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Evaluating the effect of coronary atherosclerosis on the occurrence of atrial fibrillation through coronary computed tomography angiography

Short title: Coronary atherosclerosis and atrial fibrillation

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WHAT'S NEW?

Little is known regarding the direct effects of atherosclerotic lesions in coronary vessels on the occurrence of atrial fibrillation. Data on the application of coronary computed tomography angiography for predicting atrial fibrillation remains scant. In this study, we evaluated whether patients with coronary atherosclerosis, confirmed using coronary computed tomography angiography, and significant widening of the interventricular septum in relation to the posterior wall had an increased risk of new-onset atrial fibrillation. These patients were found to be at a greater risk of sustained atrial arrhythmias, even without a history of acute myocardial ischemia, previous myocardial infarction, or revascularization procedures.

ABSTRACT

Background: The direct impact of atherosclerotic lesions in coronary vessels on the occurrence of atrial fibrillation (AF) in patients without a history of acute myocardial ischemia, previous myocardial infarction, or revascularization procedures remains largely unknown.

Aims: To assess the risk and predictors of new-onset AF in patients with coronary atherosclerosis confirmed by coronary computed tomography angiography (CCTA).

Methods: We included consecutive patients referred for CCTA who had been observed and diagnosed with new-onset AF over 10 years.

Results: Of the 549 patients enrolled in the study, 208 (37.9%) were diagnosed with atherosclerotic lesions in the coronary vessels and 63 (11.5%) developed AF during the 10 years of observation. Patients with AF were older (61.8 [10.4] years vs. 58.3 [9.2] years; P = 0.005), had an enlarged left atrium in the anteroposterior dimension (38.2 [7.2] mm vs. 34.4 [5.4] mm; P <0.001), and had a widened interventricular septum (12.3 [2.0] mm vs. 11.0 [2.1] mm; P <0.001). We also found a significant correlation between the occurrence of AF in patients with coronary atherosclerotic lesions and with increased thickness of the interventricular septum relative to the posterior wall of the left ventricle (P = 0.017).

Conclusions: Our data indicate an association between coronary atherosclerosis and the greater risk of AF in patients with increased thickness of the interventricular septum relative to the posterior wall of the left ventricle. This finding suggests that by using CCTA we can predict which patients are at higher risk of developing AF.

Key words: atrial fibrillation, chronic coronary syndrome, computed tomography of coronary arteries, coronary artery disease, coronary atherosclerosis

INTRODUCTION

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia with a steadily increasing prevalence and coronary artery disease (CAD) remains the first cause of death worldwide [1]. The number of patients with coronary atherosclerosis and CAD complicated by AF is increasing rapidly, and a strong correlation between these diseases has been reported. Furthermore, several shared risk factors, including hypertension, diabetes mellitus, sleep apnea, obesity, smoking, inflammation, and physical inactivity, may play prominent roles in their development. Despite these common risk factors, AF and coronary

atherosclerosis can independently develop because of long-term exposure to cardiovascular risk factors [2–8]. New-onset AF (NOAF) in CAD patients has mainly been studied in the setting of acute coronary syndrome (ACS), percutaneous coronary intervention, or coronary artery bypass grafting, It was consistently found to be an independent predictor of morbidity and mortality [9–13]. In contrast, data on NOAF in patients with coronary atherosclerosis without a history of acute myocardial ischemia, previous myocardial infarction (MI), or revascularization procedures are scarce, and whether they are the result of chronic ischemia is unknown. Only single studies found an association between subclinical atherosclerosis and incident AF [14, 15] not based on an assessment of changes in the coronary arteries.

The question arises as to whether it is reasonable to investigate AF in patients with atherosclerotic lesions in the coronary vessels and whether additional factors accompany atherosclerotic changes in the coronary vessels, predisposing to AF occurrence.

Thus, this study aimed to elucidate the relationship between coronary atherosclerosis and AF that potentially contributes to the interruption of their respective disease cycles, which is fundamental in treating patients effectively and optimizing their treatment plans for increased benefits [2, 7]. We also used coronary computed tomography angiography (CCTA) to measure the sizes of the left atrium (LA), interventricular septum, and posterior wall of the left ventricle to assess their significance in the onset of AF.

Having the ability to predict NOAF in these patients would greatly impact treatment planning, patient outcomes, and, finally, the cost to the healthcare system.

