Chapter 9

Exposure Keratopathy in the Intensive Care Unit: Do Not Neglect the Unseen

Benjamin Bird, Stephen Dingley,
Stanislaw P. Stawicki and Thomas R. Wojda

Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/intechopen.72791

Abstract

Exposure keratopathy (EK) is a frequently overlooked complication seen in nearly 60% of sedated or intubated intensive care unit (ICU) patients. Signs and symptoms of EK often start as mild subjective complaints of eye pain and irritation, but can progress to vision loss in the most severe cases. For many critically ill patients, the presence of sedation effectively precludes their ability to communicate clinical complaints typically associated with EK. This, combined with the potentially severe sequelae, makes EK a potentially preventable complication and a patient safety issue. Clinical management of EK can be challenging for both providers and patients due to the nature of treatment with eye drops and ointments as well as the burden and expense of associated procedural interventions. Risk factors for EK have been extensively described in the literature, and wider dissemination of this knowledge should facilitate education of physicians and nurses regarding EK prevention. The most common risk factors include lagophthalmos, chemosis, Bell's palsy, and congenital deformities. Additionally, critically ill patients are less likely to be promptly diagnosed due to the focus of staff on life-threatening problems over ocular prophylaxis. However, the potential severity of complications associated with EK mandates that prevention remains a crucial component of the care of at-risk patients. The reader will explore the broad category of adverse medical occurrences included under the umbrella term, "errors of omission" (EOO): an error category that is most likely to culminate in EK. The most critical preventive measure is education of health care providers, although this may not be enough by itself. To this end, universal precautions against EK in combination with education may be used to help combat the relatively high incidence of this easily preventable ocular pathology.

Keywords: exposure keratopathy, ophthalmology, critical care, patient safety

1. Introduction

Intensivists caring for high acuity patients must be able to actively track an impressive number of clinical variables on daily basis. Failure to do so may be associated with significant morbidity and mortality in cases involving omissions or systemic failures [1, 2]. While management priorities of the critically ill patient must emphasize life-threatening problems, other issues with less immediate consequences may go unnoticed. Exposure keratopathy (EK), noted to occur in 37–57% of sedated or intubated ICU patients, is one of those "silent" morbidities [3–6]. Visual impairment or loss of vision due to EK, especially when secondary complications such as corneal infection are present, is a serious problem that can arise if proper precautions are not observed, and has the potential to cause long-term disability [7–9].

The preventability of EK is predicated on the assumption that its occurrence is frequently a result of omission, broadly included under the category of, "errors of omission" or EOO [8, 10]. Closely related to EOO is the associated companion phenomenon of "delay to treatment" (DTT) which results in further propagation of the potentially preventable problem amidst many competing treatment priorities [8, 10–15]. In support of the latter argument, and in an attempt to prevent overall harm, it has been suggested that implementation of relatively simple protocols can help prevent most cases of EK in the ICU. In fact, implementation of evidence-based nursing practices and protocols may be the key to significantly reducing the incidence of exposure keratopathy [9, 16]. In this chapter, we will discuss various prevention strategies, as well as explore the pathophysiology, etiology, and treatment of ophthalmic EK.

2. Patient vignettes

In the following section, the authors will introduce clinical aspects of EK by presenting two patient vignettes that demonstrate the usual genesis and course of EK in the ICU. In addition, a third patient vignette will outline a fairly typical outpatient scenario involving EK. Because the main objective of this chapter is to present patient safety (PS) considerations with a clear focus on prevention, clinical management of EK in each of the three cases will not be discussed in detail.

Clinical Vignette #1. T. W. is a young man in his mid-20s who was involved in a motor vehicle collision, and presented to the local hospital's emergency department with serious injuries that required multiple surgeries. He has required an extended, 4-month stay in the ICU, a significant portion of which was spent under deep sedation for traumatic brain injury. Upon awakening and recovering his mental status, the patient complained that his eyesight was "extremely blurry" and that he had significant pain and tearing in both eyes. An ophthalmology consult showed that the patient was noted to have >20/400 vision in both eyes, despite having 20/20 vision before the car crash. A dilated fundus exam was performed and revealed no gross abnormalities. A fluorescein stain was then performed on the lens and cornea of both eyes leading to the diagnosis of EK.

Clinical Vignette #2. V. C. is a Caucasian male in his early 50s who underwent an elective blepharoplasty. Because of his anxiety regarding "objects too close to his eyes," the patient requested general anesthesia. Unfortunately, the patient's surgery was complicated by a deep

orbital hemorrhage in the left eye requiring a stay in the hospital. Due to multiple hospital-acquired infections and procedures during the ensuing hospitalization, he required escalation of care to multiple weeks spent in the intensive care. During the ICU stay, the patient was noted to have a degree of lagophthalmos in his right eye due to the recent blepharoplasty. He developed deep redness, pain, and severely impaired vision in his right eye. An ophthalmology consultation confirmed that he has EK via fluorescein stain.

Clinical Vignette #3. L. P. is an African American female in her late 70s who presented to the ophthalmology clinic reporting 5 years of bilateral "blurred vision." She was found to have cataracts and surgical correction was recommended. The patient was noted at the time to have significant proptosis as a result of pre-existing graves' disease, but no other medical history was deemed contributory at this time. The patient had the cataracts removed during two surgeries scheduled separately over a 6-month period, without any perioperative complications. The day after her second surgery, she returned to her ophthalmologist with the complaint of "foreign body sensation" underneath the bandage over her left eye. Upon uncovering the eye and examining it, the ophthalmologist found that the patient's eyelid was not fully closed due to proptosis. The patient was noted to have a yellow film over her eye and the diagnosis of EK was made after fluorescein testing and a dilated fundus exam.

3. Summation of clinical vignettes

All three clinical vignettes presented above demonstrate fairly typical presentations of EK. For patient #1 and patient #2, the circumstances leading to the development of keratopathy included an extended stay in an ICU, in which proper ophthalmic preventive care presented significant opportunities for improvement. In the case of patient #3, the simultaneous presence of a post-operative complication and a pre-existing condition predisposed her to EK. In all cases, patients showed similar symptoms, including a change in the color of the eye, the appearance of pain, and the concurrent decrease in visual acuity. In all of the above examples, early detection was critical in terms of avoiding disease progression, instituting prompt treatment, and preventing the loss of vision. The most important question, from the etiologic standpoint, is whether these cases of EK could have been prevented. Did any EOO's contribute to the genesis of EK in one or more of these occurrences? After discussing the pathophysiology, clinical characteristics, and risk factors associated with EK, the authors will provide an overview of EOO's in the context of our case vignettes and potential preventability.

