Drugs Used in Genital Infections Table

Drug Class & Drugs	Mechanism of Action	Pharmacokinetics (PK) & Resistance	Clinical Uses	Adverse Effects (AEs) & Contraindications
Nitroimidazoles -Metronidazole -Tinidazole	-The nitro group is chemically reduced in **anaerobic bacteria and sensitive protozoans** -Reactive reduction products are responsible for antimicrobial activity	**PK:** -Can be given PO, PR, Topical & IV -Orally absorbed readily and permeates all tissues by simple diffusion -Peak plasma concentrations reached in 1-3 hours -Excreted mainly in the urine -Plasma clearance is decreased in patients with **impaired liver function** .	-**Bacterial vaginosis** -**Trichomoniasis** -**Invasive amebiasis and Giardiasis** -Anaerobic bacterial infections (intraabdominal infections, brain abscess)	-**Metallic, bitter taste**, nausea & dry mouth. -GIT irritation (vomiting, diarrhea) -Irritation of mucous membranes (dysuria, dark urine) -**Alcohol intolerance (disulfiram-like reaction)** -IV infusion may be associated with seizures and peripheral neuropathy -Better avoided during pregnancy and lactation -**Potentiates the anticoagulant effect of warfarin**
Lincosamides -Clindamycin	-**Inhibits microbial protein synthesis** by interfering with the formation of initiation complexes and aminoacyl translocation reactions -Binds to the **50S ribosomal subunit**	**PK:** -Widely distributed into body fluids/tissues, bone, placenta, and breast milk, **except brain and CSF** -Accumulates in severe	-**Infections of the female genital tract** (bacterial vaginosis, septic abortion, pelvic abscesses) -Anaerobic infections -Osteomyelitis and Lung	-GIT irritation (nausea, vomiting, diarrhea) -**Superinfection: diarrhea & pseudomembranous colitis due to Clostridium difficile** -Thrombophlebitis

		hepatic dysfunction -Metabolized in liver, excreted in bile and urine **Resistance:** -Mutation in the ribosomal receptor site or enzymatic inactivation • Resistance generally **confers resistance to macrolides**	abscess -Infections resulting from fecal spillage	-Thrombocytopenia and neutropenia
Antiherpes Agents -Acyclovir	-Requires **3 phosphorylation steps** for activation (monophosphate by **viral thymidine kinase**, di- and triphosphate by host cell enzymes) -Acyclovir triphosphate **inhibits viral DNA synthesis** via competition with deoxy-GTP and **chain termination**	**PK:** -Low bioavailability (15-20%), unaffected by food -Cleared primarily by glomerular filtration and tubular secretion -Half-life is ~3 hours in normal renal function, 20	-**Genital herpes** (mainly **HSV-2**) -Herpes labialis -Herpes zoster -Herpes encephalitis -Neonatal herpes	-Nausea, diarrhea, headache (occasional) -IV administration may be associated with **reversible crystalline nephropathy**, interstitial nephritis, and **neurologic toxicity** (tremors, delirium, seizures)

		hours in anuria **Resistance:** -Due to alteration of either **viral thymidine kinase** or **DNA polymerase**		