

Microscopic Features of Vacuosyncytial Membrane and Syncytial Knot Formation in Pregnancy Induced Hypertensive and Normotensive Pregnancies

PRABHJOT KAUR, HARSH, SANJEEV SHARMA, RASALIKA MIGLANI, SUBHASH KAUSHAL

ABSTRACT

Introduction: The placenta accomplishes the functions through its unique anatomical association with mother. Placenta links the mother and fetus by interaction with maternal blood via uteroplacental vessels. Attention towards the uterine side of the placenta might explain inexplicable pregnancy complications. By the study of placental bed new information has come to light, especially for pre-eclampsia and intrauterine fetal growth retardation.

Aim: To study microscopic features of vacuosyncytial membrane and syncytial knot formation in pregnancy induced hypertensive and normotensive parturients .

Materials and Methods: This study was conducted in the Department of Anatomy and Pathology, Government Medical College, Patiala. The placentae were collected

from gynaecological operation theatre, Rajindra Hospital, Patiala. Seventy five cases of pregnancy induced hypertension and twenty five cases of normotensive pregnancies were taken. An attempt was made to see any changes in histological features of placentae of pregnancy induced hypertensive cases and compare it with the normotensive placentae.

Results: The microscopic study showed significant paucity of vasculosyncytial membrane, increased syncytial knot count i.e. >30% in PIH placentae as compared to normotensive placentae (control).

Conclusion: Microscopic changes in placentae associated with PIH are due to occlusion or narrowing of the uteroplacental vasculature. The perinatal mortality and morbidity associated with this condition may be due to alterations in the uteroplacental flow.

Keywords: Hypertensive placentae, Syncytiotrophoblast, Uteroplacental vessels

INTRODUCTION

Development of placenta is as uniquely intriguing as the embryology of the fetus. The fetus is dependent on the placenta for pulmonary, hepatic and renal functions. This blood bathes the outer syncytiotrophoblast, allowing exchange of gases and nutrients with fetal capillary blood within the connective tissue at the villous core [1].

When there is particularly close association of the fetal blood vessels with the overlying syncytium, the term "vasculosyncytial membrane" to the region was given by Getzowa and Sadowsky [2].

After 32 weeks, clumps of syncytial nuclei are found into the intervillous space. These projections are called syncytial knots which represent apoptosis [3].

Tenney, described a large swollen type nucleus in the trophoblast of pre eclamptic placenta. This he considered

to represent one of the forms of degeneration which syncytial nuclei could undergo [4].

Fox, described "syncytial knots" as the syncytial regions with high concentration of nuclei, formation of which is due to proliferation of syncytial by amitosis. He concluded that syncytial knots formation is seen with increased frequency in the last weeks of normal pregnancy and in cases of toxemia and said that it seemed to be the result of ageing [5].

MATERIALS AND METHODS

This study was conducted in the Department of Anatomy and Pathology, at GMC, Patiala, India during period of January 1, 2008 to December 31, 2009. Total 100 placentae were collected from labour room and from gynaecological operation theatre, Rajindra Hospital, Patiala with patients consent. This study was approved by institutional ethical committee. Cases were broadly divided into two groups:

1. Group I (Study/PIH group) – 75 cases of clinically proved PIH.
2. Group II (Control group) – 25 singleton normotensive pregnancies.

Cases with period of gestation more than 35 weeks were taken for study and cases that are previously hypertensive were excluded from the study. The placentae were grouped depending on the degree of hypertension as described by Cunningham et al., [1].

1. Normotensive < 140/90 mmHg
2. Mild hypertension $\geq 140/90$ - < 160/110 mmHg
3. Severe hypertension $\geq 160/110$ mmHg

The placenta was received in adequate amount of 10% formalin.

Selection of pieces from placenta was done in accordance with Salafia and Popek, who recommended minimum sections from placenta for histopathology [6].

1. Section from membrane roll
2. From central area of fetal surface
3. From central area of fetal surface
- 4 & 5. From the two ends of umbilical cord, leaving 3cm of proximal end.

All the sections of placenta were stained with Haematoxylin and Eosin stain. Stained slides of thin section were prepared to examine under microscope. Microscopic changes like infarcts, calcifications, non formation of vasculosyncytial membrane (a vital observation through which maternal and fetal blood and metabolite exchange takes place), syncytial knots, were noted.

