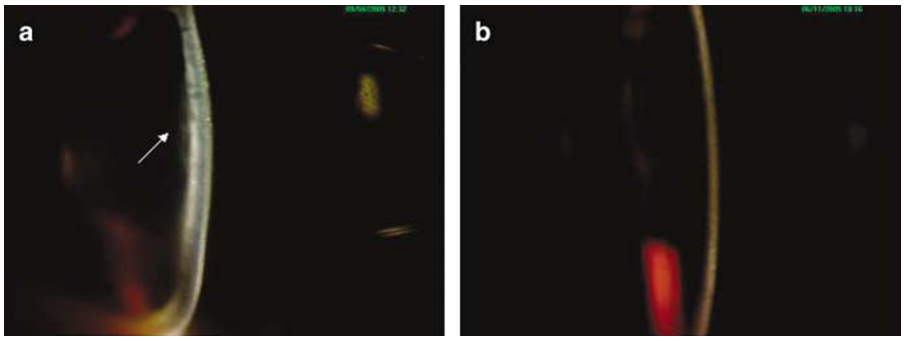

Figure 1 (a) The right eye of the patient showing central DMD (arrow) with epithelial and stromal oedema. (b) The same eye of the patient after successful repair of DMD.

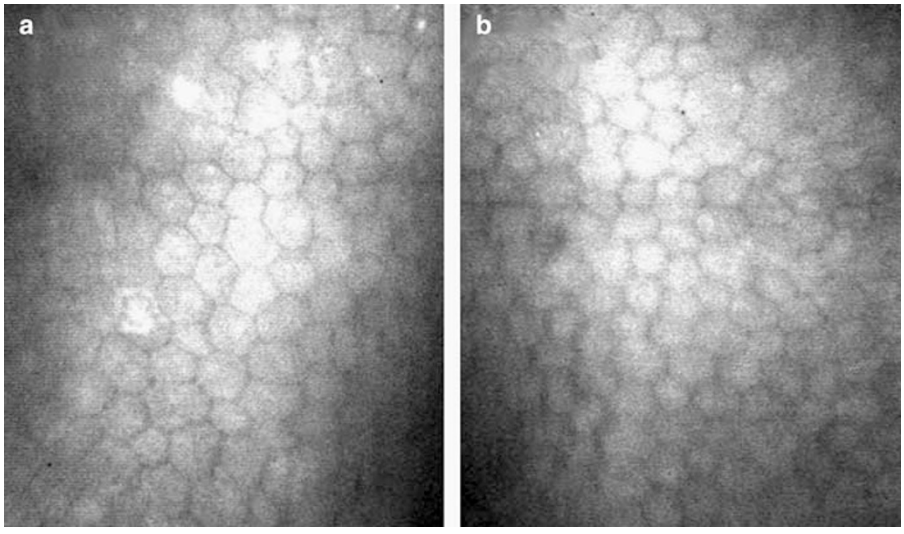

Figure 2 (a) Confocal microscopy of the right eye showing endothelial cells (cell density 1881 cells/mm^2). (b) Confocal microscopy of the left eye showing endothelial cells (cell density 2112 cells/mm^2).

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Sir,

Visual side effects after prolonged MRSA treatment

Case report

A 65-year-old man presented to eye casualty with a 2-month history of bilateral gradual loss of vision. The patient denied any other ocular or systemic symptoms.

He had no past ocular history. His past medical history revealed a left total hip replacement 3 years ago, which was complicated with a multiresistant *Staphylococcus aureus* (MRSA) infection, which, due to multidrug resistance, had been treated with long-term linezolid 600 mg b.d. for 1 year. This antibiotic had been stopped 1 week before review by the ophthalmology team.

On examination, his visual acuities were counting fingers bilaterally. Ishihara test detected a loss in bilateral colour vision, scoring 2/15 and 1/15 in the right and left eye, respectively. His pupillary responses were normal. Anterior segment examination was unremarkable and dilated fundoscopy revealed no evidence of optic atrophy or oedema.

Normal blood tests results included Hb (14.2 g/dl), WBC (8.1×10^9/l), platelets (365×10^9/l), and vitamin 12B (482 μg/l). Syphilis antibodies and mitochondrial studies were also negative.

