Mange Caused by a Novel *Micnemidocoptes* Mite in a Golden Eagle (*Aquila chrysaetos*)

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Abstract: A second-year, female golden eagle (*Aquila chrysaetos*) was live trapped in northern California because of severe feather loss and crusting of the skin on the head and legs. On physical examination, the bird was lethargic, dehydrated, and thin, with severe feather loss and diffuse hyperemia and crusting on the head, ventral wings, ventrum, dorsum, and pelvic limbs. Mites morphologically similar to *Micnemidocoptes derooi* were identified with scanning electron microscopy. The eagle was treated with ivermectin (0.4 mg/kg) once weekly for 7 weeks, as well as pyrethrin, meloxicam, ceftiofur crystalline free acid, and voriconazole. Although the eagle's condition improved, and live mites or eggs were not evident on skin scrapings at the time of completion of ivermectin treatment, evidence of dead mites and mite feces were present after the last dose of ivermectin. Two additional doses of ivermectin and 2 doses of topical selamectin (23 mg/kg) were administered 2 and 4 weeks apart, respectively. No mite eggs, feces, or adults were evident after treatment was completed. A second golden eagle found in the same region was also affected with this mite but died soon after presentation. This is the first report, to our knowledge, of successful treatment, as well as treatment with selamectin, of mites consistent with *Micnemidocoptes* species in any raptorial species.

Key words: skin disease, *Micnemidocoptes derooi*, ectoparasite, parasite, mite, raptor, bird, avian, golden eagle, *Aquila chrysaetos*

Clinical Report

A second-year, female (based on morphologic criteria),[1] 4.4-kg golden eagle (*Aquila chrysaetos*) was presented to the Veterinary Medical Teaching Hospital at the University of California, Davis, USA, for evaluation after being live trapped by a biologist. The biologist had observed the bird over a 3-week period, during which time it had occupied the northwest portion of the Altamont Pass Wind Resource Area, located about 7 miles north of Livermore, Alameda County, CA, USA. The bird was identified based on gross dermatologic abnormalities, including feather loss and crusting on the head and legs. When first observed, it was able to fly reasonably well; however, its condition deteriorated to the point where a decision was made to

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live-trap it. Within a few weeks, an additional 1-year-old, 2.7-kg, subadult golden eagle was found grounded in a nearby county with similar skin lesions and debilitation and was admitted to the Society for the Prevention of Cruelty to Animals for Monterey County, Salinas, CA, USA. This eagle had severe dehydration, weak and raspy respirations, and feather loss and crusting, and it was euthanatized because of poor prognosis and submitted for necropsy.[2] Lastly, a third eagle was found grounded in King City, CA, USA, and was also admitted to the Society for the Prevention of Cruelty to Animals for Monterey County, Salinas, CA, USA. This subadult male weighed 2.7 kg and presented with severe dehydration, weak and raspy respiration, and poor feathers. It had lesions similar to the previous eagles, and mites were present on skin scrapings. This eagle was euthanatized because of poor prognosis; however, it was not submitted for necropsy.

On presentation to the Veterinary Medical Teaching Hospital, the bird was lethargic but responsive and in thin body condition (body condition score 3/9). It was estimated to be 5% dehydrated based on brachial vein diameter and refill and on thickened saliva; however, other anatomic sites were difficult to evaluate because of the thickened skin of the eyelids and keel. On physical examination, feather loss was severe with diffuse hyperemia, lichenification, and crusting. The proliferative lesions appeared to be centered around feather follicles, and the nonfeathered skin appeared unaffected. On the head, the lesions began at, but did not involve, the cere and extended caudally to the level of the thoracic inlet and shoulders (Fig 1A). Additional affected areas were the caudal dorsum in the region of the pelvis, the ventrum extending caudally to the tail (Fig 1B), the ventral aspect of both wings extending from the distal brachium to the proximal antebrachium, and the hind limbs extending from the proximal tibiotarsus to the distal tarsometatarsus (Fig 1C). A severe feather lice infestation was also identified. The remainder of the physical examination was unremarkable with the exception of a grade II–III/VI systolic heart murmur with a regular rhythm that resolved once the patient was hydrated.

