

## ASSESSMENT OF BIRTH WEIGHT AND MORPHOMETRY OF PLACENTA IN PREGNANCY INDUCED HYPERTENSION IN SIKKIMESE POPULATION

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**ABSTRACT: BACKGROUND AND OBJECTIVES:** Birth weight is the simplest record of the new born and the most reliable indicator of foetal well-being. Low birth weight is a health hazard risking the new born to diseases in the perinatal period and failure to thrive in early childhood. The placenta, the mother and the foetus can be considered as a single entity and any adversity affecting one is bound to influence the others. The placenta is a complex organ responsible for the sustenance of the pregnancy and foetus in-utero. Pregnancy induced hypertension is the commonest complication which predisposes the foetus to birth asphyxia, intrauterine growth retardation and cardio-vascular changes and the mother to chronic hypertension and HELLP syndrome. The incidence is high in India with a fairly high number of cases in Sikkim. The study aims to evaluate the morphometric alterations of the placenta in PIH which would serve as a guide to assess the severity of the disease and analyze the cause of maternal and foetal mortality and morbidity. **DESIGN AND SETTING:** A prospective, analytical hospital based study. **MATERIALS AND METHODS:** Thirty women with PIH constituted the "cohort" while 60 normotensive pregnancies constituted the "control". **RESULTS:** Mean placental weight was significantly lower in PIH (p-value 0.014). The proportion of Low birth weight babies was significantly higher (p-value 0.0001) in cases. **CONCLUSIONS:** Reduced perfusion to placenta in PIH permanently alters the structure and physiology of the foetus and atherosclerosis in the uterine arteries causes reduced blood flow in the inter-villous spaces causing reduced placental weight and intrauterine growth retardation.

**KEY WORDS:** Pregnancy induced hypertension (PIH), HELLP (hemolysis, elevated liver enzyme, and low platelet count), Low Birth Weight (LBW), Mean Birth Weight (MBW), intrauterine growth retardation (IUGR).

**INTRODUCTION:** As per the National family health survey 2005, the prevalence of LBW babies is 22.2% in India. Low birth weight is a primary cause of infant mortality, diseases in early infancy, poor progress and development and accounts for huge amount of stress to the family. PIH is one of the foremost aetiologies of intra uterine growth retardation, foetal distress and infant mortality. The survival, development and growth of the foetus are solely dependent on the proper functioning and development of the placenta which is the foetal organ for exchange of blood gases, nutrition and excretion and is responsible for bringing about all the physiological changes of and maintaining the pregnancy to term. A thorough evaluation of placenta provides an accurate record of any complications which occurred during the prenatal life of the foetus and its consequences in future. The anatomical and pathological placental assessment becomes more precise through morphometric analysis which is non-invasive, indirect method of study of the physiology or pathology of human gestation. Rosana R.M (2008)<sup>1</sup>

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If pregnancy is terminated and placenta removed the hypertension and proteinuria resolve within a few hours though some changes take several days or weeks to resolve. The placenta is intimately involved in the aetiology of hypertensive disorders of pregnancy<sup>2</sup>. Barker D.J.P. recorded an inverse relationship between weight at birth and blood pressure. Reduced perfusion to the placenta and the foetus is one insult which can permanently alter and program both the structure and physiology of the foetus. In case of maternal hypertension atherosclerosis affects the uterine blood vessels, narrowing their lumen which leads to reduced blood flow at the inter-villous spaces<sup>3</sup>.

