Reprint from the JOURNAL of the American Veterinary Medical Association, Vol. 149, No. 7, Oct. 1, 1966, pp. 912-914.

Hypervitaminosis D in Green Iguanas

J. D. Wallach, B.S., D.V.M.

Summary

Two green iguanas, with a dietary supplement of vitamin D, calcium, and phosphorus, had gross and microscopic lesions of hypervitaminosis D characterized by massive deposition of calcium salts in the media of elastic arteries and terminal nephritis.

Hypervitaminosis is a malady that should be kept in mind in dealing with captive wild animals, especially when exact nutritional requirements are not known.

NUTRITION in captive wild animals is an important consideration in zoological parks, since the provision of natural foodstuffs for several hundred different species presents economic and storage problems. As a result of the difficulties in procuring natural foods, most of these animals are given domestic equivalents which are often supplemented with vitamins and minerals.

Nutritional difficulties most often appear as deficiencies or as a state of imbalance of micronutrients; however, hypervitaminosis can be as devastating to animals as hypovitaminosis.

Field cases of hypervitaminosis D have been recorded in cats,[6] calves,[6] cows,[3] dogs,[9] and an iguana.[7] The exact pathogenesis of hypervitaminosis D is not yet documented; however, it is known that the biochemical lesion is characterized by an increase in the serum[1,6,10] and urine[6] calcium and phosphorus levels. Vitamin D administered orally enhances both the absorption of calcium and phosphorus from the intestine[4,6] and resorption of calcium by the proximal convoluted tubules of the kidneys.[5] When there is excessive vitamin D, the amount of calcium and phosphorus in the gut then affects the degree of intoxication.[1,6] Vitamin D given by injection mobilizes the calcium stores of the skeletal system[2] and does not appear to affect intestinal absorption.[2]

Microscopically, there is a deposition of calcium salts in soft tissue,[1,6,7,10] especially in the fibroelastic components[1,3,6,7,10]; renal tubular necrosis with calcification[3,6]; and osteodystrophy that does not appear related to hyperparathyroidism[6] since analysis of parathyroid glands from a cow with hypervitaminosis D revealed a hormonal activity of only one-thirteenth that of its normal counterparts.[3] Grossly, there is fragility of bones,[2,6] calcification of soft tissue,[1,6,7,10] and nephritis[1,3,6] that is the immediate cause of death.

Case Report

Eight green iguanas (*Iguana iguana*) were displayed in a cage with 17 other lizards, including Cunningham's, shingle-backed, and blue-tongued skinks. Incandescent lighting was provided. The animals were fed on Monday, Thursday, and Saturday. For the iguanas, the ration for each of these 3 feedings was composed of ground horse meat mixed with chopped fresh vegetables, fruits, and greens topped with ½ teaspoonful of a vitamin and mineral supplement* that contained 225,000 units of Vitamin D_2 and 86,000 units of vitamin D_3 per pound. Calcium was a minimum of 28% of the supple-

From the Center for the Biology of Natural Systems, St. Louis Zoological Park, St. Louis, Mo.

Research was supported by the Center for the Biology of Natural Systems under PHS Grant No. ES00139.

*Rib-Ad No. 2, Vitamineral Products Co., Peoria, Ill.

ment and phosphorus 5%. The remaining reptiles in the cage were given ground horse meat, young mice, and chopped greens containing ½ teaspoonful of the supplement.

Two male iguanas died 11 months apart, with no history of clinical illness; they had been in the collection 4 years. The gross changes of the 1st iguana were recorded by an assistant curator[5] of the St. Louis Zoological Park. Necropsies of both iguanas revealed profound changes in the arteries and kidneys (Fig. 1, 2). The aortas grated when cut due to calcified plaques up to 2 mm. in diameter from their origin at the base of the heart to their terminal division. The general course posterior was tortuous (Fig. 1) rather than straight, and the intimal surface was dimpled. The pulmonary (Fig. 2), renal, and iliac arteries were similarly affected.

