Treatment of LRTIs

EDITING FILE

COLOR INDEX :

- MAIN TEXT
- IMPORTANT
- GIRLS SLIDES
- BOYS SLIDES
- NOTES
- EXTRA

Objectives:

- The types of respiratory tract infections (RTI)
- The antibiotics that are commonly used to treat RTIs & their side effects
- Understand the mechanism of action &pharmacokinetics of individual drugs.

FDr: pharma Question's will come like MOA&what is the drug & clinical use &side effect. PK is rare.

case بالساك راح يجي مرتبط بالمايكرو يسأل ايش الباثوجين وايش المرض وايش التريتمنت والميكانزم حقه

ANTIBIOTICS COMMONLY USED IN THE TREATMENT OF RTIS

- Tetracycline (Doxycycline it's derived from tetracycline)
- Fluoroquinolones
- Beta-lactam antibiotics (Penicillins / Cephalosporins)
- Aminoglycosides
- Macrolides

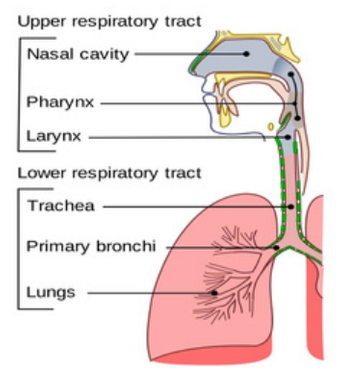
CLASSIFICATION OF RTIS

UPPER RESPIRATORY TRACT INFECTIONS (URTI)

check URTI lecture !!

LOWER RESPIRATORY TRACT INFECTIONS (LRTI)

Costly & More difficult to treat



PNEUMONIA

(Serious infection of bronchioles & alveoli)

- Community –Acquired (CAP)
- Hospital-acquired (Nosocomial)

Causes: Bacteria (just)
-S. pneumonia**(66%),
- H. influenza (20%), -M. catarrhalis (20%).

BRONCHITIS

(inflammation of major bronchi & trachea)

Acute, or Chronic, or Acute exacerbation of chronic bronchitis
Causes: viruses or bacteria
(H. influenza, Streptococcus pneumonia & Moraxella catarrhalis)

This slide is for your understanding only, but we HIGHLY RECOMMEND that you read it & it is IMPORTANT

Thank to 443 team!

Antibiotics Classification

Beta-Lactams	Aminoglycoside	Macrolides	Tetracycline	Cephalosporins	Quinolones	Sulfonamide
Penicillins Cephalosporins Carbapenems Monobactams	Suffix: Mycin	suffix: Thromycin	suffix: Cycline	Prefix: ceph or cef	suffix: Or Floxacin	Prefix: Sulfa

ANTIBIOTICS MOA

Bactericidal (kills the bacteria) by either:
- Destroying the cell wall
- Destroying the Nucleic Acid (DNA or RNA)

Bacteriostatic (stops the growth) by:
- Affecting the protein synthesis

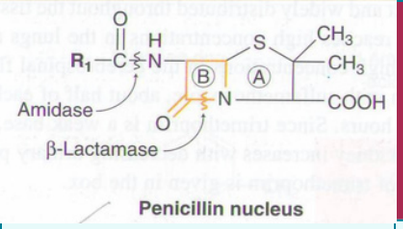
Cell Wall Synthesis	Protein Synthesis	Nucleic Acid Synthesis
Beta Lactams Note: Beta Lactams are sometimes Quinolones combined with beta lactamase inhibitors such as: clavulanic acid, sulbactam, tazobactam, this is because some strains of bacteria have evolved into species that can cleave beta lactam ring through an enzyme called beta lactamase.	30S: - Aminoglycoside - Tetracycline 50S: - Macrolides (Erythromycin) - Clindamycin - Chloramphenicol - Linezoli	DNA: Quinolones RNA: Rifampin, Rifabutin Folate synthesis: Sulfonamide, TMP-SMX

AMAZING mnemonic Thank you 439!

buy AT 30, CCEL (sell) at 50:

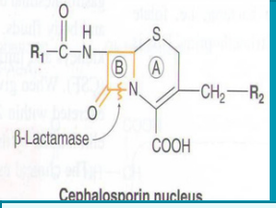
AT: Aminoglycoside, Tetracycline

CCEL : Clindamycin
Chloramphenicol,
Erythromycin,
Linezolid



Penicillins

Broad-spectrum penicillins Mix of 2 drugs to make them stronger	MAO Mechanism of action	Pharmacokinetics (PK)	ADRs	Therapeutic Uses
Amoxicillin+ Clavulanic acid (Augmentin). Ampicillin +Sulbactam. Piperacillin +Tazobactam. all beta lactamase inhibitors that comes from bacteria.	Bactericidal ***Important*** Inhibit bacterial cell wall synthesis through inhibition of peptidoglycan layer of the cell wall.	-Given orally or parenterally. - Not metabolized in human. -Relatively lipid insoluble. -Excreted mostly unchanged in urine (most of its excretion is through the kidney). - Half-life: 30-60 min (increased in renal failure).	Hypersensitivity reactions (rash,urticaria,fever). -Diarrhea -Superinfections a second infection superimposed on an earlier one especially by a different microbial agent resistant to the treatment being used against the first infection). -Nephritis (Inflammation of the kidney) -Convulsions (after high I.V dose or in renal failure).	-URTIs acute otitis media especially those produced by Group A gram positive beta-hemolytic streptococci (GAP). - LRTIs
Act on both gram +ve & gram -ve microorganisms				
443 Note: Beta Lactams are sometimes combined with beta lactamase inhibitors such as: clavulanic acid, sulbactam, tazobactam, this is because some strains of bacteria have evolved into species that can cleave beta lactam ring through an enzyme called betalactamase. Thiazolidine Ring	443 note: (it inhibits transpeptidase enzyme -> failure of cross-links -> unstable cell wall -> bacteria burst).	Probenecid Drug for gouts (uricosuric) slows their elimination and prolongs their half life by competing over tubular secretion with penicillin.		



Cephalosporin nucleus

Cephalosporins

Classified into 4 generations:

	First generation	Second generation	Third generation
e.g	Cephalexin	-Cefuroxime - Cefaclor	-Ceftriaxone -Cefixime -Cefotaxime ربط Ceftriaxone=third generation 
Route of administration	Orally	Well absorbed orally	Given by IV
Spectrum	Gram +ve bacteria	-Mainly Gram -ve Bacteria -active against β lactamase-producing bacteria (H influenzae or M catarrhalis)	More effective against Gram -ve bacilli
Uses	URTIs	Upper & Lower RTIs	In treatment of pneumonia
MOA Mechanism of action	• Bactericidal • Inhibit bacterial cell wall synthesis • (similar mechanism to Penicillins) • - more stable than penicillins to β-lactamase		
PK	• Given parenterally & orally. • Relatively lipid insoluble (like penicillins). • Do not penetrate cells or the CNS except for 3rd generation. • Mostly excreted unchanged by the kidney (glomerular & tubular secretion). • Probenecid slows their elimination & prolongs their half lives. • Half-life: 30-90 min, except ceftriaxone (4-7 hr).		
ADRs	• - Hypersensitivity reactions. • - Thrombophlebitis (inflammation and clot due to trauma of vein by IV administration) Infection of the injection site • Superinfections. • Diarrhea		

