THE PLACENTAL BED BIOPSY IN HYPERTENSIVE PATIENTS

THESIS

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By

Nahed Abd El Aleem Ali

M.B.,B.Ch

Supervised By

Dr. Mohamed Mehana

Ass. Prof. of Obstetric Gynaecology

Ain Shams University

Dr. Gamal Abd El Salam Wafa

Lecturer of Obstetric & Gynaecology

Ain Shams University

Dr. Shadia Hussein Mabrouk

Ass. Prof. of Pathology

Ain Shams University

Faculty of Medicine
AIN SHAMS UNIVERSITY
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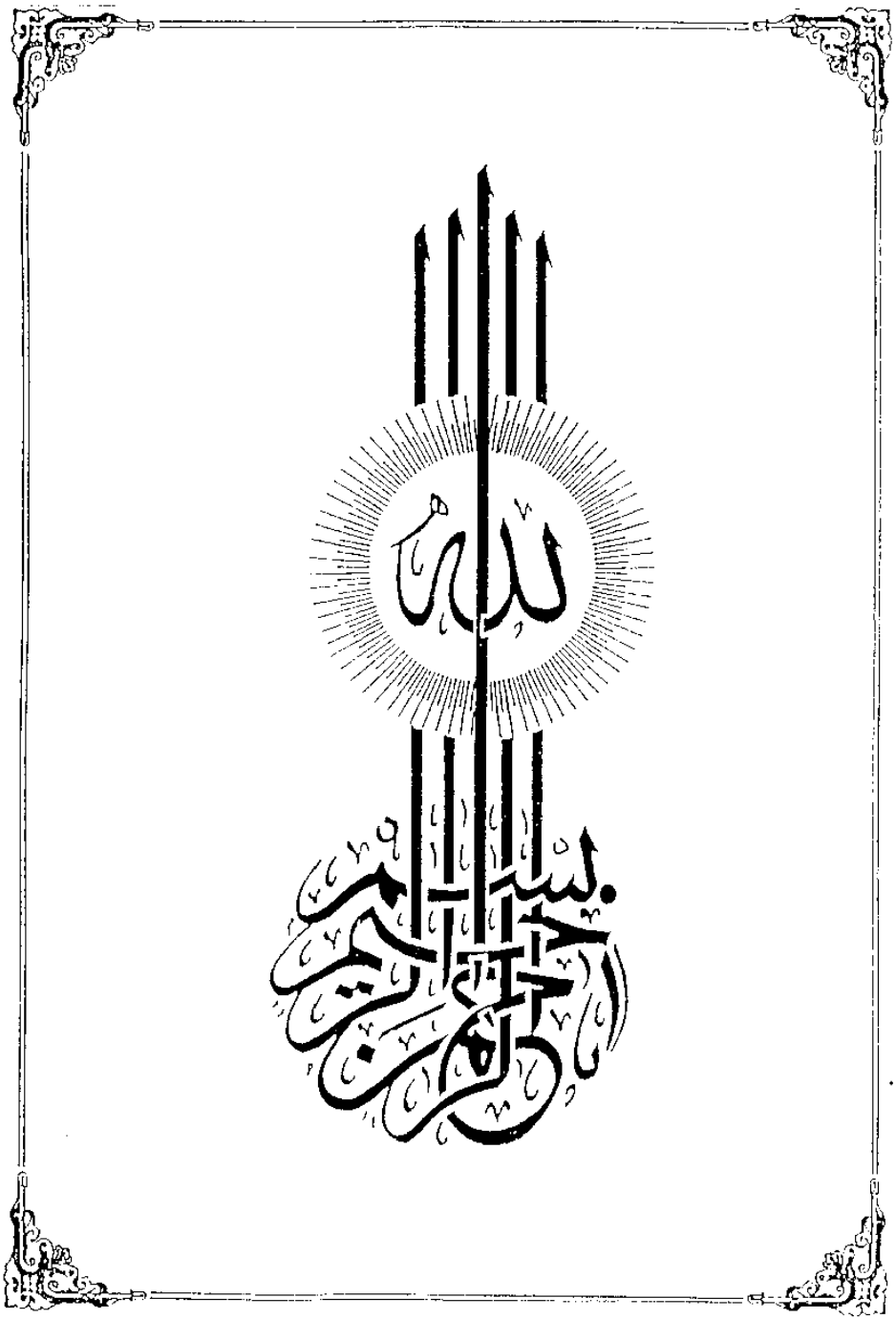

INTRODUCTION

AND AIM OF WORK

INTRODUCTION

The junctional region, or zone formed by trophoblastic invasion of basal decidua and the inner third of the myometrium had been described as the active placental battle ground and on its both sides heavy causalties occur, and it is known as the placental bed. It comprises an intimate mixture of cells and tissues from mother and foetus, beside the vessels between them (Robertson et al., 1974).

