Acutely tubular necrosis or injury (ATN) is a more common cause of acute kidney injury (AKI) among hospitalized patients than many hospitalists may realize. At least one study found that as much as half of AKI in seriously ill patients was caused by ATN (1).

The majority of AKI overall is due to prerenal causes, but prerenal AKI may itself progress to ATN (2).

The distinction between prerenal AKI and ATN has important clinical and coding implications, so precise diagnostic documentation is crucial. The confirmed or presumed underlying cause of AKI should always be clarified if possible.

Like prerenal AKI, ATN can often be promptly corrected if treated aggressively and early with IV fluid resuscitation and correction of any precipitating factors. However, patients with AKI due to ATN, even if mild, have a much greater risk of rapid progression and adverse outcomes than those without ATN. In addition, ATN is considered to have a higher severity of illness classification than prerenal AKI.

The current clinical concept of ATN has changed substantially from historical descriptions (3). According to recent studies and authoritative sources like the National Kidney Foundation and International Society of Nephrology, ATN is a functional abnormality of the renal tubules due to toxic or ischemic injury that, if severe, may sometimes progress to necrosis and sloughing of renal tubular cells. While actual histologic necrosis of renal tubules may occur in the most severe cases, the defining feature of ATN is no longer considered to be necrosis.

The term acute tubular injury (ATI) was proposed to emphasize the functional nature of this disorder but unfortunately did not catch on. Clinicians are therefore left with the misnomer of acute tubular necrosis to describe a condition in which necrosis is generally absent, creating widespread confusion about the true nature of what we call ATN.

In a large majority of cases meeting the current definition of ATN, the classic urine sediment findings of muddy-brown granular casts, epithelial cell casts, and free renal tubular epithelial cells may not be present. In fact, the urine sediment may be entirely normal. Today, the distinction between prerenal AKI and ATN is based on the clinical circumstances leading to AKI and the speed of the creatinine response to IV fluid resuscitation. Most cases of ATN are nonoliguric in nature, and prerenal AKI is typically oliguric.

ATN is associated with certain typical circumstances (listed in Table 1), in contrast to prerenal AKI, where the only identifiable precipitant may be dehydration or volume depletion. Some nephrotoxic medications that can cause ATN are listed in Table 2.

The “gold standard” for recognizing ATN is the timing of the creatinine response to effective IV fluid resuscitation. Prerenal AKI is expected to resolve within 24 to 48 hours, whereas ATN takes at least 72 hours, but often lasts seven days or more.

### Table 1. Common causes of ATN

<table>
<thead>
<tr>
<th>Common causes of ATN</th>
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<tbody>
<tr>
<td>IV radiocontrast material</td>
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<tr>
<td>Sepsis</td>
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<tr>
<td>Significant hypotension (sometimes brief)</td>
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<tr>
<td>Prolonged, difficult surgery</td>
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<tr>
<td>Obstetrical complications</td>
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<tr>
<td>Major trauma</td>
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<tr>
<td>Inadequately treated prerenal AKI</td>
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<tr>
<td>Myoglobinuria/hemoglobinuria</td>
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<tr>
<td>Nephrotoxic medications</td>
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</tbody>
</table>

Source: Author’s compilation from the references cited. AKI=acute kidney injury; ATN=acute tubular necrosis.

### Table 2. Nephrotoxic medications commonly causing ATN

<table>
<thead>
<tr>
<th>Nephrotoxic medications</th>
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<tbody>
<tr>
<td>Aminoglycosides</td>
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<tr>
<td>Cisplatin</td>
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<tr>
<td>Cyclosporin</td>
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<tr>
<td>Acyclovir</td>
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<tr>
<td>Nucleoside reverse transcriptase inhibitors</td>
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<td>Pentamidine</td>
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<tr>
<td>IV immunoglobulin</td>
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<td>Hetastarch</td>
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<tr>
<td>Mannitol (&gt;200-300 g/d)</td>
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Source: Author’s compilation from the references cited. ATN=acute tubular necrosis.