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Predictors of New-Onset Atrial Fibrillation in Patients With Chronic Coronary Artery Disease After Coronary Artery Bypass Grafting: a Prospective, Observational, Single-Centre, Non-Randomized Study

Aim	To study the factors that influence the occurrence of postoperative atrial fibrillation (POAF) in patients with chronic ischemic heart disease (IHD) after coronary artery bypass grafting (CABG).
Material and methods	This single-center prospective observational non-randomized study included 152 patients with chronic IHD. Mean age of patients was 64.4±5.9 years. All patients after CABG were divided into two groups based on the occurrence of atrial fibrillation (AF) in the early postoperative period: group 1, with POAF (n=43; 28.3%) and group 2, without POAF (n=109; 71.7%). The primary study endpoint was new-onset POAF in the early postoperative (hospital) period after CABG. The secondary study endpoint was in-hospital postoperative complications (non-fatal/fatal acute coronary syndrome (ACS), non-fatal/fatal stroke, major bleeding, death).
Results	Patients with POAF had significantly more pronounced structural and functional changes in the heart than patients with preserved sinus rhythm after CABG: larger left ventricular (LV) volume, greater LV myocardial mass, lower LV systolic function parameters and impaired diastolic function, and an enlarged left atrial (LA) cavity. Analysis of in-hospital complications did not show any differences between the groups associated with the development of POAF. The following risk factors for POAF were identified: age older than 65 years (p=0.022), body mass index ≥30.5 kg/m^2 (p=0.020), epicardial adipose tissue thickness >10.5 mm (p=0.015), indexed LA volume >33 ml/m^2 (p<0.001), LV myocardial mass index >115 g/m^2 (p=0.042), left main coronary artery disease >50% (p=0.043), duration of cardiopulmonary bypass during CABG >60 min (p=0.019), blood potassium concentration in the early postoperative period after CABG (on the first day) <3.6 mmol/l (p<0.001), and pericardial effusion volume in the early postoperative period >88 ml (p<0.001).
Conclusion	Determining the risk of developing POAF is important and necessary for the closest monitoring of a patient with chronic IHD in the postoperative period.

Keywords	Chronic ischemic heart disease; coronary artery bypass grafting; postoperative atrial fibrillation; predictors of atrial fibrillation
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Introduction

Atrial fibrillation (AF) is a prevalent complication that arises in approximately 30% of cases following coronary artery bypass grafting (CABG) procedures [1]. The mechanisms underlying the development of postoperative AF are most often associated with a combination of several factors. Patients with chronic coronary heart disease (CHD) and coronary artery stenosis frequently present with additional comorbidities, including arterial hypertension, obesity, diabetes mellitus, and heart failure (HF), which can trigger the development of postoperative AF [2–4]. Cardiac arrhythmia in the form of atrial fibrillation (AF) can exacerbate the patient's condition in the immediate postoperative period, thereby increasing the risk of thromboembolic complications, bleeding, HF, and in-hospital mortality [2, 3].

Objective

The objective of this study is to examine the factors that contribute to the development of postoperative AF in patients with chronic CHD following CABG.

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Central illustration. Predictors of New-Onset Atrial Fibrillation in Patients With Chronic Coronary Artery Disease After Coronary Artery Bypass Grafting: a Prospective, Observational, Single-Centre, Non-Randomized Study

