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ORIGINAL ARTICLE

Chronic Unilateral Uveitis as a Manifestation of Leprosy: A Case Report and Literature Review

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ABSTRACT

Purpose: To describe a case of leprosy presenting chronic anterior uveitis associated with other systemic lesions.

Methods: Case report and systematic literature review.

Results: We describe the case of a 65-year-old patient presenting clinical features of chronic uveitis and poor response to topical and intravitreal steroid treatment. Upon ocular examination, diffuse iris atrophy and macular edema were observed and laboratory tests for autoimmune and infectious diseases were within normal range. Physical examination revealed the presence of skin lesions on trunk and extremities, which were biopsied and identified as positive for leprosy.

Conclusion: The case reported herein presented atypical characteristics of uveitis due to the involvement of the posterior segment of the eye. Leprosy diagnosis could be a challenge, a systematic approach is mandatory to achieve adequate treatment.

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Leprosy; leprosy; lepromatous; uveitis; iris; macular edema

Leprosy, also known as Hansen's disease, is a chronic infectious granulomatous disease caused by *Mycobacterium leprae*, a bacillus first described in 1873 by Gerhard Armauer Hansen in Norway[1] Leprosy usually affects the peripheral nervous system, the musculoskeletal system, organs such as skin, mucosa, testicles, and eyes. Leprosy is a contagious disease transmitted by breathing airborne droplets of infected individuals; however, it is considered to be only mildly contagious.[1] The first historical records associated with this disease date back to 400 B.C. in the ancient Egyptian Empire, China, and India. During different time periods, leprosy was associated with negative beliefs, those who suffered it were exposed to great compassion and humiliation since this disease was identified with sin, dirt; patients were isolated in specialized centers designed for the sole purpose of confining "lepers."[2]

In 1941, a drug derived from dapsone was identified for the treatment of leprosy; in 1981, a multidrug chemotherapy with dapsone, rifampicin, and clofazimine revolutionized management of this disease. Since then, this multidrug regimen has been promoted by the World Health Organization (WHO).[3]

Moreover, since 1995, the WHO has distributed the multidrug treatment (MDT) free of cost causing incidence rates to be significantly reduced, so much, that leprosy was removed from the list of public health problems in the year 2000.[4] Despite this, however, incidence of leprosy remains significant in some areas, which according to reports from the year 2014 it was estimated to have a global prevalence of 174,608 cases, with an incidence rate of 3 cases per 100,000 inhabitants, is among the most affected countries Bangladesh, India, Indonesia, and Brazil.[5]

In Colombia, according to the National Institute of Health and the epidemiological surveillance system (SIVIGILA), in 2018 a total of 150 cases were reported in Colombia, of which the majority were identified in two Departments, Valle del Cauca and Santander. Nonetheless, 75.6% of the territorial entities also notified leprosy cases, most of which were of the lepromatous type with an associated disability reported in up to 40.6% of the cases.[6]

In 1966, the Ridley-Jopling classification system of leprosy was described. This system takes into account clinical, immunological, and bacteriological spectra of this disease, ranging from low count of microorganisms accompanied by a strong immune response (tuberculoid) to a high count of microorganisms accompanied by a weak immune response (lepromatous). In total, the Ridley-Jopling classification system includes six categories: tuberculoid (TT), borderline tuberculoid (BT), borderline (BB), borderline lepromatous (BL), lepromatous (L), and indeterminate (I).[7] Subsequently, in order to facilitate and determine treatment in regions with less access to healthcare, the WHO classified leprosy based on clinical manifestations and skin smears as paucibacillary leprosy (PB, five or less skin lesions and negative smears) and multibacillary leprosy (MB, six or more skin lesions and positive smear).[8]

Leprosy may cause blindness in up to 3.5% of the patients, usually as a consequence of lesions of the fifth and seventh cranial nerves due to direct bacterial invasion of the ocular globe.[9] A wide range of ocular manifestations have been reported for leprosy, including lagophthalmos, madarosis,

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corneal ulcers, cataracts, and uveitis in up to 4% of the cases. In addition, the presence of iridocyclitis has been explained by the persistence of mycobacterium in ocular structures, in its chronic form may lead to pupillary seclusion and thinning of the iris stroma.[10]

We describe the case of a patient with chronic unilateral uveitis, evaluated at an outpatient ophthalmology clinic in the city of Medellin, Colombia, who at ocular and dermatological examination exhibited clinical signs highly indicative of Hansen's disease, which were subsequently confirmed and correlated with histopathological studies.

