Uveitic Glaucoma and Hansen’s disease, A case report

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ABSTRACT

Background: The number of Hansen’s disease cases in Latin America and the Caribbean has decreased in the last decade; nevertheless, the region is still struggling with infections caused by Mycobacterium leprae. This is a case report that portrays the diagnostic and management challenges associated with atypical uveitic glaucoma that is due to Hansen’s disease.

Case presentation: A 62-year-old female was referred with a 2-year history of anterior uveitis of unknown etiology and ocular hypertension. Past medical history and general physical examination were unremarkable. Upon ocular examination, her best-corrected visual acuity (BCVA) was 20/25 in the OD and 20/60 in the OS. Tonometry showed intraocular pressures (IOPs) of 29 mmHg and 22 mmHg in her right and left eyes, respectively. The slit-lamp examination showed clinical signs of bilateral granulomatous anterior uveitis and cataracts; gonioscopy revealed open angles with some peripheral anterior synechiae for both eyes. Fundus examination and glaucoma tests revealed mild glaucomatous damage in the right eye. Given the presentation of uveitis, the respective questionnaire was completed by internal medicine and rheumatology. Four months later, after bilateral cataract surgery, the patient developed skin plaques on the face, neck, upper back, and extremities, which were biopsied and identified as positive for tuberculoid leprosy.

Conclusion: This is the first case report in Ecuador of atypical glaucoma triggered by infectious uveitis produced by Mycobacterium leprae. We describe a female patient’s clinical presentation with several ocular signs of leprosy and other non-specific and rarely seen symptoms. Uveitis is a condition that often requires a multidisciplinary team of ophthalmologists and clinicians because of the possible manifestation of an underlying systemic disease, creating a challenge for all the medical personnel involved in the management of the case.

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1. Background

Leprosy, one of the most common diseases documented throughout human history, is still present among patients with ocular morbidity. As Hansen stated, “there is no disease which so frequently gives rise to disorders of the eye as leprosy does.” The microorganism responsible for causing leprosy is Mycobacterium leprae, an acid-fast-stained bacilli, discovered by Gerhard Henrik Armauer Hansen in 1874. This disease, previously attributed to witchcraft, was responsible for millions of deaths before the arrival of antibiotics. The main features of this disease are skin ulcers, lack of skin sensitivity, muscle weakness, destruction of the nasal appendix, absence of hair on the eyebrows and eyelashes, changes in pigmentation, and diffuse involvement of the facial skin causing leonine facies, peripheral nervous system alterations and upper respiratory tract mucusa and eyes affections.

This infection is transmitted via airborne droplets of infected individuals; however, there have been reports of trauma-related transmission and zoonotic cases, which were the result of contact with armadillos and environmental reservoirs such as water sources. Mycobacterium leprae replicates at temperatures of approximately 30 °C; therefore, it has a preference for low-temperature body areas, such as the peripheral nervous system, musculoskeletal system, upper respiratory tract, skin, mucosa, testicles, and anterior chamber of the eye. Because of the deformities and disabilities associated with the infection, patients throughout history have suffered from discrimination and stigmatization. In fact, the name leprosy derives from the Latin word lepros, which means defilement.

Leprosy is a disease that has been forgotten because of its low prevalence. In 2015, World Health Organization (WHO) reported 176,176 cases, calculating a prevalence of 0.2 cases per 10,000 people.

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In Ecuador, which is located in South America, the incidence reported in 2019 was less than 1 case per 100,000 persons, and was mainly concentrated in the Amazonian region of the country. In 2015, there were 178 new cases in the United States, predominantly in the states of Arkansas, California, Florida, Hawaii, Louisiana, New York, and Texas. Centers for Disease Control and Prevention (CDC) estimates that 5000 people in the United States have been cured but suffer from long-term sequelae such as blindness.

