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ORIGINAL PAPER

INTERPLAY BETWEEN ACUTE MYOCARDIAL INFARCTION CHARACTERISTICS AND CLINICAL RISK FACTORS IN THE DEVELOPMENT OF NEW-ONSET ATRIAL FIBRILLATION

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Abstract

Introduction. The development of new-onset atrial fibrillation (NOAF) is affected by age, comorbidities, structural heart changes, and infarct characteristics, larger infarct size, and atrial branch involvement; however, the exact mechanisms and relative contributions of these factors remain poorly understood.
The aim of the study was to identify clinical and infarct-derived factors predisposing to NOAF after acute myocardial infarction (AMI).
Materials and methods. This prospective study included 150 AMI patients, randomized into NOAF (n=75) and sinus rhythm (n=75) groups. All underwent clinical, echocardiographic evaluation and were followed for two years.
Results. In the group of AMI patients, those with NOAF were older, had higher body mass index (BMI) and severe obesity, and more frequently had peripheral artery disease and grade III hypertension. Cardiovascular risk (SCORE) and cardiac biomarkers (troponin I, NT-proBNP) were higher in NOAF patients. ST segment elevation acute myocardial infarction (STEMI) prevalence and myocardial infarction type were similar, but cardiogenic shock occurred only in NOAF patients. Culprit artery distribution differed,

Résumé

Interaction entre les caractéristiques de l'infarctus aigu du myocarde et les facteurs de risque cliniques dans le développement de la fibrillation auriculaire de novo

Introduction. Le développement de la fibrillation auriculaire de novo (FADN) est influencé par l'âge, les comorbidités, les modifications structurelles cardiaques et les caractéristiques de l'infarctus, notamment sa taille et l'atteinte des branches atriales. Toutefois, les mécanismes exacts restent insuffisamment élucidés.
L'objectif de l'étude a été d'identifier les facteurs cliniques et liés à l'infarctus prédisposant à la FADN après un infarctus aigu du myocarde (IAM).
Matériels et méthodes. Étude prospective incluant 150 patients avec IAM, répartis en deux groupes : FADN (n=75) et rythme sinusal (n=75). Tous ont bénéficié d'une évaluation clinique, échocardiographique et d'un suivi de deux ans.
Résultats. Les patients avec FADN étaient plus âgés, avec un IMC plus élevé, une obésité sévère plus fréquente, ainsi qu'une prévalence accrue de maladie artérielle périphérique et d'hypertension de grade III. Le SCORE et les biomarqueurs (troponine I,

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with left anterior descending artery (LAD) more common in sinus rhythm, while LCx, LM, and RCA involvement and SYNTAX score were higher in NOAF. Apical infarctions were more frequent in NOAF (18.7% vs. 1.3%). These data suggest that NOAF is associated with adverse risk profiles, greater myocardial injury, and distinct infarct patterns.

Conclusions. NOAF in AMI is associated with older age, obesity, severe hypertension, higher cardiovascular risk, and greater myocardial injury. It is also linked to distinct infarct localization, higher coronary complexity, and increased incidence of cardiogenic shock, emphasizing its prognostic significance and the need for careful monitoring and tailored management.

Keywords: acute myocardial infarction, new-onset atrial fibrillation, heart failure.

List of abbreviations:
AMI – acute myocardial infarction
STEMI – ST segment elevation acute myocardial infarction
LAD – left anterior descending artery
LCx – left circumflex artery
LM – left main artery
NOAF – new-onset atrial fibrillation
RCA – right coronary artery

Résumé

Introduction. La fibrillation auriculaire de novo (FADN) est fréquente après un infarctus aigu du myocarde (IAM) et est associée à un pronostic défavorable. Cette étude examine les différences cliniques, angiographiques et échocardiographiques entre les patients présentant une FADN et ceux en rythme sinusal.

Méthodes. Cette étude rétrospective a inclus 1 000 patients hospitalisés pour IAM. Les données démographiques, cliniques, angiographiques et échocardiographiques ont été analysées. Les patients ont été répartis en fonction de la présence ou de l'absence de FADN. Des analyses univariées et multivariées ont été réalisées pour identifier les prédicteurs indépendants.

Résultats. La FADN est survenue chez 7,7 % des patients. Ceux-ci étaient plus âgés, plus souvent obèses, hypertendus et présentaient des taux plus élevés de facteurs de risque cardiovasculaire. Ils présentaient également une fonction ventriculaire gauche plus altérée et des marqueurs de lésions myocardiques plus élevés (troponine, NT-proBNP). Le STEMI était similaire entre les groupes, mais le choc cardiogénique n'est survenu que dans le groupe FADN. L'atteinte coronarienne différait, avec une complexité plus élevée (score SYNTAX) et une implication plus fréquente de la circonflexe, du tronc commun gauche et de la coronaire droite. Les infarctus apicaux étaient plus fréquents (18,7 % vs 1,3 %).

Conclusion. La FADN est associée à un profil de risque défavorable, une atteinte myocardique plus sévère et une complexité coronarienne accrue, soulignant son importance pronostique.

Mots-clés: infarctus aigu du myocarde, fibrillation auriculaire de novo, insuffisance cardiaque.

