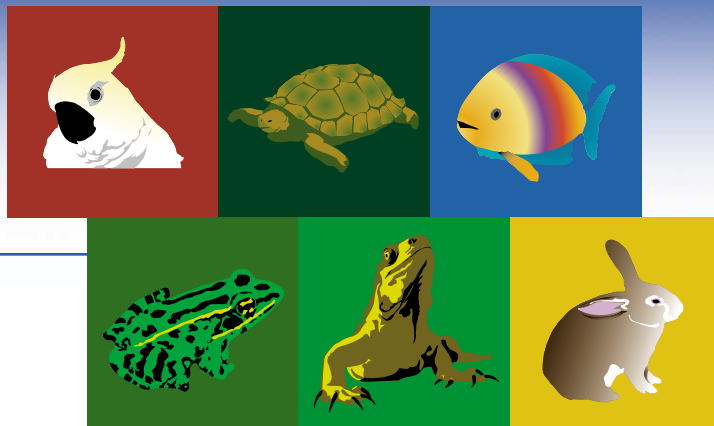


Diagnostic Challenge



History

An adult female savannah monitor (*Varanus exanthematicus*) weighing 987g was presented with a 2-week history of anorexia, malaise, and proprioceptive deficits in all four limbs. The owners had rescued the monitor from a pet store 6 months before presentation, and the monitor was housed in a 91.44 cm (36 inch) × 45.72 cm (18 inch) × 45.72 cm (18 inch) glass tank that was covered with a screen. The owners stated they maintained a temperature gradient using radiant light from 75 to 86°F (24-30°C) day time to 70 to 75°F (21-24°C) at night. Orchid bark was used as the substrate in the enclosure, which also included a large piece of driftwood. The monitor was fed live adult mice once weekly, and fresh water was provided in a bowl.

On physical examination, the patient was emaciated, nonambulatory, and depressed. The monitor was estimated to be 10% dehydrated, based on its pale, tacky mucous membranes and sunken eyes. Cachexia was clinically evident in the significant atrophy of the sagittal muscles on the head, epaxial muscles, and large muscles of the limbs. Coelomic palpation revealed a mass in the left caudal quadrant of the coelomic cavity.

The animal was hospitalized for diagnostic evaluation and treatment. Initial diagnostic tests included a complete blood count (CBC), plasma chemistry panel, direct fecal parasite examination, fecal flotation, and survey radiographs. Blood for the CBC and plasma chemistry panel was collected from the ventral coccygeal vein. Peripheral vasoconstriction attributed to the severe dehydration made jugular catheter placement impractical. Instead, warmed isotonic fluids (maintenance at 25 mL/kg/d plus 10% deficit corrected over 48 h), were given intracoelomically. Because of the severe emaciation and relative negative energy balance, enteral support was initiated to provide maintenance plus additional replacement calories. In this case, the basal metabolic rate was calculated and the caloric needs of the monitor were multiplied fourfold to compensate for the condition of the patient. An enteral carnivore diet (Walkabout Farm's Quantum Series Enteral Carnivore Diet, Nutritional Support, Virginia 24,136, USA) was provided for nutritional support at 26.12 Kcal/d. Approximately 50% of the initial calories were provided over the first 48 h to prevent re-feeding syndrome. Enrofloxacin at 10 mg/kg was administered once intramuscularly (Baytril®, 22.7 mg/mL, Bayer, Shawnee Mission, KS) then the treatment modified to oral dosing at 10 mg/kg q24 hour. Pain management was provided with an oral carprofen suspension (Rimadyl®, 10 mg/mL, Pfizer animal Health, New York City, NY; compounded by Louisiana State University School of Veterinary Medicine Pharmacy, Baton Rouge, LA) at 2 mg/kg q 12 h. The animal was depressed and in poor body condition; therefore, anesthesia was not warranted for radiographic positioning (Figs 1 and 2). Significant laboratory results are listed in Table 1.

At this time, please evaluate Figures 1 and 2, the history, results of the physical examination, and diagnostic tests. Make a list of differential diagnoses before continuing.

