

THE VALUE OF ATRIAL ELECTROMECHANICAL DELAY IN PREDICTING ATRIAL FIBRILLATION DEVELOPMENT AFTER CORONARY ARTERY BYPASS SURGERY

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Abstract

Objective: Predicting postoperative atrial fibrillation (PoAF) in the preoperative period will provide a serious advantage in preventing the morbidity and mortality associated with this arrhythmia and in planning the treatment. In this study, we investigated the value of atrial electromechanical delay (AEMD) in predicting the development of PoAF. Methods: A total of 93 patients who underwent isolated coronary artery bypass grafting (CABG) operation were included in this prospective study. Patients' demographic characteristics, laboratory parameters, echocardiographic data, and AEMD durations that could be measured by the co-use of electrocardiography and echocardiography were recorded. The patients at sinus rhythm during the postoperative period were identified as "Group 1", and those who developed PoAF were identified as "Group 2". Results: PoAF incidence was 26.88% (n=25). Left ventricle (LV) lateral AEMD, LV medial AEMD, right ventricle lateral AEMD, and left atrium (LA) lateral AEMD durations of Group 2 were significantly higher than Group 1 ($p<0.001$, $p=0.004$, $p=0.004$, $p<0.001$; respectively). In Univariate Logistic Regression Analysis, the age, hypertension, LA maximum volume, LA lateral AEMD and pulmonary artery pressure were significantly associated with PoAF development ($p=0.01$, $p=0.004$, $p=0.004$, $p=0.001$, $p=0.01$; respectively). However, only LA lateral AEMD was found as an independent predictive factor for the development of PoAF in the Multivariate Logistic Regression Analysis (OR:1.03, 95% CI:1.001-1.06, $p=0.04$). AUC was 0.741 for LA lateral AEMD in ROC Curve Analysis (95% CI:0.633-0.849, $p<0.001$). Conclusions: The development of PoAF can be predicted by AEMD durations measured in the preoperative period in patients undergoing isolated CABG.

Introduction

The most common arrhythmia following cardiac surgery is atrial fibrillation (AF), and its incidence is 20-40%. Despite advances in surgical techniques and myocardial protection methods, and innovations in anesthesia, postoperative atrial fibrillation (PoAF) is still common after coronary artery bypass grafting (CABG) especially due to the advanced age of the patient population (1). It is known that predicting PoAF development prior to operation provides a significant reduction in morbidity, mortality and hospitalization times (2,3). The pathophysiology of PoAF is multifactorial. Although it is considered that it develops due to advanced age, electrolyte imbalance, hypertension, atrial fibrosis, atrial adhesions, intraoperative ischemia, increase in postoperative sympathetic activity, and volume loading, its pathophysiology has not yet been explained fully. For this reason, there is no proven prophylactic treatment strategy (4,5).

Atrial electromechanical delay (AEMD) defines the time interval between the initial point of P wave in 12-lead electrocardiography (ECG) and the formation of the late diastolic wave in Tissue Doppler Imaging (TDI). This delay between the electrical stimulation and mechanical contraction, in other words, atrial electrophysiological changes, cause AF development by affecting the anatomy of atrium (6). Our aim in

this study was to investigate the predictive value of AEMD duration, which can be easily detected by pre-operatively routinely performed ECG and transthoracic echocardiography (TTE) (using TDI), in the development of PoAF in isolated CABG patients.

Material and Methods

Patients and study design

The data of 93 patients who underwent cardiopulmonary bypass (CPB) with isolated CABG between October 2016 and May 2017 were examined in this prospective study. Our study was approved by the Ethics Board of Clinical Research of University of Health Sciences Bursa Yuksek Ihtisas Training and Research Hospital; and the written consents of the patients who were included in the study were obtained.

The patients between the ages of 18 and 80, scheduled for isolated CABG and who had sinus rhythm were included in the study. Those with valvular heart disease requiring surgical intervention, those who had previously undergone open heart surgery, those with severe low left ventricular ejection fraction (EF) (EF <35%), those with severe cardiac segmental wall activity abnormalities, those with severely dilated left atrium (LA > 50 mm), those with a history of ablation due to AF, those with preoperative\out, bradyarrhythmia or tachyarrhythmia were not included in the study.

