

A Study Evaluating Morphology of Placenta and Fetal Outcomes in Hypertensive Pregnancies

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Abstract

Normal formation, full development and functions of the placenta determine the survival and growth of foetus. Study of placenta gives insight into the health condition of fetus. *Materials & Methods:* A total of 500 subjects, each in hypertensive and normotensive group were included in this study conducted in Anatomy Department of SBKS Medical College and Research Centre, Vadodara. *Results:* In the hypertensive groups, 80% females were primigravida while 20% females were multigravida. In the hypertensive group, there was significant increase in the number of deliveries occurring at pre-term (35%) and a significant proportion of deliveries were by lower section caesarean section (45%). Mean birth weight of the babies was 2.82 ± 0.43 in control and 2.33 ± 0.84 kg in hypertensive group. On examining the morphometry of the placentas, it was observed that mean placental weight was 541.4 ± 30.62 g in control group as against 478.4 ± 30.62 g in the hypertension group. The mean placental diameter was 19.87 ± 1.47 cm in control group while it was 17.87 ± 1.47 cm in another group. In the hypertension group the placental area, volume and thickness were significantly less as compared to control group. Mean number of cotyledons were 18.46 ± 1.54 in control group as against 16.46 ± 1.54 in hypertension group. Fetoplacental ratio and placental co-efficient were 5.23 ± 0.86 and 0.19 ± 0.02 in control group and 4.88 ± 1.79 and 0.17 ± 0.06 in the hypertension group. *Conclusion:* Hypertensive disorders of pregnancy have significant effect on the morphology and morphometry of placenta and also influences the fetal outcomes.

Keywords: Placenta; Fetus; Morphology; Morphometry; Hypertensive Pregnancies.

Introduction

Reproduction has two important aspects: the Fetus and the Mother that are connected to each other by a vital organ "the placenta" [1]. Normal formation, full development and functions of the placenta determine the survival and growth of foetus [2]. As the pregnancy progresses, the placenta undergoes morphological and morphometric changes such as changes in weight, volume, structure, shape and function in order to support the prenatal life [3]. Placenta is a reflection of maternal and fetal status. It is a report of the infant's prenatal experience. Thus the complications of pregnancy that are associated

with high perinatal morbidity and mortality are reflected in a significant way in the placenta.

Preeclampsia is an important complication of pregnancy that affects anywhere between 5 and 10% of pregnancies and leads to maternal and fetal morbidity and mortality. It is unique to pregnancy and its origin is unclear and is one of the leading cause of maternal and/or fetal death [2,3].

Pre-eclampsia is associated with hypertension and proteinuria. Its primary cause is cytotrophoblast cells apoptosis as a result of which decidua is penetrated only superficially which causes anywhere between 30 to 50% of spiral arterioles in placental bed to escape remodeling by trophoblast [3,4]. These arterioles have intact myometrial segments, this in addition to unaffected adrenergic nerve supply causes these vessels to remain undilated. Thus the formation of freely communicating sinusoids that is pertinent to adequate blood flow is hampered. It is observed that in women with preeclampsia, the mean luminal diameter of uterine spiral arterioles is less than one third to that seen in normal pregnancies. There is resultant decrease in uteroplacental perfusion and infarction of placenta with progress of gestation and ultimately fetal hypoxia that causes IUGR which

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contributes to premature delivery and fetal death [5,6].

A thorough examination of placenta, gives information that is useful in management of complications in mother and the new born [6-9]. With these aspects in mind, this study was conducted. Present study has been undertaken to assess the morphology and histology of placenta from mothers with PIH and to correlate the findings with those from normal pregnancies.

Materials & Methods

This prospective, observational study was carried out in the Anatomy Department of SBKS Medical College and Research Centre, Vadodara and Department of Obstetrics and Gynaecology of Dhiraj General Hospital Dhiraj General Hospital, Vadodara, a tertiary health care centre after obtaining ethics committee approval. A total of 500 subjects with hypertensive pregnancies (test group) and 500 subjects with normotensive pregnancies (control group) that met following inclusion criteria and none of the exclusion criteria were included in the study:

Inclusion Criteria

Normotensive mothers and mothers with pregnancy Induced Hypertension (Pre-eclampsia and Eclampsia), gestational hypertension.

Exclusion Criteria

All other maternal conditions which lead to small placental size, placental infarcts and Intra-uterine growth retardation.

The subjects willing to participate in the study were explained about the purpose and method of the study in the language they understood and written consent was obtained before including them in the study. Confidentiality of participating mothers was maintained at all levels. Following delivery, on being informed by the department of Obstetrics and Gynaecology, placentae were collected and brought to the Anatomy Department. Placental collection continued till 500 placentae of normotensive mother and 500 placentae of hypertensive mother were collected.

