## SERUM COPPER IN NORMAL AND ABNORMAL PREGNANCY

by

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# Introduction

Diagnosis of failing placental function has been the concern of obstetricians for long. Determinations of some hormones and enzymes have proved useful but are expensive and time consumming. Serum ceruloplasmin has been found to be particularly useful in this respect. However, is has been suggested that serum copper can provide the same information and its determination is comparatively simpler. Therefore, we evaluated the utility of serum copper in monitoring normal and abnormal pregnancies.

## Material and Methods

The study was conducted on 126 subjects taken from the OPD, ante-natal clinic and wards of Zenana Hospital, Udaipur and 15 healthy women of comparable age. The subjects included 60 healthy pregnant women at different periods of gestation, 25 women with abor-

tion, 5 women with intrauterine death, 10 women with premature labour, 5 women with postmature pregnancy, 8 women with pre-eclamptic toxaemia (PET), 10 women with eclampsia and 3 women with hydatiform mole.

Detailed clinical examination and routine investigations were carried out in all cases. Women suffering from diseases known to affect serum copper were excluded.

A venous blood sample was collected from each subject and was analysed for serum copper (Varley, 1975). The data were analysed statistically by Student's t-test.

#### Results

Serum copper in normal non-pregnant women was  $122.0 \pm 15.0 \ \mu g/100 \ \text{ml}$ . There was a progressive and significant rise in serum copper in normal pregnant women from 5th week of gestation onwards (Tables I and II). Serum copper was significantly lower in women with abortion, missed abortion and intrauterine death as compared to normal pregnancy of same duration. In women with threatened abortion in whom pregnancy continued, serum copper was lowered only at sixteen weeks of gestation but not at 8, 12 and 20 weeks (Table III).

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TABLE I

Serum Coppper in Non-pregnant Women and in Pregnant Women in Different

Trimesters (All values are mean ± S.D.)

Group	Number	Serum copper (µg/100 ml.)	P value (Vs Non-pregnant)
Non-pregnant	15	122.0 ± 15.0	alam E a No
Pregnant			
(I trimester)	20	$153.0 \pm 22.7$	< 0.001
Pregnant	the court capital parties and		upolilinkt 2 see - 14
(II trimester)	20	$251.0 \pm 16.2*$	<0.001
Pregnant		and an institute to	
(II trimester)	20	271.0 ± 32.4**	< 0.001

- \* Difference from I trimester significant (P <0.001)
- \*\* Difference from II trimester significant (P <0.001)

TABLE II

Serum Copper at Different Periods of Gestation (All values are mean ± S.D.)

Weeks of gestation	Number	Serum copper (µg/100 ml)	P value (Vs preceding group)
upto 4	4	110,5 ± 11.8	(60/18)
5 - 8	8	153 0 ± 8.4	< 0.001
9 - 12	8	$172.8 \pm 7.0$	< 0.001
13 - 16	. 6	214.7 ± 8.8	< 0.01
17 - 20	6	$231.7 \pm 4.8$	< 0.01
21 - 24	8	$246.3 \pm 10.5$	< 0.01
25 - 28	6	$250.0 \pm 7.2$	>0.05
29 - 32	6 .	$260.0 \pm 3.2$	< 0.02
33 - 36	8	$296.3 \pm 32.9$	< 0.01

TABLE III

Serum Copper in Abortion and Intrauterine Death (All values are mean ± S.D.)

Group	. Weeks of gesta-		Serum copper (µg/100 ml)	P value*
	tion	No.	(1-8)	
Threatened abortion; pregnancy continued	8	. 3	142.0 ± 8.3	>0.05
Pregnancy aborted	8	2	$137.0 \pm 5.3$	< 0.01
Threatened abortion; pregnancy continued	12	5	155.0 ± 18.1	>0.05
Threatened abortion; pregnancy continued	16	4	198.8 ± 3.4	< 0.01
Pregnancy aborted	16	1	180.0	
Threatened abortion; pregnancy continued	. 20	4	223.8 ± 50.1	>0.05
Pregnancy aborted	20	1	180.0	
Missed abortion	12	2	$130.0 \pm 10.7$	< 0.001
Missed abortion	16	2	$202.0 \pm 3.6$	>0.05
Missed abortion	20	1	166.0	
Intrauterine death	32	5	234.0 ± 8.6	<0.001

<sup>\*</sup> As compared to normal pregnancy of same duration.

There was no significant change in serum copper when premature labour occurred at 24 and 28 weeks of gestation but there was a significant decrease in serum copper when premature labour occurred at 32 weeks of gestation. Postmaturity up to 42 weeks did not produce any significant change but at 43 weeks, there was a significant decrease in serum copper. Serum copper was decreased in PET and eclampsia but the decrease was significant only in PET. Serum copper increased in hydatiform mole but not to significant levels (Table IV).

ing that serum copper might be used as a test for pregnancy.

