SERUM MAGNESIUM, ZINC AND COPPER IN ACUTE MYOCARDIAL INFARCTION

A THESIS

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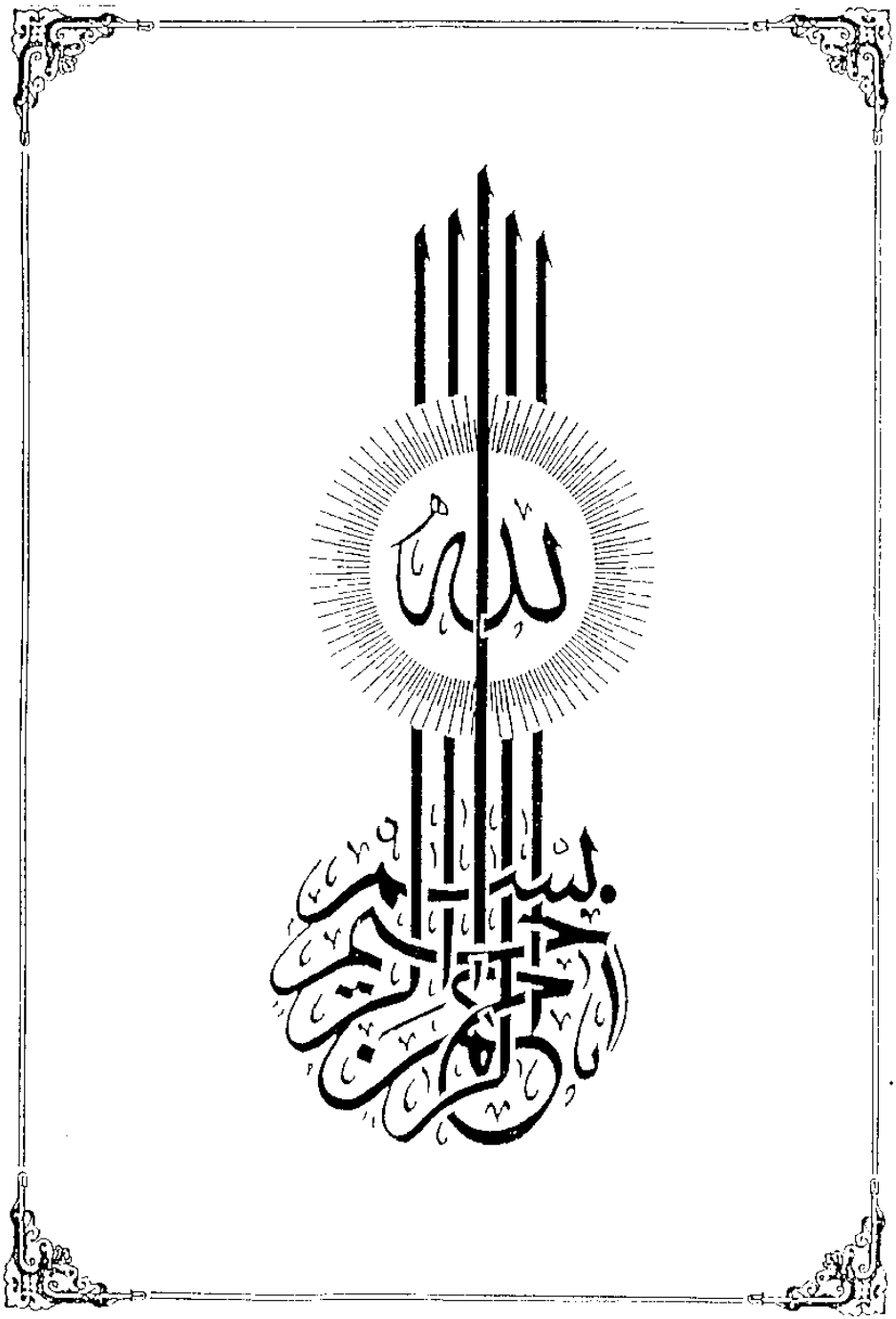

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Aim Of The Work

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Myocardial infarction in a considerable percentage of cases remain clinicaly undiagnosed despite the availability of multitude diagnostic modalities especialy in developing countries, where the majority live in rural areas and patients report late to specialized centers due to lack of transport facilities.