The Ridley and Jopling classification takes into account clinical, pathological, bacilloscopic, and immunological criteria to classify leprosy into six forms: tuberculoid (TT), borderline tuberculoid (BT), mid-borderline (BB), borderline lepromatous (BL), lepromatous (LL), and indeterminate (I). Lepromatous leprosy patients tend to have more ocular complications and vision impairment \((p = .037)\) than patients with tuberculoid leprosy, borderline tuberculoid, and indeterminate leprosy.

The WHO has proposed a more straightforward classification for treatment purposes, based on cutaneous manifestations and skin smears, which categorizes leprosy into 1) paucibacillary (PB): \(\leq 5\) skin plaques and negative smears and 2) multibacillary (MB): \(\geq 5\) skin plaques or positive smears. Up to 75% of individuals with leprosy have ocular manifestations, and 39.40% have a visual disability. There are a wide variety of ocular manifestations, such as lagophthalmos, madarosis, corneal ulcers, cataracts, uveitis, and iridocyclitis. It has been proposed that *Mycobacterium leprae* has a preference for the iris, which is due to its safety from the immune system and systemic treatment. The diagnosis of ocular leprosy is challenging in the absence of characteristic skin lesions because of its diverse ocular manifestations and extensive time for development. We describe the first case of a patient with bilateral cataracts and uveitic glaucoma secondary to tuberculoid leprosy confirmed by histopathological studies in Quito, Ecuador.

2. Case presentation

A 62-year-old female was referred to our clinic by an ophthalmologist, as she was diagnosed with anterior uveitis of unknown etiology and ocular hypertension. During the past two years, the patient had suffered from elevated intraocular pressure (IOP) in both eyes (OU) refractory to topical therapy and bilateral anterior uveitis refractory to corticosteroid therapy. For her IOP, the patient was on a fixed combination of timolol-dorzolamide bid in OU, and only timolol bid in her right eye (OD). The previous corticosteroid treatment that the patient followed whenever a crisis reappeared was topical prednisolone acetate every 3 h. This medical regimen was maintained for a year. She was not on systemic medications.

The patient presented to our clinic complaining of conjunctival injection in OU, persistent headache, eyelid irritation, and a decrease in visual acuity in the OS. The physical exam revealed mild ptosis in OU and no other findings. Upon ocular examination, her best-corrected visual acuity was 20/25 in the OD and 20/60 in the OS. IOP was 29 mmHg and 22 mmHg in her OD and OS, respectively.

The slit-lamp examination in OU showed the following findings: erythema and scales at eyelid margins, decreased tear break-up time, cornea with diffuse punctate epitheliopathy, mutton-fat keratic precipitates, symmetric, round and reactive pupils, sparse patches of iris atrophy, anterior chamber cells +, posterior subcapsular opacities in both eyes denser in the OS, and clear vitreous. Gonioscopy showed open angles in OU and peripheral anterior synechiae (PAS) in the inferior quadrant in the OD and in the superior quadrant in the OS (Fig. 1).

The fundus examination revealed a cup disc ratio of 0.55 \(\times\) 0.45 in the OD with thinning of the inferior neuroretinal rim and localized loss of the retinal nerve fiber layer (RNFL) in the inferior quadrant; the cup disc ratio in the OS was 0.4 \(\times\) 0.3. The macula, blood vessels, and peripheral retina were unremarkable as well as fluorescein angiography of the retina in OU. The visual field showed a mild superior arcuate defect and a glaucoma hemifield test (GHT) “outside normal limits” in the OD. The GHT in the OS showed “general depression of sensitivity.” The RNFL deviation map in the optical coherence tomography (OCT) showed a loss of nerve fibers in the inferior quadrant in the OD and no abnormal OS results. The average RNFL thickness was within normal parameters OU; however, RNFL symmetry was 71%. The deviation map in the macular ganglion cell analysis revealed mild thinning in the temporal quadrant in the OD and no abnormal findings in the OS. The average ganglion cell layer (GCL) plus inner plexiform layer (IPL) thickness was in the normal range in OU. The macular thickness OCT showed a central thickness of 241 \(\mu\)m in the OD and 247 \(\mu\)m in the OS. Specular microscopy indicated a cell density of 2387 cells/mm\(^2\) in the OD and 2625 cells/mm\(^2\) in the OS.

