

## SERUM CYSTINE AMINOPETIDASE LEVELS AND IUGR

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### SUMMARY

Serum cystine aminopeptidase (CAP) level was determined in 172 samples from 65 normal pregnant women as control group and 96 samples from 20 patients of clinically diagnosed IUGR between 28 to 41 weeks of gestation. Sixteen patients out of 20 (80%) clinically diagnosed IUGR showed significantly low levels of serum CAP as compared to control group.

### INTRODUCTION

The trophoblast, besides oestrogen and progesterone also produces oxytocinase, an enzyme specific to pregnancy. This is cystine aminopeptidase (CAP). Ryden (1972) has shown that serum oxytocinase activity is mainly derived from placental tissue and James (1966) has demonstrated by histochemical means that this activity originates from syncytiotrophoblast. In a study involving immunological inactivation of serum oxytocinase activity, Watkins and Small (1972) also concluded that serum oxytocinase originates from the placenta.

Several workers like Ryden (1972), Malkani et al. (1971) and Shahani and Merchant (1979) have shown that a gradual increase in

serum oxytocinase occurs with advancing gestation, the highest level being found at term.

The present study was carried out to assess the usefulness of serum CAP estimation for the diagnosis for timely intervention could be taken.

### MATERIALS AND METHODS

The patients for our study were selected from the O.P.D. and indoor wards of U.I.S.E. Maternity Hospital. 65 normal healthy pregnant women (172 serum samples collected from them) were controls and 20 patients with IUGR (96 samples obtained from them) were taken as subjects for study.

The patients with poor maternal weight gain, who were sure of the date of their last menstrual period and in whom the fundal height was less than the period of amenorrhoea by at least 4 weeks were included in this group. These patients had regular menstrual cycles prior

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to conception. 3 patients gave history of chronic smoking. Serial serum samples were taken from them from 28 to 41 weeks of gestation. Out of 20 patients 18 patients (90%) were having mild to moderate IUGR and 2 patients (10%) had severe IUGR (in severe cases FH was 6 cm less than the normal).

Serum CAP was measured spectrophotometrically by the method of Christensen and Hagelid (1975). The enzyme activity was calculated in IU/L serum.

### OBSERVATIONS

Table I shows CAP levels in normal and IUGR patients according to period of gestation. Since all the patients did not turn up for weekly follow up, the number of samples in each week is different and mean values have been adopted for various weeks of gestation.

### DISCUSSION

Serial CAP estimations done in 65 normal pregnant women from 28 to 41 weeks of gesta-

tion showed a progressive rise in enzyme activity with advancing period of gestation (being  $5.6 \pm 1.7$  IU/L at 28 weeks to  $18.0 \pm 3.9$  IU/L at term). Akin to our studies Riad (1962), Josephides and Turkington (1967), Malkani et al. (1971) Hurry et al. (1972) and Shahani and Merchant (1979) have also observed a progressive rise in CAP levels with advancing gestation.

In the present study of 20 cases of IUGR significantly low levels of serum CAP were seen. Out of 16 patients with values below normal limits 14 patients had live births. In all the 14 patients of this group a slowly rising curve was seen though the values were below the normal range. All these patients were having mild to moderate IUGR. In 2 patients full term still birth occurred. Both of them showed continuously decreasing values. Hence it is evident that serially increasing CAP activity, though below the normal range, is associated with lesser degree of placental insufficiency as compared to static or falling levels which indicate poor placenta function and fetal jeopardy. The placenta in all these cases with low CAP levels was found either

TABLE I

CAP levels in normal and IUGR patients according to period of gestation

	28 to 29		30 to 31		32 to 33		34 to 35		36 to 37		38 to 39		40 to 41	
	N	IUGR	N	IUGR	N	IUGR	N	IUGR	N	IUGR	N	IUGR	N	IUGR
No. of Samples	20	16	22	10	27	18	24	15	25	12	28	14	26	11
Mean	5.6	4.2	8.2	6.8	10.0	6.2	12.6	9.7	14.8	11.6	16.2	12.8	18.0	13.0
S.D.	1.7	1.7	2.0	1.4	1.8	2.0	1.9	1.8	2.4	1.6	3.0	2.4	3.9	2.4
SED	0.5701		0.701		0.572		0.8254		0.7769		0.9227		2.1	
t	2.455*		2.279*		3.143**		4.636***		4.118***		3.684***		2.349*	
D.F.	34		30		43		37		35		49		36	

N = Normal

INUR = Intrauterine growth retardation

\* = Significant at  $p < 0.05$

\*\* = Significant at  $P < 0.01$

\*\*\* = Highly Significant at  $p < 0.001$

**TABLE II**  
Outcome of pregnancies in IUGR

Outcome of Pregnancy	Total	Percentage
Fullterm normal delivery	14	70.0
Premature live birth	4	20.0
Full term still birth	2	10.0

**TABLE III**  
Outcome of pregnancy in relation to CAP levels in IUGR

Outcome of Pregnancy	Serum CAP levels in IU/L	
	Within Normal Limits	Below Normal Limits
Full term delivery	2	12
Premature live birth	2	2
Full term still birth	—	2
<b>Total</b>	<b>4</b> (20.0%)	<b>16</b> (80.0%)

infarcted or very small.

Hurry et al. (1972) measuring leucine aminopeptidase and cystine aminopeptidase respectively indicated that serial estimation of this group of enzymes was helpful in the diagnosis of intrauterine fetal growth retardation. Ryden (1972) found that in patients with placental insufficiency the CAP values were consistently low.

Petrucchio et al. (1973) found that patients who delivered growth retarded infants, whether hypertensive or not, failed to show the progressive increase in serum oxytocinase. They also reported that serum oxytocinase is found to be superior to urinary oestriol assays for the prediction of intrauterine fetal growth retardation.

Shahani and Merchant (1979) showed a significant correlation between the placental weight, its degree of infarction and serum oxytocinase levels. Low levels were associated with high perinatal mortality.

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