Original Article

A CROSS SECTIONAL MORPHOMETRIC STUDY OF HYPERTENSIVE WITH NORMAL PLACENTAE AND ITS CORRELATION WITH FETAL OUTCOME

Sabita Singh *[1], T S Gugapriya [2].

*[1] Dept. Of Anatomy, Velammal Medical College Hospital & Research Institute, Madurai, Tamil Nadu, India.

[2] Dept. Of Anatomy, Chennai Medical College Hospital & Research Centre, Trichy, Tamil Nadu, India.

ABSTRACT

Introduction: Placenta "the vital organ" for maintaining healthy pregnancy is unique in its development, being derived from both mother and fetus. Thus hypertensive disorder affecting mother has a deleterious impact on placenta which may lead to poor fetal outcome.

Aim: A cross-sectional descriptive study was undertaken to analyze and assess the morphological changes in hypertensive placentae and to clinically correlate it with fetal and maternal parameters.

Materials and Methods: 50 placentae were freshly collected from pregnancy induced hypertension (PIH) cases (study group) and 50 from normal pregnancy (control group). Study group was divided into three categories depending upon severity of the disease.

Observations and Results: 70% in study group were primigravida and were from rural area.46% were in age group of 15 – 20yrs.50% under low socio-economic status and 60% were without any regular antenatal check-up. Preterm, IUGR, still birth and neonatal death accounted to 10%, 12%, 18% and 10% respectively in study group. The average diameter, thickness, volume and number of cotyledons in study group were 15.91±2.11cm, 2.39±.54cm, 297.64±67.90ml and 10.02±4.13respectively. Mean placental weight was 376.41±17.198gm (mild PIH), 330.72±2.90gm (severe PIH), 329.73±3.19gm (eclampsia) and mean birth weight was 2680.29±198.46gm (mild PIH), 2212.06±36.41gm (severe PIH) and 2073.60±9.47gm (eclampsia) respectively in study group. Various pathological changes like retro placental hematoma, infarction and calcification had been noticed.

Conclusion: Placental morphometric parameters were significantly reduced (<.001) in study group as compared to control group. Decreased placental weight was associated with reduced birth weight and feto-placental ratio with increase in severity of hypertension significantly (<.001). A significant increase (<.05) in incidence of preterm, IUGR, still birth and neonatal death were found in study group. Assessment of morphological changes and its clinical relevance can be correlated with transactional study so as to provide the safe confinement and reducing the fetal morbidity and mortality.

KEYWORDS: Placental morphometry, Fetoplacental ratio, Placental coefficient, Gravida,Perinataloutcome, Pregnancy induced hypertension (PIH).

Address for Correspondence: Dr. Sabita Singh, D.No. 6, Plot No. 13, Kuruvikaran Salai, 3rd Cross Street, Anna Nagar, Maduri Dist. – 625020. Tamil Nadu, India. **E-Mail:** drsabita1234@gmail.com

INTRODUCTION

Placenta "After birth" evoked great interest among obstetricians, pathologists and recently anatomists to unveil its mysterious, complex structure in hypertensive pregnancy which can be correlated clinically. The placenta acts as a "diary of intrauterine life" which has the potency of focusing the developmental process going on during the pregnancy [1]. Placenta was often regarded as the waste product of the birth process, but obstetricians consider it to be the most important fetal organ. Being an organ of

vital importance for continuation of pregnancy and fetal nutrition, it has evoked great interest among the obstetricians as well as anatomists to understand the unique biological status of this complex organ [2]. Any pathology of placenta has direct hazardous impact over fetal outcome. Still it continues to be an underutilized and improperly handled surgical material [3].

Hypertension, a global epidemic in 21[st]century complicates pregnancy and became one among the deadly triad along with hemorrhage and infection resulting in large number of maternal deaths and there off fetal deaths. PIH is a pregnancy specific, multi-systemic disorder characterized by hypertension, edema and proteinuria after twenty weeks of gestation [4]. Pregnancy complicated by hypertension is commonly associated with placental insufficiency [5]. It has been recorded that maternal utero-placental blood flow is decreased in preeclampsia because there is maternal vasospasm [6]. Hypoxia in inter-villous space due to reduction of blood flow in placenta and myometrium is involved in the mechanisms of morphological alterations of the placenta because of maternal hypertension [7]. The main feature of abnormal placentation in PIH is inadequate trophoblastic invasion of maternal spiral arteries that results in persistence of muscular and elastic tissues in tunica media. Vessels fail to dilate, remain responsive to vasomotor influences that lead to high resistance low flow chorio-decidual circulation [8]. This results in morphological and histological changes in ischemic placentae and fetal hypoxia leading to IUGR which contributes to premature delivery and fetal death [9]. Hence the thorough examination of the placentae in utero as well as postpartum, gives valuable information about the state of fetal wellbeing [10].

