SERUM CERULOPLASMIN IN ECLAMPSIA

by

N. S. Das,* M.D.

and

K. B. Sadasiva Rao,** M.D., D.G.O.

Introduction

Eclampsia and pre-eclampsia became a topic of interest as the 'Disease of Theories' (Jeffcoate, 1966). The aetiopathogenesis of eclampsia is yet to be understood. Hepatic function in eclampsia is impaired. Delayed excretion of Bromosulfonphthalein and elevation of serum glutamic oxaloacetic transminase level is noticed. Serum alkaline phosphatase is increased and usually the raised fraction was that of heat stable nature. Possibly the increased alkaline phosphatase activity of heat stable nature is due to placental function.

Haemorrhagic necrosis in the periphery of the liver lobule was commonly identified at autopsy of eclamptic patients. The hepatic manifestations of eclampsia may be due to the associated fall in the platelet count and decrease in fibrinogen and prothrombin concentrations (Page, 1972).

Ceruloplasmin is a copper transporting protein formed in the liver. This protein is in the concentration of 32.3 ± 4.9 mg per cent in normal adult women with a range of 25 to 43 mg per cent. The serum ceruloplasmin level is of great importance in the diagnosis of hepatolenticular degeneration. In all the diseases associated with hepatic dysfunction, such as hepatitis or obstructive lesions, the ceruloplasmin levels are raised. Walshe and Briggs, 1962 found very low levels of ceruloplasmin in fetal hepatitis.

It was agreed by most of the authorities that changes observed in liver in fatal cases of eclampsia have seldom been demonstrated in non-fatal cases. Keeping this in view, an endeavour is made to study the serum ceruloplasmin in eclamptic patients, non-eclamptic pregnant women and in healthy non-pregnant women.

Material and Methods

Pregnant women with eclampsia were admitted. On the first day, before treatment was initiated serum ceruloplasmin, Blood urea were estimated. Seven days after therapy and after the control of hypertension, serum ceruloplasmin was re-estimated. The patients were re-evaluated one month after delivery. A control group of pregnant women and non-pregnant women were evaluated for serum ceruloplasmin. The ceruloplasmin was estimated by the method of Ravin, 1961.

Results

Fourteen eclamptic patients were studi-

*Addl. Professor of Bio-chemistry.

**Previously Resident Postgraduate in Obstetrics and Gynaecology.

Departments of Biochemistry and Obstetrics and Gynaecology, Andhra Medical College, Visakhapatnam.

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ed. Patients were in the age group of 20 to 30 years and mostly primigravida. The ceruloplasmin content in serum of eclamptic, non-eclamptic pregnant and non-pregnant women is presented in Table I.

TABLE I

Sr. No.	Subject	Ceruloplasmin mg. %	Blood urea mg. %
1.	Healthy non-pregnant women (n = 100)	32.3 ± 4.9 (25 to 43)	20 ± 5 (14 to 35)
2.	Pregnant non-eclamptic (n = 5)	48.4 ± 7.5 (40 to 60)	20 ± 5 (14 to 35)
3.	Eclamptic (n = 14)	74.7 ± 14 (59 to 103)	33 ± 17 (16 to 78)

The patients with eclampsia after being treated have shown slight fall in ceruloplasmin content which however returned to normal one month after delivery in all non-fatal cases. The alteration in ceruloplasmin content from the first day of admission to 7th day and one month after delivery are presented in the Table II and Fig. 1.

TABLE II

Sr. No.	Time	Ceruloplasmin content mg %
1.	At the time of admission	74.7 ± 14
2.	7th day after admission	65.6 ± 3.5
3.	One month after delivery	40.2 ± 3.9

No fatal cases were reported during the period of study.

Discussion

In the present study, ceruloplasmin content in the serum is increased significantly in eclampsia when compared to normal pregnancy ($p < 0.001$). Blood urea also increased in concentration in eclampsia. Pregnant women have shown slight increase in serum ceruloplasmin over the normal non-pregnant women. The raised levels of serum ceruloplasmin in eclampsia have slightly decreased after

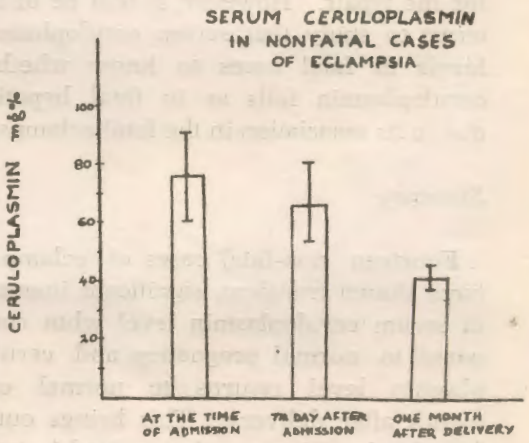

FIG.1

7 days by which time patient has dtlivered and blood pressure returned to normal. However, serum ceruloplasmin returned to normal range one month after delivery. This indicates that increased ceruloplsamin is of transient nature associated with phase of eclampsia. It was well documented in the earlier study of Scheimberg *et al* 1954, that there is a gradual increase in ceruloplasmin level in normal pregnant women during the pregnancy. It was also shown by Russ and Raymond 1956, that the oestrogen administration

leads to elevation of serum ceruloplasmin level. Since there is increased production of oestrogens in prtgnancy the increased content of ceruloplasmin may be due to the raised level in the oestrogens.

The significant raise observed in eclampsia over and above the levels observed in pregnant women may be due to the transient hepatic necrosis resulting in canalicular obstruction in the periportal areas of liver. The pathological changes were confirmed by Page, 1972. As the ceruloplasmin level returns to normal one month after delivery, it has to be presumed that the changes in liver are of transient nature and approximately take one month for the rtpair. However, it will be of interest to study that serum ceruloplasmin levels in fatal cases to know whether ceruloplasmin falls as in fatal hepatitis due to its association in the fatal eclampsia.

Summay

Fourteen non-fatal cases of eclampsia have shown transient, significant increase in serum ceruloplasmin level whtn compared to normal pregnancy and ceruloplasmin level returns to normal one month after delivery. This brings out a fact that hepatic necrosis reported in non-fatal cases may bring about a transient intracanalicular obstruction in the periportal tissue of liver and increase in ceruloplasmin. This alteration is corrected a month after delivery as indicated by normal ceruloplasmin content.

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