Clinical evaluation of patients and routine haematological and biochemical tests were also considered.

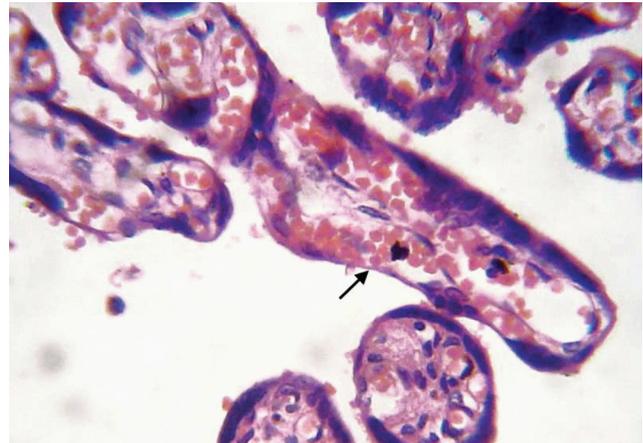
Data was compiled in performa and statistically analysed. The main observations and interpretations were done according to Salfia and Popek [6].

RESULTS

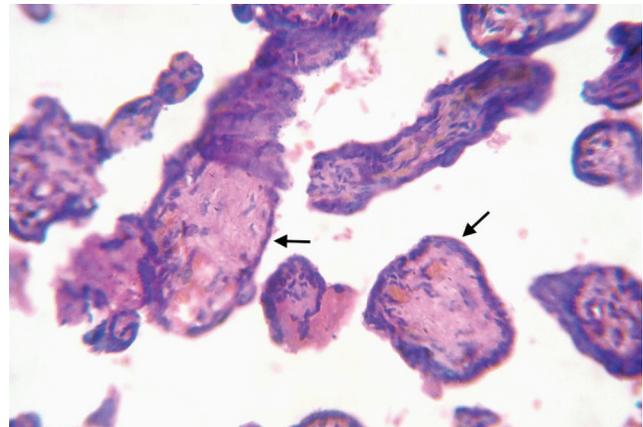
1. Vasculosyncytial Membrane

Placenta showing vasculosyncytial membrane in >6% of villi is considered normal. Gradation of formation of vasculosyncytial membrane was done according to its presence in number of villi as 0-5 %, 6-30% and >30% [Table/Fig-1&2].

[Table/Fig-3] shows that in study group, 55 (73.33%) placentae had deficient vasculosyncytial membrane formation i.e. less than 5% of villi showed vasculosyncytial membrane formation. None of the cases of control group had shown villi deficient in vasculosyncytial membrane formation. Out of 25, 3(12%) cases of control group had >30% of villi showing vasculosyncytial membrane formation.



[Table/Fig-1]: Microphotograph of placenta showing normal formation of vasculosyncytial membrane.(H and E, 400X).



[Table/Fig-2]: Microphotograph of placenta showing thickened basement membrane and non formation of vasculosyncytial membrane.(H and E, 100X).

Vasculosyncytial Membrane	Group I (Study)		Group II (Control)	
	No.	%age	No.	%age
0 – 5%	55	73.33	0	0
6 – 30%	20	26.67	22	88
> 30%	0	0	3	12
Total	75	100	25	100
Statistical Analysis				
χ^2	p value		Significance	
44.1	<0.0001		HS	

[Table/Fig-3]: Formation of vasculosyncytial membrane in study and control groups.

The statistical difference between two groups for the paucity of vasculosyncytial membrane formation was highly significant (<0.0001).