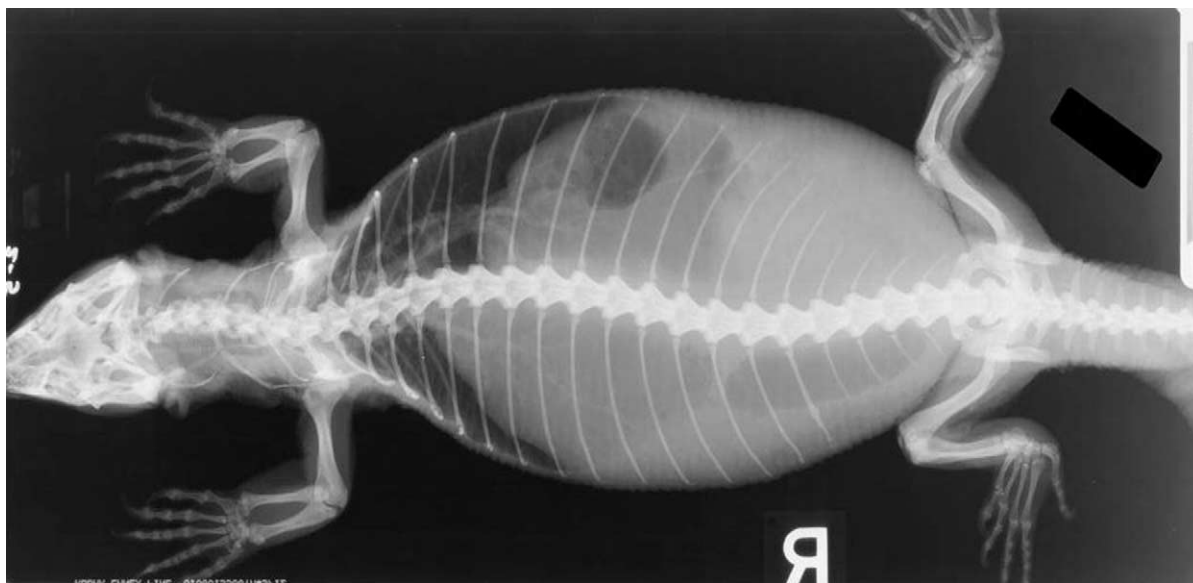


Figure 1. Dorsoventral radiograph of an adult female savannah monitor that presented with a 2-week history of anorexia, malaise, and proprioceptive deficits in all four limbs.

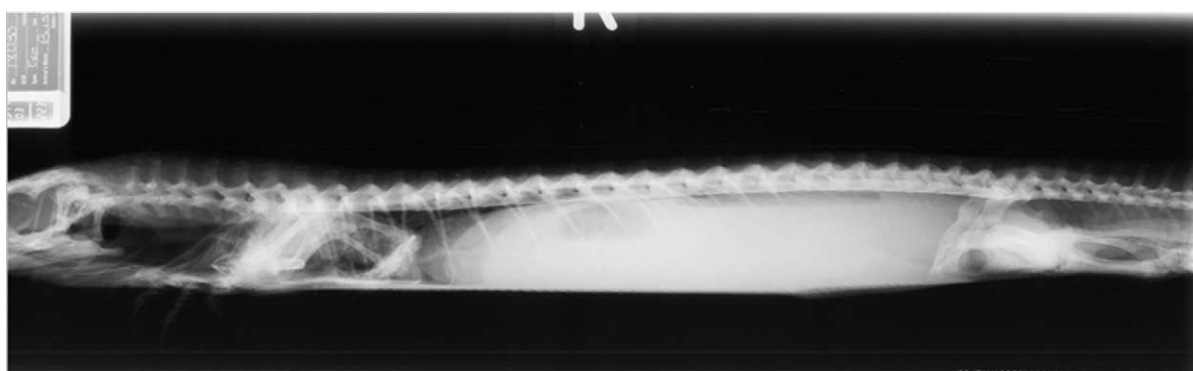


Figure 2. Lateral radiograph of the savannah monitor described in Fig 1.

