

CASE REPORT

Equine eosinophilic keratitis in horses: 28 cases (2003–2013)

Sydney Edwards¹, Alison B. Clode² & Brian C. Gilger²

¹Department of Surgical and Radiological Sciences, School of Veterinary Medicine, University of California-Davis, One Shields Avenue, Davis, California, 95616

²Department of Clinical Sciences, North Carolina State University College of Veterinary Medicine, 1052 William Moore Drive, Raleigh, North Carolina, 27607

Correspondence

Present address: Alison B. Clode, Port City Veterinary Referral Hospital, 215 Commerce Way, Suite 100, Portsmouth, NH 03801.
Tel: (603) 433-0056; Fax: (603) 433-0029;
E-mail: alisonclode@gmail.com

Funding Information

No sources of funding were declared for this study.

Received: 4 January 2015; Revised: 9 April 2015; Accepted: 29 July 2015

Clinical Case Reports 2015; 3(12): 1000–1006

doi: 10.1002/ccr3.350

Introduction

EK (Eosinophilic keratoconjunctivitis) is an inflammatory condition affecting primarily the cornea, with coinciding involvement of the conjunctiva and third eyelid [1], that causes discomfort and visual impairment associated with the development of unilateral or bilateral whitish corneal plaques and frequent corneal ulceration and edema. Alternatively, EK may present as a superficial stromal yellow perilimbal infiltrate [2, 3]. Regardless of the clinical presentation, cytologic evaluation identifies eosinophils when examined microscopically. Eosinophilic keratitis has been reported in horses, [3–10], cats [11–16], and rabbits [17].

While the exact etiology of EK is unknown, an underlying type I or IV hypersensitivity reaction to parasitic or environmental allergens has been suggested [18, 19]. Additionally, environmental factors such as geographical location and weather conditions [5] have been implicated, as EK has a predilection for presentation in summer months. One study reported 78% of EK diagnoses occurred in the summer months, with the majority during July (41%), followed by June (22%), and August (15%) [3]. It has been suggested that, in combination

Key Clinical Message

This retrospective case series evaluates husbandry and environmental conditions in relation to eosinophilic keratitis in horses. While no associations were found between disease and husbandry practices or specific environmental factors such as humidity or temperature, an increased prevalence of presentation in summer months was identified in this population of horses.

Keywords

Eosinophilic conjunctivitis, eosinophilic keratitis, eosinophilic keratoconjunctivitis, equine.

with topical therapy, controlling husbandry factors, specifically keeping fields cut short, using fly masks during turn out, increasing stall confinement to reduce time on pasture, and administering topical fly repellent under the eyes may be associated with resolution of clinical signs in all cases [3].

A diagnosis of EK is made by observing nonspecific clinical signs (blepharospasm, chemosis, conjunctival hyperemia, and epiphora) along with identifying eosinophils on cytological and/or histological examinations of corneal specimens [4, 5]. Treatment of EK is limited in that most of our guidelines are extrapolated from human literature, or based on a few case reports in the veterinary literature. Treatment of EK is either medical or surgical, with reported medical treatments of EK including topical corticosteroids [5, 6], a tapering regimen of systemic corticosteroids [3], topical nonsteroidal anti-inflammatory medications, topical mast cell stabilizers, and topical antihistamines [19]. Immune-modulating drugs such as cyclosporine have also been used in feline patients, with improvement seen in 88.6%, and a recurrence rate of 22.6% in affected cats [16]. Surgical treatments for EK include superficial keratectomy and corneal debridement using a diamond burr, used anecdotally in horses in a

manner similar to that described in dogs for treatment of indolent ulcers [20]. The outcome for a comfortable globe and remission from EK is variable, but with an appropriate treatment protocol the prognosis is good. One study reports out of 46 eyes, all retained vision, with the most common sequelae being mild corneal fibrosis [3].