Introduction

Acute myocardial infarction (AMI) remains a leading cause of global mortality. Despite advances in contemporary therapies, new-onset atrial fibrillation (NOAF) is a common complication of AMI and is associated with worse clinical outcomes, including prolonged hospitalization and increased all-cause mortality[1]. Coronary artery disease and atrial fibrillation often exacerbate each other, creating a potentially self-perpetuating cycle. Therefore, identifying clinical factors associated with NOAF development during AMI is of considerable clinical importance. Although evidence is accumulating, uncertainties remain regarding the mechanisms and relative contribution of previously identified risk factors to NOAF in AMI patients[2]. Both conditions share overlapping risk factors that trigger diverse pathophysiological responses, ultimately promoting the development of one or both diseases. Diabetes mellitus, arterial hypertension, advancing age, dyslipidemia, obesity, smoking, and physical inactivity represent the main risk factors for coronary artery disease, as extensively documented in the literature. These same factors also appear to contribute to the initiation and progression of atrial fibrillation[3]. Type 2 diabetes mellitus is a well-established independent risk factor for atrial fibrillation, associated with more severe symptoms, reduced quality of life, and higher hospitalization rates[4]. NOAF induces structural atrial remodeling, particularly dilation and fibrosis, which facilitates arrhythmia. As myocardial fibrosis is independently linked to type 2 diabetes, diabetic patients present a substrate that predisposes them to atrial fibrillation development[5]. Arterial hypertension remains the most prevalent risk factor for both atrial fibrillation and coronary artery disease, increasing the risk of NOAF by more than 30% across all age groups[6]. Hypertension-related left ventricular hypertrophy and diastolic dysfunction contribute to atrial dilation and impaired function. Additionally, activation of the renin–angiotensin–aldosterone system and elevated proinflammatory cytokine expression promote atrial fibrosis[7]. These pathological changes can facilitate ectopic foci through disrupted calcium homeostasis and the formation of re-entry circuits within fibrotic atrial regions, which may manifest clinically or remain subclinical[8]. Obesity is another common risk factor for both AMI and NOAF[9]. It increases atrial fibrillation risk directly and indirectly through comorbidities such as hypertension, diabetes, and heart failure. Obesity-related structural and electrical remodeling promotes NOAF via mechanisms

including increased adiposity, inflammation, fibrosis, oxidative stress, ion channel alterations, and autonomic dysfunction. Notably, excessive epicardial adipose tissue has been proposed as a key trigger of NOAF through paracrine signaling and direct myocardial infiltration[10]. Patients with NOAF display elevated inflammatory markers, including interleukin-6 (IL-6), interleukin-1β (IL-1β), C-reactive protein (CRP), and tumor necrosis factor-α (TNF-α). Except for IL-1β, these markers are also elevated in otherwise healthy obese women, indicating that adipose tissue contributes to systemic inflammation. Inflammatory cytokines promote oxidative stress, collagen deposition, and disturbances in calcium handling, leading to tissue injury, localized fibrosis, and altered atrial electrophysiology[11]. For instance, IL-6 drives naive T cells to differentiate into Th17 effector cells, contributing to myocardial fibrosis mediated by IL-17. Epicardial adipose tissue can release proinflammatory cytokines and adipokines, including chemerin and resistin, which are implicated in atrial remodeling[12]. Although elevated lipoprotein levels are associated with increased atherosclerosis and coronary artery disease—which in turn can raise NOAF risk[13] –several studies suggest that higher levels of total cholesterol, LDL-cholesterol, and HDL-cholesterol are paradoxically associated with lower NOAF risk. The clinical significance and pathophysiological mechanisms of this inverse relationship remain unclear[14]. In a study by Ding et al. including over 65,000 adults aged 45–60 years without prior cardiovascular disease, participants were followed for up to 35 years using ICD codes for discharge diagnoses and causes of death. Higher total cholesterol and LDL-cholesterol levels were associated with lower AF risk during the first five years (hazard ratios [HR]: 0.61, 95% CI: 0.41–0.99; HR: 0.64, 95% CI: 0.45–0.92), but this effect diminished after five years. Conversely, low HDL-cholesterol levels, high triglycerides, and elevated triglyceride-to-HDL-cholesterol ratios were consistently associated with increased atrial fibrillation risk over three decades of follow-up (HR range: 1.13 [95% CI: 1.07–1.19, $p < 0.001$] to 1.53 [95% CI: 1.12–2.00])[15]. Recent evidence from the last five years indicates that NOAF occurring during AMI is not a random epiphenomenon but rather reflects a distinct and more severe ischemic phenotype.

