

EFFECTS OF DIFFERENT GRADES OF PIH ON PREGNANCY OUTCOME

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ABSTRACT

Background: Placenta is a mirror of gestational life. It provides the reflection of the hazards, the foetus has been subjected to during its growth and development. Pregnancy induced hypertension (PIH) contribute greatly to maternal and fetal morbidity and mortality.

Aims and Objectives: This study has been taken to evaluate the effect of PIH on pregnancy outcome and to correlate the placental and foetal weight.

Materials and Methods: In our study 100 placentae were taken. 50 placentae were of control group. 32 placentae were of mild PIH, 13 placentae belonged to severe PIH and 5 to eclampsia. Placentae were weighed and foetus birth weights were noted. Fetoplacental weight (F/P) ratio and placental foetal (P/F) weight ratio i.e (placental coefficient) were calculated.

Observations and Result: 23 cases of PIH belonged to 20-25 years age group. More cases of PIH were of primigravida. Maximum cases of mild PIH 13(40.62%) belonged to 37-38 weeks of gestation. In eclampsia placental weight was less than 400 gms. All the cases of eclampsia had birth weight less than 2.5 kg. Mean placental and foetal weight was less in eclampsia than other grades of PIH. Foetal loss was 2(40%) in eclampsia.

Conclusion: PIH is a common medical disorders especially among young primigravidas. Placental and foetal weight decreases as the severity of PIH increases. The fetoplacental weight decreases with increased grades of PIH. Placental coefficient was more in severe PIH and eclampsia. Due to the impact of PIH on foetal growth there is increased risk of preterm, small for gestational age (SGA) and foetal loss.

KEY WORDS: Placenta, PIH, fetoplacental weight ratio, placental coefficient.

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INTRODUCTION

The placenta –‘the life of the foetus in utero’ – functions diversely to support the growth of the foetus, interacts with the two individuals, the mother and the developing fetus [1].

It is a mirror, which reflects the intrauterine status of the fetus [2]. The foetus, placenta and the mother form a composite triad of the

dynamic equilibrium, and dysfunction to any one of them can affect the others [3].

Hypertensive disorders in pregnancy contribute greatly to maternal and foetal morbidity and mortality. Pregnancy induced hypertension (PIH) is a multisystem disorder of unknown etiology characterized by extent of blood pressure 140/90 mm Hg or more with oedema or proteinuria

or both after 20th week of pregnancy [4].

One of the prevailing hypothesis of its pathogenesis is that the placenta plays a unique role, and the reduced placental perfusion is the point of convergence of diverse pathogenic processes in the development of preeclampsia. Because of the decrease uteroplacental blood perfusion, it is intuitive that preeclampsia will result in a detrimental effect on fetal growth, leading to intrauterine growth restriction and low birth weight [5].

The placenta provides a reflection of the hazards, the foetus has been subjected to during its growth and development. The impact of PIH on fetal growth is complex and is associated with a significantly increased risk of preterm births, low birth weight (LBW) and small for gestational age (SGA) births [6]. The primary objective of this study was to evaluate the effect of PIH on pregnancy outcome and to find the relation between placental weight and birth weight.

MATERIALS AND METHODS

This study was carried out at Jawaharlal Nehru Medical College, Sawangi (Meghe), Wardha over a period of 2 years from 2008 to 2010 after taking clearance from institutional ethical committee. Fifty placentae were collected from normal deliveries and fifty from PIH patients from Obstetrics & Gynaecology department. All the cases varied from 20- 35 years. The data like height, weight, pulse, BP, Hb % & urine examination reports were noted from patients' case sheets. Patients having blood pressure ranging from 140/90 mm Hg and above with and without oedema and/ or proteinuria were included in the study. Haemoglobin level, blood sugar, blood urea, serum bilirubin and creatinine for both the groups were noted from the investigation reports. If there is any abnormality found they were excluded from the study. The birth weights were noted from neonatal case sheets. The neonates were inspected for any congenital anomalies. Already subdivided cases like mild, severe & eclampsia were taken for the study. 32 cases of mild PIH, 13 cases of severe PIH and 5 cases of eclampsia were included in the study.

