ORIGINAL ARTICLE

Gross Morphology of Placenta in Preeclampsia

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Abstract:

Background: Preeclampsia is a pregnancy peculiar disorder with an incidence of 5-7%. It leads to placental insufficiency that results in fetal and maternal morbidity and mortality. Its etiology however remains unclear. It develops during pregnancy and subsides after delivery, implicating the placenta as the main culprit. As placenta is the mirror of maternal and foetal status, it reflects the changes due to maternal hypertension.

Objectives: This study intends to compare the changes in gross morphology of placenta in preeclampsia with those of normal placenta.

Materials & Methods: 25 normal and 50 placentae from preeclamptic patients were obtained from Lady Aitchison hospital Lahore. The placentae were distributed into four group on basis of diastolic blood pressure ranging from 90-130 mm Hg. Weight, diameter, thickness & volume of placentae were measured. Pathological findings like infarction & calcification were also noted.

Results: The study showed that the diameter, weight and volume of placentae reduced significantly with a rise in blood pressure in all preeclamptic groups. The thickness of the placentae decreased significantly in severe preeclamptic group D (diastolic B.P 116-130 mm Hg) only. Infarction of placentae was present in 16.6% cases in Group C (diastolic B.P 101-115) & 20% cases in Group D. calcification was present in 12.5% in Group C & 13.3% cases in Group D.

Conclusions: Preeclampsia significantly affects the placenta by reducing its weight, diameter, thickness and volume. Pathological changes appear in placenta with mounting diastolic blood pressure

Key words: *preeclampsia, placenta, diastolic blood pressure*

Introduction

Pre-eclampsia is a human pregnancy-specific disorder with an incidence of 2-8%[1] worldwide. The incidence being 3 to 7 % in nulliparous and 1 to 3 % in multiparous. It is directly associated with 10 to 15 % of maternal deaths[2]. It is defined as a rise of blood pressure with proteinuria with or without edema that develops after 20 weeks of gestation. The diagnostic criteria for preeclampsia is systolic blood pressure of >140mm Hg or diastolic blood pressure of > 90mm Hg measured at rest on two different occasions at least six hours apart accompanied by proteinuria of > 0.3 g in a 24-hour urine specimen[3].

Preeclampsia remits with the delivery of the placenta. Placenta is a fetomaternal organ essential for maintenance of pregnancy and normal growth and development of fetus[4]. Placenta undergoes changes in its shape, weight, volume, histological structure and function throughout pregnancy to

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support the developing fetus[5]. The embryo originally implants in maternal decidua (pregnant endometrium). The maternal blood vessels are invaded by the blastocyst derived trophoblast cells resulting in dramatic modification of these vessels. The spiral arterioles, which are normally small muscular arteries, dilate at their endometrial end. The dilated portions of the vessels show disruption of their endothelium, tunica media and internal elastic lamina. This modification extends to the inner third of the myometrium. As a result in this portion of the spiral arterioles becoming modified to into flaccid large bore vessels that fail to constrict in response to neural or humoral stimuli[6]. No such changes occur in preeclampsia. In preeclampsia some arterioles undergo remodeling at their endometrial segments but the changes do not extend as far as the myometrial part of the vessel. 30-50% of these arterioles in placental bed do not undergo trophoblast remodeling[7].

Maternal vasospasm leads to fetal hypoxia. Normal fetal growth depends on the proper development and function of the placenta. Preeclampsia has its effect on the fetus i.e. produces growth restriction, prematurity etc. hence contributing largely to perinatal mortality and morbidity[8].

The study was carried out to determine the effects of preeclampsia on the gross morphology of the placenta.