Weighing machine for placenta, vernier caliper, bucket of water, tray, scalpel with surgical blade and graph paper were the materials used.

Placentae with membranes were collected. Amnion and chorion were trimmed from the placenta. Umbilical cord was cut 50mm from insertion site. This was followed by washing and mopping.

Gross assessment: weight of each placenta was measured using a weighing scale, surface area of the maternal surface of placenta was calculated after taking its impression on graph paper. Using a measuring tape, diameters were recorded along the two axes that were at 90 degree to each other (mean of two diameters was obtained); vernier caliper was used in calculating thickness; and area was estimated in cm^2 using the formula described by Davies and Beazley.

Placental volume was calculated using the Archimedes principle (water displacement method). Following parameters were recorded: shape, weight (gram), diameter (cm), site of umbilical cord insertion, number of cotyledons, presence or absence of calcification, fetoplacental ratio (calculated by dividing the fetal weight by the placental weight) and placental coefficient (placental coefficient is obtained when placental weight is divided by fetal weight).

Results

A total of 1000 subjects, 500 in normotensive and 500 in hypertensive groups (Table 1), in the age range 20 to 35 years, were evaluated for placental characters in the labour room of department of Obstetrics and Gynecology, Dhiraj general hospital and in the anatomy department of SBKS MIRC.

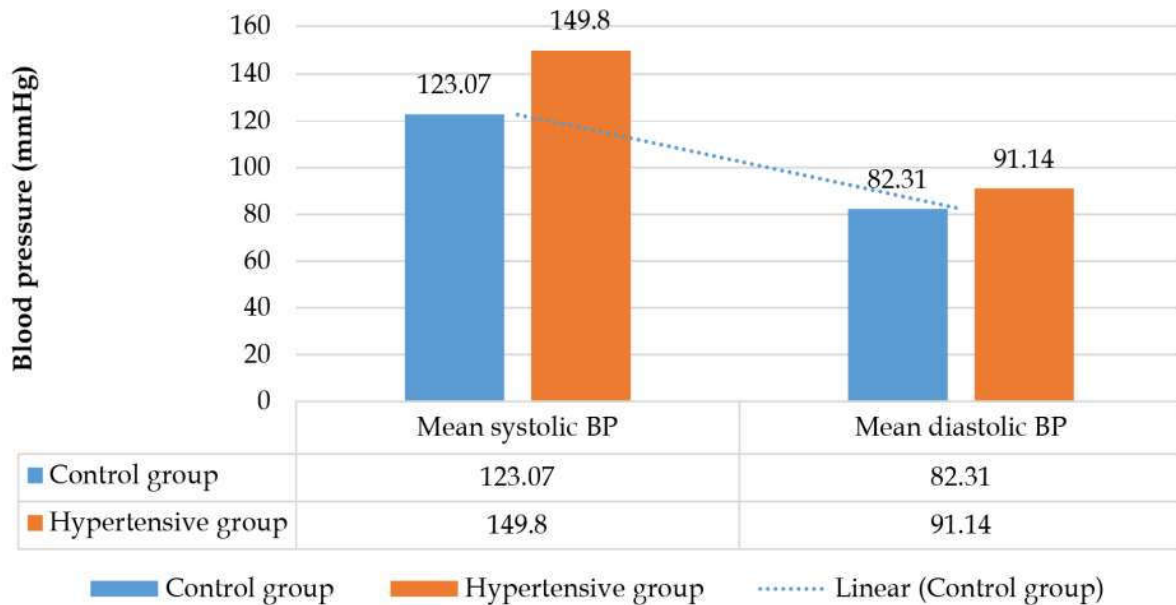
Majority of cases were in the age group of 20-25 years (46.2%) in control, whereas in hypertension group the majority of cases fell in the age group of 26-30 years (42.4%). In the normotensive group, 88.2% (n=441) were primigravida and 11.8% (n=59) were multi gravida. On the other hand in the hypertensive groups, 80% (n=400) females were primigravida while 20% (n=100) females were multigravida (Table 2).

