A COMPARATIVE STUDY OF AMNIOTIC FLUID UREA AND BLOOD UREA IN NORMAL PREGNANCY AND PRE-ECLAMPTIC TOXAEMIA

by

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Introduction

Since the foetus lives surrounded by amniotic fluid, the idea has grown that estimation of this milieu ēxterieur might give information about its metabolism, a similar philosophy to the estimation of expired air or of urine in the adult extra-uterine human. Amniotic fluid in late pregnancy, is largely made by the foetus and contains many products of his metabolism.

The volume of amniotic fluid is used by clinicians as an estimate of the foetal state.

Abnormal constituent of the liquor amni will probably be of increasing diagnostic as well as of prognostic importance. Foetal urine reflects foetal renal functions. Estimation of the osmolarity is a guide to the electrolytes present in the amniotic fluid. By cytological study of the amniotic fluid, sex and certain chromosomal abnormalities can be determined in early weeks of pregnancy. Estimation of bilirubin by spectrophotometry, one can predict the severity of the haemolytic disease.

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Material and Methods

The clinical material consists of two groups of pregnant subjects.

Group 1: Normal pregnant female—This group includes 65 cases and they were further divided into four according to the period of gestation.

(a) First Group—16-22 weeks of gestation.
(b) Second group—22-32 weeks of gestation.
(c) Third group—32-40 weeks.
(d) Fourth group—Includes cases who were in early labour with bag of water intact.

Group 2: Group includes cases in which pregnancy was complicated by toxaemia. In this group, 51 cases were studied. In all, period of gestation ranged from 30 weeks to 40 weeks of pregnancy. The diagnosis of toxaemia was based on presence of any two of the three, viz., raised blood pressure, edema over feet, albuminuria.

According to severity of toxaemia cases were classified under 4 groups: mild, moderate, and severe toxaemia, and eclampsia.

Blood was collected from antecubital vein and amniotic fluid was collected either by trans abdominal amniocentesis, or by vaginal route in those cases where labour was being induced by artificial rupture of membrane or cases who were already in labour.

Results and Discussion

Results of the present study are shown in Tables I and II.

Table I depicts that inspite of the greatly increased demands for protein involved in the mother for foetus, the body is able to maintain the lower limits of normal blood urea. The mean concentration of blood urea ranged from 21.43 to 22.06 mg%.

Amniotic fluid urea levels show a small rise over maternal serum urea levels in early weeks of pregnancy, but rises considerably in the later weeks of pregnancy

TABLE I

Showing the blood urea and amniotic fluid urea obtained in different periods of gestation

	16-22 weeks	22-32 weeks	32-40 weeks	In labour	Total
No. of cases	16	20	11	18	65
Blood urea (mg%)					
Range	18.6-24.6	19.4-24.0	20.2-24.4	19.30-26.20	18.6-26.20
Mean	22.06	21.435	21.8181	21.6944	21.7246
S.D.	1.78	1.2516	1.2456	2.0057	1.6044
p	<.001	<.001	<.001	<.001	<.001
Amniotic fluid urea (mg%)					
Range	24.6-38.8	30.0-44.40	32.60-39.60	30.40-44.20	24.6-44.40
Mean	31.03	35.725	36.309	37.3722	35.1076
S.D.	3.77	3.3944	2.6015	3.5858	4.1083
p	<.001	<.001	<.001	<.001	<.001

TABLE II

Showing the Blood Urea and Amniotic Fluid Urea Obtained in Toxaemic and Eclamptic patient

	Total	Mild toxaemia	Moderate Toxaemia	Severe Toxaemia	Exclampsia
No. of cases	51	23	14	10	4
Blood urea (mg%)					
Range	20.0-38.4	20.0-30.0	22.8-30.4	32.4-38.4	32.0-36.8
Mean	21.7901	24.1478	26.6857	34.91	34.80
S.D.	4.9808	2.3651	2.2241	2.0496	2.1725
p	<.001	<.001	<.001	<.001	<.001
Amniotic fluid urea (mg%)					
Range	28.40-59.7	28.40-46.0	32.2-48.4	41.0-48.6	41.3-59.7
Mean	40.5784	36.1913	41.50	45.53	50.20
S.D.	5.8995	3.2861	4.3115	2.2900	7.7127
p	<.001	<.001	<.001	<.001	<.001

from a mean level of 31.03 mg% at 16 weeks to 37.37 mg% at term. Correlating the blood urea level vs. amniotic fluid urea level, it was found to be negatively correlated in early pregnancy and in labour patient while positively in 22 to 40 weeks of pregnancy (Table III).

