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شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم

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شبكة المعلومات الجامعية

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التوثيق الالكتروني والميكروفيلم

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جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

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بالرسالة صفحات لم ترد بالأصل

ASSESSMENT OF NUTRITIONAL FACTOR IN PATIENTS WITH ISCHEMIC HEART DISEASE

Thesis

Submitted to the faculty of Medicine,
University of Alexandria
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By

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INTRODUCTION

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Definition of ischaemic heart disease:

Ischaemic heart disease (IHD) is defined by the World Health Organization (WHO) as "myocardial impairment due to imbalance between coronary blood flow and myocardial requirements caused by changes in the coronary circulation.[1]

Epidemiology:

- The cardiovascular disease (CVD) are emerging as a major health problem all over the world. in the United States coronary artery disease CAD accounts for fully one half of the nearly one million deaths each year from (CVD).[2] In the United Kingdom, CAD is responsible for about one third of all male deaths and one quarter of all female deaths.[1]
- CVD were reported as a leading cause of death in Egypt[3] and the proportionate mortality rate due to CVD has dramatically increased in the last decades from 12.4 to 42.5% of all deaths.[4]
- Risk factors that are responsible for the development of CAD varies between rural and urban areas.
- Patients with schistosomal hepatic fibrosis (SHF) are not common subjects of atherosclerosis. This has been attributed to the reduced levels of serum lipids, hyperestrogenaemia and to high ration of α tocopherol to total lipids in SHF patients.[5] However in the last few years, schistosomal patients started to develop CAD without any evident risk factor known before. This could be attributed to environmental,

socioeconomic and nutritional factors as well as changes in life style of population towards urbanization.[6]

Coronary circulation

Anatomy of the coronary circulation: Figure (1)

Right coronary artery:

It arises from the anterior aortic sinus and runs forwards in the atrioventricular groove supplying branches to the right atrium, sinoatrial node, most of the anterior surface of the right ventricle and usually ends as the posterior interventricular branch.

Left coronary artery:

It arises from the left posterior aortic sinus and passes forwards between the pulmonary trunk and the left auricle and it has two branches:

1. Anterior descending branch: runs in the anterior interventricular sulcus supplying small part of the right ventricle and apical part of the left ventricle.
2. Circumflex branch: runs in the atrioventricular goove giving of one or more marginal branches supplying the lateral wall of the left ventricle and a branch to the left atrium.

The left and right coronary arteries anastomose by small branches at the apex of the heart and in the interventricular septum.[7]