The blood pressure in the control group was in the normal range mean systolic blood pressure was $123.07\text{mmHg} \pm 4.14\text{mmHg}$ and mean diastolic blood pressure was $82.31\text{mmHg} \pm 4.08\text{mmHg}$. In the hypertensive group majority patients had both systolic as well as diastolic blood pressure raised, while some patients had only raised systolic pressure and few had only raised diastolic pressure. The mean systolic blood pressure in the

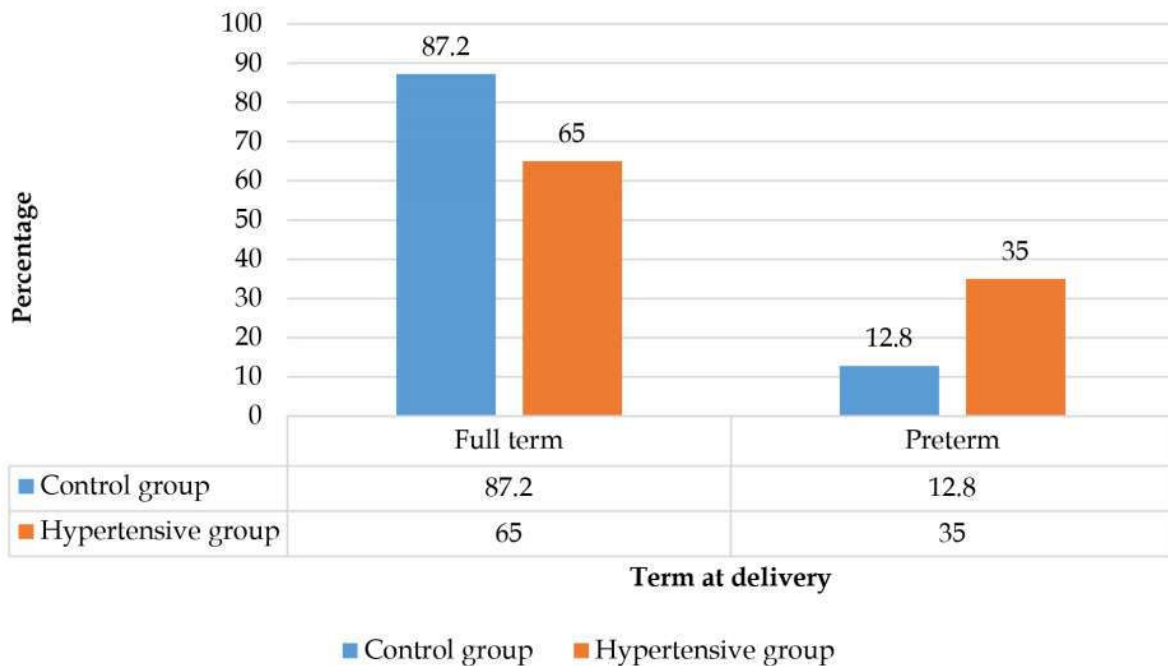
hypertensive group was $149.8\text{mmHg} \pm 7.48\text{mmHg}$, while mean diastolic blood pressure was $91.14\text{mmHg} \pm 4.14\text{mmHg}$. (Graph 1). In the control group majority of the deliveries completed full term (87.2%) while in the hypertensive group there was significant increase in the number of deliveries occurring at pre-term (35%) (Graph 2).

In the control group majority of the deliveries (96%) were by vaginal route, while in the hypertensive group a significant proportion of deliveries were by lower section caesarean section (45%) (Graph 3).

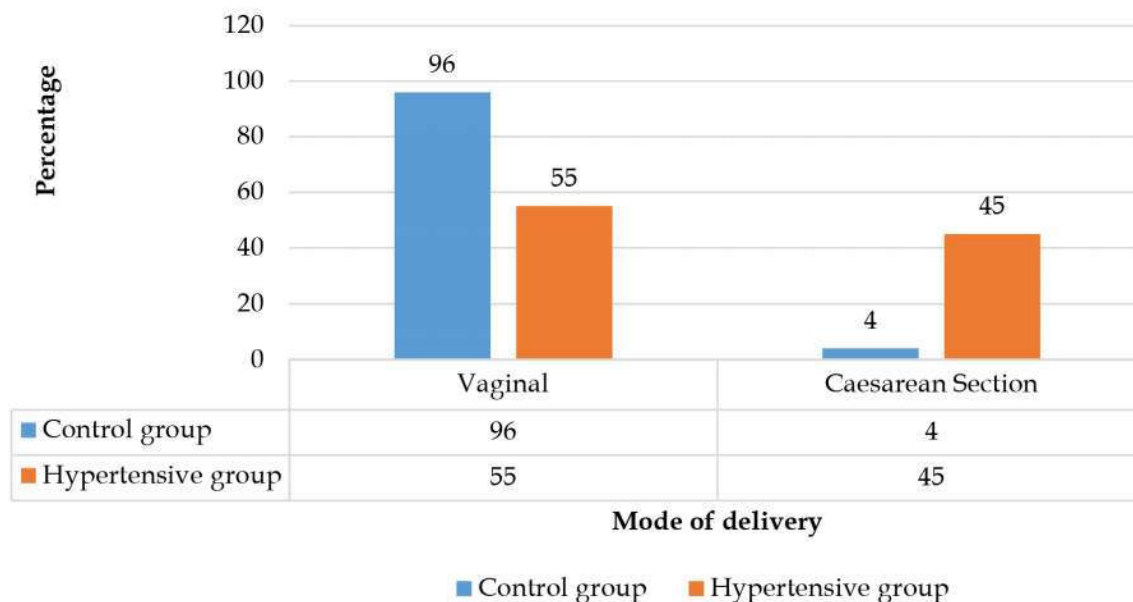
Majority of the patients in both group gave live birth viz., 98.6% in control group and 90% in



