Epicardial Pad of Fat as a Predictive of No-reflow following Primary PCI for STEMI and its Correlation with Mortality Risk using GRACE Score

Thesis

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بسم الله الرحمن الرحيم

قالوا

سبحانك لا علم لنا
إلا ما علمتنا إنك أنت
العليم الحكيم

صدق الله العظيم

سورة البقرة الآية: ٣٢

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Amr Salah

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List of Abbreviations

Abb.	Full term
ACEI	*Angiotensin converting enzyme inhibitor*
ACS	*Acute coronary syndrome*
AHA	*American heart association*
AMI	*Acute myocardial infarction*
BB	*B receptor blocker*
BMI	*Body mass index*
CABG	*Coronary artery bypass graft*
CAD	*Coronary artery disease*
CKD	*Chronic kidney disease*
DM	*Diabetes mellitus*
EAT	*Epicardial adipose tissue*
ESC	*European society of cardiology*
FH	*Family history*
GP IIb IIIa inhibitors	*Glycoprotein IIb IIIa inhibitor*
GRACE	*Global Registry of Acute coronary events*
HF	*Heart failure*
HR	*Heart rate*
HTN	*Hypertension*
IC	*Intracoronary*
IV	*Intravenous*
JVP	*Jugular venous pressure*
LV EF	*Left ventricular ejection fraction*
LV	*Left ventricle*
LVEDD	*Left ventricular end diastolic diameter*
LVESD	*Left ventricular end systolic diameter*

List of Abbreviations (Cont…)

Abb.	Full term
MACE	*Major adverse cardiac events*
MBG	*Myocardial blush grade*
MR	*Mitral regurgitation*
MS	*Metabolic syndrome*
MVD	*Multivessel disease*
NSTACS	*Non ST segment elevation acute coronary syndrome*
PCI	*Percutaneous coronary intervention*
PVC	*Pulmonary venous congestion*
ROC	*Recover operating characteristic curve*
SBP	*Systolic blood pressure*
STEMI	*ST segment elevation myocardial infarction*
TIMI	*Thrombolysis in Myocardial infarction*
TTE	*Transthoracic echocardiography*

ABSTRACT

This study was conducted on 113 patients who presented to the cardiology department of Ain Shams university hospital, with first acute anterior STEMI and underwent primary PCI. Patients admitted to the coronary care unit were analyzed thoroughly and data was recorded {Full history taking, clinical examination, 12 lead surface ECG, CK total and CK-MB, coronary angiographic details and transthoracic echocardiography in the first 48 hours}.Medical treatment of STEMI was given to subjects as per hospital protocol. All patients, during admission after AMI were subjected to echocardiographic evaluation of epicardial adipose tissue and LVEF. Patients were further divided into two groups using epicardial adipose tissue thickness of 5 mm as a cut off point, this number was derived from the ROC curve. **Group I**: Included patients EAT thickness less than 5 mms and it included 44 patients (38.9%). **Group II**: Included patients with EAT thickness greater than 5 mms and it included 69 patients (61.1%). Both groups were studied as regard epicardial adipose tissue thickness relation to the risk of no-reflow and to the mortality risk using GRACE score. Both groups were age and sex matched with homogenous risk factors for CAD. At univariate analysis for clinical data, group II (EAT > 5mm) showed statistically significantly increase in mortality risk using GRACE mortality risk, with no statistically significant difference as regard the location of infarction (anterior vs non anterior) BMI and pain to door time. Meanwhile for coronary angiographic data, univariate analysis showed statistically significant lower TIMI flow and statistically significantly greater multivessel affection in group II patients. The current study showed that epicardial fat thickness was significantly correlated with prognosis in patients with ACS. This suggests that echocardiographic epicardial fat thickness could be applied as predictive marker of prognosis in patients with ACS. EAT thickness determined on echocardiography may provide additional and substantial information on risk of no-reflow in STEMI patients treated with primary PCI.

