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Changes in Placenta in Preeclampsia and Eclampsia -A Study of 144 Cases in Rural Tripura

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ABSTRACT

Background: *Preeclampsia and eclampsia are the two leading cause of maternal mortality and are two important factors in fetal wastage. This study was designed to investigate the morphological and histopathological changes in placenta from pregnancies complicated with preeclampsia and eclampsia.*

Objectives: *To study the morbid changes in placenta in cases of preeclampsia and eclampsia and to correlate the findings with normotensive mothers, because these changes serve as a guide to the duration and severity of disease.*

Materials and Methods: *The study was done from November 2013 to April 2016 (21/2 yrs) in the rural hospitals of Tripura, mainly Sub divisional and District hospitals in collaboration with Dept of Pathology TMC and Dr BRAM Teaching Hospital on 144 placentas, out of which 70 were collected from normotensive mothers and the remaining 74 from preeclampsia and eclampsia cases. All the placentas were studied morphologically and histologically. The birth weight of neonates was recorded.*

Results: *It was observed that weight and dimensions of placenta was less in study group when compared with control group. The mean neonatal birth weight was more in normal pregnancy and feto-placental weight ratio was significantly high in hypertensive group. Gross abnormalities noted were the placental infarcts, retroplacental hematoma, and calcification. Histopathological study showed significant number of syncitial knots , cytotrophoblastic proliferation , thickening of the villous basement membranes , fibrinoid necrosis , hyalinization , calcification , endarteritis obliterans and decreased villous vascularity in preeclampsia and eclampsia group.*

Conclusions: *These changes in placenta may cause placental insufficiency as a result of compromised utero-placental blood flow. Therefore has an adverse affect on the fetus. Preeclampsia and eclampsia have definite influence on morphology, histology of placenta, and thus affects the growth of the fetus. The gross abnormalities and villous lesions in the preeclampsia ($P < 0.05$) were significant and eclampsia syndrome ($P > 0.05$) were not significant.*

KEYWORDS : Calcification, eclampsia, infarction, preeclampsia, villous abnormalities

INTRODUCTION

Placenta is a unique and wonderful organ that arises de novo. Being an organ of vital importance for the continuation of a pregnancy and fetal nutrition, it has evoked great interest among the pathologists and the obstetricians as well, and much work has been done to understand the "unique biological status" of this complex organ. [1]

The root cause of preeclampsia is the placenta. Preeclampsia begins to abate with the delivery of the placenta and can occur in the absence of a fetus but with the presence of trophoblast tissue with hydatidiform moles. In view of this, study of the placenta should provide insight into the pathophysiology of preeclampsia. In this presentation we examine placental pathological and pathophysiological changes with preeclampsia and fetal growth restriction (FGR).[2]

Pregnancy complications like preeclampsia and eclampsia are reflected in placenta in a significant way both macroscopically and microscopically. Several studies have shown that utero-placental blood flow is decreased in preeclampsia and eclampsia due to maternal vasospasm.[3] This leads to constriction of fetal stem arteries and has been associated with the changes seen in the placenta of preeclamptic women.[4] Maternal vasospasm leads to fetal hypoxia and accordingly it may lead to fetal distress and fetal death.[5] A significant increase in syncytial knot formation in placental villi indicates the disturbance in the hormonal factors, which may probably lead to altered blood flow. 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. The preeclamptic women will have a lower mean gestation, so the proportion of fetal capillaries will be lower. The capillaries become larger as the gestation proceeds. This relative increase in fetal capillary volume with decrease in proportion of connective tissue will lead to smaller parenchymal volume leading to decrease in placental weight .[6] Present study has been undertaken to record the data on the morphology, morphometry, and histology of placenta from mothers with preeclampsia and eclampsia .This study was done to find out the morbid changes of the placenta of hypertensive mothers in comparison to normotensive mothers. As placenta is the mirror of maternal and fetal status, it reflects the changes due to preeclampsia and eclampsia .

METERIALS AND METHODES

Study was done from November 2013 to April 2016 ($2^1/_2$ yrs) in the rural hospitals of Tripura, mainly Sub divisional and District hospitals in collaboration with Dept of Pathology TMC and Dr BRAM Teaching Hospital. The study was approved by Institutional Ethics Committee.

The study was done in 144 placentas, which were collected from Obstetrics and Gynecology department of Sub divisional and District hospitals of Tripura. Out of the 144 placentas collected, 70 placentas were from uncomplicated full term deliveries and served as control group. Another 74 placentas were collected from preeclamptic and eclamptic cases and served as study group.

The age of the women varied from 18 to 38 years. The cases were divided into three groups, namely, normal, preeclampsia, and eclampsia group.

