# EPIDEMIOLOGIC STUDY OF ECLAMPSIA

by

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Eclampsia is a preventable disease but is still frequently encountered in developing countries that lack effective systems of antenatal care. In rural areas, eclampsia accounted for 14 per cent of total maternal deaths (Upadhyay, 1975). At Post graduate Institute of Medical Research and Education (PGIMER), Chandigarh, eclampsia was responsible for 3.3 per cent of total maternal deaths (1969-78). This report presents our experience with eclampsia cases over a period of 10 years.

*Materials and Findings*

During the 10 years period from Jan. 1969-till Dec. 1978, 116 cases of eclampsia were managed at PGIMER, Chandigarh, giving an incidence as 0.58 per cent of all pregnancies. Among 363 twin deliveries, the incidence was 0.82 per cent which is approximately 1½ times that of singleton pregnancies.

*Religion*

Fifty eight per cent of the eclampsia patients were Hindus, 39.6 per cent were Sikhs and 2 were Muslims (1.72 per cent).

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*Residence*

The patients in this series belonged to the northern India, 65.5 per cent (76) patients had rural background as compared to 29 per cent among all obstetrical patients delivered in this hospital (unpublished data).

*Socioeconomic Status*

Socioeconomic status (ICMR-1966) showed that except 2 patients, all were in low or middle income group; only 18 patients ever went to school.

*Nutritional States*: The haemoglobin level was less than 11 g/100 ml in 24.1 per cent of eclamptics as compared to only 12 per cent of the 19683 women who delivered at this hospital. Determination of serum proteins in 66 eclamptic patients gave lower than normal figures in 43 (65.15 per cent) of them.

*Age and Parity*: Of the 116 patients, 62 per cent (72) were nulliparous and 38 per cent (44) were multiparous, of the latter, 8 were grand multiparous with parity ranging from 5 to 9. The age of the patients ranged from 16 to 44 years, with 18 per cent eclamptic above 30 years and 31 per cent were less than 20 years of age.

*Type of Antenatal Care*

Ninety two per cent of the eclamptics did not receive antenatal care. Five of 9

who did seek any, failed to follow instructions for attending the antenatal clinic or earlier hospitalisation. Four were advised to rest at home.

*Onset in Relation to Delivery*

Eclampsia occurred ante-partum in 53.54, per cent, intrapartum in 37 per cent and postpartum in 9.48 per cent. The mean number of convulsions that each patient developed prior to institution of therapy was 6 with a range of 1-41. Nine patients had their first convulsion in the hospital. History of fits was lacking in 1 case of postpartum eclampsia who presented with coma and died (autopsied).

The onset of postpartum convulsions was within 24 hours of delivery in 9 patients, 5 of them had within 4 hours of delivery. Two remaining had convulsions at 36 hours and 51 hours following deliver.

*Gestational Age*

The first convulsion occurred prior to 37 weeks in 72 (62 per cent) cases. In a few patients it occurred at or before 28 weeks of pregnancy, the earliest being at 26 weeks gestation.

*Symptoms of Eclampsia*

Headache and/or blurred vision was present in 61.2 per cent (71) of patients before convulsion developed.

*Blood Pressure*

It is pertinent here to point out that eclampsia can occur with normal blood pressure, though hypertension is commonly met with. Table 1 shows the blood pressures of eclamptic patients on admission.

There were 11 cases of eclampsia with normal blood pressure not exceeding 140/90, while 8 cases were admitted in shock. Twenty-three patients had systolic blood pressure exceeding 200 mm.Hg.

*Proteinuria*

Proteinuria was invariably present. Only 2 cases had 'trace' proteinuria, 10.3 per cent (12) had 1+ to 2+ while 87 per cent (101) had 3+ or 4+.

No consistant relationship was found between the amount of proteinuria and blood pressure. Absence of proteinuria was recorded in only 1 case of eclampsia.

*Microscopic Examination (M/E)*

M/E of catheter specimen of urine showed epithelial casts in 1, hyaline casts in 8, granular casts in 15, and combination of granular and hyaline casts in 13 cases. Gross haematuria was noted in 27 (23.27 per cent) cases while in additional 22 patients (18.96 per cent) significant microscopic haematuria was domonstrated. Significant pus cells were noticed in 4 eclamptics.

Blood urea and uric acid increased as the disease worsened. Following delivery a dramatic improvement was seen in blood biochemistry, urine findings and blood pressure.

Fundoscopy revealed normal finding in 47, grade I or II in 55, grade III in 5 (including 2 cases of retinal detachement) and grade IV changes in 5 cases. Fundus examination could not be done in 4 cases.

