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Original Research Article

## Placenta in pregnancy induced hypertension

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### ABSTRACT

**Background:** Placenta is a predictor of outcome of pregnancy. Placental examination holds an important role in improving outcome of pregnancy. It is strategically located at fetomaternal interface and it acts like a record of pregnancy in which the cumulative effects of pregnancy related events and changes reflecting the intrauterine environment can be scrutinized. Objective of present study was to diagnose presence, if at all, of lesions of placenta and their nature in cases of pregnancy induced hypertension specifically in 2<sup>nd</sup> and 3<sup>rd</sup> trimester of pregnancy and to classify the lesions based on clinical presentation and to compare the results.

**Methods:** This study is based on histopathological examination of placenta in cases of PIH over a period of three years, from May 2011 to April 2014. The study was carried out at a tertiary care hospital.

**Results:** There were 280 (19.4%) cases of PIH amongst a total of 1440 cases. In these 280, 170 (60.72%) cases had severe PIH, 110 (39.28%) cases had mild PIH. Also, 180 (16.28%) cases revealed increased morbidity and mortality.

**Conclusions:** Evaluation of placenta is an extremely important predictor, that helps to improve the outcome of pregnancy.

**Keywords:** Placenta, PIH, Predictor

### INTRODUCTION

The entire existence of fetus in utero is solely dependent on only one vital organ "The Placenta". Placenta is essential for both maintenance and promotion of growth and development of fetus.<sup>1</sup> The most accurate record of the infant's prenatal experience is the placenta.<sup>2</sup> Pregnancy-induced hypertension (PIH) is the leading cause of maternal, fetal and neonatal mortality.

Pregnancy complications reflect in a significant way, both macroscopically and microscopically, in the placenta. Several studies have shown that there is uteroplacental insufficiency in PIH due to maternal vasospasm.<sup>3</sup> This leads to vasoconstriction of fetal stem arteries and hence the changes seen in the placenta of preeclamptic women.<sup>4</sup>

Maternal vasospasm leads to fetal hypoxia and also may lead to fetal distress and fetal death.<sup>5</sup> The placenta is the mirror of maternal and fetal status. This study was carried out to diagnose changes, gross and microscopic, in placenta in cases of pregnancy induced hypertension over a period of 3 years from May 2011 to April 2014.

### METHODS

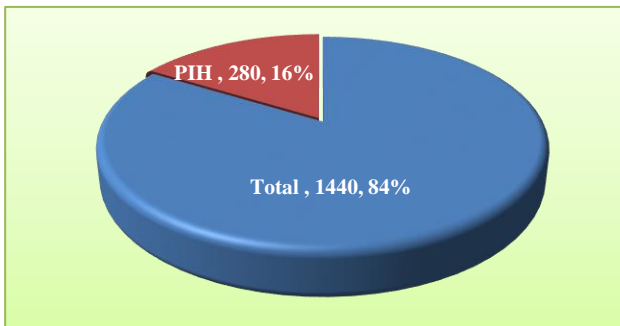
This study consists histopathological examination of placentae in cases of PIH from May 2011 to April 2014. The study was carried out at a tertiary care hospital.

All the placentae in cases of pregnancy induced hypertension, in 2<sup>nd</sup> and 3<sup>rd</sup> trimesters of pregnancy were included in the study. Placentae of first trimester of pregnancy were excluded from the study.

Placenta, membranes and umbilical cord were submitted to the pathology department for examination. After receiving the specimen it was washed in running tap water, weighed and cut into vertical segments of 1-2 cm thickness from maternal to fetal surface to ensure proper fixation and then it was fixed in adequate volume of 10% formalin for 1 week. All the placentae were handled in accordance to Universal precautions. Thorough gross examination of the placenta was done with careful review of the umbilical cord, placental membranes, fetal and maternal surfaces. All significant lesions were noted. The diagnosis was further made on microscopy in the light of clinical details and investigations.

**RESULTS**

We observed 280 (19.4%) cases of PIH amongst a total of 1440 cases (Figure 1).



