

Placenta as a reflection of maternal disease-Pregnancy induced hypertension (PIH)

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Citation this Article: Dr. Archana Joshi, Dr. Aparna Manjarkhede,“Placenta as a reflection of maternal disease-Pregnancy induced hypertension(PIH)”, IJMSIR- June - 2021, Vol – 6, Issue - 3, P. No. 08 – 14.

Type of Publication: Original Research Article

Conflicts of Interest: Nil

Abstract

Background: Placenta is the mirror of maternal and foetal status. It reflects changes due to maternal causes like Anaemia, diabetes mellitus and hypertension. Pregnancy induced hypertension are the leading cause of maternal mortality and an important factor in fetal wastage. The incidence is high in developing countries with malnutrition, hypoproteinemia and poor obstetric facilities.

Material and Methods: A total of 100 placentae were collected.50 belonged to normal pregnancy and 50 cases were of pregnancy induced hypertension. All placentae were examined for various gross and microscopic parameters.

Results: Placentae were found to be of less weight in PIH than normal pregnancy. The histological parameters like cytotrophoblastic proliferation, increased syncytial knot formation, stromal fibrosis etc. were found more commonly in diseased group.

Conclusion: Morphological features were more prominent in placentae of pregnancy induced hypertension than normal placenta.

Keywords: Placenta, Pregnancy induced hypertension (PIH),Villous abnormalities.

Introduction

Placenta is a unique and wonderful organ directly related to the growth and development of the fetus in the uterus.^[1]

Being an organ of vital importance for the continuation of a pregnancy and fetal nutrition, it has created great interest among the pathologists. Many persons have done a lot of research work to understand the complexity of this unique organ.^[1]

Pregnancy complications like hypertension or gestational diabetes is reflected in the placenta significantly both macroscopically and microscopically. It has been observed and recorded that the maternal uteroplacental blood flow is decreased in pregnancy induced hypertension due to maternal vasospasm. Reduced maternal uteroplacental blood flow leading indirectly to constriction of fetal stem arteries has been associated with different changes seen in placenta of Hypertensive pregnancies^[2,3] Pregnancy induced hypertension is a frequent disorder with a reported incidence of 2-8% among pregnancies. It accounts for many maternal deaths every year worldwide. Placenta is a natural focus of the source of this disease as all the signs and symptoms of this disease resolve after delivery of placenta.

PIH is the serious disease of the pregnancy occurring in mid to late stages of gestation.

In India the hypertensive disorders complicating pregnancy are quite common. The review of literature reveals that this group exerts its deleterious effect on the placenta.^[4] Thus there is need for a thorough examination of placenta.

Material and Methods

This is a hospital based cross sectional study carried out in the department of Pathology of a tertiary care hospital over a period of 2 years.

Total 100 cases were collected; 50 cases with uncomplicated (normotensive) pregnancy and 50 cases with pregnancy induced hypertension were selected randomly from patients of Department of Obstetrics. History of past illness was recorded and patients were examined clinically for weight, blood pressure, Anaemia etc. The laboratory investigations like blood sugar, urea, creatinine, haemoglobin, urine for proteins and pus cells were done along with ophthalmoscopic examination.

Samples were divided into two groups

Group A-Control group

Group B-Study group

Inclusion criteria

Placentae were collected from all pregnant women of all ages and parity.

Group A-Uncomplicated full term deliveries with normal blood pressure without proteinuria and edema.

Group B-Pregnant women with blood pressure equal to or more than 140/90 mm of Hg at least on 2 occasions; 6 or more hours apart after 20 weeks of present gestation with or without proteinuria, edema and convulsions.

Exclusion criteria

Cases with hypertension prior to pregnancy and hypertension secondary to other causes were excluded.

Cases associated with Diabetes mellitus, anaemia, renal disease, cardiac disease were also excluded.

Placental Examination

The standard protocol for examination was followed for placentae received in the department of Pathology

Placentae were studied grossly for different parameters like weight, shape, diameter, thickness, membranes, presence of infarction, calcification, retro placental haematoma length of umbilical cord, insertion of umbilical cord.

