Maternal deaths from neurological complications of hypertensive crises in pregnancy

A. M. RICHARDS, J. MOODLEY, M. R. R. BULLOCK, J. W. DOWNING

Summary

Fourteen maternal deaths from eclampsia or severe pre-eclampsia where disturbed cerebral function, as evidenced by prolonged unconsciousness, was given as the main cause of death are reviewed. Prolonged duration of seizures, hypotensive/hypoxic episodes, cerebral oedema and intracranial haematomas were most frequently identified as causative agents in the development of cerebral dysfunction. Failure to maintain an airway and iatrogenically induced hypotension were the two most important contributory factors to the patients' deaths. Management recommendations to prevent this type of maternal death are given.

Results

Fourteen maternal deaths were attributed to eclampsia or severe pre-eclampsia during the 2-year period 1 July 1983 to 30 June 1985 at this hospital were reviewed.

For purposes of the study eclampsia was defined as convulsions associated with proteinuric hypertension, and severe pre-eclampsia was diagnosed when symptoms such as headache, epigastric pain, blurred vision and hyperreflexia with clonus were associated with proteinuric hypertension. All patients had proteinuria of 2+ or more and a diastolic blood pressure level of 110 mmHg or higher on admission to hospital. They were all managed according to a standardised protocol described elsewhere. Briefly, this included prevention or termination of convulsions with magnesium sulphate, lowering of blood pressure with dihydralazine and delivery by caesarean section when vaginal delivery was not imminent (within 6 hours of admission). Persistence of a depressed consciousness level for longer than 6 hours after delivery was considered an indication for cerebral CT. When raised intracranial pressure (ICP) was suspected on CT and clinical features, the patient was managed in the intensive care unit where measures were undertaken to reduce ICP. Patient details are given in Table I.

When permission was granted autopsy was performed, and in certain instances detailed neurohistological examination was undertaken.

Patients and methods

King Edward VIII Hospital, acting as an obstetric referral centre for approximately 8 million people, delivers on average 17,000 patients a year. Maternal deaths due to eclampsia and severe pre-eclampsia during the 2-year period 1 July 1983 to 30 June 1985 at this hospital were reviewed.

Pregnancy-induced hypertension (PIH) is common among the black population in Natal, and because of inadequacies in antenatal care this condition frequently progresses to eclampsia. Figures for 1983 from King Edward VIII Hospital in Durban indicate the development of eclampsia in approximately 9 out of every 1,000 patients delivered, with a maternal mortality rate of 10%.

Prolonged unconsciousness subsequent to eclamptic seizures carried a particularly poor prognosis in our institution; before 1982 up to 50% of such patients died. Because of postmortem studies it was previously thought that intracerebral haematomas were the most common cause of death in these patients. However, recent work with cerebral computed tomography (CT) has demonstrated cerebral oedema rather than haematoma in the majority of eclamptic patients dying from cerebral dysfunction.