METHODS

This study included patients hospitalized in our cardiology clinic between January 1, 2009, and December 31, 2011, and diagnosed with atherosclerotic changes in the coronary vessels through CCTA (Figure 1). From the available medical records, patients without previously diagnosed AF, symptomatic heart failure, left ventricular ejection fraction <50%, significant valvular defects, congenital heart disease, cardiomyopathies, decompensated diabetes mellitus, severe impairment of the liver or kidney (encompassing acute and chronic diseases), or inflammation (autoimmune, connective tissue, and tumors) were selected. Additional exclusion criteria were as follows: ACS, history of MI, percutaneous coronary revascularization or surgical treatment of CAD. The follow-up duration was 10 years. Moreover, during the observation period, patients who had MI, hemodynamic intervention, heart surgery, occurrence of symptoms of heart failure, diagnosis of oncological disease, and inflammatory disease, or contact with whom either at the outpatient clinics of the National

Institute of Cardiology or by telephone was lost, were excluded. The primary outcome was the evaluation of the effect of coronary atherosclerosis on the occurrence of AF through computed tomography (CT) angiography from a group of 549 patients. The diagnosis of cardiac arrhythmia was based on available medical documentation, medical history, clinical history, and tests performed during hospitalization, consistent with the diagnoses at the National Institute of Cardiology of Warsaw. The extracted variables included demographic data, medical history, physical examination results, resting electrocardiograms, and routine transthoracic echocardiograms. The study protocol was approved by the local ethics committee and the patients provided written informed consent to participate in the study.

CCTA protocol

During the study period, two generations of dual-source CT scanners were used (Somatom Definition CT and Somatom Definition Flash CT, Siemens, Erlangen, Germany). Unless contraindicated, an intravenous or oral dose of metoprolol was administered to achieve a heart rate of <65 beats/min, and sublingual nitroglycerin was administered before CCTA. Contrast transit time was estimated by injecting a test bolus. To acquire the volume dataset, we injected 80–120 ml iodinated contrast material (Iomeron 400, Bracco Altana Pharma, Konstanz, Germany), followed by a mixture of 20% contrast agent and 80% saline. The scan parameters varied according to the scanner type and were as follows: beam collimation, 64 × 0.6 mm; tube voltage, 100 or 120 mV; gantry rotation time, 330 or 280 ms; and tube current, 330–438 mA/rotation or 320 mA/rotation. Dose-reduction strategies, including electrocardiogram (ECG)-gated tube current modulation and prospective axial triggering, were used to reduce the radiation dose whenever possible. Routine reconstructions of the scan data were performed in diastole (65–75% of the R–R interval) with a slice thickness of 0.6 mm and an increment of 0.4 mm.

CCTA analysis

In our study, we present CCTA as a diagnostic tool for the assessment of coronary atherosclerosis, indicated for patients with low to intermediate risk of CAD [16, 17]. Normal sex- and age-specific reference ranges for left ventricular (LV) mid-diastolic wall thickness (LV-MDWT) on prospective ECG-triggered mid-diastolic CCTA have been established. CCTA data were evaluated offline by a highly experienced reader on a dedicated workstation. The CCTA datasets were analyzed using multiplanar reconstructed images. The study assumed that patients diagnosed with coronary atherosclerosis had coronary lesions estimated

to be >30%. For each patient, a 3-chamber view parallel to the LV outflow tract and a 4-chamber view parallel to the interventricular septum were generated as previously described [18]. In the 3-chamber view, the anterior–posterior LA diameter was measured, whereas, in the 4-chamber view, the following measurements were obtained: diameter of the superior–inferior and thicknesses of the interventricular septum and the posterior wall of the left ventricle.

Statistical analysis

All results for nominal variables are reported as counts and percentages. A χ^2 independence test or Fisher's exact test was used to compare proportions. Numerical variables are presented as mean (standard deviation), and the significance of the differences between the means of the two groups was verified using an independent Student's t-test. Univariate and multivariable binary logistic regression with a backward selection of variables was used to identify independent factors for the occurrence of fibrillation events. Variables with univariate *P*-values <0.15 were entered into a multivariable analysis. Odds ratios (ORs) and prediction accuracies (c-statistics) were calculated using 95% confidence intervals. The comparison of the usefulness of explanatory variables in the model defining the occurrence of AF was presented using receiver operating characteristic curves and c-statistic. All hypotheses were 2-tailed, with a type I error of 0.05.

RESULTS

Of the 549 patients admitted to exclude or confirm and evaluate atherosclerotic plaques in coronary vessels, who were analyzed (Figure 1) 45 developed de novo AF during 5 years of observation, and 63 developed de novo AF during the 10 years of observation. These patients were older (61.8 [10.4] vs. 58.3 [9.2]; *P* = 0.005), had a larger anteroposterior dimension of the LA (38.2 [7.2] vs. 34.4 [5.4]; *P* <0.001), and had a thicker interventricular septum (12.3 [2.0] vs. 11.0 [2.1]; *P* <0.001). An interesting observation was the significant ratio of the dimensions of the interventricular septum to the posterior wall of the left ventricle (1.37 [0.21] vs. 1.20 [0.20]; *P* <0.001; Table 1, Figure 2).

Moreover, patients diagnosed with AF and coronary atherosclerosis were older (66.0 [9.8] vs. 61.4 [8.5]; *P* = 0.017) and had a larger anteroposterior dimension of the LA (39.3 [5.8] vs. 35.2 [5.7]; *P* = 0.002), a thicker interventricular septum (13.0 [1.7] vs. 11.4 [2.0]; *P* <0.001), and a higher ratio of the dimension of the interventricular septum to the posterior wall of the left ventricle (1.41 [0.21] vs. 1.21 [0.20]; *P* <0.001; Tables 2–3, Figure 3).

