followed by systemic antibiotics and steroids. Intravitreal antibiotics were given only a week after the onset of his symptoms, on admission to the referral hospital.

The rapid loss of vision despite subsequent aggressive intraocular treatment with antibiotics, bears testimony to the pathogenicity of *Pseudomonas* endophthalmitis. This case illustrates once again that intravenous antibiotics are the wrong treatment for acute infectious endophthalmitis even where the organism is sensitive to the antibiotic.

The mainstay of bacterial endophthalmitis treatment is intravitreal antibiotics. The antibiotics used for intravitreal injection include amikacin (0.4 mg in 0.1 ml), vancomycin (1 mg in 0.1 ml), cefuroxime (1 mg in 0.1 ml), gentamicin (0.2 mg in 0.1 ml), and clindamycin (1 mg in 0.1 ml).[15] Vancomycin is considered the drug of choice for Gram-positive infection and acts by inhibiting the synthesis of peptidoglycan, a major component of the bacterial cell wall. Aminoglycosides such as amikacin and gentamicin which inhibit intracellular protein synthesis are used in the management of Gram-negative infections. Ceftazidime is also a useful alternative against Gram-negative organisms and acts by inhibiting the transpeptidase reaction which cross links the bacterial cell wall.[16] The Endophthalmitis Virectomy Study,[17] which looked at the management of post-operative endophthalmitis, showed there was no advantage in the concurrent administration of topical antibiotics unless there are specific problems such as microbial keratitis or wound infection. The study also established that addition of systemic antibiotics did not confer any advantage in post-operative endophthalmitis. However, in the management of endogenous endophthalmitis, especially in cases with proven bacteremia, systemic antibiotics have been used as an adjunct to intravitreal antibiotics.[18] While exogenous endophthalmitis and endogenous endophthalmitis in the immunocompromised host are well-recognised entities, a high index of suspicion is vital in diagnosing endogenous endophthalmitis in the immunocompetent patient. This case once again underlines the need for early diagnosis and administration of intravitreal antibiotics in the management of endophthalmitis.

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Sir,

Cortical blindness following pre-eclampsia

Pre-eclampsia is a disease characterised by hypertension, proteinuria and oedema in late pregnancy. It occurs in as many as 10% of all pregnancies and is a major cause of maternal and fetal morbidity.[1] It is a multisystem disorder that affects hepatic, renal and coagulation systems. Until its terminal phase it is asymptomatic but can rapidly proceed to generalised seizures (eclampsia).

Visual disturbances are reported in 20% of women with pre-eclampsia and in as many as 50% with eclampsia.[2] Isolated transient cortical blindness is rare in pre-eclampsia, reportedly occurring in 1-3% of cases.[3,4]

We describe a unique case of isolated cortical blindness in a woman with pre-eclampsia resulting in a degree of permanent visual loss. Typical occipital and parietal lobe cerebral oedema were seen on computed tomography (CT) and magnetic resonance imaging (MRI).