Graph 1: Mean blood pressure in Control and Hypertensive group



Graph 2: Term of delivery



Graph 3: Mode of delivery

hypertension group. Intra uterine death was seen in 10% of the deliveries in hypertension group. Fetal outcomes were as show in table III. Mean birth weight of the babies was 2.82 ± 0.43 in control and same was 2.33 ± 0.84 kg in hypertension group. Mean APGAR score at birth was 7.7 ± 1.38 in control group while the same was 6.32 ± 2.23 in hypertension group; mean APGAR score at 5 minutes was 8.1 ± 2.82 in hypertension group as compared to 9.23 ± 1.38 in control group. A significant proportion of new borns of hypertensive mothers (58.6%) required NICU admission.

In the control group majority of the new born had weight between 2.6 to 3 kg, while in the hypertension group majority of the patients had weight between 2 to 2.5kg.

When the attachment of the placenta was studied, it was observed that in control group majority of the placental attachment was eccentric (51.2%) while in the hypertension group majority of the placentas were attached marginally (46.8%). The data on the location of insertion of umbilical cord in three groups are presented in figure IV. In the control group (71.8%) as well as the hypertension group (59.2%) majority of the placentas were discoid in shape as show in table IV.

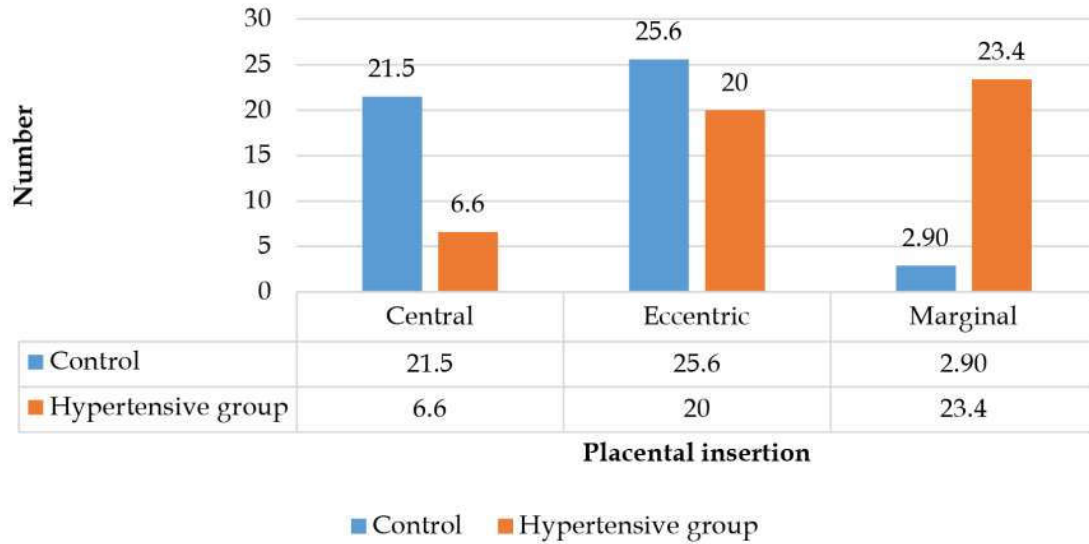
On examining the morphometry of the placentas, it was observed that mean placental weight was 541.4 ± 30.62 g in control group as against 478.4 ± 30.62 g in the hypertension group. The mean placental diameter was 19.87 ± 1.47 cm in control group while it was 17.87 ± 1.47 cm in another group.

