

Eclampsia in Kelantan

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Summary

A review of eclampsia in Kelantan was undertaken from 1983-1988. There were 146 documented cases in the state (66 per 100,000 deliveries). Eight maternal deaths occurred. Sixty seven (45.9%) were primigravida. Six of the 79 multiparous women developed eclampsia for the first time following remarriages to new partners. The multisystem dysfunction resulting from eclampsia resulted in varied maternal complications. Fatal cerebral haemorrhage (3 cases), acute pulmonary oedema (8 cases), acute renal failure (6 cases), HELLP Syndrome (8 cases) and acute abruptio placentae were the commoner complications. The average number of convulsions per patient was 1.3. The mean gestation of mothers who delivered prematurely (28.2%) was 34.6 weeks and that for those at term (71.8%) was 39.1 weeks. The caesarean section rate was 42.5 %. The perinatal mortality rate was 185.9 per 1000. The implications of this high maternal and fetal mortality and morbidity are discussed in the light of the health delivery system and patient education. A team approach to medical management of eclampsia with the need for intensive care monitoring is suggested.

Key Words: Eclampsia, maternal complications, fetal mortality, intensive care monitoring.

Introduction

Eclampsia remains a common cause of maternal morbidity and mortality in the east coast states of Peninsular Malaysia¹. The multisystem dysfunction resulting from the severe sequelae of pre-eclampsia has been predominantly under the purview of obstetricians and primary health care workers. Despite the unsettled controversies surrounding the aetiology of pre-eclampsia, the pathophysiology and clinical manifestations are well understood. Equipped with better understanding of the consequences of the disease there appears a need to involve intensive care specialists like anaesthesiologists and neurologists, to obtain good maternal outcome in eclampsia patients in developing countries².

The convulsive state and its severe consequences can be prevented if pre-eclampsia and hypertensive diseases are adequately managed and appropriately referred³. The occurrence of eclampsia in both primigravidae and multiparous patients in Kelantan with predictable frequency

led us to review the disorder. The aim of the study was to review the pattern of the disease and to highlight the magnitude of the problem in Kelantan.

Materials and methods

Pregnant mothers beyond 24 weeks of pregnancy, up to two weeks after delivery who had well documented generalised convulsions between January 1983 to December 1988 were studied. The case records of parturients admitted to either the General Hospital, Kota Bharu or the five district hospitals in Kelantan from 1983 to 1985 were studied. Beyond 1986, with the Obstetric Department functioning only at the University Hospital, the remaining cases were studied prospectively.

Predetermined management protocols were used in managing eclampsia in all patients admitted to the University Hospital. The following parameters were noted: age, parity, pre-existing hypertension or medical disorders, blood pressure at first antenatal visit, gestational age at first onset of hypertension, blood pressure recording on admission to hospital, antihypertensive drug therapy and eclampsia management, complications of eclampsia and related therapy, period of gestation at delivery and mode of delivery.

The perinatal and neonatal outcome were studied and paediatric evaluation was sought if the neonate was not admitted for intensive care to the special care nursery.

All patients who had recovered from eclampsia were followed up at the postnatal clinic for up to six weeks postpartum. In cases where the blood pressure remained elevated beyond this period were placed under a physician's care.

Investigations done on admission included full blood picture, coagulation screen, renal and liver function tests. Coagulation screening was routinely done for all severe pre-eclampsia cases and eclampsia only after late 1986. Biophysical and biometric evaluation by ultrasonography was resorted to when deemed necessary.

Intravenous diazepam was the only anticonvulsant used in the study. Hydralazine was administered to control severe hypertension. Labetolol, both orally and by infusion was employed in some cases where immediate control of hypertension to desired levels was not adequately accomplished⁴. Caesarean section was done after stabilisation if delivery was not imminent within a few hours of convulsions and the cervix remained unfavourable.

