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Common Ophthalmic Emergencies

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Abstract and Introduction

Abstract

Ophthalmic emergencies are immediate threats to the visual system that can lead to permanent loss of visual function if left untreated. These emergencies should be detected by physicians and immediately treated and referred to an ophthalmologist if necessary. This article reviews the most common ophthalmic emergency room presentations, the history and physical examination for an ophthalmic emergency, and the diagnosis and management of each condition.

Review Criteria: Information was collected for this review via the following methods: Pubmed search of all literature on ophthalmic emergencies; consultation with common ophthalmic texts; advice and practice from the expertise at Wilmer Eye Institute.

Message for the Clinic: The ophthalmic presentations listed in our article are very common in the primary care setting. It is important that every primary care physician be able to recognise these emergencies, and either treat or refer urgently as described to avoid ophthalmic or even systemic morbidity.

Introduction

Ophthalmic emergencies include conditions that involve sudden threats to the visual system that left untreated can lead to permanent loss of visual function and severe threats to the life or function of the patient. It is of paramount importance that physicians are able to recognise the associated signs and symptoms of such emergencies and are able to instigate urgent components of the work-up and treatment. It is also important that physicians are able to recognise which patients require referral to an ophthalmologist.

This article will review the most common ophthalmic emergency room presentations, will discuss the history and physical examination for an ophthalmic emergency and then summarise the diagnosis and management of the individual conditions that are responsible for the greatest burden of morbidity.

Epidemiology of Emergency Room Presentations

The burden of ophthalmic disease received by emergency departments depends on the social context and healthcare environment in which they exist. Several studies have investigated the most common ophthalmic diagnoses in patients seeking aid in emergency rooms.^[1-5] Many emergency room visits are due to common diseases of relatively minor severity: conjunctivitis,^[1,4,5] corneal abrasions,^[1-5] dry eye^[4] and blepharitis.^[5] Open globe injuries, a vision-threatening condition that requires immediate ophthalmology consultation, made up a substantial number of the presentations in a study from Pakistan,^[2] but were only a minor fraction of the presentations in the other studies.^[1,3-5]

History

Although it may be difficult to determine which ophthalmic presentations are vision threatening and require emergent care, a careful patient history may provide several important symptoms including reduced visual acuity, visual field changes, floaters, photopsia, head, orbital or ocular pain, changed appearance of the ocular adnexae, ptosis, diplopia and alterations in the pupil size. If the symptoms are severe or rapidly progressive, urgent referral to an ophthalmologist is appropriate.

Past ophthalmic and general medical history gives the background for the current presentation. It is important to determine whether the current presentation could be a recurrence or a complication of a previous ophthalmic condition. Always ask about any recent ophthalmic or orbital surgery. Possible complications of ophthalmic surgery include endophthalmitis and other infections; corneal epithelial defects; raised or lowered intraocular pressure (IOP); hyphema; vitreous haemorrhage; and,

retinal detachment. Orbital surgery may be complicated by infection, haemorrhage, and damage to the extraocular muscles or optic nerve. Nasal and sinus surgery may also have orbital complications.

It is also important to be aware of systemic diseases which may have ophthalmic manifestations. Common systemic conditions that affect the eyes include diabetes and thyroid disease, hypertension, autoimmune and inflammatory diseases, infectious disease and malignant disease.

Physical Examination

One of the keys for the non-ophthalmologist to perform a thorough eye examination is to look for asymmetry between the two eyes. Ophthalmic examination is a very specialised area, and referral to an ophthalmologist is often required because of difficulty in interpretation of findings.

Visual Acuity

Visual acuity is the most easily measured element of visual function and should always be assessed before administration of any diagnostic test or treatment. Visual acuity is most commonly graded using a Snellen chart. In ophthalmic clinics the Early Treatment Diabetic Retinopathy Study chart is also often used. In the emergency room an estimate of Snellen visual acuity can be made with a hand-held card. A diagnosis must be made to explain any substantial decline in visual acuity or the patient must be referred to an ophthalmologist.

Visual Field

In an emergency room, visual fields should be evaluated by confrontation testing with the examiner's fingers or hands. Acutely diminished visual fields are most frequently because of retinal (e.g. retinal detachment) and neurological diseases (e.g. stroke).