Contemporary observational studies consistently demonstrate that NOAF is closely linked to infarct characteristics suggesting greater myocardial injury, advanced structural remodeling, and heightened inflammatory activation. The type and extent of myocardial infarction appear to play a significant role in the development of NOAF. Cohort studies have reported a higher incidence of NOAF in patients with ST-segment elevation myocardial infarction (STEMI) compared with non-STEMI, which is associated with elevated cardiac troponin and natriuretic peptide levels, longer hospitalization, and more frequent in-hospital complications[16]. These observations suggest that larger infarct size and abrupt loss of contractile myocardium are important triggers for atrial electrical instability. Left ventricular dysfunction and acute cardiac remodeling have also been consistently linked to NOAF. Reduced left ventricular ejection fraction has repeatedly been identified as an independent predictor of both early and late NOAF after AMI[17]. Beyond ventricular impairment, recent echocardiographic evidence emphasizes the role of atrial structural changes. In particular, the left atrial minimum volume index has emerged as a strong predictor of NOAF, surpassing traditional measures of diastolic dysfunction and highlighting the importance of atrial stretch and decreased compliance in arrhythmia development[18]. The relationship between the culprit coronary vessel, infarct localization, and the development of NOAF in the setting of AMI remains incompletely defined. Observational studies have indeed reported a higher incidence of NOAF in patients presenting with STEMI, a clinical scenario frequently related to proximal left anterior descending artery (LAD) occlusion and extensive myocardial injury, although the culprit vessel itself is rarely included as an independent variable in multivariable models[16]. In contrast, inferior myocardial infarctions, typically related to right coronary artery (RCA) or left circumflex artery (LCx) involvement, have been hypothesized to promote NOAF through different mechanisms, including autonomic imbalance and potential ischemia of atrial tissue. Importantly, atrial myocardial perfusion is supplied by small atrial branches arising predominantly from the RCA and, less frequently, from the LCx or LAD. Angiographic studies have suggested that occlusion of atrial branches may be associated with early NOAF after AMI, supporting the concept of direct atrial ischemia as a trigger for atrial fibrillation. However, these observations are largely derived from small cohorts or mechanistic analyses rather than contemporary large-scale clinical studies[17]. Despite these plausible mechanisms, contemporary evidence comparing the incidence of NOAF according to the specific culprit artery (LAD versus RCA versus LCx) is scarce.

The objective of the study was to comprehensively elucidate the clinical determinants and infarct-related characteristics that predispose to the development of NOAF following AMI.

Materials and methods

Study design and patient assessment methods

This prospective study was conducted at the Institute of Cardiology in Chisinau, Republic of Moldova. The number of participants included in this study was determined based on preliminary findings and evidence from previous research to ensure sufficient statistical power for detecting meaningful clinical differences. Data were collected consecutively from 2019 to 2024. The study population included 150 adult patients (≥18 years) diagnosed with AMI, all of whom provided written informed consent.

The study was approved by the Ethics Committee of the Nicolae Testemitanu State University of Medicine and Pharmacy (approval number 54, April 12, 2018) and included patients diagnosed with AMI with symptom onset within 24 hours. The study was conducted in accordance with the principles of the Declaration of Helsinki. Exclusion criteria were a life expectancy of less than 2 years due to non-cardiovascular pathology, cognitive disorders, drug abuse, previous atrial fibrillation.

The study population was divided into two equal groups: Group I (n = 75) included patients with AMI who developed NOAF during the acute phase, while Group II (n = 75) comprised patients with AMI who maintained sinus rhythm. All participants underwent comprehensive clinical, paraclinical, and echocardiographic assessments. Patients were enrolled consecutively and followed prospectively for two years to evaluate predefined endpoints, including heart failure–related hospitalizations, cardiovascular mortality, and non-cardiovascular mortality.

Statistical analysis

Statistical analyses were performed using RStudio (version 2024.09.1+394) and Python (version 3.12.3), ensuring a fully reproducible analytical workflow. Continuous variables were described using standard summary measures, including range, mean, standard deviation, median, and interquartile range, and between-group comparisons were carried out using the Mann–Whitney U test. Categorical variables were reported as absolute counts and percentages with corresponding 95% confidence intervals. Associations between categorical variables were assessed using Pearson's chi-square test, with p-values estimated through a Monte Carlo simulation approach based on 100,000 iterations. A two-sided α level of 0.05 was considered statistically significant.

Results

A total of 150 patients were enrolled, with a mean age of 67.4±10.6 years (95% CI: 66–69). Patients in Group I (AMI with NOAF) were significantly older than those in Group II (70.0 ± 9.9 years, 95% CI: 68–72 vs. 64.7 ± 10.6 years, 95% CI: 62–67; p = 0.001). Sex distribution did not differ significantly between groups (p = 0.12). Women were more frequently represented in Group I than in Group II (40.0%, 95% CI: 29–51% vs. 26.7%, 95% CI: 17–37%), although male patients predominated in both cohorts. Patients with NOAF exhibited significantly higher body weight and body mass index (BMI). Mean body weight was 87.6±15.7 kg (95% CI: 84–91) in Group I compared with 81.4±14.7 kg (95% CI: 78–85) in Group II (p = 0.005). Similarly, BMI was significantly higher in Group I (30.5±6.3 kg/m^2, 95% CI: 29–32) than in Group II (28.3±5.0 kg/m^2, 95% CI: 27–29; p = 0.009). Obesity was significantly more prevalent among patients with NOAF. When BMI categories were examined, severe obesity (class III) was markedly more frequent in the NOAF cohort (10.7%; 95% CI: 3.7–18%) than in sinus rhythm patients (1.3%; 95% CI: 0–3.9%).

