A MORPHOMETRIC COMPARATIVE STUDY OF PLACENTA IN NORMAL AND PREECLAMPSIA PREGNANCIES DELIVERING AT 36 TO 38 WEEKS OF GESTATION AND ITS IMPACT ON BIRTH WEIGHT

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ABSTRACT: BACKGROUND: Placenta is a remarkable organ which functions as a vital organ for the developing fetus. Evaluation of placenta conveys the intrauterine environment of the fetus. Examination placenta in many diseases, including preeclampsia, and its comparison to placenta in normal pregnancies reveals placental pathology as a cause of fetal growth disorders. OBJECTIVE OF THE STUDY: To correlate effect of preeclampsia on placental development and subsequently its effect on fetal growth. MATERIALS AND METHODS: Across-sectional study was conducted in a tertiary care center. The placentas from pregnancy delivered between 36 to 38 weeks were included in the study. Morphometric measurements of 100 placentas from pregnancies that had preeclampsia were compared with 100 placentas from pregnancies that were normotensive. The placental morphometry was correlated with fetal birth weight. OBSERVATIONS AND RESULTS: Study showed that the placental development was significantly affected with relation to shape in preeclampsia group. 97% were discoid in normotensive group whereas in preeclampsia group it was 88%. Attachment of cord were more eccentric in preeclampsia than normotensive group (26% vs. 19%) but the difference was not statistically significant (p >0.05). The mean number of cotyledons, mean placental diameter, mean placental weight and mean placental surface area were significantly (p<0.0001) reduced in preeclampsia group when compared to normotensive group (14.07+/- 2.04 and 17.04+/- 2.06, 15.31+/-1.08cm and 16.00+/-0.75 cm,357.45+/-61.66 g and 423.11+/-45.85g, 183.79+/-25.94 cm²and 201.40+/-19.19cm² respectively). The fetal weights were also significantly (p<0.0001) affected in preeclampsia group (2360+/-0.40 g) as compared to in normal cases (2590+/-0.29 g). The correlation coefficient of number of cotyledons, placental diameter, placental weight and surface area were calculated with respect to birth weight was found to be significant in both study group and control group. CONCLUSION: The placenta is significantly affected in preeclampsia in its development leading to adverse outcome on fetal intra uterine growth. KEYWORDS: Placenta, Preeclampsia, Normotensive, Morphometric comparison, Correlation coefficient.

INTRODUCTION: Human placenta is uniquely intriguing and functions as pulmonary, hepatic and renal organ for the fetus. These are accomplished by placental anatomical association with maternal interface.¹

Proper development of placenta and fetus requires adequate establishment of uteroplacental circulation. One of the major transition that occurs is the conversion of circulation in spiral arteries from high resistance and low flow to high flow, low resistance circulation. The endovascular trophoblasts are vital to these changes that occur in both the segments of spiral artery that lie in decidua and myometrium. This occurs in 2 stages. The first stage of invasion occurs in first trimester.
and involves the decidual component of spiral artery and second wave occurs in 2nd trimester which involves the myometrial component and thus converting the whole length of spiral artery into a funnel like channel.

In contrast to normal pregnancy, pregnancies complicated by preeclampsia or fetal growth restriction demonstrate inadequate maternal vascular response to placentation. The vascular changes in preeclampsia are limited only to decidual component of spiral artery, thereby leaving the myometrial components of spiral artery with intact musculoelastic architecture which are responsive to vasopressor effects of hormones. Many complication occur as a result of incomplete invasions leading to defective uteroplacental circulation. In mild cases it may result in preeclampsia, fetal growth restriction or combination of both. In severe cases may lead to early abortions.

Preeclampsia is a form of hypertension unique to human pregnancy with heterogeneous etiology and pathogenesis which manifest as maternal and fetal syndrome. The placenta is, like other organs, is effected by vasospam of uterine and spiral arteries in preeclamsia. Preeclamsia is associated with developmental disorder of placenta and fetus. The examination of placenta gives valuable information about state of fetal well-being in utero.

This study was done to emphasize the importance of examination of placenta after delivery and its clinical relevance.

