بسم الله الرحمن الرحيم

MONA MAGHRABY

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

MONA MAGHRABY

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

Role of Colchicine in Reduction of No Reflow Phenomenon in Patients with Acute ST-Segment Myocardial Infarction, Undergoing Primary Percutaneous Coronary Intervention

.

Thesis

Submitted for Partial Fulfillment of
Master's Degree in **Cardiology**

By
Nardine Youhanna Lotfy Sadek
M.B.B.Ch. Faculty of Medicine, Ain Shams University

Under Supervision of

Prof. Dr. Ahmed Abd Elrahman
Professor of Cardiology
Faculty of Medicine, Ain Shams University

Dr. Viola William
Consultant of Cardiology
Faculty of Medicine, Ain Shams University

Dr. Khalid Karem
Lecturer of Cardiology
Faculty of Medicine, Ain Shams University

Faculty of Medicine - Ain Shams University
Cairo - Egypt – 2021

Acknowledgments

List of Contents

Title	Page No.

List of Tables

i

List of Figures

List of Abbreviations

Abb.	Full term
ALCAPA	Anomalous origin of the left coronary artery from the pulmonary artery
CAD	Coronary artery disease
CBC	Complete blood picture
DM	Diabetes Mellitus
ECG	Electrocardiogram
ESRD	End stage renal disease
FH	Family history
FMF	Familial Mediterranean fever
HTN	Hypertension
IC	Intracoronary
INR	International normalized ratio
IS	Infarct size
IV	Intravenous
MBG	Myocardial blush grade
MI	Myocardial infarction
MVO	Micro vascular occlusion
PTD	Pain to door
PCI	Percutaneous coronary intervention
PPCI	Primary percutaneous coronary intervention
SD	Standard deviation
SPSS	Statistical Package for the Social Science
STEMI	ST-segment elevation myocardial infarction
TIMI	Thrombolysis in myocardial infarction risk score

INTRODUCTION

Acute myocardial infarction is the most severe manifestation of coronary artery disease, which causes more than 2.4 million deaths in the USA, more than 4 million deaths in Europe and northern Asia, and more than a third of deaths in developed nations annually [1-2]. Primary PCI is the treatment of choice for acute coronary syndrome with STEMI[3]. The reopening of the culprit coronary artery, however, does not necessarily translate into improved tissue perfusion, despite imaging evidence that the target stenosis was adequately removed or bypassed. This phenomenon is known as **No-Reflow.**

Inflammatory Process is the main pathway of the deleterious effects of the ischemia/reperfusion injuries in ST-segment Elevation Myocardial Infarction patients.

Despite that, only a few studies started to tackle this major problem including the effects of anti-inflammatory treatment in these patients. Maybe this is because of the lack of anti-inflammatory agents without the adverse cardiovascular safety profile of corticosteroids and non-steroidal anti-inflammatory drugs.

Colchicine is a substance with potent anti-inflammatory properties, having a unique mechanism of action, which allows for safe use in patients with cardiovascular disease.

Colchicine is an inexpensive, orally-administered, potent anti-inflammatory medication. Its mechanism of action is through the inhibition of microtubule generation and, possibly, has effects on cellular adhesion molecules, inflammatory chemokines, and the inflammasome [4-5-6]. This anti-inflammatory effect of colchicine has been recently shown to improve cardiovascular outcomes in patients with STEMI, at a daily dose of 0.5mg [7].

AIM OF THE WORK

The aim of this work is to study the efficacy of colchicine administration as a preventive tool for No-reflow phenomenon in patients presented with acute STEMI undergoing primary PCI.

Chapter 1

ST-ELEVATION MYOCARDIAL INFARCTION (STEMI)

Myocardial infarction (MI) is a clinical entity involving myocardial ischemia that manifests with ECG changes and chest pain [8]. The current 2018 clinical definition of MI is based on the confirmation of the myocardial ischemic injury with abnormal cardiac biomarkers [9]. An acute ST-elevation myocardial infarction (STEMI) entails transmural myocardial ischemia that results in myocardial injury or necrosis [10].

Etiology

An ST-elevation myocardial infarction occurs from occlusion of one or more of the coronary arteries that supply the heart with blood. This abrupt disruption of blood flow is a result of an obstructing thrombus on top of plaque rupture, erosion, fissuring, or dissection of coronary arteries.

Dyslipidaemia, diabetes mellitus, hypertension, smoking, and a family history of coronary artery disease are the major risk factors for STEMI [11-12].

Epidemiology

In the United States, the estimated annual incidence of MI is 550,000 new and 200,000 recurrent patients. In 2013, a fatal MI was diagnosed in 116,793 persons in the United States

with 57% occurring in men and 43% in women. The average age of incidence of a first MI is 65.1 years for men and 72 years for women. The ST-elevation myocardial infarction represents 38% of acute coronary syndrome [13].

According to the latest WHO data published in 2018 Coronary Heart Disease Deaths in Egypt reached 163,171 or 29.38% of total deaths [14].

Pathophysiology

For an acute thrombotic coronary event to cause ST-segment elevation on a surface ECG, there should be a complete and persistent occlusion of blood flow in the culprit coronary vessel.

Sudden onset plaque rupture can occur on top of Coronary atherosclerosis in the presence of high-risk thin cap fibroatheroma (TCFA) [15]. This results in changes in vascular endothelium resulting in a cascade of platelet stimulation including adhesion, activation, and aggregation resulting in thrombus formation [16].

History-Taking and Physical Examination

Prior to performing an ECG and collecting troponins the initial history and physical examination provide the only clues that lead to a diagnosis of myocardial infarction. Information should be collected about the characteristics of the pain and associated symptoms, risk factors or history of cardiovascular disease, and

recent drug therapy [17]. Age, gender, family history of premature coronary artery disease, tobacco use, dyslipidemia, diabetes mellitus, hypertension, abdominal obesity, sedentary lifestyle, a diet low in fruits and vegetables, psychosocial stressors are risk factors for an ST-elevation myocardial infarction [18]. Cocaine use can cause an ST-elevation myocardial infarction regardless of risk factors [19]. History of congenital abnormalities as anomalous origin of the left coronary artery from the pulmonary artery (ALCAPA) syndrome may be suggestive of the cause [20-21].

Evaluation

Evaluation of patients with acute chest pain should begin with an electrocardiogram (ECG) and troponin level. The American College of Cardiology, American Heart Association, European Society of Cardiology, and the World Heart Federation committee established the ECG criteria for ST-elevation myocardial infarction (STEMI) (Table 1) [9].

Table 1: ECG criteria for ST-elevation myocardial infarction [9]:

▪ New ST-segment elevation at the J point in 2 contiguous leads with the cut-off point as greater than 0.1 mV in all leads other than V2 or V3
▪ In leads V2-V3 the cut-off point is greater than 0.2 mV in men older than 40 years old and greater than 0.25 in men younger than 40 years old, or greater than 0.15 mV in women
✓ Patients with a pre-existing left bundle branch block can be further evaluated using Sugarbush's criteria ✓ ST-segment elevation of 1 mm or more that is concordant with (in the same direction as) the QRS complex