



Avian renal disease: pathogenesis, diagnosis, and therapy

Michael Lierz, Dr Med Vet

*Institute for Poultry Medicine, Free University of Berlin,
Koserstrasse 21, 14195 Berlin, Germany*

Anatomy and physiology

The following anatomic and physiologic sections are based on a review of the works of Skadhauge, Siller, Waibl and Sinowatz, Braun, Goldstein and Skadhauge, and Carretero et al [1–7]. The kidney excretes nitrogenous waste products, and is central to body water and solute homeostasis. During embryonic development of the bird within the shelled egg, nitrogenous waste products accumulate and cannot be voided. Insoluble uric acid is the main byproduct, rather than urea. Different renal anatomy and physiology lead to different renal diseases in birds compared to mammals.

In birds, the left and right kidneys are positioned on either side of the vertebral column, and are in contact with the pelvis and synsacrum dorsally. Each kidney is divided into three divisions and each section contains several functional lobes. Each lobe is divided into lobules and each lobule has a cortex and a medulla. A lobe is defined as a group of lobules draining through their medullary cones into the same secondary branch of the ureter. Blood vessels and collecting tubules are located between the lobules. In the center of each lobule an intralobular vein collects blood from the peritubular plexus. Intralobular arteries branch from the renal arteries to supply the glomeruli. Efferent glomerular arterioles join branches of the interlobular vein (renal portal blood) and form the peritubular plexi.

The kidneys have afferent arterial and afferent venous blood supplies. The arterial supply of the cranial division of the kidneys is from a branch of the aorta. Branches of the ischiatic arteries supply the middle and caudal divisions. Arterioles form the capillary tufts of the glomeruli, which are responsible for the ultrafiltration of blood.

E-mail address: lierz@zedat.fu-berlin.de (M. Lierz).

A renal portal system with afferent venous blood is important in birds but does not exist in mammals. The external and internal iliac veins and the ischiatic and caudal mesenteric veins collect venous blood from the capillary system of the limb, tail, pelvis, and caudal part of intestine and spine. These vessels combine to form the caudal portal vein. The cranial portal vein (a branch of the external ischiatic vein) and caudal portal vein form a venous ring located ventral to the kidney. The common iliac vein carries venous blood to the caudal vena cava. A renal portal valve is located within the common iliac vein to regulate venous blood flow through the kidney. When the valve is closed, most of the venous blood from the capillary system is directed through the kidney. This happens when the bird is at rest. When the valve opens venous blood is directed into the caudal vena cava, bypassing the kidney (Fig. 1). Blood from the venous ring can still bypass the kidney by flowing into the caudal mesenteric vein or internal vertebral venous sinus. Right and left renal portal valves can operate independently. Valvular smooth muscle has sympathetic and parasympathetic innervation. Adrenergic stimulation causes relaxation of the valve muscles while cholinergic causes contraction. The smooth muscle of the renal portal vein contracts under adrenergic stimulation. These autonomic responses govern flow of renal portal blood. Under sympathetic stimulation the valve is open and blood bypasses the kidney. Parasympathetic stimulation increases valve resistance and blood enters the renal portal system.

Afferent venous blood enters the peritubular capillary network to mix with efferent glomerular arteriolar blood. Intralobular veins converge to form the caudal and cranial renal veins proximal to the renal portal valves

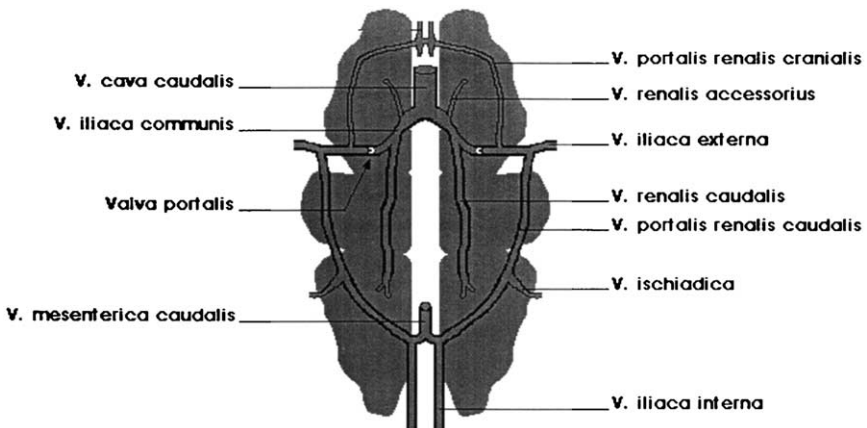


Fig. 1. Schematic drawing of the avian renal blood supply. The renal portal valve within the common iliac vein regulates the amount of blood shunted into the caudal caval vein. (Courtesy of T. Hoffman, modification of Rickert-Mueller CR. *Das Blutgefäßsystem der Niere des Haushuhns (Gallus dom.)*. Veterinary Thesis, Giessen, Germany; 1968.)

in the common iliac vein. The peritubular capillaries reabsorb large amounts of solute and water from the renal tubules.

Birds have two types of nephron. Cortical (reptilian type) nephrons are most common (up to 90%) and are located in the lobule cortex. Their glomeruli are smaller, and they do not possess the loop of Henle. Medullary (mammalian type) nephrons have long loops of Henle reaching far into the medulla. Bundles of collecting ducts form the rest of the medulla. All lobules in the same lobe drain into the same secondary branch of the ureter, which then joins a primary ureteral branch. All branches of the ureter are lined with mucous-secreting epithelium, and urates are excreted as a colloidal suspension. About 65% of uric acid is chemically bound to proteins. Urate precipitates must remain in this supersaturated suspension. Water is needed to flush the suspension through renal tubules. Smooth muscle fibres coordinate ureteral peristaltic motion to move urates towards the cloacal urodeum. Birds have no urinary bladder.

Within the mammalian kidney urea concentration (creating hypertonicity) within the medulla is the key factor enabling concentration of urine. As urea is not an important product of avian metabolism and loops of Henle are absent in 90% of avian nephrons, the capacity of the avian kidney to concentrate urine is less than mammals. Water is conserved by resorption within the coprodeum and rectum

Solutes can be secreted into the renal tubules, adding to the glomerular ultrafiltrate. Uric acid is mainly excreted through this mechanism, and is independent of both ultrafiltration and tubular urine flow. Plasma urate concentration remains normal even if the bird is dehydrated and the renal tubules are full of urate crystals [8–10]. Urates are still excreted into the tubules but cannot be flushed through the kidneys due to lack of ureteral water flow (Fig. 2). Early and aggressive fluid therapy is needed to reverse the pathologic changes. By contrast, urea is excreted by glomerular filtration. A dehydrated bird may reabsorb up to 99% of tubular water [11]. Reduced ultrafiltration following low blood pressure can reduce urea excretion. In addition, tubular reabsorption increases during dehydration, as urine flow through the tubules is slow and plasma urea concentration increases [10].

The kidneys also regulate potassium and sodium levels. Potassium can be actively reabsorbed or secreted in the distal tubule. Some species, for example, glaucous-winged gulls (*Larus glaucescens*), have salt glands to aid water and electrolyte homeostasis. Calcium is largely reabsorbed while phosphate is mostly excreted. Calcium reabsorption and phosphate secretion is regulated by parathyroid hormone, calcitonin, and vitamin D₃ [9].

