Avian Kidney Disease

Part III: Treatment of Renal Disease

M. Scott Echols, DVM, Diplomate ABVP - Avian Practice

Director of Avian Medical and Surgical Services, Westgate Pet and Bird Hospital, 4534 Westgate Blvd, Suite 100, Austin, Texas 78745, USA.

Treating avian renal disease is best based on the patient's history, physical examination and laboratory findings and kidney histopathology or a strong presumptive diagnosis. Due to the complex surrounding neurovasculature, the vast majority of renal diseases are managed medically as avian kidney surgery (aside from biopsy) is often contraindicated. Therapeutic options are offered and differ based on the patient and type of confirmed or presumed renal disease present

Avian Renal Disease Treatment

Therapeutic considerations

Treatment options for renal disorders in birds depend upon the cause and type of kidney disease and secondary complications present. Most renal disease patients are medically managed, as kidney surgery is difficult and often not needed.

Because of the location within the renal fossae, avian kidneys are difficult to surgically remove. The close associations with the lumbar and sacral plexuses and extensive vascular network surrounding the kidneys lead to the high probability of significant hemorrhage expected during surgery and possible neurologic damage. With that said, focal therapeutic surgery (including endoscopic biopsy) for superficial renal lesions and the ureters may be useful in some cases. Given the concern of serious hemorrhage, most surgical renal disease cases are managed medically.

A few accounts of therapeutic renal surgery exist. Post-renal failure due to urolithiasis or some other obstruction of the ureters or cloaca may be noted. Cloacaliths and other masses within the cloaca may be easily removed, relieving a potential ureteral obstruction. Wideman and Laverty describe the effects of renal vein and ureter ligation on kidney function in domestic fowl. Except for a 'small island' of tissue adjacent to the testes and cranial renal artery, the cranial, and portions of the middle, renal divisions atrophied significantly without compromising overall kidney function. Such a study is worth reviewing if considering renal division ablation or other similar radical procedures. Renal stones were successfully removed via extracorporeal shock wave lithotripsy in a Magellanic penguin (*Speniscus megalanicus*). Although multiple anesthetic procedures were required, ureteral stones were successfully removed from a 21 year-old male double-yellowheaded Amazon parrot (*Amazona ochracephala*).

The author has also used minor surgery in articular gout cases. In effort to speed the removal of (stabilized) articular gout, make small incisions over the gouty lesions, which are often on the feet. Express the thick material out. Anesthesia is ideal as this can be quite painful. Also, this procedure tends to be bloody and the feet often require minor bandaging to help prevet continued bleeding and

secondary infection.

Another poorly explored area is renal cancer therapy. Clearly as treatment options advance and are tested in avian species, renal cancer therapy will likely become more prevalent. For example, carboplatin at 5 mg/kg IV q 1 month was used to manage a renal adenocarcinoma (diagnosed at necropsy) in a budgerigar. The bird died approximately 3 months after initiating treatment but did temporarily show improvement of clinical signs (decreased grip in one foot and lameness changed to almost normal perching, 1 month after starting therapy). It was concluded that while carboplatin may be nephrotoxic in birds, this drug could possibly be useful in treating early renal tumors that have not progressed to renal failure.

As a general note in any bird with organ dysfunction, patients should be monitored with routine physical and laboratory evaluation, especially when taking any medication(s) chronically. The intervals between recheck examinations will vary on the patient's condition and clinician experience in handling the given case.

Diuresis and fluid therapy

As in other animals with renal disease, maintaining hydration is important in birds with most kidney disorders. Acid/base and electrolyte disorders may likely be present in birds with renal disease. At this time, only general statements concerning diuresis and fluid therapy can be made.

