Histopathological variations inthe placenta in pregnancy-induced hypertensive females with special reference to low birth weight babies.

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Abstract

The foetus, placenta, and, mother constitute the vital triad in pregnancy. Placenta is the most accurate record of the infant's prenatal experience. It is derived from both fetal and maternal tissue. Maternal tissue is decidua basalis and the fetal portion is chorionfrondosum.

Preeclampsia is a serious medical condition that affects pregnant women, and it is characteriz ed by high blood pressure and damage to organs, typically the liver and kidneys. One of the ways to diagnose preeclampsia is by examining the placenta after delivery.

Reduction in uteroplacental perfusion is thought to be the only pathogenic process contributing to the development of preeclampsia, according to the most common etiologic hypothesis. Lowered uteroplacental blood flow would lead to lighter babies at delivery.

The present study aims to find out histopathological changes in the placenta of pregnancy-induced hypertensive females and compare the results with normotensive antenatal females.

A total of 85 cases (blood pressure 140/90 or above) were taken as the study group and 60 normotensives were taken as the control group. A detailed histopathological examination was performed by two trained histopathologists. The p-value is calculated and results were compared between the two groups.

Histological study showed a significant increase in syncytial knot formation, avascular villi, hyalinized villi, perivillous fibrin deposition, stromal fibrosis, and infarcted villi in the study group as compared to the control group. There was a decrease in mean birth weight in babies of the study group as compared to the control group.

We can infer a connection between the placenta, PIH, and the foetus. Ischemic damage is caused by uteroplacental vascular anomalies. The modifications lessen newborn birth weight and disrupt fetoplacental blood flow. A useful understanding of the mechanism behind placental malfunction is gained through meticulous examination.

Keywords: Histopathology, preeclampsia, pregnancy inducted hypertension, stomal fibrosis, hyalinized villi.

Introduction

Placenta provides insight into the growth of the foetus.[1] Hypertension is one of the leading causes of preterm and stillbirths, especially, in developing countries like India. [2] Hypertensive disorders,along with infection and hemorrhage, are common in pregnancy.They are the deadly triad causing large no of maternal and fetal deaths. Due to incomplete vascular remodeling in the placenta, there are some pathological changes like intrauterine growth restriction, preeclampsia, etc.[3]

Naeye and Friedman stated that foetal deaths in women with hypertension are due to large placental mosaicism.It may be associated with inadequate placentation and retroplacental ischemia.[2] Hypertensive disorders complicate 5-10% of all pregnancies and contributegreatly tomaternal morbidities and mortalities. Of these disorders, preeclampsia syndrome either alone or superimposed on chronic hypertension isthe most dangerous. Pregnancy complications like hypertension are reflected, both macroscopically and microscopically, in the placenta in a significant way. [3]

Preeclampsia is a multisystem disorder of unknown etiology. In previously normotensive and nonproteinuric women,with proteinuria after the 20th week, it is characterized by the development of hypertension to the extent of 140/90 mm Hg or more. Eclampsia-preeclampsia when complicated with generalized tonic-clonic convulsions and/orcoma is called eclampsia.[4]The inadequate trophoblast invasion of maternal spiral arteries leads to decreased placental perfusion and is associated with the changes seen in preeclamptic women. As the placenta reflects the intrauterine status of the foetus, examination of the placenta gives a clear idea of what had happened within, and what is going to happen with the foetusin the future. [5]

Aims and Objectives

1. To investigate the histopathological changes in the placenta of women with pregnancy -induced hypertension (PIH) and their association with low birth weight (LBW) babies
2. To determine the prevalence of different histopathological lesions in the placentas of PIH women with LBW babies.
3. To evaluate the correlation between the severity of histopathological changes in the placenta and the severity of PIH.

Methods and Material

The present study was carried out at GMC Bhopal with due clearance from the institutional ethical committee. The study was carried out on data over a period of two years.A total of 85 placentas were collected from patients with preeclampsia and eclampsia. In addition,60 placentas were collected from normotensive patients as the control group.Thebirth weights of babies of both groups were recorded.

Inclusion criteria: Patients with eclampsia and preeclampsia.

Exclusion criteria: Autolyzed samples and samples of patients with other manifestations (anemia, diabetes, hypothyroidism,etc) were excluded from the study.

