

Atrial artery involvement in myocardial infarction and new-onset atrial fibrillation: two-center retrospective cohort study

Daria A. Shebonkina^{1,2}, Larisa V. Shekhovtsova^{3,4}, Maria S. Vasilyeva^{1,2}, Alexey L. Titov¹, Maxim I. Boyarintsev^{3,4}, Ilya R. Vasilenok³, Temur V. Kislukhin¹, Dmitriy V. Duplyakov^{1,2}

Corresponding author: Daria A. Shebonkina,
dariagnilomedova@mail.ru

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Contribution of the authors

Conception and study design: D.A. Shebonkina, L.V. Shekhovtsova, T.V. Kislukhin, D.V. Duplyakov
Data collection and analysis: D.A. Shebonkina, L.V. Shekhovtsova, M.S. Vasilyeva, A.L. Titov, M.I. Boyarintsev, I.R. Vasilenok
Statistical analysis: D.A. Shebonkina
Drafting the article: D.A. Shebonkina
Critical revision of the article: D.A. Shebonkina, D.V. Duplyakov
Final approval of the version to be published: all authors

ORCID

D.A. Shebonkina,
<https://orcid.org/0000-0001-8460-6058>
L.V. Shekhovtsova,
<https://orcid.org/0000-0003-1222-2674>
M.S. Vasilyeva,
<https://orcid.org/0009-0009-3233-0104>
A.L. Titov, <https://orcid.org/0000-0002-7549-5437>
M.I. Boyarintsev,
<https://orcid.org/0000-0003-3250-9023>
I.R. Vasilenok,
<https://orcid.org/0009-0002-4008-5769>
T.V. Kislukhin,
<https://orcid.org/0000-0003-2107-7499>
D.V. Duplyakov,
<https://orcid.org/0000-0002-6453-2976>

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¹ V.P. Polyakov Samara Regional Clinical Cardiology Dispensary, Samara, Russian Federation

² Samara State Medical University, Ministry of Health of the Russian Federation, Samara, Russian Federation

³ Limited Liability Company "Heart Clinic", Stary Oskol, Belgorod Region, Russian Federation

⁴ Belgorod State National Research University, Belgorod, Russian Federation

Abstract

Introduction: Predicting the development of new-onset atrial fibrillation in patients with acute myocardial infarction is challenging, and the search for potential predictors continues. Recent evidence suggests that atherosclerotic or embolic lesions in the arteries supplying the atria may influence the development of atrial fibrillation.

Objective: To assess the incidence of lesions in the arteries supplying the atria in patients with acute primary myocardial infarction who develop new-onset atrial fibrillation.

Methods: This two-center retrospective cohort study included 141 patients hospitalized in cardiology departments between 2020 and 2023 with acute myocardial infarction and new-onset atrial fibrillation. The coronary angiography protocol additionally included examination of the arteries supplying the atria and assessment of their involvement in areas of acute or chronic ischemia.

Results: Right-dominant coronary circulation was the predominant pattern, observed in 125 (88.7%) patients. In 46 (32.6%) patients, the atrial arteries were not involved in an area of acute or chronic ischemia; 34 (24.1%) patients had involvement or lesions of one atrial artery; two atrial arteries were located in an ischemic area in 35 (24.8%) patients; combined involvement of three atrial arteries was observed in 17 (12.1%) patients; and all four atrial arteries examined were located in an area of acute or chronic ischemia in 9 (6.4%) patients.

The sinus node artery was normal in 123 (84.8%) patients. The atrioventricular nodal artery was located in an area of acute ischemia in 38 (27.0%) cases and in an area of chronic ischemia in 29 (20.6%) cases; one additional patient (0.7%) had 90–99% stenosis of the artery itself. The right interatrial artery was located in an area of acute ischemia in 25 (17.7%) cases and in an area of chronic ischemia in 30 (21.3%) patients; 90–99% stenosis of this artery was detected in 11 (7.8%) additional patients. The left interatrial artery was normal or anatomically undeveloped in 106 (75.2%) patients, located in an area of acute ischemia in 8 (7.1%) patients, and located in an area of chronic ischemia in 24 (17.0%) patients; one (0.7%) patient had 90–99% stenosis.