Results

Incidence of eclampsia:

There were a total of 234,672 deliveries in Kelantan during the period of study. One hundred and forty-six cases were admitted for management of eclampsia. The incidence of eclampsia in the state was 66 per 100,000 deliveries. The incidence of hypertensive diseases complicating pregnancy in the hospital population was 8.3 per cent (1988). Three of the 23 maternal deaths in 1988 was due to eclampsia.

Demographic characteristics:

The incidence of eclampsia by age and parity is shown in Table I. The youngest patient was 16

years and the oldest 43 with a mean of 30.3 years. Of the 79 parous women, 12.3 per cent were in the high parity ⁶ group. Six of the multiparous mothers had eclampsia following remarriages and uneventful previous pregnancies.

Nutritional anaemia was seen in 18 (12.3%) cases. Other medical disorders included congenital heart disease in four, thyrotoxicosis in three and nephrotic syndrome in two. Of the multiparous patients, 32 (21.9%) were known to be hypertensive prior to pregnancy.

Table 1 Eclampsia in Kelantan by Age and Parity

| Age (Years) | Parity | | |
|-------------|----------|----------|----------|
| | 1 (%) | 2-5 (%) | 6 (%) |
| 15-19 | 25(37.3) | 0 | 0 |
| 20-24 | 22(32.8) | 12(19.7) | 0 |
| 25-29 | 14(20.9) | 18(29.5) | 2(11.1) |
| 30-34 | 4(6.0) | 18(29.5) | 7(38.9) |
| 35-39 | 1(1.5) | 13(21.3) | 7(28.9) |
| 40 | 0 | 0 | 2(11.1) |
| TOTAL | 67(45.9) | 61(41.8) | 18(12.3) |

Maternal complications:

The maternal complications seen are listed in Table II. Fatal cerebral haemorrhage occurred in three cases. Postmortem was denied in all these patients. Transient disorientation and loss of recent memory lasting from 12 hours to three days were noted in six (4.1%) cases. Transient postictal blindness was confirmed in two patients. Loss of vision lasted for eight hours post partum in one and 36 hours in another. Long term evaluation of the two cases of puerperal psychosis confirmed them to be suffering from schizophrenia.

Acute pulmonary oedema was confirmed in eight, two of which proved to be fatal. Laryngeal oedema giving rise to respiratory distress in two cases was probably under-reported. One of these patients had ventilatory support for 48 hours following caesarean section. The other improved after Frusemide and steroid therapy without the need for intubation.

Post-operative jaundice in five patients was ascribed to multiple blood transfusion. Following the introduction of routine coagulation screening for all severe hypertension and eclampsia, eight patients fulfilled the criteria for the 'HELLP' Syndrome ⁵. Two maternal deaths were due to coagulopathy and DIOC. Sixty-two (42.5%) caesarean sections were performed. Difficulties at intubation were recorded in 7%. Observable laryngeal oedema requiring a smaller intubation tube ⁶ was documented in eight cases. Abruptio placentae causing increased maternal morbidity and stillbirth was seen in 12 (0.7%) cases.

Table 2 Maternal Complications in Eclampsia

| | | | |
|-------------------------|------------------|----------------------------|-----------|
| 1. | CNS | i) Cerebral Haemorrhage | 3 (2.1%) |
| | | ii) Loss of Memory | 6 (4.1%) |
| | | iii) Postictal Blindness | 2 (1.4%) |
| | | iv) Puerperal Psychosis | 2 (1.4%) |
| 2. | Renal | i) Renal impairment # | 12(8.2%) |
| | | ii) Acute Renal Failure | 6 (4.1%) |
| 3. | CVS/Resp. | i) Bronchopneumonia | 4 (2.7%) |
| | | ii) Acute Pulmonary Oedema | 8 (5.5%) |
| 4. | Hepatic | i) Jaundice | 5 (4.1%) |
| | | ii) 'HELLP' Syndrome | 8 (5.5%) |
| 5. | Placenta | i) Abruptio Placenta | 12 (8.2%) |
| 6. | Coagulopathy | | 11 (7.5%) |
| # Documented cases only | | | |
| * | Maternal Deaths: | Coagulopathy | 2 |
| | | Cerebral Harmorrhage | 3 |
| | | A. Pulmonary Oedema | 3 |