In total, 120 patients were evaluated in this study, and 93 patients met the study criteria. Seven patients with poor image quality, 4 patients with arrhythmia before the procedure, 5 patients with severe valve dysfunction, 7 patients who were given antiarrhythmic during or after the procedure, were excluded from the study.

The demographic characteristics, comorbidities, preoperative and postoperative laboratory parameters, preoperative TTE parameters, preoperative AEMD values, perioperative and postoperative variables of the patients were recorded.

Atrial Electromechanical Delay Measurement

TTE (EPIQ 7 Cardiology Ultrasound - Philips Ultrasound, Netherlands, using 3.5 MHz probe) was performed in the left lateral decubitus position by the same cardiologist the day before the surgical procedure. Standard measurements were made from parasternal long and short axis, and 2 and 4 chamber apical views. The measurements were made by considering the American Society of Echocardiography (ASE) Guideline (7).

The EF was calculated according to the modified biplane Simpson's method (7). Mitral flow velocities were recorded from four chamber apical views with pulsed wave Doppler (PWD) with a 5-mm sample volume placed at the mitral valve tip levels. Peak early diastolic mitral velocity (E), and late diastolic mitral velocity (A) were recorded.

TDI is an echocardiographic technique that measures the velocities of cardiac structures. TDI records were made in four chamber apical views. The gain settings were minimized and TDI filter and Nyquist limits were set as 16-20 cm/s. Systolic mitral annular velocity (Sa), early diastolic mitral annular velocity (Ea) and late diastolic mitral annular velocity (Aa) values were recorded from lateral and medial mitral annulus and lateral tricuspid annulus. (Figure 1) Atrial late diastolic tissue Doppler velocity (Aaa) were recorded from left atrial (LA) and right atrial (RA) lateral wall and interatrial septum. (Figure 1). The time interval between the beginning of the P wave in the surface ECG and the beginning of Aa/Aaa record detected with TDI was called as 'AEMD'. Ventricular annular measurements were specified as lateral mitral annulus (left ventricle (LV) lateral AEMD), medial mitral annulus (LV medial AEMD) and lateral tricuspid annulus (right ventricle (RV) lateral AEMD). Atrial wall measurements were specified as lateral LA wall (LA lateral AEMD), inter-atrial septum (LA medial AEMD), and lateral right atrium (RA) wall (RA lateral AEMD).

Surgical Technique

IV midazolam 0.1 mg/kg, fentanyl 5 pg/kg, pancuronium bromide 0.1 mg/kg were used for anesthesia induction. When needed, 5 pg/kg fentanyl, 0.05 mg/kg pancuronium, or vecuronium bromide were used

to maintain anesthesia. All patients underwent median sternotomy. The left internal thoracic artery and saphenous vein were used as graft. Heparinization ACT (Activated Clotting Time) was provided to over than 400 seconds. The CPB was performed by arterial cannulation through the ascending aorta and venous cannulation with two-stage cannula through the right atrium. Myocardial protection was provided with antegrade blood cardioplegia. Moderate hypothermia (28°C-32°C) was preferred. Roller pump and membrane oxygenator were used in all cases. The CPB was adjusted to keep pump flow to 2.2-2.4 lt/min/m², mean arterial pressure 50-60 mmHg, and hematocrit 20-25% during the aortic cross-clamp. After cross-clamping, CPB was discontinued after body temperature reached 36 ° C, cardiovascular stability was achieved and sufficient blood pressure was established.

Postoperative Follow-ups

The patients were taken to the cardiovascular surgery intensive care unit after the surgeries and were followed-up there. During the intensive care process, the patients were followed-up with standard procedures as arterial pressure, blood gas, and D-II derivation continuous ECG. The electrolyte levels were closely monitored and corrected in the postoperative period. The 12-derivation ECG monitoring was performed continuously for patients who had arrhythmia or who had arrhythmia sensitivity, and arrhythmias were analyzed with 12-derivation ECG taken during arrhythmia. ECG telemetry was used in the follow-ups in the wards. The patients with AF episode for more than 5 minutes were accepted as PoAF (8).