Lumbar and sacral nerve plexi are located between the different kidney divisions (Fig. 3). The femoral, obturator, and ischiatic nerves have their origins within these plexi. Kidney diseases that cause renomegaly (eg, swelling or neoplasia) might lead to pressure on these nerves resulting in lameness or leg paralysis (Fig. 4) [12].

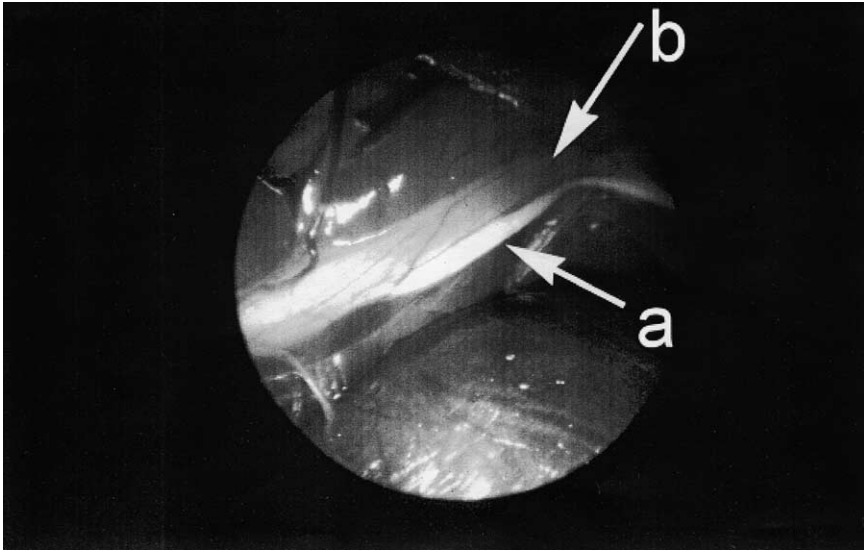


Fig. 2. Endoscopic view of a filled ureter in a sparrow hawk (a) (*Accipiter nisus*). This is a common finding in free-ranging birds found in a dehydrated and debilitated state. Oviduct (b).

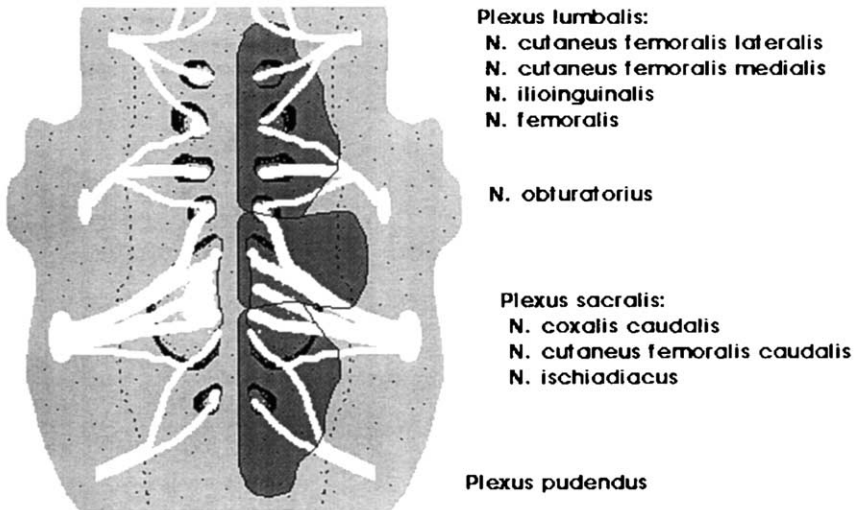


Fig. 3. Schematic view of the nerve plexus located between the three kidney divisions. Swellings or neoplasias of the kidney can increase the pressure on the nerves, leading to limb paralysis (T. Hoffmann, modified from Harcourt-Brown, 2000) [46].

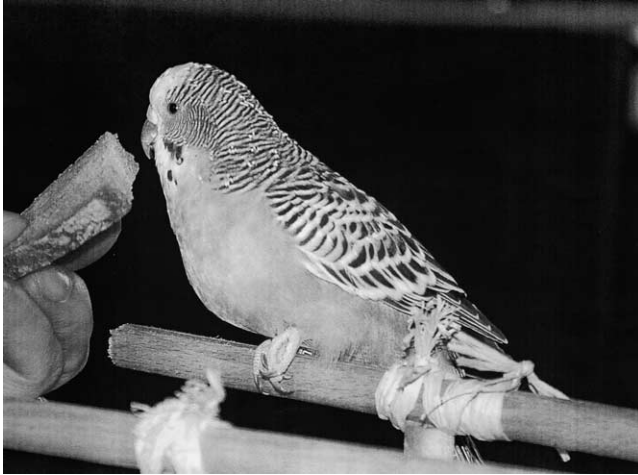


Fig. 4. Budgerigar (*Melopsittacus undulatus*) with leg paralysis due to a renal neoplasia.

Diagnosis

Diagnosis of renal disorders is difficult as pathognomonic signs are rare. In addition, renal disorders are often seen in conjunction with other diseases. Some diagnostic tests can provide immediate indication of renal dysfunction. In most cases more than one diagnostic test is necessary to confirm a renal disorder and make a definitive diagnosis.

Plasma chemistry

The use of plasma chemical parameters to diagnose renal disorders is limited. Reference values are missing or inadequate for most avian species. Reference ranges have been produced for some species, but small numbers of birds have been used and these may have been kept under different conditions to the patient. A single parameter falling outside the reference range has limited diagnostic use, and several reasons could explain an aberrant value. Consistent and repeatable abnormal results for several parameters produces a clearer picture and may help direct further investigation such as radiology, endoscopy, and biopsy.

Uric acid and urea

Uric acid and urea values can be assessed, but normal physiologic variations have to be considered. Postprandial values rise in healthy raptors with peaks up to 8 hours after feeding [13]. Therefore in raptors a 24-hour fasting period prior to blood sampling is recommended for assessment of renal function. In addition, pathologic increases in uric acid can only be detected if 70% or more of kidney function is lost making this parameter useless for

early detection [12]. Urea can be used to detect dehydration but not to confirm renal dysfunction.

The ratio of plasma urea and uric acid can be used to differentiate prerenal and renal causes of azotemia [10]. Prerenal azotemia with elevated urea levels produces a high urea:uric acid ratio. The ratio is calculated: plasma urea concentration [mmol/L] \times 1000: plasma uric acid concentration [μ mol/L]. In peregrine falcons (*Falco peregrinus*) the ratio is >6.5 [10]. Such normal ratios must be established for each species. Renal failure is likely when uric acid concentration is above the species reference range in a fasted individual. Severe tissue damage could lead to an increased uric acid concentration following release of nucleic acids [14].

Creatinine

Creatinine has limited diagnostic value. In birds, creatine is mostly excreted in urine before it is converted to creatinine so levels of plasma creatinine are low [15]. Creatinine is excreted by glomerular filtration and reabsorbed in the tubules. Both mechanisms keep the plasma concentration constant, and postprandial elevations have not been observed [13]. Greater amounts of creatinine are released in cases of severe muscle damage, but excretion appears to remain constant resulting in an elevated plasma concentration [14]. Reduction of glomerular filtration can also lead to an increased creatinine concentration [16]. In theory, if the glomerular filtration rate is preserved but tubules are damaged, plasma creatinine concentration will fall.