Diagnosis

The CBC revealed a chronic inflammatory response ($16.1 \times 10^3 \mu\text{L}$) characterized by a monocytosis ($3.5 \times 10^3 \mu\text{L}$). Initial biochemistry results showed elevated creatinine kinase (2010 IU/L), uric acid (25.4 mg/dL), and a decreased glucose (39 mg/dL) and albumin (1.3 g/dL) values. No parasites were observed on the direct fecal examination and flotation. Differential diagnoses for this monitor included abscess, granuloma, neoplasia, gastrointestinal impaction, inspissated egg/ectopic ova and cystic calculi. Dorsoven-

tral and lateral survey radiographs revealed a diffuse, soft tissue opacification in the left caudal quadrant of the coelomic cavity (Fig 3). Once the animal was rehydrated and stabilized, an exploratory coeliotomy was planned to further evaluate the coelomic mass. Sedation was achieved with 5 mg/kg tiletamine/zolazepam IM (Telazol®, 100 mg/mL, Fort Dodge Laboratories, Fort Dodge, IA) and 0.5 mg/kg butorphanol tartrate (Torbugesic, 10 mg/mL, Fort Dodge Laboratories, Fort Dodge, IA). After 15 minutes, the monitor was intubated and maintained on 1 to 2% isoflu-

rane/1L O₂ (Isoflo™, Abbott Laboratories, Abbott Park, IL). A 9-cm ventral paramedian incision was made. When the left fat pad was reflected cranially, a 7.5 cm × 3.5 cm large, firm, encapsulated polycystic mass was found filling the left caudal coelomic area and the liver appeared cystic and mottled (Figs 4 and 5). The mass was resected using 4-0 polydioxanone suture (PDS, Ethicon, Inc., Westwood, MA). Liver biopsies were taken and submitted for histopathology. The coelomic cavity was gently lavaged with 100 mL of sterile saline. The body wall was closed in a simple continuous

Table 1. Hemogram and Serum Biochemistry Profile for the Savannah Monitor

Parameter	Measurement	Reference Value (Mean \pm SD) ¹
Packed cell volume (%)	28	32.3 \pm 6.5
Leukocytes ($10^3/\mu\text{L}$)	16,100	4.753 \pm 2.481
Heterophils ($10^3/\mu\text{L}$)	4,500	1.802 \pm 1.345
Lymphocyte ($10^3/\mu\text{L}$)	8,100	2.058 \pm 1.258
Monocytes ($10^3/\mu\text{L}$)	3,500	0.980 \pm 1.936
Eosinophils ($10^3/\mu\text{L}$)	0	0.115 \pm 0.000
Basophils ($10^3/\mu\text{L}$)	0	0.213 \pm 0.370
Morphologic cell characteristic	1 ⁺ polychromasia	
Glucose (mg/dL)	39	109 \pm 24
Aspartate aminotransferase (IU/L)	23	30 \pm 32
γ -glutamyltransferase (IU/L)	28	6 \pm 6
Alkaline phosphatase (IU/L)	26	77 \pm 136
Alanine aminotransferase (IU/L)	32	58 \pm 88
Creatine Kinase (IU/L)	2010	1540 \pm 2325
Total protein (g/dL)	5.1	6.9 \pm 1.2
Albumin (g/dL)	1.3	2.1 \pm 0.5
Globulin (g/dL)	3.3	4.9 \pm 0.8
Calcium (mg/dL)	14	14.3 \pm 1.4
Phosphorus (mg/dL)	4.3	4.9 \pm 2.3
Sodium (mmol/L)	150	155 \pm 5
Potassium (mmol/L)	4.4	4.6 \pm 1.6
Chloride (mmol/L)	110	115 \pm 7
TCO ₂ (mmol/L)	14.1	18.3 \pm 3.1
Uric acid (mg/dL)	25.4	7.0 \pm 4.1

pattern and the skin closed in a horizontal mattress pattern using a 3-0 polydioxanone suture.

Fluid therapy was continued postoperatively. In addition, metronidazole at 25 mg/kg

(50mg/mL, Schein Pharmaceuticals, Inc., Florham Park, NJ) PO, q 24 h was initiated to man-

