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Pathology in Practice

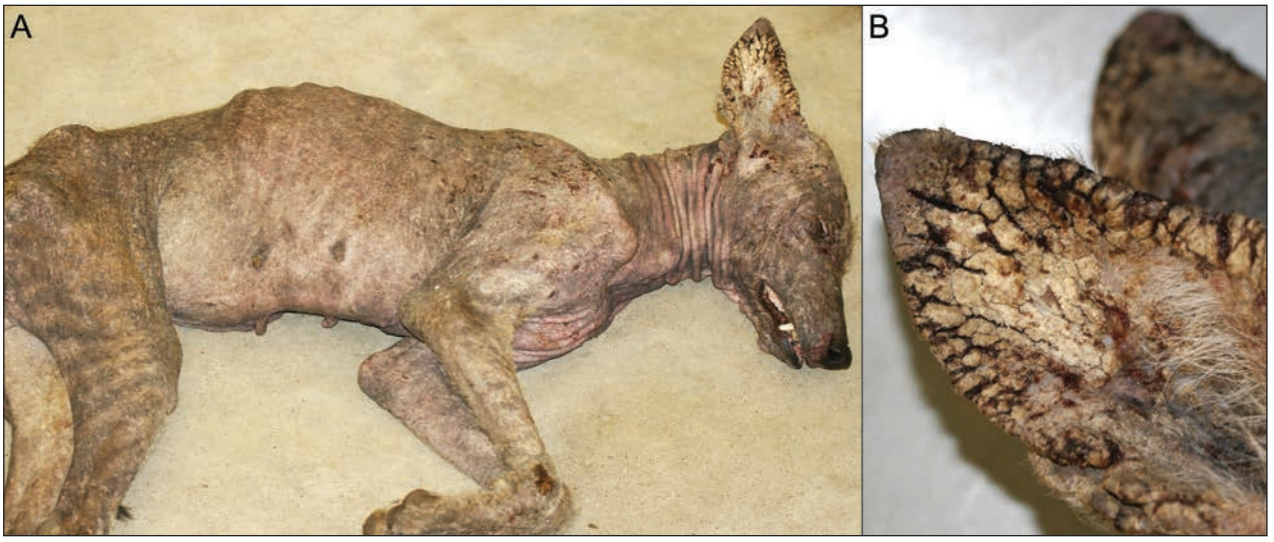

Figure 1—Photographs of the body (A) and pinnae (B) of a wild canid with severe generalized alopecia and hyperkeratosis that was shot because it was in poor physical condition and acting aggressively. A—Notice the mottled skin with severe muscle atrophy. B—The distal aspects of both pinnae were thickened with a crust up to 3 mm in thickness.

History

A wild canid of unknown species was found in a storeroom at a horse racing track. The animal was acting aggressively, and when law enforcement officials arrived at the scene, the animal was shot and killed because of its temperament and physical condition. The wild canid was reported as a possible chupacabra, and the story made national media headlines.

Clinical and Gross Findings

The body of the canid was sent to the Texas A&M Veterinary Medical Teaching Hospital. On examination, it was severely emaciated with a body condition score of 1 of 9. Several teeth were broken, and the canid was diffusely hairless with severe hyperkeratosis, especially on the pinnae (**Figure 1**). The skin was diffusely thickened and mottled gray and tan with dark gray striping on the hind limbs (lichenification and hyperpigmentation). The distal aspects of both pinnae were thickened with crusts; crusts were up to 3 mm in thickness. White to tan crusts and flaking were present along the dorsal midline in the lumbosacral area. Thick cream to white exudate was detected within the external ear canals bilaterally (otitis externa). Additional gross findings included bony proliferations on the right dorsal aspects of ribs 4 through 12 (previous fracture sites); dozens of 5-mm-long and 1-mm-diameter, tightly coiled, pink roundworms (*Physaloptera* sp) in the distal portion of the esophagus, stomach, and proximal portion of the jejunum; 4 yellow flatworms (tapeworms), with segmented bodies ranging from 19 to 23 cm long, in the distal portion of the jejunum; and ten to fifteen 5.5-cm-long white roundworms with thin anterior ends (*Trichuris vulpis*), also in the distal portion of the jejunum. Peripheral lymph nodes were diffusely enlarged. Various tissue samples were obtained for histologic examination.

Formulate differential diagnoses from the history, clinical findings, and Figure 1—then turn the page →

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Histologic Findings

The histopathologic changes in the skin were most severe on the pinnae (**Figures 2 and 3**). The epidermis of the pinnae had severe irregular epidermal hyperplasia and parakeratotic hyperkeratosis with a thick crust composed of keratin, serum protein, degenerate neutrophils, and erythrocytes. Numerous mites were embedded within the crust as well as mite eggs and large colonies of cocci. The epidermis had multifocal areas of erosion and ulceration. The superficial to mid dermis had a moderate inflammatory infiltrate composed of lymphocytes, plasma cells, eosinophils, and fewer macrophages, mast cells, and neutrophils. In sections of nonpinnal skin, mites were not evident, and the histopathologic changes included moderate irregular epidermal hyperplasia, mild orthokeratotic hyperkeratosis, hyperpigmentation and pigmentary incontinence, follicular atrophy, surface yeast, and much milder dermal inflammation and serocellular crusting than observed in the pinnae. The lymph nodes had severe, diffuse, follicular hyperplasia with medullary plasmacytosis, sinus histiocytosis, and sinus eosinophilia. Other findings included splenic plasmacytosis, mild lymphoplasmacytic interstitial nephritis, and chronic testicular atrophy. Full cross sections of the brainstem and cerebellum were submitted for rabies virus testing, which was done by means of a fluorescent antibody method. Results of rabies virus testing were negative.