GLYCOPEPTIDES

• **Vancomycin & Teicoplanin** are tricyclic glycopeptide antibiotics	**Vancomycin**	**Teicoplanin**
•A number of derivatives, the lipoglycopeptides including telavancin, oritavancin, and dalbavancin	is a tricyclic glycopeptide antibiotic produced by Streptococcus orientalis	a glycopeptide antibiotic produced by Actinoplanes teichomyetius, is available in Europe
Antimicrobial Activity	-broad spectrum of gram+ bacteria - is active against S. aureus, S. epidermidis, streptococci, Corynebacterium & Clostridium spp. -Essentially all species of gram- bacilli & mycobacteria are resistant to it.	-Active against methicillin-susceptible & methicillin-resistant staphylococci, Listeria monocytogenes, Corynebacterium spp., Clostridium spp., & anaerobic gram+ cocci.
MOA	inhibit the synthesis of the cell wall in sensitive bacteria by binding with high affinity to the D-alanyl-D-alanine terminus of cell wall precursor units. Because of their large molecular size, they are unable to penetrate the outer membrane of gram-negative bacteria 	
Resistance to Glycopeptides	Enterococcal vancomycin-resistant S. aureus strain resistance to glycopeptides is the result of alteration of the D-alanyl-D-alanine target to D-alanyl-D-lactate or D-alanyl-D-serine, which bind glycopeptides poorly, due to the lack of a critical site for hydrogen bonding.	

PK	Poorly absorbed after oral administration -administered IV, never IM. -Approximately 30%is bound to plasma protein. -it appears in various body fluids, including the CSF -About 90% excreted by glomerular filtration ha-s a serum elimination t1/2 of ~6h -accumulates if renal function if impaired, and dosage should be adjusted	-administered safely by IM & IV -highly bound by plasma proteins (90-95%) -has an extremely long serum elimination t1/2 (up to 100 hours in patients with normal renal function).
Side Effects	-Rabid IV infusion of Vancomycin may cause erythematous or urticarial reactions, flushing, tachycardia, and hypotension.(it should be taken slowly)The extreme flushing that can occur is sometimes called "red-neck" or "red-man" syndrome. This reaction is not observed with teicoplanin -Careful dosing and monitoring is necessary to balance the risks and benefits	-
	Among the hypersensitivity reactions produced by vancomycin & teicoplanin are macular skin rashes and anaphylaxis. -Ototoxicity is associated with excessively high concentrations of these drugs in plasma. -Nephrotoxicity, with more aggressive dosing regimens increase nephrotoxicity risk.	

GLYCOPEPTIDES cont....
Therapeutic Uses

Therapeutic Uses

- **Respiratory Tract Infections**
  - Vancomycin is employed for the treatment of pneumonia when MRSA is suspected, either because of healthcare-associated acquisition or in patients with community acquired pneumonia. Because vancomycin penetration into lung tissue is relatively low, aggressive dosing is generally recommended
- **Skin/Soft-Tissue and Bone/Joint Infections**
  - where gram-positive organisms including MRSA are the leading pathogens
- **CNS Infections**
  - Because of vancomycin's excellent activity against Str. pneumoniae, it is a key component in the initial empirical treatment of community-acquired bacterial meningitis in penicillin-resistant Str. pneumoniae
- **Endocarditis and Vascular Catheter Infections**
  - Vancomycin is standard therapy for staphylococcal endocarditis when the isolate is methicillin resistant or patients have a severe penicillin allergy.

Carbapenems

Imipenem

MOA	• Inhibits bacterial cell wall synthesis **(bactericidal)** • Has a wide spectrum of activity (aerobic & anaerobic gram negative & gram-positive bacteria, including pseudomonas) • resistant to most **β-lactamases.**
Pharmacokinet-ics	• Not absorbed orally, **taken by I.V**. • Penetrates body tissues & fluids including CSF • Excreted primarily by the kidney • Doses must be reduced in renal failure • Half- life about 1 hr.
Administration	• It should be used in combination with cilastatin? Why? Imipenem is inactivated by **de-hydropeptidase** in renal tubules to a less active & nephrotoxic metabolite, so it is co-formulated with the **dehydropeptidase inhibitor** cilastatin for clinical use (Imipenem/cilastatin).
Advers effect	• GIT (Nausea, vomiting, diarrhea) • Skin rash & reaction at the site of infusion • High doses may **cause seizure** in patients with renal failure • Patients allergic to penicillins may be allergic to carbapenems.

