

# VASCULOSYNCYTIAL MEMBRANE IN PLACENTAL VILLI OF NORMOTENSIVE AND HYPERTENSIVE PREGNANCIES

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## ABSTRACT

Vasculosyncytial membranes appear as thin blister like protrusion on the villous surface and form the only physical barrier between fetal and maternal blood. Paucity of vasculosyncytial membrane leads to fetal hypoxia and appears to subject the fetus to considerable risk.

This study was conducted to see the incidence of vasculosyncytial membrane in placental villi of normotensive and hypertensive pregnancies.

A total of 40 patients were selected from Obstetrics and Gynaecology Department, J.N.M.C.H., A.M.U., Aligarh and they were categorized into normotensive and hypertensive groups having 20 cases in each group respectively. Most of cases in normotensive group were multigravidae and aged between 25 to 35 yrs while cases in hypertensive group were above 35 yrs of age and mostly nulliparous. Placentas of each group were fixed in 10% formalin solution and processed for section cutting. 5 $\mu$  thick sections were prepared and stained with haematoxylin & eosin and observed under light microscope. A total of 200 villi per high power field (hpf) were counted in each slide and obtained data were expressed as mean  $\pm$  standard deviation. It was found that there was complete absence of vasculosyncytial membrane in placental villi of hypertensive patients.

Key words: fetal hypoxia, basement membrane, trophoblast, pre-eclampsia

**Key words:** aplastic anaemia, nephromegaly, hepatomegaly, retroaortic left renal vein, Myelofibrosis, leukemia

## INTRODUCTION

The placenta is the organ that facilitates nutrient and gas exchange between maternal and fetal components. Placental exchange does not take place in all villi, however it occurs only in those villi whose fetal vessels are in intimate contact with the covering syncytial membrane<sup>1</sup>. In all mature normal placentas the villous syncytiotrophoblast is clearly not homogenous structure<sup>2</sup> but they are thin measuring 1 to 0.5  $\mu$ m in thickness, free of nuclei and poor in organelles, they are directly opposed to sinusoidally enlarged parts of fetal capillaries<sup>3</sup>. Capillary basement membrane and trophoblast basement membrane may come into such close contact that they fuse to form a 'Vasculosyncytial membrane'<sup>4</sup>. These membranes appear as thin blister like protrusion on the villous surface and here form the only physical barrier between fetal and maternal blood. Paucity of vasculosyncytial membrane leads to fetal hypoxia and appears to subject the fetus to considerable risk<sup>5</sup>.

Pregnancy induced hypertension is one of diseases met during pregnancy and it leads to maternal and fetal morbidity and mortality<sup>6</sup>.

This study was a quantitative study to see the incidence of vasculosyncytial membrane in placental villi of normotensive and hypertensive pregnancies.

## MATERIALS AND METHODS

Patients were selected from OPD and emergency ward of Obstetrics and Gynaecology Department, Jawaharlal Nehru Medical College, Aligarh Muslim University, Aligarh. A total of 40 cases were selected and they were grouped into two categories, each having 20 cases respectively. In normotensive group, only those cases with normal blood pressure without edema or proteinuria were included. Most of the cases in this group were multigravidae and aged between 25-35 yrs. In hypertensive group, only those cases having blood pressure ranging 140/90 mm of Hg and above, with or without edema, and/or proteinuria were included and patients who were on antihypertensive drug or having any associated diseases like diabetes were excluded. In this group, most of the patients were nulliparous and above 35 yrs of age. All cases in both groups were delivered at full term pregnancy. After delivery there placentas

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were collected and fixed in 10% formalin and processed for section cutting. 5µ thick sections were prepared and stained with haematoxylin & eosin and observed under light microscope. A total of 200 villi per high power field (hpf) were counted in each slide and obtained data were expressed as mean ± standard deviation.

**OBSERVATIONS**

Groups	Percentage of villi having vasculosyncytial membrane
Normotensive	12.65 ± 1.81
Hypertensive	0.00

**Table - I**

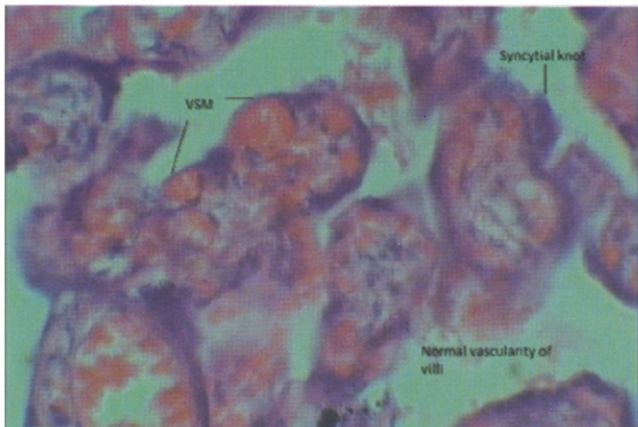


Fig i: Photomicrograph of placental villi (normotensive group) showing vasculosyncytial membrane (VSM), syncytial knot and normal vascularity. H&E X 400x

