Histo-pathological changes of placenta in normal and pregnancy induced hypertension

Dr.Parth R. Goswami[1], Dr.Shaila N.Shah[2], Dr.RekhaIyer[3]

ABSTRACT

Introduction: Placenta is the most important and vital organ of intrauterine life.Hypertension is one of the common complication met within pregnancy and contributes significantly to maternal and fetal morbidity and mortality. **Objectives:** This study is undertaken to assess the morphology of placenta in pregnancy induced hypertension and compare the same with that of normal pregnancy. **Material and method:** Fifty mothers with uncomplicated pregnancy and fifty mothers with pregnancy induced hypertension were selected randomly from in-patients of Obstetrics and Gynecology Department, Sir Takhtasinhji General Hospital,Bhavnagar, Gujarat. Gross and microscopic histopathological examination was carried out on placenta. Comparative study was done between two groups. The data were statistically analyzed using Chi square test by using EPI INFO 7 software. **Results:** Microscopic examination of placenta in Pregnancy induced hypertension show excessive syncytial knots, increased fibrinoid necrosis**,** increased villous stromal fibrosis and increased basement membrane thickening of the Villi along with decreased vasculosyncitial membrane as compare to normotensive group. **Conclusion:** Pregnancy Induced Hypertension alters the placental histomorphology. The histopathological changes among PIH group were statistically significant as compared to Normal. This will contribute to the better understanding, treatment and finally optimal management of the problem.

Keywords: Eclampsia; hypertension, Placenta; Preeclampsia

[1] Resident (R3), [2]Associate Professor, [3]Assistant Professor

Department of pathology,Government medical college, Bhavnagar, Gujarat, India.

Corresponding author Email: goswamiparth42@yahoo.com Conflicting Interest: None

INTRODUCTION

Pregnancy induced hypertension including gestational hypertension, pre-eclampsia or eclampsia, complicates a significant proportion of all pregnancies and contributes significantly to increased maternal and perinatal morbidity and mortality.

Pregnancy associated complications like hypertension are reflected in the placenta in a significant way (both macroscopically and microscopically). It has been observed and recorded that, the maternal utero-placental blood flow is decreased in pre-eclampsia due to maternal vasospasm. Reduced maternal utero-placental blood flow leading indirectly to constriction of foetal stem arteries has been associated with the changes seen in the placenta of pre-eclamptic women.

Placenta being a foetal organ shares the same stress and strain, to which the foetus is exposed. Thus any disease process affecting the mother and foetus also has a great impact on 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 the future. With this objective the present study was carried out.[1]

OBJECTIVES:

-To study the histomorphology of placenta in cases of pregnancy induced hypertension.

-To contrast the features of placenta in toxaemia with that of normal and conclude with respect to its significance.

-To correlate the pathological changes in placenta with the severity of toxaemia and degree of hypertension.

MATERIALS AND METHODS:

Fifty mothers with uncomplicated pregnancy and fifty mothers with pregnancy induced hypertension were selected randomly from in-patients of Obstetrics and Gynecology Department.

They were divided into two groups as Control group and PIH group.

Control Group comprised of pregnant women with normal bloodpressure, no proteinuria or edema.

PIH Group comprised of pregnant women with blood pressure at or above 140/90 mm of Hg on at least two occasions, six or more hours apart after 20weeks of present

pregnancy together with or without proteinuria, edema, convulsions and coma.

Clinical data were collected from the case records along with laboratory investigations.The placenta were collected immediately following delivery and washed in tap water.The shape,fetal surface,placental membrane and umbilical cord insertion was determined.

The maternal surface of the placenta was inspected.The whole placenta was left for fixation in 10% formal saline for 24-48 hours.

Following variables were studied… weight,diameter,thickness and number of cotyledon.

Tissues for microscopic examination were taken from selected areas and tissue bits were then processed according to the tissue processing protocol.

Tissue sections of 5 μm thickness were cut from paraffin embedded blocksand stained by conventional haematoxylin and eosin stain andaccording to findings special staining techniques like PAS (Periodic Acid Schiff) for basement membrane thickening and Van Gieson for stromal fibrosis, were employed.

