Original Research Article

Placental changes and its fetal implication in pregnancy induced hypertension

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Abstract

Background: In developing countries like India, pregnancy induced hypertension(PIH) accounts for a sizable number of perinatal deaths. In PIH because of placental insufficiency, the placental coefficient is higher and has been adequately correlated to the fetal outcome, associated with fetal growth retardation. **Methods:** 200 consecutive births to mothers with pregnancy induced hypertension which took place between March 2000 February 2001 in HQH Hospital, Bellary were included in the present study. **Results:** There was decrease in the mean placental and birth weight with increasing severity of PIH. With placental weight less than 300 gm, there was 80% of incidence of IUD and 100% incidence of prematurity and neonatal deaths. **Conclusion:** PIH significantly affects the placenta by reducing its weight and dimensions, by increasing extent of placental infarcts resulting in poor fetal growth and perinatal deaths.

Key Words: Pregnancy induced hypertension(PIH), Placenta, Perinatal outcome, IUD

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INTRODUCTION

In developing countries like India, pregnancy induced hypertension(PIH) accounts for a sizable number of perinatal deaths. In addition to the adverse effects of drugs used in the management of PIH Some non-therapeutic factors like maternal age, parity, socio economical status, maternal anemia, proteinuria etc. are also found to have their role in perinatal outcome in PIH.[1] In the presence of maternal disease, the fetus will be at risk due to decreased placental reserve, the maternal vascular responses to placentation will be inadequate. Placental infarcts is the most familiar of all placental lesions with uterovascular disease. A fetus may survive 20% to 30% infarction of the placenta, provided the remaining has good maternal blood supply, where as in severely ischaemic placenta associated with fetal growth retardation, the infarction of a single cotyledon may be enough to precipitate fetal death. Any marked deviation in placental coefficient from 0.08 to 0.2 is associated with abnormally large or small placenta respectively. In PIH because of placental insufficiency, the placental coefficient is higher and has been adequately correlated to the fetal outcome.[2] The weight of placenta and the weight of the newborn are readily accessible data and much can be learnt from their proper interpretation. In PIH maternal blood flow to the placenta is reduced which results in unduly small fetus. Since placenta is the fetal organ, it shares in the depression of fetal growth. The small placenta is regarded as manifestation of poor fetal growth.[3] So the present study was under taken as to know the placental changes and its fetal implication in pregnancy induced hypertension.

MATERIALS AND METHODS

200 consecutive births to mothers with pregnancy induced hypertension which took place between March 2000 February 2001 in HQH Hospital, Bellary were included in the present study. Mothers were diagnosed to

have pregnancy induced hypertension as per the standard criteria laid down by American College of Obstetric and Gynecology. Mothers with other major illness like diabetes, heart disease, renal disease, were excluded from the study. In all cases, placenta was examined soon after the delivery keeping it in a large pan. Placenta was washed and weighed after removing any retroplacental clots. Both fetal and maternal surfaces were examined. The number and extent of placental infarcts was recorded.

RESULTS

Total of 200 consecutive births to mothers with pregnancy induced hypertension out of 2325 deliveries were studied. The incidence of preterm was 31% and SGA was 17.7%. Perinatal mortality rate in the study was 345/1000.

Table 1: Mean Placental and Birth weight In relation to severity Of PIH

PIH GRADING	MEAN PLACENTA WEIGHT (gms)	MEAN BIRTH WEIGHT(gms)	FETO PLACENTAL RATIO
Gestational Hypertension	515.667	2670.74	5.2
Mild	508.810	2357.14	4.6
Servere	481.193	2067.39	4.3
Eclampsia	432.093	1726.51	4.0

Table 1 shows mean placental and birth weight in relation to severity of PIH. There was decrease in the mean placental and birth weight with increasing severity of PIH, with corresponding decrease in feto placental ratio.