4. Exposure keratopathy: pathophysiology, diagnosis, symptoms, and risk factors

Corneal epithelium helps defend the eye from external exposure and related insults [17, 18]. It is composed of avascular, stratified, nonkeratinized epithelium, which is intimately associated with the maintenance of physiologic homeostasis of lachrymation [19–21]. Tears provide lubrication to the surface, oxygen to the cornea, wash away pathogens, and adhere to the eyes via mucins produced by the corneal epithileum [22]. Lysozyme, lactoferrin, tear lipocalin, and

secretory Immunoglobulin A (IgA) help prevent infection [23, 24]. The palpebral conjunctiva moves over the cornea during blinking and dispenses/distributes tears uniformly over the ocular surface, thus inhibiting evaporation [25–27]. Tear evaporation modifies the conjunctival sac milieu, making bacterial growth difficult [28]. Orbicularis oculi contraction and levator palpebrae superioris inhibition protects the cornea from dryness by shutting the eyelid [29–31]. When any of the above components of this highly intricate and interconnected innate eye protection mechanism are interrupted, alone or in combination, whether by disease processes or natural aging mechanisms, the risk of EK increases significantly.

McHugh et al. demonstrated in a study of ICU patients that poor or inadequate eyelid closure was associated with 70% incidence of EK when compared to 29% incidence among patients able to fully close their eyelids [4]. The use of pharmacologic-induced paralysis or heavy sedation may inhibit this important natural mechanism of eye protection [6]. Fluid imbalances, increased vascular permeability, and positive pressure ventilation (PPV) may increase conjunctival edema, leading to difficulties with eye closure [32]. It has also been noted that the use of high flow oxygen through face mask or nebulizer can lead to desiccation damage of the corneal epithelium [33]. To further complicate the issue, it has also been pointed out that ICU-related reductions in rapid eye movement sleep (REM) may elevate the probability of prolonged direct corneal exposure [12]. Additional clinical factors associated with ICU-related EK include, but are not limited to: low Glasgow Coma Scale (GCS < 8), ICU stay duration of >1 week, and the presence of significant metabolic imbalances [13]. **Table 1** provides a more complete appraisal of various associated risk factors.

Exposure keratopathy is primarily a clinical diagnosis. This makes the identification and management of EK especially challenging in the critically ill and neurologically impaired patients. Also, because of the generally more vulnerable status of ICU patients, any care-related omissions that lead to EK reflect potential opportunities for improvement in overall care quality. Therefore, the incidence of EK should be considered as either direct or indirect PS indicator [9, 34]. Optimally, surveillance and prevention efforts aimed at EK should be incorporated into evidence-based nursing practice, where awareness of the problem is coupled with appropriate education that helps facilitate around-the-clock attention to the specific PS issue [9, 35]. It is important to remember that even small amount of lagophthalmos – the inability to close the eyelids completely – has negative effects on the corneal epithelium, yet is easily overlooked [36]. In addition, ointments and eye drops used in an effort to protect the eyes can be harmful in the event of an infection, with the potential for microbial transmission when using the same medication tube or applicator for treating both eyes [36]. Further, if the clinical staff is unaware, left-in-place contact lenses can increase the risk of corneal drying and infection [37].

For non-sedated patients, corneal damage usually results in severe pain due to the presence of rich innervation of this highly sensitive anatomic area, with robust nerve networks located between the epithelial cells of the corneal surface [22]. Unfortunately, in the ICU, symptoms may not be readily communicated by the patient or promptly detected by healthcare personnel, leading to delayed detection and treatment of EK. As soon as EK is suspected, the physician should check for any trauma, contour malformation, and other causes of eyelid malposition. Further, the patient's past medical history should be reappraised for

Pre-existing risk factor	Description
Bell's Palsy	A seventh cranial nerve palsy localized to one side of the face affecting the eyelids.
Blepharitis	An inflammatory condition affecting the sweat glands of the eyelids, leading to swelling.
Blepharoplasty	A surgical procedure that corrects either congenital, functional, or esthetic issues related to the eyelids.
Chemosis	A swelling of the outer conjunctival membrane(s) covering the inner eyelid and the eye.
Coloboma	A congenital or traumatic full-thickness defect of the eyelid, often leading to incomplete eye closure.
Ectropion	An outward turn of the eyelid, either upper or lower. Causes include congenital (Treacher-Collins) conditions, acquired (trauma, Bell's palsy) etiologies, and aging-related processes.
Entropion	An inward turning of the eyelid, involving either upper or lower lid. Trauma, aging, and conjunctival scarring are all causes of entropion.
Facemask ventilation	A method of short-term administration of high-flow oxygen, which possibly directs airflow over the eyes.
Floppy eyelid syndrome	Chronic conjunctivitis of the upper eyelid, more prevalent in patients with a history of sleep apnea or snoring.
Graves' disease	An autoimmune disorder of the thyroid, known to cause severe proptosis. In some cases, it can lead to incomplete closure of the eyelids.
Iatrogenic	Pharmaceutical agents leading indirectly to EK. Propofol, benzodiazepines, and other sedative hypnotics in susceptible patients can contribute to the development of EK, particularly among ICU patients with eyelid dysfunction.
Lagophthalmos	Any state or condition of the eyelids leading to incomplete closure. Examples include severe proptosis or paralysis of the eyelids.
Myasthenia gravis	An autoimmune myopathy, which may lead to issues with full closure of the eyelids.
Parkinson's disease	Gradual deterioration of control over the eyelids leads to impaired blinking mechanism.
Sjogren's syndrome	An autoimmune disorder leading to dryness of the mucous membranes throughout the body. Sjogren's syndrome is often associated with other autoimmune disorders such as systemic lupus erythematosus and rheumatoid arthritis.
Symblepharon	Adhesion of the conjunctiva of the eyelid to the conjunctiva of the eyeball itself.

Legend: EK = Exposure keratopathy; ICU = Intensive care unit.

Table 1. Various factors associated with increased risk of exposure keratopathy.

any conditions that may result in malposition or proptosis. In order to aid the diagnosis, a fluorescein stain may be applied to the cornea to highlight any erosion under a black light lamp (**Figure 1**) [5]. Microepithelial defects are pin-point epithelial elevations or slightly depressed erosions in the cornea, whereas macroepithelial defects (e.g., corneal abrasions) are larger confluent zones of epithelial loss [36]. Additionally, a penlight using blue filter after the administration of fluorescein dye may help outline the epithelium and detect corneal abrasions or ulcers at bedside [16].