Anuric and oliguric patients should be diuresed. Although manitol and lasix have been recommended to induce diuresis in birds, these drugs are poorly studied in avian species. Wideman et al used mannitol (added to a solution containing inulin and para-amino hippuric acid) to induce diuresis in chickens at a dose of 2.5% given at a rate of 0.2 ml/kg/min. Furosemide given IV (1 mg/kg BID) along with SQ saline for 72 hours was used to successfully treat a red-tailed hawk (*Buteo jamaicensis*) with acute obstructive uric acid nephropathy. Some birds, especially lories, may be sensitive to the effects of furosemide and its use should be judicious. Furosemide may also cause increased urinary excretion of Na^+, K^+ and Cl^-. If furosemide is used, electrolyte replacement may be needed. Clinically, providing parenteral fluids often induces diuresis in birds, even with most forms of renal disease.

Until acid/base and electrolyte disorders are better evaluated in birds with renal disease, balanced electrolyte solutions, such as Lactated Ringer's Solution (LRS), should be used to maintain hydration, replace fluid losses and/or induce diuresis as needed. The estimated daily fluid requirement for most birds is 40-60 ml/kg/day. Lumeij recommends that 10% of the bird's body weight should be given in fluids when the patient is in renal failure. Once a dose has been determined, warmed fluids are given either with food (tube/syringe fed), SQ, IV or intraosseously (IO). The IV and IO routes are most appropriate for critically ill patients. While appropriate in many cases, subcutaneous fluids are not adequate to rehydrate patients with severe dehydration, shock or hypothermia. Oral fluids are reserved for stable patients with mild dehydration that have normal gastrointestinal function and are contraindicated in critically ill birds.

Lactated Ringer's Solution is the fluid of choice for most critically ill birds. Besides providing fluid support, the lactate in LRS is converted to bicarbonate in the avian liver. Bicarbonate helps to reverse acidosis, which is common in critically ill patients.

The author typically diureses ill and severely hyperuricemic renal disease patients. While the definition is debatable, the author generally considers 'severe hyperuricemia' is present when one or more of the following conditions are met in clinically ill non-carnivorous and appropriately fasted carnivorous birds:

1. uric acid levels exceed 30 mg/dl
2. uric acid levels are elevated (> 10 mg/dl for most species) and rising over a period of several days (even if below 30 mg/dl)
3. there is evidence of rapidly progressive articular or visceral gout

Depending on the patient's condition, the author will typically give 50-100 ml/kg of fluid BID via the SQ, IV, IO or combination routes. Fluid therapy (combined with other medications if needed) is generally continued until the blood uric acid level drops to either normal or mildly elevated levels (10-20 mg/dl) and the bird is showing signs of improvement (eating, more active, etc). Lower amounts of parenteral fluids are given if overhydration is either suspected or a concern.

Antibiotics

Antibiotics are indicated in patients with known or suspected bacterial nephritis. Bacterial renal infections in birds may result from an ascending ureteritis, extension from local tissues (periotonitis, oophoritis, salpingitis, etc) and hematogenously. Because of the renal portal system and possible shunting of blood from the intestines directly to the kidneys, alimentary tract organisms may contribute to kidney disease and should be considered when using antimicrobial therapy. Drug choices are based on an isolated renal organism (ie: identified during kidney biopsy sampling) or a suspected infectious agent (blood, ovarian, salpinx, or cloacal/fecal cultures and/or supportive histopathology). Clinical consideration regarding potential antimicrobial-induced toxicities is important.

The distribution, elimination and toxicities of many antimicrobials are poorly defined in most bird species. Frazier et al has provided an excellent review of antimicrobial use in birds with specific consideration toward the renal system. Although mammalian literature warns of potential nephrotoxicity with amphotericin B, cephalosporin, flouroquinolone, trimethoprim/sulfonamide and tetracycline use, only aminoglycosides have been consistently and definitively associated with renal disease in birds. Those drugs with known potential nephrotoxicity should be used cautiously in birds with renal impairment. Until additional studies are completed in birds, antimicrobials that reach high concentrations in the renal tissue and urine without inducing toxicity should be chosen and used cautiously in kidney disease patients.

