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Ocular Burns, Etiology and Treatment

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Summary

This thesis is a narrative review of the topic of ocular burns. The thesis reviews the existing literature regarding the presentation, mechanisms, evaluation and treatment of ocular burns.

Ocular burns can be physical burns, either thermal or radiation burns, or chemical, caused by either alkali or acid. The different types of ocular burns present differently and operate in different mechanisms, which are detailed in this thesis. I chose, in adherence to the prevalence in the population and the interest of the medical community, to focus more in this thesis on chemical ocular burns.

Following the review of presentation and mechanism, the thesis presents the acceptable systems for evaluating and categorizing different ocular burns. This evaluation determines the course of treatment the patient should receive according to the medical protocol.

Treatment of ocular burns is done in phases, starting with emergency first aid treatment, followed by primary and secondary treatment by a physician and according to its severity and status of healing, medical and even surgical interventions.

The aim of this thesis is not only to review the current body of research regarding the topic of ocular burns, but also to emphasize the innovative and ongoing research.

Keywords

Ophthalmology; Ocular burns; Chemical ocular burns; Thermal ocular burns.

1. Introduction

Ocular burns are approximately 8-18% of reported causes of ocular trauma, and it is among the most frequent ones [1]. Ocular burns occur mostly due to workplace accidents, but also as a result of at-home accidents or during leisure activity [1,2]. Ocular burns are classified by the agent causing the burn, and can either be Physical or chemical [1].

Physical burns can be thermal, i.e., caused by a heat agent, or a radiation burn, caused by exposure to radiation, mainly ultra-violet radiation [1,2].

Chemical burns to the eye or ocular adnexa are a serious form of injury and a common form of ocular injury, especially in the workplace [3,4]. It has potential long-term consequences that may affect a person's vision and quality of life, and be associated with short- and long-term ocular pain, decreased visual acuity, ectropion, and extended risk for infection [3]. Chemical ocular burns can range in severity from very mild to severe, that are potentially blinding. They can be caused by either acids or alkalis [2].

Ocular chemical burns are the second most common workplace eye injury, with mostly male patients, aged 18 to 64, presenting with it [5]. In addition, children, especially three years old and younger are also at high risk for chemical burns, mainly alkali burns that are considered more severe and have more long-term effects [4].

In this thesis I would like to present the topic of ocular burns, focusing mainly on chemical burns, both alkaline and acidic, as this type is more common. As the literature indicates, many researchers and clinicians are looking for better, more effective ways of treating and managing this type of ocular burns. The aim of this thesis is to present traditional and innovative treatments alike.

1.1 Literature search strategy

In this thesis, I performed a narrative review. In order to do so I developed a literature search strategy, as follows:

1. Sources to search: databases such as Medscape, Medline, and Embase; Google scholar, while searching for the most recent studies (years 2012 and later); Medical textbooks; Other thesis and doctoral dissertations.

2. Keywords: Ocular burn; chemical burn; thermal burn; Ocular trauma; Surgical interventions AND ocular trauma; Evaluation AND Ocular burns.

2. Clinical presentation of the condition

2.1 Physical ocular burns

2.1.1 Thermal burns

Thermal burns can vary in severity, which then concludes the clinical presentation. Most thermal ocular burns include injuries to the face and eyelids, but in severe cases, there can also be a burn to the cornea, which may result in severe corneal scarring [2]. Usually, cell death is limited to the superficial epithelium, but thermal necrosis and penetration are possible [6].

2.1.2 Radiation burns

Main symptoms include moderate to severe ocular pain, foreign body sensation, red eye, tearing, photophobia, and blurred vision. Presentation will be dense, confluent punctate epithelial defects in an interpalpebral distribution highlighted with fluorescein staining. One might also observe conjunctival injection, mild to moderate eyelid edema, mild to no corneal edema, relatively miotic pupils that react sluggishly, and mild anterior chamber reaction. [7]

2.2 Chemical ocular burns

Chemical injuries to the eye vary in severity from mild irritation to complete destruction of the ocular surface and adnexa, which results in corneal opacification, loss of vision, and even loss of the eye [8]. The typical presentation of an eye after a chemical injury is a sudden onset of severe pain, reduced visual acuity, epiphora and blepharospasm [9,10].

