Case Report

Herpes zoster disciform keratitis – a case report

Abstract

Background: Disciform Keratitis is one of the many presentations that can occur in Herpes Zoster Ophthalmicus. It is a form endotheliitis that is accompanied by corneal swelling due to cellular dysfunction. Management of these cases involves oral antivirals and topical corticosteroids; extended prophylactic antiviral therapy should also be considered.

Case report: A 57-year-old white male who was immunocompromised due to leukemia presented with decreased vision accompanied with central corneal swelling and a circular area of keratic precipitates. He had an ocular history significant for skin lesions involving the ipsilateral eyelid about one month prior. When treated with oral valacyclovir and topical prednisolone, he responded well and had an excellent visual recovery. Due to herpes zoster recurrence and his immunocompromised status, we advised this patient to use prophylactic antivirals for one year following the event.

Conclusion: Herpes Zoster is a common disease, and ocular involvement is frequently seen. This case outlines a classic presentation of disciform keratitis due to herpes zoster. Additionally, this case highlights prophylactic treatment that should be considered, especially when the affected patients are immunocompromised and prone to recurrence. If not managed appropriately, patients may suffer from profound vision loss. Timely diagnosis and treatment are essential to help patients obtain their best visual outcome.

Keywords: herpes, zoster; herpes zoster ophthalmicus, infectious keratitis, disciform keratitis, endotheliitis, antivirals, valacyclovir

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Introduction

Herpes Zoster, colloquially known as Shingles, is a disease caused by a reactivation of the previously dormant varicella-zoster virus. This virus can lay dormant within sensory neuronal cell bodies following the primary infection. When this virus reactivates, it typically affects the skin of a particular dermatome, but if the reactivated virions are within the trigeminal ganglion, the eye can also be involved. This viral reactivation has the potential to involve all components of the eye, but corneal involvement is most frequent. Herpetic keratitis may involve the epithelium, the corneal stroma, or the endothelium.

Treatment and management depend on clinical presentation but often center around antiviral medication. Drug delivery can be topical or oral, though not all presentations can be treated with topical. Additionally, some presentations necessitate use of a topical corticosteroid to minimize ocular inflammation. Proper diagnosis and early treatment are paramount not only to decrease disease duration but also to obtain a favorable visual outcome. Prophylactic year-long antiviral treatment should be considered to potentially decrease recurrence of disease.

Case report

A fifty-seven-year-old Caucasian male presented to the eye clinic for an urgent evaluation with complaints of "cloudy" vision in the left eye over the last two weeks. He denied any ocular pain, flashes, or floaters. His past ocular history was significant for one episode of Herpes Zoster Virus (HSV) blepharitis one month prior involving the ipsilateral superior eyelid. At his exam one month prior, he had no ocular involvement, and he was treated with 500mg oral famciclovir three times a day for one week.

His past medical history was significant for Philadelphia-chromosome-positive acute lymphoblastic leukemia, chronic pain, psoriasis, obstructive sleep apnea, anxiety, & depression. His current medications for these conditions were codeine, gabapentin, and supplements of calcium and vitamin D.

At this urgent visit, the patient's corrected visual acuity was 20/25 in the right eye and 20/80 in the left eye; the left eye improved to 20/30 with the use of a pinhole occluder. Both pupils were round, reactive to light, and equal in size; no afferent pupillary defect was present. The patient had orthophoric ocular alignment and their extraocular motilities were smooth, full, and extensive in both eyes. Confrontation fields were full in both eyes.

Slit lamp examination of the right eye was unremarkable. Examination of the cornea of the left eye revealed a circular area of hazy stromal edema with a few granulomatous keratic precipitates in a circular pattern on the endothelium correlating to this area of edema (Figure 1). Additionally, there were finer non-granulomatous keratic precipitates diffusely spread across the inferior third of the corneal endothelium of this eye. There was no epithelial defect nor dendrite present. There were no cells nor flare present in the anterior chamber. The remainder of the anterior segment was unremarkable.

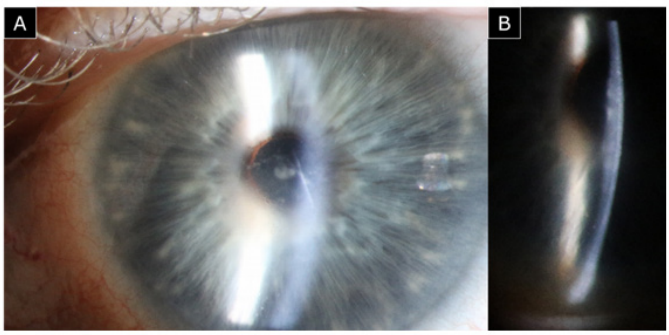

Figure 1 Anterior segment photos of the left eye at the initial visit (Day 00).

