All About Kidney Disease: ARF, CRF, and Beyond

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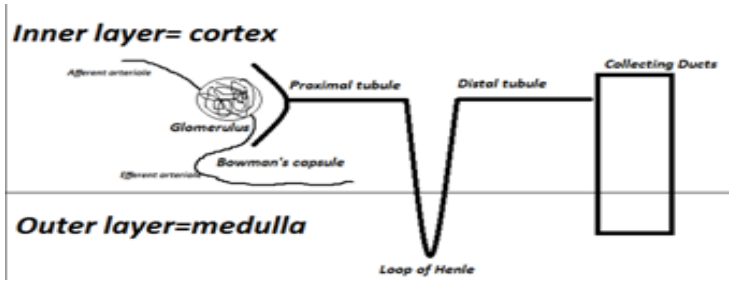
Renal disease is a commonly encountered presentation to the veterinary hospital. Both cats and dogs alike can become afflicted with the various classes of this group such as: acute or chronic, tubular, interstitial, or glomerular. Kidney insufficiency and kidney failure are also two different things. This lecture will serve to clarify and explain kidney anatomy, physiology, pathophysiology, and treatment tenets of kidney disease.

Anatomy and physiology

The kidney is a small bean shaped organ that lives in the retroperitoneal space located in the latter two thirds of the abdomen. Felines and canines each have two kidneys. The canine and feline kidneys are typically easily palpable and the right kidney lies more cranial than the left. The gross anatomy of the kidney includes the capsule, cortex, medulla, and renal pelvis. The capsule is a tough, fibrous, sheath that encloses the kidney and keeps it relatively free from movement. The cortex is the outer layer of tissue, the medulla is the inner part. The renal pelvis is a large collecting area for urine which empties into the ureters. The caudal abdominal aorta branches into the renal arteries which each go to one kidney. The caudal vena cava branches off as well, forming the renal vein. The individual cellular unit of the kidney is the nephron. Below is the structure of a nephron:

Figure 1: Nephron



Blood flows to the nephron from the afferent arteriole. Within the glomerulus, high pressures force water and small electrolytes out into Bowman's capsule. Larger molecules such as proteins are kept within the vasculature. Blood then travels away from the Glomerulus via the efferent arteriole. The "ultrafiltrate" crosses the proximal tubule, where around 70% of Na, Cl, K, and HCO3 are reabsorbed. Most of the urea is also excreted here. This makes it easier for water to move into or out of the loop of Henle. This concentrated pre-urine product then crosses the loop of Henle, where water is reabsorbed or excreted as needed depending on hydration. Lastly, the "almost" urine product finds the distal tubule, where some sodium is reabsorbed and potassium excreted, based on the action of Aldosterone. It then congregates in the collecting ducts and passes into the renal pelvis, and into the ureters.

Renal blood flow is very important. The kidney only has one artery and one vein, so blood supply to each and every nephron relies on the patency, and continuity of flow. Should cardiac output drop suddenly for any reason, the kidney's perfusion could be highly compromised. This is why monitoring blood pressure under anesthesia is very important! The kidneys are able to independently manage their own blood pressure (autoregulate) at a range of blood pressures. There are various numbers, but 60-160 mmHg is a good range. This means that below 60mmHg (mean pressure) the kidneys cannot perfuse themselves adequately. Above 160mmHg as well can cause renal damage.

Types of renal disease

There are two major categories of renal failure, acute and chronic. Renal insufficiency represents >70% loss of nephrons, required for azotemia to develop, but does not necessarily manifest as a clinical syndrome. Renal failure means a syndrome of either acute or chronic nature, causing uremia, acid/base disturbances, and abnormal fluid balance. Acute renal failure is a sudden decline in GFR (glomerular filtration rate) which is potentially reversible. Chronic renal failure is slower process of destruction of healthy renal tissure. CRF is not reversible. Acute on chronic renal failure represents a sudden decline in GFR associated with pre-existing renal disease.

Whether the renal disease is acute or chronic it can be associated with renal parenchyma (tissue), or interstitium, the tubules, the glomerulus, or more than one section of the nephron. Some examples of each disease process are listed below.

Table 1. Various forms of renal disease

Various forms of renal disease		
Disease	**Part of the nephron**	**Acute or chronic**
Glomerulonephritis	Glomerulus	Can be acute, usually chronic
Acute tubular necrosis	Tubules	Acute
Intersitial nephritis	Interstitium	Chronic
Amyloidosis	Glomerulus	Chronic
Toxicity: Ethylene Glycol	Tubules	Acute
Toxicity: Lillies/Grapes/Raisins	Interstitium	Acute
Pyelonephritis	Interstitium +/- tubules	Acute
Aminoglycoside toxicity	Tubules	Acute

Effort is often made to clarify the type of renal failure as actually diagnosing a disease can be challenging. For example, if a patient has azotemia that is renal in origin, they will have intrinsic renal failure as opposed to a renal failure caused by a vascular or extra-renal event.

Presentation

Animals with renal failure are often pretty sick. They may present with a history of anorexia, weight loss, vomiting, or diarrhea. They may have had a known toxin ingestion, or may have been potentially exposed to toxins in the home. They may also be polyuric/polydipsic. Initial interventions include a full history and physical exam.

Diagnostics

A full set of laboratory tests should be run including: Chemistry, CBC, PCV/TS, venous blood gas, and a urinalysis.

Chemistry results may reveal azotemia (hallmark of decreased GFR). Azotemia is characterized by an increase in nitrogenous waste products, namely BUN and Creatinine. Azotemia can be pre-renal, renal, or post-renal. A urine specific gravity will help differentiate between the two. The chart below helps break down the difference in azotemia.

