Periocular burns: a literature review of classification, management protocols and outcomes of treatment

BY ANNA GKOUNTELIA

The author provides a review of the current literature regarding the principles of classification, management protocols of acute ocular and periocular burns and the role of the burn and oculoplastic surgeon involved in their care.

More than two-thirds of facial burns involve the eye or periocular area and 7.5-27% of all patients treated for burns have ocular involvement. Eighty-four percent of these are due to chemicals and 16% due to thermal injury. Reflex blinking of eyelids, Bell's phenomenon, in response to heat and smoke, and protective movements of the arms and head, usually protect the cornea and eyelid margins. Frequent ocular injuries seen as a result of facial burns include lid burns, corneal burns, foreign bodies, abrasions, perforations and contracture leading to ectropion. Because of the life-threatening nature of severe burn injuries to the face and the associated massive swelling of eyelids, ocular injuries may not be noticed early and treatment may be delayed. Appropriate early intervention can have a significant effect on the final outcome for the burn patient. Permanent visual impairment is rare if prompt management is done. Superficial lid burns usually heal spontaneously and can be managed conservatively with ophthalmic antibiotic ointments, artificial tears. However, in deeper burns, early surgical intervention in the form of eschar debridement or release of contracted lids and resurfacing defects with split skin grafts can prevent secondary corneal damage. This paper is a review of the current literature regarding the principles of classification, management protocols of acute ocular and periocular burns and the role of the burn and oculoplastic surgeon involved in their care.

Background

Eyelids are important structures and play a role in protecting the globe from trauma, brightness, in maintaining the integrity of tear films and moving the tears towards the lacrimal drainage system and contribute to the aesthetic appearance of the face. Periocular burns involving eyelids and adjacent structures have been found to have increased recently. A comprehensive classification of periocular burns would help in stratifying these injuries, as well as study outcomes.

Etiopathogenesis

Burns to the eyelids may be caused by thermal, electrical, chemical or ionising radiation sources. The most serious injuries are due to chemical burns by strong acid or bases. Chemicals can be classified as either acidic or alkaline agents (Figure 1); a new classification of ocular surface burns. Many of these are used in homes, industries and agriculture, causing burns when they come into contact with the eye, resulting in a significant threat to vision, especially those that are alkaline. The extent and severity of the injury is influenced by various factors, such as the nature, quantity and concentration of the solution, the contact duration, solution penetrability and pH. While most burns occur from direct contact with the outer eye surfaces, chemicals can also reach the ocular tissue through systemic absorption via the skin, lungs or digestive tract. The intact cornea can resist a wide range of pH without injury, but a pH <4 or >10 results in an increase in permeability, which can cause severe ocular complications. The purpose of managing these burns is to eliminate or limit the causative agent from penetrating the ocular structures by irrigation; and, promoting ocular surface healing through medical and surgical intervention.

Acid		
Substance	**Chemical Composition**	**Found In**
Sulfuric acid	H_2SO_4	Car batteries
Sulfurous acid	H_2SO_3	Bleach and refigerant
Hydrofluoric acid	HF	Glass polishing and mineral refining
Acetic acid	CH_3COOH	Vinegar, glacial acetic acid
Hydrochloric acid	HC1	Swimming pools

Alkali		
Substance	**Chemical Composition**	**Found In**
Amonia	NH_2	Cleaning agents, fertilisers, refigerants
Potassium Hydroxide	KOH	Caustic potash
Lye	NaOH	Drain cleaners, airbags
Magnesium Hydroxide	$Mg(OH)_2$	Firework sparklers, flares
Lime	$Ca(OH)_2$	Plaster, mortar, cement, white wash

Figure 1: Common causes of alkali and acid injuries.

Classification

Eyelid burns are classified depending on how deep and severe they penetrate the skin's surface (Table 1). Two major classification schemes for corneal burns are the Roper-Hall (modified Hughes) classification and the Dua classification (Figure 2). The Roper-Hall classification is based on the degree of corneal involvement and limbal ischaemia. The Dua classification is based on an estimate of limbal involvement (in clock hours) and the percentage of conjunctival involvement. In a randomised controlled trial of acute burns, the Dua classification was found to be superior to the Roper-Hall in predicting outcome in severe burns. However, both classification schemes are commonly employed in daily practice. A reliable system to address eyelid injuries, the system for periocular trauma classification for periocular injuries (SPOT) (Figure 3), has taken into account the periocular soft tissue injuries, i.e. upper eyelid, lower eyelid, medial and lateral canthus injuries, based on observed clinico-anatomical patterns of eyelid injuries. This classification scheme provides guidance for ophthalmic and facial reconstructive surgeons to provide optimal outcomes in eyelid injuries. Based on the classification scheme and review of existing literature, an algorithm is presented to facilitate repair and reconstruction (Figure 4).