Conclusion: In 67.4% of cases, the development of new-onset atrial fibrillation in patients with myocardial infarction was associated with acute or chronic disease of the atrial arteries. In 95% of patients with an infarction-related right coronary artery and new-onset atrial fibrillation, the atrial arteries – the atrioventricular nodal artery and the right interatrial artery – were located in an area of acute or chronic ischemia.

Keywords: atrial arteries; atrial ischemia; coronary angiography; myocardial infarction; new-onset atrial fibrillation



Поражение артерий предсердий при инфаркте миокарда и впервые возникшей фибрилляции предсердий: двухцентровое ретроспективное когортное исследование

Д.А. Шебонкина^{1,2}, Л.В. Шеховцова^{3,4}, М.С. Васильева^{1,2}, А.Л. Титов¹, М.И. Бояринцев^{3,4}, И.Р. Василенок³, Т.В. Кислухин¹, Д.В. Дупляков^{1,2}

Для корреспонденции: Дарья Александровна Шебонкина, dariagnilomedova@mail.ru

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Вклад авторов

Концепция и дизайн работы: Д.А. Шебонкина, Л.В. Шеховцова, Т.В. Кислухин, Д.В. Дупляков
Сбор и анализ данных: Д.А. Шебонкина, Л.В. Шеховцова, М.С. Васильева, А.Л. Титов, М.И. Бояринцев, И.Р. Василенок
Статистическая обработка данных: Д.А. Шебонкина
Написание статьи: Д.А. Шебонкина
Исправление статьи: Д.А. Шебонкина, Д.В. Дупляков
Утверждение окончательного варианта статьи: все авторы

ORCID

Д.А. Шебонкина, <https://orcid.org/0000-0001-8460-6058>
Л.В. Шеховцова, <https://orcid.org/0000-0003-1222-2674>
М.С. Васильева, <https://orcid.org/0009-0009-3233-0104>
А.Л. Титов, <https://orcid.org/0000-0002-7549-5437>
М.И. Бояринцев, <https://orcid.org/0000-0003-3250-9023>
И.Р. Василенок, <https://orcid.org/0009-0002-4008-5769>
Т.В. Кислухин, <https://orcid.org/0000-0003-2107-7499>
Д.В. Дупляков, <https://orcid.org/0000-0002-6453-2976>

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¹ Государственное бюджетное учреждение здравоохранения «Самарский областной клинический кардиологический диспансер им. В.П. Полякова», Самара, Российская Федерация

² Федеральное государственное бюджетное образовательное учреждение высшего образования «Самарский государственный медицинский университет» Министерства здравоохранения Российской Федерации, Самара, Российская Федерация

³ Общество с ограниченной ответственностью «Клиника Сердца», Старый Оскол, Белгородская область, Российская Федерация

⁴ Федеральное государственное автономное образовательное учреждение высшего образования «Белгородский государственный национальный исследовательский университет», Белгород, Российская Федерация

Аннотация

Актуальность. Прогнозирование развития впервые возникшей фибрилляции предсердий у пациентов с острым инфарктом миокарда является сложной задачей, продолжается поиск возможных предикторов. В последнее время появляются данные о влиянии атеросклеротического или эмболического поражения артерий, кровоснабжающих предсердия.

Цель. Изучить частоту встречаемости поражения артерий, кровоснабжающих предсердия, при развитии впервые возникшей фибрилляции предсердий у пациентов с острым первичным инфарктом миокарда.

Методы. В двухцентровое ретроспективное когортное исследование включен 141 пациент, госпитализированный в кардиологические отделения в период 2020–2023 гг. с острым инфарктом миокарда и впервые возникшей фибрилляцией предсердий. Протокол коронарографии дополнительно включал изучение артерий, кровоснабжающих предсердия, и их вовлечение в зоны острой или хронической ишемии.

Результаты. Доминирующим оказался правый тип кровоснабжения, отмеченный у 125 (88,7 %) пациентов. У 46 (32,6 %) пациентов артерии предсердий не были вовлечены в зону острой или хронической ишемии, у 34 (24,1 %) пациентов имелось поражение или вовлечение одной артерии предсердий, у 35 (24,8 %) пациентов в зоне ишемии находились две артерии предсердий, у 17 (12,1 %) пациентов наблюдалось сочетанное поражение трех артерий, а у 9 (6,4 %) пациентов все четыре исследуемые артерии предсердий находились в зоне острой или хронической ишемии.

