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Study of Morphological Changes of Placenta in Pregnancy Induced Hypertension

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Abstract

Pregnancy induced hypertension is the hypertension that develops as a direct result of gravid state, characterized by development of hypertension to a extent of 140/90mmHg or more with/ without proteinurea. It includes Gestational hypertension, Pre-eclampsia, and Eclampsia. In these hypertensive conditions, maternal uteroplacental blood flow is decreased, indirectly causing constriction of foetal stem arteries leading to foetal hypoxia or foetal growth retardation. Thus the documentation of macroscopic findings of placenta becomes essential. Present study was carried out to study the macroscopic features of placentae in normal pregnancies and in pregnancy induced hypertension which included total 100 patients (50 normotensive and 50 from PIH). All the placentae were examined macroscopically for placental weight, number of cotyledons, infarction, calcification, insertion of umbilical cord along with fetal weight. Morphological examination of placenta is important in improving the management of subsequent pregnancies, understanding the specific etiologies of various conditions and their specific treatment along with preventive measures.

Keywords: Calcification, Cotyledons, Foetal weight, Infarction, Placental weight, Pregnancy induced hypertension.

Introduction

The placenta is an ephemeral organ interposed between the mother and fetus and is vital for the survival of the fetus as it is responsible for the respiratory, nutritional, excretory, endocrinal and immunological functions [1,2]. Pregnancy complications like gestational diabetes or hypertension are reflected in the placenta in a significant way, both macroscopically and microscopically which form one of the deadly triad along with hemorrhage and infection. The examination of placenta gives a clear idea of pathology and reflects as the mirror of perinatal and maternal mortality [3,4].

Pregnancy induced hypertension is the hypertension that develops as a direct result of gravid state with blood pressure of 140/90mmHg or more with/ without

proteinurea. It includes Gestational hypertension, Pre-eclampsia, and Eclampsia which causes decrease in maternal uteroplacental blood flow, indirectly causing foetal hypoxia or foetal growth retardation [5,6,7]. Pathological examination of placenta reveals the specific etiology of stillbirth, preterm delivery, intrauterine growth restriction (IUGR), and neurodevelopmental impairment and subsequent foetal outcome, pathological aspects of twin pregnancy, conditions with the risk of recurrence can be recognized, resulting in adequate treatment, may have medicolegal aspects [8,9]. The hypertensive disorders complicating pregnancy are quite common. In India, the incidence of preeclampsia is 1.5% resulting in foetal distress, intrauterine foetal deaths and placental abnormalities. In earlier studies, gross abnormalities of placenta have received less attention and undeserved status. Recently, morphological changes in the chorionic villi have proved a relationship between placental pathology and fetal well-being.

Hence there is a need to observe the effects of pre-eclampsia and eclampsia on the morphology of placenta in such cases and compare them with normal morphology of placenta so as to understand better the pathophysiological processes of pre- eclampsia and eclampsia.

Aim and objectives

To study the macroscopic features of placentae in normal pregnancies and in pregnancy induced hypertension cases.

Material and methods

The present study of placenta in pregnancy induced hypertension was done over the period of 2 years which included total 100 placentae of which 50 were from normotensive cases and 50 were from pregnancy induced hypertension women of age group 20- 38 years and of any parity. Placentae in disease i.e chronic hypertension, pre-eclampsia or eclampsia superimposed on chronic hypertension and gestational diabetes were excluded from the study. After due consent, the relevant clinical information of mother and babies were noted. The general shape and appearance of placenta was assessed by naked eye. Maternal surface for Completeness or dark areas, Foetal surface for Transparency of Amnion & Umbilical cord for Insertion, Membranes: Transparency, Colour (Fig. 1.)

A complete gross examination including weight, colour, infracted and calcified areas was carried out along with fetal weight (Fig. 2). Number of cotyledons are counted as shown in Fig 3.