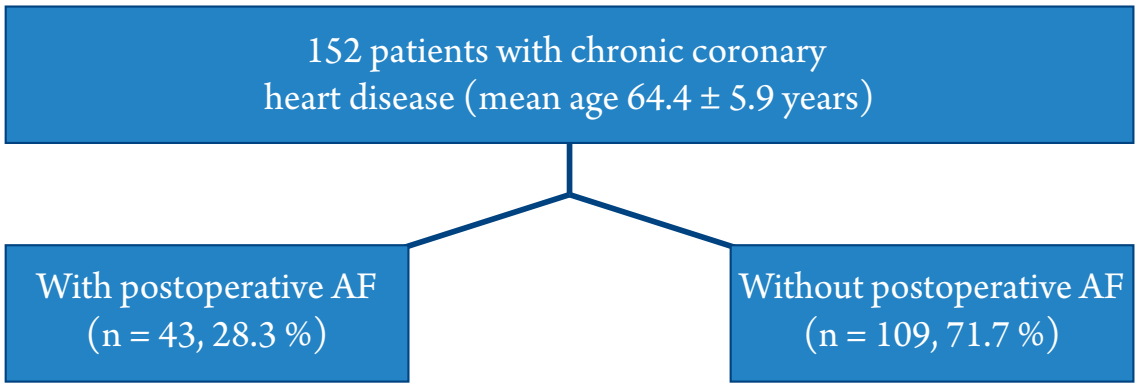

Table. Analysis of the factors associated with the risk of postoperative paroxysmal AF in patients who underwent open-heart myocardial revascularization via CABG

Parameter	B*	p
Straight line segment	1.59	0.0038
Age > 65 years	1.011	0.022
Sex (female)	0.685	0.338
BMI ≥ 30.5 kg/m2	1.254	0.020
LVMI > 115 g/m2	1.338	0.042
LAVI > 33 mL/m2	1.449	< 0.001
EAT thickness > 10.5 mm	0.998	0.015
LCA stenosis > 50 %	1.014	0.040
CPB time (during CABG with CPB) > 60 min	1.480	0.019
Potassium < 3.6 mmol/L	–0.756	< 0.001
Magnesium < 0.6 mmol/L	–0.239	0.065
Volume of pericardial effusion > 88 mL	1.147	< 0.001

* B, regression coefficient; AF, atrial fibrillation; CABG, coronary artery bypass grafting; BMI, body mass index; LVMI, left ventricular mass index; LAVI, left atrial volume index; EAT, epicardial adipose tissue; LCA, left coronary artery; CPB, cardiopulmonary bypass.

Material and Methods

A single-center prospective observational non-randomized study was conducted. The material consisted of the outcomes of treatment and examination of 152 patients with chronic CHD who underwent myocardial revascularization through CABG. The mean age of the patients was 64.4 ± 5.9 years (ranging from 36 to 79 years).

The inclusion criteria were the presence of chronic CHD and indications for myocardial revascularization through CABG.

The exclusion criteria were as follows: age under 18 years; cardiac arrhythmias in the form of supraventricular extrasystole (not characteristic of healthy individuals); history of atrial tachyarrhythmias; history of AF; acute period of myocardial infarction (MI), a period of less than 3 months following MI; left ventricular (LV) aneurysm; severe systolic LV dysfunction (left ventricular ejection fraction (LVEF) < 35%); pronounced valve dysfunction, including in the context of CHD; hemodynamically significant carotid artery lesion (stenosis of more than 70%); thyroid gland diseases; history of open cardiac surgery; history of alcohol or other substance abuse.

The patients were enrolled in the study between April 2019 and March 2021. Informed consent was obtained in accordance with the protocols of the ethics committee (extract from the protocol No. 5 of the ethics committee of the Tver State Medical University, dated March 29, 2021).

The surgical procedure was performed via coronary artery bypass grafting (CABG; n = 152), which was conducted in accordance with the established standard technique utilizing autologous arterial grafts in cardiopulmonary bypass (CPB) and an off-pump setting [5].

All patients who had undergone CABG were divided into two groups based on the presence of new-onset postoperative AF: Group 1 with postoperative AF (n = 43, 28.3%), Group 2 without postoperative AF (n = 109, 71.7%). Table 1 presents the baseline preoperative characteristics of patients depending on the onset of postoperative AF.

Table 1. Baseline characteristics of patients who underwent CABG depending on the presence of new-onset postoperative AF