The placenta with attached membranes and umbilical cord was collected soon after delivery, washed in running tap water, labeled, and then fixed with 10% formalin and sent to the Dept of Pathology TMC and DR BRAM Teaching Hospital, Agartala. Sample was kept for fixation for 4-6 weeks. Gross and microscopic examination of the placenta was carried out. The size, shape, weight, thickness at centre, number of cotyledons, and site of insertion of umbilical cord were noted down. The birth weights of newborn babies were documented and

feto-placental weight ratio was calculated. Infarction and perivillous fibrin deposits were quantified based on the surface area of involvement. These were further confirmed by histology. Two sections each from central and peripheral areas were taken. Additional sections were taken from grossly abnormal lesions. Sections were stained with the hematoxylin and eosin (H and E) stain. One hundred villi were counted from each of the four sections obtained, under light microscope and histological changes expressed as percentage.

OBSERVATIONS & RESULTS

The incidence of various gross and histological features was compared with that of normal pregnancies by using the unpaired-type Student '*t*' test.

From the study, it was observed that the weight of the placenta was less in preeclampsia and eclampsia when compared with normal placenta. The majority of placenta were oval (73%) followed by round placenta (27%). The insertion of the umbilical cord central in 18% of cases and eccentric in 82% of cases. The number of cotyledons varied from 18 to 23 in all groups[Table 1].

Table 1: Comparison of gross morphological features of placenta in different groups in present study

Parameters	Normal pregnancy	Preeclampsia	Eclampsia
Average Weight	505.75 ±43.30	399.60 ± 57.46	393.56 ± 66.66
Average Length	19.65 ± 2.58	18.50 ± 1.68	16.34 ± 1.87
Average Width	16.5 ± 1.8	14 ± 2.2	13 ± 1.5
Average Thickness at centre	2.75 ± .25	2.5 ± .27	2.6 ± .23
Average Cotyledons no.	21.45 ± 1.54	21.34 ± .57	20 ± 1.2
Cord haematoma	21(%)	4	7
Average Neonatal wt	2.8 ± .68	2.4 ± .41	2.5 ± .12
Feto- placental wt ratio	5.53±0.45	6.01±0.66	6.36±1.27
Total no of cases	70	47	27

In our study it is seen that the mean neonatal birth weight was more in normal pregnancy (2.8 kg) and less in preeclamptic and eclamptic cases (≤ 2.5 kg). The feto-placental weight ratio was significantly higher in the hypertensive group than in the control group [Table1]

Histopathological study of placenta in our study showed significant number of syncytial knots, cytotrophoblastic proliferation, villous basement membrane thickening, fibrinoid necrosis, areas of calcification and hyalinization and areas of medial coat proliferation of medium sized blood vessels or endarteritis obliterans in the hypertensive group, whereas the control group showed normal histological features.

Table 2: Comparison of histopathological features of placenta in different groups in present study

Parameters	Normal pregnancy	Preeclampsia *	Eclampsia **
Syncitial knots <40% 40-90% >90%	 66% 34% -	 53% 23% 24%	 15% 16% 69%
Cytotrophoblastic proliferation <20% 20-40% >40%	 100% - -	 - 69% 31%	 - - 100%
Villous basement membrane thickening <3% >3%	 - -	 19% 81%	 2% 98%
Fibrinoid necrosis <3% >3% >15%	 63% 28% -	 16% 60% 24%	 - 72.5% 27.5%
Hyalinization	13%	60%	69%
Calcification	-	27%	23%
Endarteritis obliterans	-	69.6%	95.75%
Decreased villous vascularity	8%	82%	79%
Total no of cases	70	47	27

* P < 0.05 – Statistically significant. **P > 0.05 – Not significant.

The gross abnormalities and villous lesions in the preeclampsia ($P < 0.05$) were significant and eclampsia syndrome ($P > 0.05$) were not significant [Table 2]

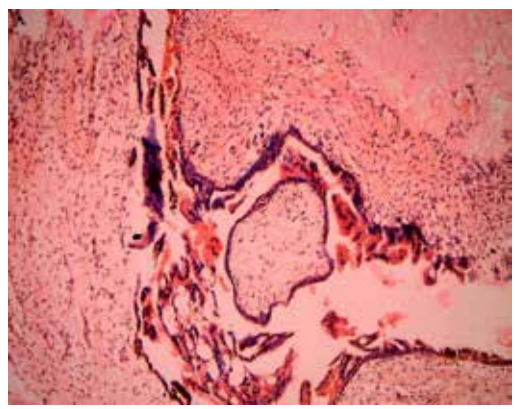

Fig: 1 Photomicrograph of villi showing intervillous hemorrhage (H and E, ×100)

