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A study on structural changes of placenta in pregnancy induced hypertensive pregnancies in eastern India

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Abstract: *Background:* Complications of pregnancy are reflected in placenta. Placenta is the mirror of maternal and foetal status. So a study of placenta and new born will help to identify the disease by which mother is affected in pregnancies of pregnancy induced hypertension. *Aim:* Our study aimed in study of morphology, morphometry and histology of placenta in normal pregnancy and in pregnancy induced hypertensive (PIH) mothers. This study also correlates the birth weight and height of the new born babies of both the mothers of normal pregnancy and PIH. *Materials and Methods:* 50 booked cases (25-normal pregnancy and 25 PIH) have been studied in an institution in eastern India. Antenatal check up of these cases were made routinely. After delivery, placentas were examined for morphological, morphometric, histological and histopathological parameters and features. Birth height and weight of the newborn babies were documented in each case. Finally, parameters were analyzed in between both the groups. *Results:* Placental weight and volume reduced significantly in P.I.H. mothers and the reduction of placental area was suggestive of pregnancy induced hypertension. Incidence of gross calcification in placenta, retro placental blood clots and infarction increased markedly in pregnancy induced hypertensive mothers. There has been increased incidence of syncitial knots and syncytio-trophoblast in PIH. Thickening of basement membrane, increased intervillous space and fibrinoid necrosis were associated with PIH. *Conclusion:* Structural changes in the placenta, macroscopic ad well as microscopic, during pregnancy induced hypertension has been detrimental to the foetus, which is the main focus of our study.

Keywords: Normal pregnancy, Pregnancy induced hypertension, Placenta.

Introduction

All Eutherian mammals possess placenta. Human placenta is a discoid chorio deciduate organ which connect the fetus with the uterine wall of the mother. It is a structure where maternal and foetal tissue come in direct contact without rejection, suggesting immunological acceptance of the foetal graft by the mother.

The placenta had a rough irregular maternal surface developed from decidua basalis and smoothfoetal component developed from chorion frondosum. Whole of the nutrition and oxygen exchange to the fetus from mother through placenta which also reveal as an organ of respiration and excretion of the fetus. Proper intra- uterine foetal growth and development depend on the adequate gaseous exchange, nutritive transfer and excretion of waste products of fetus. It also maintains and modifies the growth and development of the fetus. Both placenta and fetus are equally affected in condition of altered homeostasis of mother as are seen in hypertensive and diabetic condition. Placenta is the most accurate record of the infants' prenatal experience as stated by Benirschke [1]. Hypertensive disorder in pregnancy (either pregnancy induced hypertension or pre-eclampsia or eclampsia or chronic hypertension aggravated by pregnancy) may lead to intra-uterine growth retardation of

fetus, foetal distress, even intra- uterine death of foetus due to reduced utero-placental circulation. Poor development, inadequate adaptation of the utero placental arteries in pre-eclampsia may explain reduced chorio-decidual blood supply in hypertensive pregnancy.

In normal pregnancy proliferating cytotrophoblastic cell invade the spiral arteries in a retrograde direction deep in the myometrium. The muscular and elastic tissues of the media are destroyed and replaced by fibrinoid material resulting in dilatation funneling of spiral arteries [2]. On the basis of placental bed biopsies Brosen et al [2] reported that in pre-eclampsia there is failure of second wave of trophoblastic invasion so that the musculo-elastic media of the spiral arteries in the myometrium is retained, the vessels fail to dilate and remains responsive to vasoconstrictor stimuli resulting in a decreased chorio-decidual flow. Acute atherosis in some spiral arteries may be associated with thrombosis and possibly placental infarction in hypertensive placenta. So placenta is the organ of communication between the mother and fetus through the umbilical cord. Placenta is considered to be mirror of maternal and foetal status. It is proved that maternal hypertension (pregnancy induced or pregnancy aggravated) affect the placenta and fetus with significant evidence. So if examination of placenta is undertaken thoroughly, the degree of diseases or complications of diseases can be assessed which provides information in the field of children and maternal welfare.

