Original Article

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Placental Changes in Anemia, Gestational Diabetes and Pregnancy Induced Hypertension

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Abstract

Background: To assess the changes occurring in the placenta due to anemia, pregnancy induced hypertension (PIH), Gestational Diabetes Mellitus (GDM) and whether these changes were reversible with treatment when they were diagnosed in early stages. **Subjects and Methods**: The study is conducted in the Department of Obstetrics and Gynecology, DVVPF's Medical College, Ahmednagar on 200 anemic patients, 180 PIH patients and 650 GDM patients. Placentae were collected, stored in a 10% formalin solution. Morphological changes in respect to shape, weight, diameter, thickness, number of cotyledons and site of insertion of the cord are observed and documented. **Results:** In anemic patients who has undergone treatment, there were a smaller number of irregular (37/100) and round (63/100) round placentae were seen, whereas in the untreated anemic group, round (09/100) round and irregular (91/100) irregular placentae were seen. In PIH group with treatment, round (57/90) placentae and irregular (33/90) placentae were seen, whereas, in the untreated group, 13/100 round/oval placentae were observed and 77/100 irregular placentae were seen. **Conclusion:** Morphology of placenta is on error mode in untreated anemia, PIH and GDM. Diagnosis in the first trimester and on consequent treatment till the delivery is resulting in decreasing of irregular morphology of placenta and wellbeing of newborns.

Keywords: Placenta, Anemia, Pregnancy Induced Hypertension, Gestational Diabetes Mellitus.

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Introduction

The placenta is a mirror that reflects the intrauterine status of the fetus. promotes normal fetal development. It is an organ that connects the developing fetus to the uterine wall, thereby allowing nutrient uptake, waste elimination, and gas exchange via the mother's blood supply. Proper vascular development in the placenta is fundamental to ensuring a healthy fetus and successful pregnancy required for spontaneous induction of labor.[1] Oxygen availability has proved as a critical factor in the development of the placenta and placental blood vessels too.[2] It is known to have important functions, as an endocrine organ and in maternal-fetal exchange.[3,4]

Anemia in pregnancy is well recognized and often observed in developing countries. The severity of anemia among expectant mothers is judged by criteria suggested by the world health organization (WHO). Severe anemia is a perilous hematological disorder, due to which late abortions, prematurity, low birth weight and stillbirths are occurring. The global prevalence of anemia in pregnancy is 55.9% and in India, the incidence has been noted as high as 40-80%.[5]

Hypertension (HTN) in pregnancy may be chronic hypertension (onset before pregnancy) or may be induced due to pregnancy, preeclampsia and eclampsia. These disorders form one of the deadly triads along with hemorrhage and infection resulting in a substantial number of maternal deaths and thereby fetal deaths. It affects 7-10% of pregnancies throughout the world. HTN in pregnancy is found to be associated with variable histomorphological changes in the placenta, which shows a clear reflection of poor foetal outcome.[6]

Gestational diabetes mellitus (GDM) is described as glucose intolerance of varying severity with the onset or first recognition during pregnancy and disappears with deliverycommon metabolic problem complicates approximately 2-4% of pregnancies and it is the major cause of macrosomia and perinatal mortality and usually associated by clinical hyperglycemia, hyperlipidemia, hyperinsulinemia and placental endothelial dysfunction.[7–9] In India the prevalence of GDM is 4-11.6% and varies according to geographical areas and diagnostic

methods employed.[10,11]

With a multifaceted trait, the placenta is exposed to the regulatory influence of hormones present in both fetal and maternal circulations and is affected by changes in any of these. As a fundamental organ with complexities of intrauterine life, the placenta grabs a pliant response and tries to remunerate to prevent any fetal complications. It remodels in weight, volume, structure, shape and functions tirelessly throughout gestation to maintain prenatal life.[12]

Considering the outcomes of these diseases on the development of the placenta and further that of foetus and also the changes that occur microscopically in the placenta, we decided to study this topic using different staining methods. Going through the literature, we observed earlier researchers had done the morphological and histological studies of placenta only. In our study, we aim to study the changes in the placenta with and without treatment.

Subjects and Methods

After getting Institutional Ethics Committee clearance, this cross-sectional study was carried out in the department of Obstetrics and Gynaecology, DVVPF's Medical college and hospital.

The study participants were divided into seven groups as follows:

- Controls (n=325)
- GDMNT (n=325) : Gestational diabetes without treatment
- GDMT (n=325) : Gestational diabetes with treatment
- PIHNT (n=90) : Pregnancy-induced hypertension without treatment
- PIHT (n=90) : Pregnancy-induced hypertension with treatment
- ANENT (n=100) : Anemia without treatment
- ANET (n=100) : Anemia with treatment

Inclusion Criteria

Only the pregnant women of 18-40 years of attending the obstetrics and gynecology and willing to participate in the study by signing an informed consent form were included. Patients who deliver in normal and Caesarean sections were included.

Exclusion Criteria

Patients who are less than 18 years and above 40, Patients with blood-borne infections like HIV, Hepatitis, Patients with drug abuse, alcohol and smoking were excluded.

