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Clinical Report

Chemical Corneal Burning due to an Elective Otoplasty in a Boxer Puppy

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Abstract

Case Description- A 3-month-old female intact Boxer dog was presented to the Veterinary Hospital of Shahid Bahonar University of Kerman with a complaint epiphora, photophobia and corneal opacity of both eyes.
Clinical Findings- Ophthalmic examination revealed mild blepharospasm and mucoid discharge of both eyes, with severe bilateral chemosis and conjunctival hyperemia. Severe corneal edema and a white haze to the corneal stroma in the affected area were also observed.
Treatment and Outcome- The dog was admitted to the hospital for 14 days for intensive topical treatment including atropine eye drop, acetylcysteine eye drop, tetracycline eye ointment, ciprofloxacin eye drop, diclofenac eye drop and topical autologous therapy, Three weeks after the injury, the epitheliums were covering entire of the corneal surface and the corneas were fluorescein negative.
Clinical Relevance- Chemical injuries of the eye rarely occur in the animals based on the scarcity of publications on the subject in the veterinary literature. In the present case, chemical eye burns due to accidental exposure to the povidone iodine occurred during an elective surgery.
Key Words- Chemical Injuries, Corneal Burnt, Dog.

Case Description

A 3-month-old female intact Boxer dog was presented to the Veterinary Hospital of Shahid Bahonar University of Kerman with acomplaint epiphora, photophobia and corneal opacity of both eyes. This dog had history of elective otoplasty5 day later. Detailed history delineated that the animal eyes were not covered during surgery due to the carelessness of the operation team. So, they were exposed to the caustic fluid (surgical scrub povidone iodine 7.5%). The referring veterinarian (RV) treated the dog with the following medications: subconjunctival methylprednisolone acetate (Depo-Medrol), topical Prednisolone eye drop, and oral amoxicillin.

Clinical Findings

Ophthalmic examination revealed mild blepharospasm and mucoid discharge of both eyes, with severe bilateral chemosis and conjunctival hyperemia. Marked and extensive corneal edema and a white haze to the corneal stroma in the affected area were also observed. Furthermore, deposit staining was determined by fluorescein eye test and approximately eighty percent of the corneal surface was superficially ulcerated (Fig. 1). The pupils were also meiotic. Menace response, dazzle, and the pupillary light reflex (both direct and indirect) were absent. Moderate aqueous flare was seen in both eyes. Moreover, intraocular pressure measurements by using Vintage Shiotz Tonometer, were recorded as OD (right eye), 18mmHg and OS (left eye): 20mmHg. The pH of the lower conjunctival sac was also assessed using urine test strip as pH=7and therefore, within normal limits of course five days after injury. The corneal edema did not allow examination of the intraocular structures; therefore, the ocular sonography was recommended but the owner refused this non-invasive diagnostic procedure to evaluate intraocular structures.

Treatment and Outcome

The dog was admitted to the hospital for 14days for intensive medical treatment including 1% atropine eye

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drop once as an cycloplagic drug; autologous serum eye drops q4h for 8 days, 5%Acetylcysteineeye drop q2h for 7 days, then q6h for 2 weeks, tetracycline eye ointment q12h for 14 days and 0.1%EthyleneDiamineTetraAcetic Acid(EDTA) eyedrops q12h for 7 days, all as a matrix metalloproteinase inhibitor agents; 2% Dorzolamide/ Timolol eye drop q8h for 7days, then q12h for 2 months to control intra ocular pressure, 0.3% Ciprofloxacineye drop q4h for 7 days, then q8h for8 weeks, 0.5% Diclofenac eye drop q6h for 7days and cyclosporine eye drop q12h for 7 days, then at reducing dose over 2 months, with anti-inflammatory and analgesic effects.
Additionally patient was treated by Tramadol as a strong pain relief agent, 0.2 mg/kg PO q12h for 5 days and parenteral ascorbic acid, 50mg/kg IM as A-synthetic metalloproteinase inhibitor to reduce corneal ulceration and prevention of corneal melting in the alkali-injured eye. Ofloxacin, 15mg/kg q12h PO, was also administrated according to the synergistic effect with topical ciprofloxacin for3 weeks. During hospitalization, some fibrin strands were found in the anterior chambers, which resolved slowly on the prescribed medication. At reexamination, 15 days after the initial presentation, corneal vascularization was present from the dorsolateral limbus of the eyes. Three weeks after the injury, the epitheliums were covering entire of the corneal surface and the corneas were fluorescein negative; while, corneal vascularization was advancing further. The corneal edema remained unchanged with some vascularization and bullous keratopathy in both eyes (Fig.2). The eyes were not visual, even though light reflexes, including dazzle and consensual pupillary light reflex were absent. Treatment options, including a thermokeratoplasty and enucleation of the eyes were discussed with the owner to improve the dog's comfort, but declined.