The patient was diagnosed with anterior uveitis and cataracts in OU, uveitic glaucoma in the OD, and ocular hypertension in the OS. Given the presenting chronic granulomatous uveitis of unknown etiology, the respective questionnaire and evaluation were performed by internal medicine and rheumatology. This multidisciplinary team ordered laboratory tests to assess immunological and infectious profiles (Table 1). The serological antibodies for toxoplasmosis, rubella, syphilis, tuberculosis, and cytomegalovirus (CMV) showed no active infection. Aqueous tap was also performed to study different infectious etiologies with DNA-PCR. CMV, HSV-1, HSV-2, and Toxoplasma gondii had undetectable levels, meaning a negative result. A smear and culture of the aqueous humor were not done due to insufficient sample quantity; therefore, the DNA-PCR tests were prioritized. The immunological tests were negative for rheumatoid factor (RF) and antinuclear antibodies (ANAs). Anti-neutrophil cytoplasmic antibodies (c-ANCA and p-ANCA) were positive. Because of these results, the patient underwent a complete evaluation and imaging workup that did not reveal a conclusive diagnosis.

To manage the ocular conditions, her treatment was changed to a fixed combination of timolol-dorzolamide-brimonidine bid in OU, lowering the IOP to the low teens in OU. For her anterior uveitis, loteprednol was indicated qid in OU for one month, obtaining optimal control. The patient underwent femtosecond laser-assisted cataract surgery (FLACS) in OU, with an interval of one month between each eye. There were no complications. The postoperative IOP was 15 mmHg in the OD and 14 mmHg in the OS with the same topical medication. The BCVA was 20/25 in the OD and 20/20 in the OS. There were no postoperative changes in structural and functional glaucoma tests.

Four months after her last cataract surgery, the patient started to present systemic and ocular symptoms, such as headache, skin lesions, arthralgias in fingers and wrists, ocular pain, blepharitis conjunctival injection, blurry vision, and photophobia. The physical exam revealed erythematous skin plaques with irregular and poorly defined borders with clear centers over the face, neck, upper back, forearms, legs, and dorsum of hands (Fig. 2). Some of the skin plaques were anesthetic. At the ocular examination, the BCVA was 20/30 in the OD and 20/40 in the OS; the IOP was 32 mmHg in OU, despite maximal topical treatment.
nerve fascicles (Fig. 4B). The findings were consistent with tuberculoid (Fig. 4A). Fite Faraco staining revealed scarce acid-fast bacilli within.

...disease. The rarity of cases disease.

Diagnosis of ocular tuberculoid leprosy was made based on discard and skin incisional biopsies.

With the diagnosis, comprehensive treatment with a multidrug regimen of rifampicin, clofazimine, and dapsone was initiated. With this treatment, the patient’s systemic symptoms, such as skin lesions and arthralgia, as well as uveitis, went under control. However, her IOPs in OU were in the high teen mmHg despite being on maximal topical therapy, which was due to the damage in the TM and the increased amount of synechia in the angles. The structural and functional tests showed progression of glaucomatous damage in OU (Fig. 5). To ensure further control of the IOPs, the patient underwent trabeculectomy with mitomycin-C in OU without complications.

3. Discussion and conclusions

Leprosy is a chronic granulomatous disease with a decreasing incidence, which is mainly due to efforts and campaigns coordinated by the WHO. Since 1981, multidrug treatment (MDT), consisting of dapsone, rifampicin, and clofazimine, has been the standard therapy, and since 1995, the WHO has distributed this MDT free of cost. The rarity of cases has caused medical providers to be unfamiliar with it, leading to a misdiagnosis or a late one. With the increasing number of people living in unsanitary conditions and with limited healthcare access that is due to global immigration, refugee crises, and homeless situations, the incidence and prevalence patterns of leprosy might change. Because the ocular manifestations can be as severe as blindness, it is relevant to address this forgotten disease.

