Original Article

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Anatomy Section

Gross Changes of Placenta in Pregnancy Induced Hypertension and its Clinical Relevance: A Cross-sectional Study

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ABSTRACT

Introduction: Placenta establishes connection between mother and foetus through umbilical cord. Pregnancy Induced Hypertension (PIH) is one of the most common complication of pregnancy. Gross study of placenta in PIH gives an insight into prenatal health of baby and mother, thereby preventing maternal as well as foetal morbidity and mortality.

Aim: To study the gross changes of placenta in PIH cases with its clinical relevance.

Materials and Methods: A prospective cross-sectional study was done at tertiary care hospital of Osmania and Gandhi Medical College, Telangana, India. The study was carried over for a period of six months (from October 2019 to March 2020) with a sample size of 50 placentae in PIH mothers aged between 20-35 years who were inpatients at the Obstetrics Department. They were followed until delivery. Postdelivery, the gross anatomy of the collected placentas was studied. Varied parameters of placenta, data of newborn and clinical picture of mother were compared with normal standards and statistically analysed by using Unpaired t-test.

Results: Significant reduction of mean placental weight and mean foetal weight (p-value<0.0001) and significant increase in foetoplacental weight ratio was found. There was an average reduction of number of cotyledons by 23% in PIH cases when compared to normal placentae. An incidence of mean infarct areas of 9.6 and mean calcified areas of 13 were found on the maternal surface of placenta in PIH. An incidence of 68% of marginal type of placenta was observed in patients with PIH. Clinically, incidence of headache, pedal oedema, convulsions, visual disturbances and proteinuria were found.

Conclusion: Gross study of placenta in PIH reflects maternal and foetal status and can be taken as confirmative evidence by radiologists to prevent Intra Uterine Growth Retardation (IUGR) and guide obstetricians during management of pregnant mother with PIH. Thereby, preventing the maternal and foetal morbidity and mortality.

Keywords: Calcification, Infarcts, Pre-eclampsia, Umbilical cord

INTRODUCTION

Placenta is essential for maintenance of pregnancy and for promoting normal growth and development of foetus [1]. During foetal development, until the organs become functional, the placenta does most nutritive and respiratory supportive function to foetus. These include maintenance of homeostasis, provision of oxygen and gaseous exchange, waste removal, hormonal secretion and haemopoiesis [2]. The placenta is defined as a fusion of the foetal membranes to the uterine mucosa for the transfer of oxygen and metabolites between maternal and foetal blood [3].

Pregnancies complicated by pre-eclampsia are reflected in the placenta both macroscopically and microscopically and may be diagnosed early by thorough serial placental ultrasound examinations [4]. Pre-eclampsia remains a common complication of pregnancy that leads to unacceptable increase in foetal and maternal morbidity and mortality, particularly in less developed nations [5].

In pre-eclampsia, the normal endovascular invasion of cytotrophoblast into spiral arteries fails to occur beyond decidua-myometrial junction [6]. Maternal vasospasm leads to foetal hypoxia and accordingly, it may lead to foetal distress and foetal death [7]. Most common foetal anomalies associated with PIH are microcephaly and hypospadias [8].

From the studies of Salmani D et al., Kambale T et al., Goswami PR and Shah SN done in India, it was inferred that there are changes of placental morphometry and circulatory changes in placenta with PIH, which is adversely affecting the neonatal birth weight and perinatal outcome of the mother [9-11]. This relationship between foetus, placenta and PIH provides useful adjunct in planning and management of future pregnancy in hypertensive women. The present study aimed to find out the morbid morphological changes of the placenta in pregnancy induced hypertension with its clinical relevance.

MATERIALS AND METHODS

A prospective cross-sectional study was done at tertiary care hospital of Osmania and Gandhi Medical College, Telangana, India. The study was carried over for a period of six months (from October 2019 to March 2020). Sample size was calculated accordingly 50 placentae of PIH cases as done in previous study conducted by Goswami PR and Shah SN [11]. The Ethical clearance taken from Institutional Ethics Committee of Osmania Medical College, Hyderabad (with Reg. No. ECR/300/Inst/AP/2013/RR-19 and Ref. no. IEC/OMC-46/(Acad)/06/2020/24) was taken.

Inclusion criteria: Placentae along with umbilical cord of PIH mothers, age between 20-35 years with normal vaginal delivery at term and newborns completing nine months of gestational age without any congenital anomalies were included.