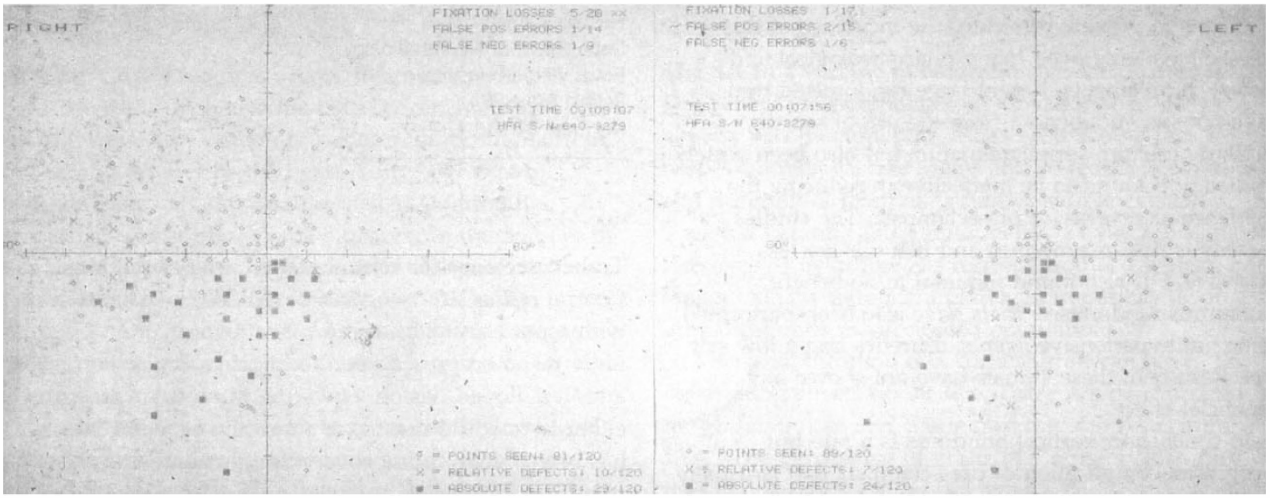

Fig. 1. *The 120 point screening visual fields showing bilateral inferior altitudinal defects.*

Case report

A 31-year-old woman (para-0) developed pre-eclampsia manifest as hypertension, oedema and proteinuria at 29 weeks gestation. She was delivered by caesarean section. Her blood pressure remained elevated following delivery and she was treated with labetalol. One week post-partum she described transient blurring of vision. This blurring of vision became a permanent feature and she was admitted to the local hosipital. Her blood pressure returned to normal during admission, but the blurred vision persisted.

Ophthalmology consultation recorded counting fingers vision in both eyes. Pupils, eye movements, and anterior and posterior segment examination were normal. The 120 point screening visual fields showed bilateral inferior altitudinal defects abutting fixation and extending into the left inferior quadrant in both eyes (Fig. 1). There were no other demonstrable neurological findings.

A CT scan demonstrated areas of low density bilaterally in the occipital lobes. MRI confirmed the CT findings, showing a high-intensity signal in the occipital areas and also in the right parietal lobe on the T2-weighted images (Fig. 2).

Three months later visual acuity was 6/5 bilaterally although she still comments on bilateral inferior scotoma, which is evident on repeat visual field-testing.

Comment

Blindness is a rare visual complication of pre-eclampsia. Most cases are transient. Parieto-occipital lesions such as oedema and infarct can be the cause. The exact pathogenesis of hypertensive encephalopathy occurring in severe pre-eclampsia and eclampsia is not fully understood. However, imaging studies consistently reveal symmetrical confluent lesions with patchy enhancement centred in the immediate subcortical white matter in the distribution of the posterior cerebral circulation.[5,6] Most lesions seen at MRI are reversible.[7]

It is suggested that vascular spasm producing transient ischaemia explains the findings in these cases. A number of theories attempt to explain this phenomenon. One observes that the anterior circulation is well innervated whereas the posterior circulation is not, and cerebral autoregulation is required to maintain appropriate perfusion. Hence over-regulation in which severe vasoconstriction occurs results in the multiple occipital lobe lesions, and breakdown in autoregulation results in vasodilatation causing focal vasogenic oedema.[8]

Additionally it has long been known that vascular reactivity is altered in patients with pre-eclampsia.[9] The causes for the differences in reactivity between vessels from patients with pre-eclampsia and normal pregnant women is not known; however, evidence for vascular endothelial involvement in this condition abounds. This evidence suggests that impaired endothelial cell function in pre-eclampsia results from one or more, as yet unidentified circulating factors released by an ischaemic placenta.[10]

