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Clinical Features and Factors Influencing the Course of Herpetic Keratitis, As Well as Optimization of its Treatment

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Annotation: Herpetic keratitis is an infection of the cornea with the herpes simplex virus. It can also affect the iris. Characteristic symptoms and signs include foreign body sensation, lacrimation, photophobia, and conjunctival hyperemia. Recurrences are common and can lead to corneal hypoesthesia, ulceration, permanent scarring, clouding, thinning of the corneal stroma, and visual impairment. Diagnosis is based on clinical suspicion, slit-lamp examination, and sometimes virological testing. Treatment is with topical and systemic antiviral agents.

Keywords: Clinical manifestations, Diagnostics, Treatment.

Introduction: Herpes simplex keratitis usually affects the corneal surface, but sometimes the stroma (deep layers of the cornea) or the inner surface of the cornea (endothelium), anterior chamber, and iris are involved. Stromal involvement is probably an immunological response to the virus.

As with all herpes simplex virus infections, the primary infection is followed by a latent phase in which the virus infiltrates the nerve roots. The latent virus can reactivate, causing a recurrence of symptoms.

Herpetic simplex keratitis is a leading cause of blindness worldwide.

Signs and symptoms of herpetic simplex keratitis

Primary infection

The primary infection is usually a nonspecific, self-limiting conjunctivitis, often occurring in early childhood and usually without corneal involvement. When the cornea is involved, symptoms include lacrimation, photophobia, and conjunctival hyperemia. This is followed in some cases by vesicular blepharitis (blisters on the eyelids), which is accompanied by worsening symptoms and blurred vision; the blisters rupture and ulcerate, then resolve within a week without leaving a scar.

Research methods and materials: Reactivation of latent herpes simplex is triggered by ultraviolet radiation (e.g., intense sunlight, corneal crosslinking (ultraviolet light therapy that hardens the cornea) or laser refractive procedures), fever, menstruation, severe systemic physical stress (e.g., burns or multiple immune fractures), , periocular injections, intraocular injections, or systemic). Relapses usually take the form of epithelial keratitis (also called dendritic keratitis), with lacrimation, foreign body sensation, and characteristic branching (dendritic or serpentine) lesions of the corneal epithelium that stain with fluorescein. Multiple recurrences can result in corneal hypoesthesia or numbness, corneal ulceration, permanent scarring, corneal opacity, thinning of the corneal stroma, and visual impairment.

Most patients with disciform keratitis, which primarily involves the corneal endothelium, have epithelial keratitis. Disciform keratitis is a secondary corneal edema associated with anterior uveitis and is characterized by a deeper, disc-shaped localized area of opacity. This form can cause pain, photophobia, and reversible vision loss.

Stromal keratitis can lead to stromal necrosis and severe pain, photophobia, foreign body sensation, ulceration, permanent scarring, opacification, neovascularization, thinning of the corneal stroma, and irreversible vision loss.

Slit-lamp examination of the patient is mandatory. In most cases, the detection of a tree-like corneal defect characteristic of herpesvirus dendritic keratitis is sufficient to make the diagnosis. The diagnosis of herpetic discoid keratitis and herpetic stromal keratitis is primarily clinical, based on a history of herpetic ocular disease and slit-lamp examination. Herpetic stromal keratitis is characterized by stromal opacification, often accompanied by neovascularization and inflammation. Herpetic discoid keratitis results in stromal edema due to dysfunction of the corneal endothelium.

If the diagnosis is difficult to make based on external appearances, the diagnosis can be confirmed by seeding the virus on viral cultures or by examining the smear using a nucleic acid amplification test (NAAT).

Dendritic (epithelial) keratitis can be treated with topical medications (such as ganciclovir or trifluridine). Topical therapy is usually effective, with the dosage of medication gradually reduced over 2-3 weeks.

