Eclampsia – A Study of its Occurrence and Maternal Sequelae

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More than two hundred years ago, Alexander Hamilton described Eclampsia as 'a disease which is always attended with the utmost hazard, and frequently kills the woman like a fit of apoplexy'.

Although a rare disease in the developed countries where good antenatal care is available, it still remains an important cause of foetal and maternal morbidity and mortality, in the developing world. Eclampsia does not come as a bolt of lightning as the term implies, but is often insidious in onset in the form of pre eclampsia. Good antenatal care with early detection and treatment of pre eclampsia can prevent this serious condition which not only carries a high perinatal mortaltiy, but is also a major threat to the mother's life.

Materials and Methods

The patients in this study are from admissions to the University Obstetrics and Gynaecology Unit of the General Hospital, Galle, Sri Lanka, during the two year period October 1st 1980 to September 30th 1982. The criteria for diagnosis of Eclampsia were generalised convulsions, hypertension, gross oedema and albuminuria in pregnancy. All other convulsive states such as epilepsy, cerebro vascular accidents and neurological lesions were excluded

Results

(*a*) *Incidence*

Out of a total of 3441 deliveries, there were 13 Eclamptics, giving an incidence of 0.378%. Of the total number who delivered only 32% were primigravidae. Seven out of the 13 Eclamptics were primigravidae, giving an incidence of 0.636% compared with the 0.256% incidence in the multigravidae.

Table 1: Distribution of cases according to age and parity

Age / *Gravidity*	≤20 *Yrs.*	20-24 *Yer.*	25-29 *Yer.*	30-35 *Yrs.*	*Total*	%	*Average Age*	*Incidence*
Primi	2	3	1	1	7	54	21.7	0.636%
Multi	—	4	1	1	6	46	23.7	0.256%

(*b*) *Antenatal care*

Four cases had no antenatal care.

Four had limited care (less than 3 attendances at a midwife only clinic).

Five had good antenatal care (seen by a medical officer on 3 or more occasions).

Of the last category pre eclampsia had been detected in 4 of them. Two had refused admission while the other two developed post partum eclampsia following delivery by induction. The other case presented with severe pre eclampsia and developed intrapartum eclampsia, following induction of labour. None of the patients had been seen prior to 20 weeks of gestation.

(*c*) *Relation to gestational age*

Table II: Distribution of cases according to gestational age

Occurrence	*Period of gestation (in weeks)*					*Total*	%
	31	31-35	35-40	40	**Not known*		
Ante partum	—	1	2	—	4	7	53.8
Intra partum	—	1	1	1	—	3	23.1
Post partum	—	1	1	—	1	3	23.1

Mean gestational age = 32 weeks

* Clinical estimation of the gestational age (according to the fundal height) in the cases where the dates were not known:
< 31 weeks — 1
31-35 weeks — 3
36-40 weeks — Nil (Mean = 32 weeks)

(*d*) *Blood pressure, oedema and albuminuria*

Significant generalised oedema was present in all the cases. The highest recorded blood pressure was 210/140 mm Hg, and the lowest 130/90mm Hg. The mean diastolic pressure in the series was 114 mm Hg, with 9 out of the 13 cases having a diastolic pressure of 110 mm Hg or more.

Except the two cases of moderate to severe pre eclampsia admitted for induction, the rest had significant albuminuria on admission. Of the former two, one developed albuminuria during labour and the other, only with the onset of post partum eclampsia.

(*e*) *Convulsions*

10 patients (76.9%) had only the presenting fit which was quickly controlled. One patient had a history of convulsions at home but none since admission. One patient had two post partum fits occurring within 1/2 an hour of delivery. One patient had a total of seven convulsions occurring over a period of 5 1/2 hours. She was delivered within 5 hours of the first.

(*f*) *Drugs used*

The drugs used for the initial control of convulsions and for subsequent sedation varied according to the case and our previous experience. The drugs commonly used were Morphine, Sodium Amytal, Diazepam, Pethidine and Largactil (often intravenously and sometimes intramuscularly).