Parameter	Patients with chronic CHD after CABG (n = 152)		p
	with postoperative AF (n = 43)	without postoperative AF (n = 109)	
Age, years (M ± m)	65.8 ± 4.7	60.5 ± 5.5	0.007
Male, n (%)	24 (55.8)	71 (65.1)	0.188
Smoking, n (%)	35 (81.4)	84 (77.1)	0.364
BMI, kg/m^2, (M ± m)	32.3 ± 3.3	29.5 ± 3.7	0.061
BMI > 30 <35 kg/m^2, n (%)	29 (67.4)	54 (49.5)	0.034
BMI > 35 kg/m^2, n (%)	11 (25.6)	17 (15.6)	0.117
Hypertension, n (%)	34 (79.1)	82 (75.2)	0.392
Diabetes mellitus type 2, n (%)	11 (25.6)	21 (19.3)	0.258
Myocardial infarction, n (%)	29 (67.4)	72 (66.1)	0.515
Chronic kidney disease, n (%)	8 (18.6)	15 (13.8)	0.302
COPD, n (%)	6 (14.0)	12 (11.0)	0.399
Peripheral artery disease, n (%)	6 (14.0)	11 (10.1)	0.337
Chronic heart failure, n (%)	9 (20.9)	12 (11.0)	0.093
History of CVA/TIA, n (%)	4 (9.3)	7 (6.4)	0.379
History of PCI, n (%)	8 (18.6)	17 (15.6)	0.409
EuroSCORE II (M ± m)	2.4 ± 0.6	2.1 ± 0.5	0.642
SYNTAX (M ± m)	27.9 ± 3.8	26.9 ± 4.0	0.284
CHA2DS2 VASc (M ± m)	3.2 ± 1.2	2.7 ± 1.4	0.059

CABG, coronary artery bypass grafting; AF, atrial fibrillation; CHD, coronary heart disease; BMI, body mass index; COPD, chronic obstructive pulmonary disease; CVA, cerebrovascular accident; TIA, transient ischemic attack; PCI, percutaneous coronary intervention.

The primary endpoint of the study was new-onset postoperative AF during the early postoperative (hospital) period following CABG. The secondary endpoint of the study was the incidence of hospital postoperative complications, including non-fatal/fatal acute coronary syndrome (ACS), non-fatal/fatal cerebrovascular accident, major bleeding, and death.

Throughout the surgical procedure and on the subsequent day, the patient was monitored continuously via electrocardiogram (ECG) in the intensive care unit. In the event of suspected cardiac arrhythmias and/or patient-reported symptoms, an additional 12-lead ECG was performed. In accordance with the 2012 HRS/EHRA/ECAS consensus and confirmed examination data, AF was diagnosed in 2020 based on Holter monitoring data, which revealed an arrhythmic episode of at least 30 seconds [6].

All patients underwent a comprehensive examination prior to and immediately following the CABG procedure. Standard examinations were conducted during the preoperative period. ECG was recorded using a 12-lead electrocardiograph ECG-1012 (Dixion, Russia), and a 24-hour ECG monitoring was conducted using a MYOCARD-HOLTER device (NIMP ESN, Russia).

Echocardiography was conducted using a Philips CX50 device (USA) in accordance with standard protocols. Moreover, the maximum thickness of epicardial adipose tissue (EAT) at end systole was assessed in the parasternal view along the long axis of the LV between the right ventricle and the ascending aorta [7]. Angiography was performed using a Philips Integris device (Netherlands) under local anesthesia (20 mL of 0.5% novocaine solution).

Following surgical intervention, all patients were administered the best possible drug therapy with blood lipid profile control, as well as antihypertensive and antiplatelet therapies [8, 9]. No differences were observed in the pharmacological treatments administered to the study groups, which included beta blockers (BBs), antiplatelet agents, statins, and angiotensin-converting enzyme (ACE) inhibitors.

The statistical processing of the obtained data was conducted using the IBM SPSS Statistics 23.0 (Armonk, USA) and WinPEPI Portal 11.61 (J. H. Abramson) software packages.

The dependent variable was the presence or absence of postoperative AF. The data are presented as mean and standard deviation (M ± SD) and absolute values and percentages. To ascertain the magnitude of the discrepancy in quantitative variables between the two samples, a Student's t-test was employed for independent samples. In lieu of a preliminary estimation of the distributions of expected values within the samples, bootstrap versions of the test were employed. The Fisher exact test was employed to ascertain the statistical significance of the observed difference between qualitative variables. Subsequently, all independent variables that were statistically significantly associated with the dependent variable were included in the multiple logistic regression model. Prior to this, all

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Table 2. Characteristics of the structural and functional parameters of the heart following CABG, depending on the presence of new-onset postoperative AF