Until relatively recently morphologic studies of human placentation in normal and abnormal pregnancy were based on the delivered placenta, on sporadic hystrectomy specimens during pregnancy, and on specimen derived at autopsy after maternal death. The maternal side of the placenta, particularly in pregnancy disorders, remains something of an enigma as post partum involution of the uterus ablates any evidence of defective placentation that may be a determinant of abnormal pregnancy (Robertson et al., 1986).

Histologic studies of human hemochorial placentation must include material deep in the placental site as it is there that two important phenomena occur, one being the controlled invasion of maternal tissues by migratory extravillous trophoblast and the other the conversion by these cells of maternal spiral arteries to uteroplacental

arteries supplying blood to the intervillous space of the placenta (Harris JWS, Ramsey EM, 1966: Brosens et al, 1967; Dixon HG, Robertson WB., 1958).

Dixon and Robertson (1958) in an investigation of the hypertensive disorders of pregnancy, introduced a new technique for obtaining tissue from the pregnant uterus at Caesarean section, which they called the placental bed biopsy, which must include not only basal decidua but also underlying myometrium containing the origins of the uteroplacental (spiral) arteries. These vessels, particularly in hemochorial placentation, are likely to be the target of lesions in such disorders as hypertension, intrauterine fetal growth retardation, and renal diseases where there is evidence of impaired uteroplacental blood flow (Browne JCM, Veall N., 1953, Dixon HG, et al., 1963 ; Lunell NO et al., 1979; Olavero Nunez JA., 1980, Lunell NO et al., 1982; Campbells et al., 1983; Trudinger BJ et al., 1985, Pearce et al., 1986; Abitbol M.M. et al., 1987; Al Znazali et al., 1988).

During pregnancy development, some changes appear in this area including vascular changes and extra vascular reaction in both foetal and maternal tissues, and these changes are termed to be the physiological changes of normal pregnancy (Brosens et al., 1972). In the basal plate, the intimate contact between trophoblast and decidua is accompanied by cell degeneration, necrosis, production

of fibrinoid substance, and appearance of the placental bed giant cells from trophoblast.

The trophoblastic invasion breaches the spiral arteries, thus establishing the intervillous circulation and modifies the structure of the spiral arteries to be the uteroplacental arteries and converts them in to distended, non reactive, low resistance and high conductance vessels capable of coping with the vast increase in the uteroplacental blood flow during pregnancy.

Failure of the previous changes to occur, for whatever reason, can thus be expected to intefer with normal growth of the placenta (Gerretsen et al., 1983).

Brosens and his calleagnes (1972) found that the absence of these changes is a pre-requisite for the development of acute atherosis, but even witrout acute atherosis, the absence of physiological changes in the spiral arteries will impede the blood supply to the placenta. Thus the non occurance of the physiological changes could be the basis of a process leading to foetal growth retordation (Gerretsen et al., 1981).

Lesions of the uteroplacental vasculature may be involved in the pathogenesis of placental insufficiency in pregnancies complicated by hypertension, diabetes, systemic lupus erythematosus and idiopathic foetal growth

retardation (Kitzmiller et al., 1981).

The use of the placental bed biopsy technique, whether taken at Caesarean section first described by Dixon and Robertson 1958, or vaginaly first described by Brosens 1964, as part of the investigation of placentation in normal and abnormal pregnancy has proved to be safe and is now well established with a sizable literature on the subject. The sampling problems, and the inherent interpretative difficulties have resulted in conflicting reports leading to confusion among readers who are unfamiliar with the placental bed biopsy technique and those with little experience of morphologic studies. It is timely therefore to review all aspects of the technology, to highlight these limitations and to encourage accuracy and precision in the interpretation of placental bed biopsies Robertson et al., 1986).

The trophoblastic invasion is a primary component of human placentation, and its result in the exposure of fetal tissue to maternal blood supply. To date, the evaluation of this process has relied mainly on the morphologic evaluation of standard hematoxylin and eosin stained sections or on histochemical or electron microscopic data. The advent of sensitive and specific immunohistochemical methods opens new approaches to the simultaneous study of the antigenic structure and morphology of cells in histologic sections. Tuttle et al., 1985).

The uterine blood flow has been found to increase ten fold from the first weeks of gestation untill delivery, and the uteroplacental vessels, especially the spiral arteries, to dilate with advancing gestational age. This morphologic adaptation of the spiral arteries is essential to facilitate the increasing blood flow to the placenta. The Doppler ultrasoundmethod is now increasingly being used in obstetrics, and intra uterine growth retardation and fetal distress, with the advent of possible non invasive measurement of blood velocities in the maternal uterine arcuate artery and fetal vessels ; and in combination with real time and pulsed Doppler equipment, it has become possible to monitor flow in the fetal aorta and umbilical vien (Gudmundsson S. and Marsal K. 1988).

