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Abstract: Feline herpesvirus type 1 (FHV-1) infection in domestic cats is most commonly manifested as an upper respiratory tract infection and is know as feline viral rhinotracheitis (FVR). Clinical signs include serous to mucopurulent ocular and nasal discharge, conjunctivitis, and corneal and oral ulceration. Ulcerative skin lesions attributable to FHV-1 in domestic cats have not been commonly reported. The possibility of genetic susceptibility to viral disease has been proposed for cheetahs on the basis of the low genetic diversity in the species. Morbidity and mortality in cheetahs with feline coronavirus infections is much higher than reported in domestic felids. A similar increased sensivity to FHV-1 may result in a higher incidence of cutaneous ulcers in cheetahs than in domestic cats.

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Persistent cutaneous ulcers associated with feline herpesvirus type 1 infection in a cheetah

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Feline herpesvirus type 1 (FHV-1) infection in domestic cats is most commonly manifested as an upper respiratory tract infection and is known as feline viral rhinotracheitis (FVR). Clinical signs include serous to mucopurulent ocular and nasal discharges, conjunctivitis, and corneal and oral ulceration.[1] Ulcerative skin lesions attributable to FHV-1 in domestic cats have not been commonly reported.[2,3] Feline viral rhinotracheitis has been described in clouded leopards,[4] cheetahs, jaguars, African and Asian leopards, mountain lions, and fishing cats.[5] Isolation of FHV-1 from cutaneous ulcers in 2 cheetah littermates has been reported.[6]

Two cheetah littermates (1 male, 1 female) were born to a 4-year-old primiparous female, which had been vaccinated annually with a killed herpesvirus, calicivirus, and panleukopenia virus vaccine.[a] The cubs were dam-reared in an outdoor grass yard and vaccinated when they were 3, 6, 9, and 12 weeks old, with the same product as used in the dam.[a] A mild serous ocular discharge began in both cubs when they were 3 weeks old. Conjunctival scrapings and ocular swab specimens tested by indirect fluorescent antibody technique were negative for evidence of chlamydia and FHV-1. Ocular swab specimens taken for virus (Crandell-Rees feline kidney cell culture) and chlamydia isolation (McCoy cell culture) also were negative. Neither FeLV antigen nor FIV antibodies were detected in serum samples. Both cubs were treated topically once daily with an ophthalmic antibiotic ointment.[b] When they were 1 month old, both cubs developed bilateral corneal ulcers, and the treatment was switched to subconjunctival administration of chloramphenicol[c] (20 mg/eye, q 72 h). No additional samples were submitted for culture.

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[a]Fel-O-Vax PCT, Fort Dodge Laboratories, Fort Dodge, Iowa.

[b]Acromycin ophthalmic, Lederle Laboratories, Wayne, NJ.

[c]Chloromycetin sodium succinate, Parke-Davis, Morris Plains, NJ.

Figure 1—Raised, circumscribed plaque-like lesions on the palpebral margins, lateral aspect of the muzzle, and chin of a cheetah cub with feline herpesvirus type 1 infection.

When he was 2 months old, the male cub was removed from the dam because he had a nonhealing and infected puncture wound on the nape of the neck. The puncture wound healed without complication after debridement and suturing. The corneal ulcers as well as the conjunctivitis resolved with continued treatment.

The female cub remained with the dam until she was 3 months old. At that age, she developed raised plaque-like lesions on the upper and lower palpebral margins (Fig 1). By the time the cub was 4 months old, the conjunctivitis and corneal ulcers had resolved with treatment. The plaque-like lesions regressed and were replaced by multiple cutaneous ulcers ranging from 1 to 10 mm in diameter on the lateral muzzle and chin, and ulcers also appeared on the tail tip and cranial aspect of the right forelimb. The oral mucosa remained uninvolved. Punch biopsy specimens from representative lesions were taken after surgical preparation of the site. Histologic examination revealed deep ulceration of the epidermis with a marked suppurative inflammatory cell infiltrate, a plasma cell infiltrate in the underlying dermis, and pseudoepitheliomatous hyperplasia of the ulcer borders. Numerous Cowdry type A intranuclear inclusions, typical of herpesviruses, were observed in islands