Macrolides

Erythromycin

Dr note: Erythromycin is the prototype Clarithromycin & azithromycin are semisynthetic derivatives of erythromycin

	Azithromycin	Clarithromycin (you can find Dr note in 15 slide)
MOA	Inhibit bacterial **protein** synthesis by binding to 50-S subunit of the bacterial ribosomal RNA **Bacteriostatic** **Bactericidal** at high concentrations (kill the bacteria)	
Pharmacokinetics	• More effective on G-ve bacteria • Stable at gastric acidity • Undergo some hepatic metabolism (inactive metabolite) • Biliary route is the major route of elimination • Only 10-15% excreted unchanged in the urine • Half-life (3 days) • Once daily dosing • No effect on cytochrome P- 450.	• More effective on G+ve bacteria • Stable at gastric acidity • Inhibits cytochrome P450 system • Metabolized in liver to active metabolite • Biliary route is the major route of elimination • Only 10-15% excreted unchanged in the urine • Half-life 6-8 hours.
Clinical uses of Macrolides	• Chlamydial pneumonia :is a type of respiratory infection caused by the bacterium Chlamydia pneumoniae. • Legionella pneumonia :is a severe form of pneumonia caused by the bacterium Legionella pneumophila.	
Adverse effects	• GI disturbances • Hypersensitivity reactions.	Dr note: Anorexia, nausea, vomiting, & diarrhea are common. GI intolerance, which is due to a direct stimulation of gut motility, is the most common reason for discontinuing erythromycin & substituting another antibiotic.

Fluoroquinolones

Fluoroquinolones are synthetic antibiotics & may be classified into “generations” based on their antimicrobial targets:

1

The 1st generation :
nalidixic acid, a narrow spectrum of susceptible organisms

2

The 2nd generation:
Ciprofloxacin & norfloxacin. They have activity against aerobic gram-negative & atypical bacteria. They also exhibit significant intracellular penetration, allowing therapy for infections in which a bacterium spends part or all of its life cycle inside a host cell (for example, chlamydia, mycoplasma, and mycobacteria)

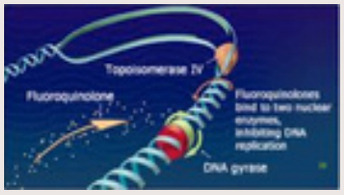
3

The 3rd generation:
Levofloxacin with increased activity against gram-positive bacteria

4

The 4th generation:
moxifloxacin against anaerobic & gram-positive organisms.

Fluoroquinolones cont...



MOA

- Fluoroquinolones enter bacteria through porin channels and exhibit antimicrobial effects on DNA gyrase bacterial (topoisomerase II) and (topoisomerase IV)
- Inhibition of DNA gyrase results in relaxation of supercoiled DNA, promoting DNA strand breakage
- Inhibition of topoisomerase IV impacts chromosomal stabilization during cell division, thus interfering with the separation of newly replicated DNA
- In gram-negative organisms (Pseudomonas aeruginosa), the inhibition of DNA gyrase is more significant than that of topoisomerase IV.

Ciprofloxacin has higher affinity for topoisomerase II & is the most active agent of this group against gram-negative organisms, P aeruginosa in particular. & should not be used for Str. pneumoniae infections

- In gram-positive organisms (Str. pneumoniae), Levofloxacin, has superior activity against gram-positive organisms, including Str. pneumoniae
- while those with more topoisomerase IV activity (for example, moxifloxacin) should not be used for P. aeruginosa infections.

Dr note:
Gm –ve pseudomonas aeruginosa . inhibition of DNA gyrase > topoisomerase IV

Gm +ve str pneumonia inhibition of topoisomerase IV > DNA gyrase

Antimicrobial spectrum : the range of microorganisms that an antibiotic can effectively target and kill.

- Bactericidal activity is more pronounced as serum drug concentrations increase to approximately 30-fold the MIC of the bacteria
- Fluoroquinolones are effective against gram-negative (Escherichia coli, P. aeruginosa, H influenzae), atypical (Legionellaceae, Chlamydiaceae), gram-positive organisms (streptococci), and some mycobacteria (Mycobacterium Tuberculosis)

Antimicrobial spectrum : the range of microorganisms that an antibiotic can effectively target and kill.	• are typically not used for the treatment of Staphylococcus aureus or enterococcal infections. They are not effective against syphilis and have limited utility against Neisseria gonorrhoeae due to disseminated resistance worldwide • Levofloxacin & moxifloxacin are sometimes referred to as “respiratory fluoroquinolones,” because they have excellent activity against Str. pneumoniae, which is a common cause of community-acquired pneumonia (CAP). • Fluoroquinolones are considered alternatives for patients with a severe β-lactam allergy.

