Diseases of the Eyelids and Orbit

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KEYWORDS

• Eyelid • Orbit • Oculoplastic surgery • Thyroid eye disease

KEY POINTS

- The eyelids and orbit house, protect, and maintain ocular health, enabling normal visual function.
- Common eyelid disorders include lesions and drooping (ptosis). Causes range from benign conditions to life-threatening diseases, the most common of which are included in this article.
- Trauma can affect any portion of the eye and periorbita. The urgency of and need for repair and treatment depend on the severity and extent of injury.

INTRODUCTION

The human eyelid is a dynamic structure that overlies the orbit, a bony compartment that houses the visual apparatus and supportive soft tissue. Compromise of normal eyelid function can jeopardize ocular health and vision, and orbital disorders can lead to permanent functional deficits. The present article provides an overview of eyelid and orbital anatomy, common disorders, and trauma-related considerations.

PERIORBITAL ANATOMY

Eyelid anatomy is intricate, dynamic, and exquisitely intertwined with function. The eyelid consists of 7 layers: skin, orbicularis muscle, orbital septum, orbital fat, retractor muscles, tarsus, and conjunctiva. The skin of the eyelid is the thinnest in the body, with no subcutaneous fat.[1,2] Sensory innervation comes from the ophthalmic branch of the trigeminal nerve in the upper eyelid and from the maxillary branch in the lower eyelid.[3] The orbicularis muscle, innervated by the temporal branch of the facial nerve, closes the eyelids. The orbital septum is a fibrous connective tissue layer that extends from the orbital periosteum at the orbital rim to the superior border of the tarsus in the upper eyelid and to the capsulopalpebral fascia just at the border of the tarsus in the lower eyelid. The septum creates an orbital compartment along with the bony walls,

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enclosing the orbit anteriorly. In the upper eyelid, there are 2 orbital fat pads: medial and central. In the lower eyelid, there are 3: medial, central, and lateral. The inferior oblique muscle runs between the inferior medial and central fat pads. The upper eyelid retractors consist of the levator palpebrae superioris muscle and the superior tarsal muscle, also known as the Muller muscle. The levator muscle is innervated by cranial nerve 3, whereas the sympathetic nervous system innervates the Muller muscle. The analogous structures in the lower eyelid are the capsulopalpebral fascia and the inferior tarsal muscle. The tarsus is a firm connective tissue plate that provides structural support to the eyelids. It measures 10 to 12 mm vertically in the upper eyelid and 4 mm in the lower eyelid; both are approximately 29 mm in horizontal length and 1 mm in thickness. Meibomian glands, which produce the oil component of tears, are embedded within the tarsal plates and open onto the surface of the eye. The conjunctiva is a membranous layer of nonkeratinized squamous epithelium that lines the inner surface of the eye and extends onto the anterior surface of the eyeball as 1 continuous layer that is folded on itself. The conjunctiva contains goblet cells, which secrete the mucin component of tears. The blood supply to the upper eyelid comes from the internal carotid artery, whereas the lower eyelid receives blood from the external carotid artery. The 2 systems have branches that anastomose to form the marginal and peripheral arterial arcades in the eyelid.[1,2] The preseptal venous system drains into the angular vein medially and the superficial temporal vein laterally. Deep to the orbital septum, orbital veins drain into the anterior facial vein and the pterygoid plexus.[2] The lymphatics of the medial upper and lower eyelid drain into the submandibular lymph nodes, whereas the lateral eyelids drain into the preauricular lymph nodes.[1,2]

CHALAZION/HORDEOLUM

Obstruction of the meibomian glands can lead to focal inflammation and swelling, known as a chalazion. Acutely, the area of inflammation is tender, erythematous, and can be warm to touch. Risk factors include blepharitis and rosacea. Treatment in the acute phase includes warm compresses with digital massage toward the eyelid margin and eyelid hygiene. The focal inflammation is not an infection and does not require antibiotic therapy.[2] Adjuvant management options include oral flaxseed oil supplementation, a short course of topical steroids limited to 2 weeks maximum, topical tea tree oil to the lashes, topical cyclosporine, and oral doxycycline for its antiinflammatory effect.[4–7] The chalazion may open on its own and produce sterile purulent drainage. More invasive treatment options include intraeyelid injection of a steroid or 5-fluorouracil. Risks of steroid injection include depigmentation of the overlying skin and retrograde migration resulting in central retinal artery occlusion, although the latter is extremely rare.[5] A chalazion may require several serial steroid injections to achieve complete resolution. The most aggressive management option is incision and curettage, which consists of eyelid eversion and marsupialization of the chalazion from the posterior surface of the eyelid. This treatment may be combined with a steroid injection, as well.[2,5] Timing of the interventions depends on patient and practitioner preferences. In general, it is reasonable to consider a trial of conservative therapy for 3 to-4 weeks before referral to ophthalmology for further management and possible procedural intervention.

