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ORIGINAL ARTICLE

Serum Uric Acid/ Albumin Ratio and Short-Term Outcomes in ST Elevation Myocardial Infarction

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ABSTRACT

Background: Serum Albumin (SA) and uric acid (UA) production and excretion could be correlated with major adverse cardiovascular events (MACE) among cases who had ST Elevation Myocardial Infarction (STEMI) and undergone primary Percutaneous Coronary Intervention (PCI). We aimed to assess the relation between high Uric Acid Albumin Ration (UAR) and short-term outcomes among patients who had STEMI and undergone primary PCI

Methods: we carried out this cohort study on 80 patients who had STEMI (and confirmed by cardiac enzymes and ECG) divided into two groups: Group I: patients with MACE. (n=33) and Group II: patients without MACE. (n=47), as determined by the modified Dukes criteria. Both groups undergone primary percutaneous intervention. Serum Uric acid, Serum Albumin, and cardiac enzymes (CK, CK-MB, Troponin) were assessed on admission with follow up of cardiac enzymes post procedure.

Results: MACE group had significantly higher uric acid and UAR levels than the non-MACE group (P value <0.01). UAR could significantly predict development of MACE (AUC = 0.65) at cut-off: 1.6 with 81% sensitivity, 76 % specificity. A statistically significant correlation was revealed between the 2 groups in Killip classification (p<0.001), UA, UAR, RWMSI & LVEF (p=0.012, 0.016, 0.002 and 0.014 respectively). Positive correlations were found between Killip Classification, UA, UAR, RWMSI and MACE were revealed (p=0.012, 0.016, and 0.014 respectively) while an inverse relationship between LVEF and MACE was found (p=0.002).

Conclusions: Uric acid-to-albumin ratio could significantly predict development of MACE among patients who had STEMI undergoing PCI with high sensitivity and specificity. UA, UAR, RWMSI and Killip classification were positively correlated with MACE while LVEF was inversely related with MACE.

Keywords: Uric Acid/ Albumin Ratio;Short-term outcomes; ST Elevation Myocardial Infarction

INTRODUCTION

Acute myocardial infarction, the most serious kind of coronary artery disease, is responsible for more than 2.4 million fatalities annually in the US, more than 4 million in Europe and northern Asia, and more than one third of all deaths in developed nations [1]. Primary percutaneous coronary intervention (PCI) is the therapy of choice for acute coronary syndrome involving STEMI. Because of this, risk stratification is an absolute must in the management and prevention of STEMI [2].

The metabolism of purines culminates in uric acid. The kidneys are in charge of excreting uric acid from the body, which accounts for two-thirds of the total daily uric acid, while the intestines remove one-third. Blood uric acid levels in adults should be within the

normal range of 3.5 to 7 mg/dL. Blood uric acid levels can be influenced by a wide range of medical diseases and medications, including hypertension, diabetes, obesity, CKD, diuretics, and anti-hypertensive medications. Recent research found a correlation between hyperuricemia and the increased risk of chronic kidney disease (CKD), cardiovascular death, and death from any cause. But the results of such research have been completely contradictory. Thus, the link between uric acid levels in the blood and death rates is still up for debate [3,4].

When it comes to both normal and abnormal physiological processes, serum albumin (SA) plays a crucial role. The typical range for serum albumin is 3.5-5.2 g/dL. Numerous hormones, medications, bioactive components, free fatty acids, calcium, sodium, and iron are transported by it, and it is the primary factor in determining intravascular oncotic pressure [5]. Albumin is more than just a transporter; prior research has demonstrated its anti-inflammatory, anti-oxidative stress-induced injury-protecting, and anti-apoptotic properties [6].

It is challenging to employ SA and UA as standalone indicators of ACS because of the wide variety of potential influences on their production and excretion [7]. So, we aimed to assess the relation between high Uric Acid Albumin Ration (UAR) and short-term outcomes among patients who had STEMI and undergone primary PCI.

METHODS

We carried out this cohort study on 80 patients who had acute coronary syndrome with ECG criteria diagnostic of STEMI who have been admitted to the Department of Cardiovascular diseases, Zagazig University hospitals, from January 2023 to January 2024. The patients were categorized into two groups: The first group involved patients who developed MACE while the second group patients without MACE. Primary percutaneous intervention and medication were part of the usual therapy for both groups of patients with acute STEMI. The research ethics board of the medical school at Zagazig University gave its blessing to the study, and all participants gave written informed consent. A component of the Code of Ethics for Research Involving Humans, the Declaration of Helsinki ensures that the study was carried out in compliance with its provisions. Before this study could begin, we obtained the approval from the Institutional Review Board (IRB#10226).

