CLINICAL SIGNIFICANCE OF PLASMA THROMBOMODULIN LEVEL IN DIABETES MELLITUS

Thesis

Submitted for The Fulfillment

Of

Master Degree in Internal Medicine

By

Salah Eldin Ahmed Nasr
M.B., B. Ch.

Supervisors

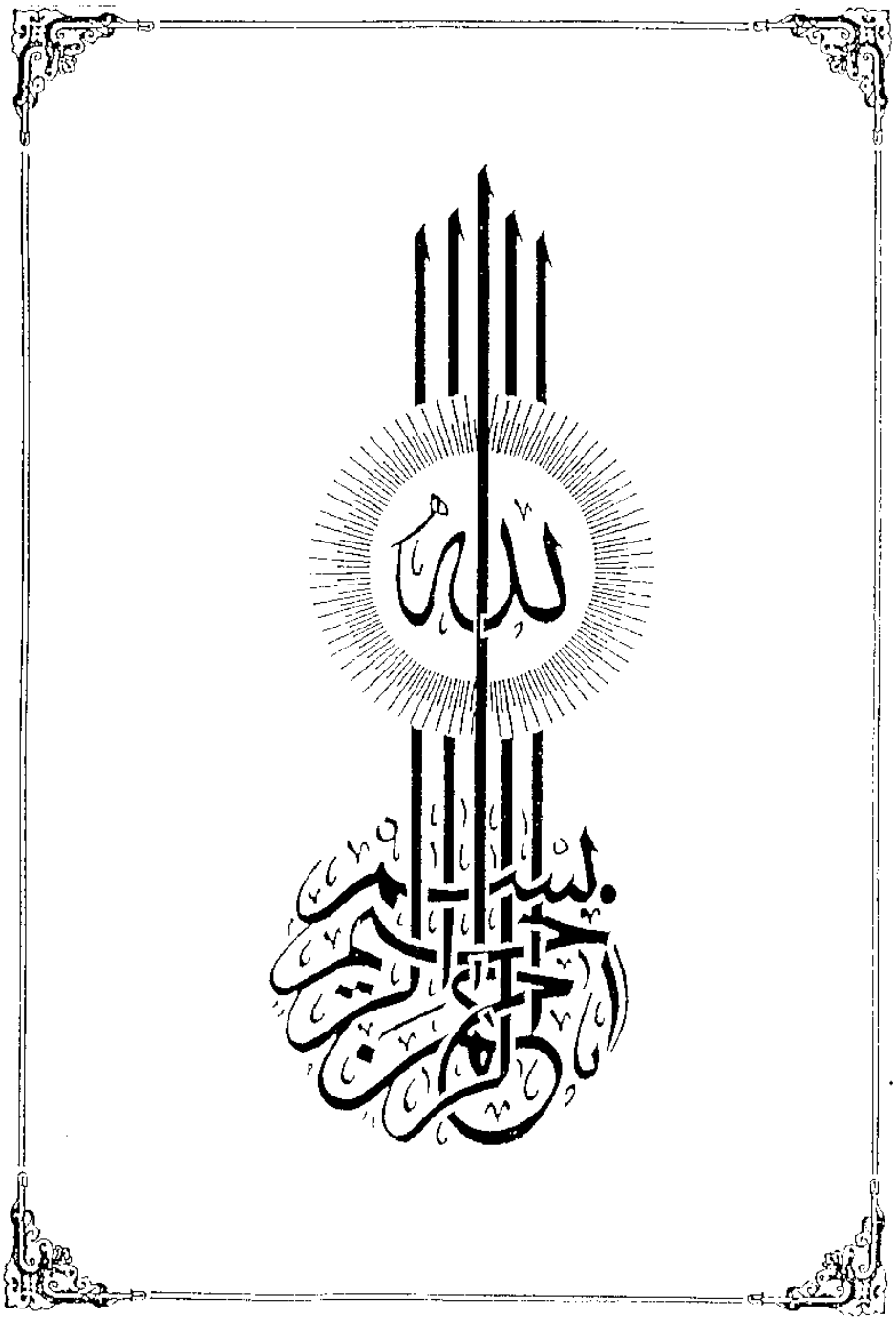
Dr: Enas. A. Assfour
Professor of Internal Medicine
Ain Shams University

Dr. Noura M. Elkholy
Assistant Professor of Biochemistry
Ain Sham University

Dr. Hanan Hamed
Lecturer of Internal Medicine
Ain Shams University

Department of Internal Medicine
Faculty of Medicine, Ain Shams University

1997



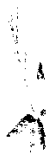

بسم الله الرحمن الرحيم

قَالُوا سُبْحَانَكَ
لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا
إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ

صدق الله العظيم
سورة البقرة آية ٣٢


To..... *My Mother*

CONTENTS



INTRODUCTION

INTRODUCTION

Thrombomodulin (TM) is a surface glycoprotein which neutralizes thrombin clotting and platelet aggregatory activity and acts as a cofactor for thrombin catalyzed activation of anticoagulant protein C.

Circulating thrombomodulin is not constantly secreted by endothelial cells but results from cellular damage. Unlike von Willebrand factor and tissue plasminogen activator, circulating TM is not a maker of endothelial cell stimulation, and the endothelial membrane TM is hardly leaked in absence of endothelial injury. The liver is considered the major site of clearance of circulating TM, and patients with liver disease especially fulminant hepatitis were reported to have elevated plasma TM levels **(Takahashi et al., 1992)** In addition the change in urinary excretion of TM may modify its plasma **(Ishii and Majerus, 1985)**.

Plasma TM has been frequently reported to be elevated in a variety of diseases including DIC, pulmonary thromboembolism, acute respiratory distress, chronic renal failure, collagen diseases, hematological malignancies, as well as in thrombotic diseases. These

findings suggest that the endothelial damage may occur more frequently than hitherto supposed **(Ta Kahashi et al., 1992).**

Subclinical elevation of urinary albumin, microalbuminurea, is well described as being a predictor for cardiovascular disease in both diabetic and nondiabetic subjects. It reflects the presence of generalized vascular damage and represents a crucial event in the natural histories of diabetes mellitus and essential hypertension **(Yudkin et al., 1988).** An increased risk of cardiovascular mortality in both insulin - dependent (IDD) and noninsulin-dependent (NIDD) diabetics has been recorded in association with microalbuminurea and the risk becomes 20 - to 40 fold higher with development of clinical proteinuria, macroalbuminurea in IDD **(Borch et al., 1985).**

AIM OF THE WORK