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شبكة المعلومات الجامعية التوثيق الالكتروني والميكروفيلم





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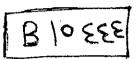
بالرسالة صفحات

لم ترد بالأصل



Plasma Fibronectin in Preeclampsia and Intrauterine Growth Retardation

Thesis Submitted for Partial Fulfillment of Master Degree In Obstetrics and Gynaecology



By

Ahmed Hosny Ahmed.

M.B.B. Ch (Ain Shams University)

Under Supervision of

Prof. Dr. Mohamed Nagy El-Makhzangy
Prof. of Obstetrics and Gynaecology

Faculty of Medicine, Ain Shams University

and

Dr. Ahmed Mohamed Nor El Din Hashaad

Lecturer of Obstetrics and Gynaecology
Faculty of Medicine

Ain Shams University

Dr. Nahla Mohamed Zakaria

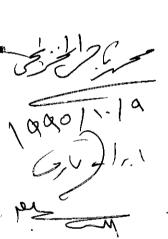
Lecturer of Clinical Pathology

Faculty of Medicine

Ain Shams University

Cairo

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بالفالقالك عَلَمُ الإنسَيَّانَ مَإلَمْ يَعَبُّلُمُ

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ARARIC SIMMADV

INTRODUCTION

Fibronectins are large, usually dimeric glycoproteins (molecular weight approximately 450,000D), that are found both in a soluble form in plasma (plasma fibronectin) and in a larger non soluble form in the extracellular matrix (cellular fibronectin) (Moses et al., 1980, Rouslahti et al., 1988).

Plasma fibronectin is synthesized primarily hepatocytes (Tamkun et al., 1983), and is involved opsonic activities with the reticuloendothelial system and in clot stablization (Schwarzbeur et al., 1984). Cellular fibronectin synthesized by fibroblast, endothelial cells, and astroglial cells among others, is involved in cell adhesion, migration, growth and differentiation (Rouslati et al., 1973). The various different fibronectins are drived from a signle fibronectin gene and are composed of multiple domains, the sequence and the presnece or absence of the various domains accounts for different functional the activities of different fibrenctins (Moses et al., 1980).

There are at least 12 versions of fibronectin. although the firbonectin gene itself does not vary (Hynes et al., 1986). Different cells types (hepatic, fibroblast, and

----- Introduction (1) -----

endothelial) may synthesize different fibronectin variants, one of these variants is a soluble plasma fibronectin containing an extra type III domain (ED I+), predominantly in large vessel endothelial cells (Vartio al., 1987), and in very small quantities in platelets (Paul et al., 1986). A growing body of evidence indicates that endothelial injury or activation is an important pathophysiologic feature of pre-eclampsia (Friedman et al., The Cellular iso form of fibronectin (cFN), is present in endothelial cells and endothelial matrix in vivo (Vartio al., 1987), and is elevated in clinical situations characterized by substantial endothelial injury, such as vasculitis, sepsis and crush injury (Rouslahti et al., 1988). The pathogenesis of this endothelial injury is poorly understood, but appears to be related to antecedent trophoblastic hypoperfusion (Mosses et al., 1980). Impaired trophoblastic invasion of maternal spiral arteriols (which presumbly results in reduced trophoblastic perfusion), has been observed in pregnancies complicated by preeclampsia and intrauterine growth retardation (Rouslahti et al., 1973). Data demonstrating the presence or absence of endothelial injury in pregnancies with IUGR are limited.

----- Introduction (2) ------



AIM OF THE WORK

The aim of this study, is to determine the presence and the degree of endothelial injury, by measuring plasma concentrations of fibronectin, in pregnancies complicated by preclampsia and/or intrauterine growth retardation.

----- Aim of the work (3)