Potassium

Hyperkalemia can be seen in acute renal failure, and can cause severe electrocardiographic changes including cardiac arrest. Sample handling can influence the potassium result, and cells should be separated from the plasma within 1 minute of collection [17].

Sodium

Hypernatremia is seen after an increased sodium intake or during dehydration. In renal disease sodium loss can be high and uncompensated. Hyponatremia can be an indicator of renal failure [9]. Diarrhea can also lead to significant sodium losses [18].

Calcium

Calcium is reabsorbed after glomerular filtration. In renal failure calcium losses are high, resulting in hypocalcemia. Nutritional and alimentary disorders should also be considered. Hypocalcemia can cause tetany. Interpretation of total and ionized blood calcium should only be done in conjunction with albumin concentration. Hypoalbuminemia reduces the quantity of

bound calcium and decreases total calcium concentration, although the level of biologically active ionized calcium may be normal [19].

Phosphorus

Reduced glomerular filtration rates can lead to high plasma concentrations of inorganic phosphorus [18,20]. Decreased phosphorus levels may reflect an alimentary problem such as hypovitaminosis D₃ or malabsorption.

Urinalysis

In contrast to plasma chemistry, urinalysis can be very valuable in diagnosing avian renal disorders. Although plasma chemistry changes occur later in the course of renal disease, urinary changes can give an earlier indication. For diagnostic purposes a quality urine sample should be nearly free of urates. Obtaining such a sample can be difficult in normal birds, but many with renal disease are polyuric, and collection of urate-free samples is possible. Nonabsorptive paper or plastic substrate allows collection of voided urine from the bird cage floor. A trained bird of prey may provide a stool sample when the hood is removed or food offered. Urine can be collected with the aid of a Petri dish. Birds in a veterinary practice are usually stressed. Stress polyuria can cause urine to be voided before retrograde entry to the intestine where absorption of water and electrolytes occurs [9]. The sediment of collected urine samples should first be examined by microscopy. High amounts of urate will reduce the diagnostic value considerably. The presence of urinary casts is a sign of renal disease. In nonpolyuric patients an intravenous infusion can increase the urine fraction but will dilute the urine components and may reduce diagnostic value. Abnormal results may suggest a diagnosis, but normal findings do not exclude nephropathy. Halsema et al [21] describes a method of cloacal cannulation to collect urine and gives reference values for pigeon urine. Unfortunately, these values are not established for other bird species. The cloaca is cleaned of feces before the cannula is introduced. It remains for at least 20 minutes until enough urine is collected. A perfect urine sample can be obtained, but the long time involved in this method (up to 200 minutes) can make it unsuitable for sick or weak birds. The urine sample is routinely examined for color, clarity, osmolality, pH, hemoglobin, glucose, and protein. In addition Gram stain and cytology can be helpful.

Color

Urine is usually white or pale yellow. Dark yellow urine may occur following the use of Vitamin B-complex. Blue discoloration is rare, but might occur if certain berries are fed. Chronic hepatitis might cause a yellow,

sulphur-colored urine while a light green color occurs in liver diseases due to biliverdinuria, and is often associated with late stage of systemic aspergillosis. Hematuria is only visible when 0.1% of the urine contains blood [9]. Chemical test sticks (Multistix[®], Bayer Diagnostics, 511 Benedict Ave, Tarrytown, NY) show positive reactions with blood concentrations of 0.001–0.002% in the urine and, along with microscopy, provide a sensitive method of blood detection. Blood within the urine is not necessarily a result of kidney damage. Blood from the cloaca, genital tract or the caudal intestine can mix with urine. In carnivorous birds with short digestion times, especially if starved or stressed, stick reactions can be positive as still undigested hemoglobin is present [10]. Sticks can detect hemoglobinuria, which suggests erythrolysis. High amounts of plasma hemoglobin result in excretion of hemoglobin by the kidney.

Myoglobinuria also causes positive reactions, and can only be distinguished from hemoglobinuria by spectrophotometry [9]. It is rarely seen but reported in flamingos (*Phoenicopterus* spp.) and ostriches (*Struthio* spp.) following rhabdomyolysis. Rosskopf et al [22] reported a red urine color in lead-poisoned amazons, probably caused by porphyrinuria rather than hemoglobinuria.

Osmolality

Bird urine has low osmolality. High values are common only in birds adapted to desert areas with maximum values between 500–1000 mOsmol [23]. Urine collected by cannula has a lower osmolality compared to excreted samples following water reabsorption in the intestine [5]. Urine osmolality provides a direct value for the ability of the patient's kidneys to concentrate urine. In polyuric birds the osmolality should be low. Alberts et al [24] developed a test for differentiation of polyuric disorders in racing pigeons. The study suggested an osmolality of 450 mOsmol/kg as the normal concentration capacity. After 24 hours of water deprivation the osmolality increases in both healthy pigeons and polyuric pigeons without kidney failure. It is thought that this can be applied to other avian species.

Uric acid, pH, and ammonia

Uric acid concentration should be measured in supernatants, as the sediments have much higher concentrations. Calcium, sodium, potassium, and magnesium values are of limited diagnostic value but might be helpful if found in high concentrations, suggesting renal dysfunction or excessive intake. Large quantities of cations are trapped within uric acid suspension [25]. This varies from 3–75% for sodium, 8–84% for potassium, and 17–32% for calcium and magnesium.

Urine ammonia concentration is not influenced by dietary protein intake but increases in dehydrated birds [9,26].

Protein

Protein concentrations up to 2 g/L are considered normal. Detection of proteinuria is difficult, as urine sticks are often too insensitive. Experience with particular stick types will be helpful. As an alternative the Ponceau S-method can be used for more accurate evaluation [27]. Proteinuria is a sign of increased glomerular permeability, and is often associated with glomerulonephritis.

Glucose

Glucose urine concentrations are normally low, and cannot be detected with urine sticks. Glucosuria is diagnosed if sticks react positively. Polyuria, polydipsia, and glucosuria are signs of diabetes mellitus, but only if the plasma glucose concentration is also elevated. Repeated tests are necessary, as stress can also cause high urine glucose concentrations. Pancreatitis is a cause of glucosuria [16].

Urine enzymes

Kidney tissue contains Lactate-Dehydrogenase, Glutamate-Dehydrogenase, Aspartate-Aminotransferase, Alanine-Aminotransferase, Creatine Kinase, and Alkaline Phosphatase. It is known that released enzymes from kidney cells are excreted with urine and do not enter the circulation [10]. High concentrations of these enzymes signify renal damage, but reference limits are not established.

Microscopic examination of urinary sediment

Most important in the diagnosis of early renal diseases is the microscopic demonstration of urinary casts and cells. Cellular casts might contain blood cells, bacteria, fungi, or tissue cells. Cast bacteria signify kidney infection. Bacteria found outside the casts are likely contaminants, and microbiologic examination of urine is not useful [9]. Rosskopf demonstrated yellow-orange material without cellular elements, suggesting hemoglobin casts [4]. Granular casts are composed of degraded cellular components. Rosskopf [4] described the transition of granular and cellular casts to hemoglobin casts as a prognostic sign for the resolution of an inflammatory process. Bermudez and Hopkins [28] demonstrated eosinophilic casts in a case of hemoglobinuric nephrosis in a rhea (*Rhea* sp.). Finding microorganisms such as cryptosporidium or coccidia is often significant.

