CK-MBmass AS AN INDICATOR OF ACUTE MYOCARDIAL INFARCTION SIZE

Thesis

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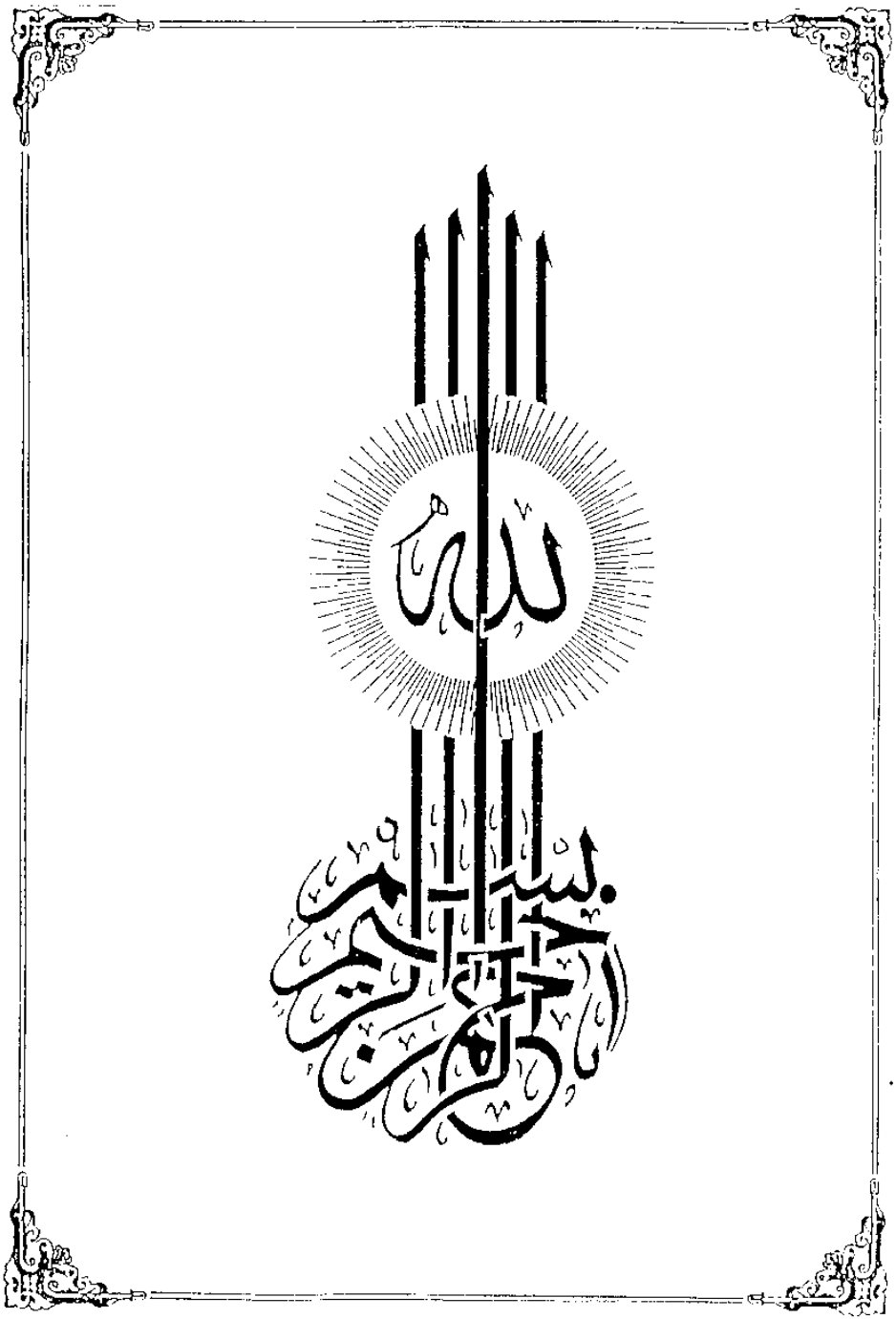

