Original Research Article

Study of placental morphological changes in chronic hypertension and its relation with fetal outcome

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Abstract

Background: Although many women with chronic hypertension do well in pregnancy, they are at increased risk for several pregnancy complications, including fetal growth restriction, placental abruption and preterm birth. **Aim:** To study the placental morphological changes in chronic hypertension and its relation with fetal outcome. **Material and Methods:** The total number of placentae studied was 400 of which 200 placentae from normal term pregnancy were taken as control group and200 placentae were from patients having medical disorder. Atotal of seven placentae were from patients having chronic hypertension. Gross examination was carried out on the fresh placentae for any abnormality and then sent for histopathological examination. Fetal outcome was compared with the placental pathology. **Results:** In chronic HTN cases, macroscopically areas of infarction and calcification were observed in 57.14% and 71.42% cases as compared to 11% and 20% of control group respectively. Microscopically increased syncytial knots and cytotrophoblastic cell proliferation were seen in 85.7% and 71.42% of cases respectively. Fibrinoid degeneration and increased basement membrane thickening were seen in 71.42% of cases. Hyper or hypovascular villi were not seen in these cases in our study. Hypermature villi were seen in 71.42% of cases which was significant. Areas of infarction and calcification were seen in 57.14% of cases. The adverse foetal outcome was statistically not significant in chronic HTN cases. **Conclusion:** The majority of the placentae from chronic hypertension in pregnancy appear to show pathological changes from which aetiology can be fairly predicted but detail quantitative estimation of those placental changes is essential.

Key Words: Pregnancy, chronic hypertension, placenta, fetal outcome.

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Received Date: 19/05/2018 Revised Date: 14/06/2018 Accepted Date: 10/07/2018
DOI: https://doi.org/10.26611/1012714

Access this article online	
Quick Response Code:	Website: www.medpulse.in
	Accessed Date: 14 July 2018

INTRODUCTION

Placenta is an organ, so vital in its function to the development of baby, is commonly discarded after birth but the careful study of this 'after birth' may yield information of prognostic significance for the new born.[1]It is fanciful to assume that every adverse perinatal outcome is associated with an abnormal placenta and equally fanciful to expect that every abnormal placenta will result in a poor perinatal outcome. It is ultimately the responsibility of the pathologist to recognize when a placenta is abnormal and to identify those which are associated with perinatal morbidity and mortality. Although many women with chronic hypertension do well in pregnancy, they are at increased risk for several pregnancy complications, including superimposed preeclampsia, fetal growth restriction, placental abruption, preterm birth, and cesarean section. Placental surface area is significantly less in pregnancy induced hypertension, but none of the researchers have mentioned the exact surface area.[2,3] Reduced maternal uteroplacental blood flow leading indirectly to constriction of foetal stem arteries has been attributed to these changes.[4] Maternal vasospasm leads to foetal hypoxia, fetal distress and death, which is fairly common towards the later part

How to cite this article: Shailendra V Mangnale, Ashwini S Mangnale. Study of placental morphological changes in chronic hypertension and its relation with fetal outcome. *MedPulse – International Journal of Gynaecology.* July 2018; 7(1): 13-16.
http://medpulse.in/Gynacology/index.php

of pregnancy.[5] The present study was carried out to study the placental morphological changes in chronic hypertension and its relation with fetal outcome.

MATERIAL AND METHODS

This prospective study was carried out in the Department of Obstetrics and Gynaecology in collaboration with the Department of Pathology in Swami Ramanand Teerth Rural Medical College, Ambajogaiover a period of two years. The total number of placentae studied was 400 of which 200 placentae from normal term pregnancy were taken as control group and 200 placentae were from patients having medical disorder during or associated with pregnancy that were randomly selected. A total of seven placentae were from patients having chronic hypertension.

Inclusion Criteria

- **Control Group:** These were the cases of uncomplicated pregnancies not having any medical disorder in pregnancy
- **Chronic Hypertension:** Chronic hypertension in pregnancy is defined by the American College of Obstetrics and Gynecology (ACOG) as blood pressure ≥140 mm Hg systolic and/or 90 mm Hg diastolic before pregnancy or, in recognition that many women seek medical care only once pregnant, before 20 weeks of gestation, use of antihypertensive medications before pregnancy, or persistence of hypertension for >12 weeks after delivery.[1]
- Patients with clinical diagnosis of chronic hypertension included in the study were categorized as -

Exclusion Criteria

- Cases having multiple pregnancies
- Cases having medical disorder in pregnancy other than chronic HTN.

