



Quantitative Analysis of Gross Morphology of Placenta in Normal and Hypertensive Patients

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Background: Placenta is an important organ and the growth and development of fetus is dependent on it. The different pregnancy complications as antepartum haemorrhage, postpartum haemorrhage, coagulopathy etc. in mothers and IUGR (intrauterine growth retardation), prematurity etc. in foetus are reflected in the placenta & are associated with pregnancy induced hypertension (PIH) . They form the major causes of maternal and fetal mortality & morbidity.

Objectives:

Quantitative analysis of the changes in gross morphology of placenta in normal and hypertensive pregnancies.

To study the resultant effect on the foetal outcome.

Material & Methods: We had taken 100 placentas of normal & hypertensive group patients each for study .We collected them from the labour room & operation theatre of Dept. of Obstetrics & Gynaecology of our medical college. Gross morphological features of placentas like size, weight, no. of cotyledons , areas of infarction, retroplacental clot, calcification and insertion of umbilical cord etc. were noted and simultaneously the histological study was also done.

Observations & Results: The gross morphological parameters like diameter, size, surface area,

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number of cotyledons and weight were reduced in hypertensive placentas than normal placentas while areas of infarction, retroplacental clot, calcification were more in hypertensive placentas than normal placentas. ($p < 0.005$) The observed differences were statistically significant.

Keywords: PIH(Pregnancy Induced Hypertension); infarction; retroplacental clot; calcification; intrauterine growth retardation; preterm delivery.

1. INTRODUCTION

Each and every organism exhibits reproduction as a feature. We all know that 'mother and foetus' are the two important ends of this process. The existence of foetus in the intrauterine life is dependent on the important organ i.e. 'Placenta'.

The hypertensive disorder in pregnancy also known as preeclampsia is a multisystem disorder having symptom complex of raised blood pressure, oedema and / or proteinuria [1] and affects 5-7% of all pregnancies worldwide.

Generally we study the hypertensive disorders in pregnancy as five types :

- Gestational hypertension
- Preeclampsia
- Eclampsia
- Preeclampsia superimposed on chronic hypertension
- Chronic hypertension

The development of placenta as well as the development of foetus is affected by the PIH. The size and weight of placenta decreases as pregnancy induced hypertension severity increases [2].

There is also a significant rise in the incidence of intrauterine growth retardation and still birth when the placental weight is on lower side. With low placental weight, low birth weight is observed [3]. Preeclampsia begins to abate with the delivery of the placenta because the root cause of preeclampsia is the placenta i.e. presence of trophoblast tissue [4].

It is generally believed that in preeclampsia there is impairment of trophoblast invasion. So "preeclampsia" is associated with abnormally shallow placentation [5]. That means the hypoinvasive placental phenotype is the characteristic of preeclampsia [6].

This reduced invasion of the trophoblasts is supposed to be the main important factor in the

development of intrauterine growth retardation of the foetus and preeclampsia in mother.

This ultimately results into unfavorable outcome of the pregnancy increasing the maternal and perinatal morbidity and mortality. In this study, we observed gross changes in hypertensive placentas so as to compare them with the placentas of normal pregnancies both quantitatively and qualitatively and also study their effect on foetal outcome.

2. MATERIALS AND METHODS

The placentas were collected from the labour room and operation theatre of Department of Obstetrics and Gynaecology of our medical college. The gross features of the placentas such as weight, size, infarction, calcification or any retro placental clot were noted. Foetal gross observation done and findings noted like APGAR score, weight, reflexes, maturity etc. APGAR score is calibrated by colour, cry, activity, heart rate and muscle tone of the baby. Low APGAR generally denotes birth asphyxia of the baby.

Also, for the histological study, biopsies of 5mm. size were taken from the centre as well as from the peripheral part of each placenta.

The following steps were followed:

- 1] Fixation
- 2] Dehydration
- 3] Clearing
- 4] Wax Impregnation After this the tissues underwent Section Cutting:(5-7 micron thick sections were cut.)

& Staining: Following stains were used for the present study:

- i Haematoxylin and Eosin(H&E Stain) &
- ii. Masson's Trichrome Stain

3. OBSERVATIONS AND RESULTS

The present study entitled was carried out in the department of Anatomy. The placentas were

collected from the labour room and operation theatre immediately after parturition and stored in 10% formal saline and subjected to gross morphological study and subsequently tissues were collected for histological study. Two sets having 100 placentas each were taken for the study.