Initial diagnostic testing consisted of blood collection for a complete blood cell count (CBC), a plasma biochemical analysis, and measurement of whole blood lead concentration. A mild leukocytosis (26.6×10^3 cells/μL; reference mean, $24.31 \pm 1.97 \times 10^3$ cells/μL^3) was present with a marked heterophilia (20.9×10^3 cells/μL; reference mean, $4.4 \pm 0.22 \times 10^3$ cells/μL^3), a marked lymphopenia (1.3×10^3 cells/μL; reference mean, $16.81 \pm 0.65 \times 10^3$ cells/μL^3), and a moderate monocytosis (2.39×10^3 cells/μL; reference mean, $0.99 \pm 0.19 \times 10^3$ cells/μL^3). Results of the plasma biochemical panel were unremarkable with the exception of a moderate hyperuricemia (18.4 mg/dL; reference mean, 7.7 ± 1.6 mg/dL^3). Differentials for the hyperuricemia included dehydration, postprandial increase, and renal insufficiency. Lead was not detected within the whole blood sample.

Additionally, skin scrapings, fecal parasitologic examination, and full body radiographs were performed. Mites consistent with Knemidokoptidae were identified by light microscopy in skin scraping materials of the head, neck, coelom, and hind limbs (Fig 2). A fecal flotation was positive for *Capillaria* species, *Isospora* species, and *Strongyloides* species in moderate numbers (2–3 per ×10 field). The eagle was sedated with midazolam (1 mg/kg IM) and butorphanol (1 mg/kg IM; Torbugesic, Fort Dodge Animal Health, Fort Dodge, IA, USA) for whole-body right lateral and ventrodorsal radiographs. No unusual findings were visible on radiographs. Morphologic characteristics of the mites from this eagle and the deceased eagle were further evaluated with scanning electron microscopy (SEM).[2] The mites from both eagles were morphologically similar to *Micnemidocoptes derooi* based on dorsal striation pattern, size of the propodosoma, location of the anus dorsoterminally, and the positioning of the anterior legs dorsally and the posterior legs ventrally on SEM.[4] Frozen skin tissue from the deceased eagle and crusts from this eagle were submitted for polymerase chain reaction and DNA sequencing. The DNA was extracted from 2 pools of approximately 5–10 mites with a Qiagen Tissue Kit (Qiagen, Valencia, CA, USA). Polymerase chain reaction amplification of a fragment of the cytochrome oxidase subunit I gene was performed,[5] modified to include 12.5 μL GoTaq Green Master Mix (Promega, Madison, WI, USA), 1.0 M of each primer, 2.5 μL of water and 5 μL of DNA. A Qiagen gel extraction kit was used to clean 715 base pair bands before DNA sequencing (Davis Sequencing, Davis, CA, USA). The DNA sequences were BLAST-searched against GenBank (NCBI, 94 Bethesda, MD, USA). There was 100% sequence homology between the pools and 88% homology to *Knemidokoptes jamaicensis* (GenBank accession JQ037816.1), the only Knemidokoptidae accession in the database. Samples from both eagles were