Артерия синусового узла у 123 (84,8 %) пациентов оказалась без патологии. Артерия атриовентрикулярного узла в 38 (27 %) случаях находилась в зоне острой ишемии, а в 29 (20,6 %) случаях – в зоне хронической ишемии, еще у одного (0,7 %) пациента зарегистрирован 90–99 % стеноз в самой артерии. Правая межпредсердная артерия в 25 (17,7 %) случаях находилась в зоне острой ишемии, у 30 (21,3 %) пациентов артерия находилась в зоне хронической ишемии, еще у 11 (7,8 %) пациентов обнаружен ее 90–99 % стеноз. Левая межпредсердная артерия у 106 (75,2 %) пациентов была без патологии или анатомически не развита, у 8 (7,1 %) пациентов находилась в зоне острой ишемии, у 24 (17 %) пациентов артерия находилась в зоне хронической ишемии, у одного (0,7 %) пациента имелся 90–99 % стеноз.

Заключение. В 67,4 % случаев развитие впервые возникшей фибрилляции предсердий у пациентов с инфарктом миокарда сочеталось с острым или хроническим поражением артерий предсердий. У 95 % пациентов с инфаркт-связанной правой коронарной артерией и впервые возникшей фибрилляцией предсердий артерии предсердий (артерия атриовентрикулярного узла и правая межпредсердная артерия) находились в зоне острой или хронической ишемии.

Ключевые слова: артерии предсердий; впервые возникшая фибрилляция предсердий; инфаркт миокарда; ишемия предсердий; коронарография



Introduction

Atrial fibrillation (AF) is the most common cardiac arrhythmia. Electrophysiologically, AF is characterized by a high rate of asynchronous depolarization of atrial cells, resulting in loss of atrial contractile function and irregular ventricular contractions [1]. Two major mechanisms are distinguished in the pathogenesis of AF: electrical and structural remodeling. Electrical remodeling results from changes in ion-channel properties that affect activation and conduction in the atrial myocardium, whereas structural remodeling refers to changes in tissue architecture, including fibrosis and atrial enlargement [2]. Certain areas of the atrial myocardium – the distal portions of the pulmonary veins and the surrounding atrial myocardium – are involved in the initiation and maintenance of AF. This structural myocardial abnormality is caused by the arrhythmia itself and is often associated with cardiovascular disease. Such remodeling can maintain AF, thereby creating a kind of pathogenic vicious circle [3]. During the acute phase of myocardial infarction (MI), AF may also be promoted by left atrial volume overload [4] and the development of active myocardial inflammation [5].

New-onset AF (NOAF) occurs in 6–21% of patients with acute MI [6]. Minder J. et al. reported that NOAF developed in nearly one in ten patients with ST-segment elevation myocardial infarction (STEMI), resulting in more frequent stroke, major bleeding, and death than in patients without AF [7]. The development of NOAF in patients with acute MI is associated with a more severe clinical course and worse outcomes, particularly higher in-hospital mortality [8].

Currently, data on predictors of NOAF in patients with coronary artery disease remain conflicting. According to a multicenter study by Biccirè F.G. et al., prognostically significant risk factors for NOAF in acute coronary syndrome include the combination of coronary artery disease (or chronic obstructive pulmonary disease) with arterial hypertension, older age (≥ 75 years), systolic heart failure, and thyroid disease [9].

In addition to traditional risk factors for NOAF in patients with MI, the role of atherosclerotic or embolic lesions in the arteries supplying the atria has received increasing attention in recent years. Álvarez-García J. et al. [10] showed that occlusion of an atrial artery is relatively common after elective angioplasty of the right or circumflex coronary artery in patients who develop AF after percutaneous coronary intervention (PCI). Risk factors for AF included the diameter of atrial branches, the presence of ostial plaques, and the maximum balloon inflation pressure during stenting. Biccirè F.G. et al. also demonstrated that occlusion of atrial artery branches

was associated with early NOAF (within 24 hours) after STEMI [11]. Thus, atrial artery disease may be one of the predictors of NOAF in patients with acute MI.

The aim of this study was to assess the incidence of lesions in the arteries supplying the atria in patients with acute primary MI who develop NOAF.