Radiology

Mammography units are useful for imaging bird kidneys, providing better detail and resolution than standard equipment. Full-body ventrodorsal

and laterolateral radiographs are routinely produced and are always recommended, as renal disorders might result from or cause other abnormalities. In particular, cardiomegaly might be directly linked to kidney disease (or vice versa) as blood pressure and ultrafiltration are linked via the renin–angiotensin mechanism (Fig. 5). Kidneys are superimposed by gut and intestine in the ventrodorsal radiograph, so the laterolateral view is more useful. In case the kidneys are visible in ventrodorsal radiographs the density must be high and a pathologic finding is presumed. In normal laterolateral radiographs the kidneys are visible as bean-shaped shadows caudal to the last ribs. The caudal kidney is overlapped by the pelvis, making interpretation difficult. Active gonads might superimpose the cranial part of the kidney and resemble neoplasia. If differentiation is difficult, contrast radiography to demonstrate intestine or kidney is recommended. In most bird species, healthy individuals demonstrate a small rim of air above the kidneys. In cases of kidney enlargement, this might not be visible. Incorrect lateral positioning of the bird may obscure this small border of air. Kidney enlargements are common with disease but are nonspecific. A change in kidney shape can be a sign of neoplasia or cysts, but in most cases a contrast radiograph is necessary for differentiation [8]. Increased density of the kidney is often a sign of dehydration or hypovitaminosis A. Gout might be suspected if crystals (uric acid precipitation) are distributed through the kidney but must be confirmed. The radiograph might suggest articular gout with urate tophi within the joints [29].

Urography

Indications for urography in birds are few. Differentiation of the kidney and ureter from other structures, obstruction of the ureter, or the demonstration of functional disturbance of the renal system are examples. Organic iodine compounds given intravenously are used as contrast media (Urografin[®] 70, Schering Berlin Inc., Montville, NJ). They are excreted very quickly via the renal portal system, especially if administered via the femoral vein, but can be slowed by use of the ulnar vein. As dosage 2 mL/kg of a 70–80% solution of organic iodine compound (300–400 mg iodine/mL) is recommended [8]. Urograph quality depends on the chosen contrast medium and the concentrating capacity of the kidney. Contrast agents are irritant, and perivascular spillage should be avoided. Agents should be used at body temperature. Newer contrast media are based on nonionic agents and therefore preferable. Side effects are reduced, and in particular they are not irritant if injected perivascularly. Most commonly Iopamidol (Solutrast 200[®], Bracco-Byk Gulden, 78467 Konstanz, Germany) or Iohexol (Accupaque[®], Amersham Health, Princeton, NJ) are used at a dose of 400 mg iodine/kg.

The first radiograph should be taken 30 seconds after injection, as in most cases the kidney will be visible. One minute after injection kidney and the

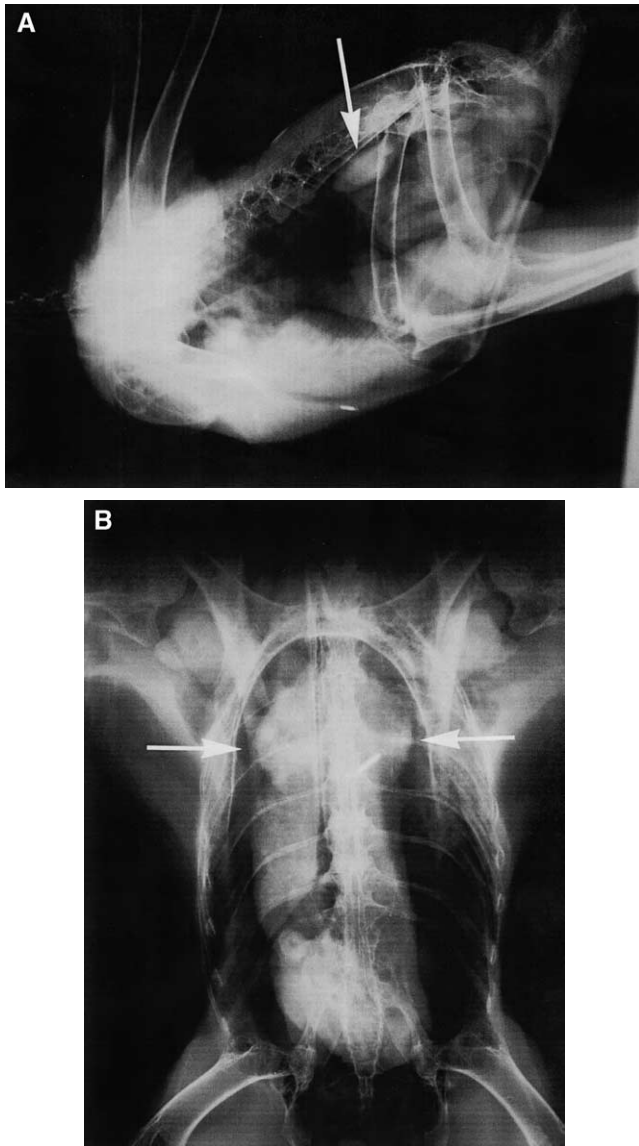


Fig. 5. Radiograph of a Steppe eagle (*Aquila nipalensis*) illustrating increased soft-tissue density in the region of the kidneys (A) and cardiomegaly (B).

ureters are visible [8]. In some cases the cloaca can be demonstrated within 2 minutes of contrast administration (Fig. 6). Fast multiple exposures (every 0.5–2 seconds) are useful, especially for angiography, but require suitably equipped radiology units. Prolonged elimination times indicate renal insufficiency. Urography should be avoided in cases of severe renal dysfunction

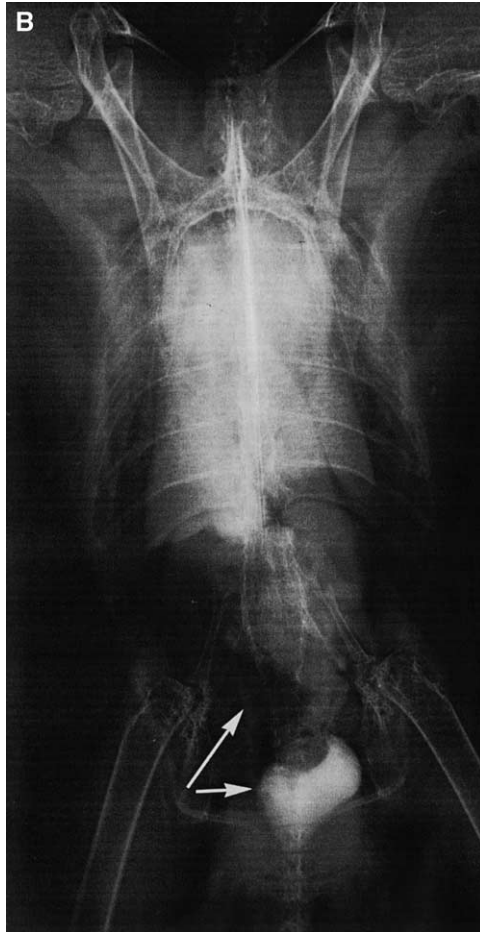
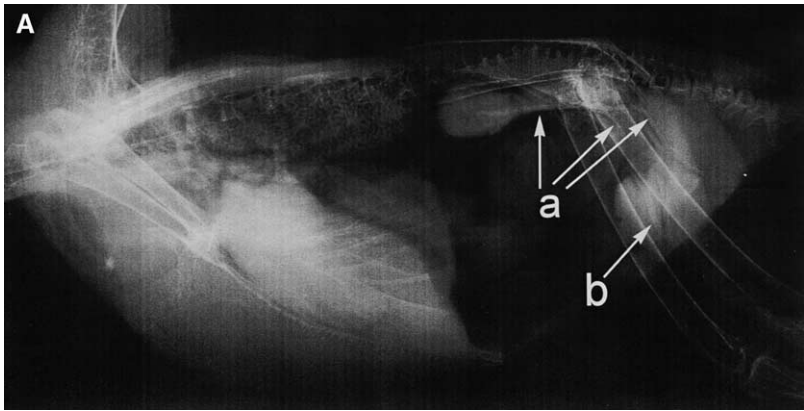