After delivery placentae were collected, the blood clots were removed during washing and

the placentae were dried with blotting paper. The rest of the membranes were trimmed off by sharp scissors near the margin and the umbilical cords were cut at about 2 cm from their insertions. The placentae were weighed by weighing machine. Examination of placentae was conducted as per proforma by Yetter JF (1998) [7]. Fetoplacental weight (F/P) ratio and placental foetal (P/F) weight ratio i.e (placental coefficient) were calculated. The statistical analysis was done by using SPSS 17.0 version.

OBSERVATIONS AND RESULTS

Table 1: Age wise distribution of cases.

Groups	20 –25 yrs	26 -30 yrs	31 - 35 yrs	Total
Control	38	10	2	50 (100%)
Mild PIH	23	6	3	32 (64%)
Severe PIH	9	4	0	13 (26%)
Eclampsia	1	3	1	5 (10%)

Table 2: Distribution of cases according to gravida.

Groups	Primi gravida	Second gravida	Third gravida	Fourth gravida
Control	14	25	9	2
Mild PIH	16	11	3	2
Severe PIH	9	4	0	0
Eclampsia	1	2	1	1

Table 3: Distribution of cases according to gestation.

Gestation (Weeks)	Groups			
	Control (50)	Mild (32)	Severe (13)	Eclampsia (5)
33 – 34	-	-	-	2 (40%)
35 -36	-	8 (25%)	6 (46.15%)	2 (40%)
37 -38	21 (42%)	13 (40.62%)	7 (53.85%)	1 (20%)
39 -40	26 (52%)	11 (34.37)	-	-
41 -42	3 (6%)	-	-	-

Table 4: Placental weight in control & different grades of PIH.

Placental weight (gms)	Groups			
	Control	Mild	Severe	Eclampsia
≤ 300	-	-	4(30.76%)	3(60%)
301 – 400	-	16(50%)	6(46.15%)	2(40%)
401 – 500	40(80%)	15(46.87%)	3(23.07%)	-
>500	10(20%)	1(3.13%)	-	-

Table 5: Foetal weight in control & different grades of PIH.

Foetal weight (Kg)	Groups			
	Normal	Mild	Severe	Eclampsia
< 2.5 kg	5 (10%)	18(56.25%)	10(76.92%)	5 (100%)
>2.5 Kg	45(90%)	14(43.75%)	3(23.07%)	-

Table 6: F/P & P/F ratio in control & different grades of PIH.

Groups	Mean foetal weight	Mean placental weight	F/P weight ratio	P/F weight ratio (placental coefficient)
Control	2813.60±258.06	464.80±40.28	6.05	0.16
Mild	2619.06±408.43	445.15±60.48	5.88	0.16
Severe	2146.15±378.14	398.07±79.36	5.39	0.18
Eclampsia	1658.80±446.39	318±81.28	5.21	0.19

Table 7: Foetal outcome in different grades of PIH.

Toxemia	Preterm	Small for gestational age (SGA)	Fetal loss
Mild	8 (25%)	10 (31.25%)	0
Severe	6(46.15 %)	4(30.76%)	0
Eclampsia	2 (40%)	1 (20%)	2 (40%)

Table 8: Mean Foetal and Placental weight of foetal outcome.

Fetal outcome	Mean Foetal weight	Mean Placental weight
Preterm	2055.31±270.97	361.12±43.72
Small for gestational age (SGA)	2254.33±117.47	416.13±38.88
Fetal loss	1274±387.49	250±70.71

Fig. 1: Showing PIH placenta.

Table no.1: In our study, maximum no. of cases were of mild PIH i.e; 32 (64%) and minimum cases of eclampsia i.e; 5 (10%). Most cases of PIH belonged to 20-25 years age group.

Table no.2: In Primigravida, 14 cases were in control group and 16 in mild PIH, 9 in severe PIH and 1 in eclampsia. The number of cases of PIH were more in primigravida.

Table no.3: Maximum no. of cases i.e; 13 (40.62%) of mild PIH were in 37- 38 weeks of gestation and minimum i.e; 11 (34.37%) were in 39-40 weeks of gestation. In severe PIH 7 (53.85%) cases belong to 37-38 weeks of gestation and in eclampsia 2 (40% cases) belong to

33-34 weeks of gestation.

