بسم الله الرحمن الرحيم

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CORONARY ANGIOGRAPHY FINDINGS IN Q-WAVE VERSUS NON-Q-WAVE MYOCARDIAL INFARCTION

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BY

MOHAMMED ABD EL-AZIZ ISMAIL AFIFY

M.B., B.Ch.

UNDER SUPERVISION OF

DR. OSAMA SANAD ARFA
ASSIST. PROF. OF CARDIOLOGY

DR. SAAD MAHMOUD AMMAR
ASSIST. PROF. OF CARDIOLOGY

DR. MOHAMMED AHMED HAMOUDA
LECTURER OF CARDIOLOGY

BENHA FACULTY OF MEDICINE
ZAGAZEG UNIVERSITY

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CONTENTS

INTRODUCTION AND AIM OF THE WORK

The stratification of patients with myocardial infarction (MI) into electrocardiography (ECG) subsets based on the presence or abscence of abnormal Q-wave has important clinical and prognostic utility. Previous studies showed the low incindence of early mortality between non-Q-wave myocardial infarction (NQWMI) but it carries increased risk of recurrent ischemia or reinfarction or even sudden death. Indeed NQWMI late mortality may equal or exceed that of Q wave MI patients (QWMI). *(Gibson , 1988)*

However, pathophysiological insights gained from autopsy and angiographic studies are very limited. Some controversy exists among the fewer trails between the similarity or the difference of outcome between Q wave and non Q wave MI patients *(DeWood et al., 1986).* So, the coronary angiography and left ventriculography appeared as important tool in identification of risk factors between coronary heart diseased (CHD) patients *(GUSTO, 1993)*

The present work aims to identify coronary lesion anatomy and left ventricular function among Q-wave versus non-Q-wave myocardial infarction patients using angiographic studies to gain further insight into pathoanatomy of these two ECG subsets of MI to help in development of treatment strategy.

Pathogenesis of acute myocardial infarction

Myocardial infarction, generally, occurs secondary to an abrupt decrease in coronary blood flow following a thrombotic occlusion of narrowed atherosclerotic artery. ***(Fuster et al.,1992)***

The fact that atherosclerotic-plaque disruption leads to thrombus formation has been known for many years. Pathological, angiographic and angioscopic studies have clearly established on association between primary plaque fissuring or ulceration and the development of acute myocardial infarction or sudden ischemic death. ***(Falk , 1985; Richardson et al., 1989; Davies et al., 1989)***

There are important contributors to plaque disruption: disruption of small atherosclerotic plaques, the presence of lipid-rich plaques, the activity of macrophages and the effect of stressors on the vessel wall in plaque disruption **(Fig., 1)** . ***(Fuster et al, 1992)***

Small plaque disruption

The severity of coronary-artery stenosis and the number of diseased vessels are markers for future cardiac morbidity and mortality ***(Moise et al., 1984).***

Lipid- rich plaque and plaque disruption

Some pathological studies revealed that the relatively small atherosclerotic plaques are commonly composed of a cresentric mass of lipids, separated from the vessel lumen by fibrous cap. ***(Stary, 1989; Richrdson et al., 1989)***

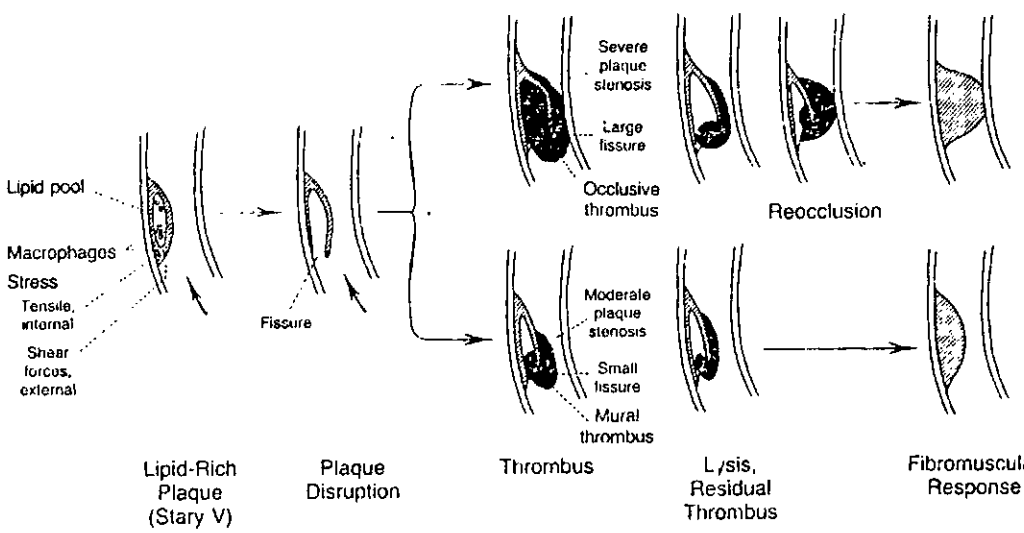

Figure (1): Typical dynamic evolution of the complicated plaque.

