RENAL DISEASE IN THE GREEN IGUANA, *Iguana iguana*

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Abstract. As green iguanas, *Iguana iguana*, continue to live for longer periods, the incidence of renal disease also increases. Although this may occur at any age, it is more commonly seen in adult iguanas. There are many predisposing factors, and probably many that have not been identified to date. Evaluation of clinical presentation, physical examination, diagnostic evaluation, biopsy techniques, and treatment will be discussed.

Key words: green iguana, *Iguana iguana*, renal disease, gout, metastatic mineralization, nephritis, renal biopsy, tophi.

INTRODUCTION

As husbandry and management of pet iguanas improve, the popular green iguana, *Iguana iguana*, is living longer in captivity. Renal disease is one of the more common causes of death in middle-aged and older iguanas, but can occur in reptiles of any age. Many predisposing factors are proposed, and it is likely that some predisposing factors still remain unrecognized. Feeding of diets containing animal protein, excessive protein, excessive vitamin and mineral supplementation, decreased environmental humidity, and lack of appropriate ultraviolet light have all been implicated. Ultimately, the etiology is probably multifactorial. Early recognition of clinical signs and biochemical abnormalities can aid in earlier diagnosis and therapeutic intervention, and also may help the veterinarian to more accurately prognosticate. Appropriate initial dietary and husbandry counseling for juvenile iguanas may also minimize the risk of renal disease and associated complications.

CASE REPORT

Clinical Signs and Physical Examination

Clinical signs associated with renal disease may vary but usually include lethargy, inappetance or anorexia, depression, and general weakness. In some cases, muscle fasciculations (particularly of the digits) can be elicited with activity or by 'pinching' one of the digits. Physical exam will reveal dehydration, as evidenced by the presence of thick ropey saliva. Constipation may be noted, but is generally secondary to compression of the colon by the enlarged kidneys. Rectal palpation should be performed in any patient suspected of renal disease.

Diagnostic Evaluation

Traditionally, uric acid concentrations in blood have been considered the best assessment for renal function in reptiles. For most reptile species, uric acid levels rarely elevate above 6.0-7.0mg/dl (356.0-416.0mmol/L) and since uric acid is eliminated by tubular secretion rather than glomerular filtration, the uric acid will not significantly increase with dehydration alone. While an elevated uric acid still remains a reliable indicator of renal pathology, it may not elevate until the later stages of renal disease. Leukocytosis may be present, but is generally moderate and due to inflammatory changes in the kidneys, unless the renal disease is due to infection, in which case leukocytosis will be severe. Total protein levels will elevate in response to dehydration, and so can further support the diagnosis.
Biochemical abnormalities that may be present along with the changes in the calcium and phosphorous include elevated CPK, often due to muscle breakdown or injury from seizuring; elevated AST (SGOT), which can be released from breakdown of kidney as well as liver parenchyma; and elevations in sodium and chloride, further supporting dehydration.

Further noninvasive diagnostics include ultrasonographic evaluation of the coelomic cavity. Bilaterally symmetrical masses of uniform echogenicity (often hyperechoic) craniodorsal to the pelvic inlet are usually representative of enlargement of the cranial aspect of the kidneys. The kidneys normally do not extend cranial to the pelvic canal. Caudal enlargement can also be detected by imaging the tail base just caudal to the pelvic inlet, at the junction of the legs and the tail.

From this point, diagnostic options vary, but all should be directed towards specific diagnostic evaluation of the kidney. If renal enlargement can be palpated cranial or caudal to the pelvic canal, a fine needle aspirate can perform. Ultrasound can enhance the diagnostic ability of this test. Needle aspirate is the least invasive method to obtain sample of the affected renal tissue, but is also the least diagnostic due to the limited sample size. Further limitations occur if the diseased areas are focally arranged within the enlarged renal parenchyma, as a needle aspirate may not penetrate the diseased areas.

