Blepharitis

Clinical features of blepharitis include itching, burning, foreign-body sensation, and sticky, crusted eyelids on waking. This constant irritation results in unconscious rubbing of the eyes (causing reddened rims) or continual blinking. Other signs include waxy scales in seborrheic blepharitis; and flaky scales on lashes, loss of lashes, and ulcerated areas on lid margins in ulcerative blepharitis. In association with KCS, dry eyes may also be a problem.

Exophthalmos

The obvious effect is a bulging eyeball, commonly with diplopia, if extraocular muscle edema causes misalignment. (See Recognizing exophthalmos.) A rim of the sclera may be visible below the upper lid as lid retraction occurs, and the patient may blink infrequently. Other symptoms depend on the cause: pain may accompany traumatic exophthalmos; a tumor may produce conjunctival hyperemia or chemosis; retraction of the upper lid predisposes to exposure keratitis. If exophthalmos is associated with cavernous sinus thrombosis, the patient may exhibit paresis of the muscles supplied by cranial nerves III, IV, and VI; limited ocular movement; and a septictype (high) fever.

Ptosis

An infant with congenital ptosis has a smooth, flat upper eyelid, without the eyelid fold normally caused by the pull of the levator muscle; associated weakness of the superior rectus muscle isn't uncommon. The child with unilateral ptosis that covers the pupil can develop an amblyopic eye from disuse or lack of eye stimulation. In bilateral ptosis, the child may elevate his brow in an attempt to compensate, wrinkling his forehead in an effort to raise the upper lid. Also, the child may tilt his head backward to see. In myasthenia gravis, ptosis results from fatigue and characteristically appears in the evening, but is relieved by rest. Ptosis due to oculomotor nerve damage produces a fixed, dilated pupil; divergent strabismus; and slight depression of the eyeball.

Orbital cellulitis

Orbital cellulitis generally produces unilateral eyelid edema, hyperemia of the orbital tissues, reddened eyelids, and matted lashes. Although the eyeball is initially unaffected, proptosis develops later (because of edematous tissues within the bony confines of the orbit). Other indications include extreme orbital pain, impaired eye movement, chemosis, and purulent discharge from indurated areas. The severity of associated systemic symptoms (chills, fever, and malaise) varies according to the cause. Complications include posterior extension, causing cavernous sinus thrombosis, panophthalmitis, meningitis, or brain abscess and, rarely, atrophy and subsequent loss of vision secondary to optic neuritis.

Dacryocystitis

Dacryocystitis is extremely painful for the patient. The hallmark of both the acute and chronic forms of dacryocystitis is constant tearing. Other symptoms of dacryocystitis include inflammation and tenderness over the nasolacrimal sac; pressure over this area may fail to produce purulent discharge

from the punctum.

Chalazion

A chalazion occurs as a painless, hard lump that usually points toward the conjunctival side of the eyelid. Eversion of the lid reveals a red or red-yellow elevated area on the conjunctival surface. Otherwise, it's seen as an indurated bump under the skin of the upper eyelid.

Stye

Typically, a stye produces redness, swelling, and pain. An abscess frequently forms at the lid margin, with an eyelash pointing outward from its center. A stye is a localized red, swollen, and tender abscess of the lid glands.

Inclusion conjunctivitis

Inclusion conjunctivitis develops 5 to 12 days after contamination (it takes longer to develop than gonococcal ophthalmia). In a neonate, reddened eyelids and tearing with moderate mucoid discharge are presenting symptoms. In neonates, pseudomembranes may form, which can lead to conjunctival scarring. In adults, follicles appear inside the lower eyelids; such follicles don't form in infants because the lymphoid tissue isn't yet well developed. Children and adults also develop preauricular lymphadenopathy, and children may develop otitis media as a complication. Inclusion conjunctivitis may persist for weeks or months, possibly with superficial corneal involvement.

Conjunctivitis

Conjunctivitis commonly produces hyperemia of the conjunctiva, sometimes accompanied by discharge, tearing and, with corneal involvement, pain and photophobia. It generally doesn't affect vision. Conjunctivitis usually begins in one eye and rapidly spreads to the other by contamination of towels, washcloths, or the patient's own hand. Acute bacterial conjunctivitis (pinkeye) usually lasts only 2 weeks. The patient typically complains of itching, burning, and the sensation of a foreign body in his eye. The eyelids show a crust of sticky, mucopurulent discharge. If the disorder is due to N. gonorrhoeae, however, the patient exhibits a profuse, purulent discharge. Viral conjunctivitis produces copious tearing with minimal exudate, and enlargement of the preauricular lymph node. Some viruses follow a chronic course and produce severe disabling disease; others last 2 to 3 weeks and are self-limiting.Itching is the hallmark of allergy. Giant papillae resembling cobblestones may be seen on the palpebral conjunctiva

Trachoma

Trachoma begins with a mild infection resembling bacterial conjunctivitis (visible conjunctival follicles, red and edematous eyelids, pain, photophobia, tearing, and exudation). After about 1 month, if the infection is untreated, conjunctival follicles enlarge into inflamed papillae that later become yellow or gray. At this stage, small blood vessels invade the cornea under the upper lid.Eventually, severe scarring and contraction of the eyelids cause entropion; the eyelids turn inward and the lashes rub against the cornea, producing corneal scarring and visual distortion. In late stages, severe conjunctival scarring may

obstruct the lacrimal ducts and cause dry eyes.

