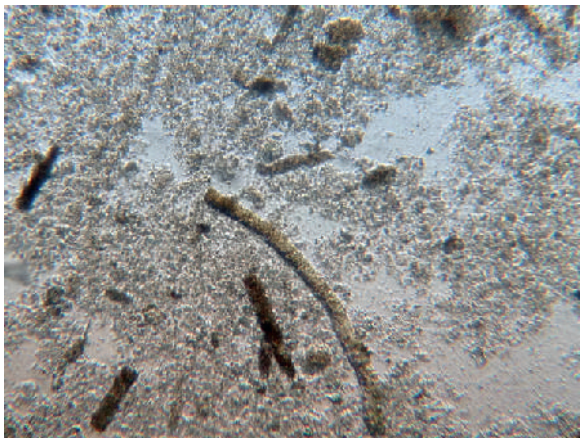
E-TABLE 31-2 RECORD REVIEW IN A HOSPITALIZED PATIENT WHO DEVELOPS ACUTE KIDNEY INJURY

FINDING	COMMENTS
Prior kidney function	Determination of whether the azotemia is acute; patients with prior kidney disease are particularly susceptible to AKI due to use of contrast dyes
Presence of infection	Sepsis is a possible cause of AKI, even in the absence of hypotension
Nephrotoxic agents	Aminoglycosides (e.g., gentamicin) are an important cause of ATN in hospitalized patients, typically nonoliguric ATN during first 2 wk of therapy; antibiotics are a possible cause of AIN; cytotoxic drugs (e.g., cisplatin) are a possible cause of AKI
Contrast studies including oral cholecystography, IVP, angiography	Important cause of ATN in hospitalized patients; typically causes oliguric ATN within 24-48 hr after study
Episodes of hypotension	Suggestion of prerenal AKI or ischemic ATN
History of blood transfusions	Incompatible blood transfusion is an unusual cause of ATN
History of loss or sequestration of extracellular fluid volume, intake and output, serial weights	Important clues to the possibility of prerenal AKI
Type of surgery	Patients with prior cardiac or vascular surgery or with obstructive jaundice are particularly susceptible to ATN
Type of anesthesia	Methoxyflurane and the related, less toxic enflurane are causes of nonoliguric ATN
Amount of blood loss during surgery and whether associated with hypotension	Suggestion of prerenal AKI or ischemic ATN

AIN, Acute interstitial nephritis; AKI, acute kidney injury; ATN, acute tubular necrosis; IVP, intravenous pyelography.



E-FIGURE 31-1 The presence of muddy brown casts in the urine sediment of a patient with acute kidney injury often points to acute tubular necrosis or apoptosis from ischemic or nephrotoxic tubular injury.