Parameter	Patients with chronic CHD after CABG (n = 152)		p
	with post-operative AF (n = 43)	without post-operative AF (n = 109)	
LVEDV, mL	114.2 ± 41	95 ± 31	0.002
LVMI, g/m2	128 ± 34	107 ± 28	< 0.001
LVEF, %	50.0 ± 9.2	57.4 ± 10.4	0.015
E, cm/sec	83.3 ± 21	74.8 ± 17.4	0.012
E/A	1.12 ± 0.4	0.94 ± 0.27	0.002
E/E'	8.8 ± 2.4	6.5 ± 2.2	< 0.001
E/E' > 9, n (%)	30 (69.8)	58 (53.2)	0.045
LA dimension, cm	43.8 ± 5.9	38.7 ± 5.3	< 0.001
LAVI, mL/m^2	37.7 ± 8.8	30.1 ± 7.3	< 0.001
EAT thickness, mm	12.3 ± 0.7	8.4 ± 0.4	< 0.001
RAVI, mL/m^2	27.7 ± 9.0	24.1 ± 5.8	< 0.001
TAPSE, mm	22.2 ± 3.8	21.6 ± 2.3	0.236
PASP, mmHg	34.2 ± 7.7	28.3 ± 6.8	< 0.001
PASP > 35 mmHg, n (%)	22 (51.2)	32 (29.4)	0.010
HR, bpm	67.3 ± 10.1	65.5 ± 9.8	0.314
P duration, ms	112.2 ± 14.8	113.1 ± 12.5	0.705
PR interval, ms	185.4 ± 28.8	163.7 ± 29.4	< 0.001

CABG, coronary artery bypass grafting; AF, atrial fibrillation; CHD, coronary heart disease; LVEDV, left ventricular end-diastolic volume; LVMI, left ventricular mass index; LVEF, left ventricular ejection fraction; LA, left atrium; LAVI, left atrial volume index; EAT, epicardial adipose tissue; RAVI, right atrial volume index; PASP, pulmonary artery systolic pressure; HR, heart rate.

Table 3. Intraoperative characteristics of patients who underwent CABG depending on the presence of postoperative AF

Parameter	Patients with chronic CHD after CABG (n = 152)		p
	with posto-perative AF (n = 43)	without post-operative AF (n = 109)	
CABG with CPB, n (%)	35 (81.4)	57 (52.3)	0.001
Patients who underwent CABG with CPB (n = 92)			
CPB duration, min	68.6 ± 10.3	53.7 ± 4.5	< 0.001
Duration of aortic clamping, min	54.2 ± 9.3	45.8 ± 2.49	< 0.001
Patients who underwent CABG (n = 152)			
Duration of mechanical ventilation, h	7.5 ± 2.6	6.3 ± 0.44	< 0.001
Time in ICU, h	33.95 ± 10.1	22.2 ± 2.8	< 0.001
Laboratory parameters on day 1 after surgery (n = 152)			
Potassium, mmol/L	3.72 ± 0.97	4.49 ± 1.0	< 0.001
Magnesium, mmol/L	0.72 ± 0.33	1.1 ± 0.47	< 0.001

CABG, coronary artery bypass grafting; AF, atrial fibrillation; CHD, coronary heart disease; CPB, cardiopulmonary bypass.

quantitative variables were converted to dichotomous variables. The forced incorporation method was employed.

A type I error value of 5% (p = 0.05) was employed as a threshold for assessing statistical significance.

Results

A total of 152 patients were included in the study, of whom 92 (60.5%) underwent CABG with the use of CPB and 60 (39.5%) were subjected to off-pump CABG. As demonstrated in Table 1, the mean age of patients with postoperative AF was significantly higher than that of patients with preserved sinus rhythm following CABG: 65.8 ± 4.7 and 60.5 ± 5.5 years, respectively (p = 0.007).

Patients with postoperative AF exhibited significantly more pronounced structural and functional alterations in the heart compared to those with preserved sinus rhythm following CABG (Table 2). These included an increase in LV volume, LV mass, and decreased indicators of LV systolic functions, and LV diastolic function impairment, as well as left atrial (LA) enlargement. The EAT thickness was found to be significantly greater in patients with postoperative AF compared to those with a preserved sinus rhythm following CABG: 12.3 ± 0.7 mm and 8.4 ± 0.4 mm, respectively (p < 0.001).