The patient provided informed consent for the publication of his clinical case and photographs.

For the literature review, a search was performed in PubMed, ClinicaKey, and SciELO regional databases using the following descriptors: Leprae, Leprosy, Hansen disease. Articles reporting on aspects of leprosy including background, epidemiology, definition, classification, clinical manifestations, pathogenesis, etiology, treatment, course of disease, and prognosis were selected for review.

Clinical case

A 65-year-old man, who worked as a distributor of agricultural products, was referred to our clinic by a rheumatologist as he was diagnosed with chronic anterior uveitis in his right eye. During the previous 3 years, he had been under multidisciplinary management by specialists in rheumatology, neurology, dermatology, and ophthalmology due to the following diagnoses:

- Demyelinating polyneuropathy in upper and lower limbs;
- Urticarial vasculitis, treated with azathioprine (50 mg bid) and cyclosporine (50 mg qd), without improvement.
- Chronic uveitis in right eye, initially diagnosed as Fuchs heterochromic iridocyclitis, treated with steroids, without improvement. Subsequently was diagnosed as cystoid macular edema and treated with intravitreal dexamethasone implant, without improvement, then, was referred to our service for further evaluation.

The patient presented to our clinic with systemic symptoms explained by his past medical history referring only decreased visual acuity. Upon ocular examination, his best-corrected visual acuity was 20/150 in the right eye and 20/20 in the left eye. Slit-lamp biomicroscopy of the right eye showed diffuse keratic precipitates, anterior chamber cells grade 0.5+, diffuse iris atrophy (Figure 1a), and intraocular lens in capsular bag. Ocular examination of the left eye was unremarkable (Figure 1b).

Fundus examination of the right eye showed areas of local and diffuse atrophy of the retinal pigment epithelium (RPE), with no evidence of active choroiditis or signs of peripheral vasculitis. In addition, a central macular thickening with presence of epiretinal membrane (ERM 0) was also observed. Fundus examination of the left eye was unremarkable. Intraocular pressure (IOP) was of 18 mmHg and 12 mmHg in his right and left eye, respectively.

Physical examination revealed erythematous-violaceous skin lesions with irregular and poorly defined borders, of variable sizes ranging from 1 to 2 cm, some of which merged and formed larger lesions, located in posterior region of neck, upper back, extensor surface of arms, and forearms. Some of the lesions exhibited loss of hair follicles and were anesthetic (Figure 2).

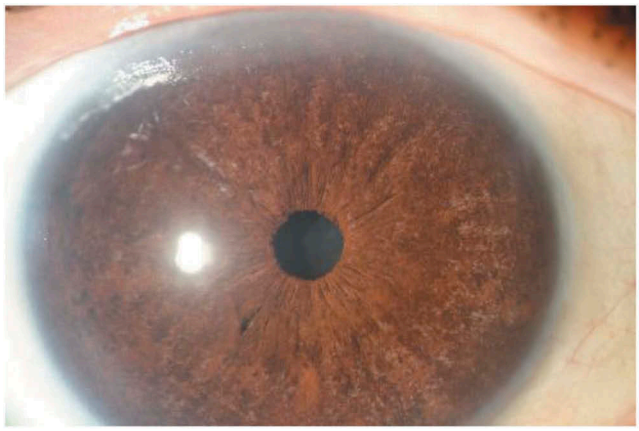

Figure 1. (a) Right eye. Diffuse iris atrophy. (b) Left eye.

