Recurrent corneal erosion (RCE) secondary to lattice dystrophy in a patient with acquired immune deficiency syndrome (AIDS)

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Background: We present a case of an Acquired Immune Deficiency Syndrome (AIDS) patient with recurrent erosion (RCE) secondary to lattice corneal dystrophy. As a human immunodeficiency virus (HIV)-infected patient becomes more immunocompromised, the ocular surface defense mechanisms may be compromised. Lattice dystrophy, RCE, and modifying approaches to the management of RCE in an HIV-positive or AIDS patient are reviewed.

Case Report: A 49-year-old man presented with RCE. His ocular history included lattice corneal dystrophy OU, recurrent corneal erosion O.S., and herpes simplex virus keratitis O.S. Systemic history included hepatitis C and HIV infection with a diagnosis of AIDS with secondary *Pneumocystis carinii* pneumonia. Viral load was 35533 HIV-RNA (ribonucleic acid) molecules/ml of plasma and CD4 lymphocyte count 99 cells/mm^3. Acuities were O.D. 20/80 and O.S. 20/50. The abrasion was treated with cycloplegia and bacitracin/polymyxin B ointment q.i.d. O.S. and it resolved in 3 days.

Conclusion: Management of lattice dystrophy with secondary RCE in an AIDS patient requires that the clinician be familiar with the patient's immune status. As the CD4 count declines and the viral load increases, the patient is at higher risk for opportunistic anterior segment infections. Clinicians need to monitor these patients closely for potential complicating ocular sequelae of AIDS such as herpes zoster ophthalmicus, herpes simplex keratitis, fungal/bacterial keratitis, and keratoconjunctivitis sicca. Our patient responded well to antibiotic therapy and cycloplegia. The importance of daily monitoring of immunocompromised patients is emphasized.

Key Words: Acquired immune deficiency syndrome (AIDS), human immunodeficiency virus (HIV) infection, recurrent erosion syndrome, lattice corneal dystrophy, corneal abrasion

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Patients infected with human immunodeficiency virus (HIV) can present with various corneal insults such as herpes zoster keratitis, herpes simplex keratitis, bacterial keratitis, microsporidial keratitis, fungal keratitis, and keratoconjunctivitis sicca keratitis.[1,2] Most of these sequelae are related to opportunistic infections with the exception of keratoconjunctivitis sicca. HIV-infected patients, like patients in the general population, can manifest a corneal dystrophy and associated complications such as recurrent corneal erosions. We present a case of a patient with acquired immune deficiency syndrome (AIDS) who had recurrent erosion secondary to lattice dystrophy. Management of a healthy patient with recurrent corneal erosions from lattice dystrophy can be clinically challenging. The factors causing a patient to be immunocompromised may further complicate the course of therapy. As the CD4 (T4 lymphocyte) count declines in an AIDS patient, there is a resultant increase in immunosuppression and vulnerability to opportunistic infections. As the systemic infection progresses, the defenses of the ocular surface may be altered, and this will influence how RCE secondary to lattice dystrophy is managed.[3,4] Our case is unique because this patient had recurrent erosion secondary to lattice dystrophy, and he was immunocompromised owing to AIDS. Our case exemplifies the importance of careful monitoring of an AIDS patient who presents with a compromised cornea, which, in this case, was caused by a recurrent erosion secondary to lattice dystrophy.

Case Report

A 49-year-old man presented with a chief complaint of tearing, photophobia, and watery discharge in the left eye (O.S.) for 1 day. He also reported having difficulty opening his eyes in the

morning. He denied redness or pain in the eyes or any recent trauma or surgery. His ocular history was significant for lattice corneal dystrophy in both eyes (OU), recurrent corneal erosion O.S., and herpes simplex keratitis (HSV) O.S. He was a poor historian, and most of his history was obtained from his hospital chart. He was unaware of any other family members with "corneal problems." Systemic history was remarkable for hepatitis C and HIV with secondary *Pneumocystis carinii* pneumonia. Medications reported were fexofenadine HCl, amoxicillin, fluconazole, abacavir sulfate, lamivudine, nefinavir, pentamidine, oxycodone/aspirin, phenytoin, sertraline HC1, and resperidone. Allergies reported include dapsone and bactrim. Viral load was reported as 35533 HIV-RNA molecules per milliliter of plasma and CD4 count 99 cells/mm^3. Best-corrected visual acuities were 20/80 in the right eye (O.D.) and O.S. 20/50. Pupils were equally round and reactive to light without an afferent defect. Extraocular muscle movements were full and unrestricted. Slit lamp examination found mild blepharitis OU, conjunctival melanosis, and lattice corneal dystrophy O.D. and O.S. with a 1.5- x 1.5-mm abrasion O.S. (*see* Figures 1 and 2). The right eye had prominent lattice dystrophy but no abrasion (*see* Figures 3 and 4)