Materials and Methods

Twenty five placentae from women with normal pregnancies and fifty placentae from women suffering from preeclampsia were obtained from operation labour room/ operation theater of Lady Aitchison hospital Lahore. These women were examined in the emergency. Their medical history i.e. history of past illness, previous pregnancies, and history of treatment was recorded on a proforma. These patients were checked for blood sugar, blood urea, creatinine, haemoglobin and proteinurea. Patients with diastolic blood pressure between 70-85mm Hg without proteinurea and oedema were allocated the normal pregnancy group & served as control. Patients with diastolic blood pressure ranging between 86-130 mm Hg, oedema and protienuera were included in the preeclamptic group. Primigravida & multigravida of all age groups & of gestational age more than 28 weeks were included in the study. Patients on medications or those who were suffering from diseases e.g. diabetes mellitus, hypertension before pregnancy, ischemic heart disease, drug addicts etc. were excluded from the study. Blood pressures of these women were checked half an hour after the arrival in the emergency and later after every half an hour until delivery. Mean of these readings was calculated and patients were divided into four groups. Group A, the control group had diastolic blood pressure ranging from 70-85 mm Hg, Group B had a range of B.P. between 86-100 mm Hg, Group C between 101-115 mm Hg and Group D had a range of B.P. between 116-130 mm Hg. After delivery, placentae were washed & mopped to remove clotted blood. The umbilical cord was removed 2 cm from the surface of the placenta. The membranes were also removed

Each placenta was examined for its colour & presence of any pathological lesion such as infarcts & calcification.

Weight of each of these placenta was measured with the help of weighing scale; placental diameters were recorded along two axes at right angles to each other with the help of measuring tape & mean of the two readings was calculated. Thickness of the placenta was taken at the centre with a long needle inserted in it. The embedded part of the needle was measured by a scale. Volume of the placentae was calculated from their diameter & thickness using the formula $\pi r^2 \times h$ (π=3.14).

Statistical analysis was carried out using Statistical Package for Social Sciences (SPSS). Chi Square test was applied to analyze qualitative data. ANOVA & Post hoc Scheff test were applied for comparison between different groups.

Results:

Out of the 50 preeclamptic placentae 11 (22%)

fell into Group B, 24 (48%) in Group C and 15, (30%) in Group D.

The placentae in Group A were bright pink in colour. The fetal membranes were shiny, transparent and freely mobile over placentae. No pathological lesions were noted in placentae in this group.

The placentae in Group B were pink and shiny to look at. The fetal membranes were transparent and moved freely over the placentae. No gross pathological lesions were noted in this group.

The placentae in Group C were dark red in colour. They lacked luster. The fetal membranes were less shiny compared to the other two groups but they were transparent and freely mobile over the placentae. Four out of the 24 placentae (16.6%) had localized infarcts which appeared as dark, firm, irregular areas sharply demarcated from the rest of the tissues. Areas of calcification was observed in 3 out of 24 placentae (12.5%).

The placentae in Group D were bluish red in colour. They lacked luster. The membranes were freely mobile over the placentae. In 3 out of 15 (20%) placentae there were localized infarcts, calcification was observed in two placentae (13.3%).

Among both cases and controls central cord insertion was observed most commonly (83.5%). Out of 25 cases in control group A, 23 (92%) had central insertion of umbilical cord. Similar findings were observed in 9 out of 11 (81.8%) placentae in preeclamptic group B, 21 out of 24 (87.5%) in group C&11 out of 15 (73%) in group D. Battledore insertion was observed in one placentae (4%) in Control group A, one (9%) in preeclamptic Group B, two (8.3%) in group C & two (18.1%) in Group D. vilamentous insertion of the cord was noted in one (4%) placenta in Control group A, one (9%) in group B, one (4%) in group C & two (18%) in group D.

As shown in Table 1&2 morphometric features of placenta like its weight, volume, thickness, diameter values are less in severe preeclamptic groups C & D compared to mild Preeclamptic group B and controls. Findings became statistically significant as the severity of the disease increased (Table 3).

