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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 nonspecific 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.

1. Background

Leprosy, one of the most common diseases documented throughout human history, is still present among patients with ocular morbidities.[1] As Hansen stated, "there is no disease which so frequently gives rise to disorders of the eye as leprosy does".[2] The microorganism responsible for causing leprosy is *Mycobacterium leprae*, an acid-fast-stained bacilli, discovered by Gerhard Henrik Armauer Hansen in 1874.[3] 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 mucosa and eyes affections.[4,5]

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.[6,7] *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.[3,6,8] Because of the deformities and disabilities associated with the infection, patients throughout history have suffered from discrimination and stigmatization.[3] In fact, the name leprosy derives from the Latin word *lepros,* which means defilement.[3]

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.[9]

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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.[10] 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.[11] 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.[12]

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).[7,13,14] Lepromatous leprosy patients tend to have more ocular complications and vision impairment ($p = .037$) than patients with tuberculoid leprosy, borderline tuberculoid, and indeterminate leprosy.[15]

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$6 skin plaques or positive smears.[13]

Up to 75% of individuals with leprosy have ocular manifestations, and 39.40% have a visual disability.[8,16] There are a wide variety of ocular manifestations, such as lagophthalmos, madarosis, corneal ulcers, cataracts, uveitis, and iridocyclitis.[6] 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.[17] 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.[18] 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 her left eye (OS) 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 1+, 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

Fig. 1. Gonioscopy exam reveals peripheral anterior synechiae (PAS) in OS.

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 μm in the OD and 247 μm in the OS. Specular microscopy indicated a cell density of 2387 cells/mm^3 in the OD and 2625 cells/mm^3 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 anesthesic. 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.

Table 1
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
IgM (U/mL)	<0.9	Reactive: > 1.1 Undetermined: 0.9–1.1 Not reactive: < 0.9
Mycobacterium tuberculosis		
PPD (mm)	<5	Positive: ≥ 10 Negative: < 10
Cytomegalovirus		
IgG (U/mL)	8.82	Reactive: > 1.1 Undetermined: 0.9–1.1 Not reactive: < 0.9
IgM (U/mL)	0.185	Reactive: > 1.0 Undetermined: 0.7–1.0 Not reactive: < 0.7
ANA		
	0.7	Positive: > 1.2 Undetermined: 1.0–1.2 Not reactive: < 1.0
ANCA		
	6.1	Positive: >1.0 Negative: < 1.0
Rheumatoid factor		
(U/mL)	32.6	Positive: > 60 Negative: < 60
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.

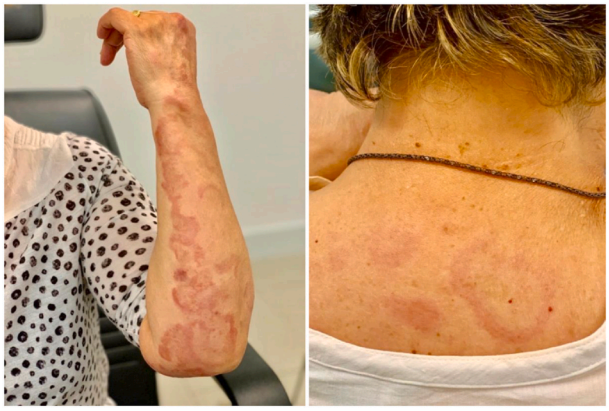

Fig. 2. Disseminated erythematous skin plaques with irregular borders and clear centers in arms and upper-back.

There were severe signs of inflammation on the anterior chamber in OU (Fig. 3) and opacification of the posterior capsule in the OS.

The patient was referred to dermatology for skin incisional biopsies, which revealed dense dermal lymphohistiocytic infiltrates along the superficial and deep vessels that compromised the nerve fascicles (Fig. 4A). Fite Faraco staining revealed scarce acid-fast bacilli within nerve fascicles (Fig. 4B). The findings were consistent with tuberculoid leprosy, which can explain both the skin and ocular symptoms. The 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 synechiae 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.[6] The rarity of cases has caused medical providers to be unfamiliar with it, leading to a misdiagnosis or a late one.[18] 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.[19] Because the ocular manifestations can be as severe as blindness, it is relevant to address this forgotten disease.[18]

Even though the majority of patients are asymptomatic, those with symptoms can develop two possible spectra of the disease.[6,7] The tuberculoid leprosy spectrum is associated with a strong cellular immune response, while the lepromatous leprosy spectrum is associated with a humoral immune response.[6] 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.[6,18,20] Regarding immunological tests, there are reported cases of positive *ANA*, *ANCA*, and RF, with *c-ANCA being* the most common antibody.[21]

The ocular signs and symptoms of leprosy arise from different mechanisms, such as direct bacterial infection and trigeminal or facial nerve involvement.[8] 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 ectropion, punctate keratitis and lagophthalmos.[8,15] All these consequences could cause neurotrophic keratitis, which is responsible for corneal ulcers and scarring and can lead to blindness.[8] The direct infiltration of unmyelinated nerves may also cause corneal hypoesthesia, similar to the glove-and-stocking anesthesia seen in the extremities.[6] On the ocular surface, conjunctivitis, conjunctival scarring, and pterygium can develop.[6,8] Other adnexal effects of *Mycobacterium leprae* are entropion, blink reflex alteration, dacryocystitis, and blockage of the nasolacrimal duct.[6,8]

