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ORIGINAL ARTICLE

Impact of Atherogenic Index of Plasma on Spontaneous Reperfusion and its Implications on mid-term Outcomes in Patients with ST-Segment Elevation Myocardial Infarction

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ABSTRACT

Objective: Our aim was to assess the correlation between atherogenic index of plasma (AIP) and spontaneous reperfusion (SR) in patients with ST elevation myocardial infarction (STEMI) and also to evaluate its relationship with in-hospital & mid-term outcomes.

Methods: Our study included 259 STEMI patients. AIP was calculated from the lipid profile of all participants, who underwent coronary angiography. SR was defined as achievement of grade 3 TIMI fl0w in the infarct-related artery (IRA) before PCI. We categorized patients into a SR group [57 (22%)] and a non-SR group [202 (78%)]. Gensini scoring system was used to evaluate the severity of coronary artery disease.

Results: The AIP value was higher in the Non-SR group than in the SR group [0.71 ± 0.11 vs. 0.56 ± 0.03; p <0 .001]. AIP had positive correlation (0.53, P<0.001) with severity of CAD. The in-hospital and mid-term adverse outcome was lower in patients with SR. AIP was found to be correlated with diabetes, high sensitive C-reactive protein, brain natriuretic peptide (BNP), left atrial (LA) volume index and E/e'. Furthermore we observed that AIP was the strongest independent predictor for non-SR in STEMI patients. The cut-off value of AIP for predicting non-SR was >0.61, (AUC: 0.89).

Conclusion: Lower AIP is associated with the development of SR in STEMI patients. AIP was positively correlated well with the severity of CAD. High AIP is also associated with adverse outcomes. We found that higher AIP value was an independent predictor of non-SR. Hence, AIP could be advocated for routine measurement in clinical practice.

Key words. Atherogenic index, spontaneous reperfusion, STEMI.

INTRODUCTION

The main aim of percutaneous coronary intervention (PCI) for acute myocardial infarction (AMI) is gaining rapid myocardial perfusion. Two types of myocardial reperfusion are recognized; spontaneous reperfusion (SR) and reperfusion which is related to either primary percutaneous coronary intervention (PPCI) or thrombolysis (Non-SR). In subjects with SR, earlier myocardial salvage is frequently achieved as SR of occluded coronaries is attained preceding PCI, getting a good chance of survival [(1)].

Numerous angiographic studies indicated that about 30% of the subjects with acute **STEMI** have SR. Clinically, SR is a phenomenon, in which more than 50% resolution of ST-segment elevation on 2

consecutive ECG leads associated with by substantial relief of chest pain, whilst, angiographically, it is defined as attainment of thrombolysis in myocardial infarction **(TIMI)** 3 flow before any PPCI or thrombolysis. [2, 3]

The attainment of timely TIMI 3 flow in the infarcted related artery **(IRA)** improved function of the left ventricle and decreased incidence of mortality. Amongst **STEMI** subjects, who underwent PPCI, spontaneous reperfusion on coronary angiography (CAG) was associated with a good prognosis. [4]

Plasma atherogenic index, the logarithm of molar ratio of triglycerides (TG) to high density lipoprotein cholesterol (HDL-C) has been established as an index of plasma atherogenicity and coronary atherosclerosis. [5] While several lipid parameters are associated with the degree of coronary artery disease (CAD), the TG/HDL-C ratio displayed the stoutest association. The plasma atherogenic index is strictly related to the size of low density lipoprotein (LDL) C particles, which ultimately replicates small density lipoprotein levels. Small density lipoprotein has been recognized as a key to predict plasma atherogenicity and coronary artery disease. [6] So, a smart understanding of spontaneous reperfusion in individuals with STEMI will be of noticeable clinical value.

The aim of this work was to uncover the relationship between AIP and SR in **STEMI** patients and to discover its relation to lesion complexity and clinical outcomes, following AMI.

