

HYPERTENSIVE ENCEPHALOPATHY IN ECLAMPSIA — A CASE REPORT

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SUMMARY

A case of hypertensive encephalopathy in eclampsia is described. Complete recovery from the neurological deficits took three and a half weeks.

INTRODUCTION

Eclampsia is not an uncommon problem in our part of the world. Among the complications that can occur is hypertensive encephalopathy where recovery is usually complete and the immediate prognosis is good. Here we present and discuss such a case.

CASE HISTORY

A 27 year old primigravida, was referred to the University Hospital after having three episodes of fits at home and seven further episodes at a District Hospital. She was then 33 weeks pregnant with previous antenatal care in an estate clinic. There was no known episode of raised blood pressure, epilepsy or head injury prior to admission. Examination revealed a drowsy patient who was restless. The pupils were equal and reactive and the blood pressure was 160/100 mm Hg. There was no pallor. No abnormality was detected in the cardiovascular, respiratory and nervous systems. Abdominal examination revealed a pregnant uterus of 30 weeks gestational size with a single fetus

in longitudinal lie and cephalic presentation. The fetal heart was heard beating regularly at 140 beats per minute. The patient was sedated with intravenous Valium and a Nepresol (Apresoline) drip was started, following which she was commenced on parenteral Ampicillin. Chest X-ray was within normal limits. Vaginal examination done when the patient was heavily sedated revealed a tubular cervix. The station of the presenting part was high.

Preparations for Caesarean section delivery was made.

During the pre-anaesthetic induction phase the patient was noted to be confused and having hallucinations. Induction of anaesthesia was uneventful and the blood pressure was maintained around 120mm Hg systolic. A male baby weighing 1200gms with an Apgar score of 2/7 was delivered.

The Caesarean section and reversal of anaesthesia was uneventful.

The patient was observed in the Intensive Care Unit and extubated the next day. She was conscious and rational then, thus she was transferred to the Labour Ward for observation. The following day she was noted to have a blank look and was unable to answer any questions. The blood pressure then was 140/100mm Hg. The neurological deficit noted was quadriplegia of varying degrees. The plantars were down going and the fundi appeared normal. She was subsequently started on oral Aldomet (methyl Dopa). She was able to answer simple questions on the 3rd post-operative day. Over the next two weeks she began to improve, with increase in motor power in the upper limbs and good level of consciousness. Motor power returned to the lower

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limbs starting with the left lower limb on the 12th post-operative day to the right lower limb on the 17th post-operative day. There was no sensory loss noted. CT scan done during this period was normal.

On discharge from the ward on the 25th day she was able to walk unaided. Her blood pressure was 110/80mm Hg without any antihypertensives.

On follow up in the post-natal clinic a month later her blood pressure and neurological status were normal.

DISCUSSION

In this country eclampsia is still an important cause of perinatal and maternal mortality despite the significant advances in treatment and better health services.

The incidence of eclampsia in a study at the University Hospital, Kuala Lumpur, was 1 : 476 deliveries.¹ The maternal mortality in this series was 6.3% and 2/3 of these cases had hypertensive encephalopathy but died of cerebral and cardiovascular complications.

In this patient the diagnosis was made from the transient nature of the neurological deficits and a normal CT scan. Hypertensive encephalopathy can occur in pre-eclampsia, superimposed on essential hypertension, and give rise to generalised or focal cerebral symptoms.²

The other interesting feature in this case is the presence of hallucinations before the induction of anaesthesia. Hallucinatory states have been noticed

in 10 percent of eclampsia patients.³ Psychosis in eclampsia usually affects young primiparas after they have had a series of convulsions⁴ as noted in this case.

The fundi in our patient did not reveal the presence of papilloedema, haemorrhages or exudates, which can occur in hypertensive encephalopathy and thought to be due to cerebral oedema.

Eclampsia can recur in about 2 percent of cases in subsequent pregnancies.⁵ This patient has been advised on contraception and early antenatal care in her subsequent pregnancy, so that, if pre-eclampsia should develop, early treatment can prevent recurrence of eclampsia.

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