Methods

This two-center retrospective cohort study included patients hospitalized in cardiology departments between 2020 and 2023 with acute MI and NOAF.

Inclusion criteria: age ≥ 18 years; STEMI or non-ST-segment elevation myocardial infarction (NSTEMI); no history of AF; NOAF during the MI; coronary angiography with or without stenting; and consent to participate in the study.

Exclusion criteria: conservative management; previous MI, PCI, or coronary artery bypass grafting; a history of thyroid disease; active malignancy; and lack of consent to participate in the study.

The coronary angiography protocol additionally included examination of the atrial arteries (Table 1): the sinus node artery (SNA), atrioventricular nodal artery (AVNA), right interatrial artery (RIAA), and left interatrial artery (LIAA). Blood-flow parameters in the atrial arteries, the arterial location relative to the territories of the major coronary arteries, and the presence of stenosing atherosclerotic plaques or thromboemboli were assessed. Acute ischemia in the territory of an atrial artery was defined as its location within or distal to a stenosis in the territory of the infarct-related artery (IRA). Chronic ischemia was defined as involvement of an atrial artery in the territory of a major coronary artery with a chronic hemodynamically significant stenosis, or the presence of 90–99% stenosis in the atrial artery itself. The relationship between the IRA and the atrial arteries was also analyzed.

The study was conducted in accordance with Good Clinical Practice guidelines and the principles of the Declaration of Helsinki. Informed consent was obtained from the patients for the use of their medical data for research purposes.

Statistical analysis

Statistical analysis was performed using Statistica 10.0. The Shapiro – Wilk test was used to assess the normality of quantitative variables. Quantitative variables with a non-normal distribution are presented as the median (Me) and interquartile range [Q1; Q3]. Categorical variables are presented as frequencies with absolute values and percentages, n (%).

Table 1. Parameters used to assess the atrial arteries

Assessment domain	Parameters
Atrial arteries	SNA; AVNA; RIAA; LIAA
Acute/chronic ischemia of an atrial artery	No signs of acute or chronic ischemia (1); area of acute ischemia (2); area of chronic ischemia; 90–99% stenosis of the atrial artery; no-reflow phenomenon (failure to restore blood flow)
Blood flow before revascularization	Artery not visualized; TIMI grade I; TIMI grade II; TIMI grade III
Blood flow after revascularization	TIMI grade 0; TIMI grade I; TIMI grade II; TIMI grade III
Territory	Right coronary artery territory; circumflex artery territory
Origin relative to stenosis/occlusion	Proximal to the stenosis/occlusion; at the level of the stenosis/occlusion; distal to the stenosis/occlusion (3)

Note. AVNA, atrioventricular nodal artery; LIAA, left interatrial artery; RIAA, right interatrial artery; SNA, sinus node artery; TIMI, Thrombolysis in Myocardial Infarction flow grade.

Results

Between 2020 and 2023, a total of 9,504 patients with acute coronary syndrome were hospitalized at the V.P. Polyakov Samara Regional Clinical Cardiology Dispensary (Samara) and Heart Clinic LLC (Sary Oskol). Various forms of AF were present in 1,891 (19.9%) patients, including paroxysmal AF in 745 (39.4%) patients. The inclusion criteria were met by 141 patients (19.8% of all patients with paroxysmal AF occurring during acute MI); 83 (59%) were men, and the median age was 68 [61; 76] years. Arterial hypertension was present in 132 (93.6%) patients, diabetes mellitus in 45 (31.9%), including insulin-dependent diabetes in 6 (13.3%), and a body mass index ≥ 30 kg/m² in 65 (46%) patients. Twelve (8.5%) patients had a history of stroke. Because the study was conducted during the COVID-19 pandemic, acute respiratory illnesses were documented during hospitalization in 10 (7.1%) cases, including confirmed COVID-19 in 5 (3.5%) patients (Table 2).

STEMI was diagnosed in 107 (75.8%) patients: 55 (51.4%) had anterior MI and 52 (48.6%) had inferior MI. A further 34 (24.1%) patients had NSTEMI. PCI of the IRA was performed in 121 (85.8%) patients; 16 (11.3%) patients had indications for coronary artery bypass grafting, while in 4 (2.8%) patients coronary artery stenoses were hemodynamically insignificant.