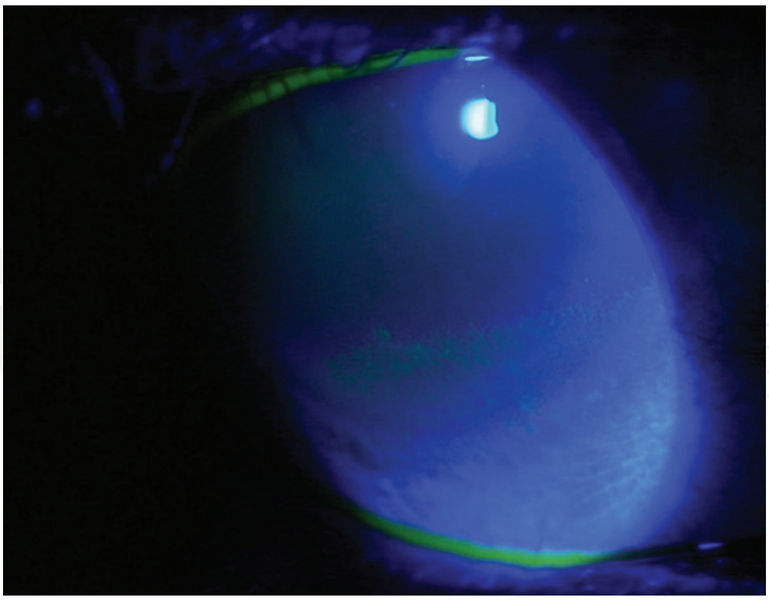

Figure 1. Punctate corneal lesions are seen upon administration of fluorescein dye under a black light lamp.

A comprehensive list of physical signs and clinical symptoms of EK can be found in **Tables 2** and **3**. It is important to note that these signs are not specific to EK and therefore the examining healthcare provider must be well-versed in other disorders which may cause similar signs and/or symptoms. A comprehensive differential diagnosis listing can be found in **Table 4**. Once the patient is alert, EK often resolves spontaneously; however, it may also lead to corneal scarring and vision loss, especially if sufficiently advanced [16, 36, 38]. Consequently, any evidence of EK, especially an opacity or haziness of the cornea, should prompt consultation

Symptoms	Description
Pain	Particularly common in the morning when the patient first awakens, especially if the underlying problem is an issue with the eyelid. It should be noted that in select patient populations presenting to ophthalmology clinics, there might be a lack of pain sensation in the eye due to other underlying or co-morbid condition(s).
Corneal irritation	May present as redness of the eye. Some cases may feature a yellow-green film over the cornea.
Foreign body sensation	Associated with irritation of nerve receptors supplying the cornea. Patients may report the feeling of an "eyelash caught" in the affected eye.
Excess tear production	Epiphora is another commonly reported symptom in EK patients experiencing significant corneal irritation. The discharge from the affected eye is often mucopurulent, particularly if the patient has acquired a superinfection during their hospital stay.
Photophobia	Corneal erosions, such as those seen in EK, can commonly allow too much light to enter the eye, causing the patient to complain of "sunlight hurting their eyes."
Blurred vision	Corneal abrasions may also contribute to an acute decrease in visual acuity, which may become permanent if the patient's condition is allowed to progress to corneal scarring. Prompt treatment is critical to healing and to regaining lost vision, thus avoiding significant long-term morbidity.

Table 2. Symptoms of exposure keratopathy.

Signs	Description
Punctate corneal erosion	A result of the cornea of the affected eye experiencing unusual dryness, culminating in epithelial cell loss. This sign can be visualized via fluorescein stain. In EK, these erosions may appear small or coalesce into a single, much larger lesion.
Chemosis	A swelling of the conjunctiva, leading to edema of the eyelids. This sign, along with lagophthalmos, is highly correlated to the development of EK in ICU patients.
Decreased tear meniscus	A normal eye should have a thin tear layer along the margins of the eyelid. Absence of this indicates dry eyes.
Corneal filaments	An appearance of "mucoepithelioid" areas of adherence involving the cornea, related to corneal epithelial dysfunction in the setting of dry eyes due to an increased mucus-to-tear ratio. Lubricating drops may help remove them, or these can be removed during a slit lamp exam.
Corneal ulceration	An open wound that appears on the cornea due to severely dried eyes. This condition carries a serious risk of corneal scarring and thus must be treated promptly.

Legend: EK = Exposure keratopathy; ICU = Intensive care unit.

Table 3. Clinical signs of exposure keratopathy.

Condition	Description
Exposure keratopathy	A result of severe eye dryness over a significant period of time. Signs include pain, redness, blurred vision, and a mucopurulent discharge from the affected eye. Must be treated immediately in order to avoid vision loss.
Corneal abrasion	An injury to the eye resulting in breach of the corneal epithelial layer, but does not progress. Commonly present as a foreign body sensation in the affected eye. Typically heals without intervention, though antibiotics may be given in select cases involving contact lenses.
Vernal keratoconjunctivitis	A dry eye condition that arises in dry climates with a seasonal periodicity. It features the appearance of giant papillae in the upper tarsals. Treatment includes artificial tears and removal of allergens.
Corneal foreign body (FB)	Irritation of the eye resulting from presence of a FB, commonly an eyelash or material related to the patient's occupational exposure (e.g., dust, metal or wood fragments). Treatment includes removal of the FB under slit lamp exam.
Toxic irritation	Redness of the eye associated with the presence of a toxic irritant. Appears as a bilateral infiltrate, usually associated with contact lenses. A similar form of this condition appears within a day after laser assisted *in situ* keratomileusis (LASIK) surgery, and resolves without intervention.
Hypersensitivity reaction	Redness of the eyes and mucopurulent discharge usually due to seasonal allergies/hay fever. Can be treated with cool compresses and antihistamines as needed.
Ulcerative keratitis	Thinning of the cornea related to autoimmune disease, such as systemic lupus erythematosus or rheumatoid arthritis. These patients are vulnerable to such complications as superinfection and perforation. Management tends to be medical, although surgery is indicated for patients with impending or current perforation.
Mooren's ulcer	An idiopathic corneal thinning that may be unilateral or bilateral. It may lead to corneal ulceration. This condition carries important associated risks, including glaucoma, perforation, and blindness, but is a diagnosis of exclusion.
Epithelial keratopathy	Punctate erosive lesions of the cornea that do not progress to EK, given that there are no other predisposing factors.
Band keratopathy	Corneal scarring that leads to a subepithelial opaque calcified plaque over the lens of the eye. These mostly appear in the elderly and have a particular horizontal deposition pattern, along with a chalky appearance. Treatment is typically conservative and requires only observation. However, in severe cases surgery is ~95% effective.

*In all of these cases, cultures yielded from corneal scraping must be negative in order to rule out infectious causes.

Table 4. Differential diagnosis of keratopathies.

with an ophthalmologist [38, 39]. Such expert evaluation is critical to confirming the diagnosis of EK and the initiation of appropriate therapy, as exemplified in this chapter's clinical vignettes.

5. Epidemiology of exposure keratopathy

Although the reported incidence of EK varies, some studies have estimated the rates to be as high as 57% in mechanically ventilated ICU patients who do not receive proper prophylactic eye care [3]. These clinical studies have also shown that up to 75% of patients with EK will show signs of lagophthalmos and chemosis before developing the condition [16]. Following proper precautions has been shown to reduce the risk of developing EK in ICU patients by more than 40% [16].