(*g*) *Mode of delivery*

Table III: Mode of delivery in relation to labour

Mode of delivery / *Labour*	*Normal vaginal*	*Forceps*	*Caesarian section*	*Total*
Spontaneous	4 + 1 twin delivery	3	1	10
Induced	2	1	1	4
Total	8	4	2	14
Percentage	57.1	28.6	14.3	

(*h*) *Foetal outcome*

Table IV: Foetal outcome in relation to birth weight

Birth Weight	**Still Births*		*Live Births*	*Neonatal deaths*	*Total foetal loss*	*Percentage foetal loss*
	A	*B*				
< 1.0 Kg	—	—	—	—	—	—
1.0 — 1.5 Kg	1	2	1	—	3	75
1.6 — 2.0 Kg	2	0	3	1	3	60
2.1 — 2.5 Kg	1	0	2	0	1	33
2.6 — 3.0 Kg	0	0	2	0	0	00
> 3.0 Kg	—	—	—	—	—	—
Total	4	2	8	1	7	50

Still Births A — Death in utero prior to admission to the unit
B — Death in utero after admission to the unit

Average birth weight in the series — 1.9 Kg.

(*i*) *Maternal complications*

Table V:

Complications	*Number of Cases*
Pulmonary oedema	01
Prolonged coma	01
Respiratory depression	01
Abruptio placentae	01
Acute renal failure	01
Hyper Pyrexia	Nil
Uncontrollable convulsions	Nil
Disseminated intravascular coagulation	Nil
Cerebro vascular accident	Nil
Trauma	Nil
Urinary tract infection	06
Chest infection	05

Three cases (23%) presented with major complications and one of them had pulmonary oedema, prolonged coma and respiratory depression.

(*j*) *Maternal mortality*

Nil

Discussion

Thirteen cases of Eclampsia were diagnosed during the two year period of study. The incidence of 0.378% is considerably higher than that reported by workers in the more developed countries.

Donald (1979) — In Queen Mother's Hospital, England

1964 — 1965 : 0.25%

1970 — 1971 : 0.11%

Wightman et al (1978) in Cardiff, Wales, 1965-1974: 0.072%

Although in the study the relative proportions of primigravidae to multigravidae were almost equal the actual incidence (which depends on their relative proportions in the total number of deliveries) was found to be more than two and a half times higher in the promigravida. Dawn and Sinha (1979) in India, found only 19 multigravidae out of a total of 141 cases of eclamptics. Wightman et al (1978) in Wales found that the incidence in primigravidae was more than 4 times that in multigravidae (0.136% and 0.031% respectively).

Since eclampsia is essentially a preventable condition, poor antenatal prophylaxis is the primary factor responsible for its occurence. This was well seen in this study where 61.5% of patients had no proper antenatal care, half of whom had no care at all. Even with proper antenatal care, some mothers refuse admission to hospital. As Dewhurst (1981) suggests this is due to either the mother being unable to understand the gravity as she has no symptoms, or she is unable to abandon the husband and children temporarily (due to various reasons). In our study, antepartum eclampsia remained the commonest presentation (53.8%) and all of them had their initial convulsion at home. The rest were equally distributed between the intrapartum and the post partum periods.

In countries where antenatal care has vastly improved, ante partum eclampsia has become relatively less common and post partum eclampsia more so.

Table VI: Occurrence of Eclampsia

Source	*Ante partum*	*Intra partum*	*Post partum*
Queen Charlotte's Hospital, London 1967-1969 (Dewhurst 1976)	1	8	17
Confidential Enquiry in the United Kingdom 1973-1975 (R.H.S.S.1979)	10	1	7
Bankura Sanmilani Medical College Hospital, India, 1975-1977 (Dawn and Sinha 1979)	104	13	22
Cardiff, Wales 1965-1974 (Wightman et al 1978)	14	17	12
Queen Mother's Hospital, England 1964—1971 (Donald 1979)	8	12	11
Present study 1980—1982	7	3	3

The stress of labour is thought to aggravate a moderate situation which then progresses to eclampsia. In our study it was noted that in the patients developing intra and post partum eclampsia the sedation was inadequate inspite of early warning signs. With early detection and treatment of pre eclampsia, and early delivery of cases with impending eclampsia, the ante partum onset of eclampsia could be prevented; but if the same vigilance is not continued during and soon after labour, intra and post partum eclampsia cannot be prevented.

