Gastrointestinal Disturbances in a Bearded Dragon (*Pogona vitticeps*)

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HISTORY

A 2.5-year-old male bearded dragon, *Pogona vitticeps*, presented with a history of scant fecal production, vomiting, lethargy, and darkening of the beard. At the age of 6–8 months, this animal discontinued eating so the owner started force-feeding it phoenix worms (*Hermetia illucens*), silkworms (*Bombyx mori*), butterworms (*Chilecomadia moorei*), squash (*Cucurbita* sp.), and zucchini (*Cucurbita pepo*). Force-feeding was continued to the time of presentation. Five months prior to presentation the owner reported that the animal had watery–mucoid diarrhea, which resolved spontaneously after 4 months. One week prior to presentation, large amounts of fecal material were palpated in the intestines, and treatment with enemas, lactulose, and a change of diet was instituted. The owner became increasingly alarmed when the lethargy worsened, the animal began to vomit, and the beard turned black.

Physical examination of the bearded dragon indicated that the animal was slightly overweight (426.3 g, with a body condition score of 6/9); large fat pads could be palpated on both sides of the coelomic cavity. The dewlap area was black. A firm, movable mass was palpated in the cranial abdomen and a tubular, soft tissue structure was palpated between the fat pads, which proved to be an enlarged colon with feces and gas on lateral and dorsoventral survey radiographs. Other radiographic findings were unremarkable. Husbandry was considered appropriate except for a diet too rich in high-fat animal matter. A blood sample was taken from the ventral coccygeal vein and submitted for hematology and chemistry determination. Abnormal values included anemia (hematocrit 18%, reference range 24–36%), hyperglycemia (glucose 1,429 mg/dL, reference range 149–253 mg/dL), hypophosphatemia (phosphorus 1.0 mg/dL, reference range 3.1–8.1 mg/dL), and hyponatremia (sodium 118 mmol/L, reference range 145–167 mmol/L).

BASED ON HISTORY AND THE RESULTS OF THE PHYSICAL EXAMINATION AND BLOODWORK, MAKE A LIST OF DIFFERENTIAL DIAGNOSES AND DETERMINE IF ADDITIONAL DIAGNOSTIC TESTS ARE INDICATED

The list of differential diagnoses for this case included:

1. Hepatic lipidosis. A diet based on high-fat animal items combined with good body condition, large fat pads, and anorexia is indicative of hepatic lipidosis (Hernandez-Divers and Cooper, 2006).
2. Neuroendocrine gastric carcinoma. This recently described malignant neoplasia affects young bearded dragons, and clinical signs include anorexia, vomiting, hyperglycemia, and anemia (Ritter *et al.*, 2009). Although other neuroendocrine carcinomas (pheochromocytoma, glucagonoma, islet somatostatinoma) could produce similar clinical signs, these have not been reported in bearded dragons.
3. *Adenovirus*. Infections by *Adenovirus* have been described in bearded dragons causing anorexia, lethargy, and hepatic necrosis (Julian and Durham, 1982; Frye *et al.*, 1994; Moormann *et al.*, 2009).
4. Constipation. Constipation is a common clinical problem in bearded dragons and is usually associated with anorexia (Wright, 2008). Causes are multiple and include inappropriate substrate, insufficient provision of green, leafy vegetables, inadequate water intake, onset of follicular development, poor hygiene, stress (new cagemates, recent acquisition), old age, urates obstructing the colon, anatomic malformation of the pelvis or spine, gastrointestinal foreign body, renomegaly, and parasitism (Wright, 2008).
5. Gastrointestinal ulcers and parasitism. As in other animals, these condition could lead to vomiting and anemia (Campbell, 2006; Funk, 2006).

From the list of differential diagnoses, neuroendocrine gastric carcinoma was the only condition consistent with all the clinical findings. Most of the other conditions, even when combined, would not account for the severe hyperglycemia. An endoscopic approach, including coelioscopy and upper gastrointestinal endoscopy, was recommended to further evaluate this patient and obtain biopsies for a definitive diagnosis.

Further diagnostic tests were delayed temporarily and the bearded dragon was discharged with a treatment regimen to manage a suspected hepatic lipidosis and constipation. Liver protectants (L-carnitine 250 mg/kg PO q24h and methionine 50 mg/kg PO q24h), oral fluids (5 mL PO q24h of an electrolyte solution), and force-feeding with a soft, vegetable-based food every 1–2 days was instituted to treat the hepatic lipidosis. Fecal retention was treated with warm water soaks once daily and metoclopramide (1 mg/kg PO q24h for 7 days).