In the multivariable analysis, the ORs for AF in patients with coronary atherosclerosis, along with indexed ORs (95% confidence intervals) for female, a thicker interventricular septum, and the dimension of the interventricular septum to the posterior wall of the left ventricle were 3.90 (1.27–12.0; $P = 0.017$), 1.541 (1.12–2.12; $P = 0.008$), and 1.029 (1.005–1.054; $P = 0.017$), respectively.

DISCUSSION

Little is known regarding the direct effects of atherosclerotic lesions in coronary vessels on the occurrence of AF. Moreover, data on the application of CCTA for the prediction of AF remains scant. Previous studies have shown a correlation between the occurrence of AF in patients with ACS, after MI, or after coronary revascularization procedures. However, no data exist on the occurrence of NOAF in stable patients with atherosclerotic lesions in the coronary vessels and without any history of coronary procedures. The difficulty in proving and assessing the direct impact of coronary atherosclerosis on the occurrence of AF probably results from the overlapping risk factors for both conditions.

Atrial fibrillation occurs in approximately 2% of the general population and increases with age, from 0.14% in patients aged <50 years and 4% in those aged 60–70 years to 14% in those aged >80 years [2, 4]. Patients with AF have been reported to be more likely to have coronary artery lesions [7, 19]. The prevalence of CAD in patients with AF ranges from 17% to 46.5%. In contrast, AF prevalence among patients with CAD is low and is estimated to range from 0.2% to 5% [2, 20], reaching 28% in patients with acute MI (AMI) [21, 22]. In patients with advanced age, a longer history of CAD, and coexistent heart failure the prevalence is twice as high [23]. NOAF followed by AMI may occur in more than 50% of these patients [7, 19, 24]. However, disturbances in homeostasis accompanying MI are significantly different from those in patients with chronic coronary syndrome.

Coronary atherosclerosis may act as an independent risk factor for AF through direct mechanisms, such as LA ischemia, microcirculation disorders, endothelial dysfunction, progressive fibrosis, or thinning of the LA muscle. Additionally, indirect pathways may be implicated, for instance, an increase in LA pressure secondary to episodes of LV ischemia [25–27]. Ischemic damage to heart muscles can lead to heart failure, which also increases the risk of AF [28].

Atherosclerotic changes in the coronary vessels can be assumed to suddenly, as in ACS, or gradually reduce the blood supply to the sinus node and the myocardium of the atria and ventricles. Consequently, this reduction may lead to disturbances in the spread of

electrical impulses in the LA (electrical remodeling). The reduction may also contribute to fibrosis and scarring of the vestibule muscle (muscular remodeling), leading to the formation of areas with slow conduction. LA ischemia may also shorten the refractive period of the LA and develop gap junction uncoupling. Similar to the presence of areas with slow conduction, this could facilitate the establishment of micro-re-entry waves and an increase in spontaneous atrial ectopic activity contributing to AF occurrence [25, 29–32].

According to previous epidemiological studies, coronary atherosclerosis and AF mutually promote the occurrence and progression of each other, forming a vicious cycle. AF can induce atherosclerosis, creating a mismatch between blood supply and oxygen consumption, and thrombosis. This promotes or exacerbates coronary heart disease with endothelial dysfunction that may be associated with the development of AF, which in turn may lead to systemic inflammation and affect plaque stability [33]. Thus, coronary heart disease can be both a cause and a consequence of AF [7]. It has been proven that, in patients diagnosed with AF and stable CAD or AMI, the location of the atherosclerotic lesion in the coronary vessel or the effect of the number of atherosclerotic coronary lesions is irrelevant to the occurrence of arrhythmia [34]. Moreover, revascularization of the coronary arteries proximal to the arteries supplying blood to the atrial myocardium was not associated with lower AF rates [29].

In our study, we also focused on the thickening of the LV wall which may be associated with a lack of or inadequate treatment of hypertension and in patients with coronary atherosclerosis, it can significantly increase the risk of AF. A history of hypertension increases the risk of AF by 34% [16]. Thickening of the subaortic segment of the interventricular septum occurs in approximately 6%–10% of patients. Recent studies indicate that its presence may be an early marker of hypertensive remodeling [35]. In addition to these structural changes, inflammation and the renin-angiotensin-aldosterone system are involved in the reentry mechanism of arrhythmia [17, 36]. We found that benchmarks, such as the dimension of the interventricular septum to the posterior wall of the left ventricle, can expand the diagnostic and prognostic roles of CT angiography in searching for NOAF, extending beyond its role in identifying coronary atherosclerosis.

To the best of our knowledge, this is the first study to demonstrate the relationship between the presence of atherosclerotic lesions in the coronary arteries, as assessed using CT, and the occurrence of AF episodes in patients without a history of AMI, previous MI, or revascularization procedures. Previous studies assessing these dependencies were often guided only by clinical history indicating coronary atherosclerosis, ECG records indicating

myocardial ischemia, or exercise tests. However, none of the studies used coronary artery CT scans to assess the staging of coronary vessels.

