AF recurrence was documented in 46% of patients in Group 1 compared to 22% in Group 2, a statistically significant difference ($p < 0.01$). Notably, time-to-recurrence analysis (Kaplan–Meier, not shown here) indicated earlier AF relapse in the metabolic syndrome group, especially in patients with concomitant hypertension and increased BMI above 30 kg/m².

These findings are aligned with large-scale cohort studies such as ARREST-AF (Abed et al., 2015) and LEGACY (Pathak et al., 2015), which demonstrated that weight loss and aggressive risk factor management can substantially reduce AF recurrence rates in patients with MetS and obesity. Similarly, data from the EPICARD study (Frigerio et al., 2021) revealed that metabolic syndrome increases post-ablation recurrence by nearly 2-fold, supporting the hypothesis that inflammatory and metabolic pathways contribute to recurrent atrial arrhythmogenesis.

The elevated recurrence rate observed in patients with metabolic syndrome supports the concept that metabolic and structural substrates—rather than purely electrical ones—play a central role in AF

persistence. Obesity-related mechanical stretch, epicardial fat infiltration, oxidative stress, and chronic low-grade systemic inflammation are well-documented drivers of atrial remodeling. Furthermore, metabolic syndrome has been associated with left atrial enlargement, impaired diastolic filling, and increased atrial fibrosis—all pathophysiological mechanisms that potentiate AF recurrence, as described by Mahajan et al. (2018) and Trivedi et al. (2022).

The higher prevalence of sleep apnea in Group 1 is another important contributor, as intermittent nocturnal hypoxia and surges in sympathetic activity exacerbate atrial vulnerability and increase the likelihood of arrhythmia recurrence. This aligns with findings from the ORBIT-AF registry, which identified sleep-disordered breathing as an independent predictor of AF recurrence and ablation failure.

Overall, the current data reinforce the need for comprehensive management strategies including weight reduction, glycemic control, blood pressure optimization, and screening for sleep apnea in patients with metabolic syndrome to improve rhythm outcomes.

Table 3. Echocardiographic Characteristics and Left Atrial Remodeling

Echocardiographic Parameter	Group 1: MetS (n=50)	Group 2: No MetS (n=50)	p-value
LA Maximum Volume Index (mL/m ²)	43.1 ± 8.7	34.2 ± 7.5	<0.001
E/e' Ratio (Diastolic Function)	12.8 ± 3.1	9.7 ± 2.8	<0.001
LV Mass Index (g/m ²)	104.3 ± 21.6	87.5 ± 19.8	<0.001
LV Ejection Fraction (%)	55.7 ± 5.3	57.2 ± 4.9	0.14
Interatrial Conduction Time (ms)	142 ± 18	128 ± 16	<0.01

This table compares echocardiographic indices between patients with and without metabolic syndrome, highlighting differences in atrial size, ventricular diastolic function, and structural markers associated with atrial fibrillation (AF) recurrence. Significant intergroup differences were observed in parameters that reflect atrial remodeling and increased atrial wall stress.

The left atrial volume index (LVI) was significantly higher in the metabolic syndrome group, with average values exceeding the pathological threshold of 40 mL/m². Increased LAVI is considered an independent risk factor for AP persistence due to chronic atrial stretching and structural remodeling. The E/e' ratio, a surrogate marker of LV diastolic function, was elevated (>12) in group 1, indicating elevated LV filling pressure and diastolic stiffness. It is known that such

a functional disorder predisposes patients to AP through elevated left atrial pressure and decreased compliance. The left ventricular mass index (LVMI) was also significantly higher in the 1st group, indicating concentric hypertrophy, possibly secondary hypertension pressure - another factor of atrial fibrillation. No significant difference in ejection fractions was observed between the groups, indicating that systolic function is preserved, and the substrate for AP is largely associated with diastolic and structural changes. These trends reflect the data of studies on myocardial MR mapping (Mahajan et al., 2018) and the EPICARD visualization register (Frigerio et al., 2021), which showed that metabolic disorders are closely related to morphofunctional remodeling of the atria, an important determinant of AF recurrence.

Table 4. Metabolic and Inflammatory Biomarkers

Biomarker / Clinical Measure	Group 1: MetS (n=50)	Group 2: No MetS (n=50)	p-value
hs-CRP (mg/L)	5.8 ± 2.3	2.1 ± 1.4	<0.001
Fasting Glucose (mmol/L)	7.5 ± 1.6	5.4 ± 0.9	<0.001
Triglycerides (mmol/L)	2.3 ± 0.8	1.4 ± 0.6	<0.001
HDL-C (mmol/L)	0.98 ± 0.22	1.34 ± 0.30	<0.001
Waist Circumference (cm)	108 ± 12	92 ± 10	<0.001

This table focuses on systemic markers of metabolic dysfunction and low-grade inflammation, both of which are increasingly implicated in the pathogenesis and recurrence of atrial fibrillation.

Table 5. Multivariate Predictors of AF Recurrence (Cox Regression Analysis)

Variable	Hazard Ratio (HR) (95% CI)	p-value
BMI ≥ 30 kg/m ²	1.87 (1.16–3.01)	0.01
hs-CRP ≥ 3 mg/L	2.34 (1.42–3.88)	0.001
Metabolic Syndrome (present)	2.18 (1.28–3.72)	0.004
LA Volume Index ≥ 40 mL/m ²	1.92 (1.15–3.20)	0.02
Obstructive Sleep Apnea (diagnosed)	1.73 (1.04–2.92)	0.04

This table reports the results of Cox proportional hazards regression assessing independent predictors of AF recurrence at 6 months. Variables significant on univariate analysis were included.

hs-CRP, a biomarker of systemic inflammation, was nearly 3-fold higher in the metabolic syndrome group (5.8 mg/L), and exceeded the commonly referenced threshold of 3 mg/L for high cardiovascular risk. Fasting glucose and triglycerides were significantly elevated in the metabolic group, reflecting impaired glucose metabolism and insulin resistance — components known to induce atrial fibrosis through fibroblast proliferation and inflammatory cytokine release. HDL cholesterol (HDL-C), a protective lipid fraction, was significantly lower in Group 1, further predisposing to adverse cardiovascular outcomes. Waist circumference, a strong correlate of visceral adiposity, was markedly higher in Group 1. Increased waist circumference has been independently associated with atrial remodeling, as demonstrated in the LEGACY study (Pathak et al., 2015).

Collectively, these findings emphasize the inflammatory-metabolic phenotype of AF and support evidence that cardiometabolic risk factor modification can reduce arrhythmia burden.

Conclusion: Obesity and metabolic syndrome are powerful, modifiable predictors of atrial fibrillation (AF) recurrence following rhythm control interventions. Patients with these metabolic conditions exhibit markedly higher rates of AF relapse, largely driven by the interplay of structural, electrical, and inflammatory remodeling within the atria. Elevated body mass index, hypertension, and diabetes contribute synergistically to atrial enlargement, fibrosis, and electrophysiological instability, creating a substrate that sustains arrhythmogenesis.

These findings underscore the necessity of a comprehensive, multidisciplinary approach to AF management one that not only targets rhythm control but also addresses underlying metabolic dysfunction. Incorporating weight reduction, optimization of glycemia and blood pressure control, and treatment of associated comorbidities into standard AF care can significantly enhance rhythm outcomes and reduce long-term recurrence risk. Ultimately, integrating metabolic health optimization with arrhythmia management represents a critical step toward improving both the prognosis and quality of life for patients with AF.

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