Complications

(1) *Pulmonary oedema*

This results from a combination of hypertension, exhaustion, and fluid and electrolyte imbalance. All three factors were thought to be responsible in our patient who responded to large doses of frusemide. Gedekoh et al (1980) recorded 3 out of 52 eclamptics developing pulmonary oedema due to excessive intravenous fluid administration, while Dawn and Sinha (1979) had an incidence of 12.8% in their series of 141 cases. A specific myocardial lesion due to hypoxia, has also been incriminated (Lawson, 1982).

(2) *Prolonged coma and respiratory depression*

This is very sinister, and may follow a cerebro vascular accident or raised intracranial pressure and cerebral oedema following convulsions. The patient in this study who developed this complication had been given

intravenous Morphine, for the control of convulsions, and the condition was reversed with Lethidrone. Morphine is known to cause a raised intracranial pressure, and acidosis by respiratory depression. On account of this both Lawson (1982) and Donald (1979) advise against its use.

(3) *Abruptio placentae*

In this condition the maternal prognosis is serious as it was in our patient who went into a state of shock soon after delivery and had to be resuscitated with rapid blood transfusions. Because of their pre-existing hypovolaemia and haemoconcentration, eclamptics tolerate blood loss very badly. The hypovolaemia is related to the reduction in the capacity of the intravascular compartment by generalised vaso spasm (Lawson 1982). Hence replacement of fluid should be carefully monitored to prevent circulatory overload (Hibbard et Rosen 1977). Our patient developed convulsions only after resuscitation, by which time the blood pressure was noted to have risen to 160/110 mm. Hg. from 130/90 mm Hg. prior to delivery. In this case, injudicious intravenous infusions may have precipitated eclampsia.

(4) *Acute renal failure*

Oliguria is a common accompaniment of eclampsia, and reflects its renal effects. It needs no specific therapy immediately, the termination of pregnancy being the first priority as powerful diuretics such as frusemide reduce the blood volume further, and aggravates the impaired renal perfusion (Lawson 1982). Sometimes this can progress to acute tubular necrosis or massive cortical necrosis. One patient in this study presented, thus, with persistant oliguria and rising blood urea levels 48 hours after delivery. Since there was no response to Mannitol and Frusemide, she needed dialysis.

(5) *Hyperpyrexia*

Not seen in this study. In the absence of any infection it is thought to be due to anoxic changes in the temperature regulating centres in the midbrain (Lawson 1982). Since it can be detrimental to both heart and brain it requires urgent therapy. Dawn and Sinha (1979) reported an incidence of 9% of cases in their study developing a temperature of more than 105°F.

(6) *Uncontrollable fits*

Our patient who had 5 convulsions within 5 hours cannot be strictly included under this category because the first were soon controlled by the delivery of the baby. Various measures have been adopted in this

including intravenous Pentothal sodium. Chan and Dillikon in 1970 (as quoted by Lawson 1982) have reported a case where 17 fits had occurred within eight hours and paralysis with D-Tubocurrarine was necessary before they could be brought under control.

(7) *Disseminated intravascular coagulation*

The hypercoagulable state seen in a normal pregnancy seems to be exaggerated in pre eclampsia and eclampsia (Bonar et al 1971). This is probably due to the release of thrombo plastic substances which may arise from blood platelets or from the release of trophoblastic tissue and amniotic fluid into the uterine circulation. However the deposition of fibrin could be secondary to tissue damage following immunological reactions (Studd 1977). It is now believed that the coagulopathy which is seen in eclampsia and pre eclampsia is more a cause rather than an effect of the condition (Symonds 1979). In the present study there was no clinical evidence of this condition, which is said to be rare in Sri Lanka. Wightman et al (1978) had two cases out of 43 who developed clinical manifestations of this condition. Gedekoh et al (1980) recorded one death due to this condition and foumd 24 out of 46 eclamptics to have thrombocytopenia.

(8) *Cerebro vascular accidents and other neurological sequelae*

In our group of eclamptics, there were no cases with these complications which occur primarily due to the hypertension and secondarily due to the convulsions. Gedekoh et al (1980) found 9 of their 52 cases developing significant neurological sequelae, of which two died of cerebro vascular accidents.