Abnormal blood tests included a low folate level (2.7 μg/l) and hence the patient was commenced on folic acid supplements.

Visual-evoked potential (VEP) of the left eye showed a prolonged latency of 128.7 and diminished amplitude of 3.13 uV. The right eye VEP also showed a prolonged latency of 125.1 ms with diminished amplitude of 2.39 uV (Figure 1) (normal amplitude values for laboratory: 4–20 uV).

An MRI scan of his brain and orbits was performed and detected no abnormality.

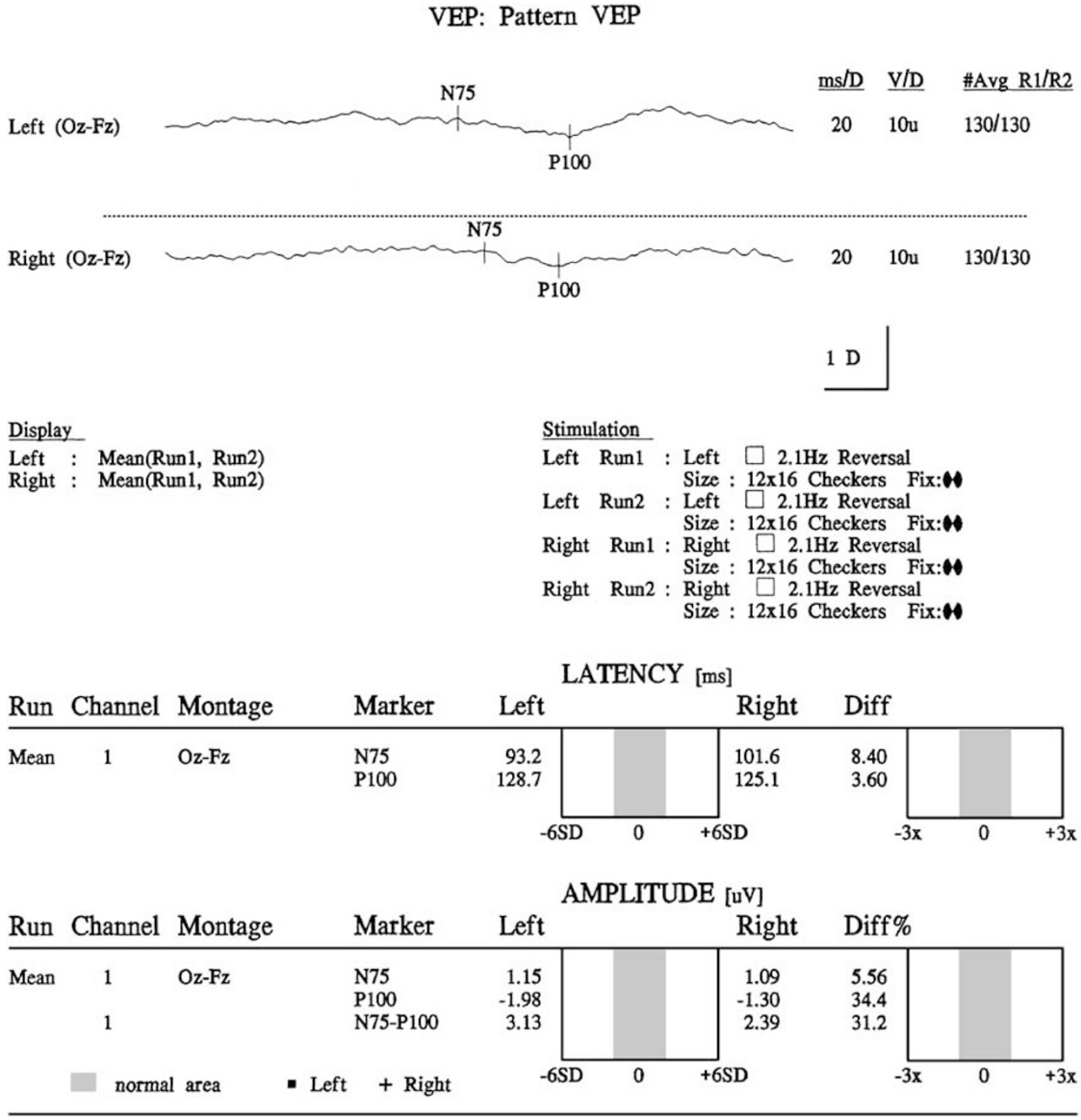

Figure 1 First VEP.