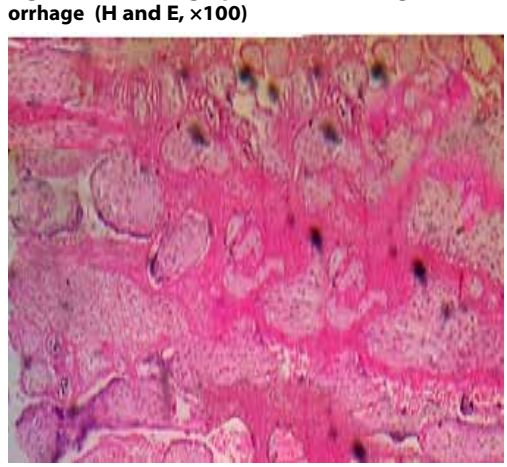

Fig :2 Photomicrograph of villi showing areas of Fibrinoid necrosis (H and E, ×100)

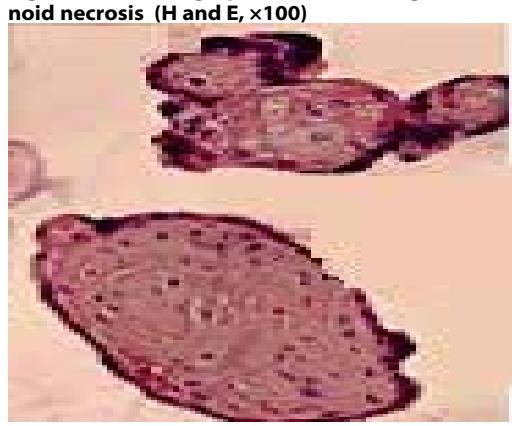

Fig:3 Photomicrograph of villi showing Syncitial knots (H and E, ×100)

DISCUSSION

The weights of the placenta in our study groups were below 400 g. The lowest weight was 230 g. In the control group, majority of the placenta weighed more than 500 g, the heaviest being 635 g. The length and width was also less in study groups.

The similar finding seen in studies done by various workers. Mallik *et al*. reported five cases of toxemia with placental weight less than 300 g.[7] Nobis and Das, in their study, observed that the placental weight in toxemic cases varies from 279 to 407 g.[8] Bhatia *et al*, in their study, have shown reduced placental weight in severe toxemia, the lowest recorded was 280 g.[9] The change in placental weight observed in the present study between the control group and 2 study groups were statistically significant.

The neonatal weight was significantly less in study groups when compared with control group ($P < 0.01$). The average weight was 2.8 kg in normal pregnancies, 2.4 kg in preeclampsia, and 2.5 kg in eclampsia cases. An earlier study by Palaskar had shown the reduced mean weight of the neonates in cases of PIH.[10]

The average feto-placental weight ratio in normal pregnancy was 5.53, in preeclamptic cases it was 6.01 and it was 6.36 in eclamptic cases. The values are similar with the study by Majumdar *et al*.[11]

Syncytial knots were seen in all the placentas from the control group while the increased incidence of syncytial knots in toxemic cases in the present study.

Cytotrophoblastic proliferation seen in all the cases of the control group within the normal range of villi containing cytotrophoblastic cells (<20%); while the study group had high villous counts of these cells, with the rise in counts with increasing severity of the toxemia.

None of the placentae from the control group showed basement membrane thickening, while the incidence of basement membrane thickening in the present study was high in two study groups.

Significant increase of fibrinoid necrosis was seen in preeclapsia and eclampsia cases which is similar with the study done by Sayeed M et.al [12] and . Kalra VB et.al[13]

Increase incidence of hyalinization and calcification and decreased villous vascularity seen in preeclampsia and eclampsia cases which was evidenced by Syeed[12] and Kalra et al.13

In the present study endarteritis obliterans was seen in patients with preeclampsia and eclampsia. Similar findings were observed by Kalra[13]and Davey.14

CONCLUSION

In the present study of placental pathology, the incidence of infarction, calcification, intervillous thrombus, and perivillous fibrin deposit was high in preeclapsia and eclampsia cases as compared to normal.

The gross and microscopic abnormalities in the preeclampsia group was significant ($P < 0.05$) and not significant in eclampsia group ($P < 0.05$) .Probably due to less number of cases in eclampsia group is the reason of this insignificance.

It seems incontrovertible that the placenta will provide insights into preeclampsia and eclampsia.

Attention to these considerations we believe will provide the optimum opportunity to fully utilize the power of placental analysis to unravel these (and the other) "great obstetric syndromes"[15]

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