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Overall, the *Quick Reference Guide to Unique Pet Species* is very informative and user friendly. It can be recommended to veterinarians in clinical practice as well as those responsible for the health care of animals found in petting zoos, nature/wildlife centers, and other zoological collections; animals in laboratory settings; and classroom pets.

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1557-5063/12/2104-530.00  
http://dx.doi.org/10.1053/j.jepm.2012.09.013

ABSTRACTS

Galindo-Cardiel I, Opriessnig T, Molina L, et al: Outbreak of mortality in psittacine birds in a mixed-species aviary associated with *Erysipelothrix rhusiopathiae* infection. *Vet Path* 49(3):498-502, 2012

*Erysipelothrix rhusiopathiae* is a ubiquitous Gram-positive rod that can infect mammals, reptiles, amphibians, birds, and invertebrates. Among avian species, turkeys appear most susceptible to *Erysipelothrix* infection, which results in a septicaemia characterized by thromboembolism and bacterial endocarditis. Erysipelas, the disease caused by *E rhusiopathiae* in animals, has been rarely described in psittacine birds. This report describes the deaths of 2 rainbow lorikeets and an eclectus parrot that developed minimal to no overt clinical signs over a 23-day period. The birds were housed in a mixed-species aviary. One of the lorikeets and the eclectus parrot were submitted for necropsy examination. The lorikeet was found on the aviary floor ataxic and unable to fly shortly before death. The eclectus parrot had been removed from the aviary because of cage-mate aggression and was being treated with dexamethasone, vitamins, and meloxicam before dying 3 days later with no overt clinical signs of illness. Sections of all organs from the necropsied birds were stained with hematoxylin-eosin and Gram’s stain and submitted for histopathologic evaluation. Frozen sections of liver were submitted for bacterial culture, and a real-time polymerase chain reaction (PCR) assay and an immunohistochemistry assay with a polyclonal antiserum for *E rhusiopathiae* serotypes 1a, 1b, and 2 were performed on paraffin-embedded tissues. Histopathologic lesions common to both birds included multifocal thrombosis, multifocal bacterial thromboembolism, and necrotizing vasculitis and bacterial and fungal thromboembolism, and locally extensive rhabdomyonecrosis with bacterial embolism. Intravascular bacteria were Gram positive in both birds. In the lorikeet, liver samples yielded a pure growth of *E rhusiopathiae*, and *Escherichia coli* was isolated from the liver of the eclectus parrot. Immunohistochemistry for *E rhusiopathiae* antigen showed mild (eclectus parrot) to marked (lorikeet) positive staining of bacteria-like organisms, and real-time PCR detected *E rhusiopathiae* in paraffin-embedded tissues from the lorikeet but not from the eclectus parrot. *Erysipelothrix rhusiopathiae* can survive for up to 5 years in the environment and is transmitted horizontally by subclinical carriers, contaminated soil, mechanical vectors, and fomites. The source of infection in this case was unknown. Pathologic lesions reported in the lorikeet are similar to those reported in chickens experimentally infected with *E rhusiopathiae*—congestion, organomegaly (liver, spleen, and kidney), and vascular damage in several organs—which is highly suggestive of disseminated intravascular coagulation and shock.

Eshar D, Wyre NR, Brown DC: Urine specific gravity values in clinically healthy young pet ferrets (*Mustela furo*). *J Small Anim Pract* 53(2):115-119, 2012

Despite the advancements in ferret medicine, values for urine specific gravity (USG) have not been previously reported for this species. This prospective study included 69 intact client-owned ferrets (*Mustela furo*) (24 males and 45 females), which were presented for routine neutering. The ferrets were aged 4 to 12 months (median age, 7 months) and had no history of disease. Physical examination, a complete blood count, whole blood biochemistry, and urinalysis were performed as part of the preoperative medical evaluation. All test results were within reference ranges. No fluids were administered before urine collection, which was achieved by either free catch (voided or via manual expression) or cystocentesis with an ultrasound-guided technique.
after sedation. Urinalysis included dipstick testing, urine microscopy, and USG measurement immediately after sample collection. The USG was measured with a hand refractometer (ATAGO MASTER-SUR/Na Urine S.G. Clinical Refractometer; ATAGO, Tokyo, Japan) with increments of 0.001. The USG range for all ferrets in the study was 1.026 to 1.070. The USG for female ferrets (mean ± SD, 1.042 ± 0.008; minimum-maximum, 1.026-1.060) was significantly lower than that for male ferrets (mean ± SD, 1.051 ± 0.009; minimum-maximum, 1.034-1.070). There was no significant difference in USG measurements between urine samples collected by free catch or cystocentesis, and no significant association was found between USG and measurements of the packed cell volume and total protein. Dipstick analysis showed trace protein (< 0.3 g/L) in 80% of urine samples, with a prevalence of 92% in male ferrets compared with 73% in female ferrets, a difference not considered significant by the authors. Bilirubin (+1) was demonstrated by urine dipstick in 16% of samples with no significant difference between genders. Bilirubinuria in the absence of liver disease is considered normal in ferrets. Urine pH, as measured by urine dipstick, was most commonly 6.0 (minimum-maximum, 5.0-7.5) and had no significant association with USG values.