Renal biopsy can be performed endoscopically. This method enables visualization of both kidneys from one approach, enables identification of affected areas and appropriate selection of a site for sampling, and has the added advantage of allowing visualization and evaluation of other organs in the coelomic cavity. The peritoneum covering the kidney must be penetrated to enable biopsy, both of which can be performed using a 3.5 or 5.0mm biopsy instrument (Storz, Inc., Galena, CA, USA). This method is minimally invasive and highly diagnostic, and is the preferred technique for renal biopsy in the author's practice if the kidneys are enlarged cranially. Limitations include a very small sample size and inability to control bleeding from the biopsy site; fortunately, because the biopsy sites are so small, there is generally minimal bleeding.

If endoscopic sampling is not available, or if renomegaly is only evident caudally, then renal biopsy can be obtained via a keyhole approach. For the cranial approach, the skin is incised cranial to the renal pelvis, just dorsal to midline. Blunt dissection through the thin musculature enables rapid exposure of the affected kidney. A vaginal speculum or refractors can be used to maintain the opening during biopsy. The author suggests pre-placing a horizontal mattress suture through the area to be biopsied; an assistant can apply digital pressure laterally on the contralateral side of the body to keep the kidney elevated in the surgical site. For caudal renal biopsy, the tail approach has been described. The incision is made at the proximal aspect of the tail base, just ventral to midline, and involves blunt dissection through the muscles to expose the kidney. Bleeding is controlled by closure of the skin and pressure from the surrounding musculature.

Histopathologic changes can vary. The kidneys may be normal or have mild changes, or may have evidence of tubulointerstitial nephritis, tubular necrosis, and interstitial nephritis with active inflammation, calcification, tophi formation, or fibrosis. Metastatic mineralization or tophi may be present in other organs or in the vasculature, particularly the heart and aorta. It is important to differentiate between metastatic mineralization and tophi formation, as the etiologies and treatments are quite disparate.
Therapy

Treatment is aimed at the underlying etiology, and therapy must be directed toward normalizing biochemical and hematologic abnormalities. If evidence of infection is present, then appropriate antimicrobial therapy should be initiated. Chronic dehydration may play a role, and fluid therapy should be initiated. (Maintenance fluid therapy is 20.0ml/kg q24hr, and fluids should be adjusted to compensate for dehydration and ongoing losses). Intravenous or IO fluids are ideal, but ICe or SC fluid administration are appropriate. If phosphorous is elevated, oral phosphorous binders should be initiated. (Amphojel or aluminum hydroxide is empirically dosed at 1.0ml/kg q12hr q8hr). Feeding of high phosphorous foods such as baby food should be avoided. Often, calcium is severely decreased. Although calcium is low, calcium supplementation should be avoided until the Ca:Phos ratio approaches or is greater than 1:1. This is because of the solubility coefficient and law of mass action - Ca x Phos <or = 50.0-70.0 (9 if measured in SI units); at higher levels, the calcium and phosphorous bind and precipitate, causing mineralization of the great vessels. Administration of calcium at this time may lead to further mineralization. In most cases, aggressive fluid therapy and phosphorous binders, along with calcium, will lead to decrease in phosphorous and increase in calcium. Once the ration approaches 1:1, oral calcium carbonate can be administered. It is a calcium supplement and a phosphorous binder.

Gout is truly an elevation of uric acid, and in some cases can lead to formation of tophi, uric acid deposits in the viscera or joints. The actual elevation in serum uric acid may be temporary and may remain undetected even in the presence of tophi. Chronic dehydration may play a role in development of this condition. Therapy is directed at decreasing further tophi formation and limiting subsequent fibrosis. Fluid therapy should be administered. Dietary management consisting of restricting purines and dietary acidification may be of benefit, although medical management is considered more effective than dietary management. Medications suggested include allopurinol (to decrease uric acid formation), probenicid (promotes urate excretion), and colchicine (to minimize fibrosis of renal and hepatic parenchyma).

During treatment for any etiology, it is important to avoid foods with high protein content (pellets, meat, and meat products); high phosphorous foods (meats, baby food); and vitamin/mineral supplementation. Several enteric formulas have been proposed, or the owners can feed blended leafy green vegetables. Administration of appropriate ultraviolet rays are also essential to recovery.

REFERENCES