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Sir,

Visual field loss and Alzheimer's disease

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Alzheimer's disease (AD) is a degenerative neurological condition of unknown aetiology affecting approximately 10% of the UK population aged 65 or over.[1] Clinical features include disturbances of memory, language, praxis and conceptual abilities. We describe a case of Alzheimer's disease presenting, unusually, with visual field loss.

Case report

A 52-year-old female teacher presented to her optometrist with an 18-month history of difficulty in writing. Optometric examination showed visual acuities of 6/5 in each eye. No abnormality of the anterior segments or fundi was detected and intraocular pressures were 20 mmHg bilaterally. Automated perimetry revealed a left inferior field defect in both eyes. These findings prompted referral to the glaucoma clinic, where a more detailed history was elicited. She first noticed problems with poor spelling and spidery handwriting. More recently she had become aware of an impaired ability to iron, orientate her dress, interpret the time on a clock and to read the schoolchildrens' handwriting. Occasionally her mind would go blank in mid-conversation and she had problems remembering the plot of a film.

She denied headache or other neurological symptoms. Her general health was good and she was not on medication.

The findings recorded by the optometrist were confirmed. In addition, Goldmann intraocular pressures were 16 mmHg in each eye and the optic disc appearances were normal (cup:disc ratio 0.4). A slightly incongruous left inferior homonymous quadrantanopia was clearly defined by Goldmann and Humphrey full threshold visual field testing (see Figure 1). Apkarian flash visual evoked responses were normal from the left occipital lobe and reduced and delayed from the right. An electroencephalogram showed non-specific changes reflecting generalised cerebral dysfunction, suggesting the possibility of Jacob–Creutzfeld disease to be unlikely. On the basis of the long history, the nature of the presenting symptoms and the striking MRI findings a diagnosis of Alzheimer's disease was made after consultation with a neurologist.

Comment

Disturbances of the visual system in Alzheimer' disease (AD) are well documented, and importantly, can predate other manifestations of dementia.[2] Unfortunately, because of characteristically vague symptomatology and normal examination findings at presentation, the diagnosis may be overlooked.

Patients with AD usually seek an ophthalmic consultation because of disturbances of pattern processing and recognition. The commonest example of this is difficulty with reading which may present as skipping of words or lines on a page, 'dancing print' and blurred vision, and can progress to alexia in advanced disease.[2,3] Other well-documented difficulties include an inability to recognize faces (prosopagnosia), to pick out individual objects in a group, or disorders of visuospatial processing,[4] eg inability to grasp objects within the field of view (optic ataxia), neglect of objects to one side of the object of regard (simultanagnosia), dress disorientation (optic apraxia), and becoming lost in familiar surroundings.

Conventional examination findings are often unremarkable in patients with AD.[4] Central visual acuity generally remains normal, at least early in the disease. Colour vision disturbances are not uncommon but may not always be apparent on formal testing. Optic disc pallor has been reported, but is not a characteristic finding. Subtle ocular motility disturbances have been demonstrated, but patients are usually asymptomatic.

Visual fields, though sometimes difficult to assess in AD patients, are usually full in the early stages.[4] Abnormal perimetry measurements have been identified by Trick *et al* during apparently reliable automated field testing.[5] However, the reported defects were mainly generalised loss of sensitivity or arcuate scotomata, and none had a clearly defined homonymous quadrantanopia. Our patient was young,