Fig. 6. Radiographs of a urography, 2-minute postinjection of contrast media (Solutrast 200) of a sparrow hawk (*Accipiter nisus*). In the lateral view (A) kidney and ureter (a) is clearly visible. The os pubis of the pelvis (b) should not be confused with the ureter. The ventrodorsal view (B) demonstrates the ureter and the cloaca filled with contrast media.

detected by plasma chemistry. Urography is most useful in assessment of kidney morphology.

Sedation or anaesthesia is recommended for the patient undergoing urography to prevent struggling and ensure intravenous injection. Overdosage and cold contrast media may cause vomiting, and stressed or weakened birds may suffer circulatory problems [8]. Ketamine hydrochloride should be avoided in birds with renal insufficiency as it is renally excreted.

Ultrasound

Ultrasonography is a familiar diagnostic tool in veterinary medicine but, due to the air sac system of birds, is not very popular in avian medicine. The outstanding advantage of this modality is its ability to view deep organ structures. Unlike endoscopy, ultrasound is noninvasive, and can be used safely in weak patients where anaesthetic risks might be high, and the bird is not stressed by handling. An ultrasound examination is recommended in cases of polyuria, polydypsia, elevated renal blood parameters, lameness, or where radiologic appearance of the kidney is abnormal. Endoscopy can follow if more information is required.

A 64 gray-scale computerized ultrasound system with videorecorder and videoprinter is recommended, and a 7.5-MHz, 60° sector transducer is preferred [30]. The probe head should be very small as the contact area in birds is limited. With a probe working surface area of 1.5 × 2.5 cm birds of 40 g up to 1 kg can be examined [30]. Distance from the scanner to the target organ is short so a gel pad standoff should be used. Ultrasound gel is essential.

Patients must be fasted prior to ultrasound examination. The fasting period depends on the size of the bird, but should be at least 3 hours to prevent interaction of food-filled intestinal slings between scanner and kidney [30]. In debilitated patients liquid food might be an alternative. The fasting period should be increased in birds of prey, and it is better if the bird casts prior to examination. For kidney examination the contact area is ventral between the xiphoid process and the ossa pubis [30]. An assistant should hold the patient in dorsal recumbency. The normal kidneys cannot be evaluated by ultrasonography, as they lie ventral to the vertebra with a small line of air interposed [31]. Air sacs are found laterally, preventing visibility. In the recommended ventromedial approach intestinal loops might cause problems, but in cases of increased kidney size including neoplasia, structures become more visible. Renal neoplasia can be seen more frequently in budgerigars. If lameness is evident an ultrasound exam might help indicate a renal mass. They appear as heterogeneous structures (Fig. 7). According to Krautwald-Junghanns and Enders, cysts may also be diagnosed using this technique [30]. They can be demonstrated as round anechoic structures with marked posterior acoustic enhancement. If the kidney can be demonstrated the bony fossa renalis appears as a white “W”-shaped line [32].

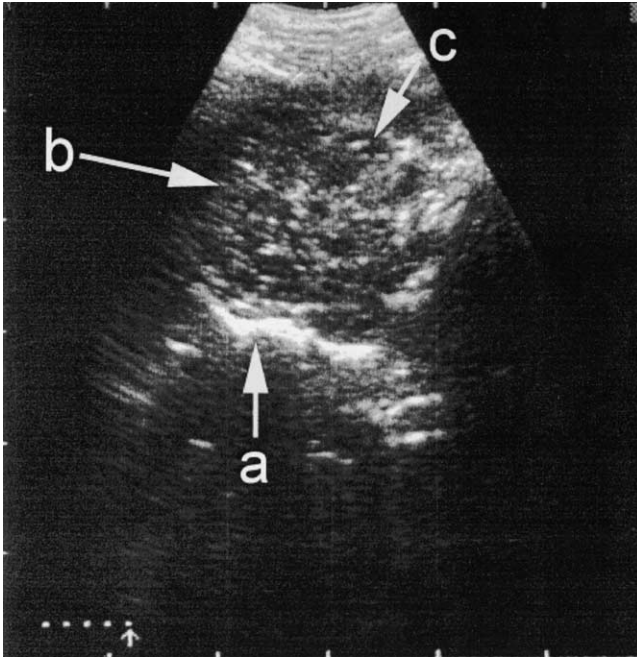


Fig. 7. Ultrasonogram of a soft tissue mass, later diagnosed as a renal adenocarcinoma (b) in a budgerigar (*Melopsittacus undulatus*). The white line (a) represents the spine bone and the fossa renalis. The hypochoic areas represent cystic and necrotic lesions (c). (Courtesy of Prof. M.E. Krautwald-Junghanns.)

Endoscopy

Endoscopy is invaluable in diagnosing visceral organ disease. It allows direct visualization and interpretation. In particular the kidney size, color, shape, and surface appearance can be assessed. Filling of the ureter can be estimated and, if parts of the kidney are abnormal, visually guided biopsies can be taken.

In general, the routine endoscopic approach into the left caudal thoracic air sac is recommended [33]. With the limb retracted caudally, the insertion point is the middle of the triangle formed by the last rib, the iliotibial muscle, and the caudal border of the synsacrum. The abdominal air sac can be entered by puncturing its wall, allowing a direct view to the kidneys. Not only the urinary system can be assessed, but so too can most of the other organs. Visceral gout on the pericardium or liver capsule can be detected. Primary diseases causing radiologic changes can be seen.

Anatomical differences between species must be considered during assessment of the renal system. Size, shape, and surface vary between species.