Norfloxacin	Levofloxacin
• Is infrequently prescribed due to poor oral bioavailability and a short half-life • It is effective in treating non-systemic infections, such as urinary tract infections (UTIs), prostatitis, and infectious diarrhea	• Due to its broad spectrum of activity, levofloxacin is utilized in a wide range of infections, including prostatitis, skin infections, CAP, & nosocomial pneumonia • Unlike ciprofloxacin , levofloxacin has excellent activity against Str. pneumoniae respiratory infections • Levofloxacin has 100% bioavailability and is dosed once daily.

Fluoroquinolones cont...

Ciprofloxacin

- Is effective in the treatment of many systemic infections caused by gram-negative bacilli
- It has the best activity against P. aeruginosa & is commonly used in cystic fibrosis patients
- With 80% bioavailability, the IV and oral formulations are frequently interchanged
- Traveler's diarrhea caused by E. coli as well as typhoid fever caused by Salmonella typhi can be effectively treated with ciprofloxacin
- Is also used as a second-line agent in the treatment of tuberculosis
- Although typically dosed twice daily, an extended-release formulation

is available for once-daily dosing, which may improve patient adherence to treatment.

Dr note about clarithromycin:

Erythromycin base is destroyed by stomach acid and must be administered with enteric coating. Clarithromycin is derived from erythromycin by addition of a methyl group and has improved acid stability and oral absorption compared with erythromycin.

Erythromycin & clarithromycin inh CytP 3A4

Erythromycin is active against susceptible strains of gram-positive organisms, especially pneumococci, streptococci, staphylococci, & corynebacteria. Mycoplasma pneumoniae, L pneumophila, Chlamydia trachomatis, Chlamydia psittaci, Chlamydia pneumoniae, H pylori, Listeria monocytogenes, & certain mycobacteria (Mycobacterium kansasii, Mycobacterium .scrofulaceum) are also susceptible

Clarithromycin & erythromycin are similar with respect to antibacterial activity except that clarithromycin is > active against .Mycobacterium avium complex

.The advantages of clarithromycin compared with erythromycin are lower incidence of GI intolerance & less frequent dosing

Fluoroquinolone Resistance

High levels of fluoroquinolone resistance have emerged in gram-positive and gram-negative bacteria, primarily due to chromosomal mutations.

-Note: Cross-resistance exists among the quinolones.

The mechanism for this resistance include the following:

1. **Altered Target:** Chromosomal mutation in bacterial genes (e.g., gyrA or parC) have been associated with decreased affinity for fluoroquinolones at their site of action.

   -Note: Both topoisomerase IV and DNA gyrase may undergo mutations.

   -Explanation: Fluoroquinolones inhibit bacterial cell division by inhibiting topoisomerase and DNA gyrase enzymes, so if those enzymes are mutated, fluoroquinolones will not recognize it.

2. **Decreased Accumulation:** Reduces intracellular concentration.

Fluoroquinolone Pharmacokinetics

Absorption

- **Orally:** 80% to 99% of fluoroquinolones are absorbed.
  - **-Exception:** only 35% to 70% of orally administered Norfloxacin is absorbed.
- **Intravenous and Ophthalmic:** of Ciprofloxacin, Levofloxacin, and Moxifloxacin are available.
- **Note:** Ingestion of fluoroquinolones with Sucralfate, Alimnum- or Magnesium containing antacids, or dietary supplements containing iron or zinc can reduce the absorption.