The ideal duration recommendable for treating renal infections has not been established in birds. In cats and dogs, greater than 4 to 6 weeks of antimicrobial use is generally recommended for treating bacterial kidney infections. The author's clinical experience with bacterial nephritis suggests that response is best when a minimum of 6 weeks of antibiotic therapy is administered. These suggested guidelines are based on renal histopathologic evaluation supporting the presence of infectious nephritis, post treatment resolution of clinical pathology abnormalities and improved follow-up kidney biopsy and histopathology in a small number of avian renal disease cases. There are no controlled studies evaluating antibiotic therapy in active bacterial nephritis cases in birds. Additionally, the author will generally treat concurrent colitis (based on culture and sensitivity results of fecal and/or cloacal cultures) for 5-7 days, or until signs abate, in renal disease patients.

Managing hyperuricemia, renal fibrosis and amyloidosis

Allopurinol:

Allopurinol's main action is to decrease uric acid production. Specifically, allopurinol inhibits xanthine oxidase, which is required to convert hypoxanthine to xanthine and subsquently to uric acid. In chickens, xanthine dehydrogenase, closely related to xanthine oxidase, is the actual enzyme used in this pathway. Allopurinol has been specifically shown to prevent renal synthesis of urates and allow the

excretion of unchanged xanthine. Regardless, both clinical and experimental data show decreased plasma/serum and/or urinary uric acid levels in birds treated with allopurinol. Interestingly, allopurinol does not appear to affect pancreatic xanthine dehydrogenase activity suggesting differing mechanisms of uric acid metabolism in the pancreas and kidney.

Specifically in red-tailed hawks (*Buteo jamaicensis*), allopurinol has been shown to be toxic at 50 mg/kg PO SID with clinical signs of vomiting and laboratory supported significant hyperuricemia and a 'renal function disorder'. The renal toxicity was even worse and included visceral gout when red-tailed hawks were given 100 mg/kg followed by 50 mg/kg of allopurinol. The toxic signs were attributed to oxypurinol, the active metabolite of allopurinol. Allopurinol given at 25 mg/kg SID PO to red-tailed hawks was shown to be safe, but had no significant effect on plasma uric acid concentrations. The authors concluded that allopurinol has a very low therapeutic ratio, at best, in red-tailed hawks and that other means of controlling hyperuricemia, such as urate oxidase, in this species should be considered. ith the exception of red-tailed hawks, allopurinol use is reported to be non-toxic in birds (in studied chickens), including chicks. Although the long-term effects are not clear, allopurinol given to chickens increases oxidative activity by lowering plasma uric acid, an important avian antioxidant.

The author uses allopurinol as a first-line drug to lower uric acid when fluid therapy and diet modification alone are not sufficient or when hyperuricemia is severe. Clinical experiences suggest that allopurinol is safe to use at published doses in Psittaciformes and Columbiformes, even when used chronically (3-6 months +). Because of the noted toxicities in red-tailed hawks and until further studies are conducted, it is reasonable to assume that allopurinol should be used judiciously, if at all, in birds of prey.

Colchicine:

Theoretically, colchicine can reduce serum uric acid levels in birds and be used to control hyperuricemia. In chicken livers, colchicine reversibly inhibits xanthine dehydrogenase (compared to a 'pseudo-reversal' with allopurinol). Colchine prevents the progression of renal disease in humans with familial Mediterranean fever, a disease of recurring fever often complicated by amyloidosis. In humans, colchine is best known for its anti-gout activity. In small animals, colchicine blocks the synthesis and secretion of serum amyloid A and decreases the formation and increases the breakdown of collagen. For these reasons, colchicine has been used to treat amyloidosis and hepatic fibrosis, respectively.

Clinical use of colchicine suggests possible benefit in reducing hyperuricemia in birds with renal disease. The author has also used colchine to reduce renal (and hepatic) fibrosis in birds and has had good success based on pre- and post-treatment tissue biopsies. As such, the author uses colchicine as a second-line drug to reduce hyperuricemia and a primary medication for histologically confirmed tissue fibrosis. Allopurinol and colchine are well tolerated when given together in most birds. If diagnosed pre-mortem, colchicine may be used in birds with amyloidosis. No controlled studies were found using colchicine in birds with renal disease.

