Eclampsia—an 11-year experience

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A retrospective study of all deliveries from 1983 to 1993 at the Tsan Yuk Hospital was performed to evaluate the incidence and outcome of eclampsia. Eclampsia occurred in 12 patients. The incidence was approximately 2 per 10 000 deliveries and was relatively constant during the study period. Most convulsions occurred during the intrapartum (41.7%) and post-partum (50.0%) periods, and about half of them had no warning signs. There were no maternal or perinatal deaths. Prophylactic anticonvulsants should be given to all patients with warning signs; however, eclampsia is not always preventable.

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Introduction

Eclampsia is a major cause of maternal and perinatal mortality and morbidity, accounting for 8.5% of all maternal deaths in the United Kingdom between 1991 and 1993.[1] Eclampsia is the occurrence of convulsions in association with signs and symptoms of pre-eclampsia. The syndrome of pre-eclampsia can affect all maternal organ systems and is usually detected by the presence of hypertension, proteinuria, and oedema during pregnancy. Hypertension and proteinuria, however, are not the only or necessarily the most important signs of pre-eclampsia. The occurrence of such signs are determined by the site and extent of involvement of the maternal organs.[2] Eclampsia can thus occur without such signs, and is not always preventable.

In developed countries, the incidence of eclampsia has fallen considerably.[3-5] This has been attributed to improvements in antenatal care facilities and the management of pre-eclampsia (Corkhill's hypothesis). With the rapid development of Hong Kong over the past 20 years, we expected that the incidence of eclampsia would have decreased. We also expected that the pattern of the disease would have changed. In this retrospective study, patients with eclampsia who were treated at the Tsan Yuk Hospital between 1983 and 1993 were reviewed. The clinical manifestations, use of anticonvulsant prophylaxis, and maternal and neonatal outcomes were evaluated. The data were also compared with those from a previous study performed between 1976 and 1981 at the same hospital.[6]

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Subjects and methods

All deliveries at the Tsan Yuk Hospital from 1983 to 1993 were reviewed. Eclampsia was defined as the occurrence of generalised convulsions during pregnancy, during labour, or within 7 days of delivery, that were not caused by epilepsy or other convulsive disorders. Cases of eclampsia were identified from the hospital statistical records by the coding 'eclampsia'. The maternity records were then retrieved and reviewed to verify the diagnosis. Data were collected on antepartum, intrapartum, and post-partum care; the presence of symptoms of imminent eclampsia; the presence of hypertension and proteinuria; the eclamptic episode; the use of prophylactic anticonvulsants; and the maternal and perinatal outcomes.

Results

During the 11-year period from January 1983 to December 1993, there were 53 514 deliveries at the Tsan Yuk Hospital. Twelve patients developed eclampsia—an incidence of approximately 2 per 10 000 deliveries. The incidence was relatively constant during the study period, with no more than three cases each year (ranging from none to three per year).

Among the 12 patients with eclampsia, four (33.3%) were Vietnamese refugees and eight (66.7%) were Hong Kong Chinese citizens. All had received antenatal care at the Tsan Yuk Hospital from the first or early second trimester. Ten patients were nulliparous and two

patients had had a previous delivery. Their ages ranged from 17 to 31 years and the mean age was 23.9 years (Table 1). One Vietnamese patient had a convulsion at the detention centre before the onset of labour. The other patients had convulsions after admission to the hospital. Five patients had convulsions during labour and six patients had convulsions after delivery. The mean gestational age was 37 weeks.

The clinical features of the patients are listed in Table 1. Patient A had a convulsion at the detention centre before the onset of labour (at 36 weeks of gestation) and without any clinical symptoms. The patient's blood pressure was normal and there was no proteinuria. After admission, combined induction was performed for maternal eclampsia, and emergency lower-segment caesarean section was performed for failure to progress in labour.