The Placentas were studied in the following Groups:

Group I - Normal Pregnancy
 Group II - Pregnancy with history of PIH (Pregnancy Induced Hypertension).

In our study, in hypertensive pregnancy 62 % of patients were below 36(32-36) weeks of gestation i.e. preterm and In normal group , 63 % were between 37-41 wks i.e. full term. This shows that the proportion of preterm gestation and prematurity is more in patients of hypertension in pregnancy.

Table 1. Distribution of study participants according to Gestational Age

Gestational age (weeks)	Mean(weeks)	SD	P value
Normal Patients	38.22	1.750	0.000
Hypertensive Patients	36.79	2.281	

Distribution of study participants according to Gestational Age

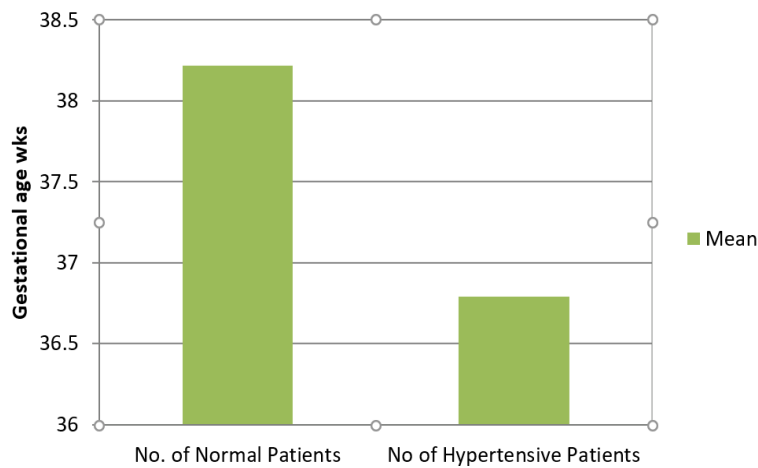


Fig. 1. Observed difference was statistically significant
t value = 4.97 , P value = 0.000

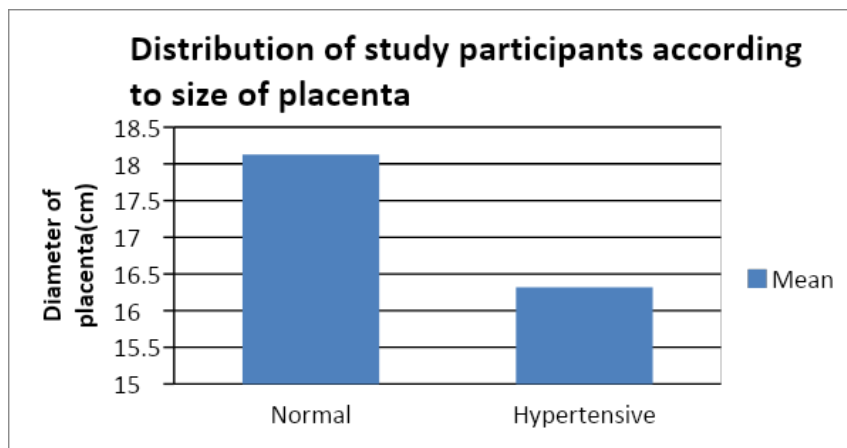


Fig. 2. Observed difference was statistically significant

Table 2. Distribution of Study participants according to size of placenta

Size of Placenta (cm)	Mean(cm)	SD	P value
Normal	18.13	1.81	
Hypertensive	16.32	2.14	0.000

Table 3. Distribution of Study Participants According To Weight of Placenta

Weight of Placenta (gm)	Mean weight (gm)	SD	P value
Normal	469.60	59.35	
Hypertensive	388.60	93.70	0.000

We found the mean diameter of the placenta in hypertensive patients is smaller i.e. 16 cm (2.14 sd) and that in normal placenta was bigger i.e.18 cm (1.81 sd)

We found the mean weight of the placenta in hypertensive patients is smaller i.e. 388 gm (93.70 sd) and that in normal placenta was bigger i.e.469gm (59.35 sd)

So, when we studied the morphology of placenta, we recorded the weight (gm) and the size i.e. diameter (cm) of placenta and observed that in both the parameters, the values were lower in

the hypertensive group as compared with the normal.