Table 1: Eyelid burns classification.

Epidermal burns (first-degree burns)

This corresponds to the zone of hyperaemia in Jackson's model. Severe sunburn is the most common first-degree burn. By definition, this affects only the epidermis, and blistering is not common. Pain is due to local vasodilator prostaglandins, and healing is usually complete within a week.

Partial-thickness burns (second-degree burns)

Partial-thickness burns involve the dermis and epidermis. This corresponds to the zone of stasis in Jackson's model. It is commonly divided into superficial and deep dermal injury.

1. *Superficial partial-thickness burns*
   Injury to the epidermis and superficial papillary dermis results in thin-walled, fluid-filled blisters with a moist red base. The exposure of superficial nerves makes these injuries painful. A burn of this depth heals within two weeks by regeneration of epidermis keratinocytes within sweat glands and hair follicles with minimal scarring.
2. *Deep partial-thickness burns*
   These burns have a pale white or mottled base beneath the blisters. The healing takes three or more weeks, and is accompanied by scarring and contraction. These injuries are of concern in the eyelid region, often necessitating early surgery for contraction and eyelid retraction.

Full-thickness burns (third-degree burns)

These destroy epidermis, dermis and all regenerative elements and correspond to the zone of coagulation in Jackson's burn wound model. The skin is dry, leathery and, as a result of heat coagulation of dermal blood vessels, the affected tissue is avascular and white. Such burns are typically painless due to loss of sensation in the involved area. Healing only occurs from the edges and is associated with significant contraction. Early excision of affected tissue and skin grafting is almost always required to resurface the burnt area and prevent secondary corneal complications.

Deep (fourth-degree burns)

These are full-thickness burns with destruction of the underlying muscle, bone and vital structures. Such burns require extensive and complex management and often result in severe contracture and prolonged disability.

Based on these definitions and the likelihood of surgery, eyelid burns may be classified as minimal, moderate or major. Minimal burns describe superficial partial-thickness burns that usually heal without the need for surgery. Moderate burns refer to deeper partial-thickness burns that are delayed in healing but may not require surgery. Major eyelid burns are deep-partial or full-thickness and invariably require early surgery with skin grafts.

Roper Hall Classification for Ocular Surface Burns

Grade	Prognosis	Cornea	Conjunctiva/Limbus
I	Good	Cornea epithelial damage	No limbal ischaemia
II	Good	Cornea haze, iris details visible	<1/3-1/2 limbal ischaemia
III	Guarded	Total epithelial loss, stromal haze, iris details obscure	1/3-1/2 limbal ischaemia
IV	Poor	Cornea opaque, iris and pupil obscured	>1/2 limbal ischaemia

Dua Classification for Ocular Surface Burns

Grade	Prognosis	Clinical findings	Conjuctiva involvement	Analogue Scale*
I	Very good	0 clock hours of limbal involvement	0%	0/0%
II	Good	<3 clock hours of limbal involvement	<30%	0.1-3/1-29.9%
III	Good	3-6 clock hours of limbal involvement	30-50%	3.1-6/31-50%
IV	Good to guarded	Between 6-9 clock hours of limbal involvement	50-75%	6.1-9/31-50%
V	Guarded to poor	Between 9-12 clock hours of limbal involvement	75-100%	9.1-11.9/75.1-99%
VI	Very poor	Total limbus (12 clock hours) invloved	Total conjunctiva (100%) involved	12/100%

Figure 2: The two major classification schemes for corneal burns: the Roper-Hall (modified Hughes) classification and the Dua classification.