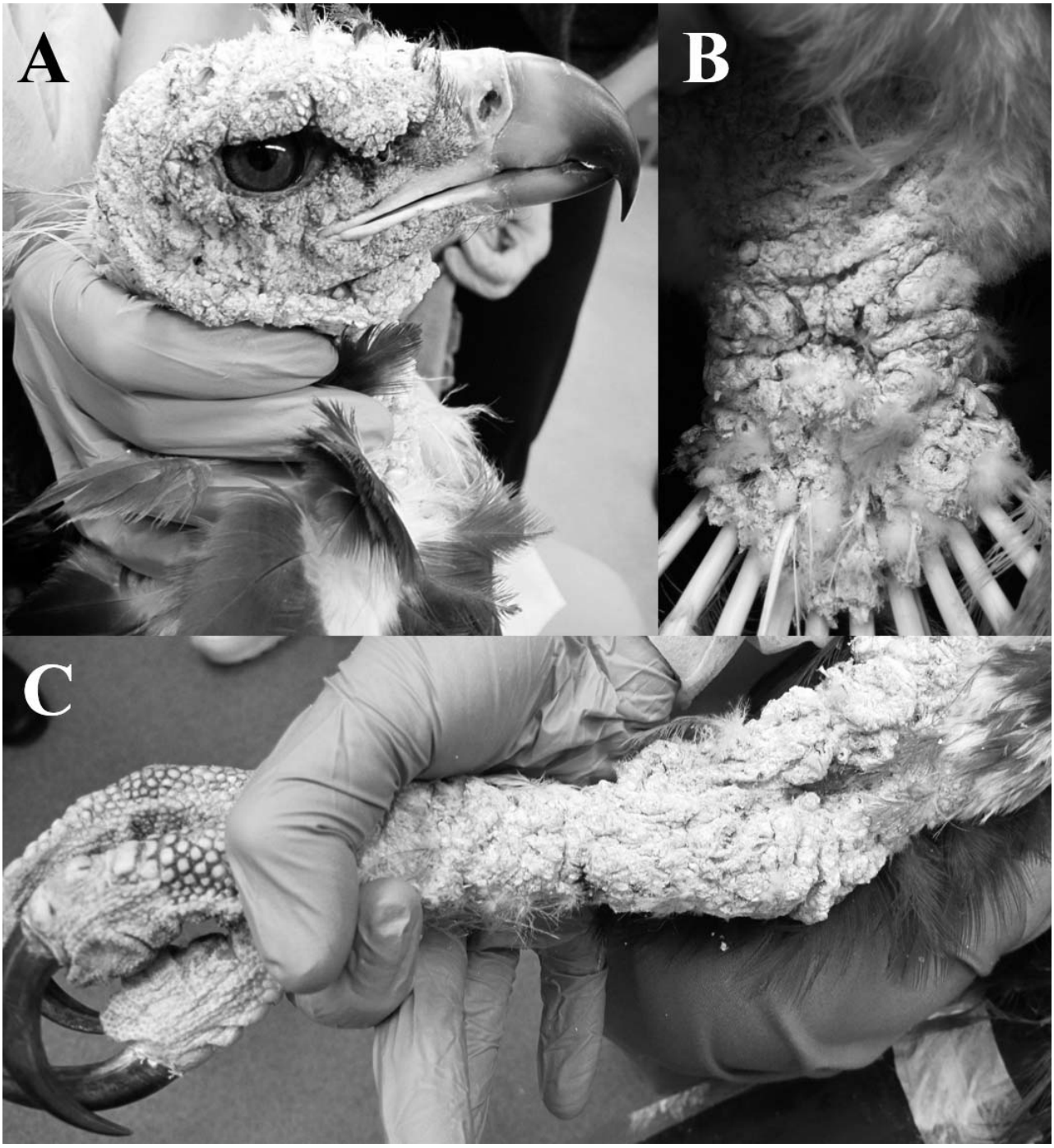

Figure 1. Head, ventrum, and hind limb of a golden eagle affected by *Micnemidocoptes derooi*–like mites. Lichenification, crusting, and feather loss are present. Note that the cere, beak, and digits are unaffected, which differs from lesion distribution reported for some *Knemidokoptes* species infections in other species of birds.

submitted to GenBank (GenBank accession KJ787640).[2]

Fluids (lactated Ringer's solution, 60 mL/kg SC per day for 2 days, then increased to 100 mL/kg SC per day for 2 days), ceftiofur crystalline free acid (20 mg/kg IM q3; Excede, swine injectable 100 mg/mL, Zoetis, Madison, NJ, USA), tramadol (5 mg/kg PO q12h), meloxicam (0.5 mg/kg PO q12h), ivermectin (0.4 mg/kg IM q7d for 3 treatments, then PO q7d for 4 treatments; Ivomec, Merial Limited, Duluth, GA, USA), and a topical pyrethrin flea spray (applied once) were adminis-