The specimensconsisting of a placental disc with an attached umbilical cord and membrane were collected.A photographic record was taken prior to grossing. The lesions were grossly described and the dimension of each lesionwas measured. One block section includesa roll of extraplacental membrane and twocross-sections of the umbilical cord, one from the foetal end and the other from the placental insertion end. These blocks were prepared from the full-thickness section of the normal-appearing placenta.The sections were processed and stained with HarrisHaematoxyline and Eosine stain.

These sections were then examined microscopically for: fibrinoid necrosis, stromal fibrosis, areas of trophoblastic cellular proliferation, syncytial knot formation, avascular villi, infarcted villi, hyalinized villi, calcified areas, median coat proliferation of medium-sized blood vessels, perivillous fibrin deposition, physiological modification of decidual arteries.

All these findings were evaluated and compared with the placentas of normotensivesplacentae.Then the results were observed and recorded.

Statistics: The study and control group placentas were examined by two observers both macroscopically and microscopically. The results were tabulated.

Results

The mean birth weight and standard deviation of babies inthe study group are 2.11kg and 0.31respectively. The same in the control group are2.67kg and 0.20 respectively.These are presented in Table 1.It was found that 100% of the cases in the control group had a baby birth weight of 2.5- 3kg. 88.23 % of the cases of the study group had a baby birth weight of 1.6-2.5kg.

A comparison of histological changes between normotensive and hypertensive placentais presented in Table 2. An increase in syncytial knot formation, as shown in Figure 1a, was seen in 58 (68.2%) cases and in 12 (20%) cases inthe control group. The results were significant with $p< 0.00001$. Fibrinoid necrosis was seen in 38 (44.7%) cases and 30 (50%) cases in the control group.With a p-value of 0.0529,the results were not significant. The area of trophoblastic cellular proliferation was seen in 10 (11.75%) cases. It ishistopathologically explained,using Haematoxylin and eosin staining, in Figure 1b. These were not seen in the control group. Similarly, lack of physiological transformation of decidual arteries and median coat proliferation of medium-sized blood vessels were not present in the control group. In the

study group, these were seen in 20(23.3%) cases. Histopathological features are depicted inFigure 1c. Asignificant increase in stromal fibrosis in the study group as compared to the control group is observed. Figure 1d shows the stromal fibrosis areas. Calcified areas were seen in 35(41.1%) cases of the study group and 34(56.6%) cases of the control group. The p-value of 0.06584 makes the result insignificant. Hyalinized villi Figure 1ewere seen in 54(63.52%)cases of the study group and 04(6.6%)cases of the control group. The results were significant with a p-value was<0.0001. Perivillous fibrin deposition was found in 18(21.7%) cases of the study group and 02(3.3%) cases of the control group. Infarcted villi were found in 47 (53.5%) cases of the study group and 20(33.3%) cases of the control group. Histopathological features are depicted in Figure 1f. Avascular villi were present in 18(21.1%) cases of the study group and 02(3.3%) cases of the control group. The results forperivillous fibrin deposition, infarcted villi, and avascular villi were significant.

Table 1: Comparison of birth weight of babies between Study & Control groups

Birth weight	Study Group	Percentage	Control Group	Percentage
1 - 1.5kg	10	11.76 %	00 (00 %)	00 %
1.6 - 2.5kg	75	88.23 %	00 (00 %)	00 %
2.5 - 3kg	00	00 %	60 (100 %)	100 %
Total	85	100 %	60	100 %

Table 2:A comparison of histological changes in normotensive and hypertensive placenta.

S.No	Histological Feature	Study Group		Control Group		p-value
		Numbers	Percentage	Numbers	Percentage	
1	Increased syncytial knot formation	58	68.2	12	20	p < 0.00001
2	Fibrinoid necrosis	38	44.7	30	50	p = 0.05242

3	Areas of trophoblastic cellular proliferation	10	11.75	00	00	
4	Calcified areas	35	41.1	34	56.6	p = 0.06584
5	Hyalinized villi	54	63.52	04	6.6	p< 0.00001
6	Perivillous fibrin deposition	18	21.7	02	3.3	p = 0.002149
7	Infracted villi	47	53.3	20	33.3	p = 0.008992
8	Avascular villi	18	21.1	02	3.3	p = 0.00214
9	Lack of physiological transformation of decidual arteries	20	23.3	00	00	
10	Median coat proliferation of medium sized blood vessels	21	24.7	00	00	