Table 1: *Results of Gross Parameters in Control and Preeclemptic Placentae*

Groups	Diastolic Blood Pressure (mm Hg)	Range of Weight (gms.)	Range of Diameter (cm.)	Range of Thickness (cm.)
A (n= 25)	70-85	Min. 485.50 Max. 710	15 18.6	2.3 3.90
B (n= 11)	86-100	Min. 490 Max.576.40	11.75 15.45	2.8 3.6
C (n= 24)	101-115	Min. 312.40 Max.528.20	7.50 13.60	2.3 3.4
D (n= 15)	116- 130	Min. 340 Max.518	9 13.8	1.6 3.6

Table 2: *Mean Values of Gross Parameters In Control And Preeclemptic Placentae*

Groups	Diastolic Blood Pressure (mm Hg)	MEAN WEIGHT (gms.)	p value	MEAN DIAMETER (cm.)	p value	MEAN THICKNESS (cm.)	p value	MEAN VOLUME (cm^3)	p value
A (n= 25)	70-85	588.3 ± 63.3		16.5± 1.09		3.3 ± 0.51		705 ± 3.41	
B (n= 11)	86-100	539.5 ± 23.4	.057	14.9 ± 1.21	.000	3.1 ± 0.26	.524	534.7 ± 3.55	.000
C (n= 24)	101-115	462.9 ± 54.6	.000	11.6 ± 1.60	.000	2.9 ± 0.29	.326	306.1 ± 3.40	.000
D (n= 15)	116- 130	424. ± 67.2	.000	11.0 ± 1.22	.000	2.62. ± 0.64	.04	249 ± 3.62	.000

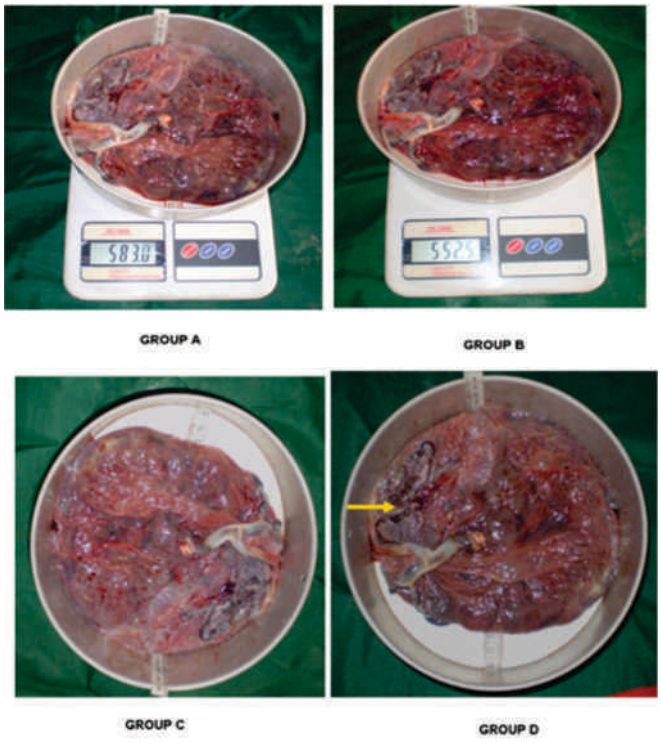

Fig1: *Placentae showing weight, diameter, thickness in all groups*

Discussion:

Preeclampsia adversely affects the morphology of placenta. The present study was carried out to assess the morphometric variations in weight, diameter, thickness and volume of placentae in preeclemptic patients.

In this study, the mean weight of the placentae in females with a mean diastolic B.P. 90 mm Hg is 537.5 gms as compared to 588 gms in control group A (Table 2). The weight significantly decreased to 463 gms and 424 gms with mean diastolic blood pressure 100 mm Hg of 110 mm Hg. respectively. Dokras et al[9] mentions a mean placental weight of 435.6 gms. Similar findings were also observed by other workers10. Placental weight decreases with mounting hypertension due to utero-placental vascular insufficiency.

The increase in diastolic blood pressure is also accompanied by a progressive decrease in placental diameter in all preeclamptic groups compared to normal (Table 2). Sultana[11] in her study have documented a mean diameter of 11.5 cm in severe preeclampsia. We have recorded a diameter of 11.6 cm in patients with mean diastolic blood pressure of 100 mm Hg and 11 cm in patients with 110 mm Hg as compare to normal i.e. 15-17 cm. Shevade et al[12], Maimoona[13], Agarwal[14] have also observed similar findings.

