

Histological Study Of Human Placenta In Normal And Pregnancy Induced Hypertension (PIH) Cases

Mrs Pooja Dhabhai¹, Dr Ghanshyam Gupta², Dr Neelam Bapna³

^{1,2} (Department of Anatomy, R.N.T. Medical College, Udaipur, Raj. India)

³ (Department of Anatomy, NIMS, Medical College, Jaipur, Raj. India)

ABSTRACT: The placentas of 30 mothers with PIH were compared to Placentas collected from mothers who had uncomplicated pregnancies. This study demonstrated that, on a quantitative histological basis, the placentas of the PIH mothers were morphologically very similar to the control placentas in terms of parenchymal and cellular content. The findings of this study support the hypothesis that, in PIH some alterations in uteroplacental blood flow and possibly umbilical blood flows causes significant changes in placental structure and function.

KEYWORDS: Fibrosis, Syncytiotrophoblast, Syncytial knots, Toxaemia, villi.

I. INTRODUCTION

PIH, still a major complication of pregnancy, jeopardizes Both mother and fetus. However, many gaps still exist in our knowledge concerning the cause and pathophysiology of this disease. A wide variety of morphologic changes has been reported in the placentas of PIH Women, however the definitive pathogenesis of these structural abnormalities, their correlation with placental function, and their relationship to perinatal loss and morbidity in these high risk pregnancies have not been clearly established.

II. MATERIAL AND METHODS

The study of placentae in normal and Pregnancy induced hypertension cases was carried out at S.M.S. Medical College & Hospital, Jaipur. The placentae were collected from sixty women admitted to the labour Rooms of the hospital (either directly or through the antenatal wards). All the cases were within the age group of 18-40 years, of average height and weight and includes both primigravida and multigravida.

GROUP 1- NORMAL PREGNANCY 30 patients included in this group, normal Hb and urine analysis, not associated with any disease.

GROUP 2-PIH CASES 30 cases of PIH including both mild and severe, only those cases having Blood pressure ranging 140/90 mmHg and above, with and without oedema, and with proteinuria were included.

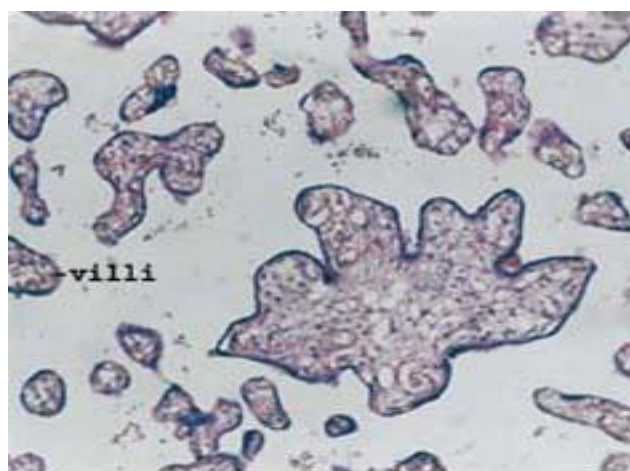


Figure 1-Normal mature Placenta showing the villi

For histopathological studies tissues were taken from the following placental sites.

- 1.Near the implantation of the umbilical cord
- 2.Center of the placenta.
- 3.Fibrotic area if any.
- 4.Infarcted area,if any.
- 5.Umbilical cord at placental junction and cut end of the stub.