In mild to moderate chemical ocular burns presentation will be corneal epithelial defects that range from scattered superficial punctate keratopathy (SPK) to focal epithelial loss, to sloughing of the entire corneal epithelium, and there will be no significant areas of perilimbal ischemia. In addition, one might observe focal areas of conjunctival epithelial defect, chemosis, hyperemia, or hemorrhages (one or more of these), mild eyelid edema, mild anterior chamber reaction and 1st and 2nd degree burns of the periocular skin with or without lash loss. In severe chemical burns, the presentation will include, additionally to all that was mentioned above, pronounced chemosis and

conjunctival blanching, corneal edema and opacification, a moderate to severe AC reaction, that may not be appreciated if the cornea is opaque, and might also include increased intraocular pressure (IOP), 2nd and 3rd degree burns of the surrounding skin, and local necrotic retinopathy as a result of direct penetration of alkali through the sclera [11].

3. Mechanisms and pathology

3.1 Physical ocular burns

3.1.1 Thermal burns

Thermal ocular burns result from accidents associated with flames, gasoline explosions, firework explosions, boiling water, molten metals, hot tar or steam [1,12]. Mostly, these types of burns are rare since the eye is largely protected from thermal exposure (by the facial structure or by the blinking reflex, for example) and the thermal agent is usually removed within seconds. The burn becomes more severe when the duration of contact with the heat agent is longer [1,12]. The extent of damage to the eye is also affected by the degree of the heat agent and the conductance of the tissue [1].

3.1.2 Radiation burns

Radiation burns occur usually immediately after the absorption of radiant energy when exposure exceeds 1 second. After the initial exposure, biological repair responses may play a significant role in determining the extent of the damage. While inflammatory, repair responses are intended to reduce the sequelae, which sometimes can result in scarring that could affect negatively on the biological function [13].

3.2 Chemical ocular burns

Chemical ocular burns can be caused by either acidic or alkaline agents [3]. Both types of chemical burns share some common elements. The pathophysiology of chemical ocular burns starts with the pH change of the ocular tissue [10]. The initial phase of the contact with the chemical agent is followed by a rush of inflammatory cells that produce detergent enzymes such as the matrix metalloproteins, which aggravate the destruction of the ocular structures. Then, in the scarring phase, there is a regrowth of healthy tissue surrounding the burn [1]. It is important to note that

while most ocular burns are caused by direct contact of the burning agent with the eye, chemical ocular burns may happen also due to a systemic absorption of the chemical agent through the skin, lungs or digestive system and it reaching the eyes via those systems [1].

The denaturation of proteins is one of the mechanisms of the body to protect and repair itself following any burn, but chemical burns have some specific effects that thermal and radiation burns do not have. The exposure to the chemical is usually longer and may continue even after the person is rushed to the emergency room. In addition, in a chemical burn the structure of biological proteins involves not only a specific amino acid sequence, but also a 3-dimensional structure that is dependent on weak forces, such as hydrogen bonding or van der Waal's forces. These 3-dimensional structures are key elements for the biological activity on the proteins, and are easily disrupted by external factors. Application of heat or chemicals, especially pH disturbances, can cause the structures to fall apart. In thermal injuries, there is a rapid coagulation of protein due to irreversible cross-linking reactions, whereas in chemical burns the protein destruction is continued by other mechanisms, mainly hydrolysis. These mechanisms may continue so long as traces of the offending agent are present, especially in deeper layers. In addition, chemical agents may act in a systemic fashion if their components are circulated throughout the victim, with potential toxicity [14].