A. A parallelepiped beam illuminating a disciform lesion of the endothelium with a hazy view through the affected cornea. A large granulomatous

keratic precipitate can be seen centrally, and smaller non-granulomatous ones can be seen near the inferior cornea.

B. An optic section through the affected cornea showing increased corneal thickness around the disciform lesion.

Corneal sensation was evaluated with a short strand of dental floss and was found to be symmetric with an equally reactive blink response, though the patient subjectively reported increased sensation in the left eye. After this, corneal staining was assessed with 1 drop of 0.25%/0.4% fluorescein sodium/oxybuprocaine (Altafluor Benox) which verified that the epithelium was intact and there were no true nor pseudo dendritic lesions present. Intraocular pressures (IOPs) were obtained with Tono-Pen and were 16 in the right eye and 19 in the left eye.

To rule out any posterior segment involvement, the patient was dilated with 1 drop each 1% tropicamide and 2.5% phenylephrine, and the posterior segment was found to be unremarkable in both eyes. We also obtained a corneal pachymetry analysis using anterior segment OCT. The central corneal thickness was 558µm in the right eye and 808µm in the left eye. OCT pachymetry was obtained at every follow-up and the pachymetry series can be seen in Figure 4 below.

Based on this patient's clinical presentation in combination with his history of ipsilateral HZV blepharitis, the diagnosis of HZV disciform keratitis of the left eye was made. The patient was thoroughly educated concerning these findings and advised on the importance of compliance with treatment and follow-up. We started the patient on 1000mg oral valacyclovir three times per day and 1 drop 1% prednisolone acetate in the left eye hourly while awake until his follow-up scheduled for two days later.

At the first follow-up, the patient reported no change in subjective symptoms despite good compliance with the prescribed therapy. His corrected visual acuity was 20/20 in the right eye and had improved to 20/40 in the left eye. The anterior segment presentation remained unchanged in comparison. We repeated corneal pachymetry analysis and the central corneal thickness was stable in the right eye but had increased by 20µm in the left eye. We also obtained HD OCT scans of both corneas (Figure 2). Even though our patient's vision improved in the left eye, given his static objective presentation and small increase in central pachymetry in the left eye, we decided to keep our treatment the same. We advised him to continue valacyclovir and topical prednisolone following the same regiment and scheduled him to return in five days.

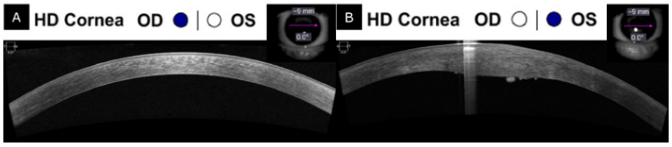

Figure 2 Anterior segment OCT at Day 02. High-definition OCT line scan through the cornea.

A. A normal cornea is seen in the right eye.

B. The scan of the left eye shows a localized area of edema and a few keratic precipitates can also be seen on the endothelial surface.

At the second follow-up, the patient reported a notable improvement in vision in the left eye with a mild residual blur. His corrected visual acuity was 20/20 in the right eye and 20/20 in the left eye, though reading was comparatively slower in the left eye at his finest acuity. Anterior segment evaluation showed a notable improvement with markedly decreased corneal edema (Figure 3). The left eye's pachymetry had a notable decrease and was now 580µm. There were only very few remaining keratic precipitates. This response to treatment was reassuring and we advised him to continue valacyclovir, and to begin a slow taper of the steroids. We educated the patient thoroughly on the importance of compliance to tapering in order decrease likelihood of rebound inflammation. We scheduled him to return in one week.

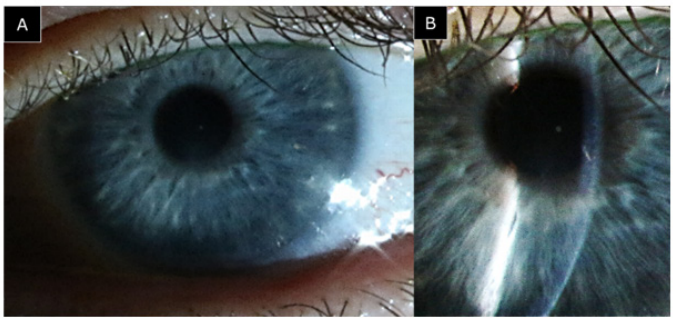

Figure 3 Anterior segment photos of the left eye at Day 07.

A. Diffuse illumination of the left eye showing a clear cornea.

B. An optic section through the previously affected cornea showing uniform thickness; a faint Descemet fold can be seen illuminated by this beam with good contrast over the bottom right edge of the dark pupil.