Small yellow or white spots within the kidney are signs of gout or a disturbed excretion process (Fig. 8). A filled and swollen ureter might be due to

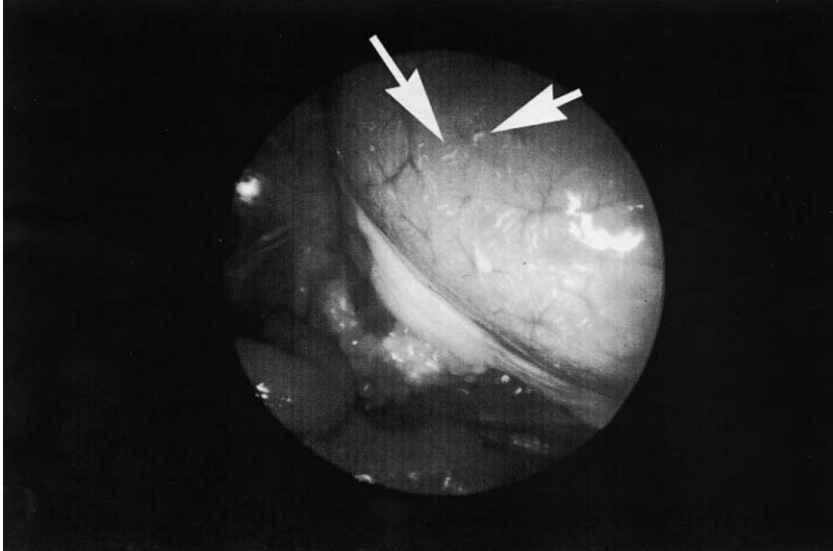


Fig. 8. Endoscopic view of urate deposits in the kidney of a black kite (*Milvus migrans*). This change is not visible on radiographs and often reversible with fluid therapy.

obstruction or dehydration. This might be reversible by fluid therapy (Fig. 2). Loss of the typical surface structure of the kidney is often a sign of glomerulonephritis while larger single yellow masses may be pyelonephritis abscesses or lymphosarcoma (Fig. 9) [33]. Kidney biopsies are necessary to differentiate different conditions.

Kidney biopsy

Kidney biopsies are recommended in cases of persistent polyuria, polydypsia, elevated uric acid levels, or abnormal gross appearance of renal parenchyma [34]. In most cases a kidney biopsy is performed during endoscopic examination. Suedmeyer and Bermudez [35] described a dorsal pelvic approach to the kidney for taking biopsies, but tissue damage is greater than with endoscopy and the kidney is not visualized. Endoscopy-guided kidney biopsy through a working channel (67065 CC, Karl Storz Veterinary Endoscopy America, Inc., Goleta, CA) with a cup-shaped flexible biopsy forceps (5 F, 67161Z, Karl Storz Veterinary Endoscopy America, Inc.) is recommended. The location of larger vessels should be kept in mind.

Computed tomography

Computed tomography has limited value for renal investigation. The procedure is expensive, complicated (taking the patient to the equipment,

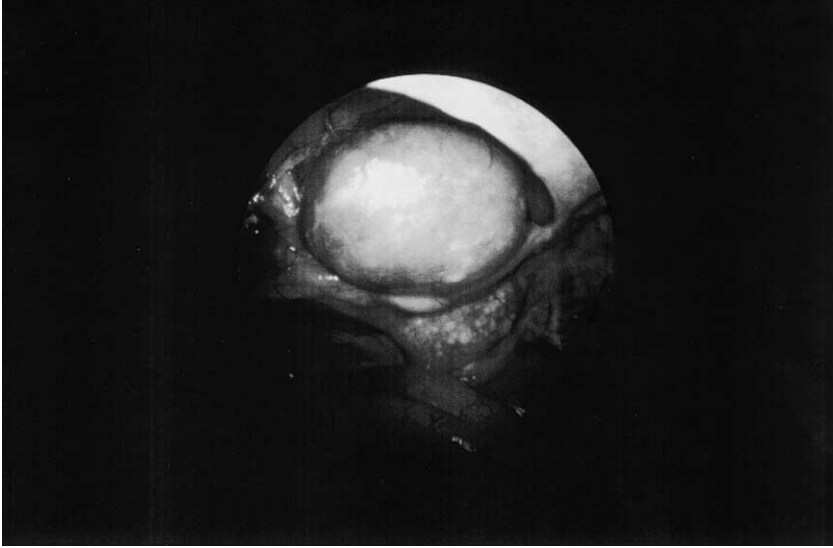


Fig. 9. Endoscopic view of a swollen, altered kidney with yellow spots, diagnosed by histopathology as pyelonephritis in an African gray parrot (*Psittacus errithacus*).

sedation, etc.), and impractical for the veterinary surgeon in practice but can be helpful for investigation of neoplasia. Sedation is essential often requiring injectable drugs with higher narcotic risk where inhalation anesthesia is not be available in the tomography center. Technical factors have been inadequately studied in birds. Krautwald-Junghanns [36] described slice section thickness from 2 mm to 5 mm nonoverlapping with varying window settings (Fig. 10).

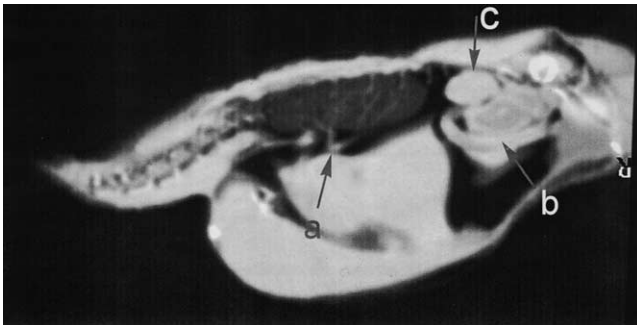


Fig. 10. Normal computed tomography picture of a Goshawk (*Accipiter gentiles*). Apart from the kidney (c) the lung vessels (a) and the intestine (b) is visible.

Renal disorders

Renal disorders are common in birds and mostly secondary to other diseases or management problems. Renal diseases can be categorized as infectious and noninfectious.

Congenital defects

Complete absence of the kidneys has been reported but leads to early embryonic death [37]. Hypoplasia with compensatory hypertrophy of the intact parts can be found more often [38]. Clinical signs can be found only if a large part of the kidney is dysfunctional.

Atrophy or hypotrophy must be differentiated from congenital hypoplasia. Hypotrophic kidneys may be found in birds with ascites, displaced eggs, or impacted oviducts where pressure on the organ is increased. Renal atrophy can block the ureter [39]. Clinical signs occur if the hypertrophic parts cannot compensate the loss of function.

Kidney cysts are thought to be congenital but may have other causes [40]. Diagnosis of aplasia or hypoplasia can be made by endoscopy or urography confirmed by histologic examination. Renal cysts can be detected by ultrasonography. Curative therapy may be impossible. Single large cysts may be punctured but a successful outcome is not guaranteed.

Gout

Gout is more a sign of renal failure than a distinct disease. Gout occurs if plasma sodium urate concentration exceeds its solubility. The solubility is estimated as approximately 600 $\mu\text{mol/L}$ as a minimum level [9]. As uric acid is excreted via tubular secretion 70% of the kidney must be malfunctioning to cause a hyperuricemia [9]. Apart from renal failure, dietary protein above the bird requirements might also cause a hyperuricemia [9]. In dehydration the kidney is still able to secrete uric acid, but low urine flow within the tubules allows crystals to precipitate, which causes postrenal obstructions, followed by anuria or oliguria [39]. This is known as “renal gout.” Hyperuricemia can result in visceral or articular gout. In cases of renal failure without precipitation in the renal tubules, articular gout, or less commonly, visceral gout, occurs alone. According to Lumeij, joints might be predilection sites for uric acid deposits as the uric acid solubility is lower than in other areas [9].

Visceral gout

Visceral gout is the deposition of urate crystals on the visceral membranes, mostly on pericardium, liver, and spleen. Urate deposits are also visible within the kidney. In acute renal failure visceral gout might occur alone [9]. Inflammatory reactions are often not detected, as birds die rapidly.