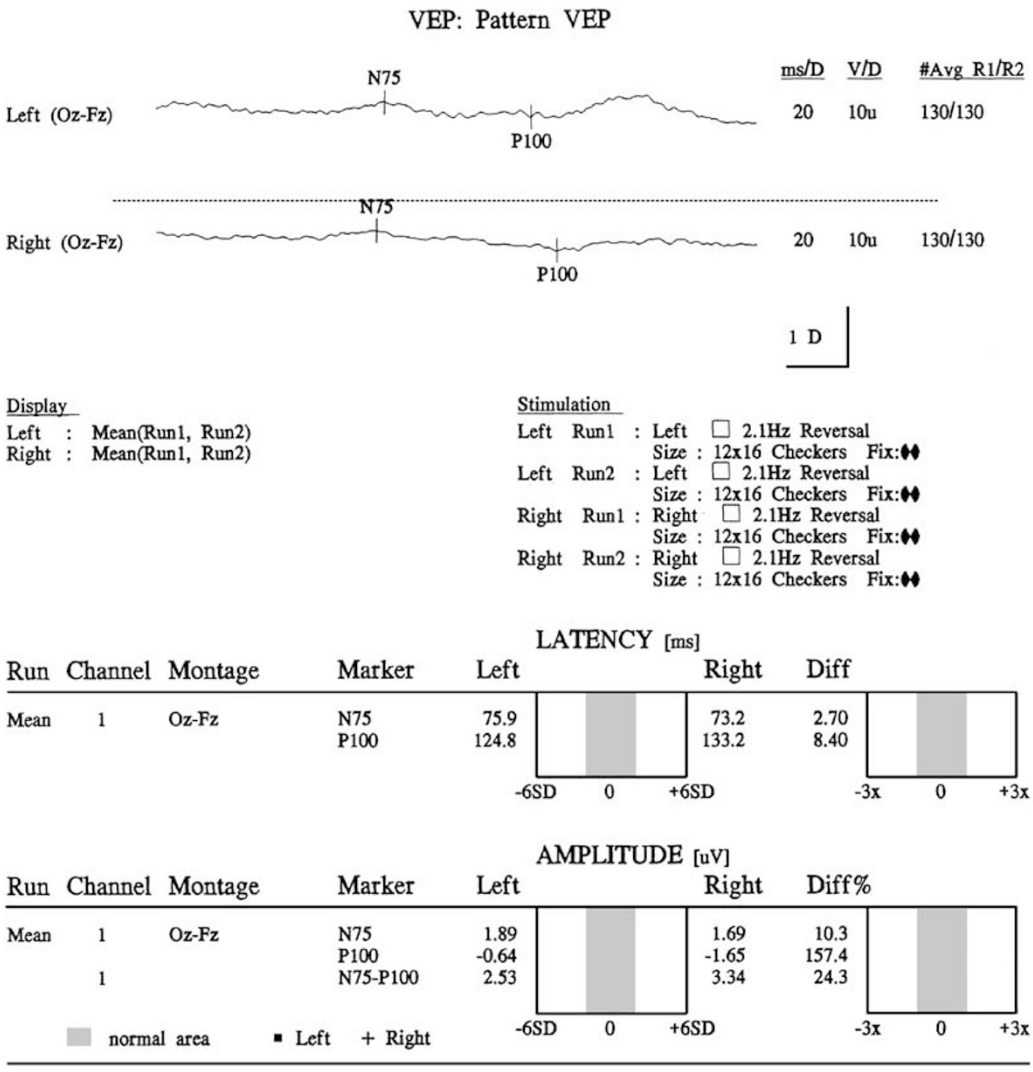

Figure 2 Second VEP.

Visual field test showed a right superior scotoma and a left superior arcuate scotoma.

Over the next 4 months, his visual acuities gradually improved to 6/6 in the right eye and 6/9 in the left. This patient still however had abnormal VEP studies showing prolonged latencies and diminished amplitudes bilaterally (Figure 2) (right eye: latency 133.2 ms, amplitude 3.34 uV; left eye: latency 124.8 ms, amplitude 2.53 uV).