The primary prevention strategy for AF aims to address and mitigate risk factors. These include lifestyle changes, such as increased physical activity, avoidance of smoking, and control of body mass index, blood pressure, total cholesterol, and blood glucose, as well as comorbidity treatments that can reduce the risk of AF. These measures also have a similar effect on preventing coronary atherosclerosis [7, 37, 38]. Thus, the prevention of AF is a relevant secondary objective for patients with chronic coronary syndrome, which has received little attention in the clinical literature to date [8, 39]. This is particularly important given the higher incidence of cardiovascular events, including cardiac death and stroke, in patients with AF and CAD than in those without CAD [9, 40–42], as recently confirmed in the CLARIFY registry [1].

For coronary atherosclerosis, the key treatments include lipid-regulating therapy and plaque stabilization to prevent plaque progression and lesion aggravation. In patients with AF, statin use can reduce the incidence of CAD by 2.7% per 30% reduction in low-density lipoprotein cholesterol [7, 43]. Therefore, appropriate lifestyle changes and comorbidity treatment could prevent the initiation of the cycle and weaken the connection between the two diseases, making primary prevention of CAD with AF the most powerful tool to break the vicious cycle [7]. Moreover, patients with confirmed changes in the coronary vessels, assessed using CCTA, even if seemingly insignificant, may require early, more intensive and aggressive pharmacological treatment, particularly in cases of LV septal dilation. Whether more intensive preventive measures and more systematic screening for AF would improve prognosis in this population deserves further investigation.

Limitations

Prospective ECG-triggered CCTA restricts the evaluation of the entire cardiac cycle, precluding traditional assessments of LV end-diastolic volume, LV end-diastolic wall thickness, and LV ejection fraction commonly performed with echocardiography and cardiac magnetic resonance imaging [44]. However, studies comparing LV wall thickness using CT and cardiac magnetic resonance imaging have demonstrated a stronger association between the two imaging modalities than with echocardiography [36, 45, 46]. This correlation holds clinical significance, especially given the widespread availability of cardiac CT. Despite the limitations inherent to prospective ECG-gated mid-diastolic acquisition in measuring these parameters, the assessment of LV-MDWT is feasible. It shows a strong correlation with the

end-diastolic and end-systolic phases, enabling the identification of an abnormally thick LV wall (Figure 4). However, no established upper limit for normal LV-MDWT using a large patient population exists currently, although post-processing software readily enables LV wall thickness measurements [47–49].

Another limitation was the lack of use of long-term monitoring equipment to detect AF. The inclusion of extended ECG monitoring or utilization of implantable/non-implantable continuous loop recorders for arrhythmic event monitoring could have enhanced our analyses.

CONCLUSIONS

Our data indicate an association between coronary atherosclerosis and the increased risk of NOAF in patients with increased thickness of the interventricular septum relative to the posterior wall of the left ventricle. We determined that these patients were at a greater risk of AF even without a history of acute myocardial ischemia, previous MI, or revascularization procedures, as indicated by the literature. This finding suggests that by using CCTA we can predict which patients are at higher risk of developing AF. Lifestyle modifications and intensification of pharmacological therapy for coronary atherosclerosis may contribute to the primary prevention of AF. Measures for preventing further dilation of the ventricular septum in these patients are necessary. Whether more aggressive pharmacological treatment of patients with coronary atherosclerosis and LV septal dilation would improve prognosis in this population deserves further investigation.

The ability to predict NOAF in patients with coronary atherosclerosis would significantly impact treatment planning, patient outcomes, and, ultimately, the cost to the healthcare system.

Article information

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Table 1. Comparison of characteristics between patients with and without AF during the 10-year follow-up

	Total n = 549 (100%)	AF+ n = 63 (11.5%)	AF– n = 486 (88.5%)	*P*-value

Age, years, mean (SD)	58.7 (9.4)	61.8 (10.4)	58.3 (9.2)	0.005
Women, n (%)	358 (65.2)	42 (66.7)	316 (65.0)	0.81
CA, n (%)	208 (37.9)	23 (36.5)	185 (38.1)	0.81
HT, n (%)	430 (78.3)	55 (87.3)	375 (77.2)	0.07
DM, n (%)	123 (22.5)	19 (30.2)	106 (21.8)	0.14
Dyslipidemia, n (%)	404 (73.9)	47 (74.6)	357 (73.8)	0.88
Echocardiography				
LA, mean (SD)	37.9 (6.1)	40.9 (4.7)	37.1 (6.2)	0.001
LAA, mean (SD)	21.0 (4.6)	23.7 (6.1)	20.3 (3.8)	0.02
IVS, mean (SD)	11.4 (1.8)	11.5 (1.3)	11.4 (1.9)	0.77
CCTA				
LA AP, mm, mean (SD)	34.9 (5.8)	38.2 (7.2)	34.4 (5.4)	<0.001
LA SI, mm, mean (SD)	52.2 (5.8)	53.4 (6.5)	52.1 (5.7)	0.09
IVS, mm, mean (SD)	11.2 (2.1)	12.3 (2.0)	11.0 (2.1)	<0.001
PW, mm, mean (SD)	9.3 (1.6)	9.1 (1.8)	9.3 (1.6)	0.49
IVS/PW mean (SD)	1.22 (0.21)	1.37 (0.21)	1.20 (0.20)	<0.001