The bearded dragon was re-examined 10 days after the initial presentation. While the animal was still lethargic, anorexic, and had another episode of vomiting, fecal production had improved. Changes in the physical examination compared with the initial presentation included weight loss (40.1 g weight loss; current weight 386.2 g) and a reduction in the fecal content palpated in the colon. A second blood sample was collected from the ventral coccygeal vein and submitted for glucose determination, which remained markedly increased (1,489 mg/dl). Upper gastrointestinal endoscopy was scheduled that same day, and the bearded dragon was anesthetized with 10 mg/kg propofol (PropoFlo, Abbott Laboratories, Abbott Park, IL) IV into the ventral coccygeal vein. After intubation, a rigid 2.7-mm cystoscope was used to visualize a mass protruding into the lumen of the stomach. The mass occupied approximately 25% of the stomach, and there were no signs of gastric ulcerations. Several biopsies were taken from the mass, which revealed that it was a well-vascularized mass. The owner was informed of the findings and elected euthanasia based on a grave prognosis.

Necropsy was performed the following day. The stomach contained a 0.3-cm-diameter white, round mass that protruded into the gastric lumen (Fig. 1). The liver was moderately enlarged, diffusely yellow, and contained a myriad of white, round masses ranging from 0.2 cm to 1.5 cm (Fig. 2). Histologically, the gastric submucosa was markedly expanded by a highly cellular neoplasm that infiltrated through the muscularis mucosa and extended into the lamina propria (Fig. 3). The neoplasm was composed of sheets and nests of densely packed polygonal cells that frequently palisaded along a fine fibrovascular stroma and occasionally formed pseudorosettes. Neoplastic cells had indistinct cell borders, a scant amount of granular, eosinophilic cytoplasm, and round to oval nuclei with coarsely granular chromatin and occasionally one prominent eosinophilic nucleolus. There was moderate anisocytosis and marked anisokaryosis, with occasional macronuclei. There was an average of 16 mitotic figures per high-power field ($\times 40$). The same neoplasm was found in nodules

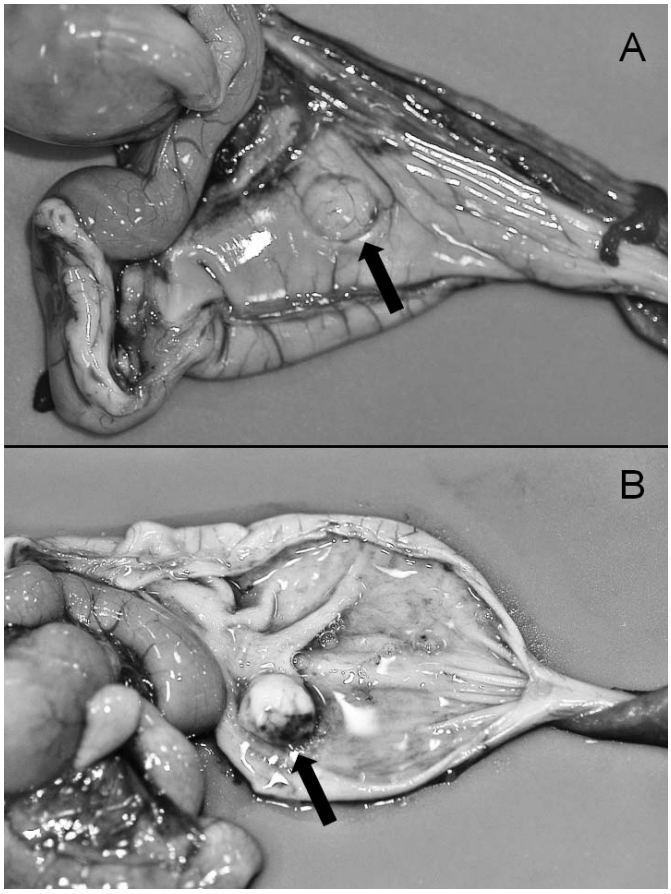

Figure 1. Necropsy examination of a bearded dragon that presented with scant feces, vomiting, anorexia, and darkening of the beard. A gastric mass (arrows) can be seen protruding into the lumen. (A) Serosal view. (B) Mucosal view.

