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A sonographic study of fetal in normal and hypertensive pregnant female in Central India

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Abstract

Background: Hypertensive disorders in pregnancy are a significant cause of maternal and fetal morbidity. The placenta's aging and senescence, reflected in these changes, are critical markers in assessing pregnancy complications. This study aims to sonographic study of fetal between hypertensive and normotensive pregnancies in Northern India, aiming to elucidate differences that could contribute to better management and outcomes in high-risk pregnancies.
Objective: A sonographic study of fetal in normotensive and hypertensive pregnant women in Central India.
Methods: The present prospective case-control study involving 200 pregnant women (100 normotensive, 100 hypertensive) of third trimester gestation. Placental grading was assessed using ultrasonography.
Results: General examination findings, however, highlighted significant differences in pulse rate, systolic and diastolic blood pressure, and respiratory rate, all higher in the case group ($p < 0.001$). Placental grading showed most patients in both groups had Grade II, and no significant difference was observed between groups. Fetal weight was significantly lower in the case group (2.5 ± 0.2 kg) compared to controls (2.7 ± 0.3 kg). Perinatal morbidity and mortality were higher in the case group but not statistically significant.
Conclusion: This study demonstrates placenta is mirror image of the fetal outcome. Examination of placenta by ultrasonography, there is definite evidence of changes in placental morphology and grading in pregnancy-induced hypertensive mothers.

Keywords: Placental grading, Hypertension, Pregnancy, Ultrasonography, IUGR.

Introduction

Worldwide around 76,000 pregnant ladies pass on every year from pre-eclampsia and related hypertensive issue.[1] Ultrasound reviewing arrangement of placenta in light of its development. This essentially influences the degree of calcifications. Placental grade III maturity is associated with placental insufficiency due to chronic hypertension. This may lead to intrauterine growth restriction (IUGR), abnormal fetal growth, fetal distress and hyaline membrane disease. Birth weight depends on the mother's body size and growth of placenta.[2] In hypertensive pregnancy, the preterm placental calcifications have adverse effects on uteroplacental blood flow, fetal growth and fetal death.[3] The arteries affected by hypertension which are carrying blood to placenta. In the unlikely event that the placenta receives insufficient blood, the child's intake of oxygen and nutrients may be reduced. This can prompt moderate development, low fetal weight (IUGR).[4] There is progressive decrease in the mean diameter & surface area of placenta with increase in severity of pregnancy induced hypertension.[5] The risk factor of hypertension is placental abruption that may cause premature birth.[6] The morphological and histological changes in placenta driving component to ischemia because of low course which prompts diminished oxygen supply to the hatchling prompting intrauterine growth restriction (IUGR) contributing to premature birth and fetal death.[7] When sonographers examined the fetal, they also examined the placenta as secondary object. The importance of sonographic examination and documentation of the placenta must be conscious for ultrasound professionals. In two-dimensional ultrasound techniques, the location and perimeters of placenta easily discovered. In three-dimension ultrasound techniques have opened frontier of placental examination. Placental maturity can be assessed by ultrasound to visualize the changes in placental substance. The placental grades are the amount of calcium deposition.[8] For the fetus to grow and develop normally in pregnancy, the placenta must operate properly. The fetal organ known as the placenta experiences the same levels of stress and strain as the fetus. Therefore, the placenta is greatly impacted by any illness condition that affects the mother and fetus, and vice versa. The placenta's morphology changes during the course of its brief existence. Changes in the placenta associated with the "aging" phenomenon are most likely a result of maturation and are closely related to pleacenta's ongoing growth. Numerous elements influence the fetus's health, but the most crucial one for creating a healthy child is a healthy placenta. Many people have the strongly held belief that the placenta ages gradually throughout a typical pregnancy and is about to enter a state of morphological and functional senescence.[9,10] There correlation between certain micro and microscopic Placental alterations and a variety of pregnancy problems. Increased prenatal morbidity and mortality are virtually definitely the result of compromised placenta perfusion from uterine vasospasm, which is linked to pregnancy-induced or pregnancy-aggravated hypertension.[11,12]

Diagnosis of gestational hypertension is made in female whose BP reaches 140/90 mm Hg or greater for the first-time during pregnancy but in whom proteinuria is not identified. Gestational hypertension is also called transient hypertension if preeclampsia does not develop and the blood pressure returns to normal by 12 weeks postpartum. Thus, the gestational hypertension is a diagnosis of exclusion.[11] In underdeveloped nations such as India, the primary causes of maternal death include infections, hemorrhage, and hypertensive pregnancies, which result both maternal and perinatal morbidity and mortality.