In normal group 63% placentas had above 20 cotyledons and 37% had below 20 of cotyledons . According to our study ,we observed that in hypertensive group there were less no of cotyledons i.e. only16% placentas had above 20 cotyledons while 84% of the placentas had below 20 cotyledons. (some even had **less than 15** cotyledons.) So the mean no. of cotyledons in normal group was 21. 45 and in hypertensive group was 18. 28.

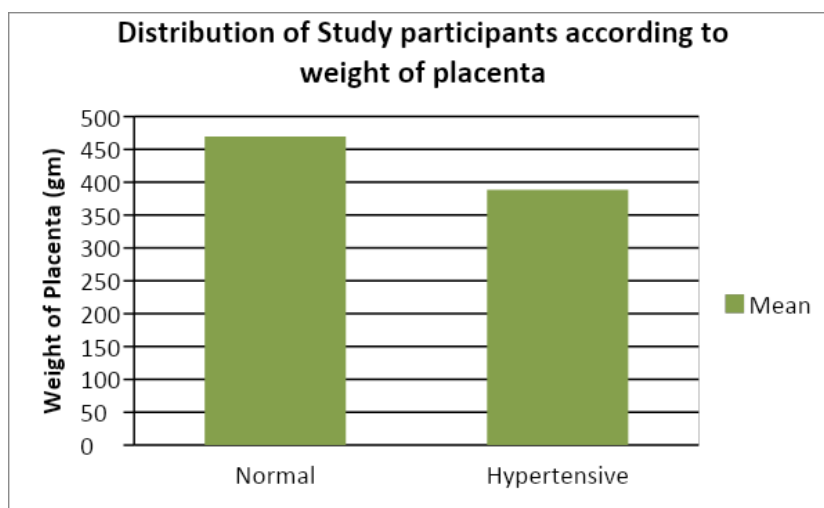


Fig. 3. Observed difference was statistically significant

Table 4. Distribution showing mean Number of cotyledons in normal & hypertensive patients

No. of Cotyledons	Mean	SD	P value
Normal	21.45	2.45	
Hypertensive	18.28	2.62	0.000

Table 5. Distribution showing insertion of umbilical cord in normal & hypertensive

Insertion of umbilical cord	Normal(%)	Hypertensive(%)
Central	91	80
Eccentric	8	2
Marginal	1	18

Chi square test=19.2 df =2, p value =0.00005



Fig. 4. Small atrophic placenta

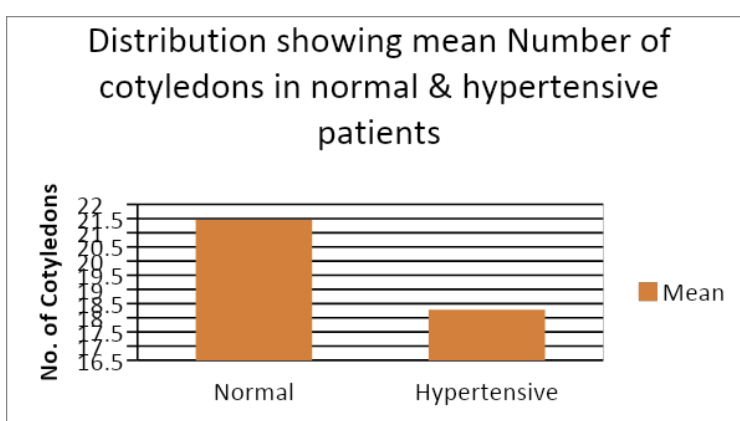


Fig. 5. Observed difference was statistically significant

In **normal** patients 91% patients had central insertion of cord and 8% patients had eccentric insertion of cord and **1% only with marginal insertion**. While in **hypertensive** patients there were 80% placenta with central insertion of cord, 2% with eccentric and **18% with marginal insertion**. We noted that **marginal insertion was a characteristic feature of hypertensive placentas**.

When we inspected the maternal surface of the placentas we found, there was only **one%** patient

with retroplacental clot in the **normal** group and there were **14 %** patients with retroplacental clot in **hypertensive** group.

In our study, **33** out of 100 patients were having calcification in **normal** group, while in **hypertensive** group, there were **47** patients with calcification of the placenta.

The mean birth weight in newborns of normal group was 2830 gm and in hypertensive gp was 2350 gm.