The anterior chamber was deep and quiet without cells or flare. Intraocular pressures as measured by Goldmann applanation tonometry were 10 mmHg O.D. and 9 mmHg O.S. Dilated fundus examination was unremarkable with no evidence of retinal opportunistic infections or HIV retinopathy. The patient was prescribed bacitracin zinc/polymyxin B 4 times a day O.S. and was scheduled for 24-hour followup examination. During that time he was cyclopleged with 1% cyclopentolate hydrochloride drops that he took once daily in O.S. He was followed up on a daily basis, and re-epithelialization of the cornea occurred after 3 days of treatment.

Discussion

A corneal dystrophy is defined as a "primary corneal disease unassociated with prior inflammation, trauma, or systemic disease."[5] Lattice dystrophy is a stromal dystrophy, which consists of amyloidal deposits of the cornea. Amyloid is an extracellular complex of chondroitin, sulfuric acid, and protein.[6-9] The origin of this amyloidal material is unknown; however, it is theorized that it may be the result of collagen degeneration[10,11] or caused by abnormal synthesis by keratocytes.[12] Three

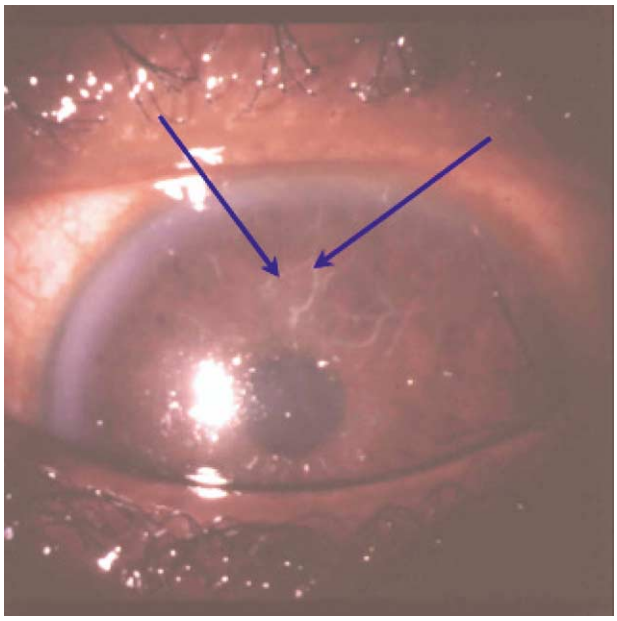

Figure 1 Prominent lattice dystrophy O.S. was visible on gross examination of the patient (*arrows*).

main types of lattice dystrophy have been described (*see* Table 1). Based on the appearance of the dystrophy (central and full thickness), our patient appears to have type I. Type I lattice dystrophy is an autosomal dominant condition that occurs bilaterally and symmetrically. It generally appears between the first and second decades of life. Its appearance consists of corneal haze, small refractile lines, dots, and dashes. Our patient, a poor historian, was unaware of any family members with corneal dystrophies. Type II lattice dystrophy is associated with systemic manifestations. Its appearance is that of coarse, translucent, stromal lattice lines radiating from the limbus, sparing the central cornea. Type II usually occurs in the second and third decade of life with systemic involvement such as nerve palsies, skin disorders, and facial abnormalities. Decreased corneal sensation and open angle glaucoma are common associations. Recurrent corneal erosions are uncommon. Type III lattice dystrophy is autosomal recessive with a late adult onset. It may be unilateral or bilateral, with thick, ropy lattice lines from limbus to limbus. Visual acuity is not usually affected until the sixth decade of life. Corneal erosions are not typically reported with type III lattice dystrophy.[5]

Lattice dystrophy is usually not the reason a patient presents to an eye care physician. Recurrent erosions are commonly associated with type I lattice dystrophy. As was the case with our