Method of Collection

Placenta specimens were collected from the obstetrics and gynecology department and stored in a 10% formalin solution and then morphometrical studies were conducted on it.

Variables

In placentae, the following parameters were measured:

Morphological changes in respect to

Shape, Weight, Diameter in the above disease conditions.

Statistical Analysis

Was carried out by using SPSS14. Data expressed as mean, standard deviation and percentages as applicable. Kolmogrov smrinov test was used to assess the normality. The between-group analysis was done by using an independent t-test. Null hypothesis was rejected at 0.05.

Results

To study the role of treatment in placental and umbilical cord changes due to anemia, gestational diabetes and pregnancy-induced hypertension, three hundred and twenty-five controls, three hundred twenty-five pregnants with gestational diabetes with treatment, without treatment, ninety pregnancy-induced hypertensives with treatment and without treatment, one hundred pregnants with gestational diabetes on treatment and without treatment were studied.

Baseline details like age, height, weight, the information about parity and type of delivery in between the groups were given in [Table 1].

[Table 2] shows the shape of the placenta in study groups. The Control group has 298 round/oval-shaped and twenty-seven irregular placentas. In the anemic group with treatment, there was less number of irregular 37/100 and 63/100 round placentas were seen, whereas in the untreated anemic group contains 09/100 round/oval placentas and 91/100 irregular placentas were seen. In the pregnancy-induced hypertension group, those who are treated had 57/90 round placentas and 33/100 irregular placentas were seen, whereas in the untreated pregnancy-induced hypertension group 13/100 round/oval and 77/100 irregular placentas were seen.

The weight of the placenta in study participants was depicted in [Table 3]. The untreated anemic group had the highest value when compared to control and all other patients.

[Table 4] shows the diameter of the placenta among various groups of the study. The lowest diameter was seen in the untreated pregnancy induced hypertension group. The thickness of the placenta in study groups was depicted in [Table 5, Figure 6]. The untreated anemic group had the lowest thickness.

Table 1: Baseline information of study participants

S.No	Parameter	Group						
		GDMNT (n=325)	GDMT (n=325)	PIHNT (n=90)	PIHT (n=90)	ANENT (n=100)	ANET (n=100)	Controls (n=325)
1	Age	27.20 (3.31)	26.98 (3.30)	27.23 (3.35)	26.94 (3.11)	27.40 (3.31)	27.10 (3.28)	26.94 (3.34)
2	Height	156.59 (5.52)	156.64 (5.55)	156.70 (5.55)	156.36 (5.72)	156.54 (5.71)	155.62 (6.53)	156.36 (5.62)
3	Weight	62.53 (10.53)	64.30 (11.51)	65.67 (12.91)	66.07 (14.20)	65.89 (12.55)	66.65 (11.80)	63.75 (11.03)
4	Parity P1 P2	88 237	94 231	38 52	29 61	38 62	42 58	78 247
5	Type of delivery Caesarean Vaginal	295 30	273 52	83 07	78 12	93 07	89 11	136 189

Data of age, height and weight was expressed as mean (SD), parity and type of delivery data is the actual number.
GDMNT: Gestational Diabetes without treatment; GDMT: Gestational Diabetes with treatment; PIHNT: Pregnancy-induced hypertension without treatment. PIHT: Pregnancy-induced hypertension with treatment. ANENT: Anemia without treatment.

Table 2: Shape of the placenta in study participants

S.No	Parame-	Group						
		GDMNT (n=325)	GDMT (n=325)	PIHNT (n=90)	PIHT (n=90)	ANENT (n=100)	ANET (n=100)	Controls (n=325)
1	Round/oval	33	125	13	57	09	63	398
2	Irregular	292	200	77	33	91	37	27

Data expressed as mean (SD)
GDMNT = Gestational diabetes without treatment. GDMT = Gestational Diabetes with treatment. PIHNT= Pregnancy-induced hypertension without treatment. PIHT = Pregnancy-induced hypertension with treatment. ANENT: Anemia without treatment. ANET= Anemia with treatment.

Table 3: Weight of placenta in study participants

Group						
GDMNT (n=325)	GDMT (n=325)	PIHNT (n=90)	PIHT (n=90)	ANENT (n=100)	ANET (n=100)	Controls (n=325)
597.55 (50.76)	469.52 (27.37) **	372.72 (45.08)	482.40 (17.29) **	594.65 (50.43)	488.14 (14.63) **	486.13 (12.26)

Data expressed as mean (SD)
** p<0.001
GDMNT = Gestational diabetes without treatment. GDMT = Gestational Diabetes with treatment. PIHNT= Pregnancy-induced hypertension without treatment. PIHT = Pregnancy-induced hypertension with treatment. ANENT: Anemia without treatment. ANET= Anemia with treatment.

