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The Morphological and Histopathological changes in placenta in hypertensive pregnant mothers - A Prospective study

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Abstract

Placenta undergoes both macroscopic and microscopic changes in pregnancy complicated with hypertension. The ageing of placenta is intensified which subsequently affects the pregnancy outcome. The fetus depends on placenta for its normal development, thus changes in placenta adversely affect the blood flow to the fetus producing uteroplacental insufficiency. Maternal or fetal disorders may have placental sequelae since the maternal and fetal interface occurs at its site. Conversely, both maternal and fetal health can be affected by primary placental abnormalities hence the examination of placenta yields important information regarding the impact of maternal disorders on the fetus. This can be in the form of preterm delivery, fetal growth restriction or neurodevelopment impairment.

Keywords: Pregnancy, Hypertension, placenta.

Introduction

The intrauterine existence of fetus is dependent on one vital organ “The Placenta”. Placenta is essential for maintenance of pregnancy and for promoting normal growth and development of fetus.[1]In recent years, it has been revealed that there is clear relationship between morbid histological changes of placenta of hypertensive mothers and fetal growth restriction[1,5].It is the most accurate record of the infant's prenatal experience. During the first half of pregnancy, the placenta not only increases its surface area but reaches its maximum. By term, placental weight is approximately one sixth of fetal weight.[4]The placenta is a cadecuous organ which after performing its functions is discarded by the body. At various stages during development, the placenta performs a remarkable range of functions, until the fetal organs become functional.Hypertension is a global epidemic of general population and pregnancy is no exception to this rule. The hypertensive disorders complicating pregnancy include gestational hypertension, pre-eclampsia, eclampsia and chronic hypertension. Pregnancy induced hypertension is associated with macroscopic and microscopic changes structure of placenta. Hypertension in pregnancy intensifies morphological changes of ageing in placenta and subsequently affect outcome of pregnancy. The resulting ischemia causes fetal hypoxia, which may lead to fetal growth restriction, fetal distress and death.[1,5]

Aim: To study the morphological and histopathological changes in placenta in hypertensive pregnant mothers.

Materials and Methods

A total number of 400 primigravidae mothers, 200 subjects of hypertensive pregnant mothers and 200 normal pregnant mothers admitted in BebeNanki Hospital, Department of Obstetrics and Gynaecology, Govt. Medical College, Amritsar have been included in this study. Data was collected based on detailed morphological study of placentae and histopathological examination after preserving placenta in 10% formalin.

Technique:

Paraffin embedded tissue sections were stained with routine haemtoxylin and eosin (H&E) and diagnosis was made on the basis of the histopathological findings

A data was collected including patient particulars, antepartum clinical findings, laboratory tests, ultrasonography, intrapartum and postpartum outcome as well as perinatal outcome of the fetus. The newborn babies were inspected for APGAR score. Their birth weights noted and the feto-placental weight ratio was calculated in each case.

Results

Table I Placental shape variations

Placental Shape	Group		Total
	Study Group	Controls	
Discoid	150 (75.0%)	200 (100.0%)	350 (87.5%)
Irregular	50 (25.0%)	-	50 (12.5%)
Total	200 (100.0%)	200 (100.0%)	400 (100.0%)

Majority of hypertensive150 (75%) subjects had discoid shaped placentae and normotensive placenta200 (100%) had discoid placentae. Only 50 (25%) of hypertensive placentae were irregular in shape.

