**Eyelid Disorders**

Last updated: May 9, 2019

**LID EDEMA**

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<td>Lid edema</td>
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**ETIOLOGY**

1. Allergies:
   - acute type (seasonal allergic lid edema) - hypersensitivity to airborne pollens or direct hand-to-eye application of pollens.
   - chronic type - contact sensitivity to topical drugs (e.g. atropine, neomycin), cosmetics, metals (e.g. nickel); perennial allergic lid edema - hypersensitivity to molds or to animal or dust mite dander.

2. Trichiasis - chronic bilateral lid edema (resembles allergic type); fever and other systemic symptoms may not be present initially; eosinophilia > 10% is characteristic.

3. Hereditary angioedema - acute lid edema.

**TREATMENT**

For allergic lid edema:
1. removal of offending cause.
2. cold compresses over closed lids may speed resolution.
3. corticosteroid ointments (for not more than 7 days) if swelling persists > 24 h.

**BLEPHARITIS**

**ULCERATIVE BLEPHARITIS** - acute bacterial infection (usually staphylococcal).

**SEBORRHIC BLEPHARITIS** - chronic blepharitis; associated with seborrheic dermatitis (Pityrosporum ovale).

**MEIBOMIAN GLAND DYSFUNCTION (MEIBOMITIS)** - chronic blepharitis caused by abnormal meibomian gland secretions, often associated with acne rosacea.

**CLINICAL FEATURES**

1. On lid margins: itching, burning, redness (red-rimmed eyelids), thickening, scales & crusts clinging to lashes.
2. Lid edema
3. Conjunctival irritation (lacrimation, photophobia).

**Symptoms of seborrheic blepharitis & meibomian gland dysfunction**

- Ulcerative blepharitis: small pustules in lash follicles → break down → shallow marginal ulcers with dry adherent crusts (leave bleeding surface when removed; during sleep, lids become glued together by dried secretions); may result in loss of eyelashes and eyelid scarring.

- Seborrheic blepharitis: greasy, easily removable scales of lid margins; secondary bacterial colonization occurs on scales.

- Meibomian gland dysfunction: meibomian gland orifice impissated (plugged) with hard waxy plug.

Patients with seborrheic blepharitis and meibomian gland dysfunction often have:
- secondary keratitis sicca.
EYELID DISORDERS

EYE66 (2)

− history of repeated styes and chalazia.
− exacerbations that are uncomfortable & unsightly but do not result in central corneal scarring or visual loss.

TREATMENT

Ulcerative blepharitis - antibiotic ointment (e.g. bacitracin/polymyxin B or gentamicin or sulfacetamide for 7-10 d).

Schorbehc blepharitis - eyelid hygiene (scrubbing lid margin daily with cotton swab dipped in dilute baby shampoo); occasionally, antibiotic ointment is indicated.

Meibomian gland dysfunction - normalizing meibomian gland secretions:
1) doxycycline tapered over 3-4 mo.
2) warm compresses (melt waxy plugs and allow trapped secretions to flow out).

HORDEOLUM
- acute localized pyogenic infection of eyelid gland:
  a) ciliary (Moll) gland (EXTERNAL HORDEOLUM, STYE) - modified apocrine sudoriferous glands that open into follicles of eyelashes.
  b) Zeis gland (EXTERNAL HORDEOLUM, STYE) - sebaceous glands that open into follicles of eyelashes.
  c) tarsal (meibomian) gland (INTERNAL HORDEOLUM, MEIBOMIAN STYE, ACUTE CHALAZION) - sebaceous glands embedded in tarsal plate, discharging at lid edge near posterior border.
    • usually staphylococcal
    • polymorphonuclear leucocytes and necrosis with pustule formation.
    • often secondary to blepharitis.
    • recurrence is common.

CLINICAL FEATURES

EXTERNAL HORDEOLUM – superficial, at eyelid base: begins with pain, redness, tenderness, foreign-body sensation → small, round, tender area of induration → small yellowish spot in center of induration (pointing) → abscess soon ruptures with pus discharge and pain relief.

INTERNAL HORDEOLUM (very rare) – deeper, more severe.
  • conjunctival lid side shows small yellow elevation (site of affected gland).
  • abscess points on conjunctival lid side (sometimes points through skin); spontaneous rupture is rare!!!
  • recurrence is common.

CHALAZION (MEIBOMIAN CYST)
- chronic granulomatous inflammation (lipogranuloma) of meibomian gland.
  • due to duct occlusion (often after internal hordeolum) - lipid breakdown products, possibly from bacterial enzymes, leak into surrounding tissue and incite granulomatous chronic inflammation (with lymphocytes and lipid-laden macrophages [Touton-type giant cells]).
  • contrary to popular opinion, research has not shown that eyelid cosmetic products either cause or aggravate condition.
  • hormonal influences on sebaceous secretion and viscosity (androgenic hormones increase sebum viscosity) may explain clustering at puberty and during pregnancy.