No significant differences were observed in the prevalence of metabolic syndrome. No significant differences were noted between groups regarding a history of stroke or an aggravated family history. Peripheral artery disease was identified only among patients with NOAF (9.3%; 95% CI: 2.7–16%), a difference that reached statistical significance (p = 0.014). With respect to lifestyle characteristics, alcohol consumption was more common in patients in sinus rhythm (44.0%; 95% CI: 33–55%) than in those with NOAF (13.3%; 95% CI: 5.6–21%; p < 0.001). Smoking prevalence did not differ significantly between groups, although a higher proportion of smokers was observed in the NOAF group (40.0%; 95% CI: 29–51% vs. 25.3%; 95% CI: 15–35%; p = 0.082). Hypertension was more pronounced among NOAF patients, with grade III hypertension affecting 40.0% (95% CI: 29–51%) compared with 22.7% in sinus rhythm (95% CI: 13–32%; p = 0.004). Other comorbidities, including prior myocardial infarction and diabetes mellitus, did not differ significantly between groups.

The overall cardiovascular risk profile was higher in NOAF patients. The mean SCORE was 25.8% (95% CI: 23–29) versus 17.8% (95% CI: 16–20) in the sinus rhythm group (p < 0.001), and very high cardiovascular risk was present in 94.7% of NOAF patients (95% CI: 90–100%) compared with 69.3% of patients in sinus rhythm (95% CI: 59–80%; p < 0.001).

Cardiac biomarkers were elevated in NOAF patients, with higher troponin I levels (10.2 ± 7.7 vs. 8.8 ± 9.3; p = 0.002) and significantly increased NT-proBNP concentrations (1,728 ± 2,206 vs. 1,005 ± 1,453; p = 0.012), consistent with greater myocardial

Table 1. Patient characteristics and comorbidities by study groups

	Gorup I N = 75[1]	*95% CI*	*Group II N = 75*[1]	*95% CI*	*Statistic Test*[2]	*p-value*[2]
BMI > 30 kg/m²	43 (57.3%)	46%, 69%	22 (29.3%)	19%, 40%	12	<0.001
					14	0.008
Obesity class I	24 (32.0%)	21%, 43%	12 (16.0%)	7.7%, 24%		
Obesity class II	7 (9.3%)	2.7%, 16%	7 (9.3%)	2.7%, 16%		
Obesity class III	8 (10.7%)	3.7%, 18%	1 (1.3%)	0.00%, 3.9%		
Overweight	21 (28.0%)	18%, 38%	34 (45.3%)	34%, 57%		
Aggravated medical history	6 (8.0%)	1.9%, 14%	10 (13.3%)	5.6%, 21%	1.1	0.4
Previous stroke	9 (12.0%)	4.6%, 19%	6 (8.0%)	1.9%, 14%	0.67	0.6
Peripheral arterial disease	7 (9.3%)	2.7%, 16%	0 (0.0%)	0.00%,0.00%	7.3	0.014
Smoking	30 (40.0%)	29%, 51%	19 (25.3%)	15%, 35%	3.7	0.082
Alcohol consumption	10 (13.3%)	5.6%, 21%	33 (44.0%)	33%, 55%	17	<0.001
Arterial hypertension					12	0.004
Absence	27 (36.0%)	25%, 47%	21 (28.0%)	18%, 38%		
grade I	2 (2.7%)	0.00%, 6.3%	1 (1.3%)	0.00%, 3.9%		
grade II	16 (21.3%)	12%, 31%	36 (48.0%)	37%, 59%		
grade III	30 (40.0%)	29%, 51%	17 (22.7%)	13%, 32%		
History of myocardial infarction	4 (5.3%)	0.25%,10%	11 (14.7%)	6.7%, 23%	3.6	0.10
Diabetes mellitus type 2	22 (29.3%)	19%, 40%	15 (20.0%)	11%, 29%	1.8	0.3
SCORE					17	<0.001
Very high risk	71 (94.7%)	90%, 100%	52 (69.3%)	59%, 80%		
High risk	4 (5.3%)	0.25%,10%	17 (22.7%)	13%, 32%		
Moderate risk	0 (0.0%)	0.00%,0.00%	6 (8.0%)	1.9%, 14%		
Anemia	24 (32.0%)	21%, 43%	4 (5.3%)	0.25%, 10%	18	<0.001
Dyslipidemia	53 (70.7%)	60%, 81%	71 (94.7%)	90%, 100%	15	<0.001
Metabolic syndrome	19 (25.3%)	15%, 35%	17 (22.7%)	13%, 32%	0.15	0.8
Troponin I	10.2 (7.7) 8.8 (5.1) 3.2 49.0	8.4, 12	8.8 (9.3) 5.5 (6.8) 0.5 55.8	6.6, 11	3618	0.002
NT-proBNP	1,728.4 (2205.7) 675.0 (2223.5) 55.0 – 7654.0	1221, 2236	1,005.2 (1452.5) 252.0 (1353.0) 24.0 – 6004.0	671, 1339	3482	0.012

[1]n (%); Mean (SD) Median (IQR) Min Max
[2]Pearson's Chi-squared test with simulated p-value (based on 1e+05 replicates); NA; Wilcoxon rank sum test.
CI – confidence Interval

injury and hemodynamic stress compared with patients in sinus rhythm (Table 1).

