

Cytotrophoblastic changes in PIH

Rasalika Miglani¹, Prabhjot Kaur², Harsh^{3,*}, Subhash Kaushal⁴

¹Demonstrator, Gian Sagar Medical College, Patiala, Punjab, ²Associate Professor, ³Assistant Professor, MMMCH, Solan, Himachal Pradesh, ⁴Professor, Govt. Medical College, Patiala, Punjab

***Corresponding Author:**

Email: harshsharma2581984@yahoo.in

Abstract

Introduction: Placenta is related to mother and fetus by indirect interaction with maternal blood that sprouts out of uteroplacental vessels. Preeclampsia and intrauterine fetal growth retardation are the disorders which provide information by the study of placental bed.

Aims: To study changes in cytotrophoblastic cells of placenta in normotensive and pregnancy induced hypertensive parturients.

Materials and Method: This study was conducted in the Department of Anatomy and Pathology, Government Medical College, Patiala. The placentae were collected from labour room, Rajindra Hospital, Patiala which include seventy five cases of pregnancy induced hypertension and twenty five cases of normotensive pregnancies. An attempt was made to find out changes in histological features of placentae of pregnancy induced hypertensive cases and compare it with the normotensive placentae.

Results: Histopathological study showed significant paucity of CTB proliferation in hypertensive group.

Conclusion: Histological changes in placenta associated with PIH are due to occlusion of the uteroplacental vasculature and thus mortality and morbidity associated with this condition is probably related to alterations in the uteroplacental flow.

Keywords: Placenta, Cytotrophoblast, Syncytium.

Introduction

The placenta is related to mother and fetus by indirect interaction with the maternal blood that spurts out of uteroplacental vessels. Blood bathes the outer syncytiotrophoblast, resulting in exchange of gases and nutrients with fetal capillary blood within the connective tissue at the villous core.⁽¹⁾

The endothelial lining of the fetal capillary and its basement membrane, the mesenchymal stroma of the villous, the cytotrophoblast and its basement membrane, and syncytiotrophoblast. The syncytiotrophoblast is relatively thick during the first four months of pregnancy but gradually becomes thinner as pregnancy advances until it forms a thin membrane in the later months.⁽²⁾

From the fourth month onwards the cytotrophoblast on the terminal villi dwindles and eventually, during second half, it is only exceptionally observed on thicker portions of the villi. In the later months of pregnancy the smaller villi show only an extremely thin membrane, not more than 0.002 mm. in thickness separating the fetal from maternal blood. Five layers of membrane, i.e., the fetal endothelium, its basement membrane, the reticular network and the syncytium with its basement membrane persist till the end of pregnancy. In many parts of a villous the endothelial basement membrane of the fetal vessels is closely or applied to the syncytial basement membrane.⁽³⁾

Microscopically, cytotrophoblastic proliferation, can be seen not only in human toxemia, but also in animals with experimentally induced toxemia or with spontaneous toxemia.⁽⁴⁾

Materials and Method

This study was conducted in Department of Anatomy and Pathology, Government Medical College, Patiala. 100 placentae were collected from labour room and from gynaecological operation theatre. Cases were divided into two groups:

1. Group I (Study 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 were grouped depending on the degree of hypertension as described by Cunningham et al.⁽¹⁾

1. Normotensive < 140/90 mm Hg
2. Mild hypertension > 140/90 - < 160/110 mm Hg
3. Severe hypertension > 160/110 mm Hg

The placenta were received in 10% formalin.

Selection of pieces from placenta was done in accordance with Salafia and Popek (1996), 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. From umbilical cord's two ends, leaving 3 cm 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 in cytotrophoblast, were noted.

The present study correlates the changes in cytotrophoblasts in normotensive and PIH cases.

The main observations and interpretations were done according to Salfia and Popek.⁽⁵⁾

Results

Cytotrophoblast Cell Proliferation: In villi of term placenta, cytotrophoblast cell proliferation is normally not present. Presence of CTB cell proliferation was graded as upto 20% and >20%.

