IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 14, Issue 4 Ver. IX (Apr. 2015), PP 11-15
www.iosrjournals.org

Placental Morphology and Fetal Outcome in Preeclampsia And Normotensive Pregnancies

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Abstract:*Placenta is a vital organ playing central role in pregnancy. It maintains pregnancy and promotes normal fetal development and serves as a major organ for transfer of essential elements between mother and fetus. Placental function is highly influenced by its anatomical structure. Present study aims to correlate morphological changes in preeclampsia with fetal birth weight and compare it to normotensive placentae . A cross sectional descriptive study was undertaken in our medical college. Total 100 placentae,50 from normotensive pregnancies and 50 from preeclampsia cases were collected.The morphometric parameters of placentae were recorded. Fetal weight and APGAR score was recorded as fetalparameters.Morphometric parameters of placenta such as weight, diameter, thickness and fetal weight was lower in study group as compared to control group. Placental weight had significant correlation with fetal birth weight. Preeclampsia is associated with placental dysfunction and definite morphological changes of placenta due to reduced uteroplacental blood flow.*

Keywords:*Morphology,Placenta, Fetal weight, Preeclampsia*.

I. Introduction

Placenta is an essential organ for maintenance of pregnancy and for promotingnormal growth and development of fetus[1].It gives the most accurate record of infants prenatal experience[2].Placenta undergoes different changes in weight, structure, volume, shape and function continuously throughout gestation to support the prenatal life[3].Intrauterine growth of fetus mainly depends on adequate function of placenta. Placenta is a fetal organ. It shares same stress and strain to which the fetus is exposed. Thus any disease which affects the mother has a great impact on placenta. Anatomical structure of placenta greatly influences in function. Thus study of placental morphology is considered essential[4].There are many well established causes of IUGR and preeclampsia being one of them. Preeclampsia is a systemic disorder defined as development of hypertension and proteinuria after 20 weeks of gestation in previously normotensive woman. Preeclampsia affects 5 to 7 % of women worldwide and is a major cause for maternal and neonatal morbidity and mortality [5]. It is one of the disorders of pregnancy which is accompanied by gross pathological changes in placenta and is associated with high perinatal morbidity and mortality [6]. Architecture of placenta is found to be changed in many maternal diseases such as hypertension, preeclampsia and ecclampsia. Preeclampsia is a one of the pregnancy induced metabolic disease that may jeopardise the health of mother and the fetus[7]. Preeclampsia contributes to complications like preterm birth, perinatal death, IUGR and is directly associated with 10 to 15 % of maternal deaths. The incidence being 3 to 7 % in nulliparas and 1 to 3 % in multiparas[8].Pregnancy complications like preeclampsia are reflected on placenta both macroscopically and microscopically.The fetus depends on placenta for normal development, thus pathological changes in placenta result in reduced blood flow across placenta and uteroplacentalinsufficiency[9,2]. Present study aims to correlate morphological changes in preeclampsiawith fetal birth weight and compare it to normotensive placenta.

II. Materials and Method

Consecutive convenient sampling method was done. 50 normal and 50 preeclamptic placentae were collected immediately after delivery from department of Obstetrics and Gynecology of our medical college. Institutional ethical committee clearance was taken.

Samples were divided into two groups as Group A and Group B.

Group A (Control): This group comprised of pregnant women without preeclampsia. These women had normal blood pressure, no proteinuria and no edema.

Group B (Preeclampsia) : Placentae were obtained from known preeclamptic cases who had no history of hypertension before pregnancy or during first 20 weeks of gestation, who had consistently recorded systolic and diastolic blood pressure of 140 / 90 mm of Hg or above and proteinuria at least '+1' in freshly voided urine. Detailed menstrual and obstetric history and past history to exclude pre-existing hypertension and other

complications was obtained.Fetal weight, sex, any congenital anomaly and APGAR score at 1 and 5 minutes after delivery was recorded as parameters of fetal outcome.

Placental parameters:

Placental Weight: Placental weight was measured by directly placing the placenta over standardised weighing scale.Placental Diameter: The placenta was placed on a flat surface after trimming and mopping. At first the maximum diameter was measured with a metallic scale graduated in centimetres. Then second maximum diameter was recorded at right angles to the first one. The mean of two diameters was considered as the diameter of placenta.Placental Thickness: Placenta was placed on a flat surface. Two circles were drawn from the centre of the placenta. Thick point needle was inserted to the full thickness of placenta at five points.

Centre of the inner circle, two different points between inner and outer circle and two different points outside the outer circle.Mean of all five points was taken as the thickness of placenta.

Number of cotyledons: Gentle pressure was applied on centre of the fetal surface of placenta. As a result the cotyledons on the maternal surface became prominent. The placenta was then placed on a flat surface with maternal side facing upwards and total number of cotyledons was recorded.