Table II Placental morphology in study and control group

	Group	N	Mean±S.D.	Statistical Significant
Placental Weight (gm)	Study Group	200	400.00±37.02	$<0.01^{S}$
	Control Group	200	483.45±29.01	
Placenta Diameter (cm)	Study Group	200	16.76±8.08	$<0.01^{S}$
	Control Group	200	18.43±1.416	
Placenta Thickness (cm)	Study Group	200	2.28±0.21	$<0.01^{S}$
	Control Group	200	2.41±0.22	
Number of Cotelydons	Study Group	200	15.32±9.36	$>0.05^{NS}$
	Control Group	200	15.21±1.13	
Feto Placental Ratio	Study Group	200	6.54±0.80	$<0.01^{S}$
	Control Group	200	6.17±0.46	

S = Significant; NS= Non Significant; $p<0.01$ = Significant; $p>0.05$ = Non Significant

The mean placental weight of study group was 400.00±37.02, whereas, placental weight of control group was 483.45±29.01 which was significant (p<0.01).

Mean placental diameter of study group was 16.76±8.08 and that of control group was 18.43±1.416 which was significant (p<0.01).

Mean placental thickness of study group and control group was 2.28±0.21 and 2.41±0.22; respectively, which was again significant (p<0.01).

The mean number of cotelydons of placenta in study group was 15.32±9.36 and that of control group was 15.21±1.13. The observed difference was statistically non-significant (p>0.05).

The feto placental ratio of study group was 6.54±0.80 and that of control group was 6.17±0.46 (p<0.01) which was significant.

Table III Morphological changes in placentae of study and control group

		Group		Total
		Study Group	Control	
Retroplacental Haematoma	Present	26 (13.0%)	7 (3.5%)	33 (8.3%)
	Absent	174 (87.0%)	193 (96.5%)	367 (91.8%)
Infarction	Present	69 (34.5%)	10 (5.0%)	79 (19.8%)
	Absent	131 (65.5%)	190 (95.0%)	321 (80.3%)
Calcification	Present	90 (45.0%)	34 (17.0%)	124 (31.0%)
	Absent	110 (55.0%)	166 (83.0%)	276 (69.0%)
	Total	200 (100.0%)	200 (100.0%)	400 (100.0%)

In the study group, 26 (13%) placentae showed retroplacental haematoma as compared to 7 (3.5%) of control group. Only 174 (87%) of study group and 193 (96.5%) of control group did not show any retroplacental haematoma.

Infarction was present in 69 (34.5%) study (hypertensive) group and 10 (5%) of (non-hypertensive) control group. Majority of non-hypertensive placentae (95%) showed no infarction.

Calcification of placenta was present in 90 (45%) hypertensive (study) group and 34 (17%) of non-hypertensive (control) group. Majority of non-hypertensive placenta (control group) i.e. 166 (83%) and 110 (55%) in study group showed no calcification.

Table – IV Histopathological changes in placentae of study and control group

		Group		Total
		Study Group	Control	
Syncytial Knot	Present	123 (61.5%)	37 (18.5%)	160 (40.0%)
	Absent	77 (38.5%)	163 (81.5%)	240 (60.0%)
Fibrinoid Necrosis	Present	106 (53.0%)	0 (0%)	106 (26.5%)
	Absent	94 (47.0%)	200 (100.0%)	294 (73.5%)
Villous hyalinization	Present	23 (11.5%)	0 (.0%)	23 (5.8%)
	Absent	177 (88.5%)	200 (100.0%)	377 (94.3%)
	Total	200 (100.0%)	200 (100.0%)	400 (100.0%)

Syncytial Knot was present in 123 (61.5%) of the study group and in 37 (18.5%) of control group.Majority of control group(81.5%) did not show Syncytial Knot.

Fibrinoid necrosis was present only in 106 (53%) study group. Rest of the hypertensive placentae 94 (47%) and all the non-hypertensive placentae i.e. 200 (100%) showed no fibrinoid necrosis.

Villous hyalinization was present only in 23 (11.5%) (hypertensive placentae) study group. Rest of the hypertensive placenta 177 (88.5%) and all the non-hypertensive placentae i.e. 200 (100%) showed no villous hyalinization.

