# PREGNANCY AFTER ECLAMPSIA

by

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Information about the immediate influence of eclampsia on reproductive performance is limited. Lopez-Llera and Horta (1974) followed up 110 women in subsequent pregnancies after eclampsia. Toxaemia recurred in 35.4 per cent of subsequent pregnancies. Susequent pregnancies tended to end before term with a 22 per cent incidence of intrauterine growth retardation (IUGR). There is no Indian report on the clinical behaviour of eclamptic women during their subsequent pregnancies. We have carried out a prospective study of 47 women who had eclampsia, during their subsequent pregnancies in an attempt to assess the immediate sequelae of eclampsia on blood pressure, renal functions and pregnancy outcome in following pregnancies.

## *Material and Methods*

The present series consisted of 48 subsequent pregnancies in 47 women who had eclampsia in previous pregnancies. Thirty women who had normotensive pregnancy with matched age served as control group. These patients were followed up after their eclamptic pregnancy in order to determine the underlying organic disease as well as their subsequent pregnancies were carefully supervised. The diagnosis of hypertensive disorder of pregnancy (HDP) was made according to the criteria laid down by the American Committee of Maternal Welfare. IUGR was diagnosed in infants whose birth weight for the corresponding gestational age was less than 1 standard deviation from the mean. Correction in the perinatal mortality rate (PMR) was made for birth weight of less than 1 kg. and/or for major congenital malformations. Statistical evaluation of the results was done using Chi square test with or without Yates correction. Women in the control group were of matched age, parity and the year of earlier delivery. Blood pressure, renal function tests and careful clinical examination was done in the subsequent pregnancies.

## *Results*

The mean age of the patient in the study group was 24.3 years. The average parity was 2.36, since 78.72 per cent of women were primigravidas at the time of their previous eclamptic pregnancy.

*Underlying Renal Disease:* Nine women

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(19.14 per cent) were diagnosed to have organic renal disease on detailed investigations after eclamptic pregnancy (Table I).

*Prematurity, Birth Weight and IUGR*: Prematurity rate (less than 37 weeks) in subsequent pregnancies in study group was significantly higher (29.1 per cent) when compared to the control group (3.33 per cent), but was found to be less than half when compared to corresponding figures of 66.6 per cent in their eclamptic pregnancies.

TABLE I

*Clinical Behaviour of Cases in Subsequent pregnancies with Underlying Renal Disease*

| Renal lesion | No. of patients | Behaviour in subsequent pregnancy |
|---|---|---|
| Chronic pyelonephritis | 4 | Albuminuria 1, Mild PET, 2, Hypertension 1 |
| Focal Proliferative glomerulonephritis | 1 | Albuminuria 1 |
| Mesangiocapillary GN | 1 | Albuminuria 1 |
| Minimal change GN | 1 | Albuminuria 1 |
| Focal and segmental glomerulosclerosis and hyalinosis | 1 | Albuminuria with hypertension 1 |
| Acute tubular necrosis | 1 | Hypertension in both subsequent pregnancies |

*Hypertensive Disorders of Pregnancy*: Various hypertensive disorders in subsequent pregnancy in the remaining 38 cases in study group is shown in Table II. There was no statistical difference when data was analysed according to the type of eclampsia in their previous pregnancy i.e. antepartum, intrapartum and postpartum.

*Weight Gain*: Weight gain after 20 weeks of pregnancy in the study group averaged 6.7-2.6 kg (Mean standard deviation) which is no different from the weight gain in the control group.

The mean gestational age in the study group was 36.84 weeks, which is more than their previous eclamptic pregnancy but was less than the control group (Table III). The mean birth weight in the study group was more than their eclamptic pregnancy while the incidence

TABLE II

*Hypertensive Disorders of Pregnancy in Study and Control Groups*

| Classification | Study group | | Control group | |
|---|---|---|---|---|
| | No. | Perinatal deaths | No. | Perinatal deaths |
| Mild PET | 6 | 1 NND | 1 | 0 |
| Severe PET | 5 | 1 MSB | 0 | |
| Essential hypertension | 7 | 1 MSB | 0 | |
| Superimposed PET on hypertension | 3 | 1 NND | 0 | |
| Normotensive | 17 | 1 NND | 29 | 0 |
| Total | 38 | 5 | 30 | 0 |

MSB—Macerated still birth. NND—Neonatal death.

TABLE III

*Gestational Age, Birth Weight, IUGR and PMR in Eclamptic and Subsequent Pregnancy*

| | Study group | | Control group N 30 |
|---|---|---|---|
| | Eclamptic preg. | Subsequent preg. | |
| Gestational age (weeks) | 34.85 ± 2.6 (N = 44) | 36.84 ± 2.12 | 39.1 |
| Birth weight (kg.) | 1.75 ± 0.55 (N = 38) | 2.35 ± 0.63 | 2.9 |
| IUGR (per cent) | 68.42 (N = 38) | 43.75 | 6.66 |
| Corrected PMR (per 1,000) | 608 | 62.5 | Nil |

N is the number of patients in whom data was available.

of IUGR was lower when compared to their previous eclamptic pregnancies.

There was no perinatal death in the control group, while there were 2 macerated stillbirths (0.85 kg, 0.8 kg) and 3 neonatal deaths (1.6 kg, 1.2 kg and 15 kg) in the study group. The corrected PMR in the study group was 62.5 per 1,000 which is one tenth of the corrected PMR of their eclamptic pregnancies. Complication by various hypertensive disorders of pregnancy adversely affected the foetal prognosis in the study group (Table II). Prematurity. low birth weight and extreme IUGR were additional factors contributing to these perinatal deaths.

*Discussion*

Controversy exists on after effects of eclampsia. Chesley *et al* (1976-78) believe that eclampsia is neither a sign of latent hypertension nor causes hypertension or its sequelae. There is another group which believes that one thirds of patients who have PET or eclampsia develop residual hypertension (Epstein. 1964). Pollak and Nottles (1960) in their study have shown a clear correlation between previous toxaemia and subsequent hypertensive disease. Fourteen patients (29.78 per cent) in the present study. including 4 women with underlying renal disease, were found to be hypertensive at the start of the subsequent pregnancy. Chesley *et al* (1976) have suggested high incidence of hypertension to multiparity. However, in our study multiparity is unlikely to be a contributing factor as majority of the patients were primigravidas (78.72 per cent) and young at the time of eclamptic episode. It is quite tempting to speculate that ecplampsia predisposses to the development of residual hypertension. However, larger studies with a carefully recorded past history are necessary to confirm this impression.

It is still not resolved whether or not recurrent toxaemia is a distinct entity or occurs in women with an underlying tendency to hypertension or pre-existing renal disease or else is the result of the damage sustained during the first episode of eclampsia (Theobald, 1965; Lopez-Llera and Horta, 1974). Our findings suggest that 39.42 per cent (11 cases) women in the study group who were normotensive at the start of subsequent pregnancy and did not have underlying renal pathology, developed recurrent toxaemia

(Table II) which is significantly higher than the control group (3.33 per cent).

Our observations also suggest that an episode of eclampsia does affect the perinatal outcome in subsequent pregnancies due to the complications associated with high rate of hypertensive disorders of pregnancy. Prematurity and IUGR were significantly commoner in the study group as compared to the control group. Similar trends were noted by Lopez-Llera and Horta (1974) in their study. It is suggested that further prospective studies should be undertaken in the developing countries to resolve the controversies regarding the clinical behaviour of these patients.

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