The mean systolic and diastolic blood pressure prior to the acute episode was 168 mm Hg and 108 mm Hg respectively. The highest blood pressure recorded was 240/150 mm Hg. This patient did not have any neurological deficit at the time of discharge. The average number of convulsions per patient was 1.3. Forty-four (28.2%) patients were less than 37 weeks pregnant at the time of convulsions. In this group the mean gestation was 34.6 weeks (range 29.1 - 36.5). In those mothers who delivered term infants (71.8%), the mean gestation was 39.1 weeks (range 38.2 - 42.1). The timing of convulsions with respect to delivery is shown in Table III. In 8/82 (56.2%) with antepartum convulsions, there was an interval of two days prior to termination because of delay in seeking treatment. Recurrent fits inspite of anticonvulsant therapy occurred in nine (4.8%). Two of these had generalised convulsions after instrumental deliveries.

The mean time interval between delivery and onset of initial convulsions in the postpartum group was 1.8 hours (0.4 - 5.2). Delayed convulsions due to cortical vein thrombosis occurred in two cases six and ten days postpartum respectively.

A total of 156 babies including five sets of twins were born. All the twins were premature. One hundred and thirty-nine (89.1%) were liverborn and 17 (10.9%) stillborn. Of the latter, seven were fresh stillbirths. Forty four percent of the neonates were less than 37 weeks gestation. Twelve of these weighed less than 1500 gms and died within twelve days of birth. The mode of delivery is shown in Table IV.

The perinatal mortality rate was 185.9 per 1000. The uncorrected perinatal mortality rate for the hospital population during this period was 40.2 per 1000. The main causes of perinatal deaths were

Table 3 Timing of Eclampsia

| Fit | n | % |
|--------------------------|----|-------|
| Antepartum | 82 | 56.2 |
| Intrapartum | 24 | 16.4 |
| Antepartum & Intrapartum | 7 | 4.8 |
| Postpartum | | |
| i. Immediate (2hr) | 20 | 13.7 |
| ii. Delayed (2 - 12 hrs) | 11 | 7.5 |
| (12 hrs) | 2 | 1.4 * |

* Cortical Vein Thrombosis

Table 4 Mode of Delivery in Eclampsia

| | Preterm * | | Term | |
|-----------------|-----------|-----------|-----------|------------|
| | SB | LB | SB | LB |
| Normal Delivery | 3 | 15 | 1 | 47 |
| Breech | 1 | 5 | 1 | 1 |
| Forceps | 0 | 3 | 6 | 11 |
| Caesarean | 2 | 15 | 3 | 42 |
| TOTAL | 6 | 38 | 11 | 101 |

* Preterm 37 weeks

SB Stillbirth

LB Livebirth

prematurity, birth asphyxia and septicemia.

Discussion

Eclampsia remains an important contribute to maternal and perinatal mortality in both developed and developing countries. The incidence of eclampsia in Kelantan is higher than developed coun-

tries but appears to be much lower than that of neighbouring southern Thailand (6,7).

The death rate in eclampsia of 4.8 per cent resulted from a combination of several factors that show wide variability. Delay in referral because of socio-cultural reasons remains a formidable problem. The absence of medical retrieval teams in the state and inadequate resuscitation while in transit contributed to considerable deterioration in the haemodynamic state of the eclamptic mother.

Primigravidae are said to be 15 times more likely to develop pre-eclampsia⁸. They accounted for 70 per cent of all eclampsia cases in Sweden⁶. In our study, more than half were multiparous.