Visceral gout usually develops without obvious clinical signs prior to death. In cases where renal urate deposition occurs prior to visceral gout, anorexia and lethargy may be noted for hours or days. Hyperkalaemia can develop, and this, rather than the uric acid, might lead to cardiac arrest and the sudden death seen with visceral gout. In postmortem examinations pericardium and liver membranes are white, resembling powdered sugar. Sometimes subcutaneous deposits are present (Figs. 11 and 12). Histologic examination confirms the diagnosis by demonstrating urate tophi.

Articular gout

Articular gout is a common problem in psittacines, and may be diet related. Urate deposits are found in the joints. The disease develops slowly. Small nodules (tophi), mostly at the metatarsophalangeal area are reported first. This condition is very painful and lameness is often present. Blood values are often increased in these birds. Radiographs often show increased kidney opacity. Urate deposits can be demonstrated in chronic cases. Articular gout can be diagnosed using the murexide test. White, toothpaste-like material (typical for gout), can be aspirated from the joint. It is mixed with nitric acid and dried over a flame. If a drop of concentrated ammonia is added, a mauve color will develop if uric acid is present [9].

Urate deposits in the kidney

Urate deposits within the kidney (Fig. 13) are seen in dehydrated birds. On radiographs, the renal density is increased. Small yellow spots are visible on



Fig. 11. Gout tophi on the pericardium and liver of a peregrine falcon (*Falco peregrinus*).

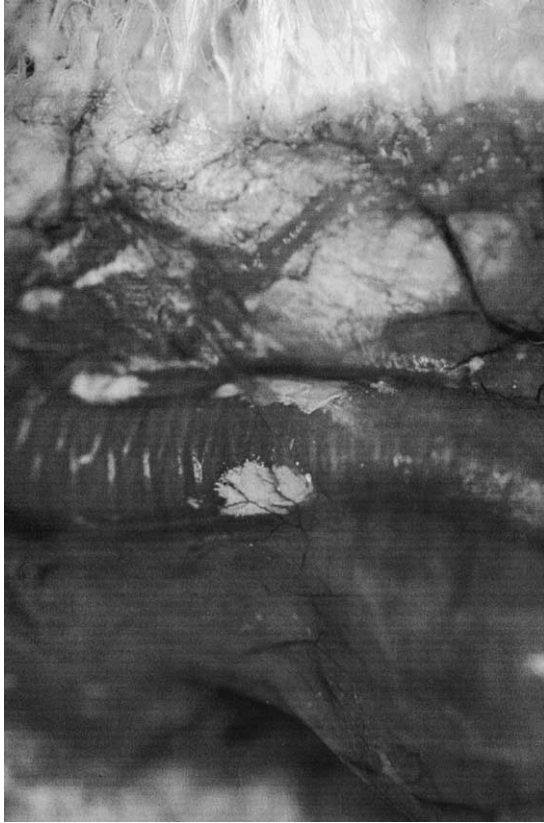


Fig. 12. Urate deposits on the trachea of a peregrine falcon (*Falco peregrinus*).

the kidney during endoscopy (Fig. 8). Clinical signs do not occur in early stages but later there may be anorexia, vomiting, and lethargy. In most cases this state is reversible with fluid administration. Therapy of gout is very difficult. Visceral gout is not treatable, and in most cases is diagnosed postmortem.

Urate precipitation within the tubules caused by dehydration is treatable. Fluid therapy is rewarding. Expanding the circulatory volume with intravenous fluids normally restores renal function in a short time. Maintenance fluid requirements are generally 5–6% body weight, but 10% should be administered intravenously, intraosseously, or subcutaneously in divided doses for rehydration process [3]. Once hyperuricemia is diagnosed prognosis is poor. Mannitol 20% (1000 mg/kg) every 15–20 minutes, and sodium bicarbonate supplemented by intravenous furosemide is recommended in humans to initiate a high alkaline urine flow, and might be useful in birds as well [10]. Vitamin A is useful, as hyperuricemia may be caused by hypovitaminosis A. In cases of gout allopurinol, as a xanthine oxidase inhibitor is recommended at a dose of 40 mg/kg/day to reduce plasma uric acid



Fig. 13. Urate deposits in the kidney (c) in a Peregrine falcon (*Falco peregrinus*). The ureter (b) is also filled with urates. For orientation the ovary is marked (a). Prognosis in such severe cases is poor.

concentrations [12]. In contrast, Lumeij and Redig [41] observed the induction of gout in red-tail hawks after using this drug.

Acute and chronic renal failure

Shock is often the cause of acute renal failure and resulting anuria. If the cause is treated, the renal failure is often reversible. Chronic renal failure can be caused by nephrotoxic agents, chronic dehydration, and ureter obstruction. Clinical signs often develop late. Nonspecific signs such as anorexia, vomiting, and lethargy are reported [9].

Restoration of renal function in acute renal failure is possible if treatment occurs immediately. Treatment considerations are mentioned above (see gout). In most renal disorders fluid administration is very important, and intravenous or intraosseous catheters are recommended. In anuric or oliguric birds fluid administration must be performed carefully. Fluid loss can be assessed by calculating the renal, gastrointestinal, and insensible loss (20 ml/kg/day) [9]. Daily weight control can be of additional help. Furosemide (0.15–2 mg/kg once a day) can be given intramuscularly or intravenously. Anuric or oliguric birds are unable to maintain their electrolyte balance. Sodium, potassium, and protein intake should be discontinued. Low protein liquid diets assist in therapy [42]. Monitoring plasma electrolytes is useful. Where treatment is successful a polyuric phase follows the renal failure. Now fluid therapy and electrolyte assessment are important to prevent

dehydration, hypokalemia, and hyponatremia. It is most important to find the cause of the disease. In parrots, vitamin A deficiency is often implicated so prophylactic treatment is recommended (5000 IU/kg intramuscular, once daily). Bacterial infections (primary or secondary), must be considered and treated appropriately. Sources of possible intoxications (lead, sodium, zinc) must be eliminated. Prognosis for chronic renal failure is poor. Treatment considerations are similar to acute renal failure.

Metabolic disorders

The kidney is involved in various metabolic systems. Well known in poultry but also occurring in pet birds is fat deposition within the renal tubuli as a result of lipidaemia, toxic influence, or lack of energy or oxygen [39]. Biotin deficiency might be another cause [43].

Hypervitaminosis D₃ and/or excessive dietary calcium often leads to hypercalcemia and soft tissue mineralization of the renal parenchyma, or nephrocalcinosis. Ureteral concretions can cause postrenal renal failure [9]. Radiographs, urography, endoscopy, and ultrasonography can lead to a diagnosis.

Hypovitaminosis A is thought to cause metaplasia of renal tubules and ureteral epithelium, leading to ureteral obstruction by urate precipitates. Urography might show the obstruction, but specific diagnosis is difficult unless other disorders caused by a hypovitaminosis A are noted. In some cases histologic examination of biopsies may confirm the diagnosis.

Deposition of amyloid at the tubular and glomerular basal membranes is well known in ducks [44]. In most cases it is part of a generalized amyloidosis, which causes severe glomerular damage resulting in nephrotic syndrome, and possibly gout. It often follows chronic inflammation, for example, in falcons with a long history of pododermatitis. Despite good appetite, birds lose weight and appear lethargic. Clinical signs may include ascites or edema of the limbs, and diagnosis is confirmed at postmortem or via kidney biopsy [9]. If metabolic disorders are diagnosed, treatment should focus on the primary problem. Symptomatic treatments of the renal problems are listed above (renal failure).

