

Renal Disease

Renal Disease—Case-Based Approach to Acute Renal Failure, Chronic Renal Failure and Protein-Losing Nephropathy

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Presenting Complaint/History

The most consistent presenting complaint for patients with chronic renal failure (CRF) and protein-losing nephropathy (PLN) is polyuria/polydipsia (PU/PD), though occasionally the owner does not recognize these clinical signs. Other historical findings for chronic renal failure may include weight loss and decreased appetite. Intermittent vomiting may be seen if secondary uremic gastric ulcers are present. In many cases, a CRF patient will be asymptomatic (other than PU/PD) until dehydration (potentially due to an unrelated cause) leads to decompensation, leading to a more acute history from the owner's perspective. Although many PLN patients are asymptomatic early in the course of disease, history may include weight loss, PU/PD, lethargy, signs related to secondary CRF, and/or signs associated with an underlying cause (e.g., lameness). Occasionally, an animal with PLN will present with a distended abdomen because of ascites or dyspnea secondary to pleural effusion; these clinical signs result from a low colloid oncotic pressure secondary to hypoalbuminemia. While CRF and PLN can show up as an incidental finding on annual well-check bloodwork, the patient with acute renal failure (ARF) generally presents acutely ill. Common findings with acute renal failure may include sudden onset of lethargy, anorexia, and/or vomiting. Unlike chronic renal failure patients who are usually polyuric, acute renal failure patients are frequently oliguric or anuric. Patients that are oliguric or anuric usually have a poorer prognosis than ARF patients that are polyuric.

Physical Exam Findings

Physical exam findings in CRF patients reflect the chronic nature of the disease and include poor body condition, unkempt hair coat and small irregular kidneys. Oral exam may reveal ulcers, uremic breath odor and/or pale mucous membranes. Many PLN patients have normal physical exams. As the disease progresses, physical exam findings consistent with CRF may develop. Lameness, fever and other evidence of chronic infectious, inflammatory or neoplastic underlying conditions may be present. Secondary systemic hypertension may be reflected in retinal hemorrhages,

arterial tortuosity or detached retinas in both CRF and GN patients. ARF patients, in contrast, often have very good body condition. Unlike CRF and PLN patients, the kidneys may be enlarged and painful. Fever and oral ulcerations may also be seen.

Minimum Database

• Complete Blood Count (CBC)

Red Blood Cells

- Anemia occurs in up to 50% of dogs and cats with chronic renal failure and is often normocytic, normochromic and non-regenerative reduced production of or response to erythropoietin.
- On occasion, relative polycythemia due to dehydration will mask a concurrent anemia, but once fluid therapy is successful the PCV will then reflect true red cell numbers. Rarely, renal diseases associated with hypoxia or autonomous erythropoietin production can cause secondary absolute polycythemia.
- Although unusual, anemia may be detected in patients with acute renal failure, typically developing as a result of blood loss such as gastrointestinal hemorrhage.

White Blood Cells

- Neutrophilic leukocytosis can be seen with a variety of inflammatory lesions of the renal system. Depending on the severity and chronicity of the lesion, neutrophilia can be variably associated with a "left shift" or a peripheral leukocytosis may appear to resolve when bone marrow production capacity meets demand.
- Chronic renal failure can be associated with lymphopenia, which reflects the effects of endogenous glucocorticoids or stress of chronic disease. Mild mature neutrophilia is commonly seen associated with this glucocorticoid effect, too.

- **Chemistry**

- **Urea**

- Increases in urea in renal failure are caused by impaired ability to excrete proteinaceous catabolites because of marked reduction in glomerular filtration rate (GFR). Urea is also directly related to the protein content of the diet. Urea can also be increased by gastrointestinal hemorrhage, enhanced protein catabolism, decreasing urine volumes (due to prerenal factors such as dehydration) and certain drugs (e.g., glucocorticoids).

- **Creatinine**

- Increases in creatinine are also a result of decreased renal excretion. Because many extrarenal factors may influence urea concentration, creatinine is often used as a more reliable indicator of glomerular filtration rate in patients with renal disease.

