PRESUMED HYPOVITAMINOSIS A SECONDARY TO CHRONIC BILE DUCT OBSTRUCTION IN A HERMANN’S TORTOISE (Testudo hermanni)

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ABSTRACT

A 10-yr-old, 4-kg, female Hermann’s tortoise (Testudo hermanni) presented for bilateral nictitating membrane prolapse and keratoconjunctival dryness, with intermittent blepharocoelema. Hematologic analysis demonstrated mild leukocytosis and anemia. Plasma retinol concentration (0.078 ug/ml) was low based on a reference interval for the spur-thighed tortoise (Testudo graeca) of 0.61 ± 0.16 ug/ml. Initial treatment consisted of increasing enclosure humidity, topical ophthalmic lubricants, diet modification, and a vitamin-mineral supplement (2 g p.o. SID; ReptiVite™ with D3, Zoo Med Laboratories, Inc., San Luis Obispo, CA 93401 USA). Parenteral vitamin A (1750 – 2000 IU/kg s.c. q 7 – 14 days) was started 3 mo after dietary changes; ocular lesions resolved within 1.5 mo, though plasma vitamin A concentration remained low. The tortoise’s chronic anemia did not respond to treatment with an erythropoiesis-stimulating agent (100 U/kg s.c. q 3 days for 3 doses, then q 4 days for 4 doses, then q 7 days for 5 doses; Epogen®, Amgen, Inc., Thousand Oaks, CA 91320 USA) and a whole blood transfusion (36 ml) from a red-footed tortoise (Geochelone carbonaria). Computed tomography and magnetic resonance imaging revealed hepatomegaly with irregular margins and a distended gallbladder. The tortoise was euthanized due to poor prognosis and response to therapy. Gross necropsy findings correlated well with advanced imaging results. No bile duct was identified grossly or histologically. A severely distended gall bladder, intrahepatic bile stasis, bridging portal fibrosis, and nodular regeneration were secondary to chronic biliary outflow obstruction. It was theorized that biliary outflow obstruction prevented adequate absorption of dietary vitamin A.

LITERATURE CITED