Table 6. Distribution showing Retroplacental clot in normal & hypertensive patients

Retroplacental clot	Normal(%)	Hypertensive(%)
Present	1	14
Absent	99	86

Chi square test = 10.38 P value = 0.0006

Table 7. Distribution showing Infarction in normal & hypertensive patients

Infarction	Normal(%)	Hypertensive(%)
Present	4	44
Absent	96	56

Chi square test = 41.69 P value = 0.00001

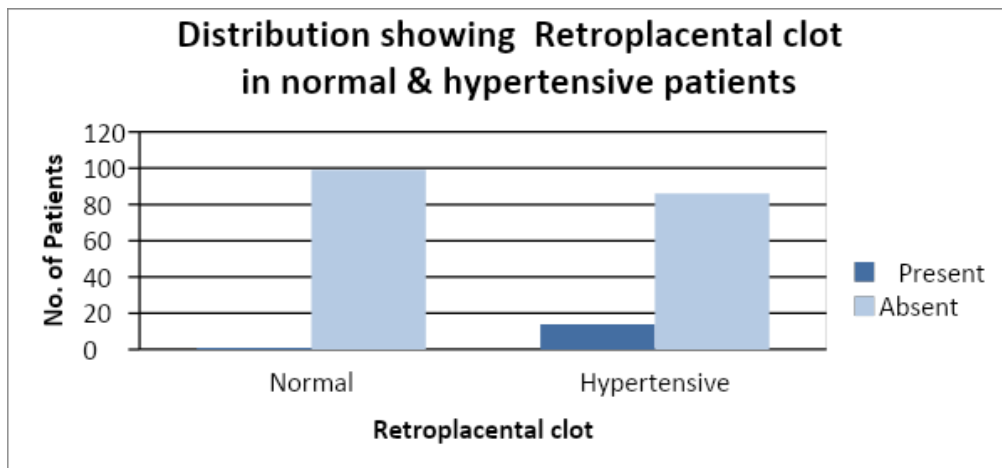


Fig. 6. Observed difference is statistically highly significant



Fig. 7. A large retroplacental clot on maternal surface

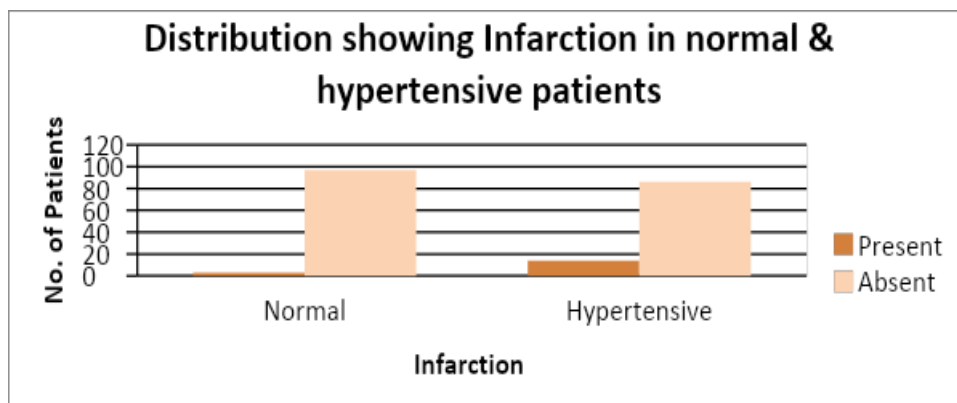


Fig. 8. Observed difference is highly statistically significant

Table 8. Distribution showing Calcification in normal & hypertensive patients

Calcification	Normal(%)	Hypertensive(%)
Present	33	47
Absent	67	53

Chi square test=3.5 P value =0.03



Fig. 9. We see here the white areas of infarction and the areas of hemorrhage near it