- **Phosphorus**

- Hyperphosphatemia may be caused by decreased renal excretion of phosphate, translocation of phosphate from intracellular to extracellular fluid, and increased intake of phosphate.
 - Compensatory renal secondary hyperparathyroidism maintains serum phosphorus concentration within the reference interval until $\geq 85\%$ of nephrons are non-functional. Hyperphosphatemia is not observed in renal failure until after the onset of azotemia or loss of $\geq 75\%$ of the nephron population.
 - Serum phosphorus concentration often is increased in acute renal failure with severe reduction in GFR ($< 15\%$ of normal).

- **Potassium**

- Hyperkalemia: may be seen in acute anuric or oliguric renal failure, urethral obstruction or ruptured bladder due to decreased urinary excretion. Therapy with ACE-inhibitors may also lead to hyperkalemia.
 - Hypokalemia: commonly seen in chronic renal failure and in post-obstructive diuresis due to increased loss through the kidneys. Decreased dietary intake may be a contributing factor in inappetent patients. Gastrointestinal loss may also be contributory in patients with vomiting or diarrhea.

- **Calcium**

- Hypercalcemia may be caused by acute or chronic renal failure and accentuated by concurrent dehydration.

- Renal failure-induced hypercalcemia, as indicated by increased total serum calcium concentration, will typically be associated with a concurrent ionized serum calcium concentration within or just below the reference interval.
 - Most dogs and cats with chronic renal failure ($> 90\%$) will have total serum calcium concentration within the reference interval.
 - Hypercalcemia can further damage the kidneys by causing renal vasoconstriction and renal interstitial mineralization. Possible mechanisms of hypercalcemia in renal failure include:
 - Reduced urinary excretion of calcium due to low GFR
 - Decreased renal degradation of PTH
 - Autonomous parathyroid gland secretion of PTH
 - Increased PTH set point for calcium
 - Increased intestinal sensitivity to low concentrations of calcitriol
 - Total serum calcium concentrations are decreased in approximately 10% of dogs with chronic renal failure.
 - Decreased serum ionized calcium concentration is found in 40% of dogs with chronic renal failure and mechanisms include:
 - "Mass law" effect due to increased serum phosphorus concentration (i.e., the amounts of calcium and phosphorus that can remain in solution together are defined by the $[Ca] \times [P_i]$ product).
 - Decreased production of calcitriol by kidneys resulting in impaired intestinal absorption of calcium.
 - In cases of uremia there can be skeletal resistance to the action of PTH.
 - Hypocalcemia may be caused by hypoalbuminemia (correction factor doesn't apply to cats), acute or chronic renal failure or ethylene glycol intoxication.
 - Hypocalcemia in chronic renal failure usually is asymptomatic, since concurrent metabolic acidosis of renal failure can lead to an increased ionized component of serum calcium.
 - Hypocalcemia also may occur in acute renal failure as a result of severe hyperphosphatemia and "mass law" effect.

Total CO₂ or HCO₃⁻

- The total CO₂ content is a measure of all sources of CO₂ that contribute to plasma or serum.
 - If the sample is handled aerobically, dissolved CO₂ is released to the atmosphere and the total CO₂ measurement is essentially equal to the concentration of HCO₃⁻ in the sample. In routine clinical practice the total CO₂ determination often is considered synonymous with [HCO₃⁻].
 - Chronic renal failure is accompanied by a mild to moderate well-compensated metabolic acidosis from decreased renal excretion of fixed acid. Decreased Total CO₂ may be seen.
 - In acute renal failure, metabolic acidosis may be more severe, because of insufficient time for renal compensatory responses. Decreased Total CO₂ may be seen.

Albumin

- Hypoalbuminemia can occur secondary to renal loss in PLN. Hypoalbuminemia may also be seen with renal inflammatory disease (albumin is a negative acute phase reactant).
- Hyperalbuminemia can occur secondary to hemoconcentration from dehydration in any type of renal disease.

