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A Study on

THE FOETAL UMBILICAL-PLACENTAL VASCULAR ARCHITECTURE IN PRE-ECLAMPSIA

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BY

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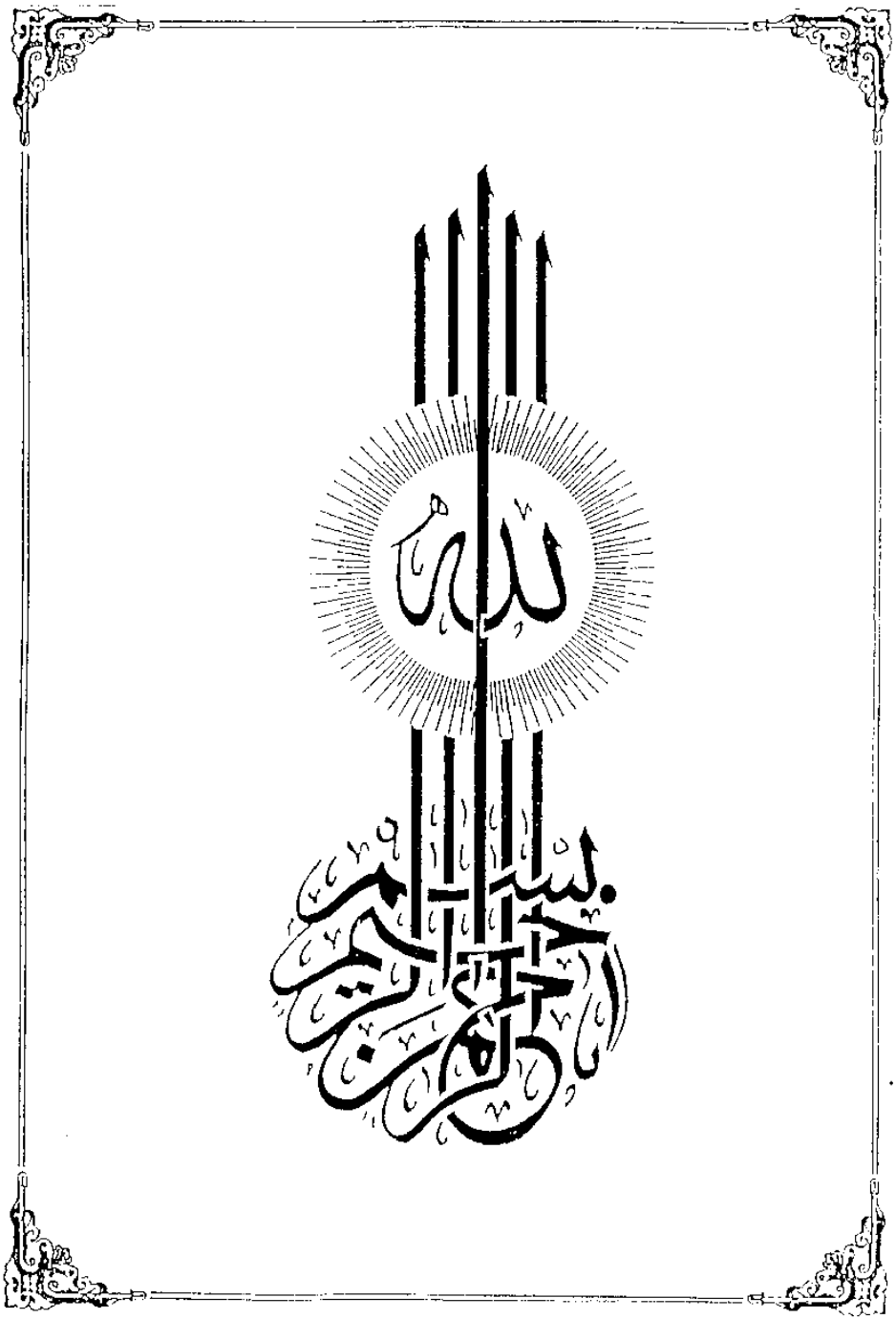

