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EVALUATION OF MACROSCOPIC AND MICROSCOPIC CHANGES OF PLACENTA COMPLICATED BY PREGNANCY INDUCED HYPERTENSION WITH INTRA UTERINE GROWTH RESTRICTION OF FOETUS VERSUS PREGNANCY INDUCED HYPERTENSION WITHOUT INTRA UTERINE GROWTH RESTRICTION OF FOETUS - A COMPARATIVE STUDY

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ABSTRACT

Background: The placenta serves as a crucial feto-maternal organ that mediates nutrient, gas, and waste exchange between mother and fetus. Pregnancy-Induced Hypertension (PIH) is a major cause of perinatal morbidity and mortality and is frequently associated with Intrauterine Growth Restriction (IUGR). Placental morphology and histopathology play an essential role in understanding the pathophysiological basis of these complications. **Aim:** To evaluate and compare the macroscopic and microscopic changes in placentas from pregnancies complicated by PIH with IUGR and PIH without IUGR.

Materials and Methods: A hospital-based cross-sectional observational study was conducted at the Departments of Obstetrics & Gynaecology and Anatomy, Burdwan Medical College & Hospital, from January to December 2014. Eighty placentas were examined — 40 from PIH mothers with IUGR (Group A) and 40 from PIH mothers without IUGR (Group B). Detailed gross and histopathological examinations were performed, and parameters such as placental weight, volume, surface area, cord insertion type, syncytial knots, villous fibrosis, calcification, atherosis, and fibrin thrombi were recorded. Statistical analysis was carried out using SPSS v20, employing Student's *t*-test and Chi-square test.

Results: Placental weight, volume, surface area, and number of cotyledons were significantly reduced in PIH mothers with IUGR compared to those without IUGR ($p<0.05$). Marginal cord insertion was more common in the IUGR group, while central insertion predominated in non-IUGR cases ($p<0.05$). Birth weight and Apgar scores were significantly lower in PIH with IUGR. Severe hypertension was observed exclusively among PIH mothers with IUGR. Microscopically, diffuse syncytial knots, focal villous fibrosis, moderate calcification, and atherosis were markedly increased in PIH with IUGR (all $p<0.05$). Fibrin thrombi were less frequent in the IUGR group, while hypervascularity differences were not statistically significant.

Conclusion: Placental changes are more pronounced and deleterious in PIH complicated by IUGR compared to PIH without IUGR. Reduced placental weight and vascular lesions such as atherosis, villous fibrosis, and calcification likely contribute to placental insufficiency and subsequent fetal growth restriction. Routine placental examination in hypertensive pregnancies can

provide valuable insights into fetal outcome prediction and maternal management.
Keywords: Placenta, Pregnancy-Induced Hypertension, Intrauterine Growth Restriction, Syncytial Knots, Villous Fibrosis, Calcification, Atherosis.

INTRODUCTION

The placenta is a foeto-maternal organ that connects the developing foetus to the uterine wall to allow nutrient uptake, waste elimination and gas exchange via the mother's blood supply, fight against internal infection and produce hormones to support pregnancy. The placenta was first described by early Egyptian, while the term first coined by Realdus Columbus in 1559. The word placenta comes from the Latin word for flat cake, from Greek plakóenta/plakoúnta, accusative of plakóeis/plakoús, "flat, slab-like", in reference to its round, flat appearance in humans.[1,2]

The placenta is a dynamic organ which is unique in its development and functions. It is the only organ in the body which is derived from two separate individuals, the mother and the foetus.[3,4] Short lived by design, its brief existence ensures survival of human embryo or foetus in the intrauterine environment.[1]

As a foetomaternal organ the placenta develops from two components: the foetal component (Chorion frondosum), which develops from the same blastocyst that forms the foetus, and the maternal component (Decidua basalis), which develops from the maternal uterine tissue.

