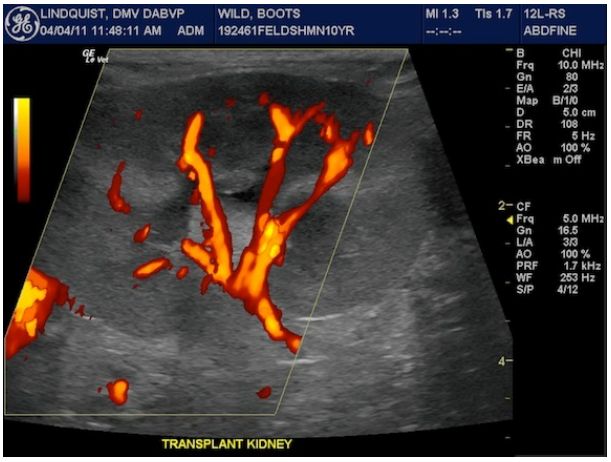
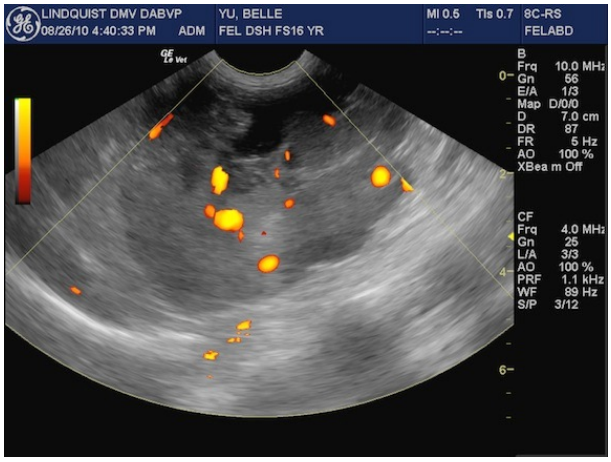
Animal Sounds
Mobile Veterinary Ultrasound

APRIL 2014

ACUTE RENAL FAILURE

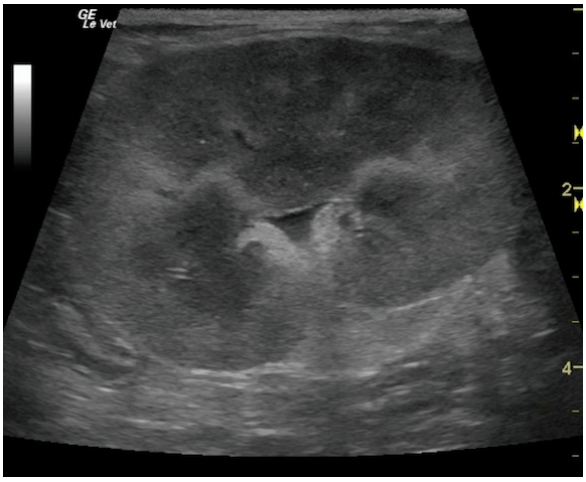




Description: Acute renal failure (ARF) also referred to as acute kidney injury is defined as a rapid deterioration in renal function sufficient to result in the accumulation of metabolic wastes in the body and is characterized by impaired regulation of water and solute balances. ARF may result from pre-renal, post-renal, and/or primary renal causes.

Pre-renal azotaemia reflects a reduction in glomerular filtration rate (GFR) resulting from renal hypo-perfusion and does not result from structural renal damage. Immediate restoration of renal blood flow results in reversal of the azotemia over a period of time. However, if the hypo-perfusion is severe or prolonged, or if there is prior renal dysfunction, acute primary renal failure due to ischemic acute tubular necrosis will result. Post renal azotemia occurs with obstruction to urine flow or rupture of the excretory pathway with subsequent urine reabsorption. Urinary obstruction that persists may cause irreversible renal damage. Early recognition of post renal azotemia will result in complete restoration of renal function. Acute tubular necrosis accounts for the majority of acute primary renal failure cases. This is characterized by the abrupt and sustained reduction in GFR resulting from an ischemic or toxic renal insult. Conditions predisposing to the development of ischemia are the same as that for pre-renal azotemia, however, the duration of the ischemia is important

Nephrotoxins are a frequent cause of tubular necrosis. The high rate of blood flow, high rate of metabolic activity and excretory function of the kidneys predisposes them to the toxic effects of drugs, and endogenous or exogenous toxins.



Clinical Signs: the clinical course in acute tubular necrosis can be divided into an initiating phase, a maintenance phase and a recovery phase.

The initiation phase begins with onset of renal injury and continues through onset of oliguria. The greatest potential for preventing or reversing tubular damage and progression to overt renal failure exists in this phase, because it is during this phase that renal cell damage develops. This phase may only be evident in retrospect because it lacks characteristic signs.