Colour Vision

Defects in the red-green axis of colour vision are most easily tested using Ishihara plates. Unilateral red desaturation can be tested by asking the patient to compare the colour intensity of a red target, such as the top of a bottle of mydriatic eye drops. Asymmetry of colour perception is usually a sign of optic nerve pathology.

Eye Movements

In the emergency department, eye movements including adduction, abduction, and up- and down-gaze should be checked, as well as combinations of these. In cases of diplopia where an ocular motility problem cannot be determined by these manoeuvres alone, cover and alternate-cover tests at near and distance should be performed. It is sometimes worth checking whether diplopia is present when one eye is covered. Monocular diplopia generally results from abnormalities of the affected eye's optical system.

Pupils

The pupillary light reflex is of great importance in assessing retinal and optic nerve function. Examination of the pupils is also an important part of the evaluation of the oculomotor nerve and of the sympathetic nervous system in Horner's syndrome. Of particular importance is to evaluate for a relative afferent pupillary defect or Marcus-Gunn pupil using the 'swinging light test'. Presence of a relative afferent pupillary defect should lead to ophthalmologic referral.

Intraocular Pressure

Elevated IOP can lead to glaucomatous visual field loss. The rate of field loss depends on the pressure and on the susceptibility of the individual. The most commonly used instruments for accurately measuring pressure are the Tonopen and the Goldmann applanation tonometer. The Tonopen is a small, portable device that is easy to use and is appropriate for use in the ER by a non-ophthalmologist. If no tonopen is available, a crude gauge of IOP can be gained by palpation of the globe through the eyelid.

Anterior Segment

The anterior segment of the eye is best examined using the slit lamp. Biomicroscopic examinations of the lids, conjunctiva, cornea, anterior chamber, iris, lens and anterior vitreous should be performed. Gonioscopy is a highly specialised examination technique used particularly in the assessment of the anterior chamber angle, especially in cases of suspected angle-closure glaucoma. If no slit lamp is available, a hand-held penlight or flashlight can assist in viewing the cornea, iris and pupil.

Posterior Segment

The posterior segment can be examined at the slit lamp using a 90D or similar lens, and by direct and indirect ophthalmoscopy. Most non-specialists are most familiar with direct ophthalmoscopy and this examination should be performed to assess the retina and optic disc head. The retina should be checked, as appropriate, for abnormalities of the vasculature, haemorrhages, oedema, lipid exudates, retinitis, retinal elevation, retinal tears or breaks and retinal detachment. The vitreous should be checked for inflammatory changes, haemorrhage and posterior vitreous detachment. The optic nerve should be evaluated for cupping, atrophy, elevation and swelling, as well as for vascular abnormalities at the nerve.

Adnexae

Lids

Trauma to the lids must be assessed for damage to the tarsal plate, levator palpebrae superioris and canaliculi as well as for other orbital and ocular trauma. Repairs to anything other than skin wounds should be made by a physician experienced in repairing the lid structures. In injuries disrupting the lid margin, perfect apposition of the wound edges must be achieved in order to preserve proper lid function, and these cases are best referred to an ophthalmic surgeon.

Conjunctivitis

Acute conjunctivitis is usually of viral, bacterial or allergic origin. It presents with ocular pain and redness and epiphora and may be accompanied by reduced visual acuity. Viral conjunctivitis may be associated with the presence of a known contact, or a recent upper respiratory tract infection. It usually becomes bilateral and is typically associated with subtarsal follicles and a preauricular lymphadenopathy. There may be corneal subepithelial infiltrates, in which case ophthalmic referral is appropriate. The collection of a swab for diagnostic culture may be appropriate for epidemiological purposes, and is useful in cases of blepharoconjunctivitis where herpetic infection is likely. Care is generally supportive. The patient must take contact precautions to avoid spread of the disease. The physician should ensure that all examination equipment is cleaned with a viricidal cleanser to prevent further spread of the virus.

Bacterial conjunctivitis is most commonly seen in children. It is usually treated with erythromycin ointment. When a purulent conjunctivitis is present, possible exposure to *Neisseria gonococci* should be suspected and bacterial culture should be performed.