Changes in thickness of placentae in different preeclamptic groups were not well marked except for severe preeclamptic cases (Table 2). Ashfaq[15] and Sultana[11] have mentioned a mean thickness of 2.7 cm and 3.1 cm respectively in patients with higher diastolic blood pressures. Our findings corroborated with those of Salmani[16], Keche[17] & Dadhich[18] who have mentioned a thickness of 2.7, 1.57 & 2.02 cm respectively. The thickness of placentae is attributed to chorionic villi which undergoes compensatory hyperplasia due to hypoxia. Apoptosis also increases in hypoxia. The two processes i.e. apoptosis and compensatory hyper-plasia run parallel and balance each other in such a way that no significant difference in thickness of placentae in preeclampsia was found[15]. With a diastolic blood pressure between 116-130 mm Hg, however there is a failure of these compensatory changes with apoptosis overriding ischemia resulting in decreased thickness of placentae[19].

The volume of placentae as calculated from the above mentioned results of diameter and thickness was reduced significantly in all the preeclamptic groups (Table 2). Bhavina[20] mentions a volume of 375.9 gms and Pomorski et al[21] have recorded a significant decrease in volume with a 'p' value <0.05 in patients with diastolic blood pressure more than 100 mm Hg., Singh[10] & Maimoona[13] observed similar findings in their studies.

In this study, placental infarcts were present in 17% placentae with a range of diastolic blood pressure 101- 115 mm Hg and in 25% placentae with diastolic blood pressure ranging between 116-130 mm Hg. In previous studies 28% and 35% of placentae showed infarcts[20,22]. A significant increase in incidence of infarction was reported in a study (50% in mild PIH, 70% in severe PIH)[16]. Placental

infarct is the result of an occlusion of utero-placental artery. The inter-villous flow ceases, the inter-villous space collapses and the villi become compressed and undergo ischemic necrosis which is visible as a dark, firm, irregular area sharply demarcated from the rest of the tissue[19].

Different studies have recorded their observation on calcification of placentae. Higher number of hypertensive placentae showed calcification in a study[23] but another study[14] reported 62.1% normal placentae with calcification. On the other hand, frequency of calcification was same in control as well as hypertensive group in yet another study[24]. Navbir[25] observed placental calcification in 73.34% cases of preeclamptic pregnancies in his study. However, 10% of the placentae from the control group also showed calcification in the same study. Thus, placental calcification was seen more commonly in placentae from hypertensive patients. In the present study areas of calcification were observed in 12.5% placentae in group C & 13.3% placentae in Group D

Conclusions

It is concluded that preeclampsia has a definite adverse influence on the morphology of placenta. The weight, diameter, thickness & volume decreased with mounting diastolic blood pressure. Pathological findings like infarction & calcification appeared as preeclampsia aggravated.

This study will help the obstetrician to know the level of hypertension at which the deleterious changes appear in the placentae and thence will stress the need for early diagnosis and treatment.

