

Intermediate uveitis and Lyme borreliosis

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Abstract

A case of chronic intermediate uveitis and associated classic snowbanking (pars planitis) with severe cystoid macular oedema probably due to Lyme borreliosis is reported. Despite a disease duration of 10 years the patient's ocular symptoms and visual acuity responded promptly to intravenous ceftriaxone treatment. This case demonstrates that periodic re-evaluation of patients with intermediate uveitis is necessary to obtain a specific diagnosis which may include Lyme borreliosis.

Ophthalmic manifestations of Lyme borreliosis can occur in every stage of the disease either in the presence or absence of other organ involvement.^{1,2} The ocular involvement includes keratitis, uveitis, endophthalmitis, and various neuro-ophthalmologic symptoms. The cases of intermediate uveitis due to Lyme borreliosis reported so far have been associated with posterior synechiae and granulomatous anterior chamber inflammation.¹ We report on a patient with classic intermediate uveitis (pars planitis) and Lyme borreliosis.

Case report

In 1982 a 29-year-old man noticed a decline of visual acuity in both eyes. He consulted several ophthalmologists from 1982-84 but no ocular abnormalities were found which could explain his complaints. In 1984 he suffered from presumed bacterial keratitis in his left eye which was treated topically elsewhere.

In February 1985 he visited our department with complaints of conjunctival redness and blurred vision. History disclosed an amblyopic right eye due to esotropia. He had a refractive error of $+6+1.5 \times 120^\circ$ RE and $+6+1.5 \times 170^\circ$ LE. The corrected vision was 0.4 RE and 0.6 LE. Further ocular examination revealed a slight follicular conjunctivitis in both eyes, peripheral blue-dot lens opacities in both eyes with a clear visual axis, and no signs of intraocular inflammation. No abnormalities except conjunctivitis were encountered and no treatment was given.

In November 1985 he complained again of decreased vision. On examination a corrected vision was 0.4 RE and 0.5 LE was found and except for the blue-dot lens opacities there were no pathological findings.

In July 1987 he consulted us again with a progressive visual decrease. Visual acuity was 0.3 in both eyes. Further examination revealed flare and cells in the anterior chamber, blue-dot lens opacities, vitritis, peripheral snowbanking, and cystoid macular oedema (CMO) in both eyes (Fig 1). The fluorescein angiography confirmed the pars planitis and CMO and disclosed no signs of vasculitis or chorioretinitis. The results of the

screening programme for uveitis (serum angiotensin converting enzyme, serum lysozyme, serological test for syphilis, Mantoux test, chest x ray) were all within normal range.

The diagnosis of idiopathic intermediate uveitis was made. Oral prednisone therapy was instituted because of the marked CMO. The visual acuity slowly improved to 0.4 both eyes and the inflammatory signs regressed within several weeks.

In December 1987 progressive visual loss occurred in LE due to massive retinal oedema with associated peripheral intra- and subretinal exudates. Cryocoagulation of the peripheral retina (snowbanks) LE was performed with favourable effect. In the following years the patient also received subconjunctival corticosteroid injections depending on his disease activity and his visual acuity varied between 0.3-0.5 RE and 0.3-0.6 LE.

In 1990 re-evaluation of the cause of uveitis revealed no abnormalities except for a positive antibody titre against *Borrelia burgdorferi* (indirect immunofluorescence assay 1:320). A detailed history concerning Lyme borreliosis was obtained. The patient recalled no tick bite. However in 1980 he noticed a droplet of blood on his left thigh. Several days later an expanding erythematous skin lesion occurred on this spot. Within 1 week severe radicular pain in both legs occurred. Because of this pain he could not walk nor sleep for 1 week. The pain abated in 1 month without any specific treatment.

Neurological examination in 1990 revealed no abnormalities. Analysis of cerebrospinal fluid showed normal leucocyte count, protein, and glucose concentration. No signs of intrathecal antibody production were found.

The history of the skin lesions was compatible with erythema migrans and the radicular pain

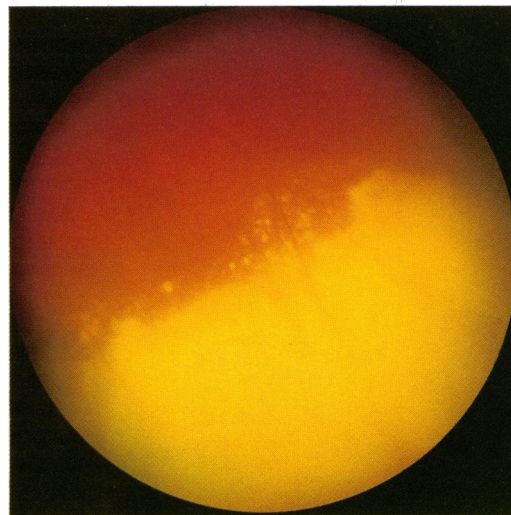


Figure 1 Intermediate uveitis with peripheral snowbanking in a patient with Lyme borreliosis (LE).

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might have been meningoradiculitis. A presumptive diagnosis of Lyme borreliosis was made according to CDC criteria.³ Subsequently the patient was treated with ceftriaxone intravenously 4 g/day for 2 weeks with prompt response of ocular symptoms. In 2 months his visual acuity was improved to 0.5 RE and 0.8 LE and the active inflammatory signs vanished.

Discussion

Intermediate uveitis associated with Lyme disease has been described but in these patients the uveitis was complicated by posterior synechiae and granulomatous anterior chamber inflammation.¹ These anterior chamber symptoms are not regularly encountered in classic intermediate uveitis (pars planitis).⁴

Our case demonstrates that even a classic intermediate uveitis with a protracted course

may be due to Lyme borreliosis. Our patient responded promptly to specific antibiotic therapy with ceftriaxone and despite the CMO over several years his visual acuity rapidly increased. We report this case to emphasise the following points: periodic re-evaluation of patients with intermediate uveitis is necessary to obtain a specific diagnosis which may include Lyme borreliosis. Furthermore chronic cases suffering from intermediate uveitis and long standing CMO due to Lyme borreliosis may benefit from adequate treatment.

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