Globulin

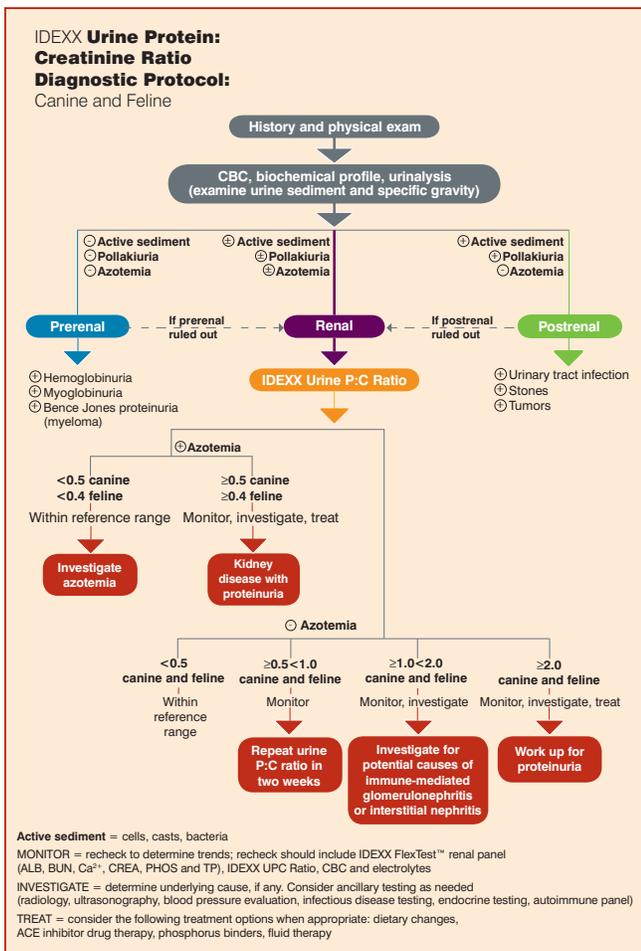
- Globulin levels are typically normal or elevated in PLN since these proteins are too big to be lost through the kidneys.
- Hyperglobulinemia can occur secondary to a chronic pyelonephritis or a chronic inflammatory, infectious or neoplastic disease which might be the underlying cause of a glomerulonephritis or amyloidosis.

Cholesterol

- Hypercholesterolemia is very common in dogs with glomerulonephritis and amyloidosis. The pathogenesis is complex and not fully understood.
- **Urinalysis:** Complete urinalysis should include observation of color and clarity, urine specific gravity measured by refractometry, urine pH, bilirubin, glucose, occult blood, ketones and screening for proteinuria. Urine sediment should be examined for red blood cells, white blood cells, epithelial cells, casts, organisms and crystals.
 - Patients in renal failure can have isosthenuric urine (specific gravity [SG] 1.008-1.012). Cats and dogs with early renal insufficiency may

have minimal ability to concentrate urine. A SG of less than 1.035 in a dehydrated feline patient or less than 1.025 in a dehydrated canine patient is considered suspicious for decreased renal function. Concentrating ability generally decreases before the development of azotemia. Patients with PLN may maintain some concentrating ability before the development of secondary tubular damage.

- An active sediment (bacteriuria, white blood cells, red blood cells or casts) may suggest infection. An attempt should be made to localize the infection to the upper or lower urinary tract because this may affect therapy.
- Casts are cylindrical molds of the renal tubules and are composed of aggregated proteins or cells. The presence of casts in the urine sediment localizes activity to the kidney itself. Occasional hyaline and rarely seen fine granular casts per low power field may be considered normal if there are no other findings associated with renal disease. Cellular, coarse granular and waxy casts are always pathologic.
- **Urine Protein:Creatinine Ratio (UPC):** Urine protein screening tests include urine dipsticks, sulfosalicylic acid (SSA) precipitation test and microalbuminuria assay. Urine dipsticks are prone to false positive results in highly concentrated or alkaline urine (pH>7.5) and may not be sensitive enough to detect small amounts of protein. If protein is detected by an SSA test on urinalysis or by microalbuminuria assay and the sediment is inactive, a UPC should be performed. The UPC offers a technique for quantitative measurement of proteinuria in dogs and cats, and is available through reference laboratories, as well as select in-house chemistry analyzers. The UPC can be used to screen for early renal disease, confirm other proteinuria screening tests, quantify protein loss, aid in discovery of protein origins (localization), prove persistence, monitor disease progression and evaluate therapeutic response. Recent studies of dogs and cats with persistent proteinuria show that these patients are at greater risk to become ill or die from renal disease, and are more likely to die from other, non-renal, diseases. Proteinuria has varied causes and clinical consequences. Distinguishing between physiologic or pathologic causes and extra-renal or renal origins of proteinuria is of utmost importance.
- **Urine Culture and Sensitivity:** The absence of an active sediment in isosthenuric urine does not rule out the presence of infection. Likewise, not all pyelonephritis patients have an abnormal