Focal obstruction of the sebaceous meibomian glands can also result in an acute infection, known as a hordeolum or stye. The causative organism is typically a gram-positive bacterium such as *Staphylococcus aureus*. They may resolve with diligent warm compresses and lid hygiene but require systemic antibiotics if they progress to cellulitis. If the inflammation forms an abscess, it typically requires incision and drainage in addition to oral antibiotics.[2,5]

PREMALIGNANT AND MALIGNANT EYELID TUMORS

Actinic keratosis is the most common precancerous lesion of the skin. It typically presents as a scaly, keratotic plaque on sun-exposed skin in a fair-skinned, elderly patient. The risk of malignant transformation to squamous cell carcinoma (SCC) is 0.24% per year, with up to 25% of actinic keratoses resolving spontaneously over the course of 1 year. Nevertheless, suspected actinic keratosis should undergo biopsy for pathologic confirmation followed by treatment with topical 5-fluorouracil or imiquimod. A patient with multiple actinic keratoses has a 12% to 16% incidence of SCC.[8]

Lentigo maligna is a precancerous lesion consisting of uncontrolled melanocyte proliferation. It appears as a flat area of patchy pigmentation and can be distinguished from benign senile and solar lentigo by its irregular borders, uneven pigmentation, and slow progressive growth. From 30% to 50% of lentigo maligna lesions transform into invasive melanoma. These lesions require complete excision with wide surgical margins and close surveillance for recurrence.[2,9]

Basal cell carcinoma (BCC) accounts for 90% to 95% of eyelid malignancies. It typically occurs in fair-skinned, blue-eyed elderly patients who may already have a history of prior BCCs.

Risk factors include extensive sun exposure in the first 2 decades of life and cigarette smoking. Younger patients may have a systemic syndrome, including basal cell nevus syndrome, also known as Gorlin syndrome, and xeroderma pigmentosa. The most commonly affected periocular location is the lower eyelid (50%–60%), followed by the medial canthus (25%–30%), upper eyelid (15%), and lateral canthus (5%).[2] Clinical presentation varies from chronic eyelid inflammation to a rapidly expanding growth. Nodular BCC is the most common type, appearing as a firm, pearly nodule with telangiectasias and central ulceration. The most aggressive form is morpheaform BCC, which can have poorly defined margins on examination. Clues to malignancy on physical examination include madarosis (loss of eyelashes), ulceration, nonhealing scabbed wounds, and focal chronic inflammation.[10] Incisional or excisional biopsy of suspicious lesions confirms the diagnosis. Treatment is complete surgical excision with clear margins, which may require Mohs micrographic surgery to preserve as much normal eyelid tissue as possible to enable optimal reconstruction.[11] Recurrence, orbital invasion, and metastasis are rare.[10] Extensive disease may require oral vismodegib, a hedgehog pathway inhibitor.[12]

Although SCC is less common than BCC in the periocular region, it is a more aggressive malignancy known for perineural extension, lymphatic spread, and direct invasion. Risk factors include history of prolonged sun exposure, actinic keratosis, and immunodeficiency. SCC that arises from actinic keratosis tends to be less aggressive. Clinical appearance typically includes disruption of normal eyelid architecture, such as madarosis and ulceration. Patients with perineural involvement may report pain. Management and treatment consist of surgical excision with wide margins and pathologic confirmation. Suspicion for orbital invasion or systemic spread warrant further work-up, which may include radiographic imaging and neck dissection with lymph node biopsy.[13] Medical therapy with epidermal growth factor receptor inhibitors, anti–programmed cell death protein 1 (PD-1) antibodies, and hedgehog pathway inhibitors is an alternative treatment option for patients who are poor surgical candidates.[2,14,15] SCC may recur, warranting lifelong surveillance.[13]

Keratoacanthoma is low-grade SCC that occurs in middle-aged and elderly patients, with a higher incidence among those who are immunosuppressed. It presents as a dome-shaped nodule with rolled edges surrounding a central keratin-filled crater.

