

Hordeolum and chalazion treatment

The full gamut

Hordeola and chalazia are some of the most common inflammatory eyelid disorders encountered in optometric practice. Many patients try to treat these lesions conservatively using home remedies or over-the-counter medication. Often, such treatment is efficacious and the lesion resolves as intended. In those individuals where the condition persists, the optometrist may be consulted for more definitive care.

Internal hordeolum

Signs and symptoms

An internal hordeolum (meibomian sty) is a small abscess caused by an acute staphylococcal infection of the meibomian glands of the tarsus (Figure 1)¹. These lesions may occur in conjunction with acute or chronic blepharitis. They point posteriorly and often rupture spontaneously and drain through the conjunctival surface². A specific change in meibomian gland secretion has been linked to internal hordeolum formation³.

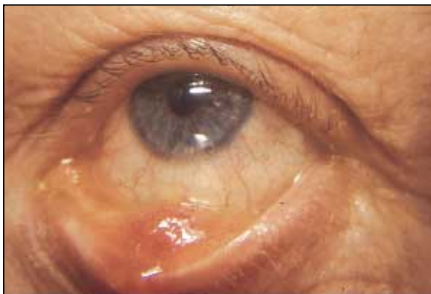


Figure 1 Internal hordeolum

These lesions characteristically occur abruptly with a painful swelling and erythema, often of the entire eyelid. Eversion of the eyelid will show a more localised lesion and in advanced cases, a yellowish nodule can be seen through the tarsal conjunctival surface². The eyelid margin surrounding the orifice of the involved meibomian gland is usually inflamed. Any secretions within the orifice are purulent when expressed. The inflammation can spread to other adjacent glands or to the apposing or contralateral eyelid⁴. Recurrences are common, especially if any underlying conjunctivitis or blepharitis is not adequately treated.

Treatment

Because the infection is deep within the lid tissue, the topical application of antibiotics is usually ineffective⁵. The patient should be instructed to apply hot compresses for five to 10 minutes, two to four times a day, in order to liquefy the stagnant secretions and facilitate drainage through the meibomian orifice². Lid

scrubs with a mild shampoo also helps to remove any debris, which may have accumulated on the eyelid margin surface, and in those patients with blepharitis. Because staphylococcus species are usually the underlying causes of the infection, primary medical therapy should consist of a penicillinase-resistant penicillin such as dicloxacillin. Dosages of 125mg to 250mg every six hours, usually result in prompt resolution of the infection⁵. Patients who are allergic to penicillin can try oral erythromycin, chloramphenicol or the aminoglycosides². Finally, in cases which resist medical therapy, incision and drainage using a sterile needle or blade may be necessary⁶.

External hordeolum

Signs and symptoms

An external hordeolum (common sty) is a purulent inflammation of infected eyelash follicles and surrounding sebaceous (Zeis) and apocrine (Moll) glands of the lid margin (Figure 2)⁴. It is usually due to a staphylococcal infection and may be associated with staphylococcal blepharitis. The lesions are often associated with fatigue, poor diet and stress and can be recurrent⁶.

External hordeola present as tender inflamed swellings in the lid margin, which points



Figure 2 External hordeolum

anteriorly through the skin¹. In most cases, the lesion drains spontaneously within three or four days after pointing⁵. More than one lesion may be present and, occasionally, minute abscesses can involve the entire lid margin. Pain, particularly on manipulation of the eyelid, is the most notable symptom. As with any skin abscess, the nodule is usually red and warm to the touch.

Treatment

Hot compresses several times a day accelerate the pointing of the lesion and its spontaneous drainage. If an eyelash is seen to extend from the involved lesion, then epilation of the lash can initiate drainage of the lesion by creating an effective drainage channel⁷. Bacitracin or erythromycin antibiotic ointment, applied four

times a day during the acute phase and continued twice daily for one week thereafter, may prove helpful, especially in preventing the infection spreading to the surrounding lash follicles⁸. Systemic antibiotics such as oral erythromycin or dicloxacillin may be necessary if there is severe preseptal cellulitis¹. Finally, for resistant lesions, an incision can be made with a sterile needle or blade into the area of pointing, which allows the abscess cavity to drain^{6,7}.

Chalazion

Signs and symptoms

A chalazion is a localised lipogranulomatous inflammatory response involving the sebaceous glands (meibomian or Zeis) of the eyelid. It occurs secondary to obstruction of the gland duct⁴. The obstruction can be the result of inflammation or infection (acne rosacea or seborrheic dermatitis), or of neoplastic lesions (sebaceous gland carcinoma or Merkel cell tumour) of the lid margin⁹. Chalazia occur spontaneously or may follow an episode of acute internal hordeolum.