CBC. In the absence of bacteriuria, consider urine culture if the urine is poorly concentrated (SG ≤1.015) or has >5 WBC/hpf or if the patient has an endocrine disorder (i.e., diabetes mellitus, hyperadrenocorticism or hyperthyroidism).

Differentiating ARF, CRF and PLN

Early PLN is differentiated from CRF and ARF by the absence of azotemia and the presence of an elevated UPC. A lack of urine-concentrating ability usually precedes azotemia in CRF—this stage is termed early renal insufficiency. PLN eventually progresses to CRF in most patients. Late stage PLN (with progression to CRF) will have all the clinical signs of CRF in addition to significant proteinuria. As progressive damage occurs in PLN, the UPC may drop somewhat reflecting the decreased number of functional nephrons. Proteinuria can also be seen in CRF secondary to tubulointerstitial nephritis (the form of CRF more commonly seen in older cats), but it is usually present to a lesser degree than that seen with PLN. Differentiation of ARF and CRF should be possible in most cases based on the presenting complaint/history, physical exam and minimum database results (see table at top of right-hand column).

	ARF	CRF	GN
Body condition	Good	Poor	Variable
Clinical signs	Acute onset, severely ill. Vomiting, anorexic, lethargic/depressed.	Chronic, gradually progressive signs. Weight loss; decreased appetite; chronic vomiting. Often not very ill for degree of azotemia.	Asymptomatic until CRF; weight loss, signs related to underlying disease (e.g., lameness, neoplasia); ascites/pleural effusion
PU/PD	May be poly-, an-, or oliguric	+++	+ → +++
Kidneys on PE	Often enlarged/painful	Small, irregular	N to small
Azotemia	+	+	+/-
Proteinuria	Usually N, depending on cause	N to slight increase	+++
Anemia	Not usually unless gastrointestinal ulceration and hemorrhage	Usually depending on severity of renal disease	+/-

Differential Diagnoses

- Acute Renal Failure:** (Investigate the potential for Addisonian crisis) Acute pyelonephritis, Leptospirosis, ureteral obstruction, lymphoma, FIP, toxins (e.g., Ethylene glycol, NSAIDs, lilies, heavy metals, aminoglycoside therapy etc.), secondary to hypotensive episode (e.g., anesthesia, shock, severe dehydration).
- Protein-losing nephropathy:** (Investigate the potential for prerenal and postrenal causes of proteinuria.) It has been accepted that amyloidosis and glomerulonephritis (GN) are the most common glomerular diseases in dogs and cats. Glomerulonephritis can be further characterized as membranous glomerulonephropathy, membranoproliferative glomerulonephritis and proliferative glomerulonephritis. Glomerulonephropathies may be idiopathic or may be a secondary process. Any chronic inflammatory, infectious or neoplastic condition has the potential to induce a PLN and many can induce amyloidosis. Infectious diseases that should be considered include bacterial endocarditis, pyometra and other chronic bacterial infections including gingivitis, heartworm infection, systemic mycotic infections, (e.g., coccidiomycosis), rickettsial infections (*Ehrlichia* sps., *Anaplasma* sps., RMSF), brucellosis, *Borrelia burgdorferi*, and *Bartonella* sps. White blood cell cancers (lymphoma, multiple myeloma) are especially likely to cause GN through

production of immunoglobulins by the cancer cells. Glomerulonephritis can be associated with immune-mediated diseases and can be an important component of systemic lupus erythematosus. In many cases, an underlying cause is not determined, and the condition is considered idiopathic.