The gross abnormalities were noted as 'absent' when no visible lesion was noticed and 'present' when gross lesions were visible.

Sections were taken from the different sites for histopathological study

- 1) Centre of the placenta
- 2) Margins of the placenta
- 3) Near insertion of umbilical cord
- 4) Infarcted area if any
- 5) Calcified area if any
- 6) Fibrotic area
- 7) Umbilical cord
- 8) Membranes

The sections were processed in an automatic tissue processor. Tissues were embedded in paraffin wax and cut at 4 microns. The sections were stained with Haematoxyline and Eosin (H & E) stain.

One hundred villi were counted from sections obtained and histological changes expressed as percentage.

The histological study of placenta was done in detail which consists of number of syncytial knots, cytotrophoblastic proliferation, fibrinoid necrosis, villous vascularity, basement membrane thickening,

endarteritis obliterans, villous stromal fibrosis, inter villous haemorrhage, extra villous cytotrophoblastic cells.

All the gross and histopathological features were compared with placenta of control group.

Statistical analysis was done with Chi square test.

A p value of <0.05 was considered as statistically significant.

Results

The placentae from patients with pregnancy induced hypertension weighed <500 gm, the lowest weight being 180 gm whereas the placentae from normal pregnancies weighed >500 gm, the heaviest being 700 gm in the present study.

Table1: Gross features of placenta

Gross Features	Control group N=50 No. of cases (%)	Hypertensive group N=50 No. of cases (%)
Average weight of Placenta	454gm	336 gm
Average Diameter	23 cm	16 cm
Infarction Present	9(18%)	22(44%)
Calcification Present	30(60%)	18(36%)
Retro placental haematoma Present	6(12%)	10(20%)
Per villous fibrin Present	19(38%)	28(56%)

Various villous lesions like vascularity, syncytial knots, cytotrophoblastic proliferation, basement membrane thickening, vasculosyncytial membrane, endarteritis obliterance, villous stromal fibrosis and fibrinoid necrosis were studied microscopically. The changes

Table 2: Various Villous abnormalities

Villous lesions	Control group N=50 No. cases (%)	PIH group N=50 No. of cases (%)
Villous vascularity decreased	22(44%)	35(70%)
Increased syncytial knots	19(38%)	45(90%)
Cytotrophoblastic proliferation	14(28%)	45(90%)
Basement membrane thickening	11(22%)	44(88%)
Paucity of vasculosyncytial membrane	30(60%)	46(92%)

The size and thickness of the placenta was less in PIH as compared to normal.

Most of the placentae were round. Bilobed placenta was seen in 1%of cases.

The umbilical cord showed central insertion (76%) followed by eccentric insertion. In the present study there was no membranous or vilomembranous insertion. The gross features like calcification were seen in control as well as in hypertensive group. The incidence of infarction (Fig.1), retro placental haematoma and per villous fibrin was higher in PIH than normal group.

between control and study group were noted. (Fig.2- Fig.8)

Enderteritis obliterance	7(14%)	23(46%)
Villous stromal fibrosis	12(24%)	46(92%)
Fibrinoid necrosis	22(44%)	48(96%)

Statistical analysis-The gross abnormalities in the study group were statistically significant ($p < 0.001$) when compared to normal. Villous abnormalities in the PIH group when compared to control group were statistically significant ($p < 0.001$).

Discussion

Placenta is the most important organ which plays key role in pregnancy. It is the mirror of perinatal status. Examination of placenta is important for both mother and infant. It gives valuable information which is important for further management. There are various maternal disorders where placenta plays a fundamental role. The pregnancy induced hypertension is one of such disorders which exerts its deleterious effect on the placenta.^[3] So the present study was undertaken to analyse the placental changes in pregnancy induced hypertension. The study was carried out to assess the significance of gross placental changes and various histopathological parameters as these changes serve as a guide to the duration and severity of the disease. In the present study the commonest age range of the mothers in PIH was 21-30 years. In the present study average weight of normal placenta was 454.4gm and in PIH cases the weight was 335.8gm. In various studies there are variations in placental weight. Mallick et al, have reported five cases of toxemia of pregnancy with placental weight < 300gm. Study done by Uдания A, Jain M.L. placental weight was significantly less in hypertensive group than the control group.^[2] Similar findings were observed with other studies.^[4]

Bhatia et al, in other study showed reduced placental weight in severe PIH, the lowest being 280gm.^[5]

The maximum number of PIH placentae in the present study showed diameter significantly lower than control group. This was similar to studies done by Goswami P, Salman O, Mallik et al.^[6]

The gross pathological abnormalities of the placenta were discussed in various studies.