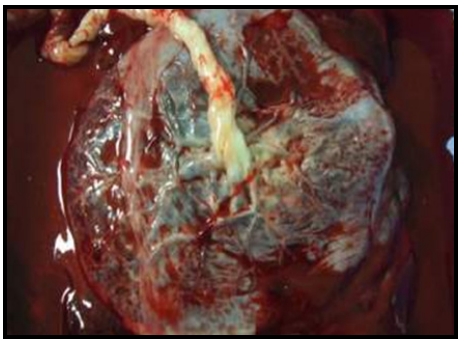
Material and Methods

A total number of 50 booked cases were included in this study. The cases were divided into two groups (Normal pregnancy-25 cases & Pregnancy with hypertension-25 cases). Clinical study of the cases was done in pregnancy and in labour. Antenatal checkups of these cases were made routinely. L.M.P.(Last menstrual period), EDD (Expected date of delivery, gestational periods and other important and relevant histories were documented properly in all the cases. General examinations were done properly with special note for presence of pedal oedema. Blood pressure, blood sugar levels, haemoglobin percentage, urinary presence of albumin and other important systemic examinations were done and findings were recorded carefully.

Morphometric & morphological examination of placenta after delivery: Fresh placentae were collected from mothers who either had undergone Caesarean section in the operation theatre or normal delivery in the labour room. Each placenta was examined and parameters were documented. Shape, Dimension (maximum and minimum diameter were measured), Area, Weight and Volume (estimated by water displacement method) were recorded for each placenta.

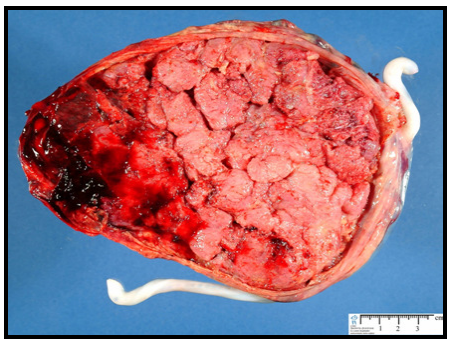
Foetal surface (Figure 1) of placenta was examined with due importance to the following points: mode of insertion of the cord,mode of insertion of membranes, colour and translucency of amnion, presence or absence of amnion nodosum, cyst, subchorionic fibrosis, calcification and infarction.

Fig-1: Fetal surface of placenta in P.I.H



Maternal surface (Figure 2) of placenta was also examined for complete or incomplete or any succenturiate cotyledon and the colour of the maternal surface was also noted. Presence or absence of blood clots, depressed area, calcification infarctions were also noted and finally the number of cotyledons were documented.

Fig-2: Maternal surface of placenta in P.I.H



Histolological examination of placenta: After proper fixation and processing of the procured specimen, blocks were made from those placental tissue. Sectioning was done properly by using rotary microtome and ultimately properly made slides were stained by haematoxylin and eosin dye and mounted. These slides were finally examined under binocular light microscope with 10X, 40X & 100X magnification. Birth height & birth weight of the newborns were recorded for all the cases.

Results

Mean weight of placentas in normotensive mothers were 442 gms, volume- 391 ml, and the area of the placenta- 245 cm2. There were more or less same in both sexes. Though placenta vol. were slightly lower in female than male, but weight of placentae was heavier in (443-442) = 1gm in case of female than male. Placentae of male babies were bigger than female babies (248 – 241) = 7 cm2. Average number of cotyledons were same in both sexes. Mean birth weight of babies was 2.48 kg with male being (2.56 – 2.40) = 0.16 kg. = 160 gm. Heavier than female. It shows that the mean height of babies in normotensive mothers were 45cm. These have been shown in Table 1.

Table-1: Birth weight & height and placental Morphometric study in normotensive mothers at terms

Sex of baby	No of cases	Avg. Birth Weight (kg)	Avg. Birth Height (cm)	Avg. placenta Weight (gm)	Avg. placental Volume (ml.)	Avg. Placental Area(cm^2)	No. of Cotyledon
Male	13	2.56	46	442	395	248	16
Female	12	2.40	44	443	391	241	16
All cases	25	2.48	45	442	391	245	16

Average weight, volume and area of placenta in P.I.H. mothers are 383 gms, 362 ml. respectively. These values were more or less same in both sexes. Average female placentas were bigger in area (233- 220) = 13cm2 and average weight of female placentas were more (386 – 378) = 8 gm. But placental volume was less in female (363-358) = 5 ml. Average no. of cotyledon in both sexes are same. The average body weight of baby in P.I.H. mothers were 2.5 kg. though the female were mild heavier than male (2.55-2.48)= 0.07 kg. = 70 gm. Height of babies in case of male were higher than female. All of the findings are depicted in Table 2.