In view that the patient was presenting non-granulomatous chronic uveitis of unknown etiology, we requested immunological

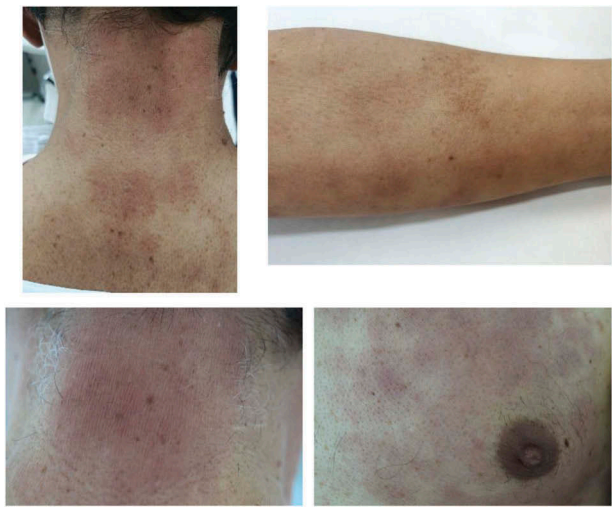

Figure 2. Erythematous-violaceous skin lesions, some of the lesions exhibited loss of hair follicles.

Table 1. Laboratory tests.

Test	Result	Test	Result
HLA B27	Negative	ANAS	Negative
PPD	Negative	ENAS	Negative
Ac VIH	Negative	Ac VHB	Negative
FTA-ABS	Negative	Ac VHC	Negative

and infection profile (Table 1), macular optical coherence tomography (OCT), and biopsies of skin lesions. Laboratory tests to assess immunological and infection profile were unremarkable.

Macular OCT revealed a central thickness of 345 m, with an abnormal anatomical distribution and multiple cystic spaces involving the inner and outer nuclear layers, confirming a macular edema of probable mixed etiology (uveitis and ERM) (Figure 3).

Skin biopsy revealed findings consistent with Hansen's disease, thus clearly and decisively establishing the etiology of the observed systemic, dermatologic, and uveitic symptoms (Figure 4a). Skin smear examination showed bacterial index of 2+ and presence of globi (Figure 4b).

Therefore, a diagnosis of multibacillary borderline lepromatous leprosy was made, other members of the multidisciplinary team were immediately informed about our findings and diagnosis. Comprehensive management and treatment of his disease were initiated with rifampicin, clofazimine, and dapsone.

To date, the patient is followed by neurology, rheumatology, dermatology, and ophthalmology. His uveitis has significantly improved, currently is undergoing treatment for his systemic disease.

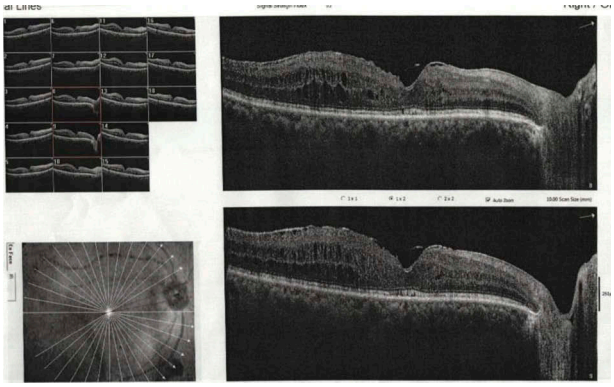

Figure 3. Macular OCT. Right eye. Showing a macular edema.

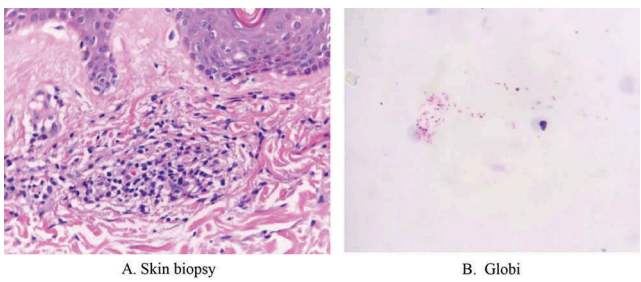

Figure 4. (a) Skin biopsy. (b) Globi.

Discussion

Leprosy is a chronic granulomatous disease with high morbidity. Despite multiple campaigns aiming to eradicate this disease, leprosy is still endemic in 122 countries, some of them in Latin America. For instance, Brazil needed to implement programs for early detection and treatment in order to reduce the number of patients with physical disabilities associated with leprosy.[11] In Colombia, leprosy cases are usually reported in municipalities in which the majority of the population resides (66%), meaning that almost 70% of the Colombians could be exposed to this bacillus and thus possibly get infected. In our case, this patient was required to travel to multiple places within Colombia for work-related reasons, thus being constantly exposed to risk of infection given the local epidemiology of leprosy.[12]

