Effect of Chronic Pretreatment with β-Blocker on No-Reflow Phenomenon in Diabetic Patients with Acute ST-Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention

Thesis Submitted as Partial Fulfillment for Degree of Master of Cardiology

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بسم الله الرَّحمن الرَّحيم

صَدَقَ اللهُ العَظيم

(سورة طه : من الآية 114)

This work is dedicated

To

The Soul of my Mother and Father

Acknowledgement

The ability to achieve this work is attributed first and foremost to Allah, the Gracious, who helped me to accomplish this endeavor, and for granting me with a teamwork that, without their magnificent effort, this work would have never been brought to light.

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Ali Jabari

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List of Abbreviations

AMI	Acute Myocardial Infarction
CABG	Coronary Artery Bypass Grafting.
CAD	Coronary Artery Disease
CHF	Congestive Heart Failure
CE-MRI	Contrast-Enhanced Magnetic Resonance Imaging
COPD	Chronic Obstructive Pulmonary Diseases
cTnI	Cardiac Troponin I
cTnT	Cardiac Troponin T
CVD	Cardiovascular Disease
CXR	Chest X-Ray
EPD	Distal Embolic Protection Devices
DM	Diabetes Mellitus
ECG	Electrocardiography
GPIIb-IIIa	Glycoprotein IIb/IIIa
HDL	High-Density Lipoprotein.
IRA	Infarct-Related Artery
LDL	Low-Density Lipoprotein
LV	Left Ventricle.
LBBB	Left Bundle-Branch Block
MCE	Myocardial Contrast Echocardiography
MI	Myocardial Infarction.
MVO	Microvascular obstruction
PCI	Percutaneous coronary intervention
RAAS	Renin-Angiotensin-Aldosterone System
STEMI	ST Segment Elevation Myocardial Infarction
TIMI	Thrombolysis In Myocardial Infarction
VF	Ventricular Fibrillation

Introduction

Primary percutaneous coronary intervention (PCI) in patients with ST-elevation myocardial infarction (STEMI) has been used as an important therapeutic method since the last decade of the 20th century and has gradually become the method of choice in many medical centers. Various studies have shown that primary PCI is associated with lower rates of mortality, reinfarction and cerebral hemorrhage in comparison with thrombolytic treatments.[1]

Although shown to be extremely important in maintaining epicardial artery patency in acute myocardial infarction (AMI), the attention has shifted recently from epicardial artery patency to the status of the microvasculature.[2]

Previous studies have shown that 5–30% of patients treated with primary PCI fail to achieve thrombolysis in myocardial infarction (TIMI) flow grade 3 after successful opening of the artery without angiographic evidence of the mechanical obstruction. This phenomenon is deemed as no-reflow, which determines the prognosis in patients after AMI.[3]

Several mechanisms responsible for no-reflow have been identified in experimental models, including extravascular compression, microvascular vasoconstriction, and platelet/leukocyte capillary plugging.[4]

Previous evidence suggests that Beta blockers have multiple favorable effects on the vascular system not directly related to their effect on blood pressure.[5]

Clinically, no-reflow is important as it predicts a poorer outcome and is associated with ongoing symptoms and persistent electrocardiographic (ECG) changes. In comparison to patients attaining TIMI 3 flow, patients with no-reflow have an increased incidence of ventricular arrhythmias, early congestive cardiac failure, cardiac rupture and cardiac death. As such, it is of paramount importance to consider strategies to prevent the occurrence of no-reflow phenomenon.[6-8]

However, to the best of our knowledge, there is insufficient data regarding the effects of prior Beta blocker use on coronary blood flow after primary PCI in patients with AMI.

Aim of the study

The aim of this study is to test the hypothesis that Beta blocker treatment before admission would have beneficial effects on the development of the no-reflow phenomenon after acute myocardial infarction.

Review of Literature

Chapter 1

ST-segment Elevation Myocardial Infarction

Definition

Myocardial infarction, from pathologic point of view, is myocyte cell death, usually due to prolonged myocardial ischemia.[9]

STEMI is a clinical syndrome defined by characteristic symptoms of myocardial ischemia in association with persistent electrocardiographic ST elevation and subsequent release of biomarkers of myocardial necrosis. Diagnostic ST elevation in the absence of left ventricular (LV) hypertrophy or left bundle-branch block (LBBB) is defined by the European Society of Cardiology/ACCF/AHA/World Heart Federation Task Force for the Universal Definition of Myocardial Infarction as new ST elevation at the J point in at least 2 contiguous leads of ≥2 mm (0.2 mV) in men or ≥1.5 mm (0.15 mV) in women in leads V2–V3 and/or of ≥1 mm (0.1 mV) in other contiguous chest leads or the limb leads. The majority of patients will evolve ECG evidence of Q-wave infarction. New or presumably new LBBB has been considered a STEMI equivalent.[10]

It is a life threatening event and a true medical emergency. The risk of morbidity and mortality associated with STEMI increases