Statistical Analysis

Our data were analyzed by using the SPSS 23.0 Statistics Package (SPSS Inc, Chicago, Ill, USA). The variables were expressed as percentage, numeric variables, and mean \pm standard deviation. The Student t-test was used to compare normally distributed variables, and the Mann-Whitney U-test was used to compare the variables that did not show normal distribution. The Chi-Square test or Fisher's Exact Chi-Square test were used to compare categorical variables. The risk factors that affected the PoAF development were identified with univariable and multivariate logistics regression analysis. The sensitivity and specificity of AEMD in showing PoAF development was evaluated with the Receiver Operating Characteristic (ROC) Analysis. Intra-observer agreement was assessed 20 randomly selected patients by analyzing the echocardiographic data measured (AEMD parameters, two times in different time) by the same operator, using Pearson's correlation coefficients. The statistical significance level was taken as $p < 0.05$ in all assessments. 10 randomly

Results

A total of 68 patients followed with sinus rhythm after CABG were defined as "Group 1", and a total of 25 patients developing PoAF (patients who developed AF episode lasting at least 5 minutes during the postoperative period) were defined as "Group 2".

The PoAF development rate was 26.88% ($n = 25$) in 93 patients who were included in our study, and the average AF development time was 2.80 ± 1.77 days. The demographic and clinical characteristics of the patients are presented in Table 1. The mean age, hypertension rate and diuretic use rate of patients in Group 2 were significantly higher than those in Group 1 ($p = 0.014$, $p = 0.03$, $p = 0.01$; respectively).

The preoperative laboratory data of the patients are presented in Table 1. No statistically significant differences were detected between the groups in terms of hemogram parameters, renal function tests, electrolytes, and lipid profiles. When the preoperative TTE measurements of the patients were evaluated, it was detected that the mean LA maximum volume and pulmonary artery pressure (PAP) were significantly higher in Group 2 ($p = 0.002$, $p = 0.012$; respectively); the mean LV lateral Ea velocities and LV medial Ea velocities were significantly lower ($p = 0.006$, $p = 0.02$; respectively) (Table 2).

The only intraaortic balloon pump support need, which is among the assessed perioperative variables, was significantly more in Group 2 ($p = 0.02$). No significant differences were detected between the groups in terms of the CABG times, CPB times, cross-clamp times, and the number of grafts used, which indicated that similar surgical procedures were performed. The average length of stay in the intensive care unit, which is among the postoperative variables, was significantly higher in Group 2 ($p = 0.009$). Also, no significant

differences were detected between the groups in terms of the postoperative laboratory parameters that were evaluated (Table 3).

The average AEMD durations of the patient groups are presented in Table 2. The LV lateral AEMD, LV medial AEMD, RV lateral AEMD and LA lateral AEMD durations were significantly longer in Group 2 than in Group 1 ($p < 0.001$, $p = 0.004$, $p = 0.004$, $p < 0.001$; respectively).

The variables that were associated with the development of PoAF were included in the Univariate Logistic Regression Analysis. Age, hypertension, LA maximum volume, LA lateral AEMD and PAP were found to be significantly associated with PoAF development ($p = 0.01$, $p = 0.004$, $p = 0.004$, $p = 0.001$, $p = 0.01$; respectively). These variables, which were found to be statistically significant, were included in the Multivariate Logistic Regression Analysis. The LA lateral AEMD variable was found to be an independent predictive factor for PoAF development (OR: 1.03, 95% CI: 1.001-1.06, $p = 0.04$) (Table 4).

ROC Curve Analysis was conducted to determine the predictive effect of AEMD in PoAF development. For LV lateral AEMD, the Area Under the Curve (AUC) was 0.764 (95% CI: 0.652-0.876, $p < 0.001$). For LV medial AEMD, AUC was 0.686 (95% CI: 0.567-0.806, $p = 0.006$). For RV lateral AEMD, AUC was 0.706 (95% CI: 0.588-0.823, $p = 0.002$). For LA lateral AEMD, AUC was 0.741 (95% CI: 0.633-0.849, $p < 0.001$) (Figure 2).