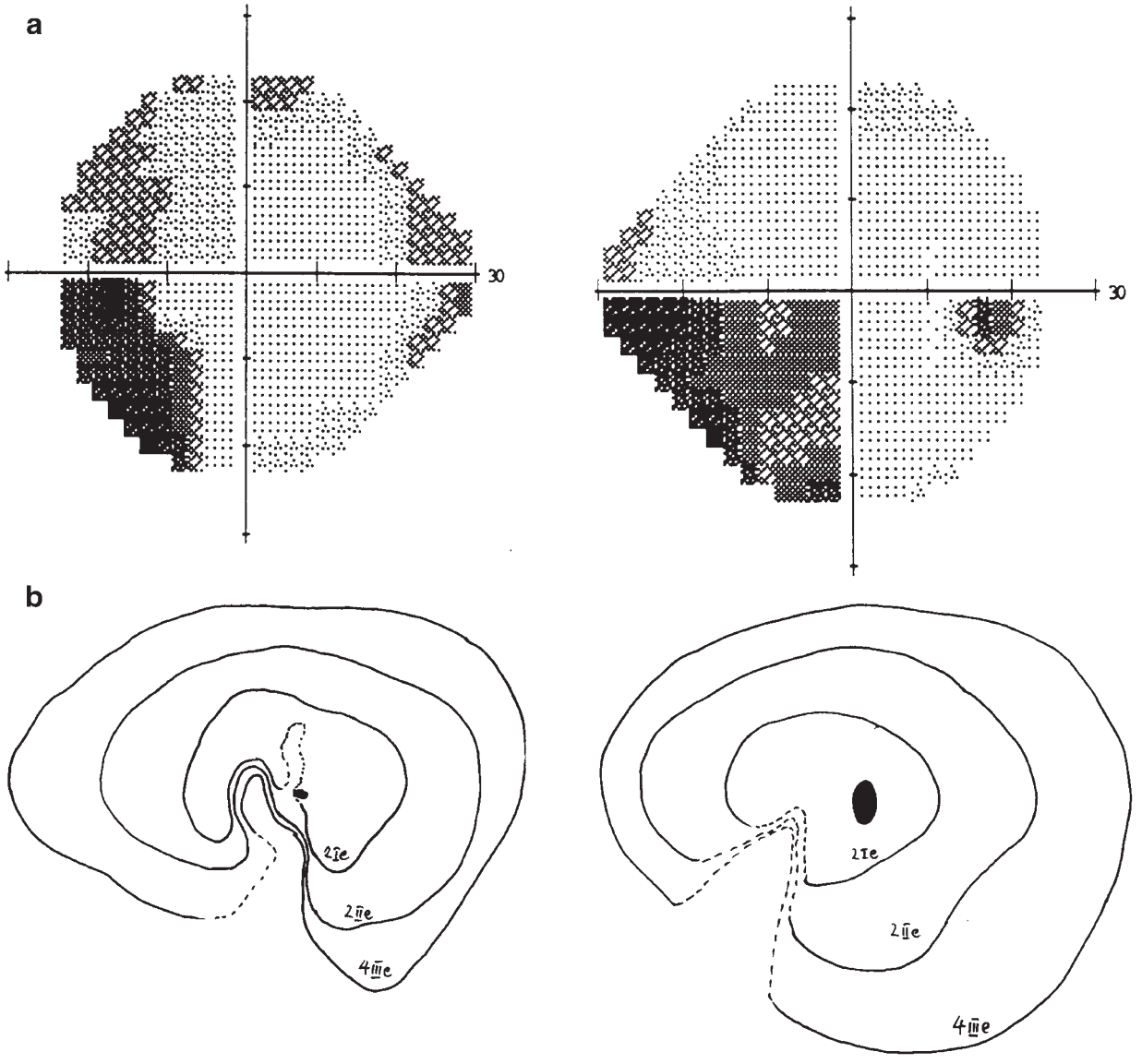

Figure 1 (a) Full threshold 24–2 perimetry. (b) Goldmann perimetry.

with no cardiovascular risk factors, and there was no sign on neuroimaging of a lesion in the contralateral optic radiation, parietal lobe or optic tract such as thromboembolic infarction, cerebral hemorrhage or a space-occupying lesion. However, histopathological studies have shown AD patients may exhibit significant selective loss of neural elements within the visual cortex, which could account for the pattern of field loss in this case.[6]