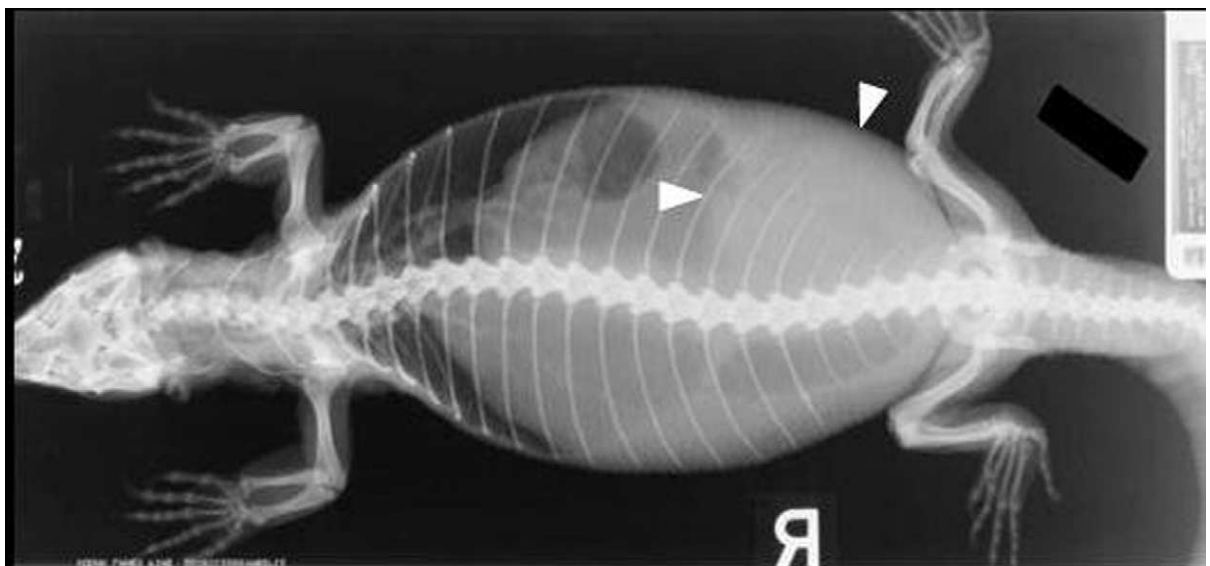


Figure 3. Same as Fig 1, showing increased soft tissue opacity in the left caudal coelomic quadrant (triangles) displacing the viscera cranially into the lungs.



Figure 4. Surgical resection of the Schwannoma in the savannah monitor. Note the encapsulated polycystic mass filling the left caudal coelomic area.



Figure 5. Note the large cystic structures on the liver.

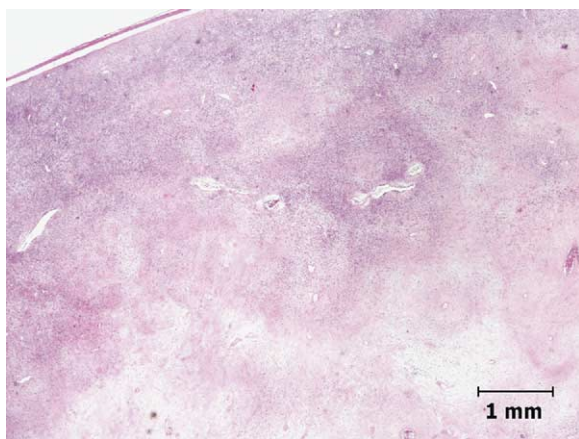


Figure 6. Microscopic examination of the mass with large areas of necrosis and fibrosis. Note the outline of the periphery of the encapsulated mass. Hematoxylin and eosin stain.

age potential anaerobic infections.

Microscopic examination of the mass revealed large areas of

necrosis and fibrosis with firm regions comprised of neoplastic cellular spindle-type cells resembling neural pericytes (Figs 6

and 7). All neoplastic cells were positive for S-100a and vimentin, but were negative for desmin and smooth muscle actin via immunohistochemistry. The histologic lesion, which appeared to be contained within the fibrotic encapsulation, was consistent with a benign peripheral nerve sheath tumor (schwannoma). The liver biopsy revealed areas of vacuolated fat and a multilocular cystic structure with the occasional remnants of hepatic cells. The liver biopsies were consistent with polycystic hepatitis. The patient's general condition had markedly worsened. The CBC was repeated, and the monitor still had a monocytosis ($1.9 \times 10^3/\mu\text{L}$). Red blood cell morphology revealed the presence of mitotic figures and a 3^+ polychromasia. Flaccid paralysis and a lack of proprioception were more pronounced after surgery. The patient was found dead 18 days after presentation.

At necropsy, the liver had two nodular masses that measured 1- to 1.5-cm diameter and were surrounded by caseous necrosis. Microscopic findings of the liver revealed areas of coagulative necrosis surrounded by small numbers of heterophils and macrophages. Marked fibrosis with hepatocellular loss, microthrombi in the sinusoids, and vacuolated hepatocytes were also noted. No related or significant gross lesions were observed in other organs.

