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Types of Renal Disease in Avian Species

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Renal disease in birds is frequently encountered. Like most other animals, birds are susceptible to a full spectrum of renal insults, such as toxins, tumors, infections, and degenerative conditions. Accurate diagnosis of renal disease is based on a complete history, physical examination, and laboratory evaluation of the patient. Because it is often required for a more definitive diagnosis, special attention is given to histopathologic evaluation of renal tissue, whether through a premortem biopsy or collection at gross necropsy.

Congenital disease

Although not frequently reported, congenital renal lesions are occasionally seen in birds [1]. Renal hypoplasia or aplasia occurs sporadically in birds. It is usually diagnosed as an incidental necropsy finding. Divisional aplasia is common in some breeds of chickens. The cranial division is most likely to be absent. Compensatory hypertrophy of the opposite kidney is generally present.

Renal cysts may be solitary or multiple. If the lesion is severe, the result will be renal failure. Glomerular hypervascularity has been reported in canaries [2]. It leads to glomerular deformation but does not result in immediate renal failure.

Infectious disease

Bacterial

Bacteria can enter the kidney either by ascending the ureters or by haematogenous spread. In either type of infection, the kidneys may be grossly enlarged with variable degrees of necrosis. Histologic lesions suggestive of bacterial nephritis include tubular dilatation and impaction with inflammatory cells [3]. Acute ascending infections are characterized by abundant

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bacteria found in tubules and occasionally in the interstitium. As nephritis becomes chronic, tubular necrosis, cyst formation, distortion, and interstitial fibrosis with mononuclear cell infiltration become evident. Initial hemogenous lesions may be present in glomeruli. Organisms associated with necrosis and a pleocellular inflammatory infiltrate may be seen.

Necrosis of the tubular epithelium is sometimes prominent, but inflammation may be minimal or nonexistent in cases of bacterial nephritis. Distal collecting tubules and cortical collecting ducts are primarily affected by bacterial infections. Subacute ascending infections have a marked inflammatory response with heterophils in the lumen of the tubules. Tubulointerstitial lesions are locally extensive. With severe locally extensive lesions, it may be difficult to determine whether the infection began in the tubules and involved the interstitium or vice versa.

A wide range of gram-positive and gram-negative bacteria are known to cause kidney disease, either as an ascending infection or as part of a systemic disease. Staphylococci and streptococci are common pathogens [4,5]. Other bacteria that can affect the kidney include members of Enterobacteriaceae, *Listeria* sp, *Erysipelothrix rhusiopathiae*, and *Pasteurella* sp [6].

Mycobacterial and *Chlamydophila psittaci* infections are generally systemic infections. They may cause lesions in the kidney [7] but often do not. Mycobacterial lesions are similar to those found in other tissues. Renal lesions caused by *C psittaci* are characterized by interstitial inflammation composed primarily of histiocytes, plasma cells, and lymphocytes, with intracytoplasmic organisms seen in histiocytes in some cases. Any bacterial organism associated with sepsis may be found in avian renal tissue.

Mycotic

Fungal infection of the kidney occurs either as an extension of a fungal infection of the abdominal air sacs [8,9] or as a component of fungemia where a fungus has invaded a vessel, resulting in fungal thrombosis of blood vessels. Fungal hyphae in the lesion give it specificity.

Parasitic—protozoal

Isospora sp and *Eimeria* sp are found in the kidneys of nearly all species of wild ducks and geese [10–14], as well as in other avian species [15–17]. Organisms are predominantly found in the epithelium of perilobular collecting ducts and the medullary collecting tract. *Eimeria truncata* may cause a more severe disease in juvenile waterfowl. Organisms are found in tubular epithelial cells or free in the lumen associated with inflammatory cells and necrotic debris.

Cryptosporidial infection of the kidney of birds is reported [18–21]. Kidneys appear swollen and pale. Slight proliferation of tubular epithelial cells may be seen, and organisms are present on their surface.

Encephalitozoon hellem is a potential cause of renal disease. Lesions are most commonly seen in lovebirds and budgerigars but have been reported in other psittacine birds [22,23]. Gross changes may be absent, or small pale foci may be present in the renal parenchyma. Histologically, small protozoal organisms are present in cells and may be free in necrotic foci and the lumen of the renal tubules.