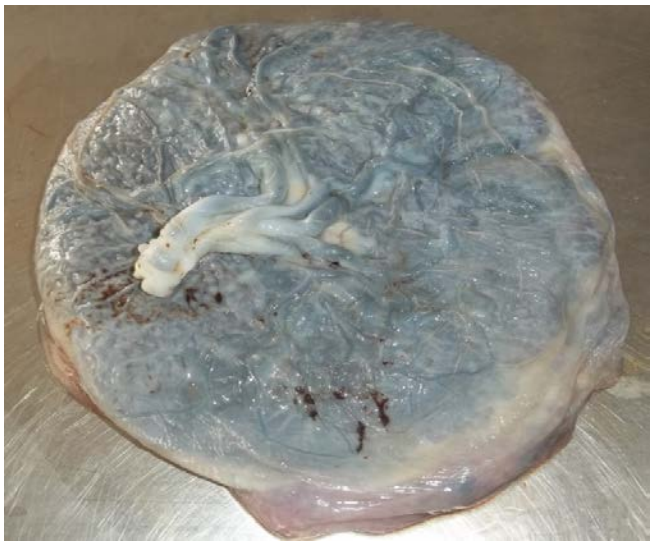

Figure 1

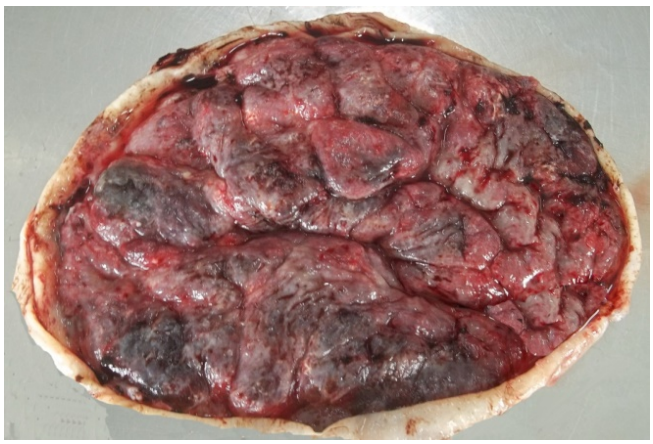

Figure 2

Figure 3

Results

The data was analyzed by SPSS 16.0 for windows evaluation version software. The independent t-test and Mann-Whitney U- test were used for statistical difference determination between the 2 groups.

Note: 1) p-value of 1.48E-07 is 1.48 X 10-07 or 0.000000148

2) IQR= Interquartile Range (i.e. 75th Percentile-25th Percentile).

Macroscopic Findings

Table No. 1: showing comparison of placental weights, fetal weights, F/P ratio, no of cotyledons, calcified areas and infracted areas in Group I and Group II:

(Unpaired t-test applied. ^ Data failed 'Normality' test. Hence Mann-Whitney test was applied. t-value replaced by Z-value.)

Insertion of umbilical cord – In group I 38 % had central insertion of cord and while 62% had eccentric insertion of cord. In group II 24% had central while 69% had eccentric insertion. Pearson chi square test was applied and found to be non significant.

In the present study, the average weight of the placenta in normotensive cases was 495 gm and 422 gm in PIH cases. The lowest recorded weight was 240 gm for PIH group. Range of placental wt in Group I was 370- 610 gm while that of Group II was 240-780 gm. The median value for Group I was 505gm while that Group II was 412gm.

Range of foetal wt for Group I was 2000-3480gm having mean value of 2654.30gm and for Group II it was 1000-3325gm having mean value of 2229.80gm. Foetoplacental weight ratio range in group I was 3.60 – 7.40 and in Group II it was 2.00- 7.60. Range of No of cotyledons in both group I & II was same 14 to 19. Median value for Group I & II was 17 and 16 respectively. Range of no of calcified areas in Group I was 2-8 having median value of 4 while that in Group II it was 4-15 having median value of 10. Range of no infarcted areas in Group I was 2- 8 and in Group II was 7-16.

Discussion

Pre-eclampsia has definite adverse influence on the morphology of the placenta. The placenta gives important information regarding fetal outcome. Thus placenta has a vital role to play in the survival of foetus. The present study was carried out to analyze and assess the gross features of the placenta like placental weight, number of cotyledons, insertion of umbilical cord, number of infarcted areas, number of calcified areas, and foetal weight, foetoplacental weight ratio.

In the present study mean placental weight in Group I was 495.5±68.00gm, while in Group II it was 422±111.5 gm. It reveals that placental wt in PIH group is significantly less than in normotensive group. This finding corroborates with studies of Sankar K.D. et al, Rehman MZ et al, H. H. Modi et al, Kulandaivelu R. A. et al, Kartha S. et al as follows [1,10,11,12,13].

Sr. No	Author	Mean placental weight (gms)	
		Group I	Group II
1.	Sankar K.D. et al	470	401
2	[1]Rehman MZ et al	502	293
3	H. H. Modi et al	>400	<400
4	Kulandaivelu R. A. et al	550	476
5	Kartha S. et al	570	370
6	Present study	495.50Median(505gm)	422.20Median(412gm)

In the present study mean foetal wt in Group I was 2654.30±364gm while in Group II it was 2229.80±518gm. Difference between two groups was statistically significant. This reveals that mean foetal birth wt in Group II (PIH Group) is significantly lower than Group I. This corroborates with study of other workers Rehman MZ et al, Kulandaivelu R. A. et al, and Kartha S. et al as follows [10,12,13].

Sr. No	Author	Mean foetal birth weight (gm)	
		Group I	Group II
1.	Rehman MZ et al	3296	2373
2	Kulandaivelu R.A. et al	2950	2480
3	Kartha S. et al	2900	2100
4	Present study	2654	2229

In PIH there is increased pressure on blood vessels, consequently foetoplacental blood flow is severely impaired and transplacental gas and nutrition exchange is poor, placing the foetus at risk of hypoxia, acidosis, and low birth weight.

There is also evidence that fetal growth depends on the placental weight. When the rate of uteroplacental flow is chronically reduced placental weight also decreases. The findings of the present study show that birth weight in PIH group is low.