Clotting time and clot retraction time done at bed side were within normal limits in 114. Detailed coagulation status was investigated in 18 subjects. Two of them had disseminated intravascular coagulopathy with clinical manifestation. One patient died, while in another case the disease was mild and spontaneous correction occurred after resuscitative measure and one unit of fresh blood. Six patients had demonstrated laboratory data

suggestive of intravascular coagulation in absence of obvious clinical symptoms. Platelet count was consistently lower than normal in cases of eclampsia (unpublished data).

*Severity of Disease*

The majority of the patients were antepartum or intrapartum and thus arrived at the hospital in severe eclamptic state having had multiple convulsions during the transport. There were 65 case of severe eclampsia (Eden's Criteria). Thirty-nine cases presented with intrauterine death at admission.

*Management*

On admission the patient was placed in an air-conditioned intensive care room within the labour room complex. Lytic—cocktail therapy (Menon, 1961) was used for control of convulsions. Additional sedatives and anticonvulsants (diazepam-28, paraldehyde 6, I/V pentothal 1) were supplemented if convulsions were not controlled within 3-4 hours of start of therepy. When blood pressure was above 160/110 mm Hg despite deep sedation, injectable serpasil was used. I/V frusemide was used for pulmonary odema and to produce diuresis in 14 cases. Supportive treatment included suction of throat, supplemental $O_2$ and antibiotics alongwith continuous nursing and medical supervision.

Immediate control of convulsions occurred in 66.3 per cent (77) patients. Control of convulsions was not possible withing 8 hours of start of treatment in 8 cases. Blood pressure became normal in 48 hours in 71 cases and within 1 week in another 17 cases. Twenty-eight cases required oral antihypertensive agents for control of blood pressure. Eighty-eight (75.86 per cent) cases had a urinary output above 1000 ml/24 hours and 21 had an output of 400-1000 ml/24 hours.

*Mode of Delivery*

One patient died undelivered and 4 had delivered at home. 84.4 per cent (98) patients had vaginal delivery. Cesarean section (C.S.) was performed in 13 cases (11.20 per cent) for uncontrolled fits in 5, uncontrolled fits and oliguria in 2, unfavourable cervix in 2, failed induction in 2, total placenta previa in 1, and imminent eclampsia in 1 (1 fit postpartum) were the indications for C.S.

*Maternal Morbidity and Martality*

Twenty-three cases were in coma for more than 72 hours and 9 had hyperpyrexia. Other complications (Table II)

TABLE I

*Blood Pressure Recordings in* 116 *Patients of Eclampsia on Admission*

| Blood Pressure mm of Hg | Number of patients | | |
|---|---|---|---|
| | Antepartum | Intrapartum | Postpartum |
| Shock (less than 80) | 4 | 3 | 1 |
| Normal (Systolic less than 140 and diastolic less than 90) | 6 | 4 | 1 |
| Systolic 140 to 160 and/or diastolic 90 to 110 | 10 | 5 | 4 |
| Systolic above 160 and/or Diastolic above 110 | 42 | 31 | 5 |
| Systolic above 200 | 14 | 6 | 3 |
| Diastolic above 120 | 28 | 16 | 5 |

TABLE II
*Complications in 116 Patients of Eclampsia*

| Complication | Number of patients | | |
|---|---|---|---|
| | Antepartum | Intrapartum | Postpartum |
| Coma of 72 hours or more | 11 | 11 | 1 |
| Temperature above 105°F | 3 | 6 | - |
| Pulmonary edema | 1 | 2 | - |
| Oliguria | 1 | 2 | 1 |
| Anuria | 2 | 1 | - |
| Disseminated intravascular coagulation | 1 | 1 | - |
| Postpartum psychosis | 6 | 7 | - |
| Shock | 4 | 3 | 1 |
| Cerebrovascular accidents | 4 | - | - |
| Respiratory infection | 6 | 9 | - |
| Urinary tract infecton | 9 | 9 | - |
| Acute hepatic failure | 1 | 1 | 1 |
| Aspiration pneumonia | 1 | 1 | - |
| Atonic P.P.H. | 2 | 1 | - |
| Abruptio placentae | 3 | 1 | - |

included pulmonary odema in 3, oliguria in 4, acute renal failure 3 (2 acute tubular necrosis, 1 cortical necrosis), cerebrovascular accident in 4, postpartum psychosis in 13 and acute hepatic failure in 3.