As with other disease conditions, recognition of potential predisposing factors or other underlying causes may allow modifications in both treatment and prevention strategies that may decrease the prevalence and severity of the disease. The purpose of this study was to determine if signalment, husbandry practices, or environmental factors predispose horses to develop EK in NC (North Carolina).

Materials and Methods

Study population

Records of horses diagnosed with EK that were presented to the NCSU-VTH (North Carolina State University Veterinary Teaching Hospital) between 2003 and 2013 were reviewed. Only horses receiving a complete ophthalmic examination, including topical fluorescein dye application (Ful-Glo fluorescein sodium ophthalmic strips, USP, 1.0 mg; Akorn, Inc., Lake Forest, IL 60045), applanation tonometry (TonoPen XL, Medtronic, Jacksonville, FL), slit-lamp biomicroscopy (Kowa SL-14 slit lamp; Kowa Company, Ltd., Tokyo, Japan), and indirect ophthalmoscopy (Volk indirect lens; Volk Optical, Inc., Mentor, OH) after mydriasis (Tropicamide Ophthalmic Solution; USP, Akorn, Inc.), by a board-certified veterinary ophthalmologist were eligible for inclusion in the study population. Horses were included in the study population based on a confirmed diagnosis of EK made via identification of eosinophils on corneal cytology and/or histopathology. Clinical signs that were variably present included mild secondary uveitis (miosis, flare, ocular hypotony), uncomplicated superficial corneal ulceration, and evidence of concurrent bacterial and/or fungal keratitis on corneal cytology, culture, or histopathology specimens.

Procedures

Information retrieved from the medical records included signalment, city of residence, year of presentation to the NCSU-VTH, and month clinical signs were first observed. Information on specific husbandry practices was obtained, including whether the horse was pastured, stalled, or a combination of the two; if a combination, was the horse stalled between 8:00 and 20:00 h (hereafter referred to as day) or stalled between 20:01 and 7:59 h (hereafter referred to as night); the diet fed (specific types of hay,

grain, and supplements). When possible, information regarding vaccination and deworming protocol was also obtained. Information obtained from the NOAA (National Oceanic and Atmospheric Administration) for environmental factors was based on the city of residence and year and month clinical signs were first observed (as obtained from the medical record) for each individual horse, and included average rainfall in “in” (inches), average high temperature (°C), average low temperature (°C), and average monthly relative humidity (%). Because the majority of horses presented from NC, we attempted to further evaluate if region within the state played a role in EK development by further dividing horses that presented from NC into three geographic groups based on whether they presented from the mountain, piedmont, or coastal region (Fig. 1). Regions from other states were not examined simply due to the low number of horses presenting from outside NC.

Results

Signalment

Twenty-eight horses were diagnosed with EK between 2003 and 2013 and met the inclusion criteria, with 20 presenting from NC and the remaining presenting from South Carolina ($n = 4$), Virginia ($n = 3$), and New York ($n = 1$). Ten geldings and 18 mares were affected. Breeds represented were Thoroughbred ($n = 8$), Quarter Horse ($n = 7$), Arabian ($n = 4$), Hanoverian ($n = 2$), Warm Blood ($n = 2$), and one each from the following breeds: Walking Horse, Appaloosa, Mule, Miniature Horse, and Standardbred. Quarterhorses and Thoroughbreds (13% and 15%, respectively) were the most common breed affected with EK. Ages of horses with EK ranged from 3 weeks to 27 years, with a mean (\pm SD) of 10.8 (\pm 7.4) years. Signalment data are summarized in Table 1.

Clinical course of disease

Twenty-one of 28 horses had received topical treatment prescribed by their primary veterinarian prior to presentation to the NCSU-CVM. The following medications were used: topical antimicrobials (21 horses), topical corticosteroids (five horses), topical cyclosporine (two horses), topical antifungals (seven horses), topical NSAIDS (two horses), systemic NSAIDS (10 horses), and systemic antibiotics (three horses).