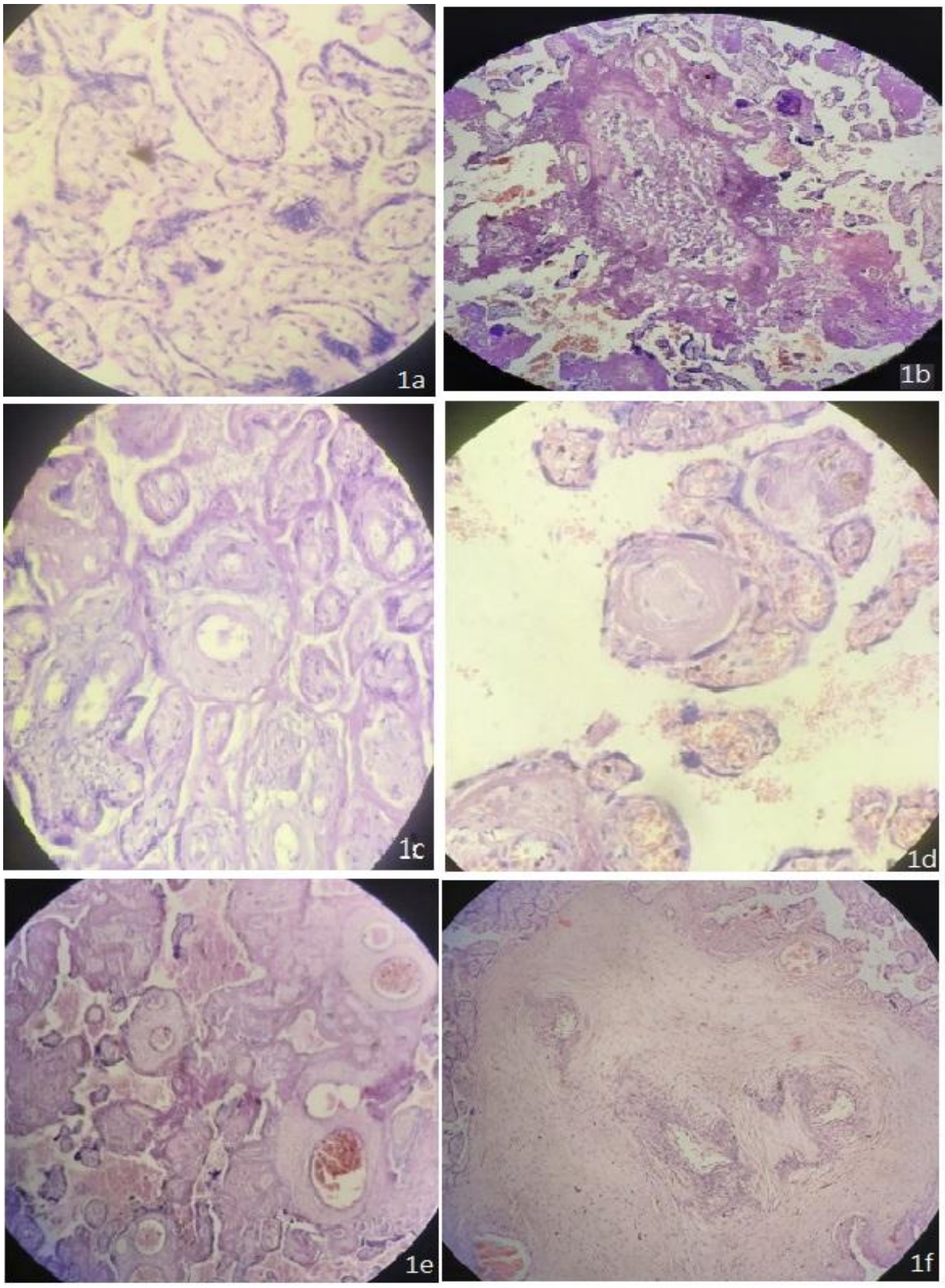

Figure 1:

1a: Microscopic view showing increased syncytial knot formation
1b: Microscopic view showing areas of trophoblastic cellular proliferation
1c: Microscopic view showing villus stomal fibrosis
1d: Microscopic view showing hyalinized villi
1e: Microscopic view showing infarcted villi
1f: Microscopic view showing lack of physiological transformation of decidual arteries.

