Nutritional Degenerative Myopathy in a Population of Captive Bred *Uroplatus Phantasticus* (Satanic Leaf-Tailed Geckoes)

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Nutritional degenerative myopathy in a population of captive bred *Uroplatus phantasticus* (satanic leaf-tailed geckoes)

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Abstract. Severe generalized degenerative myopathy was diagnosed in a population of captive bred satanic leaf-tailed geckoes (*Uroplatus phantasticus*). The diagnosis was based on characteristic histological changes and response to dietary therapy. This is the first reported case of nutritional myopathy in the satanic leaf-tailed gecko.

Muscular degeneration and necrosis are relatively common conditions in veterinary medicine.[12] Of all the causes of muscular degeneration, vitamin E/selenium deficiency (nutritional myopathy) remains an important condition, particularly in production animals. Nutritional myopathy is most commonly reported in swine,[11,13,16] cattle,[1,10,20] sheep,[2] intensively reared salmonids,[8] and chickens.[3] In nonproduction animals there have been sporadic reports of a nutritional myopathy–like syndromes involving pelicans,[4] rabbits,[19] iguanas,[7] a cat,[6] horses,[18] goats,[14,15] and sea lions.[5]

Two dead, juvenile geckoes of the species *Uroplatus phantasticus* were submitted for necropsy. The specimens were 3–4 cm in length, weighed 3.2 and 3.4 g, and were unremarkable grossly. Histologically in both cases there were moderate to marked autolytic changes. In 1 specimen there was moderate to marked multifocal skeletal myofibrillar degeneration and necrosis. A tentative diagnosis of generalized muscular degeneration was reached. However, because of the autolytic changes present and the significant potential impact on this vivarium, further live specimens were requested for examination, sacrifice, and necropsy. Four more animals were submitted, alive, for complete necropsy examination. In all cases the owner/breeder noted a marked decline in appetite and activity, with waxing and waning multifocal cutaneous red to white discolorations. Table 1 indicates weights and the distribution of histological lesions. In case 1 there were multiple focal regions of myocardial mineralization. In addition, there were severe, extensive, multifocal to coalescing areas of skeletal muscular degeneration and necrosis (Fig. 1) with loss of striations, individual fiber hypereosinophilia, fiber fragmentation, flocular degeneration, and a moderate increase in endomysial fibrous tissue. Focal areas of skeletal muscle mineralization were present (Fig. 2). Inflammatory infiltration was notably absent. More severe changes were present in case 2, in which distinct fiber dissolution and mineralization was common. Although the affected muscle distribution varied between individual cases (Table 1), all cases displayed marked myofibrillar degeneration. In addition, corresponding to the gross appearance of

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Table 1. Necropsied cases of *Uroplatus phantasticus*. The sex and body mass of individual unfixed animals and the main distribution of myofibrillar degeneration and necrosis are indicated.

Case no. and sex	Body mass (g)	Incidence and distribution of dermal granulomas	Main muscle regions affected
1, Male	3.4	numerous and generalized	forelimb, myocardium, and tongue
2, Female	2.9	occasional, tail, and ventral inguinal region	severe and generalized; most marked in paraveterbral and tongue musculature
3, Male	2.7	none noted	severe and generalized
4, Female	2.4	occasional, tail	severe and generalized

the focal skin lesions, there were multiple granulomas within the dermis, composed of abundant accumulates of histiocytes with lesser heterophilic infiltration, and a surrounding often angiocentric infiltration of plasma cells and lymphocytes. Further staining of the granulomas with modified acid fast, Giemsa, periodic acid–Schiff, and gram stains were consistently negative. Mycotic and aerobic cultures were attempted on multiple subcutaneous lesions and were negative in all instances.

A final diagnosis of multifocal to generalized skeletal muscular degeneration was reached. The lesions noted were consistent with those reported commonly in cases of nutritional myopathy due to vitamin E/selenium deficiency. Similar lesions, including the minimal inflammatory infiltrates and low levels of mineralization are consistently noted in salmonids fed vitamin E/selenium–deficient diets and in intensively cultured fish fed rancid fats.[8] A report of nutritional myopathy in iguanas found multifocal muscular degeneration with fibrous replacement of fibers and variable degrees of inflammation. Other causes of myopathy included those caused by protozoan or metazoan parasites[9]—none of which were noted in the current cases.

