Pharmacologic Causes of Acute Kidney Injury

Cause	Examples
Prerenal	
Volume depletion	SGLT2 inhibitors, diuretics
Intrarenal/afferent arteriolar vasoconstriction	NSAIDs (including COX-2 inhibitors); amphotericin B; calcineurin inhibitors; iodinated radiocontrast agents
Efferent arteriolar vasodilation	Renin inhibitors; ACE inhibitors; ARBs
Intrinsic	
Acute tubular necrosis	Aminoglycosides; vancomycin, particularly in combination with piperacillin-tazobactam; polymyxins; lithium; amphotericin B; pentamidine; cisplatin; foscarnet; tenofovir; cidofovir; carboplatin; ifosfamide; zoledronate; contrast agents; sucrose; immune globulins; mannitol; hydroxyethyl starch; dextran; synthetic cannabinoids; amphetamines
Acute interstitial nephritis	Etiologies of acute interstitial nephritis are similar to those for chronic tubulointerstitial nephritis. Acute interstitial nephritis may lead to chronic tubulointerstitial nephritis with protracted exposure (see Table 19)
Acute glomerulonephritis	ANCA-associated drugs, such as minocycline and levamisole (veterinary antihelmintic used in some cocaine preparations)
Acute vascular syndromes	Drug-induced TMA: quinine; cancer therapies (gemcitabine, mitomycin, bortezomib, sunitinib); calcineurin inhibitors (cyclosporine, tacrolimus); drugs of abuse (cocaine, ecstasy, intravenous extended-release oxymorphone); clopidogrel; anti-angiogenesis drugs; interferon; mTOR inhibitors
Intratubular obstruction	Crystals: sulfonamides; triamterene; ciprofloxacin; ethylene glycol; acyclovir; indinavir; atazanavir; methotrexate; orlistat; large doses of vitamin C; sodium phosphate purgatives

AKI = acute kidney injury; ARB = angiotensin receptor blocker; COX = cyclooxygenase; mTOR = mammalian target of rapamycin; SGLT2 = sodium-glucose cotransporter 2; TMA = thrombotic microangiopathy.