6. Treatment

Successful treatment of EK begins with a thorough assessment of the patient's eyes, including the corneas and a complete slit-lamp and fundoscopic exam (**Figure 2**). This is essential to ruling out several other conditions, as listed previously in **Table 4**. The conjunctival fornices should also be swabbed and the resulting sample sent out for bacterial culture testing, as microbial superinfection is a major complication that can be associated with this condition.

Effective management of EK necessitates the restoration of proper lubrication of the eye to prevent further damage. Lenart et al. studied 50 patients who each had one eye that

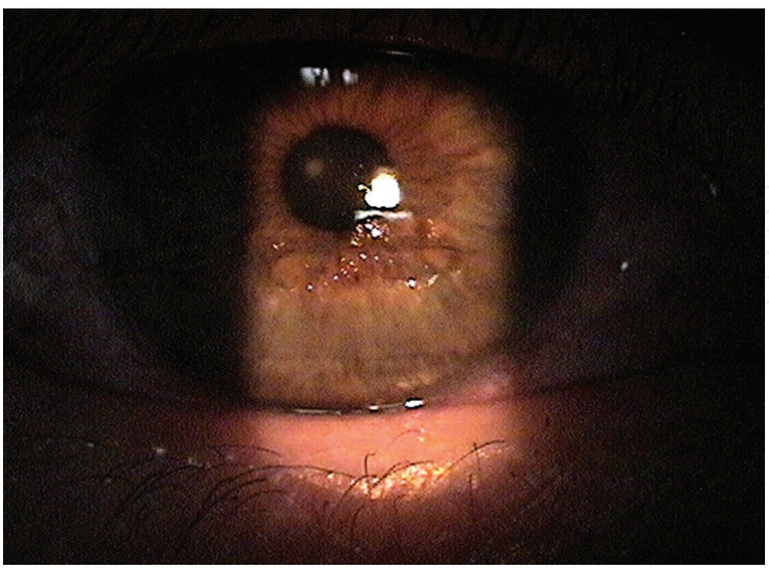

Figure 2. Appearance of punctate corneal lesions characteristic of exposure keratopathy, illuminated via slit lamp exam.

received artificial tear ointment every 4 h, while the other eye was passively closed by nurses when it was found open. The authors found that there were nine abrasions in the passively closed eyes, compared with only two abrasions in the ointment-treated eyes [40]. Ezra et al. [41] compared "eye toilet" using two alternative treatments – Geliperm versus Lacrilube. Twenty-four patients comprised the "eye toilet" group, 13 of which (53%) acquired EK. The group receiving Geliperm was found to have EK in 90% (9 out of 10) cases, while only 2 out of 13 (15%) patients in the Lacrilube group (15%) developed EK. The authors concluded that Lacrilube was more effective at preventing EK than "eye toilet" or Geliperm [41]. Nonetheless, a 2003 survey found that despite the above findings, 75% of British ICUs were using Geliperm [42]. If artificial tears are unavailable, the use of punctal plugs in the lacrimal ducts may be used [3]. Further lubrication with ointment up to four times daily is also recommended [3]. Strict adherence to these steps allows for the eye to re-establish its homeostatic moisture levels and promote healing of the erosions.

The maintenance of "moisture chambers" may protect the cornea when the eye remains open. In a study of 60 critically ill patients with a limited or absent blink reflex, half of participants were assigned to receive "lubricating eye drops" every 2 h while the remaining participants' eyes were covered with polyethylene film to create a "moisture chamber." Eight of thirty "lubricating eye drop" patients demonstrated corneal staining with fluorescein, which signified EK, contrasted with only 1 of 30 moisture chamber patients [43]. Koroloff et al. [44] studied 110 ICU patients with reduced or absent blink reflex. Groups either received hypromellose drops and Lacrilube every 2 h or had polyethylene covers on the eyes to create a "moisture chamber." Eyes in both patient groups were cleaned every 2 h with saline. The study showed that none of the patients developed corneal ulceration in the polyethylene group while four patients experienced ulceration in the hypromellose group [44]. In another report, 146 patients were assigned to receive treatment with either a "moisture chamber" created with swimming goggles and gauze soaked in sterile water (closed chamber), or ocular lubricants with securing tape over the eyes (open chamber). Thirty-nine (32%) eyes of the open chamber group versus ten (8%) of closed chamber eyes acquired EK [39]. Lastly, a meta-analysis comparing "moisture chambers" versus ocular lubricants was conducted, including three randomized trials that cumulatively enrolled nearly 300 patients [16]. Rates of EK were significantly lower when "moisture chambers" were used to protect the eye (7.1%) when compared to lubricating ointment use (21.2%). Despite some heterogeneity between component studies, cumulative results of this meta-analysis (Odds Ratio, 0.208 with 95% Confidence Interval 0.090–0.479) strongly support the use of "moisture chambers" [16].

Finally, if all of the previously outlined management steps fail and the patient's EK continues to progress, there are surgical options available that have been proven effective. Tarsorrhaphy may be performed, but it may significantly interfere with serial examinations of the affected eye(s) [39]. Consultation with Ophthalmology is strongly recommended. Additional procedures are described in **Table 5**.

Surgical technique	Description
Partial tarsorrhaphy	A surgical procedure in which the eyelid of the affected eye is sewn shut, either laterally or medially. Complete tarsorrhaphy is also used when functional vision is not immediately necessary
Eyelid reconstruction	A surgical procedure used to reconstitute the integrity of the eyelid itself, usually in cases of ectropion/entropion or coloboma.
Gold or platinum weight implant	A gravity-assisted method of eyelid closure accomplished by the implantation of a weight on the upper eyelid. This procedure is most useful against palsy-related causes of eyelid defects.
Orbital decompression	A gravity-assisted method of eyelid closure accomplished by the implantation of a weight on the upper eyelid. This procedure is most useful against palsy-related causes of eyelid defects.
Conjunctival flap	Grafting of the conjunctiva in order to provide pain relief and assist with healing of the cornea.
Sutured amniotic membrane graft	A procedure used for severe refractory EK when other procedures have failed to resolve the problem. This procedure has been shown to have some benefit and assist healing.

Table 5. Surgical procedures to reduce exposure keratopathy.

7. Complications of exposure keratopathy

If the eye is allowed to become too dry, small deficiencies in corneal epithelium may develop and lead to superficial keratopathy. This can be detected on slit lamp examination. As superficial keratopathy worsens, the corneal epithelium becomes more permeable [45, 46]. Of note, superficial keratopathy has been found in as many as 60% of intubated and sedated ICU patients [3, 28]. Moving further along the continuum of acuity, eyelid swelling, conjunctival swelling with hyperemia, and eyelid crusting or discharge are the primary signs of infection in an ICU patient [3, 37, 47, 48]. Slit lamp examination typically shows evidence of the presence of bacterial corneal ulcer while penlight examination reveals ulcerated corneal epithelium with a gray or white infiltrate [16, 49, 50].