2. Syncytial Knots

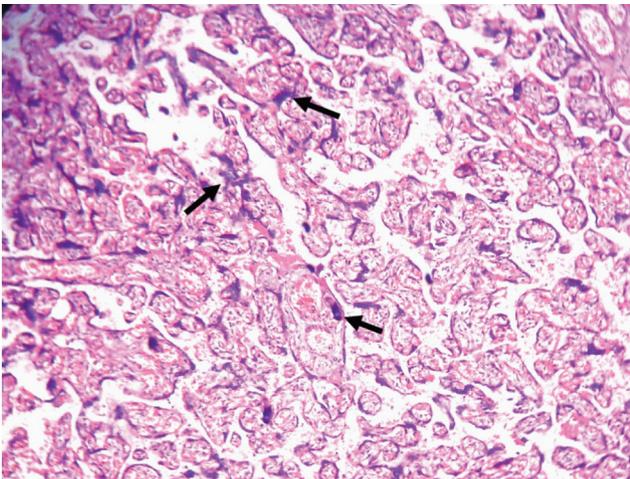
Formation of syncytial knots in 0-30% of villi is considered as a normal placental maturation phenomenon.

[Table/Fig-4] shows that in study group, syncytial knots formation in 0-30% of villi was present only in 21(28%) cases and majority of cases i.e. 54(72%) had syncytial knot formation in > 30% of villi [Table/Fig-5]. In the control group, out of 25 cases only 2(8%) cases had syncytial knot formation in >30% villi while the rest of the 23(92%) cases showed syncytial knot formation in 0-30% villi.

The statistical difference between two groups was significant (<0.001).

Formation of Syncytial Knots	Group I (Study)		Group II (Control)	
	No.	%age	No.	%age
0 – 30%	21	28	23	92
> 30%	54	72	2	8
Total	75	100	25	100
Statistical Analysis				
χ^2	p value		Significance	
31.2	<0.001		S	

[Table/Fig-4]: Syncytial knot formation in study and control groups.



[Table/Fig-5]: Microphotograph of placenta showing syncytial knot count. (H&E, 40X).

DISCUSSION

In this study, the emphasis has primarily been made on microscopic changes in placenta in PIH and normal cases and its relation with fetal outcome which in future will be helpful in managing pregnancies complicated with hypertension.

Microscopic Changes

1. Vasculosyncytial membrane

Placenta with 6-30% of the villi showing vasculosyncytial membrane formation were said to have a normal count. Fox, observed vasculosyncytial membrane count of 5.3% in toxemia and 2.9% in essential hypertension [7].

In the present study, 73.33% cases of study group (PIH) showed deficient VSM formation while 88% cases of control group had normal VSM formation i.e. in 6-30% villi and 12% cases were showing VSM formation in > 30% villi [Table/Fig-1-3].

Tewari et al., observed 0-5% vasculosyncytial membrane in 92.8% grade III placentas of pre-eclampsia cases [8].

Sodhi et al., [9] and Kurdukar et al., [10] noticed paucity of VSM in placentae from PIH.

The villous syncytio trophoblast undergo regional differentiation to form VSM in mature villous to serve synthetic as well as transfer function. Deficiency of VSM areas can be considered as a failure of trophoblastic differentiation in PIH – a failure that appears to subject the fetus to considerable risk as reduction in the density of VSM results in a decrease materno-fetal gaseous exchange.

2. Syncytial Knots

The presence of syncytial knots has been taken as an index of placental maturity (Benrischke) [11].

In the present study, 72% cases of PIH (study group) had syncytial knot count > 30% whereas in control group majority of the placentae (92%) had shown count between 0-30% [Table/Fig-4,5].

Bajaj et al., [12], Masodkar et al., [13] Avasthi et al., [14] found 100% cases of PIH, 69% of cases of PIH and 80% cases of PIH associated with excessive syncytial knot formation. Majumdar et al., [15] and Artico et al., [16] also observed significant increase in syncytial knots in PIH as compared to control [Table/Fig-6].

Syncytial knot formation is seen with increased frequency in last weeks of pregnancy and more villi show syncytial knots

Authors (Year)	Group	% of cases with SK count >30%	Significance
Bajaj et al., (1979) [12]	PIH	100%	-
	Control	44%	
Masodkar et al., (1985) [13]	PIH	69%	-
	Control	0%	
Avasthi et al., (1991) [14]	PIH	80%	-
	Control	0%	
Majumdar et al., (2005) [15]	PIH	-	S
	Control	-	
Artico et al., (2009) [16]	PIH	-	S
	Control	-	
Present Study (2009)	PIH	72%	S
	Control	8%	

[Table/Fig-6]: Comparison of syncytial knot formation > 30% of this study with various studies.

in prolonged pregnancy and toxemia. These are the result of ageing changes which are more due to accelerated maturation of placenta in PIH.