Hypertensive disorders in pregnancy pose a significant threat to maternal and fetal well-being, affecting approximately 8-10% of pregnancies worldwide.[13] In Central India, the prevalence of hypertensive disorders is notably higher, with limited access to early detection and management. Placental dysfunction is a critical factor in the development of hypertensive disorders, and accurate assessment of placental morphology through ultrasonography can facilitate timely intervention. However, the current understanding of placental changes in hypertensive pregnancies is largely based on Western populations, with scarce data on regional Indian demographics. The lack of standardized placental grading criteria and limited expertise in ultrasonography compounds the issue, leading to delayed diagnosis and increased morbidity. Furthermore, the correlation between placental grading and maternal blood pressure levels, as well as the predictive value of placental grading for hypertensive disorders, remains poorly understood. This knowledge gap hinders the development of targeted interventions, exacerbating the already high rates of maternal and fetal complications in Central India. Thus, purpose of study is to sonographic study of fetal in difficult hypertensive pregnancy to that in normotensive pregnancy.

Martial and Method

The present prospective case control study was conducted in department of anatomy in LNCT UNIVERSITY BHOPAL for the period of 1.5 year from August 2020-February 2022 in Lucknow. A minimum of 200 patients were chosen, with 100 pregnant women with hypertension serving as the case group and the other 100 as the control group. All singleton pregnant women with hypertensive disorders presenting in labour in third trimester with blood pressure more than 140/90 mmHg were enrolled in this study. Pregnant women with Multifetal gestation, Epilepsy, Bone disorder of any multivitamin intake, Renal diseases, Liver disease, Thyroid disease or any endocrinal disease, Haemorrhagic disorder and Diabetes mellitus were excluded from the study. A detailed clinical history including age, sex, occupation, socio – economic status and any associated risk factors contributing for the illness was elicited from the case and controls.

Patients were having to meet American College of Obstetricians & Gynecology (ACOG) criteria for diagnosing hypertensive disorders pregnancy (Diastolic Blood Pressure >= 90 mmHg or Systolic Blood Pressure >= 140 mmHg or both). Hypertensive Disorders of Pregnancy (HDP) could be divided into 4 subgroups according to ACOG:

1. **Preeclampsia (PE)-Eclampsia (EC)**

PE is defined as blood pressure ≥140/90 and proteinuria ≥+1, measured at least twice 4-6 h apart after gestational weak 20. Eclampsia was defined as the onset of convulsion in woman with EP that could not be attributed to other cases.

2. **Chronic hypertension**

Chronic (preexisting) hypertension was defined as hypertension (systolic blood pressure ≥140mmHg or diastolic blood pressure ≥90 mmHg or both) that present before 20 weeks gestational or prior to pregnancy.

3. **Chronic hypertension with Supremeimposed preeclampsia**

When preeclampsia develops in women with chronic (preexisting) hypertension, the classification of disease was chronic (preexisting) hypertension superimposed preeclampsia.

4. **Gestational hypertension**

GH was defined as development of hypertension (i.e., systolic BP ≥140 mmHg and diastolic BP ≥90 mmHg) for the first time after mid pregnancy (after 20 weeks) gestation without proteinuria or other features of preeclampsia; this terminology replaces the term “pregnancy included hypertension. All women who fit into the inclusion criteria would be informed about the study protocol and their written & informed consent would be taken. Detail history of these women with include age, religion, literacy, occupation, residence socioeconomic status would be noted. Obstetrics history, past history, any history of previous pregnancy affected by hypertension disorder of pregnancy (HDP), family history, pre-existing medical conditions, gestational age, education, socioeconomic status, smoking status. Equal number of healthy pregnant women would be taken under control group.