III. Results

The mean weight of placenta was 502.26gms in control group and 430.38gms in study group. The mean placental diameter and thickness was 18.7cmsand 2.3cmsin controlgroup, 17.2cms and 1.8cms in study group respectively. The mean number of cotyledons was18.9 in control group and 16 in study group. All the morphometric parameters were significantly reduced in study group as compared to control group.Out of 50 placentae 6% showed marginal attachment in control group and 24%in study group. Placental infarcts were observed in 8% placentae of control group and 66% cases in study group.The mean fetal birth weight and APGAR score was also found to be lower in study group as compared to control group. The mean fetal weight was 2.1 kg and 2.8 kg in study group and control group respectively. The Mean APGAR score was 6.5 in study group and 8.9 in control group.

Table I: Gross morphology of placenta in control group and preeclampsia

Placental Parameters	Control Group Mean ± SD	Study Group Mean ±SD
Placental weight (grams)	502±58.42	430 ±50.69
Placental diameter (cms)	18.7±1.55	17.2±1.70
Placental thickness (cms)	2.3±0.43	1.8 ± 0.49
Number of cotyledons	18.9±1.98	16 ± 2.29
Marginal insertion of cordn(%)	3(6%)	12(24%)
Placental infarcts n (%)	4(8%)	33(66%)

Table II: Placental weight in control group and study group

Placental weight (grams)	Control Group %	Study Group%
300-400	6 (12%)	19 (38%)
400-500	15 (30%)	26 (52%)
500-600	25 (50%)	5 (10%)
600-700	4 (8%)	—

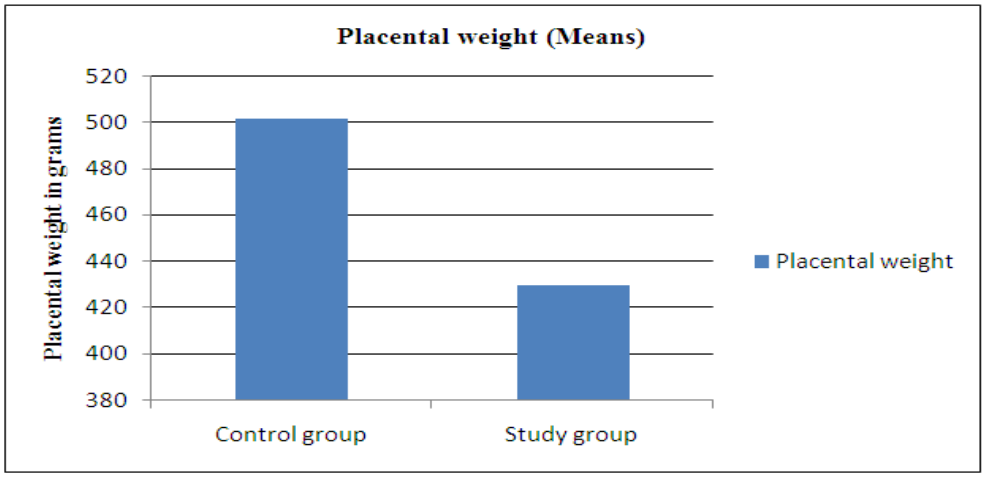

Figure I: Means of placental weight(grams)

Table III: Fetal parameters of control group and preeclampsia.

Fetal parameters	Control Group Mean ± SD	Study Group Mean ± SD
Birth weight (kg)	2.8±0.46	2.1± 0.39
APGAR score	8.9±0.76	6.5 ±0.95

Table IV: Fetal weight n(%)

Fetal weight (kg)	Control Group	Study Group
Below 2.5	9(18%)	39 (78%)
Above 2.5	41(82%)	11 (22%)

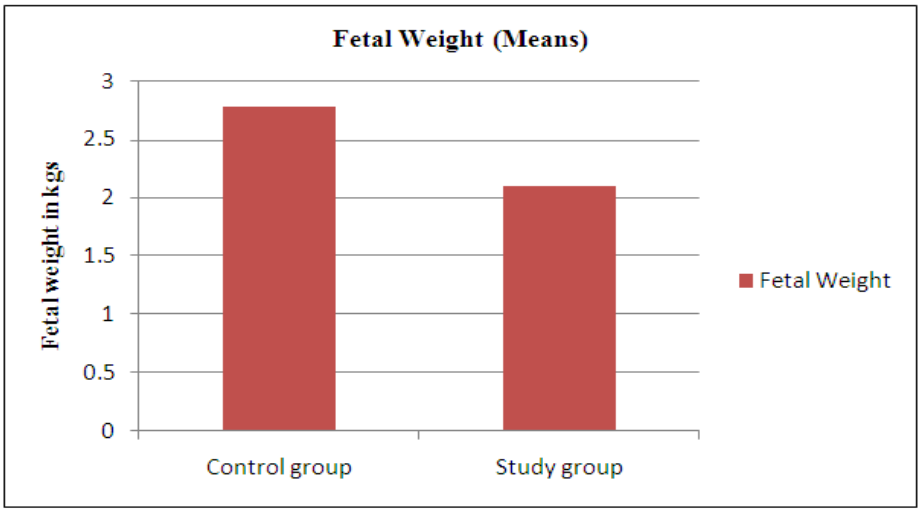

Figure II: Means of fetal weight (kg)