Cases with the following criteria were included: Patients who had ACS with ECG criteria diagnostic of STEMI (The ST-segment elevation in leads V2-V3 and/or other leads must be at least 1.5 mm among females, 2.5 mm in males under 40 years old, 2 mm in men 40 years and older, and 1 mm in all other leads) who undergone primary PCI [8].

Cases with the following characteristics were excluded: Cases who had post Thrombolytic therapy, Late presentation after 24 hours of the onset of chest pain, Post CABG, Pregnant females, chronic kidney disease (creatinine clearance $\leq$ 60ml/min), Patients already treated of hyperuricemia, Patient refusal to PCI, as well as patients presented with cardiogenic shock primary PCI [9].

All study populations had the following: Careful history taking, Full cardiac examination including Vital signs including

Blood pressure, heart rate and respiratory rate as well as Cardiac and chest auscultation.

Investigations: 12 lead surface ECG: (Pre-procedure, post-procedure immediately within 12-24 hours and daily), Serum Uric acid, Serum Albumin, complete blood count (CBC), serum creatinine, sodium, potassium, liver enzymes, INR and cardiac enzymes (CK, CK-MB, Troponin) on admission and follow up cardiac enzymes post procedure. Pre-discharge echocardiography: Echocardiography was done to all patients after the PCI procedure to assess the MR, regional segmental wall motion abnormality, LV dimensions and systolic function. In hospital follow up for MACEs including heart failure, cardiac mortality and reinfarction.

Intervention phase:

Coronary angiography, primary angioplasty, as well as stenting: Before the coronary angiography, all patients were given ticagloror (180 mg) or clopidogrel (600 mg loading dosage) and aspirin (300 mg) chewable. Angioplasty and emergency coronary angiography were carried out. Initial injections were made into the artery that was thought to be clear of obstructions. The TIMI classification, the SYNTAX score, and the Gensini score for coronary artery disease severity were used to rate the blood flow in the infarct-related artery (IRA). Thrombus grade was also used to define thrombus presence. Upon initial determination of the coronary architecture, heparin was delivered at a dose of 100 IU/kg.

The coronary arteries on the left and right sides can then be seen. An angiographic evaluation was conducted through visible examination. Patients with IRA will only have primary angioplasty, which may involve balloon angioplasty and/or stent placement, based on the type of lesion. The operator was charged with the responsibility of using aspiration devices and glycoprotein IIb/IIIa inhibitors [10]. Interventional success during the acute period was determined for each procedure as the presence of residual stenosis to less than 30% of obstruction. Patients were admitted to the coronary care unit after angioplasty and were all prescribed aspirin and either clopidogrel 75 mg once daily or ticagrolor 90 mg twice daily.

The severity of coronary artery disease was evaluated using a lesion-based angiographic grading technique known as SYNTAX score [11]. To measure the extent of atherosclerosis, the Gensini score was employed [12].

Statistical Analysis:

We used SPSS 23.0 for Windows (IBM Inc., Chicago, IL, USA) to conduct all of our statistical reports. For the quantitative variables—mean and standard deviation (SD)—we compared the two groups using unpaired Student's t-test. Quantitative variables were presented as frequency and percentage (%), and qualitative variables were analyzed with the Chi-square test or Fisher's exact test as needed. A two-tailed P value below 0.05 was used to demonstrate statistical significance, and a P value below 0.001 was used to characterize a highly significant result. Each test's overall diagnostic performance was assessed using analysis of ROC curves. The AUC was a measure of the test's overall performance. As a statistical threshold, a p-value of less than 0.05 was utilized.

RESULTS

Patients included in our study ranged from 24 years and 87 years with mean ± SD

(56.43±11.38) 65 patients were males (81.2 %), 39 patients (48.8 %) were hypertensives, 37 had diabetes (46.3 %), 47 (58.8 %) were smokers, and 14 (17.5 %) had dyslipidemia, 33 patients (41.2 %) suffered MACE (eg. 26 cases had heart failure, 4 cases had Post MI Angina, 3 cases had no reflow), 15 cases had cardiogenic shock, 3 had mechanical complications) and 47 (58.8 %) were free of MACE, with non-statistically significant difference between the two groups regarding as regards age, gender or comorbidities of the patients (Table 1).