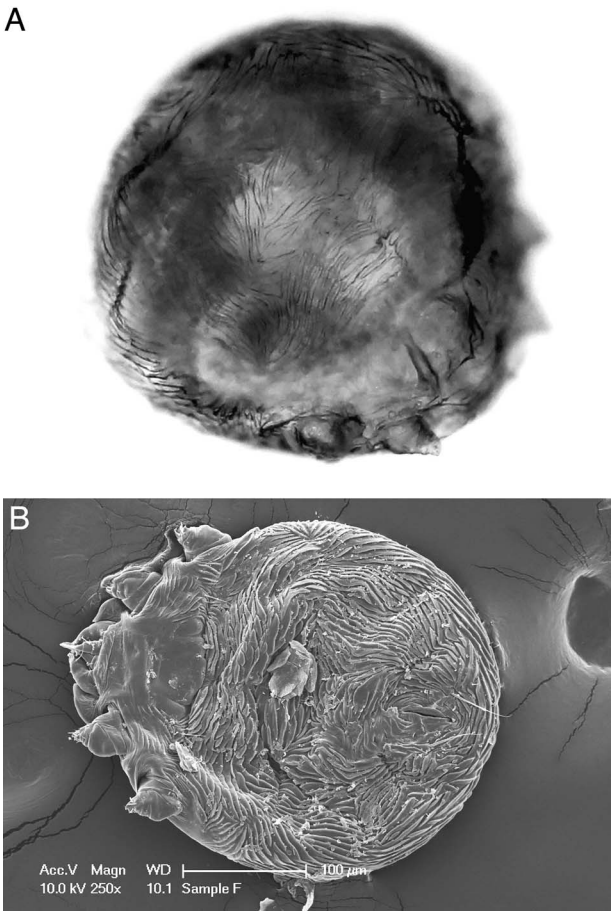

Figure 2. (A) Light microscopy and (B) scanning electron microscopy (SEM) images of *Micnemidocoptes derooi*–like mites from a golden eagle from northern California, USA. Morphologic characteristics of the mites from both eagles in this report are consistent with *M derooi* on SEM, including dorsal striation pattern, size of the propodosoma, location of the anus dorsoterminally, and the positioning of the anterior legs dorsally and posterior legs ventrally. Mites collected by skin scraping in mineral oil (×100, light microscopy).

tered. Because of the heavy endoparasite infestation and risk of developing opportunistic fungal infection while in captivity, fenbendazole (20 mg/kg PO q24h for 5 days; Panacur, Intervet Inc, Millsboro, DE, USA) and voriconazole (11 mg/kg PO q24h; Mylan Pharmaceuticals Inc, Morgantown, WV, USA), respectively, were also administered.

During the next 3 weeks, the bird's skin abnormalities gradually improved and it had no evidence of feather lice or endoparasites after treatment, based on visual examination and repeat fecal flotation, respectively. A CBC was repeated 24 days after presentation to evaluate response to antibiotic, antiparasitic, and antifungal therapy. The heterophilia (17×10^3 cells/μL), lymphopenia (5.25×10^3 cells/μL), and monocytosis (1.75×10^3 cells/μL) had improved. Differentials for the continued inflammation included ongoing mite infestation and reactions to repeated intramuscular injections (ceftiofur crystalline free acid and ivermectin). The antibiotics were discontinued, and the route of the ivermectin was changed to oral administration.

On day 44 after presentation, the ivermectin treatment was discontinued; a total of 7 doses, each a week apart, had been administered. The oral voriconazole was continued. On day 72 after presentation, a CBC was repeated, results of which were unremarkable. A repeat skin scraping of the head, neck, and coelom was performed, and mite eggs were visible; however, no appreciable live adults were present. Because of the presence of mite eggs, and the potential for live adults, ivermectin was reinstated (0.4 mg/kg PO q14d for 2 treatments).

On day 84 after presentation, a skin scraping was performed of the head, neck, and coelom to assess response to ivermectin therapy. Dead adult mites were seen; however, no eggs were found. The ivermectin therapy was again discontinued, and a repeat skin scraping to assess for presence of mites, eggs, or mite fecal material was planned for 2 weeks later.

On day 98 after presentation, dead adult mites and mite feces were present on the skin scraping of the head, neck, and coelom. Because of the lack of complete resolution with ivermectin therapy, topical selamectin (23 mg/kg topically q4wk for 2 treatments; Revolution, Pfizer, New York, NY, USA) was initiated. The bird was regularly monitored for any adverse effects, including neurologic abnormalities; no abnormalities were identified throughout the course of treatment.

On day 147 after presentation, a skin scraping was performed of the head, neck, and coelom to assess response to selamectin and showed the presence of 3 dead, adult mites; however, the area sampled was a crust within a proliferative follicular region. Because of the lack of evidence of live mites and the marked improvement that had been made by the patient, no further treatment was instituted.