**Figure 1: Proportion of PIH.**

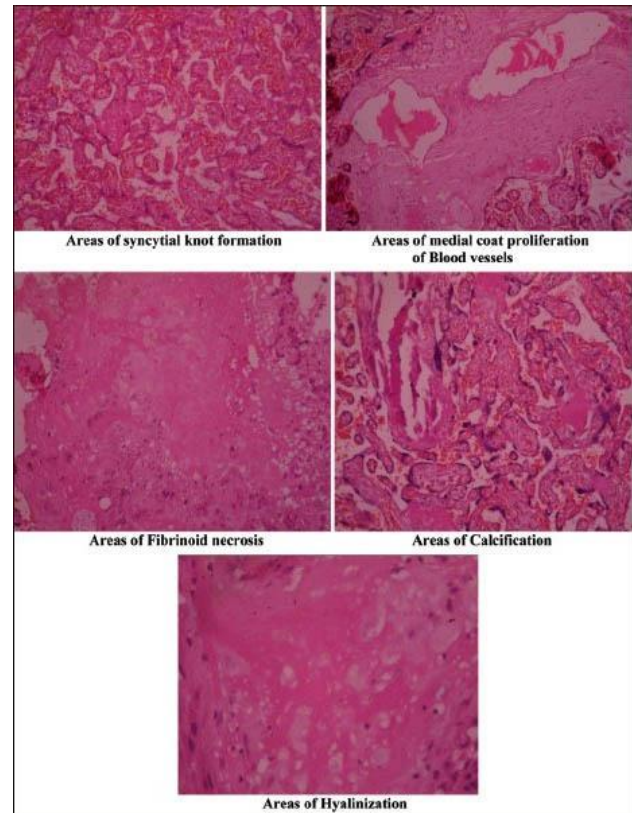
170 (60.72%) were having severe PIH, while 110 (39.28%) were having mild PIH. Out of total 280 cases of PIH, 180 (64.28%) cases showed high morbidity and mortality which included, abruptio placentae 80, DIC 20, HELLP syndrome 10 intra uterine losses 52, still births 9, eclampsia 9 cases (Table 1).

**Table 1: Complications.**

Pathology	Number	Percentage
Abruptio placentae	80	44.40
DIC	20	11.11
HELLP	10	5.56
Intra-uterine losses	52	28.89
Still births	9	5
Eclampsia	9	5
Total	180	

Amongst severe PIH cases, 130 (76.4%) placentae showed low placental weight, in mild PIH only 20 (18.2%) placentae showed the same for the corresponding gestational age. It revealed that low placental weight was significantly associated with PIH (Chi-square=6.930 and P value 0.08). Weight of baby was smaller than expected, for the corresponding gestational age in 140 (82.3%) cases of severe PIH and 40 (35%) cases of mild PIH. It revealed that low birth weight was significantly

associated with PIH (Chi-square=4.312 and P value 0.03).



**Figure 2: Histopathological changes.**

Placentae were smaller in PIH. Commonest pathology seen was large multifocal infarcts, in 120 (42.85%) cases, followed by retro- placental clot in 80 (28.50%) cases, abruptio placentae 80 (22.40%) cases and calcification in 20 (7%) cases (Figure 2). The base and edge infarcts were most extensive. Infarcts were seen in 100 (58%) cases of severe PIH and 20 (18.2%) cases of mild PIH. Uteroplacental insufficiency (UPI) was the major pathological diagnosis in 78.6% cases as documented in literature.<sup>6</sup> Uteroplacental insufficiency (UPI) reflected increased infarcts, syncytial knots, maternal vessel thrombosis and fibrinoid necrosis.

**DISCUSSION**

The study included 280 (19.4%) cases of PIH. 60% of cases revealed severe PIH. Similar observations found in study by Narasimha A et al.<sup>7</sup> In this study increased morbidity and mortality included abruption placentae 80, DIC 20, HELLP syndrome 10, intra uterine losses 52, still births 9, eclampsia 9 cases. An almost similar clinical outcome was obtained by Alexander et al who studied 6518 patients with PIH.<sup>8</sup>

On gross examination, small placentae and large multifocal infarcts in 120 (42.85%) cases is the commonest pathology, followed by retroplacental clot 80

