BLOOD PLATELETS IN PREGNANCY INDUCED HYPERTENSION

THESIS

Submitted in partial fulfilment of M.D. Degree

IN

(Obstetrics and Gynaecology)

BY

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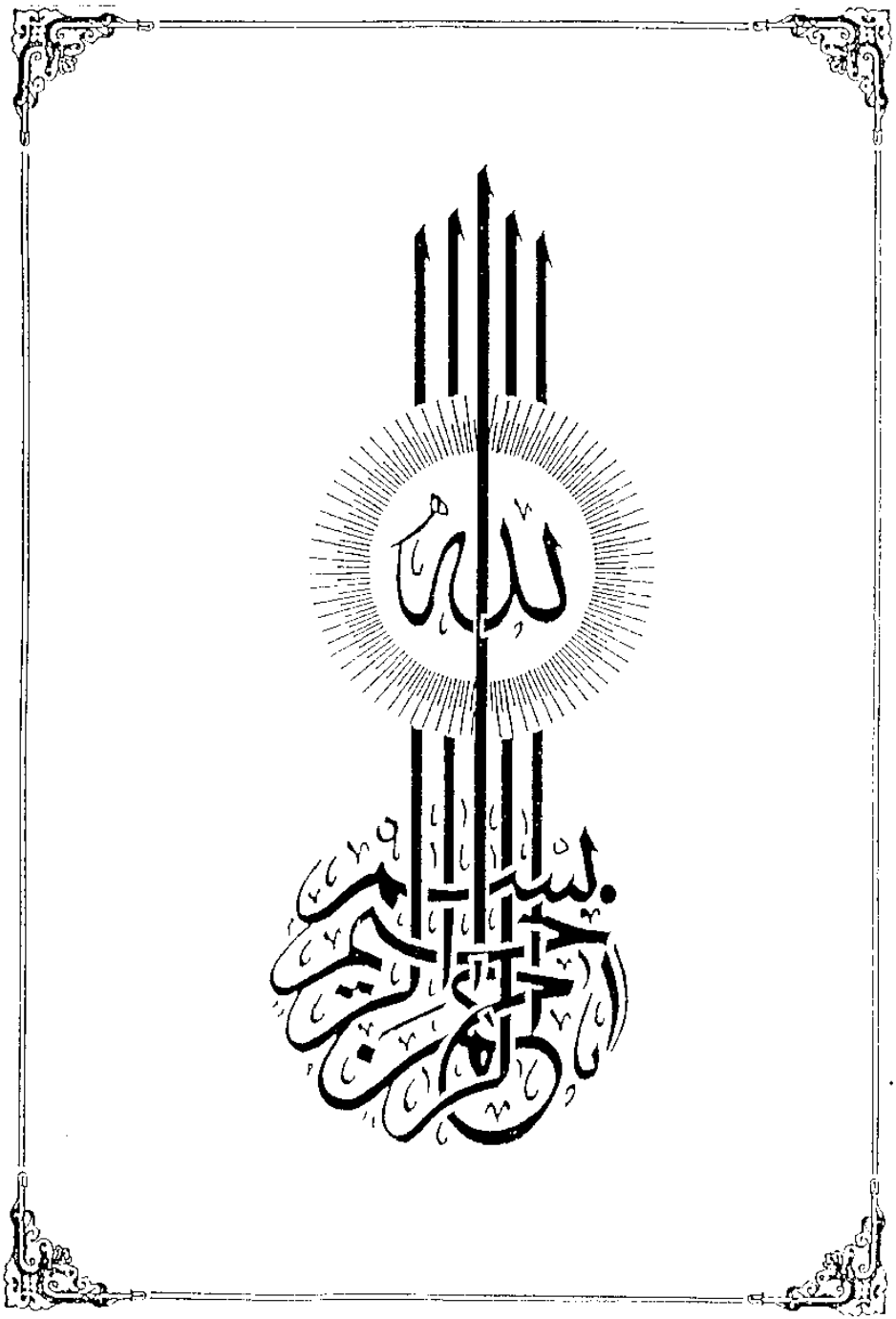

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CHAPTER (I)

INTRODUCTION

Aim of the work

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INTRODUCTION

Pregnancy induced hypertension is a hypertensive disorder unique to pregnancy occurring primraily in primiparae. Its complications are well understood, but the pathogenesis remains obscure (Gibson, 1982)[81]. Pregnancy induced hypertension involves mild or severe pre-eclampsia and eclampsia (Symonds, 1979)[235]. Platelet consumption is an early feature of the disease (Redman and Bonnar, 1978)[187].