Table (2) showed that in MACE group had significantly higher Uric Acid and UAR levels compared to the non-MACE group (8.4 ± 2.3 mg/dl and2.77 ±1.2 versus 7.15 ± 2.05 mg/dl and 2.19 ± 0.9 with P value <0.01).

The MACE group had a considerably lower left ventricular ejection fraction (LVEF) than the non-MACE group (P 0.002), but the MACE group had a higher Regional Wall Motion Score Index (RWMSI). There was no statistically significant difference between the two groups in terms of coronary angiographic parameters (Table 3).

ROC curve analysis was performed on UAR and revealed that it can significantly predict Development of MACE (P <0.01 and AUC = 0.65) at cut-off : 1.6 with 81% sensitivity, 76 % specificity (Table 4, Figure 1).

Statistically significant correlation using Spearman's rank between the two groups in Killip classification (p<0.001) (Table 5).

Statistically significant differences were revealed between the two groups in UA, UAR, RWMSI & LVEF (p=0.012, 0.016, 0.002 and 0.014 respectively). Positive correlations were found between Killip Classification, UA, UAR, RWMSI and MACE were revealed (p=0.012, 0.016, and 0.014 respectively) while an inverse relationship between LVEF and MACE was found (p=0.002). (Table 6).

Regarding short term outcomes other than MACE,Short term outcomes including All-cause mortality at 30 days up to 1 year (AF, Atrial flutter, VT & VF) and Cardiogenic shock patients requiring Hemodynamic support. No Statistically significant regarding vascular complications (Supplementary Table 1).

Table 1: Demographic data and comorbidities of both groups

	Group I MACE (No. 33)	**Group II No MACE (No. 47)**	**P**
Age (mean ±SD)	57.7±10.47	55.5±12	0.3
Gender Males (n= 65) Females (n= 15)	 26 (40 %) 7 (46.7 %)	 39 (60 %) 8 (53.3 %)	0.64
HTN	12 (36.4%)	27 (57.4%)	0.06
DM	15 (45.5%)	22 (46.8%)	0.90
Dyslipidemia	8 (24.2%)	6 (12.8%)	0.18
Smoking	23 (69.7)	24 (51.1%)	0.22
known CAD	10 (30.3%)	11 (23.4)	0.49

HTN: hypertension, DM: Diabetes mellitus, CAD: coronary artery disease

Table 2: Laboratory parameters of the studied groups

Mean ± SD	Group I MACE (No. 33)	Group II No MACE (No. 47)	P Value
HGB(g/dl)	13.5±1.9	12.96±1.8	0.15
TLC (10^3/µL)	12.6 ± 5	11.02 ± 3.9	0.13
PLT (×10^9/L)	271.3 ± 61	267.94 ± 73	0.8
Serum Cr. (mg/dL)	1.05 ± 0.38	1.1 ± 0.7	0.6
eGFR	82.68 ± 25	84.97 ± 28	0.7
INR	1.18 ± 0.2	1.11 ± 0.1	**0.03**
CK(u/L)	431 ± 726	394 ±521	0.4
CKMB (ng/mL)	146 ± 163	115±117	0.3
Tn(ng/L)	2.83 ±3.38	2.25 ± 2.8	0.4
UA (mg/dl)	8.4 ± 2.3	7.15 ± 2.05	**0.01***
SA (g/dl)	3.32 ±0.8	3.45±0.670	0.4
UAR	2.77 ±1.2	2.19 ± 0.9	**0.01***

HGB: haemoglobin, TLC: total leucocytic count, PLT: platelets, Serum CR: Serum creatinine, eGFR: estimated glomerular filtration rate, INR: International normalised ratio, CK: Creatine kinase-. CKMB:Creatine kinase-MB ,Tn: troponin, UA: Uric acid, SA: serum albumin, UAR: uric acid albumin ratio *: significant as P value≤0.05. TLC: Total leucocyte count. CK-MB: creatine kinase-myocardial band