Even though the majority of patients are asymptomatic, those with symptoms can develop two possible spectra of the disease. The tuberculoid leprosy spectrum is associated with a strong cellular immune response, while the lepromatous leprosy spectrum is associated with a humoral immune response. In histologic cuts with Fite Faraco and Ziehl Neelsen stains, tuberculoid leprosy presents with inflammatory infiltrate in the dermis and epidermis, epithelioid histiocytes surrounding small cutaneous nerves, and scarce bacilli, as was seen in our patient.

Regarding immunological tests, there are reported cases of positive ANA, ANCA, and RF, with c-ANCA being the most common antibody.

The ocular signs and symptoms of leprosy arise from different mechanisms, such as direct bacterial infection and trigeminal or facial nerve involvement. Direct invasion of hair follicles is responsible for madarosis and trichiasis, invasion of the eyelids and CN III invasion is responsible for ptosis, and invasion of CN VII is responsible for entropion, punctate keratitis and lagophthalmos. All these consequences could cause neurotrophic keratitis, which is responsible for corneal ulcers and scarring and can lead to blindness. The direct infiltration of unmyelinated nerves may also cause corneal hyposthesia, similar to the glove-and-stocking anesthesia seen in the extremities. On the ocular surface, conjunctivitis, conjunctival scarring, and pterygium can develop. Other adnexal effects of Mycobacterium leprae are entropion, blink reflex alteration, dacryocystitis, and blockage of the nasolacrimal duct.

Uveitis is a common presentation in these patients because of the preference of Mycobacterium leprae invasion to the iris and ciliary body. There are three possible mechanisms of iridocyclitis: direct invasion, sympathetic denervation, and autoimmune response. Direct invasion is associated with photophobia, pain, reduced visual acuity, and keratic precipitates. The sympathetic denervation of the iris, secondary to a chronic inflammatory process, is associated with iris atrophy, synchiae, punctiform pupils, and the presence of iris pearls. Iris atrophy is present in more than 25% of patients, making it the most common ocular lesion. The iris pearls, present in 4.8% of patients, are spherical white-yellow lesions considered pathognomonic of Hansen’s disease. Last, the autoimmune response is associated with the appearance of granulomas in the iris.

| Laboratory results for plausible immunological and infectious etiologies. | Result | Reference values |
|---|---|---|
| **VDRL** | Not reactive | |
| **Toxoplasma gondii** | | |
| IgG (U/mL) | 17.70 | Reactive: ≥ 6.5 |
| Not reactive: < 6.5 | |
| IgM (U/mL) | <0.9 | Reactive: > 1.1 |
| Not reactive: < 0.9 | |
| Undetermined: 0.9-1.1 | |
| **Rubella** | | |
| IgG (U/mL) | 15.20 | Reactive: > 10 |
| Undetermined: 5-9 | |
| Not reactive: < 5 | |
| **ANA** | | |
| IgG (U/mL) | 0.185 | Reactive: > 1.0 |
| Undetermined: 0.7-1.0 | |
| Not reactive: < 0.7 | |
| **ANCA** | | |
| IgG (U/mL) | 6.1 | Positive: ≥ 60 |
| Not reactive: < 60 | |
| **Rheumatoid factor** | | |
| IgG (U/mL) | 32.6 | Positive: > 100 |
| Not reactive: < 100 | |
| **Aqueous Humor PCR-DNA** | | |
| Cytomegalovirus | Not detectable | |
| HSV-1 and HSV-2 | Not detectable | |
| Toxoplasma gondii | Not detectable | |

VDRL: Venereal Disease Research Laboratory; PPD: Purified Protein Derivative. PCR: polymerase chain reaction.

