Eye signs in anaesthesia and intensive care

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Abstract

Ophthalmology can be unfamiliar territory for the anaesthetist and intensivist. This article describes the signs associated with iatrogenic injury to the eye in the operating theatre and the intensive care unit. Patients are at risk of corneal abrasions, exposure keratopathy and chemosis. The prone patient encounters an additional risk of ischaemic optic neuropathy and acute glaucoma. In the intensive care setting, the intensivist should be alert to signs of ocular infection, for example, conjunctivitis, microbial keratitis and endophthalmitis. In the trauma patient, careful evaluation of the eye is required to rule out sight-threatening conditions such as retrobulbar haemorrhage, globe rupture, orbital fractures and retinal detachment.

Keywords Endophthalmitis; exposure keratopathy; globe rupture; prone position; retrobulbar haemorrhage; trauma

Royal College of Anaesthetists CPD Matrix: 2C03, 2C04, 2F01, 3A12

Learning objectives

After reading this article, you should be able to:

- recognize the signs of ocular damage that can arise during anaesthesia and intensive care
- identify signs of infection in the eye on the intensive care unit
- recognize the signs of ocular trauma, including sight-threatening pathology

Signs of ocular damage in anaesthesia and intensive care

Corneal abrasion

Accidental injury to the cornea can result in an abrasion, a superficial scratch to the surface epithelium, which manifests as a painful red watery eye. This epithelial defect is best seen with fluorescein eye drops, especially under blue light, where it stains green. Careful closure and taping of the eyelid reduce the risk of accidental corneal abrasion. Abrasions tend to heal rapidly and are treated with chloramphenicol ointment and lubrication. Mydriatic drops and an eye patch may be used to help with discomfort.

Exposure keratopathy

The health of the corneal surface relies on moisture from tears. Incomplete closure of the eye or reduced blinking can lead to rapid evaporation of the tear film which leads to a dry and compromised ocular surface. This can be exacerbated by high oxygen flows through ill-fitting masks or nasal cannulae and even fans used in the intensive care unit.[1] Exposure keratopathy can manifest as a red eye, and the epithelial defect will look like a corneal abrasion under fluorescein drops and blue light.

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Exposure keratopathy is common in the anaesthetized, paralysed patient, especially in the intensive care setting, where the risk increases with length of stay and duration of mechanical ventilation.[1] Prolonged exposure keratopathy can lead to progressive corneal thinning, and even corneal perforation in extreme cases. Moreover the cornea, without its protective epithelium, becomes vulnerable to infection – microbial keratitis (a type of corneal ulcer) may occur.

The treatment for exposure keratopathy includes frequent lubrication with drops or ointments. Moisture chambers using a plastic wrap can also be used to reduce evaporation from the ocular surface. The eyelid may also be closed mechanically, by using tape or with sutures. This can be performed by the ophthalmologist at the bedside.

Chemosis

Chemosis is bulging of the conjunctiva caused by oedema. In anaesthesia, this can be a side effect of eye blocks such as sub-Tenon block. It is more likely in certain positions, such as prone and steep Trendelenburg positions. In the intensive care setting, risk is increased with poor venous return and oedematous states.

Chemosis can lead to exposure keratopathy due to the prolapsed conjunctiva protruding through the closed eyelids. Severe chemosis can also impair physiological lubrication of the adjacent cornea, leading to localized corneal dryness and thinning known as a dellen. If untreated, the dellen can in severe cases progress to corneal perforation.

Anaesthesia in the prone position

The patient in the prone position is vulnerable to ocular damage of varied aetiology, including chemosis, exposure keratopathy and corneal abrasions. Direct eye compression or raised orbital pressure can occur. This, combined with severe or recurrent hypotension, can lead to ischaemic optic neuropathy and central retinal artery occlusion. This will present with painless visual loss.

Rarely, patients in the prone position can develop acute primary angle closure glaucoma, where a rise in intra-ocular pressure leads to retinal or optic nerve ischaemia.[1] The cornea becomes cloudy and grey and the pupil is fixed in the mid-dilated position and is unresponsive to light. This requires immediate ophthalmological treatment.

Signs of infection

Conjunctivitis

A sticky discharge from the eye, with or without redness, may indicate the presence of conjunctivitis. Eye swabs should be taken due to the increased likelihood of atypical organisms. Infection control precautions should be strictly followed.

Microbial keratitis

Colonization of the eye with bacteria occurs in a time-dependent fashion on the intensive care unit, mostly as a result of exposure to respiratory secretions.[1]

Bacterial invasion of the damaged cornea in exposure keratopathy can occur rapidly. The patient may have a red, sticky eye and a corneal ulcer. The cornea may also become dull or develop a white patch. This requires urgent ophthalmological review.

Although bacterial infections are more common, immunocompromised patients can also develop herpes simplex keratitis.[1] This has the typical appearance of dendritic corneal ulcers, most visible under fluorescein and blue light.

Endophthalmitis

Endophthalmitis is a serious, sight-threatening intra-ocular infection and an ophthalmological emergency. An awake patient may complain of severe pain and worsening vision. The patient may have a red eye, although this may be more subtle than expected. The presence of a hypopyon, a white line visible in front of the iris, is highly suspicious of endophthalmitis. Immediate ophthalmological review should be sought.