One of the most feared complications associated with EK is microbial (or infectious) keratitis, which may lead to perforation, scleritis, endophthalmitis, and even blindness [46, 51–53]. In severe cases of corneal infections refractory to maximal medical therapy, treatment of microbial keratitis may require corneal transplantation [52, 54–56]. Of note, survival rates of grafts performed for this indication tend to be significantly lower than comparable rates for other conditions [56].

Bacterial superinfection is another serious complication that can occur in an ICU patient with EK. Thus, it is prudent to be aware of the clinical characteristics associated with the most common agents of superinfection (**Table 6**) [3, 53, 57, 58]. When left untreated, these infections can progress to complications including perforation, scleritis, endophthalmitis, and loss of vision [16, 57–59]. In terms of other factors associated with the risk of corneal infection, it has been postulated that exposed corneas may also be susceptible to aerosol droplets spread via tracheal suctioning [60–62]. If nurses hover at the head of the bed, direct inoculation may occur if the suction catheter is withdrawn directly over the patient's eye [62].

Bacteria	Associated sign
Streptococcus spp.	Purulent or crystalline infiltrate that can follow either a fulminant or indolent course. Indolent course is associated with chronic steroid use.
Staphylococcus spp.	Well-defined stromal infiltrate that can progress to an abscess.
Pseudomonas spp.	Rapidly progressive necrotic infiltrate, usually associated with the use of contact lenses.
Moraxella spp.	Indolent inferior corneal infiltrates that occur in the setting of immune deficiency.

Table 6. Clinical characteristics of superinfections seen in the setting of exposure keratopathy, including associated signs, symptoms, and clinical course.

Anecdotally, because right-handed nurses generally remove the suction catheter over the left eye, it has reported that higher bacterial contamination may be seen in the left eye than the right eye [60]. Finally, in cases involving severe infections leading to visual loss, corneal transplantation may be required [60–63].

A further major complication of EK is corneal ulceration (**Figure 3**), which represents a progression of this pathological state [3, 64]. The ulceration is often peripheral and displays corneal thinning that presents upon slit lamp examination [3, 65, 66]. If this occurs, the treating physician should be careful with the administration of steroids, which may exacerbate the ulceration [45, 53, 67, 68]. If the patient's ulceration progresses to the point that a perforation is imminent, then corneal transplantation surgery or amniotic membrane graft may be indicated [45, 69]. Prevention of ulceration is therefore critically important in patients with EK; for prophylaxis, the use of bandage contact lenses and concurrent broad-spectrum antibiotics can help decrease the incidence of this morbidity [49, 53]. Treating providers must remember that the above procedures can be a source of

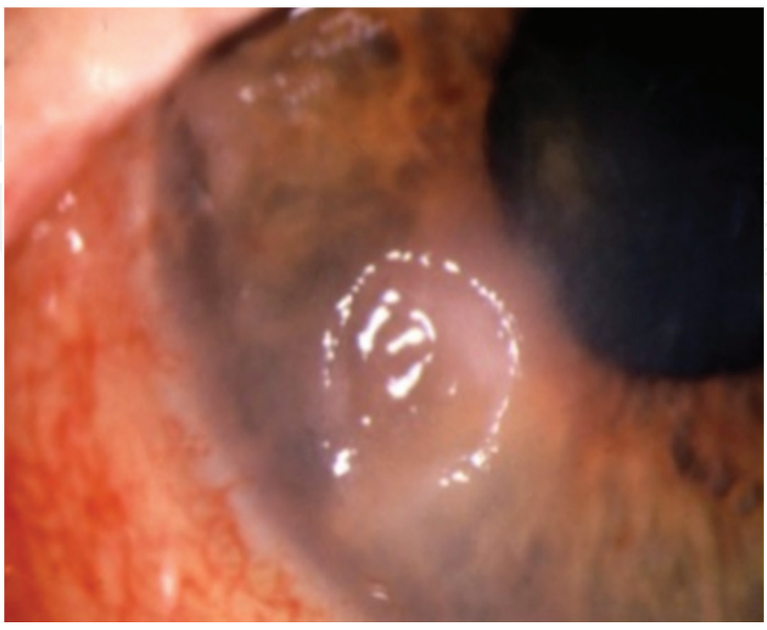

Figure 3. Corneal ulceration becomes apparent under examination as a saucer-like thinning of the epithelium.

significant stress to the patient, thus highlighting the importance of limiting the overall risk of EK through good clinical practices and the developing and closely following protocols aimed at prevention. This chapter's clinical vignettes touch upon both the short- and long-term sequelae of EK, with emphasis on prevention, early identification, and prompt treatment. The overall complexity of care, especially in high acuity environments such as the ICU where "competing priorities" are the norm, can contribute to a variety of errors of omission (EOO) – a topic discussed in greater detail in the subsequent sections of the chapter.

8. Exposure keratopathy: preventive strategies and patient safety considerations

Although maintenance of a favorable ocular environment may not be critical to saving the patient's life, attention to this important aspect of patient care is both an indirect measure of quality of care provided and an essential component of preventing significant morbidity following extended ICU stays. Despite this, one ICU audit discovered that only about one in four patients received appropriate eye assessment, and only about 55% had the provision of eye care properly documented [51]. What is more, even experienced nurses have difficulty following eye-care guidelines [52], likely due to the above-mentioned large number of competing priorities. Despite these barriers, progress is being made and numerous prophylactic/preventive measures, screening techniques, procedures, and guidelines have been designed, implemented, and reported to help reduce the incidence of EK.

In one study, McHugh et al. [4] set out to determine if specialization enhanced ocular disease detection. Two junior ICU physicians, twice a week, examined lid position and ocular surfaces of all patients continuously sedated for >24 h. An ophthalmologist performed similar examinations with a slit lamp. Cumulatively, 48 examinations were performed on 18 patients. ICU doctors had 77.8% sensitivity and 96.7% specificity in detecting EK, with all "missed" cases having erosions involving <5% of the corneal surface. The authors concluded that using regular eye checks, ICU staff should be able to adequately diagnose EK and facilitate ocular therapy [4].

Suresh et al. [70] created a protocol where patients with closed lids received no treatment, those with exposed conjunctiva received lubricants, and patients with exposed corneas or ventilated patients in the prone position received both lid taping and lubricants. Of 34 patients examined, 11 were excluded (4 because of protocol non-compliance and 7 because of errors in initial assessment of lid position). The 23 patients who were followed demonstrated an 8.7% rate of ocular surface disease, compared to 42% prevalence in historical controls [70].

In another study, authors concentrated on reducing the risk of ocular *Pseudomonas aeruginosa* infection [29]. Their clinical guideline stipulated that unconscious patients should receive eye care every 2 h. Any swelling, conjunctival hyperemia, corneal clouding, and epithelial loss were noted and recorded. Exposed corneas were lubricated every 2 h and patients at risk for corneal exposure had their eyes taped shut. Tracheal suctioning was performed at bedside, with the patient's eyes covered and daily swabs of the eyes obtained. If eye swab cultures grew *P. aeruginosa*, topical gentamicin was started and ophthalmology consultation was requested.