Abbreviations: AF, atrial fibrillation; CA, coronary atherosclerosis; CCTA, coronary computed tomography angiography; DM, diabetes mellitus; HT, hypertension; IVS/PW, dimension of the interventricular septum to the posterior wall of the left ventricle; LA AP, left atrial anteroposterior diameter from 3-chamber view; LA SI, left atrial superior-inferior diameter from 4-chamber view; PW, posterior wall of the left ventricle; SD, standard deviation

Table 2. Multivariable analysis of the occurrence of AF in patients with coronary atherosclerosis

	Univariable analysis		Multivariable analysis	
	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value
Age (per 1-year increase)	1.068 (1.011–1.128)	0.02	–	
Women	2.31 (0.870–6.111)	**0.08**	3.90 (1.27–12.0)	0.02
HT	0.919	0.89	–	

	(0.292–2.895)			
DM	1.736 (0.707–4.261)	0.23	–	
Dyslipidemia	0.530 (0.193–1.458)	0.22	–	
CCTA				
LA AP	1.121 (1.041–1.207)	0.002	–	
LA SI	0.999 (0.936–1.066)	0.98		
PW	0.948 (0.729–1.232)	0.69	–	
IVS/PW	1.045 (1.023–1.068)	<0.001	1.029 (1.005–1.054)	0.02
AUC (95% CI)			0.815 (0.734–0.897)	

Abbreviations: AUC, area under the curve; CI, confidence interval; OR, oddas ratio; other — see Table 1

Table 3. Multivariable analysis of atrial fibrillation occurrence in patients without coronary atherosclerosis

	Univariable analysis		Multivariable analysis	
	OR (95% CI)	*P*-value	OR [95% CI]	*P*-value
Age (per 1-year increase)	1.037 (0.999–1.076)	0.06	–	
Women	0.677 (0.341–1.346)	0.27	–	
HT	3.314 (1.144–9.602)	0.03	–	
DM	1.458 (0.674–3.157)	0.34	–	
Dyslipidemia	1.455	0.33	–	

	(0.684–3.096)			
CCTA				
LA AP	1.109 (1.049–1.174)	<0.001	1.080 (1.013–1.151)	0.02
LA SI	1.084 (1.013–1.159)	0.019	1.075 (1.003–1.152)	0.04
IVS	1.293 (1.104–1.515)	0.001	1.424 (1.169–1.735)	<0.001
PW	0.943 (0.763–1.166)	0.53	0.678 (0.515–0.893)	0.006
IVS/PW	1.032 (1.017–1.048)	<0.001		
AUC (95% CI)			0.738 (0.652–0.824)	

Abbreviations: see Tables 1 and 2

Table 4. Comparison of characteristics between patients with and without coronary atherosclerosis

	CA+ n = 208 (37.9%)	CA– n= 341 (62.1%)	*P*-value
Age, years, mean (SD)	61.9 (8.8)	56.7 (9.3)	<0.001
Women, n (%)	119 (57.2)	239 (70.1)	0.002
HT, n (%)	174 (83.6)	256 (75.1)	0.02
DM, n (%)	59 (28.4)	66 (19.3)	0.02
Dyslipidemia, n (%)	172 (83.1)	232 (68.2)	<0.001
CCTA			
LA AP, mm, mean (SD)	35.7 (5.9)	34.4 (5.7)	0.012
LA SI, mm, mean (SD)	52.1 (5.3)	52.4 (5.3)	0.59
IVS, mm, mean (SD)	11.6 (2.0)	10.9 (2.1)	<0.001
LVPW, mean (SD)	9.5 (1.7)	9.1 (1.6)	0.006
IVS/LVPW, mean (SD)	1.23 (0.21)	1.21 (0.21)	0.17

Abbreviations: LVPW, left ventricular posterior wall; other — see Table 1

Table 5. Comparison of characteristics between patients with and without AF during the 10-year follow-up depending on coronary atherosclerosis

	CA+			CA–		
	AF+ n = 23 (11.1%)	AF– n = 185 (88.9%)	*P*-value	AF+ n = 40 (11.7%)	AF– n = 301 (88.3%)	*P*-value
Age, years, mean (SD)	66.0 (9.8)	61.4 (8.5)	0.02	59.3 (10.1)	56.4 (9.1)	0.06
Women, n (%)	17 (73.9)	102 (55.1)	**0.09**	25 (62.5)	214 (71.1)	0.26
HT, n (%)	19 (82.6)	155 (83.8)	1.00	36 (90.0)	220 (73.1)	0.02
DM, n (%)	9 (39.1)	50 (27.0)	0.22	10 (25.0)	56 (18.6)	0.34
Dyslipidemia, n (%)	17 (73.9)	155 (84.2)	0.24	30 (75.0)	202 (67.3)	0.33
CCTA						
LA AP, mm, mean (SD)	39.3 (5.8)	35.2 (5.7)	0.002	37.5 (7.9)	34.0 (5.2)	0.008
LA SI, mm, mean (SD)	52.0 (6.3)	52.1 (6.7)	0.98	54.2 (6.6)	52.1 (5.0)	0.02
IVS, mm, mean (SD)	13.0 (1.7)	11.4 (2.0)	<0.001	11.9 (2.1)	10.8 (2.1)	0.001
PW, mean (SD)	9.4 (2.1)	9.5 (1.7)	0.69	9.0 (1.7)	9.1 (1.6)	0.59
IVS/LVPW, mean (SD)	1.41 (0.21)	1.21 (0.20)	<0.001	1.34 (0.20)	1.19 (0.20)	<0.001