SPOT classification for periocular injuries

Type	Zone	Anatomical region	Injury
I	I	Upper eyelid	A=Superficial B=Partial thickness 1=without tissue loss 2=with tissue loss C=Full thickness 1=<1/4 tissue loss 2=<1/4-1/2 tissue loss 3=>1/2 (near total / total loss of eyelid)
II	II	Lower eyelid	A=Superficial B=Partial thickness 1=without tissue loss 2=with tissue loss C=Full thickness 1=<1/4 tissue loss 2=<1/4-1/2 (Subtotal) 3=>1/2 (near total / total loss of eyelid)
III	III	Medial canthus	A=Superficial B=Partial thickness (periosteum intact) 1=without tissue loss 2=with tissue loss C=Full thickness (periosteum breached) 1=without tissue loss 2=with tissue loss (Presence of injuries to the lacrimal canalicular system is represented by an 'L')
IV	IV	Lateral canthus	A=Superficial B=Partial thickness (periosteum intact) 1=without tissue loss 2=with tissue loss C=Full thickness (periosteum breached) 1=without tissue loss 2=with tissue loss
V	Any combination of above involving more than 1 zone		

Figure 3: SPOT classification.

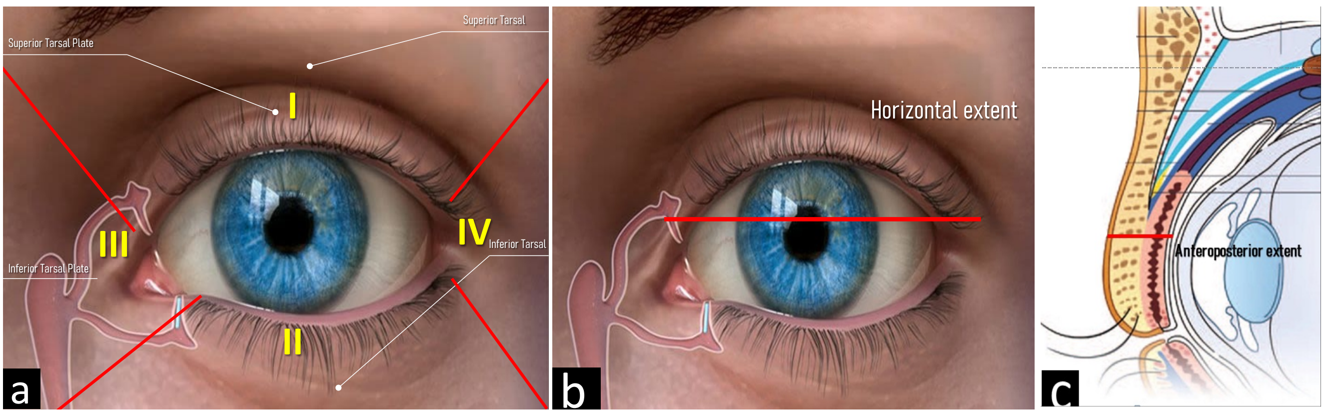

Figure 4: Zone topography according to the SPOT classification.

Table 2: The initial treatment of chemical injury based on the Roper-Hall grade of injury.

Grade I

- Topical antibiotic ointment (erythromycin ointment or similar) four times a day
- Prednisolone acetate 1% four times a day
- Preservative free artificial tears as needed
- If there is pain, consider a short acting cycloplegic like cyclopentolate three times a day

Grade II

- Topical antibiotic drop like fluoroquinolone four times daily
- Prednisolone acetate 1% hourly while awake for the first 7-10 days. Consider tapering the steroid if the epithelium has not healed by day 10-14. If an epithelial defect persists after day 10, consider progestational steroids (1% medroxyprogesterone four times daily)
- Long acting cycloplegic like atropine
- Oral vitamin C, 2 grams four times a day
- Doxycycline, 100mg twice a day (avoid in children)
- Sodium ascorbate drops (10%) hourly while awake
- Preservative free artificial tears as needed
- Debridement of necrotic epithelium and application of tissue adhesive as needed

Grade III

- As for Grade II
- Consider amniotic membrane transplant / Prokera placement. This should ideally be performed in the first week of injury. Experienced surgeons have emphasised placement of the amniotic membrane to cover the palpebral conjunctiva by suturing to the lids in the operating room, not just covering the cornea and bulbar conjunctiva

Grade IV

- As for Grade II/III
- Early surgery is usually necessary. For significant necrosis, a Tenonplasty can help re-establish limbal vascularity. An amniotic membrane transplant is often necessary due to the severity of the ocular surface damage

