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Investigation of Placental Modifications in Patients with Preeclampsia: Examination using Light and Electron Microscopy

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Abstract: *Preeclampsia can have an effect on both the mother's health as well as the unborn child, and it only occurs during gestation and after delivery. Proteins in urine as well as high levels of blood pressure are two signs of this disease that advances swiftly. The study's goal was to look at the histology and to examine for ultrastructural alterations in the placentas of preeclampsia-affected pregnancies. Slices of paraffin were made from placenta specimens used for light microscopic analysis while an electron microscope with scanning technology was employed to produce and observe specimens for ultrastructural analysis. The syncytiotrophoblast layer nuclei were found to be arranged in many sprouts and lengthy anastomosing strands, according to light microscopic examinations. The foetal placental capillaries receded up to total absence as the villus connective tissue core gradually contracted. Endothelial degeneration and atheromatous changes were present in placental stem arteries, whereas endothelial degeneration, escalating fibrosis, and obliteration were seen in lower decidual arterioles. These results are compatible with a rise in fetoplacental vascular impedance, which was shown to be present when lacking end-diastolic flow velocity was established that it existed in the umbilical artery just before birth. The results clarify how this issue has interfered with the movement of nutrients and gas.*

Key Words: Placental Modification, Preeclampsia, Microscopy, Ultrastructural Alteration

Introduction

The placenta's healthy growth and functioning, which keeps the mother and foetus linked for the exchange of waste, nutrients and blood gases are essential for normal embryonic development and long-term survival [Weissgerber, et al., 2006, Red-Horse et al., 2004, Baschat, et al., 2004]. Preeclampsia is a medical condition, caused by abnormal placental formation. The cause of this common prenatal sickness, which affects 9%–12% of pregnancies, is still a mystery. It is a major contributor to mother and foetal mortality as well

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as morbidity [Livingston, et al., 2000, Errera, et al., 2013, Huppertz, et al., 2011, Smith, et al., 2015].

Like other epithelia, the villous placenta is constantly renewed, and the syncytiotrophoblast is kept in place by fusing with the cytotrophoblasts beneath it. In conditions like preeclampsia, fewer arterioles are broken by invasive cytotrophoblasts because of anomalies in adhesive molecule changing, demonstrating that this subgroup of trophoblast cell populations failed to mature normally. In these situations, the outermost decidua is the only area where cytotrophoblast penetration occurs [Huppertz, et al., 2010, Han, et al., 2006, Zhou, et al., 1993].

Materials and Methods

Twenty pregnancies (study group) were found to have severe intrauterine growth retardation (IUGR) using a set of prenatal criteria. Reduced amniotic fluid volume and estimated foetal weight that is below the 10th percentile (defined as three centimetres or less being the highest vertical depth of amniotic fluid devoid of cords) Preeclampsia, which was defined as having blood pressure that exceeded 140/100 mmHg on a minimum of two occasions, followed by an increase in the diastolic blood pressure that exceeded 25 mmHg, occurred after 20 weeks of gestation throughout each pregnancy in the study group. A positive finding of +3 on dipstick testing or a protein excretion of 0.3 g/24 hours were further signs of proteinuria. The research group's entire membership was born by scheduled caesarean surgery.

All newborns exhibited IUGR symptoms. The control group included a singleton pregnancy that produced live births, structurally healthy neonates, and fetuses with normal growth rates (and a comparable mean gestational age) as the study group. The middle portion's full thickness was sampled, and processing was done as previously mentioned. Regular paraffin embedding procedures were followed, and samples were sliced at 5m to 7m and hematoxylin and eosin stained. Digital SEM was used for scanning electron microscopic (SEM) research. The specimen pieces were fixed in 2.5% glutaraldehyde at 4 C for 2 hours after being post-fixed in 2% osmium tetroxide and rinsed with a cacodylate buffer solution. The samples were dried in various ethanol concentrations solutions. A portion of every specimen was laid out on a stub as well as marked using gold (3 nm) for SEM analysis. Toluidine blue semithin segments were sliced and formed, and the leftover pieces of these samples were inserted in epoxy glue.

Results

Age and gestational age at birth was similar in preeclamptic women and controls. Nevertheless, birth, mean arterial pressure, Preeclamptic women's weight, placental weight, and other characteristics were substantially different as compared to normal patients ($P < 0.01$). Control placentas exhibited a distinct appearance, showing trophoblastic cell types arranged in thin layers surrounding a central connective tissue core. Each villous was densely packed with foetal capillaries. Also, intervillous gaps containing maternal blood separated the villi from one another.

Preeclamptic placentas showed specific histological modifications. The nuclei of the syncytiotrophoblasts had a distinct distribution and a propensity to cluster, especially in regions where the syncytial layers protrude through the intervillous spaces. The villous tree has a pseudo-labyrinthine look because of the long, slender syncytial threads that span the intervillous intervals linking one villus to another. Sectioned syncytial strands or sprouts can be recognized as there is no villous core.