Keratitis

Keratitis is usually unilateral. The patient presents with decreased vision, discomfort ranging from mild irritation to acute pain, tearing, and photophobia. On gross examination with a penlight, the corneal light reflex may appear distorted. When keratitis results from exposure, it usually affects the lower portion of the cornea.

Corneal abrasion

A corneal abrasion typically produces redness, increased tearing, discomfort with blinking, a sensation of "something in the eye" and, because the cornea is richly endowed with nerve endings from the trigeminal nerve (cranial nerve V), pain disproportionate to the size of the injury. It may also affect visual acuity, depending on the size and location of the injury.

Corneal ulcers

Typically, corneal ulceration begins with pain (aggravated by blinking) and photophobia, followed by increased tearing. Eventually, central corneal ulceration produces pronounced visual blurring. The eye may appear injected. If a bacterial ulcer is present, purulent discharge is possible.

Uveitis

Anterior uveitis produces moderate to severe unilateral eye pain; severe ciliary injection; photophobia; tearing; a small, nonre-active pupil; and blurred vision (due to the increased number of cells in the aqueous humor). It sometimes produces deposits called keratic precipitates on the back of the cornea, which may be seen in the anterior chamber. The iris may adhere to the lens, causing posterior synechiae and pupillary distortion; pain and photophobia may occur. Onset may be acute or insidious. Posterior uveitis begins insidiously, with complaints of slightly decreased or blurred vision or floating spots. Posterior uveitis may be acute or chronic, and it may affect one or both eyes. Retinal damage caused by lesions from toxoplasmosis and retinal detachments may occur. Refer the patient to an ophthalmologist for dilated fundus examination and treatment for local systemic diseases

Retinal detachment

Initially, the patient may complain of floating spots and recurrent flashes of light (photopsia). However, as detachment progresses, gradual, painless vision loss may be described as a veil, curtain, or cobweb that eliminates a portion of the visual field.

Vascular retinopathies

Central retinal artery occlusion produces sudden, painless, unilateral loss of vision (partial or complete). It may follow amaurosis fugax or transient episodes of unilateral loss of vision lasting from a few seconds to minutes, probably due to vasospasm. This condition typically causes permanent blindness. However,

some patients experience spontaneous resolution within hours and regain partial vision. Central retinal vein occlusion causes reduced visual acuity, allowing perception of only hand movement and light. This condition is painless, except when it results in secondary neovascular glaucoma (uncontrolled proliferation of weak blood vessels). The prognosis is poor—some patients with this condition develop secondary glaucoma within 3 to 4 months after occlusion. Nonproliferative diabetic retinopathy produces changes in the lining of the retinal blood vessels that cause the vessels to leak plasma or fatty substances, which decrease or block blood flow (nonperfusion) within the retina. This disorder may also produce microaneurysms and small hemorrhages. Nonproliferative retinopathy causes no symptoms in some patients; in others, leakage of fluid into the macular region causes significant loss of central visual acuity (necessary for reading and driving) and diminished night vision.

Age-related macular degeneration

The patient notices a change in central vision. Initially, straight lines (for example, of buildings) become distorted; later, a blank area appears in the center of a printed page (central scotoma).

Cataract

Characteristically, a patient with a cataract experiences painless, gradual blurring and loss of vision. As the cataract progresses, the normally black pupil appears hazy, and when a mature cataract develops, the white lens may be seen through the pupil. Some patients complain of blinding glare from headlights when they drive at night; others complain of poor reading vision, and of an unpleasant glare and poor vision in bright sunlight. Patients with central opacities report better vision in dim light than in bright light because the cataract is nuclear and, as the pupils dilate, patients can see around the lens opacity.

Retinitis pigmentosa

Typically, night blindness occurs while the patient is in his teens. As the disease progresses, his visual field gradually constricts, causing tunnel or “gun-barrel” vision. Many people retain this tunnel of useful vision until quite late in life. The speed of vision loss varies considerably from person to person. However, blindness follows invasion of the macular region.

Optic atrophy

Optic atrophy causes abrupt or gradual painless loss of visual field or visual acuity, with subtle changes in color vision.

Extraocular motor nerve palsies

The most characteristic clinical effect of extraocular motor nerve palsies is diplopia of recent onset, which varies in different visual fields, depending on the muscles affected. Typically, the patient with third nerve palsy exhibits ptosis, exotropia (eye looks outward), pupil dilation, and unresponsiveness to light; the eye is unable to move and can't accommodate. The patient with fourth nerve palsy displays diplopia and an inability to rotate the eye downward or upward. The head is tilted to the side opposite the involved area in superior oblique palsy. Sixth nerve palsy causes one eye to turn; the eye can't abduct

beyond the midline. To compensate for diplopia, the patient turns his head to the unaffected side and can develop torticollis.

Glaucoma

Chronic open-angle glaucoma is usually bilateral, with insidious onset and a slowly progressive course. Symptoms appear late in the disease and include mild aching in the eyes, loss of peripheral vision, seeing halos around lights, and reduced visual acuity (especially at night) that isn't correctable with glasses. Acute angle-closure glaucoma typically has a rapid onset, constituting an ophthalmic emergency. Symptoms include acute pain in a unilaterally inflamed eye, with pressure over the eye, moderate pupil dilation that's nonreactive to light, a cloudy cornea, blurring and decreased visual acuity, photophobia, and seeing halos around lights. Increased IOP may induce nausea and vomiting, which may cause glaucoma to be misinterpreted as GI distress. Unless treated promptly, this acute form of glaucoma produces blindness in 3 to 5 days.