- **Chronic Renal Failure:** In most cases, by the time chronic renal failure has been diagnosed, it is not possible to identify the cause of the renal disease. Some causes that might be identified or suspected based on signalment or imaging include polycystic kidney disease (ultrasound diagnosis; Persians, cairn terriers, beagles, other long-haired cats), renal dysplasia (young animals with abnormal kidney architecture on ultrasound), amyloidosis (Shar-Peis, Abyssinians), Fanconi syndrome (Basenjis), and glomerulonephropathy with secondary CRF. Chronic pyelonephritis should also be considered as a differential for CRF. Biopsy findings in both dogs and cats most commonly reveal chronic tubulointerstitial nephritis with glomerulonephropathy being the second most common finding.

Additional Diagnostics

- **Blood Pressure:** Systemic hypertension is a common complication of CRF and especially of PLN. Uncontrolled hypertension can cause further damage to the nephrons, leading to more rapid progression of the disease. Control of hypertension, therefore, is an important component of therapy. Systemic hypertension is usually detected by indirectly measuring blood pressure. The doppler ultrasonic method is the method of choice, but oscillometric techniques can be useful in dogs.
 - **Antithrombin III** (Hypoalbuminemic PLN patients): Antithrombin III is important in the prevention of thromboembolism. It is similar in size to the albumin molecule. When protein loss through the kidneys leads to hypoalbuminemia, decreased antithrombin III levels are likely to also be noted. Serum levels can be measured or patients who are hypoalbuminemic can be given antithrombotic therapy as a precaution.
 - **PTH/Ionized Calcium:** This evaluates for renal secondary hyperparathyroidism, which is usually accompanied by normal or elevated total serum calcium. Ionized calcium is typically low normal to decreased with normal to elevated PTH levels. Additionally, in patients with elevated total serum calcium, determination of ionized calcium and parathyroid hormone levels are recommended to determine if the hypercalcemia is secondary to the renal disease or perhaps causing the renal disease (e.g., hypercalcemia of malignancy, vitamin D intoxication).
- **Imaging:**
 - **Radiographs:** The presence of small, irregular kidneys on radiographs provides strong evidence for chronic renal disease. However, normal or enlarged kidneys do not exclude CRF. The presence of unilateral renal enlargement may represent neoplasia, compensatory hypertrophy, perirenal cysts or hydronephrosis secondary to ureteral obstruction (particularly common in cats) and should be further evaluated by ultrasound. Kidney size may be increased in acute renal failure. Areas of nephrocalcinosis may also be seen in chronic renal disease.
 - **Ultrasound:** Ultrasound is often superior to radiography in obtaining accurate renal measurements. Hyperechogenicity of the renal cortex and loss of corticomedullary definition are supportive of primary renal disease. Familial renal dysplasias and glomerulopathies may be accompanied by grossly abnormal architecture. Specific causes for chronic and acute renal failure may be detected by ultrasound including polycystic kidney disease, neoplasia, ureterolithiasis and pyelonephritis (although findings may be normal in pyelonephritis). Infiltrative diseases of the kidneys may also be suspected; e.g., renal lymphoma, FIP based on ultrasonographic findings. Normal findings on ultrasound do *not* exclude the possibility of renal disease.
 - **Biopsy or Fine Needle Aspiration:** Cytology and histopathology are rarely performed in the management of renal disease in small animals. Fine needle aspiration may be indicated to diagnose a suspected lymphoid neoplasm. Histopathology of PLN may allow differentiation of amyloidosis, glomerulonephritis, familial nephropathies or minimal change syndrome. Collection of urine from a dilated renal pelvis by ultrasound guidance may yield a better sample for urine culture in cases of suspected pyelonephritis.
 - **Ancillary Testing:** Renal disease has a variety of inciting causes that may eventually lead to renal failure. In addition to the Minimum Laboratory Database (e.g., CBC, biochemistry panel, fecal analysis, complete urinalysis), testing for the following diseases may be indicated based on signalment, history, physical examination and MDB findings:
 - Immune-mediated or autoimmune disorders
 - Neoplasia
 - Bacterial sepsis