So, the placenta includes chorionic plate of the foetal side and the basal plate of the maternal side. After fertilization in the ampullary part of the uterine tube fertilized ovum reaches the uterine cavity. Due to the process of cell division morula is formed which soon gets converted into the blastocyst. Due to presence of zona pellucida, the trophoblasts of the blastocyst fail to stick to the uterine wall. After disappearance of zona pellucida, trophoblasts of the blastocyst get attached to the uterine wall (implantation). Due to proteolytic activity, trophoblasts invade and go deeper in the decidua basalis. This is known as interstitial implantation .[5]

Intravillus circulation is foetal while the intervillus circulation is maternal Cytotrophoblast entering the trabeculum fails to penetrate the layer of syncytiotrophoblast and hence does not reach the decidua. However, cytotrophoblast succeeds in coming out of the syncytium at the apex of the villus. The cells of the cytotrophoblast join the cytotrophoblastic arm of adjoining villus and form the trophoblastic shell. Due to the intervention of cytotrophoblastic shell, syncytiotrophoblast gets isolated from the decidua and also divide into the outer and the inner layers.[6,7]

In the present study it was intended to detect the typical histological findings of placenta in PIH with IUGR and also in PIH without IUGR. The findings provide a record and can be used to plan the future care of mother and child. This study is designed to compare the significance or importance of morphological and histological changes of placentas in pregnancies complicated by PIH with IUGR and PIH without IUGR. This study aims to throw light to any gross morphological and structural peculiarities of placenta that might contribute to development of intrauterine growth restriction in mothers with pregnancy induced hypertension.

MATERIALS AND METHODS

It was a hospital based observational study with a cross-sectional design. The research proposal was approved by the Institutional Ethics Committee. Proper informed consents in convenient language were taken from the participating patients. The following study was undertaken from January - 2014 to December – 2014. Among Pregnant patients attending antenatal OPD for antenatal check up & delivered in the Department of Obstetrics & Gynaecology, Burdwan Medical College & Hospital. and Department of Anatomy in Burdwan Medical College.

Inclusion Criteria

1. Pregnant mothers who belong to Primigravida and 2nd gravida were selected in this study.
2. Patients were included in 18 years to 30 years of age groups.
3. Patients having gestational age between 28 weeks to 34 weeks attending at the outpatient department of Burdwan Medical College & Hospital were included and clinically examined for detection of intrauterine growth restriction.
4. Only pregnancies induced hypertensive (PIH) mothers and outcome with or without intrauterine growth restriction (IUGR) were included.

Exclusion Criteria

1. Patients having gestational age less than 28 weeks and more than 34 weeks at the time of recruitment were excluded.
2. Patients suffering from co-morbid condition leading to IUGR such as elderly gravida, diabetes mellitus, anaemia, thrombotic disease, heart disease, chronic renal disease, collagen vascular disease etc. were excluded.
3. Foetuses with congenital anomaly were excluded.
4. Mothers unwilling to participate were not included.
5. Any maternal infection that has effects on placental morphology (eg. syphilis) was excluded.
6. Pregnant mothers suffering from chronic hypertension were also excluded.

7. Pregnant woman with multiple pregnancies and multigravida having 3 or more pregnancies were not included.
8. Pregnant mothers suffering from congenital uterine anomaly were excluded.
9. The patients who were admitted through emergency without proper investigation reports were not included.

The names of mothers, address, age, parity were noted and height and weight were measured. The period of gestation was calculated from first day of last menstrual period (L.M.P). General examination of the patient including pedal oedema was noted. Blood pressure of every patient was measured during antenatal check up. Haemoglobin percentage, blood group and Rh typing, blood sugar (F), urinary albumin were also noted. Presence of respiratory diseases, cardio - vascular diseases or any other systemic diseases was carefully looked for. Height of uterus was measured in centimetre (Gravidogram). Presentation, position and foetal heart sound (FHS) were also noted.

After delivery of placenta it was examined meticulously regarding foetal surface, maternal surfaces, the membrane and the umbilical cord respectively. Presence or absence of blood clot was carefully noted. Then membranes of placenta were trimmed off by a sharp scissors near the margin. Now the whole placenta with umbilical cord was thoroughly rinsed under clean tap water to remove the blood clots. After washing properly the placenta was placed over a blotting paper and was fully covered by it and pressed. This process was repeated for three to four times and the placenta became relatively dry. Now the placenta was ready for macroscopic examination. Before studying the size, shape and weight, the cord was cut very close to the placenta, so that its weight cannot influence the weight of the placenta.

