SERUM MUCOPROTEIN LEVELS IN NORMAL AND TOXAEMIC PREGNANCIES

by

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Introduction

Toxaemia has always been classified as a disease of varying aétiology. In recent years, an interest has been shown regarding the immunological basis of toxaemia of pregnancy. Various observations have led to this understanding. Platt et al (1958) postulated a single gene locus of maternal antigen passing into the foetus and causing toxaemia. Stevenson and Dawson (1971) found the incidence of consanguinity less in toxaemia patients as compared to the general population. Bardwil and Toy (1959) produced pre-eclampsia experimentally by injection of antisera prepared against placental homogenates. Histologically, the decidua of toxaemic patients shows a marked lymphocytic infiltration in puerperium (Robertson *et al* 1967). Jenkins *et al* (1973) observed a higher transformation index of lymphocytes in mixed lymphocytic culture after washing away the serum of toxaemic patients which contained higher levels of seromucoid. These authors believe that the immunological disparity in toxaemia was proved by the higher transformation index and the raised levels of seromucoid was a protective reaction or failure of normal protective mechanism.

Serum seromucoid levels increase in pregnancy, especially near term and are higher at term and puerperium in some cases of pre-eclampsia and eclampsia (Jenkins *et al* 1973).

Methods and Material

The subjects for the present study were selected from the indoor and outdoor wards of UISE Maternity Hospital, Kanpur.

The control group had 48 normal healthy adult females, in whom pregnancy was carefully excluded.

Twenty-nine cases of pre-eclampsia and eclampsia were studied which were further sub-divided into 3 groups.

A. Mild PET—BP between 140/90 and 160/100 after the 20th week without proteinuria.

B. Severe PET—B.P. 160/100 with oedema and proteinuria.

C. Eclampsia.

Mucoprotein levels were estimated in:

(i) Amniotic fluid (collected by amniotomy or during rupture of membranes).

(ii) Maternal blood, and (iii) Cord blood.

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TABLE I

Mean Total Protein in Normal Pregnancies

Specimen	No. of estimations	Total protein in gm/dl ± SD
1. Mat. Sera	90	5.65 ± 0.05
2. Cord Sera	90	4.92 ± 0.08
3. Amniotic fluid	90	0.75 ± 0.02

TABLE II

Variation in Total Protein at Delivery and Puerperium in Normal Pregnancy, PET and Eclampsia

Type of case	No. of estimations	Mean total protein in gm/dl ± S.D.
Control	40	6.6 ± 0.82
Normal pregnancy (3rd trimester)	18	5.6 ± 0.60
Normal pregnancy at delivery	50	5.4 ± 0.70
PET in 3rd trimester	23	5.18 ± 0.60
Eclampsia in 3rd trimester	6	4.2 ± 0.80
PET mothers at delivery	23	5.06 ± 0.02
Eclampsia mother at delivery	6	5.04 ± 0.04

Note:—The fall in total protein is more marked in pregnancy associated with toxaemia. Mean S.M.P. level in normal controls (40) was 50.31 mg/dl ± 15.45 the range being 37.5—67.5 mg/dl.

The samples were analysed for total protein content by the Biuret method (Wooton. 1969). The SMP concentration was estimated by the method of Weimer and Mohsin (1953) in terms of its protein content.

Observations

The mean SMP levels in the 3rd trimester showed a steady rise with increasing degree of toxaemia.

TABLE IV

Mean SMP Levels in Cord Blood

Type of case	No. of estimations	Mean SMP in mg/dl ± S.D.
Normal pregnancy	38	19.8 ± 0.04
Mild PET	13	22.8 ± 2.26
Severe PET	10	24.9 ± 3.21
Eclampsia	6	32.7 ± 6.12

A gradual rise of cord SMP was seen with increasing degree of toxaemia.

A significant (sigt) rise of SMP levels is seen in all the three groups. Level's in PET and eclampsia are persistently higher than normal pregnancy.

TABLE III

Mean SMP Levels in the Third Trimester of Pregnancy in Normal Pregnancy, PET and Eclampsia

Type of cases	No. of cases and %	Mean SMP in mg/dl	± S.D.
Normal pregnancy	38 (56.70)	59.10	± 1.28
Mild PET	13 (19.50)	126.50	± 4.41
Severe PET	10 (14.90)	125.80	± 3.82
Eclampsia	6 (8.90)	129.36	± 6.23
Total	67		
X_2	71.32		
P value	< 0.05 (Significant)		

TABLE V

Mean SMP Levels of Puerperium in Normal Pregnancy and Toxaemia

Puer-peral day	Normal pregnancy			PET			ECLAMPSIA		
	No.	Mean	± S.D.	No.	Mean	± S.D.	No.	Mean	± S.D.
0-2	12	78.80	± 2.8	6	198.60	± 4.2	1	200.20	± 8.9
3-4	20	156.50	± 4.9	8	214.70	± 3.3	3	224.50	± 12.2
5-7	18	188.16	± 6.2	9	220.8	± 7.2	2	246.50	± 4.8
Total	50			23			6		
X_2	72.41			88.70			7.26		
P value	< 0.05 (Significant)			<0.05 (Significant)			<0.05 (Significant)		

TABLE VI

Mucoprotein Levels in Amniotic Fluid

Type of Cases	Meconium stained samples (A)			Unstained samples (B)			't' ratio of difference of Mean A & B	P Value
	No. %	Mean SMP mg/dl	± S.D.	No. %	Mean SMP mg/dl	± S.D.		
Normal Pregnancy	50 (56.20)	223.2	±10.6	50 (56.20)	171.5	±11.2	23.46	0.05 Sigt.
Mild PET	13 (14.60)	428.4	±13.2	13 (14.6)	218.0	± 9.1	45.46	0.05 Sigt.
Severe PET	10 (11.23)	435.0	±20.6	10 (11.23)	230.0	±15.8	23.68	0.05 Sigt.
Eclampsia	6 (6.74)	446.0	±16.5	6 (6.74)	256.0	±10.2	21.90	0.05 Sigt.

