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Evaluation of a D.dimer latex agglutination slide test as a rapid test for abruptio placentae

A thesis submitted for the partial fulfillment of master degree in obstetrics and gynaecology

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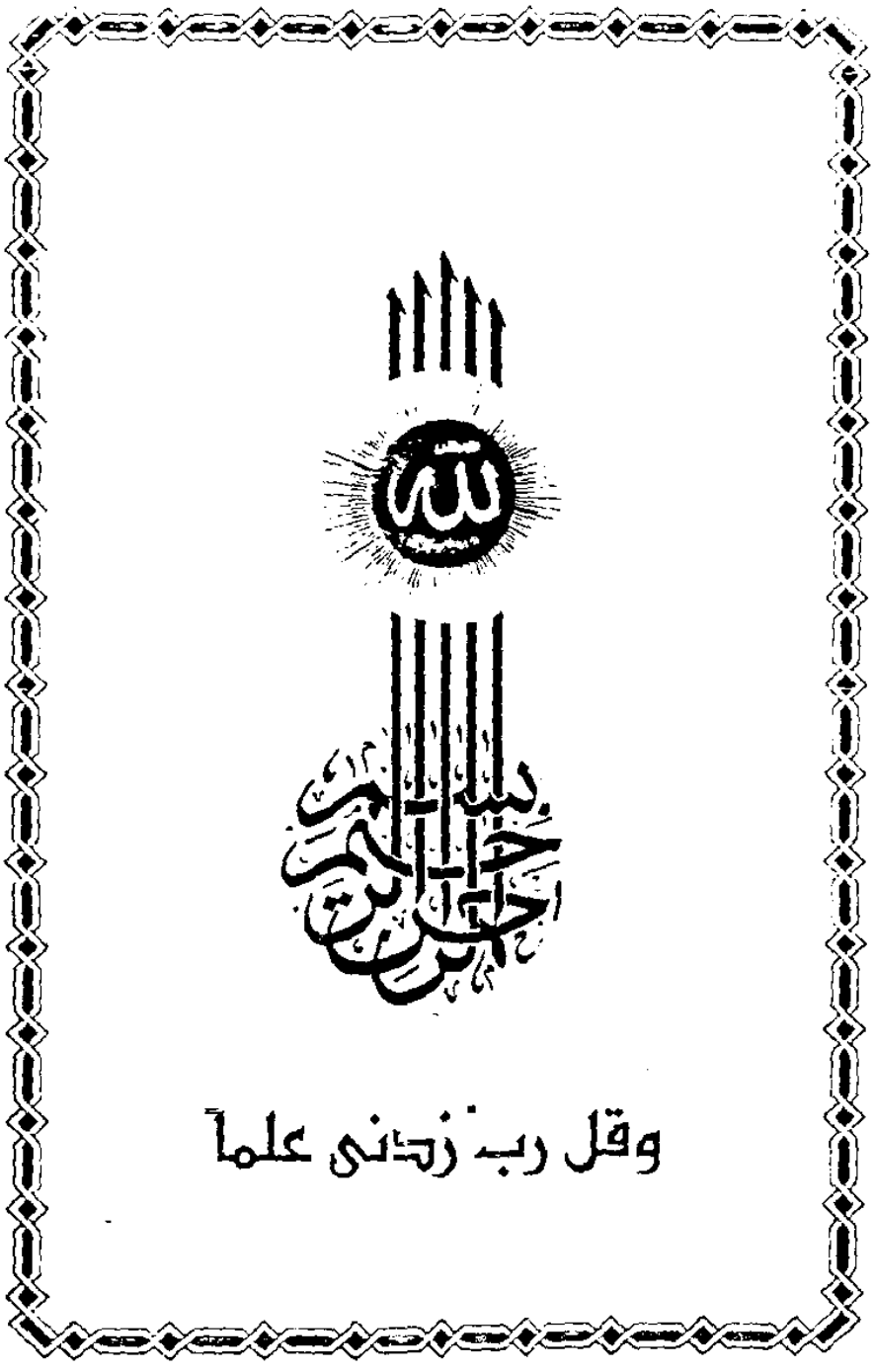

