

# ALPHA FOETO PROTEIN (A.F.P.) IN HIGH RISK PREGNANCIES

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## SUMMARY

In the present study, 50 high-risk pregnancies during the period of April 80 to March 81 in Safdarjang Hospital, New Delhi were included. Levels of A.F.P. (Alfa Foeto Protein) were measured in sequential cord blood and maternal sera by counter immuno electrophoresis (C.I.E.P.). Quantitative assay was done by Radial Immuno diffusion technique in cord sera. While in cases of maternal sera, C.I.E.P. positive cases (with a sensitivity of 250 nanogm/ml) were further quantitatively analysed by Radio-immuno assay.

In pre-eclamptic toxæmia, pregnancies associated with congenital defects and hydramnios, cord blood A.F.P. levels were comparable to those found in normal pregnancies. While in pregnancies complicated by diabetes mellitus, Rh isoimmunisation or multiple pregnancies, A.F.P. cord blood levels were higher than normal. High maternal A.F.P. level is associated with foetal damage in the form of either intrauterine or neo-natal death.

## Introduction

Alpha foeto protein (A.F.P.) or foetoglobulin have no corresponding compound in the adult. This is used in pregnancy as a means of monitoring foetal development. It has been used in the antenatal diagnosis in different trimesters of pregnancy. The indications for A.F.P. assay vary in all three trimesters of preg-

nancy in contrast to most other biochemical foetal monitors which are useful in only one period of gestation. A.F.P. concentration varies from mgm per ml. in foetal serum to micrograms per ml. in amniotic fluid to nanograms per ml in maternal serum. Least concentration being in maternal serum.

Circulating foetal A.F.P. is affected by certain pregnancy disorders. Levels tend to be higher than normal in infants born to Rh immunised or diabetic mothers and lower than normal in infants born to toxæmic mothers. These pregnancy dis-

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sera were positive by C.I.E.P. There is steady decline in cord blood levels in both males and females throughout last trimester of pregnancy in P.E.T. At term, males had greater A.F.P. levels than females.

Sequential cord and maternal blood were collected in 15 Rh negative mothers, but only 2 showed evidence of isoimmunisation. One of these 2 cases, had detectable A.F.P. levels by C.I.E.P. which was also associated with intrauterine foetal death. Out of 2 cases of diabetes mellitus, 1 had high maternal A.F.P. levels and A.F.P. was detected in both cord sera by C.I.E.P. This child had foetal distress during parturition and died during neonatal period. There were 8 cases of multiple pregnancies, 7 pairs of twins and 1 triplet delivery.

There was definite correlation between maternal and cord blood A.F.P. levels. The highest maternal level, 620 nano gm/ml. was associated with abruptio placenta seen in association with twin pregnancy. Both twins died two days after delivery. In cases where maternal A.F.P. levels were below 250 nanogm/ml. neonatal mortality was 16.6%. While in cases where maternal A.F.P. levels were high, neonatal mortality was 36.5%. There were 10 cases with congenital malformations in the newborn, out of which 5 had detectable maternal A.F.P. (50%). All were associated with neural tube defects.

In all there were 11 intrauterine foetal deaths (all groups combined) but maternal A.F.P. could be detected in only 5 (45.5% by C.I.E.P.). These included 2 cases of severe P.E.T., one of severe Rh isoimmunisation and 8 associated with neural tube defect.

#### Discussion

The present pilot study of A.F.P. levels in sequential cord blood and mater-

nal sera was done on limited number of patients. Although the number was small, yet significant variation in maternal and cord sera in each high risk, pathological pregnancy was noted.

The largest group in this study of 50 high-risk pregnancy was of 25 patients with pre-eclamptic toxemia. Out of 25, there were 14 preterm deliveries before 37 weeks of gestation and 11 term deliveries. The mean cord blood level of A.F.P. in these cases was 11.36 mgm/100 ml.

Out of 25 maternal sera, 1 patient had A.F.P. 620 nanogm/ml. This primigravida with PET had a macerated still birth of a female baby at 40 weeks. This unexplained rise in A.F.P. could only be as a result of admixture of maternal and foetal blood during parturition. Coheen *et al* (1973) detected A.F.P. in maternal sera by C.I.E.P. in only 3 out of 83 cases and they also gave a similar reason for the unexplained rise. In the remaining 23 cases (92%) no precipitation line was observed by CIEP.

Seppala (1975) has reported A.F.P. levels above the normal median in maternal sera in only 35% of cases. Khoo *et al* (1978) compared maternal serum A.F.P. levels in normal patients and patients with PET and found significantly lower mean A.F.P. and majority of the individual values were lower than mean for normal pregnancy. Rodock *et al* (1976) indicate the maternal A.F.P. levels less than 5th percentile in 16% of cases as compared to 5% in normal pregnancy. In our study 92% of the maternal sera in third trimester in cases of PET had A.F.P. levels less than 250 nanogm/ml. Toxaemia of pregnancy is characterised by decreased uteroplacental circulation and this explains the decreased transfer of A.F.P. from foetus to mother. Khoo *et al* (1978) have suggested that in PET,

the disease itself may affect the foetus in such a way that it produces less amounts of A.F.P. Low A.F.P. in cord sera may be due to accelerated maturation of foetal liver and hence decreased production (Seppala *et al* 1972). In our study, 52% of the cord sera had A.F.P. below the median.

Seppala (1975) found A.F.P. levels greater than median in 65% of maternal sera and 62% of cord sera in patients with diabetes mellitus. In our study cord blood A.F.P. levels were high in both the cases of diabetes mellitus while only 1 maternal serum had higher levels of A.F.P. (More than 250 nanogram/ml.).

From the results obtained in our study, it is concluded that a pronounced increase in A.F.P. levels may take place in severe Rh immunisation associated with intrauterine foetal death.

Vivian and Ward (1974) did not predict intrauterine death in Rh disease by maternal sera A.F.P. Levels.

Coheen *et al* (1973) predicted increase in A.F.P. in maternal sera before intrauterine death. Bock *et al* (1976) reported high maternal A.F.P. levels in 3 out of 12 cases and in all eventually foetal death occurred.

Ishiguro (1973) in cases of multiple pregnancy found A.F.P. levels twice as high as the normal levels. Garof and Seppala (1975) found A.F.P. levels greater than in normal single pregnancies. There was no correlation be-

tween the birth weight and maternal AFP levels in twins.

A large number of congenital anomalies have been found to be associated with high maternal A.F.P. levels. In our study of 10 newborns with congenital defects, A.F.P. was more than 250 hanaogm/ml. in 5 cases (50%).

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