A Fresh Look at Metabolic Bone Diseases in Reptiles and Amphibians

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Metabolic bone diseases (MBDs) are a group of conditions that continue to plague and perplex veterinarians and pet owners. Although the reptile and amphibian (herptile) veterinary knowledge base has grown understanding of the causes and diagnostic and treatment options are often extrapolated from human or other mammalian medicine models. Although the roles of UV-B radiation (280–315 nm wavelength), calcium, phosphorus, and cholecalciferol (vitamin D3) are better understood in some MBDs, there remain many “X” factors that are not. Likewise, quantitative diagnosis of MBDs has been difficult not only in terms of staging a disease but also regarding whether or not a condition is present. Treatment options also present challenges in corrective husbandry and diet modifications, medication/modality selection, and dosing/regimen parameters.

Green iguanas (Iguana iguana) have provided the classic presentations of the most commonly seen MBDs, nutritional secondary hyperparathyroidism (NSHPT) and renal secondary hyperparathyroidism (RSHPT), for many years. Although the popularity of the green iguana as a pet has waned, other herptiles have now filled the void, with MBDs still a common primary presentation to veterinarians for those animals. Lizards and chelonians are the most common groups represented, but snakes, anurans, and urodeles/caudates may also develop and present with MBDs.

With a switch in the common species presenting with MBDs comes new challenges in how to identify and how to correct these diseases. I have seen MBDs in bearded dragons (Pogona vitticeps), Asian water dragons (Physignathus cocincinus), various chameleon species, leopard geckos (Eublepharis macularius), uromastyx (Uromastyx spp), Testudo spp, and Geochelone spp. Instead of the typical diurnal basking, herbivorous green iguana habitat, the entire spectrum of potential environments and dietary

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options now needs to be addressed for MBDS. Although the classic presentation of rubber jaw and popeye arms and legs is noticeable in some species, in other species there are myriad clinical signs to be aware of.

Radiographs and initial blood work are the standards to start better quantifying the diagnosis, but it is critical to try to apply evidence-based medicine techniques to initially assess, diagnose, treat, and prevent these conditions if veterinary medicine is to advance in addressing them. Anecdotal reports are often helpful as starting points, but peer-reviewed scientific studies will continue to be necessary to support and validate theories, test results, and treatment regimens or to debunk those that are ineffective or even potentially harmful. Where possible, this article references such studies, few though they are at this time. In other cases, well-respected anecdotal herp veterinary articles or review articles are referenced, with extrapolations directly from human/mammalian studies and personal anecdotal observations referred to at times as necessary.

CALCIUM HOMEOSTASIS

Although each MBD is different, there are some basic concepts that address the physiology behind why these diseases occur. It is critical for this to be understood by veterinarians, veterinary staff, and owners, particularly in terms of future prevention. Fig. 1 shows these basics facts of calcium/phosphorus/cholecalciferol homeostasis and how when one part goes wrong, the ramifications affect the other parts and even parts outside the standard system. Fig. 2 is a handout provided by me to all herp-owning clients, whether or not their pet has an MBD. Readers are welcome to contact me for a copy of this to use with their own clients, as long as appropriate credit is provided and it is not modified.

As a review, calcium is a mineral that needs to be consumed in a herp’s diet (perhaps as part of the environmental water). Calcium is critical for bone development

Fig. 1. Calcium and cholecalciferol homeostasis and disease pathophysiology.