References

1. Akhlaq M, Nagi A. H, Yousaf A.W. Placental morphology in pre-eclampsia and eclampsia and the likely role of NK cells. Indian J Pathol Microbiol. 2012;55:17-21
2. Uzan J, Carbonnel M, Piconne O, Asmar R and Ayoubi J.M. Preeclampsia: Pathophysiology, Diagnosis and management. Vasc Health Risk Manag. 2011;7:467-474.
3. Subramaniam V. Seasonal variation in the incidence of preeclampsia and eclampsia in tropical climatic conditions. BMC Women's Health. 2007;7: 1-18.
4. Udaina A, Bhagwat S.S, Mehta C.D. Relation between placental surface area, infarction and foetal distress in pregnancy induced hypertension with its clinical relevance. J.Anat. Soc . India. 2004; 53(1):27-30
5. Roberts J.M, Gammill H.S. Preeclampsia: recent insights. Hypertension. 2005;46(6):1243–9.
6. Pijnenborg R, Vercruysse L, Hanssens A. The uterine spiral arteries in human pregnancy: Facts and controversies. Placenta. 2006; 27(9–10): 939–58.
7. Roberts J. M and Escudero C. The placenta in preeclampsia. Pregnancy Hypertens. 2012 Apr 1; 2(2): 72–83.
8. Vijayalakshmi B, Kittali S. Morphological changes of placenta in cases of pre-eclampsia and perinatal outcome. Int J Sci Stud 2015;3(5):137-142.
9. Dokras A , Hoffmann D. S , Eastvold J.S , Kienzle M.F , Gruman L.M , Kirby P.A , Weiss R.M , and Davisson R.L. Severe Feto-Placental Abnormalities Precede the Onset of Hypertension and Proteinuria in a Mouse Model of Preeclampsia. Biology of Reproduction. 2006; 75: 899–907
10. Singh S, Gugapriya T S. A Cross Sectional Morphometric Study of Hypertensive with Normal Placentae and its Correlation with Fetal Outcome. Int J Anat Res 2014;2(2):437-42.
11. Sultana S, Hossain GA, Rahman H, Hasan N, Mannan S, Zannat S. Gross morphometry of human placenta in eclampsia. Mymensingh Med J. 2006 Jan;15(1):10-4
12. Shevade S, Arole V, Bharambe V, Paranjape V. Placental Morphology and Fetal Outcome in Preeclampsia And Normotensive Pregnancies. IOSR Journal of Dental and Medical Sciences. 2015; 14(4): 11-15.
13. Maimoona A, Rekha D.G. Study of placental changes in pregnancy induced hypertension. International journal of Reprod Contracept Obstet Gynecol. 2013;2(4):524-527
14. Agarwal GC, Jain A. Morphological study of placenta in normal and hypertensive pregnancies. 2015;2(5): 121-128.
15. Ashfaq M, Janjua M.Z, Channa M.A. Effect of Festational Diabetes and Maternal Hypertension on gross Morphology of Placenta. J Ayub Med Coll

Abbottabad. 2005 Jan-Mar;17(1):44-7

16. Salmani D, Purushothaman S, Somashekara S.C, Gnanagurudasan E, Sumangaladevi K, Harikishan R, et al. Study of structural changes in placenta in pregnancy-induced hypertension. J Nat Sc Biol Med. 2014;5:352-5.
17. Keche H.A, Keche A.S. Morphometric differentiation between placenta in PIH and normal pregnancy. Int J Med Sci Public Health. 2015;4: 250-255
18. Dadhich A et.al. Study of effect of eclampsia and chronic hypertension on gross morphology of placenta Int J Biol Med Res. 2012; 3(2):1771-1773
19. Ahmed M, Daver R.G. Study of placental changes in pregnancy induced hypertension. Int J Reprod Contracept Obstet Gynecol., 2013; 2(4): 524-527
20. Bhavina K, Sundara Pandian S, Priya G. A study on morphology of placenta and umbilical cord in Hypertensive pregnancy with and without Proteinuria. International Journal of Biomedical Research. 2013;04(01):50-54
21. Pomorski M, Zimmer M, Florjanski J, Michniewicz J, Wiatrowski A, Fuchs T, Milnerowicz-Nabzdyk E. Comparative analysis of placental vasculature and placental volume in normal and IUGR pregnancies with the use of three-dimensional Power Doppler. Arch Gynecol Obstet (2012); 285:331–337
22. Aparna Narsimha, Ds Vasudeva. Soectrum of changes in placenta in toxemia of pregnancy. Indian Journal of Pathology and Microbiology. 2011;54 (1):15-20.
23. Roberts D.J. Placental Pathology: A survival guide. Arch Pathology Lab Med. 2008; 132: 641-51.
24. Ghodke S, Dharwadkar S. Hypertensive disorders affecting the morphometry of placenta. J Sci Soc. 2012;39:17-8.
25. Navbir P. Placental Morphology and its Co-relation with foetal Out-come in Pregnancy-Induced Hypertension. International Journal of Basic and Applied Medical Sciences. 2012;2(3):120-125

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