Patients B to F developed eclampsia during the intrapartum period. Patients B, C, and D were known to have raised blood pressure prior to eclampsia. Patient B also suffered from proteinuria and headache, which are signs of imminent eclampsia. Patients C and D had only mildly elevated blood pressures, with a diastolic blood pressure of 90 mm Hg. Patients E and F did not show any signs or symptoms prior to eclampsia. None of the five patients received prophylactic anticonvulsant therapy. Lower-segment caesarean section was performed for three of the patients: one for foetal distress and two for cephalopelvic disproportion.

Patients G to L developed eclampsia during the post-partum period. All episodes occurred within 24 hours of delivery. Five patients (patients G to K) had premonitory symptoms and signs of eclampsia. Only three of them, however, were given prophylactic anticonvulsant therapy. Patients G and H received diazepam and patient I received a 'lytic cocktail' (pethidine, promethazine, and chlorpromazine). The choice of anticonvulsant mainly depended on the obstetricians' preference. Nevertheless, these three patients developed eclampsia despite the use of anticonvulsants. Eclampsia developed in patient L without any warning signs.

Among the 12 patients, four (33.3%) patients had normal blood pressure prior to the eclamptic fit and two (16.7%) patients had a diastolic pressure of 90 mm Hg. Significant proteinuria (ie proteinuria ≥300 mg/day or an albustix of ≥1+ on two occasions more than 4 hours apart) was present in only four (33.3%) patients. All patients had severe hypertension (ie blood pressure ≥160/110 mm Hg). Only three (25%) patients had symptoms suggestive of imminent eclampsia (headache or vomiting).

There were no maternal or perinatal deaths. Maternal complications included postnatal anaemia, postnatal pyrexia, wound haematoma and chest infection. Caesarean section was performed in seven (58%) patients. All 12 patients were followed up in our postnatal clinic and their blood pressures returned to normal. There were

Table 1. Summary of clinical features of patients with eclampsia

Period	Patient/ age (y)	Parity	Gestation (wk)	Antenatal problem	Maximum blood pressure (mm Hg)
Antepartum	A/17	0	36	—	115/70
Intrapartum	B/23	0	38	Induced labour, pre-eclampsia	150/110
	C/25	0	38	Intrapartum HT[§]	130/90
	D/31	0	38	Gestational HT	150/90
	E/28	0	39	—	120/80
	F/30	0	41	—	130/85
Post-partum	G/26	1	33	IUGR[‖], pre-eclampsia	200/120
	H/26	0	32	Pre-eclampsia	160/110
	I/21	0	35	Gestational HT	180/130
	J/20	0	37	Pre-eclampsia	170/110
	K/22	1	38	—	180/100
	L/18	0	40	—	120/85

*LSCS lower-segment caesarean section
†AGA appropriate for gestational age
‡AS Apgar score
§HT hypertension
‖IUGR intrauterine growth retardation
¶CS caesarean section
**SGA small for gestational age
††NSD normal spontaneous delivery