Abbreviations: see Tables 1 and 4

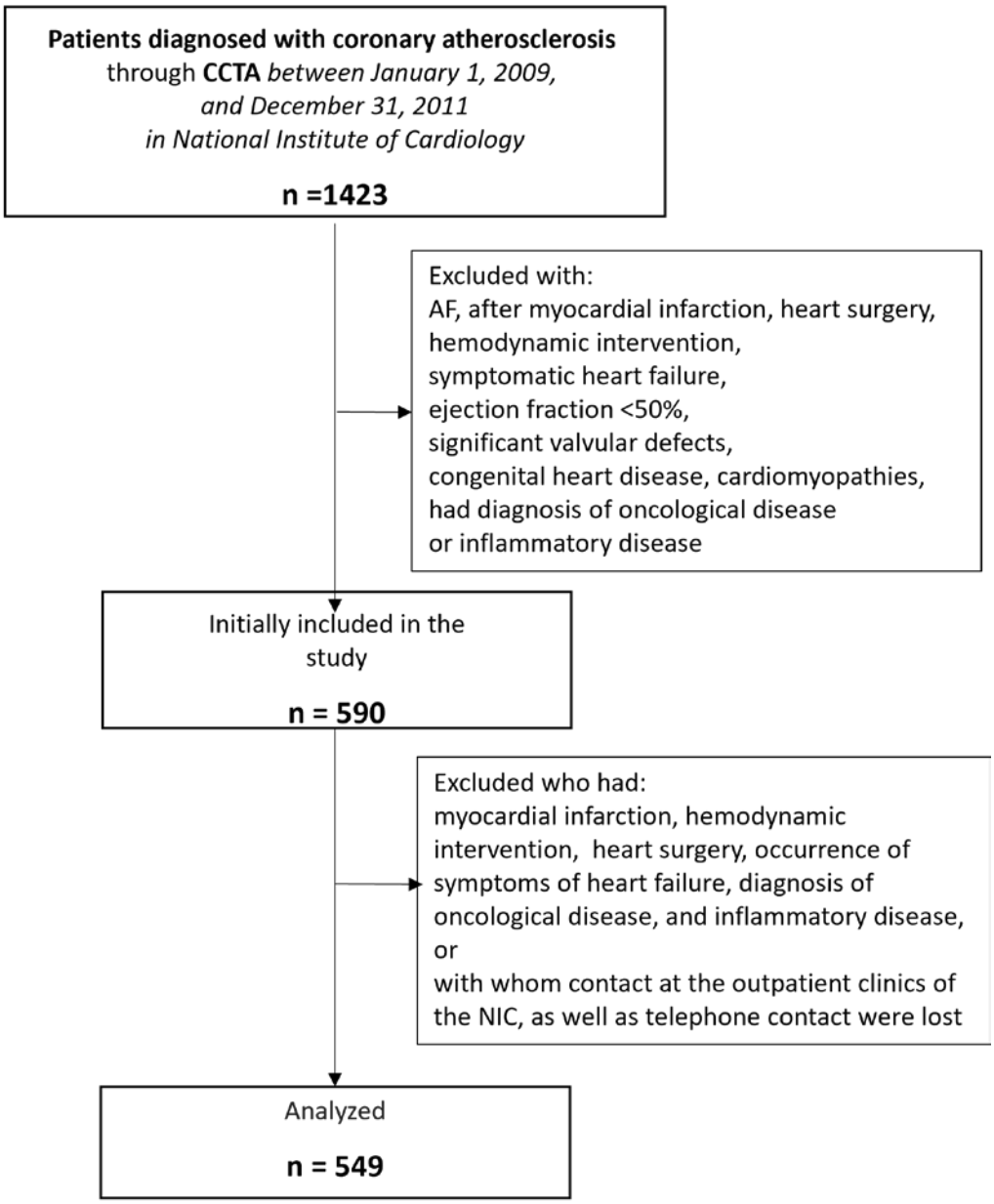

Figure 1. Flow chart of patient selection

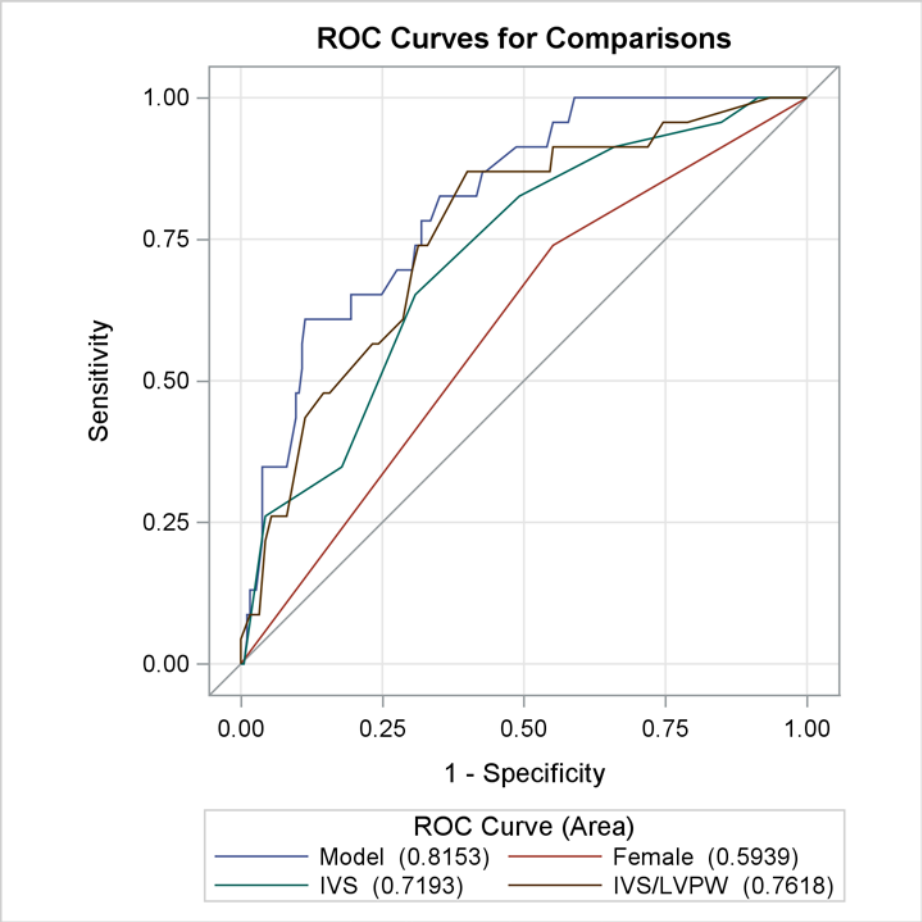

Figure 2. An impact of independent factors on the prediction of atrial fibrillation in patients with coronary atherosclerosis