On day 226 after presentation, a skin scraping of the head, neck, and coelom, a CBC, and a plasma biochemical panel were performed. No mites, eggs, or mite fecal material was detected, and all clinical signs were considered resolved. Results of the CBC and plasma biochemical panel were unremarkable with the exception of a mildly increased aspartate aminotransferase (AST) concentration (501 U/L;

reference mean, 293 ± 19 U/L[3]). The uric acid concentration was mildly decreased (4.4 mg/dL). Bile acid concentration was measured (12 μmol/L) and interpreted to be unremarkable, but to our knowledge, bile acid reference intervals for this species have not been reported. Because of the potential for hepatotoxicity associated with the chronic use of voriconazole, it was discontinued.[6]

On day 279 after presentation, the eagle was released back to the wild in the same geographic region where it was trapped (Fig 3). Approximately 1 week before release, the bird began to molt over the head and pectoral region. Before release, a high-frequency global positioning system mobile communications satellite transmitter (Cellular Tracking Technologies, LLC; http://celltracktech.com) was fitted and placed. Approximately 60 days after release, tracking of the eagle showed a normal flight history, suggesting the bird was acclimating well back into the wild and was near its capture location.

Figure 3. Golden eagle previously affected with *Micnemidocoptes derooi*–like mites after treatment with ivermectin and selamectin, immediately before release. Note that the eagle still has evidence of molting feathers on the head and neck.

Discussion

This is the first case report, to our knowledge, to document the successful treatment of mange associated with Knemidokoptes or Micnemidocoptes sp. mites in a golden eagle. There were no appreciable adverse effects after selamectin treatment, and the eagle was successfully released after 279 days in captivity.

The Knemidocoptidae are submacroscopic ectoparasites that burrow into the skin of birds.[7] Several *Knemidokoptes* species have been identified in birds, most commonly, *Knemidokoptes pilae*, which affects legs and face of psittacine birds, *K jamaicensis*, which affects legs and beak of passerine birds, and *Knemidokoptes mutans*, which causes feather breakage and loss, typically in chickens.[8] Knemidokoptic mange is reportedly most prevalent in budgerigars (*Melopsittacus undulatus*) and passerines (Passeriformes),[9] but this may partially reflect a bias away from observation of free-ranging wild birds. Pathogenesis resembles that of sarcoptid mites in mammals, where mechanical trauma from mite burrowing and excretory and secretory products from the mites contribute to skin disease.[7] The entire life cycle of the mite is reported to be spent on the host; therefore, transmission requires direct contact between birds,[8] but fomites as a source of infection in confined quarters, such as the nest, have not been ruled out. Clinical disease commonly accompanies some degree of immune compromise secondary to stress, malnutrition, or immunosuppression; a genetic predisposition has been suggested.[9]

Previous reports of knemidokoptic mange in raptors are scarce and include *K mutans* in a great-horned owl (*Bubo virginianus*)[10] and unidentified *Knemidokoptes* species in a snowy owl (*Nyctea scandiaca*),[11] a Swainson's hawk (*Buteo swainsoni*),[12] and in accipiters in Great Britain.[11] To our knowledge, no Micnemidocoptes mite has been found on a raptor previously and the congeneric, *M derooi*, was only described once in African palm swifts (*Cypsiurus parvus*).[4] Another golden eagle identified concurrently with this case was infested with the same mite, based on SEM and DNA testing.[2]

Knemidokoptidae typically have a predilection for the nonfeathered areas of the bird, with the face, bill, and cere or legs and feet (ie, "scaly face" and "scaly leg") being most commonly affected.[7] The mites burrow to the level of the stratum germativum on the face, legs, and feet but can also burrow into the feather follicles on the wings and thighs.[7] The mites can cause pronounced hyperkeratosis of the cere and adjacent tissue, as well as proliferation and inflammation of the beak.[9] Often, there is a powdered appearance to the beak and a raised honeycomb mass, either on the cere, eyelids, beak, feet, or other body location. With magnification, pinpoint tunnels where the mites live can be seen in the powdery masses.[9] More severe lesions include sloughing of the claws, loss of digits, or traumatic amputation of the entire foot.[13]

