

Comparison of Basement Membrane in Normotensive and Pregnancy Induced Hypertensive Placentae

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ABSTRACT

Introduction: Placenta is related to mother and foetus via indirect interaction with maternal blood that spurts out of uteroplacental vessels. By study of placental bed new information has come to light, especially for pre – eclampsia and intra uterine fetal growth retardation.

Aim: To study changes in basement membrane in pregnancy induced hypertensive and normotensive placentae.

Materials and Methods: The present study was conducted in the Department of Anatomy, MMMCH, Kumarhatti, Solan to study the histological changes in placenta in pregnancy induced hypertensive parturients as compared to normotensive subjects. It was based on 100 cases, 25 from normotensive subjects (control group) and 75 from clinically proven cases of pregnancy induced hypertension (study group).

An attempt was made to see any changes in histological features of placentae of pregnancy induced hypertensive cases and compare it with normotensive placentae.

Results and Conclusion: The microscopic study showed

significant thickening of basement membrane in PIH placentae. As compared to normotensive placentae (control) and are due to occlusion or narrowing of utero placental vasculature suggesting thereby compromised foeto – placental circulation.

Keywords: Basement Membrane, Placenta, PIH.

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INTRODUCTION

Placenta is the marker of biological events occurring during pregnancy.¹ The placenta has the shape of the flattened cake (plakuos = placenta = cake) at full term. Roughly the mature placenta has diameter of 15 – 20 cm, thickness 3 cm, volume 500ml and weight about 500gms. During the 2nd half of pregnancy, it increases in weight less rapidly than fetus, so that at term it becomes insufficient to meet the nutritional requirements which are compensated by “placental barrier” thinning separating the fetal and maternal blood streams, thus increasing its efficiency in transmission.² Fetal growth and metabolism depends on the adequate exchange across placenta. Placental inadiquence and altered placental function may result from primary pathological alternations in the mother, fetus or placenta leading to intrauterine growth retardation.³

MATERIALS AND METHODS

The present study was conducted in the Department of Anatomy, MMMCH, Kumarhatti, Solan during period of July 2016 to December 2018. Total of 100 placenta were collected from labour room and from gynecological operation theater, MMMCH;

Kumarhatti, Solan. Ethical approval was taken prior to the study from the institutional ethical committee, MMMCH, Kumarhatti, Solan. Cases were divided into 2 groups:

- Group I (Study/ PIH Group): 75 cases of clinically proved PIH
- Group II (Control group): 25 singleton normotensive pregnancies

Cases with period of gestation more than 35 weeks were taken for study. The placenta were grouped depending on the degree of hypertension as described in the cunningham et al (2005).

- Normotensive < 140/90 mmHg
- Mild hypertension ≥ 140/90 - <160/110 mmHg
- Severe hypertension ≥ 160/110 mmHg

The placentae were received in adequate amount of 10% formalin. The placentae were examined grossly and following morphological features were noted.

1. Weight of the placenta
2. Volume of placenta was recorded by water displacement technique.
3. Assuming the placenta to be a perfect circle, the mean diameter was estimated.

4. Birth weight of infant was recorded in each case to calculate fetoplacental ratio.

5. Site of insertion of umbilical cord

6. Any gross abnormality in size and shape, gross infarcts, hemorrhages, necrotic areas and calcification was observed.

Selection of pieces from placenta was done in accordance with Salafia and Popek (1996), who recommended minimum sections from placenta for histopathology.

1. Section from membrane roll

2. From central area of maternal surface

3. From central area of fetal surface

4&5. 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 like thickening of basement membrane, avascular fibrosed villi and fibrinoid deposits were noted. Besides gross morphological and histopathological examination of placenta, clinical evaluation of patients and routine hematological and biochemical tests were also considered.

Data was compiled in a Performa. The main observations and interpretations were done according to Salafia and Popek (1996).⁴