Systemic sarcosporidial infection may lead to interstitial nephritis with an infiltrate that is primarily lymphoplasmacytic [24]. Organisms are usually not seen. Schizonts of leukocytozoon can also be found in the kidney [25].

Parasitic—metazoal

Trematodes may be incidental findings or lead to clinical renal disease in some birds. These infections are most common in waterfowl. The flukes are found in collecting tubules in the medullary cone [26,27]. Severe infections result in obstruction of the tubules, variable inflammation and necrosis, and secondary dilatation proximal to the obstruction.

Visceral larval migrans due to *Baylisascaris procyonis* may lead to necrotic tracts and larvae within the kidneys [28].

Viral

Adenovirus infection of the kidney is seen in a variety of birds [29,30]. Grossly there may be some nonspecific renal enlargement. Microscopic lesions are usually minimal, ranging from mild interstitial mononuclear cell infiltration to tubular epithelial cell vacuolation and necrosis. Scattered tubular epithelial cells have karyomegalic nuclei containing large, darkly eosinophilic or basophilic inclusion bodies.

Polyomavirus infection may be acute and cause the kidneys to be slightly swollen. Virus may be recovered from the kidney [31]. Histologically, renal tubular epithelial cells may have karyomegaly, with affected nuclei containing clear or amorphophilic, slightly granular inclusion bodies. These may be differentiated from those of adenovirus by their tinctorial properties.

Both primary and secondary lesions may occur in nonbudgerigar psittacine birds with avian polyomavirus disease [32,33]. Intranuclear inclusion bodies and accompanying karyomegaly are commonly seen in mesangial cells. Glomeruli will appear swollen. As many as 70% of these birds will develop a secondary glomerulopathy. This lesion is caused by the deposition of dense aggregates of immune complexes.

Finches with polyomavirus infection may have both renal tubular epithelial and mesangial karyomegaly with intranuclear inclusion bodies.

Nonsuppurative inflammation may be present in the renal interstitium in other viral infections, including reovirus [34] and paramyxovirus [35,36]. Paramyxovirus-1 in pigeons may cause an interstitial lymphoplasmacytic

nephritis and tubular necrosis, with granular and hyaline casts present in the tubules. West Nile virus causes a variable lymphocytic interstitial nephritis as part of generalized disease [37].

Other viruses that may cause renal disease include coronavirus, togavirus, and influenza A virus [38].

Inflammatory disease of undetermined cause

Except as a sequela to polyomavirus infection (discussed in the previous section), immune complex glomerulopathies are infrequently documented in birds, but autoimmune glomerulonephritis has been produced [39–41]. In chronic cases, there may be proliferation of parietal epithelium and glomerular crescent formation. Eventually glomerular shrinkage, fibrous connective tissue proliferation, and sclerosis occur.

Noninfectious disease

Avian renal disease has a wide variety of noninfectious causes. Many of these result in chronic disease with a common gross and histologic appearance.

Dehydration results in reduced urine flow and sludging of the urate crystals within the tubules. Gross lesions are characterized by multifocal white to yellow-white foci or streaks that represent urate deposits. The gross appearance is similar to that of mineralization. Microscopically, urates are dissolved during the fixation process but leave behind needle-shaped and amorphous spaces surrounded by an eosinophilic protein matrix [42,43].

Disorders of protein metabolism may lead to elevation of uric acid; however, whether this in turn may result in urate deposition has not been established [44–46].

Other nutritional problems may result in renal disease. Metastatic mineralization of the kidney is a common lesion in nestling parrots and to a lesser extent in adult birds. A nutritional imbalance is suspected. Renal lipidosis may be secondary to a high-fat diet or chronic hepatic disease. On a gross level the kidneys are pale, and microscopically there is fat in tubular epithelial cells. Lipid-containing macrophages are usually present in glomerular capillaries. Vitamin A deficiency leads to squamous metaplasia of the epithelium of the ureters and collecting ducts, which in advanced cases results in the transformation of the ureteral epithelium to a keratinized epithelium [25,47–51]. High-cholesterol diets have also been associated with diffuse renal disease, including proliferative glomerulopathy, periglomerular fibrosis, multifocal interstitial nephritis, and lipid-laden cells within the glomeruli of pigeons [52].