Table 2. Classification of azotemia

Types of Azotemia		
Type of azotemia	**USG results**	**Patient condition**
Pre-renal	Concentrated <1.030	Patient is dehydrated or hypovolemic. This results in a decline in GFR as the body preserves fluids for vascular volume.
Renal	Isosthenuric 1.007-1.012	If patient is dehydrated/hypovolemic: The kidneys are not doing their job and concentrating urine. The urine is the same USG as plasma, meaning it is being excreted without any water retention.
Post-renal	Varies: usually concentrated	Hard to simply diagnose post-renal azotemia from USG but if any evidence of osbtruction present this would result in a post-renal azotemia.

Other lab work abnormalities may reveal:

Hyperphosphatemia, Hyperkalemia (inappropriate retention)- acute, Hypokalemia- chronic, Anemia (lack of erythropoeitin production)- non-regenerative, Glucosuria, proteinuria if glomerular damage, Casts on sediment analysis- if tubular function damaged, Metabolic acidosis from uremic acids

Additional workup options include:

Ultrasound with fine needle aspirate and cytology, Leptospirosis titers (acute), Ethylene Glycol tests (acute), Tick borne disease titers (acute and/or chronic). All patients should also have baseline blood pressures as many of these patients are hypertensive

Treatment- acute

Treatment of the ARF patient is very labor intensive. These patients need fluids and volume so a peripheral IV catheter should be placed. Any intravascular volume deficits should be addressed immediately via fluid blouses. After volume replacement hydration,

maintenance and urine production should be of concern. A central line may be an option as well for CVP monitoring. They should have regular blood pressures, weights, and a urinary catheter placed to monitor output. An increase in weight after hydration, elevated blood pressures, CVP's, or decreased urine output should alert you to potential volume overload. Urine output should be 1-2ml/kg/hr on maintenance fluids. Ins should equal outs. If the patient is polyuric, fluids should be increased to meet demand. If anuric, fluids slowed to prevent iatrogenic fluid overload. They should potentially have broad spectrum antibiotics started in the face of a potential infectious process. If Lepto is suspected, care should be taken to vigilantly avoid urine. Life-threatening electrolyte disturbances (hyperkalemia and hypocalcemia) should be addressed with insulin/glucose, and/or parenteral calcium. If the patient is hypokalemic, postassium should be added to their fluids. Do not exceed 0.5meq/kg/hr. Early nutrition is also important in the renal failure patient. Acid-base abnormalities (typically metabolic acidosis) will often resolve or improve with fluid therapy. Sodium bicarbonate therapy is only reserved for life threatening academia. These patients should receive stress ulcer prophylaxis, including an H2 blocker and/or a proton pump inhibitor (omeprazole or Pantoprazole). Because emesis can be caused by uremia, a centrally acting anti-emetic may be necessary. Metoclopramide, Maropitant, or Ondansetron/Dolasetron are efficient in managing emesis in these patients. Hyperphosphatemia can be nauseating so phosphorus binders (Aluminum hydroxide) should be given if the patient can tolerate enteral medication. If the patient is anemic, and blood transfusion may be necessary, however, the acute renal failure patient is usually not anemic. Renal function should be assessed with fluid therapy, if the patient decompensates into oliguria or anuria, dialysis should be considered. These patients should be monitored for fluid overload as well. This can manifest as chemosis, peripheral edema, tachypnea, hypoxemia, and/or pulmonary edema. Constant auscultation of lung fields may help catch development of wet lungs.

Treatment-chronic

Patients hospitalized for chronic renal failure should receive all of the monitoring an acute renal failure patient does. However, they may also be anemic. Some patients compensate well for anemia, but some may require a transfusion. The decision to transfuse is not always easy and should be based not solely on the PCV values. These patients may also have GI blood loss as uremia affects platelet function and predisposes to gastric ulceration. Chronic renal failure patients are typically hypokalemic and should receive potassium supplementation. The philosophy of treatment with chronic cases is to treat the acute crisis and then focus on long term management. Nutritional support may be necessary in the form of an E-tube, PEG tube or other longer term feeding device. Low protein and phosphorus diets truly slow progression of the disease and enhance survival. Hypertension should be managed typically with calcium channel blockers, and proteinuria managed with ACE inhibitors.

Table 3. Renal pharmacology

Drug	Drug Class	Frequency	Route	Indications
Famotidine	H2 blocker	SID	IV, SQ	Uremia, gastritis
Pantoprazole	Proton pump inhibitor	SID	IV	Uremic gastritis
Aluminum Hydroxide	Phosphorus binder	SID	PO	Hyperphosphatemia
Furosemide	Diuretic	As needed	IV	Anuria/Fluid overload
Mannitol	Diuretic	Once	IV slow	Anuria/Fluid overload
Metoclopramide	Anti-emetic	CRI	IV	Vomiting
Sucralfate	Gastric coating agent	QID	PO	Gastric ulceration
Amlodopine	Anti-hypertensive	SID-BID	PO	Hypertension
Omeprazole	Proton pump inhibitor	SID	PO	Uremic gastritis
Benazapril	ACE Inhibitor	SID-BID	PO	Proteinuria

Prognosis

For chronic renal failure, prognosis depends on the severity of the disease. With mild-moderate disease (Creatinine <3-5 mg/dL) survival is 1-3 years. Proteinuria and hypertension are negative survival indicators. With ARF, mortality is 60%. 2/3 of the patients that survive ARF have some degree of chronic renal failure.

Summary

Kidney disease is complex. This arises from the complex nature of the urogenital anatomy of the kidney, and the variability with presentations. With a greater understanding of kidney disease, veterinary technicians are more prepared to institute intricate and top notch nursing care to these often quite critical patients.

References available upon request