Despite the significant achievements made by developing country in health, maternal and neonatal morbidity and mortality continues to be high. Presently maternal mortality is 212 per one lakh live births and 5% of it is due to PIH which is much higher in comparison to developed countries [11]. Though India is one among the developing country, pregnant mother from rural areas belonging to low socio-economic status are still devoid of proper antenatal check up. This study can help not only obstetricians by correlating the placental changes clinically in PIH but can also create awareness about the health status of our country.

MATERIALS AND METHODS

This study was done on 100 cases obtained from the department of obstetrics and gynecology, M.K.C.G. Berhampur,Oridsha. 50 placentae from uncomplicated full term deliveries taken as "control group" and rest 50 placentae from PIH as "study group". In PIH only those cases of more than 20 weeks gestation having BP>140/90 mm of hg and above with or without edema and/or proteinuria were included. Study group was categorized into 3 group- mild PIH [17], severe PIH [18] and eclampsia [15]. Mother's medical examination (age, weight , blood pressure, edema,convulsion), required investigations (blood sugar,urea, creatinine, urine for albumin) along withother relevant history like socio-economic status, habitat, type of gravid, antenatal-checkup were recorded. The placenta with cord and membrane were collected soon after delivery both from labor room or Operation Theater. Placenta along with membrane and umbilical cord were examined for any pathological lesions. It was washed thoroughly under running tap water to remove the blood clots and soaked with the help of blotting paper. Membranes were trimmed and umbilical cord was cut at a distance of 5cm from the site of insertion. Placentae were coded for their identification. Morphometry of placentae (Diameter, thickness, volume, number of cotyledons, weight) were done. Placental diameter was calculated by metallic scale (cm) as mean of two diameter perpendicular with each other. Thickness was taken as average of five points from three circular zone of placenta (one from central, two each from middle and peripheral zone). Volume was measured by water displacement method and cotyledon was counted in loop manner. Placental weight was recorded after squeezing the placenta to make it free from blood. Fetal weight was measured and finally feto-placental ratio and placental coefficient was recorded. Pathological lesions like infarction, retroplacental hematoma and calcification were recorded.

The type of delivery and fetal outcome (Preterm, IUGR, still birth and neonatal death) were also noted.

Statistical Analysis

The collected data was entered in the excel spreadsheet, processed and analyzed using the statistical software SPSS version 16.0. Data was expressed as percentages and mean values (with standard deviations). Differences between two groups were analyzed using Independent sample t-test and for more than two groups, ANOVA was used for means and Chi-square test and Fisher's exact test for proportions.Results were defined as statistically significant when the P value (2-sided) was less than 0.05.

RESULTS

In the study group, forty six percentages were in age group of 15-20yrs, seventy percentages from rural area, fifty percentages were under low socio-economic status, only forty percentages took regular antenatal checkup and seventy percentages were primigravida (Table 1).

Gross examination of placentae was done and recorded. The average diameter, thickness, volume and number of cotyledons in study group were 15.91±2.11cm, 2.39±.54cm, 297.64±67.90ml and 10.02±4.13respectively. Mean placental weight was 376.41±17.198gm (mild PIH), 330.72±2.90gm (severe PIH), 329.73±3.19gm (eclampsia) and mean birth weight was 2680.29±198.46gm (mild PIH), 2212.06±36.41gm (severe PIH) and 2073.60±9.47gm (eclampsia) respectively in study group. Gross placental pathology was also observed and noted. The study group presented with more gross pathological features when compared to control group (Table 2 & 3).