MATERIALS AND METHODS: After obtaining Institutional Ethical committee clearance and informed consent from the woman who were included in this study, a cross sectional study was conducted over a period of 18 months at Department of OBG of tertiary care teaching hospital.

We studied 100 placentas whose pregnancies were complicated by preeclampsia and delivered between 36-38 weeks and 100 placentas from normal pregnancies who also delivered between 36-38 weeks. Examination of placenta was done as per methodology of Joseph Yetter. The data collected was entered in Microsoft excel 2007 and analyzed using Epi info 3.4.3. Descriptive statistics such as Mean, SD and proportions was calculated. Z test for difference between means and correlation coefficient value for continuous variables was calculated. P value <0.05 was considered as statistically significant.

RESULTS: The placental shape in normotensive group were discoid in 97 cases compared to 88 in preeclampsia group. Chi square value was 5.83 and p value <0.015. Attachment of cord were more eccentric in preeclampsia than normotensive group, 26 Vs. 19. Chi square value was 2.13 and p value was 0.34. The mean number of cotyledons, mean placental weight, mean placental diameter, and mean placental surface area were significantly (p <0.0001) reduced in preeclampsia group when compared to normotensive group (14.07+/- 2.04 and 17.04+/-.206, 357.45+/-.61.66 g and 423.11+/- 45.85 g, 15.31+/-1.08 cm and 16.00+/-0.75cm, and 201.40+/-19.19cm² respectively) (Table1). The fetal weights (Table1) were also significantly (p<0.0001) affected in preeclampsia group (2360+/-0.40g) as compared to normal cases (2590+/-.029g). The correlation coefficient of number of cotyledons, placental diameter, placental weight and surface area were calculated with respect to birth weight was found to be significant in both pre-eclamptic group and normotensive group (Table 2).

DISCUSSION: Improper placentation is regarded as one of the main etiologies for preeclampsia. The normal placentation involves 2 stages of spiral artery invasion by endotrophoblast.
The absence of 2 stage of spiral arterial invasion causes dysfunctional perfusion of intervillous spaces with oxidative and hemodynamic stress. The damaged placenta releases excessive pro inflammatory and angiogenic factors into maternal circulation. However now it has been observed as a six stage disease by a recent study. It has now become more clear that the antecedents to poor placentation is immunological in origin. The morphometric measurements which are used as parameters to assess placental development.

The placental shape in normotensive group were more normal, being discoid, than in preeclampsia group 97 versus 88 in the recent study. Chi square value and p value were significant. The attachment of cord in relation to the fetal surface of placenta were found to be more eccentric type in preeclampsia cases than normal cases 26 versus 19 but P value was 0.34 and Chi square vale was 2.13 and not statistically significant. However some studies found statistical significance.

The preeclampsia has significant effect on uteroplacental circulation and subsequently fetal circulation, leading to underdevelopment of placenta and fetus. Preeclampsia not only affects placental weight but also its dimensions. The reduction in number of cotyledons, placental weight, diameter, and surface area in the present study is consistent with some of the previous studies (Table 3).

The explanation to negative effect of preeclampsia to placental development can been seen in microscopic and ultramicroscopic structural study of placenta. Microscopic changes are nonspecific and not necessary for diagnosis but if found in large numbers, they are suggestive of preeclampsia. The characteristic lesions are vascular lesion which atherosis of decidual spiral arteries, thrombosis, infarction and retro placental hematoma. Other changes included syncitial knots, cytotrophoblastic proliferation, thickening trophoblastic basement membrane, obliterated enlarged endothelial cells in villous capillaries, hyalinization, and calcification. There is reduction of total volume of parenchyma and villous surface area. A pilot study done to compare morphometry of preeclamptic and normotensive placenta showed that density of placental villi and carrying capacity of stem villi decreases and thickness of stem villi arterial walls and extent of fibrosis increased in preeclampsia placentalae.

However recent studies found out that the placenta was mostly normal or larger with no fetal growth retardation in late onset preeclampsia but was hypoplastic and associated with fetal growth retardation when preeclampsia was early onset, suggesting probably early onset preeclampsia is a placental disorder whereas late onset is a maternal disorder.