- Rickettsiae (e.g., *Ehrlichia* spp., *Anaplasma* spp., RMSF)
- Spirochetes (e.g., *Borreliosis*, *Leptospirosis*)
- Viral infections (e.g., FeLV, FIV)
- Heartworm infection
- Systemic mycotic infections
- Brucellosis
- Severe inflammatory disorders
- Metabolic disorders (e.g., hyperadrenocorticism, hyperthyroidism)

Therapeutic Management

• Address Underlying Causes (if known)

- Leptospirosis: antibiotic therapy (ampicillin, penicillin, amoxicillin, doxycycline)
- Pyelonephritis: antibiotics chosen based on culture and susceptibility and ability to penetrate renal tissue
- Lyme disease (*borreliosis*): antibiotic therapy (doxycycline, amoxicillin) plus therapy for PLN
- Ethylene Glycol toxicity: Fomepizole (dogs), ethanol (cats); hemodialysis if available locally; peritoneal dialysis
- Shar Pei Fever: colchicine
- Ureteral obstruction: surgery and medical management to prevent recurrence
- PLN: treatment of underlying systemic inflammatory, infectious or neoplastic disease

• Specific Therapy for Proteinuria

- ACE inhibitors (enalapril, benazepril, lisinopril): 0.25 to 0.5 mg/kg SID to BID. Lowers the amount of protein loss through the kidneys by causing vasodilation of the efferent renal arteriole, thus reducing intraglomerular pressure. ACE inhibitor therapy has been proven in a multicenter clinical trial to limit progression and in many cases results in improvement of PLN.
- Moderately protein-restricted diet
- Low dose aspirin: used in patients with decreased albumin or antithrombin III levels to help prevent thromboembolic events. 0.5 to 5 mg/kg PO given in dogs q24h-BID, in cats q48hr.
- Omega-3 fatty acid supplementation: Early studies show promise for antioxidant therapy.
- Immunosuppressants (azathioprine, cyclophosphamide, mycophenolate, cyclosporine): although glomerulonephritis is an immune-mediated process, there are no controlled clinical trials that demonstrate

the efficacy of immunosuppressive drugs in the treatment of canine glomerulonephritis. Corticosteroids can increase proteinuria. Treatment with immunosuppressants; e.g., cyclosporine, may be beneficial when glomerulonephritis is a component of a primary autoimmune disorder such as systemic lupus erythematosis.

• Specific Therapy for Anuria/Oliguria

- Hospitalization, IV fluids with diuresis and/or dialysis if available. Monitor for evidence of fluid overload.
- Measure urine output. Should be > 1–2 ml/kg/hr.
- Once rehydrated, if still oliguric, give mannitol 0.5–1.0 g/kg as a slow IV bolus over 15–20 min. If effective diuresis results, start CRI of 1.0–2.0 mg/kg/min (60–120 mg/kg/hr). Continue for 24–48 hours. Do not use mannitol if anuric, showing evidence of fluid overload, or hyperkalemic.
- Alternatively, furosemide 2–4 mg/kg IV bolus, repeat prn (+/- dopamine 1–3 µg/kg/min CRI in dogs) can be used. This is the treatment of choice in hyperkalemic or overhydrated animals.

• General Supportive Care

Azotemia:

- Hospitalization and IV fluids for correction of contributing prerenal azotemia due to dehydration. Generally recommended if concurrent issues have led to an acute exacerbation of CRF. Important part of therapy for ARF.
- Subcutaneous fluid therapy at home or on an outpatient basis
- Low protein diet
- H₂ receptor antagonist (e.g., famotidine) to help prevent GI ulceration secondary to azotemia

Hyperphosphatemia:

- Phosphorus-restricted diet (generally a low-protein diet)
- Phosphate binders: must be given with meals [e.g., Amphojel® (aluminum hydroxide) at 30 to 90 mg/kg/day divided with meals; Epakitin® (calcium carbonate/chitosan) at 1 mg/ 5 kg BW twice daily with food. DO NOT use if animal on calcitriol]. Newer generation phosphate binders [Fosrenol™ (lanthanum carbonate) and Renagel® (selvemer hydrochloride)] used in human medicine have not been evaluated for use in canine and feline patients.