The onset and progression of this lesion is usually slow and associated with few symptoms. They are more common in the upper lid, appearing as a hard, immobile, painless, roundish lump in the tarsal plate¹⁰ (Figure 3). The chalazion may produce pain if it grows very large and cause distention of sensory nerve endings. An upper lid chalazion may press on the cornea and cause blurred vision from induced astigmatism¹¹. At least 25% of chalazia resolve spontaneously within six months of onset, but most require treatment¹².



Figure 3 Chalazion

Treatment

Topically or systemically administered antibiotics are ineffective because the lesion is not infectious in origin¹³. The application of hot compresses followed by gentle massage may evacuate stagnant secretions. This prevents further chalazion formation and encourages drainage along the duct of the involved

gland – which may be of benefit if the lesion is small². Vigorous massage can cause further extravasation of the meibomian secretions into the surrounding tissue, spreading the granulomatous inflammation². Regrettably, this treatment is not very effective, resolving only around 40% of these lesions^{13,14}.

Chalazia which fail to resolve with conservative management may be treated with an intralesional injection of steroid¹⁴. This technique increases the resolution rate to 80%, while combining the conservative therapy with steroid injection increases the resolution rate to 90%¹⁴.

Since the chalazion is encapsulated by connective tissue, there is little room for space-occupying steroid medication. Therefore, a steroid of increased concentration such as triamcinolone acetamide (Kenalog-40), a 40mg/ml concentration works well since only a 0.10-0.20cc dose needs to be injected (Figure 4).

The chalazia can be injected through the skin surface or the conjunctival side using a 1ml tuberculin syringe with a 27-gauge or 30-gauge needle. The steroid suspension should be injected into the centre of the lesion. If injection is performed from the conjunctival side, several drops of a topical anaesthetic to numb the puncture site and minimise blinking. Injection through the skin surface of the eyelid requires no anaesthesia. Some practitioners prefer to use a chalazion clamp, but this is not always necessary. Chalazia typically resolve within one or two weeks after a single injection, but larger chalazia may require a second injection.



Figure 4
Setup for steroid injection for chalazia

Locally injected steroid suspension works because a chalazion is composed of steroid-sensitive histocytes, multi-nucleated giant cells, lymphocytes, plasma cells, polymorphonuclear leukocytes, and eosinophils⁴. The injected steroid suppresses additional inflammatory cells and impedes chronic fibrosis.

This technique is safe and effective. There has been one reported case of a serious complication resulting in both retinal and choroidal vascular occlusion from embolisation of the injected steroid¹⁵. To minimise the chances of this occurring, practitioners should aspirate for blood before injecting, take care to inject slowly, and avoid heavy digital pressure during and after injection¹⁶. Other less serious complications include pain on injection, depigmentation of the eyelid at the injection site, temporary skin atrophy and subcutaneous white (steroid) deposits (Figure 5)³.



Figure 5
Subcutaneous white (steroid) deposits after intralesional triamcinolone injection

The most reliable therapy involves surgical excision of the affected meibomian gland (Figures 6-9). The surrounding eyelid tissue needs to be injected with the anaesthetic



Figure 6
Injection of eyelid with the anaesthetic, Xylocaine (lidocaine)



Figure 7
Chalazion clamp and traction suture in place



Figure 8
Curette adjacent to granulomatous debris scraped from inside the meibomian gland



Figure 9
Forceps pointing to excised gland after it has been cleaned of debris

Xylocaine (lidocaine). The eyelid is everted and a traction suture is placed through the eyelid margin. Then a chalazion clamp is positioned over the lesion. This helps stabilise the eyelid and assists in hemostasis. A surgical #11 or #15 straight blade or a circular trephine blade is used to incise the involved meibomian gland through the conjunctival surface. A curette is then used to scrape out the chronic granulomatous debris.

The chalazion clamp and traction suture are removed and the eyelid is repositioned. Digital pressure is applied until all the bleeding has stopped. The eye is treated with antibiotic ointment, which the patient should continue to use two times a day for five to seven days. The patient should be re-evaluated after about two weeks.

There are usually few complications from this surgery. The eyelid may be swollen and discoloured after the surgery for several days to one week. Occasionally, a subconjunctival haemorrhage can also develop, but this will resolve without incident (Figure 10). On rare occasions, the chalazion may recur if the surgical excision was incomplete.