Leprosy is a contagious disease transmitted by breathing airborne droplets of infected individuals; however, the majority of patients do not develop the disease, largely because the host immune response is the one responsible for the emergence of symptoms and clinical manifestations of leprosy. Although humans have been described as the major reservoir for *Mycobaterium leprae*, in regions such as Latin America, armadillos have also been found to serve as reservoirs of infection.[13]

The type of leprosy that clinically develops in each patient depends on the host immune response, which is taken into account in the Ridley-Jopling classification system, as it is based on the type of skin lesion and bacterial load. Patients with tuberculoid leprosy have a good cellular immune response, in general present fewer lesions and a low bacterial load. On the other hand, patients with lepromatous leprosy have a humoral immune response and multiple skin lesions. The simplified classification of leprosy proposed by the WHO, aiming to facilitate and determine treatment in regions with less access to healthcare, refers to paucibacillary leprosy (PB, five or less skin lesions and negative smears) and multibacillary leprosy (MB, six or more skin lesions with positive smear). Therefore, under this system, our patient was classified as having multibacillary lepromatous leprosy.[14,15]

Mycobacterium leprae is an obligate intracellular rod-shaped bacterium, affecting mainly peripheral nerves due to its tropism for Schwann cells, macrophages, and skin. In addition, this bacillus is commonly found in cooler places of the body such as the nasal mucosa and anterior eye chamber. However, as previously mentioned, clinical manifestations are a direct consequence of the host immune response.[16]

Histopathological diagnosis can be achieved by using dyes such as Fite-Faraco or hematoxylin-eosin stains, which expose the pathogen despite it being an acid- and alcohol-fast bacterium. Lepromatous leprosy is characterized by the presence of numerous bacilli that form clusters referred to as globi[17], as was the case in our patient whose skin smear was reported as grade 2+ and globi was also identified.

Of bacterial infections, leprosy presents the highest incidence of ocular involvement, mostly in the anterior chamber due to its lower temperature compared to the rest of the eye. It has been proposed that *M. leprae* enters via blood vessels of the ciliary body, reaching the iris as an immune sanctuary site through small autonomic nerves and being able to extend as a retrograde axonal degeneration.[18]

In leprosy, inflammation episodes may be of two types: Type I, also known as reversal reaction, and Type II, or erythema nodosum. In type I there is a delayed hypersensitivity reaction that is associated with tuberculoid and borderline leprosy, in which the most frequent ocular is lagophthalmos. On the other hand, in the erythema nodosum reaction, there is a strong immune response that is associated with lepromatous leprosy, the ocular involvement is mostly uveitic and corneal.[19]

Ocular damage in leprosy is achieved by four mechanisms: 1) direct mycobacterial infection, 2) involvement of fifth and seventh cranial nerves, 3) reversal reaction due to increased activity of the immune system, 4) by erythema nodosum.[20]

Involvement in eyelids and annexes may be extensive, initiating with madarosis or loss of eyebrows due to the direct infiltration of the bacilli into the hair follicles and atrophy, which can be followed by other anomalies such as trichiasis, ectropion, entropion, blink reflex alteration, and ptosis. Parikh and colleagues described a series of patients with multibacillary leprosy treated with multidrug therapy in which the prevalence of lagophthalmos was higher in older patients and in those presenting the tuberculoid form of the disease.[21]

On the ocular surface, conjunctivitis, although rare, is produced by primary infection. However, there is major corneal involvement in patients presenting lepromatous leprosy, since the bacilli can directly infiltrate unmyelinated nerves of the corneal stroma producing small calcification and in addition, significant corneal hypoesthesia, particularly in patients with the multibacillary form of the disease.[22]

Furthermore, treatment with clofazimine has been linked with crystalline keratopathy in leprosy patients, associated with duration of treatment.[20]

Uveitic involvement in leprosy patients predominantly affects the iris, due to the preference of the bacilli for cooler places in the body. Three pathogenic mechanisms have been described to cause iridocyclitis: by persistence of *M. leprae* bacilli in ocular structures, through neuroparalysis and an autoimmune response. When the cause of inflammation is direct invasion of this pathogen, the clinical presentation is usually acute with florid symptoms such as photophobia, pain, reduced visual acuity, keratic precipitates – which are more common in lepromatous and borderline types of leprosy.[23] On the other hand, when the inflammatory process is chronic and dormant, the pathophysiological mechanism is by sympathetic denervation of the iris that leads to its atrophy and is also known as neuroparalytic uveitis. Clinical manifestations include, in addition to atrophy of the iris, synechiae, miosis (punctiform pupils) and presence of iris pearls, which are considered by some authors to be pathognomonic of iriditis caused by *M. leprae* bacillus.