Phase	Day	Recovery
Initial	0	Clinical findings relate to the severity of injury and can be graded according to degree of limbal, corneal and conjuctival involvement.
Acute	0-7	Epithelial regrowth begins to occur if there is a sufficient amount undamaged limbal stem cells. Treatment should be directed at encouraging growth while quelling inflamation
Early Repair	7-21	Cornea / conjunctival epithelium and keratocytes prolifirate during this stage. Mild injuries show complete re-epithelialisation while more severe injuries can have persistent epithelial defects. Activity of collagenases peaks by day 14-21 while collagen synthesis continues. Treatment should attempt to maximise collagen synthesis while minimising collagenase activity
Late Repair	After day 21	In mild injuries, where the limbal stem cell population is intact, repair is completed. In grade II injuries, where there is focal stem cell loss, there may be a focal conjunctivalisation of the cornea, ultimately leading to either repopulation by conjunctival epitheliem or stromal ulceration and permanent scarring. In cases of severe limbal damage, despite optimal management, the eye often cannot be salvaged

Figure 5: Stages of ocular recovery following chemical injury.

Recommended treatment

While there is variability in treatment strategies of chemical burns, most authors recommended a graded approach depending on the severity of injury. Mild burns (Roper-Hall grade I) respond well to medical treatments and lubrication, while more severe burns necessitate more intensive medical therapies and surgery. Table 2 shows a paradigm for the initial treatment of chemical injury based on the Roper-Hall grade of injury.

Conclusion

The goal in the management of periorbital burns is preservation of vision, prevention of future complications and restoration of an acceptable aesthetic outcome. Total visual loss is thankfully rare, but early ophthalmology intervention is vital given the evidence of corneal damage as a brief therapeutic window exists.

The final outcome of an eye burn depends on the cause of the burn, the depth of the injury, which structure(s) of the eye were involved, whether other parts of the body were burned, and the development of complications. With eye burns of any type, close follow-up care is important during the first several weeks to prevent scarring, exposure keratitis and other complications of ocular and periocular burns. Primary prevention and patient counselling on proper eye protection, first aid measures, and initial management is essential because most of the ophthalmic complications can be avoided with these measures.

The medical team working in the burn casualty must prioritise first aid measures in all facial burn patients, like irrigation of the eyes, trimming of the charred eyelashes, use of artificial tears, padding of the eyes and topical antibiotics. A thorough ophthalmological examination in the first 24 hours of burn injury must be made mandatory. Consequent eye examinations must be made to look for any adverse consequences and any intervention made as early as possible to prevent adverse outcomes in these patients.

Classification systems are necessary in order to provide a framework in which to scientifically study the aetiology, pathogenesis and treatment of diseases in an orderly fashion. Based on classification schemes, the ophthalmic, oculoplastic and facial reconstructive surgeons will be guided to provide optimal outcomes in eyelid injuries in order to facilitate repair and reconstruction.

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Reading list available on the website www.eyenewsuk.com or on request to diana@pinpoint-scotland.com

