Erythema Multiforme in a Ferret (*Mustela putorius furo*)

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KEYWORDS

• Ferret • Erythema multiforme • Hypersensitivity • Immune disease
• Dermatopathology • Adrenal disease • Hyperadrenocorticism

KEY POINTS

- Erythema multiforme is an uncommon skin disease that most dermatopathologists think is a host-specific hypersensitivity reaction induced by various antigens that alter keratinocytes, making them targets of an aberrant immune response.
- In veterinary medicine, erythema multiforme has been more commonly reported in dogs than in cats, and is typically associated with drug administration (eg, ampicillin, cephalexin, trimethoprim-sulfonamide, griseofulvin, acepromazine, enrofloxacin, and lincomycin), although many cases remain idiopathic.
- The onset of clinical signs is typically acute with erythematous macules and papules developing rapidly and often becoming annular or serpiginous as they coalesce in partially symmetric patterns.
- On skin biopsy histology, individual cell necrosis of keratinocytes, or apoptosis, and lymphocyte satellitosis are the most characteristic histologic lesions of erythema multiforme.
- Adrenal disease should be considered as a primary underlying cause of erythema multiforme in the ferret.

INTRODUCTION

Erythema multiforme (EM) is an uncommon skin disease that most dermatopathologists think is a host-specific hypersensitivity reaction induced by various antigens that alter keratinocytes, making them targets of an aberrant immune response. Reported in humans and various animal species, including the dog, cat, and horse,[1] it is currently thought that the process of cell death occurring in EM results from an initiation of a death program through cell-mediated or unknown factors.[2] It has been documented that the cell-mediated response in EM has a Th1 cytokine pattern.[3,4] The T cell–mediated response is directed at keratinocytes that may express antigens

Disclosures: The author has nothing to disclose.
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Vet Clin Exot Anim 16 (2013) 599–609
http://dx.doi.org/10.1016/j.cvex.2013.05.001 **vetexotic.theclinics.com**
1094-9194/13/$ – see front matter

in a novel way because of drug administration, infection, or neoplasia resulting in apoptosis (single-cell necrosis) of keratinocytes.[3] The more severe the clinical presentation of EM, the more likely it is to be associated with an adverse drug reaction.[3,5]

Among animals, EM has been more commonly reported in dogs than in cats, but in both species the disease is:

- Most commonly associated with drug administration; ampicillin, cephalexin, trimethoprim-sulfonamide, griseofulvin, acepromazine, enrofloxacin, and lincomycin all have been reported as initiating factors.

Other less common initiators include:

- Neoplasia[2]
- Bacterial infection[6]
- Fungal infection[7]
- Viral infection[8]
- Idiopathic[6]
- Insecticidal dips[9]
- Food hypersensitivities[10]

Breed or sex predilection has not been noted for any of the forms of EM.[2]

Lesions of EM usually develop after 7 to 18 days of exposure to the causative agent,[6,11] although lesions may develop as soon as day 1 after exposure if the animal has been previously exposed.[5,6] The ferret reported here developed lesions 12 weeks after having undergone surgery for gastric trichobezoars and partial resection of a grossly enlarged right adrenal gland, during which time it was exposed to various drugs including dexmedetomidine, burtorphanol, isoflurane, famotidine, cefazolin, and meloxicam. Polydioxanone suture (PDS) was used during surgery. Because of the length of time between drug exposure and onset of clinical signs, it is unlikely that drug administration was responsible for the EM in this ferret.

CLINICAL HISTORY AND PHYSICAL EXAMINATION FINDINGS

A 5-year-old neutered male with a body weight of 1.2 kg initially presented with a sudden onset of scabbing and erythema of the ventral inguinal area. The skin of the dorsum was not affected and there were no dermal signs typical of ferret hyperadrenocorticism. On initial presentation the body temperature was normal and the ferret was bright and alert. Pruritus was not reported, and external parasites were not seen on physical examination or multiple skin scrapings. On gross examination, several papules were seen with varying degrees of scale and scabbing. Dermal tape preparation cytology showed cornified epithelial cells and occasional cocci bacteria. Because of the close resemblance of the ferret's gross dermal lesions to those of epidermal collarettes commonly associated with superficial pyoderma typical of the canine, the ferret was placed on cefadroxil (Cefadrops, Fort Dodge Animal Health, Fort Dodge, IA) at 10 mg/kg every 12 hours with instructions to reevaluate clinical signs in 1 week.

CLINICAL COURSE

A medical progress examination 1 week later revealed a worsening of the dermatitis. The dermatitis, manifested as erythematous papules and scabs, had progressed to involve the inguinal (**Fig. 1**A) and axillary regions (see **Fig. 1**B) and ventromedial aspect of the limbs. In addition, all four feet showed foot pad thickening with hyperkeratosis as well as dermal inflammation between the pads (**Fig. 2**A). The ear pinna were