Renal amyloidosis is most frequently observed in waterfowl and small passerine birds. Multiple organs in addition to the kidney are generally

involved. Grossly the kidneys may be enlarged, pale, and somewhat friable. Histologically, the amyloid is eosinophilic or amphophilic and may be deposited in glomerular or tubular basement membranes and the walls of renal arteries and arterioles [25,53,54].

Iron storage disease primarily affects the liver, but iron pigment is also seen in renal tubular cells in many affected birds. The iron does not cause an inflammatory or degenerative response [25,55].

Renal disease and lesion formation may be secondary to a wide variety of conditions [56–59]. Ischemic (hemoglobinuric, myoglobinuric) nephrosis is secondary to a number of problems. Hemoglobinuric nephrosis is infrequent because of the rarity of hemolytic disease in birds. Myoglobinuric nephrosis may be a sequela to exertional rhabdomyolysis or severe crushing injury to muscle [60]. In both cases, the kidneys may be dark brown. Tubular degeneration and luminal accumulation of amorphous eosinophilic material resembling myoglobin are seen microscopically in proximal convoluted tubules, and eosinophilic casts are noted in collecting tubules.

Toxic nephropathies

History is important in making a diagnosis of toxic nephropathy, because most renal toxins cause similar gross and histologic lesions; therefore, a definitive causative diagnosis is often not possible based on gross and histopathologic changes alone. Sometimes the tentative diagnosis is made by excluding all other possibilities [61].

Vitamin D₃– and vitamin D₃ analogue–based rodenticides are toxic in birds. These rodenticides cause increased intestinal absorption of calcium and hypercalcemia. Decreased urinary calcium excretion may also occur. Calcium is deposited in soft tissues, including the kidney [25].

Gentamicin sulfate and amikacin are two aminoglycoside antibiotics that are commonly used in birds. Gentamicin sulfate is more nephrotoxic than amikacin. Aminoglycoside toxicity results in kidney enlargement and changes resembling those seen with other causes of renal failure [62].

Lead and zinc toxicity both may cause acute tubular necrosis [63–67]. Gross changes vary from none to swollen, pale kidneys. Cadmium, mercury, and arsenic are also nephrotoxic [25].

Several mycotoxins, including oosporein, citrinin, and ochratoxin, have been shown to cause disease in poultry or domestic waterfowl [68–70]. Aflatoxins may also be a problem; they can cause degeneration of the proximal convoluted tubules and thickening of glomerular basement membranes [25,71].

Excessive salt ingestion leads to renal problems that result in urate deposition and gross and histologic lesions [72,73].

A variety of other nephrotoxins have been reported to affect birds [61,66,74,75].

Physical/other nephropathies

Acute renal hypoxia/ischemia is usually related to a localized or generalized vascular problem. The results are tubular necrosis, protein leakage, and urate deposition. Lesions are similar to the various problems discussed under metabolic disorders, and differential diagnoses include many of these conditions. Renal hemorrhage may be secondary to trauma, ischemia, or a variety of primary disease conditions. The hemorrhage may be visible grossly and may affect both interstitium and tubules. The end result of many of these conditions is chronic or end-stage renal disease with severe fibrosis [25].

Neoplastic disease

Renal tumors are reported in many species of birds but are particularly common in the budgerigar [76–78]. Renal carcinoma is the most common tumor of the kidney; however, adenoma, nephroblastoma, cystadenoma, fibrosarcoma, lymphosarcoma, and other neoplasms are also reported in the avian kidney [25].

The most common presenting sign of renal neoplasia is unilateral or bilateral lameness or paralysis [79]. These symptoms result from compression of the lumbar and sacral nerve plexi as they pass through or dorsal to the kidney, respectively, or from tumor growth into and adjacent to the synsacrum. Skeletal muscle atrophy and osteopenia may also be seen. Metastasis is occasionally reported [80].

Embryonal nephromas (nephroblastomas) are most commonly reported in chickens but are also found in psittacine and small passerine birds. They are usually unilateral but may be bilateral and are grossly similar to carcinomas [25].

Lymphosarcoma may be isolated to the kidney but is usually a part of generalized neoplastic disease. Grossly, the kidneys are pale, mottled, and moderately firm with nodular or diffuse cell infiltration. Myeloproliferative disease and histiocytosis are also reported in the avian kidney [81–83].

Other primary sarcomas are possible but are rarely reported, and metastatic sarcomas are infrequent. Malignant melanoma may affect the kidney, usually as a part of multicentric neoplastic proliferation [25].

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