In the past, the pH change was considered the key element to understanding the extent of the damage caused to the ocular tissue in a chemical burn, but current research shows that other factors, such as the temperature of the chemical, also determine this damage [10]. Other factors that determine the extent and severity of the damage caused by a chemical ocular burn are the consistency of the agent – liquids tend to be easier to irrigate while solids may become entrapped in the conjunctival papillae and steadily resolve in the tear meniscus, thus leading to prolonged exposure and irritation of the ocular tissues, as well as the concentration of the agent and the length of time of the exposure, which both can increase the extent of the damage. In addition, a forceful impact may also increase the damage [15].

3.2.1 Acid burns

Acidic agents tend to cause less severe injuries than alkaline agents, because they tend to precipitate proteins in the eye, causing the coagulation of protein, which may serve as a barrier for further invasion of intraocular structures and penetration to deeper layers, thus limiting the damage

[3,9]. Nonetheless, acids can still cause severe damage to the eye, depending on the variables mentioned above, like temperature, consistency, concentration etc. Acidic agents cause coagulative necrosis [15]. Despite this barrier, that limits the damage to superficial layers, ocular lesions that are caused by acids with a pH below 2.5 are deep and necrotizing, and can affect the conjunctival and limbal vessels [1].

For example, hydrofluoric, hydrochloric, chromic, acetic and sulfuric acids are very concentrated and have a pH of 1.0-3.5. This makes them cause damage to the superficial tissue structure very fast, but they are neutralized quickly due to the protons' bind with the tissue protein and its precipitation and denaturation of it. Another example is battery acid, which is a very common cause of acid ocular burns due to explosions of car batteries, an event that causes damage due to the forceful nature of impact [1].

3.2.2 Alkali burns

Alkaline agents tend to cause more severe injuries than acidic agents, because basic structures are lipophilic and penetrate the eye faster than acidic chemicals [9]. Alkaline substances have the ability to cause cellular membrane lysis, penetrate the cornea and anterior chamber and soften tissues [3]. As a result, they may cause damage to the trabecular meshwork, ciliary body, and the lens [9]. Alkali cause liquefactive necroses. Alkaline agents contain hydroxyl moieties that saponify the fatty acids enclosed in the superficial cell membranes. As mentioned above, once the function of the cell membranes is interrupted, cell death begins and the damaging agent reaches the underlying connective tissue, where the matrix proteoglycans are readily hydrolyzed leaving collagen fibrils especially susceptible to enzymatic degradation. These enzymes are produced by damaged or regenerating epithelial cells and immune cells which would have invaded the tissue immediately after exposure [15].

4. Diagnosis

4.1 Evaluation

An evaluation of the eye after an ocular burn should include:

- Visual inspection of the surrounding skin, lids, lashes and the exterior structure of the eye [16].

- Fluorescein staining and slit-lamp examination [16]: A slit-lamp is a microscope with a bright light, and this exam allows detailed examination of the anterior segment of the eye – the sclera, the cornea and the lens. It is also known as biomicroscopy [17,18]. Fluorescein is a nontoxic, water-soluble, synthetic organic hydroxyxanthene dye. It creates a stain that is easily detected by a cobalt blue light, and is used for the evaluation of the tear film and for the detection of disruptions of intercellular junctions and stains punctate and macroulcerative epithelial defects [8].
- Measurement of intraocular pressure
- Visual acuity test

It should be noted that laboratory testing is not mandatory for isolated ocular burns. Maxillofacial or orbital CT may be useful, particularly with burns caused by an explosion or by projectile trauma to the face where significant injury to the globe or surrounding structures is suspected [16].

4.1.1 Grading of severity of the burn

4.1.1.1 Roper-Hall system

By this system, chemical burns are graded by severity, based on corneal clarity and severity of limbal ischemia. The grade of the burn indicates both prognosis and treatment plan [2]:

- Grade 1 burn is characterized by a clear cornea (only epithelial damage) and no limbal ischemia. The prognosis is excellent.
- Grade 2 is characterized by a hazy cornea but a visible iris detail and less than one third of the limbus being ischemic. The prognosis is good.
- Grade 3 is characterized by total loss of corneal epithelium, stromal haze obscuring iris detail and between one third and one half limbal ischemia. The prognosis is guarded.
- Grade 4 is characterized by an opaque cornea and more than one half of the limbal ischemia. The prognosis is poor.