At the third follow-up, he reported improved vision since their last visit and good compliance to his steroid taper. His corrected visual acuity was 20/20 in both eyes with notable subjective improvement compared to his last appointment. Anterior segment evaluation showed trace resolving fine keratic precipitates in the left eye but was otherwise unremarkable in both eyes. The corneal pachymetry had normalized with 557µm in the right eye and 548µm in the left eye. Since the patient had now completed the treatment regimen for valacyclovir, we decreased this to a prophylactic dose of 1000mg by mouth daily for the next year. We advised the patient to continue his steroid taper and he was scheduled for a final follow up one month later at the end of the taper.

At his final follow-up evaluation, the patient's vision was 20/20 in both eyes and the anterior segment was unremarkable in both eyes. He reported completing his steroid taper and was continuing his prophylactic treatment (Figure 4).

Discussion

The varicella zoster virus (VZV) is the causative agent responsible for Herpes Zoster. After primary infection (Chickenpox), the virus can lay dormant in the neuronal cell body located in the dorsal root ganglia or cranial nerve nuclei. Herpes Zoster (HZ), or Shingles, is due to a reactivation of the dormant VZV which is transported down the sensory axon causing eruptive vesicular skin lesions restricted to a specific dermatome. The yearly incidence of HZ is around 0.1% in younger patients but increases to around 1% in patients over 65 years old.[1] It is reported that around 33% of adults will be affected by HZ in their lifetime.[2] Additionally, HZ recurrence can occur anywhere from 1.2-9.6% of the time; immunocompromised state, female sex, advanced age, and long-lasting post-herpetic pain all increase the risk of recurrence.[3]

Herpes Zoster Ophthalmicus (HZO) is when the viral reactivation occurs within a sensory neuron from the ophthalmic branch of the trigeminal nerve. Prevalence is variable, but up to 20% of HZ reactivations are HZO, and ocular involvement occurs in around 50% of HZO cases.[4] All components of the eye can be affected in HZO. It

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may present as conjunctivitis, keratitis, uveitis, scleritis, or retinitis; the first three of these being the most common.[4]

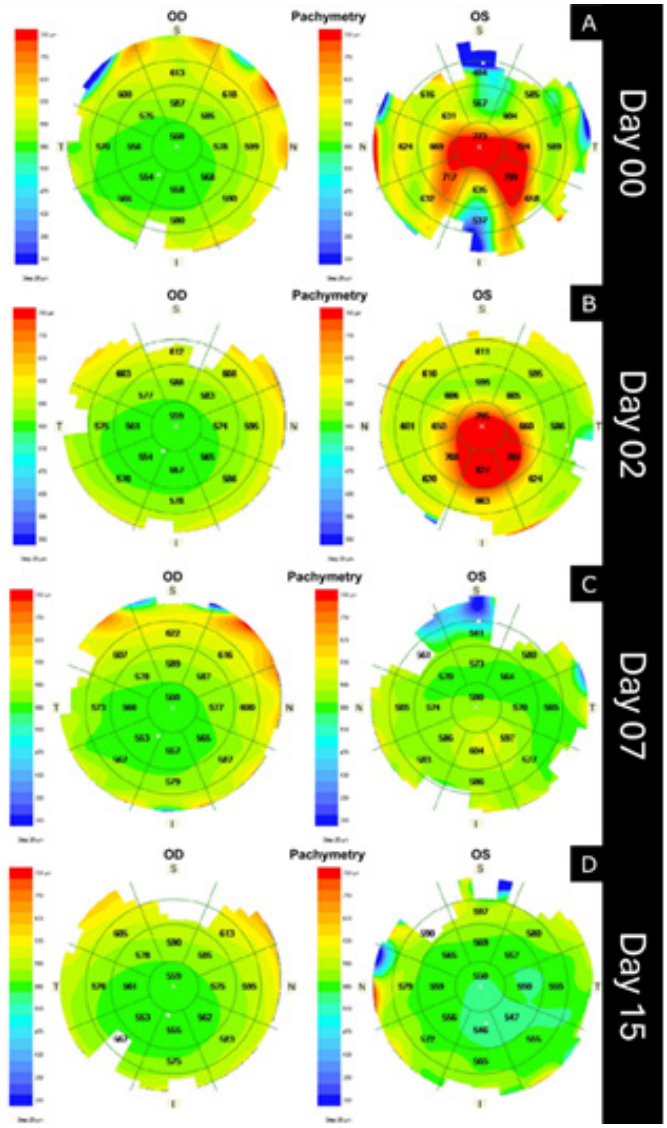

Figure 4 Anterior segment OCT pachymetry analysis. A series of pachymetry analyses from our patient's first visit [A] to their third follow-up [D]. Central pachymetry was slightly increased at his day-two evaluation [B]. The pachymetry had notably decreased at the seven-day mark [C]. By day fifteen, corneal pachymetry had normalized and was equal to the fellow eye [D].