In recent years attension has been drawn towards trace element like copper which is an important component of numerous cuperoenzimes present in the myocardium, and are involved in maintenance of myocardial integrity.

Also zinc is needed by the myocardium for synthesis of enzymes dependant on this metal ion, because these enzymes function in repair of myocardial damage.

While magnesium (which is not one of trace elements) has an important role in protection against ischemic heart disease.

Also magnesium is known to influence the excitable membrane, and a variety of electrocardiographic changes have been reported in association with magnesium depletion.

The aim of our work is to study the level of magnesium, zinc and copper in cases of myocardial ischaemia and infarction and the possible role of these elements in complications of myocardial injury.

Review Of Literature

REVIEW OF LITERATURE

MYOCARDIAL INFARCTION

Necrosis of the cardiac muscle due to irreversable myocardial ischemia is called infarction (Hurst, 1982).

Myocardial infarction can be diagnosed when two of the following three clinical features are present which are chest discomfert that is characterestic of myocardial ischemia, QRS, ST and T changes, and elevated serum enzyme (Hurst, 1982).

Myocardial infarction signifies necrosis or death of a portion of heart muscle because of an interruption of its blood supply. It may occurs 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 hematological disturbance (Friedberg, 1969).

In a study made by (Ribeiro et al, 1984) the diagnosis of coronary artery disease was based on at least one of the following criteria :-

Coronary angiography demonestrating more than 50 % narrowing of at least one major coronary artery.

A myocardial infarction documented by enzyme elevation, significant Q wave in the ECG,

A prevous coronary by pass operation.

A history which was completly typical for angina pectoris or an exercise test with both one or more millimeters of ST depression and chest discomfort typical for angina

Coronary atherosclerotic heart disease indicates that an abnormality of the coronary arteries is present.

Atherosclerosis is a metabolic disease of multiple causes in which altered lipid metabolism plays an important role, lipid accumulation most prominantly in the intima of major arteries, has been recognized as a cause of atherosclerosis.

Circulating lipoprotein particularly cholesterol with low density lipoproteins are the primary sources for the accumulated cholesterol in atherosclerotic arteries (Goldstein, 1973).

Raul (1978) revealed that the incidence of coronary disease was related to serum cholesterol level even in rural samples, in which the incidance rate of this disease is lowest. Triglyceride values, unlike cholesterol, were not significantly related to this incidance in either rural or urban men.

Total plasma cholesterol, and more specifically, low density lipoprotein (LDL) cholesterol are powerful predictors of both the prevalence and incidance of coronary heart disease as observed in many counteries throughout the

world. High density lipoprotein (HDL) cholesterol on the other hand is negatively associated with coronary heart disease, so that the higher the level of HDL cholesterol, the lower the risk (Castelli, 1984).

Another study had also shown that HDL cholesterol is inversely and strongly related to coronary atherosclerotic raised lesions (Holme at al., 1981).

In the last 20 years epidemiological and experimental studies have provided considerable evidence linking certain risk factors to the development of atherosclerotic lesions, the prevention and managment of coronary atherosclerosis obviously require the identification of the major factors contributing to the atherosclerotic process, and carefull effort to correct or remove the modifiable risk elements envolved (Hurst, 1974).

Risk factors for coronary heart disease

According to Hurst (1974) risk factors are classified into

(1) Non modifiable risk factors

1- Age

2- Sex

3- Family history of premature coronary atherosclerotic heart disease

(2) 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- Cigarattes smoking

5- Carbohydrate intolerance

(B) Minor modifiable risk factors

1- Obesity

2- Sedentary living

3- Personality type

4- Psychosocial tension

Age, sex and blood pressure were found to be major risk factors predictive of coronary heart disease mortality in lipid research clinics (LRC) follow up study (1980).