Systemic Hypertension:

- Treatment is recommended for patients with systolic blood pressure readings consistently over 160 mm Hg (or > 180 mm Hg in stressed patients; particularly cats), and patients with elevated pressure readings who have evidence of hypertensive retinopathy.
- Cats: First choice is amlodipine with a starting dose of ¼ of a 2.5 mg tablet/cat PO q24 hr. Additional medications may include atenolol and ACE inhibitors.
- Dogs: First choice are ACE inhibitors at 0.25 to 0.5 mg/kg SID to BID, particularly if PLN is present. Amlodipine may also be used at a dose of 0.2–0.4 mg/kg PO q24h. Additional medications may be indicated if hypertension cannot be controlled.

Acidosis:

- Treatment is recommended if TCO₂ is less than 14 mmol/L.
- Bicarbonate dose (mmol) = body weight (kg) x 0.3 x bicarb deficit (desired-measured bicarb)
- Hospitalized patients on IV fluids: calculate bicarbonate dose required to bring the patient up to a TCO₂ of approx 16. Give half of the required amount of sodium bicarbonate slow IV over 20–30 minutes. If in-clinic evaluation of TCO₂ is available, retest and give second half in IV fluids over the course of 2–4 hours if indicated.
- Outpatient therapy: occasionally metabolic acidosis will be severe enough to require oral alkalization therapy in CRF patients. This can be supplemented with potassium citrate at 40 to 60 mg/kg/day divided (which also provides commonly needed potassium) or with a baking soda slurry at 8 to 12 mg/kg q 8 to 12 hours.

Renal Secondary Hyperparathyroidism:

- Vitamin D analogues: calcitriol or 1,25-dihydroxyvitamin D
- Control existing hyperphosphatemia prior to starting vitamin D analogues; must keep phosphorus <6 mg/dl. Use aluminum-based phosphate binders rather than calcium-based binders when given concurrent with vitamin D analogues.
- If creatinine is 2–3 mg/dl, then initiate calcitriol therapy at 2.5 ng/kg/day. Assess serum calcium on day 7 and day 14 and then every 6 months.
- If creatinine is >3 mg/dl, then measure baseline parathyroid (PTH) level. Initiate calcitriol therapy at 3.5 ng/kg/day. In addition to monitoring

calcium levels as described, measure PTH level after 4 to 6 weeks. If the PTH level is still elevated, increase calcitriol dose by 1–2 ng/kg/day, but do not exceed 6.6 ng/kg/day. If higher calcitriol doses are required, pulse therapy may be necessary.

Anemia: Common in CRF patients due to decreased production of erythropoietin. May also be seen secondary to gastric ulceration and anemia of chronic disease.

- H₂ receptor antagonists (for gastric ulceration)
- Human recombinant erythropoietin; consider concurrent iron supplementation.
- **Dialysis:** Expensive, available only at select locations. Can be used as part of management of CRF if finances are unlimited. Very helpful in management of ARF to allow time for recovery of renal function.
- **Kidney Transplantation:** Expensive, only available at select locations, limited by availability of an appropriate donor. Most transplant programs are selective for patients with a high chance of success.

Monitoring

- **Short-term, hospitalized ARF patients:** Monitoring of azotemia, electrolytes, acid-base status, lung sounds/respiratory rate for volume overload, urine output and blood pressure. Azotemia may continue to increase initially depending on cause, then may slowly improve over the course of days to weeks.
- **Short-term, hospitalized CRF patients:** BUN/creatinine may be checked daily to help determine length of inpatient therapy—initially these values should drop as dehydration is corrected, and then values will stabilize. At this point, tapering of IV fluid rate is initiated in preparation for discharge. Monitor PCV as correction of dehydration may reveal preexisting anemia. Recheck BUN, creatinine, phosphorus and electrolytes prior to discharge once eating.
- **Long-term CRF/PLN patients:** After initial diagnosis, monthly monitoring of a renal profile for two to three months to determine progression of disease. If rapid changes are seen, continued frequent monitoring is recommended. If disease appears fairly stable, monitoring may be decreased to q3 months. Renal profile should include a CBC, BUN, creatinine, electrolytes, albumin and TP, phosphorus, calcium and TCO₂. In addition, animals with PLN should have a UPC measured. Blood pressure should be monitored as indicated and urine

cultures should be considered every 3 months in animals with CRF.