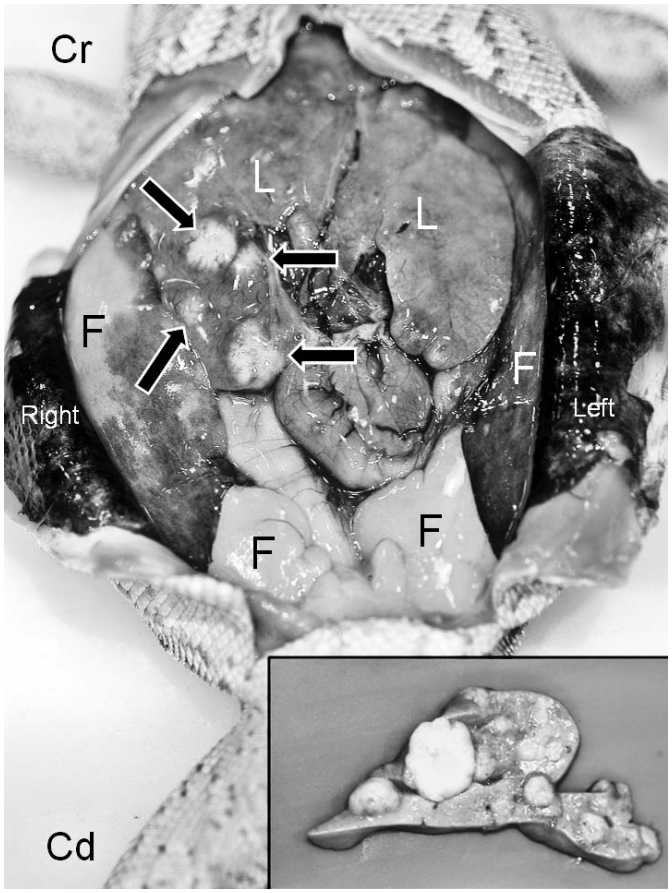

Figure 2. Necropsy examination of the bearded dragon described in Figure 1. Multiple masses can be seen within the liver (arrows). L = liver, F = fat pad, Cr = cranial, Cd = caudal. Inset: Liver on cut section showing multiple masses.

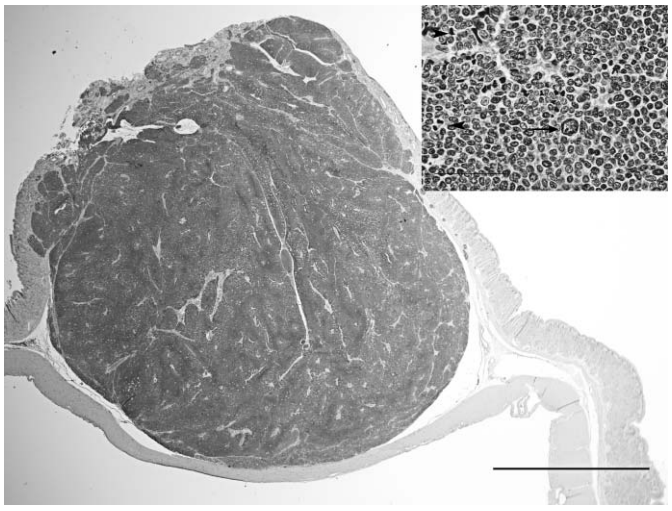

Figure 3. Histologic examination of the gastric mass. The neoplasm in the gastric submucosa is composed of polygonal cells in a fine fibrovascular stroma (bar = 2 mm). Inset: The gastric neoplasm exhibits marked anisocytosis and anisokaryosis with macronuclei (arrow) and numerous mitotic figures (arrowheads) (bar = 50 μm). Histologic diagnosis of this neoplasm is a gastric neuroendocrine carcinoma.

of the liver and kidney. In addition, all hepatocytes were disrupted by large vacuolization. The histologic diagnosis was gastric neuroendocrine carcinoma with metastases to the liver and kidney, and severe diffuse hepatic lipidosis.

Discussion

Gastric neuroendocrine carcinoma is an emerging entity in bearded dragons that produces clinical signs that may include anorexia, weight loss, weakness, and vomiting. Clinicopathologic abnormalities have only been reported in a few animals and included anemia and hyperglycemia. These malignant tumors readily metastasize. Multihormonal expression is common; however, many of these tumors express somatostatin (Ritter *et al.*, 2009). In the present case, immunohistochemistry to characterize hormonal production by the tumor was not done. The present case shared most clinical signs and clinicopathologic findings with the article that described this malignancy, including anemia, hyperglycemia, and weight loss; characteristics that are also shared with human somatostatinoma (Ritter *et al.*, 2009). However, glucose levels in the present case (1,429 mg/dL and 1,489 mg/dL) were markedly higher than in the previous report (Ritter *et al.*, 2009). Hyponatremia and hypophosphatemia were also noted in this case; however, the authors suspect that these findings were the result of metabolic derangements secondary to the carcinoma and anorexia.

Endoscopy and biopsy were the most appropriate tools for an antemortem diagnosis of this malignancy. Euthanasia was indicated in this case, as all described cases have developed metastasis in multiple organs, particularly the liver. It is important to note, however, that no study has been performed to rule out the possibility of early detection and treatment based on the clinicopathologic abnormalities described. This report also shows the problem associated with using plasma chemistries to diagnose liver disease in reptiles. Despite the severe hepatic lipidosis and infiltration of the liver with tumors, AST (25 U/L), bile acids (58 μmol/L), cholesterol (284 mg/dL), and triglycerides (43 mg/dL) were all considered within normal limits, probably due to the chronic nature of the disease or the presence of functional hepatic reserve.

In conclusion, veterinary clinicians should consider neuroendocrine gastric carcinoma in their differential diagnoses list for bearded dragon cases when anorexia, vomiting, hyperglycemia, and anemia are found. Prognosis of this condition is grave because metastases are a common finding.

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