Sledge DG, Bolin SR, Lim A, et al: Outbreaks of severe enteric disease associated with *Eimeria furonis* infection in ferrets (*Mustela putorius furo*) of 3 densely populated groups. J Am Vet Med Assoc 239(12):1584-1588, 2011

Severe outbreaks of enteric coccidiosis were described in 3 unrelated, dense ferret populations regularly receiving new ferrets. Outbreaks were characterized by severe diarrhea and substantial mortality rates. The ages of affected ferrets in each group ranged from less than 1 year to greater than 6 years, with ferrets of all ages similarly affected. There was no apparent sex predilection, and ferrets with underlying diseases (e.g., hyperadrenocorticism, presumptive insulinoma) were not disproportionately affected. All ferrets in each group had been previously vaccinated against canine distemper and rabies viruses. The first outbreak involved a ferret rescue group in the Detroit area, which had a total population of 42 ferrets ranging in age from less than 1 year to greater than 5 years. During the course of the outbreak, more than half of the ferrets had clinical disease signs and 7 died. Four of the ferrets that died were submitted for necropsy. Most clinically affected ferrets were identified during the first 2 to 3 weeks of the outbreak, but diarrhea sporadically developed in ferrets over the next 4 months despite attempts to quarantine affected ferrets and undertake thorough environmental cleanings. Three years after the initial outbreak, there was a similar but more isolated outbreak of bloody diarrhea. The second affected group included 63 ferrets from a private ferret breeder and shelter in western Pennsylvania. During a 2-month period, 13 ferrets died and at least 21 ferrets had clinical signs of enteric disease and recovered. Necropsies were performed on all ferrets that died. In this facility, ferrets were separated into 3 groups, each of which occupied 1 of the 3 floors of the shelter. During the first 3 weeks of the outbreak, clinical signs affected only ferrets on the first floor, but ferrets on all 3 floors were diagnosed with the disease. The third affected group was from a shelter in eastern Pennsylvania that housed 62 ferrets. An outbreak of severe diarrhea over the course of 2.5 months affected at least 29 ferrets, 4 of which died. Two of the ferrets were submitted for necropsy. As with group 1, most of the diseased ferrets were identified during the first 3 weeks of the outbreak. Clinical signs of enteric condition were similar for all 3 groups. The most common clinical disease sign was foul-smelling diarrhea, ranging in color and consistency from beige and pasty or gelatinous to dark black and tarry. Occasionally, hematochezia developed and ferrets were lethargic, whereas weight loss, dehydration, anorexia, and weakness were common clinical conditions. The clinical course of disease in any ferret was generally 5 to 10 days before recovery or progressive worsening and eventual death. Fecal samples from diseased ferrets examined by direct smear techniques and fecal floatation testing sporadically showed low numbers of oocysts in group 1 only. Gross and histopathologic lesions were similar in all ferrets. Characteristic histologic lesions included erosion, blunting, and occasional fusion of intestinal villi and sloughed epithelial cells containing intracytoplasmic coccidia, often in high numbers and representing multiple life stages. The morphology of sporulated oocysts identified from group 1 was consistent with *Eimeria furonis*, and PCR assays of formalin-fixed tissue specimens from ferrets in all 3 groups confirmed the identification. Results of PCR assays for coronavirus, rotavirus, and influenza virus, as well as immunohistochemistry testing for ferret enteric coronavirus,
were negative. Bacterial cultures of intestinal sections yielded no pathogenic organisms. When coccidia were identified during outbreaks, ferrets were treated with sulfadimethoxine (25 mg/kg orally every 24 hours) for 21 days in addition to supportive care. Treatment was moderately effective in controlling clinical signs of enteric disease but failed to eliminate fecal shedding of oocysts in group 1 despite multiple treatments. In some cases, ferrets died despite treatment. *Eimeria furonis* is the species of coccidia most commonly reported in ferrets, but it is generally thought to cause subclinical infections. Overt disease has rarely been reported in individual young ferrets, and this is the first report describing outbreaks of severe enteric disease associated with *E furonis* affecting multiple ferrets at different locations. Serial examinations of both individual and pooled fecal samples are recommended to identify the causative coccidial organism in the face of outbreaks of enteric disease in ferrets.