Table no.4: 16 (50%) mild PIH cases has placental weight ranging from 301 – 400 gms. 4 (30.76%) severe PIH cases has placental weight \leq 300 gms. In eclampsia placental weight ranging from 301-400 gms were seen in 2 (40%) cases.

Table no.5: Foetal weight < 2.5 kg was present in 10 (76.92%) of severe PIH cases. All the cases of eclampsia has foetal weight < 2.5 kg.

Table no.6: With the increase in severity of PIH, there is gradual decrease in the F/P weight ratio and P/F weight ratio increases simultaneously.

Table no.7: In mild PIH, preterm and SGA babies were 8 (25%) and 10 (31.25%) respectively. In severe PIH, preterm and SGA babies were 6 (46.15 %) and 4 (30.76%) respectively. Fetal loss was 2 (40%) in eclampsia.

Table no.8: The mean foetal weight was 1274±387.49 and mean placental weight was 250±70.71 in foetal loss.

DISCUSSION

The Placenta has been described as a “diary of intrauterine life” and it may elucidate many aspects of intrauterine life. Impaired placental function is a major cause of fetal growth restriction, so the present study was undertaken to analyze the effect of different grades of PIH on pregnancy outcome and to correlate placental and foetal weight.

In the present study we had 32 (64%) cases of mild PIH, 13 (26%) cases of severe PIH and 5 (10%) cases of eclampsia. We had only 1 case of eclampsia in 31-35 years range. Maximum number of cases were of mild PIH (23) belonging to 20-25 years age group (Table no 1). Goswami P et al in their study had maximum 20 (50%) cases in 17-20 years age group while 21-24 years age group had 12 (30%) cases of PIH [8]. Kambale et al had maximum number of cases belonging to 20-25 years age group. They also had only 1 case of eclampsia above 30 years of age [9]. Mean maternal age in preeclampsia is 24.0 (±4.7) years in the study by Boyd P A and Scott A [10].

In our study we had 14 cases of primigravida in

control group and 16, 9, and 1 case in mild PIH, severe PIH and eclampsia respectively (Table no 2). The number of cases of PIH were more in primigravida. Kambale et al had 24 cases of primigravida, where in 14 cases were of mild PIH. 7 and 3 cases were of severe PIH and eclampsia respectively [9]. We had more number of primigravida cases as it is one of the etiological factor of PIH.

We studied maximum number of cases i.e; 13 (40.62%) of mild PIH in 37 - 38 weeks of gestation. In severe PIH 7 (53.85%) cases were reported in 37-38 weeks. In 33 -34 weeks of gestation only 2 (40%) cases of eclampsia were present (Table no 3). Asgharnia M et al in their longitudinal cross- sectional study which includes cases of known gestational age ≥ 37 weeks had mean gestational age (days) 274.5 days [11]. XK Chen et al in their study had mean gestational age (weeks) 38.11 ± 2.89 in PIH group [6].

15 (46.87%) cases of mild PIH had placental weight ranging from 401-500 gms, while in 301-400 gms range 6 (46.15%) cases were of severe PIH. In 2 (40%) cases of eclampsia, placental weight ranged from 301-400 gms (Table no 4). Raghavendra A.Y. et al had 24.10% cases in mild PIH in 401-500 gm placental weight range while in eclampsia 8% placenta had weight >500 gms [12]. Navbir P had only one placenta in 400-600 gms weight range while 5 (83.33%) placenta had weight <400 gms in eclampsia [13]. In our study mean placental weight in mild PIH, severe PIH and eclampsia is 445.15, 398.07 and 318 gms respectively which is less as compared to control group (Table no 6). Navbir P in his study found mean placental weight 329.17, 379.5, and 412.5 in eclampsia, moderate and mild pre-eclampsia respectively [13]. Das B in their study had placental weight more reduced in proteinuric patients and also in cases where the duration of hypertensive disorder was prolonged [14].

Dutta DK found decreased placental weight in PIH and also commented that the weight of the placenta decreases with the increasing grades of PIH [15]. Aherne considered the weight of the placenta to be "functionally significant" because it is related to villous surface area and to total foetal weight. Walker and Turnbull stated that risk of foetal hypoxia is increased if pla-

cental function is impaired in preeclampsia. The cause of small placentae may be genetic or some external influence such as uteroplacental circulation of low capacity [16].