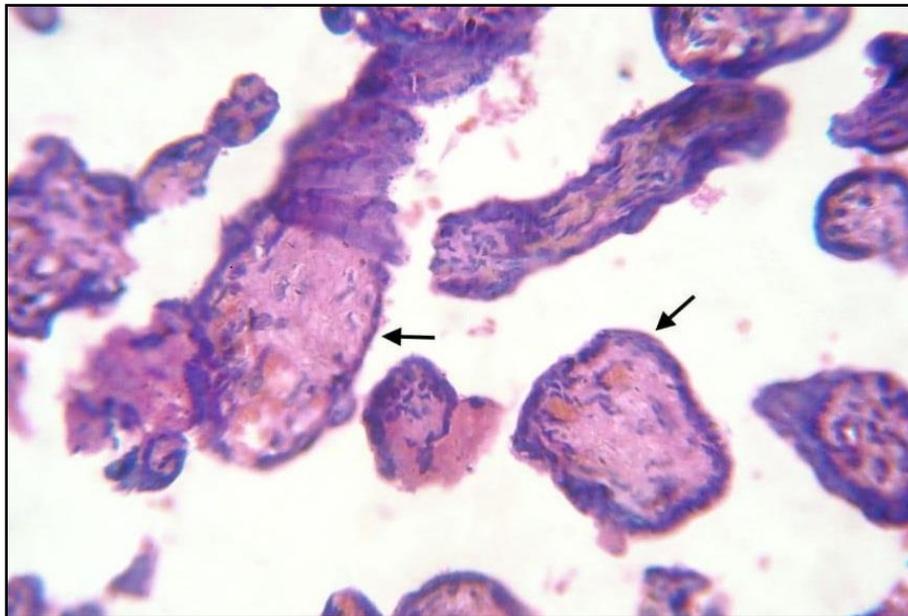


Figure 1: Microphotograph of placenta showing thickened basement membrane and non – formation of vasculo syncytial membrane.

Table 1: Thickened basement membrane in study and control groups

Thickened basement membrane	Group I (Study)		Group II (Control)	
	No.	%age	No.	%age
Absent	17	22.67	25	100
Present	58	77.33	0	0
Total	75	100	25	100
Statistical Analysis				
χ^2	P value		Significance	
46	<0.0001		HS	

Table 2: Thickened basement membrane (Comparison with other studies)

Authors (Year)	Group	% of cases	Significance
Masodkar et al (1985)	PIH	90.4	-
	Control	0	-
Avasthi et al (1991)	PIH	60	-
	Control	0	-
Present Study (2009)	PIH	77.3	HS
	Control	0	-

RESULTS

The present study was conducted in the Department of Anatomy, MMMCH, Kumarhatti, Solan. The placentae were collected from labour room and from Gynaecological Operation Theatre, Department of Obstetrics and Gynaecology, MMMCH, Kumarhatti, Solan. Histological features of placentae were studied in pregnancies complicated by pregnancy induced hypertension and compared that findings with those of control group.

Table 1 shows that thickened of basement membrane was present in 58 (77.33%) cases in the study group (Figure 1). Thickened basement membrane was not observed in any of the cases of control group. The statistical difference between two groups was highly significant.

DISCUSSION

Pregnancy complicated by hypertension not only affects maternal health but also jeopardizing fetal normalcy. The placenta being the bridge between maternal fetal activities, this structure is considered as a window through which understanding of maternal dysfunction as well as of their impacts on fetal wellbeing can be obtained. The present study was conducted in the Department of Anatomy, MMMCH, Kumarhatti, Solan to study the histological changes in placenta in pregnancy induced hypertension as compared to normotensive subjects. It was based on 100 cases, 25 from normotensive subjects (control group) and 75 from clinically proven cases of PIH (study group).

In the present study, thickened basement membrane was found to be associated with 77.3% PIH placentae. Thickened basement membrane was not observed in any of the cases of the control group. The statistical difference between two groups was highly significant (Table 1, Figure 1).

Thickened basement membrane was observed in 90.4% cases of PIH by Masodkar et al⁵ (1985) and 60% cases of PIH by Avasthi et al⁶ (1991). No thickened basement membrane was observed by them in placentae of control group.

LiC et al (2000)⁷ and Wang et al (2002)⁸ found significant thickening basement membrane in pregnancy-induced-hypertension group, when compared to normal term pregnancy.

Thickening of villous trophoblast thick membrane is usually is response to placental ischemia causes by pregnancy induced hypertension.

SUMMARY AND CONCLUSION

Placenta is one of the most challenging organ. Pregnancy induced hypertension (PIH) continue to be a significant source of fetal mortality and morbidity and recent evidence suggests that incidence of PIH is increasing. The present study was conducted in the Department of Anatomy, MMMCH, Kumarhatti, Solan to study the histological changes in placenta in pregnancy induced hypertension as compared to normotensive subjects. It was based on 100 cases, 25 from normotensive subjects (control group) and 75 from clinically proven cases of PIH (study group).

An attempt was made to see any changes in histological features of placentae of pregnancy induced hypertensive cases and compare it with normotensive placentae.

The microscopic study showed significant thickening of basement membrane 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 associated with this condition is probably related to alterations in the uteroplacental flow.

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