There were 9 maternal deaths in this series, giving mortality rate of 7.75 per cent. The maternal death rate was 6.45 per cent for antepartum eclampsia, 9.3 per cent for intrapartum and 9.09 per cent for postpartum eclampsia. Maternal mortality among multiparas was 3 times that of nulliparas (13.63 per cent versus 4.16 per cent). Older eclamptics over 30 years had much higher maternal mortality than younger eclamptics (19.04 per cent versus 5.26 per cent).

Eden's Criteria was found to be a good prognostic marker of severity. All deaths occurred in severe eclampsia (13.8 per cent). Maternal mortality was 17.3 per cent in patients in whom blood pressure (systolic) exceeded 200 mm hg as compared to 5.37 per cent in whom blood pressure was less than 200 mm hg. Likewise the presence or absence of FHS at admission was found to be a good index of the chances of survival of the patient. Of 77 patients admitted with positive FHS, there were only 4 maternal deaths (5.19 per cent) compared to 5 maternal deaths in 39 patients admitted with no FHS (12.8 per cent).

No deaths occurred among the patients who developed a first convulsion in the hospital. Thirteen caesarean sections were done in this series with 1 death, not attributable to operation. It was of interest to note that maternal mortality increased 10 times (50 per cent) when eclampsia occurred earlier than 30 weeks than when it occurred after 30 weeks (5.45 per cent) of gestation. There was no loss of life in eclamptics with twin pregnancy.

Necropsy was denied in 2 of 9 maternal deaths. One of these patients had signs of pulmonary odema, hypotension and hyperpyrexia; while other succumbed to acute respiratory failure 70 minutes following CS. The principal findings at necropsy were acute liver necrosis in 3,

*Evaluation of the Shake Test for the Fetal Well-being—Sharma et al. pp.* 21-24

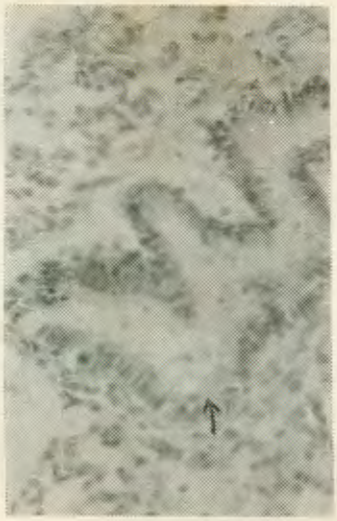

Fig. 1
Hyaline membrane lining the terminal air-passages.

*Abdominal Pregnancy after M.T.P.—Jha et al. pp.* 144-145

Fig. 1
Photograph showing the foetus with specimen of uterus.

*Florid Immediate Post-Operative Pulmonary Oedema—Shahul & Lalla pp.* 124-126

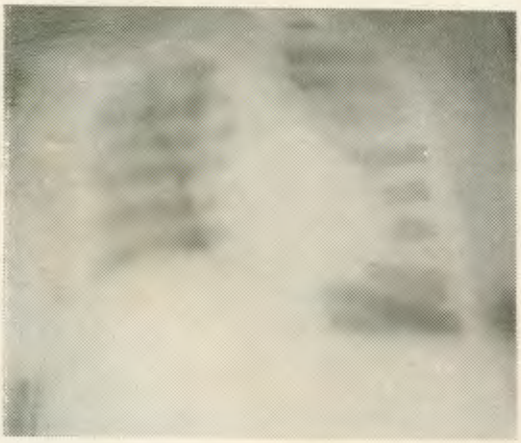

Fig. 1
Before treatment "Florid pulmonary oedema".

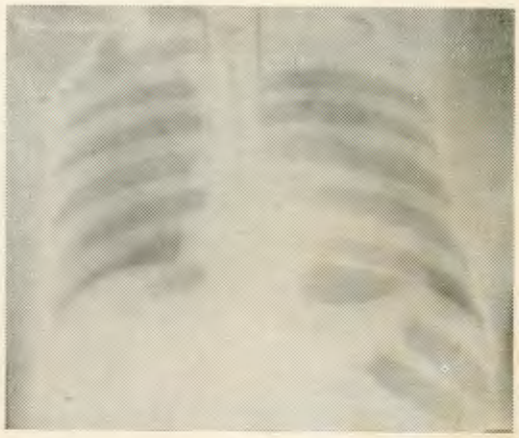

Fig. 2
After treatment "Marked regression of pulmonary oedema."

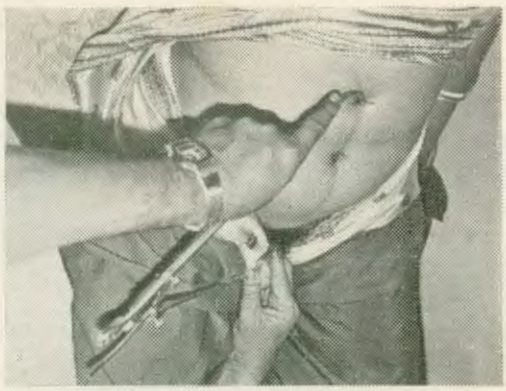

Fig. 1
Symphysis fundal vertical uterine length (L) by pelvimeter in cm.