Table 3: Echocardiography and Coronary angiographic parameters of both groups

	Group I MACE No 33	Group II No MACE No. 47	P
LVEF	42.24 ±9.7	48.15 ±7.35	**0.002****
RWMSI	1.53 ± 0.3	1.36 ± 2.8	**0.01***
Pain to wire in hours	9.52 ± 3.11	9.26 ±3.7	0.7
Aggrastat	9 (27.3%)	8 (17%)	0.2
Asap	2 (6.1%)	0	0.1
Culprit Vessel **LAD** **LCX** **RCA**	 21 (63.6%) 3 (9.1%) 9 (27.3%)	 20 (42.6%) 9 (19.1%) 18 (38.3%)	0.1
No of Diseased Vs **1 vs Dse** **2 vs Dse** **3 vs dse**	 11 (33.3%) 14 (42.4%) 8 (24.2%)	 20 (42.6%) 18 (38.3%) 9 (19.1%)	0.6
TIMI Flow **TIMI 0** **TIMI 1** **TIMI 2** **TIMI 3**	 3 (9.1%) 1 (3%) 6 (18.2%) 23 (69.7%)	 0 4 (8.5%) 5 (10.6%) 38 (80.9%)	0.5

	Group I MACE No 33	Group II No MACE No. 47	P
MBG			0.9
1	4 (12.1%)	5 (10.6%)	
2	10 (30.3%)	16 (34%)	
3	19 (57.6%)	26 (55.3%)	
Syntax score	14.48±7.9	14.58±9.27	0.9
Gensini score	32±42.7	31±45.04	0.7

LAD: Left Anterior Descending Artery.,LCX: Left Circumflex Artery,RCA: Right Coronary Artery, 1 vs Dse: This means there is one vessel with significant disease (narrowing or blockage). 2 vs Dse: There are two vessels with significant disease. 3 vs Dse: There are three vessels with significant disease.TIMI stands for Thrombolysis In Myocardial Infarction grading.MBG: Myocardial Bridging Angiography, LVEF: Left ventricular ejection fraction. RWMSI: Regional Wall Motion Score Index.

Table 4: Roc curve for UAR to predict development of MACE.

Cut-off	Sensitivity	Specificity	95 % confidence interval	AUC	P Value
1.6	81 %	76 %	0.52-0.78	0.65	0.019*

AUC: area under the curve. *: significant as P value≤0.01

Table 5: ***Spearman's rank correlation between both studied group parameters***

	r	p
Gender	.053	.641
Killip Class	.684	.000**
No of diseases vs	.095	.402
MBG	.013	.909

**. Correlation is significant at the 0.01 level (2-tailed
MBG: Myocardial Bridging Angiography

Table 1: Pearson Correlations among different parameters between both studied groups

	r	p
Age	.096	.395
Pain to wire	.037	.745
Syntax score	-.006-	.960
Gensini_score	-.035-	.755
HGB	.159	.158
TLC	.170	.131
PLT	.024	.830
S.cr	-.046-	.687
eGFR	-.042-	.712

	r	p
INR	.157	.164
UA	.278	**.012***
S.Alb	-.083-	.467
UAR	.268	**.016***
CKMB	.111	.326
CK	.029	.796
Tn	.094	.405
LVEF new	-.340-	**.002****
RWMSI	.274	**.014***

HGB: haemoglobin, TLC: total leucocytic count, PLT: platelets, Serum CR: Serum creatinine, eGFR: estimated glomerular filtration rate, INR: International normalised ratio, CK: Creatine kinase-. CKMB: Creatine kinase-MB ,Tn: troponin, UA: Uric acid, SA: serum albumin, UAR: uric acid albumin ratio *: significant as P value≤0.05. TLC: Total leucocyte count. CK-MB: creatine kinase-myocardial band, LVEF: Left ventricular ejection fraction. RWMSI: Regional Wall Motion Score Index.