Table 1: Distribution of cases

Normotensive group		Hypertensive group				Total			
No.	%	Gestational Hypertension No.	%	Pre-eclampsia No.	%	Eclampsia No.	%	No.	%
500	50	125	12.5	250	25	125	12.5	500	50

Table 2: Gravida status of the patients enrolled

Gravida status	Normotensive group		Hypertensive group	
	No.	%	No.	%
Primigravida	441	88.2	400	80
Multi gravida	59	11.8	100	20
Total	500	100	500	100



Graph 4: Distribution cases according insertion of placenta

In the hypertension group the placental area, volume and thickness were significantly less as compared to control group. Mean number of cotyledons were 18.46 ± 1.54 in control group as against 16.46 ± 1.54 in hypertension group (Table 5).

In the control group majority of the placental weight was between 500 to 550g while in the hypertension group majority of the placental weight was between 451 to 500gm (Table 6).

Table 3: Fetal Outcomes

Parameters	Control group	Hypertensive group
Live Birth	493 (98.6%)	450 (90%)
IUD	7 (1.4%)	50 (10%)
Birth weight (kg)	2.82 ± 0.43	2.33 ± 0.84
Mean Apgar score at birth	7.7 ± 1.38	6.32 ± 2.23
Mean Apgar score at 5 minutes	9.23 ± 1.38	8.1 ± 2.82
NICU admission required	14 (2.8%)	293 (58.6%)

Table 4: Placental Shape

Shape	Control	Hypertensive group
Discoid	359 (71.8%)	296 (59.2%)
Heart	86 (17.2%)	42 (8.4%)
Oval	52 (10.4%)	85 (17%)
Irregular	3 (0.6%)	77 (15.4%)

Table 5: Placental Morphometry

Parameters	Control group	Hypertensive group	p-value
Mean placental weight (g)	541.4 ± 30.62	478.4 ± 30.62	<0.05
Mean placental diameter (cm)	19.87 ± 1.47	17.87 ± 1.47	<0.05
Mean placental area (cm ²)	266.24 ± 19.75	204.77 ± 16.89	<0.05
Mean placental volume (cc)	425.19 ± 31.53	239.02 ± 19.74	<0.05
Mean placental thickness (cm)	2.16 ± 0.16	2.01 ± 0.11	<0.05
Mean number of cotyledons (n)	18.46 ± 1.54	16.46 ± 1.54	<0.05

Table 6: Distribution of cases according to placental weight

Placental weight	Control group	Hypertensive group
400-450	0	106 (21.2%)
451-500	62 (12.4%)	235 (47%)
501-550	236 (47.2%)	159 (31.8%)
551-600	202 (40.4%)	0

Table 7: Fetoplacental ratio and Placental coefficient

	Control	Hypertension group
Fetoplacental ratio	5.23±0.86	4.88±1.79
Placental coefficient	0.19±0.02	0.17±0.06

Table 8: Gross examination of placenta

Gross examination finding	Status	Normotensive group		Hypertension group	
		N	%	N	%
Calcification	Present	179	36%	269	54%
	Absent	321	64%	231	46%
Infarction	Present	63	13%	212	42%
	Absent	437	87%	288	58%
Placental cyst	Present	28	6%	84	17%
	Absent	472	94%	416	83%
Accessory placental lobe.	Present	34	7%	135	27%
	Absent	466	93%	365	73%

Fetoplacental ratio and placental co-efficient were calculated, in the control group they were 5.23±0.86 and 0.19±0.02 while in the hypertension group they were 4.88±1.79 and 0.17±0.06 respectively which was significantly less as compared to control group. (Table 7) Gross examination of placenta revealed the features shown in table 8.

Discussion & Conclusion

Placenta is the pathway for nutrient flow and gas exchange between the mother and the fetus. Any abnormality of the placenta significantly impacts the fetal growth. Hypertensive disorders of pregnancy exert profound impact on the morphology and morphometry of placenta and thereby have impact on fetal outcomes.

In the current study 500 normotensive and 500 hypertensive females were enrolled. In the hypertensive group, 25% had gestational hypertension, 50% had pre-eclampsia and 25% had eclampsia. In the study by Siva Sree Ranga. M.K et al., 40%, 56.7 and 3.3% for gestational hypertension, pre-eclampsia and eclampsia respectively [10]. In the

study by Kambale T et al., maximum of 57.7% cases belonged to mild PIH, 33.3% cases belonged to severe PIH, and minimum of 8.8% cases were of eclampsia [11]. In the study by Kheir AEM, et al., of the patients diagnosed with hypertensive disorder of pregnancy, 29 (42%) were pregnancy induced hypertension while 15 (21.7%) and 3 (4.3%) had preeclampsia and eclampsia respectively [12].

