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Case Report-Post Cataract Surgery Herpes Simplex Keratitis and its Management

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Abstract: Herpes simplex keratitis (HSK) is a leading cause of corneal opacification and infection related visual loss. Even though an individual may not have had the clinically apparent disease, high fever, immune-suppression, and sometimes surgery can reactivate latent herpes1. Its presentation can be distinctively divided in to two types, epithelial keratitis or stromal keratitis due to a difference in pathogenesis, this inevitably postulates treatment difference. In this report, a case of postoperative herpes simplex virus (HSV) keratitis after a cataract surgery is described. The diagnosis and medical management of herpes simplex keratitis are discussed.

Keyword: Herpes Simplex, Corneal Blindness, Topical Antiviral.

INTRODUCTION

Herpes simplex keratitis is one of the leading causes of infectious corneal blindness in the world. It remains latent in the human host 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 is dependent upon the pathogenesis of the disease. Topical antiviral, corneal debridement can be used in the case of the herpetic epithelial disease. This report describes a case of herpes simplex keratitis that is possibly triggered by surgical trauma.

CASE REPORT

A 48 year old male presented with painful, red and watering right eye 2 weeks post cataract surgery. His uncorrected VA 1 day post surgery was 6/60.There was no other relevant past and medical history. Visual acuity was 6/24 right eye and 6/6left eye. Slit lamp examination Right eye showing dendritic epithelial corneal ulcer inferior central, grade 1 corneal oedema. Anterior chamber grade1 cells. Diagnosis Based on the signs and symptoms, the patient was diagnosed with herpes simplex keratitis. This patient was treated with acyclovir eye ointment (3%) five times a day. After 1 week patient best corrected visual acuity was reduced to 6/60, corneal oedema remained grade1, anterior chamber grade 2 cells, and mild improvement on fine keratic precipitates were present. The patient was advised to continue with the same treatment after 1 week Patient symptoms were reduced. Best corrected visual acuity was improved to 6/9.Dendritic corneal Ulcer was healed, no keratic precipitates were present, the anterior chamber was normal.

Figure 1: **Photograph Showing Dendritic Ulcer on Fluoroscein Staining**

DISCUSSION

HSV is a DNA virus that commonly affects humans. Infection occurs by direct contact with skin or mucous membrane with virus-laden lesions or secretions. HSV type 1 (HSV-1) is primarily responsible for orofacial and ocular infections, whereas HSV type 2 (HSV-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 transported 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 (ie, 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 primary ocular HSV infection[1]. A prospective, multicenter trial failed to find an association between anecdotal environment triggers (eg, stress, systemic infections, sunlight exposure, menstruation, contact lens wear, and eye injury) and ocular HSV recurrence.[2, 3, 4]

Herpes simplex is the leading cause of infectious corneal blindness in the United States. In its epithelial form, dendritic keratitis is the most common presentation to the primary care optometrist. 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.

Currently, no treatment has been proven to remove the virus from the ganglia, therefore the goals of treatments 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.

In the case of herpetic epithelial keratitis, the corneal epithelial disease is effectively controlled by topical antiviral agents, clinical trials showed that topical drugs trifluridine and acyclovir were equally effective in treating the condition,[5]approximately 97% of patients with dendritic ulcer treated with topical trifluridine healed within 2 weeks[6]. Other studies showed that ganciclovir 0.15% gel heals herpes simplex dendritic ulcers as effectively and rapidly as acyclovir 3%ointment but with less effect on vision blurring[7, 8]. As antiviral may cause 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[9, 10]. In the case of possible drug adverse effects to the corneal epithelium, the use of oral valaciclovir has been reported as a safe and effective alternative to topical antiviral agents.[11]

This drug 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. Although the literature expresses caution at treatment periods longer than 21 days in September 2009, the FDA approved Zirgan (ganciclovir gel 15%, Sirion Therapeutics) for acute herpetic keratitis (dendritic ulcers). European practitioners have had access to ganciclovir ophthalmic gel for more than 10 years. Its dosage is one drop five times daily until resolution of the ulcer, then three times daily for another seven days. Research and clinical experience will hopefully prove the effectiveness of this product now that it is available in the United Such treatment may at least reduce the viral load in the ciliary ganglion and associated nerves, even if there isn't a large effect in the corneal epithelium.
On the other hand, some researchers have suggested that an orals only approach is clinically effective in the absence of the potentially toxic topical drugs.[5] Incontrovertible research on this topic is lacking. 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

Our treatment choice is to use topical trifluridine or the newer ganciclovir, with conservative monitoring, as well as the oral agent. Hopefully, a large sample study will vindicate the orals only approach to herpes simplex keratitis

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