PERINATAL MORTALITY AND MORBIDITY ASSOCIATED WITH ECLAMPSIA

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The multiplicity of treatment regimens currently used for the treatment of eclampsia results in essentially the same low maternal mortality (0-15%) but do not yield substantial fetal survival figures. The uncorrected fetal mortality ranges from 30-60 per cent, main reasons being the toxaemia itself, prematurity and the sedation used in the treatment (Mudaliar and Menon, 1972). Unless proper attention is given to the problems affecting the infant, further reduction in perinatal mortality (PMR) is unlikely. We have carried out a retrospective survey of births in eclampsia patients during 1969-78 to review the PMR and discuss the various causal and contributory factors for high perinatal loss.

Material and Findings

Perinatal deaths included still births (SB) and all neonatal deaths (NND) occurring within 7 days of life. Correction in PMR was made for newborns weighing less than 1 kg., born with less than 28 weeks of gestation and for major congenital malformations. A cause of death was assigned after considering the clinical and pathological data including the necropsy findings.

Between 1969-78, 116 cases of eclampsia were treated at PGIMER, Chandigarh, giving an incidence of 0.58 per cent. One hundred and seven patients had their first fit at home and 92 per cent had received no antenatal care. Convulsions occurred before labour in 62, during labour in 43 and postpartum in 11 cases. 69 per cent women were 20 years or above, and 38 per cent had previous deliveries. In 20 of them there was past bad obstetrical history. One patient had eclampsia in a previous pregnancy. Eight women reported having toxaemia in previous pregnancy. Family history of eclampsia (mother and/or sister) was elicited only in 3 cases. In 44 (38 per cent) cases eclampsia occurred after 37 weeks of gestation, while in 72 (62 per cent) it occurred prior to 37 weeks, the earliest being at 26 weeks gestation.

One patient died undelivered and 4 patients had delivered at home. The 111 women delivered had 114 babies, including 3 sets of twins. Of the total 58 perinatal deaths, there were 48 SBs and 10 NNDS. In 39 cases fetal heart sounds (FHS) were absent at admission while in another 9, intrauterine death occurred in hospital. There were 33 males (56.8 per cent) and 25 females (43.1 per cent).

Table I summarises the PMR in relation to birth weight and gestational age. Prematurity emerged as the single largest

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Accepted for publication on 24-3-81.

TABLE I

Distribution of Perinatal Deaths According to Gestational Age and Birth Weight (Figures in parenthesis indicate total number of infants)

Birth Weight in grams	Gestational Age in weeks				
	Less than 28	29 to 32	33 to 36	37 & above	Total
Less than 1000	5 (5)	5 (5)	—	—	10 (10)
1001 to 1500	1 (1)	5 (6)	6 (10)	1 (1)	13 (18)
1501 to 2000	—	5 (7)	8 (17)	2 (4)	15 (28)
2001 to 2500	—	1 (1)	4 (12)	5 (18)	10 (31)
2501 & Above	—	—	2 (5)	8 (22)	10 (27)
Total	6 (6)	16 (19)	20 (44)	16 (45)	58 (114)

factor for the perinatal loss 72.4 per cent of total fetal loss occurred in preterm infants (< 37 weeks). Heavier the infant better was the chance for survival. There were 48 perinatal deaths in the low birth weight (< LBW- < 2.5 kg) group, and only 10 in the normal weight group. This brings out LBW of fetus as other major factor for the perinatal loss.

Another major contributing factor to PMR was intrauterine growth retardation (IUGR) i.e. birth weight below-1 standard deviation (-ISD) for gestational age. IUGR occurred in 56, including 6 who were twins, 11 infants (9.64 per cent) had birth weight which was less than-2SD and 45 (39.4 per cent) had birth weight of -1SD of the mean for the corresponding gestational age (based on charts prepared in our institution). Five of 11 infants with birth weight of -2SD died (45.45 per cent), whereas the mortality figure for infants with birth weight of -1SD was 53.33 per cent. Thus 29 of 56 growth retarded infants were lost, 21 being preterm, while 8 were term (Table II).

TABLE II

Foetal Outcome in 108 Single and 3 Twin Pregnancies in Women with Eclampsia

Time of onset of Eclampsia		Intrauterine growth retardation		Well grown		Total	
		Preterm	Term	Preterm	Term		
Antepartum	Survivors	9	8*	7	6	30	64
	Deaths	15** (4 NNDs)	1	14 (4 NNDs)	4	34	
Intrapartum	Survivors	3	5	7	5	20	42
	Deaths	5	6	7	4 (2 NNDs)	22	
Postpartum	Survivors	0	1	1	4	6	8
	Deaths	1	1	0	0	2	

* I pair of twin, ** 2 pairs of twins, NNDs Neonatal Deaths.

There were 8 perinatal deaths among the 23 mature well grown babies, 4 of whom came with absent FSH and in 1 intrauterine death occurred in the hospital. Causes of death in rest were congenital malformation in 2 (hydrocephalus 1, 1, tracheo-esophageal fistula) and severe birth asphyxia in 1.

The causal factor for 10 NNDs were birth asphyxia in 3, extreme low birth weight (< 1 kg.) in 3, respiratory distress syndrome (RDS) in 2-tracheo-esophageal fistula in 1, bronchopneumonia and septicemia in 1. Of these, 2 were term and average for gestational age (AGA), 4 were growth retarded and preterm, and 4 were preterm but AGA. (Table II).

Thirteen of 56 surviving infants had complications. Two had minor congenital abnormality in the form of talipes, 2 infants had cynotic spells but grew out of it without neurological sequelae, 3 growth retarded infants suffered from hypoglycemia, 3 preterm infants had hyperbilirubinemia (one needed exchange transfusion), 1 each had bronchopneumonia, RDS and diarrhoea.