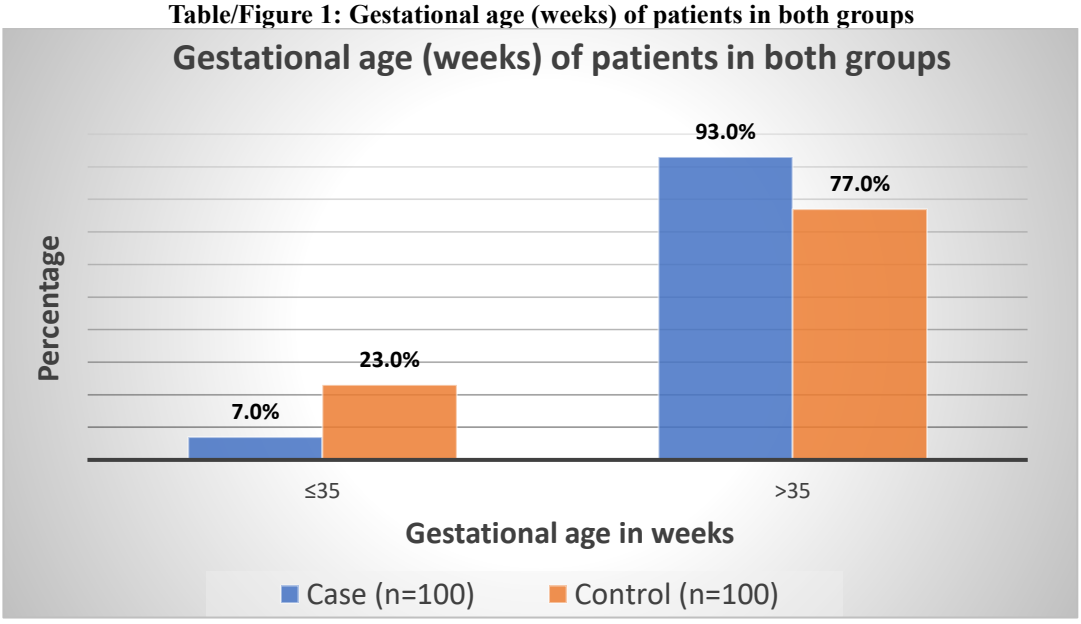
After a written informed consent form was obtained, a detailed history of the presenting symptoms and their onset was recorded. Detail histories of all the women was obtained (like demographic, age of patient, age of menarche, previous menstrual history) and radiological finding was noted on patient proforma. Each participant underwent ultrasonographic examination to estimate the gestational age, routine haemogram and other biochemical investigation was carried out as and when required. Both the control and study groups had their placental grades recorded using ultrasound technology, and the research's results were examined in terms of placental grading, fetal distress, birth asphyxia, delivery method, fetal maturity, and prenatal morbidity.

Ultrasonographic assessment is performed on a GE LOGIQ PRO5, using a high frequency 7-12MHz linear electronic array transducer. All ultrasounds were performed by a single radiologist to avoid inter-observer variation. Patient in supine position, Jelly was applied over the abdomen and examination was carried out. The placenta's morphology was examined in the following headings in order to scan it for placental grading: Chorionic plate, echo-texture of placental substance and basal layer.
Microsoft Excel was used in creating the database and producing graphs, while the data was analysed using the statistical Package for the Social Science (SPSS) version 23.0 for Windows. Mean and standard deviation (±SD) was used to describe quantitative data meeting normal distribution. Both control and research groups had their ultrasonic placental grading recorded. The outcome was examined in relation to baby's birth weight, fetal maturity, prenatal morbidity and mortality, and placental grading. Result was compared using a chi square test of significance. The student "t" test was used to determine whether there was a statistical difference between two groups and the parameters measures.

Result:

In the case group, only 7% had gestational age ≤35 weeks, while in the control group, 23% had gestational age ≤35 weeks. Most patients in both groups had gestational age more than 35 weeks. The mean age of the case group was 25.10±4.3 years, which was similar to the control group (24.71±4.2 years), with a p-value of 0.517. Additionally, there were no significant differences in height (158.5±7.5 cm vs 158.2±8.1 cm, p=0.786), weight (60.97±5.3 kg vs 60.6±5.6 kg, p=0.631), and body mass index (BMI) (24.2±1.0 kg/m2 vs 24.10±1.1 kg/m2, p=0.501) between the case and control groups, respectively.
The case group had a significantly higher pulse rate (89.7±5.8) compared to the control group (75.4±6.0), with a p-value of <0.001. Similarly, the case group had higher systolic blood pressure (SBP) (153.3±10.2) and diastolic blood pressure (DBP) (92.4±3.5) compared to the control group (120.3±3.3 and 80.4±3.4, respectively), with p-values of <0.001. The respiratory rate (RR) was also higher in the case group (20.4±3.4) compared to the control group (16.6±2.4), with a p-value of <0.001.
In this study we noted that the sex distribution was similar, with 52% males in the case group and 53% males in the control group. The mean birth weight was significantly lower in the case group (2.5±0.2 kg) compared to the control group (2.7±0.3 kg), with a p-value of <0.001. The incidence of birth asphyxia was slightly higher in the case group (15%) compared to the control group (11%), but the difference was not statistically significant. However, the case group had significantly lower rates of perinatal morbidity (14% vs 25%, p=0.049) and perinatal mortality (9% vs 25%, p=0.003) compared to the control group.
None of the patients in either group had a placental grading of 0. Placental grading I was observed in 5% of the case group and 10% of control group. The majority of patients in both groups had a placental grading of II, with 52% in case group and 60% in control group. Placental grading III was observed in 43% of the case group and 30% of control group. Overall, the majority of patients in both groups had a placental grading of II. There was non-significant difference in placental grading between the two groups (p-value = 0.207).