During PCI, glycoprotein IIb/IIIa receptor inhibitors were used in 26 (18.4%) cases. Their administration was prompted by no-reflow/slow-reflow in 53.8% of cases, primary extensive IRA thrombosis in 23.1%, stent thrombosis in 11.5%, technically complex PCI in 7.7%, and intraprocedural thrombosis in 3.8%.

Table 2. Baseline patient characteristics (n = 141)

Characteristic	Value
Age, years, Me [Q1; Q3]	68 [61; 76]
Men, n (%)	83 (59)
Women, n (%)	58 (41)
COVID-19, n (%)	5 (3.5)
Acute respiratory illness, n (%)	3 (2.1)
Pneumonia, n (%)	2 (1.4)
Current smoking, n (%)	34 (24.1)
Former smoking, n (%)	15 (10.6)
Diabetes mellitus, n (%)	45 (31.9)
Insulin-dependent, n (%)	6 (13.3)
Arterial hypertension, n (%)	132 (93.6)
Stroke, n (%)	12 (8.5)
Obesity, n (%)	65 (46)
Anterior STEMI, n (%)	55 (39)
Inferior STEMI, n (%)	52 (36.9)
NSTEMI, n (%)	34 (24.1)
IRA stenting, n (%)	121 (85.8)

Note. IRA, infarct-related artery; NSTEMI, non-ST-segment elevation myocardial infarction; STEMI, ST-segment elevation myocardial infarction.

Thrombolytic therapy was administered to 28 (19.9%) patients, predominantly before hospital admission (96.4%). It was effective in 12 (42.8%) patients. PCI after thrombolytic therapy was performed in 23 (82%) patients.

Table 3. Timing of NOAF onset after MI, *n* (%)

Setting	Within 24 h	24–48 h	>48 h	Unknown
Before CAG/PCI, <i>n</i> = 92 (65.3%)	79 (85.9)	6 (6.5)	1 (1.1)	6 (6.5)
During CAG/PCI, <i>n</i> = 7 (5%)	6 (85.7)	0 (0)	0 (0)	1 (14.3)
After CAG/PCI, <i>n</i> = 42 (29.8%)	11 (26.2)	18 (42.9)	10 (23.8)	3 (7.1)

Note. CAG, coronary angiography; MI, myocardial infarction; NOAF, new-onset atrial fibrillation; PCI, percutaneous coronary intervention.

Echocardiography was performed on days 1–2 after MI. The mean left atrial (LA) diameter was 40 mm [37; 43]; the mediolateral LA dimension was 42 mm [38; 46], the superoinferior dimension was 55 mm [50; 60], the LA volume was 62 mL [50; 80], and the LA volume index was 40.95 mL/m² [36.6; 49.5]. The left ventricular ejection fraction was 49% [42; 56].

Drug therapy was administered in accordance with the clinical practice guidelines of the Ministry of Health of the Russian Federation [12; 13]. During hospitalization, 55 (39%) patients did not receive oral anticoagulants; 47 (33.3%) of them received parenteral anticoagulants, while 8 (5.7%) patients received dual antiplatelet therapy without anticoagulants. Anticoagulant therapy was not recommended after discharge for 17 (13.2%) patients because AF was considered a complication of MI; these patients had no AF recurrence during hospitalization and were in sinus rhythm at discharge.

Characteristics of new-onset atrial fibrillation

NOAF was documented in 135 (95.7%) patients; an additional 6 (4.3%) patients had a combination of NOAF and atrial flutter. In 84 (59.6%) patients, NOAF was documented on admission; in 57 (40.4%) patients, it developed during hospitalization, including after PCI in 42 (29.8%) patients. The timing of AF onset is presented in detail in Table 3.

Tachysystolic AF was recorded in 125 (88.7%) patients, the normosystolic form in 13 (9.2%), and bradysystolic AF in 3 (2.1%) patients.

At hospital discharge, sinus rhythm had been restored in 122 (86.5%) patients: pharmacologically in 114 (93.4%) cases, by electrical cardioversion in 6 (4.9%), and spontaneously in 2 (1.6%). Seven (5%) patients died before sinus rhythm was restored.

AF recurred during hospitalization in 21 (14.9%) patients; sinus rhythm was restored in 14 (66.7%) of them. AF persisted at discharge in 12 (8.5%) patients; in 8 (5.7%) patients, sinus rhythm could not be restored either pharmacologically or by electrical cardioversion.