Once implemented in the ICU, conjunctival *Pseudomonas* isolation rates decreased from 0.8 to 0.05% [29]. This particular intervention very nicely highlights the powerful effects of simple, easy-to-follow protocols on both PS and care quality.

A review by Alansari described a nurse driven protocol for eye care in the ICU. For patients with risk factors as previously described, clinical assessments for lid closure were recommended every 8 h [71]. In addition, the following recommendations were made:

- In the presence of incomplete closure, application of Duratears (lubricating eye ointment) every 4 h or polyethylene coverage, depending on the severity of the lack of closure, was recommended.
- Lid cleanliness should be inspected every 4 h, with basic eye hygiene implemented if any issues are identified.
- Eye dryness should be inspected every 4 h and Duratears added as needed.
- Assessment for ocular surface disease should be made every 4 h, and the supervising care provider should be notified with any findings of concern.

It is this and other, similar protocols that are most likely to result in significant and sustainable reduction in the incidence of EK. In the modern PS and care quality paradigm, continuous self-improvement and evidence-based, protocol-driven care are the cornerstones of ensuring optimal clinical results for our patients.

Providers should remember that patient management does not end in the acute care setting. Therefore, during the post-acute phase of treatment, the follow-up examination frequency will depend on each patient's individual case of EK, the length of stay in the ICU or hospital, and the details of any surgical procedures performed. If the lesion(s) on the patient's eye are mild and vision is not threatened, then follow-up eye examinations on a weekly or even monthly basis may be appropriate [3]. If EK has progressed to corneal ulceration, then eye examinations should occur daily, or every 2 days at most [3]. However, even if daily examinations are not needed, proper precautions (i.e., the provision of eyedrops and appropriate ocular ointment) to prevent EK or worsening thereof must be observed.

9. Errors of omission: a focused discussion

Errors of omission (EOOs) can be defined as actions that lead to adverse events that happen because of the healthcare provider (or team) not having done something, whether intentional or not [72]. On the other hand, errors of commission involve performing an action, but not carrying it out correctly. For example, prescribing the incorrect dosage of a medication or carrying out a procedure that is unintentionally different from the one originally intended, may be considered in the latter category of errors.

Figure 4 demonstrates the key differentiation between errors of omission versus those of commission. **Figure 5** shows the taxonomy of errors of omission and commission. In terms of frequency,

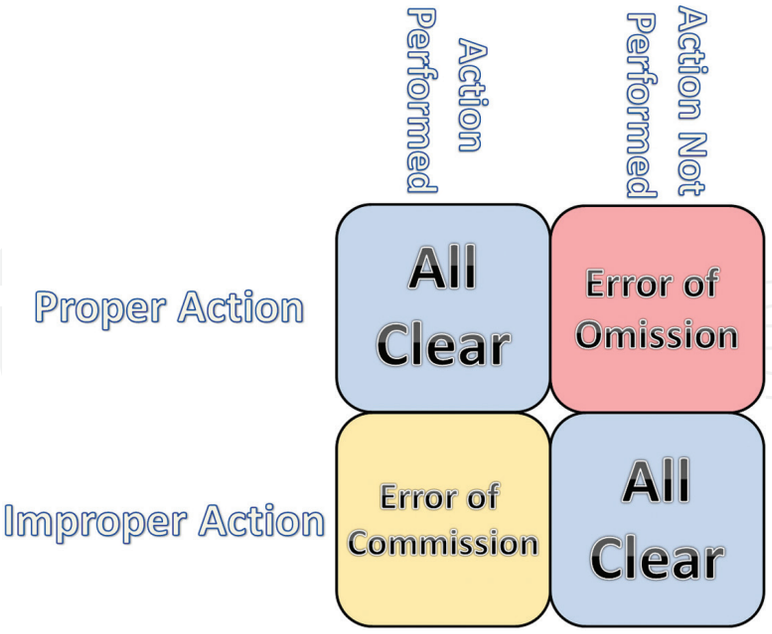

Figure 4. Schematic representation of the interplay between the appropriateness of a specific action, its performance, and the presence and type of error. Errors of omission occur when proper action is not carried out.

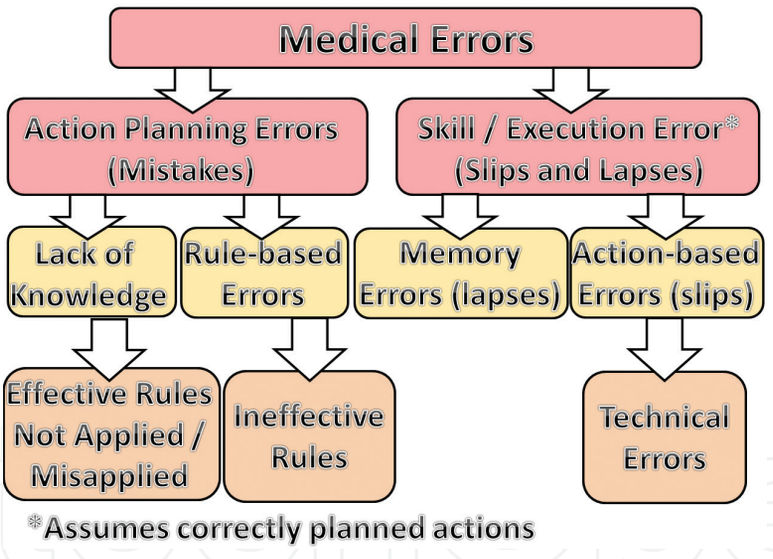

Figure 5. Schematic taxonomy of different types of medical errors (Adapted and compiled from: http://cecourses.org/preventive-care/preventing-medication-errors/ and http://www.thebestmedicalcare.com/patient-safety/when-why-and-how-things.html).

errors of omission tend to be more common than errors of commission (see the "Introductory Chapter" of *Vignettes in Patient Safety, Volume 1*) [72–74]. Factors that contribute to EOOs include deficient or lack of education [75], faulty communication [75, 76], insufficient labor resources [75, 77], and the absence of necessary tools (e.g., checklists, technological support, etc.) [75].

It has been shown that EMR implementation can lead to significant improvements in compliance with protocolized care paradigms and the reduction of EOOs [72]. In addition, omissions associated with documentation of medical history and clinical events may lead to error

propagation and escalation [72], further highlighting the importance of process standardization and protocol compliance. Finally, the importance of education and skills maintenance must be strongly emphasized. In addition to ensuring adequate medical knowledge, practitioners should remain acutely aware regarding the ever-present possibility of an error as well as ways to prevent adverse events [78].