Other features that should be taken into account in the initial assessment are the extent of corneal and conjunctival epithelial loss, iris changes, the status of the lens and the IOP [2].

4.1.1.2 Dua et al classification system

This classification system is newer than the Roper-Hall systems, and takes into consideration other characteristics of the burn [12]:

- the extent of limbal involvement in clock hours – the limbus is divided into 12 clock hours to determine how many parts are involved in the burn. It expands the term limbal ischemia so it also includes areas of the limbus where a complete or full thickness loss of limbal epithelium has occurred without significant ischemia, which is also a possible situation.
- the percentage of conjunctival involvement – only involvement of bulbar conjunctiva, up to and including the conjunctival fornices, is considered.
- Analogue scale – the limbal involvement in clock hours of affected limbus, divided by the percentage of conjunctival involvement.

This classification offers not four but six grades of burns [12]:

- Grade 1 is characterized by 0 clock hours of limbal involvement, 0 percent of conjunctival involvement and 0/0% on the analogue scale. The prognosis is very good.
- Grade 2 is characterized by up to 3 clock hours of limbal involvement, up to 30 percent of conjunctival involvement and 0.1-3/1-29.9% on the analogue scale. The prognosis is good.
- Grade 3 is characterized by 3 to 6 clock hours of limbal involvement, 30 to 50 percent of conjunctival involvement and 3.1-6/31-50% on the analogue scale. The prognosis is good.
- Grade 4 is characterized by 6 to 9 clock hours of limbal involvement, 50 to 75 percent of conjunctival involvement and 6.1-9/51-75% on the analogue scale. The prognosis is good to guarded.
- Grade 5 is characterized by 9 to 12 clock hours of limbal involvement, 75 to 100 percent of conjunctival involvement and 9.1-11/75.1-99.9% on the analogue scale. The prognosis is guarded to poor.
- Grade 6 is characterized by total limbus involvement (12 clock hours), total conjunctival involvement (100 percent) and 12/100% on the analogue scale. The prognosis is very poor.

4.1.1.3 Comparison between Roper-Hall and Dua et al grading system for ocular burns

A study was conducted in order to examine the predictive outcome of ocular burns using the two different prognostic classification systems. First, the study noted the main differences between the two [19]:

- Roper-Hall system offers 4 grades while Dua et al system offers 6 grades.
- Grades 2 and three in both systems are exactly the same.
- While the Roper-Hall system assigns the same grade, grade 4, to all burns with more than 50% limbal ischemia, the Dua et al system offers three grades of burns with more than 50% limbal involvement.
- Another key difference is the inclusion of conjunctival involvement, which is missing from the Roper-Hall system, in the Dua et al system.

As far as the clinical outcomes, that focused on burns that are classified as grade 4 in the Roper-Hall system and grades 4,5 & 6 in the Dua et al system, the study has found the following [19]:

- There was no difference in the time taken and the epithelial defect between grades 4, 5 & 6 in the Dua system, which were all included in grade 4 in the Roper-Hall system.
- There was a significant difference in the extent of corneal vascularization between grades 4, 5 & 6 in the Dua system, that were all included in grade 4 in the Roper-Hall system.
- There was a significant difference in the extent of corneal vascularization between grades 4, 5 & 6 in the Dua system, that were all included in grade 4 in the Roper-Hall system.

This leads to the conclusion that the Dua et al system provides significant additional information regarding the burns that are in grade 4 of the Roper-Hall system, that can help determine the prognosis of a burn a patient presents with.

4.2 Differential diagnosis

- Corneal abrasion
- Corneal ocular and ulcerative keratitis in emergency medicine.
- Fluoride toxicity
- Globe rupture
- Ultraviolet keratitis.