Table V The mean birth weight in study and control group

Birth Weight	Group	N	Mean±S.D	Statistically significance
Birth Weight	Study Group	200	2.60±0.28	P<0.01[S]
	Control Group	200	2.98±0.25	

The mean fetal weight of study group was 2.60±0.28 and that of control group was 2.98±0.25. The birth weight of neonates was statistically low in hypertensive (study) group (p<0.01;) which was significant.

Table –VI fetal outcome in study and control group

Fetal Outcome	Component	Group		Total
		Study Group	Control Group	
Fetal Status	Live	197 (98.5%)	200 (100.0%)	397 (99.2%)
	IUD	3 (1.5%)	-	3 (0.8%)
Intrauterine Growth Retardation	Present	47 (23.5%)	2 (1.0%)	49 (12.3%)
	Absent	153 (76.5%)	198 (99.0%)	351 (87.8%)
Neonatal Intensive Care Unit Admission	Admitted	38 (19.0%)	-	38 (9.5%)
	Not admitted	162 (81.0%)	200 (100.0%)	362 (90.5%)
Meconium Stained Liquor	Present	65 (32.5%)	19 (9.5%)	84 (21.0%)
	Absent	135 (67.5%)	181 (90.5%)	316 (79.0%)

Out of 200 hypertensive subjects, 3 (1.5%) had stillborn babies and rest of the study group and all the control group subjects delivered live babies.

About 47 (23.5%) babies born in study group had growth retardation (IUGR), whereas only 2(1%) of control group babies were growth retarded. 198(99%) of babies in control and 153 (76.5%) in study were normal for gestation.

In study group, 38 (19%) of babies required admission and treatment in Neonatal Intensive Care Unit (NICU). But none was admitted in NICU from control group.

Discussion

In present study,on morphological examination majority of hypertensive and normotensive placentae were discoid in shape. Only 50 (25%) of hypertensive placentae were irregular in shape.This correlates with study done by Navbiret al[6]who found the shape of the placenta was discoidal in 73.33% in the study group and 83.33% in the control group.

Weight of placenta is functionally significant as it is related to villous surface area and fetal metabolism. Normally placental weight ranges from 400gms to 600gms.

In present study it was observed that mean placental weight was less in hypertensive placentae as compared to normotensive placentae. The weight of the placentae in study group was below 500 g. The least weight recorded being 300 g. In the control group, majority of the placenta weighed more than 500 g, the heaviest being 550 g. There was significant reduction in the weight of placenta in hypertensive one as compared to control group. This was similar to the observation made by Harshmohan et al.[7]Similar results are reported by Udaina et al[1], Mallik,Mirchandani and Chitra et al[9], Majumdaret al[3] and Artico et al[4] who found reduced placental weight in hypertensive cases as compared to normotensive pregnancies.

In present study morphometric parameter of placental diameter was significantly reduced in study group as compared to normal group ($P < 0.01$). The study by Mallik*et al*[8] reported that the mean diameter of placenta was 17.54 cm. Cibils et al [9]and Kishwaraet al[10] reported that placenta from hypertensive cases were smaller than normal indicating an underlying pathological process interfering with the normal growth of placenta.

The present study noted that the mean placental thickness was 2.28 cm in hypertensive placentae and 2.41 cm in normotensive placentae. Hypertensive placentae were smaller and thinner as compared to normotensive placentae. Similar findings have also been reported by Modiet al[13], Londhe et al[12]. Teasdale et al[11] found significant reduction in transverse diameter of preeclamptic placentae.

In the study, the average feto-placental weight ratio in normal pregnancy was 6.17 ± 0.46 and 6.54 ± 0.80 in hypertensive group. The values correlate with the earlier study by Majumdar*et al*[3]. The same ratio was found to be less in the hypertensive group than control group by Priyaet al.[14]

In present study, the commonest lesions observed macroscopically were infarction (34.5%). Infarction occurs due to thrombotic occlusion of maternal uteroplacental blood vessels and is seen in pregnancies complicated by hypertensive disorders. It worsens the uteroplacental blood flow.