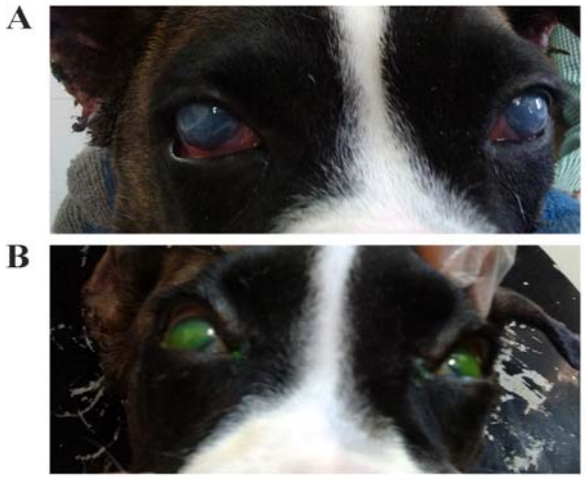

Figure 1- bilateral chemosis, conjunctival hyperemia, Marked corneal edema (A), corneal ulcer was determined by fluorescein eye test and approximately eighty percent of the corneal surface was superficially ulcerated (B).

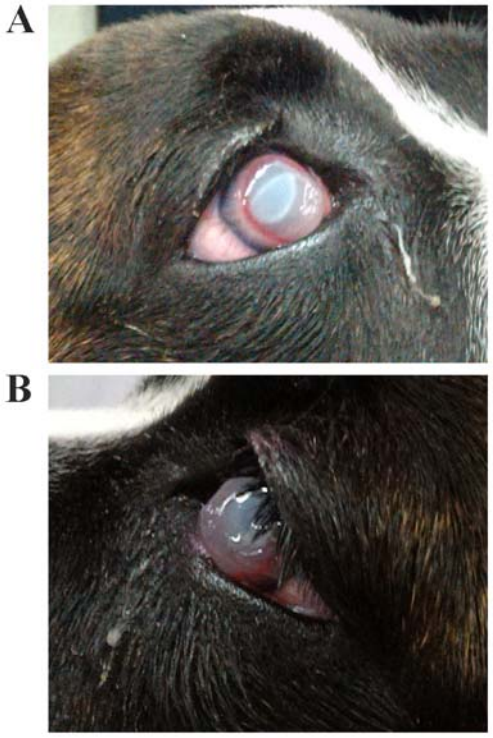

Figure 2- Three weeks after the injury, the epitheliums were covering entire of the corneal surface and the corneas were fluorescein negative. The corneal edema remained unchanged with some vascularization and bullous keratopathy in both right (A) and left (B) eyes.

Clinical Relevance

Alkaline injuries of the eye rarely occur in the animals based on the scarcity of publications on the subject in the veterinary literature.[9]In the present case, chemical eye burns due to accidental exposure to the Povidone Iodine 7.5% occurred during an elective surgery.
Alkaline chemicals cause injuries via two different chemical processes: the saponification of triglycerides found in the cell membranes by the hydroxyl ions, which leads to acute lysis of these cell membranes and the denaturation and hydrolysis of proteins due to the change in pH. The hydrolysis leads to a loss of the quaternary, tertiary, and secondary structures of proteins and therefore, to a loss of function of structural as well as transport proteins and enzymes. Consequences are cell death, reduced mechanical strength, and impaired repair mechanisms. Damage to the corneal epithelial stem cells at the limbus will particularly influence the corneal healing process and therefore, the visual outcome.[3]Perilimbal whitening is a useful indicator in humans to judge the extent of corneal stem cell damage and indirectly the injury to the underlying ciliary body and trabecular meshwork.[8]The alkaline molecules may rapidly penetrate the cornea and enter the anterior

chamber. This usually destroys the keratocytes of the affected stroma and damages the corneal endothelial cells. Damage to intraocular structures, including iris, ciliary body, and lens due to penetrated alkaline molecules to the cornea has been reported in humans.[8]An acute, increase in intraocular pressure has been directly measured in rabbits and monkeys after the alkaline molecules penetrate the cornea and destroy anterior chamber structures.[7]In accordance with this, a raised intraocular pressure was detected in the present case. Afibrinous uveitis, as observed in this case, may result from pH-induced cell lysis and release of necrotic debris into the aqueous humor that might be the cause of increased IOP. Acute clinical signs including, blepharospasm, chemosis, and conjunctival hyperemia were observed in this dog. Furthermore, there was an extensive destruction of the corneal epithelium exposed to the alkaline substance. Saponification and denaturation of the stroma were also visible as fine white opacities subsequently; mild to moderate corneal edema was present too. In cases of intraocular involvement, Synechiae, ocular hypertension, iris paralysis, cataract and phthisis bulbi may result.[8]Long-term complications in the mentioned case include mild corneal pigmentation, vascularization and fibrosis, bilateral permanent keratoconus, and corneal edema.

