AMNIOTIC FLUID SERUM ALKALINE PHOSPHATASE (HEAT STABLE) LEVEL IN NORMAL AND TOXAEMIAS OF PREGNANCY

by

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That the foetus can be in jeopardy in intrauterine life long before the onset of labour is well known due to large number of stillbirths in which the only abnormal autopsy findings are those of intrauterine asphyxia. Therefore, a dependable and reliable test of placental function is one of the most urgent needs in obstetrics. Various placental enzymes as an index of placental function are being used for this purpose.

Curzen and Morris (1965) showed that the heat stable alkaline phosphatase in the serum rises slowly in 3rd trimester of normal pregnancy and higher level was seen in pregnancy induced hypertension. With liberal use of amniotic fluid analysis various authors claimed that the HSAP level in amniotic fluid was a more reliable index of foeto-placental unit than the serum HSAP level. A statistically significant correlation seems to exist between maternal and amniotic fluid HSAP.

The present investigations were undertaken to study the Heat Stable Alkaline Phosphatase (HSAP) in amniotic fluid as compared to maternal and foetal blood in normal and abnormal pregnancies.

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Material and Methods

The present study consisted of 238 cases of pregnant women after 34 weeks of gestation. All the cases were selected from the antenatal wards or labour room of Patna Medical College Hospital during the period from July, 1977 to October, 1979. The cases were divided into the following groups:

(A) Normal pregnant women (between 34 to 40 weeks of gestation).

(B) Pregnant women with pre-eclampsia having previous essential hypertension (B.P. 140/90 mm. Hg).

(C) Pregnant women with severe pre-eclampsia (B.P. more than 160/100 mm. Hg) associated with oedema with or without albuminuria developing in second half of pregnancy.

(D) Cases of I.U.G.R. where measured fundal height was less by 3 weeks of actual fundal height calculated from the L.M.P.

In every case one sample each of maternal blood, cord blood and amniotic fluid were obtained at the time of delivery, and weight of the placenta was noted.

Biochemical estimations were carried out within 4 to 6 hours of collection of

samples so that the enzyme deterioration was minimum.

Observations

maternal serum and cord blood serum did not show any significant change, whereas amniotic fluid HSAP level showed statistically significant rise (p value less than .05).

TABLE I
Distribution of Cases in Different Age Groups

Group of cases	Total No. of cases	Age groups in years with No. of cases			
		16-19	20-24	25-30	31-35
Normal pregnancy	128	18	70	34	6
Post-maturity	20	—	10	8	2
Pre-eclamptic toxaemia with essential hypertension	30	8	16	6	—
Pre-eclamptic toxaemia (severe)	30	4	10	12	4
Intra-uterine growth retardation	30	4	10	12	4

Table I shows that the cases have been distributed in age groups to establish that the age has no effect on the Heat Stable Alkaline Phosphatase.

The statistical analysis of Table II revealed that the mean HSAP level in

Table III shows statistically significant rise in the level of HSAP in both the maternal serum and amniotic fluid and p value is .05 and .04 respectively. However, cord blood serum value of HSAP

TABLE II
Comparison of Mean Values of HSAP Between Mild PET Cases and Normal Pregnancy Between 36-38 Weeks of Gestation

	Normal	PET	Difference	p value	t value
Maternal serum	11.21	11.80	0.59	.06	3.19
Amniotic fluid	4.53	5.86	1.33	.005	3.24
Cord serum	3.33	3.84	0.51	.06	2.11

TABLE III
Comparison of Mean Values of HSAP in Severe PET Cases With Normal Pregnancy in 36-38 Weeks of Gestation

	Normal	PET	Difference	t value	p value
Maternal serum	11.21	13.1	1.89	2.3168	.05
Amniotic fluid	4.53	6.7	2.17	2.5112	.04
Cord blood	3.33	3.2	0.10	1.1296	.1

did not show any significant rise.

From statistical analysis of Table IV it could be well appreciated that the level of HSAP in amniotic fluid and cord blood serum showed a significant fall, but more marked in amniotic fluid, whereas maternal serum did not show any significant fall (p value .06).

(Table V). In cases of post-maturity significant rise in level of HSAP was observed in amniotic fluid. Therefore, it can be inferred that the level of HSAP in amniotic fluid serves as a better reliable index of placental function.

