

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

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SUMMARY

The present study was undertaken to establish the reliability of mean arterial pressure in second trimester (MAP_2) of pregnancy in predicting the development of pregnancy induced hypertension (PIH) before its clinical onset. There was a rising trend in occurrence of PIH with each 5 mm of Hg increment of MAP_2 with a distinct increase noted at 90 mm of Hg, the critical point. 57.1% of the patients with MAP_2 more than 90 mm of Hg developed PIH while 88.3% of patients with MAP_2 of 90 mm of Hg or less remained normotensive.

Introduction

Pregnancy induced hypertension is one of the most serious complications of pregnancy. Its prevention and treatment would be better if we could identify the patients who are likely to develop pre-eclampsia.

The impact of blood pressure upon the circulation dynamics is best expressed by utilizing mean arterial pressure where a single figure is derived from systolic and diastolic blood pressure readings. The formula advocated by Burton (1965) is used i.e.

$$\text{Mean arterial pressure (mm of Hg)} = \frac{\text{Systolic} + 2 \text{ diastolic}}{3}$$

The present study has been undertaken to provide a definite evaluation of this criteria as a diagnostic aid in predicting pregnancy induced hypertension.

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Material and Methods

In this study a total of 100 pregnant patients in the age group of 19-40 years coming for antenatal check up at Government Hospital for Women/Medical College, Amritsar, were closely followed up to study blood pressure changes and development of PIH. All the patients studied carried singleton pregnancy of 12-16 weeks gestation and were normotensive at the onset of pregnancy.

Blood pressure was recorded with the patient lying down, relaxed, her forearm horizontal and well supported, upper arm level with her heart, blood pressure cuff applied firmly without creasing and inflated and deflated smoothly. The systolic pressure was recorded when Korotkoff's sounds appeared and diastolic pressure with disappearance of sounds i.e. phase V. MAP was then calculated by using the Burton's formula. Blood pressure recordings at 16th, 20th and 24th week were averaged to obtain mean

arterial pressure of second trimester (MAP_2) and correlated with blood pressure readings at 28th, 32nd, 36th and 40th week to predict the development of PIH according to criteria of American Committee of Maternal Welfare.

Observations

Mean arterial pressure of second trimester (MAP_2) bears a linear relationship to the occurrence of PIH irrespective of parity (Table I).

TABLE I
Showing Linear Relationship of Incidence of PIH with Each 5 mm of Hg Rise in MAP_2

MAP_2 (mm of Hg)	Total patients	Patients with PIH
Less than 80	55	0%
81-85	18	5 (27%)
86-90	13	5 (38.5%)
91-95	6	3 (50%)
96-100	6	4 (66.6%)
More than 100	2	1 (50%)

When MAP_2 was more than 90 mm of Hg 57.1% women developed PIH while only 11.7% of women developed PIH when MAP_2 was 90 mm of Hg or less. The results were statistically significant ($p < 0.01$).

Patients with increasing parity were at increasing risk to develop PIH when

MAP_2 was more than 90 mm of Hg. Primiparae were at 50% risk to develop PIH whereas multiparae were 66.6% prone to develop PIH (Table II). When MAP_2 was less than 80 mm of Hg there was no risk at all but with MAP_2 between 80-90 mm of Hg, 11.7% women developed PIH.

The increasing age was not found to influence the development of PIH. The incidence was rather higher in patients of less than 20 years in age and then showed a decline.

Twenty per cent of patients with a past history of PIH showed positive correlation in the present pregnancy and 44.5% of these patients had haemoglobin less than 10 gm%. Five per cent of this study group developed proteinuria of 2+ or more.

Discussion

PIH can be recognised early in gestation long before the patient has any subjective complaints. Increased vascular response to pressor substances could be seen as early as 22 weeks of gestation (Gant *et al*, 1972). Reduced plasma volume was reported to precede the development of PIH (Chesley, 1972; Gallery *et al*, 1979). However, these types of biochemical studies are expensive, require

TABLE II
Showing Correlation of MAP_2 and PIH, Paritywise

MAP_2 (mm of Hg)	PIH			
	Para 1	Para 2	Para 3	Para 4 and above
Less than 80	19.0 (0)	20.0 (0)	8.0 (0)	8.0 (0)
81-85	12.4 (33)	4.1 (25)	2.0 (0)	—
86-90	2.1 (50)	4.2 (50)	5.2 (40)	2.0 (0)
91-95	4.2 (50)	2.1 (50)	—	—
96-100	4.2 (50)	—	1.1 (100)	1.1 (100)
More than 100	—	—	—	2.2 (100)
Total	41.9 (21.9)	30.3 (10)	16.3 (18.8)	13.3 (23.1)

sophisticated laboratory equipment and trained personnel. Therefore, the attention was directed towards clinical screening of antenatal patients.

Gant *et al* (1974) employed roll over test for antenatal screening of the patients. Thompson and Mueller (1978) studied the efficacy of this test and found 90.95% accuracy for a negative test and 60% accuracy for a positive test.

Page and Christianson (1976) observed that second trimester mean arterial pressure could accurately detect patients at risk for PIH before its clinical onset. Bhatia and Jain (1983) reported that with MAP₂ of more than 90 mm of Hg, 50% of the patients developed PIH. In our series, a positive test accurately predicted the development of PIH in 57.1% of patients while a negative test predicted that PIH will not develop in 88.3% of patients.

The rising trend in occurrence of PIH with each 5 mm increment of MAP₂ seen

in the present study is in agreement with the reports of Page and Christianson (1976) and Bhatia and Jain (1983).

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