METHODS

We prospectively recruited a total of 259 consecutive patients **(174 men; mean age 59 ± 13 years),** presented with STEMI according to the following guidelines [7]**:** (i) new ST-segment elevation at the J-point in two contiguous leads with the cut-off value greater than 0.1 mV in all leads other than V_2 or V_3 and (ii) in leads V_2-V_3, a cut-off value of ≥0.2 mV in males aged ≥40 years, ≥0.25 in males aged <40 years, or ≥0.15 mV in females. We defined spontaneous reperfusion as the occurrence of **TIMI** 3 flow grade in the IRA on initial **CAG**.

We excluded subjects with atrial fibrillation, those with history of anticoagulant and/or antithrombotic medication within the previous month, advanced kidney disease, patients with malignancy, septicemia, previous myocardial infarction, former PCI, open-cardiac surgery and patients with cardiac valve disease.

Written informed consent was obtained from all participants. The study was done according to The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans. Approval for the research was given by the ethical and scientific faculty committee under the **IRB number [9459].**

Laboratory assessment: Venous blood samples were collected from all participants at the time of admission to the emergency department, analyzed by using an automated chemistry analyzer (Roche Diagnostic Modular Systems, Tokyo, Japan). Before the angiography, blood samples were collected for blood chemistry analyses. Plasma total cholesterol (TC), triglycerides (TG), low density lipoprotein-cholesterol **(**LDL-**C)**, high density lipoprotein-cholesterol (HDL-**C**), fasting glucose and creatinine levels were analyzed by commercial kits, using (Abbott Diagnostics, USA). LDL-**C** was calculated by straight LDL-**C** analyses. After that, plasma atherogenic index was obtained by the base 10 logarithm of the ratio of the TG level to HDL-C level. [8] Additional tests were obtained as high sensitive C-reactive protein (hs-CRP), levels of Brain Natriuretic Peptide (BNP) and troponin.

Echocardiography: Evaluations were performed in accordance with the American Society of Echocardiography **(ASE)** guidelines by a **Vivid 7 GE Medical System (GE Healthcare, Horten, and Norway).** The left ventricular ejection fraction and left atrial volume were measured using modified Simpson method. The following parameters were obtained baseline and after 6 months of follow-up; left ventricular ejection fraction (LVEF %), Left atrial volume index (LAVI mL/m^2), **E/e'**, stroke volume (mL/m^2). Moreover, adverse left ventricular remodeling index at the 6-month follow-up was assessed and recognized when at the six-month follow-up the LV end-systolic volume (LVESV) increased by more than 15% from the baseline dimension.[9]

Coronary Angiography: All participants underwent CAG based on the standard approach. For the left coronary system, 5 typical views were obtained (left anterior oblique cranial, left anterior oblique caudal, antero-posterior cranial, right anterior oblique cranial, and right anterior oblique caudal views), while for the right system, 2 views were obtained (left anterior oblique and right anterior oblique). All views were recorded to digital memory. Coronary lesions were assessed by two experienced interventional cardiologists. A significant and severe coronary artery stenosis was

defined as the presence of a lumen reduction of ≥50% in at least one major epicardial coronary artery. [10]

Severity of CAD was determined according to Gensini Scoring (GS) system [11]. GS is an angiographic scoring system for quantification of the severity of CAD. GS <20 means less severe and GS ≥20 means more severe CAD. **TIMI** was graded as follows: grade 0: no perfusion, grade 1: penetration without perfusion, grade 2: partial perfusion and grade 3: complete perfusion**.** Spontaneous reperfusion was defined as the attainment of TIMI grade three flow in the infarcted related artery on the baseline CAG [12,13]. Our cases were classified into two groups, a **SR** group and **non-SR** group, according to baseline CAG. Data of both groups were compared and statistically analyzed.

Follow-Up and Outcomes: All participants were followed-up for 6 months for cardiovascular adverse outcomes and echocardiographic changes.