INTRODUCTION

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The study of the placenta in cases of toxemia attracted the attention of many investigators. It is believed that utero placental ischaemia plays an important role in pre-eclampsia. The impact of this ischaemia on the foetal-placental vessels however, is not yet elucidated. McKay, Hertig, and Co-workers, reported thickening of the basement membrane of the foetal capillaries as an initiative factor in endarteritis. A similar observation has been ascribed to the process of physiological placental ageing. It is clear that the placenta plays an integral and fundemental role in the pathogenesis of pre-eclampsia, the disease occurs only when a placenta is present, can develop in the absence of a fetus as in hydatidiform mole and can only be cured with certainty by removal of the placenta. It is true that occasional cases of post partum eclampsia do occur, but there is no aetiological theory currently available that explains these rare and obscure cases. The histological lesions of the placenta in toxemia are inconstant even in severe cases and many reports emphasize the often observed discripancy between the clinical state of the disease and the grade of the pathomorphologic findings in the placenta. There is evidence indicating that the duration of toxaemia symptoms is as significant as the

severity of the lesion and the histologic alterations of the placenta in toxaemia are not constant because they depend upon the duration and severity of the disease and consist, in large part, of an intensification of the degenerative vascular processes or of the degenerative phenomena in the villi including infarction seen in the normal pregnancy. It is concluded that the changes found in the placenta in cases of toxaemia are identical with, but occur more frequently and are more extensive than, those found in the placentas of normal pregnancy.

AIM OF THE WORK

The aim of this work is to study the foetal umbilical and placental vascular changes in pre-eclampsia and to correlate them with the severity of the clinical presentation .

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REVIEW OF LITERATURE

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The placenta is an organ consisting of both fetal and maternal tissues which are interlocked in such a manner as to provid an extensive surface of contact between them without any mixing of the circulation between maternal blood in the intervillous space and the surface of the fetal villi.

Development of the human placenta:

The ovum is fertilised in the Fallopian tube and reaches the uterine cavity as a morula which rapidly converts into a blastocyst and loses its surrounding zona pellucida. The outer cell layer of the blastocyst then proliferates to form the trophoblastic cell mass, from which cells infiltrate between these of the endometrial epithelium; the latter degenerates and the trophoblast thus comes into direct contact with the endometrial stroma, this process of implantation being completed by the 10th or 11th. Postovulatory day.

In the seven-day ovum the trophoblast forms a plaque which rapidly differentiates into two layers an inner layer of large clear mononuclear cytotrophoblastic cells

and an outer layer of multinucleated syncytiotrophoblast. That the syncytitrophoblast is derived from the cytotrophoblast, not only at this early stage but also throughout gestation, is now well established, for even when the trophoblast is growing rapidly, synthesis of DNA occurs only in the nuclei of the cytotrophoblastic cells (Richart, 1961; Galton, 1962), and it is only in these cells that mitotic figures are seen. It would appear that the syncytiotrophoblast is formed by fusion of cytotrophoblastic cells for although no intercellular membranes can normally be seen in the synctial layer, remnants of such membranes can occasionally be found with the aid of the electron microscope (Carter, 1964; Enders, 1965). Cells and nuclei of a type intermediate in morphology between those usually seen in the two trophoblastic layers have also been identified by electron microscopy (Tighe, Garrod and Curran, 1967).