Intra-observer agreement was higher than 0.90, $p < 0.001$, for all AEMD parameters.

Discussion

In our study, the value of AEMD durations measured with the co-use of preoperative ECG and TTE (using TDI) in predicting PoAF was investigated in isolated CABG patients. It was found that the AEMD durations were statistically significantly longer in our group with PoAF. Although the difference between the groups in the LA antero-posterior diameters was not detected in TTE measurements, LA maximum volume was significantly higher in the group with PoAF. LV lateral Ea and LV medial Ea velocities were found to be lower in the group with PoAF, and this was an indication that diastolic dysfunction was more common in the group with PoAF. Also, LA lateral AEMD was found as an independent predictive factor in the multivariate logistic regression analysis we conducted to determine predictors for PoAF development.

PoAF is a cardiac arrhythmia that increases morbidity and mortality by affecting ventricular functions, causing hemodynamic impairment and increasing the risk of thromboembolic events. Its incidence after cardiac surgery is 20-40% (1). The PoAF incidence was 26.88% in our study, which included isolated CABG patients. Several studies were conducted to identify the risk factors of PoAF development. Advanced age, high body surface area, white ethnicity, hypertension, LV hypertrophy, use of preoperative digoxin, obstructive pulmonary disease, presence of postoperative LV diastolic and systolic dysfunction, preoperative high LA volume measurement, presence of postoperative low atrial filling fraction, and increased adrenergic drug use after surgery were associated with PoAF development (9-16). Also, the use of P wave duration with signal-averaged ECG to predict PoAF development was evaluated, and it was found that patients developing PoAF had preoperative prolonged P wave durations (17). In fact, when all these risk factors are evaluated, all of these parameters appear before us as the cause or result of atrial remodeling. Atrial remodeling triggers the development of PoAF by causing the development of multiple reentrant waves that initiate and possibly maintain AF (18).

The main finding of our study was that the AEMD durations measured by ECG and TTE (using TDI), which is a noninvasive method, was significantly longer in the group with PoAF. It is recently acknowledged that AEMD duration is a non-invasive evaluation method of atrial conduction time, and AEMD duration measurement is an easy, fast and reliable method. AEMD is defined as the delay between the detected electrical activity onset (P wave onset in superficial ECG) and the contraction in atrial myocardium ("late diastolic wave = Aa wave" evaluated with TDI). It was emphasized in many studies that the prolongation of AEMD durations was the independent marker of new-onset or recurrent AF (19-21). Determining the P wave duration in signal-averaged ECG is also useful for predicting the atrial electromechanic conduction

times, but its use in clinical practice is limited because it requires special equipment, and evaluation of its results is time-consuming. However, AEMD, which is a new echocardiographic parameter, was confirmed to be correlated with P wave durations detected by using signal-averaged ECG (17,20).

In a study conducted by Özlü et al., it was reported that LA maximum volume and AEMD duration were the independent predictors of PoAF development after CABG surgery (23). Although their results support the results of our study, unlike our study, only inter-atrial electromechanical delay (the difference between LV lateral AEMD and RV lateral AEMD) was evaluated in this study. In another study conducted by Fujiwara et al., 88 off-pump CABG patients were included, AEMD durations were measured with preoperative TTE (using TDI), and in addition, left atrial volume index (LAVI) was calculated (24). Patients were monitored with ECG telemetry for 7 days postoperatively. As a result, they concluded that PoAF was detected in 35 patients (39.8%), and the AEMD duration (OR: 1.11, 95% CI: 1.06-1.16, $p = 0.0001$) and LAVI (OR: 1.11, 95% CI: 1.02-1.20, $p = 0.01$) were independent predictors of PoAF development with multivariate logistic regression analysis. They also conducted ROC Curve Analysis, and reported that AUC: 0.85 for AEMD (sensitivity: 74.3% and specificity: 86.8%) (23). Although a different CABG technique was applied in this study compared to our study, our results were similar. In our study, we also found the LA lateral AEMD was an independent predictive factor for PoAF development (OR: 1.03, 95% CI: 1.001-1.06, $p=0.04$); and AUC: 0.741 for LA lateral AEMD in ROC Curve analysis (95% CI: 0.633-0.849, $p < 0.001$). LA lateral AEMD duration is significantly correlated with LA diameter and LA maximum volume (25). These data may explain the importance of AEMD durations, which is an indicator of pathological changes in the atrium, for predicting PoAF. The LV lateral AEMD parameters were not included in the univariate and multivariate analysis because EMD parameters were similar in atrial and ventricular site.