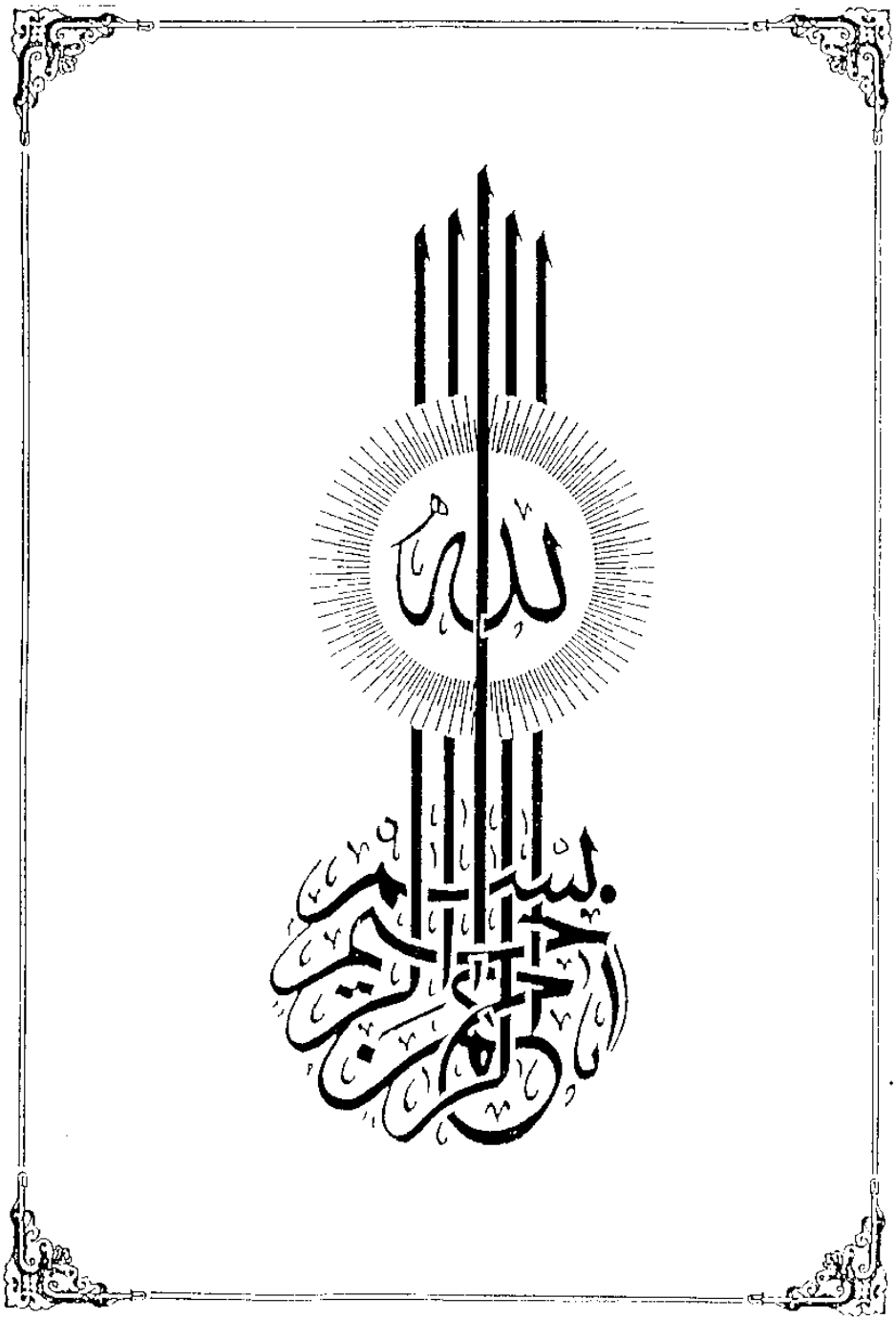

To

MY PARENTS
AND
MY SONS

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Contents

Introduction

Introduction

The separation of the placenta from its site of implantation before the delivery of the foetus has been variously called: placental abruption, abruptio placentae, ablatio placentae, accidental haemorrhage and premature separation of the normally implanted placenta (williams, 1993)

It is emphasized that the signs and symptoms with abruptio placentae can vary considerably, as external bleeding can be profuse, yet placental separation may not be so extensive as to compromise the foetus directly or there may be no external bleeding but the placenta may be completely sheared off and the foetus dead as a direct consequence. Back pain, uterine tenderness, rapid uterine contraction or persistent uterine hypertonus were detected singly or more often in combination. Retroplacental haematoma can be recognized sonographically in only 1 of 59 cases. Only 25 percent of women with placental abruption could be confirmed by sonography. Importantly negative findings with ultrasound examination do not exclude life threatening placental abruption (Hurd and co-workers, 1983).

By far the most commonly associated condition is either pregnancy induced or chronic hypertension. In 50 percent of women with severe placental abruption so as to kill the foetus maternal hypertension was apparent once the depleted intravascular compartment was adiquately refilled. (Pritchard and co-workers, 1991).

Abruptio placentae is the most frequent obstetric cause of coagulation failure which appears to be related to the degree of placental separation. (Michael de swiet, 1989).

An important consequence of intravascular coagulation is the activation of plasminogen to plasmin which lyses fibrin microemboli to maintain patency of the microcirculation.

The coagulation defects most often develop within the first few hours and perhaps even minutes, after the onset of pain or bleeding. (Bonnar J, 1969)

As abruptio placentae progress, there is an increasing placental site haemorrhage and local consumption of factors responsible for clot formation, stabilization and lysis.