The majority of horses ($n = 19$) presented with all of the following clinical signs at presentation: blepharospasm, epiphora, and corneal ulceration. All horses had unilateral involvement at time of presentation. Ten horses also presented with a superficial stromal perilimbal

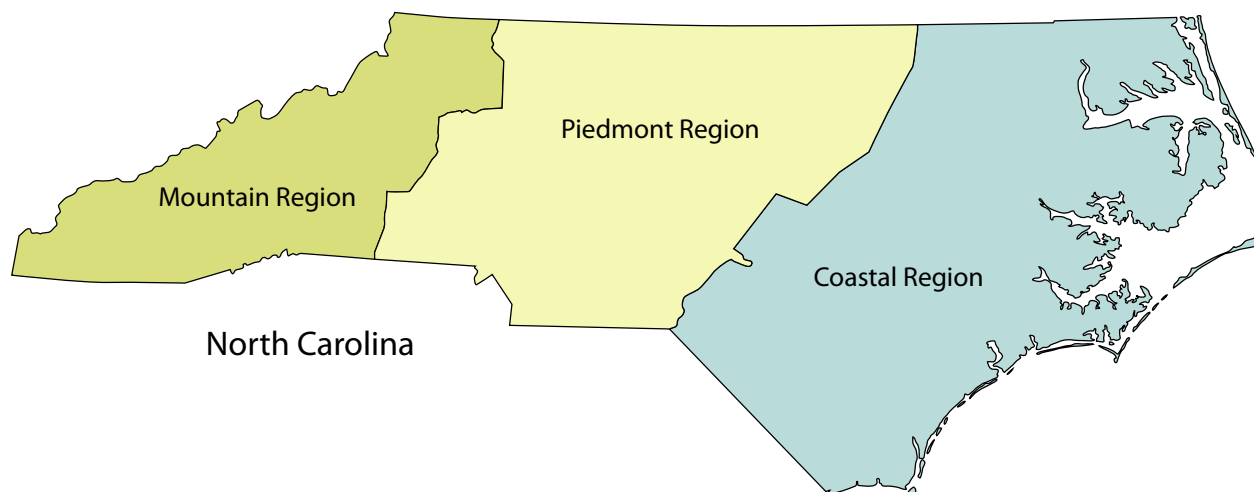


Figure 1. Geographical regions of North Carolina. Adapted from E. Ralsa. Landforms of the United States (Waltham Blaisdell Publishing Co. 1940).

Table 1. Signalment data of EK (eosinophilic keratoconjunctivitis) horses that were presented to the North Carolina State University Veterinary Teaching Hospital Equine Ophthalmology Service between 2003 and 2013.

	EK (n = 28)
Breed	
TB	8
QH	7
Other	13
Age (years)	
mean (±SD)	10.8 (±7.4)
Gender	
Male	10
Female	18

yellow infiltrate. Eight horses presented only with conjunctivitis characterized by a caseous exudate.

At admission and before initiation of treatment, all horses had corneal cytology and culture samples taken. Cytology in 10 horses revealed predominantly eosinophils, and mixed eosinophil and neutrophil populations in the remaining 18 horses. Fungal cultures were positive in 10 horses, eight with *Aspergillus* spp. and two with *Fusarium* spp. Eight horses with positive fungal cultures also had secondary bacterial infections, confirmed via culture as *Staphylococcus haemolyticus* (n = 5) and *Stenotrophomonas maltophilia* (n = 3).

Although the prevalence of eosinophils and clinical picture in most horses was supportive of EK and thus treatment with topical corticosteroids was indicated, based on the potential for fungal infections associated with the time of year and geographic location, most patients (18 horses)