**METHODS:** Analytical Study with Prospective cohort design which was carried out from May 2007 to April 2008, at Central Referral Hospital, Sikkim Manipal Institute of Medical Sciences, Tadong. Due to feasibility constraints convenient sample sizes was taken for the study. A total of 90 pregnant women in the age group of 18years and above who consented to participate were included. Pregnant women with blood pressure ranging from 140/90 to 160/110mmHg with proteinuria were included as “cohort” and 60 pregnant women with normal blood pressure were included as “control”. All these women were followed up throughout their pregnancy till delivery to study the final outcome of their pregnancies. Age matching was done to identify each Cohort with two corresponding Controls. The study of gross morphology of placenta was conducted in the Department of Anatomy. Pregnancy induced hypertension was diagnosed as per the WHO criteria in the department of obstetrics and gynaecology. The mothers and neonates were given tags with code numbers for identification. After delivery the placenta were collected from the labour room and operation theatre, washed, dried and examined and fixed in 10% formalin. The newborn babies were examined for any congenital anomalies, weight, and the apgar scoring were noted. The sizes, shape, weight, volume, thickness, number of cotyledons, cord length of the placenta were noted. The placenta was inspected for calcifications, infarctions, infections, site of cord insertion and membranes.

The collected data were tabulated and analyzed using the SPSS (Statistical Package of social sciences), version 10.0 for windows. Findings were expressed in terms of proportion and depicted in the form of Tables. Z-test and Chi-Square Test were applied for univariate analysis to study the effect of each variable over the outcome. A p-value<0.05 was considered to be statistically significant All these 90 participants had singleton pregnancies and their outcome was live births. Pregnant women who did not give informed verbal consent and twin pregnancies were excluded.

## RESULTS:

**Affects on new born:** Though the number of babies with foetal distress, and low Apgar was higher in PIH however this did not show statistical significance.

The Mean birth weight in (MBW) of new born babies in controls was 2.8(±0.32), with a maximum of 3.9kg and minimum of 2.1kg. The mean birth weight in cases (PIH) was 2.6(±0.3) with a maximum of 3.1kg and 2.0kg. Out of the 30 cases 18 babies were of low birth weight (<2.5kg) i.e. 60%, while 12 cases were of normal birth weight (40%). The birth weight ranged from 2.0 to 3.0kg in cohorts. The birth weight was significantly less in PIH cases (p=0.0001).

The proportion of Low Birth Weight babies was significantly higher (60%) in PIH. The proportion of LBW babies was found to be significantly higher among the pregnant women in the age group of 30 years and above(45.5) as compared to those in the age group of 25-29 years(17.6%)

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and less than 25 years(8.9%).Chi-square for linear trend with increase in maternal age was also found to be statistically highly significant.

The number of infarcts, calcifications, and marginal insertion of placenta was more in mothers with PIH compared to normotensive mothers however this was not statistically significant.

## **OBSERVATIONS ON PLACENTA:**

**Gross anatomy:** The placental weight in controls ranged between 425 -700 g m. The mean placental weight in hypertensive mothers (PIH) was 511.2( $\pm$ 51.5) with a maximum of 600 gm and minimum of 380 gm. Mean placental weight in normotensive mothers was 548.6( $\pm$ 72.8) gm with a range of maximum 700 gm and minimum 425gm. It was recorded that 14 placentas of PIH cases weighed between 300-500 gm i.e.46.6% while 13 placentas weighed >500 gm i.e.43.3% and 3 placentas weighed less than 300gm i.e. 10%. In their study Udainia and Jain(2001) recorded that 65.33% of placenta weighed in the range of 300-500 gm while 21.33% weighed less than 300 gm and 13.34% weighed more than 500gm. Jain et al (2007) revealed that 67% placental weight less than 500 gm in PIH cases. In the present study 56.6% of the placenta in PIH weighed less than 500gm.

## **DISCUSSION:**

**Birth Weight:** Birth weight is the simplest parameter which serves as the most reliable indicator for the growth, development and sustenance of the neonate. Low birth weight is a major health problem in developing countries like India and hypertension in pregnancy is one of the aetiologies of intra uterine growth reduction. 20 to 25% of babies born to mothers with hypertensive disorders are small for gestational age (SGA). Pre term deliveries occur in 50% of pregnancies with hypertension. Major anomalies occur in 3% off springs and there is increased risk of congenital viral infections especially with cytomegalovirus and hepatitis B. The perinatal mortality rate is around 5- 10% in hypertensive disorder of pregnancy.