Placental infarct is considered pathological if it is seen in more than 5% of surface area. In toxemia of pregnancy it is commonly seen because of thrombotic occlusion of maternal uteroplacental vessels.^[7]

In present study infarction is seen in 44% of cases, which is comparable with studies done by Narsimha et al (41%), Mirchandani et al (37%), Masodkar et al (40%).^[1,8]

Uдания et al observed that there is increase in the incidence of placental infarction with severity of toxemia.^[9]

Calcification is considered as evidence of placental degeneration.

In present study the incidence of calcification in normal placenta is more as compare to PIH. In PIH cases, calcification is seen in 36% of cases.

Narsimha and Vasudeva in their study noted that incidence of calcification in normal placenta was 62%. In toxemia cases the overall incidence was 26.9%

The retro placental haematoma was seen in 20% case in present study whereas it is 11.1% in study carried out by Narsimha.^[1]

The wide spectrum of villous lesions was observed in PIH patients. The villous lesions were mainly due to

decreased maternal uteroplacental blood flow in patients of PIH due to maternal vasospasm.

Villous vascularity

Decreased vascularity of villi was observed in cases of severe PIH in present study. This is supported by various studies like

1) Narsimha 2) Kalara et al 3) Dhabhai P et al.^[10]

Cytotrophoblastic proliferation is seen in patients with increasing severity of blood pressure. In the present study in control group 28% of villi show cytotrophoblastic proliferation and in study group 90% of villi show increased proliferation. This is similar to study done by Narsimha et al. Endarteritis obliterance is seen in placenta from PIH patients. In present study 46% of PIH cases show endarteritis obliterance.

Paucity of vasculosyncytial membrane is an index of fetal hypoxia.^[11] In the present study paucity of vasculosyncytial membrane is seen in 92% cases of study group.

Basement membrane thickening

The incidence of villi showing thickened basement membrane was considered as abnormal and was a common feature of placentae from pregnancy induced Hypertension. In the present study it is seen in 88% of cases of control group.

Fox^[11] considered that it is because of proliferation of cytotrophoblastic cells which secreted the basement membrane as a response to placental ischemia.

Syncytial knots

A significant increase in the syncytial knot formation in the placental villi may indicate disturbance in the hormonal factors. It is an indication of excessive aging due to either post maturity or disease state causing placental insufficiency. In the present study increased incidence of syncytial knots was seen in 90% cases of PIH.^[12,13]

Villous stromal fibrosis

It is due to reduced uteroplacental blood flow.

In our study villous stromal fibrosis was seen in 92% of PIH cases whereas 245 seen in control group.

Almost similar findings were seen in study done by Fox and Kalra et al.^[9,11]

Fibrinoid necrosis

Significant villous fibrinoid necrosis was noted in cases of PIH which is comparable with other studies.^[14,15]

Conclusion

From the present study it can be concluded that, the hypertensive disorders of pregnancy adversely influence the morphology of the placenta which ultimately influence the foetal outcome.

The gross and histopathological features are more significant in placenta of hypertensive group. These changes can be attributed to the reduced uteroplacental blood flow which occurs in PIH cases.