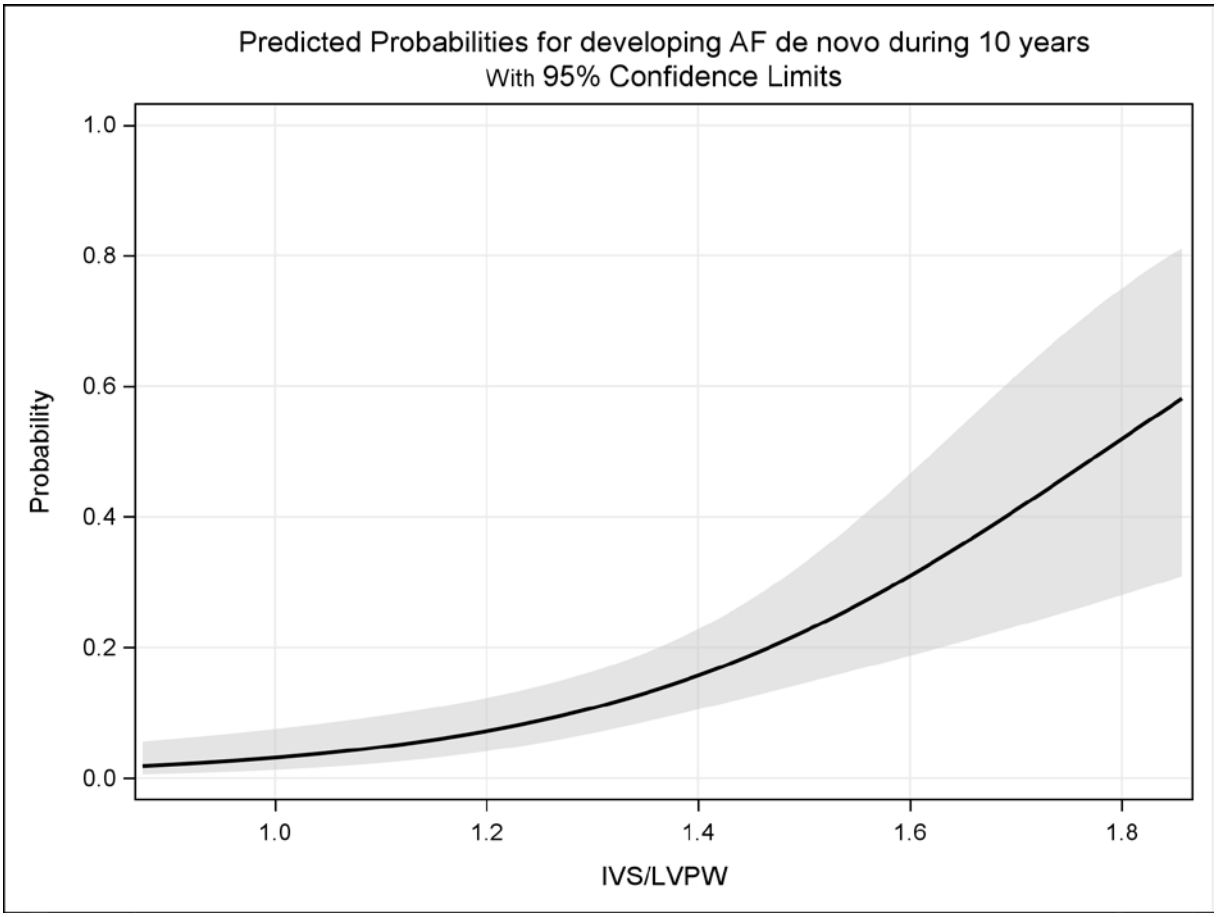

Figure 3. Predicted probabilities for developing atrial fibrillation *de novo* during 10 years in patients with