In the present study, mean F/P ratio in Group I was 5.39±0.90 and in Group II it was 5.35±1.01. The difference was not statistically significant. Present study corroborates with studies of Kulandaivelu R. A. et al who found mean F/P ratio 5.39 in group I and 5.20 in group II. But in other studies, difference between two groups was significant like Kartha S.et al and Raghunath G. et al ,the mean F/P ratio was 5.7 in group I and 6.5 in group II while 5.35 in group I and 6.03 in group II respectively. Present study found that the foeto-placental unit is adversely affected in PIH. Due to placental insufficiency foetal growth is affected. According to Little and Thomson et al, for evaluation of foetus, weight of placenta is not enough but the foeto-placental weight ratio is important [12,13,14].

Mean No of cotyledons for Group I was 16.86±1.11 and for Group II it was 16.34±1.51. The difference was not statistically significant. Findings of present study corroborates with the studies of Rehman MZ et al, Akther F. et al, who found 30 in group I and 22 in group II and 17 no of cotyledons in group I and 16 in group II respectively. The number of cotyledons in PIH group were lower than control Group I and this contributed to the insufficient blood supply due to PIH.

Placental calcification has shown variable values among both groups. Placental calcification is regarded as evidence of placental senescence or degeneration [10,15,16,17].

In the present study mean no of calcified areas in Group I was 3.80±1.44 and in Group II it was 10.22±2.71 with median value in Group I as 4 and in Group II as 10. This suggests that there is significant increase in calcification areas in placentae of pregnancy induced hypertension Findings of present study corroborates with studies of other workers like Kartha S. et al, Motwani R.et al who found 10% in group I, 21% in group II while 26.66 % , 70 % of no of calcified areas in group I and group II and respectively [13,18].

Placental infarction is a zone of ischaemic necrosis of a group of villi due to complete interference with their blood supply in the deciduas or in the local state by thrombosis of a spiral arteriole or a retroplacental haemorrhage. In pregnancies complicated by pre eclamptic toxaemia, the incidence of placental infarction was very considerably raised, than in uncomplicated pregnancies thus the incidence of placental infarction was related to the severity of PIH in these cases and not to any other maternal factor [19]. Mean No infarcted areas in Group I was 4.26±1.54 and in Group II was 10.98±2.54 in present study. There is significant difference. Kulandaivelu R. A et al observed placental infarction in 70 % cases of pregnancy induced hypertension cases. Kartha S. et al found placental infarction was more common in placentae of pregnancy induced hypertension cases (26%) than in placentae of normotensive cases (10%). Motwani R. et al considered placental infarction of more than 5% surface area as pathological. In this study author found infarction was more common in placentae of pregnancy induced hypertension (43.33 %) than in placentae of normotensive cases (3%) [12,13,18].

Central insertion of umbilical cord was present in 19 (38%) & 12 (24%) in Group I and Group II placenta respectively while Eccentric insertion of umbilical cord was present in 31(62%) & 38 (76%) in Group I and Group II placenta respectively. Overall eccentric insertion of umbilical cord was more frequent about 69% as compared to central insertion of umbilical cord which was about 31% only. This finding corroborates with study of Rath G & Garg. K. Narasimha A. & Vasudeva et al found central insertion as obvious finding than eccentric insertion of umbilical cord [20,21].

Conclusion

Pregnancy induced hypertension is one of the leading cause of maternal mortality and emerging cause of foetal wastage. Placental examination is important in evaluating perinatal morbidity and mortality. PIH affects placentae by decrease in weight, foetal weight and foetoplacental weight ratio, increasing number of calcification areas and infarcted areas as compared to normal placentae. Thus one should have a responsibility to recognize the placental abnormalities that contribute to an adverse perinatal outcome.

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Legends Table

Table 1: showing comparison of placental weights, fetal weights, F/P ratio, no of cotyledons, calcified areas and infracted areas in Group I and Group II: (Unpaired t-test applied. ^ Data failed 'Normality' test. Hence Mann-Whitney test was applied. t-value replaced by Z-value.)

Variable	Groups	Mean	SD	Median	IQR	t/z - value	p-value
Placenta weight(gms) ^	Group I	495.50	68.00	505.00	112.50	Z-value- 4 .307	1.66E-05
	Group II	422.20	111.52	412.50	127.50	Difference is significant	
Foetal weight (gms)	Group I	2654.30	364.98	2630.00	471.25	t value- 4.732	7.48E-06
	Group II	2229.80	518.80	2307.50	556.25	Difference is significant	
F/P ratio	Group I	5.39	0.90	5.30	1.13	t value- 0.199	0.843
	Group II	5.35	1.01	5.45	1.25	Difference is not significant	
No. of cotyledons	Group I	16.86	1.11	17.00	2.00	Z value-1.629	0.103
	Group II	16.34	1.51	16.00	3.00	Difference is not significant	
No. of calcified areas	Group I	3.80	1.44	4.00	1.25	Z value-8.351	6.79E-17
	Group II	10.22	2.71	10.00	5.00	Difference is significant	
No. of infarcted areas	Group I	4.26	1.54	4.00	3.00	Z value- 8.605	7.62E-18
	Group II	10.98	2.54	10.00	4.00	Difference is significant	