In 1922, Stahnke[229] stated that thrombocytopenia was present in severe cases of toxemia of pregnancy associated with increased intravascular coagulation and haemolysis.

It was realized that platelets are not merely trapped passively in pregnancy induced hypertension but their aggregation and the release of their constituents may contribute to the pathogenesis and deterioration of the disease (Wigham, 1978)[268].

Aggregation of platelets in pregnancy induced hypertension may be the result of intravascular coagulation (Schneider, 1947)[208] or interaction between platelets and endothelium (Pritchard, 1976)[184] or autoimmune reaction (Bern, 1981)[17].

Platelets' 5-hydroxytryptamine might play a role in the pathogenesis of pregnancy induced hypertension. Increased levels of 5-hydroxytryptamine have been reported in the placenta of women with severe pre-eclampsia (Senior ,1963)[212].

Research in the role of prostaglandins in vascular disease and in the physiology and pathology of the maternal and placental circulation has renewed interests in platelets and their significance in the pathogenesis of pregnancy induced hypertension (Wallenburg,1981)[245].

Speroff (1973)[227] proposed that inadequte prostaglandin synthesis might be a factor in the development of pregnancy induced hypertension.

Hamberg(1975)[93] demonestrated conversion of prostaglandin endoperoxides to thromboxane A_2 in lungs and platelets.Thromboxane A_2 is a powerful platelet aggregating agent and vasconstrictor (Moncada and Vane,1979)[150].

Wallenburg and Rotman(1981)[248] demonstrated enhanced reactivity of platelet thromboxane A_2 pathway in normotensive and hypertensive pregnant women complicated by chronic placental insufficiency.

Scott (1984)[210] found that in normal placenta production rates of thromboxane A_2 and prostacyclin were equivelant but in placentas of pregnancy induced hypertension, thromboxane A_2 production was twelve fold greater than prostacyclin production.

Walker(1984)[244] reported that labetalol could control blood pressure and reduce platelet consumption in pregnancy induced hypertension.

Aim of the Work

Cases of pregnancy induced hypertension were investigated for platelet function (i.e. aggregation) and structure using Electron Microscopy in comparison with normotensive non pregnant and normotensive pregnant women.

Thesis Contents

In chapter II, morphological description for blood platelets, physiology of blood platelets and prostaglandins.

Chapter III : Review of literature about blood platelets in pregnancy induced hypertension, Antithrombin III, prostaglandins and uteroplacental vasculature.

Chapter IV : Subjects and description for different methods of investigations especially platelet aggregation and preparation of blood platelets for Electron Microscopy.

In chapter V. the results are presented and discussed in chapter VI. In chapter VII summary and conclusions of the present study are reported.

CHAPTER (II)

Review of morphology and physiology of blood platelets

Prostacyclin and thromboxane

Platelet inhibitor drugs

BLOOD PLATELETS

Life Cycle of Platelets:

Platelets are formed in the golgi region of the cytoplasm of megakarocytes and released into the blood by fragmentation of the cytoplasm. Most of the platelets are produced by megakarocytes in the bone marrow, but few may be derived from pulmonary megakarocytes (Oberling, 1973)[174].

Thrombocytopiesis is controlled by thrombopoietin. After acute platelet depletion 4-14 days are required to restore the platelet count to normal (Schulman et.al., 1960) [209].

After release from megakarocytes, the platelets enter a non exchangeable pool in the spleen for two days, then they enter the circulation, where they live for seven to ten days until, they are destroyed by reticulo-endothelial system. Platelets die as they get older, but they also are randomly destroyed in the process of haemostasis and also, perhaps, in endothelial cell repair (Johnson , 1971) [114].

Light Microscopy of Platelets :

Platelets are little ovoid non nucleated bodies, two