Sections were prepared with paraffin embedding and H & E staining

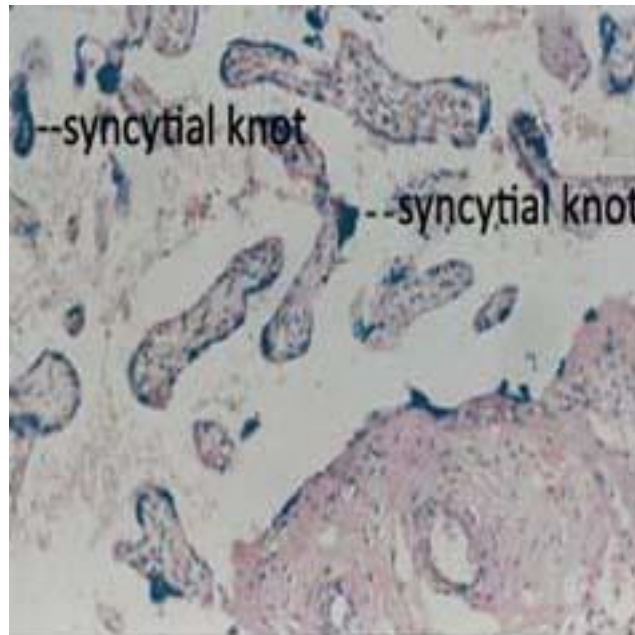


Figure 2-Numerous Syncytial knots in PIH Placenta

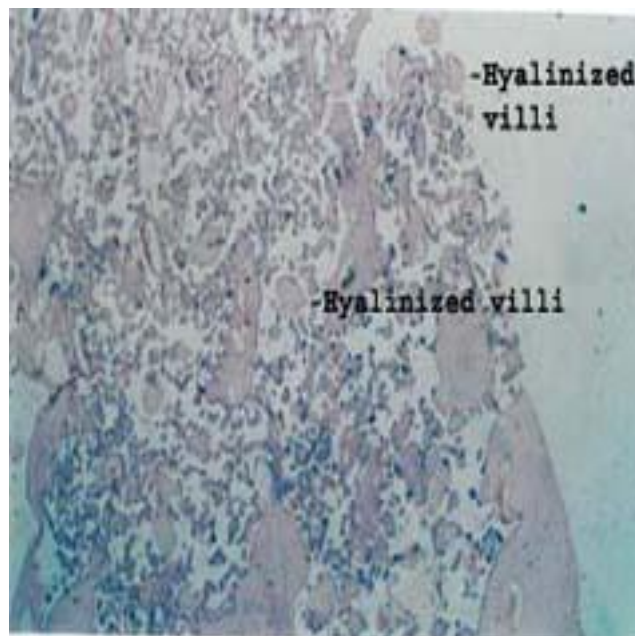


Figure 3-PIH Placenta showing Hyalinized villi

III. OBSERVATIONS & RESULT

Each histological lesion is counted in per Low power field.The findings observed in abnormal cases were compared with that of normal cases.Significant finding in histology was the premature aging as seen in placentae which increased with the increase in severity of the disease.Syncytial knots were present in as high as 25 cases out of the total 30 cases(83%).increase in no. Of hypovascular villi and fibrinoid necrosis was seen in very large no.of cases. Villi showed increased fibrosis contributing to the premature aging (33%).

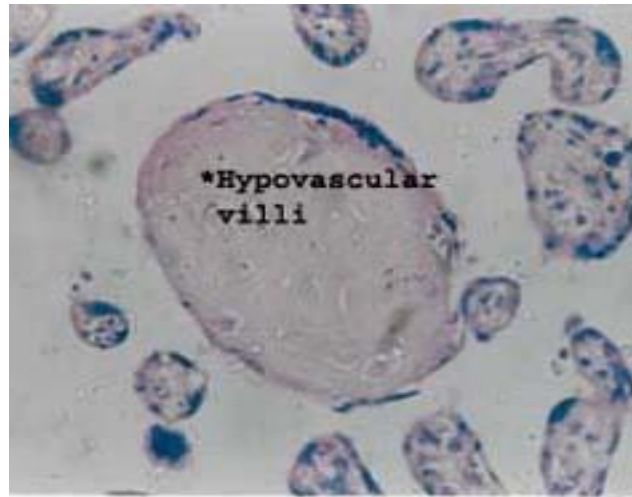


Figure 4-PIH Placenta showing Hypovascular Villi.



Figure 5-PIH Placenta showing Stromal fibrosis.

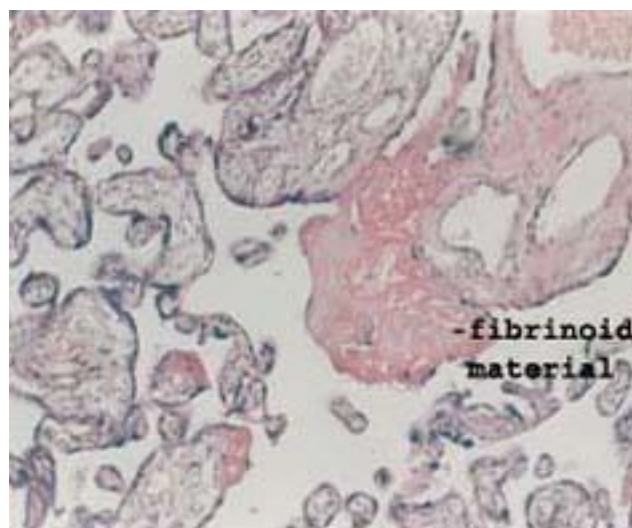


Figure 6-PIH Placenta showing Fibrinoid necrosis.



Figure 8-In PIH Placenta complete villous fibrinoid necrosis, remnants of syncytiotrophoblast seen in some areas.

TABLE NO-I

Histopathological findings	No.of cases	% of cases
Fibrinoid necrosis	17	57%
Syncytial knots	25	83%
Villous fibrosis	10	33%
Hypovascular villi	15	50%

TABLE NO-II

Birth weight	Total no.of cases	Hypovascular villi	% of cases	With increased fibrinoid necrosis	% of cases
Below 2000 gms	9	8	89%	8	89%
2001-2500 gms	14	2	14%	4	29%
2501-3000 gms	6	2	33%	1	16%
Above 3000 gms	1	1	100%	1	100%