Data Analysis: All datas were analyzed by the using SPSS version 20 and Students t-test and Chi-square test for statistical analysis.

RESULTS

Table 1: Distribution of Macroscopic Features of placenta among PIH mothers with IUGR (Group A) and PIH mothers without IUGR (Group B). (n= 80)

Macroscopic features of placenta	PIH mothers with IUGR (Gr. A) Mean ± (SD)	PIH mothers without IUGR (Gr. B) Mean ± (SD)	P value
Placental Weight (gm)	366.03 ± (12.33)	446.33 ± (30.84)	0.000*
Placental Volume (ml)	214.13 ± (12.57)	258.98 ± (17.77)	0.000*
Placental Surface Area ($cm^{2)}$	154.8 ± (28.17)	214.87 ± (41.25)	0.000*
Placental Maximum Diameter (cm)	15.60 ± (1.63)	18.05 ± (1.81)	0.000*
Placental Minimum Diameter (cm)	12.25 ± (1.46)	14.78 ± (2.12)	0.000*
Placental Thickness (cm)	1.23 ± (0.39)	1.39 ± (0.48)	0.287
Number of Cotyledon	10.58 ± (1.60)	15.33 ± (2.23)	0.000*

Students t-test, *p value<0.05= statistically significant

Table 1 shows that the mean placental weight, volume, surface area, diameter and cotyledon number are significantly low in PIH mothers with IUGR. So, it is observed that all the parameters of PIH mothers with IUGR are statistically significant decreased in comparison to PIH mothers without IUGR except placental thickness, where statistically significant difference is not found.

Table 2: Distribution of Insertion of Umbilical Cord in placenta among PIH mothers with IUGR (Group A) and PIH mothers without IUGR (Group B). (n= 80)

PIH mothers			Insertion of Umbilical cord				Total	p value
			Central	Eccentric	Marginal	Velamentous		
IUGR status	Yes (Gr. A)	Count	20	0	19	1	40	0.001*
		% within IUGR status	50.0%	0.0%	47.5%		100.0%	
	No (Gr. B)	Count	33	3	4		40	
		% within IUGR status	82.5%	7.5%	10.0%		100.0%	
Total		Count	53	3	23	2.5%	80	
		% within IUGR status	66.2%	3.8%	28.8%	0	100.0%	
						0.0%		
						1 1.2%		

Chi-square test, *p value<0.05= statistically significant

In this table it is found that percentage of central insertion of umbilical cord is significantly high in PIH mothers without IUGR. One important finding is that percentage of marginal insertion of umbilical cord is significantly high in PIH mothers with IUGR. Another finding is that eccentric insertion of umbilical cord is absent in PIH mothers with IUGR. Velamentous insertion of umbilical cord is absent in PIH mothers without IUGR. There is statistically significant difference present in different types of insertion of umbilical cord between group A and group B.

Table 3: Distribution of Birth weight, Apgar score, BMI of mother, Gravida, Gestational duration among PIH mothers with IUGR (Group A) and PIH mothers without IUGR (Group B). (n= 80)

	PIH mothers with IUGR (Gr. A) Mean ± (SD)	PIH mothers without IUGR (Gr. B) Mean ± (SD)	p value
Birth Weight (gm)	1995 ± (283.93)	2950 ± (312.35)	0.000*
Apgar Score	6.70 ± (1.07)	7.73 ± (0.96)	0.000*
BMI of Mother	23.78 ± (2.05)	25.01 ± (2.39)	0.015*
Gravida	1.23 ± (0.42)	1.28 ± (0.45)	0.611
Gestational Duration (Days)	260.78 ± (16.38)	273.43 ± (6.23)	0.000*

Students t-test, *p value<0.05= statistically significant

Table 3 shows that all the parameters of group A are statistically significant decreased in comparison to group B except gravida, where statistically significant difference is not found. It is observed that the mean birth weight and Apgar score have significantly lower value in PIH mothers with IUGR. It is also noted that the mean gestational duration and BMI of mothers are significantly less in PIH mothers with IUGR.