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Legend Figures

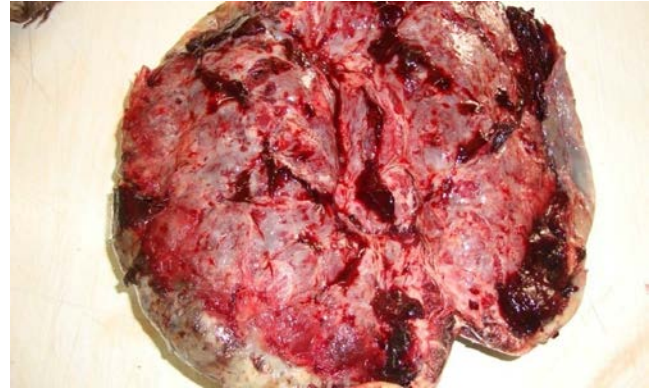


Fig.1: Gross photograph showing infarction of Placenta

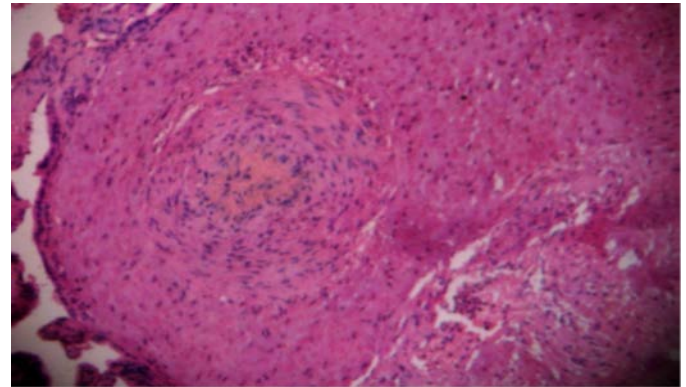


Fig. 2: photomicrograph showing endarteritis Obliterance (H&E, 40X)

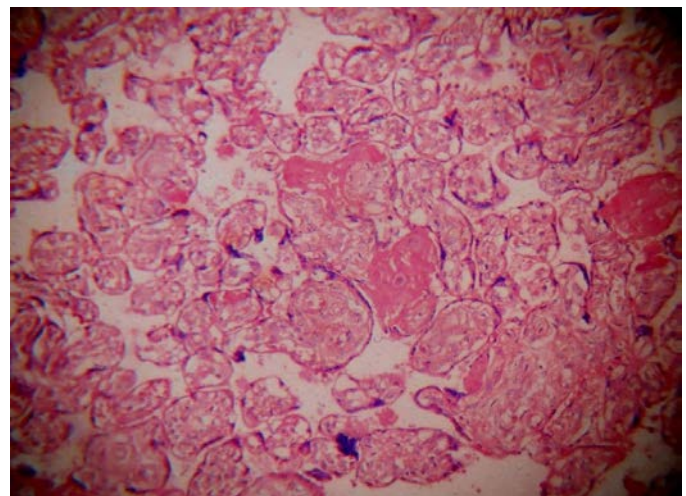


Fig.3: Photomicrograph showing few hyalinized and fibrotic villi (H&E, 10X).

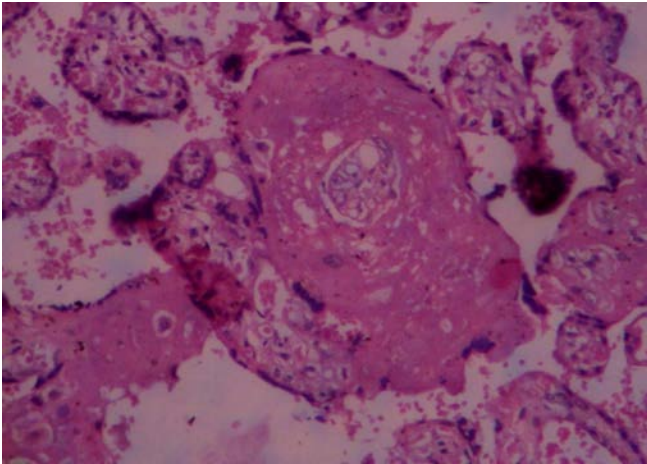


Fig.4: Photomicrograph showing villous stromal fibrosis (H&E,40X).