In cases of acute ocular alkaline exposure, treatment should be initially focused on immediately removing the alkaline agent and neutralizing the ocular surface and intraocular pH to prevent ongoing damage by removing visible, particles and vigorous flushing of the ocular surface.[12]

Irrigation should be continued until the normal tear pH of 7.5 is reached. The use of purpose-made eye flushes for chemical burns is limited in veterinary medicine due to their high costs. Fluids with low osmolarity such as tap water can be used instead. They facilitate corneal edema and therefore, dilute the agent in the stroma.[4]Necrotic tissues have to be removed from the eye surface, as it increases inflammatory mediators that attract and stimulate polymorph nuclear cells, which potentially cause damage to the normal tissue by releasing a large amount of proteinases. Topical broad-spectrum antibiotics and matrix metalloproteinase inhibitors (MMPI) drugs should be used to prevent secondary bacterial infection and corneal degradation from proteinases. Proteases or proteinases are classified into Matrix metalloproteinase (MMPs), serine proteinase, aspartic proteinase, and cysteine proteinase.[3]MMPs and serine proteinases have been investigated in the horse, rat, rabbit, and canine corneas. [5]In the normal cornea, the degradatory properties of these enzymes are antagonized by tissue inhibitors of metalloproteinase. However, when imbalance occurs between MMPs and their inhibitors, disruption, disintegration or digestion of the corneal extra-cellular matrix occurs, leading to keratomalacia.[6] Thus, exogenous MMP inhibitors are commonly used in the clinical setting in an attempt to avoid this complication. Matrix metalloproteinases inhibitors (MMPI) such as acetylcysteine, EDTA, synthetic inhibitors of metalloproteinases (SIMP) and topical autologous serum have been used therapeutically on corneal chemical wounds and proved efficient in preventing corneal ulceration and perforation.[11] Application of autologous serum also could providean extra supply of the necessary proteins, suchas fibronectin, albumin, and epidermal growth factor, for epithelial regeneration after alkali injury of the cornea.[11]Tetracycline and Doxycycline can protect the cornea against proteolytic degradation after moderate to severe ocular chemical injury and have MMPI effects.[10]It is believed that the MMPI effects of tetracycline have more therapeutic effects in treatment of corneal ulcer than their antibacterial effects. In this patient, we used tetracycline, Acetylcysteine and ascorbic acid as MMPI agents.

Pain receptors are often destroyed by the injury, leading to a short-lived initial excruciating pain.[8] So, we used systemic opioid (Tramadol) and topical atropine 1% to decrease the pain and the spasm of the ciliary body muscles. Cyclosporine might be beneficial in suppressing inflammatory cytokine expression, as shown in a rat model.[1] According to this, topical cyclosporine was also administered in our case. This dog presented with persistent corneal edema, corneal scarring and conjunctivalization and corneal pigmentation following the alkaline injury and remained visually impaired six months after that due to the lack of regenerative capacities of the canine corneal endothelial cells. Late phase management should be addressed to surgical procedures that might include superficial keratectomy, limbalauto-graft, and penetrating keratoplasty.[12] Unfortunately, surgery was declined by the owner.

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چکیده

سوختگی شیمیایی قرنیه بدلیل اتوپلاستی قرنیه در یک قلاده توله سگ نژاد باکسر

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توصیف بیمار– یک قلاده سگ ماده سه ماهه نژاد باکسر، با علائم ریزش اشک، ترس از نور و تاری قرنیه به بیمارستان دامپزشکی دانشگاه شهید باهنر کرمان مراجعه نمود.
یافته‌های بالینی- پس از انجام معاینات بالینی، اسپاسم ملایم پلک، ترشحات موکوئیدی، کموزیس دو طرفه شدید، پرخونی ملتحمه، ادم قرنیه و ترشحات سفید رنگ کدر در استرومای قرنیه‌ی مناطق آسیب دیده مشاهده شد و با توجه به تاریخچه‌ی اخذ شده، سوختگی شیمیایی تشخیص داده شد.
درمان و نتیجه- بیمار طی ۱۴ روز تحت درمان گسترده‌ی موضعی شامل استفاده از قطره‌های چشمی آتروپین، استیل سیستئین، سیپروفلوکساسین، دیکلوفناک، پماد چشمی تتراسایکلین و درمان‌های موضعی دیگر از این قبیل قرار گرفت.
سه هفته پس از شروع درمان، لایه‌ی اپیتلیال منطقه‌ی آسیب دیده را پوشانده و جواب تست فلوئورسین منفی بود.
کابرد بالینی– براساس مطالعات مربوطه‌ی موجود در نشریات دامپزشکی، سوختگی‌های شیمیایی در حیوانات به ندرت اتفاق می‌افتد، در بیمار مورد نظر، سوختگی در اثر تماس تصادفی بتادین یده با چشم حین جراحی انتخابی گوش بری اتفاق افتاده است.
کلید واژگان- آسیب شیمیایی، سوختگی قرنیه، سگ.