Fig. 1: Relationship between severity of pregnancy induced hypertension and mean placental weight.
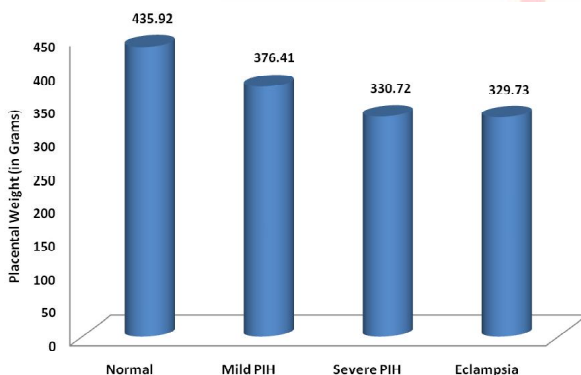
Fig. 2: Relationship Between severity of pregnancy induced hypertension and mean weight of the new born baby.
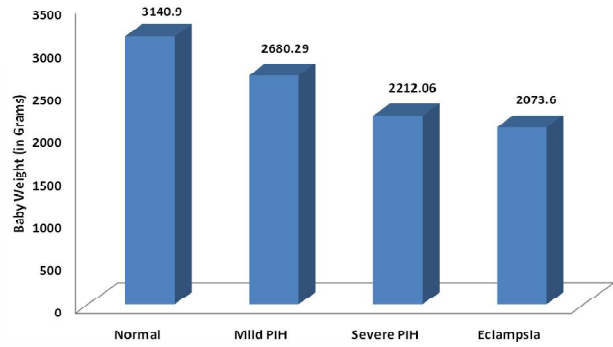
Fig. 3: Cross section of placenta showing area of red infarction.

Fig. 4: Cross section of placenta showing area of white infarction.

Fig. 5: Cross section of placenta showing area of calcification.

The maternal and fetal outcomes of both the study and control groups were observed and analyzed which showed more incidence of LSCS, preterm, stillbirth and dead born deliveries in study group (Table 4).

Table 1: Demographics of control and study group.

	Control Group (n=50)	Hypertensive Group (n=50)	p- value	Mild PIH (n=17)	Severe PIH (n=18)	Eclampsia (n=15)	p- value
Age Group (in years)							
15-20	14 (28.0)	23 (46.0)	0.363	6 (35.3)	10 (55.6)	9 (60.0)	0.412
21 – 25	19 (38.0)	16 (32.0)		4 (23.5)	6 (33.3)	4 (26.7)	
26 – 30	10 (20.0)	8 (16.0)		4 (23.5)	2 (11.1)	2 (13.3)	
31 – 35	3 (6.0)	1 (2.0)		1 (5.9)	-	-	
>35	4 (8.0)	2 (4.0)		2 (11.8)	-	-	
Habitat							
Rural	9 (18.0)	35 (70.0)	<0.001	7 (41.2)	13 (72.2)	15 (100.0)	0.01
Sub Urban	18 (36.0)	7 (14.0)		5 (29.4)	2 (11.1)	-	
Urban	23 (46.0)	8 (16.0)		5 (29.4)	3 (16.7)	-	
Socio Economic Status							
Upper	13 (26.0)	4 (8.0)	<0.001	4 (23.5)	-	-	0.007
Upper Middle	18 (36.0)	5 (10.0)		3 (17.7)	2 (11.1)	-	
Middle	6 (12.0)	6 (12.0)		3 (17.7)	3 (16.7)	-	
Lower Middle	8 (16.0)	10 (20.0)		4 (23.5)	4 (22.2)	2 (13.3)	
Lower	5 (10.0)	25 (50.0)		3 (17.7)	9 (50.0)	13 (86.7)	
Antenatal Check up							
No/Irregular ANC	8 (16.0)	30 (60.0)	<0.001	5 (29.4)	11 (61.1)	14 (93.3)	0.001
Regular	42 (84.0)	20 (40.0)		12 (70.6)	7 (38.9)	1 (6.7)	
Type of Gravida							
Primigravida	21 (42.0)	35 (70.0)	0.005	11 (64.7)	12 (66.7)	12 (80.0)	0.596
Multigravida	29 (58.0)	15 (30.0)		6 (35.3)	6 (33.3)	3 (20.0)	

Table 2: Comparison of placental and fetal weight in control and study group.