In this case, colony and individual animal size restrictions ruled out the possibility of further sacrifices for selenium and vitamin E assays that would have required the sacrifice of a significant proportion of the vivarium. The vivarium owner/breeder was requested to consider the diet, and any management changes associated with the animals, including the use of potential toxins such as pesticides. The geckoes were fed a diet primarily of live or frozen crickets, tropical fish flakes, and various scraps of fresh vegetables and fruit. Isopods were fed to the geckoes on a weekly basis and when in season, liver snails and slugs were offered. Until approximately 2 months before this disease outbreak, waxworms (raised by the vivarium owner) were also fed to the geckoes regularly. When raised, waxworms are fed a diet composed almost entirely of honey and wheat germ. Wheat germ in particular is a rich source of vitamin E, and its removal was considered a highly likely cause of nutritional myopathy. Empirical vitamin E supplementation was implemented, and there have been no further deaths or lesions. Based on response to treatment and the history of the dietary modification, the diagnosis was extended to nutritional

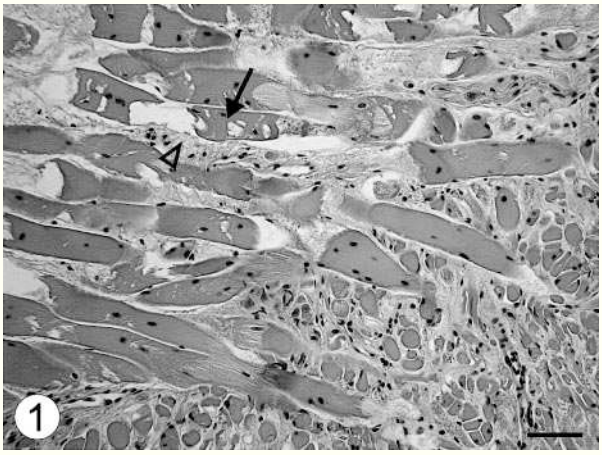

Figure 1. Skeletal muscle from the epaxial musculature of *Uroplatus phantasticus*, case 2. There is extensive fragmentation, degeneration and necrosis of myofibers (arrow) with interstitial fibrosis (open arrow head). Note the minimal inflammatory response. Bar = 75 μm. Hematoxylin and eosin stain.

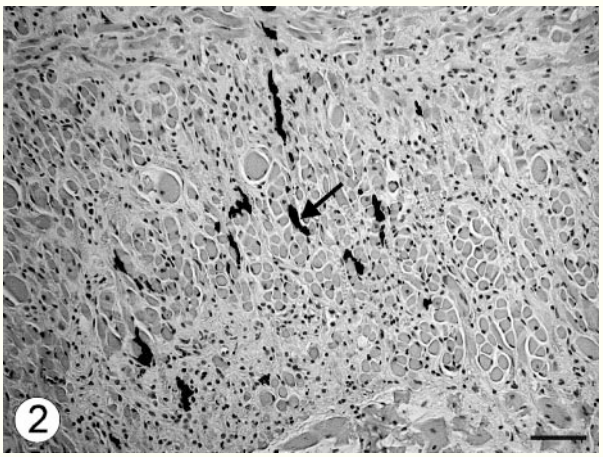

Figure 2. Section of tongue from *Uroplatus phantasticus*, case 1. There is mineralization (arrow) of myofibers, with minimal inflammatory response. Bar = 150 μm. Von Kossa stain.

myopathy, consistent with vitamin E/selenium deficiency. Cost restraints excluded the possibility of quantitative vitamin E and selenium analysis of the diet.

Uroplatus phantasticus are uncommon to those outside of herpetological circles because of their fastidious habits and relative rarity outside of their natural habitat of Madagascar. To the author's knowledge, this is the first report of this species in the English language veterinary literature. Anecdotally, nutritional myopathy is not uncommon in captive reptiles; however, it is more likely noted in monitors and snakes. (M. M. Garner, personal communication) The literature contains very few reports of white muscle disease in reptiles, and typically the final diagnosis is tentative or corroborated with a combination of clinical signs, histopathology, and if appropriate, response to treatment.[7,15] The etiology of the dermal lesions remains uncertain. It is possible, however, that the low levels of vitamin E/selenium led to multifocal, localized adipose tissue necrosis beneath the surface of the epidermis. Why the lesions would appear so focal, as opposed to more generalized, is unclear. Alternatively, it has been shown that vitamin E/selenium–deficient lambs have reduced lymphoproliferative responses in vitro.[17] It is plausible that such a reduction could be involved in the formation of these lesions in other species, including reptiles.

This case highlights the challenges associated with diagnosis of vitamin E/selenium deficiency in nonproduction species. In the more traditional veterinary production species, defined ranges and testing procedures are generally available, and detailed reports of both spontaneous and experimentally induced disease exist. With regard to exotic/nonproduction species, numerous challenges exist in arriving at a definitive diagnosis. In many instances, such as this case report, there is an almost insurmountable challenge related to the size of the specimens. To harvest sufficient liver at necropsy or blood antemortem would require the sacrifice of large numbers of animals. Second, although normal ranges exist, they are often based on small numbers of animals and are of questionable value.

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