Another option is oral treatment (such as acyclovir or valacyclovir). Acyclovir or valacyclovir may be prescribed as suppressive therapy to prevent frequent recurrences and preserve vision in patients at risk of vision loss. The dosage of each drug should be adjusted for patients with impaired renal function.

Treatment of immunocompromised patients usually involves intravenous administration of antiviral agents (e.g., acyclovir).

Topical glucocorticoids are contraindicated in epithelial keratitis, but they may be effective when used in combination with antiviral agents in the later stages of the disease when stromal (discoid) keratitis or uveitis are present. In such cases, patients may be prescribed prednisolone acetate.

If the epithelium surrounding the dendritic defect is loose and swollen, removing it with a cotton-tipped applicator before starting drug therapy may speed healing. Topical treatment for photophobia includes atropine 1% solution and scopolamine 0.25% solution 3 times daily.

Herpes simplex viral keratitis is a recurrence of a primary ocular herpes simplex infection that usually presents as a nonspecific, self-limited conjunctivitis.

The characteristic feature is corneal opacity with branching dendritic or serpentine lesions (indicating dendritic keratitis) or disc-shaped localized corneal edema and anterior uveitis (indicating disciform keratitis) or stromal scarring (indicating stromal keratitis).

The diagnosis is confirmed by a characteristic clinical picture, virological study results, or a smear using NAAT.

Treatment involves antiviral agents, most often topical ganciclovir or trifluridine, or oral acyclovir or valacyclovir.

Results: Ophthalmic herpes zoster is a reactivated latent varicella-zoster virus (VZV) infection affecting the eye (herpes zoster). Symptoms and signs, which can be severe, include a unilateral dermatomal rash on the forehead and painful inflammation of all tissues of the anterior and, rarely, posterior structures of the eye. Diagnosis is based on the characteristic appearance of the anterior segment of the eye with herpes zoster-like dermatitis along the 1st branch of the trigeminal nerve (V1 branch). Treatment includes oral antiviral drugs, mydriatics, and topical corticosteroids.

After primary infection, latent infection persists in the sensory ganglion. VZV-specific T cell-mediated immunity maintains VZV in a latent state. Reactivation of the virus occurs when immunity is weakened by age, illness, or immunosuppression. Patients with herpes zoster of the forehead with nasociliary nerve involvement (as indicated by nasal tip involvement) have a 3-fold higher risk of ocular involvement than patients without nasal tip involvement (1). Overall, the eyeball is involved in half of cases (2). Varicella virus is highly contagious and can be transmitted by direct contact with a skin lesion or by airborne droplets.

Prodromal pain or tingling in the forehead may occur. In the acute phase of the disease, in addition to the forehead rash, symptoms such as severe pain and marked swelling of the eyelids may occur; conjunctival, episcleral, and pericorneal injection; corneal edema, and photophobia.

Keratitis and/or uveitis can be severe and can lead to scarring. Late sequelae include glaucoma, cataracts, chronic or recurrent uveitis, corneal scarring, neovascularization, and hyperesthesia, all of which are common and can cause reduced visual acuity. Postherpetic neuralgia may develop later. Patients may develop episcleritis (with no significant risk of visual loss) and/or retinitis (with significant risk of visual loss).

The diagnosis is based on the presence of either the typical rash on the forehead and/or eyelids and tip of the nose, or on the nature of the pain, which is accompanied by signs of a previous herpes infection in the form of residual rashes (e.g., atrophic hypopigmented lesions). Both signs identified on the skin are unilateral (i.e., do not cross the midline). Vesicular or bullous lesions in this distribution do not yet clearly involve the eye, still requiring ophthalmological consultation to determine the involvement of the organ of vision in the pathological process. Skin culture and immunological or polymerase chain reaction tests are performed at the beginning of the examination, or serial serological tests are performed only if the pathological changes are atypical and the diagnosis is uncertain.