Patients in the age range of 20 to 35 years were selected for the study. Majority of cases were in the age group of 20-25 years (46.2%) in control, whereas in hypertension group the majority of cases fell in the age group of 26-30 years (42.4%). Similar to our study, in the study in Sudan, most of the women affected by hypertensive disorders of pregnancy were aged 26-30 years (36.2%) and 31-35 years (28.9%), indicating that the incidence of hypertension is higher in pregnant women in their 3rd and 4th decades [12]. Our findings were also comparable to the study by Siva Sree Ranga. M.K et al. maximum patients in the hypertensive group were in the age group of 25-29 years (53.33%) and in the normotensive group were in the age group of 20-24 years (50%) [10]. In the study by Kambale T et al., most cases belonged to 20-25 years age group, of which 16 cases were of mild PIH. Minimum numbers of cases were present in the age group of 30-35 years. There

Table 9: Comparison of Placental Morphometry with published literature

Parameters	Current study	Keche HA et al [20]	Kishwar a S et al [27]	Siva		Ghodke S et al [28]	Baloch AH et al [26]	B Vijayalakshmi et al [29]	Dadhich A et al [30]	Shevade S et al [31]									
				SreeRanga. M.K et al. [10]	Londhe PS et al [22]														
Control	Hypertension	Control	Hypertension	Control	Hypertension	Control	Hypertension	Control	Hypertension	Control	Hypertension								
Mean placental weight (g)	541.4 ± 30.62	478.4 ± 30.62	464.80 ± 40.21	387.00 ± 72.54	-	455.7 ± 59.9	516.7 ± 25.7	312.93 ± 70.14	401.80 ± 54.62	410.60 ± 67.86	320.00 ± 64.80	520.31 ± 39.02	307.12 ± 49.13	478.80 ± 292.122	371.70 ± 85.316	491.44 ± 46.14	326.20 ± 86.93	502 ± 58.42	430 ± 50.69
Mean placental diameter (cm)	19.87 ± 1.47	17.87 ± 1.47	18.53 ± 0.87	15.72 ± 0.98	18.80 ± 2.32	16.08 ± 2.08	14.1 ± 0.7	19.1 ± 0.9	-	-	-	16.39 ± 1.14	15.82 ± 0.92	20.33 ± 1.446	17.94 ± 1.963	17.77 ± 1.26	16.37 ± 1.37	18.7 ± 1.55	17.2 ± 1.70
Mean placental area (cm ²)	266.24 ± 19.75	204.77 ± 16.89	270.89 ± 27.47	194.85 ± 23.86	-	-	-	182.80 ± 57.47	212.48 ± 54.51	219.40 ± 40.46	185.04 ± 33.72	269.32 ± 15.87	179.93 ± 38.45	-	-	-	-	-	-
Mean placental volume (cc)	425.19 ± 31.53	239.02 ± 19.74	452.20 ± 44.01	374.40 ± 72.18	389.83 ± 81.45	292.80 ± 71.81	-	372.87 ± 150.36	439.48 ± 135.14	268.48 ± 79.01 (ml)	194.70 ± 81.49 (ml)	-	-	420.45 ± 140.816	238.20 ± 93.197	-	-	-	-
Mean placental thickness (cm)	2.16 ± 0.16	2.01 ± 0.11	1.96 ± 0.17	1.57 ± 0.19	1.59 ± 0.39	1.51 ± 0.37	1.9 ± 0.9	2.4 ± 0.1	-	-	-	2.11 ± 0.31	2.04 ± 0.22	2.02 ± 0.199	1.77 ± 0.423	2.10 ± 0.60	1.79 ± 0.27	2.3 ± 0.43	1.8 ± 0.49
Mean no. of cotyledons (n)	18.46 ± 1.54	16.46 ± 1.54	-	-	15.77 ± 2.80	14.30 ± 2.47	16.1 ± 2.5	19.1 ± 1.2	-	-	-	17.10 ± 0.98	16.92 ± 0.91	10-15	16-20	17.72 ± 1.70	14.36 ± 1.82	18.9 ± 1.98	16 ± 2.29

Table 10: Fetoplacental ratio of current study as compared to available literature

Current study		Keche HA et al [20]		Siva SreeRanga. M.K et al. [10]		Londhe PS et al [22]		Salmani D et al [34]		B Vijayalakshmi et al [29]	
Cont rol	Hyperten sion	Cont rol	Hyperten sion	Cont rol	Hyperten sion	Cont rol	Hyperten sion	Cont rol	Hyperten sion	Cont rol	Hyperten sion
5.23±0.86	4.88±1.79	6.05 ± 0.07	5.51 ± 0.18	5.5 ±1.9	5.6± 0.7	6.79± 2.04	7.23± 1.90	5.72 ± 0.93	6.35± 2.05	5.89±0.769	6.40±0.888

Table 11: Calcification and infarction seen in placenta

	Current study		Siva SreeRanga. M.K et al. [10]		Narasimha A et al [35]	
	Control	Hypertension	Control	Hypertension	Control	Hypertension
Calcification	35.8%	53.8%	23.3	56.7	8.10%	77.7%
Infarction	12.6%	42.4%	6.7	36.7	10.8%	22.2%

was only one case of eclampsia above 30 years of age. In the control group, 30 cases belonged to 20-25 age group and three cases were present in the age group of 30-35 years [11].