Table 4: Distribution of Grading of Hypertension among PIH mothers with IUGR (Group A) and PIH mothers without IUGR (Group B). (n= 80)

PIH mothers			Grading of Hypertension			Total	p value
			Mild	Moderate	Severe		
IUGR	Yes (Gr. A)	Count	5	10	25	40	0.000*
		% within IUGR status	12.5%	25.0%	62.5%	100.0%	
	No (Gr. B)	Count	37	3	0	40	
		% within IUGR status	92.5%	7.5%	0.0%	100.0%	
Total		Count	42	13	25	80	
		% within IUGR status	52.5%	16.2%	31.2%	100.0%	

Chi-square test, *p value<0.05= statistically significant

Table 4 shows that there is statistically significant difference present in different grading of hypertension between group A and group B. In this table the most important finding is that the percentage of severe hypertension is high among PIH mothers with IUGR and this type of hypertension is absolutely absent in PIH mothers without IUGR. It is noted that the percentage of moderate variety hypertension is also significantly high in PIH mothers with IUGR in comparison to PIH mothers without IUGR. Another finding is also noted that the percentage of mild hypertension is very much high among PIH mothers without IUGR.

Table 5: Distribution of number of Syncytial Knots among PIH mothers with IUGR (Group A) and PIH mothers without IUGR (Group B). (n= 80)

PIH mothers			Syncytial Knots			Total	p value
			Absent	Diffuse	Focal		
IUGR status	Yes (Gr. A)	Count	1	30	9	40	0.000*
		% within IUGR status	2.5%	75.0%	22.5%	100.0%	
	No (Gr. B)	Count	17	0	23	40	
		% within IUGR status	42.5 %	0.0%	57.5%	100.0%	
Total		Count	18	30	32	80	
		% within IUGR status	22.5%	37.5%	40.0%	100.0%	

Chi-square test, *p value<0.05= statistically significant

Table 5 shows that the percentage of diffuse variety of syncytial knots are very much high in PIH group of mothers with IUGR and it is absolutely absent in PIH mothers without IUGR. The percentage of focal variety of syncytial knots is high in PIH group of mothers without IUGR in comparison to PIH mothers with IUGR. There is statistically significant

difference present in number of syncytial knots between both groups.

Table 6: Distribution of Fibrosed villi of placenta among PIH mothers with IUGR (Group A) and PIH mothers without IUGR (Group B). (n= 80)

PIH mothers			Fibrosed Villi of placenta		Total	p value
			Absent	Focal		
IUGR status	Yes (Gr. A)	Count	10	30	40	0.000*
		% within IUGR status	25.0%	75.0%	100.0%	
	No (Gr. B)	Count	31	9	40	
		% within IUGR status	77.5%	22.5%	100.0%	
Total		Count	41	39	80	
		% within IUGR status	51.2%	48.8%	100.0%	

Chi-square test, *p value<0.05= statistically significant

In this table it is found that the percentage of focal variety fibrosed villi is significantly high in PIH mothers with IUGR as compared to PIH mothers without IUGR. In most of the cases of PIH mothers without IUGR, fibrosed villi in placenta are absent. Statistically significant difference is present in the distribution of fibrosed villi of placenta between both groups.

Table 7: Distribution of Hypervascularity of placenta among PIH mothers with IUGR (Group A) and PIH mothers without IUGR (Group B). (n= 80)

PIH mothers			Hypervascularity of placenta		Total	p value
			Absent	Focal		
IUGR status	Yes (Gr. A)	Count	14	26		0.115
		% within IUGR status	35.0%	65.0%	100.0%	
	No (Gr. B)	Count	21	19	40	
		% within IUGR status	52.5%	47.5%	100.0%	
Total		Count	35	45	80	
		% within IUGR status	43.8%	56.2%	100.0%	

Chi-square test, *p value<0.05= statistically significant

In this table it is seen that the percentage of focal variety hypervascularity of placenta is significantly high in PIH mothers with IUGR as compared to PIH mothers without IUGR. In 52.5% cases of PIH mothers without IUGR, hypervascularity of placenta are absent. There is no statistically significant difference between two groups.