Exclusion criteria: Mothers who underwent cesarean section, diseased mothers other than PIH, mothers with bad obstetric history and preterm deliveries and newborns with congenital anomalies were excluded.

Study Procedure

Informed consent was obtained from all subjects who participated and they were followed until delivery. Postpartum placentae were collected by cutting the umbilical cord at a distance of 5 cm from the site of insertion for observing changes in gross anatomy and also noting the birth weight of newborn excluding any congenital anomalies. Placentas were washed in running tap water dried up and labelled. The placentae were weighed before preserving to avoid error and observed for fresh infarcts. Then placentas along with the umbilical cord were identified by corresponding code numbers and were preserved in 10% formalin solution for four weeks. Gross features like type of placenta, number of cotyledons, number of infarcts, calcifications were recorded along with placental and foetal weights. From the recorded foetal birth weights and placental weights, foetoplacental ratios were calculated. The incidence of variations of placenta based on the insertion of umbilical cord (central, marginal and velamentous) in PIH was recorded [12].

The normal standards of placental weight, foetal weight, foetoplacental ratio and number of cotyledons were taken from a similar study done in southern India by Salmani D et al., [9]. The case sheets of mothers were collected and noted for clinical features of PIH-primigravida/ multigravida, blood pressure, headache, pedal oedema, convulsions, visual disturbances, proteinuria and IUGR [13]. IUGR was noted according to the ultrasound report during antenatal check-up of mother as per case records.

STATISTICAL ANALYSIS

The statistical significance between the study group and normal standards were analysed by using Student's Unpaired t-test. The p-value <0.05 was considered statistically significant. Statistical analysis was done by using Epi Info 7.1 software.

RESULTS

Of 50 PIH cases, 31 (62%) were primigravida and 19 (38%) were multipara. The means of placental and newborn weights were reduced significantly (p-value <0.05) in PIH compared with normal standard. The foetoplacental ratio was significantly higher in PIH. The average number of cotyledons was found to be significantly reduced by 23% in PIH [Table/Fig-1].

Parameters	Normal (Mean±SD)	PIH (Mean±SD)	Unpaired t-test	p-value
Placenta weight	519.80±59.23 gm	338.76±53.60 gm	16.02	<0.05
Foetal weight	3140±280 gm	2156.62±240.54 gm	18.79	<0.05
Foetoplacental weight ratio	5.72±0.93	6.58±1.14	6.64	<0.05
No. of cotyledons	21.18±1.48	14.2±1.087	4.37	<0.05

[Table/Fig-1]: Morphological parameters of placenta and foetus.
p-value <0.05 was considered statistically significant

The mean number of infarcts of 9.6 and calcified areas of 12.8 were found in PIH. Incidence of marginal type of placenta was found to be 68% and central type 32% in PIH cases [Table/Fig-2].

Parameters	Values
Mean number of infarct areas	9.64±3.27
Mean number of calcified areas	12.86±4.67
Percentage of marginal type of placenta	34 (68%)
Percentage of central type of placenta	16 (32%)
Percentage of velamentous type	0

[Table/Fig-2]: Gross features of placenta.

In this study, it was observed that the mean maternal diastolic blood pressure was 105 mmHg and mean systolic pressure was 149 mmHg. The most common symptoms observed were pedal oedema and headache and the most common signs presented were proteinuria and IUGR [Table/Fig-3].

Clinical Feature	n (%)
Headache	35 (70)
Pedal oedema	38 (76)
Convulsions	8 (16)
Visual disturbances	5 (10)
Proteinuria	50 (100)
Intra Uterine Growth Retardation (IUGR)	43 (86)
Mean blood pressure	149/105 mmHg

[Table/Fig-3]: Clinical features in PIH.

DISCUSSION

The present study showed significant reduction of placental weight, foetal weight and the number of cotyledons in PIH mothers, with increased incidence of number of infarcts and calcified areas in PIH placenta. This could be correlated clinically for monitoring health status of mother and intrauterine growth of the foetus.