Although keratoacanthomas can undergo gradual spontaneous resolution, standard treatment is complete surgical excision.[16]

Periocular sebaceous cell adenocarcinoma is a highly malignant tumor of the meibomian glands or accessory sebaceous glands with potential for aggressive local invasion, metastatic spread, and even death. It occurs more commonly in the upper eyelid, where there is a higher concentration of glands compared with the lower eyelid. Sebaceous cell carcinoma affects women more than men and tends to present in patients more than 50 years old.[17] It can occur independently or in association with Muir-Torre syndrome, an autosomal dominant condition characterized by sebaceous neoplasms and colon cancer.[18] Classically, sebaceous cell carcinoma masquerades as chronic or recurrent inflammation, often mistaken for unilateral red eye, unilateral blepharitis, or chalazion recalcitrant to treatment.[17] Recurrent chalazion in the same location or chronic focal inflammation in older patients should raise suspicion for sebaceous cell carcinoma, especially when physical examination reveals madarosis or distortion of normal eyelid architecture. Full-thickness eyelid biopsy with lipid stain on pathology confirms the diagnosis. A map biopsy of the conjunctiva helps to assess the extent of ocular surface spread, given the potential for skip lesions and pagetoid (intraepithelial) spread. Large tumors with suspicion for orbital extension require radiographic imaging. Local treatment consists of wide local excision, possibly with Mohs microsurgery. Tumors that have spread superficially may require adjuvant cryotherapy, and those that have orbital extension require exenteration (surgical removal of the eyeball and all orbital tissue, including periosteum).[17,19] Cases of spread warrant a sentinel lymph node biopsy, as well as systemic work-up for distant metastasis.[20,21]

Cutaneous melanoma is extremely lethal, accounting for greater than 65% of skin cancer deaths, and occurs very rarely on the eyelids.[22] It may arise de novo or transform from a preexisting nevus or lentigo maligna.[22,23] Risk factors include sunlight exposure, genetic predisposition, and environmental exposures.[23,24] New pigmented lesions appearing after the first 2 decades of life should alert suspicion. Features include irregular borders, patchy pigmentation, ulceration, madarosis, and bleeding. The 2 most common types affecting the eyelid are lentigo maligna melanoma and nodular melanoma. The former typically starts as lentigo maligna in the malar skin; a nodular growth arising from the surrounding pigmentation signifies malignant transformation. It can spread superiorly to the eyelid and involve the lid margin and even the conjunctiva. Nodular melanoma is characterized by aggressive vertical spread and may lack pigmentation; there may be deep invasion at the time of diagnosis.[2] Staging is determined by Breslow thickness, Clark level, and ulceration. The most important predictors of survival for localized melanoma include tumor thickness and ulceration. Melanoma with regional lymph node spread carries worse prognosis with a greater number of involved lymph nodes, larger tumor volume, and presence of ulceration. Melanoma with distant metastasis carries a worse prognosis when it involves a visceral anatomic site.[22] Treatment of localized melanoma entails wide local excision with a 5-mm margin, broadened to 10 mm for tumors with Breslow thickness 2 mm or thicker. Thick melanomas (>2 mm) should undergo further work-up with sentinel lymph node biopsy. High-dose radiation is used as adjuvant treatment, as well as therapy for regional lymph node spread and palliative management for metastatic disease. Systemic disease is managed with chemotherapy and immunomodulatory agents.[25] Tumors that are less than 0.75 mm in thickness have a 5-year survival rate as high as 98%, whereas those thicker than 4 mm with ulceration confer a survival rate less than 50%.[26] Local recurrence risk ranges from less than 1% to 25%.[25]

BLEPHAROPTOSIS

The terms blepharoptosis and ptosis refer to drooping of the upper eyelid, which obstructs incoming light rays and can cause functional visual impairment. Types include myogenic, aponeurotic, neurogenic, and mechanical forms. Levator aponeurosis dehiscence or disinsertion through age-related attenuation, trauma, or repetitive stretching (eye rubbing, contact lens use, eye surgery) is the most common cause of ptosis and warrants surgical evaluation when it impairs activities of daily living.[27] The following discussion focuses on acquired neurogenic and myogenic forms of ptosis with systemic implications.