Urate Oxidase:

Urate oxidase has also recently been discussed as an alternative method to manage hyperuricemia in birds. At least in humans, urate oxidase is reported to degrade the excess of uric acid to allantoin, which the kidneys can more easily be clear than uric acid. Urate oxidase is also very specific for urates and uric acid and does not interfere with the metabolism of purines as does allopurinol. In one study, 200 and 600 U/kg and 100 and 200 U/kg of urate oxidase were given IM to pigeons and red-tailed

hawks, respectively. When compared to controls, all dosing regimens caused a significant decrease in plasma uric acid concentrations within 2 days of the first dose. The authors concluded that 'urate oxidase is much more effective compared with allopurinol' but, this promising drug needs further evaluation to better understand it's use and potential long-term effects.

Dietary modification

As a general note, birds should be fed diets appropriate for their species. Supportive dietary therapy should always be considered in any anorexic patient. As is true with all sick birds, renal disease patients should be weighed routinely at regular intervals and monitored for weight loss.

Protein:

The question of dietary protein restriction in the face of renal disease remains controversial. The current human and veterinary literature sites arguments for and against both restriction and supplementation of protein with renal disease patients. The current human literature sites malnutrition (potentially from protein-restricted diets) as 'the most potent predictor of death in end-stage renal failure'. The resultant recommendation is that patients on protein-restricted diet should be well supervised and provided adequate calories.

Although feeding 20% protein to chicks, including young cockatiels, has been recommended as a general level for normal development, excessive protein intake for birds with renal disease has not been determined. Feeding diets consisting of 60% and 80% protein (2 separate studies) were required to induce articular gout in genetically predisposed chickens. In a study using adult cockatiels, birds fed up to 70% protein for 11 months had no evidence of visceral or articular gout or significant renal lesions. This led the authors to the conclusion that in cockatiels, 'high dietary protein levels are not associated with kidney dysfunction'. These experimental diets represent unnaturally high protein levels and do not serve as a realistic evaluation of the effect of diet on renal disease and/or gout in birds.

The management of hypoproteinemia may also be important in birds with renal disease. The identification of hypoproteinemia and association with renal disease in birds is unclear.

Until further research better defines the role of dietary protein needs in relation to renal disease, avian kidney disease patients should be fed a well-balanced diet appropriate for their respective species. If instituted, birds fed protein restricted should be carefully monitored. No current studies evaluate the effect of low or high protein diets in birds with naturally occurring renal disease were available at the time of writing. A safe recommendation is that birds with hyperuricemia and/or gout should not consume diets with protein levels greater that what is considered normal for the given species.

Nutritional supplementation

Treatment: omega-3 fatty acids:

Omega-3 fatty acids (N-3 FA) have gained popularity for their anti-inflammatory, lipid-stabilizing and anti-neoplastic effects, renal protective properties and other potential qualities. N-3 FA are polyunsaturated and are designated by their first carbon-carbon double bond occurring at the third carbon from the methyl group. N-3 FA are those rich in eicosapentaenoic (EPA), docosohexaenoic (DHA) and/or linolenic acid. Flax seed and menhaden (cold water plankton-feeding fish) oils contain predominately linolenic acid and EPA and DHA, respectively and therefore have different N-3 FA compositions. DHA and EPA are more readily incorporated into biological tissues, but also carry greater potential to create metabolic oxidative stress than linolenic acid. The clinical impact of the

differences of the various N-3 FA has not been clearly defined.

Studies evaluating N-3 FA in mammals serve as the basis for potential treatment value in birds with selected renal disease. At this time, only anecdotal information exists regarging use of N-3 FA in birds with renal disease.

