FOLLOW UP STUDY OF BIOCHEMICAL CHANGES OCCURING IN RECENT CARDIAC INFARCTION

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INTRODUCTION

The diagnosis of acute myocardial infarction using enzymatic determination is now been used routinely in most of cardiac centres. The recent concept that the determination of the size of the myocardial infarction at the onset have a bearing on prognosis is now been accepted.

The aim of this work is to correlate the enzyme level with the prognosis mainly the complications that follows the onset of infarction. This was achieved by studying cases referred to Ain Shams C.C.U. with the provesional diagnosis of myocardial infarction. Three enzymes avilable for estimations were CPK, SGOT, and LDH, and were carried out in all patients studied.

One added aim of the work was to see the effect of acute insult to the heart and circulation during the course of myocardial infarction on the blood chemistery mainly cholesterol and triglycerides.

REVIEW OF LITERATURE

Anatomy of Coronary Arteries (James, 1965):

The left coronary ostium, which is 3 to 5 mm in diameter, originates from the left coronary sinus of valsalva. The main left coronary artery lies between the body of left atrium and the pulmonary artery, and curves anteriorly toward the anterior interventricular sulcus. After a usual length of 1 to 2 cm. it divides into two or more branches. The branch that enters the left atrioventricular sulcus originates at a right angle and becomes the left circumflex artery. The branch that enters the anterior interventricular sulcus, virtually as a continuation of the main left coronary artery, becomes the left anterior descending artery. Branches originating between these two are known as diagnoal left ventricular arteries, coursing in the free wall of the left ventricle between the circumflex and anterior descending arteries. The left anterior descending artery always reaches the apex cordis and curves about it to enter the posterior interventricular sulcus. The left circumflex artery usually terminates at or just beyond the obtuse margin of the left ventricle, supplying most of the lateral

wall and usually about half of the diaphragmatic surface of the left ventricle.

The right coronary ostium is normally 2 to 4 mm in diameter and lies near the middle of the right coronary sinus of valsalva. In addition to the main right coronary ostium in about half of the human hearts there is a second ostium located about 1 mm away and giving rise to the conus artery, and its potential importance as a source of collateral circulation is obvious (Schlesinger, et al, 1949). The main right coronary artery passes between the body of the right atrium and pulmonary artery into the right atrioventricular sulcus, in which it continues about the margo acutus to the diaphragmatic surface. In about 90% of human hearts it crosses the posterior interventricular sulcus to supply nearly half of the diaphragmatic surface of the left ventricle, in addition to most of the free wall of the right ventricle. The right coronary terminates as several parallel trunks descending in or near the posterior interventricular sulcus toward the terminal branches of the left anterior descending artery at the apex. The right coronary gives a second unusual branch, which curves behind the

aorta to emerge in the left atrioventricular sulcus and become a left circumflex artery, in such cases the main left coronary artery provides only the anterior descending branches.

The right ventricular papillary muscle is supplied predominantly by penetrating branches from the left anterior descending coronary artery. The anterior papillary muscle is supplied primarily by one or more branches of the left anterior descending artery or by diagonal branches of the left circumflex artery. The posterior papillary muscle is supplied by the junction of terminal branches of the left circumflex artery and of the right coronary artery, when the left circumflex artery supplies nearly all the diaphragmatic surface of the left ventricle (10%), its branches provides the entire supply for the posterior papillary muscle.

Most of the blood supply to the interventricular septum is provided by the left anterior descending coronary artery. Branches into the septum from posterior descending arteries rarely penetrate more than 15 mm from the epicardium. The septal branches of the anterior descending artery sweep posteriorly along the right

ventricular side of the septum, sending in smaller divisions to supply local areas. The are immediately posterior to the membranous septum is supplied by the only long septal branches of the posterior descending artery. This area includes the region of the atrio-ventricular (AV) node and bundle of His.

In about 55% the sinus node artery originates from the second or third centimeter of the right coronary artery, and in about 45% from the first few millimeters of the left circumflex artery. From either right or left site of origin it courses along the body of the adjacent atrium to the base of the superior vena cava, from the left side coursing through the anterior myocardial band (Bachmann's bundle) to reach that point. The sinus node artery does not terminate in the node, but passes directly through it and sends only small nutrient branches laterally into the substance of the node.

The A-V node is supplied by the artery which crosses the crux of the heart, that point at which the atrioventricular sulci cross the junction of the posterior margins of the interatrial and interventricular sulci.

In about 90% of the human hearts this is the rig coronary artery and in the remainder it is the l circumflex. The artery crossing the crux makes unique deep U term at this point, and the artery t the A-V node arises near the apex of this penetrati turn.

Definitions:

Myocardial infarction signifies the necrosis or death of a portion of heart muscle because of an inter ruption or curtailment of its blood supply. It may occur as a result of an acute coronary occlusion, a sharp reduction in the volume or oxygen content of the coronary blood, owing to circulatory or hematologic disturbance (Friedeberg, 1969).