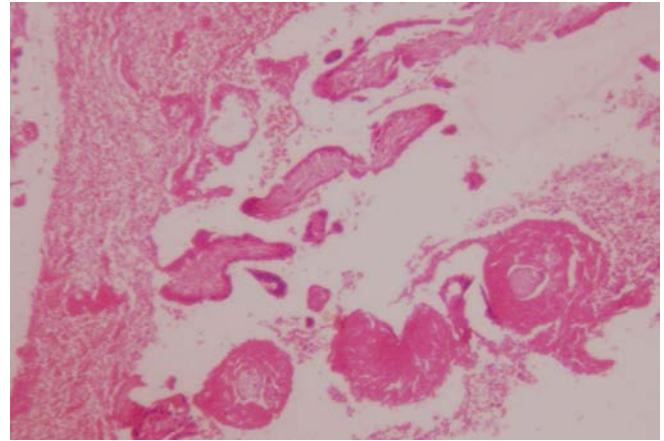


Fig.7: Photomicrograph showing perivillous fibrin deposit (H&E,10X).

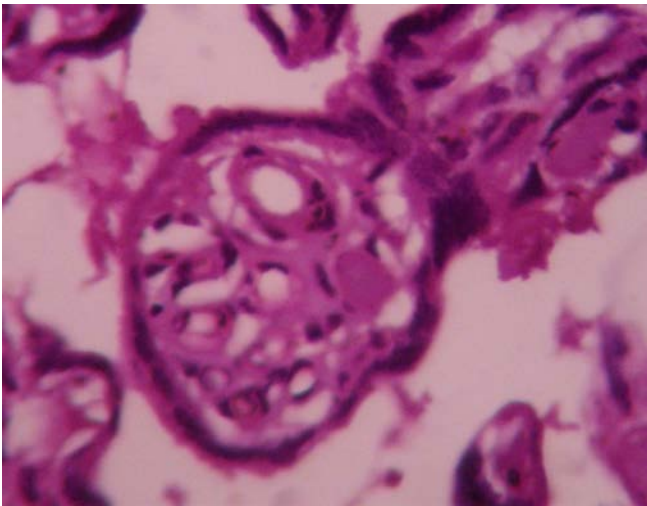


Fig.5: Photomicrograph showing basement membrane thickening (H&E,40X).

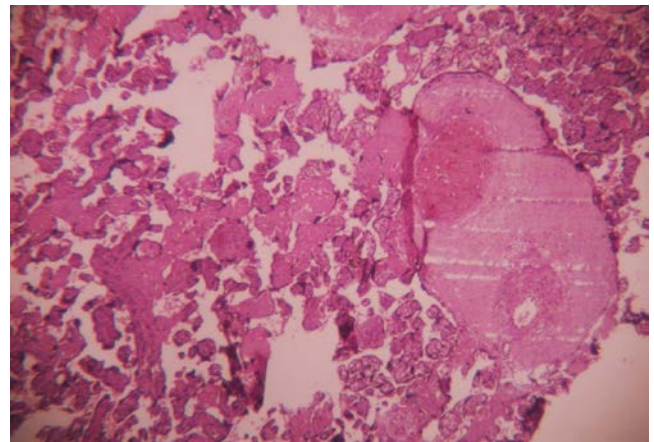


Fig.8: Photomicrograph showing infarction and focus of endarteritis obliterans (H&E,10X).

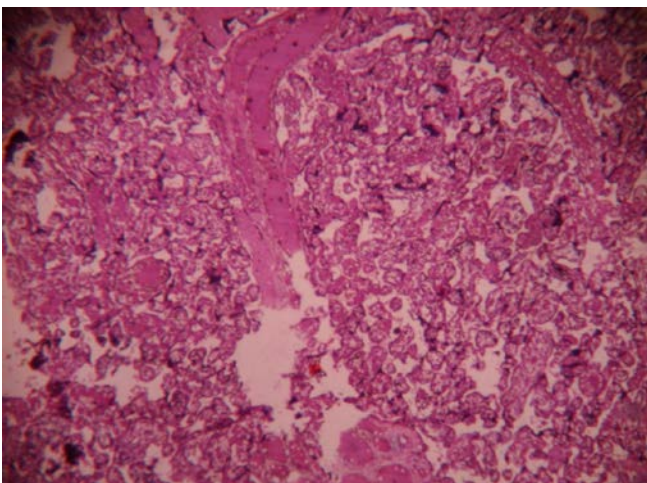


Fig.6: Photomicrograph showing infarction of villi (H&E,10X).