Table 8: Distribution of Calcification of placenta among PIH mothers with IUGR (Group A) and PIH mothers without IUGR (Group B). (n= 80)

PIH mothers			Calcification of placenta			Total	p value
			Absent	Focal	Moderate		
IUGR status	Yes (Gr. A)	Count	0	13	27	40	0.000*
		% within IUGR status	0.0%	32.5%	67.5%	100.0%	
	No (Gr. B)	Count	14	26	0	40	
		% within IUGR status	35.0%	65.0%	0.0%	100.0%	
Total		Count	14	39	27	80	
		% within IUGR status	17.5%	48.8%	33.8%	100.0%	

Chi-square test, *p value<0.05= statistically significant

From the above table it is seen that the percentage of moderate calcification is very much high among PIH mothers with IUGR but this variety is absent in PIH mothers without IUGR. It is also revealed that the percentage of focal calcification is significantly high in PIH mothers without IUGR as compared to PIH mothers with IUGR. There is statistically significant difference present in the distribution of calcification of placenta between both groups.

Table 9: Distribution of Atherosis of placenta among PIH mothers with IUGR (Group A) and PIH mothers without IUGR (Group B). (n= 80)

PIH mothers			Atherosis of placenta		Total	p value
			Focal	Moderate		
IUGR	Yes (Gr. A) status	Count	11	29	40	0.000*
		% within IUGR status	27.5%	72.5%	100.0%	
	No (Gr. B) status	Count	32	8	40	
		% within IUGR status	80.0%	20.0%	100.0%	
Total		Count	43	37	80	100.0%
		% within IUGR status	53.8%	46.2%		

Chi-square test, *p value<0.05= statistically significant

In this table it is observed that the percentage of moderate atherosis of placenta is significantly high among PIH mothers with IUGR as compared to PIH mothers without IUGR. It is also noted that the percentage of focal atherosis of placenta is significantly high in PIH mothers without IUGR in comparison to PIH mothers with IUGR. There is statistically significant difference present in the distribution of atherosis of placenta between both groups.

Table 10: Distribution of Fibrin thrombi in vessels of placenta among PIH mothers with IUGR (Group A) and PIH mothers without IUGR (Group B). (n= 80)

PIH mothers			Fibrin thrombi in vessels of placenta		Total	p value
			Focal	Absent		
IUGR	Yes (Gr. A) status	Count	11	29	40	0.006*
		% within IUGR status	27.5%	72.5%	100.0%	
	No (Gr. B) status	Count	23	17	40	
		% within IUGR status	57.5%	42.5%	100.0%	
Total		Count			80	
		% within IUGR status			100.0%	

Chi-square test, *p value<0.05= statistically significant

Table 14 shows that the percentage of focal variety of fibrin thrombi in vessels of placenta is significantly low among PIH mothers with IUGR in comparison to PIH mothers without IUGR. It is also noted that in most of the cases of PIH mothers with IUGR, fibrin thrombi in vessels of placenta is absent. Statistically significant difference is present in the distribution of fibrin thrombi in vessels of placenta between both groups.

DISCUSSION

In the present study the placental weight is low in both groups which are consistent with the study Udainia A et al (2004),[8] found that placental weight is reduced in preeclamptic cases. Significant decrease in placental weight was seen in intrauterine growth restriction which was observed in the study by S Kotgirwar et al (2011),[9] study. R Sudha et al (2012),[10] found that significant reduction of placental weight in hypertensive pregnancies was observed when compared with normotensive pregnancies except for gestational diabetes mellitus. In the study by M Hemalatha et al (2014),[11] it was seen that majority of placentas of IUGR pregnancies weighed between 300-400 grams and 20% of placentas of IUGR group weighed less than 300 grams and another 20% of placentas weighed more than 400 grams. In the present study mean placental weight of group A (PIH mothers with IUGR) is 366.03. So, this finding corroborates with this present study.