Acute allergic conjunctivitis usually presents with sudden development of chemosis and epiphora, often after known exposure to an allergen, and usually resolves in < 24 h and does not require treatment. Any foreign material should be removed.

Orbits

General

The optic nerve and the vascular supply to the eye pass through the orbit, and the orbital contents also include the extraocular muscles. When an orbital problem is suspected the function of all these structures should be evaluated. Optic nerve function should be assessed on the basis of visual acuity, pupillary light reflex and Ishihara colour plate testing. IOP should also be checked. Extraocular movements must be evaluated both for motor nerve palsies and for restriction of motility. Resistance to retropulsion should be evaluated. Proptosis or enophthalmos should be assessed using a Hertel exophthalmometer. Vertical displacement of the globe can also be assessed, and is particularly important in the case of blow-out fractures. If an orbital fracture is suspected, a computed tomography (CT) scan of the orbits with true coronal views is recommended to evaluate the extent of the fracture. If an orbital infection is suspected, the patient's temperature and vital signs should be recorded in addition to CT imaging of the orbits.

Orbital Cellulitis

Orbital cellulitis may present with erythema, tenderness, blurry vision, headache and diplopia. Conjunctival chemosis, purulent discharge, fever, proptosis and restricted ocular motility with pain upon attempted movement may be present. Orbital cellulitis may arise as a complication of orbital trauma, from direct extension of a dental or sinus infection, as a complication of orbital or sinus surgery and from haematogenous spread. Organisms that commonly cause orbital cellulites include *Staphylococcus* species, *Streptococcus* species, *Haemophilus influenzae*, Bacteroides, or gram-negative bacillirods, particularly in trauma cases, direct extension from sinus or dental infection, complications from orbital trauma or eye/paranasal sinus surgery, or spread from the vasculature are all common sources of orbital cellulitis. Mucormycosis must be considered in diabetic or immunocompromised patients, and orbital specimens must be examined when an orbital or ear, nose and throat (ENT) specialist feels that the clinical situation warrants this.

After obtaining history of trauma or focal/and local and systemic illnesses, a complete ophthalmic examination should be performed. One should look for signs of meningitis, afferent papillary defect (APD), limited eye movement, decreased skin sensation, and optic nerve/disc and fundus abnormalities. CT scans of the orbits and sinuses can confirm the diagnosis, and rule out abscesses or foreign bodies which would require surgical management. Laboratory testing of complete blood counts with differential, blood cultures, and gram stain and culture of any drainage can help to target antibiotic therapy.

If orbital cellulitis is diagnosed, an ophthalmology as well as ENT opinion is mandatory, as often the underlying pathology is located in the orbital sinuses. In these cases, broad-spectrum antibiotics, ampicillin/sulbactam or ceftriaxone plus vancomycin, should be administered by intravenous infusion for the first 72 h, and orally for 1 week thereafter. Metronidazole covers anaerobes, and should be considered as well. For patients allergic to penicillins/cephalosporins, vancomycin plus gentamicin or clindamycin plus gentamicin are good substitutes. After initial improvement, amoxicillin/clavulanate or cefaclor can be administered orally for 14 days on an outpatient basis. If symptoms worsen, a follow-up for abscesses, cavernous sinus thrombosis or meningitis should be performed.^[6]

Blow-out Fracture

Traumatic blow-out fractures present with pain that, increases upon vertical eye movement, binocular diplopia and crepitus after nose blowing. Epistaxis and ecchymosis may also be present. Enophthalmos may be present after the oedema has resolved. Trauma without a blow-out fracture may present with similar signs, in which case they usually resolve spontaneously in a week.

An examination of the orbital contents as described above should be performed. Signs of subcutaneous emphysema should be noted. The globe should be evaluated carefully for rupture, hyphema, inflammation, iridodialysis, and retinal or choroidal injury. CT scan of the orbit should be obtained to determine the extent of the trauma and to plan the surgical management if this is appropriate.