IV. Discussion

Weight of placenta is functionally significant as it is related to villous surface area and fetalmetabolism[1].Normally placental weight ranges from 400gms to 600gms. In present study we observed that mean placental weight was less in preeclamptic placentae as compared to normotensive placentae. In present study mean placental weight was 502.26 gms in normal pregnancy and 430.38 gms in preeclamptic cases. Similar results are reported by Udaina et al, Mallik et al and Mujumdar et al who found reduced placental weight in preeclamptic cases as compared to normotensive pregnancies. Comparative studies were also done by Aparna et al. However they concluded that gross abnormalities were significant in normotensive placentae. Mayhew et al reported that preeclampsia was not associated with main effects on placental morphology.

The present study noted that the mean placental diameter and thickness was 17.2cms and 1.8cms in preeclamptic placentae and 18.7cms and 2.3cms in normotensive placentae. Preeclamptic placentae were smaller and thinner as compared to normotensive placentae. Similar findings have been reported by Modi et al, Londhe et al, Salmani et al. Teasdale et al found significant reduction in transverse diameter of preeclamptic placentae. This reduction may be due to small size of placenta in preeclampsia. Ciblis et al andKishwara et al reported that preeclamptic placentae were smaller than normal indicating an underlying pathological process which interferes with normal growth of placenta.

Number of cotyledons were also less in preeclamptic placentae as compared to normotensive. The mean number of cotyledons was 18.9 in normotensive placentae and 16 in preeclamptic placentae. Similar findings were reported by Londhe et al, Nag et al and Kishwaraetal.Rath et al reported that intercotyledonous vasculature is altered in hypertensive placentae resulting in low birth weight babies.

In present study we have noted that all placental dimensions that is mean placental weight, thickness and diameter show significantly lower values in preeclamptic cases as compared to control. The main reason for reduced placental weight in preeclampsia could be uteroplacental insufficiency. Preeclampsia adversely affects placental morphology. It has been recorded that maternal vasospasm in preeclampsia leads to decreased maternal uteroplacental blood flow. Hypoxia and reduction in blood flow could be responsible for morphological alterations of placenta in preeclampsia[20,2]. The mean external diameter of uterine spiral arterioles in preeclamptic women is less than half of the diameter of these arterioles in normotensive women. This results in reduced uteroplacental blood flow due to which placenta becomes extremely ischemic as gestation continues. Placenta tries to compensate for reduced supply however these compensatory changes are insufficient and thus fails to develop adequate placental mass[21]. Kishwara et al[22] and Teasdel et al[15]have reported reduced Parenchymal components such as intervillous space, trophoblasts mass of peripheral villous

tissue and capillaries in preeclamptic placentae as compared to normotensive placentae. Sankar et al reported reduced villous diameter,surface area and vessel densities in preeclamptic placentae. Thus reduced proportional and absolute volume in preeclampsia could contribute to reduced morphological dimensions in preeclampsia.

Preeclampsia is a primary cause of placental insufficiency [24]. Abnormal cytotrophoblastic invasion leads to placental ischemia and endothelial dysfunction which characterizes preeclampsia [25]. The main impact on fetus is under nutrition due to uteroplacental vascular insufficiency which leads to growth retardation [26].Odegard et al reported that preeclampsia was associated with 5% reduction in fetal weight. The risk of small for gestational age was four times higher in infants born to preeclamptic women as compared to normotensive women. Kishwara et al[28] reported that the mean fetal weight in preeclampsia and control was 2.80 kg and 2.26 kg respectively. Boyd and Scott et al reported that the mean fetal weight was 1998 gms and 2714 gms in control and preeclamptic placentae. In present study the mean fetal weight in normotensive cases was 2.8 kg and preeclamptic cases was 2.1 kg. We observed increased incidence of IUGR and reduced placental weight with increased severity of hypertension. It was observed that smaller placentae usually accompanied low birth weight babies. Similar findings have been reported by Londhe et al, Udaina et al, Salmani et al, Sankar et al and Meyhew et al. The mean APGAR score in present study was 8.9 and 6.5 in normotensive and preeclamptic cases respectively. Similar findings were reported by Navbir et al, Kishwara et al 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. Thus leading to inadequate placental mass causing placental dysfunction and consequent chronic fetalhypoxemia.Myatt et al, Soma et al, Eskild et al suggested that placental insufficiency and impaired placental function in hypertensive pregnancy leads to lowfetal birth weight.

V. Conclusion

Our results confirm strong association with low placental weight and low fetal birth weight. This data indicates that low placental weight is associated with placental dysfunction. Thus we can conclude that severity of preeclampsia has adverse effect of morphology of placenta and consequently affects the fetal weight.

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