High levels of cellularity and fibrillar content can be seen in the connective tissue villous stoma, which causes the entire villous core to have a strong affinity for collagen staining. In most villi, fetal capillaries have typically vanished, yet on occasion they have still been discernible. Red blood cells have only been shown to be present inside the lumina of a tiny number of preserved capillaries.

The central core of connective tissue, which grew inside the ends of the villi, entirely took the role of the embryonic blood sinusoids. Despite the

lack of any sort of capillary wall structure, certain fetal nucleated red blood cells could be perceived in a few uncommon villi. These cells appeared to dwell directly in the connective tissue stroma. Atheromatous progression and endothelial wall deterioration were present at all phases in each branch of the main umbilical arteries. In the basal decidual arterioles, endothelial deterioration, escalating fibrosis, and destruction were seen. Such terminal villous structures could be identified by their profusion of small, many buds that resemble villi and their sinusoidal-shaped projections, which were identical in shape and dimensions to the apical dilatations of the capillaries loops. Capillary loops appeared more numerous and deeper in comparison to the ones in the control group. The majority of capillary loops lack coiling, while preeclamptic cases of extended loops had considerably fewer branches. Compared to controls, there were substantially fewer terminal villi, which looked like long drainpipes. In preeclamptic cases, there were huge plaques of a fibrin-like material that commonly extended across multiple villi over the superficial surfaces of the villous tissues. The villi's outside surface exhibits noticeable folds and wrinkles, as though the trophoblast were stacked up.

Table 1

Comparison of Variables between Control and Preeclampsia (PET) Groups

Parameters	PET SD	Control SD	P value
Gestational age	31.4 2.48	31.3 2.55	NS
Maternal age	36.9 5.6	30.4 4.9	
Maternal Blood Pressure (mmHg)			
Systolic	147.8 7.3	118.1 6.4	
Diastolic	103.5 3.6	70.2 9.5	
Placental weight (g)	261.0 90.3	520.25 114.2	
Birth weight (g)	1204.3 353.6	1808.4 534	< 0.01
Indication for delivery	9.0 IUGR, 9.0 IUGR+PE	8.0 CI, 8.0 PTL, 2 optional deliveries	
Outcomes of umbilical artery Doppler	AEDFV	RI, 0.68 0.02	

Discussion

Whenever the longitudinal extension of the capillaries within the matured intermediary villi exceeds the diameter of the villi, the capillaries coil to form loops that emerge beyond the villous surface, resulting in the formation of the terminal villi, whose resembled grape-like outgrowths. Their capillary divides into a complicated network of loops with multiple incorporated branching and intermittent sinusoidal expansion. Since the development of such a low-resistance capillary network coincides with the corresponding increase in the embryonic heart rate accessing umbilical arteries to around 30% at the phase, this design favours maximum food and gas exchanges as the fetus develops. A similar study was conducted by Landon et al [Landon, et al., 2020].

The increased occlusive process was shown to be occurring inside improperly grown terminal villous capillaries, Examinations of the terminal villous ultrastructure in preeclamptic patients [Krebs, et al., 1996] revealed that fetal cardiac function was diminishing as a result of increasing hypoxia and acidosis, but this not the only factor. Maternal thrombus along the periphery of the villi will affect the interchange of placenta gases in premature preeclampsia. In our preeclamptic instances, the trophoblast's crumpled look is obviously aberrant; this may be caused by a slower turnover of trophoblasts. Since syncytiotrophoblast renewal requires young cytotrophoblast integrating within the syncytium, a number of metabolic processes of villous syncytiotrophoblasts, such as the transfer of amino

acids to the cells or the creation of nitric acid, could result in risk. Poor villous syncytiotrophoblast development brought on by nitric oxide can promote the growth of parental fibrinogen plaques located on the outermost layer of such aberrant villi.

Basal decidual arterioles gradually disappeared, and stem arteries developed atheromatous alterations the proportionate frequency of hypoxic modifications [Aplin, et al., 2020]. The stem arteries were histopathologically distinguished by perivascular lipo phage infiltration and fibrinoid necrosis [Khong, et al., 2004]. Atherosclerosis' aetiology and pathophysiology are yet unknown, however, given that vascular lesions seen in transplant rejection reactions resemble atherosclerotic lesions. The higher occurrence of thrombosis formation and succeeding hypoxic alterations in the tissue of the womb can be attributed to the presence of preeclampsia in arteries that do not experience normal physiological alterations during pregnancy. Fluctuations in its levels are associated with the placental modifications observed in preeclampsia.

Conclusion

This study concluded by demonstrating that ischemia damage to placental tissue and maldeveloped terminal villi occur in preeclampsia-complicated placentas. These findings are consistent with an increase in fetoplacental vascular resistance when there was none in the umbilical artery before delivery. The disorder's poor gas and nutrient transfer is explained by these findings.

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