Additional information:

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Chapter 43

- 1. General structure of beta lactam antibiotics (why are they called beta lactam)
- 2. Classification of Penicillins
 - a. Penicillinase susceptibility
 - b. Spectrum of action
- 3. Metabolism and excretion of penicillins
 - a. Which penicillins are excreted in the urine
 - b. Which penicillins are excreted via bile
 - c. Effects of Probenecid on excretion
- 4. How are Procaine and Benzathine forms of penicillin G different from penicillin G
- 5. Condition in which most penicillins cross blood-brain barrier
- 6. Mechanism of action of Penicillins and resistance
- 7. Substances that are used in conjunction with penicillins to prevent their inactivation
- 8. Clinical uses, spectrum of action and resistance profile
 - a. Penicillin G
 - b. Penicillin V
 - c. Methicillin
 - d. Nafcillin
 - e. Oxacillin
 - f. Ampicillin and Amoxacillin
 - g. Synergistic use of ampicillin with aminoglycosides
 - h. Piperacillin and Ticarcillin
- 9. Allergy and disturbances associated with penicillins

!!!! Please see the following video: (better to know)

https://www.youtube.com/watch?v=rrFAh5-E00g

If you are interested in drug induced hemolytic anemia generally, check the following video: <u>https://www.youtube.com/watch?v=Pclx-</u>

K1k83Q&t=151s

<u> FA2020 – page 187-188 – Must know</u>

- 10. Classification of Cephalosporins
- 11. Pharmacokinetics of cephalosporins
 - a. Main way of excretion
 - b. Which cephalosporins are excreted mainly in the bile
 - c. Entrance into the CSF
- 12. Mechanism of action and Resistance (Cephalosporins)
- 13.Clinical uses of Cephalosporins (according to generation) [Ceftaroline is 5th generation)
- 14. Toxicity and adverse effects of cephalosporins

<u>FA2020 – page 189 – Must know</u>

15. Mechanism of action, Clinical uses and adverse effects of:

- a. Aztreonam (monobactam)
- b. Carbapenems (Imipenem, Doripenem, Meropenem, Ertapenem)
- c. Vancomycin
- 16.Beta-Lactamase inhibitors
 - a. Clavulanate
 - b. Sulbactam
 - c. Tazobactam

17.0ther cell wall or membrane active agents – mechanism of action, clinical uses and adverse effects (if given)

- a. Vancomycin
- b. Fosfomycin
- c. Bacitracin
- d. Cycloserine
- e. Daptomycin

Chapter 44

- 1. Definition of postantibiotic effect
- 2. Mechanism of action (MOA) of:
 - a. Chloramphenicole
 - b. Macrolides
 - c. Clindamycin
 - d. Telithromycin (not very high yield)
 - e. Tetracyclines
 - f. Streptogramins
 - g. Linezolid
- 3. How can selective toxicity of protein synthesis inhibitors be explained
- 4. Tetracyclines
 - a. Are they broad spectrum or narrow spectrum
 - b. MOA
 - c. Divalent cation effects on Tetracycline absorption
 - d. Their metabolism (briefly)
 - e. Placental penetration
 - f. Tetracycline whic can be used in patients with renal failure (is excreted fecally)
 - g. Antibacterial activity
 - h. Mechanism of resistance
 - i. Clinical and selective uses
 - i. Doxycycline
 - ii. Tetracycline
 - iii. Minocycline
 - iv. Demeclocycline
 - j. Tigecycline
 - i. How is it different from tetracyclines
 - ii. clinical uses and spectrum of action
 - iii. side effects

- k. Toxicity
 - i. GI
 - ii. Bones and teeth
 - iii. Effects on kidney and liver
 - iv. Photosensitivity
 - v. Vestibular toxicity
- 5. Macrolides: Azithromycin, Erythromycin, Clarithromycin, Fidaxomicin
 - a. MOA
 - b. Differences of Azithromycin to other macrolides
 - c. Antibacterial activity
 - d. Mechanisms of resistance
 - e. Cross resistance
 - f. Clinical uses and considerations
 - g. Toxicity/Side effects
- 6. Telithromycin
 - a. MOA
 - b. Main clinical uses
 - c. Side effects (briefly)
- 7. Clindamycin
 - a. MOA
 - b. Mechanism of resistance, Cross resistance
 - c. Clinical use
 - d. Toxicity
- 8. Streptogramins
 - a. MOA
 - b. Clinical uses
 - c. Adverse effects
- 9. Chloramphenicole
 - a. MOA
 - b. Metabolism
 - c. Mechanism of resistance
 - d. Placental/BBB penetration
 - e. Antimicrobial activity and clinical uses
 - f. Toxicity/ Adverse effects
 - i. GI
 - ii. Bone marrow (dose dependent, reversible)
 - iii. Adverse effects in neonates/infants, causes of these adverse effects
 - iv. Drug interactions
- 10. Oxazolidinone: Linezolid, (Tedizolid not very high yield)
 - a. MOA
 - b. Mechanism of resistance
 - c. Clinical uses
 - d. Adverse effects

