contact lens as often as possible; whereas she was initially aware of intermittent diplopia this improved over a period of several weeks by which stage she was noted to have a non-specific exophoria with left suppression. These findings remained stable over a period of several months and it was therefore deemed to be safe to proceed with implantation of a secondary intraocular lens combined with iris prosthesis. Post-operatively her acuity gradually improved to 6/9 with orthoptic assessment revealing a non-specific exophoria. Although she was initially aware of diplopia this gradually settled and over the following 4 months her best corrected acuity improved to 6/6 and she regained 110 arc sec (Frisby). At her most recent clinic visit 1 year after her surgery her examination is unchanged.

Comment

Recovery of a high level of stereoacuity following such a long period of visual deprivation and longstanding strabismus is a very rare occurrence. Although there have been reports of patients regaining up to 40 arc sec of stereoacuity more than 30 years following the onset of their strabismus,[1,2] in contrast to our patient they all had acuities of at least 6/9 in the squinting eye with no reported history of visual loss. It is thought by some that the ability of patients with chronic acquired strabismus to regain binocular vision is dependent upon their previous capacity for binocularity.[4] As our patient regained such a high level of stereoacuity it is reasonable to assume that this was present before her original injury. However, the fact that this occurred after such a long period of poor vision is most unusual and would indicate that the visual system has a surprising capacity for recovery.

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Sir,

Acute leber hereditary optic neuropathy in a 73-year-old man

Sudden onset of optic neuropathy in the elderly is most frequently vascular in aetiology; however, when bilateral, the diagnosis of LHON should still be considered. We report the oldest documented case of visual loss due to LHON.

Case report

A 73-year-old man presented with severe painless sequentially bilateral visual loss over the past few weeks. He recorded counting fingers vision bilaterally, but ocular examination was unremarkable with only mild dry age-related macular degeneration, normal retinal vessels, and healthy looking optic discs. Investigations including fluorescein angiography, CT scan of head and orbits, and extensive blood testing failed to provide any explanation.

He was a known hypertensive with a history of previous myocardial infarction and peripheral vascular disease requiring bilateral femoral bypass grafting. Carotid Doppler ultrasound scanning demonstrated internal carotid artery stenosis of >70% on the side of the initially affected eye, but no significant stenosis on the other side.

A family history of LHON was elicited. Genetic screening to investigate this possible diagnosis was undertaken which found a guanine to adenine mutation at position 11 778 on the mitochondrial DNA. His optic discs had become pale by the 10th week after presentation and he could see only hand movements.

Comment

LHON is a maternally inherited condition caused by point mutations in the mitochondrial DNA. Penetrance, independent of genotype, is estimated at 25–50% in males and 5–11% in females,[1] visual loss becoming bilateral within 1 year in 97% of cases. The typical interval between eyes is 2–4 months, although simultaneous bilateral involvement is reported.[2]

Of patients who eventually suffer visual loss, almost 70% will do so before the age of 30.[2] Those unaffected by the age of 50 are >95% likely to remain so, although asymptomatic carriers have been found to demonstrate significant subtle chronic changes such as peripapillary microangiopathy, retinal nerve fibre layer thickening,[3] and colour vision deficit.[4]

Metabolic or ischaemic factors are well-recognised triggers to visual loss in LHON. The precipitant of visual loss in our patient was felt to be ischaemia due to vascular disease leading to an exceptionally late clinical presentation.

This case demonstrates that there may be no upper limit to the age at which LHON should be considered in the differential diagnosis of bilateral or sequential optic neuropathy.

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Sir,

CRMP-5-IgG in patient with paraneoplastic optic neuritis with lung adenocarcinoma

Paraneoplastic optic neuritis (PON) is an ocular manifestation of the paraneoplastic syndrome that is clinically characterized by subacute vision reduction, optic disc swelling, and neurological symptoms.[1] Previously, the diagnosis was difficult because it was based only on the clinical signs and symptoms. Recently, a distinct IgG for the 62 kDa collapsin response-mediator protein-5 (CRMP-5-IgG) was demonstrated to be a serological marker for PON.[1,2]

Case report

A 55-year-old woman noticed a subacute bilateral vision reduction and was referred to our hospital. She had a history of a lung adenocarcinoma resection at 46 years of age, but the cancer reappeared 3 years later. She also had occasional epileptic seizures beginning at 54 years of age.

On examination, her visual acuity was 20/400 in each eye. Her pupillary responses, eye movements, and anterior segments were normal. The critical fusion frequencies were markedly reduced. Funduscopic examination showed a mild swelling of both optic discs (Figure 1a). There were no inflammatory cells in the vitreous cavity. Fluorescein angiography revealed hyperfluorescence and leakage on the optic disc in accord with the swelling, but not in areas away from the disc. The arm–retina time was not delayed. Visual field examination showed a central scotoma and enlarged blind spot in both eyes (Figure 1b). Full-field cone and rod electroretinograms were within normal limits.

Cranial computed tomography (CT) and magnetic resonance imaging showed neither metastatic cancer nor white matter lesions suggestive of multiple sclerosis. Cerebrospinal fluid examination revealed a slightly elevated protein level (64 mg/dl). Laboratory findings were normal except for an elevated carcinoembryonic antigen.

Methylprednisolone pulse treatment, 500 mg/day, was given for 3 days but the visual acuity and optic disc appearance did not improve. Chest CT showed nodular lesion at the apex of the right lung.

Based on these findings, a possibility of PON was considered. We performed Western blot analysis of her serum and detected a 62 kDa band corresponding to CRMP-5-IgG (Figure 2).

Comment

Most of the previous cases of PON were associated with small-cell lung carcinoma, and a lung adenocarcinoma has been reported in only one case but without CRMP-5-IgG testing.[1,3] We are unaware of previous