this group is not without risk and needs careful monitoring, many patients can take these drugs judiciously for short periods only.

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Exclude eclampsia early

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We report on a woman whose eclampsia was misdiagnosed.

Case report

A 35 year old West Indian woman was seen in the casualty department after having four seizures. She was an unmarried mother of one child and was on holiday in the United Kingdom. She was conscious and lucid and on direct questioning denied being pregnant. She was endorphic (weight 96 kg, height 163 cm), with a blood pressure of 135/65 mm Hg and 3+ proteinuria; she was considered to have generalised oedema. Findings on neurological examination were unremarkable.

Four hours after admission she had a seizure, characterised by tonic-clonic movements of her arms and legs, and was incontinent of urine. Her blood pressure was 200/130 mm Hg and her proteinuria persisted. She was given an intravenous bolus of diazepam, paracetamol, nifedipine, and frusemide. Investigations showed raised aspartate transaminase (53 U/l) and alkaline phosphatase (630 U/l) activities and a low serum albumin concentration (17 g/l). Results of haematological tests and urea and electrolyte concentrations were normal.

Because of her proteinuria, hypoalbuminaemia, and oedema she was given a high protein diet and her fluid balance was strictly controlled. Her weight fell from 96 kg to 89 kg. She was considered clinically to have ascites, and so on day 6 a drain was sited in her right flank; one litre of fluid was drained over 24 hours. A brain scan on day 7 showed a localised 2 cm lesion in the right parietal lobe with associated bilateral cerebral oedema, and a primary or secondary tumour was diagnosed provisionally. The fluid obtained by paracentesis was reported as containing chorionic or mesothelial cells. A pelvic mass was now palpable, and an ultrasound scan showed a fetus with no signs of life. She was referred to obstetricians.

A further scan showed a fetus with no signs of life and oligohydramnios. The patient's serum urate concentration was 728 μ mol/l, she had 3+ proteinuria, and her diastolic blood pressure was 128 mm Hg. Labour was induced with extra-amniotic prostaglandin, and her blood pressure was controlled with an

infusion of hydralazine. A fetus weighing 1100 g was delivered; the patient refused permission for a post-mortem examination. The postpartum period was uneventful, and she was subsequently discharged.

A repeat brain scan 21 days after the first did not show any lesion, and so a revised diagnosis of an intracerebral haemorrhage secondary to her hypertensive episode (eclampsia) was postulated.

Comment

Eclampsia is rare in the United Kingdom,¹ with an estimated incidence of less than 1 in 1000 deliveries. In black people it is more common and progresses more rapidly.² Most maternal deaths in England and Wales have been due to eclampsia or pre-eclampsia (though pulmonary embolism is now an equally common cause), with cerebral haemorrhage usually being the lethal event.³ In most cases (72%) of death due to eclampsia or pre-eclampsia the care (diagnosis and management) was considered to have been sub-standard,³ with half of the patients who died of eclampsia having had fits while in obstetric consultant units.

It is understandable that the diagnosis of eclampsia was delayed in our patient, especially as she denied being pregnant and was referred to a medical ward. The clinical diagnosis of ascites was remarkable as, although ascites is not uncommon in severe pre-eclampsia, it is generally diagnosed retrospectively at caesarean section or necropsy⁴ or prospectively by ultrasonography. The initial brain scan did not help in the diagnosis, probably because findings in eclampsia are non-specific.⁵ The revised diagnosis after repeat scanning, however, correlates with necropsy findings in most of the maternal deaths from hypertensive disease.³

This case serves to remind all doctors of the importance of considering and excluding all clinical conditions early when faced with a complex case. A regular review of reports of clinical audit, such as the Confidential Enquiry into Maternal Deaths, in leading journals might well improve matters.

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ONE HUNDRED YEARS AGO

The attention of the General Medical Council may with advantage be called during its present session to the communication from Mr J R Williams, LRCP, MRCS, which we publish in another column, concerning practice in British Columbia, from which it appears that registered British practitioners are now not entitled as such to practise in British Columbia,

but are compelled to pass an examination there in preliminary as well as final subjects. Not only are British diplomas thus disqualified from taking effect in a British colony, but it appears that a plain intimation is given that the intention is not only to set up an independent standard to judge of the fitness of medical men possessing a double British diploma to practise, but in effect to employ, also, such legislation with the view of prohibiting practice where it is considered that local interests require such prohibition. (*British Medical Journal* 1890;ii:1263.)