Fig. 2. Disseminated erythematous skin plaques with irregular borders and clear centers in arms and upper-back.
Chronic inflammation triples the risk of cataracts, especially posterior subcapsular inflammation, with an incidence of 33.2%. In leprosy eyes with iris atrophy, cataract surgery is technically challenging, and studies have shown uncertain results. In this case, FLACS was the technique used based on the patient’s uveitis history. FLACS uses less time for ultrasound and fewer levels of phacoemulsification energy. These factors generate lower levels of anterior segment inflammation, making FLACS beneficial for uveitic patients.

Leprosy patients tend to have lower IOP because atrophic areas of the iris are more permeable to aqueous humor, while atrophy of the ciliary body decreases its production. However, glaucoma was recognized in 10% of leprosy patients, mostly secondary to uveitis. In these cases, glaucoma requires close and frequent surveillance because of the risk of recurrent hypertensive peaks and progression of glaucomatous damage, as in the case of our patient.

Anterior uveitis has a wide variety of etiologies from infectious to autoimmune. The first step is to perform a complete history and physical examination; for this case, it helped make some etiologies, such as juvenile rheumatoid arthritis and Posner-Schlossman syndrome, more unlikely. Then, a detailed slit lamp and ophthalmoscopic eye examination was crucial for observing different signs, such as trabecular meshwork nodules, vitreous opacities displaying snowballs and optic disc nodules, which are pathognomonic of sarcoidosis. None of these were found in our patient, making sarcoidosis more unlikely. For infectious etiologies, aqueous tap DNA-PCR and serological antibodies were crucial, discarding CMV, HSV-1, HSV-2, toxoplasmosis, rubella, tuberculosis, and syphilis. Finally, the excisional skin biopsy smear with staining was the key for making the diagnosis of *Mycobacterium leprae*.

In terms of treatment, an essential consideration is the side effect of cumulative clofazimine dose, which may cause crystalline keratopathy. Despite the completion of MDT, 24% of patients have a relapse of ocular manifestations, even with negative smears. This is due to the ability...
of *M. leprae* to persist inside iris macrophages. Therefore, regular ocular examinations are warranted even after completion of treatment.

As ophthalmologists, we should not forget that in a high percentage of cases, uveitis is an ocular manifestation of a systemic process. Hence, a multidisciplinary approach is needed to investigate all possible etiologies. This case illustrates how challenging the diagnosis, treatment, and follow-up can be.

**Declaration of competing interest**

The authors declare that they have no conflicts of interest.

**Abbreviations**

| Abbreviation | Full Form |
|--------------|-----------|
| ANA          | Antinuclear Antibodies |
| ANCA         | Antineutrophil Cytoplasmic Antibodies |
| BCVA         | Best-Corrected Visual Acuity |
| BT           | Borderline Tuberculoid |
| BB           | Mid-borderline |
| BL           | Borderline Lepromatous |
| CDC          | Centers for Disease Control and Prevention |
| CMV          | Cytomegalovirus |
| CN           | Cranial Nerve |
| FLACS        | Femtosecond Laser-Assisted Cataract Surgery |
| IOP          | Intraocular Pressure |
| IPL          | Inner Plexiform Layer |
| GCL          | Ganglion Cell Layer |
| GHT          | Glaucoma Hemifield Test |
| HVS          | Herpes Virus Simplex |
| I            | Indeterminate |
| LL           | Lepromatous |
| MDT          | Multidrug Treatment |
| OCT          | Optical Coherence Tomography |
| OS           | Left Eye |
| OD           | Right Eye |
| PAS          | Peripheral Anterior Synechiae |
| RF           | Rheumatoid factor |
| RNGL         | Retinal nerve fiber layer |
| TM           | Trabecular Mesh |
| WHO          | World Health Organization |

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**Patient consent**

The patient consented in writing to the publication of this case and associated images.

**Contributorship statement**

ARV, ERV, and AMV all contributed to the first draft and revision and approved the final version of the manuscript. All authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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