Reading list

1. Fish R, Davidson RS. Management of ocular thermal and chemical injuries, including amniotic membrane therapy. *Curr Opin Ophthalmol* 2010;**21(4)**:317-21.
2, Barouch F, Colby KA. Evaluation and initial management of patients with ocular and adnexal trauma. In: Miller JW, Azar DT, Blodi B (eds). *Albert and Jakobiec's Principles and Practice of Ophthalmology*, 3rd ed. Philadelphia: WB Saunders Elsevier; 2008: 5071-92.
3. McCulley J. Chemical Injuries. In: Smolin G, Thoft RA (eds). *The Cornea: Scientific Foundation and Clinical Practice*. Boston: Little, Brown and Co; 1987.
4. Gupta N, Kalaivani M, Tandon R. Comparison of prognostic value of Roper Hall and Dua classification systems in acute ocular burns. *Br J Ophthalmol* 2011;**95(2)**:194-8.
5. Lin MP, Ekşioğlu U, Mudumbai RC, et al. Glaucoma in patients with ocular chemical burns. *Am J Ophthalmol* 2012;**154(3)**:481-5.
6. Hughes W. Alkali burns of the eye. I. Review of the literature and summary of present knowledge. *Arch Ophthalmol* 1946;**35**:423.
7. Roper-Hall MJ. Thermal and chemical burns. *Trans Ophthalmol Soc UK* 1965;**85**:631-53.
8. Dua HS, King AJ, Joseph A. A new classification of ocular surface burns. *Br J ophthalmol* 2001;**85(11)**:1379-83.
9. Herr RD, White GL Jr, Bernhisel K, et al. Clinical comparison of ocular irrigation fluids following chemical injury. *Am J Emerg Med* 1991;**9(3)**:228-31.
10. Rihawi S, Frentz M, Schrage NF. Emergency treatment of eye burns: which rinsing solution should we choose? Graefes Arch *Clin Exp Ophthalmol* 2006;**244(7)**:845-54.
11. Dohlman CH, Cade F, Pfister R. Chemical burns to the eye: paradigm shifts in treatment. *Cornea* 2011;**30(6)**:613-4.
12. Ikeda N, Hayasaka S, Hayasaka Y, Watanabe K. Alkali burns of the eye: effect of immediate copious irrigation with tap water on their severity. *Ophthalmologica* 200;**220(4)**:225-8.
13. Donshik PC, Berman MB, Dohlman CH, et al. Effect of topical corticosteroids on ulceration in alkali-burned corneas. *Arch Ophthalmol* 1978;**96(11)**:2117-20.
14. Pfister RR, Haddox JL, Yuille-Barr D. The combined effect of citrate/ascorbate treatment in alkali-injured rabbit eyes. *Cornea* 1991;**10(2)**:100-4.
15. Gabardi S, Munz K, Ulbricht C. A review of dietary supplement-induced renal dysfunction. *CJASN* 2007;**2(4)**:757-65.
16. Ralph RA. Tetracyclines and the treatment of corneal stromal ulceration: a review. *Cornea* 2000;**19(3)**:274-7.
17. Smith VA, Cook SD. Doxycycline-a role in ocular surface repair. *Br J Ophthalmol* 2004;**88(5)**:619-25.
18. Matsuda H, Smelser GK. Epithelium and stroma in alkali-burned corneas. *Arch Ophthalmol* 1973;**89(5)**:396-401.
19. Haddox JL, Pfister RR, Slaughter SE. An excess of topical calcium and magnesium reverses the therapeutic effect of citrate on the development of corneal ulcers after alkali injury. *Cornea* 1996;**15(2)**:191-5.
20. Pfister RR, Haddox JL, Sommers CI. Effect of synthetic metalloproteinase inhibitor or citrate on neutrophil chemotaxis and the respiratory burst. *Invest Ophthalmol Vis Sci* 1997;**38(7)**:1340-9.
21. J Gross, Azizkhan RG, Biswas C, et al. Inhibition of tumor growth, vascularization, and collagenolysis in the rabbit cornea by medroxyprogesterone. *Proc Natl Acad Sci USA* 1981;**78(2)**:1176-80.
22. Panda A, Jain M, Vanathi M, et al. Topical autologous platelet-rich plasma eyedrops for acute corneal chemical injury. *Cornea* 2012;**31(9)**:989-93.
23. Kuckelkorn R, Schrage N, Reim M. Treatment of severe eye burns by tenonplasty. *Lancet* 1995;**345(8950)**:657-8.
24. Shafto CM. A simple method of inserting amniotic membrane grafts into the conjunctival sac. *Br J Ophthalmol* 1950;**34(7)**:445-6.
25. Baum J. Thygeson lecture. Amniotic membrane transplantation: why is it effective? *Cornea* 2002;**21(4)**:339-41.
26. Tseng SCG, Di Pascuale MA, Liu D T-S, et al. Intraoperative mitomycin C and amniotic membrane transplantation for fornix reconstruction in severe cicatricial ocular surface diseases. *Ophthalmology* 2005;**112(5)**:896-903.
27. Hopkinson A, McIntosh RS, Tighe PJ, et al. Amniotic membrane for ocular surface reconstruction: donor variations and the effect of handling on TGF-beta content. *Invest Ophthalmol Vis Sci* 2006;**47(10)**:4316-22.