The exact cause for the rise of HSAP in maternal serum and amniotic fluid is still

TABLE IV

Comparison Between HSAP Level in Maternal Serum, Amniotic Fluid and Cord Blood Serum in Normal Pregnancy and I.U.G.R.

	Normal	I.U.G.R.	Difference	t value	p value
Maternal serum	13.05	12.9	0.15	1.7116	.06
Amniotic fluid	5.2	3.9	1.3	2.2134	.03
Cord serum	3.5	3.0	0.5	1.9842	.05

TABLE V

Comparison of HSAP Level Between Normal Pregnancy and Post-maturity

	Normal	Post-maturity	Difference	p value	t value
Maternal serum	14.4	14.28	0.12	.06	2.3461
Amniotic fluid	5.4	5.8	0.4	.02	3.0112
Cord serum	3.9	3.86	0.04	.08	1.2459

Statistically it is clear that there is significant rise in the level of HSAP in amniotic fluid only (p value being less than .02). No significant change is appreciated in maternal serum and cord blood (p value being .06 and .08 respectively).

Discussion

The heat stable alkaline phosphatase of amniotic fluid resembled the maternal serum qualitatively in thermal stability. Raised level of HSAP was observed in severe pre-eclampsia in serum but not in mild pre-eclampsia. But when measured in amniotic fluid significantly raised level was observed in mild pre-eclampsia also. Similarly, in cases of I.U.G.R. significant fall was observed in amniotic fluid

not known, but many hypothetical views have been put forward by many workers. The source of raised HSAP in amniotic fluid appears to be from the placenta rather than the maternal serum, since significantly higher values for HSAP were seen in placenta, but not in maternal serum in mild and severe pre-eclampsia cases. Curzen and Morris (1966) drew attention towards the elevated level cause as hyper-placentosis. Increased tendency of alkaline phosphatase enzyme in amniotic fluid indicates an indirect reflex of enhanced cellular metabolism according to Roy Chowdhary *et al* (1971). It has been proved that amniotic fluid containing fair number of cells, like other cellular granules, the major functional organelles, the alkaline phosphatase

activity is limited to these amniotic cells. Thus, the rise of HSAP level in amniotic fluid represents the cellular reaction stage of enhanced amniotic cellular metabolism under the stress of PET. On the other hand, raised level in maternal serum indicates a pathological state called by Curzen and Morris (1968) as hyper-placentasis, under the stress of severe PET. Therefore, in mild PET cases which is not capable of producing the state of hyper-placentosis which is usually seen in cases of severe PET, it is only in the amniotic fluid that the HSAP shows a statistically significant rise, signifying a near cellular reactive state. Therefore, we can conclude that amniotic fluid HSAP level is the reliable indicator of placental function in mild PET cases which shows early changes in the level of HSAP.

Similarly, in I.U.G.R. cases decrease of enzyme level is significant in amniotic fluid might be due to atrophy or decrease in syncytio-trophoblastic cells, as a result of pathological insufficiency or early placental ageing, a constant associate of majority of intra-uterine growth retardation.

In cases of post-maturity a significant rise of HSAP level was found in amniotic fluid. After 34 weeks of gestation until term, the HSAP level decreases in placenta in total quantity and in per gram of placental tissue. This trend of rising level continue after full term, hypothetical conclusion drawn from this is that enzyme leaks out of post-mature placenta while intra-cellular concentration falls as observed by Hawes and Halliday (1970). Roy Chowdhary *et al* (1971) found significantly increased enzymatic activity in amniotic fluid in cases of post-maturity.

Summary

HSAP levels were estimated after 34 weeks of gestation in normal pregnancy, mild and severe PET cases, I.U.G.R. and Post-maturity cases in K.A. unit/100 ml in serum, amniotic fluid and cord blood serum.

(1) There is gradual increase in the level of HSAP in serum and amniotic fluid with increasing weeks of gestation.

(2) Significant increase in level of HSAP is found only in amniotic fluid in mild PET cases, not in serum.

(3) Significant decrease in level of HSAP is found in only amniotic fluid, not in maternal serum in I.U.G.R. cases.

(4) In post-maturity cases significant rise in level of HSAP is found in amniotic fluid.

Therefore, HSAP level in amniotic fluid serves as a reliable diagnostic parameter of PET, IUGR cases and post-maturity i.e. early diagnosis of placental insufficiency.

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