PREDICTION OF PREGNANCY INDUCED HYPERTENSION BY MEAN ARTERIAL PRESSURE OF SECOND TRIMESTER

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Perinatal mortality rate (PMR) in pregnancy induced hypertension (PIH) is directly related to the severity of the hypertensive disease (Jain and Dhall, 1980). Whatever can be done to make an early diagnosis of PIH and prevent its progression to severe forms of the disease in late pregnancy, could improve fetal and neonatal survival rates.

Recent studies by Gant and associates (1972, 1972, 1974) have shown that PIH is chronic in development and can be recognised early in gestation before the patient has any subjective complaints by utilising the angiotensin infusion test. Dehydroisoandrosterone sulphate loading test and supine pressor test. Place of supine pressure test is controversial and its value has not been confirmed in a preliminary study carried out at our Institute (Dhall and Dhall, 1979). Other two tests are time consuming and thus have no place in developing countries where over crowding in hospitals leads to such screening for PIH impractical.

Page and Christianson (1976) observed that second trimester mean arterial pressure (MAP2) accurately detected patient at risk for PIH before its clinical onset. The present study has been carried out to find out correlations of MAP2 with the development of PIH in third trimester, proteinuria, intrauterine growth retardation (IUGR) and PMR.

Material and Methods

Five hundred and ninety-nine, initially normotensive pregnant women with singleton pregnancy with no known history of renal disease were seen at monthly intervals from 16 weeks of amenorrhoea onward and their blood pressure was measured in supine position. The women were closely followed to study blood pressure changes, development of PIH, proteinuria and fetal outcome.

The MAP was calculated by Burton's formula $MAP = \frac{\text{Systolic} + 2\ \text{diastolic}}{3}$ mm.Hg.

All blood pressure recordings of 5th and 6th months were averaged to obtain MAP2. The diagnosis of PIH was made according to the criteria of American Committee of Maternal Welfare. Proteinuria was qualitatively assessed by conventional boiling technique and expressed as 1+ to 4+. IUGR was defined as birth weight below one standard deviation (— 1 SD) of mean weight for gesta-

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tion according to growth charts of pediatrics department of our Institute.

Results

Study included 53.58 per cent primipara, 24 per cent second para, 15.52 per cent third para and 6.84 per cent para 4 and above, in the age group of 19 to 36 years.

MAP2 and PIH

Table I shows the rising trend in the incidence of PIH with each 5 mm Hg rise in MAP2 occurring irrespective of parity, with distinct increase noted at MAP2 greater than 90 mm Hg. If MAP2 was higher than 90 mm of Hg, 49.19 per cent women developed PIH while 8.73 per cent women with MAP2 of 90 or less developed PIH. The results were statistically significant ($P < 0.01$). There was no statistical difference observed in the incidence of PIH and primiparas (23.9 per cent), second (19.4 per cent) and third parous women (20.4 per cent).

Proteinuria and IUGR. 2.9 per cent of study population developed proteinuria of 1+ or more. There was only 1 case of proteinuria without PIH which was detected to have urinary tract infection. Table II shows the clear rising trend in proteinuria IUGR with increasing level of MAP2.

PMR: The fetal outcome in 599 single births is shown in Table III. No consistent correlation of perinatal loss was observed with rising level of MAP2. Unfavourable fetal outcome in women was largely

TABLE I

Correlation of MAP_2 and Pregnancy Induced Hypertension (PIH)

MAP_2 (mm Hg)	PIH (Percentage)				
	Para I	Para 2	Para 3	Para 4 and above	Total cases
Less than 80	0	0	0	0	0
81 - 85	10	3.57	10.52	0	7.75
86 - 90	18.1	16.6	5.26	16.6	15.6
91 - 95	50	46.6	33.3	11.1	43.93
96 - 100	57.1	50	50	25	50
Above 100	90	60.6	100	100	84.2
Total	23.9	19.4	20.4	9.7	21.36

TABLE II

Correlation of Proteinuria and IUGR with MAP_2

MAP_2 (mm Hg)	IUGR		Proteinuria	
	No.	Percent	No.	Percent
Less than 80	5/65	7.6	1*	1.5
81 - 85	26/232	11.2	3	1.2
86 - 90	16/115	13.9	2	1.7
91 - 95	24/132	18.18	6	4.5
96 - 100	10/36	27.7	2	5.5
Above 100	8/19	42	4	21.0

* Proteinuria without PIH.

TABLE III
Perinatal Mortality

Age	Parity	MAP_2	PIH	Gestation (weeks) at birth	Birth weight (kg)	Perinatal deaths
24	$P_2+0+0+0$	83	Yes	37+	2.4	MSB
23	$P_1+0+0+0$	86	No	33+	1.1	FSB
29	$P_1+0+4+0$	96	Yes	33	1.1	FSB
27	$P_0+2+0+0$	103	Yes	36	1.3	MSB
33	$P_1+0+2+1$	103	Yes	37	1.9	Late NND

dependent upon the subsequent development of PIH. Four perinatal deaths occurred in PIH group and 3 of these had associated proteinuria. The only stillbirth in non PIH group occurred in a woman with earlier terms macerated stillbirth, who presented with urinary tract infection, proteinuria and high fever at 32 weeks.

Discussion

Early identification of patients who develop PIH during pregnancy has been a long standing goal of clinicians. A number of different procedures have recently been developed to improve detection. While not entirely accurate as a method for detection of PIH, the MAP2 meticulously obtained during 5th and 6th months, may help to identify those patients who are destined to develop PIH in 3rd trimester. Our experience shows that a normotensive women with MAP2 higher than 90 mm Hg had a 50 per cent risk of developing PIH. Conversely, 91.27 per cent women with MAP2 of 90 mm Hg or less remained normotensive throughout the rest of pregnancy. Page and Christianson (1976) reported that MAP2 of 90 or over signifies high risk for the development of PIH. Fallis *et al* (1963) observed that 34.5 per cent women with MAP2 greater than 90 developed PIH while Karna (1979) showed such correlation in only 28.3 per cent cases.

With respect to IUGR and proteinuria, our data supports the views of Page and Christianson (1976) that linear relationship exists between MAP2 and the later development of proteinuria and IUGR. Comparison of figures of the present study and that of Page and Christianson (1976) is not done as the criteria used in the two studies are different.

Our results show that perinatal loss is directly related to the development of PIH in the third trimester. No definite relationship could be obtained with any absolute level of MAP2. We are not in a position to support or contradict Page and Christianson view that gestational hypertension without proteinuria does not place the infant to any increased risk. Larger number of women still need to be assessed.

Many obstetricians consider that PIH is a disease only of primigravidas. Present study showed that multiparous women not only developed PIH with equal frequency but also showed similar correlation of MAP2 to later development of PIH.

Summary

A prospective study on 599 women with singleton pregnancy was conducted to determine the relationship of MAP2 to

later development of PIH, IUGR, proteinuria and PMR. A linear relationship was obtained of MAP2 to later PIH, IUGR and proteinuria. No correlation of PMR was observed with rising level of MAP2.

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