Higher mucoprotein levels were seen in meconium stained samples as compared to clear samples. Even in the clear samples levels were much higher in toxaemias as compared to normal pregnancy.

Discussion

One of the hypothesis for the success of the foetus as a homograft on the mother and the absence of its immune rejection has been given by Apffel and Peters (1969) who suggested the existence of 'symbodies' which in contrast to the antibodies bring about and maintain tolerance to an antigenic stimulus. The nature of these humoral substances was most likely to be glycoproteins. Since seromucoid proteins are known to respond more to immune response than any other fraction of glycoprotein (Winzler 1960), they have been chosen for examination.

In the present series, mean total protein in control cases was 6.6 ± 0.82 gm/dl with a range of 5.2 — 7.9 gm/dl.

At delivery the level in normal pregnancy was 5.4 ± 0.7 gm/dl, in PET it was 5.18 ± 0.6 gm/dl and in eclampsia 4.2 ± 0.8 gm/dl. The reduction of total protein in toxaemias of pregnancy may be due to a decrease in the albumin fraction, as has been estimated by electrophoresis (MacCillivery and Tovey 1957). The mean SMP levels of control cases varied between 37.5 — 67.5 mg/dl (mean 50.31 ± 15.95 mg/dl). This value is comparable to the value reported by Greenspan (1954) of 52.7 mg/dl for adult females.

In the 3rd trimester of pregnancy the mean SMP of control groups was 59.1 ± 1.28 mg/dl. in PET it was 126.5 ± 4.41 mg/dl, in severe PET. It was 125.8 ± 3.82 mg/dl and in eclampsia it was 129.36 ± 6.0 mg/dl. These findings are closely comparable to the published figures of Good *et al* (1973) and Das Gupta (1975). But finding in severe PET differs from the present series, in which a steady rise of SMP is observed with an increasing degree of toxaemia.

Comparing the puerperal SMP levels of normal and toxaemic pregnancies, we found the levels persistently higher in toxaemia.

This rise of SMP in toxaemia can be explained on an immunological basis. An antigenic disparity may promote an active cell mediated immunity with higher immunnoprotective levels of SMP.

Cord SMP, Levels: In normal pregnancy mean cord SMP was 19.8 ± 0.04 mg/dl, in mild PET, it was 22.8 ± 2.26 mg/dl in severe PET 24.9 ± 3.21 mg/dl and in eclampsia it was 32.7 ± 6.12 mg/dl, thereby showing a steady rise with varying degrees of toxaemias as compared to normal pregnancy. These results are in accordance with Das Gupta (1975). Good *et al* (1973) showed that cord blood mucoprotein can not be explained only on the basis of stress during labour as has also been noted in the present study. Possibly the maternal antigen provokes an immune response in the fetal lymphocytic system, with a rise of mucoprotein. The antibodies formed in the foetus, thereby, passing into the maternal circulation may produce a severe antigen antibody reaction with a rise of BP and kidney changes leading to proteinurea and serum protein changes as shown in the present study. All these can be compared to nephrotic syndrome which has an accepted immunological basis. The SMP levels in the mother may be only secondary to stress and dysproteinemia due to a partially selective loss of proteins in the urine.

Amniotic Fluid Mucoproteins

Mean mucroprotein in normal pregnancy was 197.35 ± 27.48 mg/dl, in mild PET it was 28.2 ± 40.8 mg/dl, in severe PET it was 332.5 ± 52.6 mg/dl and in eclampsia it was 351.0 ± 46.7 mg/dl.

A steady rise of mucoprotein level was therefore observed in toxaemia as compared to normal pregnancy and the values were much higher than SMP levels in maternal blood. Sinha and Mukerjee (1973) have reported similar findings.

When a separate study of meconium stained and clear samples of liquor was conducted in all the groups, a statistically significant rise of mucoprotein was observed in the meconium stained samples. The change was also reported by Sinha and Mukerjee (1973).

Evidently this rise of mucoportein levels was due to contamination of liquor with meconium but the mucoprotein in clear samples of liquor from cases of PET and eclampsia was still seen to be much higher than in normal pregnancy.

The reason for this is still a debatable one. Considering the placental insufficiency in toxaemia as a factor causing foetal hypoxia, it is likely that mucoprotein content in clinically clear samples of liquor may be raised to a significant level by occult meconium which is not enough to stain the liquor over and above the mucoprotein obtained from the other likely sources. If the presence of meconium in the liquor is accepted as one of the indicators of foetal hypoxia, the SMP level may be a parameter of its severity.

Conclusions

If the immunological basis of toxaemia is accepted, the level of serum mucoprotein should be raised due to its immunoprotective function in cases of toxaemias as compared to normal pregnancy. This view may be supported by the present study, but since the number of cases studied is not a large one the view is subject to further confirmation.

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