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لم ترد بالاصل

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

A THESIS
SUBMITTED IN PARTIAL FULFILLMENT OF
MASTER DEGREE IN CARDIOLOGY

BY

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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)***

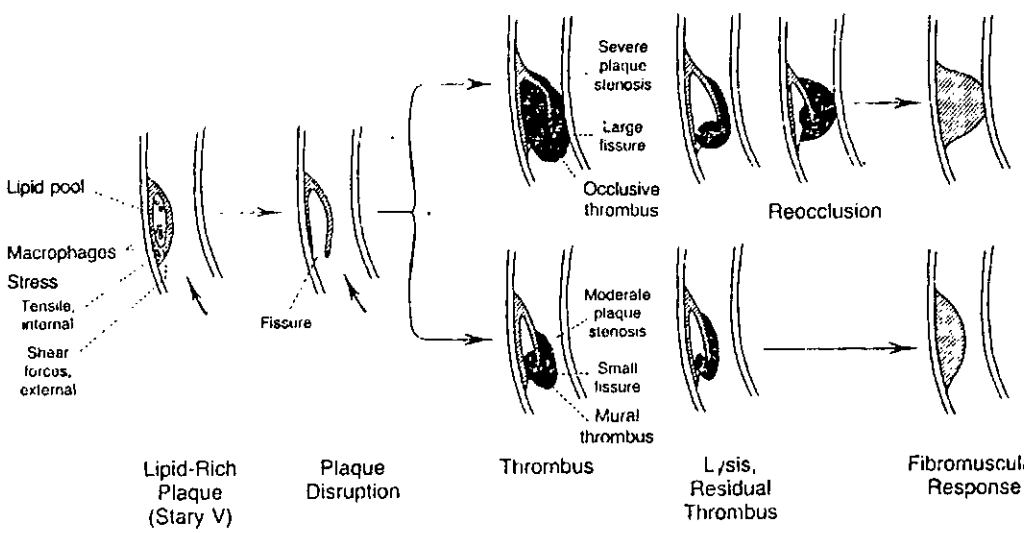

Figure (1): Typical dynamic evolution of the complicated plaque.