Perinatal Mortality

The overall PMR was 508 per 1000 (corrected 438 per 1000). This figure is of course, influenced by inclusion of 39 patients who came with intrauterine death. There were 9 fresh SBS and 10 NNDs including 2 infants with congenital malformations in eclampsia patients admitted with positive FHS giving uncorrected PMR of 253 per 1000 (corrected 160 per 1000).

For the prematures the PMR was 608 per 1000, and it was 355 per 1000 for the term infants. The PMR figures for multiple pregnancies versus singleton pregnancies were 666 per 1000 and 500 per 1000 respectively. The incidence of PMR in antepartum eclampsia, intrapartum eclampsia and postpartum eclampsia were 531, 523 and 250 per 1000 respectively. The PMR was higher among very young women (< 20 years) and in those over 30.

The initial B.P. reading were correlated with PMR, the latter being lowest in the normal B.P. range, increased when initial B.P. was extremly low (50 per cent) and, markedly increased if initial B.P. was very high. It was 826 per 1000 for infants whose mother had a systolic B.P. above 200 mm.Hg. on admission compared to PMR of 428 per 1000 per 91 infants whose mothers had a systolic B.P. below 200 mm.Hg. Increase in B.P. in hospital seemed to cause no significant alteration in PMR unless the rise was more than 50 systolic, 40 diastolic or both above the baseline. There was marked increase in PMR with severe form of eclampsia (Eden's Criteria) i.e. 753 per 1000 versus 176 per 1000 in mild form. Table III shows the PMR in relation to the onset of convulsion and delivery interval. When only cases with positive FSH were taken into consideration, there was no clear trend of increasing PMR with convulsion and delivery interval.

Thirteen had casarean section (C.S.) but 8 had C.S. performed for eclampsia per se (unpublished data). One SB occurred with C.S. done in a case of total placenta previa (absent FHS). Two deaths were attributable to major congenital malformations (hydrocephalus 1, T-O. fistula 1), 2 died in early neonatal period due to RDS and septicemia respectively. There were 7 SBs among 32 patients in whom the labour was induced. The corrected PMR in C.S. group was 230 per 1000, while it was 475/per 1000 in vaginal delivery group (Table IV).

TABLE III
Convulsion Delivery Interval and Perinatal Deaths

Time of onset of Eclampsia	Convulsion delivery interval in hours				
	12	24	36	48	More than 48
ANTEPARTUM					
Total no. of births	3	18	15	10	15
No. of PNDs	1	5	9	8	11
Absent FHS on admission	1	3	4	4	7
FHS disappeared in hospital	—	1	3	2	1
NNDs	—	1	2	2	3
INTRAPARTUM					
Total no. of births	11	22	7	2	0
No. of PnDs	4	11	5	2	0
Absent FHS on admission	4	9	3	2	0
FHS d'sappeared in hospital	0	1	1	0	0
NNDs	0	1	1	0	0

TABLE IV
Mode of Delivery and Perinatal Deaths

Mode of delivery	Total	Still Births	Neonatal deaths	Congenital malformation
Cesarean section	13	1	2	2
Vaginal delivery	98	46	7	0

Discussion

In the present series, the over all PMR in 111 eclamptics who delivered in this hospital was 508 per 1000 (corrected 438 per 1000). In 75 babies who were born where the FHS had been heard on admission 56 survived, giving PMR of 253 per 1000 (corrected 160 per 1000).

It is generally accepted that the stress of labour, traumatic delivery, and heavy sedation of the eclamptic women in labour increases the danger to the fetus. It is most disturbing to find that in 9 patients the IUD occurred in the hospital and 3 babies succumbed in early neonatal period due to severe birth asphyxia.

The perinatal deaths in eclampsia are mainly related to preterm deliveries, low birth weight IUGR, intrauterine death, and severe birth asphyxia (Tables I, II). Perinatal mortality to great extent can be minimised only with good compliance by the patient, a factor notably lacking in most of our eclamptic cases. Facility for domiciliary visiting for neonates is indicated. It is partinent to note the IUD in hospital occurred only after 12 hours of convulsion delivery interval (Table III). Therefore, it is imperative that after the

initial medicinal treatment an immediate decision should be made on the mode of delivery. If the fetal heart sounds are heard on admission, delivery must be effected as soon as posible. Because of perinatal deaths resulting from anoxia, more liberal use of CS should be seriously considered in cases with reasonable fetal maturity and weight. The time of CS is critical as much care and expertise may come to naught if CS is delayed.

The present study reveals that perinatal mortality was higher in young women (< 20) and those over 30, with multiple pregnancy, with initial BP which was extremely low or very high, and in severe form of eclampsia according to Eden's criteria. PNM was uniformly high with antepartum and intrapartum eclampsia.

The extent to which the different sedatives and anticonvulsants influence perinatal outcome is not clear. Treatment should be simple and avoiding large doses of drugs that "carry over" and might depress respirations and have adverse effects as impairment of the thermoregulation. Opinions are contradictory regarding the safety of diazepam therapy as regards the baby. Although 30 mg is now recognised as being the maximum safe maternal dose if fetal effects are to be avoided (Cree *et al*, 1973). Lean *et al* (1968) has lowered PMR to 11 per cent with diazepam in much higher doses but they performed CS in 68 per cent of their patients and promptly induced labour in others. Pritchard and stone (1967) using magnesium sulphate therapy reported fetal salvage rate of 90 per cent, if the fetus was alive at the time of admission of the mother to the hospital. However value of these regimes and C.S. has not yet been evaluated in controlled trials.

Finally, the effect of heavy sedation, prematurity and growth retardation, can probably be minimised by proper management and prompt resuscitation of newborn. Modern technique of combating drug depression and availability of neonatal intensive care facilities are essential.

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