TABLE NO.III
STATISTICAL ANALYSIS OF HISTOLOGICAL EXAMINATION OF PLACENTAL VILLI

S.NO	<i>Histological lesions</i>	<i>Normal (30) mean+_S.D.</i>	<i>PIH(30) mean+_S.D.</i>	<i>P value</i>
1	Syncytial knots	6.5+_4.9	23.1+_7.52	P<0.001
2	Hyalinized villi	0.26+_1.0	5.7+_3.04	P<0.001
3	Fibrinoid necrosis	2.96+_3.52	6.26+_2.8	P<0.001
4	Stromal fibrosis	2.23+_1.73	31.23+_42.29	P<0.001

IV. DISCUSSION

The study was carried out using following parameters-

4.1 Syncytial knot formation-(These are foetal clumps of syncytial nuclei protruding from the outer surface of villous trophoblasts. There is a disagreement regarding formation of these knots) In the present study statistically significant syncytial knot count were seen in cases of toxemia. Fox(1965) concluded from his study that only factor related with syncytial knot formation is length of gestation and suggested that within limits it can be regarded as an index of placental maturity. Statistically significant high count seen in toxemia may be related to reduced foetal villous flow in these cases. Fox concluded from his study that excess syncytial knot formation occur in generalized form whenever the foetal circulation through the villi appears to be reduced. Irrespective of the mechanism which is responsible for reducing fetal blood flow through the villi the inevitable result is stromal fibrosis and excess syncytial knot formation both of which are good indices of the degree of reduction in villous perfusion.

4.2 Villous Hypovascularity(Hyalinized villi)- The mechanism involved in producing hypovascular villi is not fully understood, In the present study statistically significant count of hyalinised villi was observed in cases of toxemia. Teasdale(1980) and Udainia et al (2004) reported that fibrinoid necrosis, endothelial proliferation of arteries and hyalinization depict the mosaicism of placenta and probably the aftermath of hypertension again the mosaicism of the placenta probably leads to placental insufficiency and ultimately to foetal growth retardation, Zacutti(1992) thus creating a vicious cycle.

4.3 Stromal fibrosis- In the present study statistically significant stromal fibrosis was seen in 34% cases of toxemia. Similar to our study Harianne et al(1976) also found statistically significant level of stromal fibrosis in cases of toxemia but they also obtained highly significant level (P<0.01) in cases of anaemia. This they explained is due to relative anoxia in this condition. The two main factors thought to be responsible for the formation of stromal fibrosis are normal aging and reduced blood flow, stromal fibrosis is due to reduced blood flow seems more conclusive as excess stromal fibrosis was seen in cases of toxemia. This hypothesis would explain the results obtained in this study, the high incidence of villous fibrosis in placentae from pregnancies complicated by toxemia is probably due to reduction of the foetal blood flow by obliterative endarteritis of the foetal stem arteries, which is a common of such placentae.

4.4 Fibrinoid Necrosis- Fibrinoid necrosis of placental villi is a highly characteristic lesion. The first step in the evolution of this abnormality is the appearance of a small "blob" of homogenous material in the trophoblast, this substance lies deep to the syncytiotrophoblast and external to the basement membrane, from which it is quite distinct. The "blob" of abnormal material gradually enlarges, the accretion always being on deep surface, so that the fibrinoid material bulges progressively into the villous stroma. It does not invade the stromal tissue, for the underlying trophoblastic basement membrane remains intact and is pushed inwards to form a gradually deepening crescent. This process continues until the whole of the villus is replaced by fibrinoid matter. The syncytium of the affected villus shows a progressive degeneration, but even in the final stages a few remnants of this tissue remain. Hence the final appearance is that of a mass of structureless, homogenous, acidophilic material around the periphery of which are a few degenerate syncytial nuclei. Many workers have thought that this lesion is due to replacement of the villus by fibrin, this being formed either from the maternal blood in the intervillous space. (Mckay et al. 1958; Wigglesworth, 1964) or from the fetal blood in the villous capillaries (Kline, 1951; Emmrich, 1966).

V. CONCLUSION

Statistically significant counts of syncytial knots $P < 0.001$ and hypovascular villi seen in cases of PIH, suggesting their presence due to reduced villous blood flow. Statistically significant counts of Stromal fibrosis and Fibrinoid necrosis were also seen in cases of PIH, it may indicate an immunological reaction within placental tissue responsible for premature onset of labour or it may be a degenerative process.

REFERANCES

- [1] Fox, H.J. *Obstet Gynaec Brit Cwith* 72:347, 1965.
- [2] Teasdale F. *Gestational changes in functional structure of the human placenta in relation to foetal growth. Am J Obst* 1980;137:560-562.
- [3] Udainia et al (2004): *Relation between placental surface area, infarction and foetal distress in pregnancy induced hypertension with its clinical relevance. J Anat Soc. Ind., 2004, 53; 1:27-30.*
- [4] Zacutti A et al. *Umbilical blood flow and placental pathology. Clin Exp Obst and Gynaecol* 1992;19:63-69.
- [5] Mckay et al (1958): *Obstet. and Gynaec.* 12, 1.
- [6] Wigglesworth, J.S.: *J. Obst. & Gynaec. Brit* 71:871, 1964.
- [7] Kline, B.S.: *Am. J. Obstet. & Gynae.* 61:1065, 1951.