Mean and standard deviation of placental volume of group A (PIH mothers with IUGR) are 214.13 ± 12.57 and of group B (PIH mothers without IUGR) are 258.98 ± 17.77 respectively which is statistically significant. In the study by S Majumdar et al

(2005),[12] mean placental volume was significantly lower values (p>0.01) in the hypertensive mothers. In the present study the placental volume is low in both groups which is consistent with the study by S Majumdar et al but, these findings are much less in PIH mothers with IUGR. Mean and standard deviation of placental surface area of PIH mothers with IUGR are 154.8 ± 28.17 and of PIH mothers without IUGR are 214.87 ± 41.25 respectively which is statistically significant. Meyur R et al (2012),[13] also reported that the surface area of placenta was lower in eclamptic mothers than normotensive mothers but this was insignificant. The decrease in mean placental area of live born and still born babies of eclamptic mothers as compared to normotensives was significant in their study which suggests that placental growth and development are hindered by mechanisms related to eclampsia.

In Table 3, mean and standard deviation of birth weight of baby of PIH mothers with IUGR are 1995 grams ± 283.93 and of PIH mothers without IUGR are 2950 grams ± 312.35 respectively. These results are statistically significant between both groups. In the present study mean birth weight of baby is significantly low in PIH mothers with IUGR in comparison to PIH mothers without IUGR. Shweta Anand et al (2011),[14] revealed that the incidence of small for gestational age (SGA) was four times more in the hypertensive group; outcome in terms of morbidity and mortality was also statistically significant. They also observed that 57.1% babies were preterm and intra-uterine growth restricted (IUGR) in the hypertensive group of mothers, 71% of them being asymmetrical.

Mean and standard deviation of Apgar score of group A (PIH mothers with IUGR) are 6.70 ± 1.07 and of group B (PIH mothers without IUGR) are 7.73 ± 0.96 respectively which is statistically significant. In the present study value of Apgar score is significantly less in group A than group B. S Majumdar et al (2005),[12] study, showed that Apgar score was below 7 in 15 babies of hypertensive group of mothers and in two such babies it was 2 and 3 only. These findings are very much similar with group A of the present study. Count and percentage of mild, moderate and severe hypertension of PIH mothers with IUGR are (5, 10, 25), (12.5%, 25%, 62.5%) respectively and of PIH mothers without IUGR) are (37, 3, 0), (92.5%, 7.5%, 0%) respectively. In the present study it is noted that most of the cases of PIH mothers with IUGR babies are caused by severe hypertension. A. Y Raghavendra. et al (2014),[15] showed that the birth weight of newborn was low with increasing grading of hypertension in PIH mothers. In this study, statistically significant difference is present in different grading of hypertension between both groups.

Count and percentage of absent, diffuse and focal varieties of syncytial knots of group A mothers (PIH with IUGR) are (1, 30, 9), (2.5%, 75%, 22.5%) respectively and of group B mothers (PIH without IUGR) are (17, 0, 23), (42.5%, 0%, 57.5%) respectively. It is seen that diffuse syncytial knots are present in 75% cases of group A and absent in group B. In the present study statistically significant difference is present in distribution of number of syncytial knots in placenta between group A and group B. Count and percentage of absent and focal varieties of hypervascularity of placenta of PIH mothers with IUGR are (14, 26), (35%, 65%) respectively and of PIH mothers without IUGR are (21, 19), (52.5%, 47.5%) respectively. In the present study, the distribution of hypervascularity of placenta between two groups is statistically not significant. In the study by S Majumdar et al (2005),[12] the stromal and villous histopathological changes in the placenta like stromal fibrosis, medial coat proliferation of medium sized blood vessels per low power field were significant (p>0.01) in the hypertensive group than in the normotensive group which might be the cause or effect of hypertension. Count and percentage of focal and moderate varieties of atherosis of placenta of PIH mothers with IUGR are (11, 29), (27.5%, 72.5%) respectively and of PIH mothers without IUGR are (32, 8), (80%, 20%) respectively. Statistically significant difference is also present in this study in the distribution of atherosis between two study groups. In Table 14, count and percentage of focal and absent varieties of fibrin thrombi in vessels of placenta of group A mothers (PIH with IUGR) are (11, 29), (27.5%, 72.5%) respectively and of group B mothers (PIH without IUGR) are (23, 17), (57.5%, 42.5%) respectively. Statistically significant difference is present in the distribution of fibrin thrombi in vessels of placenta between two study groups in the present study. Increased placental perivillous fibrinoid deposition (16.7%) was present in IUGR mothers which were observed by S Kotgirwar et al (2011).[9]

