Gonorrhea:

Doxycycline { **7** / **10** / **14**

1-Uncomplicated Gonococcal Infections:

Ceftriaxone or Cefotaxime + Azithromycin (single dose)

In case of azithromycin allergy:* *Doxycycline* *(twice a day for 7 days)***

2-Prophylactic measure to prevent opthalmia neonatorum:

0.5% Erythromycin ointment or 1% solution of Silver Nitrate or 1% Tetracycline ointment

3- Gonoccal Conjunctivitis:

Ceftriaxone + Azithromycin with **Saline irrigation** + topical antibiotic solution (single dose)

4- Gonoccal Epididymitis:

Ceftriaxone (single dose) + Doxycycline **(twice daily for 10 days)**

5- PID (Pelvic inflammatory disease):

Ceftriaxone (single dose) + Doxycycline **(twice daily for 14 days)** with or without Metronidazole **(twice daily for 14 days)**

6-DGI (Dissemmniated gonorrheal infection):

Ceftriaxone **(every 24h)** + Azithromycin (single dose)

**** All sex partners with sexual contact with patients within 60 days should be tested for gonorrhea.**

**** Sex partners with sexual contact within two weeks should be treated presumptively for gonorrhea.**

Chlamydial Urethritis:

Azithromycin (single dose) or Doxycycline **(twice a day for 7 days)**

***Alternative Regimens:** *4 2 1 - 7 days*

Erythromycin **(four times a day for 7 days)**
Ofloxacin **(twice a day for 7 days)**
Levofloxacin **(once daily for 7 days)**

Syphilis: ⟶ *Benzathine Penicillin therapy*

1- Primary, Secondary or Early latent Syphilis:

Benzathine penicillin (single dose)

2- Late latent Syphilis or latent with unknown duration:

Benzathine penicillin **(weekly for 3 weeks)**

3- Congenital Syphilis:

Benzathine penicillin (single dose)

4- for patients allergic to penicillin:

A- Tetracycline **(for 14 or 28 days)**
B- Erythromycin **(for 14 or 30 days)**
C- Azythromycin **(for 14 days)**
D- Ceftriaxone **(for 10 days)**
*in case of **neurosyphilis**, the dose is given **daily for 10-14 days**

Chancroid (Soft sore):

Erythromycin **(for 7 days) as a Main Treatment**
Ceftriaxone or Azithromycin as **Alternatives** as (single dose)

Lymphogranuloma venereum:

Tetracycline **(four times daily for 14 days)**

* Erythromycin or Doxycycline or Azythromycin can be used for treatment
* Most cases require repeated courses

Trichomoniasis: (Parasitic STIs)

Oral metronidazole	Oral Tinidazole
- Cure rates are >95%. **Treatment should include infected persons & their partners due to:** High rates of infection in asymptomatic partners and high rates of re-infection	**Single-dose taken with food.** **Cure rates** range from **86-100%.** **For resistant infections → twice daily for 14 days.** **When metronidazole fails tinidazole may be used**

HIV:

HAART → 2 NRTIs & 1 of the following (NNRTIs, Protease inhibitors, or Integrase inhibitors))

NRTI	NNRTI
Phosphorylated by host kinases Competitive inhibition of reverse transcriptase and chain termination of DNA *main component of HAART **Zidovudine** is used for general prophylaxis and for prevention of vertical transmission in pregnancy	No need for phosphorylation Not competitive (Bind to site another than NRTI) Bind to and inhibit reverse transcriptase inhibiting DNA synthesis. e.g: **Efavirenz**

	protease inhibitors (PIs)	Integrase inhibitors	Fusion inhibitors
Examples:	Atazanavir Lopinavir Ritonavir	Raltegravir	Maraviroc
Mechanism of action	inhibit HIV-1 protease (which cleaves the polypeptide products of the viral mRNA into functional parts → assembly & maturation of new viruses)	Inhibit integration of viral genome in host cell DNA.	Inhibit binding and entry of the virus into immune cells.

Genital herpes:

	Antiviral drugs	Foscarnet
Examples:	Acyclovir Famciclovir Valacyclovir	
Mechanism of action:	**Activation**: Guanosine analogs. •Mono-phosphorylated by HSV thymidine kinase (TK) **(not phosphorylated in uninfected cells → few adverse effects).** **They are further activated by host-cell kinases to the Triphosphates are (triphosphates substrates for viral DNA polymerase → incorporated into the DNA molecule → chain terminations)** **MOA: The enzyme thymidine kinase combines phosphate with nucleoside to form nucleotides which incorporate with DNA, Acyclovir will be converted by thymidine kinase to false nucleotide which block DNA synthesis by DNA polymerase, Acyclovir has no effect on cells that are not effected by virus.**	**Mechanism of action:** Inhibition of Viral DNA polymerase and RNA polymerase and HIV reverse transcriptase Doesn't require activation by viral or human kinases **clinical uses:** Acyclovir-resistant HSV infection
Note:	**Valaciclovir** is the pro-drug of acyclovir. **Valaciclovir** is converted into acyclovir by intestinal & liver enzymes resulting in improved bioavailability of acyclovir.	**Toxicity:** • Nephrotoxicity •Electrolyte disturbances that may cause seizures (hypocalcemia & hypomagnesemia

Hepatitis B:

interferon 2b&2a, Lamivudine, Adefovir, Telbivudine, Entecavir

Interferon 2b&2a	**Telbivudine**	**Adefovir**
Glycoproteins normally synthesized by virally infected cells. They have wide range of antiviral and antitumor effects **MOA:** Unclear but possibly act through: 1- Inhibition of viral penetration, translation, transcription, protein processing, maturation, and release 2- Enhanced phagocytic activity. 3- Increase proliferation and survival of cytotoxic T cells	Thymidine analog **MOA:** phosphorylated intracellularly to the triphosphate, compete with endogenous thymidine triphosphate for incorporation into viral DNA, where it serves to terminate further elongation of the DNA chain	Adefovir dipivoxil is a nucleotide analog **MOA:** phosphorylated to adefovir diphosphate, which is then incorporated into viral DNA. This leads to termination of further DNA synthesis and prevents viral replication.

Lamivudine	Entecavir
Cytosine analog Lamivudine must be phosphorylated by host cellular enzymes to the triphosphate (active) form **MOA:** an inhibitor (HBV) DNA polymerase	Guanosine analog ** Entecavir is effective against lamivudine-resistant strains of HBV **MOA:** Following intracellular phosphorylation to the triphosphate, it competes with the natural substrate, deoxyguanosine triphosphate, for viral reverse transcriptase.

Hepatitis C:

a combination of antivirals that can be used according to liver condition and type of hepatitis C virus, example:

Ribavirin : Competitive inhibition of IMP (inositol monophosphate) dehydrogenase → inhibition of guanine nucleotides synthesis. Inhibition of viral RNA polymerase

HCV protease inhibitor → ↓↓ viral replication. **Toxicity:** photosensitivity & rash.	***NS3/4A Protease inhibitors*** **e.g. simeprevir**
Inhibition of HCV RNA-dependent polymerase. **Toxicity:** sofosbuvir (Sovaldi) → headache & fatigue.	***NS5B polymerase inhibitors*** a. Nucleoside (sofosbuvir) b. Non-nucleoside (dasabuvir)
Inhibition of HCV NS5A replication complex (replicase) → ↓↓ viral replication.	***NS5A (replicase)inhibitors*** e.g. daclatasvir & ledipasvir