Postoperative Herpes Simplex Virus Keratitis After Cataract Surgery

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Introduction : Herpes simplex keratitis is one of the leading causes of infectious corneal blindness in the world. It remains latent in the human after the primary infection and can be reactivated by many factors. When activated, it travels along the trigeminal nerve to the cornea and causes recurrent infection which leads to corneal scarring. Management of the condition depends upon the pathogenesis of the disease. Topical antiviral treatment can be used in the case of the herpetic epithelial disease. This report describes a case of herpes simplex keratitis possibly triggered by surgical trauma.

Case report : A 54 year old male presented with painful, red and watering eye 3 weeks after cataract surgery. Best corrected visual acuity was 4/10 in the right eye and 10/10 in the left. Slit lamp examination of the right eye showed dendritic epithelial corneal ulcer,corneal oedema and mild anterior chamber reaction. Based on the signs and symptoms, the patient was diagnosed with herpes simplex keratitis. The patient was treated with ganciclovir ophthalmic gel (0,15%) five times a day, tobramycin eye drops (0,3%) foyr times a day and artificial tears. The patient also received oral valaciclovir 500mg two times a day. After 1 week, patient's best corrected visual acuity was 7/10, corneal oedema and anterior chamber's reaction were reduced, while the corneal ulcer was improved. 7 days later, patient's best corrected visual acuity reached 10/10, the corneal ulcer was healed and the anterior chamber was normal.

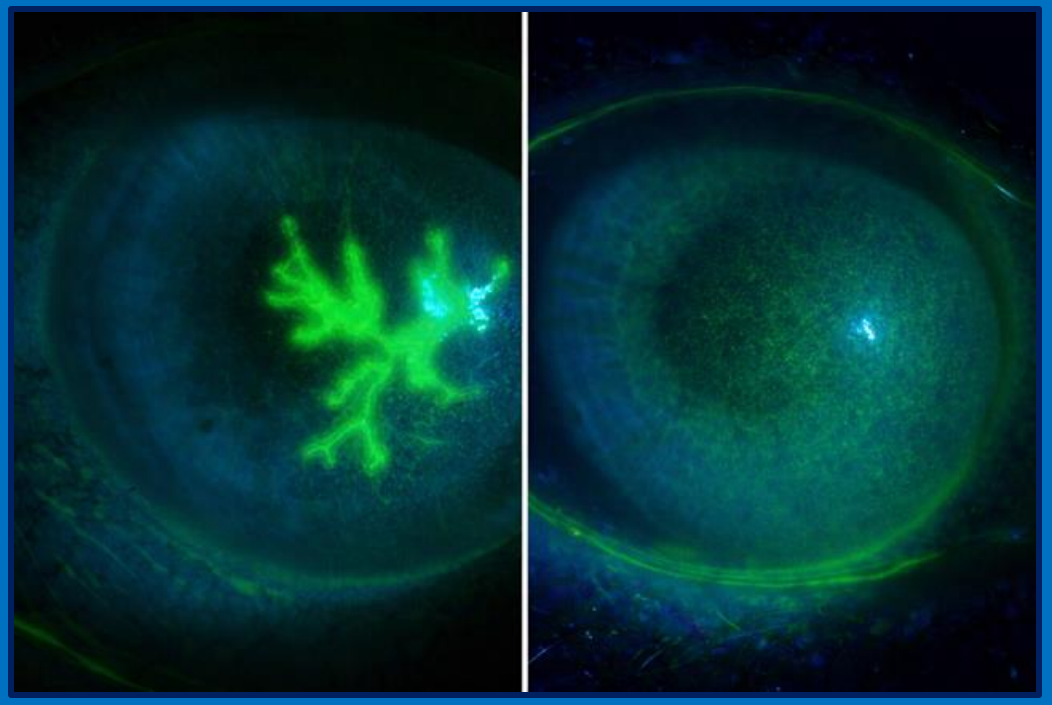

Figure 1 : Foto showing dendritic ulcer on fluoroscein staining before and after treatment.

Discussion : Herpes simplex virus (HSV) is a DNA virus that commonly affects humans. Infections occurs by direct contact with skin or mucous membrane with virus-laden lesions or secretions. HSV type 1 is primarily responsible for orofacial and ocular infections whereas HSV type 2 generally is transmitted sexually and causes genital disease. HSV-2 may rarely infect the eye by means of orofacial contact with genital lesions and occasionally is transmitted to neonates as they pass through the birth canal of a mother with genital HSV-2 infection. Primary HSV-1 infection occurs most commonly in the mucocutaneous distribution of the trigeminal nerve. It is often asymptomatic but may manifest as a nonspecific upper respiratory tract infection. After the primary infection, the virus spreads. Infected epithelial cells to nearby sensory nerve endings and is trasported along the nerve axon to the cell body located in the trigeminal ganglion. There, the virus genome enters the nucleus of a neuron, where it persists indefinitely in a latent state. Primary infection of any of the 3 (ophthalmic, maxillary, mandibular) branches of cranial nerve V can lead to latent infection of nerve cells in the trigeminal ganglion. Interneuronal spread of HSV within the ganglion allows patients to develop subsequent ocular disease without ever having had ocular HSV infection.