CONCLUSION

From the present study this can be concluded that PIH mothers with IUGR present with significantly smaller placenta as evident by almost all the macroscopic parameters of placenta (weight, volume, surface area, cotyledon number, maximum and minimum diameter), than PIH mothers without IUGR. Similarly, outcome of the neonates was significantly poor in PIH mothers with IUGR (as shown by significantly low mean birth weight of baby and Apgar score) than PIH mothers without IUGR. PIH mothers with lower BMI and smaller gestational duration were complicated with IUGR, though order of pregnancy did not show any statistically significant difference between the study groups.

REFERENCES

1. Emin M, Anna I, B, Susan J. The placenta: transcriptional, epigenetic, and physiological integration during development. J Clin Invest. 2010; 120(4): p. 1016-25.

2. Huppertz B, Kingdom JCP. The placenta and fetal membranes. In editor. IED, editor. Dew hurts Text book of Gynecology and obstetrics. 7th ed. London: Blackwell publisher; 2007. p. 19-25.
3. Ragunath G, Lakshmi V, Shenoy V. A study on the Morphology and Morphometry of the Human Placenta and its Clinical Relevance in a population in Tamilnadu. Journal of Clinical and Diagnostic Research. 2011 Apr; 5(2): p. 282-286.
4. Devi C, Neelam S, Raghupathy N. The Histological Findings in Human Placenta at Different Gestational Ages. IOSR-JDMS. 2013; 6(1): p. 29-31.
5. In Kadasne's text book of embryology. First edition ed.: ISBN 978-93-5025-152-2; 2011. p. 83.
6. Anorlu R, Nnamdi C, Odum C. Risk factors for pre-eclampsia in logos, Nigeria. ANZ Jf Obstet Gynaecol. 2005; 45: p. 78-82.
7. Gunnar K, Bjorg E, Sven G, Magnar U, Perberg S. Maternal deaths in developing countries: A preventable tragedy. Norsk Epidemiologi. 2005; 15(2): p. 141-149.
8. Udainia A, Bhagwat S, Mehta C. Relation between Placental Surface Area Infarction and Foetal Distress in Pregnancy Induced Hypertension with its Clinical Relevance. J.Anat.Soc.India. 2004; 53(1): p. 27-30.
9. Kotgirwar S, Ambiye M, Athavale S, Gupta V, Trivedi S. Study of Gross and Histological features of placenta in intrauterine growth retardation. J. Anat. Soc. India. 2011; 60(1): p. 37-40.
10. Sudha R, Sivakumar V, Jebakani CF. study of shape of placenta and its relation to placental weight in normal and complicated pregnancies. National Journal of Basic Medical Sciences. 2012; 2(4).
11. Hemalatha M, Phani MK, Jankai M, Dudala SR. Histopathological evaluation of placentas in IUGR pregnancies. ASIAN PACIFIC JOURNAL OF HEALTH SCIENCES. 2014; 1(4): p. 566-599.
12. Majumdar S, Dasgupta H, Bhattacharya A. A Study of Placenta in Normal and Hypertensive Pregnancies. J Anat Soc India. 2005; 54(2): p. 1-9.
13. Meyur R, Sadhu A, Mitra B, Ghosal AK, Biswas A, Patra DK. Morphometric and Macroscopic Observations of Human Placenta in Eclampsia. Indian Medical Gazette. 2012 November.
14. Anand S, Anand K. Perinatal Outcome in Growth Retarded Babies Born to Normotensive and Hypertensive Mothers: A Prospective Study. People's Journal of Scientific Research. 2012 Jan; 5(1).
15. aghavendra AY, Vinay KV, Veena P. A STUDY OF PLACENTAL WEIGHT AND FETAL OUTCOME IN DIFFERENT GRADES OF PREGNANCY INDUCED HYPERTENSION. International Journal of Anatomy and Research. 2014; 2(4): p. 625-29.