The kidneys were enlarged 2 to 3 times, and their cut surface varied from a hyaline to a ground-glass appearance. Samples of bone were not taken.

Microscopic examination of the aorta and other elastic arteries revealed massive deposition of calcium salts in the media (Fig. 3), making complete rings in some areas. The kidneys had swollen mitochondria, pyknotic nuclei of tubular epithelium, and cellular casts impregnated with calcium salts.

A diagnosis of hypervitaminosis D was made.

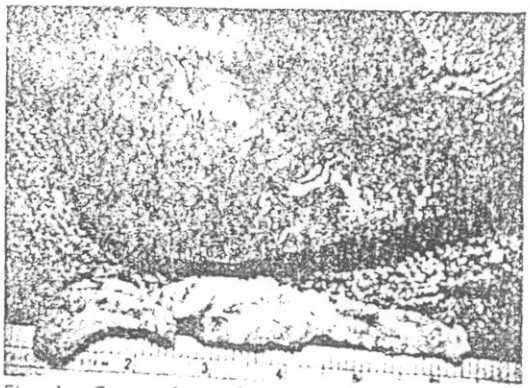

Fig. 1—Cut surface of swollen kidney from a 4-year-old male green iguana with hypervitaminosis D showing ground-glass appearance and abdominal aorta from same iguana revealing irregular course and calcified plaques.

Fig. 2—Heart and lungs from iguana described in Figure 1 revealing massive calcification of the aorta and pulmonary arteries. Notice the "clay-pipe" appearance of the pulmonary branches in the lung parenchyma.

Fig. 3—Photomicrograph of a segment of the aorta described in Figure 1 revealing deposits of calcium salts in the media. H&E stain; x 105.

Discussion

A previous case of hypervitaminosis D in an iguana[6] in a collection for 6 years resulted from dietary supplementation with a vitamin and mineral preparation. The 2 iguanas cited in this report had been in the collection for 4 years. The remaining animals in the cage were new arrivals of a year or less and were not clinically affected.

It appears that long term administration of vitamin and mineral supplements containing vitamin D, calcium, and phosphorus are deleterious to green iguanas.

A condition that may grossly mimic hypervitaminosis D in iguanas is extensive aortic deposits of cholesterol[9]; demonstration of calcium salts or cholesterol clefts microscopically will easily differentiate the 2 conditions.

References

[1]Ames, R.: Fat-Soluble Vitamins. Ann. Rev. Biochem., 27, (1958): 382.

[2]Bro-Rasmussen, F., and Hjarde: Fat-Soluble Vitamins. Ann. Rev. Biochem., 30, (1961): 456.

[3]Cole, R., Chamberlain, D. M., Hibbs, J. W., Pounden, W. D., and Smith, C. R.: Vitamin D (Viosterol) Poisoning in a Cow, from a Study on the Prevention of Parturient Paresis. J.A.V.M.A., 130, (April 1, 1957): 298-299.

[4]Gloor, U., and Wiss, O.: Fat-Soluble Vitamins. Ann. Rev. Biochem., 35, (1964): 318-319.

[5]Hoessle, C., assistant curator, St. Louis Zoological Park, St. Louis, Mo.: Personal communication, 1966.

[6]Jubb, K. V. F., and Kennedy, P. C.: Pathology of Domestic Animals. Academic Press, London (1963): 23-25.

[7]Pallaske, G.: Hypervitaminosis D in a Lizard. Berl. Münch. tierärztl. Wchnschr., 74, (1961): 132.

[8]Pitt, G. A. J., and Morton, R. A.: Fat-Soluble Vitamins. Ann. Rev. Biochem., 31, (1962): 500.

[9]Reichenbach-Klinke, H., and Elkan, E.: The Principal Diseases of Lower Vertebrates. Academic Press, London (1965): 538-539.

[10]Smith, H. A., and Jones, T. C.: Veterinary Pathology. Lea & Febiger, Philadelphia, Pa. (1961): 682.