سامية محمد مصطفى

شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم

سامية محمد مصطفى

شبكة المعلومات الجامعية

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

سامية محمد مصطفى

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها علي هذه الأقراص المدمجة قد أعدت دون أية تغيرات

يجب أن

تحفظ هذه الأقراص المدمجة بعيدا عن الغبار

سامية محمد مصطفى

شبكة المعلومات الجامعية

بعض الوثائق الأصلية تالفة

سامية محمد مصطفى

شبكة المعلومات الجامعية

بالرسالة صفحات لم ترد بالأصل

MYOGLOBIN VERSUS CARDIAC ENZYMES IN EARLY DIAGNOSIS OF ACUTE MYOCARDIAL INFARCTION

Thesis

**Submitted to the Faculty of Medicine,
University of Alexandria
In partial fulfilment of the
requirements of the degree of**

MASTER OF

CLINICAL AND CHEMICAL PATHOLOGY

By

Mona Sobhi Arian Botros
MBBCh Alex

**Faculty of Medicine
University of Alexandria**

1995

B
١٤١١٩

SUPERVISORS

Prof. Dr. Aly Ahmed Abdel Aal
Professor of Clinical Pathology
Faculty of Medicine
Alexandria University

Dr. Salah Ahmed Marzouk
Ass. Prof. of Clinical Pathology
Faculty of Medicine
Alexandria University

Dr. Mostafa Mohamed Nawar
Ass. Prof. of Cardiology
Faculty of Medicine
Alexandria University

ACKNOWLEDGEMENT

I would like to express my gratitude and deep appreciation to Prof. Dr. Aly Ahmed Abdel Aal, Professor of Clinical Pathology for his guidance and follow up and his sincere supervision, valuable suggestions enormously shared in completing this work.

Deepest thanks to Dr. Salah Marzouk, Assistant Professor of Clinical Pathology for his very close supervision, active guidance in revising this thesis.

I am greatly grateful to Dr. Mostafa Nawar, Assistant Professor of cardiology for his helpful advice, expert guidance and cooperation.

I am deeply thankful to Dr. Zeinab Ibrahim, Lecturer of Clinical Pathology for her great guidance and support.

CONTENTS

Chapter		*Page*
I.	*Introduction*	*1*
	- Enzymes	*2*
	- Myoglobin	*20*
	- Other laboratory measurements	*25*
	- D-Dimer	*27*
	- New cardiac contractile proteins	*29*
	- Electrocardiographic findings	*33*
	- Variant angina pectoris	*35*
II.	*Aim of the work*	*36*
III.	*Material*	*37*
IV.	*Methods*	*39*
V.	*Results*	*60*
VI.	*Discussion*	*86*
VII.	*Summary*	*93*
VIII.	*Conclusions*	*95*
VIII.	*References*	*96*
	Arabic Summary	—

LIST OF TABLES

No.		Page
I.	Comparison between serum total CK activity among studied cases.	61
II.	Comparison between serum total LD among studied cases.	63
III.	Comparison between serum CK-MB activity among studied groups.	64
IV.	Comparison between serum ALT level among studied cases.	65
V.	Comparison between serum AST activity between studied groups.	69
VI.	Correlation between admission time versus levels of serum parameters studied.	70
VII.	Comparison of serum turbiquant myoglobin level among the three studied groups.	71
VIII.	Comparison between latex myoglobin in the studied groups.	73
IX.	correlation between myoglobin and total CK in AMI group.	77
X.	Sensitivity and specificity of turbiquant myoglobin and total CK.	80
XI.	Sensitivity and specificity of turbiquant myoglobin and CK-MB.	81
XII.	Sensitivity and specificity of turbiquant and latex myoglobin.	82

XIII. Laboratory findings of total CK, CK-MB and CK-MB/total CK ratio in the three studied groups. 83

XIV. Laboratory findings of total LD, ALT and AST in the three studied groups. 84

XV. Results of turbiquant and latex myoglobin in the three studied groups. 85

LIST OF FIGURES

No.		Page
1.	The electrophoretic separation of serum CK isoenzyme.	8
2.	Serum LD isoenzyme pattern obtained with a thin layer agarose gel electrophoresis system.	16
3.	Model of myoglobin at low resolution.	21
4.	Bar code for turbiquant myoglobin.	42
5.	Measuring Unit in Turbi Time System.	45
6.	Immunoprecipitation reaction in Turbi Time System.	47
7.	Comparison between mean total CK level in the three studied groups.	66
8.	Comparison between mean CK-MB level in the three studied groups.	67
9.	Comparison between mean turbiquant myoglobin level in the three studied groups.	72
10.	Scattered diagram between turbiquant values and myoglobin latex in AMI.	75
11.	Correlation between total CK and turbiquant myoglobin in AMI.	78

CHAPTER I

INTRODUCTION

INTRODUCTION

Coronary heart disease is a major cause of disability and mortality in human, accounting for more than 27% of all deaths in the UK and, it is estimated that more than 300,000 people suffer heart attacks each year. In the United States nearly 1,500,000 patients suffer from acute myocardial infarction (AMI) annually that represents approximately 25% of all deaths.[1]

More than 60% of the deaths associated with AMI occur within one hour of the event and are attributable to arrhythmias and ventricular fibrillation.[2]

In the 1980, before the introduction of thrombolytic therapy, the mortality rate during hospitalization and the year following infarction were approximately 10 percent each.[3,4] In the United States, the decline in death rate from coronary artery disease has been accompanied by diminished mortality from AMI. This fall in the mortality appears to be caused by two factors: A fall in the incidence of AMI by 25 percent or more[5] and a similarly marked fall in the case fatality rate once AMI has occurred.[5-8]

Laboratory findings in AMI:

A) Enzymes.

B) Myoglobin.

C) Other laboratory measurements.

D) D-Dimer

E) New cardiac contractile proteins.

A) Enzymes:

Enzymes are proteins synthesized intracellularly, and most of them carry out their function within the cells in which they are formed.[9] The measured level of activity of an enzyme in blood is the result of the balance between the rate at which it is entering the circulation from its cells of origin and the rate at which it is being inactivated or removed.[9]

Clearance of enzymes:

Most enzyme molecules are not small enough to pass through the healthy glomerulus of the kidney, and therefore urinary excretion is not a major route for elimination of enzymes from the circulation.[9] Evidence now suggests that inactivation begins in the plasma and that inactivated enzymes are rapidly removed, probably by the reticuloendothelial system such as the bone marrow, spleen and liver (Kupffer's cells) or, to a lesser extent, by nearly all cells in the body.[10] The mechanism appears to be by