Indeed, plaques that undergo disruption tend to be relatively small and soft with a high concentration of cholesterol and its esters ***(Richardson et al., 1989).***

Increased shear forces in the area of stenosis sudden changes in intraluminal coronary pressure or tone and bending and twisting of an artery during each heart contraction may contribute to the disruption of these plaques ***(Nobuyoshi et al., 1991).***

Macrophage and plaque disruption

The earliest lesions in human atherosclerosis are composed of macrophages or foam cells which originate in circulating monocytes, lipid-laden smooth muscle cells and scattered or confluent extracellular lipid ***(Stary , 1989)***

With age, some of these lesions become fibromuscular, whereas others become fibrolipid lesions rich in macrophages and prone to disruption ***(Stary , 1989).***

The role of macrophages in atherosclerosis has been reviewed by ***Stienberg et al.(1989) & Schwartz et al.(1991).***

Macrophages participate in the uptake and metabolism of lipids. They, also, enhance the transport and oxidation of low-density lipoprotien cholesterol.

Macrophages facilitate the secretion of nitrogenic factor that lead to the proliferate of smooth muscle cells.In addition, macrophages generate the toxic products (Free radicals, products of lipid oxidation) that facilitate endothelial damage ***(Stienberg et al., 1989).***

Furthermore, macrophages can release protease (elastase and collagenase) that digest the extracellular matrix and contribute to the disruption of the plaque ***(Richardson et al., 1989).*** Indeed, macrophages can enhance local thrombogenesis by releasing tissue factors and plasminogen activator inhibitor-1 ***(Drake et al., 1989)***

Vessel wall stressors and plaque disruption

Alterations in stressors on or within plaques may be important in their disruption.

Davies and coworkers (1989) analyzed atherosclerotic plaques in patients who died with ischemic heart disease. They found that fissured plaques contained a lipid pool in the intima and occurred at the junction of the fibrous cap with adjacent normal arterial wall. They suggested that in this area, the cap's lack of underlying collagen support may make it more susceptible for rupture.

A side from stressors within the plaque, stress produced by a disturbed pattern of blood flow in areas of stenosis may be contribute to tearing of the plaque in thinner portion of the fibrous cap. ***(Richardson et al., 1989)***

If the plaque disruption is major with extensive exposure of collagen and athermatous core content to the lumen, this may lead to immediately to occlusive thrombosis, with infarction or sudden cardiac death. ***(Nobuyshi et al., 1991)***

Alternatively, the minor disruption (more common), is leading to non-occlusive thrombosis and the patient may have not symptoms or may have unstable angina or non-Q-wave myocardial infarction.***(Davies & Thomas ,1984)***

The lesion may gradually heal, with proliferation of smooth muscle cells and greater degree of stenosis. On the other hand, further increase in coagulability and vasoconstriction after thromboxan A_2 release which promote a conformational changes in glycoprotein IIbIIIa receptors. These receptors develop a high affinity for the sequence arginine-glycine-aspartaic acid on fibrinogen alpha chain and for the sequence of dodecapeptide on fibrinogen gamma chain.

Fibrinogen is a multivalent molecule produce platelet cross-linking and aggregation which may lead to occlusive thrombosis, infarction and sudden cardiac death. ***(Fuster et al., 1992; Falk et al., 1995).***

So, the characteristic of vulnerable plaque include high lipid content, a thin fibrous cap, a reduced number of smooth muscle cells, and increased macrophages activity with elaboration of metalloproteinaseas ***(Davies et al,1976; Willerson et al., 1984)***

HMG coenzyme A-reductase inhibitors may be able to stabilize plaque, thereby returning a previously vulnerable plaque to less vulnerable state ***(Brown et al., 1993).***

The risk factors for the development of atherosclerosis, such as hypercholesterolemia, cigarette smoking, diabetes mellitus, hypertension, family history and male gender, are well defined. However, the precipitating factors for myocardial infarction are not much less well understood.

Besides the customary thrombotic occlusion of coronary artery, coronary spasm ***(Lambert and Pepine, 1989),*** coronary embolism ***(Herzog et al., 1991),*** primary dissection of coronary artery ***(Bulkley and Roberts, 1973),*** and drugs such as cocaine ***(Foussas et al., 1989)*** are