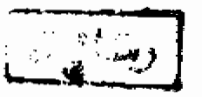
STUDY OF INCIDENCE, COURSE, AND SIGNIFICANCE OF PYREXIA AFTER ACUTE MYOCARDIAL INFARCTION (AMI)

Thesis
Submitted in Partial Fulfilment
for Master Degree of Cardiology



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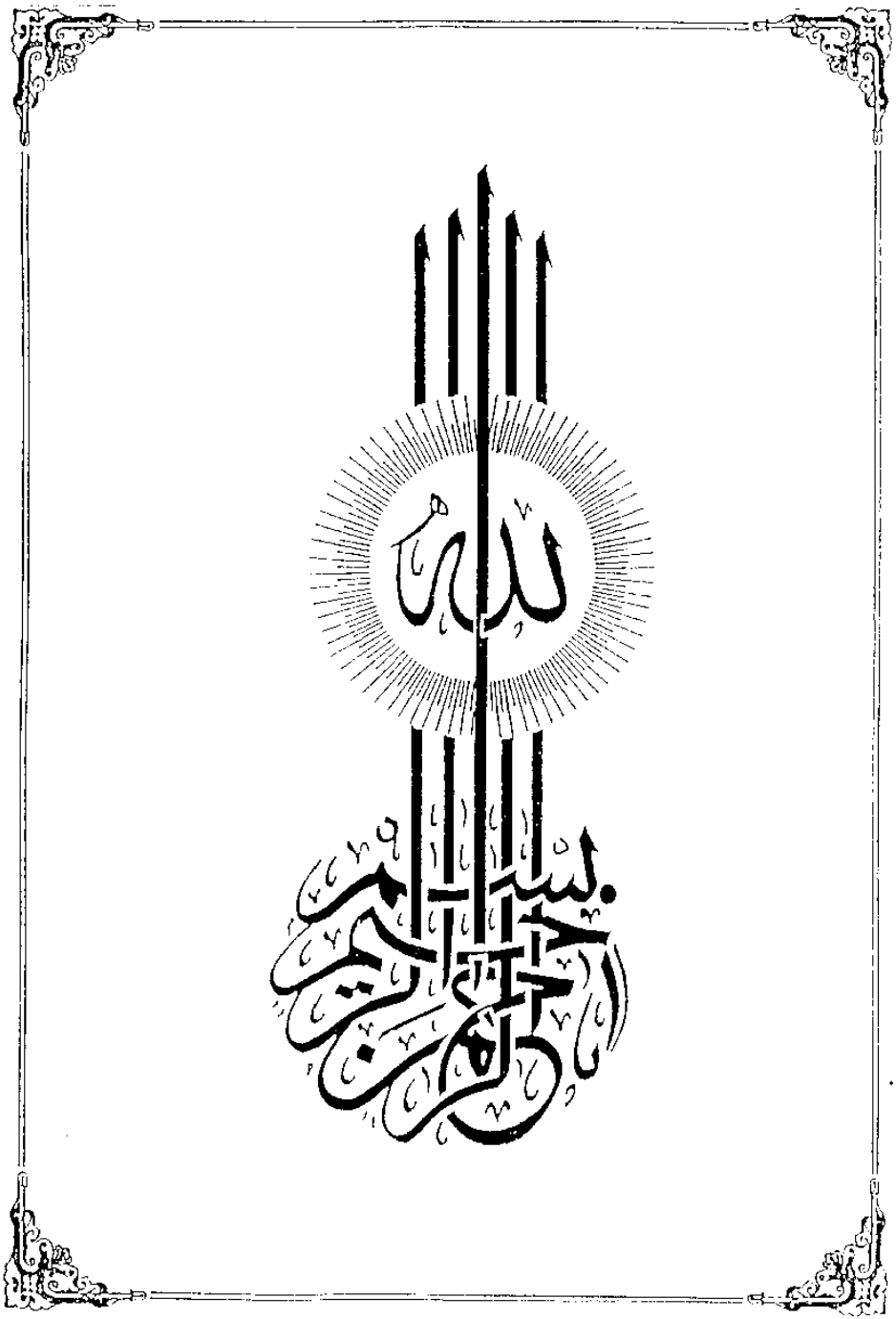

List of Abbreviations

AMI : acute myocardial infarction

CCU : coronary care unit

CHF : congestive heart failure

COP : cardiac out put

CPK : creatine phosphokinase enzyme

ECG : electrocardiogram.

EP : endogenous pyrogen.

ESR : erythrocytic sedimentation rate.

Ht.F. : heart failure.

IHD : ischaemic heart disease.

Lab. : laboratory.

LDH : lactate dehyrogenase enzyme.

MI : myocardial infarction.

PMI-syndrome: post myocardial infarction syndrome.

SGOT : serum glutamic oxalacetic transaminase enzyme.

Temp. : Temperature.

TLC : total leukocytic count.

UTI : urinary tract infection.

5-HT : five-hydroxy tryptamine.

CONTENTS

INTRODUCTION

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Fever in acute myocardial infarction (AMI) is a normal response to tissue necrosis. It usually takes a characteristic course, duration and amplitude. However, some patients hardly develop any pyrexia while others develop high and prolonged fever.

The development of complications, either cardiac or extracardiac, may be one of the factors which can alter this usual fever course in AMI.

Although some previous studies have attempted to identify this usual course of fever and its significance, little is known about the individual susceptibility, type of infarction, extent of infarction, age, sex, and other associating diseases on the severity and duration of fever, and whether it can predict complications or prognosis in patients with AMI.