In all the eclampsia cases we had foetal weight < 2.5 kg (Table no 5). Ahmed M et al recorded birth weight < 2 kg in 13 (8.6%), 23 (44%) and 36 (76.6%) cases of mild, moderate and severe PIH respectively [17]. Kambale et al had 14 (53.8%), 9 (60%) cases < 2.5 kg in mild & severe PIH respectively. They also have 100% cases of low birth weight in eclampsia [9]. In our study we had mean foetal weight of 2619.06, 2146.15 and 1658.80 gms in mild, severe PIH and eclampsia respectively (Table no 6). Navbir P found 2.16, 2.59 and 2.79 kg mean foetal weight in eclampsia, moderate and mild preeclampsia respectively [13]. Goswami P et al stated that during pregnancy, the placental mass maintains a dynamic relationship with the weight of developing foetus [8]. Shah RK commented that weight of placenta and birth weight are more decreased in preeclampsia with oedema than that without oedema but in severe preeclampsia both will be decreased probably due to marked proteinuria [18]. Rath stated that in hypertension arrangement of the intracotyledons vasculature is altered resulting in low birth weight of the babies [19]. Naeye RL explained that the metabolic abnormality in the placenta may potentiate the effects of low uteroplacental blood flow which impairs nutrient synthesis that leads to the foetal growth retardation associated with preeclampsia and eclampsia [20].

In the present study we noted foeto-placental weight ratio 5.88, 5.39 and 5.21 in mild, severe PIH and eclampsia respectively (Table no 6). We have decreasing F/P ratio with increasing toxemia. Mohan H et al had 6.0, 5.28 and 5.18 foeto-placental weight ratio in mild, severe PIH and eclampsia respectively. He observed direct proportionate relationship of foeto-placental weight ratio, being lowest in case of eclampsia and maximum in the control group. [21].

According to Macpherson's, F/P ratio was an additional means of evaluating placental weight deviation [22]. Fox noted that in many hypertensive gestations there is decreased foeto-placental weight ratio because of a compensatory hypertrophy of placenta under the influence of

unfavourable maternal environment [13]. Mukherjee B et al commented that a high correlation between the foetus and placenta is due to the fact that both, foetus and placenta share the tissues of common genetic origin [23].

Placental / foeto weight ratio (placental coefficient) in mild, severe PIH and eclampsia is 0.16, 0.18 and 0.19 respectively (Table no 6). We found P/F ratio increases with increase in grades of toxemia. Teasdale F calculated placental coefficient 0.14 and 0.17 in control and preeclampsia respectively. The increased number of high P/F value in preeclampsia could only be accounted for by greater reduction in foetal weight than placental weight [24]. In particular, reductions in placental weight and the ratio of placental to foetal weight (P/F) have been considered as possible indications of functional inadequacy [25].

The foetal outcome in different grades of PIH i.e; preterm babies are 8(25%), 6(46.15%) and 2(40%) in mild, severe PIH and eclampsia respectively in our study. Also we have 10(31.25%), 4(30.76%) and 1(20%) small for date babies in mild, severe PIH and eclampsia respectively. In eclampsia there were 2(40%) foetal loss (Table no 7). Ahmed M et al had SGA babies 19(12.6%), 34(65.5%) and 38(80.9%) in mild, moderate and severe PIH respectively [17]. George J.N. et al had preterm babies 3(6.24%) and 28(53.84%) while SGA babies 3(6.24%) and 16(40%) in mild and severe PIH respectively [26]. Kaur P had poor perinatal outcome in preeclampsia when compared with normotensive pregnancies in which all the newborns were appropriate for gestational age (AGA). He noted intrauterine growth retardation (44%) and intrauterine deaths (13.33%) were mostly associated with poorly controlled preeclamptic mothers and the difference between two groups was statistically significant [27]. In study carried out by Meshram D.P et al the incidence of IUGR in pre eclamptic patients was 19.14% while perinatal mortality in severe pre eclampsia was 28.72% [28]. In the study by Bangal VA et al, prematurity was the commonest fetal complication seen. They have 17.99%, 47.62%, and 52.63% cases of prematurity in mild, severe PIH and eclampsia respectively. IUGR was the next common complication seen in 3(7.69%), 11(26.19%) and 6(31.58%)

cases of mild, severe PIH and eclampsia respectively. Kapoor et al concluded that, the incidence of premature babies was 23% in PIH and prematurity was one of the major risk factors for increasing the perinatal mortality [29].