5 DISEASES OF THE CARDIOVASCULAR SYSTEM

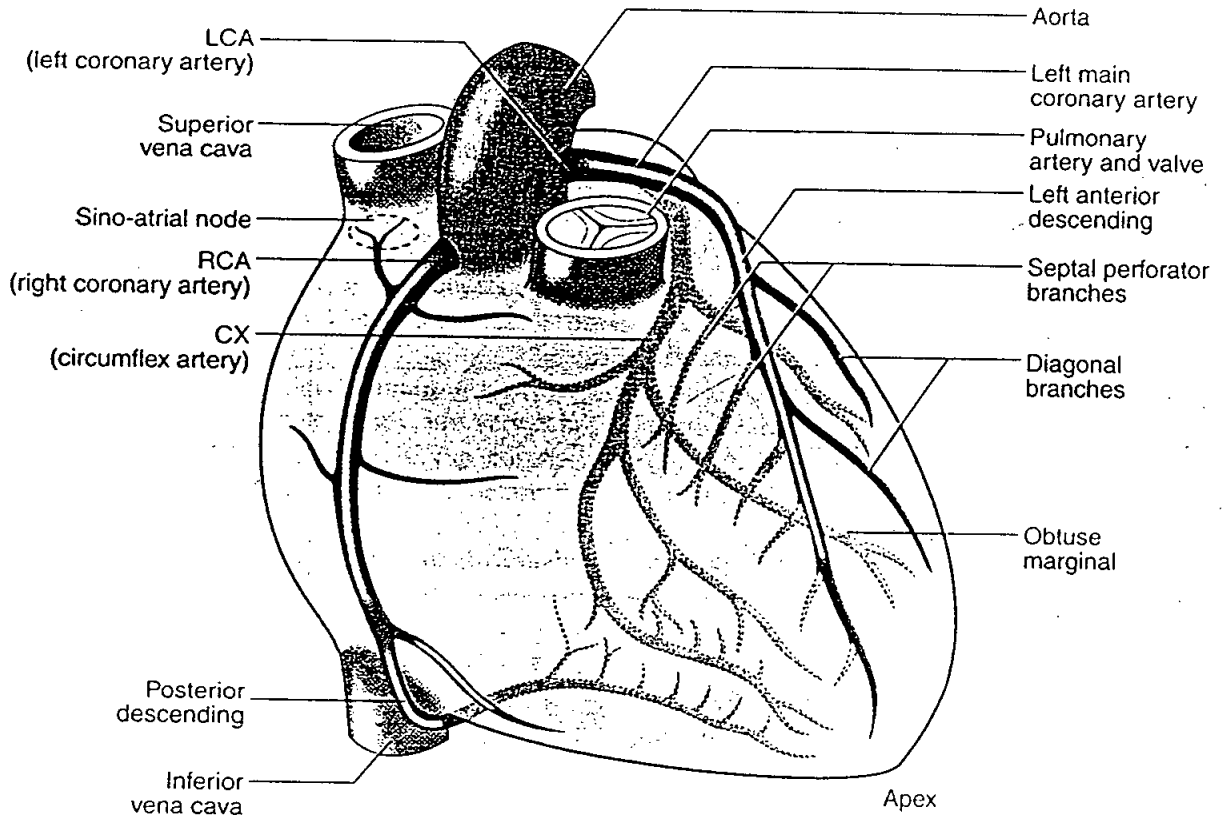

Fig. 5.1—*continued.* **Diagram of the coronary arteries of the heart** (anterior view). The left (LCA) and right (RCA) coronary arteries arise in the respective sinuses just distal to the aortic valve. The left main coronary artery divides into a left anterior descending branch which supplies the front wall and apex of the heart, and a circumflex (CX) branch which supplies the posterior wall of the heart and a variable proportion of the inferior wall. The right coronary artery gives off right ventricular branches and may give rise to the posterior descending vessel which runs in the posterior interventricular groove. The sinoatrial node (sinus node) is situated at the junction of the superior vena cava and right atrium and the atrioventricular node (AV node) is situated beneath the right atrial endocardium at the lower end of the interatrial septum.

Physiology of coronary circulation

Under resting conditions, cardiac muscle normally was mainly fatty acids for its energy insteand of carbohydrates, with about 70 percent of the normal metabolism being derived from fatty acids. Under anaerobic or ischaemic conditions, cardiac metabolism must call on the anaerobic glycolysis mechanisms for energy. This can supply little extraenergy in relation to the large energy requirements of the heart. Also glycolysis uses tremendous quantities of the blood glucose and at the same time forms large amounts of lactic-acid in the cardiac tissue, which is probably one of the causes of cardiac pain in cardiac ischaemic conditions. This ATP in turn acts as the conveyer of energy for cellular function. in sever coronary ischaemia, the ATP degrades to adenosine, much of this can be lost from the muscle cells into the circulating blood. This adenosine is believed to be one of the substances that causes dilatation of the coronary arterioles during coronary hypoxia. The loss of adenosine also has a serious cellular consequence, within as little as 30 minutes of sever coronary ischaemia, as occur after a myocardial infarct or during cardiac arrest, about one half of the adenine base can be lost from the cardiac muscle cells. Therefore, once a serious bout of ischaemia has persisted for 30 or more minutes, relief of the coronary ischaemia may be too late to save the lives of the cardiac cells. This almost certainly is one of the major causes of cardiac cellular death after myocardial ischaemia and one of the most important causes of cardiac, debility in the late stages of circulatory shock.[8]

Etiology of ischaemic heart disease (IHD)

The term ischaemic heart disease defines as a disease spectrum of divers etiology, with the common factor being an imbalance between myocardial oxygen supply and demand.

Decrease in myocardial oxygen supply occur due to decrease in quantity of coronary blood as in coronary atherosclerosis, embolism, coronary osteal stenosis, coronary artery spasm, congential anomalies of the coronary circulation and very uncommonly an arteritis of the coronary vessels or occur due to decrease in oxygen carrying capacity of the blood as in severe anaemia, hypoxia or the presence of abnormal haemoglobin.(9)

Increase in myocardial oxygen demand occurs due to volume over load on the heart as in aortic value incompetence, mitral valve incompetence and in hyperdynamic circulation as in thyrotoxicosis or occur with pressure over load due to systemic hypertension, aortic stenosis and aortic coarcitation.(10)

Pathogenesis of coronary atherosclerosis:

Atherosclerosis is a disorder of the arterial wall characterized by accumulation of cholesterol esters in the cells derived from the monocyte-macrophage line, smooth muscle cell proliferation and fibrosis. The earliest abnormality is the migration of blood monocytes to the subendothelium of the artery. Once there, they differentiate into macrophages which accumulate cholesterol esters derived from plasma LDL. Distortion of the subendothelium leads to platelet derived nitrogen as platelet derived growth factor (PDGF) which stimulate smooth muscle cell growth. Death