Between the 10th and 13th postovulatory days a series of intercommunicating clefts, or lacunae, appear in the rapidly enlarging trophoblastic cell mass; these clefts are possibly formed as a result of the engulfment within the cynsytitrophoblast of endometrial capillaries (Harris and

Ramsey, 1966). The lacunae soon become confluent to form the precursor of the intervillous space and as maternal vessels are progressively engulfed, or eroded, this becomes filled with maternal blood; at this stage the lacunae are incompletely separated from each other by trabecular columns of syncytiotrophoblast, which between the 14th and 20th postovulatory days tend to become radially orientated and come to possess a central cellular corethat is produced by proliferation of the cytotrophoblastic cells at the chorionic base. These trabecular columns are not true villi but serve as the framework from which villi will later develop , the placenta at this stage being a labyrinthine rather than a villous organ and the trabeculae being therefore best called " primary villous stems " (Boyd and Hamilton, 1970). Continued growth of the cytotrophoblast leads to its distal extension into the region of the decidual attachment and, at the same time, a mesenchymal core appears within the villous mesenchyme. Later, the villous stems become vascularised, the vessels arising within the stem. Angiogenesis within the stems is almost certainly a function of the mesenchymal tissue (Dempsey; 1972) though it has been suggested that the vessels are formed by the cytotrophoblastic cells (Cibils, 1968). In

due course the vessels within the stems establish functional continuity with others differentiating from the body stalk and inner chorionic mesenchyme. The distal part of the villous stem is formed almost entirely by cytotrophoblast which is not invaded by mesenchyme and not vascularised but which is anchored to the decidua of the basal plate. These cells, which form what is sometimes referred to as the " cytotrophoblastic cell columns", proliferate and spread laterally to form a continuous cytotrophoblastic shell which splits the syncytiotrphoblast into two layers, the definitive syncytium on the fetal aspect of the shell and the peripheral syncytium between the shell and the decidua. The definitive syncytium persists as the limiting layer of the intervillous space but the peripheral syncytium degenerates and is replaced by a layer of fibrinoid material (Nitabuch's layer).

Cytotrophoblastic cells emigrate from the shell into the myometrium where they give rise to syncytial like giant cells which extensively colonise the placental bed. Furthermore, cytotrophoblastic cells invade and partially replace the endothelium of the decidual portion of the maternal spiral arteries; this endovascular trophoblast

causes considerable disruption of the arterial wall with the replacement of most of the muscular media by fibrinoid material (Roberteson, Brosens and Dixon, 1975).

The establishment of the trophoblastic shell is a mechanism to allow for rapid circumferential growth of the developing placenta. This leads to an expansion of the intervillous space into which sprouts extend from the primary villous stems. These offshoots consist initially only of syncytotrophoblast but as they grow they pass through the stages previously seen during the development of the primary villous stems i.e, interusion of cytotrophoblast, formation of a mesenchymal core and eventual vascularisation. These sprouts form the primary stem villi and, as they are true villous structures, the placenta is by the 21st day of pregnancy a vascularised villous organ.

Between this date and the end of the 4th month of gestation those villi orientated towards the uterine cavity degenerate and form the chorion laeve whilst the thin rim-decidua covering this area gradually disappears to allow the chorion laeve to come into contact with the parietal decidua of the apposite wall of the uterus.

The villi on the side of the chorion towards the decidua basalis proliferate and progressively arborise to form the chorion frondosum which develops into the defenitive placenta. During this period there is some regression of the cytotrophoblastic elements in the chorionic plate and in the trophablastic shell where the cytotrophoblastic columns degenerate and are largely replaced by fibrinoid material (Rohr's layer); clumps of cells remain however to form the " cytotrophoblastic cell islands". Although there is cytotrophoblastic regression in the basal shell there is during the fourth month of gestation, a proliferation of the endovascular cytotrophoblast which moves retrogradely from the decidual down into the myometrial segments of the spiral arteries. Here it partially replaces the endothelium and invades the media,this process being accompanied by fibrinoid necrosis of the musculoelastic tissue of the vessel wall. The loss of this tissue results in a progressive distension of these vessels, a physiological phenomenon which allows for accommodation of the greatly augmented blood flow required as pregnancy progresses (Brosens, Robertson and Dixon, 1967; Sheppard and Bonnar, 1974; Robertson, Brosens and Dixon, 1975).