Table/Figure 1: Gestational age (weeks) of patients in both groups



Table/Figure 2: Maternal Age and Anthropometric variables of patients in both groups

	Case (n=100)	Control (n=100)	p-value
Age (years)	25.10±4.3	24.71±4.2	0.517
Height (cm)	158.5±7.5	158.2±8.1	0.786
Weight (kg)	60.97±5.3	60.6±5.6	0.631
BMI (kg/m^2)	24.2±1.0	24.10±1.1	0.501

Table/Figure 3: General examination of patients in both groups

General examination	Case (n=100)	Control (n=100)	p-value
Pulse rate	89.7±5.8	75.4±6.0	<0.001
SBP	153.3±10.2	120.3±3.3	<0.001
DBP	92.4±3.5	80.4±3.4	<0.001
RR	20.4±3.4	16.6±2.4	<0.001

Table/Figure 4: Fetal outcome in both groups

Fetal outcome		Case (n=100)	Control (n=100)	p-value
Sex	**Male**	52 (52.0)	53 (53.0)	1.000
	Female	48 (48.0)	47 (47.0)	
Weight		2.5±0.2	2.7±0.3	**<0.001**
Birth asphyxia	**Yes**	15 (15.0)	11 (11.0)	0.400
	No	85 (85.0)	89 (89.0)	
Perinatal morbidity	**Yes**	14 (14.0)	25 (25.0)	**0.049**
	No	86 (86.0)	75 (75.0)	
Perinatal mortality	**Yes**	9 (9.0)	25 (25.0)	**0.003**
	No	91 (91.0)	75 (75.0)	

Table/Figure 5: Placental grading of patients in both groups

Placental grading	Case (n=100)	Control (n=100)	p-value
0	0 (0.0)	0 (0.0)	0.207
I	5 (5.0)	10 (10.0)	
II	52 (52.0)	60 (60.0)	
III	43 (43.0)	30 (30.0)	

Discussion

Our study noted that the mean age for the case group was 25.10±4.3 and for control group was 24.71±4.2, with non-significant difference between groups. In a similar study **Nazir S et al[14]** reported that mean + S.D of maternal age was 27.7+4.3years with minimum and maximum maternal age were 20 and 40 years respectively. In a study **Chhatwal J et al[15]** reported mean age of cases: 27.60±4.37 years.

Our study noted that there was anthropometric variable (height, weight and BMI) comparable in the both groups (p-value > 0.05). In this study there were significant differences higher pulse rate, SBP, DBP and RR in the case with respect to control groups (p-value < 0.001).

In the case group, only 7% had gestational age ≤35 weeks, while in the control group, 23% had gestational age ≤35 weeks. Most patients in both groups had a gestational age of more than 35 weeks. Significant difference in gestational age between the case and control groups (p-value = 0.001). **Nazir S et al**[14] reported that the mean + S.D gestational age was 34.9+2.3 weeks with minimum and maximum were 27 and 39 in weeks respectively. In a comparative study **Zhang LY et al[16]** reported that gestation age at delivery (37.38±2.10 weeks) in case group and ((39.48±2.44 weeks) in control group (p<0.05).

In this study we noted that the fetal weight 2.5±0.2kg in case group was significantly lower than fetal weight 2.7±0.3kg in control group (p<0.05). Although there was no statistically significant difference between case and control groups terms of the number of birth asphyxia, neonatal morbidity, and neonatal death (p>0.05). In a comparative study **Zhang LY et al**[16] found that in cases with placenta premature aging, baby birth weights (2802.00±502.99g) were considerably lower in control group (3324.35±411.34g, p<0.01). In another study **Begum F et al[17]** reported that among the delivered babies 89% had birth weight of 2.5 to 3.9 kg, 8% had low-birth weight & 3% had weight of 4 kg or more.