Macroscopically infarction and calcification were noted.

Histopathologically syncitial knots,vasculosyncitial membrane,fibrinoid necrosis,fibrotic villi and basement membrane thickening were evaluated.

The different histological findings were observed against the background ofnormal standards of histology of placenta as described by Fox H and Peterson et al. [2]

The data was arranged in excel workbook undersuitable headings. They were statistically analyzed using Chi square test. This was done usingEPI INFO 7 software. In all the tests ‘p’ value less than 0.05 was taken to be statisticallysignificant and a value less than 0.01 was taken as highly significant.

RESULTS

A total of 100 placentas were studied, out of which 50 (50%) placentas werefrom normal term mothers (BP <140/80mm Hg) which formed the control group and 50(50%) placentas were from mothers with PIH (BP > 140/80mm Hg) who formed the PIH

Group. Of these 40 cases were of preeclampsia, 7 cases of gestational hypertension and 3 cases of eclampsia.

In both control and PIH groups mothers,majority belong to the age group between 20-30 years.

Of the 50 controls 20 (40%) were primigravida and 30 (60%) weremultigravida and out of 50 PIH cases 35 (70%) were primigravida and 15 (30%) were multigravida. This shows that in the present study PIH was more common in primigravida.

Mean systolic blood pressure in control group was 122 and PIHgroup was 152 mm of Hg. The mean diastolic blood pressure in controlgroup was 80 and PIH group was 98 mm of Hg.

In the PIH group placental weight (410 gm) was significantly less ($p< 0.001$) than that of control group (512 gm).

Mean diameter of placenta of control group was17.5 cms and that of PIH group was 14.5 cms. The difference in the mean diameter between thetwo groups was statistically significant ($p<0.001$).

The mean thickness of placenta of control group was 2.60 cms and that of PIH group was 2.20 cms. The difference in the thickness between thetwo groups was statistically significant ($p<0.001$).

Table 1: Incidence of various placental parameters in both the groups

Parameters	Control group (n=50)	PIH group(n=50)
Shape of placenta		
Circular	32	27
Oval	18	20
Irregular	-	03
Fetal surface	Normal	Normal
Membranes	Normal	Normal
Umbilical cordinsertion		
Central	41	20
Eccentric	08	22
Marginal	01	08
Velamentous	-	-
Single umbilical artery	Nil	Nil
Mean umbilical cord length in	26.5	22.5

cms.		
Knots in umbilical cord	Nil	Nil
Maternal surface		
Mean number of cotyledons	19	15
Calcification	08	18
Infarction of >5% area	00	10
Retroplacental hematoma	Nil	Nil

This shows that in the PIH group, eccentric insertion of the umbilical cord,irregular shaped placenta, calcification and infarction (Figure 1)involving >5% of parenchyma were more common and showed less number of cotyledons and smaller umbilical cord compared to control group.

