

Lipid Keratopathy Due to Presumed Herpes Simplex Keratitis: Case Report

Herpes Simpleks Keratitine Bağlı Olduğu Düşünülen Lipid Keratopati

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ABSTRACT Lipid keratopathy is a disease characterized by fat deposits in the corneal stroma which causes white opacification on the cornea. When it affects the center of the cornea, visual acuity decreases. The purpose of this case is to present the treatment and treatment result of a patient with lipid keratopathy. A 41-year old man was referred to our clinic with complaint of redness and decreased vision in the left eye lasting for 2 years. He received topical medications without an improvement. Ophthalmic examinations, including visual acuity and slit lamp biomicroscopy was performed before and after treatment. He received oral acyclovir treatment after the suspicion of herpetic keratouveitis due to corneal neovascularization, keratic precipitates and stromal opacification. At the end of the treatment, marked regression of corneal neovascularization and reduction in lipid deposition was noted and visual acuity increased. In conclusion oral acyclovir can be used in the treatment of lipid keratopathy due to presumed herpes simplex keratopathy.

Key Words: Keratitis, herpetic; acyclovir

ÖZET Lipid keratopati, kornea stromasında yağ birikimi ve korneanın beyaz opasifikasyonu ile karakterize bir hastalıktır. Kornea santralini etkilerse görme keskinliğini azaltabilir. Bu olguyu yayınlamaktaki amacımız lipid keratopati tanısı konan bir hastada tedavi ve tedavi sonucunu sunmaktır. 41 yaşındaki erkek hasta, gözde kızarıklık ve az görme şikâyeti ile kliniğimize başvurdu. Hastanın hikâyesinden daha önce çeşitli tedavilere rağmen şikâyetlerinde bir düzelme olmadığı öğrenildi. Tedavi öncesinde ve sonrasında görme keskinliği ölçüldü ve yarıklı lamba biyomikroskobu ile oftalmolojik muayenesi yapıldı. Gözde tekrarlayan kızarıklık şikâyeti ile muayenesinde kornea neovaskülarizasyonu, lipid keratopatiye bağlı stromal opasifikasyonu ve keratik presipitatları olan hastaya herpes simpleks keratouveiti ön tanısı konularak oral asiklovir tedavisi başlandı. Tedavi sonrasında, kornea neovaskülarizasyonunda belirgin gerileme ve lipid birikiminde belirgin azalma ve görme keskinliğinde artış görüldü. Sonuç olarak herpes simpleks keratitine bağlı olduğu düşünülen lipid keratopati tedavisinde oral asiklovir kullanılabilir.

Anahtar Kelimeler: Keratit, herpetik; asiklovir

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Lipid keratopathy is a disease characterized by fat deposits in the corneal stroma which causes white opacification on the cornea. When it affects the center of the cornea, visual acuity decreases. Primary lipid keratopathy is lipid accumulation without a systemic disease and vascularization; it is bilateral and shows symmetrical progression. The secondary form is a more common entity and associated with lipid leakage from corneal neovascularization as a result of an ocular pathology or a trauma.¹⁻⁴

This case presents a case of lipid keratopathy and its management.

CASE REPORT

A 41-year old man was referred to our clinic with complaint of redness and decreased vision in the left eye lasting for 2 years. Previously he had visited several ophthalmologists due to the same complaints and he had received topical medications (artificial eye drops, antibiotic eye drops and steroid eye drops) without an improvement. He didn't have a systemic illness. His best corrected visual acuity (BCVA) was 20/20 with a cylinder of -0,50 D in 120° in his right eye and 20/50 with a refraction of -4,00 D in 180° in his left eye. Slit lamp examination showed extensive deep stromal lipid deposits and neovascularization extending from the temporal limbus involving one-third of the temporal aspect of the pupillary area, obscuring iris details, thus affecting the visual axis (Figure 1). Keratic precipitates were also observed. The intraocular pressures were within normal limits (12 mmHg R, 10 mmHg L). The posterior segments of both eyes were unremarkable. Anterior segment optic coherence tomography (OCT) revealed mid stromal hyperreflectivity.

After the suspicion of herpetic keratouveitis due to corneal neovascularization, keratic precipitates and stromal opacification he received oral acyclovir 800 mg (Asiviral, Terra) five times per day, loteprednol etabonate 0.5 percent eye drops (Lotemax, Bausch&Lomb) three times per day, artificial eye drops (Refresh, Allergan) five times per day.

After three weeks, his vision improved to 20/20 with -0.75 -2.00 *180. Corneal opacity and vascularization reduced significantly (Figure 2).

DISCUSSION

Corneal neovascularization is a healing process resulting from various causes like inflammations, infections, traumas, allergies, immune responses and contact lens wearing. Corneal neovascularization can be associated with necrosis, haze, thinning, and lipid keratopathy.¹



FIGURE 1: Color photograph of the left eye shows the opacity which obscures iris details.

(See color figure at <http://www.turkiyeklinikleri.com/journal/oftalmoloji-dergisi/1300-0365/>)



FIGURE 2: Color photograph of the left eye at three weeks. The opacity showed improvement, clearing of the stromal infiltrate and allowing some view of the iris details.

(See color figure at <http://www.turkiyeklinikleri.com/journal/oftalmoloji-dergisi/1300-0365/>)

Herpes simplex virus (HSV) is a DNA virus that is associated with epithelial and stromal keratitis. It is an important cause of infectious blindness because it can lead to corneal neovascularization, stromal opacification and scarring.⁵

Stromal opacification in HSV keratitis is thought to be the result of the inflammatory response to the virus and of direct viral effects. Non-necrotizing and necrotizing stromal keratitis can lead to corneal neovascularization, scarring, and blindness as an end result.⁵

Inflammation is usually involved in the pathogenesis of stromal neovascularization; therefore corticosteroid therapy is usually the first-line treatment. In a study, subconjunctival and intracorneal bevacuzimab injection for corneal neovascularization showed regression of neovascularization and significant reduction in deposited lipids.^{6,7} It was shown that photodynamic therapy had potential in controlling and reducing the density of both corneal neovascularization and lipid keratopathy.⁸

In our case, lipid keratopathy was secondary form because of vascularization. Other causes of vascularization such as trauma, allergy and contact lens wearing were not seen in our patient. Presence of photophobia, recurrent episodes of redness, decreased vision, vascularization, keratic precipitates and stromal opacity made us think that the diagnosis was herpetic keratouveitis. Patient was treated conservatively. Neovascularization decreased and lipid keratopathy showed improvement.

In secondary lipid keratopathy, detecting and eliminating the underlying cause is essential. To date, no useful treatment has been confirmed but few studies show that photodynamic therapy and vascular endothelial growth factor inhibitor can be useful for the treatment of lipid keratopathy. These approaches do not eliminate the underlying cause; they only help the regress of the vascular component thus reducing the corneal lipid.

In our opinion, the improvement of neovascularization and stromal opasification is due to acyclovir treatment; because, in the history of the patient, he had corticosteroid treatment without any improvement.

In conclusion, treatment with oral acyclovir could be useful to treat lipid keratopathy due to presumed herpes simplex keratitis. Although this report presents a single clinical case, we recommend the use of oral acyclovir in lipid keratopathy due to presumed herpes simplex keratitis.

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