In this case, the feathered areas of the bird's body were exclusively affected, including the head, neck, legs, ventral wings, dorsum, and ventrum, whereas the beak, cere, and scaled areas of the hind limbs were spared. The anatomic lesions from the deceased eagle were very similar in appearance and distribution, and the mites from both eagles were confirmed by DNA sequencing to be the same knemidokoptic mite.[2] The lesion distribution reported for *M derooi* in African palm-swifts involved thickening of the surface epithelium at the lateral aspects of the internal surface of the bill near the commissures of the mouth.[4] It is unclear whether the more-unique lesion distribution in this case was caused by the severity of the infestation, whether the organism may have affected this host species differently, or whether this might be a closely related, but not previously described, mite. The mites affecting the feathered areas may have led to more severe clinical disease because of feather loss and loss of thermoregulatory abilities. Clinical signs associated with Knemidokoptidae commonly include pruritus; feather loss, especially adjacent to the nonfeathered affected areas; feather destructive behavior; self-mutilation; proliferative skin lesions; and digit necrosis.[14–17] In this case, the observed clinical signs were severe feather loss from the body with diffuse hyperemia, severe lichenification, and crusting.

Diagnosis of knemidokoptic infestation is typically based on either the demonstration of the mites with microscopy of skin scrapings, acetate tape preparations, or crusts of the lesions or by recognizing the small bore holes within the hyperkeratotic lesions.[12,14] When evaluated directly, the mites appear as tiny, white, immobile spheres.[7] Under the microscope, they can be recognized by their globoid shape and short legs that extend just beyond the body's lateral margins.[18] Scrapings and acetate tape preparations may be negative, even when mites are present.[9,12] Differential diagnoses include dermatomycoses, papillomavirus, fiber constriction (if located on the legs), and poxvirus.[14] In this case, initial diagnosis was made by evaluating a skin scraping microscopically. Mites were then evaluated more closely through the use of SEM and DNA sequence analysis. Even though morphologic characteristics were similar to *M derooi*, we are cautious in claiming that the mites we saw in the golden eagles are the same species as those identified in the palm swifts. There is very sparse coverage of DNA sequences from mites from this genus in general, and DNA is not available from the single prior report of this mite.

Treatment for knemidokoptic mange historically has involved the use of ivermectin orally, topically, or by injection.[9] In passerine birds, infestation often improves, but may not be eliminated, with ivermectin therapy, which may be because of the presence of secondary infections.[9] Plant oil, used as an adjunct topical treatment with ivermectin, has been suggested to suffocate the mites.[14] Other treatments include moxidectin and benzyl benzoate.[19,20] Selamectin has been used successfully to treat budgerigars affected with knemidokoptic mange at a dose of 23 mg/kg with a repeat treatment 3–4 weeks later.[21] Treatment in the case reported here included both ivermectin and selamectin. Although the clinical signs of the infestation improved dramatically with ivermectin treatment, the infestation did not appear to completely resolve in this patient; this may have been because of the overwhelming parasite infestation or underlying immunosuppression. After 2 treatments of selamectin, no evidence of mites was identified. The dose of 23 mg/kg topically was used empirically; therefore, the eagle was closely monitored for adverse effects associated with potential overdose and toxicosis, which could include diarrhea, vomiting, muscle tremors, anorexia, erythema, lethargy, salivation, tachypnea, pruritus, ataxia, and seizures.[22] There were no adverse effects observed after the 2 administered doses in this patient.

Epizootics of knemidokoptic mange have been reported in passerine populations in North America, Europe, Asia, and Africa, with many individuals dying or requiring euthanasia.[13] The impact of this disease is unknown because there are often other factors involved. The case reported here is 1 of 2 golden eagles definitively diagnosed over a short period, with a third bird showing strong suspicions of the same clinical mite syndrome. We (D.A.B., K.S.S.) have observed additional golden eagles with suspicious feather loss over the subsequent 1 year period in California, and others have reported them from Nevada, suggesting this may be an emerging infectious disease of golden eagles in these states. Further studies regarding the extent of this Micnemidocoptes species mite in the free-ranging raptor population, especially in golden eagles in California and Nevada, is warranted.

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