OBSERVATIONS ON THE URIC ACID CONTENT OF AMNIOTIC FLUID, MATERNAL AND CORD SERA IN PRE-ECLAMPTIC TOXAEMIA AND ECLAMPSIA

by

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Uric acid is the chief end-product of purine metabolism. Normally its level in serum is slightly increased by rich diet and pregnancy, (Cantarow and Schepartz, 1967), but markedly in labour when its concentration may be doubled (Kellar, 1963). Increased tissue breakdown, diminished renal clearance and metabolic diseases, like gout cause an accumulation of this substance in the serum.

The presence of uric acid in the amniotic fluid was demonstrated by Uyeno (1919) but he was unable to estimate it. Williams and Baren (1924) reported that the uric acid content of amniotic fluid increased as pregnancy approached term and was higher than that of maternal serum. Shrewsbury (1933) estimated the amniotic fluid uric acid in abnormal cases and found it to vary between 1.4 and 5.7 mg. per cent. Cantarow, Stuckert and Davis (1933) compared the uric acid levels of amniotic fluid, maternal and cord bloods and concluded that it was the highest in the amniotic fluid. Serr, Czacskes and Zuckermann (1963) also observed that the uric acid concentration was the highest in amniotic fluid, somewhat lower in foetal blood and the lowest in maternal blood.

The present study was undertaken to observe the changes in uric acid level in amniotic fluid, maternal and cord sera in pre-eclamptic toxaemia and eclampsia as compared with normal pregnancy.

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Material and Method

Cases of normal pregnancy, mild and moderate pre-eclamptic toxaemia (B.P. upto or below 160/110 mm Hg. with oedema and/or albuminuria), severe pre-eclamptic toxaemia (B.P. above 160/110 mm Hg. with oedema and/or albuminuria) and eclampsia between 38 and 41 weeks' of gestation, either in pregnancy or early in labour were selected for study. Liquor amnii was obtained either by transabdominal amniocentesis or by puncture of the hind bag of water with Drew Smythe Catheter (Sinha, Sen and Mukherjee, 1967). The distribution of the different types of cases is shown in Table I.

Uric acid estimation of the liquor amnii, maternal and cord sera was done by the method of Caraway (Cheyne, 1964). The weight and apgar score of the newborn were recorded.

Results

Tables II, III and IV show the changes in the mean uric acid content of liquor amnii, maternal and cord sera, respectively in different degrees of toxaemia compared with normal pregnancy.

TABLE I

Distribution of Different Types of Cases

Types of cases	Liquor Amnii	Maternal blood	Cord Blood
Normal pregnancy	33	33	33
Mild + Mod. P.E.T.	29	29	28
Severe P.E.T.	26	26	22
Eclampsia	33	33	18

Numbers and types of cases where uric acid estimation was done.

TABLE II

Comparison of the Mean Uric Acid (Mg per 100 ml) of Liquor Amnii Between Normal Pregnancy and Different Degrees of Toxaemia and Between Increasing Degrees of Toxaemia

Group of cases	No. of Cases	Mean	S.D.	S.E.	t	df	P value
Normal Pregnancy	33	5.50	0.70	0.12	2.72	60	Less than 0.01
Mild & Mod. P.E.T.	29	6.10	1.01	0.19			
Normal Pregnancy	33	5.50	0.70	0.12	5.20	57	Less than 0.001
Severe P.E.T.	26	6.80	1.14	0.22			
Normal Pregnancy	33	5.50	0.70	0.12	10.09	64	Less than 0.001
Eclampsia	33	7.90	1.13	0.19			
Mild & Mod. P.E.T.	29	6.10	1.01	0.19	2.41	53	Less than 0.05
Severe P.E.T.	26	6.80	1.14	0.22			
Severe P.E.T.	26	6.80	1.14	0.22	3.79	57	Less than 0.001
Eclampsia	33	7.90	1.13	0.19			

TABLE III

Comparison of the Mean Uric Acid (mg per 100 ml) of Maternal Serum Between Normal Pregnancy and Different Degrees of Toxaemia and Between Increasisg Degrees of Toxaemia