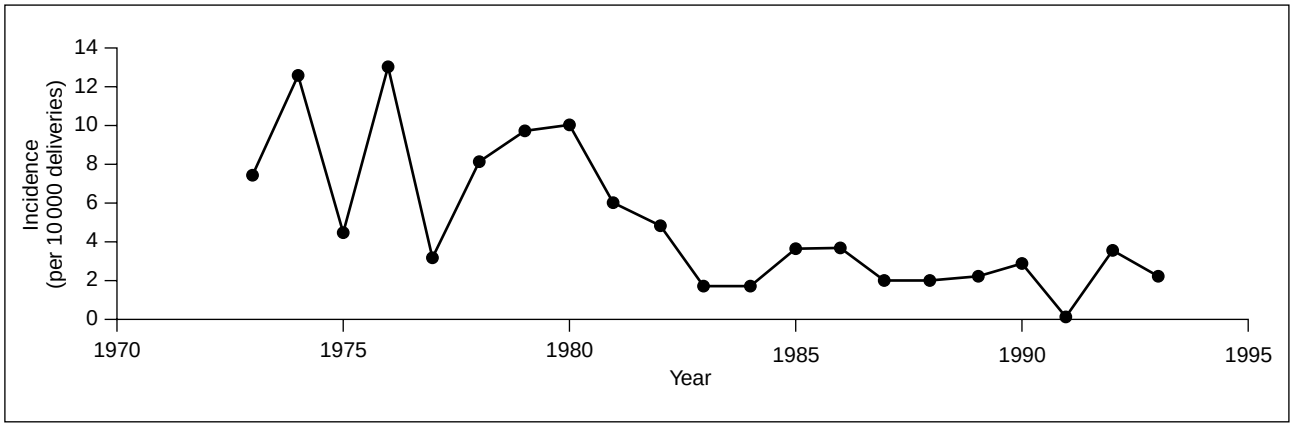

Fig. The annual incidence of eclampsia from 1973 to 1993 at the Tsan Yuk Hospital

no significant perinatal complications. One baby was small for gestational age due to intrauterine growth retardation. The mean birthweight of the other babies was 3081 g (range, 1920-3670 g).

Discussion

The incidence of eclampsia in our series was 2 per 10 000 deliveries. This was similar to the overall incidence of eclampsia in Hong Kong[7] (2.6 per 10 000) and the reported incidences in other developed countries.[3,8,9] The incidence of eclampsia per 10 000 deliveries is 4.9 in the United Kingdom,[3] 4.3 in the United States,[8] and 2.7 in Sweden.[9] The incidence from this review is lower than that reported by our unit in 1984 (10 per 10 000 deliveries).[6] We noticed that the annual incidence decreased from 1973 to 1983 but was relatively constant from 1983 to 1993 (Table 2; Fig). Because of this and the fact that our statistical system became computerised in 1994, the 11-year period was chosen for this study.

Nulliparity was one of the major risk factors identified in our series. Ten (83.3%) of the 12 patients were nulliparous and only two (16.7%) were multiparous (Table 2). About 50% of all women delivered at the Tsan Yuk Hospital during the study period were nulliparous; thus, there was a significantly higher incidence of eclampsia in nulliparous women. Teenage pregnancy was a risk factor recognised in our previous study.[6] This was not observed in the present series.

In this series, one (8.3%) patient had eclampsia during the antenatal period, five (41.7%) had eclampsia while in labour, and six (50.0%) had eclampsia during

Maximal proteinuria (albustix or g/d)	Symptoms before seizure	Prophylactic anticonvulsant	Maternal outcome	Perinatal outcome
—	—	—	LSCS*, postnatal anaemia	AGA†, AS‡ $7^{1}10^{5}$
2+	Headache	—	LSCS	AGA, AS $10^{1}10^{5}$
—	—	—	LSCS, postnatal anaemia, fever	AGA, AS $7^{1}10^{5}$
—	—	—	Vacuum extraction	AGA, AS $5^{1}9^{5}$
—	—	—	LSCS	AGA, AS $3^{1}4^{5}$
—	—	—	Vacuum extraction, postnatal anaemia	AGA, AS $4^{1}9^{5}$
4+	—	Diazepam	Classical CS¶, wound haematoma	SGA**, AS $5^{1}8^{5}$
10 g/d	Headache	Diazepam	LSCS	AGA, AS $5^{1}7^{5}$
—	Vomiting, headache	'Lytic cocktail'	LSCS	AGA, AS $8^{1}10^{5}$
3+	—	—	NSD††	AGA, AS $8^{1}10^{5}$
—	—	—	NSD, chest infection	AGA, AS $10^{1}10^{5}$
—	—	—	NSD	AGA, AS $10^{1}10^{5}$

Table 2. Distribution of cases and the incidence of eclampsia from 1973 to 1993 at the Tsan Yuk Hospital

Year	Primiparous	Multiparous	Overall*	Year	Primiparous	Multiparous	Overall*
1973	4	1	7.5	1983	1	0	1.6
1974	7	2	12.5	1984	1	0	1.7
1975	2	1	4.6	1985	2	0	3.5
1976	6	3	12.9	1986	1	1	3.6
1977	2	0	3.2	1987	1	0	1.9
1978	5	0	8.0	1988	1	0	1.8
1979	5	1	9.6	1989	1	0	2.1
1980	7	0	9.9	1990	0	1	2.6
1981	3	1	6.0	1991	0	0	0.0
1982	3	0	4.7	1992	1	0	3.5
Total	44	9	7.9	1993	1	0	2.0
				Total	10	2	2.2