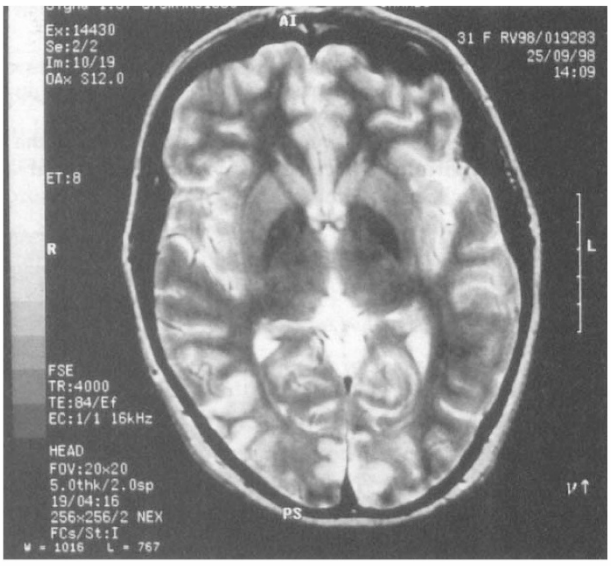

Fig. 2. *MRI scan showing bilateral occipital infarcts and right parietal infarct.*

Efforts to prevent or reduce the incidence of this disease have employed many pharmacological and dietary supplements. Recent large randomised trials have not shown a benefit from the use of aspirin. Similarly calcium supplementation has also been widely studied and found to be ineffective in reducing the incidence or severity of pre-eclampsia. The studies regarding zinc, magnesium and fish oils are less extensive, but also found minimal to no benefit. Numerous randomised trials have also been performed using antihypertensive agents, diuretics and a low salt diet. Results of these studies have not shown any beneficial effect.

In conclusion, cortical blindness is a rare but recognised complication of pre-eclampsia that is usually reversible. We report this case to emphasise that in some instances visual loss may be permanent. The postulated underlying mechanism of vascular endothelial dysfunction is depicted by the focal ischaemic changes in the brain seen in neuroimaging. However, until the pathogenesis of pre-eclampsia is well understood prevention of this condition by any means remains unlikely.[11]

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Sir,

Trabeculectomy for central retinal artery occlusion

Central retinal artery occlusion (CRAO) is associated with a poor visual outcome.[1] Treatment is often undertaken on presentation to improve ocular perfusion and, it is hoped, vision. Lowering intraocular pressure either by medical means, or surgically by paracentesis, has been shown to improve visual outcome and perfusion.[2] Repeated paracentesis has been described.[3] We report a patient who required a trabeculectomy to lower intraocular pressure after repeated paracentesis was successful but ocular perfusion worsened as intraocular pressure increased.

Case report

A 56-year-old man presented to the Eye Casualty with a 4 h history of decreased vision in his right eye. He had a visual acuity of Perception of Light (PL) with a poor pupil response, intraocular pressure 32 mmHg, and fundal appearance consistent with CRAO. The visual acuity in his left eye was No Perception of Light (NPL) due to an atherosclerotic CRAO 4 years previously. He had severe atherosclerotic disease necessitating a femoral-popliteal bypass operation and was fully warfarinised with an international ratio of 3.2.

On this occasion treatment was undertaken with immediate paracentesis using a 27 gauge needle under topical anaesthesia at the slit lamp. One to two minutes after paracentesis the intraocular pressure was 7 mmHg, and his vision improved to 6/36 with a reactive pupil. Twenty minutes later the vision began to deteriorate; the intraocular pressure had risen to 18 mmHg. A further paracentesis followed by intravenous acetazolamide 500 mg was undertaken with subsequent improvement in vision to 6/36. After half an hour there was a further deterioration of vision and a further paracentesis was performed. After paracentesis on each occasion his pupil response returned and his retinal circulation was restored. It was decided to undertake a trabeculectomy, which was performed approximately 4 h after first presentation. Sub-Tenon's anaesthesia was used with a standard technique involving a limbus-based conjunctival flap, standard scleral flap trabeculectomy and peripheral iridectomy.

The following day his intraocular pressure was 10 mmHg and his visual acuity was 6/60 with a small hyphaema. Twenty-four months later he is maintaining a pressure of 18 mmHg and a visual acuity of 6/24. Carotid Doppler sonography showed a 50–70% lumen loss and a plaque in the common carotids causing turbulent flow.