The ossociation between coronary risk factors and clinicaly manifist coronary heart disease have been established. The pathway of action has been hypothesized as being through the biulding up of coronary atherosclorotic raised lesions and stenosis finally resulting in occlusion ischemia and myocardial infarction, angina pectoris, sudden

death due to coronary heart disease (Holme et al., 1985).

Clinical Persentation of Acute Myocardial Infarction

In a survey of 194 patients with myocardial infarction undertaken in Oxford, 44 % had a history of angina, 19 % pervious infarction and 30 % high blood pressure.

Many patients provide a retrospective history of **Prodromal fatigue** during the preinfarction period, but the vague and commen nature of this symptoms make it difficult to evaluate (Pentecost 1984).

Pain is the most commen complaint in patients with myocardial infarction. In some instance the discomfort may be severe enough to be described as the worst pain the patient has ever experienced. The pain is deep and visceral described as heavy, sqeezing and crushing in character. It is similar to pain of angina but is usually more sever and lasts much longer and when it begins during a period of exertion, it does not usually subside with cessation of activity, typically the pain envolves the central portion of the chest and / or epigastrium and in 30 % of cases it radiates to the arms. Less common sites of radiation include the abdomen, back, lower jaw and neck the pain of myocardial

infarction does not radiate above the maxilla or below the umbilicus (Braunwald and Alpert, 1983).

The patient may consume large quantities of nitrates due to pain over a short period of time which fail to releive pain and produce hypotension and sometimes syncope.

A patient with no pervious experience of angia, may assume the pain as indigestion, the frequency of nausia and belching reinforces this belief with the result that he may take dyspepsia remedies before seeking medical advice.

Sweating was another commen feature of the attack (Pentecost, 1984).

15 to 20 % of myocardial infarction are painless, which is greater in patients with diabetes mellitus, atributed to diabetic neuropathy, and it increases with age.
In elderly, myocardial infarction may present as sudden onest of **breathlessness**, which may progress to pulmonary oedema.

Other less commen presentation in the absence of pain include sudden loss of consciousness, a confusional state, a sensation of profound weakness, the appearance of an arrhythmia or merely an unexplained drop in arterial blood pressure.

In many patients, the dominant feature of the patient's presentation is the reaction to the chest pain. Patients are

typically anxious and restless, attempting to releive pain by moving about in bed, belching or even induction of vomiting. In contrast to pain of angina pectoris which causes the patient remain relatively immobile for fear of making pain reappear (Braunwald and Alpert, 1983).

The patient's appearance is mainly determined by the severity of pain. This will provokes fear and anexiety leading to over activity of sympathetic nervous system irrespective of any autonomic imbalance attributable to the process of infarction itself.

The patient may present with **shock** in which there is pallor, sweating, thready peripheral pulse and poorly perfused exterimities.

If myocardial injury is seveere, evidences of left ventricular failure is present, the patient is orthopnic but physical signs are sparce, crepitation over the lung field are often abscent and the jugular venous pressure is usually normal even when left ventricular falure is severe. An atrial or fourth sound is present in the acute stages of infarction. When myocardial damage is extensive, a third heart sound is audible.

Although the patient's blood pressure will almost always fall below previously recorded levels after infarction, this may not be apparent for several hours and it must never be

assumed that maintenance of the normal blood pressure is evidence against infarction.

In patients suffering extensive myocardial damage severe systemic hypotension will develope early in the course of illness.

Fever is usually appearent within 12 hours and is occasionaly the first confirmatory evidence of infarction, being evident before return of serum enzyme levels. Pyrexia usually settle within four or five days but occasionally persists for a week.

Pericarditis is a sign of extensive infarction which involve the epicardial region of the heart and usually makes its appearance between the second and fourth days. The rub may persist and accompanied by further chest pain but is often remarkably transient (Pentecost, 1984).

An acute inflammatory response to the presence of dead myocardial tissue results in an elevated neutrophil count and a raised erythrocyte sedimentation rate, both are non specific finding and of little practical help in diagnosis.

Death of myocardial cells result in the release of intracellular enzymes that vary greatly in the time relationship between the episode of infarction and rise in plasma concentrations, as well as in their specificity for myocardial damage.