Figure 1: Thrombosed vessels causative factor for placental infarction…

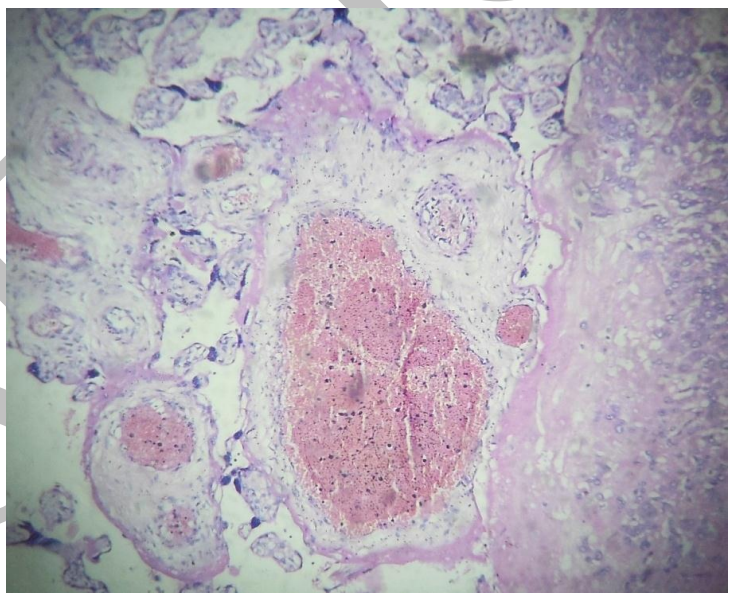

Table 2: Microscopic lesions of placenta

Microscopic lesions	No.of placenta showing microscopic changes		Percentage of placenta showing microscopic changes	
	Control group N=50	PIH group N=50	Control group N=50	PIH group N=50
Syncitial knot formation in >30% of villi(Figure 2)	03	37	06%	74%
Vasculosyncitial membrane<5 % of villi	00	40	00%	80%
Fibrinoid necrosis>3%of villi(Figure 3)	32	45	64%	90%
Stromal fibrosis>3%of villi(Figure 4)	34	48	68%	96%
Basement membrane thickening>3%of villi	01	30	02%	60%

The above table summarizes the number of placenta showing different microscopic lesions and percentage of placenta showing microscopic lesions in control and PIH group.