Fifteen months after his initial presentation, his visual acuities had returned to 6/5 and 6/6 in his right and left eye, respectively. Dyschromatopsia and visual field defects remained stable and nonprogressive (Figures 3 and 4). After treatment with folic acid supplements his folate levels had risen to within normal limits (4.3 μg/l). VEP studies at this time had returned to normal (Figure 5). The patient reported no further symptoms.

Comment

Toxic optic neuropathy is typically a progressive bilateral symmetrical painless visual loss, which may cause a central or centralcaecal scotoma. There is currently no specific treatment for this disorder; however, early detection and prompt management may ameliorate and even prevent severe visual loss.[1]

Our case demonstrates the progressive loss of vision, which returns to normal following the cessation of the causative agent. The loss of vision was accompanied by low folate levels, persistent dyschromatopsia, and severely affected VEPs. His visual field defects are not of a typical optic neuropathy picture; however, no other cause for the defects has been discovered.

Linezolid is a synthetic antibacterial agent that belongs to a new class of antimicrobials.

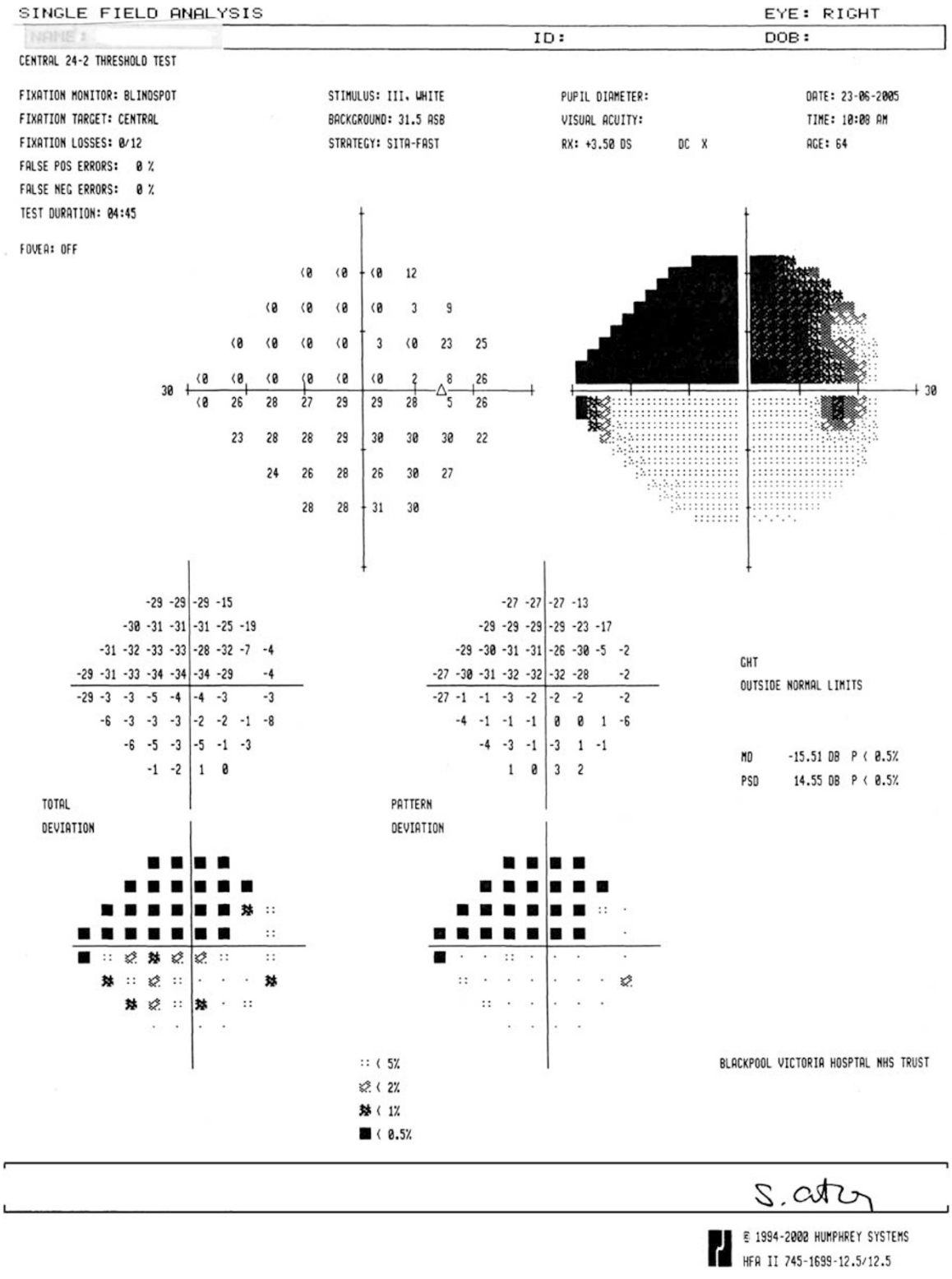

Figure 3 Right eye (last visual field test).

According to this, drugs data information leaflet, neuropathy (peripheral and optic) has been reported with use longer than the recommended duration of 28 days.[2]

There is a growing body of evidence that shows long-term linezolid use is associated with severe peripheral and optic neuropathy. In most cases, the optic