coronary atherosclerosis

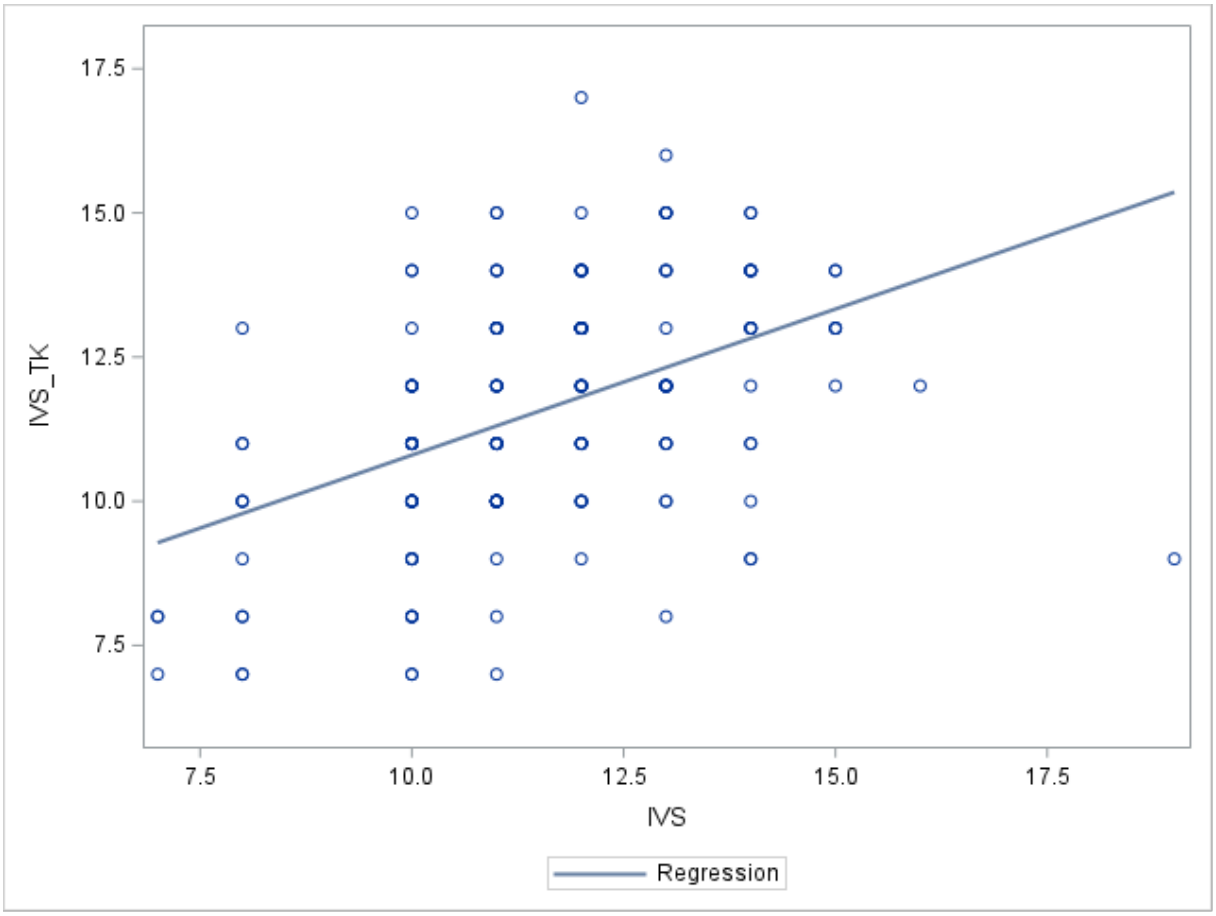

Figure 4. Pearson correlation coefficient IVS /IVS CT (IVS_TK): r = 0.916; *P* <0.001