Discussion: Early treatment with acyclovir, famciclovir, or valacyclovir reduces the risk of developing ophthalmologic complications. Patients with uveitis or keratitis should receive topical corticosteroids (e.g., prednisolone acetate). The pupil should be dilated with 1% atropine or 0.25% scopolamine 3 times daily. Intraocular pressure should be monitored and appropriate treatment should be initiated if it is significantly elevated.

The use of a short course of high-dose oral corticosteroids to prevent postherpetic neuralgia in patients over 60 years of age in good general health remains controversial.

Medications used to treat neuropathic pain (such as gabapentin or tricyclic antidepressants) can help relieve the symptoms of postherpetic neuralgia.

The recombinant herpes zoster vaccine is recommended for immunocompetent adults 50 years of age and older, regardless of whether they have had herpes zoster or have received the older, live-attenuated vaccine. This recombinant vaccine reduces the risk of herpes infection; its effectiveness is 97% for people aged 50–69 years (1) and 90% for people aged 70 years and older (2).

Interstitial keratitis is a chronic non-ulcerative inflammation of the middle layers of the corneal stroma, sometimes accompanied by uveitis. The cause is usually infectious. Symptoms include photophobia, pain, tearing, and blurred vision. Diagnosis includes slit lamp examination and serological tests to determine the cause of the disease. Treatment is etiotropic and may require the use of local glucocorticoids.

Interstitial keratitis is rare in the United States as a manifestation of certain corneal infections. Most cases occur in childhood as a late complication of congenital syphilis. Later, the disease may affect both eyes. Similar but less severe bilateral keratitis develops in Cogan syndrome, Lyme disease, and Epstein-Barr virus infection. Rarely, acquired syphilis, herpes simplex virus, herpes zoster virus, or tuberculosis can cause unilateral interstitial keratitis in adults.

Common symptoms include photophobia, pain, lacrimation, and gradual loss of vision. The lesion begins as areas of inflammation in the middle layers of the stroma. In some cases (often associated with syphilis), the cornea takes on a ground-glass appearance, which interferes with visualization of the iris. Newly formed vessels grow from the limbus (neovascularization), which leads to the appearance of the "caviar spots" sign (orange-red areas). Syphilitic interstitial keratitis often has anterior uveitis and choroiditis. Inflammation and revascularization usually begin to subside within 1 to 2 months, regardless of treatment. Corneal transparency is usually maintained, which leads to mild to moderate vision loss.

The exact etiology of the disease should be determined. The specific etiology can be determined by the presence of stigmata of congenital syphilis, vestibulo-auditory signs, a diffuse rash, or a history of contact with ticks. In addition, all patients undergo serological testing, which includes the following tests:

Conclusion: Fluorescent antibody absorption test for Treponema pallidum or microhemagglutination assay for Treponema pallidum are common screening tests for syphilis.

Newer tests such as the Treponema pallidum latex agglutination test, Treponema pallidum enzyme immunoassay, chemiluminescent immunoassay, and nucleic acid amplification test (NAAT) may also be effective in diagnosing ocular syphilis.

Patients with negative serological test results may have an idiopathic syndrome that includes Cogan syndrome, interstitial keratitis, and vestibular-auditory disorders. In cases of hearing loss, tinnitus, or dizziness, consultation with an otolaryngologist is required to prevent the development of chronic vestibular-auditory disorders.

Interstitial keratitis, which is rare in the United States, involves chronic inflammation of the middle layers of the cornea.

Characteristic symptoms are photophobia, pain, lacrimation, gradual loss of vision, the appearance of "caviar spots" and anterior uveitis.

Tests for syphilis, Lyme disease, and Epstein-Barr virus infection should be performed.

Treatment should be performed by an ophthalmologist; topical corticosteroids are occasionally prescribed.

Dry eye syndrome (DES) is a chronic bilateral disorder of the conjunctiva and cornea, characterized by decreased tear production and impaired corneal tear film stability.

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