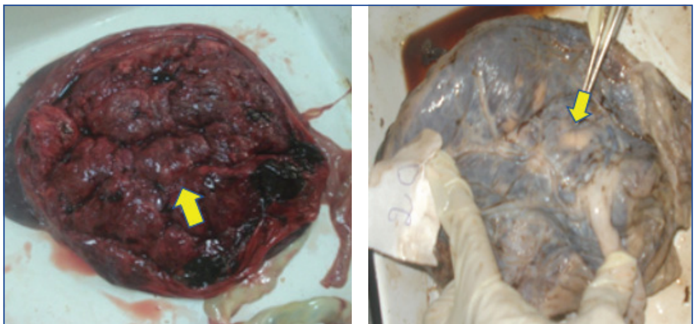
In the present study, a significant reduction of mean placental weight was observed in PIH cases. The studies of Goswami PR and Shah SN, Zhang YL et al., Kartha S and Poothiode U also reported a reduced placental weight of 179 grams, 200 grams and 102 grams, respectively in PIH cases compared to mean placental weight in normal cases [11,14,15].

In the present study, a significant reduction of mean newborn weight was observed in PIH cases. The studies of Udainia A and Jain ML, Kambale T et al., Kartha S and Poothiode U, Ilie C et al., also showed a reduction of mean newborn weight (360 grams, 660 grams, 800 grams and 260 grams, respectively) in PIH cases compared to mean newborn weight in normal cases [1,10,15,16].

The foetoplacental weight ratio was significantly increased which correlates with the other research works by Udainia A and Jain ML, Kambale T et al., Kartha S and Poothiode U [1,10,15].

Number of cotyledons in normal placenta varies from 19-23 [9]. In the present study, the number of cotyledons in PIH placenta ranged from 12-16. The mean number of cotyledons in PIH placenta was 14 which correlates with the study by Goswami PR and Shah SN [11]. Though, a study by Kartha S and Poothiode U, showed no significant difference between PIH and normal cases with respect to the number of cotyledons [15].

An incidence of mean infarct areas of 9.6 (19%) was found on the maternal surface of placenta in PIH cases [Table/Fig-4,5]. This correlates with the study by Kambale T et al., and Kartha S and Poothiode U, which showed an incidence of 26% and 28% of infarcts in placenta with PIH, respectively [10,15]. Underlying pathophysiology seems to be defective remodelling of the spiral arteries, known as decidual arteriopathy, that could contribute to a hypoxic environment and thereby, placental insufficiency which is often found in pregnancies with PIH [17].



[Table/Fig-4]: Maternal surface with cotyledons and fresh infarcts.
[Table/Fig-5]: Foetal surface with old infarcts. (Images from left to right)

In this study, approximately 13 (26%) calcified areas on an average were observed in the placenta of PIH mothers [Table/Fig-6]. Similar analysis was reported by Goswami PR and Shah SN and Kartha S and Poothiode U, with incidence of 36% and 21% of calcified areas in placentae of PIH mothers respectively [11,15].

In this study, marginal type of placenta was observed in 68% of PIH cases [Table/Fig-7]. In the studies of Goswami PR and Shah SN and

[Table/Fig-6]: Calcified areas on placenta.
[Table/Fig-7]: Marginal type of placenta. (Images from left to right)

Kartha S and Poothiode U, observed marginal type of placenta in 16% and 3% of mothers with PIH, respectively [11,15].

In the present study, 62% of PIH mothers were primigravida and 38% of PIH mothers were multiparous indicating a greater incidence of PIH in primigravidae which correlated with researches by Kambale T et al., and Jain K et al., [10,18].

The most common symptoms observed were pedal oedema and headache and the most common signs presented were proteinuria and IUGR. The clinical features as documented under case records were headache, pedal oedema, convulsions, visual disturbances, proteinuria in mothers and also, IUGR in foetus which correlates with studies of Udainia A and Jain ML, Goswami PR and Shah SN and Zhang P et al., [1,11,19].

Limitation(s)

The placentas of only normal vaginal delivery were studied (no cesarean sections were included). Histopathological correlation could not be done in the department of anatomy due to financial constraints.

CONCLUSION(S)

The placental weight and number of cotyledons of placenta will significantly reduce in PIH mothers and significant reduction of foetal weight in newborn of PIH mothers. Primigravida were mostly affected. Increased incidence of marginal placenta, infarct and calcified areas can be seen. This indicates gross study of placenta in early gestation of pregnant mothers radiologically, mothers radiologically which can serve as an early evidence of PIH and IUGR; thereby, reducing foetal and maternal morbidity and mortality.

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