Findings of Table 1 shows that in the study group, 49 (65.33%) cases showed proliferation of cytotrophoblast cell in their villi and 26 (34.67%) cases had no cytotrophoblast cell proliferation whereas proliferation of cytotrophoblast cells was absent in all the cases control group. 29 (38.67%) cases of study group showed presence of cytotrophoblast cell proliferation in >20% villi (Fig. 1 & 2).

The statistical difference between two groups was significant.

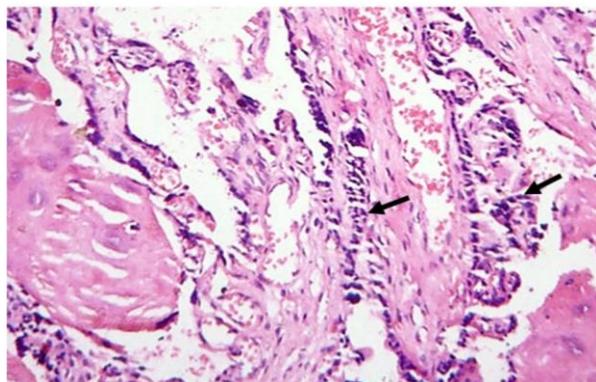
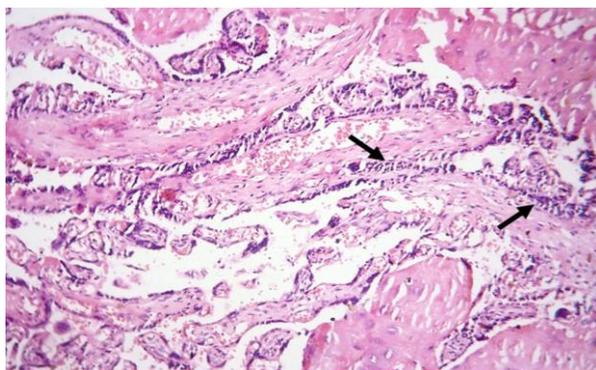


Fig. 1: Microphotograph of placenta showing cytotrophoblast cell proliferation.(H&E,40X)

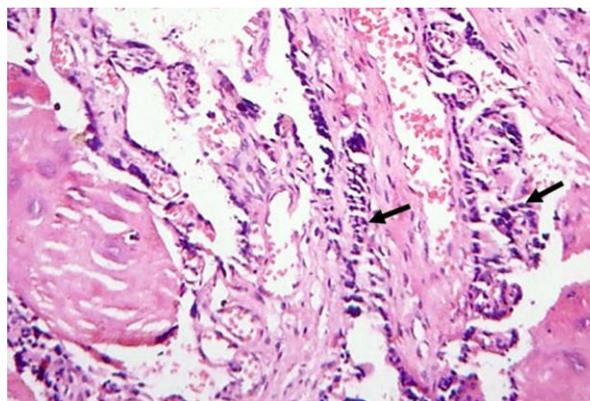
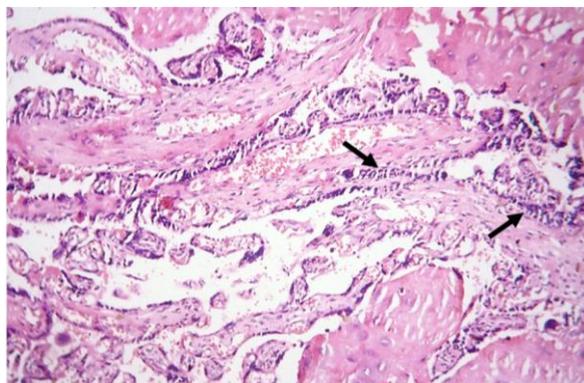


Fig. 2: Microphotograph of placenta showing cytotrophoblast cell proliferation.(H&E,100X)

Table 1: Cytotrophoblast cell proliferation in study and control groups

Cytotrophoblast cell proliferation	Group I (Study)		Group II (Control)		
	No.	%age	No.	%age	
Absent	26	34.67	25	100	
Present	Upto 20%	20	26.67	0	0
	> 20%	29	38.67	0	0
Total	75	100	25	100	