In mammals, N-3 FA can significantly reduce thromboxane A_2 (TXA) synthesis in platelets and glomerular cells and increase production of vasodilatory prostaglandins. N-3 FA partially substitute EPA and DHA acid for arachidonic acid in membrane phospholipid. This pathway decreases the release of arachidonic acid and, subsequently, the cyclooxygenase-mediated synthesis of TXA. In contrast, most animals readily convert omega-6 fatty acids (N-6 FA) to arachidonic acid and subsequently, eicosanoids (prostaglandins, TXA). As with arachidonic acid, EPA also serves as a substrate for the formation of vasodilatory prostaglandin/cyclins (PGI/PGE) and their respective products (PGI_2 /PGE_2 and PGI_3 /PGE_3) all of which have similar biologic potency. These vasodilatory prostaglandin/cyclins increase renal blood flow and single nephron GFR.

In humans and rats supplemented with N-3 FA for at least 4 to 6 weeks, single nephron GFR, plasma flow and renal blood flow increased and/or decreased renal vascular resistance occurred. In a separate evaluation, dogs on a low-fat diet supplemented with N-3 FA had preserved renal function and structure when induced with renal disease. Another study found that N-3 FA supplementation reduced glomerular capillary pressure and prevented deterioration of GFR in dogs with renal disease. Compared with controls and thromboxane synthetase inhibitor–treated dogs, beagles supplemented with N-3 FA demonstrated increased renal production and excretion of $PGE_{2/3}$, which was believed to have stabilized renal tubular lysosomal membranes. These N-3 FA supplemented dogs had decreased gentamicin-induced proximal tubular necrosis when compared to controls.

Specific 'toxicities' associated with N-3 FA supplementation are poorly described but some potential adverse effects may occur. Chickens fed diets high in N-3 FA have had reduced plasma and tissue vitamin E (the body's primary anti-oxidant) and plasma carotenoid levels due to lipid peroxidation. Therefore, supplementing the diet with N-3 FA increases the requirements for dietary vitamin E. As supported by clinical investigations, vitamin E supplementation should be considered with use of N-3 FA or any other polyunsaturated fatty acids. Specifically, 160 mg/kg of vitamin E (dl-α-tocopheryl acetate) was shown to prevent loss of α-tocopherol in tissues and normalize or increase resistance to lipid peroxidation in chickens fed a commercial diet supplemented with 3% tuna oil (N-3 FA).

Other potential side effects may be noted with N-3 FA supplementation in birds. Menhaden oil supplementation in laying chickens has been shown to contribute to hepatic lipidosis, likely via enhancing the lipogenic activity (along with estradiol) of the liver. This single study cautions the use of N-3 FA in reproductively active hens. In another study, chickens fed diets high in N-3 FA had no alteration in primary or secondary humoral response, but experienced a 50% reduction in antibody dependent cell cytotoxicity (ADCC). The concern presented therein was that reduction in ADCC-related immune functions might increase a patient's susceptibility to certain disease (Marek's). N-3 FA supplementation may also affect the ability of antigen-presenting cells to present antigen, again suggesting the potential for immune system alteration. An increased incidence of infectious disease in birds has not been definitively associated with N-3 FA supplementation.

Although specific doses have not been established, some believe that the appropriate N-6 to N-3 FA ratio is more important to inhibiting eicosanoid synthesis from arachidonic acid than is the absolute amount of N-3 FA. A dietary N-6 FA: N-3 FA ranging from 5:1 to 15:1 has been proposed as desirable for dogs and cats with renal disease. Using the above dietary guideline, 2 to 4 weeks are required to see any initial effects of the dietary change in dogs and cats. One study in chickens showed that

maximal N-3 FA tissue (egg yolk) levels were obtained after 3 to 4 weeks of supplementation. Long-term supplementation (3 to 6 months or more) is likely appropriate if N-3FA are to be used.

The author has successfully used supplements containing N-6 FA: N-3 FA of 4-5:1 to 1:3 (0.22 cc/kg body weight, PO, SID) combined with low-dose aspirin (0.5- 1.0 mg/kg PO q 12 h) together to manage histologically confirmed glomerulopathies in avian patients. 'Success' was gauged on normalized hyperuricemia, (4/4), improved clinical appearance (3/4) and repeat renal biopsy showing normal glomerular light microscopic histology (1/1) in an African grey parrot (*Psittacus erithacus erithacus*), citron-crested cockatoo (*Cacatua sulphurea citrinocristata)*, red-lored Amazon parrot (*Amazona autumnalis*) and a ring-neck dove (*Streptopelia risoria*). The author has also used a supplement containing N-6 FA: N-3 FA of 1:3 (0.22 cc/kg body weight, PO, SID) alone to manage various forms of renal disease in mixed avian species with no recognized adverse side effects. Unfortunately no clinical trials using fatty acids in avian renal disease were found, only anecdotal reports such as noted here.