	Control Group (n=50)	Hypertensive Group (n=50)	p- value	Mild PIH (n=17)	Severe PIH (n=18)	Eclampsia (n=15)	p- value
Placental Wt(gm)							
Mean±SD	435 .92±14.18	345.96±24.29	<0.001	376.41±17.19	330.72±2.90	329.73±3.19	<0.001
Min, Max	416, 456	326, 392		334, 392	327, 335	326, 334	
CoV.	3.25	7.02		4.56	0.87	0.96	
Fetal Wt (gm)							
Mean±SD	3140.90±73.38	2329.72±284.87	<0.001	2680.29±198.46	2212.06±36.41	2073.60±9.47	<0.001
Min, Max	3039, 3289	2062, 2758		2086, 2785	2068, 2229	2062, 2086	
CoV.	2.33	12.22		7.4	1.64	0.45	
FP Ratio							
Mean±SD	7.2:1±0.30	6.7:1±0.37	<0.001	7.1:1±0.28	6.6:1±0.11	6.2:1±0.04	<0.001
Min, Max	6.6, 7.9	6.2, 7.3		6.2, 7.3	6.2, 6.7	6.2, 6.3	
Placental Coefficient							
Mean±SD	0.14±0.005	0.15±0.008	<0.001	0.14±0.006	0.15±0.002	0.16±0.001	<0.001
Min, Max	0.13, 0.15	0.14, 0.16		0.14, 0.16	0.15, 0.16	0.16, 0.16	

DISCUSSION

Pregnancy induced hypertension adversely affects the morphology of placenta. Placenta brings the mother and fetus, the two important ends of reproduction in contact with each other. Therefore the placenta which is usually considered as records of infant's prenatal experience provides crucial information about the deleterious effects of pregnancy induced hypertension on fetal outcome.

Teenage pregnancy and primigravida were risk factors for preeclampsia and eclampsia [12]. 48.71 % (mild PIH), 42.85 %(severe PIH) and

Table 3: Placental Morphometry and Pathology.

	Control Group (n=50)	Hypertensive Group (n=50)	p-value	Mild PIH (n=17)	Severe PIH (n=18)	Eclampsia (n=15)	p-value
Diameter (in cm)							
Mean±SD	18.40±1.42	15.91±2.11	<0.001	18.01±1.40	16.01±0.99	13.42±0.48	<0.001
Mini, Maxi	15.6, 20.0	12.8, 19.9		14.0, 19.9	13.8, 18.3	12.8, 14.2	
Thickness (in cm)							
Mean±SD	2.77±0.51	2.39±0.54	0.001	2.80±0.53	2.47±0.34	1.84±0.188	<0.001
Mini, Maxi	1.8, 3.5	1.5, 3.5		2.0, 3.5	2.0, 3.2	1.5, 2.2	
Volume (in ml)							
Mean±SD	437.96±51.22	297.64±67.90	<0.001	351.35±55.5	309.39±44.4	222.67±22.53	<0.001
Mini, Maxi	318, 496	205, 438		218, 438	248, 412	205, 285	
No. of Cotyledons							
Mean±SD	16.26±4.14	10.02±4.13	<0.001	12.29±4.56	10.06±3.76	7.40±2.29	0.002
Mini, Maxi	11, 28	5, 20		7, 20	6, 17	5, 12	
Infarction							
Present	6 (12.0)	29 (58.0)	<0.001	6 (35.3)	10 (55.6)	13 (86.7)	0.013
Absent	44 (88.0)	21 (42.0)		11 (64.7)	8 (44.4)	2 (13.3)	
Retroplacental Haematoma							
Present	4 (8.0)	24 (48.0)	<0.001	4 (23.5)	8 (44.4)	12 (80.0)	0.006
Absent	46 (92.0)	26 (52.0)		13 (76.5)	10 (55.6)	3 (20.0)	
Calcification							
Present	7 (14.0)	22 (44.0)	0.001	5 (29.4)	6 (33.3)	11 (73.3)	0.023
Absent	43 (86.0)	28 (56.0)		12 (70.6)	12 (66.7)	4 (26.7)	

Table 4: Maternal and Perinatal Outcome.

	Control Group (n=50)	Hypertensive Group (n=50)	p- value	Mild PIH (n=17)	Severe PIH (n=18)	Eclampsia (n=15)	p- value
Mode of Delivery							
LSCS	15 (30.0)	28 (56.0)	0.009	8 (47.1)	11 (61.1)	9 (60.0)	0.657
NVD	35 (70.0)	22 (44.0)		9 (52.9)	7 (38.9)	6 (40.0)	
Preterm							
Yes	-	5 (10.0)	0.056	1 (5.9)	4 (22.2)	-	0.083
No	50 (100.0)	45 (90.0)		16 (94.1)	14 (77.8)	15 (100.0)	
IUGR							
Yes	-	6 (12.0)	0.027	2 (11.8)	4 (22.2)	-	0.148
No	50 (100.0)	44 (88.0)		15 (88.2)	14 (77.8)	15 (100.0)	
Still Birth							
Yes	-	9 (18.0)	0.003	-	5 (27.8)	4 (26.7)	0.059
No	50 (100.0)	41 (82.0)		17 (100.0)	13 (72.2)	11 (73.3)	
Neonatal Death							
Yes	-	5 (10.0)	0.022	-	2 (11.1)	3 (20.0)	0.167
No	50 (100.0)	45 (90.0)		17 (100.0)	16 (88.9)	12 (80.0)	