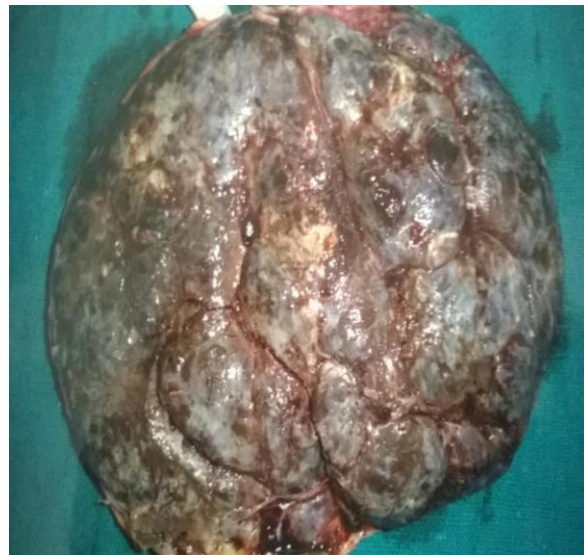


Fig. 10. Calcified placenta showing multiple white spots

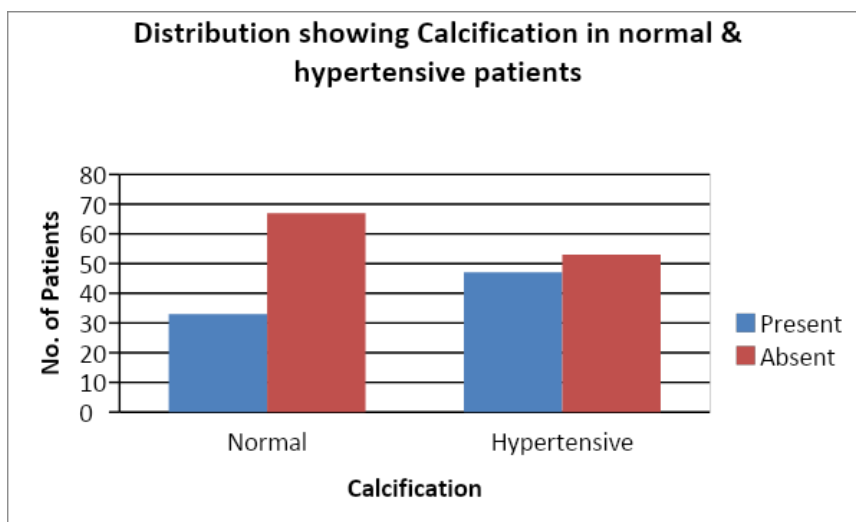


Fig. 11. Observed difference was statistically significant

Table 9. Distribution showing mean birthweight of newborn in normal and hypertensive patients

Birth weight(gm)	Mean (gm)	Std Dev	P Value
Normal	2830	46	0.000
Hypertensive	2350	56	

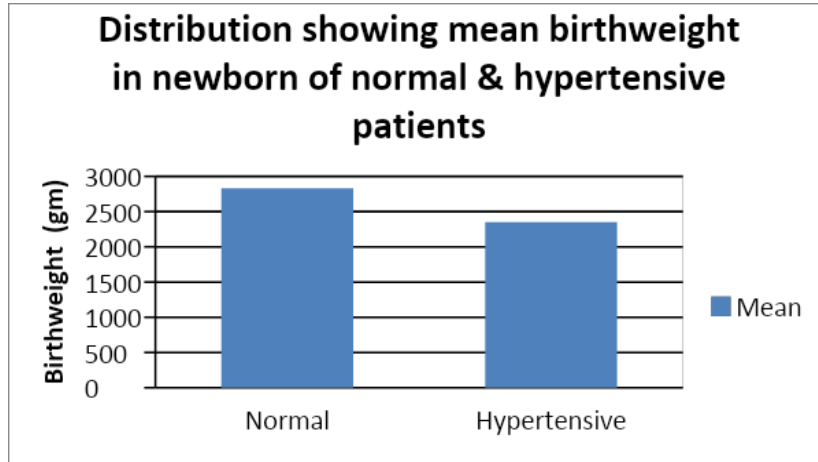


Fig. 12. Observed difference was statistically significant

Table 10. Distribution showing asphyxia at birth in normal & hypertensive Asphyxia Normal(%) Hypertensive(%)

