Avian Nephrology
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Birds have paired kidneys that are embedded within the renal fossa of synsacrum, and surrounded by air sacs (diverticula of the abdominal air sacs). There are three main divisions (cranial, middle, and caudal) to each kidney, and each division is supplied by its own artery (cranial, middle, and caudal renal arteries). Additional blood supply to each kidney is supplied by the renal portal system. In different species of birds, other than psittacines, certain divisions are fused. The renal divisions are comprised of multiple lobes, and each lobe contains multiple lobules. Because the kidneys are located deep within the renal fossa of the synsacrum, surrounded by air sacs, and supplied by multiple arteries, a nephrectomy in any avian species is very difficult, if not impossible to perform. Nerves from the sacral and lumbar plexus, specifically the ischiatic nerve, running alongside the ischiatic artery, pass through the parenchyma of the kidneys. Therefore, anything that causes renal enlargement (neoplasia, etc.) can cause nerve impingement and/or compression. This often results in paresis or paralysis of the ipsilateral leg.

The avian kidney does not have a distinct cortex and medulla. There are two main types of nephrons - mammalian and reptilian. Reptilian nephrons possess a simple glomerulus, lack a Loop of Henle, and comprise approximately 70–90% of avian nephrons. Not only do the reptilian nephrons lack the Loop of Henle, but their nephrons are also oriented perpendicular to the collecting ducts, instead of parallel as in mammals. As there is no hyperosmotic medulla, and no counter-current multiplier system, avian kidneys cannot produce hyperosmotic urine. The majority of the nephrons are located in the upper, wider portion of the kidney (equivalent to the mammalian renal cortex). The “stalk” of the kidney is referred to as the medullary cone, equivalent to the mammalian renal medulla, and contains a majority of the mammalian nephrons and the collecting ducts. The avian kidney is composed of a continuous system of ducts - whereas in a mammals the collecting ducts terminate at the renal papilla, urine is collected in the pelvis, and this drains into the ureter. There is no renal pelvis in birds or reptiles; therefore, they cannot develop pyelonephritis. No avian species possesses a urinary bladder, and the ureters empty directly into the urodeum of the cloaca, thus no urethra. Within the cloaca, the urine can undergo post-renal modification, and water reabsorption can occur. Also, urine can be refluxed further into the rectum and colon for additional reabsorption.

Birds cannot significantly concentrate their urine, for the anatomic reasons listed above. To compensate for this, birds have significant post-renal modification of urine. As previously discussed, water reabsorption can occur in the cloaca, rectum, and colon. Birds living in marine or arid environments often have nasal salt glands, which secrete a watery fluid that is hyperosmotic to plasma. This allows for removal of excessive salts without use of excessive amounts of water. Uric acid is the primary form of nitrogen elimination, but birds also produce small amounts of urea. Uric acid precipitates as a salt (urates) in urine, and is maintained in a protein-rich colloidal suspension. Uric acid is actively secreted at tubules, not freely filtered; therefore, uric acid levels should not be affected by dehydration (pre-renal azotemia). Clinically, hyperuricemia is noted in severe cases of dehydration, likely because of the depletion of blood supply to the tubules from hypovolemia. Post-renal azotemia can also occur with obstruction of the ureters such as with external compression from a large egg or cloacal disease (cloacal mass, cloacolith, etc.).

Clinical signs of renal disease are non-specific in birds, and a common presentation is the fluffed, “sick” bird. The same holds true for the physical exam - due to their location, one cannot palpate the kidneys, except if a very large kidney mass is present. And even then, it is almost impossible to determine the origin of a coelomic mass on palpation alone. Polyuria and polydipsia may be noted at home by the owners. Care must be taken when evaluating a bird’s dropping in a stressful situation, such as a veterinary hospital, as stress frequently causes polyuria due to premature voiding of cloacal contents. If articular gout is present, lameness or joint swellings may also be noted.

Uric acid is the most accurate hematologic measure of renal function in birds, and it becomes elevated when 60–70% of renal function is lost. Urinalysis and urine cultures are not commonly
performed in birds due to extensive post-renal modification of urine. Hematuria can be caused by kidney
disease, but also could be blood from cloacal, reproductive, or gastrointestinal origins. Heavy metal
toxicity is an important differential for hematuria in all avian species. Whole body radiographs are the
most common form of imaging for the kidneys. Visualization of the kidneys via coelomic ultrasound is
often limited as the kidneys are surrounded by air and bone. Endoscopy and renal biopsy is currently the
only way to definitively diagnose avian renal disease. That said, renal histologic lesions are rarely
pathognomonic for a specific disease process, with many different diseases causing similar renal lesions.

Infectious causes for renal disease include bacterial, viral, or fungal pathogens. Because the ureter
empties into the cloaca, ascending infection from fecal organisms can occur. Likewise, since the caudal
mesenteric vein (via the renal portal system) also flows through the kidneys, hematogenous infections
from enteritis can “ascend” to the kidney as well. According to one reference, approximately 50% of all
nephritis cases were associated with bacterial disease. Viral infections can cause direct damage to the
kidney, or can be found in renal histopathology as part of a generalized viremia. Fungal infections of the
kidney can occur as an extension of a fungal airsaculitis or secondary to systemic fungal infections -
*Aspergillus* sp. is the most common fungal organism identified. Parasitic infections, such as renal
coccidiosis, are most common seen in waterfowl and marine species. Other parasitic infections found in
the avian kidney include, *Sarcocystis* sp., *Microsporidia* spp., Cryptosporidia sp., and flukes. Several types
of renal disease (nephritis, renal calcification, gout) have been associated with various nutritional
imbalances - specifically hypercalcemia, hypervitaminosis D, hypovitaminosis A, and excessive amounts
of protein. Toxins, such as lead and zinc, aminoglycoside antibiotics, flunixin meglumine, and
mycotoxins have also been attributed to renal disease in several avian species. The most common
neoplasia which affect the kidneys includes renal carcinoma and renal nephroblastoma. Both of these are
commonly reported in aged budgerigars.

Gout, both articular and visceral, is a consequence of renal disease and several other compounding
factors. Increased uric acid in the blood stream (hyperuricemia) will eventually result in urate crystal
precipitation. Once present within joints (articular) or on the surface of visceral organs (visceral), it cannot
be removed. The metatarsophalangeal and interphalangeal joints are often the most commonly affected
joints diagnosed antemortem, as they are readily visible to the clinician. Both forms can be present in a
bird simultaneously; however, it is unknown why one form occurs versus the other. There are several
drugs used in human medicine to treat gout, including allopurinol, but it is important to remember that
uric acid is not the main method of nitrogen excretion in humans, which is very different than in birds
and reptiles. In addition, two studies have been performed in red-tailed hawks that examined the use of
allopurinol to decrease uric acid production. Both proved that allopurinol has a low therapeutic index in
this species. Clinicians should also consider appropriate analgesia for these patients, as well as quality of
life parameters as gout is incredibly painful and a consequence of end-stage renal disease.

**REFERENCES**

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