Ureteral obstruction (urolithiasis)

Ureteral obstructions are rare in birds but can be caused by urates or calcium salts (see above), neoplasias, inflammatory processes and cloacal prolapse. Bilateral obstructions lead to visceral gout while unilateral obstructions cause atrophy of the affected kidney and hypertrophy of the contralateral side [45–47]. In domestic fowl bilateral ureteral obstruction caused by urolithiasis has been reported to cause sudden death [39]. Clinical signs include lethargy and straining. Paralysis of one leg might occur due to the

hypertrophic kidney compressing the nerve plexus. In psittacines, bleeding from the cloaca has also been described [16]. Unilateral obstructions can be diagnosed by excretion urography. The prognosis is very poor for the affected kidney. Fluid therapy can be tried in an attempt to dissolve the blockage, but in cases of complete obstruction aggressive fluid therapy is contraindicated. Prophylactic Vitamin A should always be administered, in the case of hypovitaminosis A.

Renal hemorrhage

Renal bleeding occurs after trauma. If the kidney capsule is not damaged a hematoma will form increasing the kidney size. This may lead to pressure on the nerve plexus, resulting in lameness. Differentiation from neoplasia is very difficult, but can be achieved using endoscopy. In cases of severe kidney trauma or neoplasias birds may bleed to death. Iatrogenic renal hemorrhage can occur following endoscopic biopsy, but is usually minor and of little consequence. Petechial bleedings can be observed in toxæmia including that caused by bacterial toxins (eg, *Clostridium perfringens*) (Fig. 14) [16].

Neoplasia

Renal tumors are common in budgerigars [16]. In many cases owners report lethargy and lameness in one leg (Fig. 4). Tumor masses increase the



Fig. 14. Renal petechial hemorrhage as a result of a *Clostridium perfringens*—intoxication in a rock partridge (*Alectoris graeca*).

pressure on the nerve plexus, leading to paralysis. The bird is unable to grab the cage wire or perch. As kidney tumors are mostly cranial they are difficult to distinguish from gonadal neoplasia. If paralysis occurs at the left leg and radiographs demonstrate hyperostosis, an ovarian tumor is more likely. Only a few tumors are palpable; mostly, they are suspected from radiographs. Endoscopy or ultrasonography and biopsy supports the diagnosis. Kidney biopsy is essential for accurate diagnosis and prognosis. Adenomas, adenocarcinomas, and nephroblastomas have been reported as primary renal tumors [9,16]. Open surgical resection of renal neoplasias is seldom possible, but future developments in endoscopy may prove useful.

Nephrosis

Nephrosis describes degenerative kidney changes without inflammation. In most cases the proximal and distal tubuli are affected, rarely the collecting ducts [39]. Glomerular atrophy or hypertrophy can occur [16]. Nephrosis develops following exposure to toxins or secondary to infections that need not involve the kidney [39].

Toxin-induced nephropathies

Nephrotoxins commonly cause renal tubular necrosis. Psittacines are especially sensitive to heavy metals (eg, lead) and mycotoxins (aflatoxin, ochratoxin) [9]. Mycotoxins can be ingested with poor-quality food contaminated by *Aspergillus* spp. Some drugs, for example, gentamycin, are also known to be nephrotoxic. Pesticide contaminated fruits and vegetables can cause renal tubular necrosis. Salt poisoning can occur in pet birds, and may ensue following the feeding of salted nuts or chips [9]. Nutritional toxicosis can lead to acute renal failure with ureteral urate accumulation [39]. Salt water ingestion can cause nephropathy and heart failure [9]. A more extensive review of nephrotoxins and associated kidney lesions can be found in Siller [39].

Polydypsia and polyuria (Fig. 15) are common clinical signs, but in cases of acute renal failure anuria may occur. Birds are acutely lethargic and vomit. Nephrosis is irreversible. Treatment is symptomatic (see renal failure), but euthanasia may be required if renal function cannot be restored. Drinking water should be replaced by low-sodium water produced for humans with kidney disease. Treatment may restore renal function in nonnephrotic areas.

Nephritis

Nephritis tend to occur secondary to systemic infections, and is defined by histologic visible inflammation. Some pathogens, for example, Paramyxovirus, have a specific tropism for kidney cells. Histologically, nephritis can be divided into glomerulonephritis and interstitial nephritis, and both can



Fig. 15. Dropping from an African Gray parrot (*Psittacus erithacus*) demonstrating polyuria.

appear acute or chronic. Acute nephritis is common in septicaemic birds, due to the hematogenous spread of pathogens to the kidney [16]. Ascending infections from the cloaca also occur [16]. Pyelonephritis is more common in cases of bacterial or parasitic infections (Fig. 9). Clinical signs are dominated by the coexisting systemic infection. Polydypsia and polyuria are observed, with anuria during the terminal stage. Chronic nephritis may develop following acute disease. In most cases the interstitium, tubules, and glomeruli are affected. Urate deposits are common. Clinical symptoms are rare, as the kidney compensates for loss of functional tissue. In some cases stress leads to the observed signs of lethargy, weight loss, and anorexia. Polydypsia develops very late in the course of the disease.

Bacteria

Many kinds of bacteria can cause renal infections secondary to a systemic disease. Common pathogens such as *Staphylococcus* spp., *Streptococcus* spp., *E. coli*, *Salmonella* spp., and other enterobacteraceae, *Pasteurella* spp., and *Mycobacterium avium* are most often isolated. *Chlamydomphila psittaci* leads to necrosis within the renal tissue. Mycotic infections are rare, but toxin-induced nephropathy is often associated with aspergillosis [9].

Viruses

Many viruses cause systemic disease, and the kidney can be involved. Infectious bronchitis virus is known to have a tropism for renal cells, but paramyxovirus, reovirus, or herpesvirus may also affect the kidney [39].

Parasites

The cause of renal coccidiosis in geese (*Anser* spp.) is *Eimeria truncata*. The trematode, *Tanaisia bragai* may cause obstruction of the renal collecting

ducts, and has been reported in passeriformes, chickens, pigeons, and penguins [9,16]. *Isospora* spp., *Cryptosporidium* spp., and *Encephalitozoon* spp. can cause granulomatous nephritis in various bird species, such as galliformes, columbiformes, and passeriformes [9,16].

Diagnosis of nephrosis or nephritis can only be made following renal histology, but may be interred following radiography, urography, and endoscopy.

Nephromegaly is a common finding, and is best evaluated by radiographs, while Pyelonephritis can be detected best endoscopically. In all cases renal biopsies are recommended to make a definitive diagnosis.

Treatment should focus on the cause of the disease, making an accurate diagnosis very important. Antibiotics or antiparasitic drugs may be required. Siller described the administration of sodium and potassium bicarbonate to fowl with infectious bronchitis-nephritis. A reduction in the mortality was observed as plasma levels of sodium and potassium are decreased due to the nephritis. Therapy should be planned according to the clinical findings and correction of fluid and electrolyte imbalances.

Drugs that are nephrotoxic or excreted by the kidneys should be used with extreme caution. Intramuscular or intravenous drugs given in the pelvic limbs may reach toxic concentrations within the kidney due to the renal portal system. Injection into the pectoral muscle or ulnar vein is therefore recommended. Drugs that are excreted through the kidney may fail to reach therapeutic plasma levels in polyuric birds, or reach toxic levels if drug excretion and elimination is impaired.

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