Present	5	44
Absent	95	56
Total	100	100

Chi square test =39.03, P value = 0.0001

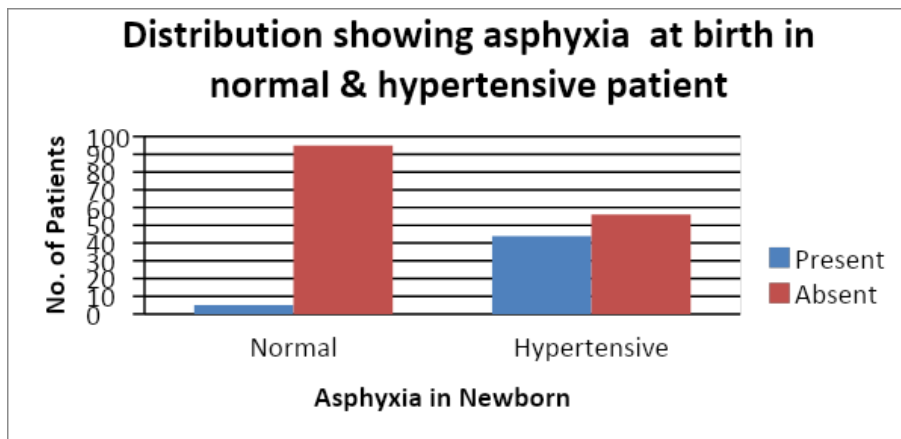


Fig. 13.

Table 11. Distribution showing stillbirth in normal & hypertensive patients

Stillbirth	Normal (%)	Hypertensive (%)
Present	2	15
Absent	98	85
Total	100	100

Chi square test =9.25 P value = 0.001

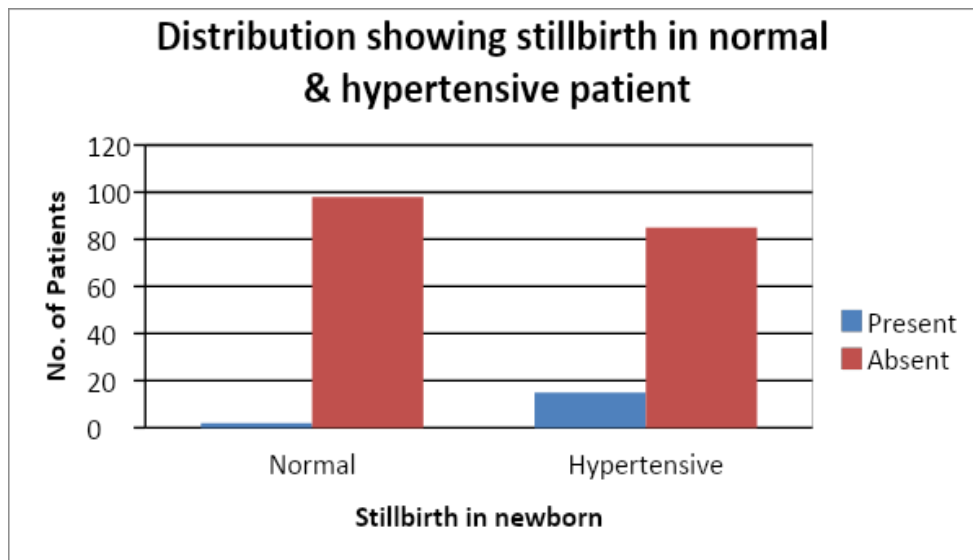


Fig. 14.

In above table, we observed that no. of babies with asphyxia in normal patients was only 5% and in hypertensive patients was 44%. So the proportion of asphyxia in newborn in hypertensive patients was more as compared with normal patients.

In our study, there were only 2% stillbirth babies in normal patients & in hypertensive patients no. of stillbirth babies were 15%. So the proportion of stillbirths was higher in hypertensive patients as compared with normal.

4. DISCUSSION

Various authors have studied the gross morphological features of placentas. In our study, the gestational age at the time of delivery ranged from 32-41 weeks in both groups. It was observed that 62% of PIH cases had preterm delivery (below 36 wks). This suggests that, maximum patients of the hypertensive group were of preterm gestational age. Our findings were similar to maham aklaq [7] and vassliki krivelessi [8].

Figen Barut And Aikut Barut [9] in their study mentioned that average placental weight in normal patients was 508 gm and in hypertensive patients was 404 gm. S. kotgiwar and N.Ambiye [10] and K. Devishankaran [11] found that in hypertensive patients the average placental weight was 291gm and in normal patients it was 573 gm. The placental weight was 427 gm in

mild PIH and 374 gm in severe PIH as was found by Vissiellekki kressie [8].

According to Thomson et al. [12] in 1969, birth weight of foetus and weight of placenta both were below average in preeclampsia but their ratios were slightly increased. Udanja Jain in 2001 [13] and Pradeep Londhe [14] in 2011, in their study found similar finding of reduced placental weight in hypertensive group than normal group.

In our study, average placental weight was 388 gm for hypertensive patients and 469 gm for normal patients.

