EFFECT OF ACUTE MYOCARDIAL INFARCTION ON SOME COMPONENTS OF THE FIBRINOLYTIC SYSTEM

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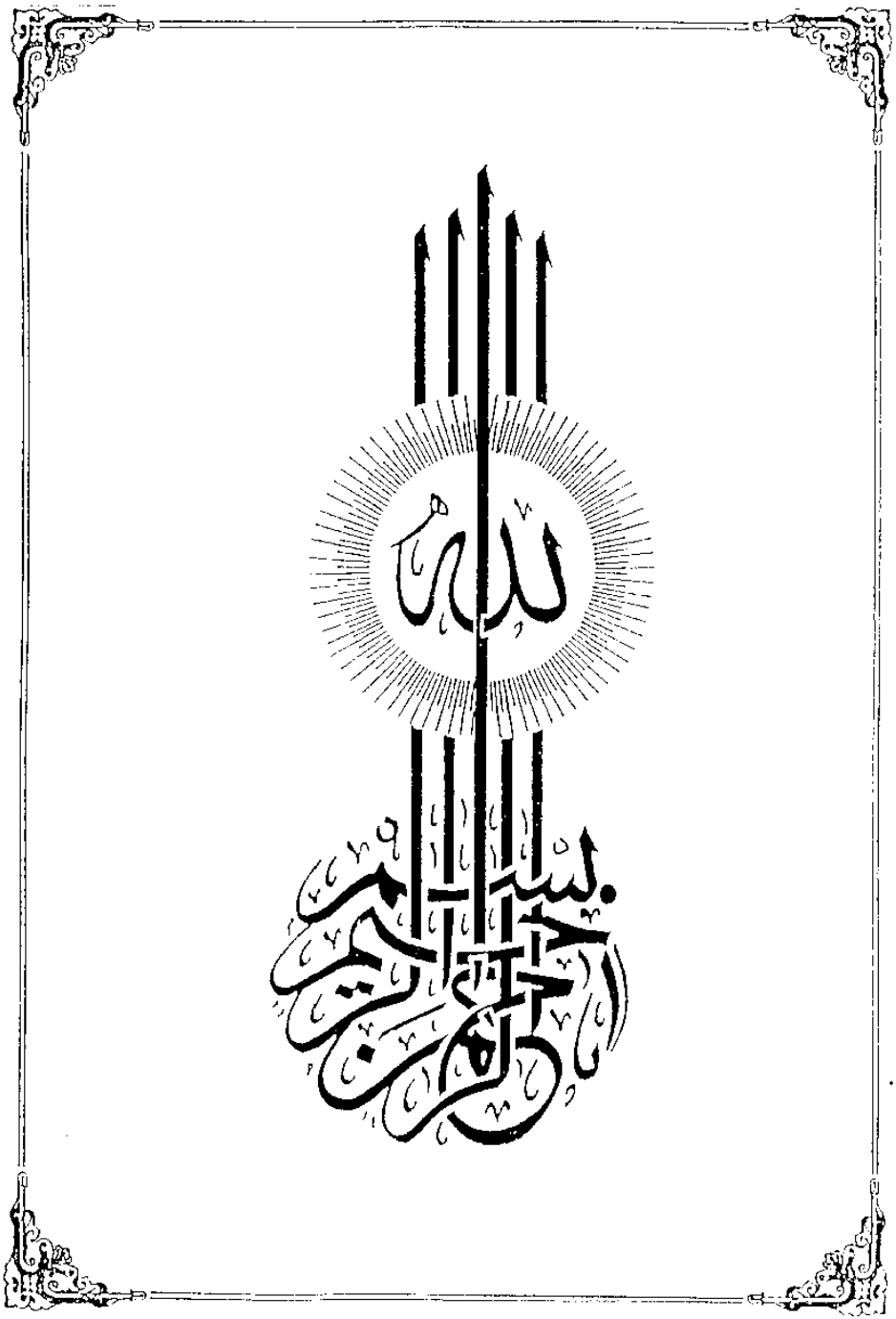

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Myocardial infarction represents one of the commonest serious health problems of contemporary medicine, and its acute condition remains to be one of the main causes of inhospital deaths, (Braunwald, 1976).

Inspite of the great advances which were achieved recently in the understanding of the pathophysiology of coronary heart disease, still the aetiology remains unclear. A therosclerotic coronary disease is the principal aetiological factor in the causation of ischaemic heart disease. The role of thrombus formation and the state of hypercoagulability in relation to pathogenesis of myocardial infarction are not yet fully understood (Sharma and Seth, 1979).

