ANIMAL EMERGENCY CENTER
at the heart of critical care

2100 W. Silver Spring Drive, Glendale, WI 53209
Phone: (414) 540-6710
Fax: (414) 540-6720
www.animalemergencycenter.com

Animal Emergency Center
Memorandum

To: Referring Veterinary Hospitals

From: Rebecca Kirby, DVM
Date: March 19, 2007

Re: CE Fax Series – Renal Failure in Reptiles

I am pleased to present you with Renal Failure in Reptiles - Part 2, by Dr. Paul Gibbons, specialist in avian, reptiles and small mammals. This paper explains the approach to the diagnosis and treatment of renal failure in your reptile patients. Please feel free to call Dr. Gibbons should you have any questions or comments regarding the CE material attached, or with any questions you may have about your avian, reptile or small mammal patients.

As always, we welcome your input and feedback regarding the CE Fax series or any other aspect of services provided to you and your clients by the staff of the Animal Emergency Center. Please contact Ms. Christina Matthews at 414-540-6710. This and other CE fax series articles can be found and downloaded from our website at www.animalemergencycenter.com.

We have a created a section on our website "What to Expect at AEC" for your clients that come to AEC with an emergency/critical care patient or for a referral appointment. The icon is on the home page. Please take a look and provide us with any feedback that you might wish to share. And please share our web address with your clients and staff to help them get the most from their visit to AEC. Thank you, as always, for your input and support!!!

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RENAL FAILURE IN REPTILES – Part 2

Paul M. Gibbons DVM, MS, DABVP (Avian)
Animal Emergency Center

Renal failure is difficult to diagnose in reptiles. An etiologic diagnosis is required to design an effective treatment plan and predict the prognosis for each individual patient.

DIAGNOSIS

Renal failure in reptiles is diagnosed by analyzing information from the presenting complaint, history, physical examination, minimum objective database, diagnostic imaging, and problem-specific diagnostic testing. Problems identified in the history can include feeding inappropriate protein sources, excessive dietary protein, high oxalate diet (including insects fed high oxalate vegetation), excess or deficient dietary vitamins or minerals, insufficient dietary moisture, inadequate environmental humidity, inappropriate water provision, inadequate ultraviolet B radiation, improper environmental temperatures, recent septicemia, past episode(s) of nutritional secondary hyperparathyroidism, recent exposure to wild-caught reptiles, or recent exposure to nephrotoxins. In addition, renal failure can sometimes occur following improper hibernation in chelonians.

Problems identified in the presenting complaint can include decreased activity, anorexia/inappetence, generalized weakness, paresis/paralysis, long bone fractures, muscle fasciculations (tremors), constipation/obstipation, dyschezia (straining to defecate), dystocia (egg binding), dysecdysis (retained shed), bloat, prolapsed tissue from the vent, chronic weight loss, poor growth, and hematuria. It is very rare for owners to report polyuria or polydipsia. Problems found in the physical examination often support the presenting complaint(s) and may also include an inability to raise the torso (in tetrapods), decreased response to stimuli, coelomic distension with fluid (ascites), palpably enlarged kidneys (per cutaneous or per cloaca), coelomic pain, reduced skin elasticity, ropey saliva, ventral edema, and cachexia. Reptiles with acute renal disease are usually in good body condition, and those in chronic renal failure are often substantially underweight.

A minimum objective database in reptiles includes a fecal direct smear, fecal flotation, complete blood count, plasma biochemistry panel, plasma ionized calcium, urine dipstick, and urine microscopy. Baseline values should be obtained on a regular basis during routine health evaluations for comparison during times of illness. Some "reference ranges" have been published for some reptile species, but wide variation occurs among laboratories, among individuals of the same species, among different species, between sexes, by season, with advancing age, among blood sample sites, and under different environmental conditions. See Table 1 for a list of clinical pathological abnormalities that can occur in renal failure. No hematological or biochemical abnormality can be expected in every case, though increased phosphorus with normal or decreased ionized calcium ("calcium:phosphorus ratio") is often found in lizards with renal failure. Creatinine measurement is not useful in reptiles, but uric acid levels will elevate with severe dehydration, end-stage renal disease, or post-renal obstruction.

Table 1. Clinical pathological abnormalities that can occur with renal failure in reptiles

Parameter	Possible abnormality
RBC count	_ to normal
PCV	_ to _
Regenerative response to anemia	None to slight
WBC	Normal to _
Heterophils	Normal to _
Azurophils	Normal to _
Monocytes	Normal to _
Albumin	Normal to _
Total calcium	_ to _
Ionized calcium	_ to _
Phosphorus	_ to __
BUN	Normal to _
Ammonia	Normal to _
Uric acid	Normal to __
AST	Normal to _
CK	Normal to _
GGT	Normal to _
LDH	Normal to _
K	Normal to _

Thoughtful consideration is required to interpret the urinalysis because reptile urine is not normally sterile, renal urine cannot be concentrated, and extra-renal organs contribute to electrolyte and water balance. Some urine abnormalities that support a diagnosis of renal disease include dark amber color, RBCs, WBCs, renal casts, parasites, epithelial cells, neoplastic cells, glucosuria, abnormal pH for the species, and proteinuria.

It is important to consider the differential diagnoses for each of the problems identified in the above process. For example, elevated plasma phosphorus can also result from excess dietary phosphorus, hypervitaminosis D, severe tissue trauma, osteolytic bone disease, hemolysis, or delayed centrifugation and separation of plasma from RBCs. Problem-specific diagnostic tests provide the data needed to rule differential diagnoses in or out. Urine and blood can be cultured for pathogenic bacteria or fungi. Renomegaly, coelomic fluid accumulation, soft tissue mineralization, skeletal demineralization, or fibrous osteodystrophy might be diagnosed with plain radiographs. Coelomic ultrasonography is used to further characterize soft tissues and fluids, or to safely guide a needle aspirate. Intravenous urography provides contrast radiographs of the urinary tract, and iohexol clearance is a valid test for renal function in green iguanas. In some cases, coelioscopy, computed tomography, or magnetic resonance imaging may be required. Coelomic fluid is characterized using clinical biochemistry tests, refractometry, microcentrifugation, cytological examination, and cultures. Renal biopsy is indicated for all cases of renal insufficiency in reptiles, and may be collected via coelioscopy, coeliotomy, or cranial dorsolateral tail keyhole. Several samples should be collected and submitted for histopathology and aerobic, anaerobic, mycobacterial and fungal cultures.

TREATMENT

Treatment is wholly based upon the etiologic diagnosis. Reptiles in renal failure usually require fluid therapy either enterally by encouraging them to drink in a shallow, tepid water bath, or parenterally though administration of subcutaneous, intravenous, or intraosseous crystalloids. Intracoelomic fluids are not recommended because it is difficult to determine whether the fluids enter circulation, and the location of coelomic structures varies among species, between individuals of the same species, and in the same individual over time. Gastric lavage should be reserved for cases with normal muscle strength. Fluid deficit should be calculated and replaced over 48 to 72 hours at a rate of less than 40 ml/kg/day. Nutritional support is delayed until after rehydration, although enteral fluids may contain simple carbohydrates. Allopurinol is indicated for gout, and antimicrobial drugs are used to treat specific pathogens. Species-appropriate environmental conditions are required for success, and usually include management of lighting, heat, nutrition, water, and humidity. Calcium and vitamin D therapy are guided by clinical biochemistry data and are usually given by mouth. Finally, oral phosphorus binders such as calcium carbonate or glubionate are used for long-term management of hyperphosphatemia.