*.Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-taile

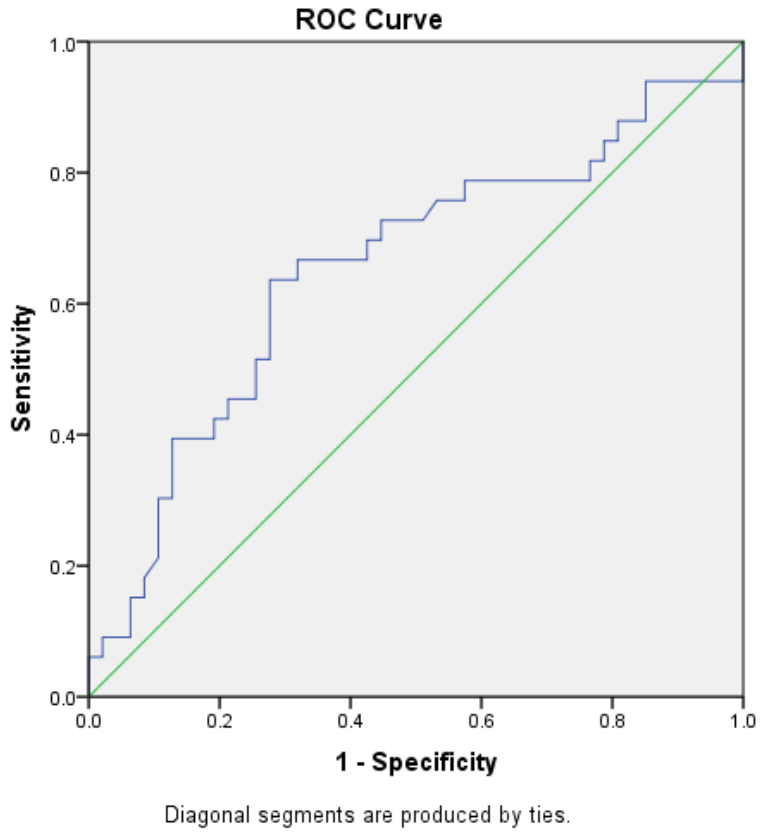

Figure 1: ROC curve For UAR to predict MACE.UAR: Uric acid albumin ratio, MACE: Major adverse cardiovascular event

DISCUSSION

New evidence suggests that UAR may be a useful indicator of inflammation and oxidative stress; both factors are strongly associated with cardiovascular diseases, according to the aforementioned research [14]. Included in the definition of major adverse cardiac events were deaths from any cause, nonfatal myocardial infarction (MI), nonfatal stroke (NFS), revascularization, and readmission to the hospital for unstable angina or heart failure [15].

Reportedly, the UAR, a novel measure of oxidative and inflammatory stress that integrates UA and albumin readings to represent the whole oxidative and inflammatory status of the body, is associated with unfavorable cardiovascular prognostic outcomes and the degree of coronary artery disease (CAD) severity [13].

The progression of heart failure and a rise in cardiovascular mortality have both been associated with elevated UA. Hyperuricemia also causes reactive oxygen species (ROS) to be produced in the cardiovascular system, which in turn activates neurohormones, lowers intracellular ATP concentration, and hinders endothelial function. Hyperuricemia is clearly associated with the onset of cardiovascular illness, including heart failure, as these side effects demonstrate. Albumin transports a number of chemicals that control acute and chronic inflammatory processes, and it is also critical for keeping the plasma colloid osmotic pressure constant. Albumin concentration drops during inflammation because to catabolism [16-20].

In the current study, non-statistically significant differences were revealed between both groups regarding mean age (57.7 ± 10.47 in MACE group vs 55.5 ± 12 years in No MACE group), and gender distribution (7 females & 26 males in the MACE group and 8 females & 39 males in No MACE group). When looking at the demographic data (age and gender), there was no discernible difference between the two groups, this was discordant with El rashidy et al. [21] as they revealed that UAR was higher in males compared to females. It is thought to be related to a higher renal clearance of urate in women, possibly due to higher plasma estrogen level (Suppressive effects of estradiol on Uric Acid).

There was no statistical difference between both groups regarding the incidence of different risk factors of CAD (smoking, hypertension, Dyslipidemia and IHD) which agreed with Yalcin et al. [7]. Except for diabetes which was higher in their study and disagreed with El rashidy et al. [21] where patients with high UAR were more dyslipidemic and smokers.

As for Laboratory parameters , both groups in our study showed no significant except for UA and UAR owing to increased UA Levels, yet SA levels in both groups was comparable, both groups had low normal SA levels mostly due to malnutrition and poor dietary habits of people in Egypt nowadays paving the way for more PLT aggregation, activation and thrombus formation which was in concordant with Yin et al. [22] and discordant with Sultan et al. [23] as the later was a retrospective study with small sample enrolling even those with normal coronaries and syntax scores about Zero as well as the UA and SA levels were normal.