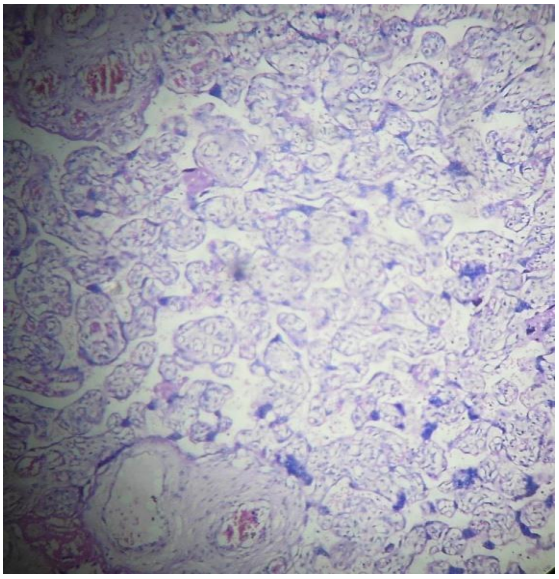

Figure 2: Excessive syncitial knots

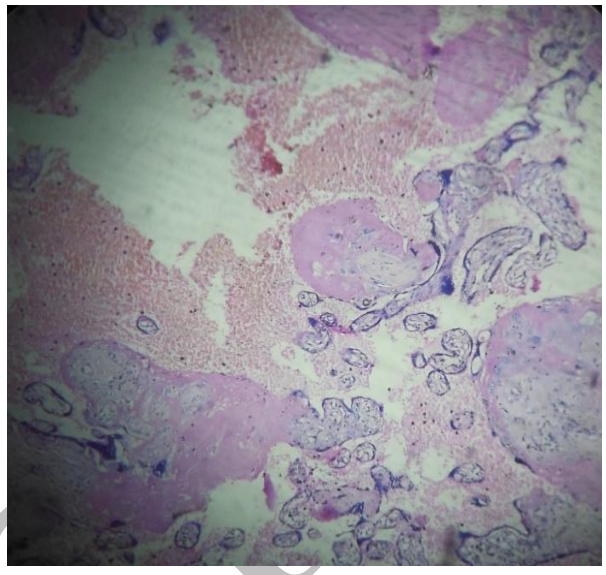

Figure 3:Fibrinoid necrosis

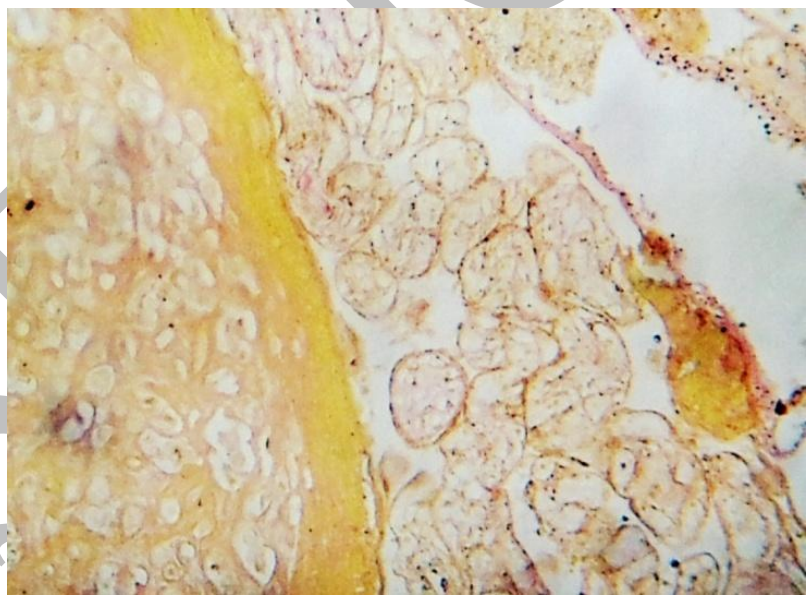

Figure 04: Villous Stromal fibrosis… (Van gieson stain)

DISCUSSION

Normally the placental morphology varies considerablyduring its short life span.

Hence in the study on placenta Fox in 1997 has stressed the importance of analyzingthe placental pathology quantitatively.[3]In the present study gross and microscopic lesions of placenta in both control and PIH group were quantified and compared.

Incidence of eclampsia (3 out of 50) was low due to good obstetric care. The incidence of preeclampsia landing in eclampsia was less similar to that reported by Menon. [4]

Jain et al and Manjunatha et al compared the number of PIH placentas delivered with **parity** and found that 45.91 % of control mothers and 54.09 % of PIH mothers were primigravidas. This is in accordance with the previous studies.[5,6]

In the present study the **shape** of placenta was round 32/50 in control groupand 27/50 in PIH group whereas it was oval in 18/50 in control and 20/50 in PIH groups.

Irregular placenta was observed in 03/50 placenta of PIH group and none of theplacenta of control group. Dadhich et al and Ashfaq found that most of placenta were round in controland PIH group, similar to the present study. [7,8] Shah et al has observed no clinicalsignificance in oval or rounded shaped placenta.[9]

The incidence of marginal and eccentric insertion of the umbilical cord in the PIH group (08 and 22 out of 50) was more than that of the control group (01 and 08 out of 50).

This is in concurrence with the findings of Fox, Pretorius, Majumdar et al, Rathetal and Udainia et al.[10,11,12,13]

The average length of umbilical cord of PIH group was 3.4 cms less than the control group similar to study by Jain et al.[05]

Mean **number of cotyledons** was found to be less in PIH group compared toplacenta of control group. This finding is similar to the findings of other studies.[14,15,16]Manjunatha et al did not find a significant difference in the mean number of cotyledons in both control and PIH groups. [06]

Kurdukar et al have reported that thrombotic occlusion of maternal uteroplacentalvessel is responsible for infarction. [17] Wigglesworth and Fox are of the opinion that extensive infarcts affects fetal outcome.[18,10] Jain et al have shown that extensive infarcts are associated with higher incidenceof fetal hypoxia and intrauterine death.[05]

In the present study, infarction involving morethan 5% of placental parenchyma was found in 10% of placenta of PIH group whilenone of the control group placenta showed extensive infarction.[05]

This finding is similar to the findings in other studies.[14,17]

Calcification was seen in both control and PIH placenta in the present study.

Increased incidence was observed in PIH placenta by Kurdukar et al and Manjunatha etal.[17,06]

None of the placenta in our study showed retroplacental hemorrhage.

Microscopic lesions:

The microscopic lesions were assessed and quantified as percentage ofplacenta in control and PIH groups showing syncytial knots in more than 30% of villi,vasculo-syncytial membrane in ≤ 5% of villi, fibrinoid necrosis, stromal fibrosis

andbasement membrane thickening in more than 3% of villi.

