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Lepromatous Uveitis Diagnosed by Iris Biopsy: A Case Report

Arash Maleki, MD; C. Stephen Foster, MD, FACS, FACR

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Introduction

Leprosy, a chronic granulomatous infectious disease caused by Mycobacterium leprae, mainly involves skin, peripheral nerves, mucous membranes, and ocular structures. As early as 1873, Bull and Hansen drew attention to leprous eye complications, which may be present in almost all patients with longstanding disease. Ocular damage occurs through four mechanisms: direct bacterial infection leading to keratitis, scleritis, and uveitis; facial and trigeminal nerve involvement; hypersensitivity reactions such as erythema nodosum leprosum (type I) or reversal reactions (type II) complicated by corneal hypoesthesia, superficial punctate keratitis, episcleritis/scleritis, nerve paralysis, and/or iridocyclitis; and secondary infections. Early subtle signs of ocular involvement are autonomic dysfunctions, including diminished pupillary reactions and reduced accommodation. Although leprosy is considered primarily a tropical disease, 2,469 cases were reported in the United States from 1981 to 1990. Imported rather than indigenous cases account for the growing incidence of leprosy in the United States, reflecting increases in immigration and international travel by American citizens.

Here, we report a male patient presenting with bilateral uveitis, glaucoma, and keratitis resistant to conventional therapy. Skin and iris biopsies, along with aqueous humor samples, disclosed abundant Wade–Fite–positive organisms consistent with *Mycobacterium leprae*.

Case Report

A 38-year-old African American male, who immigrated from St. Lucia to the United States three months prior to presentation, presented with a progressive decrease in vision in his

left eye (OS) over the preceding 8 months. His past ocular history included blunt trauma to his right eye (OD) resulting in substantial visual loss.

The patient's visual acuity was counting fingers at 2 feet (OD) and 20/100 (OS). Intraocular pressures were elevated in both eyes. Examination of the right eye revealed corneal neovascularization and edema, a fibrovascular membrane behind the endothelium, 3+ anterior chamber inflammation, Rubeosis iridis and a secluded miotic pupil. The left eye showed corneal pannus formation superiorly with inferior anterior stromal infiltrates, 3+ anterior chamber inflammation, posterior synechiae and cystoid macular edema.

Initially, the patient was treated with topical prednisolone acetate 1%, flurbiprofen sodium 0.03%, and timolol 0.5% in the left eye, along with oral diflunisal and a 40-mg transseptal injection of triamcinolone. Extensive laboratory and imaging investigations were negative or within normal limits, except for elevated circulating immune complexes and a nodular apical density on chest X-ray suggestive of granulomatous disease. A purified protein derivative (PPD) skin test for tuberculosis was negative, with appropriate positive controls. Serologic tests for syphilis were negative. The patient was referred to a pulmonologist for further evaluation of the pulmonary lesion.

Over the next 2 weeks, marked iris granulomas developed in the left eye. Vision in the right eye declined to light perception due to an intraocular pressure of 52 mm Hg secondary to complete angle closure. Bronchoscopy and possible lung biopsy were considered by the pulmonologist to further evaluate the pulmonary lesion.

At this point, the patient revealed a history of lepromatous leprosy that had been treated 15 years earlier with a two-year course of dapsone and clofazimine and he had been told he was cured. He reported hesitating to disclose his disease out of concern that it could jeopardize his efforts to obtain United States citizenship.

Biopsies of mildly hypopigmented skin of the upper extremities, as well as aqueous humor and iris of the right eye, disclosed abundant Wade-Fite-positive organisms consistent with M. leprae. Mycobacterial culture did not yield any growth; however, polymerase chain reaction (PCR) confirmed the presence of M. leprae.

Multidrug therapy with Dapsone (100 mg/day), clofazimine (50 mg/day), and rifampin (600 mg monthly) was initiated. Topical anti-inflammatory and anti-glaucomatous treatment were continued. The uveitis improved over the following six months. Rubeosis iridis regressed in the right eye, although vision remained at light perception due to corneal opacification, cataract, and presumed glaucomatous optic nerve damage. On the left, the corneal pannus and infiltrates remained stable with fluctuating intraocular pressures requiring the addition of oral antiglaucoma therapy with methazolamide. The visual field

test was within normal limits OS), with no detectable abnormalities. Visual acuity in the left eye stabilized at 20/30.