Upon examination of the ECG results, no differences were observed between the groups with respect to heart rate (HR) during the preoperative period and the duration of the P wave on the standard ECG. Concurrently, the PR interval was initially found to be statistically significantly longer in patients with postoperative AF compared to those without AF (Table 2).

A comparative assessment of the severity of coronary artery lesions between patients with AF and those with normal sinus rhythm after CABG revealed a significant difference in the frequency of left coronary artery (LCA) stenosis. This was more frequently observed in group 1: n = 19 (44.2%) in group 1 and n = 27 (24.8%) in group 2 (p = 0.017). No notable discrepancies were observed between the groups with respect to the prevalence of atherosclerotic lesions in the LAD, RCA, and LCX.

Table 3 presents intraoperative data indicating that patients with postoperative AF experienced a longer duration of CPB, mechanical ventilation, and aortic clamping, as well as a prolonged ICU stay. Furthermore, patients with postoperative AF had statistically significantly lower blood levels of potassium and magnesium (Table 3).

Table 4 presents the results of a comparative analysis of the incidence of complications in the early postoperative period between groups of patients depending on the development of postoperative AF. The majority of the evaluated indicators exhibited no significant differences, including the onset of non-fatal/fatal ACS and CVA, major postoperative

Table 4. Hospital complications depending on the presence of new-onset postoperative AF

Complication	Patients with chronic CHD after CABG (n = 152)		p
	with postoperative AF (n = 43)	without postoperative AF (n = 109)	
Myocardial infarction, n (%)	4 (9.3)	2 (1.3)	0.054
Acute heart failure, n (%)	3 (7.0)	3 (2.8)	0.138
Acute respiratory failure, n (%)	1 (2.3)	3 (2.8)	0.740
Bleeding – resternotomy, n (%)	2 (4.65)	3 (2.8)	0.437
Multiple organ system failure, n (%)	1 (2.3)	1 (0.9)	0.487
Pneumonia, n (%)	7 (16.3)	8 (7.3)	0.090
Pleurisy, n (%)	12 (27.9)	10 (9.2)	0.024
Pericarditis, n (%)	33 (76.7)	21 (19.3)	< 0.001
Pericardial effusion volume < 100 mL, n (%)	27 (62.8)	18 (16.5)	< 0.001
Pericardial effusion volume > 100 < 500 mL, n (%)	6 (14.0)	3 (2.8)	0.016
Pericardial effusion volume > 500 mL, n (%)	0	0	–
CVA, n (%)	1 (2.3)	2 (1.8)	0.634
Post-hypoxic encephalopathy, n (%)	5 (11.6)	4 (3.7)	0.073
Death, n (%)	1 (2.3)	0	0.283

AF, atrial fibrillation; CHD, coronary heart disease; CABG, coronary artery bypass grafting; CVA, cerebrovascular accident.

bleeding, and hospital mortality. Significant discrepancies were observed in the incidence of complications, including pleurisy and pericarditis.

A multiple logistic regression analysis was performed to identify the factors associated with the onset of postoperative AF following CABG (Table 5).

Discussion

Postoperative AF most frequently manifests within the initial four to five days following surgery. Thereafter, the sinus rhythm typically reestablishes itself, although the potential for paroxysmal AF persists. In the postoperative period, pro-inflammatory cytokines (C-reactive protein, interleukin-2 (IL-2), IL-6, and tumor necrosis factor alpha) are released in a maximum concentration on days 1–3 after surgery. This may serve as a substrate for the development of postoperative AF. The present study was limited by several factors, which precluded an analysis of the levels of pro-inflammatory cytokines between groups with postoperative AF and normal sinus rhythm, as well as in CABG with CPB and off-pump CABG. Cardiac surgery is frequently conducted with the assistance of CPB. The impact of CPB during CABG on the risk of developing postoperative AF remains inconclusive. The negative impact of CPB is attributed to the contact of blood with the foreign surfaces of the CPB system, which results in the release of inflammatory mediators, particularly a significant quantity of IL-6 and IL-8 [10]. In contemporary cardiac surgery, biocompatible materials and miniature circuits are employed to mitigate leukocyte aggregation, stimulate complement cascades, facilitate coagulation, and attenuate the generation of pro-inflammatory cytokines. Concurrently, the classical complement cascade can also