In the overall cohort, 104 patients (69.3%; 95% CI: 62–77%) had STEMI, and 46 patients (30.7%; 95% CI: 23–38%) had non-STEMI. Among patients with NOAF, 48 (64.0%; 95% CI: 53–75%) had STEMI and 27 (36.0%; 95% CI: 25–47%) had non-STEMI, while in sinus rhythm patients, 56 (74.7%; 95% CI: 65–85%) had STEMI and 19 (25.3%; 95% CI: 15–35%) had non-STEMI (p = 0.2).

Type 1 myocardial infarction predominated, occurring in 147 patients (98.0%; 95% CI: 96–100%). Type 4 AMI occurred in 1 patient (0.7%; 95% CI: 0–2%) and type 5 AMI in 2 patients (1.3%; 95% CI: 0–3.2%); differences between groups were not significant (p = 0.5). Cardiogenic shock was observed in 10 patients overall (6.7%; 95% CI: 2.7–11%), all in the NOAF group (10/751, 13.3%; 95% CI: 5.6–21%).

The distribution of the culprit artery (Figure 1) differed significantly between groups (p = 0.021).

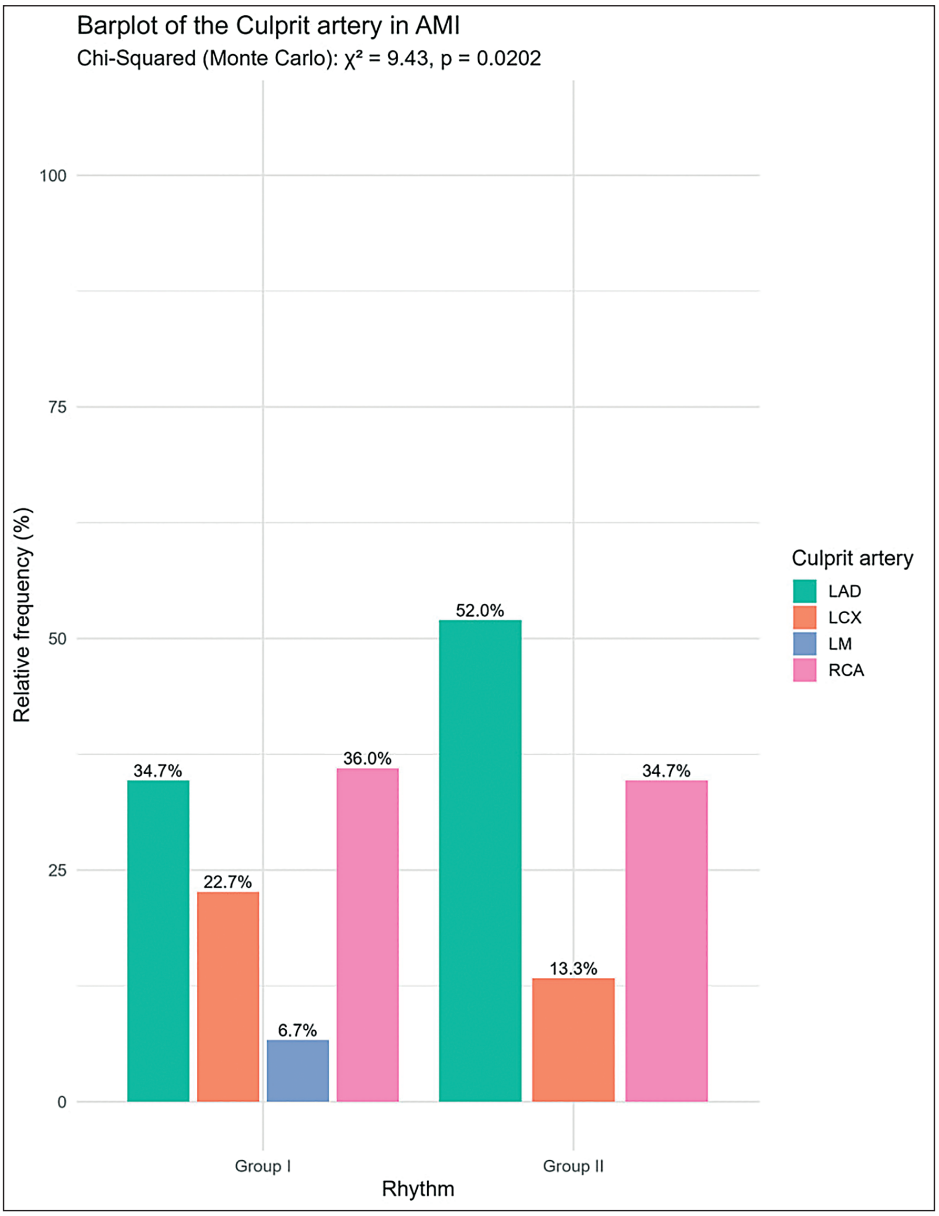


Figure 1. Distribution of culprit artery of the myocardial infarction per study groups. Group I – NOAF, Group II – sinus rhythm

In patients with atrial fibrillation, LAD was most frequently involved (34.7%; 95% CI: 24–45%), followed by the RCA (36.0%; 95% CI: 25–47%) and the LCx (22.7%; 95% CI: 13–32%). Left main coronary artery (LM) involvement was rare (6.7%; 95% CI: 1–12%). In the sinus rhythm group, the LAD artery predominated as the culprit vessel (52.0%; 95% CI: 41–63%), with similar proportions of right coronary and left circumflex artery involvement (34.7% and 13.3%, respectively). Angiographic complexity: SYNTAX score was higher in NOAF patients (30.2 ± 7.9; median 29, IQR 17–44) than in sinus rhythm (27.6 ± 8.2; median 27, IQR 14–43; p = 0.046).