<u>Chapter 45</u>

1. Difference between concentration-dependent killing, Time-dependentkilling and Postantibiotic effect (brief examples)

- 2. Different strength of Amynoglycoside effects with different means of administration (one large dose vs multiple small doses) Not very high yield
- **3.** Aminoglycosides Gentamycin, Neomycin, Kanamycin, Amikacin, Tobramycin, Streptomycin
 - **a.** MOA
 - **b.** Methods to enhance entry of Aminoglycosides in cells
 - **c.** Excretion
 - **d.** Mechanisms of resistance
 - e. Clinical uses and considerations
 - f. Uses of Spectinomycin (not very high yield)
 - g. Toxicity/ Adverse effects
 - i. Ototoxicity
 - ii. Nephrotoxicity
 - iii. Effects on neuromuscular junction
 - iv. Contraindication in pregnancy, teratogenicity
 - v. Skin reactions

 \Rightarrow For both chapters, please skim "summary table" as well.

<u>Chapter 46</u>

- 11. Antimetabolites: Sulfonamides and Trimethoprim
 - a. Mechanisms of action
 - b. Clinical use
 - c. Adverse reactions
 - d. Resistance
- 12. Meaning of sequential blockade
- **13.**Fluoroquinolones
 - a. Drug names, the list of respiratory fluoroquinolones
 - b. Interaction with probenecid
 - c. Mechanism of action
 - d. Resistance
 - e. Clinical use
 - f. Toxicity

Chapter 50

- 1. Metronidazole and Tinidazole (mechanism of action, clinical use, toxicity, drug interactions)
- 2. Fidaxomicin (mechanism of action, clinical use, the reason of low toxicity)
- 3. Rifaximin (mechanism of action, clinical use, reason of low toxicity)
- 4. Mupirocin (mechanism of action, clinical use, toxicity)
- 5. Polymixins (mechanism of action, clinical use, toxicity)
- 6. Definition and list of urinary antiseptics
- _Nitrofurantoin (mechanism of action, clinical use, side effects)
- _ Nalidixic acid (mechanism of action, clinical use, side effects)
 - 7. Definitions of antiseptic, disinfectant and sterilization
 - 8. Disinfectants and antiseptics
- _Autoclave, alcohols (isopropranol), chlorhexidine, chlorine, ethylene oxide,
- hydrogen peroxide, iodine and iodophors, quaternary amines.
 - 9. Ectoparasiticides (Lindane, permethrin, malathion)
 - 10. UV radiation (briefly)

Chapter 51

- 1. Understanding of empiric antimicrobial therapy
- 2. General principles of antimicrobial therapy
- 3. General factors influencing antimicrobial drug use
- 4. Advantage of bactericidal drugs over bacteriostatic ones
- 5. Concentration dependent vs. time-dependent killing
- 6. Understanding minimum inhibitory concentration and postantibiotic effect
- 7. The drugs that should be avoided in patients with renal failure
- 8. Drugs that should be avoided in pregnancy and neonates
- 9. Drug interactions
- 10. The reasons for using antimicrobial drug combinations
- 11. The conditions that require antimicrobial chemoprophylaxis

<u>Chapter 16</u>

- 1. Precursor Amino acid of Histamine, Its major metabolite found in urine , enzyme that metabolizes Histamine
- 2. Definition of Scombroid Poisoning (for additional info look at page 247 of First Aid)
- 3. Triple response to Histamine aka "wheal and flare" reaction
- 4. H1 & H2 receptors (High Yield)
 - a. Coupled G proteins and second messengers
 - b. Their location and Typical responses
- 5. H3 & H4 receptors, coupled G protein, briefly about their location and effects
- 6. Prototype drugs and differences between first and second generation Histamine H1 antagonists
 - a. Diphenhydramine
 - b. Chlorpheniramine
 - c. Cyclizine
 - d. Cetirizine
 - e. Fexofenadine
 - f. Loratadine
 - g. Promethazine
- 7. Mechanisms of action and effects of H1 antagonists, clinical uses, toxicity and interactions
- 8. Doxylamine+Pyridoxine (not that high yield)
- 9. H2 antagonists : Cimetidine, Ranitidine, Famotidine, Nizatidine
 - a. Mechanism and effects
 - b. Clinical use
 - c. Toxicity
- 10. Production, storage, metabolism and physiologic effects (briefly) of Serotonin, Its major metabolite found in urine
- 11. Location of Serotonin receptors
- 12. 5-HT 1D/1B agonists and their clinical application
- 13. 5-HT 2C agonists and their clinical application, reason behind being banned
 - a. know what fen-phen and dex-phen are
- 14. 5-HT4 partial agonist, reason for being restricted
- 15. Hyperthermic syndromes Table 16-2
- 16. Serotonin antagonists
 - a. Mechanisms and clinical effects
 - b. Clinical uses