STATISTICAL ANALYSIS

All statistical analyses were conducted using the Statistical Package for the Social Sciences 19.0 for Windows (IBM Corp. Released 2010. IBM SPSS Statistics for Windows, Version 19.0. Armonk, NY: IBM Corp.). The normal distribution of data was analyzed using the Kolmogorov–Smirnov test. Continuous data were expressed as mean ± standard deviation (SD). Fisher's exact test and/or Chi-square test were used to analyze categorical variables. The unpaired data were compared using Student's *t*-test. Univariate and multivariate logistic regression analyses were utilized to recognize the independent predictors of **SR**. The optimal cut-off value of the ***AIP*** with the strongest prediction for non-SR was evaluated by receiver operating characteristic (**ROC**) curve with the Youden index.

RESULTS

Out of 259 participants with ***STEMI***, 57 (22%) had SR, 39 (68%) of them were men. **Table 1** depicts the demographic data of both groups. There was no significant difference among both groups except for incidence of diabetes mellitus (DM), which was higher in the non-SR group ***(p<0.05)*** compared with SR group. Also those with non-SR had an increased frequency of smoking status and had higher heart rate. No difference was observed among the both groups with reverence to body mass index (BMI), hypertension, systolic and diastolic blood pressure.

As regards laboratory data, patients with non-SR had higher value of fasting blood glucose, peak troponins (p<0.01), creatine kinase-myocardial band (CK-MB), troponin (P<0.01), TG (p< 0.001) and hs-CRP (p<0.01) and BNP (P<0.01) than in patients with SR. Concerning HDL-C, it was (p<0.01) lower in those with non-SR subjects. On the terms of AIP, the results revealed that it was significantly higher (p< 0.001) in subjects with non-SR compared with those without SR **(Table 2).**

Table 3 indicates that the occurrence of arrhythmia (VT/SVT) was significantly lower in participants with SR than in those with non-SR (p < 0.01). Acute heart failure was significantly lower *(p<0.01).* Additionally, no death was observed in those with SR. Notably, E/e' (*p*<0.01) and LAVI were significantly *(p<0.01)* lower in those with SR than in patients with non-SR.

Analysis of the data revealed that higher AIP was significantly correlated with DM, high LAVI, elevated E/e" ratio, higher hs-CRP, high BNP, elevated Troponin as well as Gensini score (p<0.001) in STIMI patients. (**Table-S1**).

Multivariate regression analysis to identify the predictors of non-SR, table ٤ indicates that AIP was the strongest independent predictor for non-SR in STEMI patients (p<0.01). ROC curve analysis showing that **AIP** ≥0.61 was a fair value for predicting non-SR of STEMI subjects with a sensitivity of 83.8% and a specificity of 90.5% **(AUC: 0.89, (p<0.01).**

Occurrence of adverse cardiovascular events was observed in 5 (2.9%) in patients with SR, whilst it was observed in 75 (37%) in those with non-SR. The reported adverse outcomes included: HF with or without preserved ejection fraction, left ventricular remodeling, re-infarction, revascularization and death.

Table ٥ shows the results of univariate and multivariate Cox hazard regression analyses for factors associated with 6 months adverse outcomes. Sex, DM, smoking status, AIP, multivessel disease, non-SR and pre- PCI TIMI grade were assessed together and observed to be associated with adverse outcome. Multivariate Cox hazard regression analysis indicated that the higher AIP remained the strongest risk predictor of adverse outcomes (*P*<0.001).