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Discussion

The development of cerebral oedema subsequent to eclamptic seizures has been demonstrated on CT by many authors. Oedema formation may be related to the generalised vasculopathy which has been demonstrated histologically in eclamptic patients (D. I. Graham — personal communication). Vessel wall damage would allow for transudation of plasma proteins into the brain substance, with both blood pressure peaks and
<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yrs)</th>
<th>Parity</th>
<th>Duration of fits</th>
<th>BP on admission (mmHg) (proteinuria)</th>
<th>Hypotensive episode</th>
<th>Hypoxic episode</th>
<th>Sedatives in addition to routine medication</th>
<th>CT</th>
<th>Autopsy findings</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>17</td>
<td>PO</td>
<td>12 h</td>
<td>250/150 (4 +)</td>
<td>Yes</td>
<td>Yes</td>
<td>Valium</td>
<td>Not done</td>
<td>Cerebral oedema; subcapsular haematoma and centrilobular necrosis of liver; abruptio placenta; intracerebral haemorrhage; cerebral oedema; subcapsular haematoma of liver; acute tubular necrosis</td>
<td>Cardiac arrest: heavy sedation; oedematous cords and supine position; airway not protected Coagulopathy</td>
</tr>
<tr>
<td>2</td>
<td>35</td>
<td>P5</td>
<td>10 min</td>
<td>170/140 (3 +)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Large haemorrhage into ventricles</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>18</td>
<td>P0</td>
<td>8 h</td>
<td>180/120 (4 +)</td>
<td>Yes</td>
<td>Yes</td>
<td>Valium</td>
<td>Not done</td>
<td>Cerebral oedema; subcapsular haematoma and centrilobular necrosis of liver; acute tubular necrosis of kidney</td>
<td>Respiratory arrest: excessive postoperative sedation</td>
</tr>
<tr>
<td>4</td>
<td>24</td>
<td>P0</td>
<td>10 min</td>
<td>200/110 (4 +)</td>
<td>—</td>
<td>—</td>
<td>Valium</td>
<td>Occipital oedema</td>
<td>Shock lung; acute tubular necrosis of kidneys</td>
<td>Died from renal failure 7 days after delivery</td>
</tr>
<tr>
<td>5</td>
<td>19</td>
<td>P1</td>
<td>Pre-eclampsia</td>
<td>210/140 (3 +)</td>
<td>Yes</td>
<td>—</td>
<td>Not done</td>
<td>No abnormality (done 4 hrs after cardiac arrest)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>18</td>
<td>P0</td>
<td>Pre-eclampsia</td>
<td>190/120 (4 +)</td>
<td>Yes</td>
<td>—</td>
<td>Not done</td>
<td>Haematoma, cerebral haematoma, occipital lobe oedema</td>
<td>Airway not protected; pulmonary oedema</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>40</td>
<td>P2</td>
<td>2 h</td>
<td>170/115 (3 +)</td>
<td>—</td>
<td>—</td>
<td>Valium</td>
<td>Diffuse hypoxic ischaemic brain damage</td>
<td>Acute tubular necrosis of kidneys</td>
<td>Nephrol infusion after 20 min, BP 60/0</td>
</tr>
<tr>
<td>8</td>
<td>23</td>
<td>P0</td>
<td>Pre-eclampsia</td>
<td>160/120 (4 +)</td>
<td>Yes</td>
<td>Yes</td>
<td>Valium</td>
<td>No abnormality</td>
<td>Cardiac arrest: magnesium sulphate 4 g given intravenously as a rapid bolus</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>35</td>
<td>P4</td>
<td>4 h</td>
<td>210/140 (4 +)</td>
<td>—</td>
<td>—</td>
<td>Valium</td>
<td>Large brainstem haemorrhage</td>
<td>Haemorrhage probably consequent to hypertension</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>20</td>
<td>P0</td>
<td>18 h</td>
<td>160/110 (4 +)</td>
<td>—</td>
<td>—</td>
<td>Valium</td>
<td>Diffuse cerebral oedema</td>
<td>Convulsions for 18 h</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>15</td>
<td>P0</td>
<td>36 h</td>
<td>230/130 (4 +)</td>
<td>—</td>
<td>—</td>
<td>Valium</td>
<td>—</td>
<td>Large intracerebral haematoma; large intrapulmonary haemorrhage</td>
<td>Concealed pregnancy; transport difficulties; prolonged uncontrolled seizures; coagulopathy</td>
</tr>
<tr>
<td>12</td>
<td>18</td>
<td>P0</td>
<td>4 h</td>
<td>170/115 (4 +)</td>
<td>Yes</td>
<td>Yes</td>
<td>Valium</td>
<td>Occipital oedema</td>
<td>Brain oedema; acute tubular necrosis of kidneys</td>
<td>Cardiac arrest: airway not protected; heavily sedated</td>
</tr>
<tr>
<td>13</td>
<td>20</td>
<td>P1</td>
<td>10 min</td>
<td>200/140 (?)</td>
<td>Yes</td>
<td>—</td>
<td>Valium</td>
<td>Diffuse hypoxic brain damage</td>
<td>Multiple hypotensive agents, prolonged hypotension</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>20</td>
<td>P0</td>
<td>?</td>
<td>160/110 (?)</td>
<td>—</td>
<td>—</td>
<td>Valium</td>
<td>Occipital oedema</td>
<td>Pneumonia and septicaemia; cerebral oedema; acute tubular necrosis of kidneys</td>
<td>Labour induced; intrauterine infection, septicaemic death</td>
</tr>
</tbody>
</table>
seizures exacerbating the process. Since the brain is enclosed within the rigid, bony cranial, any swelling due to oedema will result in an elevation of intracranial pressure. As a consequence, a concomitant rise in mean arterial pressure is necessary to maintain adequate cerebral perfusion. If the blood pressure is overenthusiastically lowered or intracranial pressures continue to rise, cerebral perfusion will be jeopardised, resulting in ischaemic/hypoxic brain damage and ultimately the death of the patient.