Table 3: Comparison of microscopic lesions in present study with that of studiesby Narasimha et al [19] and Navbir et al. [20]

Microscopic Lesions	Percentage of placenta showing the microscopic lesions (%)					
	Present study (2013-2014)		Narasimha et al (2011)		Navbir et al (2012)	
	Control (n=50)	PIH (n=50)	Control (n=37)	PIH (n=63)	Control (n=30)	PIH (n=30)
Syncytial knots >30%	06	74	45	90.47	6.67	66.67
Vasculosyncytial membrane ≤ 5%	00	80	00	93.65	00	56.67
Fibrinoid necrosis >3%	64	90	29.72	97.82	73.33	90
Stromal fibrosis >3%	68	96	81	92	80	93.33
Basement membrane thickening >3%	02	60	00	49.25	00	70

Syncytial knot counts were found to be significantly higher in PIH group ascompared to controls and in cases of eclampsia of the PIH group. The findings of the present studycorrelated well with other studies. [19,20]

Placenta in which 6 to 30 percent of the villi show vasculo-syncytial membranes are said to have a normal count which shows ability of the placenta to supply oxygen to the fetus. The findings of the present study are comparable with otherstudies which also showed reduced vasculo-syncytial membrane counts. [20,21,10,22]

Placenta in which **fibrinoid necrosis** involving up to three percent ofplacental

villi is considered as normal and placenta in which the percentage of villi showing fibrinoid necrosis of greater than three is considered as abnormal.

The findings of present study, was in conformity with earlier studies.[19,20,17]

In term placenta ≤ 3 percent of villi may be fibrotic and if more than 3 percentof villi in a placenta are fibrotic it is considered as abnormal. Increase in **villousstromal fibrosis** (> 3% of villi) in the PIH group has been reported in all the previousstudies and in the present study. [19,20]

Microscopic lesions in eclamptic cases…..

Table 4: Comparison of microscopic lesions in eclamptic placenta of present study with that of studies by Kurdukar, [17]Narasimha et al [19] and Navbiretal[20].

Microscopic lesions	Percentage of eclamptic placenta showing themicroscopic lesions (%)			
	Present study (n=3) (2013-2014)	Kurdukar et al (2007) (n=10)	Narasimha et al (2011) (n=9)	Navbir et al (2012) (n=6)
Syncytial knots >30 % Of villi	100	100	100	100
Vasculo-syncytial membrane ≤ 5% of villi	100	70	83.33	100
Fibrinoid necrosis>3% of villi	100	100	100	100
Stromal Fibrosis >3% of villi	100	50	100	100
Basement membrane thickening	100	100	100	100

As the severity of the disease increased like in eclampsia, the microscopic lesions where seen in increased number of villi (100 %) in the present study, thesefindings concur with other studies. [19,20,101]Vasculo-syncytial membrane in ≤ 5% ofvilli was observed in all the cases of eclampsia in the present study but was observedin 83.33 % of cases by Narasimha et al and 70 % of cases by Kurdukar et al (Table 04). [19,17] This could be because of late presentation of cases of eclampsia in the present study or due to less no. of cases (n=3) of eclampsia in the present study.

CONCLUSION

- Gross examination of placenta in Pregnancy induced hypertension (PIH)shows decreased mean placental weight, thickness, diameter, number of cotyledonsand cord length. They also show increased incidence of marginal insertion ofcord, oval and irregular placenta, calcification and infarction.

- Microscopic examination of placenta in Pregnancy induced hypertensionshows excessive syncytial knots, increased fibrinoid necrosis**,** increased villous stromal fibrosis and increased basement membrane thickening of thevilli.

- Quantitative determination of placental changes is essential in study ofplacenta as normal pregnancies can also show similar placental changes due toageing.

- Hypertensive disorders in pregnancy influence the morphology of placenta which adversely affects the perinatal outcome. The early measurements of placenta by noninvasive technique like ultrasonography will be helpful in early identification of at

risk fetus and better management of such pregnancies.

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