Finding the relation of fever to different laboratory investigations in patients with AMI may be of great help in early detection and management of complications, whether cardiac or extracardiac.

AIM OF WORK

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The aim of this prospective study was to investigate the significance of fever in AMI, its relation to different factors, like age, type and extent of infarction, drug intake and complications.

Also, this work aimed at investigating the relation of this fever to different laboratory findings and whether it can predict complications, cardiac or extracardiac, and prognosis in patients with AMI.

REVIEW OF LITERATURE

GENERAL ACCOUNTS ON FEVER

Normal body temperature:

Individuals maintain their body temp. at about 37°C despite wide variations in environmental temp. For some individuals, normal body temp. can be below or above 37°C without constituting a pathologic process. Rectal temp. is usually 0.5 to 1.0°F higher than oral temp. There is a considerable variation in temp. in a given individual. During 24 hours period, body temp. varies from low point in early morning to the highest levels at 4 to 6 P.M. (Dinarello, 1985) or at 6 to 10 P.M. (Petersdrof, 1980). This diurnal variation of normal body temp. appears in a pattern that is inverse of endogenous corticosteroid release. The amplitude of this diurnal variation, also called "Circandian temp. rhythm", is about 0.6°C and individuals retain their circandian rhythm throughout life despite intervening bouts of prolonged illness. Although it has been postulated that this diurnal variation is dependent upon increasing activity during the day and rest at night, the pattern is not reversed in persons who work at night and sleep during the day for long

periods of time. Severe or prolonged exercise can produce considerable elevation in body temp. (Petersdrof, 1980 ; Hoeprich, 1980 and Dinarello, 1985).

Definition of fever:

An elevation above the normal amplitude of daily temp. for an individual is considered fever. During fever, the morning low and evening high temp. pattern can still be observed. (Dinarello, 1985).

Thermoregulation:

Fever is best understood at the hypothalamic level. The thermoregulatory center located in the anterior hypothalamous regulates internal temp. at about 37°C primarily by its ability to balance heat production and peripheral heat loss. During fever, the thermostat setting in the hypothalamic center shifts upwards e.g. from 37 to 39°C. This results in signals to increase heat production and decrease heat loss. Heat production from shivering muscles and heat coservation from peripheral vasoconstriction continue until the temp. of the blood supplying the hypothalamous matches the higher thermostat setting. (Dinarello, 1985). For example in

patients with thyrotoxicosis, there is exaggerated heat production, while in patients with congestive heart failure (CHF) there is an impairment in heat dissipation as a result of diminished cardiac out put (COP) and decline in cutaneous blood flow. Individuals taking drugs which impair sweating, as atropine, scopolamine, phenothiazine, monoamine-oxidase inhibitors, glutethemide, lysergic acid diethylamide (LSD), amphetamines and inhalation anaesthetics, may have fever in warm weather. (Petersdrof, 1980 and Dinarello, 1985).

Pathogenesis of fever:

Fever is a consequence of many stimuli, including bacteria and their endotoxins, viruses, yeasts, antigen-antibody reactions, hormonal substances, drugs and synthetic polynucleotides. These substances, which have been termed collectively "Exogenous pyrogens", are both diverse and complex. It has been postulated that they act through an intermediary substance termed "Endogenous pyrogen". (Petersdrof, 1980).

Endogenous pyrogen (E.P.):

It is a basic protein of low molecular weight, first described by Beeson in 1948. Subsequent animal and human

studies established the importance of E.P. in mediating fever and as being responsible for the upward setting of the hypothalamic thermostat. This substance is produced in response to infections, toxic substances, or immunologic reactions.

E.P. is a product of leukocytes and macrophages including Kupffer cells, splenic sinusoidal cells, alveolar macrophages and peritoneal lining cells. E.P. has not been isolated from lymphocytes, but these cells may react with antigens and through the action of lymphokinase, they may stimulate neutrophils and macrophages and possibly monocytes to release E.P.

When stored in cells, E.P. is not present in active form. Rather, its release requires synthesis of new messenger RNA and protein. Because it is present in such small amounts, E.P. has been difficult to detect in human serum or exudate. Once released, E.P. probably acts on the thermosensitive neurons in the preoptic region of the anterior part of hypothalamous. These neurons control the constancy of blood temp. and are the point where fever is initiated. The action of E.P. on the hypothalamous is by no means simple. It appears

as if there must be a release of serotonin (5-HT), a thermogenic amine, to mediate the febrile response. In addition, E.P. induces synthesis of prostaglandines E_1 and E_2 in the hypothalamous where they function as central transmitters in the initiation of fever. The action of antipyretics, as aspirin, has been shown to be the result of their ability to block prostaglandin synthesis. The prostaglandins, in turn, lead to an increase in cyclic-AMP which may be important in the metabolic processes that are operative in fever. (Petersdrof, 1980 and Dinarello, 1985).

Manifestations of fever:

The subjective symptoms of fever include sensation of feeling cold or warm, headache, myalgias, arthralgias, and general malaise. The objective signs besides elevated body temp. include increased respiratory rate, widened pulse pressure, and rapid heart rate. Laboratory findings are altered in fever. The most notable of these is elevated erythrocytic sedimentation rate (ESR) resulting from increased haptoglobin, fibrinogen, ceruloplasmin, and C-reactive protein levels (Dinarello, 1985). In some patients, the neutrophil count is elevated.