Neurogenic ptosis results from disruption of innervation to the levator muscle or Muller muscle. Conditions include acquired cranial nerve III palsy, acquired Horner syndrome, and myasthenia gravis. The most common causes of oculomotor nerve palsy are ischemia and compression. Patients with microvascular third nerve palsies typically have a history of diabetes mellitus, atherosclerosis, hypertension, smoking, and associated pain. Examination reveals complete ptosis and restriction of eye adduction, elevation, and depression (down and out).[27] Seventy-five percent of microvascular third nerve palsies spare pupil involvement.[28] Patients with anisocoria with a larger pupil on the involved side, partial third nerve palsy, no microvascular risk factors, age younger than 50 years, and persistent palsy 3 months after onset require neuroimaging to rule out an aneurysmal or neoplastic compressive lesion.[28,29] Patients older than 50 years with concern for giant cell arteritis should undergo immediate serologic work-up, including erythrocyte sedimentation rate, C-reactive protein, platelets, and possible temporal artery biopsy.[28] Horner syndrome results from disruption of sympathetic innervation to the Muller muscle, resulting in mild (2 mm) ptosis and a constricted pupil that does not dilate in dim light. Pharmacologic assessment using apraclonidine or cocaine eyedrops can confirm the diagnosis. The condition requires head, neck, and chest neuroimaging to rule out lethal conditions such as carotid dissection.[28] Myasthenia gravis (MG) is an autoimmune disease caused by autoantibodies to the postsynaptic acetylcholine receptor (AChR) of the neuromuscular junction. It presents as ocular myasthenia without systemic involvement in 20% of cases, but, of these cases, 80% develop systemic symptoms with time. It can involve any ocular striated muscle, including extraocular muscles, which results in diplopia. In patients with ptosis or diplopia that is variable or worse with fatigue, there should be a high level of suspicion for MG.[29,30] Confirmatory testing includes ice test, rest test, single-fiber electromyography, and serologic evaluation for antibodies to AChR and muscle-specific tyrosine kinase (MuSK). From 10% to 20% of patients test negative for AChR; of these, 40% to 70% test positive for anti-MuSK.[29] Patients with MG should undergo chest imaging to assess for highly associated thyroid nodules and thymoma.[28,30]

Acquired myogenic ptosis results from diseases such as chronic progressive external ophthalmoplegia (CPEO), muscular dystrophy, and oculopharyngeal muscular dystrophy.[27,29] CPEO includes a subtype known as Kearns-Sayre syndrome, a mitochondrial disease characterized by bilateral ptosis, progressive limitation in extraocular motility, retinopathy, and heart block.[31] Disease recognition enables potential lifesaving work-up and management.

ORBITAL ANATOMY

The orbit is a bony cavity that encases the eye and an intricate network of surrounding soft tissue. Seven bones (frontal, maxillary, ethmoid, lacrimal, palatine, and the greater and lesser wings of the sphenoid) form 4 walls arranged to produce a conical space

widest anteriorly and tapered at the apex. The septum defines the anterior border of the orbit, creating a closed compartment that is 30 cm^3 in volume.[32,33] The bony walls also serve as a barrier between the orbit and the surrounding frontal, ethmoidal, and maxillary sinuses and anterior cranial fossa.[33]

Orbital vessels and nerves pass through several apertures bound by the orbital bones. The superior orbital fissure, formed by the greater and lesser wings of the sphenoid bone, transmits the ocular motor nerves (oculomotor, trochlear, and abducens), branches of the ophthalmic division of the trigeminal nerve (lacrimal, frontal, and nasociliary), a portion of the sympathetic nerve fibers, and the superior ophthalmic vein.[33,34] The inferior orbital fissure communicates the branches of the maxillary division of the trigeminal nerve (infraorbital, zygomatic), the infraorbital artery and vein, the inferior ophthalmic vein, and the parasympathetics to the lacrimal gland. The optic canal, formed by the lesser wing of the sphenoid bone, carries the optic nerve, a portion of the sympathetic nerve fibers, and the ophthalmic artery. The anterior and posterior ethmoidal arteries enter the orbit through the ethmoidal foramina along the frontoethmoidal suture. The zygomaticofacial and zygomaticotemporal canals transmit the zygomatic nerve and vessel branches from the orbit to the temporal fossa and cheek.[33]