Myocardial infarction is a syndrome characterized by prolonged chest pain, QRS, ST and T wave changes and elevated serum enzymes (Hurst, 1974).

Coronary atherosclerosis implies an abnormality of the coronary arteries without indicating the part of the wall that is involved. Coronary atherosclertic heart disease indicate that a specific disease (coronary

atherosclerosis) is present and that other diseases of the coronary circulation are not. The word heart disease indicates either the clinicion or the pathologist or both has evidence that the atheroscle-rotic process has reached a degree of severity, sufficient to cause certain clinical or pathological abnormalities which result from inadequate myocardial perfusion (Hurst 1974).

In the last 20 years epidemiologic and exprei-mental studies have provided considerable evidence linking certain risk factors to the development of atherosclerotic lesions. The prevention and manegment of coronary atherosclerosis obviously requires the identification of the major factors contributing to the atherosclerotic process and a careful effort to correct or remove the modifiable risk elements involved (Hurst, 1974).

Risk Factors for Coronary Atherosclerotic Heart Disease (Hurst, 1974).

1. Nonmodifiable risk factors:
   a) age. b) sex.
   c) Family history of premature coronary athero-sclerotic heart disease.

II. Modifiable risk factors:

A. Major modifiable risk factors:

1. Elevated serum lipids (cholesterol and triglyceride).
2. Habitual diet high in total calories, total fats, saturated fats, cholesterol, refined carbohydrate and salts.
3. Hypertension.
4. Cigarette smoking.
5. Carbohydrate intolerance.

B. Minor modifiable risk factors:

1. Obesity.
2. Sedentary living.
3. Personality type.
4. Psychosocial lesion.
5. Others.

Major Medifiable Risk Factors

Elevated Serum lipids Levels:

In the last 20 to 30 years, both retrospective and prospective studies have shown a strong correlation between morbidity and mortality rates from coronary atherosclerotic heart disease and levels of circulating

lipids; Among the serum lipids, cholesterol triglyceride, and B-Lipoproteins have been found to have higher associative and predictive values (Albrink, et al, 1961, Epstein, 1965, Kannel, et al, 1971, Keys, 1970, and Keys, et al 1972).

The fact remains however that patients with cholesterol level greater than 300 mg/dl were found to have four times more risk of coronary atherosclerotic heart disease than patients with level less than 200mg/dl. According to Fredrickson (1972), cholesterol concentrations above 220 mg/dl at any age should be considered suspicious and efforts at clarification of the underlying disorders and correction of the abnormal cholesterol level appear indicated.

Hypertension:

Elevated blood pressure is a risk factor of prime importance and of established association with coronary atherosclerosis (Epstein, 1965, and Freis, 1969). It is found in about 50 percent of men and in about 75% of women with coronary heart disease (Friedberg, 1969). Evans (1965), found that atheromatous disease correlate best with the average systolic blood pressure

values. Elevation of blood pressure was associated with a two folds increase in the risk of coronary heart disease in males and a sixfold increase in females (Dawbar and Konnel, 1961). An increase in coronary heart disease occured at each successive level of blood pressure, with a seven fold increase in those with blood pressure over 180 mmHg, compared to those with pressure below 120 mmHg systolic. Induced hypertension increased the severity of experimental atherosclerosis in rabbits and dogs (Wakerlin, 1952). But the frequency of coronary heart disease in the absence of hypertension is only an accessory or aggrevating factors in atherosclerosis and not a primary cause. Gertler and Whiter, (1965) reported that at any fixed cholesterol level the coronary risk rate did not change with increasing levels of blood pressure. Keys, et al (1963), studies have shown distinct differences between the incidence of hypertension and its cerebral complications.

Smoking

Statistical evidence associating cigarette smoking with an increased risk of developing coronary atherosclerotic heart disease (CAHD) is impressive. In

general the risk of developing CAHD or the risk of death from CAHD is two to six times higher in smokers than nonsmokers, and the risk appears to be proportional to the number of cigarettes (Epstein, 1965).

An increase in blood sugar has been regularly found in experimental animals after injection of nicotine, but observations in human beings are contradictory. Oxygen consumption in the human subject, furthermore, is not increased by nicotine or by cigarette smoke. There is some evidence that platelet adhesiveness is enhanced. Platelet aggregates could cause ischemic episodes, intiate thrombosis and thus lead to atheroma formation. Anoxic damage to the vascular tunics caused by carbon monoxide could in theory favor the localization of atheroma and acclerate atherogenesis (Hurst, 1974).

Abnormal glucose tolerance

Patients with diabetes mellitus have been found to have more extensive lesions, and to have evidence of coronary heart disease at an earlier age than non-diabetic patients (Epstein, 1965, Epstein, 1967, and