Vitamin A:

Parenteral vitamin A has been recommended in birds with renal disease. Hypovitaminosis A is a reported cause of renal failure and results from metaplasia of the ureters leading to hyperkaratinization, decreased mucin production and impaction. In birds with suspected hypovitaminosis A and renal disease, appropriate diet modification and short-term parenteral vitamin A is a logical component of therapy. In such situations, the author gives a single IM vitamin A injection at the beginning of the therapy and recommends correcting the patient's diet to improve long-term nutritional status.

Non-steroidal anti-inflammatories

Non-steroidal anti-inflammatory drugs (NSAIDS) are frequently discussed for use in human and animal renal disease patients. In general, NSAIDS such as aspirin and ibuprofen are non-specific cyclooxygenase inhibitors. Low doses of aspirin may actually inhibit platelet cylcooxygenase production but allow beneficial (vasodilatory) prostacyclin formation and may be safe. Consequently, low dose aspirin therapy has been suggested to reduce platelet aggregation and subsequent thromboembolism and to minimize glomerular inflammation for mammalian patients with some glomerulopathies. More specific NSAIDS, such as thromboxane synthetase inhibitors, have been shown to attenuate renal dysfunction/damage as noted by one or more of the following: decreased proteinuria, enzymuria and tubular necrosis and preserved renal blood flow and GFR in various animals with a variety of renal diseases. Unfortunately, the beneficial effects of low dose, or specific, NSAID therapy have not been studied in birds with renal disease.

Although there are limited avian studies, most NSAIDS are eliminated by renal clearance and should be used with caution, as they have been associated with a variety of renal lesions in birds and mammals. Flunixin meglumine (Banamine®) induced glomerular lesions in bobwhite quail (*Colinus virginianus*) that increased in severity proportionally with the dose. In this short study, no biochemical or electrolyte parameters were altered, but uric acid was not measured. Aspirin has been associated with significant inhibition of prostaglandin synthesis (specifically prostaglandin $F_{2\alpha}$) in Japanese quail. In this same experiment, aspirin was shown to induce liver enlargement resulting from hepatic lipid accumulation in N-6 FA deficient Japanese quail. Acetylsalicylic acid (aspirin) injected IV into Pekin ducks induced temporary diruresis lasting 30 minutes, which is in contrast to the antidiuretic effect seen in mammals, and had no effect on GFR or peripheral blood pressure. Several *Gyps* spp. of vultures have died with renal failure and gout as a direct result of consuming diclofenac-treating livestock. The veterinary use of diclofenac has been specifically proposed as responsible for the decline of the critically endangered Oriental white-backed vulture (*Gyps bengalensis*) in Pakistan. These scattered studies only serve to

point out potential varied effects of NSAIDS in birds.

Even with the noted toxicities and lack of therapeutic studies in birds, the author feels that low dose aspirin, and possibly other NSAIDS, use can be beneficial in avian kidney disease patients. In the authors's experience, low dose-aspirin (0.5- 1.0 mg/kg PO q 12h) combined with N-3 FA supplementation is safe and may be effective at reducing the severity of some forms of avian renal disease, especially glomerular disorders. Aspirin (and N-3 FA) therapy can be used chronically and the author discontinues use once evidence of renal disease is gone or the disorder is satisfactorily managed.