Table 4: Diameter of the placenta in study participants

Group						
GDMNT (n=325)	GDMT (n=325)	PIHNT (n=90)	PIHT (n=90)	ANENT (n=100)	ANET (n=100)	Controls (n=325)
19.68 (3.23)	18.21 (2.60)*	16.09 (1.58)	17.48 (2.51)*	16.18 (1.65)	21.82 (1.29)**	22.10 (1.33)

Data expressed as mean (SD)
** p<0.001, p<0.05
GDMNT = Gestational diabetes without treatment. GDMT = Gestational Diabetes with treatment. PIHNT= Pregnancy-induced hypertension without treatment. PIHT = Pregnancy-induced hypertension with treatment. ANENT: Anemia without treatment. ANET= Anemia with treatment.

Table 5: Shows the comparison between our study and various other studies.

Parameter	Author	Author's finding	Current study
Weight	Rohini. M et.,al	410 gm in anemia	488 gm in anemia
Weight	Udainia.A, Jain.M.L.,	370-435 gm in PIH	482 gm in PIH
Weight	El Sawy.N.A., Iqbal	593.1 gm in GDM	598 gm in GDM

Discussion

This study was carried out to understand the role of treatment in placental changes due to anemia, gestational diabetes and pregnancy-induced hypertension, three hundred and twenty-five controls, three hundred twenty-five pregnants with gestational diabetes with treatment, without treatment, ninety pregnancy-induced hypertensives with treatment and without treatment, one hundred pregnants with gestational diabetes on treatment and without treatment were studied.

Some remote areas of developing countries like India have still had no access to proper medical care. Hence, this study was conducted to compare the beneficial effects of medical management in maternal anemia, gestational diabetes and pregnancy-induced hypertension. [13]

Morphometric analysis hinges on many factors such as a number of analysed placentas, interactions between anemia and malnutrition of expectant mothers. Measurement of placental volume done through ultrasound between 11^{th} and 13^{th} gestational week didn't reveal any prominent alterations among anemic mothers. [14] The baseline characteristics like age, height and weight of participants in between controls and the anemic group were same in our study.

The placentas used in our study weighted from 372.2 g to 469.52 g. The lowest weight was seen in the untreated pregnancy induced hypertension group. Variations like these and fragmentarily opposite values are earlier identified as possible and familiar among anemic mothers and maternal anemia. Thus, it shows a nondependent risk factor for maldevelopment of the placenta. Older literature indicates hypertrophy is dependent on the duration time of hypoxia, but with the advancement of pregnancy, changes to growth restriction and formation of small, hypertrophic placenta. When analogised with our study, anemic mother's placentas total volume is the same as placentas from high altitude pregnancies that are considered as a preplacental hypoxic condition. [15,16]

In a hypoxic situation, placental blood vessels continue to develop with the branching model of angiogenesis till the end of pregnancy, which finally shows short terminal villi with numerous cross-sections of blood capillaries. [17–20]

Lack of iron, not any other reason, in early pregnancy, is a causative factor for anemia, which in turn is becoming a double-sized risk for pre-term delivery of low birth weight. Contradicting data from 3^{rd} world countries where, among the well-nourished maternal population, lower iron status of expecting mothers is affiliated with higher birth weight and longer pregnancies. [21–28]

The usual term placenta is about 22 cm in diameter and 2.0 to 2.5 cm thick. However, the measurements can vary considerably, and placentas generally are not weighed in the delivery room. The maternal surface of the placenta should be dark maroon in color and should be divided into lobules or cotyledons. The structure should appear complete, with no missing cotyledons. The fetal surface of the placenta should be shiny, gray and translucent enough that the color of the underlying maroon villous tissue may be seen. [29–33]

The a placental thickness of > 40mm at term is associated with gestational diabetes, intrauterine infections and hydrops foetalis. [34] La Torre opined that at no stage of the pregnancy placental thickness exceeded 40 mm indirectly, thus indicating the cut-off value for the upper limit. [35] Among the pregnant women with CMV infections, the placental thickness was increased in about 93.3% of the subjects. [36]

There can be several variations with cord insertion into the placenta: central insertion (~90%) is the normal situation, eccentric cord insertion: lateral insertion of the umbilical cord >2 cm from the placental margin, the term sometimes used synonymously with marginal cord insertion, marginal cord insertion (~7%): insertion of the umbilical cord <2 cm from the placental margin, velamentous cord insertion (~1%): insertion of the umbilical cord on the fetal, chorioamnionitis) membranes. [37]

The form of the human placenta is generally classified as a discoid placenta. Within this, the cotyledons are the approximately 15-25 separations of the deciduas basalis of the placenta, separated by placental septa. [38] Each cotyledon consists of the main stem of a chorionic villus as well as its branches and sub-branches.

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The weight of the placenta in study participants was depicted in [Table 3]. The untreated anemic group had the highest value when compared to control and all other patients. [39]

[Table 4] shows the diameter of the placenta among the various group of the study. The lowest diameter was seen in the untreated pregnancy induced hypertension group. The thickness of the placenta in study groups as depicted in [Table 5]. The untreated anemic group had the lowest thickness. [40]

Conclusion

Morphology of placenta is on error mode in untreated anemia, PIH and GDM. Diagnosis in the first trimester and on consequent treatment till the delivery is resulting in decreasing of irregular morphology of placenta and wellbeing of newborns.

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