were started on a topical antimicrobial (Neomycin–Polymyxin B sulfates–Bacitracin zinc ophthalmic ointment: Bausch & Lomb, Inc., Tampa, FL and Akorn, Inc., Lake Forest, IL, or Ofloxacin Ophthalmic Solution USP 0.3%: Alcon Laboratories, Inc., Fort Worth, Texas 76134 for Sandoz Inc., Princeton, NJ) and a topical antifungal, either voriconazole (VFEND; Pfizer Inc., New York, NY; nine horses), natamycin (Nayacyn; Alcon Laboratories Inc., Ft. Worth TX; five horses), or both (seven horses) immediately after corneal cytology collection and coinciding corneal ulceration. Horses were also administered topical cyclosporine (Optimmune Schering Plough, Inc., Kenilworth, NJ) (n = 8), and neomycin–polymyxin–dexamethasone (Sandoz, Princeton, NJ and Alcon Labs, Fort Worth, TX) (n = 15) specifically for treatment of EK. Fifteen horses were started on oral TMS (Lannett Company Inc., Philadelphia, PA), 18 horses were started on oral doxycycline (Vibramycin; Pfizer Inc., New York, NY), and 15 horses were started on oral banamine.

The mean daily stay in the hospital for all EK horses was 7 days (± SD 4.2). The average time to resolution of clinical disease was within 4.1 months (± SD 3.2) of discharge from the hospital, with the range being 3.2–8.7 months. Five horses underwent a standing keratectomy in addition to medical treatment, a decision made at the discretion of the attending ophthalmologist. These horses tended to have a quicker resolution on disease (average 2.7 months), with the range being 0.9–3.4 months.

Husbandry practices

Most horses in the EK group (18/28) were stalled during the day and pastured during the night. The feed types

Table 2. Seasonality of presentation of horses to the North Carolina State University Veterinary Teaching Hospital Equine Ophthalmology Service between 2003 and 2013.

	Number of horses presenting with EK (eosinophilic keratoconjunctivitis) (% of total EK horses)
Winter	0 (0%)
Fall	8.0 (29%)
Spring	2.0 (8%)
Summer	18.0 (63%)

Dec, Jan, Feb = Winter; Mar, Apr, May = Spring; June, Jul, Aug = Summer; Sept, Oct, Nov = Winter.

were similar between individual EK horses, including hay, complete feeds, and supplements. The hay fed to EK horses included timothy ($n = 11$), fescue ($n = 8$), alfalfa ($n = 3$), pasture ($n = 5$), and bermuda ($n = 1$). Complete feeds were reported according to the amount of digestible energy which included maintenance feeds ($n = 5$), high energy feeds ($n = 5$), and senior feeds ($n = 2$). Supplements included fitzgerald grain ($n = 1$), granola ($n = 1$), bran mash ($n = 1$), oats ($n = 2$), bran pulp ($n = 6$), and sweet feed ($n = 4$). Most horses ($n = 22$) were currently on vaccinations for rabies, EEE, WWE, WNV, and influenza/rhinotracheitis/tetanus. Five horses were not currently vaccinated against rabies. All horses had a negative Coggins test. Information regarding specific variables such as insect control, bedding type, and proximity of hay/straw storage relative to the living space of horses were not consistently reported in medical records.

Environmental factors

The majority of EK horses (20/28) presented from the Piedmont region of NC. One EK horse presented from

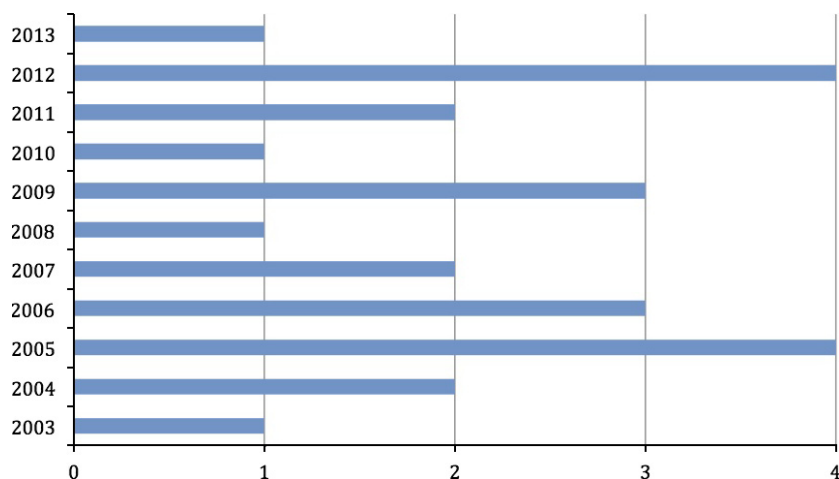
Table 3. Reported environmental factors for EK (eosinophilic keratoconjunctivitis) horses that presented to the North Carolina State University Veterinary Teaching Hospital Equine Ophthalmology Service between 2003 and 2013.