(9) *Trauma due to convulsions*

There were no serious injuries in our patients. Gedekoh et al (1980) record an impressive list of complication including fractures, dislocations, amputation of tongue and even two car collisions!

(10) *Infections*

Inspite of prophylactic antibiotics being used routinely, there was a high incidence of chest infections (38%) and urinary tract infections (46%) in this study. Collection of secretions and hypostatic pneumonia, and aspiration of vomitus was responsible for the former while catheterization was the probable cause of the latter. All patients in this study had indwelling catheters for accurate estimation of urine output. One patient developed a lung abscess probably due to aspiration of vomitus.

Mode of delivery

Vaginal delivery was preferred in the unit, and 86% of the cases were delivered vaginally, and about one third of them required assistance with 'lift out forceps'. The principle was to first control the convulsions and induce labour after 12 - 24 hours sedation, if spontaneous labour was not established by then. This conservative regime was first suggested by Strogonoff about 50 years ago. He used morphine by injection together with Chloral and Bromide per rectum for heavy sedation. The fairly high maternal mortality was drastically reduced by this method. The principles of his management are still followed save that more effective and safer drugs are used and there is a tendency for earlier delivery. In our series only two patients were delivered by caesarian section. The first for an obstructed labour due to a dead foetus with shoulder impaction, and the second for foetal distress, where the baby was salvaged. In the past, caesarián section in eclampsia was associated with a high maternal mortality, perhaps because it was used as a last resort. However Menon (1961) in Madras, Lopez Llera (1967) in Mexico, and Crichton et al (1968) in Durban have shown that it is a relatively safe procedure. But all of them have shown that the maternal mortality remains the same whether the patient is delivered abdominally or vaginally. Only the foetal outcome is significantly improved.

Menon (1961) and Lopez Llera (1967) have shown that the outlook for both mother and baby is worse, the longer the delivery is delayed. Delivery should therefore be effected promptly, by whatever means, and this ensures the best prognosis for both mother and baby. In our series of 10 mothers who developed eclampsia before delivery, the mean time duration from presenting convulsion to delivery was 15 hours, the shortest time being 1/2 an hour and the longest 47 hours. (The patient who took 47 hours to deliver presented with one convulsion at a period of gestation of approximately 30 weeks, clinically. Death in utero occurred 18 hours later, with no further convulsions, and the eclamptic state dramatically subsided. Spontaneous labour was established after about 36 hours. The foetus weighed 1 kg at birth).

Maternal mortality

Although there were no maternal deaths in this series, it still remains a major cause of maternal mortality especially in the developing world (Lawson, 1982). During the period 1973-1975, the most frequent cause of maternal death in England and Wales was hypertensive disease of pregnancy of which 54% were due to eclampsia. Eleven out of these 21 cases of maternal deaths associated with eclampsia were due to cerebral haemorrhage (Confidential Enquiries into Maternal Deaths 1979).

Maternal mortality is highest with ante and intra partum eclampsia and also tends to be higher the shorter the period of gestation is (Dewhurst, 1981).

Maternal mortality rates reported by various workers are as follows:

Menon (1961) Out of 402 patients in Madras:	2.2%
Lopez Llera (1967) Out of 107 patients in Mexico :	10.3%
Lean et al (1968) Out of 90 patients in Singapore :	3.3%
Pritchard et Pritchard (1975) Out of 72 patients in Texas :	Nil
Wightman et al (1978) Out of 72 patients in Wales:	Nil
Dawn et Sinha (1979) Out of 55 patients in India :	9.0%
and Out of 86 patients :	17.5%
Gadekoh et al (1980) out of 52 patients in Pennsylvania :	5.8%

But as these studies were carried out under very different conditions and circumstances using different modes of management, these results are not strictly comparable.

Summary

Thirteen cases of eclampsia presenting over a two year period were studied with reference to their occurrence and maternal sequelae. The relatively high incidence found and the fact that ante partum eclampsia was the commonest mode of presentation reflects poor antenatal prophylaxis. Three patients presented with serious complications and these were discussed together with other possible complications. Inspite of prophylactic antibiotics, chest infections and urinary tract infections were common. Vaginal delivery after the convulsions were controlled, was preferred to abdominal delivery. There were no maternal deaths.

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