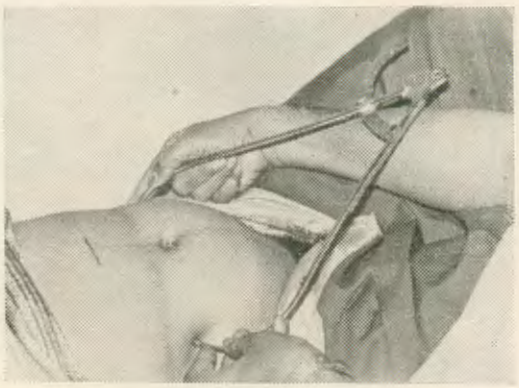

Fig. 2
Maximal uterine transverse diameter (T) measured by pelvimeter in cm.

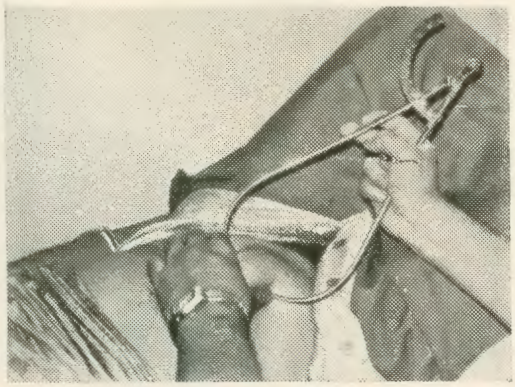

Fig. 3
Double abdominal wall thickness (DAWT) as measured by pelvimeter in cm.

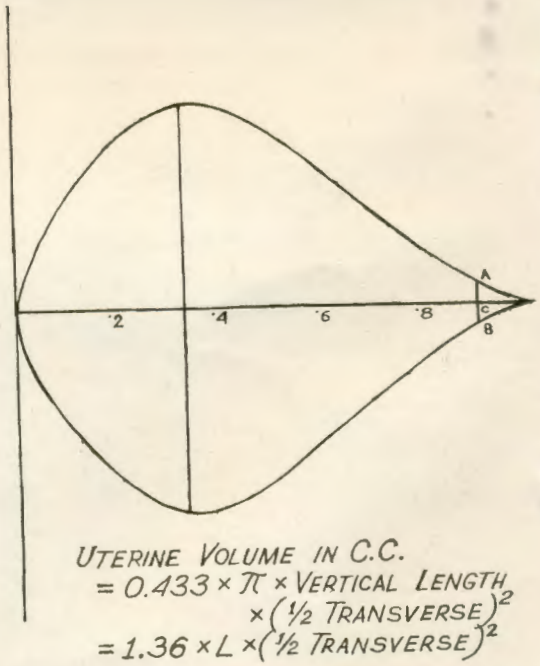


Fig. 4
Uterine value calculated by integrated calartus.

*Primary Malignant Melanoma of Vagina--Das & Mohanty pp.* 152-153

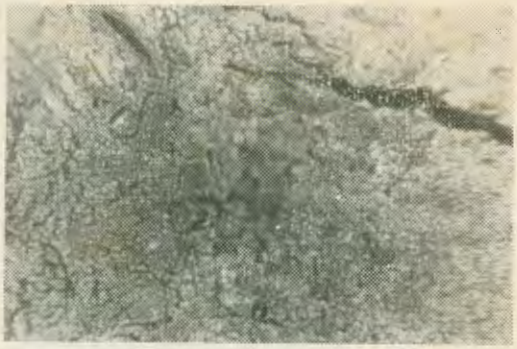

Fig. 1
Histopathology of malignant melanoma of vagina.

Fig. 2
Gross specimen of uterus, ovaries and tubes with vaginal melanoma.

*Primary Syphilis of Cervix—Sen & Chakrabarty pp.* 155-156

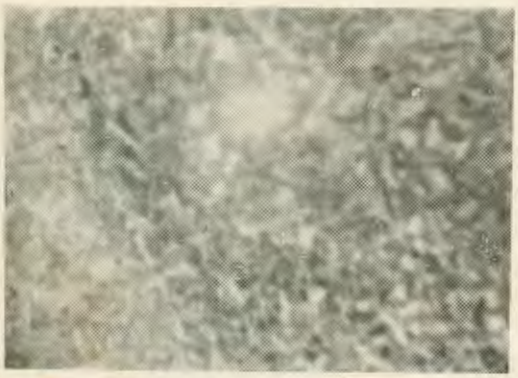

Fig. 1
Histological appearance of the lesion showing perivascular plasma cell infiltration.