There are limited studies emphasizing the importance of AEMD in predicting the development of PoAF in other cardiac operations such as aortic valve and mitral valve surgeries (26,27). Takahashi et al. monitored patients who underwent aortic valve replacement with ECG telemetry for 7 days, and reported that the PoAF incidence was as high as 65% (41 of 63 patients). They measured the AEMD duration with preoperative TTE (using TDI), and concluded that it was an independent predictor for the development of POAF (OR: 1.07, 95% CI: 1.02-1.13; $p = 0.0072$) (24). In the same center, PoAF development was investigated in patients with mitral valve replacement due to mitral stenosis, and as a result, LA lateral AEMD duration was found to be an independent predictor for the development of POAF (OR, 1.04; 95% CI: 1.01-1.07; $p = 0.0048$). Also, in ROC Curve Analysis, the AEMD duration cut-off value for the development of POAF was reported as 159.4 ms (27). Unlike our study, in these studies, the AEMD time was taken as the time between the P wave start at ECG and the mitral lateral Aa peak point measured with TDI; however, in our study, the time between the onset of the mitral lateral Aa was measured.

In the present study, it was concluded that advanced age, hypertension, LA maximum volume height and the increase in LA lateral AEMD, which is a marker of diastolic heart failure, are risk factors for the development of PoAF. The presence of LV diastolic dysfunction in predicting PoAF development comes to the forefront in many studies unlike LV systolic dysfunction (16, 19). In our study, we believe that the significantly lower LV lateral Ea and LV medial Ea velocities, among the parameters indicating LV diastolic function in the group with PoAF, is the probable cause of high PAP and diuretic usage rates in the group with PoAF. In their prospective study that included 275 elective CABG patients, Gibson et al. reported that LV systolic function was not an independent predictive factor for PoAF (19). Similarly, in our study, EF values were similar in groups with and without PoAF.

Our study had some limitations. One of the limitations of the study was that it had a single-centered, and non-randomized design with a relatively low number of patients. Another limitation was that our results are limited to postoperative new-onset, in-hospital AF, and the development of PoAF after the patients were discharged was not evaluated. Also, the development of PoAF is multifactorial; and it was another limitation that only parameters that had possible major impacts (e.g. age, hypertension, conventional TTE and TDI measurements, etc.) were included in the statistical evaluation of our study data. For this reason, the predictive effect of AEMD durations on the development of PoAF must be confirmed with broader

prospective studies with longer follow-up times in patients undergoing CABG.

Conclusion

AEMD was found to be an independent predictive factor for the development of PoAF. The triage of patients with high risk of developing PoAF can prevent PoAF, shorten the length of hospital stay, and reduce morbidity and mortality. For this purpose, measuring AEMD duration with TTE is a simple, fast and reliable method that can be used in risk the classification for the development of PoAF in patients undergoing CABG.

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Declaration of Competing Interest

The authors have no relevant conflicts of interest to disclose.

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Figure legend:

Figure 1: Atrial electromechanical delay A) LV lateral, B) LV medial, C) RV lateral, D) LA lateral, E) LA medial, F) RA lateral

Figure 2: ROC curve of AEMD parameters.

Table 1. Demographic, clinical and preoperative laboratory characteristics of the patients.