Although not applicable here, one should consider that hemisensory visual deficits have also been described in patients with corticobasal degeneration or a mixed corticobasal degeneration – AD picture.[7] In addition, some patients with AD have 'simultanagnosia' involving a defect of visual attention, in which hemifield loss or disorientation may be apparent rather than genuine.[3]

Given the diagnostic limitations of standard examination techniques, attention has focused on 'higher visual function'. Studies have shown that patients with AD have impaired form-identification and visuospatial skills in spite of preserved visual acuity and color vision.[8,9] Several tests have been designed to look for visually symptomatic patients with as yet undiagnosed AD, who may be passed as 'normal' on standardised cognitive screening tests.[9]

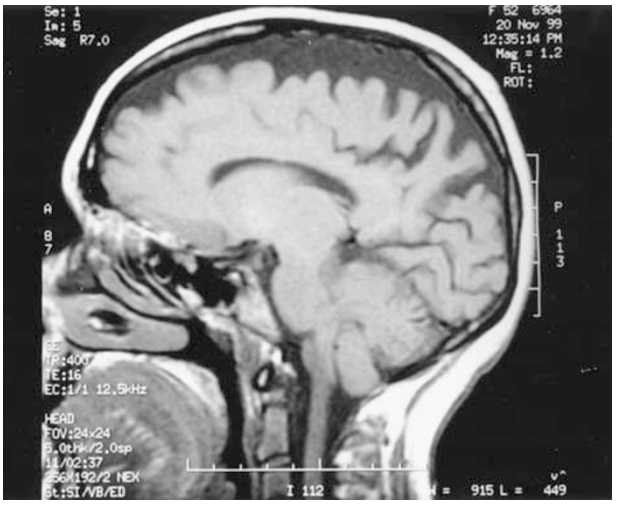

Figure 2 MRI scan of brain.

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Sir,

Subclinical hypothyroidism—increased awareness may prevent unnecessary treatment and morbidity
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A 44-year-old male lawyer was found to have a raised serum cholesterol of 8.6 mmol/l and a serum triglyceride of 1.43 mmol/l following an insurance medical. Concurrent optometric examination revealed elevated intraocular pressure (IOP). We describe the subsequent management of this patient and how an oversight of an interesting medical principle can lead to unnecessary treatment with all its sequelae.

Case report

The patient was subsequently referred to an ophthalmology department and a diagnosis of glaucoma was made on the clinical grounds of bilaterally raised IOP of 24 mmhg and the presence of abnormal optic disc appearances. Treatment with topical Timolol 0.5% b.i.d. was commenced.

IOPs remained high and after a year on treatment, topical Pilocarpine 1% t.d.s. was added. This dose was further augmented to Pilocarapine 4% t.d.s. one year later in an attempt to alleviate IOP.

At the same time this patient was attending the Lipid clinic for treatment of his hypercholesterolaemia. Diet treatment and oral Colestifol failed to reduce his levels so Prabistin 10 mg o.d. was prescribed. This caused sleep disturbance. One year later Simvastatin 20 mg o.d. was substituted which lowered the serum cholesterol level to 6.9 mmol/l, but unfortunately this drug induced seborrhoeic dermatitis and was discontinued on the advice of a dermatologist. Several visits to the Lipid clinic with further trials of Clofibrate and Tenofibrate showed little response. The patient's serum cholesterol remained elevated and Fluvastatin 40 mg o.d. was then commenced.

Five years post diagnosis, the patient came under our care. The diagnosis of glaucoma was reconsidered, as visual field testing was normal. The optic discs were not glaucomatous but dysplastic with large cups and normal nerve fibre layer. IOPs were normal in the primary position but became elevated on up gaze. In addition lid lag was present on down gaze.

There was no evidence of lid retraction, soft tissue signs, orbital proptosis or systemic features of thyroid dysfuction. We diagnosed dysthyroid eye disease and discontinued topical treatment. Thyroid function tests were performed and demonstrated a raised thyroid