In our study the mean birth weight of newborn babies of hypertensive mothers was 2.69 kg and that of normotensive babies was 2.84 kg. Jain et al reported 59.10% low birth weight in PIH i.e. (<2.5kg). Udainia and Jain (2001) observed the MBW of 2.280 in cases 2.640 in control<sup>4</sup>. Majumdar et al (2005) recorded MBW to be 2.04 in Pregnancy PIH and 2.8 kg in normotensives<sup>6</sup> with a range of maximum 3.9 kg and minimum of 2.1 kg.

The proportion of low birth weight was higher in cases (60%) compared to controls (20%) and in conformity to the study of Jain et al (59.10%)<sup>7</sup>. Chakravorty (1967) observed the MBW of 2.80 kg in controls and 2.72kg in hypertensive patients<sup>8</sup>.

According to Roberts and Gammil (2005) manifestation of maternal disorder of pre-eclampsia requires initiation by reduced placental perfusion, followed by maternal response that occurs only in the presence confounding maternal factors (genetic, behavioural or environmental)is associated with reduced perfusion not only to the foeto-placental unit but to all maternal vascular beds<sup>12</sup>. Reduced placental perfusion resulting in foetal under nutrition can lead to intra uterine growth restriction and permanent adaptive changes such as endothelial dysfunction. Thus it was noted that in placental insufficiency due to PIH the mean birth weight and the placental weight are both significantly reduced.

In the present study it was observed that the MBW in cases was higher than that of Udainia and Jain<sup>5</sup> & Majumdar<sup>7</sup> but in conformity with that of Chakraborty<sup>9</sup>. The MBW in controls was

observed to be higher in the present study which could be attributed to better socio-economic status and improved ante-natal care.

**Placental Weight:** If pregnancy is terminated and placenta removed the hypertension and proteinuria resolve within a few hours though some changes take several days or weeks to resolve. The placenta is intimately involved in the aetiology of hypertensive disorders of pregnancy<sup>2</sup>

The number of meconium stained placenta, infarcted areas, thrombosis, and calcifications was higher in PIH but these did not show statistical significance

Incidence of reduced placental weight is significantly higher in PIH which could be due to reduced placental perfusion. The decreased placental weight is attributed to atheromatous lesions in the tunica media of spiral arteries, associated with thrombosis & vascular occlusion and placental infarction<sup>11</sup>.

Udania & Jain (2001) observed that there was significant lowering of placental weight in PIH. An increase in the incidence of intrauterine growth retardation and still birth is found to be associated with lower placental weight. They recorded 12% of placentas in PIH weighed between 251-300gm and 65.33% between 301-500 gm. They observed that the placental weight ranged from 250gm - 700gm with a mean of 495( $\pm$ 114.11) gm in controls and 405.67 in cases<sup>6</sup>.

The placental weight in controls ranged between 425 -700 g in the present study. Hosemann (1946) in his study of normal term pregnancy found the placental weight of 400-1000 gm<sup>6</sup>. Majumdar et al (2005) observed that the mean placental weight in control group was 485.85( $\pm$ 47.31) and 399.10 ( $\pm$ 90.31) in cases<sup>7</sup>. K. Jain et al (2007) in their study revealed that 67% of placentas in PIH weighed less than 500 mg<sup>8</sup>. The present study reveals that the mean placental weight is 511.2( $\pm$ 51.2) in cases and 548.6( $\pm$ 72.8) in controls, and 56.6% placentas weighed less than 500gm in PIH cases while Udania & Jain(2001)<sup>6</sup> in their study reveal 77.33 % of placenta in PIH weighed less than 500gm which is slightly lower than the observations by the above authors.

Similar reduced weight of placenta in PIH was recorded by Chakravorty (1967)<sup>9</sup> Jain et al (2007)<sup>8</sup>, Armitage, Hamilton & Rowe (1967) and Majumdar (2005)<sup>7</sup> and is confirmed by the present study. It is observed that the birth-weight as well as the placental weight was higher in both controls and cases in the present study which could be attributed to improved antenatal care and better socioeconomic status of the mothers in the present times.

