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ORIGINAL ARTICLE

PLACENTAL PATHOLOGY IN PREGNANCY INDUCED HYPERTENSION

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ABSTRACT: BACKGROUND: Hypertensive disorders complicating pregnancy are common and form one of the deadly triad along with hemorrhage and infection, that results in a large number of maternal deaths and there of fetal deaths. Since all anabolites needed for foetal metabolism come from the mothers blood and foetal catabolites are passed back into the mothers circulation through the placenta, the examination of placenta gives a clear idea of what had happened with it, when it was in the mother, s womb and what is going to happen with the foetus in future. With this objective the present study was carried out. **MATERIALS AND METHODS:** Retrospective study was done for a period of 21 months from April1st 2008 to December 31st 2009..Fifty mothers with uncomplicated pregnancy (control group) and 100 mothers (test group) diagnosed as having pregnancy induced hypertension were selected from patients of our institution of the age range from 20-40 years, and parity –primi, para2 and 3.Placental morphometric parameters, gross and histopathological features were examined in both test and control groups. **STATISTICAL ANALYSIS USED:** Fishers exact test **RESULTS:** Placental morphometric parameters were significantly reduced in the control group. Acute atherosis, endothelial proliferation and fibrinoid necrosis were the significant histological findings noted in our study. **CONCLUSION:** Placental findings can be confirmatory of PIH, but its absence does not exclude the diseases. These findings will become more evident only when there is significant reduction in the uteroplacental bloodflow.

KEYWORDS: Placenta, Pregnancy Induced Hypertension, Infarction, Syncytial knots, Acute Atherosis, Chorangioma.

INTRODUCTION: Hypertensive disorders complicating pregnancy are common and form one of the deadly triad along with hemorrhage and infecton, that results in large number of maternal deaths and there of foetal deaths. Since all anabolites needed for foetal metabolism come from the mother, s blood and foetal catabolites are passed back into the mother's circulation through the placenta, the examination of placenta gives a clear idea of what had happened with it, when it was in the mother's womb and what is going to happen with the foetus in future.

The present study was carried out to analyze and study the morphometric features, gross and histological changes of placenta in pregnancy induced hypertension (PIH) irrespective of the severity of hypertension. Pregnancy induced hypertension (PIH) is defined as hypertension occurring after 20 weeks of gestation and completely resolves after delivery.

MATERIALS AND METHODS: The present study was done for a period of 21 months from April 1st 2008 to December 31st 2009 at Department of Pathology, Government Medical College Kottayam,

Fifty mothers with uncomplicated pregnancy and 100 mothers diagnosed as having pregnancy induced hypertension were selected from patients of our institutions.

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The age range was from 20-40 years, and parity –primi, para2 and 3.After delivery the placentae were taken in containers with 10% formalin. Immediate morphometric examination of placenta was made. Placentae were examined specifically for infarction, calcification, retroplacental hematoma and tumors like chorangioma.

The umbilical cord was checked regarding the insertion, length and the number of vessels. After fixation, tissues were taken from the insertion of the umbilical cord, margins-3, 6, 9, 12^0 clock positions, centre of placenta, fibrotic area, infarcted area and the umbilical cord. Routine hematoxylin and eosin sections were made for the histopathological study. Placentae were studied as that of PIH patients (Test group) and normotensive mothers (Control group).

RESULTS: The findings were studied under the headings-placental morphometry (Table I), gross anatomy (Table II) and histopathology which included villous (Table III) and stromal pathology (Table IV).

Parameters	Hypertensive group	Control group	Statistical significance (p value<0.05)
Mean Fetal weight (kg)	2.1	2.9	Significant(0.00)
Mean Placental weight (gm)	370+/_68.2	570+/_49.62	Significant(0.00)
Mean Placental area $(cm)^2$	221.2+/_30.2	283.3+/_27.2	Significant (0.00)
Mean Placental volume $(cm)^3$	397+/_59.9	567.9+/_79.8	Significant (0.00)
Fetoplacental ratio	6.5	5.7	Significant (0.01)

Table I: Placental Morphometric Study

Parameters	Hypertensive group	Control group	Statistical significance (p value<0.05)
Marginal insertion of cord	3%	0	Not significant
Infarction	26%	10%	Significant (0.032)
Calcification	21%	10%	Not significant
Retro placental hematoma	3%	0	Not significant
chorangioma	3%	0	Not significant

Table II: Gross anatomy of placenta

Parameters	Hypertensive group	Control group	Statistical significance (p value<0.05)
Syncytial knots	32%	16%	Not significant
Fibrinoid necrosis	19%	2%	Significant (0.01)
Acute atherosis	16%	4%	Significant (0.03)
Hyalinised villi	27%	14%	Significant(0.04)

Table III: Villous pathology

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Parameters	Hypertensive group	Control group	Statistical significance (p value<0.05)
Fibrosis	38%	8%	Not significant
Calcification	25%	10%	Significant (0.03)
Hyalinised areas	28%	16%	Not significant
Endothelial proliferation	13%	2%	Significant(0.04)
Inter villositis	5%	0	Not significant

Table IV: Stromal pathology

DISCUSSION:

Fetal Weight: The average fetal weight for the control group and for the hypertensive group were calculated. For those babies who were appropriate for the gestational age the average weight was 2.9kg. But for small for date babies the average weight was 2.1 Kg.