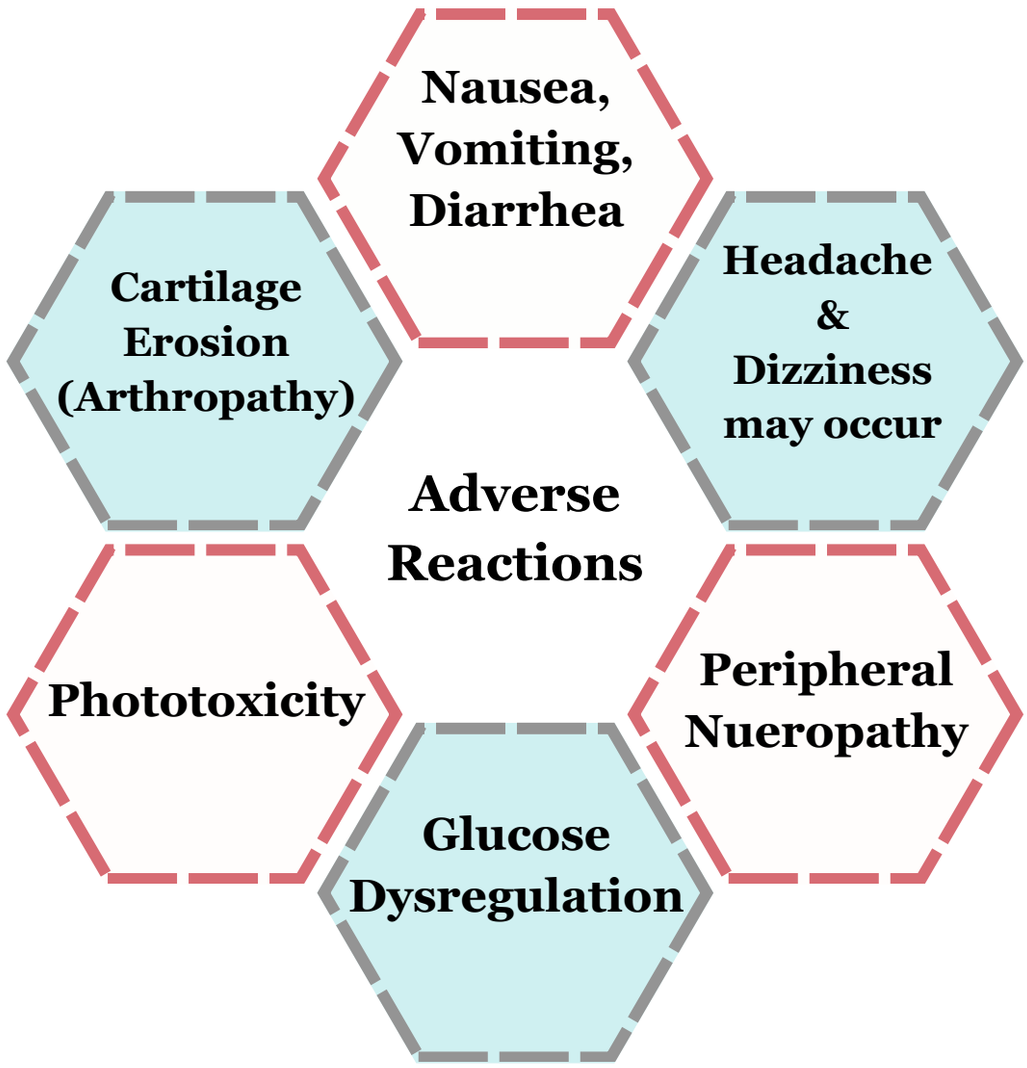
Distribution

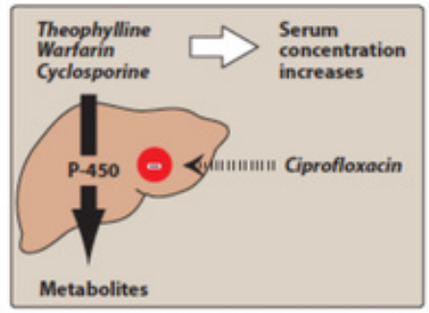
- **Binding of plasma proteins:** ranges from 10% to 40%.
- **Distribution:** The fluoroquinolones distribute well into all tissues and body fluids, which is one of their major clinical advantages.
- **Levels:** Are high in bone, urine (except Moxifloxacin), kidney, and prostatic tissue (but not prostatic fluid), and their concentrations in the lung exceeds those in serum.
- **Penetration intro Cerebrospinal Fluid:** Is relatively low except for Ofloxacin.
- **Note:** Fluoroquinolones also accumulate in Macrophages and Polymorphonuclear leukocytes.
  - **-Result:** Having activity against intracellular organisms.