The main feature of abnormal placentation is the inadequate trophoblastic invasion of maternal spiral arteries which fail to dilate and leads to high resistance low flow chorio-decidual circulation. The spiral arteries are unable to dilate to accommodate the required increase in blood flow resulting in placental dysfunction which manifests as pre-eclampsia<sup>11</sup>. The reduced placental weight in PIH as shown by Chakravorty (1967)<sup>10</sup>, Jain et al<sup>9</sup>, Udania & Jain (2001)<sup>6</sup>, Majumdar<sup>6</sup>, Boyd, Hamilton and Rowe (1967)<sup>14</sup>, Garg, Rath and Sharma (1996)<sup>16</sup> is confirmed by the present study.

**CONCLUSION:** The mean birth weight of newborns is reduced in PIH p-value0.001. The incidence of LBW babies was found to be more in preterm deliveries (38.1%) than full term deliveries (10.1%). Foetal distress, and the number of meconium stained placenta was higher in PIH cases but did not show significant variations.

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The placentas of PIH mothers weighed less than that of normotensive mothers and were statistically significant (p 0.014). The placenta of patients with PIH weighed significantly less and the mean birth weight was also reduced in PIH as compared to controls with a significant (p-0.0001). The placentas of cases were smaller with more infarcts, calcifications, and fibrinoid deposits though the number of cotyledons did not show any significant variation.

Serial No.	Obstetric Parameters	HTN Mean ( $\pm 2SD$ )	Normotensive Mean ( $\pm SD$ )	P- Value from Z test result
01	Birth Weight (kg)	2.6(0.2)	2.8(0.3)	0.0001*
02	Age of pregnant woman	26.9(4.3)	24.4(2.8)	0.001*
03	Parity	1.9(1.1)	2.3(1.4)	0.194
04	Gestational age	37.0(1.5)	38.2(1.4)	0.0001*

Table 1: Comparison of Obstetric Parameters between PIH mothers and Normotensive mothers

\*p-value<0.05 was considered to be statistically significant

Serial No	Morphology of Placenta & Cord	HTN Mean ( $\pm 2SD$ )	Normotensive Mean( $\pm 2SD$ )	P-value from Z test
01	Placental Weight(gm)	511.2(51.5)	5486(72.8)	0.014*
02	No. of Cotyledons	19.4(2.1)	19.9(2.8)	0.406
03	Placental diameter	19.2(1.8)	18.8(2.2)	0.361
04	Placental thickness	2.7(0.3)	2.7(0.5)	0.609
05	Placental volume(cc)	461.5(63.6)	450.7(90.1)	0.561
06	Cord length(cm)	55.0(2.8)	57.5(3.2)	0.782

Table 2: Comparison of Morphology of Placenta & Cord between PIH mothers and normotensive mothers

\*p-value of < 0.05 was considered to be statistically significant

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Sl. No.	CORRELATES	LOW BIRTH WEIGHT		NORMAL BIRTH WEIGHT		X <sup>2</sup> ,	P-VALUE
		n	(%)	n	(%)		
1.	<b>ANTENATAL BLOOD PRESSURE</b>						
	Cohort (PIH)	18	60	12	40	17.64	0.001
	Control (Normotensive)	12	20	48	80		
2.	<b>AGE GROUP</b>						
	<25yrs	4	8.9	41	91.1	8.55	0.014
	25-29yrs	64	17.6	28	82.4		
	≥30yrs	5	45.5	6	54.5		
3.	<b>PARITY</b>						
	≤2	11	17.2	53	82.8	0.04	0.835
	>2	4	15.4	22	84.6		
4.	<b>CORD INSERTION</b>						
	Central	6	15.8	32	84.2	2.76	0.251
	Eccentric	3	10	27	90		
	Marginal	6	27.3	16	72.7		
5.	<b>GESTATIONAL AGE</b>						
	<37weeks	8	38.1	13	61.9	9.06	0.003
	≥37weeks	7	10.1	62	89.9		

Table III: Correlates of low birth weight identified by the study

P-value < 0.05 was considered to be statistically significant

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