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بسم الله الرحمن الرحيم

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

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جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

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بالرسالة صفحات لم ترد بالاصل

CORRELATION OF POST INFARCTION MYOCARDIAL BLUSH GRADE WITH ST-SEGMENT PATTERNS AND ECHOCARDIOGRAPHIC FINDINGS

Thesis

Submitted to the Faculty of Medicine
University of Alexandria
In Partial Fulfillment of the Requirements
of the Degree of

Master of

Cardiology and Angiology

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2002

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ACKNOWLEDGEMENTS

All praise to God, master of world, the most gracious, the most merciful, the fount of all wisdom.

I would like to express my deepest gratitude and appreciation to ***Prof. Dr. Abdel Fattah El Sayed Kholeif,*** *Professor of Medicine and Cardiology for his meticulous supervision, for the whole aspects of the thesis. He had offered a lot of concern and indispensable advice, enlightening the points and paving the tracks of thoughts through the whole work.*

I am deeply indebted to ***Prof. Dr. Mohamed Ahmed Sobhi,*** *Professor of Cardiology and Angiology, for his precious effort and time spent in guidance and supervision all though this work and the golden chance he offered me by being one of this great team work.*

I am thankful and grateful for ***Dr. Magdy Abdel Moneim Rashwan****, Assistant Professor of Cardiology and Angiology for his active participation and guidance for step by step supervision and for giving me unlimited time, effort and precious advice which make this work possible.*

I would like to extend my gratitude and appreciation to Professor ***Dr. Ebtihage Hamdy,*** *head of Cardiology unit and to all members of the staff of the cardiology unit., Faculty of Medicine, Alexandria University, for their moral and educational support.*

Finally, I want to express my gratitude and deepest thanks to my parents, my sisters and my friends who gave me love and unlimited moral support.

CONTENTS

ABBREVIATIONS

AMI:	Acute Myocardial Infarction
MI :	Myocardial Infarction
CK:	Creatine – Kinase.
ECG:	Electrocardiogram
DSE:	Dobutamine Stress Echocardiography
IRA:	Infarct-Related Artery
MCE:	Myocardial Contrast Echocardiography
LV:	Left Ventricle
EF:	Ejection Fraction
PTCA:	Percutaneous Transluminal Coronary Angioplasty
TIMI:	Thrombolysis In Myocardial Infarction.
TMP:	TIMI Myocardial Perfusion.
BPM:	Beat Per Minute
LAD:	Left Anterior Descending
RCA:	Right Coronary Artery
OM:	Obtuse Marginal
CX:	Circumflex
CABG	Coronary Artery Bypass Graft
PCI	Percutaneous Coronary Intervention

Introduction

INTRODUCTION

Despite impressive strides in diagnosis and management over the last three decades, acute myocardial infarction (AMI) continues to be a major public health problem in industrialized world.[1]

A steady decline in the mortality rate from AMI has been observed across several population groups since 1960.[2-4] This drop in mortality appears to be caused by a fall in the incidence of AMI (replaced in a part by an increase in the rate of unstable angina)[5-6], and a fall in the case fatality rate once a myocardial infarction has occurred. In addition, clinicians are now more alert at identifying those patients who are at increased risk of AMI and benefit from more aggressive prophylactic cardiovascular treatments.[7]

Coronary blood flow and its regulation:

The major coronary arteries and their principle branches course across the epicardial surface of the heart. They serve as conductance vessels and normally offer little resistance to coronary blood flow. These vessels give rise to smaller penetrating vessels approximately at right angles. A large pressure drop occurs in these intramural vessels and in the coronary arteries, hence their designation as "resistance vessels". The dense network of about 4000 capillaries per square millimeter is not uniformly patent because precapillary sphincters appear to serve a regulatory function[8], in accordance with the flow needs of the myocardium. This capillary density is reduced in the presence of ventricular hypertrophy.