Endothelial dysfunction [23], oxidative stress [24], and inflammation [25] are three mechanisms via which hyperuricemia may raise the risk of CVDs, according to previous research. Other research, however, failed to find any evidence linking serum urate levels to CAD. Serum UA levels over normal have

been associated with poorer clinical outcomes and an increased risk of cardiovascular disease [26].

Epidemiological studies revealed that low serum albumin levels are an indicator of vascular endothelial dysfunction. Arques. [27] discovered a positive relation between low albumin levels and an elevated danger of inflammation, the primary cause of endothelial dysfunction. Moreover, Kinoshita.et al [28] showed that Pregnant women's serum albumin levels were positively connected with endothelial function and adversely correlated with oxidative stress. Hypoalbuminemia may contribute to the onset of CVD, and serum albumin may be an important factor in vascular oxidative stress, according to their findings. The pathophysiology of cardiovascular diseases is heavily influenced by endothelial dysfunction. Vascular tone disruption, redox imbalance, and enhanced endothelial inflammation results. One of the many cardiovascular diseases caused by impaired endothelium-dependent vasodilatation is atherosclerosis [29]. In addition, a low albumin level following a percutaneous coronary intervention is a strong predictor of poor long-term results [30].

As regard for the echocardiographic data, The MACE group had higher RWMSI & Lower Systolic functions compared to those in the No MACE group, which was in agreement with Kong et al. [31] detonating the severity of CAD at all levels associated with increased levels of serum UA and low serum Albumin levels. Consistent with the prior research, Consistent with this, we found that the MACE group had a higher RWMSI than the N-MACE group.

Considering coronary angiography in our study there was no statistically significant difference between both groups regarding pain to wire or drugs together with culprit vessel, number of vessels, thrombolysis in myocardial infarction (TIMI) flow, myocardial blush grade score (MBG) and syntax score as well as Gensini score owing that most of our patients were Multi vessel disease, this disagrees with Yalcin et al. [7], where high UAR was seen more in those with high syntax score this might be because the population studied might be much higher as well as being limited to population with chronic coronary syndrome. And disagree also with Kong et al. [31], because the Syntax score was greater in the MACE group than in the N-MACE group.

As for the Cutoff value of UAR in our study it was 1.6 and it was found to be higher in the group with MACE with a sensitivity of 81% and a specificity 76 %, a figure much higher was present in a study done by Yin et al. [22], owing to the very low levels of SA in their studied groups.

Taking into consideration short term complications regarding the different arrhythmias, People in the MACE group showed different types of arrhythmias at a higher level compared to those without MACE as well as their Killip class was higher which highlight the poor hemodynamics as well as the electrical instability in the MACE group.

Armillotta et al. [32] showed that among these AMI patients, a high Killip class was also identified as an independent predictor of unfavorable cardiac outcomes. Selçuk et al. [33] demonstrated that the UAR could be used as an independent predictor of the occurrence of new onset atrial fibrillation (NOAF) in patients with STEMI. Topaz et al. [34] demonstrated that previous atrial fibrillation, rather than newly-onset atrial

fibrillation, is a significant predictor of both the short-term and long-term death rates in patients who undergone PCI.

Limitations of the current study include small sample size that increased the risk of overlooking significant differences between the two groups, short term follows up during hospital stay, Study was limited to STEMI patients only, and Echocardiographic and ECG follow-up was limited to 4 weeks.Further larger studies are needed to validate the current study findings

Conclusions:

Uric acid-to-albumin ratio can significantly predict development of MACE among patients who had STEMI undergoing primary percutaneous coronary intervention at cut-off: 1.6 with 81% sensitivity and 76 % specificity. UA, UAR, RWMSI and Killip classification were positively correlated with MACE while LVEF was inversely related with MACE. MACE increases arrhythmia (AF, Atrial flutter, VT & VF) and cardiogenic shock patients requiring hemodynamic support.

Conflict of interest: None

Financial disclosure: None

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Supplementary Table 2: Clinical outcomes of both groups

		Group I MACE No 33	Group II No MACE No. 47	P
New Arrhythmia	**A. Flutter**	1	0	**0.002**
	AF	3	0	
	CHB	1	1	
	VT	2	0	
	VF	2	0	
Vascular complications	**Dissection**	2	1	0.13
	Hematoma	0	3	
Cardiogenic shock		14	33	**0.003**

AF: Atrial Fibrillation, CHB: Chronic Heart Block, VT: Ventricular Tachycardia, VF: Ventricular Fibrillation

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