It has been suggested that one of the causes of intravascular thrombosis might be decreased plasma fibrinolytic activity (Sherry, Fletcher, and Alkjaersig, (1959) influenced some investigators into suggesting such a cause as a possible aetiological basis for coronary artery thrombosis (Chakravarti, Rocking, and Fearnley, 1968). To them, a slight decrease in plasma fibrinolytic activity might follow the inadequate lysis of slowly

deposited fibrin strands. Yet this proposal was difficult to evaluate, as investigations of the activity of plasminogen-plasmin system in vivo are difficult to perform, and relatively insensitive, and semiquantitative (Chakrabarti et al., 1968). Recently, however, more accurate and useful methods are available and many investigators try to determine the role, if any, of defective fibrinolysis in cases of myocardial infarction.

The aim of the present work is to study fibrinolytic activity in patients after an acute attack of myocardial infarction. This has been attained by measuring some of the components of the fibrinolytic system, namely:

1. Fibrinogen,
2. Fibrin fibrinogen degradation products, and
3. Plasminogen.

REVIEW OF LITERATURE

NATURAL FIBRINOLYSIS

In 1937, Macfarlane stated that test tube thrombi formed from blood samples taken during surgical operations, sometimes dissolved spontaneously. In 1953, Fearneley and Tweed showed that there was some fibrinolytic activity in the blood of patients at rest. At the same time, Kwann and McFadzean (1956) developed the hypothesis that the vascular endothelium was the main source of activators. This was confirmed later when Todd (1959) demonstrated an activator of fibrinolysis in the walls of small blood vessels, particularly in the veins. Also, Astrup (1956) showed that there are plasminogen activators in almost all of the fluids and tissues of the body.

Two hypotheses were presented; the first is that fibrinolysis is existing in the circulation, as a counterbalance to many small thrombi, which may develop and so it keeps the normal fluidity of the blood intact (Norman, 1978). The second hypothesis is that an abnormality in the natural fibrinolysis mechanism might predispose intravascular thrombotic disease, particularly in the veins, or, exacerbate the deposition of fibrin in atherosclerosis and any other condition associated with fibrinous exudate (Norman, 1978).

It has often been suggested that fibrinolysis might be in continuous dynamic equilibrium with the process of blood coagulation. Fibrin is laid down when it is needed, to seal the defects in the endothelium, and fibrinolysis would be removing such deposits when they have served their haemostatic purpose. Although this hypothesis is not yet proved, there is a kind of undoubted interaction between the two systems at many levels.

Infact there are many appareant similarities between coagulation and the plasminogen-plasmin system (Chesterman, 1975), and there is no doubt that any abnormality of one system or the other, would lead to pathological states of coagulability (Norman, 1978).

PHYSIOLOGICAL VARIATIONS IN FIBRINOLYSIS

Physical Exercise and Emotional Stress:

Exercise and emotional stress do cause an increase in plasma activator levels. A slight effect may even be found after a moderate exercise such as walking (Cash, 1966), while a major increase is usually recorded only after severe exercise (Davis, Abildgaard, Beruauer, and Britton, 1976).

Diurnal Variations:

Fibrinolytic activity has been shown to exhibit diurnal variations, being greater during the day than at night. These diurnal variations, when present, are attributed to the stress reaction (Fearnley, 1960). On the other hand, some authors found no clear evidence for the presence of these diurnal variations, provided that there were no pathological conditions (Sawyer, Fletcher, Alkjaersig, and Sherry, 1960).

Age and Sex:

Fibrinolytic activity is greater in the elderly than in the young (Swan, 1963). Yet, Mann (1967), stated that, difference in fibrinolytic activity, attributable to age, is rather due to variations in the

methods used or to differences of the plasma fibrinogen levels.

Concerning the changes in fibrinolytic activity, there is plenty of controverce in present findings. Fibrinolytic activity, was found by Cash (1966) to be greater in females. Sawyer et al., (1960) found no significant difference in the recorded values of both sexes. Cash and Woodefield (1968) found that there was a significantly higher fibrinolytic response to exercise in the female compared to the male. Konttinen (1965) demonstrated a higher blood fibrinogen content, which may contribute to the longer euglobulin clot lysis time in the female than in the male.

Pregnancy:

Fibrinolytic activity progressively decreases as pregnancy advances, with raised plasminogen and fibrinogen levels while anti-plasmin level remains normal. The recovery from reduced to normal fibrinolytic activity takes place over a period that ranges between 15 minutes and one hour after placental delivery (Bonnar, 1973).

Obesity:

Fibrinolytic activity diminishes in obese