

Treatable blindness in temporal arteritis

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Abstract

Temporal arteritis is a common cause of blindness. Prompt steroid treatment limits unilateral visual loss while protecting the contralateral eye. Established blindness is irreversible. We report a case of temporal arteritis in which an eye with no light perception secondary to an arteritic anterior ischaemic optic neuropathy regained 6/6 vision.

A 62-year-old Caucasian woman presented to Cheltenham General Hospital in February 1987. She gave a two-month history of generalised malaise, weight loss, pyrexias, proximal myalgia, jaw ache, headache, and scalp tenderness. Corrected visual acuities were 6/5 right and 6/4 left. The erythrocyte sedimentation rate (Westergren) was 110 mm in one hour. A diagnosis of temporal arteritis was made and prednisolone 40 mg started. Seven months later the ESR was 26 mm in one hour, and her prednisolone was decreased to 10 mg daily. Soon afterwards she suffered total loss of vision in the right eye; visual acuity in the left was 6/5. The right eye showed an amaurotic pupil and a swollen optic disc. She also developed a right partial third nerve palsy. The ESR was 40 mm in one hour.

On a daily dose of 60 mg prednisolone her visual acuity in the right eye slowly improved, and three months later it was again 6/6, N 4.5. Colour vision was normal (Ishihara), though Friedmann field analysis showed slight central suppression. The third nerve palsy resolved.

In January 1988 the cycle was repeated, when right vision dropped to 6/60 with recurrent disc

swelling. After she had received intravenous methylprednisolone 500 mg daily for two days her right visual acuity again returned to 6/6, N 4.5.

In view of the extraordinary clinical course, temporal artery biopsy was performed 33 months after presentation (Fig 1). The localised loss of the internal elastic lamina and intimal proliferation was consistent with long-standing temporal arteritis.¹

So far her vision has been preserved on a regimen of combined steroid and azothioprine.

Discussion

Temporal arteritis causes blindness secondary to an ischaemic optic neuropathy, central retinal artery occlusion, or cortical infarction.² Although moderate visual recovery is well recognised in arteritic ischaemic optic neuropathy, marked improvement is rare.²⁻⁵ Once established, blindness is irreversible.⁶

Model³ reported vision improving from light perception to 'normal vision', while Rosenfeld *et al*⁷ reported improvements from 20/70 to 20/25. Both patients received methylprednisolone. Schneider *et al*⁴ tabulated 22 eyes, five of which improved from no light perception (NLP). None regained 6/6 vision.

Anterior ischaemic optic neuropathy causes an optic nerve conduction defect. Consequent visual loss will be permanent only when axonal infarction supercedes ischaemia. Although this progression usually occurs rapidly, our patient had NLP for several days, suggesting that severe, prolonged ischaemia need not result in permanent axon loss.

Intravenous methylprednisolone is a rare cause of sudden death from cardiac arrhythmias.⁸ Despite this we recommend considering its use in the treatment of visual loss secondary to arteritic anterior ischaemic optic neuropathy, especially when the second eye is affected.

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Figure 1 Photomicrograph of section of temporal artery showing fragmentation of the internal elastic lamina with cellular proliferation within the intima. ($\times 100$, elastin stain.)

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