Nasal decongestant sprays, broad-spectrum oral antibiotics, and ice packs should all be administered. Surgical repair is emergent within 24 h if CT shows entrapped muscle or tissue with signs of diplopia and gastrointestinal (nausea/vomiting) or cardiovascular symptoms (heart block, bradycardia or syncope). If no entrapped muscle is suspected, surgical repair of the orbital fracture can be delayed for 1-2 weeks, and is indicated in cases of cosmetic deformity or diplopia.

Retrobulbar Haemorrhage

Posttrauma retrobulbar haemorrhage presents with pain, tight eyelid, subconjunctival haemorrhage and proptosis resisting retropulsion. Decreased vision, eyelid ecchymosis, limited extraocular motility and increased IOP may also be present. When the vision is threatened, treatment must be commenced urgently, and full work-up delayed until after the patient's vision has been protected.

If IOP is dangerously increased or vision is threatened, urgent surgical intervention in the form of a lateral canthotomy with or without cantholysis is required and can often prevent permanent visual loss.^[6]

Cornea and Anterior Chamber

Microbial Keratitis

Bacterial keratitis often presents with a painful red eye and a mucopurulent discharge. Slit-lamp examination usually reveals a corneal epithelial defect and an inflammatory infiltrate of the corneal stroma. Signs of severe infection include an anterior chamber inflammatory reaction and hypopyon. Common offenders include *Staphylococcus*, *Streptococcus* and *Pseudomonas* species. Staphylococcal infections often present with a focal stromal infiltrate or abscess, while streptococcal keratitis is often very purulent or crystalline. Pseudomonal keratitis is common in the setting of prolonged contact lens use and often presents with an acute suppurative infiltrate.

Complete history of contact lens use and general eye care should be assessed. Slit-lamp examination with fluorescein stain can help determine any amount of epithelial loss and further characterise corneal infiltrate. Corneal and contact lens smears for bacterial, fungal, acanthamoebal and viral cultures should be performed to determine treatment.

For vision-threatening infections, fortified tobramycin alternating with fortified cefazolin should be given as eye drops every hour. Broad-spectrum antibiotics such as intensive fluoroquinolone drops can be given for uncomplicated cases of community acquired bacterial keratitis. Topical corticosteroids may help reduce inflammation after anti-bacterial therapy has been. A patch should never be placed over an eye considered to be infected as this reduces the removal of pathogens by the tear-film.^[6]

Ocular pain in a contact lens wearer should always be treated with caution. Contact lens wear can damage the ocular surface mechanically, immunologically, by hypoxic stress, by exposure to toxins in lens care media, and by providing a foreign body which can increase risk of development of infections. In particular, contact lens wearers have an increased risk of infection by *Pseudomonas* species, *Acanthameba* and *Fusarium*.^[7] Pain in contact lens wearers should generally be referred to an ophthalmologist.

Herpetic keratitis usually presents with a watery, red eye and decreased visual function. Slit-lamp examination using fluorescein classically demonstrates a dendritic ulcer. A viral swab should be collected, as documentation of herpes simplex infection may be important in subsequent management. Ophthalmic referral is necessary to start anti-viral therapy.

Corneal Foreign Body

Corneal foreign bodies present with pain, foreign body sensation and often with decreased vision. Slit-lamp examination reveals a foreign body on the surface of the cornea. Evaluation should rule out the possibility of a full-thickness corneal laceration. Most corneal foreign bodies can be removed at the slit lamp using topical anaesthesia and a 27-gauge needle by a skilled ophthalmologist or physician. Prophylaxis of microbial keratitis is usually accomplished using erythromycin ointment. Pain control may include the use of a pressure patch, non-narcotic analgesics and cold compresses.

Corneal Abrasion

Foreign body sensation in the absence of a corneal foreign body is often because of a corneal abrasion. This can usually be diagnosed at the slit lamp, and fluorescein dye can often aid in determining the area of abraded corneal epithelium. It is important to evert the upper and lower eyelids to detect a foreign body that may be causing the abrasion. Topical antibiotic eyedrops or ointment is usually recommended to prevent infection.