28. Gicquel J-J, Dua HS, Brodie A, et al. Epidermal growth factor variations in amniotic membrane used for ex vivo tissue constructs. *Tissue Eng Part A* 2009;**15(8)**:1919-27.
29. Ueta M, Kweon MN, Sano Y, et al. Immunosuppressive properties of human amniotic membrane for mixed lymphocyte reaction. *Clin Exp Immunol* 2002;**129(3)**:464-70.
30. Hao Y, Ma DH, Hwang DG, et al. Identification of antiangiogenic and antiinflammatory proteins in human amniotic membrane. *Cornea* 2000;**19(3)**:348-52.
31. Li W, He H, Kawakita T, et al. Amniotic membrane induces apoptosis of interferon-gamma activated macrophages in vitro. *Exp Eye Res* 2006;**82(2)**:282-92.
32. Kheirkhah A, Johnson DA, Paranjpe DR, et al. Temporary sutureless amniotic membrane patch for acute alkaline burns. *Arch Ophthalmol* 2008;**126(8)**:1059-66.
33. Tandon R, Gupta N, Kalaivani M, et al., Amniotic membrane transplantation as an adjunct to medical therapy in acute ocular burns. *Br J Ophthalmol* 2011;**95(2)**:199-204.
34. Huang T, Wang Y, Zhang H, et al. Limbal from living-related donors to treat partial limbal deficiency secondary to ocular chemical burns. *Arch Ophthalmol* 2011;**129(10)**:1267-73.
35. Morgan S, Murray A. Limbal autotransplantation in the acute and chronic phases of severe chemical injuries. *Eye* 1996;**10(Pt 3)**:349-54.
36. Tsai RJ, Tseng SC. Human allograft limbal transplantation for corneal surface reconstruction. *Cornea* 1994;**13(5)**:389-400.
37. Samson CM, Nduaguba C, Baltatzis S, Foster CS. Limbal stem cell transplantation in chronic inflammatory eye disease. *Ophthalmology* 2002;**109(5)**:862-8.
38. Liang L, Sheha H, Tseng SC. Long-term outcomes of keratolimbal allograft for total limbal stem cell deficiency using combined immunosuppressive agents and correction of ocular surface deficits. *Arch Ophthalmol* 2009;**127(11)**:1428-34.
39. Ma DH, Kuo MT, Tsai YJ, et al. Transplantation of cultivated oral mucosal epithelial cells for severe corneal burn. *Eye* 2009;**23(6)**:1442-50.
40. Nakamura T, Inatomi T, Sotozono C, et al, Transplantation of cultivated autologous oral mucosal epithelial cells in patients with severe ocular surface disorders. *Brit J Ophthalmol* 2004;**88(10)**:1280-4.
41. Khan B, Dudenhoefer EJ, Dohlman CH. Keratoprosthesis: an update. *Curr Opin Ophthalmol* 2001;**12(4)**:282-7.
42. Hemmati HD, Colby KA. Treating acute chemical injuries of the cornea. *Eyenet* October 2012:43-5.
43. Paterson CA, Pfister RR. Intraocular pressure changes after alkali burns. *Arch Ophthalmol* 1974;**91(3)**:211-8.
44. Le Q, Chen Y, Wang X, et al. Vision-related quality of life in patients with ocular chemical burns. *Invest Ophthalmol Vis Sci* 2011;**52(12)**:8951-6.
45. Kheirkhah A, Ghaffari R, Kaghazkanani R, et al. A combined approach of amniotic membrane and oral mucosa transplantation for fornix reconstruction in severe symblepharon. *Cornea* 2013;**32(2)**:155-60.
46. Tuft SJ, Shortt AJ. Surgical rehabilitation following severe ocular burns. *Eye* 2009;**23(10)**:1966-71.
47. Achauer BM, Adair SR. Acute and reconstructive management of the burned eyelid. *Clin Plast Surg* 2000;**7**:87-95.
48. Barrow RE, Marc KM, David H. Early release of third-degree burns prevents eye injury. *Plast Reconstr Surg* 2000;**105(3)**:860-3.
49. Bouchard CS, Morno K, Jeffrey P, et al. Ocular complications of thermal injury: a 3-year retrospective. *J Trauma* 2001;**50(1)**:79-82.
50. Cole JK, Engrav LH, Heimbach DM, et al. Early excision and grafting of face and neck burns in patients over 20 years. *Plast Reconstr Surg* 2002;**109**:1266-73.
51 Malhotra R, Sheikh I, Dheansa B. The management of eyelid burns. *Surv Ophthalmol* 2009;**54(3)**:356-71.
52. 2017-18 Basic and Clinical Science Course: Orbit, eyelids, and lacrimal system. San Francisco, CA: American Academy of Ophthalmology; 2012.
53. Mohapatra DP, Thiruvoth FM, Chittoria RK, et al. Proposal of a new classification scheme for periocular injuries. *Indian J Plast Surg* 2017;**50(1)**:21-8.
54. Clare G, Suleman H, Bunce C, Dua H. Amniotic membrane transplantation for acute ocular burns. *Cochrane Database Syst Rev* 2012;**9**:CD009379.
55. Wagoner MD. Chemical injuries of the eye: current concepts in pathophysiology and therapy. *Surv Ophthalmol* 1997;**41(4)**:275-313.