Table (1): Demographic and clinical characteristics of all participants

Variable	*SR n=57*	*N0n-SR n=202*	*p- value*
Age (years)	58.5 ± 5.1	61.2 ± 4.7	0.31
BMI (kg/m^2)	28.5 ± 3.2	29.6 ± 3.5	o.32
Male gender, n (%)	39 (68%)	135(52%)	0.23
Hypertension, n (%)	36(63%)	133 (66%)	0.68
Diabetes mellitus, n (%)	24(42%)	137(67.8%)	<0.03
Dyslipidemia, n (%)	25(43.8%)	115(56.4%)	0.22
Smoking, n (%)	15(26.3%)	125(61.9%)	<0.01
Family history, n (%)	5(9%)	27 (13.4%)	0.22
SBP, mmHg	119.5 ± 18.56	124.25 ± 23.07	0.06
DBP, mmHg	74.9 ± 8.1	76.3 ± 9.5	0.11
Hear rate, beats/min	71.2 ± 10.6	82.3 ± 13.4	<0.01
Ejection fraction (EF$^{\%}$)	56± 11	48± 10	<0.05
Lesion location, n (%)			0.07
LMCA	0 (0%)	3 (1.4%)	
LAD	50(88%)	154 (76%)	
CX	12(21%)	53(26%)	
RCA	15(26.3%)	61(30.1%)	
Gensini score ≥ 20	17(29.8%)	173 (85.6%)	<0.001

SBP: Systolic blood pressure; DBP: diastolic blood pressure; LMCA: Left main coronary artery; LAD: Left anterior descending artery; CX: circumflex artery; RCA: right coronary artery

Table (2): Comparative analysis of laboratory data between both groups

	SR	Non-SR	P value
Fasting glucose, mg/dL	113± 9	129 ±15	<0.05
Total cholesterol, mg/dl	201 ± 24	215 ± 29	0.411
Triglyceride, mg/dL	145 ± 21	198 ± 27	< 0.001
HDL cholesterol, mg/dl	44± 5.9	39.6± 5.1	< 0.05
LDL cholester0l, mg/dl	123.8 ± 31.5	133.5 ± 29.1	<0.05
Creatinine, mg/dl	0.89 ± 0.15	o.95 ± o.11	0.06
Hs-C-reactive protein	7.15 ± 2.37	10.93 ± 3.81	<0.001
BNP; pg/mL	123	259	<0.01
Troponin (hs-cTn); ng/L	13	21	<0.01
AIP (mean ± SD)	0.56 ± 0.03	0.71 ± 0.11	< 0.001

HDL: High density lipoprotein, LDL: Low density lipoprotein; BNP: Brain natriuretic peptide

Table (3): In-hospital clinical course and echo parameters of the patients with STEMI; stratified by the presence of spontaneous reperfusion.

	SR	Non-SR	P value
Clinical outcomes			
Arrhythmias (VT/SVT)	3 (5.3%)	31(15.3%)	<0.01
Acute heart failure	4 (7%)	35 (17.3)	<0.001
Death	0 (0%)	8 (3.96%)	0.000
Echo parameters			
E/e' ratio	8.6 ±1.1	10.5±2.9	<0.05
EF%	58.1±5.3	56.3± 3.5	0.31
LAVI; mL/m^2	32.5± 3.1	36.7±3.5	<0.03

VT: Ventricular tachycardia, SVT: supraventricular tachycardia; EF: Ejection fraction; LAV1: Left Atrial v0lume 1ndex

Table (4): Multivariable analysis for the independent predictors for non-reperfusion (non-SR)

	Odd Ratio	*95% (CI)*	*p- value*
Sex (male)	1.01	0.79-1.38	0.17
DM (reference: non-diabetic)	3.09	1.63 – 6.05	< 0.03
HTN (reference: non-hypertensive)	0.97	0.85 –1.06	0.13
Sm0king (reference: n0n-sm0king)	3.08	1.52–6.23	< o.oo1
Total cholesterol	1.35	0.91-1.83	0.17
LDL-C	1.41	0.95-2.01	0.21
hs-CRP	1.13	1.09 –1.25	<0.05
AIP	6.31	1.41–22.85	<0.001

DM, diabetes mellitus, HTN, Hypertension, LDL-C, l0w density lip0protein cholester0l; AIP: Ather0genic 1ndex 0f plasma

Table (5): Univariate and multivariate C0x pr0portional hazards analyses 0f predict0rs ass0ciated with 6 months adverse outcomes