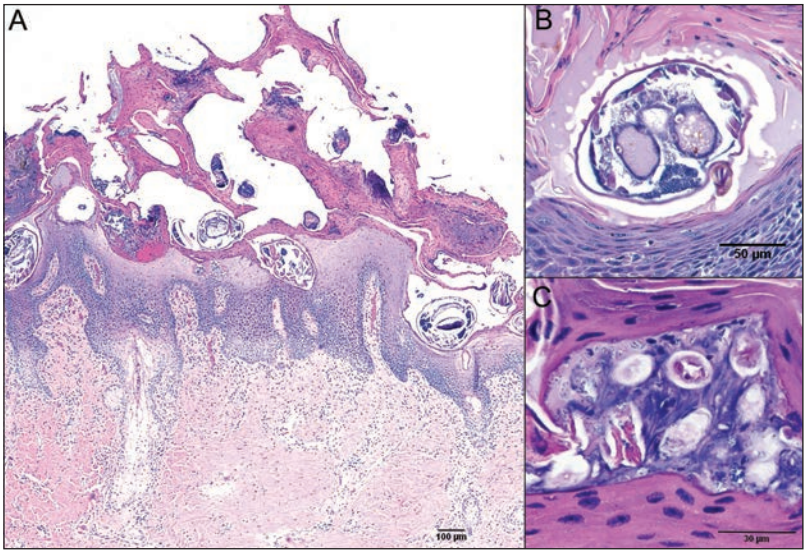

Figure 2—Photomicrographs of a section of skin from a pinna obtained from the wild canid in Figure 1. A—Notice the diffuse dermatitis with irregular epidermal hyperplasia, hyperkeratosis, thick serocellular crusts, and numerous mites. H&E stain; bar = 100 µm. B—Higher-magnification view of a mite from the same section. H&E stain; bar = 50 µm. C—Higher-magnification view revealing multiple eggs of *Sarcoptes scabiei* laid in a burrowing channel. H&E stain; bar = 30 µm.

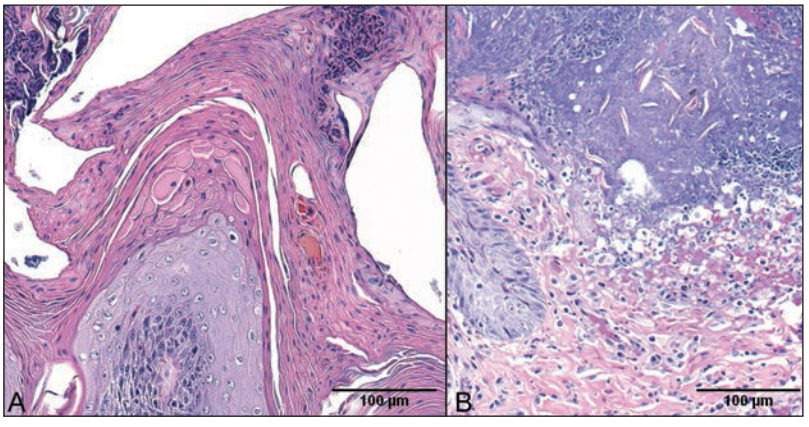

Figure 3—Photomicrographs of another section of skin from a pinna obtained from the wild canid in Figure 1. The skin has evidence of epidermal hyperplasia, severe parakeratosis, and serocellular crusting (A) and ulceration (B). H&E stain; bar = 100 µm.

Genetic Testing

To identify the species of this canid, tissue DNA was isolated by DNA purification[d] and underwent PCR amplification. Products from PCR amplification were purified[b] and cycle sequenced.[c] Samples were then analyzed on a genetic analyzer[a] with the resultant sequences edited and aligned with genomics software.[e] Phylogenetic relationships among related sequences were established and compared against all sequences on GenBank. Final analysis revealed that the DNA sequence from the sample was nearly identical to, and most closely related to, coyote (*Canis latrans*).

Morphologic Diagnosis and Case Summary

Morphologic diagnosis: severe, chronic, diffuse, proliferative, and pustular dermatitis with intralesional mites, bacteria, and yeast.

Case summary: sarcoptic mange in a coyote.

