

HYPOVITIMINOSIS A IN A CAPTIVE COLLECTION OF AMPHIBIANS

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ABSTRACT

Overview

Hypovitaminosis A is a disease of malnutrition or dysnutrition. There have been references to this disease for thousands of years and the ancient Egyptians treated xerophthalmia (night blindness) with liver from cattle or poultry that is rich in vitamin A.¹ Today hypovitaminosis A is routinely diagnosed in humans, domestic livestock, and recently with more regularity in captive amphibian species.¹

Case Report

Over a 4 month period there was a significant increase in the number of amphibian clinical cases submitted to the hospital. This was unusual as this group of animals had been relatively problem free in the past. On physical examination clinical signs included, inability to use the tongue, gastrointestinal bloat, dermal ulceration, static growth rates in juveniles, as well as septicemia and acute death. The following species were affected, Yellow and blue poison arrow frog, (*Dendrobates tinctorius*), New Guinea tree frog (*Litoria infrafrenata*), African foam nesting frog (*Chiromantis xerampelina*), Puerto Rican Crested Toad (*Peltophryne lemur*).

This etiology of clinical signs was traced to a possible mixing error in the powdered amphibian supplement used to dust invertebrate food items fed to the frogs. The base of the amphibian nutritional supplement normally has a concentration of 230,000 IU/kg of vitamin A. Crickets fed and dusted with this supplement contain approximately 4,000 IU/kg of vitamin A (3/4' crickets). It was determined by analysis that the "new" supplement being used during the time period of this case report contained only 44,000 IU/kg of vitamin A which was roughly 20% of the concentration normally provided or 800 IU/kg of vitamin A (3/4' crickets).

The first clinical signs were noted approximately 3-4 months after the "new" supplement was fed. A small number of amphibians (10 individuals) showing clinical signs were currently being treated with vitamin A, however with this information, the remainder of the collection (790 frogs) was placed on prophylactic treatment of vitamin A as described below.

Diagnostic Sampling

Blood was collected from the New Guinea tree frogs and 0.3-0.4 ml of plasma was obtained and submitted to the Michigan State University Diagnostic Center for Population and Animal Health for vitamin A analysis. Most of the other frogs were too small (< 20gm) to be able to collect the required volume of plasma for analysis.

Treatment

The total number of amphibians to be treated exceeded 800 individuals, which included over 600 juvenile African foam nesting frogs. Frogs with clinical signs were treated based on the severity of signs using the following protocols:.

Drug

Aquisaol A (Mayne Pharma (USA) Paramus NJ, 07652) 50,000 USP/ml
1:10 dilution = 5,000 USP/ml (with sterile saline)

Dose

Small Frog dose 1 drop (< 20 gm frog) = 50-100 USP per dose
Large Frog dose 2 drops (20-120 gm frog) = 100-150 USP per dose

Frequency

- 1) No clinical signs – Topically, once a week for 2 months
- 2) Mild clinical signs – Topically, every 3 days for 2 months
- 3) Severe clinical signs – Injection for first does, then topical, every 3 days for 2 months

Results

Prior to treatment, two blood samples from the New Guinea tree frogs were analyzed and were found to have concentrations of vitamin A of, 11 and 20 ng/ml. Plasma vitamin A levels drastically increased from 5-20 ng/ml to 142- 217 ng/ml over the course of the 2 month treatment. However in many cases clinical improvement was seen within days. Skin lesions were seen to be improving within two weeks but many required up to 2 months to completely heal. Frogs with “short tongue syndrome” also showed clinical improvement.² Acute cases of short tongue syndrome successfully resolved with topical treatment and supportive care in the form of hand feeding and routine weighing.

Within 3 weeks of cessation of treatment, vitamin A plasma concentrations ranged from 46-295 ng/dl. Currently a published value for serum vitamin A in wild frogs or toads could not be located. Samples from wild toads will be obtained this summer to determine baseline levels of plasma vitamin A concentration.

Topical therapy appears to increase plasma levels of vitamin A, and to produce improvements in the clinical signs of hypovitaminosis A; however duration of therapy has not been established. A comprehensive nutrition plan that includes vitamin A supplementation (minimum of 230,000 IU vitamin A/ kg of supplement) should be implemented in captive anuran collections. Current clinical trials for dose and duration of therapy are ongoing.

LITERATURE CITED

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- 2) Pessier, AP, Linn M, Garner MM, Raymond JT, Dierenfeld ES, Graffam W. 2005. Suspected hypovitaminosis A in captive toads (*Bufo* spp.). Proceedings of the AAZV, AAWV, AZA/NAG joint conference, 57.