Variables	Variables	Group 1: SR (n = 68)	Group 2: PoAF (n = 25)	p value
		Mean±sd / n (%)	Mean±sd / n (%)	
Age (year)	Age (year)	58.41 ± 10.31	64.40 ± 9.72	0.014
Gender	Male Female	54 (79.4%) 14 (20.6%)	18 (72%) 7 (28%)	0.44
Body mass index (kg/m ²)	Body mass index (kg/m ²)	28.73 ± 4.66	27.60 ± 4.11	0.28
Hypertension	Hypertension	27 (39.7%)	16 (64%)	0.03
Diabetes mellitus	Diabetes mellitus	34 (50%)	9 (36%)	0.23
Cerebrovascular disease	Cerebrovascular disease	2 (2.9%)	1 (4%)	0.79
Smoking cigarette	Smoking cigarette	31 (45.6%)	11 (44%)	0.59
COPD	COPD	6 (8.8%)	2 (8%)	0.90
Peripheral arterial disease	Peripheral arterial disease	8 (11.8%)	1 (4%)	0.26
Beta-blocker	Beta-blocker	46 (67.6%)	16 (64%)	0.74
ACEI	ACEI	13 (19.1%)	6 (24%)	0.60
ARB	ARB	7 (10.3%)	5 (20%)	0.21
Diuretics	Diuretics	6 (8.8%)	8 (32%)	0.01
Systolic blood pressure (mmHg)	Systolic blood pressure (mmHg)	120.88 ± 11.52	118.60 ± 12.54	0.90
Diastolic blood pressure (mmHg)	Diastolic blood pressure (mmHg)	73.22 ± 8.27	75.20 ± 8.71	0.31
Heart rate (bpm)	Heart rate (bpm)	74.47 ± 11.69	72.56 ± 15.00	0.52
WBC (x10 ³ µL)	WBC (x10 ³ µL)	9.29 ± 2.50	8.94 ± 2.68	0.55
Hemoglobin (gr/dL)	Hemoglobin (gr/dL)	12.96 ± 1.62	12.98 ± 1.32	0.94
Platelet (x10 ³ µL)	Platelet (x10 ³ µL)	250.77 ± 67.39	257.68 ± 69.01	0.66
Glucose (mg/dL)	Glucose (mg/dL)	144.97 ± 64.34	133.24 ± 70.91	0.45
BUN (mg/dL)	BUN (mg/dL)	17.58 ± 7.40	17.20 ± 5.43	0.81
Creatinine (mg/dL)	Creatinine (mg/dL)	0.93 ± 0.26	0.89 ± 0.30	0.58
Sodium (mEq/L)	Sodium (mEq/L)	138.60 ± 3.66	137.96 ± 3.19	0.44
Potassium (mEq/L)	Potassium (mEq/L)	4.27 ± 0.44	4.47 ± 0.54	0.07
Magnesium (mEq/L)	Magnesium (mEq/L)	1.97 ± 0.26	1.92 ± 0.17	0.32
Total cholesterol (mg/dL)	Total cholesterol (mg/dL)	191.77 ± 43.12	196.80 ± 36.17	0.60
LDL-C (mg/dL)	LDL-C (mg/dL)	117.88 ± 38.72	125.57 ± 34.16	0.37
HDL-C (mg/dL)	HDL-C (mg/dL)	42.25 ± 8.65	41.68 ± 6.93	0.76

ACEI: Angiotensin converting enzyme inhibitor; ARB: Angiotensin receptor blocker; BUN: Blood urea nitrogen; COPD: Chronic obstructive pulmonary disease; HDL-C: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol; PoAF: Postoperative atrial fibrillation; SR: Sinus rhythm; WBC: White blood cell.

Table 2. Echocardiographic characteristics of the patients.