Comments

Sarcoptic mange is a contagious disease of mammals caused by the ectoparasite *Sarcoptes scabiei*.[1] Sarcoptic mange affects hosts ranging from humans to dogs, pigs, foxes, dromedary camels, chamois, lynxes, and wombats on continents across the world.[2] Sarcoptic mange is enzootic in certain wild canid populations of North America with multiple reports of affected coyotes, timber wolves, red wolves, red foxes, and coyote–red wolf hybrids.[3] Farmers often express concern that the disease will spread to their livestock because coyotes with severe mange have been found seeking shelter in and around farm buildings during periods of colder weather.[4]

The clinical signs and pathological abnormalities of sarcoptic mange can vary markedly, not only among species but also among affected individuals of the same species.[5] In dogs, focal lesions on various areas of the body (ie, pinnae, limbs, and flank and lumbar regions) are common but have rarely been described for wild canids, either because those animals are often examined after lesions have already generalized or because sys-

temic detailed records have not been kept.[5] In many species, *S scabiei* infection is often characterized by intense pruritus accompanied by alopecia, hyperkeratosis, seborrhea, scabs, lichenification, and ulcerations.[6] However, in wild canid populations, most affected animals develop mild to severe encrustations with or without alopecia, although a small percentage develop thickened hairless skin without crusts.[5] Pruritus is often thought to be minimal in certain cases because of the lack of visible scratch wounds.[5] Given the negative results of rabies testing for the coyote described in this report, its displayed aggressiveness was presumably a consequence of fear or physical discomfort.

The coyote of the present report had severe sarcoptic mange. On the basis of scoring systems designed to classify the distribution and severity of sarcoptic mange lesions in red foxes, the coyote would have a class III type B designation with a generalized distribution of skin lesions involving thick crusts, alopecia, visible skin thickening, lymphocytes, mast cells, eosinophils, mites, bacteria, lymph node enlargement, and severe emaciation.[3,5] However, because the crusts and mites were not a major component of the more generalized lesions, one could argue that this case shares characteristics with a class III type C designation, which is considered the healing stage of sarcoptic mange.[5] The observed lymph node changes were considered to be a reactive process secondary to chronic antigenic stimulation from the primary dermatologic disease.

Differential diagnoses for *S scabiei* infection can include hypersensitivity (food or flea hypersensitivity, and atopy), *Malassezia* dermatitis, pyoderma, demodicosis, dermatophytosis, and contact dermatitis.[7] Mildly infested hosts usually do not have obvious signs of clinical disease; however, chronic and severe sarcoptic mange can cause visible deterioration of the host's physical condition, resulting in emaciation in any affected animal or reduced weight gain in growing animals, reduced milk production in cattle,[1] and even eventual death from secondary infections or hypothermia due to hair loss (particularly in wildlife species).[6] When severely affected by *S scabiei*, coyotes have even been reported to be listless, with a reduced fear of humans.[8]

Recovery from sarcoptic mange is not common in wild canids.[3] Domestic canids can be treated with systemic (selamectin, ivermectin, milbemycin oxime, or moxidectin) or topical (amitraz, fipronil spray, lime sulfur solution, or organophosphates) medications,[7] but all treatment protocols are time-consuming and rely on repeated administrations. Thus, such protocols are rarely possible to fully implement in most wildlife populations. In south Texas, the overall prevalence of sarcoptic mange in coyotes has been reported to be as high as 67%,[3] with mortality rates reported to be as high as 55%.[9] Given the reported mortality rate and the severity of disease in the coyote of the present report, its prognosis would have been guarded had treatment been feasible.

Given the severity of the skin lesions, it is understandable that there was confusion about the identity of the wild canid in this instance. The chupacabra myth appears to have first gained notoriety in Puerto Rico in 1995 and has since spread throughout the Americas.[10] The English translation of the word is goat sucker, and the name comes from the mythical animal's reported habit of attacking and drinking the blood of livestock, especially goats, although it has also been blamed for attacks on many other species.[10] Genetic testing in the case described in the present report confirmed the fact that the wild canid was a coyote.

a. ABI 3500xL Genetic Analyzer, Life Technologies, Grand Island, NY.
b. AMPure PCR Purification System, Agencourt Bioscience Corp, Beverly, Mass.
c. BigDye TerminatorCycle Sequencing Kit, version 3.1, Life Technologies, Grand Island, NY.
d. DNeasy Tissue Kit, QIAGEN Inc, Germantown, Md.
e. Geneious Pro, version 5.5, Biomatters Ltd, Auckland, New Zealand.

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3. Pence DB, Windberg LA, Pence BC, et al. The epizootiology and pathology of sarcoptic mange in coyotes, *Canis latrans*, from South Texas. *J Parasitol* 1983;69:1100–1115.
4. Samuel WM. Attempted experimental transfer of sarcoptic mange (*Sarcoptes scabiei*, Acarina: Sarcoptidae) among red fox, coyote, wolf and dog. *J Wildl Dis* 1981;17:343–347.
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6. Jimenez MD, Bangs EE, Sime C, et al. Sarcoptic mange found in wolves in the Rocky Mountains in western United States. *J Wildl Dis* 2010;46:1120–1125.
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10. Radford B. The goatsucker mystery. *Tracking the chupacabra: the vampire beast in fact, fiction, and folklore*. Alburquerque: University of New Mexico Press, 2011;3–11.