The orbital soft tissue protects and enables the eye to function in signal transduction and rotation along 3 axes. The superior oblique and the superior, inferior, lateral, and medial recti muscles originate at the orbital apex; the recti muscles run anteriorly to insert on the globe, forming a muscular cone posterior to the eye. The superior rectus enables elevation, incyclotorsion, and adduction. The inferior rectus facilitates depression, excyclotorsion, and adduction. The medial rectus provides adduction, and the lateral rectus enables abduction. The superior oblique runs in a superomedial direction toward the anterior orbit before it becomes a tendon that attaches at the trochlea; from there, the superior oblique reverses direction to course posterolaterally to insert onto the eye. This pulley system enables incyclotorsion, depression, and abduction. The inferior oblique originates from the maxillary bone periosteum and courses posterotemporally to insert onto the globe. It facilitates excyclotorsion, elevation, and abduction.[33] The oculomotor nerve (cranial nerve III) innervates the superior, medial, and inferior recti. The trochlear nerve (cranial nerve IV) innervates the superior oblique, and the abducens nerve (cranial nerve VI) innervates the lateral rectus.[32,33]

The lacrimal apparatus secretes the aqueous component of tear and provides tear drainage from the ocular surface into the inferior meatus in the nasal cavity. The lacrimal gland, which sits in the superolateral orbit in the lacrimal gland fossa created by the frontal bone, is separated into an orbital lobe and a palpebral lobe by the levator aponeurosis. The aqueous fluid produced by the glands travels in ductules that empty into the superior conjunctival fornix to mix with mucin and oil on the ocular surface.[35] The nasolacrimal drainage system begins at the puncta, which are embedded in the medial margin of each eyelid. Each opens into a canaliculus, which runs medially to the nasolacrimal sac. The sac, located in the lacrimal fossa between the anterior and posterior lacrimal crests, then drains into the nasolacrimal duct. Tears then drain from the duct into the inferior meatus. Contraction of the orbicularis oculi muscle with each blink creates a negative pressure pump system to facilitate tear drainage through the nasolacrimal system.[36]

ORBITAL EVALUATION

Comprehensive orbital evaluation starts with detailed history intake before a systematic approach to physical examination. The latter includes a focused intraocular evaluation and careful inspection of cranial nerve function, preauricular and

submandibular lymph nodes, visual acuity, pupillary reactions, intraocular pressure, extraocular motility, color vision, palpation for resistance to retropulsion, presence or absence of abnormal globe position (proptosis, enophthalmos, hypoglobus, hyperglobus), eyelid position, and visual fields. Measurement of anterior-posterior eye position can be done subjectively by having the patient tilt the head back and looking from the worm's eye view or objectively using an exophthalmometer. Baseline eye position varies among individuals, but asymmetry of greater than 2 mm between the 2 eyes suggests proptosis or enophthalmos of 1 of the 2.[2]

Orbital disease can compromise visual function, lead to permanent blindness, and/or carry systemic clinical significance. Orbital disorders can manifest from inflammatory, mass effect, structural, vascular, and functional (sensory and/or motor) conditions isolated to the orbit, extending from surrounding structures especially sinuses, or as a manifestation of systemic disease.[2] Anatomic and functional considerations can help to localize the disorder. For example, disease affecting the cavernous sinus can be differentiated from orbital apex lesions by noting normal optic nerve function. Fundamental orbital conditions, including preseptal and orbital cellulitis and thyroid eye disease, are discussed here. Important considerations in the evaluation and management of eyelid and orbital trauma are discussed later.