- **Long-term ARF patients:** In some cases, complete recovery from renal insult may occur, whereas in others, some residual renal damage remains. For those who remain azotemic, monitoring should be as for long-term CRF patients. For patients with complete recovery, periodic monitoring of BUN and creatinine is still recommended.

Glossary of Terms

- **Amyloidosis:** Deposition of beta-pleated protein sheets in various tissues occurring either as a reactive change in inflammatory conditions or as a primary idiopathic condition. Potential cause of severe protein-losing glomerulonephropathy when amyloid is deposited in the kidneys.
- **Azotemia:** Excess, non-protein, nitrogenous compounds (e.g., creatinine, urea) in the blood. Causes of azotemia are classified as prerenal, renal and postrenal.
- **Acute renal failure:** Rapid and sometimes progressive loss in renal function over a period of hours to days, marked by azotemia and some combination of hyperkalemia, hyperphosphatemia or metabolic acidosis. Severe forms of acute renal failure can manifest as oliguria and anuria, or with lesser forms, nonoliguric failure.
- **Chronic renal failure:** Caused by irreversible, intrinsic, renal parenchymal lesions present for at least two weeks and often for more than three months. Consequences of chronic renal failure are outlined under uremic syndrome.
- **End-stage renal failure:** Complete or near complete loss of kidneys, ability to concentrate urine, excrete waste and regulate electrolytes such that complications are multiple or severe and likely to cause death.
- **Glomerular disease:** Lesions affecting renal glomeruli that can lead to a nonfunctional nephron (glomerulus, Bowman's capsule, tubules) and progressive destruction of glomeruli. Glomerular disease may be associated with proteinuria and can lead to azotemia, decreased glomerular filtration rate and renal failure.
- **Glomerulonephritis:** Acquired glomerular injury due to immune-complex deposition or formation in the glomeruli and associated inflammatory reactions.
- **Glomerulonephropathy:** Disease of the glomeruli, particularly noninflammatory diseases. Can also be used as a general term for glomerular diseases when the specific type of glomerular disease (e.g., glomerulonephritis, amyloidosis, noninflammatory glomerulonephropathy, etc.) is unknown.
- **Nephritis:** Inflammatory process within the kidney, which is usually qualified by terms identifying chronicity and primary site of lesion (e.g., chronic tubulointerstitial nephritis).
- **Nephrosis:** Lesion of the epithelial lining of the renal tubules, which implies toxic damage or a non-inflammatory, non-neoplastic lesion.
- **Nephrotic syndrome:** Collection of clinical abnormalities resulting from protein-losing renal disease or chronic severe proteinuria, and include proteinuria, hypoalbuminemia, edema/ascites, hypercholesterolemia and a hypercoagulable state.
- **Protein-losing nephropathy:** Refers to a group of glomerular diseases that result in excessive protein loss in the urine. Amyloidosis and glomerulonephritis are major causes of PLN.
- **Renal disease:** Lesion affecting glomeruli, tubules, interstitium or vessels that may or may not be associated with dysfunction. In other words, kidney disease and kidney failure are not synonymous terms. Patients with kidney failure will have kidney disease, but the converse is not necessarily true.
- **Renal failure:** Impairment of kidneys' ability to perform excretory, regulatory and endocrine functions, resulting in retention of metabolic waste (e.g., azotemia) and derangements in electrolytes and acid-base balance.
- **Renal insufficiency:** Associated with inappropriate urine concentrating ability, but dysfunction is not severe enough to cause concurrent azotemia. There is a variety of non-renal diseases that can impair urine-concentrating ability without underlying intrinsic renal disease.
- **Uremic syndrome:** Multisystemic, extra-renal, toxic syndrome caused by kidney dysfunction. Components of this syndrome can be seen with acute or chronic renal failure and include anemia, stomatitis and gastroenteritis, metabolic and electrolyte disturbances, pneumonitis, hyperparathyroidism, osteodystrophy, and systemic hypertension.

REFERENCES: Available upon request.

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