Diagnosis | **Thiamine deficiency**

Typical husbandry conditions for anoles have been previously described[2,3]. Our housing conditions were consistent with these recommendations for atmospheric humidity and temperature. Our lizards' diet was also consistent with these recommendations, as they were fed insects dusted with a vitamin and calcium preparation. Given the unusual presentation of torticollis and opisthotonus, however, a tentative diagnosis of thiamine (vitamin B1) deficiency was presumed.

In reptiles, clinical signs of hypothiaminosis or hypovitaminosis B1 are generally nonspecific and include muscle twitching, incoordination, blindness, seizure activity, torticollis, abnormal posture, spiral locomotion, jaw gaping, dysphagia and, potentially, death[4]. Affected snakes may be unable to strike prey accurately; in affected chelonians, the most notable clinical sign is a sinking of the eye within the bony orbit (enophthalmos)[4]. At necropsy, usually no gross lesions are seen. Histologically, leukoencephalopathy consisting of cerebral cortical necrosis with peripheral neuritis and cardiomyopathy is frequently encountered[5]. Histologic lesions also include a diffuse eosinophilia with severe demyelination and axon sheath fragmentation; generally inflammatory cells are absent[6].

We administered 0.1 ml of vitamin B complex (Butler-Schein Animal Health, Melville, NY) intraperitoneally once per day on four consecutive days to an affected anole; clinical symptoms resolved after the second day of injection. We interpreted this response as confirmation of our initial diagnosis. Because the treated lizard responded so rapidly, we began to administer vitamin B complex to any anoles presenting with neurologic symptoms until the cause of the thiamine deficiency was ascertained.

In conversation with the laboratory personnel caring for the anoles, we learned that the Herptivite supplement and Calcium Plus had been purchased in bulk more than 2 y before we began observing symptoms in the lizards and had been stored in the dark at room temperature. Thiamine is a highly labile vitamin (as are vitamins A and C) that undergoes deterioration under normal storage conditions; this is one reason why laboratory animal diets should be used within 6 months of their milling date[7]. We recommended that laboratory personnel purchase smaller quantities of the vitamin mix, store the mix in a refrigerator at 4 °C and discard any remaining vitamin supplement 6 months after its manufacture date.

Thiamine deficiency was originally reported in silver foxes farmed for fur; the resulting neurological disease was known as Chastek paralysis[8,9]. Investigations determined that thiamine was inactivated in diets containing fishmeal[10]. Calcium Plus contains some fishmeal. Thiaminase, a naturally occurring enzyme that destroys thiamine in a diet, is present in large amounts in fish meal[11,12]. Manufacturers often correct for potential thiamine breakdown by adding 1 mg of thiamine per kg of fish meal[13]. Another potential but less common cause of opisthotonus in lizards is cerebral xanthomatosis[14].

In conclusion, we report a common nutritional cause of a neurological problem of lizards in a laboratory setting. This case emphasizes the need to pay particular attention to the composition of the diet of unusual laboratory species and the need to extend good husbandry practices to all species used in the research environment.

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