10. Summation and conclusions

Exposure keratopathy is a preventable complication that most often is due to errors of clinical omission. In addition to discussing the etiology, risk factors, management, and complications of EK, this chapter also discusses an important category of medical errors – those of omission. Among strategies to reduce errors of omission, a multi-pronged approach involving clinical education, evidence-based protocols, and hardwired quality improvement seems to be most optimal. In conclusion, with proper education of providers and establishment of protocols, the incidence of EK, and thus the incidence of any associated sequelae, should decrease.

Author details

Benjamin Bird[1]*, Stephen Dingley[2], Stanislaw P. Stawicki[2,3] and Thomas R. Wojda[3,4]

*Address all correspondence to: tuf80248@temple.edu

1 Lewis Katz School of Medicine at Temple University, Philadelphia, PA, USA

2 Department of Surgery, St. Luke's University Health Network, Bethlehem, PA, USA

3 Department of Research & Innovation, St. Luke's University Health Network, Bethlehem, PA, USA

4 Department of Family Medicine, Warren Hospital Campus - St. Luke's University Health Network, Phillipsburg, NJ, USA

References

[1] Pronovost PJ et al. Interventions to reduce mortality among patients treated in intensive care units. Journal of Critical Care. 2004;**19**(3):158-164

[2] Duwe B, Fuchs BD, Hansen-Flaschen J. Failure mode and effects analysis application to critical care medicine. Critical Care Clinics. 2005;**21**(1):21-30

[3] Jammal H et al. Exposure keratopathy in sedated and ventilated patients. Journal of Critical Care. 2012;**27**(6):537-541

[4] McHugh J et al. Screening for ocular surface disease in the intensive care unit. Eye. 2008; **22**(12):1465-1468

[5] Ezra DG, Healy M, Coombes A. Assessment of corneal epitheliopathy in the critically ill. Intensive Care Medicine. 2005;**31**(2):313-313

[6] Mercieca F et al. Ocular surface disease in intensive care unit patients. Eye. 1999;**13**(2): 231-236

[7] Foster A, Sommer A. Childhood blindness from corneal ulceration in Africa: Causes, prevention, and treatment. Bulletin of the World Health Organization. 1986;**64**(5):619

[8] Kam KR, Hayes M, Joshi N. Ocular care and complications in the critically ill. Trends in Anaesthesia and Critical Care. 2011;**1**(5):257-262

[9] Leadingham C. Maintaining the Vision in the Intensive Care Unit. Dayton, OH: Wright State University; 2014. https://corescholar.libraries.wright.edu/nursing_dnp/1/

[10] Ramírez F et al. The neglected eye: Ophthalmological issues in the intensive care unit. Critical Care and Shock. 2008;**11**(3):72-82

[11] Nichols K. Blepharitis and dry eye: A common, yet complicated combination: The diseases often co-present, which may confound treatment priorities. Review of Optometry. 2010;**147**(8):39-45

[12] Onakpoya OH, Adeoye AO, Akinpelu OV. Cost-related antibiotic dosage omissions – Challenge for orbital cellulitis management in resource poor communities. Orbit. 2009; **28**(2-3):147-152

[13] Havener WH. Emergency management of acute eye problems. Journal of the American College of Emergency Physicians. 1972;**1**(1):3356-3338

[14] Malhotra R, Sheikh I, Dheansa B. The management of eyelid burns. Survey of Ophthalmology. 2009;**54**(3):356-371

[15] Nakra T, Shorr N. Ophthalmic plastic surgery. In: Risk Prevention in Ophthalmology. New York: Springer; 2008. pp. 187-200

[16] Rosenberg JB, Eisen LA. Eye care in the intensive care unit: Narrative review and meta-analysis. Critical Care Medicine. 2008;**36**(12):3151-3155

[17] Thygeson P. Nonspecific defense mechanisms of the eye. International Ophthalmology Clinics. 1985;**25**(2):1-11

[18] Tseng SC. A practical treatment algorithm for managing ocular surface and tear disorders. Cornea. 2011;**30**:S8-S14

[19] Albert DM, Jakobiec FA. Principles and Practice of Ophthalmology. Vol. 1. Philadelphia: WB Saunders Company; 2000

[20] Trattler B. Cornea Handbook. Thorofare: Slack Incorporated; 2010

[21] Gandhi S, Jain S. The anatomy and physiology of cornea. In: Keratoprostheses and Artificial Corneas. Berlin/Heidelberg: Springer; 2015. pp. 19-25

[22] Gipson IK. The ocular surface: The challenge to enable and protect vision: The Friedenwald lecture. Investigative Ophthalmology & Visual Science. 2007;**48**(10):4391-4398

[23] McClellan K. Mucosal defense of the outer eye. Survey of Ophthalmology. 1997;**42**(3):233-246

[24] Evans DJ, Fleiszig SM. Why does the healthy cornea resist *Pseudomonas aeruginosa* infection? American Journal of Ophthalmology. 2013;**155**(6):961-970. e2

[25] Sutphin J et al. Basic and clinical science course. Section. 2006;**8**:2005-2006

[26] Bandyopadhyay P, Coffey MJ, Shawer M. Chapter 10: Development of ophthalmic formulations. In: Pharmaceutical Dosage Forms-Parenteral Medications, Formulation and Packaging. Vol. 1. New York: Informa Healthcare; 2010. p. 254

[27] Henriksson JT. Validating the Mouse Model for Ocular Surface Research and Assessing Morphological Changes in the Dry Eye Palpebral Conjunctiva. Houston, TX: University of Houston; 2011

[28] Dua HS. Bacterial keratitis in the critically ill and comatose patient. The Lancet. 1998; **351**(9100):387-388

[29] Parkin B et al. Bacterial keratitis in the critically ill. British Journal of Ophthalmology. 1997;**81**(12):1060-1063

[30] Van der Werf F, Smit AE. The world according to blink: Blinking and aging. In: Age-Related Changes of the Human Eye. Springer; 2008. pp. 319-341

[31] Skalicky SE. Protective mechanisms of the eye and the eyelids. In: Ocular and Visual Physiology. Singapore: Springer; 2016. pp. 3-12

[32] Imanaka H et al. Ocular surface disorders in the critically III. Anesthesia & Analgesia. 1997;**85**(2):343-346

[33] Maurice DM. The von Sallmann lecture 1996: An ophthalmological explanation of REM sleep. Experimental Eye Research. 1998;**66**(2):139-145

[34] Isaac T, Jha AK. Are patient safety indicators related to widely used measures of hospital quality? Journal of General Internal Medicine. 2008;**23**(9):1373-1378

[35] Creed F, Hargreaves J. Oxford Handbook of Critical Care Nursing. Oxford: Oxford University Press; 2016

[36] Hernandez EV, Mannis MJ. Superficial keratopathy in intensive care unit patients. American Journal of Ophthalmology. 1997;**124**(2):212-216

[37] Poggio EC et al. The incidence of ulcerative keratitis among users of daily-wear and extended-wear soft contact lenses. New England Journal of Medicine. 1989;**321**(12):779-783