In our study mean foetal weight was 2055 ± 270.97 , 2254.33 ± 117.47 and 1274 ± 387.49 gms while mean placental weight 361.12 ± 43.72 , 416.13 ± 38.88 and 250 ± 70.71 gm in preterm, small for gestational age babies and foetal loss respectively (Table no 8). Younoszai & Haworth noted mean birth weight 3313, 2035 and 2295 gm while mean placental weight is 420, 330 and 311gm in term normal, preterm and IUGR babies respectively [30]. In preeclampsia changes in blood flow were supposedly caused by changes in blood volume caused by plasma volume contraction. Proteinuria may be a marker for vascular damage and diminished maternal blood flow to the foetus could result in foetal hypoxia and growth restriction. These may explain why some patients with preeclampsia deliver SGA and low-birth weight infants [5].

CONCLUSION

The incidence of PIH is more in young primigravida in the age group 20-25 years. Most of the cases of different grades of PIH fall between 33-36 weeks of gestation. Placental and fetal weights are very much affected by increasing grades of PIH. F/P ratio decreases as the severity of PIH increases, simultaneously the fetal coefficient (P/F ratio) increases. The commonest complication in various grades of PIH is preterm babies along with SGA babies and fetal loss.

For safe pregnancy outcome early diagnosis, health education and timely intervention is important.

Conflicts of Interests: None

REFERENCES

- [1]. Madkar C, Musale J, Deshpande H & Shilote RA. Study of placental weight and birth weight ratio (PW/BW) and its effects on perinatal outcome. Indian journal of Obstetrics and Gynaecology. 2015; Jan – Mar ;2(1):1-6.
- [2]. Udainia A, Bhagwat SS, Mehta CD. Relation between placental surface area infarction & foetal distress in pregnancy induced hypertension with its clinical relevance. J of Anat Soc India. 2004; 53(1): 27-30.

