

Table : Morphometry of different parameters of VSM

Category	N	n	Number of V.S.M (mean±SD)	Thickness (mean±SD)		
				Endothelial B.M (nm)	Trophoblastic B.M (nm)	VSM (µm)
Control	10	100	4.01±1.37	75.59±30.08	124.86±25.61	2.32±0.53
Mild Hypertension	7	70	3.74±1.25	100.83±17.99 ³	153.76±76.44 ³	2.60±0.81 ²
Moderate hypertension	7	70	4.26±0.95	204.83±52.64 ³	505.86±184.96 ³	3.99±0.71 ³
Severe hypertension	7	70	4.96±1.1	211.49±58.12 ¹	649.79±138.10 ³	4.82±0.77 ³

N: Number of cases n: Total no. of villi B.M: Basement membrane
 Statistical Significance (Control vs Mild, Mild vs Moderate, Moderate vs Severe)
¹=non significant ²=p<0.01 ³=p<0.001

morphometric analysis of different components of VSM has been highlighted in a tabulated form (table).

DISCUSSION

Vasculosyncytial membrane is a differentiated area of placental villi that is specifically concerned with materno-fetal transfer. Becker and Bleyls⁷ reported a reduction of VSM in the placenta from the woman with pre-eclampsia. But in our study we found the increased frequency of VSM in PIH placenta. This increased frequency may be due to increase in number of capillaries to compensate the deficit of transfer of gases and nutrients through VSM. According to Fox and Blanco⁶ VSM is poor in organelles and were 0.5-1µm in thickness. In the present EM study, it was seen that syncytiotrophoblast at VSM was reduced to syncytial lamellae with few endoplasmic reticulum and lysosomes and the thickness of VSM in normotensive mother was 2.32±0.53µm which was more than the reported data. This may be due to mean thickness of VSM of all the villi studied from central parts of maternal surface of placenta. The thickness recorded by Fox and Blanco⁶ may be the thinnest part of VSM which is not clear from his study.

The basement membranes of trophoblast and endothelial cell in PIH were seen to be thicker in comparison to control and VSM was directly proportional to severity of PIH (Table). The increased thickness of basal lamina indicates an altered trophoblastic as well as endothelial cell activity. Therefore the membranous appearance of VSM is distorted because of presence of one or all of the three of the following: i.e., intervening cytotrophoblasts, thickening of both basement membranes or abundance of collagenous

connective tissue between the trophoblastic and endothelial basement membranes.

Though some reports are available about the increase thickness of trophoblastic basement membrane in PIH⁸ patients, measurement of all the layers contributing to the formation of VSM has not been recorded. According to Macara et al⁹ increased thickness of trophoblastic basement membranes and congestion of capillaries by erythrocytes in PIH results in limiting oxygen transfer from intervillous space containing maternal blood to fetal capillaries of placental villi. Therefore, it may be possible that increased thickness of the VSM could also reduce oxygen transfer in PIH cases.

Although Jones and Fox¹⁰ described hyperplasia of cytotrophoblast in cases of pre-eclampsia, its role & position at VSM has not been documented. Also syncytiotrophoblast and endothelial cells are known to express FcY receptors, while the cytotrophoblast does not express such receptors^{11,12}. In our study, it is noticed that either the lamellae of the cytotrophoblast or even the whole cell including its nuclei and organelles can contribute to the formation of VSM, in hypertensive cases. This could restrict the transport of immunoglobulins to fetal circulation leading to a risk in survival of the new born of PIH mother. Therefore it is concluded that the significant thickening of VSM may be responsible for slow transport of nutrients between mother and fetus leading to adverse effects on the fetus and new born.

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