This indicates that the weight of a newborn baby is significantly low in pregnancy induced hypertension. This is in concordance with the study conducted by Jain et al.[1] In the study conducted in Massachussets General Hospital[2] similar findings were noted.

PLACENTAL MORPHOMETRY: The placental weight of control group ranged from 520+/-49.62gms, that of test were 375+/-68.2gms. This was in concordance with the study conducted by Majumdar S et al (2005).[3] The same thing was noted by the study conducted in Massachussets dept of Pathology in 2008.[2] Hosemann (1949)[4] in his study of normal term pregnancy found the placental weight of 400-1000 grams whereas Wigglesworth (1962)[5] found placental weight to be 360-570 grams.

In our present study the placental area and volume of the control group were 283.3+/-27.2 cm^2 and 567.9 +/- 79.8cm^3 respectively. For the hypertensive group it was 221.2+/-30.2cm^2 and 397+/-59.9cm^3 respectively. Similar findings were noted in the studies conducted by Udainia et al (2004),[6] Brown and Veall (1953)[7] and Garg et al. (1996).[8]

So from the present study it can be concluded that foetal weight and placental measurements are significantly reduced in PIH.

GROSS FEATURES:

COTYLEDONS: The no of cotyledons were ranging from 16-20. There was no significant difference regarding this in the control group and hypertensive group. The study conducted by Jain et al[1] also did not find a difference in this between test and control group.

ATTACHMENT OF THE UMBILICAL CORD: Three of our cases showed marginal of the umbilical cord (Fig. 1). Others showed central attachment of the cord. In our study this was seen only in three cases of PIH group. Whereas in the study of Majumdar et al[2] this finding was seen in more number of cases.

CALCIFICATION: 21% cases of placenta of PIH group and 10% of placenta of control group showed calcification grossly. This is not a statistically significant finding.

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Even though this finding was significant in that of Majumdar et al[2] study; a study conducted by Masschusetts hospital[1] has not included this as a finding in placenta of hypertensive patients.

INFARCTION: This is a significant finding in the hypertensive group (Fig: 2). Zeek and Assali (1950)[9] defined placental infarction as a zone of ischaemic necrosis of a group of villi due to complete interference with their blood supply in the deciduas, thrombosis of a spiral arteriole or a retroplacental haemorrhage.

Fox (1967)[10] stated that in pregnancies complicated by, preeclamptictoxaemia, the incidence of placental infarction was considerably raised, above that found in uncomplicated pregnancies. The incidence of placental infarction was related to the severity of PIH in these cases and not to any other maternal factor. In the present study also, placental infarction is present in cases of PIH. Fox (1978)[11] found infarction in 25% of normal term placentae.

RETROPLACENTAL HEMATOMA: This finding was seen only in hypertensive patients with babies having growth retardation. Clinically they were cases of abruptio placentae.

CHORANGIOMA: 3 cases of chorangioma were seen (Fig: 3). All were seen in PIH patients. Chorangioma is the hemangioma of placenta. A. This was reported in 35% of the IUGR cases from Massachussets Hospital.[2] In one large study chorangiosis occurred in 3% of 1614 deliveries and was associated with placental lesions including infarcts, chronic villitis, fetal artery thrombi, and spiral artery thrombi.[12,13,14,15]

Altshuler et al.[16] found chorangiosis in 5.5% of 1350 placentae of babies admitted to the neonatal intensive care unit; among 38-40 weeks of gestation.

HISTOPATHOLOGY: Both the villous findings and stromal findings were noted separately.

VILLOUS PATHOLOGY:

SYNCYTIAL KNOTS: More than 125-150 syncytial notes per 100 villi are considered as significant. 32% of PIH cases showed this finding whereas 16% of control group showed the same finding (Fig. 4).

ACUTE ATHEROSIS: In the study conducted by General hospital of Massachussets.[2] acute atherosis was noted as a specific finding which was seen in 18% of preterm placente. In that study only 5% of the term placentae showed acute atherosis. This is considered as a part of decidual vasculopathy and is associated with severe hypertension and poor fetal outcomes.

According to Robertson,[17] acute atherosis is a lesion limited to blood vessels that have not been altered by the normal adaptive processes of implantation. However, identical vascular lesions have also been described in placentae complicated by other hypertensive disorders, diabetes, SLE and antiphospholipid antibody syndrome.