We did not find fundoscopic diagnosis of papilloedema to be a useful indicator of raised intracranial pressure, since in the majority of patients restlessness and irritability made fundoscopy impossible or inaccurate. In addition, the possibility of precipitating further seizures was high. For these reasons, and the danger of tentorial herniation (coning), lumbar puncture is contraindicated in such patients.

Recurrence seizures are known to result in hypoxic brain damage. We have recently reviewed cerebral CT done on 43 unconscious eclamptic patients; cerebral oedema was demonstrated with increasing severity proportional to the duration of seizures (unpublished observations). As the history was often inadequate it was impossible to correlate number of seizures to development of cerebral oedema, but seizures were commonly prolonged in our series of maternal mortality, suggesting that cerebral oedema was an important contributory factor in the death of these patients. Seizures should therefore be prevented or controlled as soon as possible.

The blood pressure is very labile in both eclamptic and severe pre-eclamptic patients and appears to be particularly sensitive to hypotensive agents. Seven patients in our series (50%) suffered episodes of hypotension and/or hypoxia. Marked hypotension developed in 6 patients, the fall in blood pressure being related to hypotensive agents in 2, to temporary cardiac arrest in 3, and to respiratory arrest in the remaining patient. Cardiac arrest followed magnesium sulphate toxicity in 1 patient, and in 2 patients was related to a combination of heavy sedation, partial airway obstruction and possibly supine hypotension. In both instances the arrest occurred within minutes of the patients' being placed supine on the operating table. A seventh patient (case 5), suffered a marked anoxic episode related to pulmonary oedema before death.

The normal brain possesses a complex series of autoregulatory mechanisms to ensure a constant rate of cerebral perfusion despite fluctuations in mean arterial blood pressure between extremes of approximately 50 and 150 mmHg. These mechanisms are known to be defective after a variety of causes of brain damage such as head injury, subarachnoid haemorrhage and prolonged seizures. Our findings of severe hypoxic/ischaemic brain damage would suggest that cerebral autoregulation is impaired or lost in patients with severe PIH. In this condition it is therefore important to lower blood pressure slowly and in a controlled fashion; it is suggested that the diastolic blood pressure should not be lowered by more than 30 mmHg. Drugs should be administered in small intermittent dosages or by controlled infusion, and continuous intra-arterial pressure monitoring is advantageous. Combinations of hypotensive agents should be avoided since they may have a compound effect. Postural hypotension would have a similar result, therefore patients with severe pre-eclampsia should be nursed on their sides or tilted with the aid of a pelvic wedge.

Where cerebral perfusion is endangered, optimal oxygenation is required. Heavily sedated eclamptic patients cannot maintain their own airway, particularly if nursed supine, thus allowing for partial airway obstruction by oedematous glottic tissues. Aspiration of acid stomach contents is also a danger, therefore the airway should be timeously protected. Neurosurgeons and neuro-anesthetists generally agree that the transient rise in blood pressure at the time of endotracheal intubation of the well-prepared patient is less damaging than prolonged hypoxia.

Intracerebral haematomas were present in 29% of cases (4 out of 14) in our series. Haematomas are thought to develop after rupture of small arterioles at very high blood pressures. Two of the 4 patients with intracerebral haematomas in our series had a marked clotting disorder and developed haematoma elsewhere in the body. The contribution of coagulopathy to the development of intracerebral haematomas is unclear.

Permanent neurological defects have remained in only 2 surviving eclamptic patients managed at King Edward VIII Hospital during the period of this study. An intracerebral haematoma produced hemiplegia in a member of staff who developed severe pre-eclampsia at 33 weeks' gestation. A second, unbooked, patient remains in a vegetative state following prolonged eclamptic seizures and delivery of a macerated still-born infant. Retention of the placenta may have resulted in postpartum haemorrhage and a fall in blood pressure. CT on admission to hospital demonstrated severe cerebral oedema.