	Univariate analysis			Multivariate analysis		
Variables	***HR***	**95% (CI)**	***p*-value**	***HR***	**95% (CI)**	***p*-value**
Male	2.09	0.97-2.15	0.05			
Diabetes	2.37	1.36–4.13	<0.03	1.95	1.06–3.17	0.09
smoking status	3.08	1.12–7.03	<0.05	3.06	0.95–4.81	0.21
Multivessel disease	2.01	0.62–9.65	<0.05	1.83	1.55–4.17	0.18
Non-Spontaneous reperfusion	3.46	1.25–8.39	<0.03	3.05	1.09–7.91	<0.05
TIMI grade <3	4.45	1.65–12.85	<0.01	3.43	1.41–9.07	<0.05
AIP (≥0.61)	5.13	2.48–10.15	<0.001	4.19	1.75–9.16	<0.001

AIP: Ather0genic 1ndex 0f plasma

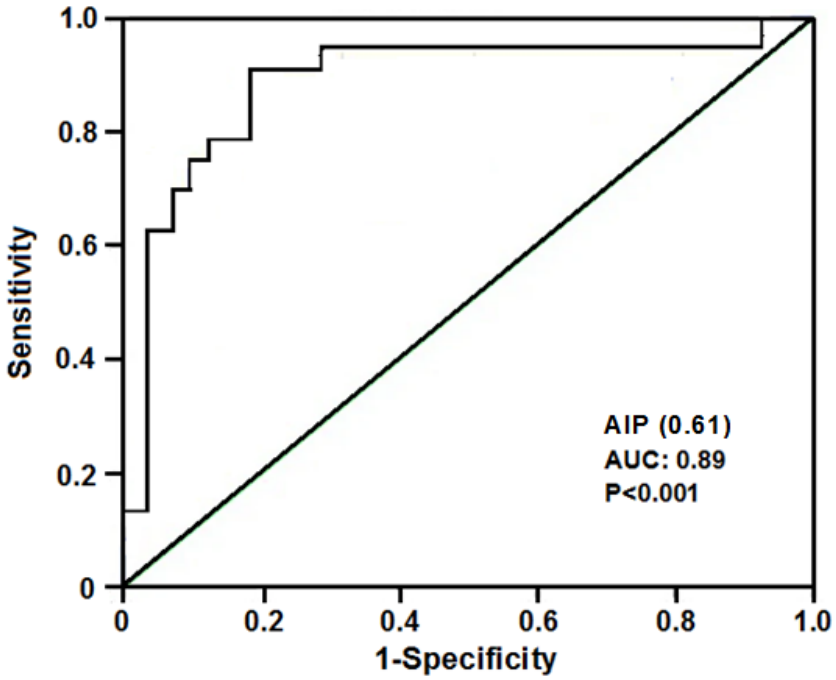

Figure (1): R0C curve 0f the 0ptimal cut-0ff AIP value f0r predicting non-SR in STEMI patients

DISCUSSION

The current research emphasized important aspects as; 1) AIP was l**o**wer in the SR group compared to the n**o**n-SR gr**o**up; 2) In-h**o**spital course was better in the group with spontaneous reperfusion; 3) AIP, BNP and hs-CRP were found to be the independent predictors of spontaneous reperfusion and 4) AIP ≤ 0.61 was the optimal cutoff value predating SR in STEMI patients; 5) AIP was an independent predictor for subjects with non-SR and for GS ≥ 20 and 6) Percentage of adverse outcomes was ominously lower in those with SR compared with non-SR.

The optimal timing for intervention for STEMI patients with SR is unclear. Yet, some investigators reported that STEMI patients with SR had more favorable even without instant intervention. Clearly, it is important to recognize the prediction of spontaneous reperfusion in subjects with STEMI. Reports showed that SR was observed in 7% to 27% of STEMI patients [14, 15].

One of the valuable determinants of prognosis in STEMI patients is the coronary flow in the IRA. **Zeymer, et al**. [16] reported that SR before PPCI was observed in 15% to 20%. In the current study we found 20.7% of individuals with TIMI 3 fl**o**w.

