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شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

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التوثيق الالكتروني والميكروفيلم

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بعض الوثائـــــــق الاصليـــــة تالفـــة

بالرسالة صفحات لم ترد بالاصل

STUDY OF THE ROLE OF PLASMA HOMOCYSTEINE AND LIPOPROTEIN (a) IN CORONARY ARTERY DISEASE

Thesis

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University of Alexandria
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By

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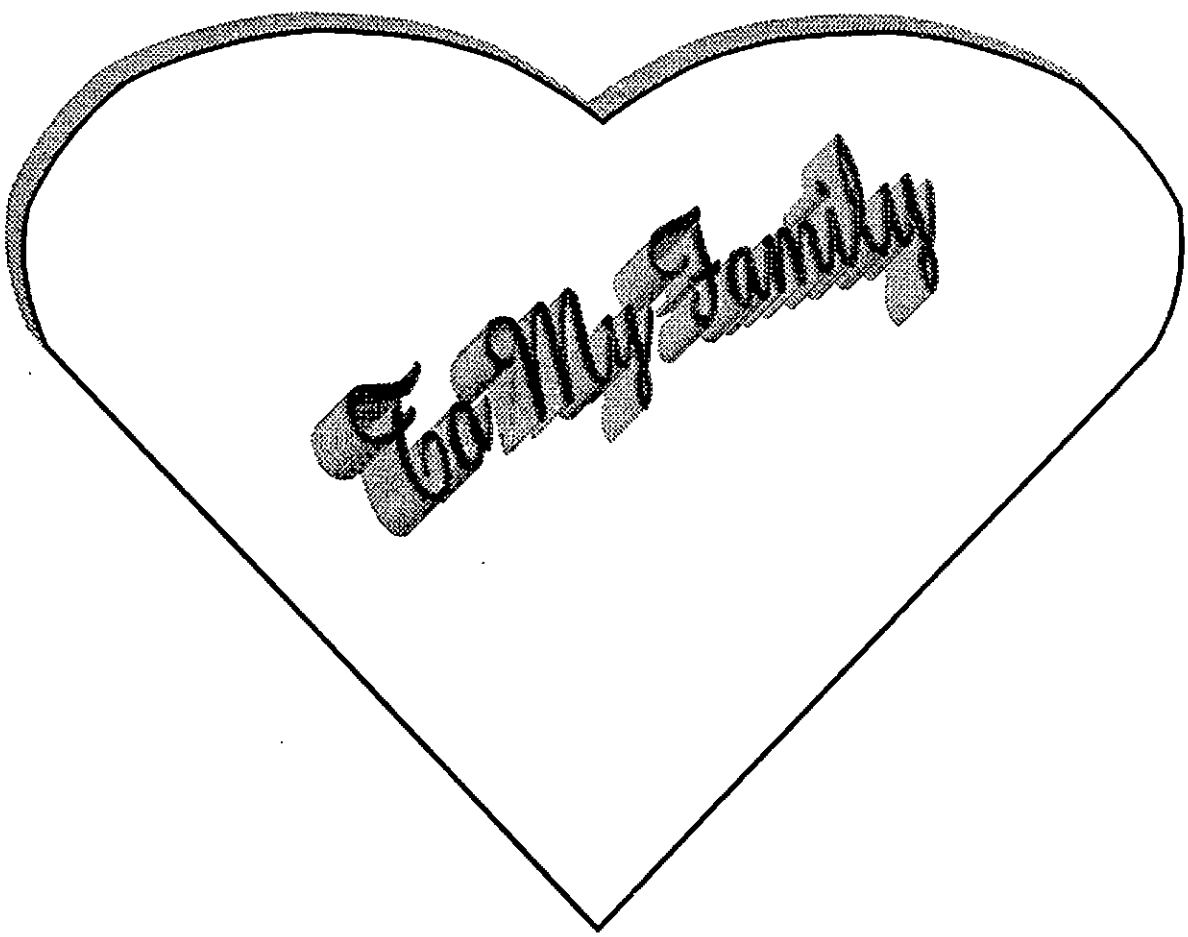
For My Family