Treatment summary

Treatments of avian renal disease should be individualized according to the patient's needs, accurate renal histologic diagnosis (if available), concurrent disorders and client considerations. Identified parasites are treated appropriately. If ova are identified in the urine, consider whether or not the eggs were actually released in the intestines. Treatment of bacterial nephritis with appropriate antibiotics should be based, in part, on culture and sensitivity results when available. Otherwise, suspected bacterial-induced nephritis should be treated with broad-spectrum, bacteriacidal antibiotics that reach high kidney concentrations and which are non-nephrotoxic. Antibacterials should also be considered when concurrent colitis is present. Nephrosis may best be managed by removing known nephrotoxicants and addressing secondary complications. Such secondary complication of any renal disease may include dehydration, hyperuricemia, fibrosis, infectious diseases and anorexia. Dietary induced renal diseases can be managed with diet change or supplementation depending on the etiology. Anti-neoplastic treatment of certain avian renal tumors may be indicated and should be considered. Specifically identifying and managing underlying diseases that may be concurrently present may best control glomerulopathies. Confirmed glomerular disorders in birds without an obvious underlying disease may be managed in some cases with low-dose aspirin and N-3 FA supplementation. Nutritional management such as weight loss, providing a balanced diet and vitamin A supplementation may also be indicated.

The following represents a quick treatment summary of some of the more common renal disease classifications. Articular gout, although not a 'renal disease', is also included. With the possible exception of 'Diet-induced renal disease of color variety psittacine birds', the patient's diet should modified as is 'appropriate' for that avian species. Secondary infections, dehydration, unacceptable weight loss, etc should be managed as needed. Combination therapy should be considered when two or more histologic renal lesions are present.

Non-descript or well-defined nephrosis: Parenteral vitamin A. Remove exposure to toxins if known. Consider N3-FA supplementation.

Glomerulopathy: If identifiable, remove/control any source of infection/inflammation. Give N3-FA and low-dose aspirin until all signs of renal disease (hyperuricemia, histologic changes, etc) are gone. N3-FA can be given chronically if needed.

Bacterial nephritis: Antibiotics for a minimum of 6 weeks.

'Diet-induced renal disease of color variety psittacine birds': Discontinue pellets and change diet over to whole grains, seeds, fruits and vegetables as is appropriate for the species. If after 3-6 months all signs of renal disease are gone, pellets (< 50% of total diet) can be cautiously added to diet.

Renal fibrosis: Use colchine until histologic fibrosis resolves. Otherwise use colchicine for 6-12 months, or until laboratory abnormalities normalize. N3-FA may also be beneficial

Articular gout: Use colchcine and allopurinol together until all signs of gout and hyperuricemia have resolved. Consider diagnosing the cause of probable underlying renal disease and manage appropriately. Give vitamin A if hypovitaminosis A is suspected. Articular gout lesions may also be surgically opened and expressed to speed removal of uric acid crystal accumulation. Again, N3-FA may be beneficial. Use aggressive fluid therapy if articular or visceral gout is accumulating rapidly.

Prognosis

The World Health Organization classification of renal disease is based on distinct glomerular pathological findings and is used for prognosis, treatment and outcome. Presently, no such classification system exists in avian medicine. In fact, there are limited studies that estimate the outcome of selected avian renal disorders. One such review noted that 'most birds live less than 3 months following a diagnosis of a renal neoplasm'. This may seem to offer a poor prognosis but only represents one form of renal diseae that is usually diagnosed late and with which there are few treatment options. Based on the author's experience, several forms of renal disease can be successfully managed, and some resolved, giving a good prognosis for long-term health to the individual patient.

Clinicians are encouraged to thoroughly evaluate each avian renal disease patient individually from diagnosis through to management or completion of treatment. Consider renal biopsy as a viable tool for diagnosing and managing disease. Dr. Robert Schmidt states 'The problem is that clinical lab tests may indicate renal disease in birds, but several kidney disorders cause similar (lab) abnormalities. If you want a definitive diagnosis, biopsy the kidney.' Treatment completion may need to be defined in some cases, as return to normal renal histology by follow-up biopsy. Until renal diseases of birds are better understood, classified and treated, the short and long term prognosis can only be estimated based on the severity of kidney lesions at that time and secondary disorders of the patient.