In this process, fibrinogen is converted to fibrin, fibrin complexes are formed and then cleaved, which results in the generation of D-dimer. Therefore the detection of D-dimer may serve as an early marker for abruptio placentae (Nolan TE, 1993).

Maternal mortality rates ranging from 0.5-5% are currently reported from various parts of the world. Most women die of haemorrhage, (immediate or delayed) or cardiac or renal failure. A high degree of suspicion, early diagnosis and definitive therapy

should reduce the maternal mortality rates to 0.5-1 percent. (Martin L. Pernoll, 1991)

In 50% foetal distress is noted early due to decreased metabolic exchange from decreased placental surface, maternal haemorrhage and decreased uterine perfusion. (Martin. L.Pernoll,1991)

Aim of the work

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To evaluate D-dimer latex agglutiation slide test as a rapid test for diagnosis of placental abruption and hence better management.

Review of Literature

CHAPTER I

Haemostasis in pregnancy :

In a normal pregnancy, the components of coagulation and blood volume are altered to facilitate haemostasis. Blood volume of the normal woman late in pregnancy expands approximately 1500 mL. Factors I (Fibrinogen), VII, VIII, IX and X are elevated, but changes in the platelet count are nlegligibe. Throughout the antepartum period, plasminogen levels are elevated, although plasmin activity is relatively normal. So pregnancy said to be hypercoagulative state as the fibrinogen is doubled, all coagulation factors increase except factor XI & XIII. On the other side the fibrinolytic system is inhibited during pregnancy.

When stress such as haemorrhage occurs, plasminogen-to plasmin conversion is stimulated, thus, coagulation and fibrinolysis are initiated. The greater amounts of available clotting factors may be initiated whenever physiologic insult occurs (Lavery, 1982, Pritchard and Mac Donald, 1980)

Normal Blood Coagulation :

The most widely accepted theory of blood coagulation is the Bennet and Ratnoff (1972) cascade theory, which divides the blood coagulation system into two components :

(1) The intrinsic system.

(2) The extrinsic system.

Common regulatory mechanisms work through the clotting system, and a common convergent pathway brings the intrinsic and extrinsic systems together for the final stages of clot formation.

Intrinsic pathway :

The intrinsic pathway is so named because its activation is dependent on a substance found in the plasma when stimulated by pathologic events, factor XII converts factor XI (Plasma thromboplstin antecedent) to its activated form. This disruption involves the release of collagen or subendothelial substances, which trigger the intrinsic system. Activated factor XI in the presence of Ca^{2+} causes activation of factor IX (Christmas factor) in turn, activated factor IX in the presence of Ca^{2-}, phospholipid, and thrombin - modified factor VIII causes activation of factor X (Stuart - Prower factor) (Bennett and Ratnoff, 1972) Figure 1.

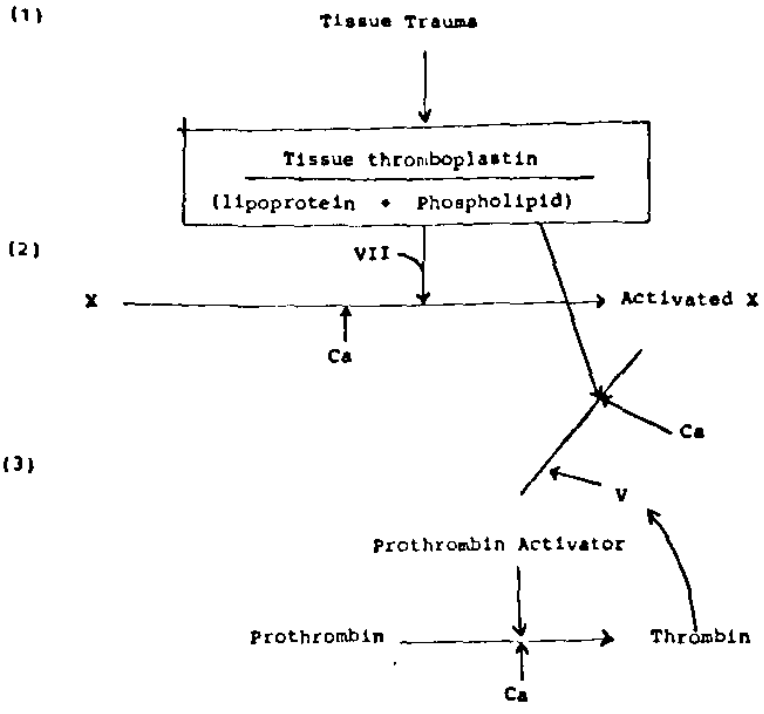

Figure (1): Clotting mechanism-intrinsic pathway (Guyton AC; 1978.)