Placenta examination

The placentae were examined after delivery. The clots adherents if any were removed and big retro-placental clots more than 50 gm weighed. Then placentae were washed thoroughly with water, membranes trimmed and cord cut 4 cm from insertion. Gross examination was carried out on the fresh placentae for any abnormality. Then the placentae were fixed in 10% formalin and sent for histopathological examination. Gross examination of the placenta included dimensions and weight. Umbilical cord was examined for length, knot and type of insertion. Membranes were examined for type of insertion and colour. Fetal surface was examined for any abnormality. Maternal surface was examined for completeness, infarction, calcification and succenturiate cotyledons. After performing gross examination of placenta by pathologist, sections from placentae were taken. Blocks from placentae included central portion and the portion where pathology was suspected. Blocks were processed routinely. Paraffin embedded blocks were trimmed and cut into 3 to 5 micron thickness. The sections were stained by routine haematoxylin and eosin staining. Microscopic examination of the placentae included villous pattern abnormalities, thickening of basement membrane, excess of syncytial knots, fibrinoid degeneration, villous vascularity, stromal fibrosis, congestion of capillaries, infarction and calcification.

Table 1: Category of chronic hypertension

Category	Systolic BP (mm Hg)	Diastolic BP(mm Hg)
Mild	140-159	90-99
Moderate	160-179	100-109
Severe	≥ 180	≥ 110
Isolated systolic(ISH)	≥ 140	< 90

Foetal Outcome: Foetal outcome was compared with the placental pathology. Correlation was made regarding mode of delivery with placental pathology. Statistical significance was analyzed by using tests standard error of difference between two proportions and standard error of difference between two means.

Statistical Analysis: Statistical significance was analyzed by using tests standard error of difference between two proportions and standard error of difference between two means in appropriate tables. In all observation tables given below * indicates significant value($p< 0.05$).

RESULTS

Out of 200 cases, 7 (3.5%) were suffering from chronic hypertension. Most of the cases i.e., 4 (57.14%) were in the 26 to 30 years age group followed by 2 (28.6%) in 31-35 years age group and one (14.3%) in 21-25 years. Out of the 7 cases with chronic HTN,3 (42.85%)cases were booked, whereas, remaining 4 (57.15%)cases were unbooked indicating that most of the patients from this rural population were not attending antenatal clinic and they ignore health check up during pregnancy. Chronic HTN was seen more commonly in multigravidas (57.14%).Among study group, only one case had preterm delivery, 36% of patients in control group had preterm deliveries. In control group only 29% of patients had max placental diameter ≤15 cms. In study group, max placental diameter ≤15 cms was observed in 5 (71.42%) cases. In cases of Chronic HTN, macroscopically areas of infarction and calcification were observed in 57.14% and 71.42% cases as compared to 11% and 20% of control group respectively. Microscopically increased syncytial knots and cytotrophoblastic cell proliferation were seen in 85.7% and 71.42% of cases respectively. Fibrinoid degeneration and increased basement membrane thickening were seen in 71.42% of cases. Hyper or

hypovascular villi were not seen in these cases in our study. Hypermature villi were seen in 71.42% of cases which was significant. Areas of infarction and calcification were seen in 57.14 % of cases. Endarteritis obliterans was seen in 14.28 % of cases of Chronic HTN (Table 2).

Table 2: Placental changes in chronic HTN

Macroscopic changes	Control group(n=200)		Chronic HTN(n=7)	
	No.	%	No.	%
Infarction	22	11	4	57.14*
Calcification	40	20	5	71.42*
Retroplacental haematoma	0	0	1	14.28
Microscopic changes				
Syncytial knots	22	11	6	85.7*
Cytotrophoblastic Proliferation	6	3	5	71.42*
Fibrinoid Degeneration	20	10	5	71.42*
Basement membrane Thickening	12	6	5	71.42*
Stromal Fibrosis	16	8	3	42.85
Villous edema	0	0	0	0
Hypervascular villi	20	10	0	0*
Hypovascular Villi	14	7	0	0*
Hypermature villi	12	6	5	71.42*
Villous immaturity	28	14	0	0
Thrombosis of villi	0	0	0	0
Infarction	34	17	4	57.14*
Calcification	36	18	4	57.14*
EndarteritisObliterans	0	0	1	14.28

In control group only 28 % of patients had placentae weighing ≤450 gm. Among cases placentae weighing ≤ 450 gm were observed in 5 (71.42%) cases. In present study, mean birth weight in control group was 2619 gm. Whereas, in chronic HTN cases, it was 2557.14gms withmean placental weight of 370 gms and feto-placental ratio of 6.91 and placental coefficient of 0.14.