Characteristics of the coronary arterial bed

Right-dominant coronary circulation was the predominant pattern, observed in 125 (88.7%) patients; left dominance was present in 13 (9.2%) patients, and balanced circulation in 3 (2.1%). The IRA was the left anterior descending artery (LAD) in 69 (49%) patients, the right coronary artery (RCA) in 42 (29.8%), the circumflex artery (LCx) in 26 (18.4%), and the left main coronary artery in 4 (2.8%) cases.

In 46 (32.6%) patients – approximately one-third – the atrial arteries were intact, that is, they were not involved in an area of either acute or chronic ischemia. One atrial artery was affected or involved in 34 (24.1%) patients; two atrial arteries were located in an ischemic area in 35 (24.8%) patients; combined involvement of three atrial arteries was observed in 17 (12.1%) patients; and all four atrial arteries examined were located in an area of acute or chronic ischemia in 9 (6.4%) patients (Table 4).

The SNA originated from the RCA in 107 (73.8%) patients and from the LCx in the remaining patients. In 2 (1.4%) patients, dual origin from both the RCA and LCx was recorded; therefore, subsequent calculations were based on 143 SNAs. The SNA was normal in 121 (84.6%) patients, was undeveloped in 7 (4.9%), was located in an area of acute ischemia in 7 (4.9%), and was located in an area of chronic ischemia in 8 (5.6%), including 7 patients with 90–99% stenosis.

On analysis of the AVNA, no involvement was found in 73 (51.8%) patients; the artery was located in an area of acute ischemia in 38 (27.0%) cases and in an area of chronic ischemia in 29 (20.6%) cases; one (0.7%) patient had 90–99% AVNA stenosis.

The RIAA showed no signs of occlusion or thrombosis in 75 (53.2%) cases, acute ischemia in 25 (17.7%) cases, and chronic ischemia in 30 (21.3%) patients; an additional 11 (7.8%) patients had 90–99% stenosis.

The LIAA was normal or anatomically undeveloped in 106 (75.2%) patients, located in an area of acute ischemia in 8 (7.1%) patients, and located in an area of chronic

Table 4. Atrial artery involvement according to the IRA territory, *n* (%)

Pattern of atrial artery involvement	Coronary artery			
	LAD	LCx	RCA	Left main coronary artery
No atrial artery involvement	33 (23.4)	9 (6.4)	4 (2.8)	0 (0)
SNA	2 (1.4)	0 (0)	0 (0)	0 (0)
AVNA	3 (2.1)	3 (2.1)	7 (5)	0 (0)
RIAA	4 (2.8)	4 (2.8)	6 (4.3)	0 (0)
LIAA	1 (0.7)	2 (1.4)	1 (0.7)	1 (0.7)
AVNA + RIAA	11 (7.8)	0 (0)	11 (7.8)	0 (0)
SNA + RIAA	3 (2.1)	0 (0)	0 (0)	0 (0)
SNA + AVNA	2 (1.4)	0 (0)	1 (0.7)	1 (0.7)
AVNA + LIAA	3 (2.1)	1 (0.7)	1 (0.7)	0 (0)
RIAA + LIAA	0 (0)	1 (0.7)	0 (0)	0 (0)
AVNA + RIAA + LIAA	3 (2.1)	3 (2.1)	5 (3.5)	0 (0)
SNA + AVNA + RIAA	0 (0)	0 (0)	3 (2.1)	1 (0.7)
SNA + AVNA + LIAA	0 (0)	0 (0)	1 (0.7)	0 (0)
SNA + LIAA + RIAA	0 (0)	0 (0)	1 (0.7)	0 (0)
SNA + AVNA + RIAA + LIAA	3 (2.1)	3 (2.1)	3 (2.1)	0 (0)

Note. AVNA, atrioventricular nodal artery; IRA, infarct-related artery; LAD, left anterior descending artery; LIAA, left interatrial artery; LCx, left circumflex artery; RCA, right coronary artery; RIAA, right interatrial artery; SNA, sinus node artery.

ischemia in 24 (17.0%) patients; one (0.7%) patient had 90–99% stenosis.