	EK population, average (\pm SD)
Rainfall (inches)	3.5 (\pm 0.45)
High temperature ($^{\circ}$ C)	28.5 (\pm 1.62)
Low temperature ($^{\circ}$ C)	16.4 (\pm 1.80)
Relative humidity (%)	70.4 (\pm 1.24)

the mountain region of NC, and zero horses presented from the coastal region of NC. The remaining EK horses presented to NCSU-CVM from regions other than North Carolina (New York: $n = 1$, South Carolina: $n = 3$, and Virginia: $n = 3$). The majority of EK horses, regardless of area of presentation (62.5%), began showing clinical signs in the summer months (June, July, and August; Table 2). Year of presentation to the NCSU-VTH is reported in Figure 2. The average rainfall, average high temperature, average low temperature, and average hourly relative humidity for EK horses are reported in Table 3.

Discussion

The objectives in the present study were to report signalment, husbandry practices, and environmental factors affecting EK horses presented to the NCSU-VTH. The majority of the reported data are from horses residing in NC, however a portion of horses presented to the NCSU-VTH were from other regions outside NC, therefore information obtained from their clinical cases may not be directly relatable to that from cases in NC. In addition, the NCSU-VTH only saw 38 cases of confirmed EK, of which 28 horses met our inclusion criteria, over the 10 years of

**Figure 2.** Number of horses with eosinophilic keratoconjunctivitis presenting to the North Carolina State University Veterinary Teaching Hospital Equine Ophthalmology Service between 2003 and 2013.

this review. These number trends are similar with previous reported cases of EK in that the overall disease prevalence, as reported in the literature, is relatively low.

The results of our study showed that there was an increased prevalence of EK in female horses, based on a male-to-female ratio of 1.0:1.8 in the EK group. As we performed no statistical comparison, conclusions on age, sex, and breed predilection need to be drawn carefully. In humans, the opposite gender predilection has been suggested, with a genetic sex-linked predisposition to eosinophilic disorders postulated in males [21–24]. One example to support this is the finding of a hematopoietic stem cell mutation involving the *PDGFRA* gene, found mainly in the male population affected with eosinophilic diseases [22]. As more female horses were affected with EK in this study, it is possible that non-sex-linked genetic predispositions may exist in mares related to the development of EK. It is important to note, however, that ocular eosinophilic disorders may also lack pathophysiological correlations with systemic eosinophilic disorders. Additionally, the small EK sample size may not truly represent the entire population of EK horses affected in other geographical regions. Given the findings in this study, the potential for sex-based differences in disease prevalence of human eosinophilic conditions [23–24] and (to the authors' knowledge) the absence of studies evaluating a genetic susceptibility to eosinophilic disorders in horses, this may be an area of study deserving of future efforts.

The age of horses presented to the NCSU-VTH affected with EK (mean 10.8 years) was similar to other studies reporting EK in horses (8.2 and 9.7) [3, 5]; however, these reports are mainly case reports and do not include a control population. In humans, children and young adults within the first third of life are more prone to developing eosinophilic disorders, suggested to be due to heightened allergen sensitization present at a younger age which results in greater levels of IgE and eosinophil recruitment to a pathological level [23]. While determining such potential connections would be ideal in identifying and controlling EK in horses, the small size of the study population makes drawing conclusions regarding age and EK development in horses difficult.

