بسم الله الرحمن الرحيم

MONA MAGHRABY

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

MONA MAGHRABY

شبكة المعلومات الجامعية

التوثيق الإلكتروني والميكروفيلم

جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأقراص المدمجة قد أعدت دون أية تغيرات

يجب أن

تحفظ هذه الأقراص المدمجة بعيدا عن الغبار

MONA MAGHRABY

The Immediate and Short term Outcomes of Patients with ST Elevation Myocardial Infarction with High Thrombus Burden receiving Intracoronary Verapamil versus Epinephrine during Primary Percutaneous Coronary Intervention

Thesis

Submitted for partial fulfillment of Master Degree in Cardiology

By

Ahmed Rafek Mohamed Fouad Al Ghazawy
M.B. B.CH, Ain Shams University

Under supervision of

Prof. Dr. Mohamed El Sayed Zahran
Assistant Professor of Cardiology
Faculty of Medicine - Ain Shams University

Dr. Mohamed Saber Hafez
Lecturer of Cardiology
Faculty of Medicine - Ain Shams University

Faculty of Medicine
Ain Shams University
2021

سورة البقرة الآية: ٣٢

Acknowledgment

First and foremost, I feel always indebted to **ALLAH,**
the Most Kind and Most Merciful.

I'd like to express my respectful thanks and profound gratitude to **Prof. Dr. Mohamed El Sayed Zahran,** *Assistant Professor of Cardiology, Faculty of Medicine - Ain Shams University for his keen guidance, kind supervision, valuable advice and continuous encouragement, which made possible the completion of this work.*

I am also delighted to express my deepest gratitude and thanks to **Dr. Mohamed Saber Hafez,** *Lecturer of Cardiology, Faculty of Medicine - Ain Shams University, for his kind care, continuous supervision, valuable instructions, constant help and great assistance throughout this work.*

Ahmed Rafek

List of Contents

List of Abbreviations

Abb.	Full term
2D	Two dimensional
ACS	Acute coronary syndrome
ARBs	Angiotensin receptor blockers
CABG	Coronary artery Bypass Grafting
CCBs	Calcium channel blockers
CCU	Coronary care unit
CFR	Coronary flow reserve
CMR	Cardiac magnetic resonance
CNR	Coronary no reflow
CVD	Cardiovascular disease
DAPT	Dual antiplatelet therapy
DES	Drug eluting stents
ECG	Electrocardiography
EMS	Emergency medical services
FFR	Fractional flow reserve
GP	Glycoprotein
HTN	Hypertension
IMR	index of microcirculatory resistance
IVUS	Intravascular ultrasound
LAD	Left anterior descending
LAO	Left anterior oblique
LCX	Left circumflex artery
LGE	Late gadolinium enhancement
LVEF	Left ventricular Ejection fraction
MACE	Major adverse cardiac events
MBG	Myocardial blush grade
MI	Myocardial infarction
MRI	Magnetic resonance imaging
MVO	Microvascular obstruction

List of Abbreviations Cont...

Abb.	Full term
NSTEMI	Non-ST-segment elevation MI
OM	Obtuse marginal
p.o.	Per os
PCI	Percutaneous Coronary Intervention
PET	Positron emission tomography
PPCI	Primary percutaneous coronary intervention
RCA	Right coronary artery
SBP	Systolic blood pressure
SCAD	Spontaneous coronary artery dissection
SPECT	Single-photon emission computed tomography
STEMI	ST elevation myocardial infarction
TFG	TIMI flow grade
TIMI	Thrombolysis in Myocardial Infarction
TMPG	TIMI myocardial perfusion grade
UFH	Unfractionated heparin

List of Tables

Table No.	Title	Page No.

List of Figures

INTRODUCTION

ST elevation myocardial infarction is causes by total thrombotic coronary artery occlusion, most of our treatment strategies focused on epicardial coronary arteries yet little interest was given to microvascular occlusion and its consequences. When a coronary artery is occluded, detrimental changes occur in the cardiac capillaries and arterioles. After relief of the occlusion, blood flow to the ischemic tissue may still be impeded, a phenomenon known as no reflow. This study attempts to provide an in-depth understanding of this phenomenon from the laboratory bench to the clinical arena and different solutions attempted at reversing it.

Several research were performed on the coronary circulation specifically on dogs, dogs were subjected to 40 or 90 minutes of proximal coronary artery occlusion. When the coronary occlusion was relieved after 40 minutes of occlusion, the blood flow was restored to the damaged myocardium as assessed by markers of perfusion such as thioflavin S and carbon black. However, after 90 minutes of coronary occlusion, there was only partial restoration of blood flow to the myocardial tissue, despite virtual elimination of the coronary occlusion. Anatomic perfusion defects were prominent in the subendocardial myocardium when thioflavin S or carbon black was injected into the vasculature after restoration of epicardial coronary flow. Electron microscopic examination of the cardiac

microvasculature within the anatomic no-reflow zones revealed significant capillary damage in the form of swollen endothelium and intraluminal endothelial protrusions and, less commonly, intraluminal platelets and fibrin thrombi. These changes, coupled with interstitial and myocardial cellular edema, could compress the capillaries and be responsible for the no-reflow phenomenon. The longer ischemia lasts, the more likely the no-reflow phenomenon is to occur. Microvascular damage did not appear to be the primary cause of myocardial cell damage because the no-reflow area appeared to be confined to areas of tissue that were already necrotic ***(Reimer et al., 2007).***

The no-reflow phenomenon is becoming increasingly recognized because of the spread of primary intervention for acute myocardial infarction and the emergence of contrast myocardial echocardiography. With the clinician focusing on both epicardial coronary arteries and the microvasculature, there is a need for a safe and effective treatment for no reflow. After prolonged cessation of coronary occlusion and restoration of blood flow to the epicardial coronary arteries, there is sufficient structural damage to the microvasculature to prevent restoration of normal blood flow to the cardiac myocytes. This may lead to inadequate healing of the cardiac scar. In addition, it may prevent the development of future collateral flow ***(Reimer et al., 2007).***

Treating no reflow may not necessarily reduce the size of myocardial infarction because the microvascular damage is usually confined well within the zone of myocardial necrosis. However, treating no reflow may enhance the delivery of blood and blood-borne elements to the necrotic area, thus speeding healing ***(Agati et al., 2001).***

Various agents have been used in management of coronary no reflow with controversial result in different studies, the most used is adenosine and verapamil, other agents also have been tried as nicorandil, sodium nitroprusside, nitroglycerine, and adrenaline and no agent of choice yet favorable in restoring the microcirculation ***(Nazir et al., 2016).***

AIM OF THE STUDY

This study is a single center randomized controlled trial designed to compare between the standard treatment strategy with two other strategies, one is adrenaline and the other is verapamil regarding the immediate and short term outcomes in patients presenting with ST elevation myocardial infarction (STEMI) with high thrombus burden during primary percutaneous coronary intervention (PPCI).