Chemical Injury

Chemical injuries range in severity from superficial punctate keratitis to corneal opacification with limbal ischemia. Acids and irritants can damage the cornea and conjunctiva. However, the most feared chemical insult to the cornea is an alkali burn, and these injuries should be managed with particular care. Ideally the management of corneal burns begins at the site of injury, where copious irrigation with tap water should be employed. In the emergency room irrigation should be commenced immediately with saline or Ringer lactate. In the case of an alkali burn the irrigation should continue for at least 30 min, or until the inferior cul-de-sac reaches a neutral pH of 7.0 by litmus paper test. Acids and alkali should never be used to neutralise each other.

After irrigation examination should include visual acuity and a slit-lamp examination to assess conjunctival and corneal damage. Lid eversion must be performed to search for foreign bodies. IOP may be increased and therefore should be checked.

Any foreign bodies or residual chemical crystals should be removed. Necrotic tissue should be debrided. Any conjunctival adhesions should be gently broken with a glass rod covered with antibiotic ointment. Depending on the injury it may be

appropriate to treat with a topical antibiotic such as erythromycin, prednisolone acetate 1%, a cyclopegic agent such as atropine or homatropine. Additional lubrication using artificial tears and oral pain medication are used as needed. Significant intraocular hypertension should be treated. An ophthalmologist should assess all alkali injuries as well as severe chemical injuries from any other cause. There is some evidence that more severe alkali burns may benefit from a regime of intensive topical steroids, ascorbate and citrate.^[8] The long-term visual rehabilitation of a severe corneal burn is complex and is usually managed by a corneal specialist.

Acute Angle Closure Glaucoma

Acute ocular hypertension or 'glaucoma' presents with ocular pain, decreased vision, frontal headache, nausea and coloured halos around lights. IOP is increased and slit-lamp examination sometimes shows corneal microcystic oedema. The most common cause of acutely raised IOP is acute angle closure. Most cases are related to narrow anterior chamber angles, often because of increased lens thickness. Other important causes of angle closure, which are important to differentiate so that appropriate cause specific treatment may be initiated are, neovascular glaucoma and some cases of uveitic glaucoma. Patients may have acutely increased IOP, corneal microcystic oedema and a shallow anterior chamber in both eyes. East Asians and the Inuit are genetically predisposed to shallow anterior chambers with narrow angles. It is sometimes triggered by mydriatic use. Complete evaluation and treatment of angle closure glaucoma require immediate ophthalmic referral. Peripheral iridotomy in the affected eye and usually also in the fellow eye are required in cases of anatomically narrow angles.

Globe

Ruptured Globe

A ruptured globe from a penetrating ocular injury usually presents with decreased vision, subconjunctival haemorrhage, hyphema and irregular shaped pupil. A traumatic cataract and full-thickness scleral or corneal lesion may also be present. The lens may become subluxed and vitreous haemorrhage may also be present.

A patient with a ruptured globe with penetrating ocular injury should be immediately seen by an ophthalmologist because urgent surgical repair is often required.^[9] Prior to surgery, the eye should be protected with a shield and systemic antibiotics should be given as soon as possible (cefazolin with moxifloxacin for adults and cefazolin with gentamicin for children under 12 years). Tetanus toxoid should be administered and an anti-emetic should be used *pro re nata* to reduce the risk of expulsive haemorrhage. CT of the orbits and brain is necessary to localise injuries, rule out foreign bodies and plan surgical repair. B-scan ultrasound should be avoided in the setting of a ruptured globe, as the injury could be made significantly worse from the pressure of the ultrasound probe.

Intraocular Foreign Body

Some intraocular foreign bodies can be very difficult for the non-ophthalmologist to detect on examination. The occurrence of any ocular problem following striking metal upon metal should lead to referral to an ophthalmologist even if the examination appears normal. Examination should be performed to assess visual acuity, the foreign body's entry site, whether the entry wound is self-sealing or still leaking, whether there is any prolapse of intraocular tissue and the extent of damage to the globe, especially to the lens and retina. An iris transillumination defect should alert the examiner to the possible presence of an intraocular foreign body in the posterior segment. Pupil irregularity or the presence of hyphema or vitreous haemorrhage are other clues. CT scanning with 1 mm or finer cuts and B-sound ultrasonography are the best imaging modalities for the detection of foreign bodies.