Thoroughbreds and Quarter Horses were the most frequently affected breeds with EK at the NCSU-VTH; however, those two breeds also represent the majority of horses that presented to the NCSU-VTH for reasons other than ophthalmic disease (data not shown). Another study of EK horses identified five affected Thoroughbreds, one affected Quarter horse, and one affected Arabian [5], thus it is possible that a true breed predilection would exist when greater numbers of affected individuals are available for study.

The seasonality of horses presenting with EK in this study is similar to that previously reported as the majority

of EK horses (62.5%) began showing clinical signs during the summer months (June–August). In one report, a large number of horses (41%) presented during the month of July [3]. Other studies report that horses included in the study presented during the warmer summer months [4, 5, 19]. Insect hypersensitivity [25], which is exacerbated in warmer months, was correlated with development of EK, however further studies are needed to fully support this finding.

When considering the possible involvement of diet in the development of eosinophilic diseases, it is generally considered that either certain dietary components are more likely to trigger an allergic immune response, or certain individuals are more genetically susceptible to developing a reaction secondary to dietary triggers. We did not statistically analyze differences in diets between the EK and general hospital populations due to wide variability, however it may be interesting in future studies to identify dietary trends among horses that develop EK relative to a control population of horses.

In addition to examining dietary factors, deworming and vaccination protocols were gathered from medical records and follow-up phone calls, however insufficient data were collected to facilitate any statement about their role, if any, in EK development. Based on data review without statistical analysis, all horses were on appropriate vaccination and deworming schedules. A bacterial infection or release of *Onchocerca* secondary to recent deworming has been suggested as a possible pathogenesis for EK development due to eosinophilic infiltration of the cornea that occurs with parasitic keratitis [18], however such a connection was not apparent in the current study.

Most EK horses were stalled during the day and pastured during night. While it would have been ideal to report further details explaining the exact time of day, and exact number of turn out hours per day for each horse, we were unable to accurately gather this information due to the retrospective nature of this report, and the reliance on owner memory for details. Therefore, we report broad categories of time divided into 12-h divisions of turnout during the day versus night. Eleven horses used in this report were deceased or no longer owned by the contacted owner, therefore exact times of turnout were difficult to recall. Recent studies have suggested that limiting daytime outdoor exposure does decrease the incidence of EK by limiting exposure to environmental allergens, UV light, and fly hypersensitivities [25]. Following the idea that outdoor exposure is linked to EK development, it is noteworthy that, consistent with a previous study [3], the majority of horses in this study initially presented with EK in the months of June, July, or August when environmental conditions can be extreme. Climate determines the flora and fauna, and thus environmental allergens, within particular

geographical regions, thus exacerbation of symptoms in patients during the spring and summer months may indicate that season-specific flowering plants and pollens are allergic triggers. For future studies, it would be beneficial to find out more information specific to composition of fields in which patients were turned out.

As an additional possible explanation, the summer months, according to NOAA, are associated with higher temperatures (average high 29.6°C), humidity levels (average 72%), and rainfall volume (average 3.2 in/month), thus it is possible that as temperature, humidity, and rainfall increase, so does the incidence of EK. To determine such a connection, however, further studies with control population from a different environment would be necessary. Alternatively, it may be possible that such environmental factors may expedite clinical signs once a horse develops EK, rather than predisposing development of disease. A final possible explanation for the rise in EK cases during the summer months may be associated with air quality in these regions during the more humid summer months. Gaseous materials such as ozone, nitrogen dioxide, and particulate matter generated in high traffic areas are greater in summer months and play a role in IgE-mediated hypersensitivity conditions in humans [23], thus it is possible that they are involved in the pathogenesis of EK in our study population. Additional data regarding detailed aspects of air quality would need to be gathered to better assess this hypothesis.