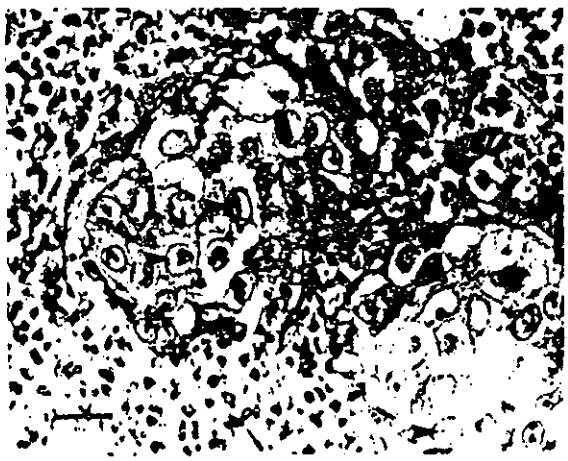

Figure 2—Photomicrograph of epidermal cells containing herpetic intranuclear inclusions (arrowheads) surrounded by suppurative inflammation. H&E stain; bar = 10 μm.

of epithelial cells within the area of suppuration (Fig 2). Transmission electron microscopy confirmed the presence of herpesvirus particles in the biopsy specimens. Virus isolation attempts by use of Crandell-Rees feline kidney cells yielded a herpesvirus from biopsy specimens. The isolate was identified as FHV-1 by a serum neutralization test and restriction endonuclease analysis of viral DNA.[6] The ulcers persisted and enlarged up to 5 cm in diameter. Oral treatment with acyclovir (10 mg/kg of body weight, q 12 h for 6 weeks) was begun when the cub was 6 months old. Mild but transient improvement was seen in the cutaneous ulcers, with decreased exudation and crusting of lesions. Results of repeated CBC and serum biochemical analyses done before and throughout acyclovir treatment were normal except for persistent eosinophilia (15 to 52%) attributed in part to ascariasis as diagnosed by fecal flotation. Because herpesvirus was isolated from the biopsy specimens taken after the completion of acyclovir treatment, persistence of the infection was confirmed.

Clinical disease caused by FHV-1 in exotic felids is similar to that in domestic cats. Onset of clinical signs at 1 month of age may be associated with loss of passive maternal immunity from the dam. Passive immunity is protective to domestic kittens until they are 5 to 8 weeks old.[7] To our knowledge, cutaneous ulcers associated with FHV-1 have not been reported in any other exotic species. Early ulcers around the eyes may be related to direct infection from ocular exudate. Lesions elsewhere are at sites accessible to normal grooming (forelimb, thigh, tail) or associated with play-derived wounds. This suggests local infection of traumatized skin with virus-containing oral secretions. The existence of a latent carrier state is probable. The dam of the cubs of this report had FHV-1 isolated from ocular secretions when she was 1 year old (1984). At the time that the litter was born (1987), the dam was clinically normal, however, she is presumed to be the source of the infection. Similar oculonasal discharges have developed in 5 of 6 litters (20 of 22 cubs) of cheetahs born at the St. Louis Zoo between 1975 and 1988. The fact that the virus was isolated from ulcers on our cub when it was 7½ months old also demonstrates the persistence and latency of the herpesvirus infection.

The lesions on the male cub resolved once he was removed from the queen, whereas the lesion of the female cub, which remained with the queen, persisted and increased in severity. The continued exposure to the source of the virus (the dam) may have contributed to the persistence and severity of the infection in this cub. Two subsequent litters (3 cubs total) from this queen were removed when they were 5 days old and were hand-raised. None of these cubs developed clinical signs of herpesvirus infection or cutaneous ulcers, suggesting that extended exposure is necessary for development of persistent infection.

The possibility of genetic susceptibility to viral disease has been proposed for cheetahs[8] on the basis of the low genetic diversity in the species.[9] Morbidity and mortality in cheetahs with feline coronavirus infections (feline infectious peritonitis) is much higher than reported in domestic felids.[10] A similar increased sensitivity to FHV-1 may result in a higher incidence of cutaneous ulcers in cheetahs than in domestic cats.

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