HZ skin lesions usually precede acute keratitis by about one month.[5] Our patient presented for his urgent visit nearly perfectly adhering to this time course following his ipsilateral skin lesions. Our patient's IOPs showed asymmetry at presentation with the pressure being higher in the affected eye. Though acute IOP elevation is not specific for HZO, it is a documented findings in these patients.[6] Though this may have been the case, the IOP of the affected eye could have been artificially elevated due to corneal edema.

In endotheliitis, there can be direct viral involvement of the endothelium, or it may be an immune response to viral particles. Both cause dysfunction of the endothelial cells which leads to a loss of corneal deturgescence overlying the affected cells. Endotheliitis is classified by its pattern of keratic precipitates (KP) and corneal edema. It can be diffuse, sectoral, linear, or disciform.[7] Our patient had a very stereotypical disciform presentation as the KPs were oriented in a circular pattern and there was corneal edema over this area.

The leading treatment for patients with ocular involvement of VZV is oral antiviral therapy with acyclovir, valacyclovir, or famciclovir following their respective dose regiment (Table 1). If the patient is immunocompromised, intravenous acyclovir at a 10mg/kg dose three times a day for seven days should be considered.[8] For our patient, oral antivirals were satisfactory given his favorable treatment response. Additionally, while topical antivirals may be effective for VZV epithelial keratitis, this treatment is not sufficient when there is involvement of deeper corneal layers.[9]

Table 1 Oral antiviral therapy for herpes zoster. This table outlines the medications with their respective recommended dose, frequency, and duration of therapy.

Medication	Dose	Frequency	Duration
Acyclovir	800 mg	5× per day	7–10 days
Valacyclovir	1000 mg	3× per day	7–10 days
Famciclovir	500 mg	3× per day	7–10 days

In addition to antivirals, patients with concomitant immune stromal keratitis or uveitis should be treated with topical steroids are recommended at 1 drop 4-8 times per day to start, then adjusting as clinically indicated with a very slow taper at the terminus of their use.[10] The use of topical corticosteroids concurrently with antiviral treatment is also recommended by the American Academy of Ophthalmology when stromal keratitis or uveitis is present in HZO cases.[4] Though these recommendations do not specifically state endotheliitis, this same treatment is used in viral endotheliitis.[1]

While valacyclovir has a good safety profile when taken at the recommended dosage, dosage should be modified if a patient's creatinine clearance is less than 30 mL/min/1.73m^2 due to primarily renal clearance.[11] Acute kidney injury is possible with valacyclovir and other oral antivirals which prescribing optometrists should be aware of; the risk is higher when patients are elderly or taking other nephrotoxic medications.[12] Our patient had normal kidney function so this was of no concern.

The Zoster Eye Disease Study (ZEDS) assessed the effects of prophylactic treatment with 500mg valacyclovir by mouth twice daily for 1 year following an episode of HZO with ocular involvement. This study showed that when compared to placebo, this treatment had similar rates of their primary end point (new or worsening keratitis or iritis) at 12 months but was protective at the 18-month assessment.[13] Additionally, the valacyclovir group was shown to have a lower risk for multiple episodes.[13] These results give good support for consideration of this therapy in patients affected by HZO with ocular involvement. Our patient is immunocompromised due to leukemia, and though the ZEDS data doesn't assess this particular patient group, given a propensity for infection, prophylactic treatment seems a safe choice for patients with impaired immunity.

Our patient was a 57-year-old immunocompromised male with an episode of HZ blepharitis one month prior. For these prior skin lesions, he presented three days after onset and then was started on antivirals. This highlights the importance of starting oral antivirals within 72 hours of symptomatic onset to lessen the likelihood of ocular involvement.[4] Patients affected by endotheliitis need antiviral therapy and topical corticosteroids. Early diagnosis and treatment can help in visual recovery and decreased likelihood of poor visual outcomes. Our patient showed good response to treatment without any persistent sequalae following this keratitis episode.

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Conclusion

This case is an example of one of the various ways varicella zoster virus can affect the eye. Disciform keratitis presents as corneal edema with an underlying circular area of keratic precipitates. Ocular involvement in HZO can have a detrimental impact on a patient's vision and quality of life. Around 10% of patients with ocular involving HZO have either moderate (worse than 20/50) or severe (worse than 20/200) vision loss, the risk of which is increased in elderly or immunosuppressed patients.[14] Prophylactic treatment with antivirals after an episode of ocular involving HZO may be beneficial at decreasing multiple recurrences.[13] As eye care providers, it is paramount that we properly identify viral keratitis and treat accordingly to favor the best visual outcomes for our patients.

Acknowledgement

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Conflicts of interest

The authors declare that they have no known competing financial interests or personal relationships that appeared to influence the work reported in this study.

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