Acute Kidney Injury in Dogs and Cats

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KEYWORDS

• Acute kidney injury • Ischemia • Nephrotoxin • Azotemia • Oliguria

KEY POINTS

- Acute kidney injury has 4 clinical phases: initiation, extension, maintenance, and recovery.
- Various pathophysiologic mechanisms contribute to renal cellular damage and death, including degradation of ATP, cytoskeletal alterations, changes in nitric oxide, and inflammation.
- Appropriate fluid administration is key to therapy.
- The prognosis varies according to the cause of renal injury.

INTRODUCTION

Acute kidney injury (AKI) is defined as an abrupt decrease in kidney function. Categories of AKI include prerenal (hemodynamic), renal (intrinsic renal disease), and postrenal (obstructive nephropathy or rupture of the urine collecting system). Only renal AKI is discussed in this article.

The clinical course of AKI can be divided into 4 phases. The first, or initiation phase, occurs during and immediately after the insult to the kidneys when pathologic damage to the kidney occurs. The second is the extension phase, during which ischemia, hypoxia, inflammation, and cellular injury continue. Clinical and laboratory abnormalities may not be evident during the first 2 phases. The third, or maintenance phase, is usually characterized by azotemia, uremia, or both and may last for days to weeks. Oliguria (<0.5 mL urine per kilogram body weight per hour) or anuria (no urine production) may occur during this stage, although urine production is highly variable. The fourth phase is recovery, during which time renal tubular repair occurs and azotemia improves. Marked polyuria may occur during this stage as the result of partial restoration of renal tubular function and of osmotic diuresis of accumulated solutes. Renal function may return to normal, or the animal may be left with residual renal dysfunction. Milder renal pathologic conditions can result in nonazotemic renal failure and are

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