Horses presenting from separate regions within NC (mountain, piedmont, and coastal) were evaluated to determine the incidence of EK development in each region. Data from NOAA suggest that overall, the mountain region tends to have lower temperature and humidity levels throughout the year when compared to the piedmont and coastal regions, with temperatures varying by 15–20° and humidity by 10–15%. The greatest number of EK horses presented from the piedmont region of NC, which could be explained by the location of the NCSU-CVM in the piedmont region, thus, making it more likely owners living in this region can bring their horses to the NCSU-VTH for veterinary care. Additionally, the piedmont region may be more densely populated with horses compared to the mountain and coastal regions. Due to the small number of horses presenting from regions other than the piedmont, potentially for the above-stated reasons, conclusions of the impact of geographical location on the incidence of EK cannot be made.

Horses diagnosed with EK experienced resolution of clinical signs within 4.1 months, which is similar to other reports of EK which ranged from 2 to 4 months [3–8, 10]. All horses were treated with a combination of a topical antibiotic, topical antifungal, and a topical mydriatic,

however, the variability in medication prescribed to all horses in this study prevented statistical analysis between medications prescribed and clinical course of disease. The authors propose that using these medications in a prospective study environment may be better to assess medical therapy on disease severity and progression.

Limitations of this study are inherent to retrospective evaluations. The small number of horses and, in some cases, incomplete information limit identification of statistically significant differences in the factors evaluated. Ideally, future studies evaluating larger numbers of horses diagnosed with EK residing in different geographical regions will allow comparison of specific environmental and husbandry factors, including those not specifically evaluated in this study, and thus yield more statistically relevant results.

Conflict of Interest

None declared.

References

1. Morgan, R. V., and K. L. Abrmas. 1996. Feline eosinophilic keratitis: a retrospective study of 54 cases (1989–1994). *Vet. Comp. Ophthalmol.* 6:131–134.
2. Clode, A. B. 2011. Diseases and surgery of the cornea. Pp. 181–266 in B. C. Gilger, ed. *Equine ophthalmology*. 2nd ed. Elsevier Saunders, Maryland Heights, MO.
3. Utter, M. L., C. Miller, and K. L. Wotman. 2013. Eosinophilic keratitis in 46 eyes of 27 horses in the Mid-Atlantic United States (2008–2012). *Vet. Ophthalmol.* 17:1–10.
4. Ramsey, D. T., H. E. Whiteley, P. A. Jr Gerding, and R. A. Valdez. 1994. Eosinophilic keratoconjunctivitis in a horse. *J. Am. Vet. Med. Assoc.* 205:1308–1311.
5. Yamagata, M., D. A. Wilkie, and B. C. Gilger. 1996. Eosinophilic keratoconjunctivitis in seven horses. *J. Am. Vet. Med. Assoc.* 209:1283–1286.
6. Jennings, A. F. 2007. Eosinophilic keratoconjunctivitis in a donkey. *British Association of Veterinary Ophthalmology Annual Conference*.
7. Kainzbauer, C., R. Steinborn, G. Mair-Scorpio, et al. 2008. PCR-mediated detection of bovine papillomavirus E5 and L1 DNA in equine eosinophilic conjunctivitis/keratitis. *39th Annual Meeting of the American College of Veterinary Ophthalmologists* 11:413–429.
8. Sandberg, C. A., I. P. Herring, J. J. Schorling, et al. 2008. Ulcerative eosinophilic keratoconjunctivitis in three horses: clinical course and characterization by electron microscopy. *39th Annual Meeting of the American College of Veterinary Ophthalmologists* 11:413–429.
9. Kafarnik, C. 2010. Equine eosinophilic keratitis. *Comp. Anim.* 15:4–6.