Serum copper was decreased in abortion, missed abortion and intrauterine death. This agrees with the observations of Friedman *et al* (1969).

However, variable results were obtained in threatened abortion when pregnancy continued upto term. Heijkenskjold and Headenstedt (1962), have reported 'that serum copper is low in threatened abortion with poor prognosis. Our data do not support this contention.

In premature labour and postmature

TABLE IV

Serum Copper in Premature Labour, Postmaturity, Toxaemia of Pregnancy and Hydatiform Mole (All values are mean ± S.D.)

Group	Weeks of gestation No.		Serum copper (µg/100 ml)	P value*
Premature labour	24	3	253.3 ± 9.4	>0.05
Premature labour	28	3	240.0 ± 8.3	>0.05
Premature labour	32	4	$241.5 \pm 12.7$	>0.05
Postmaturity	42	3	278.7 ± 4.7	>0.05
Postmaturity	43	2	$227.0 \pm 23.1$	< 0.01
Pre-eclamptic toxaemia	29-32	8	251.0 ± 11.2	>0.05
Eclampsia	36	10	270 8 ± 73.3	>0.05
Hydatiform mole	16	2	$272.0 \pm 121.0$	>0.05
Hydatiform mole	20	1	416.0	

<sup>\*</sup> As compared to normal pregnancy of some duration.

## Discussion

Serum copper in our normal non-pregnant subjects was  $122.0 \pm 15.0 \, \mu \text{g}/100 \, \text{ml}$ . This is in good agreement with the observations of De Jorge *et al* (1965), Vaidya and Mazumdar (1967), Friedman *et al* (1969) and Kapoor *et al* (1977).

The progressive rise in serum copper during normal pregnancy observed by us is similar to the data of Dokumov (1968), Friedman et al (1969) and Kapoor et al (1977). The rise in serum copper from 9th week of gestation onwards is so strik-

pregnancy, serum copper levels were variable. Friedman et al (1969) and Schenker et al (1969) found that serum copper started declining after term. We have observed a decrease only after 43 weeks.

We have observed a significant decrease in serum copper in PET and no change in eclampsia. This is contrary to the report of Screnker *et al* (1969) who observed a rise in serum copper in both PET and eclampsia.

Serum copper has been reported to rise markedly in hydatiform mole (Heijkenskjold and Hedenstedt, 1962 and Schenker et al 1969). Two of our patients at 16 weeks of gestation did not show any change in serum copper. One patient at 20 weeks had very high serum copper.

Thus our results show that normal pregnancy leads to a progressive rise in serum copper. Serum copper may help in the laboratory diagnosis of inevitable abortion, missed abortion and intrauterine death. However, variable changes were observed in threatened abortion, premature labour, potmaturity, toxaemia of pregnancy and hydatiform mole. Previous claims about the diagnostic value of serum copper in these conditions could not be confirmed in our study.

# Summary

Serum copper was estimated in 15 normal non-pregnant women, 60 normal women at different periods of gestation and 66 women with various obstetric complications. Serum copper progressively rose in normal pregnant women from 5th week of gestation onwards. Significantly lower values were found in inevitable abortion, missed abortion and intrauterine

death as compared to normal women at the same period of gestation. Serum copper could be of diagnostic importance in these conditions.

Previous cllaims about the diagnostic value of serum copper in threatened abortion, premature labour, postmature pregnancy, toxaemia of pregnancy and hydatiform mole could not be confirmed.

## References

- De Jorge, F. B., Delascio, D. and Antunes, M. L.: Obstet. Gynec. 26: 225, 1965.
- Dokumov, S. I.: Am. J. Obstet. Gynec. 101: 217, 1968.
- Friedman, S. Bahory, C., Eckerling, B. and Cans, B.: Obstet. Gynec. 33: 189, 1969.
- Heijkenskjold, F. and Hedenstent, S.: Acta. Obstet. Gynec. Scand. 41: 41, 1962.
- Kapoor, M., Kishore, N. and Gupta, K...
   J. Obstet. Gynec. India, 27: 367, 1977.
- Schenker, J. G., Jungeris, E. and Polishuk, W. J.: Am. J. Obstet. Gynec. 105: 933, 1965.
- Vaidya, R. M. and Mazumdar, B. M.: Ind. J. Med. Sci. 21: 232, 1967.
- Varley, H.: Practical Clinical Biochemistry, edition 4th, New Delhi, 1975, Arnold Heineman Publishers (India), p. 477.