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Pregnancy Induced Hypertension and Prior Trophoblastic Exposure

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OBJECTIVE – To find out if a prior trophoblastic exposure in a gravid woman is associated with a lower incidence of pregnancy induced hypertension (PIH). **METHODS** – Data from 317 cases delivered under the authors supervision from 1st January 2002 to 31st December 2002 were analysed. Incidences of PIH were noted. Parity was noted in each case. **RESULTS** – Out of 317 women, 65.6% were primigravidas. Overall incidence of PIH was 15.5%. Statistical analysis ("Z" test) of the data showed that there were no statistical differences between the incidences of PIH among the different groups based on gravidity. **CONCLUSION** – Prior trophoblastic exposure does not give protection against development of PIH in subsequent pregnancy.

Key words : pregnancy induced hypertention, gravidity

Introduction

Pregnancy induced hypertension (PIH) has been defined as hypertension developing in pregnancy as a result of the gravid state. It may or may not be associated with edema and/or proteinuria. The incidence of preeclampsia is about 6-18% of all the mulliparous pregnancies[1]. But this frequency varies depending on the diagnostic criteria and the population studied. It is theorized that a prior exposure to trophoblasts may induce some protective effect on the immunological mechanism in a subsequent pregnancy[2,3]. The present study attempts to find out if a prior trophoblastic exposure in a gravid woman is associated with a lower incidence of PIH.

Methods

Case records of 317 women delivered under the author's supervision in one year from 1st January 2002 to 31st January 2002 were analysed. During the antenatal period, all women had routine antenatal care. After delivery the following parameters were noted down for the study:

1. Gravidity : Whether the women was a primigravida or a multigravida.
2. If multigravida details of her past obstetric performance -
   a) whether there was any history of abortion and if so, whether it was induced or spontaneous.
   b) prior delivery beyond 28 weeks of gestation.

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PIH was diagnosed in the antenatal period whenever the blood pressure was elevated above 140/90 mm Hg on at least two occasions at least 6 hours apart.

Women with diabetes mellitus, known hypertension, heart disease, renal disease, collagen diseases and multiple pregnancy were excluded from the study – .

The cases were grouped as follows-

Group I – primigravid women (P_{1+0} after delivery)

Group II – women having history of abortion (P_{0+1}) –

a) those having history of spontaneous abortion

and b) those having history of induced abortion.

Group III – those having history of prior delivery beyond 28 weeks –

Group IV – those having combinations of above.

Results

The overall incidence of PIH in the present series was 15.5% (49/397).

Table I shows the distribution of the various groups of women as classified in the present study. Group I formed the largest bulk viz, (65.6%).

Table II shows the incidences of PIH as found in the various groups. There is a gradual decrease in the incidence of PIH from Group I to Group IV.

Discussion

The overall incidence of PIH was 15.5%. The incidence is highest among the women who have conceived for the first time (18.3%) and the incidence decreases if there is a history of prior exposure to trophoblasts. It is theorized that a prior trophoblastic exposure confers

in a low resource environment like ours, but majority (77.6%) of the women preferred to have prophylactic cerclage with its associated extra cost rather than choosing an insignificantly lower bill with increased chance of preterm delivery and its associated morbidity and mortality. Interestingly 84.0% (21/25) patients who opted not to have cerclage in their next pregnancy were among the controls without preterm delivery. This was not unexpected since without intervention they did not have preterm delivery and did not see any rational reason for paying extra bills and risk surgery.

Application of prophylactic cerclage in pregnant women with previous preterm delivery has been shown to confer an advantage by reducing preterm delivery rate by 27.6% at a reasonable cost acceptable to women with no additional risk to the mother and baby. Embracing this strategy may reduce the social, economic and emotional stress associated with preterm delivery especially in resource poor countries.

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Table I. Incidences of various groups of patients as classified in the present study

Group	Number of cases	Percent
Group I	208	65.6
Group II a	31	9.8
Group IIb	13	4.1
Group III	41	12.9
Group IV	24	7.6
Total	317	

Table II. Incidences of PIH as found in the various groups of patients (n=317)

PIH	Group I		Group II a		Group II b		Group III		Group IV	
	No.	Percent	No.	Percent	No.	Percent	No.	Percent	No.	Percent
Yes	38	18.3	4	13	1	7.6	1	2.4	5	20.8
No	170	81.7	27	87	12	92.4	40	97.6	19	79.2
Total	208		31		13		41		24	

Number of cases with PIH- 49 (15.5%)

some degree of immunity to the maternal system. When there is a history of induced abortion where the trophoblasts are viable and healthy (Group IIB) the degree of immunization may be more compared to when there is a history of spontaneous abortion where the trophoblasts are dead or abnormal (Group IIA). A full term pregnancy gives more protection against PIH in subsequent pregnancy (Group III). Saftlas et al[3] have found that prior birth conferred a strong protective effect against preeclampsia, whereas a prior abortion conferred a weaker protective effect. They also found that parous women who change partners in a subsequent pregnancy appear to loose the protective effect of a prior birth. They had also proposed an immune-based etiologic mechanism whereby prolonged exposure to fetal antigens from a previous pregnancy protects against preeclampsia in a subsequent pregnancy.

But it must be remembered that it is not the immunological mechanism that is solely responsible for the development of PIH. No single theory can explain all the situations. Theories that have been mentioned include poor placentation, hyper-placentosis, systemic reaction etc[4]. On comparison of Group I and II, applying "Z" test for hypothesis testing between group proportions at 95% confidence interval the calculated statistical value lies within the range of – 1.96 to + 1.96, the value is 0.75 and Odds Ratio (OR) is 1.69. Thus the difference in the incidence of PIH between Group I and Group II is not significant. Similarly, between Group III, and Group I the calculated statistical value is 1.02 and when tested within the aforesaid confidence limits with OR being 7.33, the same conclusion is drawn. Again between Group I and Group IV, the calculated statistical value is 1.37 and OR 0.85, and the same conclusion is drawn. Thus, the overall conclusion is that there is no significant difference in PIH incidences among the four groups of women.

Brown[5] had found a high recurrence rate of pregnancy induced hypertension in second pregnancy. Similarly, Parazinni et al[6] had found that parous

women were at decreased risk of PIH in comparison with nulliparous women the OR were 0.7 (95% C.I. = 0.4 – 1.0) and 0.5 (95% CI = 3 - 0.9) respectively, in women reporting 1 or > 2 births. They found no important relation between previous spontaneous or induced abortion and PIH risk.

This retrospective study of 317 women shows that the overall incidence of PIH is 15.5%. Statistical analysis of the data shows that the theory of prior trophoblastic exposure conferring protection against PIH in subsequent pregnancy is not valid

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