FIBRINOID NECROSIS AND HYALINISED VILLI: These findings were obvious in the hypertensive group compared to the control group. This is in concordance with Masschusetts general hospital[2] study. (Fig. 5)

STROMAL PATHOLOGY:

STROMAL FIBROSIS: 38% of the placentae of PIH patients showed stromal fibrosis whereas only 16% of the control group showed this finding.

CALCIFICATION: More number of hypertensive cases showed calcification making it a significant stromal finding.

ENDOTHELIAL PROLIFERATION: 11% of PIH cases have this finding whereas only 2% of the control group showed this feature. So endothelial proliferation can be considered as a more specific finding for PIH. This is considered as a part of foetal thrombotic vasculopathy.

INTERVILLOSITIS: 5% of the PIH placentae showed this finding (Fig: 6) whereas none of the normal placentae showed this feature. Taking PIH placentae as a whole, it became statistically insignificant because of less no: of cases. This is in concordance with the Massachussets study[2]. This can also be considered as a specific finding for PIH.

CONCLUSION: All the placental morphometric parameters are significantly altered in hypertensive cases. A single finding either in the gross or microscopy cannot be said as pathognomonic of PIH but acute atherosis, fibrinoid necrosis and endothelial proliferation were more specific and significant findings in our study. If present, placental findings can be confirmatory of pregnancy induced hypertension, but its absence does not exclude the disease as these findings will become more evident only when there is significant reduction in the utero placental blood flow.

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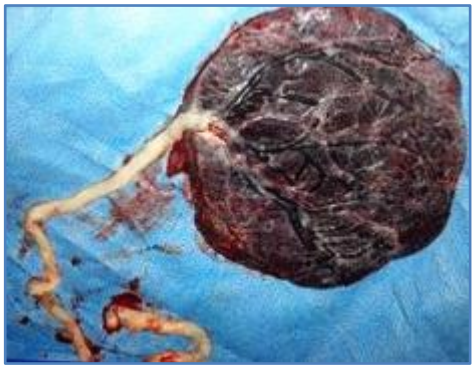

Fig. 1: Marginal insertion of the umbilical cord

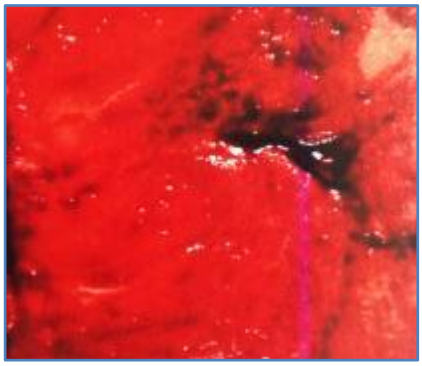

Fig. 2: Placental infarction

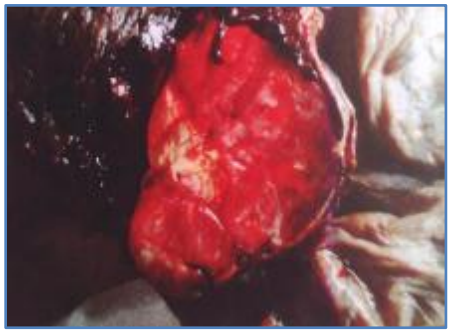

Fig. 3: Chorangioma

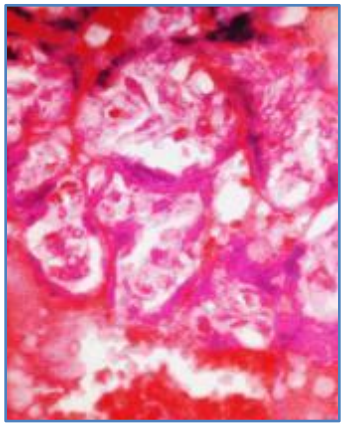

Fig. 4: Syncytial knots

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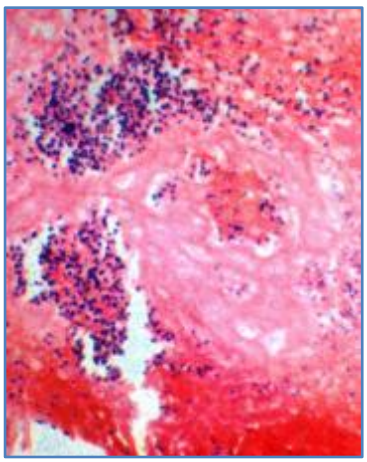

Fig. 5: Fibrinoid necrosis

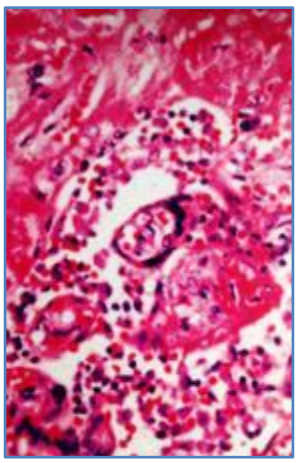

Fig. 6: Intervillositis

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