When analyzing the localization of AMI, distinct patterns emerged between patients with NOAF and those in sinus rhythm. Apical infarctions were markedly more frequent in the NOAF cohort (18.7%; 95% CI: 9.8–27%) compared with sinus rhythm patients (1.3%; 95% CI: 0–3.9%), highlighting a statistically significant difference in this less common infarct location. Anterior wall infarctions were the most prevalent overall, affecting 54.7% of NOAF patients (95% CI: 43–66%) and 44.0% of sinus rhythm patients (95% CI: 33–55%), whereas inferior wall infarctions were observed in 44.0% of NOAF patients (95% CI: 33–55%) and 37.3% of SR patients (95% CI: 26–48%). The overall comparison of AMI localization between the two groups was statistically significant (p = 0.002) (Figure 2).

These findings suggest that atrial fibrillation is associated with a higher likelihood of apical

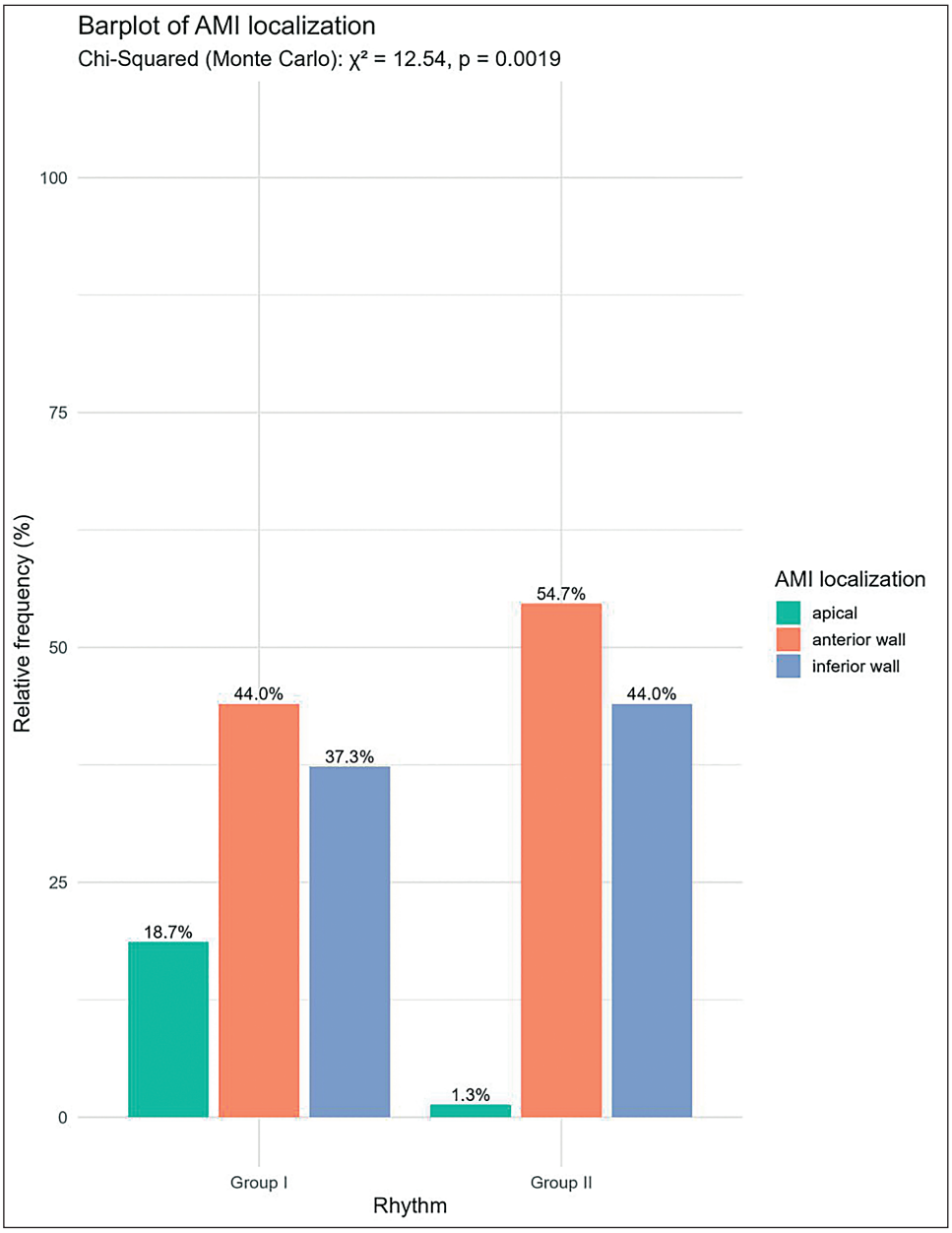


Figure 2. Localization of AMI per study groups. Group I – NOAF, Group II – sinus rhythm