CONTENTS

LIST OF ABBREVIATIONS

ADP, ATP	Adenosine di, triphosphates
ALT	Alanine aminotransferase
Apo A, B, C, E	Apolipoproteins A, B, C, E.
APPT	Activated partial thromboplastin time
AST	Aspartate aminotransferase
BH_4	Tetrahydrobiopterin
CAD	Coronary artery disease
CHD	Coronary heart disease
C DNA	Complementary deoxyribonucleic acid
DTT	Dithiothreitol
EDTA	Ethylenediaminetetraacetic acid
EGF	Epidermal growth factor
ELISA	Enzyme- linked immunosorbent assay
eNOS	Endothelial nitric oxide synthase
FH	Familial hypercholesterolemia
HDL	High- density lipoproteins
HDL-ch	High- density lipoproteins- chlesterol
HRP	Horse radish peroxidase
ICAM-1	Intercellular adhesion molecule 1
IgG	Immunoglobulin G.
LDL	Low- density lipoproteins
LDL-ch	Low - density lipoproteins - cholesterol
Lp (a)	Lipoprotein (a)
LSD	Least significant difference
mRNA	Messenger ribonucleic acid

MTHE	N5- methyl tetrahydrofolate
MTHER	Methylenetetrahydrofolate reductase
NIDDM	Non insulin dependent diabetes mellitus
NO	Nitric oxide
PABA	Para- aminobenzoic acid
PAI-1	Plasminogen activator inhibitor-1
PAs	Plasminogen activators
PG	Prostaglandins
PGA	Pteroylglutamic acid
PT	Prothrombin time
PVS	Polyvinyl sulphate
RCL	Reactive center loop
SAH	S- adenosyl L- homocysteine
SDS	Sodium dodecyl sulphate
Serpin	Serine protease inhibitor
SPS	Sample pre- treatment solution
TF	Tissue factor
TG	Tiglycerides
THF	Tetrahydrofolic acid
t-PA, u-PA	Tissue-type, urokinose-type plasminogen activators
TXA_2	Thromboxane A_2
VCAM-1	Vascular cell adhesion molecule - 1
VLDL	Very low- density lipoproteins
Vn	Vitronectin

INTRODUCTION

INTRODUCTION

CORONARY ARTERY DISEASE

The term coronary artery disease (CAD) defines a disease spectrum of diverse etiology, with the common factor being an imbalance between myocardial oxygen supply and demand.[1] The term CAD has been used synonymously with coronary heart disease (CHD), or atherosclerotic heart disease.[2]

Pathogenesis of CAD:

Myocardial ischaemia is considered as a discrepancy between myocardial oxygen requirements and the oxygen delivering capacity of the coronary circulation.[3] Atherosclerotic plaques are the most common cause of CAD.[4] When atherosclerosis significantly occludes the coronary artery, impairing the coronary flow, CAD ensues.[5]

The transition from stable to unstable angina pectoris probably results from a change in the surface of the atherosclerotic plaque, often rupture of the intima of the fibrous cap with subsequent haemorrhage, platelet aggregation, fibrin deposition, and thrombus formation or a sudden increase in smooth muscle tone, resulting in spasm of the coronary artery.[5] Thrombosis superimposed on a stenotic atherosclerotic lesion is the most frequent cause of myocardial infarction.[6]

Coronary atherosclerosis:

Atherosclerosis is a progressive disease process that generally begins n childhood and has clinical manifestations in middle to late adulthood. It is a multifactorial process which requires extensive proliferation of the smooth muscles within the intima of medium size and large size arteries.[7]

The pathological aspects of atherosclerosis:

The two principal forms of atherosclerosis are the early lesion or the fatty streak and the advanced lesion or fibrous plaque, which can become a complicated lesion.[8] Fig. (1)

The fatty streak:

This is the most common lesion of atherosclerosis and occurs at all ages. It becomes evident in the coronary arteries during the second decade.[10] The early fatty streaks appear to consist of macrophages together with a variable number of T-lymphocytes. As the lesions expand, they appear to contain smooth muscle cells that have migrated into the intima as well. Both the macrophages and the smooth muscle cells are laden with cholesterol and cholesterol oleate. These lesions are confined principally to the intima and being flat, they cause little or no obstruction of the affected artery and have no clinical sequelae.[11]

The fibrous plaque:

This is a more advanced lesion that begins to develop around the age of 25 in those populations in whom there is a high incidence of atherosclerosis. There is migration and proliferation of smooth muscle cells, forming a fibrous cap, owing to the deposition by these cells of new connective tissue matrix, including collagen, elastic fibers and proteoglycans and to the accumulation of intracellular and extracellular lipids.[11] The fibrous plaque is located in the intima and characteristically leads to eccentric thickening of the artery that often results in occlusion of the lumen.[10]