Elimination

- **Most fluoroquinolones:** Are excreted Renally.
  - **-Result:** Dosage adjustment are needed in Renal dysfunction.
- **Moxifloxacin:** Is excreted primarily by the Liver.
  - **-Result:** No dose adjustment is required for renal impairment.

Fluoroquinolone Adverse Reactions



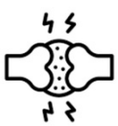


Notes

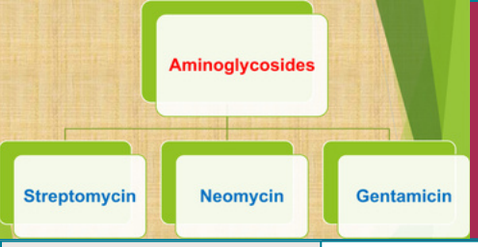
I. In Phototoxicity: patients taking these agents should be advised to use sunscreen and avoid excess exposure to sunlight.

-Note: If phototoxicity occurs, discontinuation of the drug is advisable.

II. Cartilage Erosion (Arthropathy): It has been observed in immature animals exposed to fluoroquinolone.



-Result: These agent should be avoided in Pregnancy and Lactation and in Children under 18 years of age.



Aminoglycosides

Examples	**Streptimycin - Neomycin - Gentamicin.**
MOA	**-Mechanism:** Inhibit bacterial protein synthesis by binding to 30-S subunit of the bacterial ribosomal protein. **-Type:** Bactericidal. DR. NOTE: AMINOGLYCOSIDES ARE MOSTLY USED AGAINST AEROBIC GRAM-NEGATIVE BACTERIA, ESPECIALLY WHEN THE ISOLATE MAY BE DRUG-RESISTANT & WHEN THERE IS SUSPICION OF SEPSIS. **-Activity:** Only active against Gram Negative Aerobic Organisms.
PK	• **Absorption:** Poorly absorbed orally. **-Reason:** Because they're Highly Charged. **-Result:** Given Parenterally (IM, IV). 💉 • **T1/2:** Is 2-3 hours. **-Note:** In renal impairment T1/2 increase to 24-48 hours. • **Cross Placenta.** • **Excretion:** Excerted unchanged in the urine.
Adverse Effects	• **Ototoxicity.** • **Nephrotoxicity.** • **In very high doses:** Neuromuscular blockade that results in respiratory paralysis.
Therapeutic Uses of Gentamicin	• **Severe infections caused by Gram Negative Organisms.**

Tetracyclines

Examples	**Chlortetracycline - Doxycycline - Minocycline.**
MOA & Antimicrobial Activity	**-Mechanism:** Inhibit protein synthesis by binding reversibly to 30-S subunit of the bacterial ribosome. **-Type:** Broad-spectrum Bacteriostatic antibiotic. **-Activity:** Active against many Gram-Positive & Gram-Negative bacteria (Anaerobes, Rickettsiae, Chlamydiae & Mycoplasmas).
PK	• **Doxycycline:** It's a long acting tetracyclin. 💊 **Usually given Orally.** • **Absorption:** Is 90% - 100%. **-Location:** Absorbed in the upper s. Intestine. **-Note:** Absorption is best in absence of food. **-Caution:** Food & di & tri-valentine cations (Ca, Mg, Fe, Al) impair absorption. • **Protein Binding:** 40% - 80%. • **Distribution:** Distributed well, including CSF. • **Cross Placenta & Excreted in milk.** 🫀 **Metabolism:** Largely Metabolized in the Liver.