Primigravida is one of the etiologic factors of PIH. In the current study in the normotensive group, 88.2% were primigravida and 11.8% were multi gravida. On the other hand in the hypertensive groups, 80% females were primigravida while 20% females were multigravida. There was no statistical difference in the two groups. In the study conducted in Kerela, in the hypertensive group 86.6% females were primigravida and in the normotensive group 93.3% females were primigravida [10]. In the study conducted in Grant Medical College, Mumbai the number of cases of PIH were more in primigravida group (24 cases) wherein 14 cases were of mild PIH and seven and three cases of severe PIH and eclampsia, respectively. In the control group, 18 cases were primigravida and 13 were second gravida [11]. In the study by Kheir AEM et al about 58% of the hypertensive women were multiparous [12].

In the study by Kumari P et al., the mean systolic blood pressure was 117.23±2.53mmHg and mean diastolic blood pressure was 69.32±2.48mmHg in the normotensive group and the mean systolic blood pressure was 146.00±2.70 mmHg and mean diastolic blood pressure was 94.83±3.13 mmHg in the hypertensive group [13]. In our study the blood pressure in the control group was in the normal range mean systolic blood pressure was 123.07mmHg±4.14mmHg and mean diastolic blood pressure was 82.31mmHg ± 4.08mmHg. The mean systolic blood pressure in the hypertensive group was

149.8mmHg±7.48mmHg, while mean diastolic blood pressure was 91.14mmHg±4.14mmHg.

In the control group majority of the deliveries completed full term (87.2%) while in the hypertensive group there was significant increase in the number of deliveries occurring at pre-term (35%). In the study conducted at Dr. SM CSI Medical College, Kerala, 33.33% deliveries in hypertensive group were pre-term, while all the deliveries in normotensive group were full-term. In the study by Adu-Bonsaffoh K et al. in Ghana, 80 (21.7%) pregnancies were delivered preterm [14]. In the study by Pokorna V et al, the mean duration of gestation was 37 (24-42; median, range) weeks in hypertensive mothers [15]. In the study by Kheir AEM et al the number of preterm pregnancies was high as 52% babies were preterm [12].

In the control group majority of the deliveries (96%) were by vaginal route, while in the hypertensive group a significant proportion of deliveries were by lower section caesarean section (45%). In the study by Pokorna V et al., 84% deliveries were by Caesarean section in hypertensive mothers [15]. Siva SreeRanga. M.K et al. in their study observed that in the hypertension group 60% deliveries were by vaginal route and 40% deliveries occurred by LSCS while all the deliveries in normotensive group were by vaginal route [10]. While in the study conducted in Sudan, 98.5% pregnancies were delivered by caesarean section [12].

Majority of the patients in both group gave live birth viz., 98.6% in control group and 90% in hypertension group. In the study by Siva

SreeRanga. M.K et al., all the patients in normotensive group gave live birth while in the hypertensive group 93.3% of pregnancies resulted in live birth. Allen VM et al. in their study had shown that women with any hypertension in pregnancy were 1.4 times more likely to have a stillbirth as compared with normotensive women [16]. Similar finding was observed by Ananth CV, et al., they also observed that the increased risk of stillbirth was higher in women having their second or higher order births compared with women having their first birth [17].

In the current study the mean birth weight of the babies was 2.82 ± 0.43 in control and same was 2.33 ± 0.84 kg in hypertension group. Rahman LA in their study had observed that there was a significant association of pregnancy-induced hypertension with low birth weight and that women who delivered low birth weight babies were 5 times more likely to have had pregnancy-induced hypertension [18]. However, on the other hand Xiong X in their study found that there were no differences in mean birth weight between women with gestational hypertension and women with normal blood pressure. Further analysis suggested that pre-eclampsia and gestational hypertension were associated with increased rates of both small-for-gestational-age and large-for-gestational-age infants. The majority of the babies born to mothers with different types of pregnancy-induced hypertension were appropriate-for-gestational-age or even large-for-gestational-age. Most babies born to mothers with severe pre-eclampsia or pre-eclampsia and gestational hypertension had similar fetal growth to those born to normotensive mothers [19]. In another study, the mean birth weight in babies born to hypertensive mothers was 2.5 ± 0.7 kg while that of babies born to normotensive mothers was 2.9 ± 0.4 kg. [10] In the study by Pokorna V et al, the mean birth weight was 2600 (370–4820)g [15]. In the study by Keche HA et al the mean birth weight was 2813.60 ± 258.06 g in control group, while it was 2141.00 ± 439.69 g in hypertensive group [20].