52.63 % (eclampsia) cases in age group of 15-20yrs have been reported in a study of 100 placentae. 65% of total cases were of primigravida and 80% were un-booked without antenatal checkup [13]. The present study also found the similar observation (Table1). In primigravida there is lack of effective immunization by previous pregnancy. Other cause can be due to unyielding abdominal wall leads to increased abdominal pressure results in decreased uterine blood flow by external compression of myometrium as uterine blood flow is controlled by the tone of myometrium through which all the blood vessels passes to reach the intervillous space [14].

In a study of 100 placentae, mean placental weight was 435.63±103.91 and 371.43±88.52 in mild PIH and severe PIH respectively [15]. Fetal weight also decreased as weight of placenta decreases in hypertensive pregnancy which is in accordance with the present study (table- 2, fig-1,2). Placental weight decreases with increase in severity of hypertension due to utero-placental vascular insufficiency [16]. The linear relationship of placental weight and fetal weight can be very well showed by mean feto-placental ratio and placental coefficient. There was reduction in feto-placental ratio in hypertensive placentae as 6.15:1 (mild PIH), 5.43:1 (severe PIH) and 5.21:1(eclampsia) when compared to control group and the placental coefficient in PIH was .16 in a study [17,18] which is similar to our study (table 2).   A comparative study of 60 placentae, reported the placental diameter and thickness as 16.08±2.08 vs 18.80±2.32 and 1.51±.37 vs 1.59±0.39 respectively [8]. In another comparative study carried out on 100 placentae , placental volume was 612.98±213.26 vs 375.99±173.61 and number of cotyledons was 17±2 vs 16±2 [21]. The present study also recorded the similar morphological dimensions (table-3). This reduction of morphometric parameters in hypertensive placentae may be due to placental insufficiency in hypertensive pregnancy [8].

Placental infarction in toxemia of pregnancy is due to thrombotic occlusion of utero-placental blood flow [14]. A definite increase in incidence of infarction was reported in a study amounting to 50% (mild PIH), 70% (severe PIH) and 70% (eclampsia) [17]. Studies have reported a higher incidence of retroplacental haematoma in hypertensive pregnancy [2].   Different observations on calcification were recorded by various studies. Calcification was seen in more number of hypertensive placentae in a study [17], but it was reported as higher as 62.1% in normal placentae by another study [21]. On the other hand, frequency of calcification was same in control as well as hypertensive group in yet another study [22]. The present study reported all the pathological changes significantly higher (<.001) in hypertensive placentae (Table- 3).

28.8% preterm delivery, 4.8% still birth rate and 4.8% perinatal mortality have been reported in a study of two hundred fifty cases of hypertensive pregnancy. The rate of caesarean section was 14.8% [19] in comparison to 3.5% in normal pregnancy which is lower than our study. IUGR was found in a study to be four times higher in infants born to women with preeclampsia [20]. The present study also reported poor fetal outcome in hypertensive pregnancy (Table 4). The main impact of hypertension in pregnancy is under nutrition due to utero-placental vascular insufficiency which leads to growth restriction [15].

CONCLUSION

Our study revealed that morphometric parameters of placenta were significantly reduced and incidences of pathological changes were more in hypertensive cases as compared to control group. Primigravida of low socioeconomic status from rural area among cases were the most affected group. Fetal weight was decreased with reduction in placental weight, consequently reduced feto-placental ratio and placental coefficient in study group. Thus placentae can act as effective index for maternal and fetal status in hypertensive pregnancies. The adverse perinatal outcome in hypertensive pregnancy can be improved by regular antenatal checkup, widespread use of prenatal care and proper health facility along with health education especially to rural and low socioeconomic groups. This study can help the doctors to achieve the goal of good feto-maternal outcome by clinically correlating the placental changes with fetal and maternal parameters in hypertensive pregnancies.

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