Side Effects	**1. Nausea, Vomiting, Diarrhea, Epigastric pain.** **-Reason:** If given with food. **2. Thrombophlebitis** (If given I.V). **3. Hepatic toxicity** (In prolonged therapy with high dose). **4. Brown Discoloration of Teeth** (In children). **5. Deformity or Growth Inhibition of Bones** (In children). **6. Phototoxicity.** **7. Vertigo.** (دوار) **8. Superinfections.** **(Because they have a broad spectrum activity, against wide range of bacteria, they may disrupt normal flora which create an environment for other pathogens)** **Dr. Note: Deposited in fetal teeth & bone; contraindicated in pregnancy & children.** **readily bound to calcium deposited in newly formed bone or teeth in young children. When a tetracycline is given** **during pregnancy, it can be deposited in the fetal teeth, leading to fluorescence, discoloration, and enamel dysplasia. It can also be** **deposited in bone, where it may cause deformity or growth inhibition. Because of these effects, tetracyclines are generally avoided in** **pregnancy. If the drug is given for long periods to children younger than 8 years, similar changes can result.**
Contraindications	• **Pregnancy.** • **Breast feeding.** • **Children** (Below 10 Years).
Uses	• **Treatment of Upper Respiratory Tract Infections (URTIs) caused by:** S. pyogens, S. Pneumonia & H. Influenza.

“ study smarter , not harder “

Active recall

For Anki flash cards click the icon

Take active quizzes in our team channel to test your understanding.

click the icon to get free flash cards

summary

MCQs

1 which of the following is an adverse effect of the penicillin:

- **A** Thrombophlebitis
- **B** Ototoxicity
- **C** Diarrhea superinfections
- **D** A and B

2 A patient came to the ER with respiratory tract infection, he was diagnosed with chlamydial pneumonia , which of the following antibiotics, should be used for him?

- **A** Clarithromycin
- **B** Imipenem
- **C** Moxifloxacin
- **D** Doxycycline

3 Which of the following antibiotics can cause brown discoloration of teeth in children?

- **A** Vancomycin
- **B** Penicillin
- **C** Macrolides
- **D** Tetracycline

4 Which of the following antibiotic inhibit bacterial cell wall synthesis?

- **A** Fluoroquinolones
- **B** Macrilides
- **C** Tetracycline
- **D** Carbapenems

1.C 2.A 3.D 4.D

MCQs

5 Which of the following antibiotics is contraindicated in pregnancy?

- **A** Penicillin
- **B** Tetracycline
- **C** Erythromycin
- **D** Gentamicin

6 Cartilage erosion can be seen in children taking which of the following antibiotics?

- **A** Fluoroquinolones
- **B** Imepenems
- **C** Vancomycin
- **D** Gentamicin

7 Which of the following is an example of the Aminoglycosides?

- **A** Erythromycin
- **B** Streptomycin
- **C** Vancomycin
- **D** All of the above

8 A patient came to the ER, he was diagnosed by Traveler's diarrhea which is an infection caused by E.coli, which of the following antibiotics should be used for him?

- **A** Gentamicin
- **B** Clarithromycin
- **C** Ciprofloxacin
- **D** Minocycline

1.B 2.A 3.B 4.C

SAQs

1. **What’re the mechanisms responsible for Fluoroquinolones resistance?**

   Slide 16

2. **What’s the mechanism of action of Macrolides?**

   Slide 11

3. **What’re the adverse effect of Penicillin?**

   Slide 5

4. **What’s the mechanism of action of Penicillins?**

   Slide 5

Team leaders

Ritaj Alsubaie **Raseel Aldajany**

Eyad Alzubaidi

Team members

Madaen Alarifi
Haya Alateeq
Noreen Almarabah
Janan Alsayari
Norah Alnoshan
Alanoud alnajawi
Sahar Alfallaj
Samiyah sulaiman
shaden Alotaibi
Roaa Alhajri
Rimaz Alhammad

Ali Al-Abdulazem
Waleed Alanazi
Abdulaziz Sahhari
Abdulrahman Almalki
khalid Alghamdi
Abdulaziz Alanazi
Abdulrahman Alnafisah
Abdullah Alzoom
Ahmed Alabbad

Contact us at : pharmacology.444ksu@gmail.com