5. Management

5.1 Prevention

Studies have shown that most ocular burns can be prevented by taking appropriate precautions and following safety guidelines in the workplace and home environment, as follows [3]:

- Proper storage of dangerous agents in the home, to prevent child injuries. Household chemicals like bleach should be stored in high, locked cabinets that are inaccessible to children.
- Counseling and raising awareness among parents to the dangers of chemical burns in children in the household environment, to encourage them to take said precautions.
- Education for first responders such as police forces and paramedics regarding appropriate immediate treatment of chemical ocular burns, as will be described below. This, in order to minimize the damage caused by such injuries should they occur.
- Setting workplace regulations regarding handling and storing such chemicals.
- Enforcing guidelines mandating employers to purchase proper safety equipment for workers.

5.2 First Aid

5.2.1 Irrigation

First aid for ocular chemical burns is copious irrigation to dilute the chemical [20]. Irrigation should be with isotonic solution and should continue until the eye's pH is between 7.0-7.4. It is important to note that no substance should be used to neutralize the chemical exposure since the exothermic reaction may cause secondary thermal injury [16].

Traditionally, irrigation is done using tap water, 0.9% normal saline, isotonic buffered phosphate solution or Ringer's lactate. This is called passive decontamination of the chemical burn. Researchers suggest that active decontamination, which means adding an amphoteric, polyvalent and chelating components, will be more effective [20,21]. This is explained by the neutralizing effect of this solution without producing heat [20]. The studies on that matter were conducted using Previn® (Diphoterine®) solution. These studies have shown that the active decontamination resulted in significantly lower severity of corrosive chemical burns [20,21]. It is important to stress

that especially in case of an alkali burn, irrigation should be as immediate as possible, and should not be delayed in order to retrieve any special solution [15].

5.2.2 Clinical examination

The initial examination will take place only after irrigation, as will be described below in the management section. The examination will include [9]:

- Complete eye examination.
- Making sure no foreign bodies are embedded in any part of the ocular structures.
- Evaluation of the condition, as previously explained.

5.3 Medical Treatment

5.3.1 Traditional medical treatment

Traditional medical treatment includes [22]:

- Topical steroids – reduce inflammation and neutrophil infiltration and address anterior uveitis. One must note that they also have an impairing effect on stromal healing [2].
- Topical antibiotics – for prophylaxis of bacterial infection [2].
- Mydriatic cycloplegics – for improving comfort [2] if there is significant photophobia, pain or AC inflammation [11].
- Antiglaucoma therapy
- Citrate – for the inhibition of neutrophil activity and the reduction of intensity of the inflammatory response [2].
- Ascorbate – improves wound healing by promoting synthesis of mature collagen by corneal fibroblasts [2].

5.3.2 Umbilical cord serum drops

Autologous serum eye drops have been shown to be effective in treatment of many kinds of topical surface disorders such as severe dry eye and neuropathic keratitis. Umbilical cord serum eye drops have been shown to be effective in similar conditions. Both of these types of drops are efficient due to the presence of various growth factors like epidermal growth factor (EGF), acidic and basic fibroblast growth factor (FGF), platelet-derived growth factor, hepatocyte growth factor, vitamin

A, transforming growth factor (TGF)-β, substance P, insulin-like growth factor (IGF)-1, nerve growth factor (NGF), fibronectin, and serum antiproteases such as α2-macroglobulin. There is a higher concentration of some of these growth factors in the umbilical cord serum drops, and for these reasons researchers decided to explore the possibility of these drops in treating and healing the ocular surface in chemical ocular burns. They have found that umbilical cord serum therapy is more effective than autologous serum eye drops or artificial tears in ocular surface restoration after acute chemical injuries, and suggest it will be introduced to the medical treatment protocol [22].