[38] Hashish A, Awara AM. One-stage reconstruction technique for large congenital eyelid coloboma. Orbit. 2011;**30**(4):177-179

[39] Sivasankar S et al. Eye care in ICU. Indian Journal of Critical Care Medicine. 2006;**10**(1):11

[40] Lenart SB, Garrity JA. Eye care for patients receiving neuromuscular blocking agents or propofol during mechanical ventilation. American Journal of Critical Care. 2000;**9**(3):188

[41] Ezra D et al. Preventing exposure keratopathy in the critically ill: A prospective study comparing eye care regimes. British Journal of Ophthalmology. 2005;**89**(8):1068-1069

[42] King D, Healy M. Prevention of eye disease in intensive care – A telephone survey. In: Intensive Care Medicine. New York: Springer-Verlag; 2003

[43] Cortese D, Capp L, McKinley S. Moisture chamber versus lubrication for the prevention of corneal epithelial breakdown. American Journal of Critical Care. 1995;**4**(6):425-428

[44] Koroloff N et al. A randomised controlled study of the efficacy of hypromellose and Lacri-lube combination versus polyethylene/cling wrap to prevent corneal epithelial breakdown in the semiconscious intensive care patient. Intensive Care Medicine. 2004;**30**(6):1122-1126

[45] Pfister RR, Murphy GE. Corneal ulceration and perforation associated with Sjögren's syndrome. Archives of Ophthalmology. 1980;**98**(1):89-94

[46] Hutchinson K, Apel A. Infectious keratitis in orthokeratology. Clinical & Experimental Ophthalmology. 2002;**30**(1):49-51

[47] Cervantes LJ, Mah FS. Clinical use of gatifloxacin ophthalmic solution for treatment of bacterial conjunctivitis. Clinical Ophthalmology (Auckland, NZ). 2011;**5**:495

[48] Rumelt S. Overview of common and less common ocular infections. In: Advances in Common Eye Infections. Rijeka: InTech; 2016

[49] Gangopadhyay N et al. Fluoroquinolone and fortified antibiotics for treating bacterial corneal ulcers. British Journal of Ophthalmology. 2000;**84**(4):378-384

[50] Gwin NT. Overview of Ocular Disorders. Thorofare: SLACK Incorporated; 1999

[51] Dawson D. Development of a new eye care guideline for critically ill patients. Intensive & Critical Care Nursing. 2005;**21**(2):119-122

[52] Cunningham C, Gould D. Eyecare for the sedated patient undergoing mechanical ventilation: The use of evidence-based care. International Journal of Nursing Studies. 1998;**35**(1-2):32-40

[53] Gokhale NS. Medical management approach to infectious keratitis. Indian Journal of Ophthalmology. 2008;**56**(3):215

[54] Chen W-L et al. Therapeutic penetrating keratoplasty for microbial keratitis in Taiwan from 1987 to 2001. American Journal of Ophthalmology. 2004;**137**(4):736-743

[55] Sharma N et al. Therapeutic keratoplasty for microbial keratitis. Current Opinion in Ophthalmology. 2010;**21**(4):293-300

[56] Sit M et al. Corneal graft outcome study. Cornea. 2001;**20**(2):129-133

[57] Roberts A. Eye Infections. 2010. Available online at: https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/384497/eye_infections_20.pdf

[58] Sharma N, Vajpayee RB. Corneal Ulcers Diagnosis and Management. New Delhi: Jaypee Brothers/Medical Publishers Pvt. Limited; 2008

[59] Jackson TL. E-Book-Moorfields Manual of Ophthalmology. Philadelphia, PA: Elsevier Health Sciences; 2007

[60] Hilton E et al. Nosocomial bacterial eye infections in intensive-care units. The Lancet. 1983;**321**(8337):1318-1320

[61] Hutton WL, Sexton RR. Atypical pseudomonas corneal ulcers in semicomatose patients. American Journal of Ophthalmology. 1972;**73**(1):37-39

[62] Ommeslag D, Colardyn F, De Laey J-J. Eye infections caused by respiratory pathogens in mechanically ventilated patients. Critical Care Medicine. 1987;**15**(1):80-81

[63] Kirwan J et al. Microbial keratitis in intensive care. BMJ: British Medical Journal. 1997; **314**(7078):433

[64] Cosar CB et al. Tarsorrhaphy: Clinical experience from a cornea practice. Cornea. 2001; **20**(8):787-791

[65] Garg P, Rao GN. Corneal ulcer: Diagnosis and management. Community Eye Health. 1999;**12**(30):21

[66] Wilson DM, John GR, Callen JP. Peripheral ulcerative keratitis – An extracutaneous neutrophilic disorder: Report of a patient with rheumatoid arthritis, pustular vasculitis, pyoderma gangrenosum, and Sweet's syndrome with an excellent response to cyclosporine therapy. Journal of the American Academy of Dermatology. 1999;**40**(2):331-334

[67] Davis A et al. Topical steroid use in the treatment of ocular alkali burns. British Journal of Ophthalmology. 1997;**81**(9):732-734

[68] Guidera AC, Luchs JI, Udell IJ. Keratitis, ulceration, and perforation associated with topical nonsteroidal anti-inflammatory drugs. Ophthalmology. 2001;**108**(5):936-944

[69] Forrester JV, Kuffova L. Corneal Transplantation: An Immunological Guide to the Clinical Problem (with CD-ROM). Singapore: World Scientific; 2004

[70] Suresh P et al. Eye care for the critically ill. Intensive Care Medicine. 2000;**26**(2):162-166

[71] Alansari MA, Hijazi MH, Maghrabi KA. Making a difference in eye care of the critically ill patients. Journal of Intensive Care Medicine. 2015;**30**(6):311-317

[72] Overhage JM et al. A randomized trial of "corollary orders" to prevent errors of omission. Journal of the American Medical Informatics Association. 1997;**4**(5):364-375

[73] Weingart NS et al. Epidemiology of medical error. British Medical Journal. 2000; **320**(7237):774-777

[74] Tam VC et al. Frequency, type and clinical importance of medication history errors at admission to hospital: A systematic review. Canadian Medical Association Journal. 2005;**173**(5):510-515

[75] Kalisch BJ, Landstrom G, Williams RA. Missed nursing care: Errors of omission. Nursing Outlook. 2009;**57**(1):3-9

[76] Sutcliffe KM, Lewton E, Rosenthal MM. Communication failures: An insidious contributor to medical mishaps. Academic Medicine. 2004;**79**(2):186-194

[77] Kalisch BJ, Tschannen D, Lee KH. Do staffing levels predict missed nursing care? International Journal for Quality in Health Care. 2011;**23**(3):302-308

[78] Stawicki S et al. Fundamentals of Patient Safety in Medicine and Surgery. New Delhi: Wolters Kluwer Health (India) Pvt Ltd; 2014

IntechOpen