(28.5%) cases, abruption 60 (21.42%) cases and calcification 20 (7%) cases. The basal and edge infarcts of placenta were extensive and more commonly seen in cases of severe PIH. An alteration in the hormonal factors probably leads to altered blood flow and hence a significant increase in syncytial knot formation in placental. According to Robertson, the cause of reduction in blood flow is due to vasculopathies of spiral arteries, which in turn causes reduction in the weight of placenta. It has been recorded that maternal utero-placental blood flow is decreased in preeclampsia because of maternal vasospasm. Reduced maternal utero-placental blood flow indirectly leads to constriction of fetal stem arteries.<sup>6 (9)</sup>

It revealed that low placental weight was significantly associated with severe PIH. These findings correlated with other studies.<sup>6,10,11</sup> Weight of baby was smaller than expected gestational age in 140 cases (82.3%) of severe PIH, and 40 cases (35%) of mild PIH. It revealed that low birth weight was significantly associated with severe PIH which correlate with findings of other studies.<sup>6,10-14</sup>

## CONCLUSION

PIH contributed to 19.4% of cases amongst all the placentae received. Placental examination helps reveal increased morbidity and mortality in the form of low placental weight and associated low birth weight of baby. The histopathological changes of PIH and uteroplacental insufficiency revealed various structural changes such as significant number of syncytial knots, areas of fibrinoid necrosis, areas of medial coat proliferation of medium sized blood vessels, areas of calcification, and areas of hyalinization. Thus placental changes occurring in PIH directly affect the growth and nutrition of fetus in utero. A detailed history, clinical examination and investigations of mother and ultrasonography play an essential role in the interpretation of histopathological diagnosis of placental lesions.

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## REFERENCES

1. Udainia A, Jain ML. Morphological study of placenta in pregnancy induced hypertension with its clinical relevance. J Anat Soc India. 2001;50:24-7.
2. Benirshke K. The placenta: How to examine it and what you can learn. Contemp Obstet Gynecol. 1981;17:117-9.

3. Bewly S, Cooper D, Campbell S. Doppler investigation of utero-placental blood flow resistance in the second trimester. A screening study for pre-eclampsia and intra-uterine growth retardation. Br J Obst Gynaecol. 1991;98:871-9.
4. Stock MK, Anderson DF, Phernetton TM, McLaughlin MK, Rankin JH. Vascular response of the maternal placental vasculature. J Dev Physiol. 1980;2:239-46.
5. Thomson AM, Billewicz, Hytten FE. Placenta in relation to birth weight. J Obstet Gynecol Br CW. 1969;76:865-72.
6. Majumdar S, Dasgupta H, Bhattacharya K, Bhattacharya A. A study of placenta in normal and hypertensive pregnancies. J Anat Soc India. 2005; 54(2):1-9.
7. Narasimha A, Vasudeva DS. Spectrum of changes in Placenta in toxemia of pregnancy. Indian J Pathol Microbiol. 2011;54 (1):15-20.
8. Cunningham F, Leveno K, Bloom S, Hauth J, Rouse D, Spong C. Pregnancy Hypertension. Williams Obstetrics: 23rd Ed. United States. McGraw-Hill; 2010;Chapter 34:706-756.
9. Boyd PA, Scott A. Quantitative structural studies on human placenta associated with preeclampsia essential hypertension and intrauterine growth retardation. Br J Obstet Gynaecol. 1985;92:714-21.
10. Udainia A Jain M.L Morphological Study of Placenta in Pregnancy Induced Hypertension with its Clinical Relevance. J Anat Soc India. 2001;50(1):24-7.
11. Palaskar A, Chaudhary KR, Mayadeo NM. etoplacental weight relationship in normal pregnancy and preeclampsia-eclampsia. Bombay Hospital J. 2001;43(3):361-3.
12. Thanawala U, Khopkar K. Intrauterine growth restriction, Diagnosis and management. In: Desai P, Malhotra, Shah-editors. Principles 127 and Practice of Obstetrics and gynecology. 3<sup>rd</sup> Ed. Jaypee brothers. New Delhi;2008:204.
13. Mohite SS, Umarji BN, Doshi MA, Karambelkar RR. Normal full term human placenta-morphological study. Res J Krishna Institute, Karad. 2009 July;2(2):45-7.
14. Chakravorty AP. Fetal and placental weight changes in normal pregnancy and pre-eclampsia. J Obstet Gynaecol Br Commonwealth. 1967;74:247-53.

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