The tumor most likely originated from either a paravertebral muscle along the coelomic wall or a peripheral nerve/ganglion around the intercostal space.

Comments

The packed cell volume in this monitor was above the reference

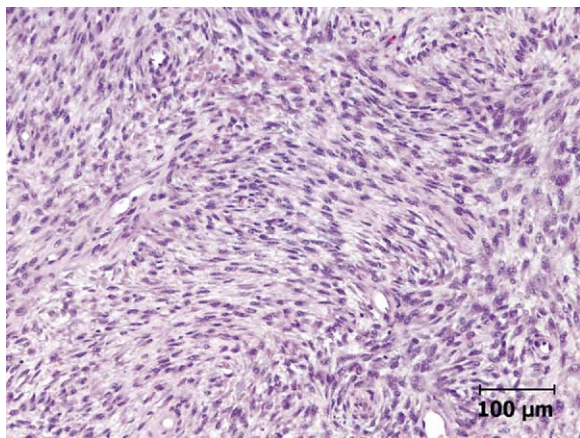


Figure 7. Photomicrograph examination of the mass arranged in partial whorls patterns, bundle of cells, and spindle-shaped nuclei. Hematoxylin and eosin stain.

of 26%.¹ However, this animal was severely dehydrated and, therefore, the 28% was falsely elevated. In addition, the 1⁺ and 3⁺ polychromasia suggested that there were immature cells being released in the peripheral pool. When interpreting a CBC for a reptile, it is important to know the general distribution of their cells. Some animals are primary heterophilic, while others are primary lymphocytic. Savannah monitor's are primary lymphocytic. Not having this information can lead to the misclassification of a leukogram. The CBC revealed a chronic inflammatory leukogram that was characterized by the monocytosis. The plasma biochemistry abnormalities resolved following provision of the enteral and fluid therapy. The chronic liver disease and hepatic lipidosis surely contributed to the death of this animal. In this monitor, the hepatic lipidosis was most likely do to a chronic anorexia and impaired metabolism. The AST was found to be within normal limits in this animal. With the severe degree of liver pathology noted, it would have been expected that liver enzymes would be elevated. If we had suspected liver disease before the surgery, we would have performed a bile acid assay.

Peripheral nerve sheath tumors of the abdominal autonomic ganglia and the myenteric plexus are rare, but examples have been reported in humans, cattles, horses, dogs, rats and fish.²⁻⁸ Schwannoma is a tumor of the nerve sheath or Schwann cells. It is characterized by being solitary and encapsulated. Histologically, the tumor contained long spindle cell, partial whorls, and pallasiding nuclei. Immunohistochemically, the tumor stained positive for S-100, a protein which is specific for nervous tissue.^{9,10}

In humans, neurofibromatosis type 2 (NF2) is an autosomal, dominant inherited disorder characterized by the presence of tumors, especially schwannomas. The NF2 is characterized as a bilateral vestibular schwannoma affecting cranial nerve eight.¹¹ The tumor accounts for 8% of all human intracranial tumors and 29% of human primary spinal tumors.^{11,12} Solitary large tumors have been found in the duodenum and the cecum of dogs, and in the colon of horses.^{4,6} In reptiles, malignant peripheral nerve sheath tumors have been reported in a water moccasin (*Agkistrodon piscivorus*),⁷ a Korean viper (*Agkistrodon halys brevicaudu*),⁵ and a bearded

dragon (*Pogona vitticeps*).⁹ Benign peripheral nerve sheath tumors have also being described in two related bearded dragons,¹⁰ and a golden tree snake (*Chrysepelae ornate*).¹³ Although these tumors usually do not metastasize, they are locally invasive. There are no published reports on specific treatments of peripheral nerve sheath tumors in reptile species. Depending on the size, the location and the type of peripheral nerve sheath tumor involved, surgical correction is the preferred treatment. Even though the tumor was benign, it carried a poor prognosis because of its location. Ultimately, we believe this animal's death resulted from liver failure.

This case was submitted by **Orlando Diaz-Figueroa, DVM** and **Mark A. Mitchell, DVM, MS, PhD**, from the Department of Veterinary Clinical Sciences, and **E. Donald Roberts, DVM, PhD**, and **Dae-Young Kim, DVM, PhD**, from the Department of Pathobiological Sciences, Louisiana State University School of Veterinary Medicine, Skip Bertman Drive, Baton Rouge, LA 70803.

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