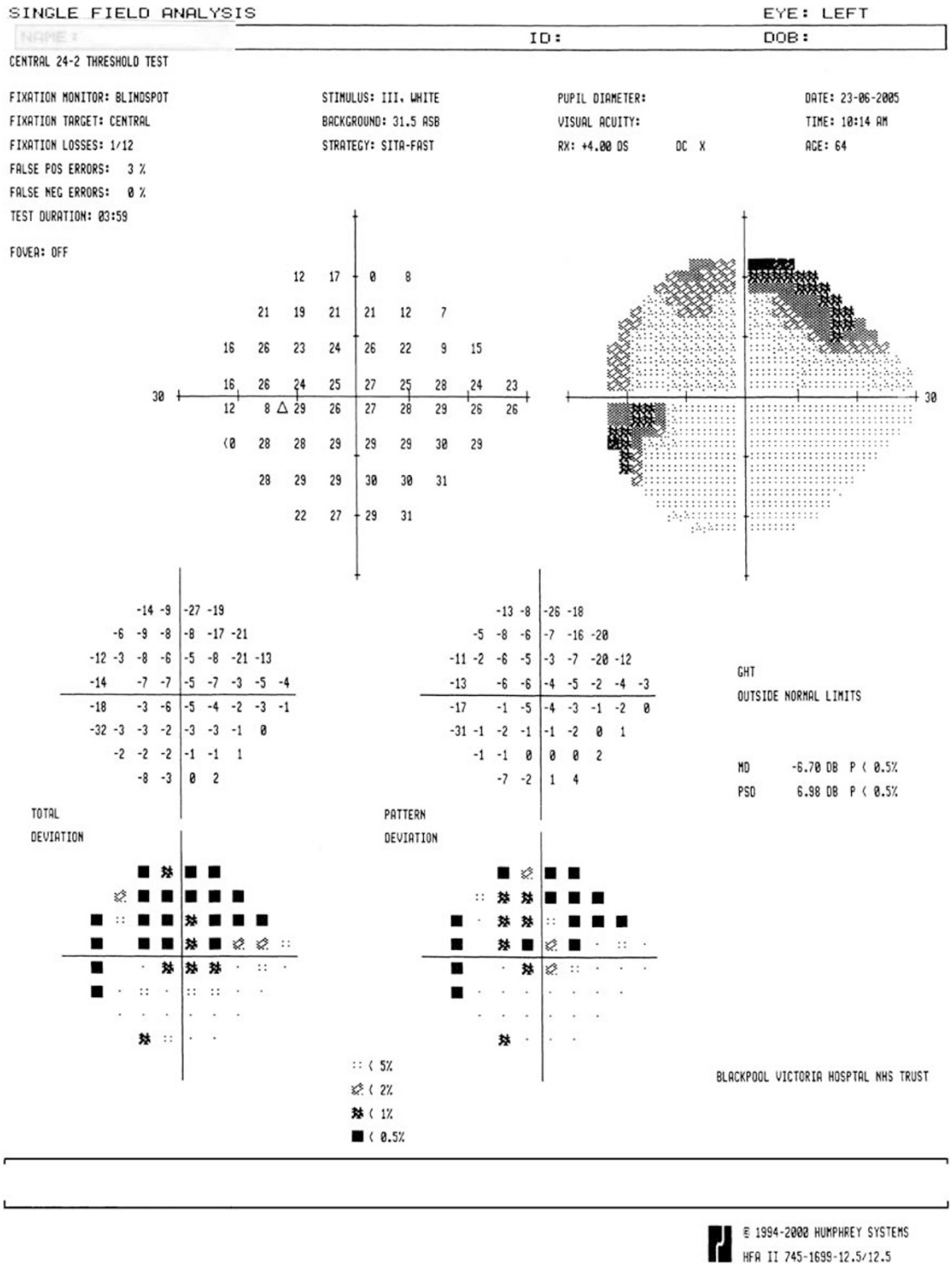

Figure 4 Left eye (last visual field test).

neuropathy resolved with drug cessation, leaving a residual deficit in central visual acuity.[3–8] The mechanism of toxicity remains unclear although previous reports of linezolid associated optic neuropathies suggest mitochondrial dysfunction as the most possible cause of neurotoxicity. In particular, low folate levels subsequently cause elevated plasma homocysteine, which can lead to inhibition of neuronal mitochondrial function.[9–11]

