Managing Fluid and Electrolyte Disorders in Kidney Disease

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KEYWORDS

• Acute kidney injury • Chronic kidney disease • Renal failure

KEY POINTS

- A personalized, carefully constructed fluid plan that is frequently assessed and revised as needed is an important component of treating patients hospitalized for kidney disease.
- Accurate assessment of hydration and water balance is critical for successful fluid therapy.
- The goals of fluid therapy in kidney disease are (1) acute resuscitation to restore effective intravascular volume, organ perfusion and tissue oxygenation; (2) maintenance of intravascular volume homeostasis without excessive fluid accumulation; and (3) fluid removal during convalescence to remove hemodynamically unnecessary volume.
- Diuretics to increase urine flow do not improve renal function.

Because of the role of the kidneys in maintaining homeostasis, kidney failure may lead to derangements of fluid, electrolyte, and acid-base balance. Reversing these derangements is the goal of treatment.

Kidney disease is classically compartmentalized into acute and chronic disease. Decompensation of chronic disease presents as an acute crisis, and the principles of therapy are similar, although clinical manifestations of acute kidney injury (AKI) or decompensated chronic kidney disease (CKD) may be quite different. Many patients with AKI require hospitalization for optimal management. Patients with CKD may need hospitalization or their fluid and electrolyte management may occur on an outpatient basis.

When blood flow to the kidney is diminished, as may occur with hypovolemia, hypotension, decreased cardiac output, or increased renal vascular resistance, azotemia may develop. This hemodynamic azotemia is typically rapidly reversible when the underlying cause is corrected. Intrinsic kidney injury occurs when damage to the renal

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Vet Clin Small Anim ■ (2016) ■–■
http://dx.doi.org/10.1016/j.cvsm.2016.09.011 **vetsmall.theclinics.com**

parenchyma occurs. The damage may be reversible or irreversible, and includes damage to the glomeruli, tubules, interstitium, or renal vasculature. Post-renal azotemia occurs when there is an obstruction to urine flow, from the level of the renal pelvis to the urethra, or when urine leaks into surrounding tissue and is reabsorbed (ie, ruptured bladder, ureter, or urethra). Post-renal azotemia can also be rapidly reversed by diverting the urine either by a urinary catheter or peritoneal catheter (in cases of an intra-abdominal rupture). With both hemodynamic and post-renal causes of azotemia, long-standing problems may progress to intrinsic renal failure. Although significant kidney disease can be present without azotemia, fluid therapy is generally not necessary in those situations. In fact, fluid therapy is not necessary in compensated CKD with mild to moderate azotemia.

FLUID TREATMENT

Normal fluid losses consist of insensible and sensible losses. Insensible losses are those that are not consciously perceived, such as water lost via respiration, normal stool, or sweating. Sweating is a negligible volume in dogs and cats. There is variation in respiratory losses in dogs, which may lose considerable amounts of fluid by excessive panting, but 22 mL/kg per day is the average.[1] The main sensible fluid loss in the healthy patient is urine output. Additional sensible losses include the volume lost from vomiting, diarrhea, body cavity drainage, or burns. In healthy animals, these losses are replaced by drinking and the fluid contained in food. In sick animals, not voluntarily consuming food or water, or in which water intake is compromised by vomiting, fluid therapy is necessary to replace these losses. With renal disease, urine volume is frequently abnormally high or low, or inappropriate for the situation, and fluid therapy is tailored for the individual patient to maintain fluid balance.

Fluid Therapy for Hospitalized Patients

Many definitions of AKI exist. In the International Renal Interest Society (IRIS) AKI grading scheme, an increase in serum creatinine concentration of 0.3 mg/dL over $\leq$48 hours defines Grade I, with progressive increases at higher stages. Attempts to lessen further renal damage are more likely to be successful in early stages of injury. Although oliguria or anuria are the classic manifestation of AKI, the patient may present with polyuria, which frequently portends a less severe renal injury.[2,3] Patients with CKD may present in a decompensated uremic crisis, which may represent AKI superimposed on chronic disease.

Many drugs have been evaluated for their benefit in treating AKI, and some are helpful in certain settings. However the most effective therapy of AKI is careful management of fluid balance, which involves thoughtful assessment of hydration, a fluid treatment plan personalized for the specific patient, repeated and frequent reassessment of fluid and electrolyte balance, and appropriate changes in the treatment plan in response to the rapidly changing clinical situation of the kidney disease patient.

Assessing Hydration

The key feature to an appropriate fluid plan is accurate determination of hydration status. Blood volume can be measured using indicator dilution techniques, radioactive tracers, bioimpedance spectroscopy, or other methods. Unfortunately, readily available accurate measurement of blood volume is not feasible in general practice settings.

Despite a lack of precise objective data, there are many ways to estimate hydration. A deficit of the extravascular fluid compartment (interstitial and intracellular) causes dehydration. A severe deficit may decrease the intravascular compartment, leading