infarctions, while anterior and inferior wall infarctions remain common across both groups. The observed distribution may reflect differences in coronary artery involvement, hemodynamic conditions, or microvascular susceptibility in NOAF patients. Recognizing these patterns is clinically relevant, as infarct localization influences both immediate management strategies and prognostic assessment, particularly in the context of atrial arrhythmias. Multivessel disease was frequent in both groups: NOAF 64/75 (85.3%; 95% CI: 77–93%), sinus rhythm 61/75 (81.3%; 95% CI: 73–90%; p = 0.7). Revascularization: NOAF patients underwent more percutaneous intervention (55/75, 73.3%; 95% CI: 63–83%) than sinus rhythm (34/75, 45.3%; 95% CI: 34–57%; p < 0.001). The rates of complete revascularization did not differ significantly between groups (38.7% vs. 25.3%; p = 0.12). Overall, cardiovascular mortality occurred in 10.0% of the cohort (n = 15; 95% CI: 5.2–15%), with similar incidence in patients with atrial fibrillation (10.7%; 95% CI: 3.7–18%) and those maintaining sinus rhythm (9.3%; 95% CI: 2.7–16%; p > 0.9). In contrast, hospitalizations for heart failure (Figure 3) affected 28.7% of the total population (n = 43; 95% CI: 21–36%) and were significantly more frequent in the NOAF group (37.3%; 95% CI: 26–48%) compared with patients in sinus rhythm (20.0%; 95% CI: 11–29%; p = 0.030).

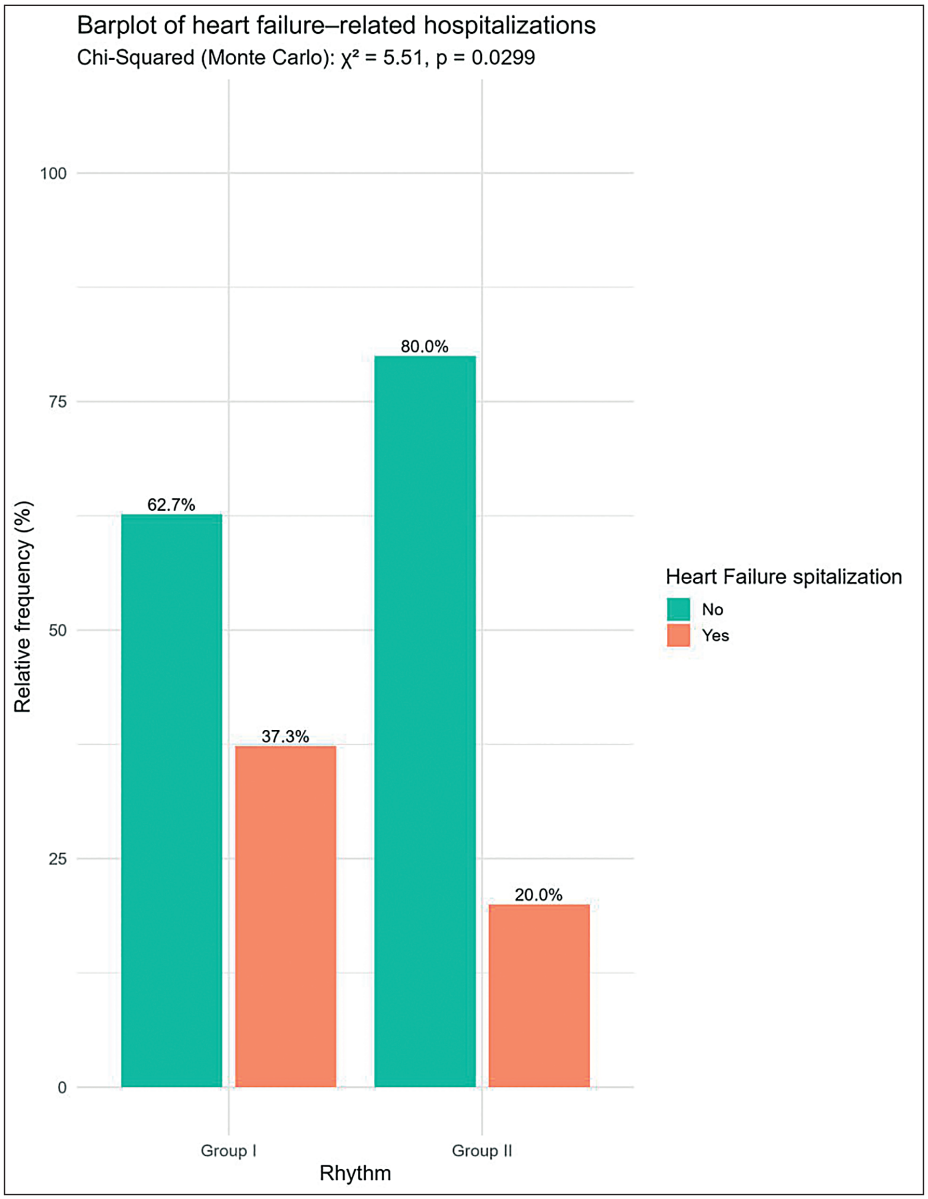


Figure 3. Rate of heart failure hospitalizations per study groups. Group I – NOAF, Group II – sinus rhythm