Endogenous bacterial endophthalmitis can be caused by spread of systemic infection through the blood stream, and a source of sepsis should be sought.

Treatment is with intravitreal antibiotics. A vitrectomy may be required.

Fungal endophthalmitis is less common but should be actively excluded by ophthalmological review if the patient has a positive blood culture or line tip for candida, aspergillus or any other fungal organism. Treatment should be with an anti-fungal agent that penetrates the eye, such as fluconazole or voriconazole — intravitreal administration might also be required.

Ocular signs in trauma

Any trauma to the globe or orbit requires a careful examination by an ophthalmologist.[2,3] The most serious conditions to be ruled out, through examination and/or imaging, include:

- retrobulbar haemorrhage
- globe rupture
- orbital fractures
- secondary raised intraocular pressure (IOP), e.g. due to hyphaema
- basal skull fracture (especially with bilateral ring periocular ecchymoses).

Retrobulbar haemorrhage can lead to ocular compartment syndrome and compression of the optic nerve. The patient may complain of reduced vision. Prompt diagnosis and treatment is essential to preserve visual function.

Orbital fractures most commonly involve the maxillary bone (orbital floor), but the medial, lateral or frontal walls can also be involved. Common features include globe displacement and diplopia, the latter occurring as a result of entrapment or damage of an extraocular muscle.

Globe ruptures can be anterior or posterior — posterior ruptures can be hard to diagnose. If globe rupture is suspected, manipulation should be kept to a minimum. Patients with disrupted globe integrity will be at high risk of endophthalmitis and need urgent surgical exploration and repair.

Table 1 describes the signs in the above conditions.

Patients with *lid lacerations* should have careful wound exploration and assessment of the integrity of the globe. The type of repair will depend upon the depth of the laceration, involvement of the eyelid margin or adnexal structures and whether there has been loss of tissue.

A *periocular haematoma* or ecchymosis (diffuse bruising) and/or *periorbital oedema* are commonly seen in blunt force trauma. It is usually benign. Severe periorbital oedema may restrict eye movements and lead to diplopia which normally resolves spontaneously.

Ocular signs in trauma

Condition	Signs
Retrobulbar haemorrhage	Proptosis Haemorrhagic chemosis Ecchymosis Raised IOP Reduced visual acuity Relative afferent pupillary defect
Globe rupture	Distorted pupil Scleral or corneal lacerations Hyphaema Reduced IOP Herniation of intraocular contents (usually vitreous or dark uveal tissue) Enophthalmos (posterior displacement of globe within orbit) OR Proptosis (protrusion of the globe), if associated with periorbital swelling/ retrobulbar haemorrhage
Orbital fracture	Periorbital haematoma or oedema Globe displacement • Enophthalmos in orbital floor or medial wall fracture. • Proptosis or axial displacement may also occur. Diplopia Sensory changes (infraorbital anaesthesia due to damage of maxillary branch of trigeminal nerve in orbital floor fracture)

Table 1

A *hyphaema* is a haemorrhage in the anterior chamber. This can be total or partial (with a fluid level in the anterior chamber).

Subconjunctival haemorrhages can look dramatic but are usually innocuous, unless associated with a dellen.

Trauma can affect the posterior segment of the eye in several ways, for example by causing vitreous haemorrhage, retinal haemorrhages or oedema, retinal breaks or detachment and other sight-threatening conditions. A detailed examination with dilated fundoscopy is required to diagnose these.

Chemical eye injury

Chemical eye injuries are a true ocular emergency.[3] Immediate irrigation is indicated before a full history and examination are performed. The ocular surface and anterior segment can sustain severe damage, especially in alkaline burns.

Acute features may include a red eye with or without chemosis. A white eye indicates blanching of the conjunctival vessels in ischemia and is a poor prognostic factor. The cornea may have a cloudy appearance due to oedema. Fluorescein staining may reveal corneal epithelial damage.

Following irrigation, initial treatment often includes adequate analgesia, lubricants, and topical antibiotics and steroids.

Thermal eye injury

Thermal injuries can affect the eyelids and ocular surface.[3] Clinical features of thermal eye injury include a red eye (or white in ischaemia), chemosis, corneal opacification or perforation.

Acute thermal injury to the eyelids is treated with lubrication, topical antibiotic ointment and dressings or a moisture chamber. If there is a risk of corneal exposure, extra procedures, such as sutures or skin grafts, may be required.

Thermal injuries involving the ocular surface are treated in a similar manner to chemical burns.

Signs in traumatic brain injury

Traumatic brain injury is associated with numerous pupillary and eye movement abnormalities, as a result of direct or indirect injury to the eye or cranial nerves II, III, IV and VI.[4] Lesions in the pons and mid-brain also contribute to these abnormalities.

Optic neuropathy as a result of direct trauma may present with immediate and irreversible blindness and is associated with a relative afferent pupillary defect.

The cranial nerves are vulnerable due to their position and intracranial course. Oculomotor paresis may be present as intracranial blood or oedema leads to uncal herniation and compression of the nerve. Abducens neuropathy may occur following damage to petrous temporal bone damage, or as a false localizing sign in raised intracranial pressure.

Bilateral pupillary dilatation is an adverse prognostic sign in severe traumatic brain injury.[5] In comparison, pupil asymmetry is associated with better prognosis and may suggest the presence of a space occupying lesion amenable to surgical intervention. ◆

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