10. Chisholm, W. H. 1989. Feline eosinophilic keratitis. *Can. Vet. J.* 30:438–443.
11. Nasisse, M. P., T. L. Glover, C. P. Moore, et al. 1998. Detection of feline herpesvirus 1 DNA in corneas of cats with eosinophilic keratitis or corneal sequestration. *Am. J. Vet. Res.* 59:856–858.
12. Andrew, S. E. 2001. Ocular manifestations of feline herpesvirus. *J. Feline Med. Surg.* 3:9–16.
13. Moore, P. A. 2005. Feline corneal disease. *Clin. Tech. Small Anim. Pract.* 20:83–93.
14. Malik, R., N. S. Lessels, S. Webb, et al. 2009. Treatment of feline herpesvirus-1 associated disease in cats with famciclovir and related drugs. *J. Feline Med. Surg.* 11:40–48.
15. Spiess, A. K., J. S. Sapienza, and A. Mayordomo. 2009. Treatment of proliferative feline eosinophilic keratitis with topical 1.5% cyclosporine: 35 cases. *Vet. Ophthalmol.* 12:132–137.
16. Grininger, P., R. Sanchez, I. M. Kraijer-Huver, et al. 2012. Eosinophilic keratoconjunctivitis in two rabbits. *Vet. Ophthalmol.* 15:59–65.
17. Schmidt, G., J. Krehbiel, S. Coley, et al. 1982. Equine ocular onchocerciasis: histopathologic study. *Am. J. Vet. Res.* 43:1371–1375.
18. Gilger, B. C. 2013. Equine ophthalmology. Pp. 1584–1585 in K. N. Gelat, ed. *Veterinary ophthalmology*. 5th ed.. John Wiley & Sons Inc, Ames, IO.
19. Moawad, F. J., G. R. Veerappan, and R. K. Wong. 2009. Eosinophilic esophagitis. *Dig. Dis. Sci.* 54:1818–1828.
20. Sila, G. H., R. J. Morreale, D. W. Lorimer, et al. 2009. A retrospective evaluation of the diamond burr superficial keratectomy in the treatment of spontaneous chronic epithelial defects in dogs from 2006 to 2008. *40th Annual Meeting of the American College of Veterinary Ophthalmologists* 40:404.
21. Straumann, A., S. S. Aceves, C. Blanchard, et al. 2012. Pediatric and adult eosinophilic esophagitis: similarities and differences. *Allergy* 67:477–490.
22. Weller, P. F., and G. J. Bublely. 1994. The idiopathic hypereosinophilic syndrome. *Blood* 83:2759–2779.
23. Miranda, C., A. Busacker, M. D. Balzer, et al. 2003. Distinguishing severe asthma phenotypes: role of age at onset and eosinophilic inflammation. *J. Allergy Clin. Immunol.* 113:101–108.
24. Vasiliou, J. E., S. Lui, S. A. Walker, et al. 2014. Vitamin D deficiency induces Th2 skewing and eosinophilia in neonatal allergic airway disease. *Allergy* 69:256–260.
25. Utter, M. E., D. Keenan, L. Makkreel, et al. 2010. Eosinophilic keratoconjunctivitis in 19 mares on a Standardbred farm. Abstracts: 41st Annual Meeting of the American College of Veterinary Ophthalmologists. *Vet. Ophthalmol.* 13:407–423.
26. Liacouras, C. A., J. M. Spergel, E. Ruchelli, et al. 2005. Eosinophilic esophagitis: a 10-year experience in 381 children. *Clin. Gastroenterol. Hepatol.* 3:1198–1206.
27. Ingle, S. B., Y. G. Patle, H. G. Murdeshwar, et al. 2011. A case of early eosinophilic gastroenteritis with dramatic response to steroids. *J. Crohn's Colitis* 5:71–72.
28. Kim, N. I., Y. J. Jo, M. H. Song, et al. 2004. Clinical features of eosinophilic gastroenteritis. *Korean J. Gastroenterol.* 44:217–223.
29. Chen, M. J., C. H. Chu, S. C. Lin, et al. 2003. Eosinophilic gastroenteritis: clinical experience with 15 patients. *World J. Gastroenterol.* 9:2813–2816.
30. Hsu, Y. Q., and C. Y. Lo. 1998. A case of eosinophilic gastroenteritis. *Hong Kong Med. J.* 4:226–228.