Table 5. Results of multiple regression analysis to identify the factors associated with the risk of postoperative AF in patients with chronic CHD who underwent CABG

Parameter	B	β (OR)	p
Straight line segment	1.59	4.90	0.0038
Age > 65 years	1.011	2.75	0.022
BMI ≥ 30.5 kg/m²	1.254	3.50	0.02
LVMI > 115 g/m²	1.338	3.81	0.042
LAVI > 33 mL/m²	1.449	4.26	< 0.001
EAT thickness > 10.5 mm	0.998	2.71	0.015
LCA stenosis > 50 %	1.014	2.76	0.04
CPB time (during CABG with CPB) > 60 min	1.48	4.39	0.019
Potassium < 3.6 mmol/L	-0.756	0.47	< 0.001
Volume of pericardial effusion > 88 mL	1.147	3.15	< 0.001

B, regression coefficient; OR, odds ratio; AF, atrial fibrillation; CHD, coronary heart disease; CABG, coronary artery bypass grafting; BMI, body mass index; LVMI, left ventricular mass index; LAVI, left atrial volume index; EAT, epicardial adipose tissue; LCA, left coronary artery; CPB, cardiopulmonary bypass.

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be triggered by heparin-protamine complexes utilized in CABG with CPB [10]. The results of our study demonstrate that a CPB duration exceeding 60 minutes during CABG is associated with an increased risk of postoperative AF ($p = 0.019$). Moreover, a correlation was identified between the risk of postoperative AF and the presence of left atrial lesions ($p = 0.040$). This finding can be associated with a more severe lesion of the coronary bed, a longer duration of CPB, and comorbidities in patients with postoperative AF compared to those with normal sinus rhythm after CABG. Nevertheless, the intergroup comparison revealed no statistically significant differences between the groups with and without postoperative AF, including the SYNTAX score and the majority of comorbidities (Table 1).

In the postoperative period, systemic inflammation is accompanied by local inflammation of the pericardial zone due to an increased volume of fluid in the pericardial cavity. This contributes to cardiomyocyte apoptosis and a change in the electrical activity of the myocardium [11]. In our study, the presence of a pericardial effusion exceeding 88 mL was identified as a risk factor for the development of AF during the early postoperative period ($p < 0.001$). Elahi et al. [12] demonstrated that open cardiac surgeries result in significant inflammatory responses, accompanied by the formation of a pro-oxidant environment within the pericardium. This environment is identified as a primary contributor to the development of AF. During CABG (in the context of pericardectomy), blood flows into the pericardial cavity. The presence of small quantities of residual blood can exert a biological effect on the pericardium, leading to oxidative stress and supraventricular arrhythmias [13].

Lancaster et al. [14] identified a correlation between the emergence of postoperative AF in patients with elevated potassium levels (4.30 and 4.21 mmol/L, respectively; $p < 0.001$) and magnesium levels (2.33 and 2.16 mg/dL, respectively; $p < 0.001$) compared to the control group. In particular, the authors observed that potassium supplementation administered prior to CABG did not result in a reduction in the incidence of postoperative AF (37% vs. 37%; $p = 0.813$). Conversely, magnesium supplementation was associated with an increased risk of postoperative AF (47% vs. 36%; $p = 0.005$). In a study by Howitt et al. [15], a mean serum potassium concentration of less than 4.5 mmol/L was identified as a risk factor for the development of postoperative AF (odds ratio (OR) 1.43; 95% confidence interval (CI) 1.17–1.75; $p < 0.001$). Conversely, the mean concentration of magnesium (1.0 mmol/L) was not found to influence the elevated risk of developing postoperative AF (OR 0.89; 95% CI 0.71–1.13; $p = 0.342$). In the present study, a potassium level of less than 3.6 mmol/L was identified as a factor associated with an increased risk of postoperative AF ($p < 0.001$).