Table 2: Perinatal outcome in relation to Placental weight

Placental weight (in grams)	No of Cases	For live births				
		Pre-term	Term	Sga	Neonatal Deaths	Iud/sb
< 300	11(5.5)	2(100.0)	0(00.0)	1(50.0)	2(100.0)	9(81.8)
301 to 500	122(61.0)	45(50.6)	44(49.4)	27(30.3))	25(28.1	33(27.1)
Above 500	67	2 (03.0)	65 (97.0)	0(00.0)	0(0.00)	0(00.0)
		P<0.001		P<0.001	P<0.001	P<0.001

Table no 2 shows the perinatal outcome in relation to placental weight. With placental weight less than 300 gm, there was 80% of incidence of IUD and 100% incidence of prematurity and neonatal deaths. Decreasing placental weight was associated with poor perinatal outcome which was statistically significant.

Table 3: Perinatal outcome in relation to Placental infarcts

PLACENTAL INFARCTS (%)	NO OF CASES	FOR LIVE BIRTHS				
		PRE-TERM	TERM	SGA	NEONATAL DEATHS	IUD/SB
0	95(47.5)	13(13.7)	82(86.3)	2(02.1)	2(02.1)	0(00.0)
1 to 5	23(11.5)	7(33.3)	14(66.7)	6(28.6)	5(23.8)	2(08.7)
6 to 10	51(25.5)	19(59.4)	13(40.6)	16(50.0)	12(38.7)	19(37.3)
Above 10	31(15.5)	10(100.0)	0(0)	4(40.0)	8(80.0)	21(67.7)
		P<0.001		P<0.001	P<0.001	P<0.001

Table no 3 shows perinatal outcome in relation to extent of placental infarction. 95 cases of PIH (47.5%) did not have any placental infarcts. There were no cases of IUD in this group, when the extent of placental infarct was more than 10%, IUD incidence was 67% and 80% of the babies died in neonatal period. Also higher incidence of SGA was observed with increasing extent of infract.

DISCUSSION

200 consecutive births to mothers with PIH were studied. The incidence of PIH in the study was 8.6%. In the study group, 83 babies were born preterm. Incidence of prematurity was 41.5%. The incidence was highest in eclamptic cases (62.8%). Incidence of prematurity was noted to be 23% by Githa *et al*[4], and 58.1% by Sibai *et al*.[5] The incidence of small for gestational age was 17%. Incidence of SGA was noted to be 29% by Brazy *et al*[6] and 20% by Sibai *et al*.[5] Though there are multiple reasons for IUGR, the increased incidence in PIH probably reflects the severity of uteros placental insufficiency. Perinatal mortality rate in the study was

345/1000 which was 4.5 times higher than general PNMR of the hospital (77.4/1000). Perinatal mortality reported by Githa *et al*[4] was 197.2/1000 and Jain *et al*[7] was 106/1000.

Placental changes and perinatal outcome

A linear correlation was observed between reduced placental weight and increased perinatal morbidity and mortality. With placental weight less than 300 gm the incidence of IUD was 82% and 100% incidence of neonatal deaths. Similar observations were made by Kher *et al* [3]in which all still born fetuses had placental weight of less than 300 gm, Mirchandani *et al*[8] also found increased incidence of fetal distress with placental weight less than 300 gm. In PIH maternal blood flow to the placenta is reduced. Limitation of placental blood flow results in unduly small fetus. It is suggested that since placenta is the fetal organ, in the depression of fetal growth. The small placenta is regarded as manifestation of poor fetal growth.[3]With increasing extent of placental infarcts, unfavourable perinatal outcome was observed. With extent of infarction more than 10% IUD incidence was 67.7% and 80% of the babies died in neonatal period. Kher *et al*[3] also noted similar observation. In their study when extent of infarction was more than 30%, 6 of 14 babies were still born. Extensive infarction indicates widespread thrombosis of maternal vessels. This lesion superimposed on a placenta whose functional reserve is already dissipated worsens uteroplacental ischemia resulting in high incidence of fetal hypoxia, intrauterine growth retardation and death.[2]

CONCLUSION

PIH significantly affects the placenta by reducing its weight and dimensions, by increasing extent of placental infarcts resulting in poor fetal growth and perinatal deaths. However, it does not have any effect on placental shape, umbilical cord insertion, and number of cotyledons on maternal surface. Hence high risk patients of PIH should be appropriately counselled and must be managed with adequate neonatal intensive care facilities to improve the outcome.

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