CLINICAL FEATURES

− onset - indistinguishable from stye; more common on upper lid.
  − after few days - painless, slowly growing round mass in lid; seen subconjunctivally as red-gray mass; overlying skin can be moved loosely.
  − large lesions have been reported to cause astigmatism or hyperopia resulting from central corneal flattening.
  − acute inflammatory exacerbation (internal hordeolum) can result in anterior rupture (beneath skin) or posteriorly (through conjunctiva); it never points to lid margin (unlike stye).
  − sebaceous dysfunction and obstruction elsewhere (e.g. comedones, oily face) are the only associated features.
EYELID DISORDERS

TREATMENT
• most disappear after few months (hot* compresses for 10-15 min qid may hasten resolution);
• early in condition, blocked glandular orifices may be opened by vigorous lid massage between 2 cotton wool buds at slit lamp (local anesthesia may be beneficial); self-administered technique is also available - called "2 fingers times 10 massage" (at conclusion of bath / shower, patient warms hands under hot water, using 1 drop of baby shampoo, patient works up lather, and then places index finger over closed lids at lid margin and vigorously massages lid back and forth 10 times; then repeats procedure with middle, ring, and little fingers).
• if there is no resolution after 6 wk:
  a) incision & curettage, after procedure, cauterization with phenol or trichloroacetic acid may prevent recurrence of small chalazia.
  b) intralacholution corticosteroid (e.g. triamcinolone diacetate). - if associated with ACNE ROSACEA, 6 month course of low dose TETRACYCLINES may help sebaceous glands to produce shorter-chain fatty acids that are less likely to block gland orifices.
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• ENTROPION AND ECTROPION
Both conditions, if persistent and bothersome, are best treated surgically!
ECTROPION - eyelid eversion
• results from:
  1) tissue relaxation with aging (LID-LAXITY ECTROPION)
  2) scar (CICATRICIAL ECTROPION)
  3) CN7 palsy (PARALYTIC ECTROPION)
  4) ichthyosis (CONGENITAL ECTROPION).
• usually involves lower lid.
• poor tear drainage through nasolacrimal system ——> epiphora.
• conjunctival / corneal exposure ——> redness, irritation, keratinization of palpebral conjunctiva, corneal ulceration.

CICATRICIAL ECTROPION:

CONGENITAL ECTROPION (in ichthyosis):

TREATMENT
• lubrication, moisture shields.
**Eyelid Disorders**

- cicatricial entropion - digital massage to stretch scar, steroid injection into scar.
- paralytic entropion - taping lateral canthal skin supertemporally provides temporary relief; external paste-on upper lid weights.

**Entropion** - eyelid inversion

1. Acute spastic entropion - orbicularis oculi spasm due to ocular irritation.
2. Involutional entropion - horizontal laxity of medial and/or lateral canthal tendons, involution of orbital fat (involutional enophthalmos with unstable eyelid position).
3. Cicatricial entropion - scar tissue of conjunctiva; digital eversion of eyelid margin is difficult.
4. Congenital entropion (very rare) - dysgenesis of lower eyelid retractors, structural defects in tarsal plate also (tarsal kink syndrome).

- causes irritation (lashes rub against globe) → corneal ulceration and scarring.

**TREATMENT**

- ocular lubrication (tear preparations).
- spastic entropion - eyelid hygiene, antibiotics, corticosteroids, botulinum toxin.

**TUMORS**

**XANTHELASMA** - common, benign subcutaneous deposit, with yellow-white, flat plaques of lipid material; associated with hypercholesterolemia; do not need to be removed (except for cosmetic reasons).

**BASAL CELL CARCINOMA** frequently occurs at lid margins, at inner canthus, and on upper cheek.

- other malignant tumors are less common; tumors simulating chronic blepharitis or chronic chalazion should be biopsied rather than treated for a long time.

**LID RETRACTION, LAGOPHTHALMOS**

Whenever lid retraction is suspected, exclude contralateral ptosis!

- etiology:
  1) thyroid-associated ophthalmopathy [see p. 2744 >>]
  2) PARS plana (dorsal midbrain) syndrome [see p. Eye64 >>]
  3) prior lid surgery / trauma.

- differentiate from CN7 palsy.

**LAGOPHTHALMOS** - condition in which complete closure of eyelids over eyeball is difficult or impossible.

- etiology: exophthalmos, mechanical obstacles, CN7 palsy.
- lubricate eyes with liquid paraffin ointment.
- corneal ulceration may develop; H: temporary tarsorrhaphy.

**BLEPHARO)PTOSIS**

Etiology:

1) weakening of levator aponeurosis due to age / trauma.
2) hypotropia (causes ptosis).  
3) Horner syndrome - both MRD 1 & MRD 2 ↓
4) CN3 palsy - MRD 1 ↓ with unchanged MRD 2.
5) myasthenia gravis; ptosis is transient; curtain sign (not specific for myasthenia gravis) - elevation of one lid causes contralateral lid to droop (explained by Hering law).

**Cogan lid twitch** - patient is asked to quickly look upward from downward position → lid overelevates and then droops.

**BIBLIOGRAPHY** for ch. “Ophthalmology” → follow this [LINK >>]

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