Discussion

Incident of leprosy has been declining, largely due to coordinated efforts and campaigns by the World Health Organization (WHO). Since 1981, multidrug therapy (MDT)—consisting of dapsone, rifampicin, and clofazimine—has been the standard treatment, and since 1995 the WHO has provided this regimen free of charge. However, even today, the social stigma of leprosy and the lack of suspicion of this rare disease on the part of physicians in developed societies may delay appropriate diagnosis and care.

Most patients with Leprosy are asymptomatic. But those who do develop symptoms may present along two distinct spectra of the disease. Although less common than keratitis, uveitis is a frequent manifestation in these patients due to the predilection of *Mycobacterium leprae* for invading the iris and ciliary body. Iridocyclitis may occur through three mechanisms: direct invasion, sympathetic denervation, and autoimmune response. This is evidenced by the reduction of T-suppressor cells during acute lepromatous uveitis and vasculitis/perivasculitis in iris biopsy with low-grade iridocyclitis, suggesting an immune-complex-mediated reaction.

The chronic iridocyclitis in our patient with borderline lepromatous leprosy could have been caused by a combination of (a) persistent organisms as demonstrated by iris biopsy, resulting in cell-mediated reactions such as iris granuloma formation by macrophages, epithelioid cells, and T-lymphocytes, and (b) autoimmune phenomena as evidenced by increased circulating immune complexes. The worsening of symptoms after the application of periocular steroids, however, suggests that infection was the main pathogenic factor in our patient's ocular disease. It is important to note that transseptal corticosteroids should be used with the utmost caution in uveitis cases in which infection has not completely been ruled out.

Miliary lepromas of the iris or "iris pearls", which developed in our patient during follow-up, are pathognomonic features of leprous eye involvement. M. leprae may localize to the iris very early during dissemination of the organisms throughout the body and may multiply in stromal mononuclear cells which take on the appearance of foam cells. Such cells containing "globi" composed of closely packed acid-fast bacilli coalesce and become clinically visible as iris lepromas. "Iris pearls", however, are rarely discovered without evidence of previous or acute iris inflammation as in our patient.

Glaucoma is supposedly an uncommon complication of leprosy, and decreased intraocular pressures were found in the majority of patients with leprous iridocyclitis in one

study. Walton and coworkers, however, reported glaucoma in 10% of their leprosy patients which in most cases was secondary to uveitis.

The eyes with lepromatous leprosy may harbor living organisms or antigens long after the skin is bacteriologically negative. In one study 24% of patients had ongoing eye problems after completion of multidrug therapy, indicating that regular ocular examinations are necessary even after the systemic disease is controlled. The appearance of new ocular lesions in a leprosy patient may be the first sign of incomplete treatment or relapse of the leprosy disease, as demonstrated in our patient.

Our patient had been treated with Dapsone and clofazimine, two bactericidal medications for M. leprae. The duration of the therapy was probably adequate to achieve an intermittent bacteriological "cure" as evidence by negative skin smears immediately after treatment; however, this treatment was inadequate to eliminate the bacteria from the eye.

The WHO study group recommends treating "borderline" lepromatous leprosy with multidrug therapy consisting of dapsone (100 mg daily), rifampicin (600 mg once monthly), and clofazimine (300 mg once monthly and 50 mg daily) for at least two years, and, when possible, until skin smears become negative. The protocol emphasizes that rifampicin should always be included in combination with dapsone because of its high bactericidal activity. However, this patient had not received rifampicin as part of his prior therapy, and his infection may therefore be dapsone-resistant. Additionally, longer treatment courses than those recommended by the WHO for systemic disease may be necessary to treat ocular involvement in leprosy.

During ocular involvement with M. leprae, acid-fast organisms have been identified in the aqueous humor and in a scleral nodule of a patient with lepromatous iridocyclitis. The diagnosis in our patient was ultimately established by demonstrating M. leprae in aqueous humor and involved iris tissue.

In conclusion, although challenging, leprosy should be included in the differential diagnosis of uveitis that is unresponsive to conventional therapies. Clinical suspicion and a systematic diagnostic approach are essential for identifying the underlying infection and ensuring appropriate treatment.

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