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.[8] The sympathetic denervation of the iris, secondary to a chronic inflammatory process, is associated with iris atrophy, synechiae, 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.[8] The iris pearls, present in 4.8% of patients, are spherical white-yellow lesions considered pathognomonic of Hansen's disease.[6] Last, the autoimmune response is associated with the appearance of granulomas in the iris.[8]

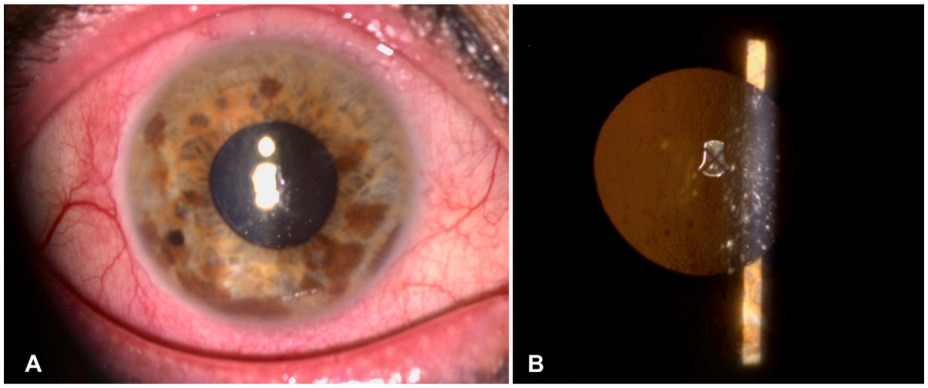

Fig. 3. A. Conjunctival injection and keratic precipitates in OD. **B.** Slit lamp exam reveals keratic precipitates in OS.

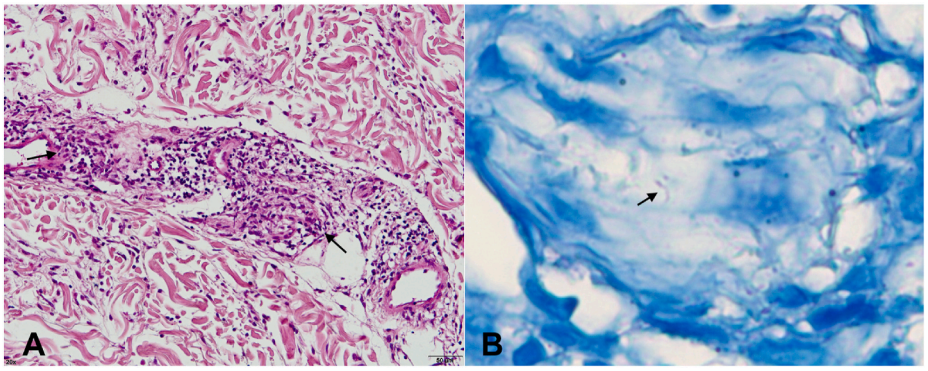

Fig. 4. A. Lymphohistiocytic perivascular infiltration (arrows) seen with Hematoxylin & Eosin stain. **B.** Bacilli in the middle of nerve (arrow) seen with Fite Faraco stain.

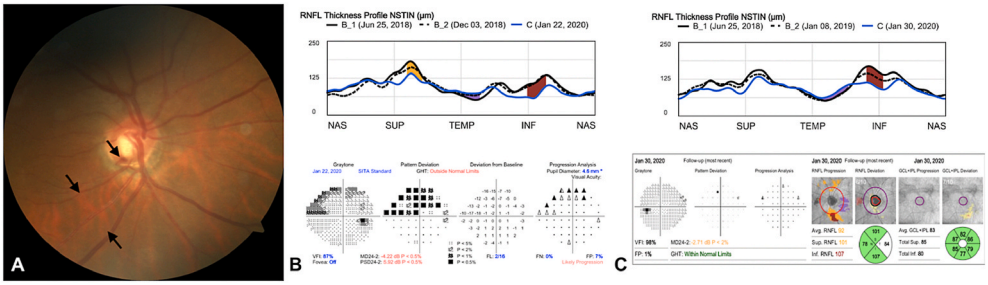

Fig. 5. A. Hemorrhage in the right optic disc with a loss of the RNFL in the inferior quadrant (arrows). **B.** Structure-Function Guided Progression Analysis (GPA) shows in the RNFL thickness profile (NSTIN) a progression of the thinning in the inferior quadrant, consistent with the progression of the superior arcuate defect in the visual field in OD. **C.** Structure-Function GPA shows in the RNFL thickness profile (NSTIN) a progression of the thinning in the inferior quadrant in OS.

Chronic inflammation triples the risk of cataracts, especially posterior subcapsular inflammation, with an incidence of 33.2%.[6,8,22] In leprosy eyes with iris atrophy, cataract surgery is technically challenging, and studies have shown uncertain results.[23] 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.[24] 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.[8,25] However, glaucoma was recognized in 10% of leprosy patients, mostly secondary to uveitis.[26] 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-Scholssman 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*.[27]

In terms of treatment, an essential consideration is the side effect of cumulative clofazimine dose, which may cause crystalline keratopathy.[8] Despite the completion of MDT, 24% of patients have a relapse of ocular manifestations, even with negative smears.[8,28] This is due to the ability

of *M. leprae* to persist inside iris macrophages.[8] 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

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
TT	Tuberculoid
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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