Discussion : Herpes simplex is the leading cause of infectious corneal blindness. In its epithelial form, dendritic keratitis is the most common presentation. Confusion of these lesions with pseudodendrites is a common problem that can best be solved by remembering the two key features of the classic dendritic lesion : true dendritic lesions show arborization and terminal end bulbs. Secondarily, the clinician can be tipped to the possibility of prior herpes infection if there exists unexplained corneal scarring, corneal hypoesthesia or iris atrophy. Pseudodendrites can be caused by contact lenses and their solutions, trauma, dry eye, and other infections, especially herpes zoster. A good history can be a key tool in differentiating such lesions.

Discussion : Currently, no treatment has been proven to remove the virus from the ganglia, therefore the goals of treatment are to interfere with viral replication to control virus multiplication, to reduce the recurrence rate and corneal scarring in order to preserve visual acuity and corneal sensitivity. Treatment of HSK is based on whether the condition is caused solely by an active virus or if it is due to an immunological reaction to viral antigens in the stroma or endothelium. It is thought the severity of HSK disease is dependent on both virus strains and host factors.

Discussion : In the case of herpetic epithelial keratitis, the corneal epithelial disease is effectively controlled by topical antiviral agents. Clinical trials showed that topical drugs ganciclovir and acyclovir were equally effective in treating the condition. Approximately 97% of patients with dendritic ulcer treated with topical drugs healed within 2 weeks. As antiviral maycause local toxicity to the corneal epithelium, the dosage frequency can be reduced as the dendritic ulcer begins to heal but should be continued for several days after healing to allow the shedding of dormant virus. In the case of possible drug adverse effects to the corneal epithelium, the use of oral valaciclovir (Valtrex 500mg 2 times a day for 10-14 days) has been reported as a safe and effective alternative to topical antiviral agents.

Discussion : Ganciclovir ophthalmic gel (0,15%) interferes with viral replication by blocking DNA transcription. It is very effective, though toxicity is a significant risk. Increased redness, pain, infiltrates and corneal staining, despite improvement in the dendritic lesion, suggest drug toxicity. Therefore, literature expresses cution at treatment periods longer than 21 days. Its dosage is one drop five times daily until resolution of the ulcer, then three times daily for another seven days. On the other hand, some researchers have suggested that an orals only approach is clinically effective in the absence of the potentially toxic topic drugs. Because the cornea has no blood supply, the orals only approach relies on the knowledge that oral acyclovir achieves serotherapeutic levels in the precorneal tear film.

Conclusion : Out treatment of choice is topical ganciclovir gel (0,15%), with conservative monitoring in order to prevent local toxicity to the corneal epithelium, as well as oral valaciclovir, the use of which remains optional based on literature. Further research and clinical experience will hopefully prove the effectiveness of an oral only approach for the treatment of herpes simplex keratitis in the absence of the potentially toxic topical drugs.

Management of Herpetic Keratitis

Type of Herpetic Keratitis	Clinical Features	Treatment	Notes / Remarks
Epithelial	• Dendritic / disciform ulcer • Superficial erosions • Ocular pain, photophobia, tearing	• **Topical:** Ganciclovir gel 0.15%, 5x/day for 7–10 days • **Systemic:** Valacyclovir 500 mg 2x/day for 7–10 days	• Avoid corticosteroids during epithelial phase. • Monitor healing within 1–2 weeks.
Stromal	• Inflammation of the corneal stroma without epithelial defect • Corneal haze, reduced vision • Often immune-mediated following prior infection • **Topical:** Corticosteroids (e.g., Prednisolone acetate 1%, 4x/day) • **Combined antiviral therapy:** Ganciclovir gel 0.15%, 5x/day or • Valacyclovir 500 mg, 2x/day		• Corticosteroids should **only** be given with antiviral coverage. • Monitor for recurrences and scarring.
Endothelial (Herpetic Endotheliitis)	• Endothelial involvement • Corneal edema, decreased clarity • Rare, may resemble stromal keratitis • **Topical:** Corticosteroids + Ganciclovir gel 0.15%, 5x/day • **Systemic:** Valacyclovir 500 mg, 2x/day		• Goal: reduce inflammation and protect endothelium. • Monitor endothelial cells and avoid worsening infection.