Mans C, Sladky KK: Endoscopically guided removal of cloacal calculi in three African spurred tortoises (*Geochelone sulcata*). *J Am Vet Med Assoc* 240(7):869-875, 2012

Urinary calculi, typically composed of urate salts, are commonly reported in captive chelonians. The underlying etiology of calculus formation remains unknown, but suggested contributory factors include vitamin deficiencies, calcium deficiency, excess amounts of dietary protein and oxalates, and bladder infection. Urinary calculi are most frequently found in tortoises native to arid climates. The urinary bladder in tortoises native to arid climates, the urinary bladder is the primary organ responsible for postrenal osmoregulation and fluid storage. Chronic dehydration, which causes delayed or insufficient emptying of the bladder, has been implicated in calculus formation. When cystic calculi migrate from the urinary bladder to the pelvic canal, they can become wedged in the cloaca. The intrapelvic location of the chelonian cloaca complicates surgical intervention for calculus removal. Plastronotomy is highly invasive, requires prolonged surgery and recovery times, and is costly. A surgical approach through the prefemoral fossa is less invasive but provides limited visibility and access, particularly for the removal of large cloacal or cystic calculi. This report describes the diagnosis and endoscopically-guided treatment of cloacal calculi in 3 female African spurred tortoises (*Geochelone sulcata*). Historical findings with retained cloacal calculi included reduced food intake, tenesmus, and lack of fecal output. Physical examination findings were unremarkable, and cloacal calculi were diagnosed by radiography and endoscopy in all 3 tortoises. One tortoise also had a cystic calculus. The tortoises were anesthetized by the following protocols: tortoises 1 and 3 received midazolam (2.0 mg/kg subcutaneously [SC]), medetomidine (0.15 mg/kg SC), ketamine hydrochloride (2.5 mg/kg SC), and morphine (1.0-1.5 mg/kg SC), whereas tortoise 2 received morphine (2.0 mg/kg SC) and midazolam (1.0 mg/kg SC). The animals were placed in dorsal recumbency at a 30-degree angle with the tail below the head. Cloacal endoscopy was accomplished with a 30°, 18-cm x 2.7-mm rigid videendoscope in a 3.5-mm diameter operating sheath (endoscope models 64019BA and 67065C; Karl Storz Veterinary Endoscopy America Inc., Goleta, CA USA) attached to a mobile endoscopy unit with an integrated image processing module (Tele Pack Vet; Karl Storz). An intravenous infusion set, attached to the 2 ingress ports of the operating sheath, was used to deliver warm tap water (sterile fluids were deemed unnecessary in the nonsterile cloacal environment), thereby facilitating visibility during cloacoscopy and allowing simultaneous irrigation of debris out of the field of view. Once the cloacal calculus was identified by endoscopy, a bur was used to drill into the stone. A plain-fissure cutting bur and soft-tissue protector (iM3, Inc., Vancouver, WA USA) were mounted to a low-speed motor dental handpiece (iM3, Inc.) that had a straight nose cone. By use of endoscopic guidance, the cutting bur was inserted into the cloaca and used to create a hole in the center of the calculus. A Babcock forceps was then used to grasp the calculus and crush it into fragments. In one case, the full calculus could not be fragmented during the first procedure (because of its cranial location) but was removed completely during a second procedure. Because there is the real possibility of soft-tissue injury with this procedure, the bur was not introduced into the urinary bladder for removal of the cystic calculus. Meloxicam was administered postoperatively (0.2 mg/kg SC), and atipamezole (0.75 mg/kg SC) and flumazenil (0.05 mg/kg SC) were administered to antagonize medetomidine and midazolam, respectively. The tortoises were able to ambulate within 10 to 15 minutes after antagonist administration and were subsequently discharged from the hospital later the same day. The patients began eating 1 to 2 days after discharge and defecated generally within 7 days after dis-
charge. Iatrogenic damage to the cloaca was not detected in any of the tortoises, and the total time for each procedure was 30 to 60 minutes.

**Kirchgessner MS, Tully TN Jr, Nevarez J, et al: Magnesium therapy in a hypocalcemic African grey parrot (*Psittacus erithacus*). J Avian Med Surg 26(1):17-21, 2012**