Variables	Variables	Group 1: SR (n = 68)	Group 1: SR (n = 68)	Group 2: PoAF (n = 25)	p value
Variables	Variables	Group 1: SR (n = 68)	Group 1: SR (n = 68)	Group 2: PoAF (n = 25)	p value
		Mean±sd	Mean±sd	Mean±sd	
Ejection fraction (%)	Ejection fraction (%)	56.89 ± 6.54	56.89 ± 6.54	55.04 ± 8.46	0.26
LVEDD (mm)	LVEDD (mm)	46.59 ± 3.82	46.59 ± 3.82	45.68 ± 4.29	0.32
LVEDS (mm)	LVEDS (mm)	30.34 ± 4.40	30.34 ± 4.40	29.80 ± 3.59	0.63
Septum (mm)	Septum (mm)	11.05 ± 1.93	11.05 ± 1.93	10.47 ± 2.25	0.22
Posterior wall (mm)	Posterior wall (mm)	10.18 ± 1.36	10.18 ± 1.36	9.88 ± 2.19	0.42
LA diameter (mm)	LA diameter (mm)	38.63 ± 4.19	38.63 ± 4.19	39.83 ± 4.49	0.23
LA maximum volume (ml)	LA maximum volume (ml)	47.55 ± 16.98	47.55 ± 16.98	61.36 ± 22.02	0.002
PAP (mmHg)	PAP (mmHg)	17.17 ± 7.39	17.17 ± 7.39	21.92 ± 9.17	0.012
Mitral E velocity (cm/s)	Mitral E velocity (cm/s)	76.19 ± 20.97	76.19 ± 20.97	74.57 ± 15.39	0.43
Mitral A velocity (cm/s)	Mitral A velocity (cm/s)	83.96 ± 17.82	83.96 ± 17.82	89.31 ± 21.45	0.22
Mitral E/A	Mitral E/A	0.94 ± 0.31	0.94 ± 0.31	0.87 ± 0.42	0.43
LV lateral Ea velocity (cm/s)	LV lateral Ea velocity (cm/s)	9.03 ± 2.04	9.03 ± 2.04	7.77 ± 1.56	0.006
LV lateral Aa velocity (cm/s)	LV lateral Aa velocity (cm/s)	11.25 ± 2.54	11.25 ± 2.54	10.93 ± 2.77	0.60
LV lateral Sa velocity (cm/s)	LV lateral Sa velocity (cm/s)	8.58 ± 2.09	8.58 ± 2.09	8.31 ± 2.14	0.58
LV medial Ea velocity (cm/s)	LV medial Ea velocity (cm/s)	7.06 ± 1.66	7.06 ± 1.66	6.13 ± 1.82	0.02
LV medial Aa velocity (cm/s)	LV medial Aa velocity (cm/s)	9.88 ± 1.73	9.88 ± 1.73	9.25 ± 1.40	0.10
LV medial Sa velocity (cm/s)	LV medial Sa velocity (cm/s)	7.37 ± 1.34	7.37 ± 1.34	7.08 ± 1.20	0.35
LV lateral E/Ea	LV lateral E/Ea	8.75 ± 3.02	8.75 ± 3.02	9.75 ± 3.18	0.16
LV medial E/Ea	LV medial E/Ea	11.14 ± 3.40	11.14 ± 3.40	12.47 ± 3.07	0.09
LV lateral AEMD (ms)		40.20 ± 14.77	55.64 ± 15.69	< 0.001	
LV medial AEMD (ms)		31.66 ± 13.70	40.92 ± 12.94	0.004	

Variables	Variables	Group 1: SR (n = 68)	Group 1: SR (n = 68)	Group 2: PoAF (n = 25)	p value
RV lateral AEMD (ms)		15.36 ± 13.02	24.24 ± 12.26	0.004	
LA lateral AEMD (ms)		39.92 ± 16.81	54.64 ± 17.46	< 0.001	
LA medial AEMD (ms)		26.23 ± 14.07	31.96 ± 14.59	0.08	
RA lateral AEMD (ms)		9.98 ± 13.48	14.08 ± 11.91	0.18	

A: Late diastolic wave; Aa: Late diastolic annular wave; AEMD: Atrial electromechanical delay; E: Early diastolic wave; Ea: Early diastolic annular wave; LA: Left atrium; LV: Left ventricle; LVEDD: Left ventricle end-diastolic diameter; LVESD: Left ventricle end-systolic diameter; PAP: Pulmonary artery pressure; PoAF: Postoperative atrial fibrillation; RA: Right atrium; RV: Right ventricle; Sa: Systolic annular wave; SR: Sinus rhythm.