Table-2: Birth weight & height and placental morphometric study in Pregnancy induced hypertensive mothers at term

Sex of baby	No of cases	Avg. Birth Weight (kg)	Avg. Birth Height (cm)	Avg. placenta Weight (gm)	Avg. placental Volume (ml.)	Avg. Placenta Area(cm^2)	No. of Cotyledon
Male	15	2.48	46	378	363	220	16
Female	10	2.55	42	386	358	233	16
All cases	25	2.50	44	383	362	225	16

It is seen that incidence of ill-defined cotyledons, sub-chorionic fibrosis and gross calcification are more of less same. But the retro-placental blood clot and gross infarction were much higher in P.I.H. mothers than normotensive mothers. Incidence of sub-chorionic fibrosis (Figure 3) were 24% and 20% and that of gross calcification were 16% and 20%.But incidence of retro-placental blood clot and infarction were much higher in P. I. H. mothers than normotensive mothers. In P.I. H. mothers 40% placenta showed retro-placental blood clots. Where as in normotensive mothers incidence were only 8%. Incidence of infarction in normotensive mothers and pregnancy induced mothers was 8% and 32% respectively. These findings have been compared in Table 3.

Fig-3: Microphotograph showing Calcifications in placenta of P.I.H

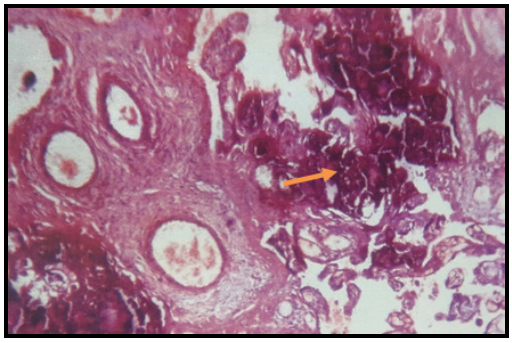


Table-3: Comparison of gross morphological changes of placenta in normotensive and P.I.H. mothers

Morphological changes	Normotensive mothers	P.I.H. Mothers
Presence of sub-chorionic fibrin	24%	20%
Ill defined cotyledons	24%	24%
Retro-placental blood clot	8%	40%
Gross calcification	16%	20%
Infarction	8%	32%

In case of pregnancy induced hypertensive mothers the incidence of fibrinoid necrosis (20%) in placenta and intervillous space (36%) was markedly increased than the normotensive mothers. No abnormality in basement membrane and cytotrophoblast in villous were found in normotensive mothers but 8% of mothers with PIH showed thickening of the basement membrane (as evident from Figure 4). Syncitial knot increased in P.I.H. mothers (16%) than that of normotensive mothers (4%). The comparison is evident from Table 4.

Fig-4: Microphotograph showing Basement Membrane proliferation in placenta of P.I.H

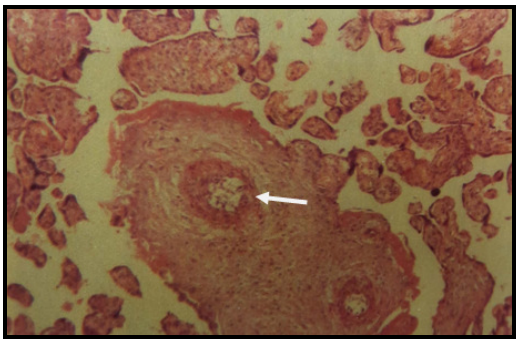


Table-4: Comparison of histological changes of placenta in normotensive and P.I.H. mothers

Abnormalities in placenta.	Normotensive mothers	P.I.H. Mothers
Abnormality of blood vessels.	0%	12%
Fibrinoid necrosis in villous.	8%	20%
Abnormality of intervilous space.	8%	36%
Abnormality in basement membrane (thickening)	0%	8%
Abnormality in cytotrophoblast in villous.	0%	8%
Syncytial knot	4%	16%

Discussion

Out of twenty-five cases of normotensive mother the placentae were either circular or ovoid in shape. This finding was same with general opinion by R. K. Shah et al [3]. The fetal growth was largely dependent on its nutrition coming from maternal blood as well as upon the surface area of the placenta available for diffusion of nutrient from mother to the fetus. Weight of the baby and weight of the placenta correspond well with chorionic villous surface area. The mean placental area in this present series was 245 sq.cm, which is slightly lower than Davies &Beazley [4] but it is higher than that of Murthy et al [5]. The mean placental volume in the present series was in case of normotensive mothers was 391ml. which is nearly equal with Aherene & Dunhil [6]. Placental weight may become the single most important factor to relate foetal growth. In this present study mean birth weight of baby was 2.48 kg and average placental weight was 383 gm. It was almost similar with the findings of RK Shah et al [3].