* overall incidence: per 10 000 deliveries

the post-natal period (Table 3). In comparison with the data obtained between 1976 and 1981, antenatal eclampsia has markedly decreased. This may be due to an increased effectiveness in the antenatal care and screening, as well as earlier intervention. There was, however, a marked increase in the proportion of cases occurring during the intrapartum period. None of the five affected patients received prophylactic anticonvulsants. Four (80%) of the patients had no warning signs or only mildly elevated blood pressure. The other patient had signs suggestive of impending eclampsia, but prophylactic treatment was not given. Atypical presentations are thus becoming a proportionately greater problem as eclampsia with a classic presentation is prevented. They also explain the relatively stable incidence of eclampsia in recent years.

The majority of cases of eclampsia occurred in the postnatal period. As reported in our previous study,[6] relaxation in the obstetrician's vigilance after a safe delivery should be considered as a contributing factor. Adequate monitoring and treatment are still necessary after delivery. One should bear in mind that a pre-eclamptic patient is still prone to eclampsia within 24 to 48 hours after delivery and effective treatment has to be continued. Moreover, it should be noted that eclampsia can occur despite the use of anticonvulsant prophylaxis.

Table 3. Distribution of cases of eclampsia in relation to labour

Period	No. of cases (%)	
	1976-1981	1983-1993
Antepartum	11 (34.4)	1 (8.3)
Intrapartum	1 (3.1)	5 (41.7)
Post-partum	20 (62.5)	6 (50.0)
Total	32	12

Further reductions in the incidence of eclampsia may be possible by increased vigilance to premonitory signs, provided that prophylactic anticonvulsants are given. In our hospital, prophylactic anticonvulsants are given to all patients having 'severe' pre-eclampsia. The risks are assessed individually according to the presence of signs and symptoms of impeding eclampsia, significant proteinuria, severe hypertension, and/or biochemical abnormalities. In this series, 6 of 12 patients presented with severe hypertension, significant proteinuria, or symptoms such as headache and vomiting. Prophylactic anticonvulsants, however, were not given to all six patients: only to three of them. If prophylactic anticonvulsants had been given, these three cases (25% of all cases) of eclampsia might have been prevented. Since the use of prophylactic anticonvulsants was not included in our database, their effectiveness in preventing eclampsia cannot be evaluated in this study. Magnesium sulphate has been shown to be effective in preventing further fits for those patients with eclampsia.[10] Further studies are needed to see whether it is effective in preventing eclampsia in patients without preceding convulsions.

The lytic cocktail, consisting of pethidine 25 mg, promethazine 25 mg, and chlorpromazine 25 mg, should no longer be used as prophylactic treatment. It has been shown that this mixture of drugs may induce seizures rather than prevent them.[11] The lytic cocktail has therefore been withdrawn from our treatment protocol.

As already noted, six patients (accounting for 50% of all cases) with eclampsia did not experience warning signs. This made prophylactic treatment difficult. Therefore, new screening and diagnostic tests for features other than hypertension and proteinuria are necessary for the prevention of eclampsia which has an atypical presentation.

There were no maternal deaths in our series. None of the patients suffered from major complications such as renal failure, pulmonary oedema, neurological deficit, or liver failure. This may be due to the small number of patients in our study—maternal mortality has been estimated to be approximately 1 in 50.[3]

Conclusions

In conclusion, eclampsia complicated about 1 in 5000 deliveries in the present series. Nulliparity was identified as a risk factor. Screening by measurement of blood pressure and analysis of urine could prevent most episodes preceded by hypertension and proteinuria, provided that timely intervention and prophylactic measures are taken. There is still room for improvement, as prophylactic anticonvulsants were not given in this series to all patients with these warning signs. Eclampsia is not always preceded by the common warning signs. Therefore, eclampsia is not always preventable.

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