AIM OF WORK

The aim of this work is to study the histopathological changes in the placental bed in hypertensive disorders with pregnancy, in comparison with the histological changes in normal pregnancy, to find out the pathological changes reflected by these diseases.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The morphological studies of the placental bed to date have been concentrated on the uteroplacental vasculature as the vessels of the placental bed are the target for lesion in normal and abnormal pregnancies especially hypertensive disorders and diabetes mellitus (Robertson et al., 1986).

The uterus derives its blood supply from two major sources, the uterine arteries which are branches of the internal iliac arteries and the ovarian arteries which take their origin from the abdominal aorta below the origin of the renal arteries, within the pregnant uterus these principle supplying arteries send branches to the myometrium, the decidua and the placenta.

There is general agreement on the anatomy of the blood supply to the human non pregnant uterus. The arcuate arteries derive from the uterine arteries and run in the outer third of the myometrium giving off two sets of vessels, the centrifugal ones which nourish the outer one third of the myometrium, and the centripecal ones which branch in a fan like manner toward the endometrium, these centripetally directed arteries are the radial arteries and they give off basal branches that supply only the basal endometrium and break up into terminal spiral arteries, these terminal spiral arteries course through the endometrium giving off numerous vessels that pass

laterally to supply the neighbouring stroma and glands. the spiral arteries terminate in the outer third of endometrium as a 'easn of arterioles(Dalgaard 1946 ; Lundgren 1957 ; Harris & Ramsey 1966 ; Boyd & Hamilton 1970 ; Khong et al., 1986).

There is disagreement about the pattern of branching of the radial artery terminates. The division of the radial artery into two or more arteries did not occur and that the spiral artery was merely a continuation of the radial artery (Dalgaard 1946).

The radial arteries are called spiral arteries only upon entering the endometrium (Harris & Ramsey, 1966).

Each radial artery divides into two or more spiral arteries. The fact that one radial artery terminates as two or three spiral arteries is important as it explains the not infrequent finding that a uteroplacental artery and an adjacent artery without physiological changes may be seen apparently emanating from a single radial artery in the placental bed biopsy Brosens et al., 1967 ; Khong et al., 1986).

In the non pregnant uterus the spiral arteries are the termination of the radial arteries, the latter traversing the myometrium as off shoots of the arcuate system, just internal to the myometrio endometrial junction the radial

artery gives off the spiral or coild arteries which are muscular arteries with a well defined internal elastic lamina which, however gradually disappear as the spiral artery penetrate into the endometrium, the radial arteries also give rise to smaller arteries known as straight arteries but some authors prefer to call them the basal arteries that ramify into the inner myometrium and terminate in the basal endometrium. These basal arteries appear to be much less hormoneresponsive than the spiral arteries, their function probably being nutritive only, and their role in the menstrual cycle and pregnancy is relatively minor (Robertson et al., 1975).

The integrity of the conceptus in human must depend directly upon the provision and maintenance of adequate supply of maternal blood to the intervillous space (Brosens et al., 1967).

The development of the fetus depends, among other things, on the quality and quantity of the maternal blood delivered to the intervillous space of the fetal placenta, and to provide the necessary blood supply for the growing conceptus, the maternal blood vessels have to undergo extensive adaptations and strcutural alteration known as 'physiological changes' including the conversion of the spiral arteries of the non pregnant uterus to the utero-placental arteries (Brosens et al., 1967 ; De Wolf et al., 1980).

During pregnancy the spiral arteries of the placental bed which start out with a diameter of 200 to 300 microns, become distended to a diameter of 1,000 u., and are capable of delivering a ten-fold increase in the supply of blood requried by the fetoplacental unit in the third trimester (De Wolf et al., 1980).

The human placenta recieves its blood supply from more 100 uteroplacental arteries, which are elaborated by the action of migratory interstitial and endovascular trophoblasts on the spiral acteries in the placental bed (Harris & Ramsey, 1966 ; Brosens et al., 1967 ; Boyd & Hamilton., 1970).

In normal pregnancy, these trophoblast incuced vascular adaptation, extend back from their openings into the intervillous space to where the spiral arteries originate from their parent racial arteries in the inner myometrium, it is thought that this vascular phenomenon is effected in two stages, the conversion of the decidual segments of the spiral arteries by a wave of endovascular trophoblast migration in the first trimester and the myometrial segments by a subsequent wave in the second trimester (Robertson et al., 1975 , 1986 ; Pijnenborg et al., 1980, 1981, 1983).

Following implantation in humans, placentation proceeds