CELLULITIS

Infection of the periocular soft tissue ranges from mild preseptal cellulitis to orbital infections with possible intracranial complications. Sources of bacteria include inoculation from skin trauma, direct extension from adjacent structures (sinusitis, dacryocystitis, endophthalmitis, dental infection), and hematologic spread from distant infection. Infection isolated anterior to the orbital septum constitutes preseptal cellulitis and can be treated with oral antibiotics tailored to the bacterial source. Adjuvant therapy includes warm compresses, nasal decongestants for sinusitis, and incision and drainage for abscess collection. Orbital cellulitis requires intravenous antibiotic therapy and close observation for complications such as optic neuropathy, abscess formation, and cavernous sinus thrombosis.[2] Subperiosteal and orbital abscesses may require surgical decompression to control the infection according to evidence-based guidelines established by Garcia and Harris[37]:

- Patient older than 8 years
- Concomitant frontal and/or chronic sinusitis
- Nonmedial location of subperiosteal abscess
- Large subperiosteal abscess
- Anaerobic infection, including infection from odontogenic source
- Recurrence of abscess after previous drainage
- Optic nerve or retinal compromise

Poorly controlled diabetic and immunocompromised patients are at risk for aggressive invasive fungal sinusitis with orbital involvement, which carries high potential to threaten both vision and life. Causative organisms include *Mucor*, *Rhizopus*, and *Aspergillus*.[38,39] Examination typically reveals proptosis, orbital apex syndrome with complete ophthalmoplegia, decreased vision, and necrotic tissue in the nasopharynx. With severely immunosuppressed individuals, such as inpatients receiving chemotherapy, these symptoms may be mild because of lack of an immune response to the fungal infection. Treatment often requires urgent local surgical debridement through transnasal endoscopic decompression and intravenous antifungals.[2,39]

THYROID EYE DISEASE

Thyroid eye disease (TED), also known as thyroid-associated orbitopathy, thyroid orbitopathy, and Graves ophthalmopathy, is an autoimmune condition characterized by orbital inflammation. Although the exact pathophysiology underlying TED remains partially undefined, research has outlined the mechanism as a loss of immune tolerance to thyroid-stimulating hormone receptor (TSH-R) and insulinlike growth factor-1 receptor (IGF-1R) along with overexpression of IGF-1R. The dysregulation leads to overactivation of orbital fibroblasts, which perpetuate a cascade of orbital inflammation, tissue remodeling, and fibrosis.[40] TED can occur in any state of thyroid function: 90% of patients have Graves hyperthyroidism, 6% are euthyroid, 3% have Hashimoto thyroiditis, and 1% have primary hypothyroidism.[41,42] It affects women 6 times as frequently as men and 7 times more often among smokers compared with nonsmokers.[42,43] Peak incidence is bimodal, occurring among women aged 40 to 44 years and 60 to 64 years and among men aged 45 to 49 years and 65 to 69 years.[41,42]

Active TED is a self-limiting disease that transitions to a chronic inactive phase with a 5% to 10% risk of reactivation. The clinical presentation of active TED varies in severity, ranging from mild dry eyes to severe loss of vision. In mild disease, patients may report foreign body sensation, dry eye, tearing (reflexive), eyelid redness and swelling, eye redness, and spontaneous eye pain. Patients with moderate disease may also notice eyelid retraction, bulging eyes, and possible double vision. Manifestations of severe TED include diplopia, decreased visual acuity and color vision, and loss of visual field in addition to the symptoms of mild and moderate disease.[40–42] Work-up of TED includes full ophthalmic examination, as well as serologic evaluation (thyroid-stimulating hormone, T3, free T4, thyroid-stimulating immunoglobulin, TSH-R antibody, and thyroid peroxidase) and orbital imaging with computed tomography or MRI.[2,43]

Management of TED depends on activity severity. An exception is smoking cessation, which is recommended for all patients.[44] Those with mild disease may be observed or require only supportive therapy, including ocular surface lubrication. For mild to moderate disease, patients may benefit from oral selenium, topical cyclosporine, eyelid taping or moisture chamber goggles overnight, and steroid therapy.[44] Treatment options for active TED causing diplopia and proptosis include oral or intravenous steroids, radiotherapy, and immunomodulatory therapies such as rituximab and tocilizumab.[40] In the chronic phase, patients with persistent proptosis may benefit from orbital decompression, those with strabismus may be managed with prism glasses and/or eye muscle surgery, and patients with eyelid malposition can undergo surgical correction.[43]

TRAUMA

Periorbital and orbital injuries range from benign contusions to devastating blinding conditions. There may be associated ocular surface and intraocular damage that require a full comprehensive ophthalmologic evaluation to assess (see Cynthia A. Bradford and Andrew T. Melson's article, "Ocular Complaints, Disease and Emergencies in the General Medical Setting," in this issue). The following discussion focuses on the evaluation and management of isolated eyelid and orbital injuries.

