

**TABLE 31-3** URINALYSIS AND MICROSCOPIC EXAMINATION OF THE URINE SEDIMENT

| TEST                       | PRERENAL                | VASCULITIS                 | GN                         | ATN                   | AIN                    | POSTRENAL                     |
|----------------------------|-------------------------|----------------------------|----------------------------|-----------------------|------------------------|-------------------------------|
| Specific gravity           | High                    | Normal/high                | Normal/high                | Isosmotic             | Isosmotic              | Isosmotic                     |
| Dipstick blood             | Negative                | Positive                   | Positive                   | ±                     | ±                      | Negative                      |
| Dipstick protein           | Negative                | Positive                   | Positive                   | Negative              | ±                      | Negative                      |
| Urine sediment examination | Negative, hyaline casts | RBC casts, dysmorphic RBCs | RBC casts, dysmorphic RBCs | Granular casts, RTECs | WBC casts, eosinophils | Negative, sometimes WBCs/RBCs |

AIN, Acute interstitial nephritis; ATN, acute tubular necrosis; GN, glomerulonephritis; RBCs, red blood cells; RTECs, renal tubular epithelial cells; WBCs, white blood cells.

With certain processes, crystals may be indicative of the underlying cause of AKI. For example, calcium oxalate crystals may suggest enteric hyperoxaluria or ethylene glycol intoxication, uric acid crystals may point to acute urate nephropathy, and various other crystals may indicate a drug-induced form of AKI.

### Urinary Indices

Spot urine chemistry testing (sodium, creatinine, and urea), along with plasma samples (sodium, creatinine, and BUN), has been used to evaluate renal tubular function in the setting of AKI, primarily to distinguish prerenal AKI from ATN. These measures allow the clinician to calculate fractional excretion of sodium ( $FE_{Na}$ ) and fractional excretion of urea ( $FE_{Urea}$ ); they are thought to be more accurate indicators than urine sodium concentration, which is less than 10 to 20 mEq/L with prerenal AKI and greater than 20 mEq/L with ATN.

The ratio of the clearance of sodium (Na) to that of creatinine (Cr) is calculated as a percentage:

$$FE_{Na} = (U_{Na}/P_{Na}) \times (P_{Cr}/U_{Cr}) \times 100$$

where U and P are the concentrations in urine and plasma, respectively. Likewise, the ratio of urea clearance to creatinine clearance is

$$FE_{Urea} = (U_{Urea}/P_{Urea}) \times (P_{Cr}/U_{Cr}) \times 100$$

The rationale for the use of these indices is that the ratio of urine to plasma creatinine concentrations ( $U_{Cr}/P_{Cr}$ ) provides an index of the fraction of filtered water excreted. Assuming that all of the creatinine filtered at the glomerulus is excreted into the urine, any increment in the concentration of creatinine in urine over that in plasma must result from the removal of water.

In prerenal AKI, because the increased stimulus for salt and water retention,  $U_{Cr}/P_{Cr}$  typically is considerably greater than it is in ATN; moreover,  $FE_{Na}$  is <1%, and urine sodium concentrations are characteristically low. In contrast, in AKI due to ATN, the nephrons excrete a large fraction of their filtered sodium and water, resulting in a lower  $U_{Cr}/P_{Cr}$ , higher urine sodium concentrations, and a higher  $FE_{Na}$  (E-Table 31-3). An important clinical exception to this finding is that  $FE_{Na}$  can be high (>1 to 2%) with prerenal AKI in the setting of diuretic therapy. To counter this effect, calculation of  $FE_{Urea}$  has been used: An  $FE_{Urea}$  greater than 35% favors a diagnosis of prerenal AKI, and an  $FE_{Urea}$  greater than 50% favors ATN.

Interpretations of these tests, therefore, must be made in conjunction with other assessments of the patient, because clinically important exceptions to these generalizations exist. As an example, prerenal AKI can manifest with an elevated  $FE_{Na}$  or

$FE_{Urea}$  in the setting of glycosuria, metabolic alkalosis, bicarbonaturia, salt-wasting disorders, or CKD. Similarly, ATN with low  $FE_{Na}$  and  $FE_{Urea}$  occurs with pigmenturia, sepsis, radiocontrast injury, severe heart or liver failure, and nonoliguric ATN.

### Renal Imaging

If either prerenal AKI or ATN is the likely cause of AKI, and if the clinical setting does not require the exclusion of another cause, then no further diagnostic evaluation is required. Further assessment may be necessary if the diagnosis is uncertain, especially if the clinical setting suggests other possibilities (e.g., obstruction, vascular accident); if clinical findings make the diagnosis of prerenal AKI or ATN unlikely; or if oliguria persists without a good reason. When indicated, diagnostic renal imaging is important in the evaluation of AKI. Retroperitoneal ultrasonography of the kidneys, ureters, and bladder is the first test used because it is readily available, noninvasive, free of radiation exposure, and fairly accurate.

Ultrasonography provides information about kidney size (large, normal, or small) and the parenchyma (normal or increased echogenicity), the status of the pelvis and urinary collecting system (normal or hydronephrotic), and the presence of structural abnormalities (e.g., stones, masses, enlarged lymph nodes). In the setting of AKI, this test can rapidly confirm or exclude the presence of hydronephrosis (E-Fig. 31-2) and a diagnosis of obstructive uropathy. Interrogation of the renal arteries by Doppler ultrasonography provides important information about renal blood flow and renal artery stenosis; however, this test is highly operator dependent.

Computed tomography (CT) of the retroperitoneum provides important information about the cause of postrenal AKI (e.g., tumor, stones, retroperitoneal fibrosis) when ultrasound findings are negative or inconclusive. CT angiography can also accurately diagnose renal artery disease and renal infarction, but there is a risk of nephrotoxicity in those patients with underlying acute or chronic kidney disease. Magnetic resonance (MR) imaging does not add much to CT scanning except in the diagnosis of retroperitoneal fibrosis. Gadolinium MR angiography can safely provide important information about renal artery stenosis or thrombosis, but it should be avoided in patients with AKI or stage 4 or greater CKD. Nephrogenic systemic fibrosis can develop in these patients, especially with nonionic or linear gadolinium contrast agents and in the setting of inflammation.

Radionuclide tests are used to assess the presence or absence of renal blood flow, differences in flow to the two kidneys, and excretory (secretory) function. However, these studies have limited utility in AKI and have reduced accuracy in quantitating absolute rates of flow.