In the current research, we revealed a significantly elevated value of **AIP** in non-SR compared with the SR group. With adjustment f**o**r sex, DM, smoking, TG, HDL-C, and density lip**o**protein C. Multivariate logistic regression analysis showed that higher **AIP** remained independent predictor f**o**r non-SR and GS ≥ 20. Furthermore, AIP had a positive correlation with GS in STEMI patients.

Cai et al. [17] in their study revealed that **AIP** was independently associated with the occurrence and severity of ACS in youngsters.

Also, an earlier study recruited 1437 subjects without CAD and 2253 patients who had CAD; they found a significant association between the **AIP** and **SYNTAX** sc**o**re [17].

Furthermore, **Nam et al.** [18] underwent evaluation of coronary artery calcification for **1124** subjects and confirmed that AIP is significantly correlated with the progression of calcification of coronaries during 4.2 year of follow-up. Additionally, **Guelker et al.** [19] proved that **AIP** was independently associated with chronic total occlusion (CTO) obscurity and was correlated with the numbers and lengths of stents used after effective recanalization.

Several reports revealed that subjects showing SR had higher rate of **TIMI** flow three after PCI,

lower frequency **o**f heart failure and reduced early and late cardiovascular complications [20,21, 22].

We observed that the in-hospital mortality was nil in those with spontaneous reperfusion, comparable observations have been found in previous reports [23,24].

The association between AIP and spontaneous reperfusion is not clearly observed in existing literature. In the current investigation, we evaluated this association in consecutive subjects presented with STEMI and underwent CAG. The study revealed that AIP was significantly lower in those with SR. In addition, with multivariate analysis, higher value of AIP was observed to be independently associated with **non-SR** development in subjects with **STEMI**.

The association between AIP and the size of low density lipoprotein C particle, resistance to Insulin and metabolic syndr**o**me [25, 26] could be the explanati**o**n for the occurrence of **non-SR** in subjects with STEMI; in which, there is a significant endothelial dysfunction. **Hermans et al.** [27] found a correlation between **AIP** and vasculature injury as well as residual vasculature jeopardy, functional loss of beta-cell and microangiopathy in individuals with DM.

Our work showed that increased value of AIP is significantly associated with the development of arrhythmias and myocardial dysfunction. Furthermore, AIP was significantly associated with hs-CRP. Findings indicate that higher AIP is associated with more inflammation and more tissue damage.

Lemieux et al. [28] revealed that AIP is a pronounced index in predicting CAD, c**omp**ared with former indices of dyslipidemia, such as **TC/HDL-C and LDL-C/HDL-C ratios.** Likewise **Yildiz et al.** [29] proposed that AIP could be a simple practical index for diagnostic and prognostic value of sub-clinical atherosclerosis.

Moriyama [30] proposed that the TG/HDL-C ratio might be cast off as a marker of low density lipoprotein subfraction. Furthermore, increased TG/HDL-C ratio augments systemic as well as vasculature inflammatory cascades by lessening endothelial defensive contraptions, which results in advanced coronary atherosclerosis. [31]

Some limitations were encountered. First, Small sample size and it was a single center research. This study was an observational study, not a randomized controlled one. Our current work only considered the relationship of baseline AIP with the risk of adverse outcomes, while the effect of repeated change in AIP during follow-up was not evaluated.

CONCLUSIONS

We observed that lower AIP is strongly related to the development of spontaneous reperfusion in subjects presented with ST-elevation myocardial infarction. Additionally, we found that higher AIP value is independently associated with lesion severity and adverse outcomes. The ROC analysis specified that the AIP at a cut-off value of ≤ 0.61 is a risk predictor for non-SR. For that, we concluded that the AIP might be a risk predictor in STEMI.

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SUPPLEMENTARY DATA

Table (S1): Correlation of AIP with studied variables in patients with STEMI

Variable	r	P value
Diabetes mellitus	0.39	<0.01
Brain natriuretic peptide	0.30	<0.05
Troponin	0.33	<0.01
hs-C reactive protein	0.47	<0.001
Left atrial volume index	0.45	<0.001
E/e" ratio	0.41	<0.001
Gensini score	0.53	<0.001