EYELID LACERATION

Penetrating periocular lacerations can extend to any level of depth and can contain retained foreign bodies. If history (projectiles, stab injury, and so forth) and physical

evaluation (fat exposure that indicates violation of the orbital septum, visible or palpable embedded foreign body, and so forth) suggest deep injury and/or retained foreign body, computed tomography is indicated for further assessment of the extent of injury. Lacerations that appear isolated to the superficial orbit may extend into the intracranial space. Contaminated wounds require debridement and prophylactic oral antibiotics, especially when the injury violated the septum. Mechanisms of injury that carry a high infection risk, such as dog bites and those involving retained organic matter, also warrant prophylactic antibiotics. Inflammatory foreign bodies typically require urgent removal, whereas inert objects without complications may be observed indefinitely. Orbital foreign bodies that incite acute and chronic inflammatory reactions include organic matter, copper, brass, and bronze. Those that remain relatively inert in the orbit include stone, glass, plastic, steel, and aluminum.[45]

The approach to eyelid laceration repair hinges on whether the injury involves the eyelid margin. Wounds that do not involve the lid margin require only superficial skin closure, even if the laceration violates the orbital septum. Topical ophthalmic antibiotic ointment to the wound helps to prevent exposed tissue from granulating before repair and promotes smooth healing after laceration repair.[45] Injuries that involve the eyelid margin warrant referral to ophthalmology for precise approximation and margin eversion of the wound edges to minimize notching and misalignment.[1,45] Complex wounds also benefit from evaluation by an ophthalmologist to rule out occult vision-threatening injuries, such as globe rupture and retinal detachment.

CANALICULAR LACERATION

Lacerations that extend medial to the punctum can signify injury to the canaliculus. Canalicular probing performed by an ophthalmologist assesses the extent of injury and can confirm the diagnosis. Canalicular lacerations should undergo repair with silicone stent insertion within 3 to 5 days to minimize the risk of stenosis, which can result in epiphora and need for additional surgery. Patients should be up to date on tetanus status and may benefit from prophylactic oral antibiotics depending on the mechanism of injury.[1,46]

ORBITAL FRACTURE

Orbital and facial trauma can result in fracture of any wall of the orbit, with or without surrounding soft tissue injury. Patients should undergo ocular examination to rule out concomitant ophthalmic injury even in the setting of blunt trauma and no visual complaints. Emergency sequelae from orbital fractures include retrobulbar hemorrhage causing orbital compartment syndrome, extraocular muscle entrapment, and ocular injuries such as ruptured globe (see Cynthia A. Bradford and Andrew T. Melson's article, "Ocular Complaints, Disease and Emergencies in the General Medical Setting," in this issue).[34] Indications for orbital fracture repair in first few weeks after injury include persistent diplopia in primary and/or downgaze and enophthalmos.[34,47] Large fractures comprising greater than 50% of the floor have a high risk of eventual enophthalmos; however, observation is an option for patients without diplopia and enophthalmos who elect to monitor. Outcomes of surgical repair performed greater than 2 weeks from time of fracture are comparable with those from fracture repair within 2 weeks.[47]

In summary, the eyelids and orbit encompass intricate structures that serve to protect and maintain the function of the visual apparatus. They can present with signs and symptoms of conditions that require urgent testing for life-threatening diseases and/or warrant referral to ophthalmology for further evaluation and management. Early

recognition and action by internists and medical subspecialists play a vital role in the management of these periocular conditions.

CLINICS CARE POINTS

- Signs of eyelid malignancy, including loss of eyelashes, nonhealing ulcers, and recurrent chalazia in the same location, warrant an incisional biopsy or possible referral to an oculoplastic surgery specialist for further evaluation and management.
- Ptosis is a common age-related condition that, nonetheless, may be the presenting symptom of a systemic condition that requires awareness and consideration.
- When evaluating a patient with bilateral proptosis, rule out TED, which is the most common cause of this finding.
- Management of patients with orbital cellulitis includes intravenous antibiotics and close monitoring.
- Patients with eyelid and/or orbital trauma should undergo urgent evaluation and possible repair.

DISCLOSURE

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