Table 3. Perioperative and postoperative characteristics of the patients.

Variables	Group 1: SR (n = 68)	Group 2: PoAF (n = 25)	Group 2: PoAF (n = 25)	p value
	Mean±sd / n (%)	Mean±sd / n (%)	Mean±sd / n (%)	
CABG time (min)	217.57 ± 56.08	215.80 ± 50.75	215.80 ± 50.75	0.89
CPB time (min)	82.74 ± 26.71	89.54 ± 27.25	89.54 ± 27.25	0.29
Cross-clamp time (min)	59.77 ± 23.68	63.95 ± 23.39	63.95 ± 23.39	0.45
Number of grafts	3.04 ± 0.87	3.28 ± 0.84	3.28 ± 0.84	0.24
Minimum body temperature (°C)	30.22 ± 0.63	30.13 ± 0.50	30.13 ± 0.50	0.49
Positive inotrop use	41 (60.3%)	19 (76.0%)	19 (76.0%)	0.16
IABP use	2 (2.9%)	4 (16%)	4 (16%)	0.02
Intubation time (h)	10.48 ± 4.45	12.04 ± 4.45	12.04 ± 4.45	0.16
ICU time (h)	51.30 ± 12.94	60.48 ± 18.48	60.48 ± 18.48	0.009
WBC (x10 ³)	11.83 ± 2.73	12.23 ± 3.45	12.23 ± 3.45	0.56
Hemoglobin (gr/dL)	9.45 ± 0.79	9.63 ± 0.82	9.63 ± 0.82	0.34
Platelet (x10 ³)	253.82 ± 86.42	283.32 ± 101.61	283.32 ± 101.61	0.17
Glucose (mg/dL)	156.60 ± 54.96	143.36 ± 50.56	143.36 ± 50.56	0.29
BUN (mg/dL)	19.70 ± 9.16	21.36 ± 12.51		0.48
Creatinine (mg/dL)	0.97 ± 0.32	1.06 ± 0.58		0.36
Sodium (mEq/L)	137.20 ± 3.84	136.00 ± 4.06		0.50
Potassium (mEq/L)	3.67 ± 0.41	3.84 ± 0.50		0.11

BUN: Blood urea nitrogen; CABG: Coronary artery bypass grafting; CPB: Cardiopulmonary bypass; IABP: Intra-aortic balloon pump; ICU: Intensive care unit; PoAF: Postoperative atrial fibrillation; SR: Sinus rhythm; WBC: White blood cell.

Table 4. Univariate and multivariate logistic regression analysis of risk factors for PoAF development.

Variables	Univariate	Univariate	Multivariate	Multivariate	Multivariate
	OR (95% CI)	p value	OR (95% CI)	OR (95% CI)	p value
Age	1.06 (1.01-1.12)	0.01	1.03 (0.97-1.09)	1.03 (0.97-1.09)	0.30
Gender, female	1.50 (0.52-4.29)	0.45			
Body mass index	0.94 (0.84-1.05)	0.28			
Hypertension	2.70 (1.04-6.98)	0.004	1.15 (0.50-4.54)	1.15 (0.50-4.54)	0.46
Diabetes mellitus	1.77 (0.69-4.57)	0.23			
Hemoglobin	1.01 (0.74-1.36)	0.94			
LA maximum volume	1.03 (1.01-1.06)	0.004	1.02 (0.99-1.05)	1.02 (0.99-1.05)	0.12
LA lateral AEMD	1.05 (1.01-1.07)	0.001	1.03 (1.001-1.06)	1.03 (1.001-1.06)	0.04
Cross-clamp time	1.008 (0.98-1.02)	0.45			
PAP	1.07 (1.01-1.13)	0.01	1.04 (0.97-1.12)	1.04 (0.97-1.12)	0.17

OR: Odds Ratio; CI: Confidence Interval

AEMD: Atrial electromechanical delay; LA: Left atrium; PAP: Pulmonary artery pressure.