Discussion

The present study hasa mean birth weight of 2.107kgin patients withpreeclampsia, which is similar to many other authors.Xiong X[6] studied that birth weights are significantly lower in mothers with preeclampsia who delivered at < 37 weeks. Majumdar S etal. [7] observed mean birth weight of babies with preeclampsia was 2.04kg. Tushar K etal. [8] found the mean birth weight of babies in patientswith preeclampsia to be 2.076kg. TS Raghuraman [9] observed 262 cases with significant antenatal obstetric problems and found that 56% of cases with low birth weight had preeclampsia. Ojha K et al.[3] studied the placenta in preeclampsia and found the mean birth weight to be 2.37kg.

The mean weights of the placenta in preeclampsia in the studies done by MajumdarS.[7],Pasricha N [10],YiebeltalW and Amenu T[11],Ojha K[3], and,Phadungkiat W [12]were399.10, 329.17, 456.20, 386.36, 372.1 gm respectively.The mean weight in our study is 371.86. gmwhich is in concordance with the findings of the previous studies.

As per Sengupta K et.al. [13], the shapes of the placenta in preeclampsia were oval in 40%, circularin 33.35%, and irregular in 26.7% of cases.YiebeltalW and Amenu T[10],the shapes of the placenta in preeclampsia were oval in 10%, circularin 72%, and irregular in 8% of cases.In the present study, the shapes of the placenta in preeclampsia were oval in 22.5%, circular in 72.2%, and irregular in 5% of cases.

As per the study done by Majumdar S. [7],the mean number of cotyledon in the hypertensive group was 16& that of control group was 17. The result found were not significant. As per YiebeltalW.and Amenu T.[10], the mean number of cotyledon was 17.24 ± 1.06 in preeclampsia and 18.66 ± 1.21 in normotensive females. The present study is similar to these with number of cotyledon in the study group being 13.88 ±4.37 and the control group as 22.37±2.70. The results were significant with a p-value<0.01.

Ananya A.[14], OjhaK et al. [3], and, Tushar K et.al. [8] found a retroplacental clots in 12 %, 5.45%& 7% of the cases of preeclampsia respectively. The present study has very similar results with 9 % of cases of preeclampsiashowingretroplacental clots.

In the present study, there is a significant increase in stromal fibrosis in patients of the preeclampsia group as compared to the control group.Similar findings have been seen in many other studies done by Majumdar S.[7] Sahay B. [15]Ananya A.[14]and Ojha K.etal. [3].

Tushar K. etal.[8],Ananya A.[14],and,OjhaK. et al. [3] observed 64.2%, 70% and 82.5% cases respectively, of preeclampsia showing an increase in syncytial knot formation. The incidence of fibrinoid necrosis in preeclampsia placenta 53.3%, 77.5% and 32.72% cases respectively.In the present study, these numbers are 68.2% and 41.1% cases, respectively. The results are very similar to other studies.

As for theMajumdar S. etal. [7] and Tushar K. etal. [8],in the present study, significant increase in areas of trophoblastic cellular proliferation is observed in contrast to that of the control group.

In the present study 63.52% of cases of preeclampsia are showing hyalinized villiwhich is statistically significant in comparison to that of the control group. Similar results were also observed in other studies, done by Majumdar S. etal [7], Farrah S. [16], and OjhaK. et al. [3]

Calcified areas, coating proliferatingmedium sized blood vessels, and lack of physiological transformation of decidual arteries is comparable to the findings of Fiona L. et al [17], Majumdar S. at al [7],and OjhaK. et al[3].

Conclusion

It can be concluded that the placenta, pregnancy-induced hypertension, and foetusare interlinked. Uteroplacental vascular abnormalities like lack of physiological transformation of decidual arteries and medial coat proliferation of medium-sized blood vessels lead to ischemic damage. Ischemic damage due to preeclampsia lead to histological changes in the placenta such as increased syncytial knot formation, fibrinoid necrosis, areas of trophoblastic cellular proliferation, calcification, perivillous fibrin deposition, avascular, infarcted and hyalinized villi, lack of physiological transformation of decidual arteries and median coat proliferation of medium-sized blood vessels. These changessignificantly compromise fetoplacental blood flow and reduce neonatal birth weight. A careful histopathological examination provides valuable insight into the mechanism of placental dysfunction.

Future Scope of Work: More detailed studies need to be done at the cellular level involving the various factors affecting the histopathological changes in the placenta in relation to pregnancy-induced hypertension.

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