5.4 Surgical treatment

5.4.1 Early surgical treatment

In order to promote revascularization of the limbus, one might need to use one of the following surgical procedures [2]:

- Advancement of Tenon capsule with suturing to the limbus is aimed at re-establishing limbal vascularity to help to prevent the development of corneal ulceration.
- Limbal stem cell transplantation from the patient's other eye (autograft) or from a donor (allograft) is aimed at restoring normal corneal epithelium [2].
- Amniotic membrane grafting to promote epithelialization and suppression of fibrosis [2]. Amniotic membrane transplantation is a surgical procedure that has been used in the ophthalmological field for many years, and recently became even more popular due to the development of new tissue preservation methods [23]. It has been studied and found that indeed amniotic membrane transplantation promotes normal conjunctival epithelization while suppressing fibrosis formation [24]. Amniotic membrane transplantation has been proven to significantly improve the rate of epithelial healing as opposed to standard medical therapy alone [25]. However, there were no significant long-term benefits found to this intervention, especially when the burn is severe [23,25,26,27].
- Gluing or keratoplasty may be needed for actual or impending perforation.

5.4.2 Late surgical treatment

Later, one might need to use one of the following surgical procedures [2]:

- Division of conjunctival bands and symblepharon.

- Conjunctival or other mucous membrane grafting.
- Correction of eyelid deformities such as cicatricial entropion.
- Keratoplasty for corneal scarring should be delayed for at least 6 months and preferably longer to allow maximal resolution of inflammation.
- In a very severely damaged eye, a keratoprosthesis may be required.

6. Conclusion

In this thesis, I aimed to present a narrative review of the existing literature concerning the topic of ocular burns.

The review has shown that the main focus of the medical and scientific community in this topic is chemical burns, since they are more prevalent and have more severe short- and long-term effects.

My aim was to review the existing knowledge and identify aspects, especially treatment options, that are in the center of interest in this field. The review has shown two main procedures that are still undergoing examination in order to understand better their benefits and mechanism. One is a medical treatment using umbilical cord serum drops, and the other is a surgical treatment, an amniotic membrane transplantation. Both of these treatments are well established in other ophthalmological conditions, but still need to be studied further in the context of chemical ocular burns. In addition, the review has demonstrated the contribution of using the Dua et al system to determine the degree of the burn, as opposed to the Roper-Hall system. Moreover, the review demonstrated that the medical community is still exploring for the best irrigation solutions for chemical burns.

All of the above-mentioned studies and examination demonstrate that even though ocular burns, and specifically chemical ocular burns, are a relatively common ocular trauma that is treated in a similar fashion for years, there is still room for development in its evaluation and treatment.

7. Bibliography

1. Mashige K. Chemical and thermal ocular burns: a review of causes, clinical features and management protocol. South African Family Practice. 2015;58(1):1-4.
2. Salmon J, Kanski J. Kanski's clinical ophthalmology. [Edinburgh]: Elsevier; 2020. Chapter 22, Trauma. P. 912-916
3. Haring R, Sheffield I, Channa R, Canner J, Schneider E. Epidemiologic Trends of Chemical Ocular Burns in the United States. JAMA Ophthalmology [Internet]. 2016;134(10):1119-1124. Available from: https://jamanetwork.com/journals/jamaophthalmology/fullarticle/2540517
4. Bunker D, George R, Kleinschmidt A, Kumar R, Maitz P. Alkali-Related Ocular Burns. Journal of Burn Care & Research. 2014;35(3):261-268.
5. Quesada J, Lloves J, Delgado D. Ocular chemical burns in the workplace: Epidemiological characteristics. Burns. 2020;46(5):1212-1218.
6. Solano J. Ocular burns and chemical injuries. Medscape Reference [Internet] 14 June 2019 [cited 29 April 2022] Available from: https://emedicine.medscape.com/article/798696-overview
7. Bagheri N, Wajda B, Calvo C, Durrani A. The Wills Eye Manual. 7th ed. Hagerstown: Wolters Kluwer Health; 2016. Chapter 4, Cornea, P. 186-188
8. Feder R. Basic and Clinical Science Course. San Francisco, CA: American Association of Ophthalmology, 2019. Section 08, external Disease and Cornea.
9. Eslani M, Baradaran-Rafii A, Movahedan A, Djalilian A. The Ocular Surface Chemical Burns. Journal of Ophthalmology. 2014;2014:1-9
10. Ramponi D. Chemical Burns of the Eye. Advanced Emergency Nursing Journal. 2017;39(3):193-198.
11. Bagheri N, Wajda B, Calvo C, Durrani A. The Wills Eye Manual. 7th ed. Hagerstown: Wolters Kluwer Health; 2016. Chapter 3, Trauma, P. 60-67.
12. Dua H. A, King A. J, Joseph A. new classification of ocular surface burns. British Journal of Ophthalmology. 2001;85(11):1379-1383.
13. Bass M. Handbook of optics. New York: McGraw-Hill; 2010. Volume 3: Chapter 7, ocular radiation hazards. P. 226-241