Hypocalcemia-induced seizure activity is a well-recognized clinical syndrome in African grey parrots. Although several theories have been advanced to explain this syndrome, the underlying pathophysiology remains unknown. Patients exhibiting mild neurologic abnormalities are often normocalcemic (calcium level, 8-13 mg/dL), whereas calcium levels during seizure activity are typically lower than 6.0 mg/dL. This report describes a 10-year-old African grey parrot presenting for abnormal neurologic behavior of 24 hours’ duration. The bird’s diet for the previous 8 years had consisted of a commercial seed mix occasionally supplemented with fruit and vegetables. On physical examination, the bird was sternal, unable to right itself, and hypertonic. Results of a plasma biochemical analysis on hospital admission included elevated levels of aspartate aminotransferase, creatine kinase, and uric acid, as well as a decreased calcium level (5.5 mg/dL). Results of a complete blood count and whole-body radiographs were unremarkable. Analysis of a heavy metal panel (lead, zinc, and copper) showed a slightly elevated copper level, which was attributed to possible species variation or dietary influence. Despite treatment with parenteral calcium and vitamins A, D, and E as well as increasing doses of orally administered calcium, the bird’s plasma calcium levels continued to decrease over the following 4 days. On day 5, the bird exhibited seizure activity after which the ionized calcium level was determined to be 0.54 mmol/L (reference range, 0.96-1.22 mmol/L). The plasma magnesium level was decreased at 1.9 mg/dL (reference range, 2.1-3.4 mg/dL). Magnesium sulfate (20 mg/kg intramuscularly once) was administered 8 days after the initial presentation, and the bird’s plasma calcium level, measured 24 hours later, had increased to 7.7 mg/dL. The bird was weaned off all parenteral medications, including calcium gluconate. Results of a plasma biochemical profile analyzed 4 days after magnesium administration showed a calcium level of 8.3 mg/dL and a magnesium level of 3.3 mg/dL. No further neurologic abnormalities were observed. The authors hypothesize that a primary dietary magnesium deficiency was the cause of the bird’s neurologic abnormalities. Magnesium functions as a cofactor for many physiological enzymes, including 1-α-hydroxylase, the enzyme involved in vitamin D₃ metabolism. Decreased plasma levels of magnesium also result in an impaired response to parathyroid hormone, whose function it is to increase blood calcium levels by stimulating renal calcium reabsorption, increasing osteoclastic bone resorption, and increasing intestinal absorption of calcium. These functions occur synergistically with calcitriol, the principal active form of vitamin D₃. Primary magnesium depletion in leghorn chicks has been shown to result in progressive hypocalcemia and insufficient production of calcitriol. Furthermore, research in leghorn chicks has shown that a normocalcemic, magnesium-deficient diet results in low plasma magnesium and calcium levels. To correct the hypocalcemia that is directly related to magnesium-deficient diets, the hypomagnesemia must first be rectified.

**Liu J, Kerr PJ, Wright JD, et al: Serological assays to discriminate rabbit haemorrhagic disease virus from Australian non-pathogenic rabbit calicivirus. Vet Microbiol 157(3-4):345-354, 2012**

The pathogenic rabbit hemorrhagic disease virus (RHDV) belongs to the *Lagovirus* genus, which also includes the benign rabbit caliciviruses (RCVs). RHDV was introduced into Australia and New Zealand as a biological control agent for wild rabbits and has resulted in a dramatic reduction in the rabbit population, although it has been less effective in the more humid and cooler regions of Australia. In some areas, antibodies cross reacting to RHDV were found in blood samples from wild rabbits before RHDV was released, suggesting that nonpathogenic RCV strains could be effectively immunizing rabbits against RHDV. In fact, the RCV-A1 strain recently isolated in Australia does elicit partial cross protection against RHDV in rabbits. Understanding the geographical distribution and seasonal occurrence of RCV-A1 strains is of particular concern in Australia, where this nonpathogenic virus appears to be serving as a natural vaccine against RHDV, thus reducing the efficacy of the biocontrol program. Conversely, other countries working toward preventing the introduction or transmission of RHDV in wild rabbit populations are interested in learning more about the potential ability of RCV exposure to protect against RHDV.
infection. Strains of RCV previously identified in rabbits were very closely related to RHDV, and using RHDV-specific assays resulted in RHDV antibodies masking antibodies to RCV, thus preventing accurate serologic identification. Before this study, no specific serologic test for RCV had been developed. However, the Australian RCV-A1 strain shows substantial genetic differences from RHDV. This study describes the development of a competition enzyme-linked immunosorbent assay (ELISA) able to discriminate between antibodies to RHDV and RCV. Although various degrees of cross reaction with RHDV antibodies were observed in isotype ELISAs, a competition ELISA using an RCV-A1–specific monoclonal antibody was shown to be specific for RCV-A1 antibodies and did not cross react with RHDV antibodies. In addition, the study describes the development of a semiquantitative mucosal secretory IgA ELISA specific for RCV-A1, which is performed on rectal swabs instead of blood samples. The combination of tests described in this study provides important tools for monitoring RCV-A1 infection when it occurs alone and for discriminating between RCV and RHDV infections within the same rabbit population.

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1557-5063/12/2104-30.00  
http://dx.doi.org/10.1053/j.jepm.2012.09.014