Complications of myocardial infarction

The course of MI was complicated by acute heart failure classified as Killip class II in 7 (5%) patients, Killip class III in 10 (7%), and Killip class IV (cardiogenic shock) in 19 (13.5%) patients. Intra-aortic balloon counterpulsation was required in 5 (3.5%) patients, temporary cardiac pacing in 9 (6.4%), mechanical ventilation in 13 (9.2%), and vasopressors in 22 (15.6%) patients. Among the 19 patients with cardiogenic shock, the IRA was the RCA in 10, the LAD in 8, and the LCx in 1. When the IRA was the RCA, atrial artery involvement was present in 9 of 10 patients, including involvement of two, three, or four atrial arteries.

During hospitalization, ischemic stroke was diagnosed in 3 (2.1%) patients. Hemorrhagic complications (Bleeding Academic Research Consortium [BARC] grades 1–3b) developed in 10 (7%) patients. The sources were the

gastrointestinal tract in 2 (20%), the urinary tract in 2 (20%), and other sites, including hemopericardium, in 6 (60%). Blood transfusions were administered to 6 (4.3%) patients. Recurrent ischemia during hospitalization occurred in 9 (6.4%) patients; PCI was not performed in one patient because of multivessel disease. In-hospital mortality was 8.5% (12 patients). Atrial artery involvement was observed in 10 of the 12 patients; in two cases, all four atrial arteries were located in an area of acute or chronic ischemia. After discharge, 3 (2.1%) patients died within 30 days of MI onset.

Discussion

Identifying the cause of NOAF in patients with MI is crucial because even asymptomatic AF requires adjustment of antithrombotic therapy and a change in the subsequent management strategy. Numerous factors that contribute to the development of NOAF have been discussed in the literature. In our opinion, the most important independent predictors of NOAF in patients with MI are the

degree of LA volume overload, as assessed by magnetic resonance imaging, and LA volume deformation [14].

It has been shown that involvement of more than 50% of at least one atrial arterial branch – the SNA, AVNA, or RIAA – as well as the RCA, is associated with a substantially higher risk of AF within one year [15]. The role of atrial arteries in the development of NOAF in patients with MI has recently been studied actively [16]. In the study by Biccirè F.G. et al., impaired atrial perfusion was an important pathogenetic mechanism underlying the development of AF, as evidenced by the higher frequency of SNA, AVNA, and RIAA occlusion in these patients than in those who remained in sinus rhythm after PCI [11].

According to our study, patients with a first MI and NOAF more often had STEMI than NSTEMI (75.9% and 24.1%, respectively). In most patients, NOAF developed before hospital admission. When it occurred during hospitalization, it generally developed after PCI within 24–48 hours.

The AVNA (43.3%) and RIAA (37.6%) were most frequently located in areas of acute or chronic ischemia, either individually or in combination. SNA blood flow was virtually unimpaired in 85% of patients, probably because of the characteristics of its origin from the parent artery. In most cases, the LIAA was undeveloped or showed no pathology. Our findings confirm the results reported by Biccirè F.G. et al. [11].

Two-thirds of patients (66.7%) who developed NOAF in the setting of MI had compromised atrial arteries, defined as arteries located in an area of acute or chronic ischemia. Among patients with an RCA as the IRA, acute or chron-

ic ischemia of the atrial arteries was recorded in 90.5% of cases. These patients also more frequently had combined involvement of two (31.6%), three (26.3%), or even four (2.6%) atrial arteries. These findings are consistent with the results of the study by Pokorney S.D. et al. [15].

At the same time, it should be noted that 46 (32.6%) patients had no involvement of any atrial artery. This situation was most common when the LAD was the IRA (71.8%).

Thus, our findings emphasize the important role of the condition of the atrial arteries in the development of NOAF in patients with MI, particularly inferior MI. However, a multicenter prospective study is required to confirm this hypothesis.

Study limitations

The limitations of this study include its retrospective design and the absence of a control group of patients with acute primary MI without AF.

Conclusion

Our findings indicate that in 67.4% of cases, the development of NOAF in patients with MI was associated with the atrial arteries being located in an area of acute or chronic ischemia. Among patients with RCA involvement and NOAF, 95% had acute or chronic ischemia in the territories of the AVNA and RIAA. However, these conclusions are based on a limited sample and require confirmation in future studies.

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