There is positive correlation between shape of placenta, attachment of cord, number of cotyledons, placental diameter, weight, surface area and birth weight confirming the placental development is of utmost importance for fetal growth and wellbeing which is affected in preeclampsia.

Placenta does not suffer much structurally in isolated preeclampsia or when preeclampsia was late onset but those with fetal growth restriction with or without preeclampsia had reduced villous diameter, surface area, length, volume and maternal vascular lesions. The effect was more in those who had preeclampsia with fetal growth restriction. Preeclampsia and fetal growth restriction are seen to be cumulative rather than synergistic. These morphological changes tend have major physiological implications in terms of placental functions and fetal growth.
CONCLUSION: The present study points to the fact that, the placental development is seriously affected in preeclampsia. The resulting placental growth retardation with its functional derangement subsequently affects the growth of fetus, causing many morbidities in neonatal and later life. Any further research to enhance or to prevent placental growth retardation may go in a long way to help the growth of fetus in utero and prevent subsequent morbidity in intrauterine and extra uterine life.

| Parameter               | Normotensive | Hypertensive | Z value | P value |
|-------------------------|--------------|--------------|---------|---------|
| No. of cotyledons       | 17.04 +/- 2.06 | 14.07 +/- 2.04 | 10.24   | <0.0001 |
| Weight of placenta      | 423.11 +/- 45.85 | 357.45 +/- 61.66 | 8.54    | <0.0001 |
| Diameter of placenta    | 16.00 +/- 0.75  | 15.31 +/- 1.08  | 5.24    | <0.0001 |
| Surface area            | 201.40 +/- 19.19 | 183.79 +/- 25.94 | 5.45    | <0.0001 |
| Birth weight            | 2590 +/- 0.29   | 2360 +/- 0.40   | 4.65    | <0.0001 |

Table 1: Comparison of parameters of placenta between normotensive and preeclampsia cases

| Characteristic                          | Correlation Coefficient |
|-----------------------------------------|-------------------------|
|                                        | Normal Pregnancy | Preeclampsia |
| Placental Weight & Birth weight         | 0.96                   | 0.98         |
| Maximum placental diameter & Birth weight | 0.91                   | 0.92         |
| Surface area of placenta & Birth weight | 0.9                   | 0.86         |
| Number of cotyledons & Birth weight     | 0.77                   | 0.82         |

Table 2: Correlation coefficient between different placental parameters and birth weight in both study groups

| Studies   | Mean placental weight * P vs N | Mean placental diameter. * P vs N | Mean placental surface area *P vs N | Mean fetal weight *P vs N |
|-----------|--------------------------------|----------------------------------|------------------------------------|--------------------------|
| Ajankar   | 306.45 +/- 78.74 vs 408.14 +/- 54.78 |                                   | 182.84 +/- 56.71 vs 221.99 +/- 50 | 2279.14 +/- 418.08 vs 2651.18 +/- 392 |
| VP 17     |                                |                                  |                                    |                          |
| Sabitha   | 345.96 +/- 24.29 vs 435.92 +/- 14.18 | 15.91 +/- 2.11 vs 18.40 +/- 1.42 |                                    | 2329.72 +/- 284.87 vs 3140.90 +/- 73.98 |
| Singh 18  |                                |                                  |                                    |                          |
| Londhe PS | 312.93 +/- 70.14 vs 401.80 +/- / |                                  | 182.80 +/- 57.47 vs 212.48 +/- / | 2260 +/- 560 Vs 2730 +/- 410 |
| 13        |                                |                                  |                                    |                          |
| This study| 357.45 +/- 61.66 vs 423.11 +/- 45.85 P value significant | 15.31 +/- 1.08 vs 16.00 +/- 0.75 P value significant | 183.79 +/- 25.94 vs 201.40 +/- 19.19 P value significant | 2360 +/- 0.40 vs 2590 +/- 0.29 P value significant |

Table 3: Different studies showing morphometric comparisons between preeclampsia and normotensive placentae

*P= Preeclampsia Cases; N= Normotensive Cases
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