Group of cases	No. of cases	Mean	S.D.	S.E.	t	df	P value
Normal Pregnancy	33	3.20	0.91	0.15	5.65	60	Less than 0.001
Mild & Mod. P.E.T.	29	4.50	0.99	0.18			
Normal Pregnancy	33	3.20	0.91	0.15	7.77	57	Less than 0.001
Severe P.E.T.	26	5.30	1.18	0.23			
Normal Pregnancy	33	3.20	0.91	0.15	17.77	64	Less than 0.001
Eclampsia	33	6.40	0.63	0.11			
Mild & Mod. P.E.T.	29	4.50	0.99	0.18	2.76	53	Less than 0.01
Severe P.E.T.	26	5.30	1.18	0.23			
Severe P.E.T.	26	5.30	1.18	0.23	4.40	57	Less than 0.001
Eclampsia	33	6.40	0.63	0.11			

TABLE IV

Comparison of the Mean Uric Acid Content (mg per 100 ml) of the Cord Serum Between Normal Pregnancy and Different Degrees of Toxaemia and Between Increasing Degrees of Toxaemia

Group of cases	No. of cases	Mean	S.D.	S.E.	t	df	P value
Normal Pregnancy	33	3.60	0.69	0.12	2.30	59	Less than 0.05
Mild & Mod. P.E.T.	28	4.70	2.36	0.45			
Normal Pregnancy	33	3.60	0.69	0.12	6.15	53	Less than 0.001
Severe P.E.T.	22	5.20	1.13	0.24			
Normal Pregnancy	33	3.60	0.69	0.12	11.25	49	Less than 0.001
Eclampsia	18	6.30	0.91	0.21			
Mild & Mod. P.E.T.	28	4.70	2.36	0.45	1.92	48	Not significant.
Severe P.E.T.	22	5.20	1.13	0.24			
Severe P.E.T.	22	5.20	1.13	0.24	3.54	38	About 0.001
Eclampsia	18	6.30	0.91	0.21			

Comparision between Tables II and III showed slightly higher uric acid level in cord sera than in maternal sera in normal pregnancy and mild and moderate pre-eclamptic toxaemia, but the maternal serum level was higher than cord serum in severe toxaemia and eclampsia. When the amniotic fluid uric acid level exceeded 8 mg per 100 ml., 12 foetuses died in utero in 33 eclampsia cases. However, a correlation between amniotic fluid uric acid level and the birth weight or apgar score of the newborn could not be established.

Discussion

Higher level of uric acid in amniotic fluid compared with maternal or cord sera with progressive increase towards term strongly suggests foetal urine as an important source of uric acid in the liquor amnii. Mean cord serum uric acid in normal pregnancy and mild and moderate pre-eclamptic toxaemia was higher than that in the maternal serum. Cantarow, Struckert and Davis (1933) and Serr, Czaczkes and Zuckermann (1933) also reported similar results in cases of normal pregnancy. Increased nucleic acid breakdown in the foetus during the process of replacement of foetal red blood cells by the adult substitute is an important cause of nucleic acid degradation in the foetal system. The excretion of uric acid through the foetal kidneys is manifested by high uric acid level in amniotic fluid.

Rise of uric acid content of amniotic fluid, maternal and cord sera in different degrees of toxaemia compared with normal pregnancy is evident from Tables II, III and IV, respectively. Rise of maternal blood/serum uric acid (Stander and Cadden, 1934; Lancet and Fisner, 1956; Prabhavati, 1957; Purandare and Agashe, 1959; McFarlane, 1963; Kishore and Tandon, 1965) and cord blood uric acid (Roy Choudhury and Chakravarty, 1964) in toxaemia of pregnancy have also been reported. In severe pre-eclamptic toxaemia

and eclampsia, the degree of rise in maternal serum uric acid level was higher than that in cord serum. The rise of maternal serum uric acid in toxaemia of pregnancy was found to be due to either increased tubular reabsorption of uric acid (Chesley and William, 1945; Seitchik, 1953) or decreased glomerular filtration of this substance (Hayashi, 1956).

Rise in the mean cord serum uric acid in toxaemia may be due to the reduced excretion of this substance through the placenta due to the diminution of blood supply through choriodecidual space (Browne and Veall, 1953), resulting in reduced clearance of uric acid from the foetus across the placenta. The rise of amniotic fluid uric acid level may be caused by a compensatory increase of uric acid excretion through foetal urine. Bruno Wolf (Quoted by Taussig, 1927) produced experimental evidence of such compensatory overfunctioning of foetal kidneys and concluded that where the urine forming substances are withheld in maternal blood, the foetal organs take an added function to eliminate them.

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