Table 3: Feto-placental characteristics

Characteristics	Control group	Study group
Placental weight (gm)		
≤350	28	02
351-450	28	05
451-550	138	00
551-650	06	00
Birth weight (gms)		
501-1000	00	00
1001-1500	06	00
1501-2000	14	00
2001-2500	44	02
2501-3000	98	05
3001-3500	38	00
3501-4000	00	00
Mean	2619	2557.14
Mean placental weight (gms)	446.3	370*
Feto-placental ratio	5.86	6.91
Placental coefficient	0.17	0.14

In present study, the adverse foetal outcome was statistically not significant in chronic HTN cases. Low Apgar score at 1 min was seen in 14.28% of cases, perinatal deaths were observed in 14.28% of cases and uneventful deliveries were seen in 85.71% of cases.

Table 4: Foetal outcome

Fetal outcome	Control group		Study group	
	No.	%	No.	%
Low Apgar at 1 min	24	12	1	14.28
Perinatal death	28	14	1	14.28
Uneventful delivery	158	79	6	85.71

DISCUSSION

The potential benefits of placental examination include clarification of pathological features, improved management of subsequent pregnancies by diagnosing pathological conditions that may have risks of recurrence or may even be preventable or treatable. According to Fox H, placental changes that occur in patients of essential hypertension and PIH are almost same except an obliterative endarteritis of the fetal stem arteries is encountered less frequently in the hypertensive placenta and there is less tendency to form excess of syncytial knots, stromal fibrosis, retroplacentalhaematomas and villous fibrinoid necrosis as compared to PIH cases.[6] Das B *et al* found that morphologically placentae of PIH are more severely affected than the placentae of essential hypertension.[7] Corrêa R *et al* found in their study that the number of knots presented a positive correlation with the length of time and severity of the hypertension during gestation and the fibrin deposit was greater in all hypertensive syndromes of pregnancy.[8] In present study, on macroscopic examination infarcted and calcified areas were seen in 57.14% and 71.42% of cases and retroplacental haematomas were seen in 14.28% of cases having chronic hypertension while on microscopic examination increased syncytial knots were seen in 85.7% cases and increased cytotrophoblastic cells, fibrinoid degeneration and increased basement membrane thickening in 71.42% of cases, infarcted and calcified areas seen in 57.14% of cases which were seen more frequently than in cases of preeclampsia and eclampsia. All these findings does not correlate with those of Fox H[6]and Das B *et al*[7]but this increased frequency of pathological changes may be due to a positive correlation with the length of time and severity of the hypertension during gestation as stated by Corrêa R *et al*, as full term deliveries occurred in 85.71% of cases having chronic hypertension in present study.[8] In present study, the adverse foetal outcome was statistically not significant in chronic HTN cases. Low Apgar score at 1 min was seen in 14.28% of cases, perinatal deaths were observed in 14.28% of cases and uneventful deliveries were seen in

85.71% of cases. Masodkar *et al* found that fetal outcome was worse when placenta weights <300 gms.[9] Microscopically placentae from cases with low Apgar score and stillbirths showed increased syncytial knots, thickening of the basement membrane in stillbirths and perinatal deaths. Kovalovszki L *et al*, observedthatthe percentage of edematous villi were significantly higher in the group of newborns requiring resuscitation.[10] The severity of the edema had a positive correlation with the need for resuscitation at birth and with the arterial blood pH values in the umbilical cord. Mardi K andSharma J found that the incidence of infarction, intervillous fibrin deposition were much higher in IUGR placentae on gross examination.[11] Highly significant increase in the incidence of infarction, intervillous fibrin deposition, stromal fibrosis and syncytial knotting were found in IUGR placentae compared to full term normal placentae on microscopic examination. The incidence of basement membrane thickening and cytotrophoblastic hyperplasia were also higher in IUGR placentae. Meejus G found that Multiple placental infarctions (49.2%) dominated in the study group (birth weight < 10^{th} percentile) but in 21 cases (33.3%) no histological abnormalities were found.[12]

CONCLUSION

The majority of the placentae from chronic hypertension in pregnancy appear to show pathological changes from which aetiology can be fairly predicted but detail quantitative estimation of those placental changes is essential. The importance of immediate gross examination of all placentae and thorough microscopic examination in those cases where placenta or pregnancy outcome is abnormal should be stressed.

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Source of Support: None Declared
Conflict of Interest: None Declared