Two interesting features were noted in this study. The occurrence of convulsions for the first time in parous women who had remarried - indicating the immunological element in the aetiology of pre-eclampsia. The other feature is the increased risk of convulsions with high parity and increasing maternal age. This was similar to the findings of BoMoller⁶. The prevalence of residual hypertension in the older parous women may be pivotal in causing eclampsia in this high risk group. Renal disease, chronic hypertension and multiple pregnancy have been recognised as predisposing factors in the genesis of pre-eclampsia. All these were recognised in the profile of the patients in the study and cognizance must be taken of these factors during antenatal care. Evaluation of mean arterial blood pressure according to the observations of Page et al^{9,10} where second and third trimester readings are distinct entities may lead to earlier recognition of the disorder. Strict adherence to looking for a blood pressure of 140/90 mm Hg prior to starting treatment has been found to be unsatisfactory in our context. Although excessive weight gain was recorded in the course of the antenatal care of patients, it was not identified as an important sign of impending pre-eclampsia. Overweight women have been found to be three to seven times more likely to develop hypertension in pregnancy¹¹

The various complications seen in this study emphasises that the syndrome is characterised by functional derangement of nearly every organ system. A generalised vasospasm due to an increased sensitivity to vasoactive substances such as Angiotensin II and catecholamines^{12,13} produces its effects on the principal target organs. In such circumstances the management of pre-eclampsia should not come only under the purview of the obstetrician. The need for intensive and sometimes, invasive haemodynamic monitoring emphasises the importance of a team approach requiring the need for calling in the anesthesiologist, paediatrician and neurologist. Although a variety of anti-hypertensive drugs are available hydralazine and labetalol proved to be effective with little compromise to foetal sustenance^{4,14}. Urgent evaluation is needed in sudden onset of blindness in eclampsia^{3,15,16}. The occurrence of blindness in our series is a rare phenomenon and has been thought to be due to an intense vasospasm of the distal posterior cerebral arteries¹⁵.

Severe abruptio placentae causing increased perinatal morbidity, mortality and life-threatening postpartum haemorrhage should be suspected in all cases of acute fetal distress and sudden uncontrolled 'settling' of hypertension in the eclamptic patient¹⁷. The hypotension caused by this phenomena was dramatic in twelve cases.

A syndrome that includes haemolysis, elevated liver enzymes and low patient counts (HELLP Syndrome) described by Weinstein⁵ was seen in eight cases. Coagulopathy requiring the need for blood component replacements and invasive haemodynamic monitoring was explicit in all the cases. Advances in coagulation technology has led to a more comprehensive evaluation of the haemostatic system in eclampsia. Consumptive coagulopathy defect (DIVC) can be eclampsia¹⁹. Although a rapid decline in maternal mortality was reported in Kelantan over the last

deaths were directly attributable to DIVC.

Effective evaluation of haemodynamic status of the patient and appropriate pharmacotherapy will be vital in patients undergoing operative deliveries^{3, 18}. The operative delivery rate in our series was high. Morbidity and mortality rates would be reduced if attention is given to correction of dehydration, hypoxemia and acidemia prior to administration of anaesthesia to the eclamptic patient.

Inevitable termination of pregnancy in eclampsia is a common cause of prematurity and neonatal deaths. The perinatal mortality from eclampsia in this study was more than four times that of the hospital population and ten times that of the state.

It is evident here that there are two subgroups of eclamptics in Kelantan i.e. primiparous and multiparous. Term pregnancies, often in the parous women had a shorter period of subclinical pre-eclampsia¹⁹. Although a rapid decline in maternal mortality was reported in Kelantan over the last six years, the pattern of disease does not seem to show a decline of incidence. There is a need for continued surveillance of health care provided by the health delivery system together with improved patient education strategies. Formalised and adequate documentation with comprehensive analysis has been possible with the introduction of the National Indicator Approach of quality assurance activities since 1986. Tertiary health care providers in referral centres need to look at eclampsia from a multisystem disorder aspect and the need for a team approach in management to obtain optimum results.

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