14. Palao R, Monge I, Ruiz M, Barret J. Chemical burns: Pathophysiology and treatment. Burns. 2010;36(3):295-304.
15. Soleimani M, Naderan M. Management Strategies of Ocular Chemical Burns: Current Perspectives. Clinical Ophthalmology. 2020; Volume 14:2687-2699
16. Bates A, Zanaboni A. Ocular Burns. [Updated 2021 Aug 11]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK459221/
17. Galan N. Slit lamp exam: Uses, procedure, results, and other eye exams [Internet]. Medicalnewstoday.com. 2018 [cited 12 May 2022]. Available from: https://www.medicalnewstoday.com/articles/322267
18. Porter D. What is a Slit Lamp? [Internet]. American Academy of Ophthalmology. 2018 [cited 12 May 2022]. Available from: https://www.aao.org/eye-health/treatments/what-is-slit-lamp
19. Gupta N, Kalaivani M, Tandon R. Comparison of prognostic value of Roper Hall and Dua classification systems in acute ocular burns. British Journal of Ophthalmology. 2010;95(2):194-198.
20. Lewis C, Al-Mousawi A, Jha A, Allison K. Is it time for a change in the approach to chemical burns? The role of Diphoterine ® in the management of cutaneous and ocular chemical injuries. JPRAS. 2017 Feb 17; 70: 563-567.
21. Wiesner N, Dutescu R, Uthoff D, Kottek A, Reim M, Schrage N. First aid therapy for corrosive chemical eye burns: results of a 30-year longitudinal study with two different decontamination concepts. Graefe's Archive for Clinical and Experimental Ophthalmology. 2019;257(8):1795-1803.
22. Sharma N, Goel M, Velpandian T, Titiyal JS, Tandon R, Vajpayee RB. Evaluation of Umbilical Cord Serum Therapy in Acute Ocular Chemical Burns. Invest Ophthalmol Vis Sci 2011;52:1087
23. Gheorghe A, Pop M, Burcea M, Serban M. New clinical application of amniotic membrane transplant for ocular surface disease. J Med Life 2016;9:177–9.
24. Shimazaki J, Yang H-Y, Tsubota K. Amniotic Membrane Transplantation for Ocular Surface Reconstruction in Patients with Chemical and Thermal Burns. Ophthalmology 1997;104:2068–76. https://doi.org/10.1016/S0161-6420(97)30057-8.

25. Tandon R, Gupta N, Kalaivani M, Sharma N, Titiyal J, Vajpayee R. Amniotic membrane transplantation as an adjunct to medical therapy in acute ocular burns. British Journal of Ophthalmology. 2010;95(2):199-204.
26. Clare G, Suleman H, Bunce C, Dua H. Amniotic membrane transplantation for acute ocular burns. Cochrane Database of Systematic Reviews. 2012;2015(4).
27. Westekemper H, Figueiredo F, Siah W, Wagner N, Steuhl K, Meller D. Clinical outcomes of amniotic membrane transplantation in the management of acute ocular chemical injury. British Journal of Ophthalmology. 2016;101(2):103-107.