If visible, penetrating foreign objects should not be removed immediately so as to avoid extrusion of intraocular contents. Any intraocular foreign body will require removal by an ophthalmologist, and in the case of posterior segment foreign bodies, the expertise of a vitreo-retinal surgeon is required. Prior to surgery, a shield should protect the eye, and tetanus prophylaxis, intravenous antibiotics (generally vancomycin and ciprofloxacin), and a cyclopegic should be given.

Retina

Retinal Vein Occlusions

Retinal vein occlusions are a common cause of sudden, painless loss of vision, but can also have a more gradual onset. Vein

occlusions are classified as either central (CVO) or branch (BVO) vein occlusions. CVOs generally occur in the vicinity of the lamina cribrosa in the optic nerve head. BVOs usually occur at the sites of arterio-venous crossings in the retina.

The extent of visual loss is extremely variable, especially in CVO. The extent of visual field affected in BVO corresponds to the area of affected retina. The most impressive fundal finding in both conditions is the presence of multiple intraretinal haemorrhages of varying severity. In the case of BVO these haemorrhages occur in the area drained by the affected venule. In the case of CVO the entire retina is affected. Cotton wool spots may occur. The obstruction site may be evident in BVO. Swelling of the optic disc frequently occurs in CVO.

Vein occlusions sometimes present with vitreous haemorrhage secondary to optic disc or retinal neovascularisation or with neovascular glaucoma because of neovascularisation of the anterior chamber angle.

While no emergency management is required, consultation with an ophthalmologist must be arranged to monitor and treat neovascularisation and macular oedema. Follow up with the patient's primary care physician is also warranted to minimise risks such as hypertension and diabetes. In the case of bilateral simultaneous CVO and CVO in people under the age of 60 years a review of possible haematological and inflammatory causes of venous thrombosis and obstruction should be performed. An internist should take a careful history and perform a physical examination and laboratory work-up should be directed by the findings on history and examination. The basic work-up could include a complete blood count, erythrocyte sedimentation rate, fasting plasma homocysteine levels, anti-nuclear antibody and anti-phospholipid antibody levels.

Central Retinal Artery Occlusion

Central retinal artery occlusion (CRAO) presents with unilateral, acute, painless loss of vision. Often, patients may reveal a history of amaurosis fugax.^[10] Embolus from the heart, aorta or carotid arteries, giant cell arteritis (GCA) (see below), other collagen vascular diseases, and hypercoagulability disorders are all causes of acute CRAO.

On examination, white retinal oedema and a cherry-red spot in the macula can be detected. There is attenuation of the retinal arterioles and box-car-shaped blood columns may be observed.

Ocular massage has been documented to lead to resolution of CRAO in some instances. Therefore CRAO is a true emergency. Acute, painless loss of vision must be triaged at high priority and ocular massage should be performed immediately once the diagnosis is made. There is no other proven treatment for CRAO. Irreversible damage to the retina occurs within a few hours of the initial occlusion, and hence the window for intervention is short. Ocular decompression by anterior chamber paracentesis sometimes leads to dislodgement of the embolus so that it moves distally in the vascular tree where it may cause less visual loss. This should be performed by an ophthalmologist. Arterial thrombolytics are under investigation for treatment of this condition but are not recommended at this time.^[10]

Blood tests, such as complete blood count, a hypercoagulability work-up, erythrocyte sedimentation rate and C-reactive protein (CRP) should be performed. Carotid artery duplex Doppler ultrasonography and complete cardiac evaluation should be performed to determine whether there is a treatable embolic source.

Retinal Tear

Tears in the retina can be precursors to retinal detachment, as they allow fluid from the vitreal cavity to enter the potential space between the retina and retinal pigment epithelium. They require semi-urgent evaluation and treatment. Retinal tears typically present with photopsia or multiple floaters. The photopsia represent the sensory perceptions because of retinal traction. Floaters may occur when a retinal tear crosses and damages a retinal vessel resulting in a vitreous haemorrhage. Retinal tears are diagnosed ophthalmoscopically. An ophthalmoscopic examination for suspected retinal tear is not complete without circumferential scleral depression by an ophthalmologist. Where the view of the retina is impaired, typically because of vitreous haemorrhage, ultrasonographic examination is required. Retinal tears are most commonly treated by an ophthalmologist using laser retinopexy or cryotherapy. The referral should be made within 24 h.