*Rectal Prolapse—Munshi et al. pp.* 157-158

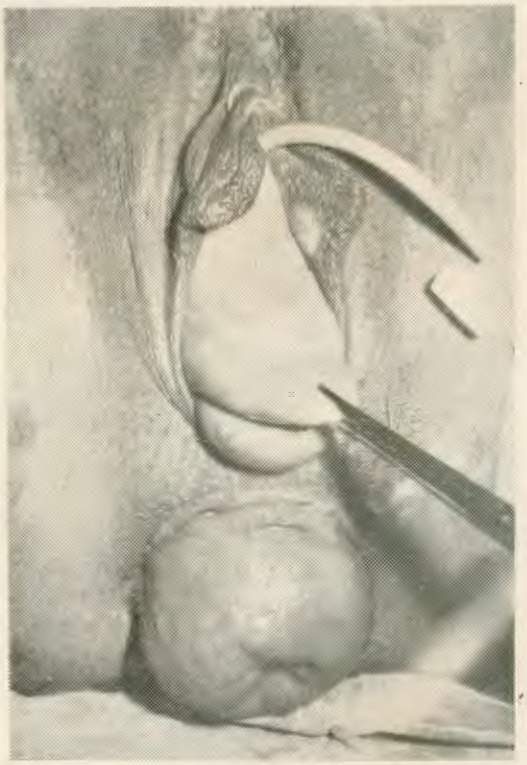

Fig. 1
Uterine prolapse with rectal prolapse cervicopexy with Grahams operation has been done.

acute cortical necrosis with features of DIC in 1, bilateral confluent bronchopreumonia in 1, pulmonary odema in 1, and intracranial haemorrhage in 1. Patient with intracranial haemorrhage could have been saved had CS performed instead of waiting for vaginal delivery once the convulsions were controlled because B.P. remained elevated (240/140) despite antihypertensives.

There was no clear trend of maternal loss noticed with increasing time interval between onset of fits and delivery in this study.

*Perinatal Mortality*

There were 48 still births and 10 neonatal deaths including 2 major congenital malformations, giving an uncorrected perinatal mortality of 508 per 1000 (corrected 438 per 1000). In 39 of 48 stillbirths, mothers were admitted with absent FHS.

*Discussion*

During the last 50 years there have been many innovations in the management of eclampsia and various drugs have become available. But the direct comparison of mortality rate at different centers using different regimens is impossible unless allowances is made for the severity of eclampsia. It is quite clear from this study that the occurrence of eclampsia with its high risks to mother and offspring is due to poor antenatal management. WHO expert committee in 1975 concluded that nutritional factors have no influence on incidence of eclampsia. Majority of our patients belong to poor socioeconomie status which is responsible for malnutrition as well as lack of proper antenatal management.

The maternal mortality from eclampsia in recent reports ranges from 0-17.5 per cent (Menon, 1961; Crichton *et al*, 1968; Lean *et al*, 1968. Pritchard and Pritchard, 1973; Lopez-Llera *et al*, 1976. Dawn and Sinha, 1979). The high incidence of deaths from eclampsia (7.75 per cent) in our study, is a direct reflection of poor antenatal care and late admissions of patient from rural areas. There were no significant changes noticed in incidence and maternal mortality due to eclampsia at PGIMER, Chandigarh between the two 5-year periods of 1969-73 and 1974-78. It is of interest to note that unlike other studies from India (Menon, 1961; Upadhyay, 1964; Dawn and Sinha, 1979), the present study reveals that maternal mortality is higher with intrapartum and postpartum eclampsia in comparison to antepartum eclampsia (9.3 per cent and 9.09 per cent versus 6.45 per cent).

The maternal mortality was significantly higher with advancing age, in multiparas, before 30 weeks of pregnancy, with systolic B.P. more than 200 mm.Hg, in mothers with absent FHS at admission, and in severe eclamptics.

Perinatal mortality in eclampsia was high in our series (corrected 438 per 1000). This is largely attributable to high proportion of eclamptics admitted with absent FHS, chronic placental insufficiency, preterm deliveries, low birth weight and sedatives used in such cases (unpublished data).

Finally, as this potential loss of life from the severe form of disease is certainly preventable, the results of this study indicated the urgent need of better public health education and importanoe of continued efforts in monitoring and reviewig the line of treatment.

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