**Recommendations**

We make the following recommendations for the management of patients with eclampsia and severe pre-eclampsia:

1. Continuous monitoring of blood pressure, pulse rate, ECG and central venous pressure is required as the cardiovascular system is extremely labile in this condition and can deteriorate in seconds.

2. The airway should be maintained and protected. Any patient with a Glasgow Coma Scale of less than 9 should be intubated. Nursing staff at peripheral clinics should be taught how to position an unconscious patient, insert an oral airway and administer oxygen.

3. An arterial partial pressure of oxygen of at least 100 mmHg should be maintained. Mechanical ventilation may be necessary.

4. Blood pressure should be carefully and slowly lowered, the diastolic pressure should be lowered by not more than 30 mmHg in order to maintain cerebral perfusion.

5. Seizures should be prevented or terminated as soon as possible. Efficient transport facilities must be available and personnel at peripheral clinics should be capable of administering anticonvulsants.

6. The fetus should be delivered within 6-12 hours of admission; caesarean section is often indicated. General anaesthesia, administered by a skilled anaesthetist, is recommended. Where these facilities are not available epidural anaesthesia would be adequate, provided a hypotensive episode is prevented with sufficient intravenous 'pre-loading' and coagulopathy is excluded by estimation of crude clotting time, fibrinogen levels and platelet counts.

7. An eclampsia team should be organised, since the problems developed by these patients are multifactorial. Personnel experienced in the management of these patients need to work together in order to improve patient outcome.

While prevention of PIH and eclampsia must await an understanding of its aetiology, improvement in antenatal care together with active management of the disease when it develops will improve the maternal prognosis.

We would like to thank Mrs S. Moodley for typing this manuscript, Professor J. van Dellen and the neurosurgical team at Wentworth Hospital and the Natal Provincial Administration Metropolitan Ambulance Service.

**REFERENCES**


Hypertrophic cardiomyopathy in infancy and childhood

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Summary

Hypertrophic cardiomyopathy (HCM) presented in 10 children under 2 years of age (group 1) and in 5 between 3 and 8 years (group 2). The clinical, ECG, chest radiographic and echocardiographic features are reviewed and prognosis over a mean follow-up period of 3.5 years is reported. Patients in group 1 had more symptoms and 7 had evidence of heart failure at some stage; all had ECG abnormalities. Group 2 patients presented with murmurs and only 1 had heart failure. Medical management of these patients is discussed and the importance of accurate diagnosis stressed, since HCM may have a poor prognosis in childhood; 3 out of 15 patients have died.

Hypertrophic cardiomyopathy (HCM) is a disease of cardiac muscle characterised by hypertrophied and disorganised myocytes. The changes occur predominantly in the interventricular septum, and less frequently in the ventricular walls and papillary muscles. Echocardiography has demonstrated that a large number of patients have either mild or no obstruction to left ventricular (LV) outflow under basal conditions. The condition was initially defined in adults and thought to be uncommon in childhood. Subsequently, HCM was recognised to have a particularly poor outcome in younger patients, although the prognosis in infants remains unclear.

The increased utilisation of echocardiography at Red Cross War Memorial Children's Hospital over the past 5 years, and the distinctive appearance of HCM on an echocardiogram, has stimulated increased recognition of this condition. Experience of HCM in all children presenting to the Cardiac Unit of this hospital over the past 8 years is described.

Patients and methods

Records of all patients admitted between June 1977 and June 1985 were reviewed, and the diagnosis of HCM was based on clinical, echocardiographic and angiographic findings in patients without other structural cardiovascular abnormalities. Out of a total of 15 patients with HCM identified, 13 had echocardiographic features of the condition. In 1 case the diagnosis was established at autopsy, no other investigations having been performed. One case was diagnosed on cardiac catheterisation (echocardiography was not available at that time).

Echocardiography was performed with either an Advanced Technology Laboratories MK 500 C or a Picker Echoview System 80 C, with routine two-dimensional echocardiographic (2DE) examination of cardiac anatomy when possible, and careful analysis of the long axis views by M-mode technique in all cases. Cardiac catheterisation was performed in 7 cases, using fluid-filled catheters with Statham pressure transducers and a Hewlett Packard Monitoring System 8890 A. Left ventriculograms were obtained for all 7 patients and showed typical features of HCM. Six of these patients also underwent echocardiography.