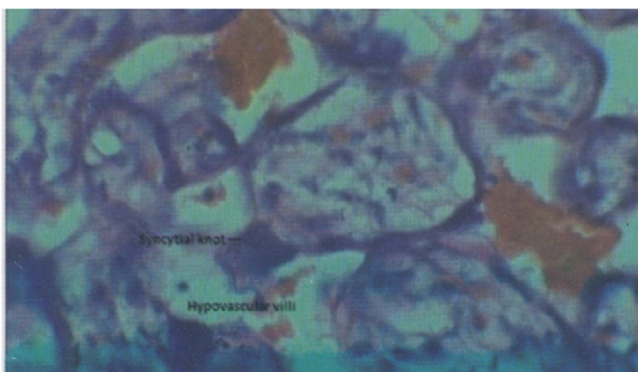


Fig ii: Photomicrograph of placental villi (hypertensive group) showing absence of vasculosyncytial membrane, syncytial knot and avascular villi. H&E X 400x.

It was observed (Table I) that in placentas of normotensive group (fig i), number of villi having vasculosyncytial membranes were 12.65 ± 1.81 % whereas in placentas of hypertensive group (fig ii), there was an absence of vasculosyncytial membrane in villi.

**DISCUSSION**

Bremer<sup>7</sup> first identified Vasculosyncytial membranes and the term 'vasculosyncytial membrane' was introduced by Getzowa and Sadowsky<sup>8</sup>.

In present study it was found that vasculosyncytial membrane was absent in placental villi of hypertensive pregnancies and this finding is in accordance with findings of Beckers and Belyl<sup>9</sup>, Fox<sup>10</sup> and Dimitrovska N<sup>11</sup>

Becker and Bleyl<sup>9</sup> have claimed that placentas from pregnancies complicated by pre-eclmptic toxemia have an unduly low proportion of villi with vasculosyncytial membrane.

Fox<sup>10</sup> found that vasculosyncytial membrane was deficient i.e. present on less than 5% of the villi in placenta of hypertensive pregnancies. This paucity of membranous area can be considered as a failure of trophoblast differentiation. He also noted a clear-cut inverse relationship between incidence of villous vasculosyncytial membranes and that of fetal hypoxia.

According to Dimitrovska N<sup>11</sup>, a low VSM count, as a reflection of villous regression, was found in placentas from prolonged pregnancies and, to a lower degree, in placentas from pregnancies complicated by preeclmptic toxemia. Mature babies whose placentas had a low VSM count suffered from a high incidence of hypoxic complications.

In contrary to our findings, Horkey<sup>12</sup> thought that in fetal hypoxia there was an increased formation of these membranes in an attempt to increase the surface available for gas transfer.

**Conclusion**

Vasculosyncytial membrane is the only physical barrier between fetal and maternal blood. In placentas of hypertensive pregnancies, vasculosyncytial membrane become deficient resulting in fetal hypoxia, which in turns leads to perinatal morbidity and mortality. Hence proper antenatal checkup in early weeks of pregnancy gives an opportunity to

provide suitable management.

#### REFERENCES

1. Sadler TW; Langman's medical embryology in third month to birth: the fetus and placenta. 10th edn. Wolters Kluwer (India) pvt. Ltd.,New Delhi 2007, pp. 97-98.
2. Amstutz E. Observations on the maturation of chorionic villi in the human placenta with special reference to epithelial plates. *Acta Anat(Basel)*.1960; 42:1230.
3. Fox H. and Balanco A. Scanning electron microscopy of the human placenta in normal and abnormal pregnancies. *European Journal of Obstetrics & Gynecology and Reproductive Biology*. 1974; 4(2): 45-50.
4. Fox H. The morphological basis of placental insufficiency. *Journal of Obstetrics and Gynaecology of India*. 1975; 25: 441-450.
5. Fox H, Sabire N; Pathology of the Placenta in histological abnormalities of the placenta. 3rd edn. W.B. Saunder & co. Philadelphia, London 2007, pp 155.
6. MacKay AP, Berg CJ, Atrash HK. Pregnancy-related mortality from preeclampsia and eclampsia. *Obstet Gynecol*. Apr 2001;97(4):533-8.
7. Bremer JL. The interactions of the mesonephros, kidney and placenta in different classes of mammals. *American Journal of Anatomy*. 1916; 19:179-209.
8. Getzowa S, Sadowsky A. On the structure of the human placenta with full-term and immature fetus, Living or dead. *J Obstet Gynaecol Brit Emp* 1950; 57:388-96.
9. Becker V, Bleyl U. Placental villi in pregnancy toxemias and fetal erythroblastosis in the fluorescent microscopy picture. *Virchows Arch Pathol Anat Physiol Klin Med*. 1961; 334:516-527.
10. Fox H. the incidence and significance of vasculosyncytial membranes in the human placenta. *Journal of Obstet. & Gynaec. Of the British Commonwealth*. 1967a; 74:28-33.
11. Dimitrovska N. Characteristics of the vasculosyncytial membranes in human chorionic villi. *Jugoslavenska ginekologija i opstetricija*. 1978; 18(3-4):279-84
12. Horkey Z. Quantitative changes in the vascularization of the chorionic villi of the diabetic placenta. *Zentralblott gynakol*. 1964; 86: 815.