

# GESTATIONAL DIABETES AND PREECLAMPTIC TOXAEMIA

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Gestational diabetes (GD) is considered to be one of the risk factors for the development of pre-eclamptic toxæmia (PET). Singh (1976) reported that 10 out of 15 patients with severe PET and 4 out of 15 cases of mild PET had abnormal glucose tolerance test (GTT). On the other hand, a large number of patients with PET do not develop GD. This discrepant observation remains unexplained by the current knowledge on the effect of PET on carbohydrate metabolism. This prospective study was undertaken to evaluate the association of PET with GD and to assess the nature of disturbance in carbohydrate metabolism in PET.

## Material and Methods

One hundred and thirty patients, diagnosed as mild to severe PET using criteria of American Committee of Maternal Welfare, formed the study group of the present study. All known diabetic patients were excluded from the study. These patients were carefully followed up in the antenatal clinic of our hospital. During the third trimester of pregnancy, all patients were subjected to oral GTT using

100 gm of glucose. The blood sugar was estimated on autoanalyser using glucose oxidase method. Criteria for the diagnosis were similar to those of O'Sullivan and Mahan (1964). 30 patients with normal pregnancy, during third trimester, constituted the control group.

## Results

Out of 130 patients of PET, GD was detected in 27 patients (20.7 per cent). Of these, it was diagnosed in 6 out of 20 patients with severe PET, and 21 out of 110 patients with mild PET as against only 1 abnormal GTT in 30 control cases (Table I). The Fasting blood sugar levels of patients with mild and severe PET and women with normal pregnancy have been compared in Table II.

The fasting blood sugar levels in severe PET group (68 mg per 100 ml) is significantly lower than the mean fasting blood sugar level in the control group and in patients with mild PET. However there was no statistical difference between the mild PET group and the controls.

## Comments

Scant literature coupled with variability in the diagnostic criteria and technique of blood sugar estimation has led to considerable controversy on the precise nature of alteration in the carbohydrate

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TABLE I  
Incidence of Gestational Diabetes in PET & Normal Pregnancy

	Number of Patients	No. of GD	Percent
Severe PET	20	6	30
Mild PET	110	21	19
Normal Pregnancy	30	1	3.33
	Severe versus normal	p <0.01	
	Severe versus mild	p >0.01	
	Mild versus normal	p <0.05	
	All PET versus normal	p <0.05	

TABLE II  
Fasting Blood Sugar Levels in Patients With Pet & Controls

	No. of patients	Mean Fasting blood sugar level mg per 100 ml with SD
Severe PET	20	68.0 ± 7.6
Mild PET	110	79.4 ± 13.2
Normal pregnancy	30	82.8 ± 11.0

metabolism in patients with PET. Controversy also exists whether GD results as a complication of PET or it in some way contributes to the occurrence of PET. Our data suggest that 20.7 per cent of the patients with PET were vulnerable to the development of GD. It has been shown that there is significantly higher perinatal

mortality in GD complicated with PET as compared to group of patients with GD but without PET (Agarwal and Gupta, 1979). The present data is in agreement with the observations of Singh (1976) and his hypothesis on the association of GD with PET. The influence of PET on carbohydrate metabolism needs further investigations to identify the precise mechanism by which PET contributes to GD.

References

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