Key words: Epicardial fat; no reflow.

INTRODUCTION

ST- elevation myocardial infarction is the most serious presentation of atherosclerotic coronary artery disease (ACAD) carrying the most hazardous consequences & patients with ST elevation are candidate for immediate reperfusion therapy [(1)].

Primary percutaneous coronary intervention (PCI) is considered the preferred reperfusion modality for patients presenting with ST-segment elevation myocardial infarction (STEMI) [(1)].

Severe Microvacular occlusion may manifest angiographically as reduced flow in the patent upstream epicardial arteries, a situation that is termed "no-reflow" or " slow flow" which is defined as less than TIMI III flow in absence of abrupt closure, high grade stenosis or flow limiting dissection and this event is associated with an increased infarct size, reduced recovery of ventricular function, further more this phenomenon is also linked to ventricular arrhythmia, early congestive heart failure or even cardiac rupture [(2)].

There are data on the relationship between epicardial adipose tissue thickness as a marker of microvascular disease and no-reflow [(3)].

Adipose tissue is known to affect the cardiovascular physiology via the release of active adipokines in paracrine fashion (via local release and diffusion) or endocrine (via the systemic circulation) manner [4].

Pericoronary EAT - released leptin promptes endothelial dysfunction, increased EAT thickness had been associated with coronary atherosclerosis progression and plaque vulnerability[5].

Epicardial fat is true visceral fat located within proximity of the myocarduim between the visceral pericardium and the myocarduim, and it shares the same blood supply as the adjacent myocarduim it also shows paracrine functions. This is the risky fat that is metabolically active[6].

AIM OF THE WORK

To demonstrate the relationship between epicardial adipose tissue (EAT) thickness and no-reflow phenomena in patients undergoing primary PCI for acute STEMI as a marker of microvascular disease and its correlation with the risk of mortality using GRACE score.

Chapter 1

CORONARY MICROCIRCULATION

I- Anatomy of coronary microcirculation

Coronary vasculature can be divided into two anatomically & functionally different segments. The extramural coronary vasculature consists of large conduit coronary arteries & veins running on the epicardial surface of the heart, whereas the intramural coronary vasculature consists of smaller coronary arteries, arterioles and capillaries along with their accompanying vein within the myocardium itself[(7)].

The intramural coronary circulation represents a complex vascular tree with a large number of branching orders and arborizations supplying the various vascular compartments of the myocardium. Ventricular branches with a perpendicular take off run vertically from the epicardium to the endocardium. Some of these branches reach the endocardium while others bifurcate at variable depths within the myocardium. In contrast, atrial branches ramify on the external surface without penetrating walls of the rather thin myocardium[(8)].

An important components of the intramural system are the arterial-arterial anastomoses, also termed coronary artery collateral vessels. These vessels link intramural branches originating either from the same coronary artery or from different coronary arteries. Also communications can be observed between intramural vessels and cardiac chambers[8].

Small coronary arteries (150-400 μm) run intramyocardially and are, similarly to epicardial vessels, characterized by a monolayer of endothelial cells embedded with the tunica intima, internal elastic lamina, and several layers of tunica media, external elastic lamina and tunica adventitia[9].

Coronary arterioles (< 150 μm) display a single layer of endothelial cells, two to three circular and concentric layers of smooth muscle cells and an outer adventitia[9].

Myocardial capillaries (5-10 μm) are thin-walled structures containing a single layer of endothelial cells frequently associated with pericytes. Capillaries run parallel to myocardial fibers; usually two capillaries can be observed surrounding a single myocardial fiber. This symmetrical pattern is similar to the capillary networks observed in the striated muscles [9].

II- Physiology of coronary microcirculation

Flow across the myocardium depends on the *pressure gradient* between the aortic root and the right atrium and the *resistance* of the coronary vasculature. Under normal conditions, the driving pressure is fully maintained along the epicardial conduit vessels with little if any pressure loss in the