Statistical Analysis

χ^2	P value	Significance
32	<0.0001	HS

Table 2: Cytotrophoblast cell proliferation: comparison with different authors

Authors (Year)	Group	% of cases	Significance
Fox (1964)	PIH	96.3%	-
	Control	-	
Avasthi et al (1991)	PIH	65%	-
	Control	0%	
Masodkar (1985)	PIH	64.2%	S
	Control	0%	
Majumdar et al (2009)	PIH	-	S
	Control	-	
Present Study (2009)	PIH	65.3%	HS
	Control	0%	

Discussion

Microscopic Changes: In the present study, cytotrophoblast cell proliferation was seen in 65.3% cases of study group (PIH) and was not observed in any case of control group Table 2, Fig. 1 & 2.

Fox (1964),⁽⁶⁾ Masodkar (1985)⁽⁷⁾ and Avasthi et al (1991)⁽⁸⁾ observed CTB proliferation in 96.3%, 65% and 64.2% cases of PIH respectively. Majumdar et al (2005)⁽⁹⁾ observed significant number of areas of CTB proliferation in PIH group as compared to control group.

The CTB cells are the stem cells of the villous trophoblast and thus function as a germinative zone from which the syncytiotrophoblast is formed. Ischemic injury in PIH is due to decreased utero placental blood flow. Ischaemic stress causes syncytium and cytotrophoblast to proliferate in an attempt to replace the damaged tissue. CTB proliferation can also occur in other conditions responsible for ischemia or anoxemia. The oxygen requirement for CTB cells would seem to be less than those of syncytium therefore this layer shows proliferation as compared to syncytiotrophoblast.

Summary and conclusion

Microscopic study showed significant paucity of CTB proliferation, thickened basement membrane, as compared to normotensive placentae.

The perinatal mortality and morbidity associated with this condition is probably related to alterations in the uteroplacental flow.

Pregnancy which is complicated by hypertension not only affects maternal health but also jeopardize fetal normalcy. The placenta being bridge between maternal fetal activities, this structure is considered as window through which understanding of maternal dysfunction as well as of their impacts on fetal well-being can be obtained and therefore can be useful in management of future pregnancies.

Reference

1. Cunningham FG, Leveno KJ, Bloom SL, Hauth JC, Glistrap L, Wenstrom KD. Implantation, embryogenesis and placental development. In: Williams O (ed.) 22nd (edn), McGraw Hill, Medical Publishing Division, 2005; p 51, 61.
2. Hamilton WJ, Mossman HW. Physiology of the placenta. In: Hamilton, Boyd and Mossman's-Human Embryology (Prenatal Development of Form and Function) by Late Hamilton WJ and Mossman HW (eds.). Printed by Macmillan Press, London, 4th edn, 1976; p 132-161.
3. Hamilton WJ, Mossman HW. The implantation of the blasocyst and the development of fetal membranes, placenta and decidua. In: Hamilton, Boyd and Mossman's-Human Embryology (Prenatal Development of Form and Function) by Late Hamilton WJ and Mossman HW (eds.). Printed by Macmillan Press, London, 4th edn., 1976; p 83-131.
4. Soma H, Yoshida K, Mukaida T, Tabuchi Y. Morphologic changes in hypertensive placenta. Contrib Gynecol Obstet 1982;9:58-75.

5. Salafia CM and Popek EJ. Placenta. Aderson's Pathology. Mosby – Year Inc. 10th edn, 1996;2:2310-2353.
6. Fox H. The villous cytotrophoblast as an index of placental ischaemia. J Obstet Gynaec Brit Cwlt 1964;71(6):885-892.
7. Masodkar AR, Kalamkar LR and Patki PS. Histopathology of placenta and its correlation with foetal outcome. J Obstet Gynaecol India, 1985;35:294-300.
8. Avasthi K, Midha U, Sabharwal BD et al: Histopathology of placenta and its correlation with foetal outcome. J Obst Gynae India, 1991;41(3):317-328.
9. 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.