In our study Mean APGAR score at birth was 7.7 ± 1.38 in control group while the same was 6.32 ± 2.23 in hypertension group; mean APGAR score at 5 minutes was 8.1 ± 2.82 in hypertension group as compared to 9.23 ± 1.38 in control group. In the study conducted in Ghana, One and 5 minute APGAR scores < 7 occurred in 125 (34.0%) and 55 (14.7%) neonates respectively [14]. In the study by Kambale et al., only 6.6% neonates in normotensive group had low APGAR score at birth, while most the neonates in hypertensive groups had low APGAR score [11]. In another study, APGAR score at birth was 6.9 ± 1.8

in hypertensive group while it was 9 in normotensive group; APGAR score at 5 minutes was 8.5 ± 0.8 in hypertensive group while it was 10 in normotensive group [10]. In the study by Pokorna V et al., observed that Apgar score was negatively correlated to proteinuria, but not to the number of antihypertensives or other parameters [15].

In the present study a significant proportion of new borns of hypertensive mothers (58.6%) required NICU admission. Similar was the observation by Adu-Bonsaffoh K et al. in a study conducted in Ghana wherein 91 (24.7%) neonates were admitted to the Neonatal Intensive Care Unit, 56 (15.2%) had neonatal respiratory distress/asphyxia with 14 (3.8%) requiring ventilatory support. [14] In a study by Hubli M et al., as compared with normotensive pregnancies, hypertensive pregnancies that delivered at 35 and 36 weeks of gestation had higher rates of neonatal intensive care unit admission (33.3% vs 10.7%). The rate of neonatal intensive care unit admission (25.6% vs 8.7%) and duration of neonatal stay (3.9 vs 2.0 days) were greater in hypertensive pregnancies that delivered at 37 weeks of gestation [21].

In the present study, in the control group majority of the umbilical cord insertion was eccentric (51.2%) while in the hypertension group majority of the placentas were attached marginally (46.8%). This was similar to study by Pretorius where in marginal insertion of umbilical cord was reported in 42% of hypertensive cases as against 11.3% marginal insertion of placenta in hypertensive cases in the study by Londhe PS et al. [22, 23]. This lateral insertion placenta has impact on uterine artery resistance and development of preeclampsia and intrauterine growth retardation [7,24].

In our study in the control group (71.8%) as well as the hypertension group (59.2%) majority of the placentas were discoid in shape. In the study by Kishwara S et al., placenta was circular (43.3%) in majority of the patients in control group while it was oval (40%) in majority of the patients in hypertensive group [25]. While it was discoid in majority of the cases in both the groups in the study by Siva SreeRanga. M.K et al. [10]. This was also the case with study by Baloch AH where in most of the placentas were round to oval [26].

Hypertension during the pregnancy significantly impacts the morphology of the placenta. Decrease blood flow due to increased resistance impacts the overall development of placenta resulting in decrease in placental weight, area, diameter, volume and also number of cotyledons, this was comparable to published literature (Table 9).

In the control group majority of the placental weight was between 500 to 550g while in the hypertension group majority of the placental weight was between 451 to 500gm. In the study by Shevade S et al, majority of the placental weight in the control group was between 500-600g while in the hypertension group same was between 400-500g [31]. In the study by Patil GV et al., majority of the placental weight in the control group was between 500-600g while in the hypertension group same was between 300-400g [32].

In a study by Yadav SK et al. the placental co-efficient was 0.19 ± 0.02 in the hypertensive group, while it was 0.18 ± 0.02 in the control group, while in the current study the placental co-efficient was 0.19 ± 0.02 in control group and 0.17 ± 0.06 in hypertension group [33]. Due to resultant decrease in the fetal weight, there was decrease in fetoplacental ratio (FPR) and placental co-efficient in hypertensive group as compared to control group. In the reported literature, FPR was higher in the hypertension group as compared to control group, this was contrary to the findings of our study (Table 10, 11).

Thus a significant impact of hypertension on placental morphometry and morphology and its resultant impact on fetal weight as compared to normotensive patients was observed. This also suggests that study of the placenta can give an insight into the health status of the mother and fetus.

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