CONTENTS

CONTENTS

CONTENTS

LIST OF ABREVIATIONS

FPIA	Fluorescence polarization immunoassay
HK	Hexokinase
ASE	American society of echocardiography
AMI	Acute myocardial infarction
ECG	Electrocardiogram
CCU	Coronary care unit
CK	Creatine kinase
LD	Lactate dehydrogenase
AST	Aspartate aminotransferase
cTn-I	Cardiac troponin-I.
cTn-T	Cardiac troponin-T.
MLC	Myosin light chain
MHC	Myosin heavy chain
GPBB	Glycogen phosphorylase BB
CABG	Coronary artery bypass grafting
PTCA	Percutaneous transluminal coronary angioplasty
CrP	Creatine phosphate
ADP	Adenosine diphosphate
ATP	Adenosine triphosphate
NAD	Nicotinamide adenine dinucleotide
G-6-P	Glucose-6-phosphate
G6PD	Glucose-6-phosphate dehydrogenase
AMP	Adenosine monophosphate
Ap5A	Diadenosine pentaphosphate
MEIA	Microparticle capture enzyme immunoassay
FEIA	Fluorometric enzyme immunoassay
2DE	Two dimensional echocardiography.

LIST OF FIGURES

LIST OF TABLES

LIST OF TABLES

Introduction and Aim of the Work

INTRODUCTION AND AIM OF THE WORK

Acute myocardial infarction is a serious common life threatening condition worldwide. And one of the most important determinants of prognosis is myocardial infarct size as it correlates with acute and long term morbidity and mortality, moreover it is important in assessing interventions in patients with acute myocardial infarction **(Roberts and Ishikawa, 1983).**

For long times trials of accurate sizing of myocardial infarcts were thoroughly investigated and laboratory as well as non laboratory methods were studied. Non laboratory methods include the electrocardiography, the echocardiography, myocardial scintigraphy and nuclear magnetic resonance **(Nixon et al., 1980).**

Non laboratory methods, however, have many technical difficulties as they require expertise on the part of the examiner and interpreter. Moreover they may be time consuming, largely subjective and sometimes inconclusive, in addition, some are very sophisticated and cannot be done on routine basis especially for a critically ill patient **(Bakker et al., 1993).**

On the other hand, laboratory methods include measurement of serum cardiac markers which are intracellular macromolecules that leak out of the cardiac muscle cells as a result of damage of their sarcolemmal membrane caused by ischemia **(Ellis, 1991).**

Several tedious and complicated formulas were invented for measurement of infarct size using total CK, and then CK-MB activity. However question arose about their validity after introduction of thrombolytic therapy **(Vatner et al., 1977)**.

The newly developed determination of the mass concentration of creatine kinase-MB represents a recent advance towards greater specificty. It represents measurement of enzyme protein quantity intead of its activity which may be altered after its release in the serum **(Delanghe et al., 1990)**. And unlike the determinations of activities of CK and its MB isoenzyme, it is not prone to interference from hemolysis or atypical and macromolecular forms of CK **(Bakker et al., 1993)**.

CK-MB mass assay was proved to be more sensitive and specific than total CK or CK-MB activity for the early diagnosis of acute myocardial infarction and for assessment of reperfusion after thrombolytic therapy **(Mair et al., 1995).**

Aim of the Work:

The aim of this study is to evaluate CK-MB mass measurement in cases of AMI as a good indicator for the size of infarcted area in comparison to the routine measurement of the enzyme activity of total CK, CK-MB , LD and AST.

Also to find how it will correlate and the other cardiac enzymes to the size of myocardial infarction as shown by Echocardiography.

Review of Literature

ACUTE MYOCARDIAL INFARCTION

Acute myocardial infarction (AMI) is one of the most serious medical events worldwide. In the United States 1,500,000 patients suffer from acute myocardial infarction annually and approximately one fourth of all deaths are due to AMI. **(American Heart Association, 1990).**

More than 60 percent of the deaths associated with AMI occur within one hour of the event and are attributable to arrhythmias, especially ventricular fibrillation.**(Pasternak et al., 1992).**

Clinicians being faced with AMI, their goal of therapy is to salvage myocardium. New therapeutic interventions, specifically thrombolytic agents such as streptokinase and tissue plasminogen activator, have become available recently to restore coronary artery blood flow (reperfusion), to limit the size of infarction and to reduce the incidence of morbidity and death **(Bang et al., 1989).**

Causes of Acute Myocardial Infarction:

Almost all myocardial infarction cases result from *atherosclerosis* of the coronary arteries, generally with superimposed coronary thrombosis **(Pasternak et al., 1992).**

Numerous other pathological processes can, on occasion, involve the coronary arteries and result in myocardial