Table/Figure 6: Compare the Placental grade in present study with other studies

		Grade-0	Grade-I	Grade-II	Grade-III
Petrucha AR et al [18](1982)		4.0%	48.0%	33.0%	15.0%
Mishra N et al[19] (2006)		0.0%	15.0%	58.0%	27.0%
Sunanda KM et al[20] (2014)		33.0%	24.0%	22.0%	17.8%
Vidyarthi A et al[21] (2017)		0.0%	8.0%	61.0%	31.0%
Nazir S et al[14] (2017)		0.0%	18.0%	56.0%	26.0%
Begum F et al[17] (2020)		6.5%	25.0%	35.5%	33.5%
Naik Aet al[22] (2021)		0.0%	0.0%	21.0%	79.0%
Gupta N et al[23] (2022)	**Case**	0.0%	7.5%	30.0%	62.5%
	Control	0.0%	12.8%	50.0%	37.5%
Present study	**Case**	0.0%	5.0%	52.0%	43.0%
	Control	0.0%	10.0%	60.0%	30.0%

The majority of patients in both groups had a placental grading of II, with 52% in case group and 60% in control group. Placental grading III was observed in 43% of the case group and 30% of control group. Non-significant difference in placental grading between two groups (p-value = 0.207). In a similar study **Naik A et al**[22] reported that when compared to the previous research, it was discovered that the placental grades of 39% and 61% of the control group's women and 21% and 79% of the study group's women, respectively, were statistically not significant (p>0.05).[18,19,20] **According to Saliha et al[24]** the impact hypertension on development procedure of placenta is identified by ultrasonography. 100 pregnant women were included. 50 normotensive and 50 hypertensive ladies were analysed by ultrasonography at three periods. Initially between 29- 32 weeks growth, second between 33-35 weeks and third following 36 weeks till 40 weeks development were included. The result, G II and G III placenta was 27 of 50 (54%) and 2 of 50 (4%) at third trimester.
Changes in placenta associated with the "aging" phenomenon are most likely a result of maturation and are closely related to pleacenta's ongoing growth. Numerous elements influence the fetus's health, but the most crucial one for creating a healthy child is a healthy placenta. Many people have the strongly held belief that the placenta ages gradually throughout a typical pregnancy and is about to enter a state of morphological and functional senescence.[10] According to our research, prenatal morbidity and mortality increased in grade three and were marginally greater in case group compared to control group (p>0.05). **Krielessi V et al[25]** estimate that the prevalence of hypertension illnesses complicating pregnancy, along with bleeding and infection, significantly contributes to the morbidity and death of both mothers and fetuses. Maternal fatalities range from 2.6% to 7.6% due to hypertension. **Begum F et al**[17] reported that the 75% of the respondent mother had normal healthy fetal outcome, 19.5% had outcome of asphyxiated babies and 5.5% had still births or IUD. **Backes CH et al[26]** revealed that preterm delivery, fetal growth restriction, low birth weight, placental abruption, caesarean delivery, liver insufficiency, sub capsular liver hematoma, cerebral edema, renal failure, thrombocytopenia, and intravascular coagulation are among the many complications associated with preeclampsia-complex pregnancies. **Misra DP et al[27]** said that anomalies in the placenta have been linked to the problems of hypertensive diseases in pregnancy. As a result, there is a lot of curiosity in the hypertensive woman's placenta. **Afzal E et al[28]** reported that the antagonistic perinatal results including development, limitation and still birth was higher in hypertensive untimely conveyances placental infarcts in typical full-term deliveries.
Thus, ultrasound reviewing arrangement of placenta light of its development. This essentially influences the degree of calcifications. Placental insufficiency brought on by long-term hypertension is linked to placental grade III maturity. This may lead intrauterine growth restriction (IUGR), fetal distress, abnormal fetal growth, and hyaline membrane disease. Birth weight depends on the mother's body size and growth of placenta.[2] In hypertensive pregnancy, the preterm placental calcifications have adverse effects on uteroplacental blood flow, fetal growth and fetal death.[3]

Limitations of the study:

1. Our study was single center with small sample size , which may not be representative of the larger population.
2. The study did not monitor the long-term survival of neonates after discharge from the Special Newborn Care Unit (SNCU).

Conclusion

In conclusion in both groups, it was found that placenta is mirror image of the fetal outcome. Examination of placenta by ultrasonography, there is definite evidence of changes in placental morphology and grading in pregnancy-induced hypertensive mothers. There is accelerated placental grading in hypertensive mothers and is associated with adverse perinatal outcome. In the obstetric community, ultrasound placental grading may be utilized as a screening method for

antepartum fetal monitoring. The obstetrician may be alerted to the emergence of maternal and neonatal problems related to hypertension if early and accelerated placental maturation is detected. Placental grading will assist us in reducing maternal and perinatal difficulties by assisting with early diagnosis, formulating a plan of care, and implementing prompt intervention.

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