- [3]. Jain K, Kavi V, Raghuvver CV, Sinha R. Placental pathology in pregnancy-induced hypertension (PIH) with or without intra uterine growth retardation. *Indian J of Pathology & Microbiology*. 2007; 50(3): 533-537.
- [4]. Dutta D.C. The Placenta and Fetal membranes, Hypertensive disorder of pregnancy. *Textbook of Obstetrics*. 4th edition. New Central book Agency (P) Ltd, Calcutta. 1998; 28-40 & 234-255.
- [5]. Xu Xiong MB, Nestor N, Demianczuk, Pierre Bueens, L Duncan Saunders. Association of preeclampsia with high birth weight for gestational age. *Am.J.Obstet Gynecol*. 2000; 183(1): 148-155.
- [6]. XK Chen, SW Wen, G Smith, Q Yang, M Walker. Pregnancy induced hypertension and infant mortality: roles of birthweight centiles and gestational age. *An International Journal of Obstetrics and Gynaecology*. 2007; 114: 24-31.
- [7]. Yetter JF. Examination of the Placenta, The American academy of family physician. 1998.
- [8]. Goswami P, Memon S, Rathore M. Fetoplacental weight relationship in normal pregnancy and pregnancies complicated by pregnancy induced hypertension and abruption of placenta. *International Journal of Research in Medical sciences*. 2015; 3(5): 1081-1084.
- [9]. Kambale T, Iqbal B, Ramraje S, Swaimul K, Salve S. Placental morphology and fetal implications in pregnancies complicated by pregnancy induced hypertension. *Medical Journal of Dr. D.Y. Patil University*. 2016; 9: 341-347.
- [10]. Boyd PA, Scott A. Quantitative structural studies on human placentas with pre-eclampsia, essential hypertension and intrauterine growth retardation. *British Journal of Obstetrics & Gynaecology*. 1985; 92: 714-721.
- [11]. Asgharnia M, Esmailpour N, Poorghorban M, Atrkar-Roshan Z. Placental weight and its association with maternal and neonatal characteristics. *Acta Medica Irania*. 2008; 46(6): 467-472.
- [12]. Raghavendra AY, Vinay KV, Pai V. A study of placental weight and fetal outcome in different grades of pregnancy induced hypertension. *International J of Anatomy and Research*. 2014; 2(4): 625-29.
- [13]. Navbir P. Placental morphology and its co-relation with foetal outcome in pregnancy induced hypertension. *International J of Basic and Applied Medical Sciences*. Sep-Dec 2012; 2 (3): 120-125.
- [14]. Das B, Dutta D, Chakraborty S, Nath P. Placental morphology in hypertensive disorders of pregnancy & its co-relation with foetal outcome. *J of Obstetrics & Gynaecology of India*. 1996; 46: 40-46.
- [15]. Dutta DK, Dutta B. Study of human placenta associated with pre eclampsia and essential hypertension in relation to foetal outcome. *J of Obstetrics & Gynaecology of India*. 1989; 13: 757-763.
- [16]. Thomson AM, Billewicz WZ, and Hytten FE. The weight of the placenta in relation to birthweight. *The J of Obst & Gynae of B Cwelfth*. 1969; 76(10): 865-872.
- [17]. Ahmed M, Daver R. Study of feto-maternal outcome in pregnancy induced hypertension. *Global Journal of Medical Research (E) Gynaecology and Obstetrics*. 2014; 14(1): 21-25.
- [18]. Shah RK, Jagiwala KS, Vyas PK. Placental morphology & foetal growth in normal and abnormal pregnancies. *J Obstet Gynae of India*. 1985; 35: 1089 - 1094.
- [19]. Rath G, Garg K et al. Vascular pattern of human placenta in complicated pre-pregnancy, a corrosive cast study. *Ann Nat Med Sc (Ind)*. 1994; 30: 17-22.
- [20]. Naeye RL. Disorders of the placenta, fetus and neonate diagnosis and clinical significanc. *Mosby Year Book*. 1992; 1st edi. 92-179.
- [21]. Mohan H, Sodhi S, Mohan PS, Jaiswal TS, Nagpal R, Rathee S. Foetal correlation with placental pathology in toxemia of pregnancy. *J of Obstetrics & Gynaecology of India*. 1989; 39(1-6): 170- 175.
- [22]. Macpherson T. Fact and Fancy - What can we really tell from the placenta? *Arch Pathol Lab Med*. 1991; 115: 672- 679.
- [23]. Mukherjee B and Ram Lal. Relation between foetal weight and placental size. *J. Anat. Soc. India*. 1983; 32(3): 124-126.
- [24]. Chakravorty AP. Foetal and placental weight changes in normal pregnancy and pre-eclampsia. *J.Obstet. Gynaec. Bri. Cwlth*. 1967; 74: 247-253.
- [25]. Davies JP, Beazley JM. A study of placental size and chorio- amnionitis in a consecutive series of hospital deliveries. *J Obstet Gynaec Brit Cwlth*. 1973; 80: 246-251.
- [26]. George JN, Amaresh A. Neonatal mortality and morbidity in pregnancy induced hypertension: A prospective observational study. *Journal of Evolution of Medical and Dental Sciences*. 2014; 3(4): 5238-5246.
- [27]. Kaur P, Kaushal S, Singh K, Sharma A. Placental weight, Birth weight and fetal outcome in preeclampsia and normotensive pregnancies. *International Journal Plant, Animal and Environmental Sciences*. 2013; 3(4): 30-33.
- [28]. Meshram DP, Chavan YH, Kadam PN, M.G, Ramteke DJ. Maternal and foetal outcomes in pregnancy induced hypertension – A hospital based study. *International Journal of Pharmaceutical sciences Invention*. 2014; 3(4): 23-26.
- [29]. Bangal VA, Giri PA, Mahajan AS. Maternal and foetal out come in pregnancy induced hypertension: A study from rural tertiary care teaching hospital in India. *International Journal of Biomedical Research*. 2011; 2(12): 595-599.
- [30]. Younoszai MK, Haworth JC. Placental dimensions and relations in preterm, term and growth retarded infants. *Am J. Obst. & Gynec*. 1969; 103 (2): 265-271.

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