TABLE III

Showing the correlations between blood urea and amniotic fluid urea

	r
16-22 weeks	— 0.0491
22-32 weeks	+ 0.4299
32-40 weeks	+ 0.4104
In labour	— 0.2184
Mild Toxaemia	+ 0.1072
Moderate Toxaemia	+ 0.2673
Severe Toxaemia	— 0.2855
Eclampsia	— 0.6346

The above observations are in accordance with those of Guthmann and May (1930), Friedberg (1968), Sozanskii (1961), but in disagreement with Purandare and Agashe (1959), Riedel *et al* (1963), Kishore and Tandon (1965).

Purandare *et al* (1959) and Riedel (1963) showed fall in blood urea levels in normal pregnancy as compared to the non-pregnant blood urea levels. Kishore and Tandon (1965) too did not report any significant alteration in blood urea concentration with the advancing period of gestation, but found low levels of blood urea in normal pregnancy as compared to normal levels in non-pregnant state. No satisfactory explanation for this low blood urea has been given.

Different explanations have been given by various workers about the presence of higher concentration of amniotic fluid urea and its significant correlation with the advancing period of gestation in normal pregnancy.

Danforin and Hull (1958) suggested that higher concentration of urea in amniotic fluid is due to its active transport from the maternal compartment into the amniotic fluid across the chorioamnion. Chez *et al* (1964), Pitkkin *et al* (1968) and Biggs and Dancan (1970) have explained that amniotic fluid is a product of fetal kidneys that is discharged into the amniotic cavity, its quantity regulated by fetal deglutition.

Guthmann and May (1930), Makepeace *et al* (1931) suggested that it could be due to osmolarity deficit with the advancing pregnancy.

Observing the blood urea and amniotic fluid urea in abnormal pregnancy, it is apparent that there is definite rise in levels of blood urea and amniotic fluid urea with the increasing severity of toxaemia when compared with normal pregnancy (Tables II, III and IV).

TABLE IV

Showing Correlation Between Blood Urea and Amniotic Fluid Urea of Normal Pregnancy (16 weeks—labour) Vs abnormal pregnancy (Toxaemia and Eclampsia)

Amniotic fluid urea		Blood urea		
p	t	p	t	
>.05	1.1424	<.05	5.4773	Normal Vs Mild toxaemia
<.05	5.2413	<.05	9.7813	Normal Vs Moderate toxaemia
<.05	7.8070	<.05	23.3328	Normal Vs Severe toxaemia
<.05	6.7666	<.05	15.5437	Normal Vs Eclampsia

Although there is rise in blood urea levels, but it is more remarkable in am-

niotic fluid urea levels in toxaemia.

So it can be concluded that high urea concentration in amniotic fluid has definite correlation with the degree of toxaemia.

TABLE V

Showing correlations between blood urea Vs. Amniotic fluid urea in normal and abnormal pregnancy

		Statistical correlation between the correlation of normal and abnormal pregnancy
Normal pregnancy (65)	—0.0212	+ 4.0104
Abnormal pregnancy (51)	+0.6562	$p < .01$

Kishore and Tandon (1965) also reported elevated values even in mild degree of toxaemia.

Saxena and Kharoliwal (1971) also were in agreement with elevated levels of amniotic fluid urea levels in toxaemia e.g. 42.35 mg% as compared to 33 mg% in normal pregnancy, but they reported that there was no significant difference in blood urea concentration in normal and toxaemic group (disagree with present study).

Rise in blood urea in toxaemia might be due to one of the following factors (Gillibrand *et al*, 1969), changes in the fluid balance, increased urea production or impared excretion.

Sinha and Mukherji (1973) suggested that the rise in amniotic fluid area concentration might be due to diminished urea clearance by the fetus through the placenta due to reduced circulation in the choriodecidual space in toxaemia of pregnancy and increased excretion of urea through the foetal urine which under normal condition would have been excreted through placenta. Sozanskii (1961) stated that rise of urea contents of amniotic fluid might be due to addition of large amount of urine in to liquor caused by disturbed intra uterine reflex retention of urination as fetus is in a state of asphyxia in toxaemia pregnancy.

The present study concludes that there is no appreciable variation in blood urea concentration during the course of normal pregnancy. On the other hand, higher values have been found in liquor urea values with the advancing pregnancy and maximum fluid urea concentration is found at full term. Both the blood and amniotic fluid urea levels showed a steady rise with the increase in the degree of toxaemia.

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