The incidence of infarction among the hypertensive placentae was 36.7% in the present study and 6.7% in cases. Incidence of calcification was 56.7% in the hypertensive group and 23.3% in the non hypertensives group. Intra uterine death and low fetal weight was noticed among cases associated with placental calcification.[22]

Udainaet al[17], Majumdar S.et al[3] and Rosana R.M. et al [15] made similar observation in their study. This is in comparison with Masodkaret al[16] who had observed a similar increase in the incidence of placental infarction (40.4%) with severity of toxaemia.

On gross examination, retroplacental haematoma was present in (13%) of study group and 7% of control group. Tangirala S, kumara D found that gross lesions like retro placental hematomas were more in the hypertensive group which adversely influence the perinatal outcome.[21]

In present study placental calcification was present in (45%) in study group. Rath[4] 2000 and MajumdarSet al[3] also observed more foci of calcification in the hypertensive group.

In the present study, syncytial knot formation was seen in 61.5% of placentae of hypertensive group and 18.5% of control group.This was similar to the observation of Maham A et al[18]. Syncytial knot is thought to be a manifestation of degenerative changes in the trophoblastic tissue as well as a reaction to uteroplacental ischemia.

It is established that the physiology of the uterine vascular pattern, notably of the spiral arteries in patients is remarkably modified following decidual implantation of blastocyst and during placentation in hypertensive patients. A significant increase in the cytotrophoblastic cellular proliferation and syncytial knot formation in the placental villi may also indicate a disturbance in the hormonal factors which may probably lead to altered morphometry of placenta resulting in hypertensive disorders in the mother and low birth weight babies[20].

The present study observed that 53% of placentae in study group showed fibrinoid necrosis, similar to the observation made byMaham A et al [18]. The aetiology of fibrinoid necrosis is obscure but the possibility that it is due to an immunological reaction within the villous cytotrophoblastic tissue is postulated.

Udainiaet al[17] reported that fibrinoid necrosis and villous hyalinization probably leads to placental insufficiency and ultimately to fetal growth retardation.

Different microscopic variants were observed on histological examination of the specimens. The striking villous lesion seen in toxaemia of pregnancy werecytotrophoblasticcell proliferation. 72.5% cases of toxemia of pregnancy showed significant villous stromal fibrosis. Increased syncytial knot formation was seen in 82.5% of placentae of toxaemia of pregnancy 77.5% of cases of toxaemia of pregnancy showed fibrinoid necrosis.[23]

A study by Majumdar[3]et al showed histological findings like syncytial knot formation and fibrin plaque formation in greater amount in hypertensive placentae.

The study conducted by Goswamiet al[19]concluded that fetal outcome in terms of birth weight of newborn of mother having pregnancy induced hypertension and their placentae showed calcification (grossly and on microscopic examination) was poor as compared to control group.

Similar findings were reported by Kishwaraet al[10] who reported lower APGAR score in preeclampsia than normotensive cases. Placenta seems to adapt well to hypoxic condition in preeclampsia, although the compensatory changes that occur are insufficient. This leads to inadequate placental mass causing placental dysfunction and consequent chronic fetal hypoxemia.

Gunasena et al studied there is increasing evidence that hypertension in pregnancy is fundamentally related to poor trophoblast invasion of the myometrium which results in maternal spiral arteries being hampered in their normal physiological vasodilatation.[24]

Conclusions

Hypertensive disorders of pregnancy show significant differences in various parameters of placental morphology and histology. Hypertensive disorders of pregnancy adversely influence fetal outcome. Placenta has a great potential to provide valuable information in the case of an adverse foetal outcome.The present study makes us understand the importance of placenta and its role in the growth of a normal fetus. Placental examination becomes important as it will help in understanding the specific etiologies of adverse outcome which will need specific treatment so that preventive measures can be taken for those at risk for recurrence in subsequent pregnancies especially in preeclampsia and eclampsia cases.

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Conflict of interest: None declared

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