In this present study mean value of number of cotyledons was 16.This finding was similar with that of Murty et al [5]. No anomalies of cord insertion were observed. It was not also associated with low birth weight baby. This is in accordance with the findings of P.-F. Plouin [7], Peckham [8] and H. Fox.[9]. Robert E.L. jr. in 1972[2] observed that sub chorionic deposition of fibrin was present in almost

every placenta after 24 weeks of gestation. However, H. Fox [10] showed the incidence of sub chorionic fibrosis to be only 20%. The incidence in this study of normal pregnancy was 24%.In 1974 Nesbitt JR [11] gave opinion that no term placenta is free from calcification. He also indicated that calcification has no clinical importance. The incidence of this present study is 16%, which is almost same that of previous studies by Gore et al [12].

The incidence of retro placental haematoma observed by H. Fox in 1966 [10] was 4.5%. However, in this present study the incidence was 4% in normal pregnancy. The increased incidence of placental haemorrhage may be associated with generalized protein and vitamin deficiency [13]. Ahmed et al [14] noted the incidence of infarction varies according to the severity of PIH. R.K. Shah et al in 1985 [3] noted the incidence of 23.8% cases. In this present study incidence was found 8%.Histology in this present study of term normotensive mothers revealed the structure of normal basement membrane, mature villous and normal blood vessels in placenta. Others important finding were presence of fibrinoid necrosis 8%, increased inter villous space 8% and syncitial knot (increased) in 4% cases. It is strongly believed that pregnancy induced hypertension is responsible for depression of both fetal and placental growth, possibly as a result of placental insufficiency. Bandana Das et al [15] observed in their study that there was reduction in both fetal and placental weight in P.I.H. mothers, fetal weight being more affected than the placental weight. This was supported by Kher et al [16] in his study.

In our present study, average birth weight and height were not affected significantly when compared between normotensive mothers and hypertensive mothers. But for comparison of placental weight of normotensive mothers at term with that of P.I.H. mothers at term, there was significant decrease of placental weight in P.I.H. mothers. According to the Kher et al [16] there were reduction of both placental weight and birth weight in P.I.H. mothers where as baby weight was more affected. Result of the present study showed that there were moderate reduction of placental volume and weight in mothers having P.I.H.

As evident from the Table 5, the incidence of syncytial knots, fibrinoid necrosis, calcifications, cytotrophoblast cell proliferation, basement membrane (BM) thickening etc are more in P.I.H than normotensive mothers, as recorded by previous authors.

Table-5: Comparative incidence of syncytial knots, fibrinoid necrosis, calcifications, cytotrophoblast cell proliferation, basement membrane (BM) thickening etc in P.I.H and normotensive mothers, as recorded by previous authors.

Study	Syncytial knots	Fibrinoid necrosis	Calcifications	Cytotrophoblast cell proliferation	BM thickening
Singh et al [17]	+++++	++++	++++	++++	+++++
Udainia et al [18]	+	+++	+	+++	++
Das B et al [15]	+++	+++	++	+++	++
Damania et al [19]	+++	++	+++	+++	+++
Kofinas A D [20]	+++	++++	+++++	++	+
Pretorius et al [21]	+	+++	++	+++	+++
Di Salvo et al [22]	+++	++	+	+++++	++++
Salmani D [23]	++	++++	++++	+	++
Rajyalakshmi et al [24]	++++	+++++	++++	+++	+++++
Present study	+	+	++	++	+

Conclusion

Modern obstetrics aims at well-being of mother as well as fetus. Pregnancy induced hypertension is one of the most important complications in obstetrics. Pre-eclampsia or P.I.H. is a condition, complicated with pregnancy associated hypertension *(BP≥ 140/90 mmHg.*

After 20th weeks of gestation) proteinuria with or without pedal oedema. P.I.H. is further complicated with convulsion. The placenta is the focus to identify the etiology of P.I.H. It is associated with reduced placental perfusion. Microanatomical changes in placenta are indirect evidence of vascular insufficiencies due to hypertension during pregnancy which is reflected in birth weight and other parameters of fetal well-being.

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