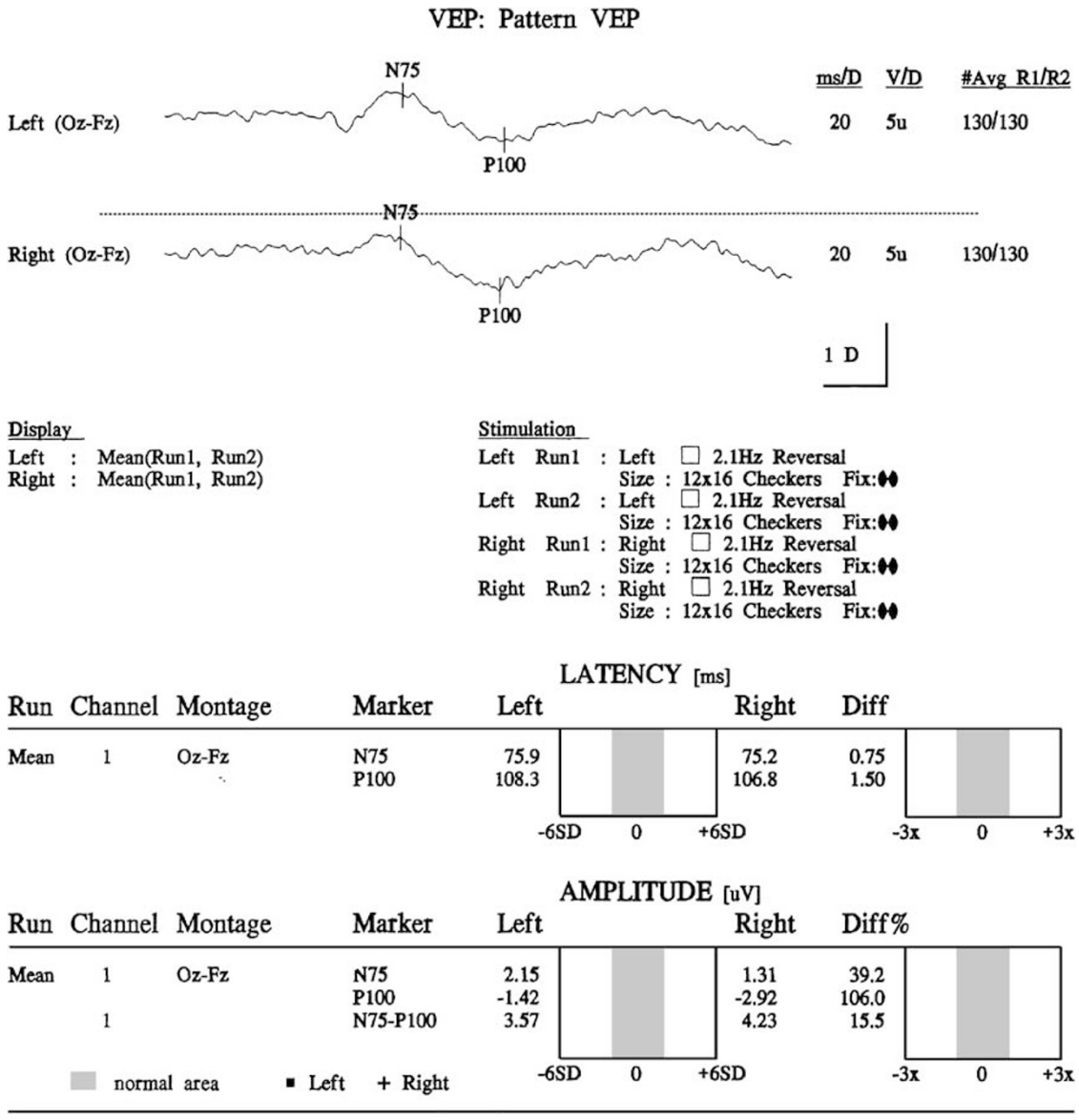

Figure 5 Third (last) VEP.

Given the high profile of MRSA infections, the use of such antimicrobial agents may be used increasingly in an attempt to control persistent infections. We therefore feel that this case should be brought to the attention of the ophthalmic community so that they are aware of its possible ophthalmic toxicity.

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Sir,

Surgical removal of massive macular hard exudate combined with intravitreal triamcinolone in diabetic maculopathy

Case report

A 65-year-old retired graphic designer who was non-insulin dependent diabetic was referred to the vitreoretinal team for consideration of surgical treatment for exudative diabetic maculopathy. He had previously had unsuccessful macular grid and focal Argon laser treatment at his referring unit. On presentation, his corrected visual acuities were right 3/60<N48 and left 6/36<N48. Anterior segments were normal. Fundoscopy revealed extensive intraretinal hard exudates at both posterior poles, more pronounced at the right macula as shown in Figure 1. Fluorescein angiogram revealed macular oedema in both the eyes.

The possibility of surgical intervention was discussed with the patient. He was keen despite a guarded prognosis and potential complications of surgery. With his informed consent, he underwent a standard right three port pars plana vitrectomy followed by a small partial thickness retinotomy made with a sharp pick just temporal to the massive macular hard exudate. The hard exudate was debulked using saline irrigation with a narrow gauge cannula and sub-retinal forceps through the retinotomy. After fluid-air exchange, 4 mg of intravitreal triamcinolone and 15% C_3F_8 gas, were injected. A face down posture was recommended for 1 week to allow time for the retinotomy site to heal.