Prabhjot et al., grossly examined the PIH Placentae and stated that PIH Placentae show gross changes are due to occlusion or narrowing of the uteroplacental vasculature which will lead to ischaemic damage and accelerated placental maturation [17].

CONCLUSION

The microscopic study showed significant paucity of vasculosyncytial membrane, increased syncytial knot count i.e. >30% in PIH placentae as compared to normotensive placentae (control).

Microscopic changes in placentae associated with PIH are due to occlusion or narrowing of the uteroplacental vasculature, placental ischaemic damage and accelerated placental maturation. The perinatal mortality and morbidity may be to alterations in the uteroplacental flow.

Pregnancy complicated by hypertension not only affects maternal health but also affect the developing embryo. Placenta being bridge between maternal fetal activities through which understanding of maternal dysfunction as well as fetal wellbeing can be obtained and can be a useful in management of future pregnancies.

REFERENCES

- [1] Cunningham FG, Leveno KJ, Bloom SL, Hauth JC, Gilstrap L, Wenstrom KD. Implantation, embryogenesis and placental development. In: Williams O (ed.) 22nd (edn), McGraw Hill, Medical Publishing Division, 2005; p 51, 61.
- [2] Getzowa S and Sadowsky A. On the structure of human placenta with full term and immature fetus, living or dead. *J Obstet Gynaec Brit Emp.* 1950; 57:388-96.
- [3] Benirschke K and Kaufmann P. The Pathology of human placenta. Springer-Verlag, New York, 4th edn. 2000; p 62-74.
- [4] Tenney B Jr. The placenta in toxemia of pregnancy. *Am J Obstet Gynaec.* 1935;29:819.
- [5] Fox H. The morphological basis of placental insufficiency. *J Obstet Gynaecol India.* 1975;25(4):441-50.
- [6] Salafia CM and Popek EJ. Placenta. Aderson's Pathology. Mosby – Year Inc. 10th edn, 1996;2:2310-53.
- [7] Fox H. The incidence and significance of vasculosyncytial membrane in the human placenta. *J Obstet Gynaec Brit Cwlth.* 1967;72:28-33.
- [8] Tewari K, Tyagi SP, Saxena K, et al. Ultrasonographic and histological study of placenta in abnormal pregnancy cases. *J Obstet Gynaecol India.* 1997;47(2):119-26.
- [9] Sodhi S, Mohan H, Jaiswal TS, Mohan PS, Rathee S. Placental pathology in pre-eclampsia eclampsia syndrome. *Indian J Pathol Microbiol.* 1990;33(1):11-16.
- [10] Kurdukar MD, Deshpande NM, Shete SS, Zawar MP. Placenta in PIH. *Indian J Pathol Microbiol.* 2007;50(3):493-97.
- [11] Benirschke K. A review of the pathologic anatomy of the human placenta. *Am J Obstet Gynec.* 1962;84(11):1595-619.
- [12] Bajaj G, Mirchandani JJ and Chitra S. Placenta in intrauterine growth retardation. *J Obstet Gynaecol India.* 1979;29(4):805-10.
- [13] Masodkar AR, Kalamkar LR and Patki PS. Histopathology of placenta and its correlation with foetal outcome. *J Obstet Gynaecol India.* 1985; 35:294-300.
- [14] Avasthi K, Midha U, Sabharwal BD, et al. Histopathology of placenta and its correlation with foetal outcome. *J Obst Gynaec India.* 1991;41(3): 317-28.
- [15] Majumdar S, Dasgupta H, Bhattacharya K, Bhattacharya A. A study of placenta in normal and hypertensive pregnancies. *J Anat Soc India.* 2005; 54(2):34-38.
- [16] Artico LG, Madi JM, Godoy AE, Coelho CP, Rombaldi RL, Artico GR. Histopathological changes in human placentas related to hypertensive disorders. *Rev Bras Ginecol Obstet.* 2009;31(1):10-16.
- [17] Kaur P, Harsh, Kaushal S. Gross features in pregnancy induced hypertensive and normotensive placentae. *Int J Med Res Prof.* 2016;2(4):91-97.

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