Figure 10
Child with eyelid ecchymosis and subconjunctival haemorrhage after surgical excision of chalazion

Pyogenic granuloma

Signs and symptoms

A pyogenic granuloma may be seen after trauma or surgery, or may form over inflammatory lesions, such as chalazia. These nodules occur rarely in the anophthalmic socket following enucleation of the eye and at the margin of corneal transplants¹⁷.

These lesions occur on the conjunctival side of the eyelid and are fleshy, red, usually sessile with a palpable rigid either non-tender or moderately tender presentation (Figure 11). Microscopically, a pyogenic granuloma is



Figure 11
Pyogenic granuloma

composed of granulation tissue with chronic inflammatory cells, fibroblasts, and endothelial cells of budding capillaries. The term pyogenic granuloma is actually a misnomer since the lesion is neither pyogenic nor granulomatous¹⁸.

Treatment

Treatment consists of complete excision and curettage of any underlying inflammatory eyelid lesion such as a chalazion. Pathologic evaluation is also recommended, since several other benign and malignant neoplasms, such as Kaposi's sarcoma, may simulate pyogenic granuloma¹⁷.

About the author

Dr Leonid Skorin Jr is a licensed optometrist and a board-certified ophthalmologist. He is fellowship trained in neuro-ophthalmology. He has numerous publications and has lectured internationally.

References

1. Kanski JJ (1991) *Clinical Ophthalmology* 4th ed. Butterworth-Heinemann, Boston, p. 12-14.
2. Kaufman HE, Barron BA, McDonald MB, Kaufman SC (eds) (2000) *Companion Handbook to the Cornea* 2nd ed. Butterworth-Heinemann, Boston, p. 29-33.
3. Shine WE, McCulley JP (1996) Meibomian gland triglyceride fatty acid differences in chronic blepharitis patients. *Cornea* 15: 340-346.
4. Bertucci GM (2001) Periocular skin lesions and common eyelid tumors. In: Chen WP (ed) *Oculoplastic Surgery: The Essentials*. Thieme, New York, p. 225-241.
5. Marren SE, Bartlett JD, Melore GG (2001) Diseases of the eyelids. In: Bartlett JD, Jaanus SD (eds) *Clinical Ocular Pharmacology* 4th ed. Butterworth-Heinemann, Boston, p. 485-522.
6. Alexander KL (1980) Some inflammations of the external eye and adnexa. *J. Am. Optom. Assoc.* 51: 142-146.
7. Hudson RL (1981) Treatment of styes and meibomian cysts. *Practical procedures. Aust. Fam. Phys.* 10: 714-717.
8. Trevor-Roper PD (1974) Diseases of the eyelids. *Int. Ophthalmol. Clin.* 14: 362-393.
9. Font RL (1986) Eyelids and lacrimal drainage system. In: Spencer WH (ed) *Ophthalmic Pathology: An Atlas and Textbook*. WB Saunders, Philadelphia, p. 2141-2336.
10. Gershen HJ (1985) Chalazion. In: Fraunfelder FT, Roy FH (eds) *Current Ocular Therapy* 2nd ed. WB Saunders, Philadelphia, p. 354-355.
11. Nisted M, Hofstetter HW (1974) Effect of chalazion on astigmatism. *Am. J. Optom. Physiol. Opt.* 51: 579-582.
12. Cottrell DG, Bosanquet RC, Fawcett IM (1983) Chalazions: the frequency of spontaneous resolution. *BMJ* 1983; 287-1595.
13. Bohigian GM (1979) Chalazion: a clinical evaluation. *Am. Ophthalmol.* 11: 1397-1398.
14. Garrett GW, Gillespie ME, Mannix BC (1988) Adrenocorticosteroid injection vs. conservative therapy in the treatment of chalazia. *Am. Ophthalmol.* 20: 196-198.
15. Thomas EL, Laborde RP (1986) Retinal and choroidal vascular occlusion following intralesional corticosteroid injection of a chalazion. *Ophthalmology* 93: 405-407.
16. Francis BA, Chang EL, Haik BG (1996) Particle size and drug interactions of injectable corticosteroids used in ophthalmic practice. *Ophthalmology* 103: 1884-1888.
17. Skorin L (2000) Corneal and eyelid anomalies. *Consultant* 40: 265-272.
18. Griffith DG, Salasche SJ, Clemons DE (1987) *Cutaneous Abnormalities of the Eyelid and Face: An Atlas With Histopathology*. McGraw-Hill, New York, p. 136-137.