At 2 months follow-up with refraction, his visual acuity was 6/60 N24 right. He stated that he had better depth perception, his vision was brighter and he could now watch the television at 3 m whereas previously he had to be within 1 m of the screen. His near vision had improved (<N48–N24). Fundoscopy showed a significant reduction in the size of the macular exudates (Figure 1c).

Visual outcome for the right eye remains satisfactory after 20 months at 6/60 N24 with subjectively increased depth perception and contrast sensitivity.

Comment

Diabetic maculopathy is an important cause of visual impairment. The current treatment for clinically significant macular oedema is focal or grid Argon laser photocoagulation to stabilise vision.[1] In advanced cases, laser treatment is ineffective. Massive deposition f macular hard exudates carries an increased risk of subretinal fibrosis.[2] With this in mind, other treatment modalities have been attempted. Yang[3] performed vitrectomy, focal endolaser, and panretinal photocoagulation, and showed regression of both macular oedema and hard exudates. Intravitreal triamcinolone alone has been reported to reduce macula oedema and the amount of hard exudates.[4,5] Takagi *et al*,[6] Sakuraba *et al*,[7] and Takaya *et al*[8] have removed macular hard exudates surgically following vitrectomy. Despite good anatomical results, visual acuity improvement was not maintained long term with surgical removal alone because of atrophic or degenerative changes.[8] A combined procedure of surgical debulking and intravitreal triamcinolone would theoretically simultaneously reduce both macular exudate and oedema in a shorter period of time thereby reducing the risk of subretinal fibrosis.