Immune-complex-mediated iridocyclitis is a result of the cellular immune response, mainly by T cells and is associated with the presence of granulomas in the iris.[24] Our patient presented keratic precipitates and diffuse iris atrophy, which is similar to that reported in the literature for patients with multibacillary lepromatous leprosy.

Iris atrophy results as a consequence of the lesion to the muscle or nerve fibers. Iris pearls, also known as military lepromas, are spherical, creamy white or pale yellow and opaque lesions. The most currently accepted hypothesis explaining the presence of iris pearls proposes that after bacillary invasion, a few mononuclear cells migrate to the stroma of the iris forming foam cells that may remain in the iris for long periods of time without eliciting a visible inflammatory response in the anterior chamber. Within these foam cells, bacilli are able to replicate and form small colonies that over time can increase in size and merge, thus becoming visible under slit-lamp biomicroscopy.[25,26] In general, size of iris pearls is usually 0.5 mm, but can reach up to 2 mm, contrary to Gilbert-Koeppe nodules, are located deep in the stroma and they develop independently of acute inflammatory signs.[27]

Pinpoint pupils have also been described in leprosy patients, explained by the autonomic denervation of the anterior segment. In addition, case series from India have described a substantial increase in incidence of cataracts, particularly posterior subcapsular cataracts, in leprosy patients. However, a direct association with the disease has not been elucidated given that the majority of patients were of older age and had previous use of steroids.[28]

As in unusual pathologies, differential diagnosis should be considered including Fuchs heterochromic iridocyclitis, which was the initial diagnosis in this case, and viral uveitis.[29] Fuchs uveitic syndrome, as mentioned in the literature, is characteristically monocular associated with central and stellar keratic precipitates. In the iris, anterior stromal atrophy can be found, it is also seen in the infection caused by leprae, although in Hansen´s disease seems to be deeper. It is infrequent to find posterior compromise as cystoid macular edema, usually, patients with Fuchs iridocyclitis are not very symptomatic or present with significant decreased vision, but present with cataract or glaucoma.[30] Our patient, although with monocular symptoms, had severe decreased vision and associated cystoid macular edema. In viral uveitis, the course is usually self-limited and is associated with the systemic infectious peak. Acute retinal necrosis caused by viruses of the Herpes family presents with severe and marked inflammatory symptoms associated with focal chorioretinal and vasculitic lesions.

Finally, it is worth to point out that even though great efforts have been made to eradicate leprosy, as it is uncommon to find patients presenting catastrophic manifestations, this infection may cause blindness not only due to the previously described lesions but also due to infiltration of the ciliary body, leading to its atrophy and subsequent phthisis bulbi.[31]

The treatment recommended by WHO for paucibacillary leprosy is based on a regimen including rifampicin and dapsone; for patients with multibacillary leprosy, clofazimine should be added. The duration of treatment is at least 6 months but may be extended to 12 months. Rates of relapse and resistance to this regime are low, being of 3:100,000 per treated patients and are usually associated with improper use of medications, for example, when used as monotherapy.[32]

In conclusion, ocular leprosy has been widely reported throughout the literature, despite its multiple ocular manifestations involving annexes, ocular surface, and uveal tract, diagnosis is challenging due to its variable clinical characteristics.

In leprosy, ocular involvement is generally found in the anterior segment, being common manifestation madarosis and lagophthalmos in the lepromatous type of leprosy. In the cornea, bacilli may directly infiltrate unmyelinated nerves of the stroma leading to small calcifications and hypoesthesia, with risk of corneal ulceration.

The clinical case herein presented, while initially requiring only ophthalmological evaluation, illustrates how challenging a diagnosis may be, and the relevance of approaching uveitis as a systemic disease, thus compelling the specialist to perform a thorough physical examination in order to reach seemingly unusual, yet accurate, diagnoses.

Disclosure statement

The authors declare no conflict of interest.

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