- c. Toxicity
- 17. Treatment of obesity
- 18. Ergot alkaloids : Classification and effects on following tissues:
 - a. Vessels
 - b. Uterus
 - c. Brain
- 19. clinical uses of ergot alkaloids & toxicity:
 - a. Ergotamine, Dihydroergotamine, Methysergide
 - b. Ergonovine, Ergotamine
 - c. Bromocriptine, Pergolide, Cabergoline

Pay attention to drug summary table at the end of the chapter (drug names)

Chapter 54

- Differentiate between characteristics of cycle specific and cycle nonspecific chemotherapeutic drugs
- Recall figure 54-1
- Describe broadly the log-kill hypothesis and distinctive features of solid tumors in humans
- List the mechanisms involved in anticancer resistance with their corresponding drugs (if given)
- Describe and differentiate between Primary induction chemotherapy, Neoadjuvant therapy and Adjuvant therapy
- Describe principles of combination and rescue therapies
 - Know the use and mechanism of Leucovorin, Mesna and Dexrazoxane
- Name alkylating agents, their mechanism of action and adverse effects/distinctive features associated with them/most common clinical application
- Name Antimetabolites, their mechanism of action and adverse effects/distinctive features associated with them/most common clinical application
- Name drugs in "Natural product anticancer drugs" section, their mechanism of action and adverse effects/distinctive features associated with them/most common clinical application
- Name Antitumor antibiotics, their mechanism of action and adverse effects/distinctive features associated with them/most common clinical applications
- Name drugs in "Miscellaneous anticancer agents" section, their mechanism of action and adverse effects/distinctive features associated with them/most common clinical applications
 - NOT high yield: Panitumumab, Ziv-aflibercept, Sorafenib, Sunitinib, Pazopinib
- Name Hormonal anticancer agents, their mechanism of action and adverse effects/distinctive features associated with them/most common clinical applications

Chapter 55

- Describe mechanism of action of Glucocorticoids, pharmacokinetics, their clinical use and toxicity
- Differentiate between mechanisms of action of Immunophilin ligands (Calcineurin,mTOR inhibitors), recall their clinical use, pharmacokinetics and toxicity
- Describe mechanism of action of Mycofenolat mofetil, its clinical use and toxicity
 - Know reason behind highest susceptibility of lymphocytes to Mycofenolate
- Describe mechanism of action of Thalidomide (+Lenalidomide), its possible clinical use and toxicity
- Recall broadly mechanism of action and clinical application of drugs listed in Table 55-2 (Fingolimod is low yield)
- Describe broadly mechanism of action of Antilymphocyte Globulin and Antithymocyte Globulin, their clinical use and toxicity
 - Understand why toxicity might manifest as anaphylaxis and serum sickness
- Describe mechanism of action of IGIV and D immune globulin, their clinical use and toxicity.
 - Know timeframe of administering D immune globulin
 - Understand why maternal Abs to Rh positive cells are not produced for subsequent pregnancy.
- Recall monoclonal antibodies, their targets, mechanism of action, clinical application and toxicities (if listed)
 - From section "Target other" high yield drugs are: Natalizumab, Omalizumab, Ustekinumab, Canakinumab
 - From table 55-3 high yield drugs are : Abatacept, Abciximab,
 Ipilimumab, Omalizumab, Palivizumab, Rituximab, Trastuzumab
- Name drugs in "Immunomodulation therapy", their clinical use and mechanism of action
- Recall mechanisms of drug allergy and name example drugs (It will be better if students become familiar with them)

Chapter 47

- 1. Mechanisms of action of the azole, polyene, and echinocandin antifungal drugs.
- 2. Clinical uses of amphotericin B, flucytosine, individual azoles, caspofungin, griseofulvin, and terbinafine.
- 3. Pharmacokinetics and toxicities of amphotericin B.
- 4. Pharmacokinetics, toxicities, and drug interactions of the azoles.
- 5. Main topical antifungal agents.

<u>Chapter 48</u>

- 1. Main targets for antiviral action in viral replication.
- 2. Action of antiherpes drugs and the mechanisms of HSV and CMV resistance.
- 3. Pharmacokinetic properties and toxic effects of acyclovir, ganciclovir, cidofovir, and foscarnet.

- 4. Mechanisms of anti-HIV action of zidovudine, indinavir, and enfuvirtide.
 - Match a specific antiretroviral drug with each of the following:
 - to be avoided in pregnancy;
 - hyperpigmentation;
 - neutropenia;
 - pancreatitis;
 - peripheral neuropathy;
 - inhibition of P450;
 - severe hypersensitivity reaction; injection site reactions.
- 5. Significant properties of 4 drugs active against HBV and HCV.
- 6. Properties of an anti-influenza drug acting at the stage of viral uncoating and another acting at the stage of viral release.