Retinal Detachment

There are three types of retinal detachment classified by aetiology: rhegmatogenous retinal detachment is due to a tear or break in the retina; exudative retinal detachment occurs when inflammatory, neoplastic or other exudative processes cause

fluid to leak into the subretinal space and tractional retinal detachment where neovascular membranes, most commonly because of proliferative diabetic retinopathy, pull the retina forward.

Retinal detachment most commonly presents with loss of vision or a visual field defect described as a 'veil' or 'fog'. Acute traction on the retina often gives the appearance of flashing lights or photopsia. A torn retinal blood vessel can lead to vitreous haemorrhage which presents with floaters. Visual acuity and visual field should be determined. The diagnosis of retinal detachment is made on ophthalmoscopy or by B-scan ultrasonography if no clear view to the retina is available.

Endophthalmitis

Endophthalmitis refers to an infection of the contents of the eye. The infection may enter the eye as a result of surgery, trauma, haematogenously or secondary to infection of a trabeculectomy bleb or of the cornea or sclera. Acute endophthalmitis presents with rapidly deteriorating vision and pain. Decreased visual acuity, hypopyon and vitritis are the most common signs.

Early identification of endophthalmitis and immediate referral to an ophthalmologist will increase the probability of saving the patient's eye. The ophthalmologist will obtain a sample of intraocular fluid to send for immediate bacterial and fungal culture. Most cases of endophthalmitis are treated with intravitreal antibiotic injections. Vitrectomy surgery is usually reserved for cases in which the visual acuity is light perception.

Common organisms cultured in endophthalmitis cases include *Staphylococcus epidermidis*, *Staphylococcus aureus*, and Streptococcal species, while traumatic endophthalmitis is more often associated with *Bacillus* species, *S. epidermidis*, gram-negative species, and mixed flora. Infection of a trabeculectomy bleb is associated with Streptococcal or gram-negative bacterial infections.

Most cases of endophthalmitis are treated with intravitreal vancomycin and ceftazidime. In most cases, intensive topical steroids (prednisolone acetate), topical fortified antibiotics (vancomycin and tobramycin), and atropine should be administered, while trauma patients may benefit from tetanus toxoid as well.^[6] The Endophthalmitis Vitrectomy Study did not demonstrate any value in treating endophthalmitis with intravenous antibiotics.^[11]

Optic Nerve and Neurological

Giant Cell Arteritis

Giant cell arteritis is an autoimmune disorder in the elastic lamina of arteries, causing luminal obstruction and ischaemia.^[2] GCA causes a rapidly progressive, irreversible optic neuropathy. The headache that accompanies GCA is typically associated with scalp tenderness because of superficial temporal arteritis and with jaw claudication during chewing. Systemic symptoms such as fatigue and malaise, chills and loss of weight are common. GCA commonly occurs in association with polymyalgia rheumatica. Transient visual obscurations maybe a symptom of impending optic neuropathy. Arteritic ischaemic optic neuropathy presents with acute, unilateral visual loss that quickly becomes bilateral. Other ophthalmic manifestations include CRAO and palsies of the third, fourth and sixth cranial nerves.

When GCA presents with a headache it must be diagnosed and treated so that optic neuropathy can be avoided. Therefore the suspicion of the diagnosis of GCA must be entertained whenever a patient over the age of 50 years present with a temporal headache. When the presentation is of unilateral arteritic ischaemic optic neuropathy then treatment must be started in an attempt to protect the other eye from developing a vascular occlusion. Work-up should include a comprehensive eye examination. In cases of arteritic anterior ischaemic optic neuropathy, a relative afferent pupillary defect is present and the optic disc is swollen. Retinal cotton wool spots may be present.^[10] Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are usually elevated. Temporal artery biopsy is diagnostic, and may be performed up to a week after initiation of steroid therapy. GCA causing an optic neuropathy is treated with intravenous methylprednisolone.

Cavernous Sinus Thrombosis

Cavernous sinus thrombosis can present with bilateral chemosis, eyelid oedema, eye movement abnormalities and proptosis. Often, patients develop fever, nausea and altered consciousness. The thrombosis usually results from extension of an infection (usually *S. aureus*), or aseptically from trauma or surgery.