As in any vascular bed, blood flow in the coronary bed depends on the driving pressure and the resistance offered by this bed. Coronary vascular resistance, in turn, is regulated by several control mechanisms include: myocardial metabolism (metabolic control), endothelial (and other humoral) control, autoregulation, myogenic control, extravascular compressive forces, and neural control. These individual control mechanisms may be impaired in a variety of conditions and contribute to the development of myocardial ischemia.[9]

Microvascular dysfunction

Pathophysiology:

Most of myocardial infarctions result from coronary atherosclerosis, generally with super imposed coronary thrombosis.[10] The atherosclerotic plaques that are associated with thrombosis and total occlusion, located in the infarct-related vessels are generally more complex and irregular than those in vessels not associated with MI.[11]

After plaque rupture and intracoronary thrombus formation, ischemia causes ultrastructural damage to myocytes and the coronary microcirculation soon after coronary occlusion.[12] Once epicardial reperfusion occurs and blood flow to the infarct zone is restored, reperfusion injury caused by neutrophil infiltration, generation of oxygen free radicals and activation of the complement system and adhesion molecules may further damage the microcirculation.[13-15]

Damaged myocytes and arterioles are thought to hinder microvascular flow by increasing distal vascular resistance, stimulating arteriolar spasm and causing endothelial dysfunction. In addition, platelet microemboli are thought to be "showed" downstream of the micro circulation after plaque rupture, causing microvascular obstruction that further limit tissue perfusion once the epicardial infract vessel in recanalized.[16] Microvascular dysfunction also appears to occur in non-infarct related vessels, suggesting that myocardial ischemia may stimulate a global inflammatory response through the release of cytokines.[17] Thus, microvascular dysfunction after epicardial reperfusions is a complex process with several probable interrelating stimuli and factors. (Fig. 1).

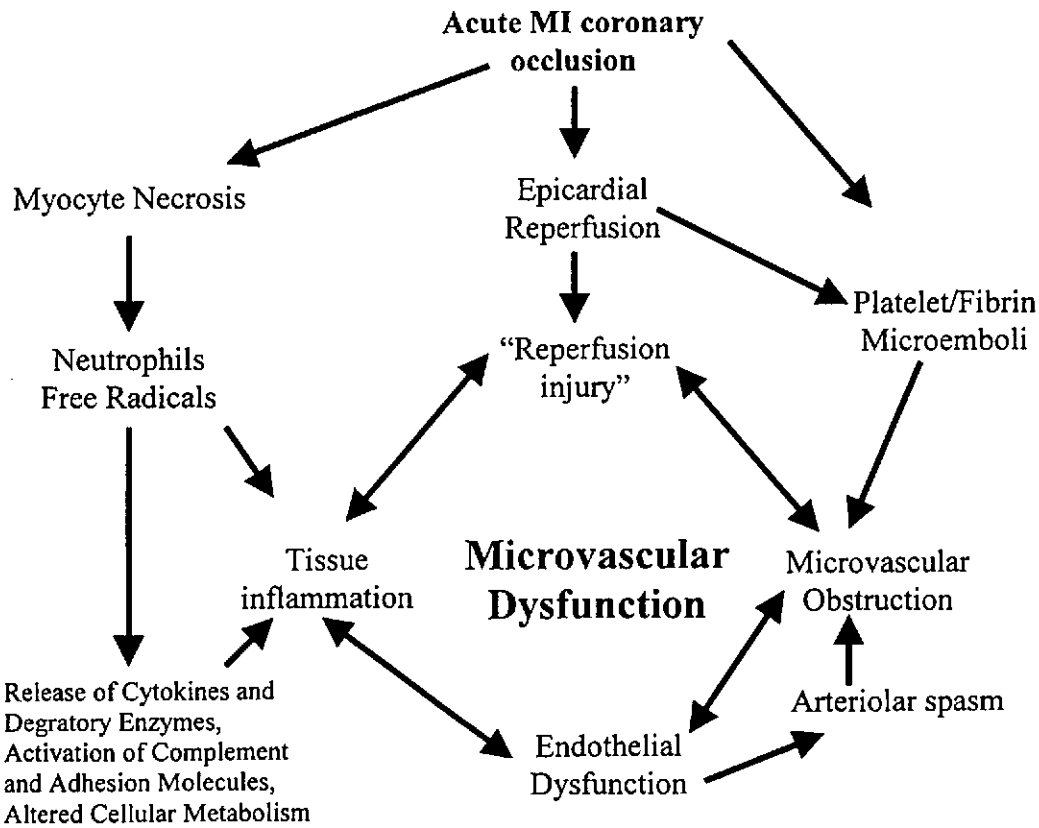

Figure 1: Pathophysiology of microvascular dysfunction after epicardial perfusion.[18]