Davies et al. [15] and Murthy et al. [16] both were of the opinion that the placental sizes assessed by area, weight or volume were significantly correlated with the weight of the neonates in case of normal delivery. The growth of the foetus is dependent upon its nutrition coming from maternal blood. The size and the surface area of the placenta available for diffusion of nutrients from mother to foetus also has effect on the foetal weight.

There were 3.5% mean areas of infarction in placentas in normal patients while 17.4% were the mean areas of infarction in hypertensive patients as studied by Dr. Pradeep Londhe [14].

There were 3.77% & 16.5% areas of infarction in normal patients and hypertensive patients respectively according to the study done by Mujumdar S. and H. Dasgupta [17].

In our study, the areas of infarction were 4% in normal patients and 44% in hypertensive patients.

The deposits of calcification were found as white or pale fine granules or clumps on the placenta. Our findings were nearly similar to Fox [18] in 1964 who found 24.6% calcification in normal group. Pushpa Goswami et al. [19] in 2012, found that mean number of calcified areas of placentas in PIH were more than those found in normal patients. Majumdar S .and Dasgupta H [17] found that mean calcified areas in placenta $4.13 + 1.15$ in normal as compared to $33.3 + 3.15$ number in hypertensive patients which were significant. Our findings were nearly similar with the above findings with 33 out of 100 patients were having calcification in normal group, while in hypertensive group, there were 47 patients with calcification of the placenta and we had significant "p value".

S. Kotgirwar, M. Ambiyee [10] in 2011, found out of 30 cases, incidence of eccentric insertion of cord was 18 in hypertensive group & in normal group, it was 14. However, *Deeplaxmi Salmani [20] in 2012, found that the eccentric insertion was more i.e. 82% in hypertensive patients while central insertion was less i.e. 18%. Pradeep Londhe [14] in 2011 found that, 11.3% of marginal insertion of cord was in hypertensive and 2.7% was in normal patients. In our study, in hypertensive group maximum no. of patients had central insertion while next common type was marginal 18% and in control group, maximum no. was central insertion followed by next common type eccentric 2%.

Our findings were similar to Fox [21] in 1978, who reported a higher incidence of retroplacental haematoma in PIH. Gunnapriya Raghunath, et al. [22] observed, 3 cases of abruption with retroplacental clots in PIH among 101 cases. Aparna Narsimha Rao [23] studied the correlation between retroplacental clots with severity of hypertension and found retroplacental clots in 22.22% cases in placentas of eclampsia & 18.5% cases in severe preeclampsia and none in placentas of normal patients. Das et al. [24] in 1996 found an incidence of 10% cases of retroplacental haematoma in the PIH group.

In our study, retroplacental clots in PIH was 14% compared to only 1% in normal group which was statistically significant ($p < 0.005$). Abbasi RM, et al. [25] found 3-4 fold increase in perinatal mortality in PIH which was a leading cause of stillbirths in 45% of the patients as reported by

Patil Yet al. [26] & perinatal mortality was 52.0% in PIH (hypertensive patients) as compared to 29.8% in normotensive. Few evidences can be recollected from a number of studies [27-36].

Our study also shows hypertensive disorders in pregnancy affect the perinatal outcome in the form of IUGR, increased no. of asphyxiated and stillborn infants. So, the perinatal mortality and morbidity was increased in hypertensive pregnancies in our study also.

5. CONCLUSION

Our study was aimed to correlate the morphological changes of the PIH (hypertensive) placentas with the outcome of pregnancy and also comparison with those of the normal group. The birth weight of fetuses in hypertensive group were on lower side than the fetuses in the normal group and the APGAR score was also lower than normal. Low APGAR generally denotes birth asphyxia of the baby.

These all studies were statistically significant. The gross changes in placentas with history of PIH were retroplacental haematoma, areas of infarction and calcification, scanty no of cotyledons. reduced size, weight and surface area of the placenta and these all changes were noted in more no. in hypertensive group than in normal group. Definitely all the above observations lead to the conclusion that there was unfavorable outcome of pregnancy in hypertensive patients. The intranatal and perinatal asphyxia was increased in hypertensive pregnancies. We know that the placenta is the mirror image of antenatal period and provides information about it. So, it is very important that we should perform the examination of placenta adequately after every delivery as a routine procedure.

CONSENT

As per international standard or university standard, patients' written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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