The ophthalmic examination should focus on identifying any cranial nerve palsies, with particular attention devoted to observing pupillary reflexes for an APD, extraocular motility, trigeminal nerve function, ptosis, exophthalmia and resistance to retropulsion. CT scan or magnetic resonance imaging (MRI) of the sinuses, orbit and brain can help diagnose this condition. Two to three sets of peripheral blood cultures as well as a culture from the source of infection should be obtained to help define treatment.

Co-management with paediatricians or internists is usually appropriate. Management generally includes hospitalisation, intravenous fluid replacement, and intravenously administered antibiotics for several weeks. Nafcillin (or vancomycin if patient is penicillin allergic) plus ceftazidime can be used initially until culture results are obtained. For non-infectious causes, systemic anti-coagulation or aspirin is recommended.

Horner's Syndrome

The classic triad presentation of Horner's syndrome is unilateral ptosis, meiosis and facial anhidrosis. The meiosis often presents as anisocoria, most apparent in dim light, with the smaller pupil on the affected side. Causes of Horner's syndrome include internal carotid dissection, trauma, cluster headaches, herpes zoster infection, Pancoast tumour and stroke.

The most important diagnosis to make or exclude in an emergent fashion is internal carotid artery dissection. This diagnosis may be excluded using catheter angiography, CT angiography, or a combination of MRI and magnetic resonance angiography. Combined carotid artery MRI and magnetic resonance angiography (MRA) are now favoured by many practitioners as they are minimally invasive and highly sensitive.^[12]

Third Nerve Palsy

A third nerve palsy presents with diplopia and ptosis. It may be accompanied by periocular pain, which is particularly frequent in cases of aneurysmal compression. The affected eye will have impaired motility, and appear turned down and out, especially in cases of nerve compression, and will often have a dilated pupil that is minimally reactive to light. The most common causes of third nerve palsy are microvascular infarcts, typically occurring in people with diabetes, hypertension or atherosclerosis, and a berry aneurysm, usually of the posterior communicating artery (PCA). Other causes of this condition include brain tumour, uncal herniation and pituitary apoplexy.

Work-up should include a complete history including risk factors for GCA, known cancer or central nervous system mass, hypertension, diabetes mellitus and recent infections. Blood pressure should be measured. A complete ocular examination, including pupillary reflexes, extraocular motility and visual fields should be performed, along with a full neurologic examination, assessing all other cranial nerves. In cases of non-pupil sparing third nerve palsy a gadolinium-enhanced MRI/MRA is best able to image a mass or aneurysm impinging on cranial nerve III (CN III). PCA aneurysms should be referred urgently to a neurosurgeon. Non-pupil sparing third nerve palsies where an aneurysm is not detected should be further evaluated by a neurologist or neuro-ophthalmologist. Other tests may include ESR, and fasting blood sugar and glycosylated haemoglobin.

Homonymous Hemianopia

Patients with homonymous hemianopia present with bilateral loss of vision of either the right or left visual field, but have normal pupillary responses. This disorder can be due to any unilateral lesion of the optic tract posterior to the optic chiasm. Stroke, tumour, haemorrhage, demyelinating disease and infection such as Progressive Multifocal Leukoencephalopathy (PML) may also present with homonymous hemianopia.^[10] A homonymous quadrantanopia has similar localising significance, but is most likely to be secondary to a lesion of the optic radiations.

This presentation calls for complete ocular and neurologic examination, including MRI of the brain, and evaluation of stroke risk factors. Emergent ECG should also be performed to rule out myocardial infarction or atrial fibrillation.^[10] Where an acute cerebral vascular accident (CVA) is diagnosed it should be managed urgently by an internist or neurologist.

Summary

Various ophthalmic emergencies may be encountered by the non-ophthalmologist. Basic knowledge of the ocular anatomy and common vision-threatening conditions are necessary to properly diagnose and triage these patients. Although

ophthalmology consultation is often required to manage complex ocular disorders, the non-ophthalmologist can play a critical role in preserving eyesight for patients who present with acute emergencies.

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