The maintenance phase is characterised by the onset of oliguria (<1ml/kg/hour of urine production). Onset of this phase typically occurs during the first 24 hours but may be delayed for up to 1 week. Duration of this phase is highly variable, but it usually persists for up to 2 weeks. It is characterised by fluid and electrolyte imbalances, including alteration in hydration, hyponatremia, hyperkalemia, high anion gap metabolic acidosis, hypocalcemia, hyperphosphatemia and azotemia. Clinical signs include gastrointestinal, hematologic and neurologic manifestations of renal failure.

The recovery phase commences when the GFR increases so that the urea and creatinine levels no longer continue to increase. There is a progressive increase in the urine volume. Tubular function, although improving, remains impaired and diuresis persists because of impaired ability of the tubules to reabsorb sodium and to respond to vasopressin. Clinical manifestations observed in the maintenance phase persist into the recovery phase. In some patients infections and/or gastro-intestinal bleeding may become evident. Sites of infection include respiratory tract, operative sites and urinary tract. Resultant septicemia can occur. Intravenous and urinary indwelling catheters may play a role in sepsis.

Diagnostics: Extra-urinary disorders producing pre-renal azotemia are associated with concentrated, hypersthenuric urine with a relatively low concentration of sodium and high concentration of creatinine. ARF typically shows enlarged or swollen kidneys, elevated haematocrit, and azotemia. Urine is isosthenuric or minimally concentrated, containing high concentrations of creatinine. There may be a proteinuria or a glycosuria. The sediment will show casts and RTE cells. Complete anuria is usually associated with post renal azotemia. Features that are typical for acute tubular necrosis are anuria in the absence of urinary tract obstruction or rupture, severe proteinuria, significant hematuria with red cell casts and prolonged oliguria. In these cases a diagnostic renal biopsy is indicated.

Treatment: Fluid therapy - most patients with ARF are volume depleted. Fluid therapy is indicated to correct dehydration, which will restore adequate renal perfusion and may prevent further renal damage. If the etiology was pre-renal in origin then urine volume will increase. In the maintenance phase the fluid therapy is directed towards maintaining fluid balance and preventing both overhydration and underhydration. Only maintenance needs and on going losses are provided for. Insensible losses are calculated at 20 mls/kg/24hrs. Aggressive fluid therapy during the recovery phase may perpetuate the polyuria. As urine volume stabilizes, the volume of fluid administered should be corresponding reduced. Because dehydration may occur during this phase, body weight and clinical assessment of hydration status should be carefully monitored as fluid therapy is reduced.

Converting oliguria to non-oliguria - therapy designed to convert oliguria to non-oliguria should be considered only for oliguria patients that are unresponsive to fluid volume replacement. Mannitol, furosemide and dopamine may be used following volume replacement in an attempt to increase GFR and urine volume.

Hyperkalemia - this is commonly associated with the maintenance phase of ARF. Concentrations > 6 mmol/l may require treatment with sodium bicarbonate, dextrose, insulin and/or calcium gluconate.

Hemodialysis - should be considered in patients with severe, persistent uremia, acidosis or hyperkalemia. It may also be used to treat overhydration and to hasten elimination of nephrotoxins.

Conclusion: As ARF can be frequently iatrogenic and associated with nephrotoxic drugs or inadequate fluid therapy prevention is the best therapy.

References:

Acierno MJ, Maeckelbergh V. Continuous renal replacement therapy. Compend Contin Educ Vet. 2008;30:264-272.

Grauer GF. Early detection of renal damage and disease in dogs and cats. Vet Clin North Am Small Anim Pract. 2005; 35:581-596.

Labato MA. Strategies for management of acute renal failure. Vet Clin North Am Small Anim Pract. 2001;31:1265-1287.

Ross L. Acute kidney injury in dogs and cats. Vet Clin North Am Small Anim Pract. 2011; 41:1-14.

"You are receiving this monthly SonoPath.com newsletter on hot sonographic pathology subjects that we see every day owing to your relationship with a trusted clinical sonography service.This service has a working relationship with SonoPath.com and sees value in enhancing diagnostic efficiency in veterinary medicine."

This article is an excerpt form the upcoming pocket guide offered by SonoPath.com: "The Curbside Guide, Diagnosis & Treatment of Common Sonographically Detected Disease." Available Spring 2014.

Ultrasound is our utility, veterinary medicine is our passion

This Communication has Been Fuelled By:

SonoPath LLC. 31 Maple Tree Ln. Sparta, NJ 07871

USA Via Costagrande 46, MontePorzio Catone (Roma) 00040 Italy

Tel: 800 838-4268

For Case Studies & More Hot Topics In Veterinary Medicine For The GP & Clinical Sonographer Alike,

Visit http://www.SonoPath.com