Discussion

In patients with AMI, NOAF identifies a distinct, clinically vulnerable phenotype. These patients were older and had a higher burden of adiposity, with severe obesity being particularly prominent. Hypertension was more prevalent and severe, and peripheral artery disease was observed exclusively in the NOAF group, reflecting a more advanced systemic atherosclerotic profile. Although rates of diabetes, prior myocardial infarction, metabolic syndrome, and cerebrovascular disease were similar between groups, NOAF patients had a markedly higher global cardiovascular risk, as indicated by elevated SCORE values and a predominance of very high-risk status. Biochemically, this adverse profile was supported by higher troponin I and NT-proBNP levels, suggesting greater myocardial injury and hemodynamic stress at presentation. Lifestyle factors differed as well: alcohol consumption was more frequent in patients maintaining sinus rhythm, while smoking showed only a non-significant trend toward higher prevalence in the NOAF cohort. The presence of atrial fibrillation did not significantly alter the distribution of STEMI versus non-STEMI or the predominance of type 1 myocardial infarction, indicating largely comparable ischemic mechanisms between groups. Nevertheless, NOAF identified patients with a more severe clinical and angiographic profile. Cardiogenic shock occurred exclusively in the AF group, highlighting a higher acute risk. Coronary anatomy was more complex, with a higher prevalence of left main and

circumflex involvement, fewer LAD-related infarctions, and higher SYNTAX scores, suggesting more diffuse coronary disease. Infarct localization differed, with apical myocardial infarctions more frequent in NOAF patients, while anterior and inferior wall infarctions were common in both groups. This pattern likely reflects differences in coronary involvement, myocardial vulnerability, and hemodynamic instability associated with atrial fibrillation, with implications for risk stratification and post-infarction remodeling. Despite a similarly high prevalence of multivessel disease, AF patients underwent percutaneous coronary intervention more frequently, likely due to greater clinical instability and anatomical complexity, although rates of complete revascularization were similar. Importantly, NOAF was not associated with increased cardiovascular mortality during follow-up, but it was linked to a higher rate of heart failure–related hospitalizations. Overall, atrial fibrillation in the setting of AMI appears less as a direct determinant of mortality and more as a marker of disease severity, hemodynamic compromise, and adverse non-fatal outcomes. These findings support the need for intensified monitoring and tailored management strategies in this high-risk population, with particular attention to preventing heart failure progression and managing complex coronary disease.

In the literature, the prognostic implications of NOAF remain controversial, particularly regarding whether its impact on short-term (in-hospital) and long-term outcomes is equivalent[19]. In a study published in 2024, involving a cohort of 616 patients admitted to the intensive care unit with AMI, NOAF was diagnosed in 87 patients (14.1%), 136 patients (22.1%) had pre-existing AF, and 393 patients (63.8%) did not develop AF. Initial episodes of NOAF were associated with greater hemodynamic instability compared with episodes occurring in patients with pre-existing AF, characterized by higher heart rates and increased catecholamine requirements (both $p < 0.001$). Independent predictors of survival during the intensive care unit stay included NOAF ($p = 0.016$), resuscitation before or during the intensive care unit admission ($p < 0.001$), and acute dialysis ($p = 0.002$)[20].

The analysis based on the ARGEN-IAM-ST registry demonstrated that heart failure—assessed using the Killip and Kimball classification—is independently associated with the development of NOAF, in line with findings from the GUSTO-I and GUSTO-III studies[21]. Similarly, a subanalysis of the international GRACE study, which included more than 21,000 patients with acute coronary syndromes and stratified them according to the presence of NOAF, pre-existing atrial fibrillation, or no atrial fibrillation, identified a Killip score ≥2 as an independent predictor of NOAF[22, 23].

Conclusions

In AMI, NOAF identifies a distinct high-risk phenotype characterized by older age, higher adiposity, more severe hypertension, advanced atherosclerotic burden, and increased myocardial injury and hemodynamic stress. Although it does not significantly affect infarct type or cardiovascular mortality, NOAF is associated with greater clinical and angiographic complexity, cardiogenic shock, and a higher risk of heart failure–related hospitalizations.

Consistent with large registries and contemporary studies, heart failure severity, reflected by higher Killip class, emerges as a key determinant of NOAF. Overall, NOAF in AMI should be regarded as a marker of disease severity and hemodynamic instability rather than an independent driver of mortality, warranting closer monitoring and targeted management to prevent adverse non-fatal outcomes.

Author Contributions:

Conceptualization, N. C, D.B,; methodology, D.B; software and statistical analysis, O.A; investigation, N.C; data curation, M.B, V.D, I.E.; writing–original draft preparation, D.B; writing–review and editing, N.C; supervision, L.D. All the authors have read and agreed with the final version of the article.

Compliance with the Ethical Requirements:

"The authors declare no conflict of interest regarding this article"

"The authors declare that all the procedures and experiments of this study respect the ethical standards in the Helsinki Declaration of 1975, as revised in 2008(5), as well as the national law. Informed consent was obtained from the patients included in the study"

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