It is important to acknowledge that EAT plays a role in the pathogenesis of paroxysmal AF. On the one hand, EAT has thermoregulatory and cardioprotective functions. On the other hand, it secretes a number of biologically active substances that trigger fibrotic processes in the atrial myocardium [16]. EAT, a depot of visceral fat situated between the myocardium and the visceral layer of the pericardium, is responsible for the production of a multitude of pro-inflammatory cytokines. These cytokines can exert an influence on the myocardium through paracrine or vasocrine mechanisms, thereby contributing to the development of AF [16, 17]. An increase in the EAT thickness is associated with a higher level of secreted inflammatory mediators and the trigger mechanism of AF [7, 17]. In a study by Petraglia et al. [18], an increase in EAT was observed to be associated with age. Our study revealed that age is a significant factor influencing the risk of postoperative AF, with a higher probability observed in individuals aged above 65 years ($p = 0.022$) The aging process is accompanied by degenerative and inflammatory changes in the atria, which are associated with a disruption of their electrophysiological properties Petraglia et al. [18] revealed that the mean thickness of EAT in patients who developed postoperative AF was greater than that observed in individuals with a preserved sinus rhythm following surgery. Furthermore, the investigators observed a significant elevation in IL-6 levels in EAT secretions among patients with postoperative AF. In our study, we found a correlation between the risk of developing postoperative AF and both body mass index (BMI; ≥ 30.5 kg/m^2; $p=0.020$) and the thickness of EAT (an elevated risk of postoperative AF in EAT > 10.5 mm; $p = 0.015$), which exceeded the threshold values in the population with postoperative AF (10 mm) [7]. The HUNT study [19], which included 93,860 residents of Norway, demonstrated a correlation between BMI variability and an increased risk of AF. Obese patients are nearly 50% more likely to develop AF than individuals of a healthy weight.

A substantial role in the incitation of postoperative AF is attributed to the LA [20], which plays a pivotal role in cardiac function. The mechanical function of the LA is comprised of three components:

1) Reservoir function during LV systole;
2) Passive conduit associated with the passage of blood from the pulmonary veins into the LV during early ventricular diastole;
3) Booster pump associated with active LA emptying during late ventricular diastole [21].

Any alterations to the LV inevitably impact the LA, ultimately leading to the impairment of its functions. The reservoir function of the LA is contingent upon the movement of the LV base during systole, which in turn determines the end-systolic volume. The conduit function is dependent

upon the compliance of the LA and is reciprocally associated with the reservoir function, as well as with the relaxation and compliance of the LV [21]. It should be noted that our study was limited in that we did not examine the mechanical function of LA in the development of postoperative AF. The present study has identified risk factors for the development of postoperative AF associated with an increase in LV mass (an increase in the LV myocardial mass index (LVMI) exceeding 115 g/m^2; p = 0.042) and an increase in the LA volume (LA volume index (LAVI) exceeding 33 mL/m^2; p < 0.001). Myocardial hypertrophy, as evidenced by an increase in LVMI and a concomitant rise in LV stiffness and impaired diastolic function, invariably compromises the functional capacity of the LA myocardium. Ozben et al. demonstrated that a LAVI of ≥ 36 mL/m^2 is linked to the onset of postoperative AF following CABG. The sensitivity and specificity of this association were 84.6% and 68.6%, respectively (p = 0.006) [22]. Rizvi et al. [23] observed a reduction in both LA global (p = 0.007) and regional (p = 0.01) longitudinal strain in patients with postoperative AF compared to those without postoperative AF. The findings demonstrated a statistically significant role of the reservoir function of the right atrium in the maintenance of sinus rhythm [23]. These findings suggest that not only is LA dysfunction a risk factor for the onset of postoperative AF, but also that structural and functional changes in the right atrium are equally important indicators of the risk of postoperative AF.

Limitations

Examination of the mechanical function of LA.

Conclusion

The risk of postoperative atrial fibrillation following coronary artery bypass grafting in patients with chronic coronary heart disease is elevated with advancing age, in obese patients, and in those with involvement of the left coronary artery. Furthermore, an increase in the thickness of epicardial adipose tissue, left ventricular myocardial hypertrophy and left atrial dilatation, electrolyte imbalance, postoperative effusion in the pericardial cavity, and prolonged use of cardiopulmonary bypass have also been identified as contributing factors.

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No conflicts of interest are reported.

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