Antibiotic Pharmacology Cheat Sheet

by happyfeet2020 via cheatography.com/144934/cs/31417/

Gram Positive Bacteria

- Plasma membrane covered by thick peptidoglycan (murein) cell wall

-Certain abx inhibit synthesis of the petidoglycan cell wall: penicillins, cephalosporins, bacitracin, vancomycin, monobactams and carbapenems

-Blue purple stain

-most common aerobic gram + bacteria w/ podiatric complications: staphylococcus (grm += staph aureus, grm - = staph epidermidis), streptococcus - penicillin sensitive/non-penicillinase producing, methicillin resistant staph aureus, methicillin sensitive/penicillinase producing, vancomycin resistant staph a and corynebacterium

Group A strep common in superficial skin infections (erysipelas)

Group B strep very common in diabetic foot infections

Group A and B strep sensitive to: penicillins, cephalosporins, clindamycin, erythromycin

Cornyebacterium minutissimum is implicated in erythrasma (treat with erythromycin)

*If C&S reports MRSA or group D strep (enterococci), consider infectious disease consult

Gram Negative Bacteria

-Plasma membrane covered by a thinner peptidoglycan cell wall which is then covered w/ an outer lipopolysaccharide membrane

-prohibits entry of most penicillins and cephalosporins

- resists the uptake of blue dye so stains pink

-Aerobic gram - pathogens: enterics like salmonella, klebsiella, proteus, etc, pseudomonas, etc

some broad spectrum synthetic penicillins and 3rd gen cephalosporins (IV) may allow some entry through outer membrane. However, space b/ cell wall and outer membrane is high in B-lactamases which is a penicillin-destroying enzymes

The addition of B-lactamase inhibitor (clavulanic acid) to a broad spectrum penicillin may increase its spectrum of activity to include some gram - coverage

*possible to have a mixed gram + and gram - bacteria (ie diabetic foot that has been exposed)

Anaerobes

-infections often composed of mixed gm+ and gm- bacteria

produce foul smelling gas and frequently encased in abscess wall

common anaerobes: clostridium which respond well to penicillin, clindamycin and tetracyline. infection may be aggressive and may need iV therapy and surgical intervention

Bacteroids are also a common anaerobe which is a gm- bacillus. They are common in diabetic foot infections. Abx therapy include PO clindamycine, amoxi clav.

inhibitors of Cell Wall Synthesis - B-Lactams

inhibitors of Cell Wall Synthesis - B-Lactams (cont)

Folate Antagonists

MOA:	The final step in bacterial cell wall synthesis is cross linking of adjacent peptidoglycan strands. B-lactams bind to transpeptidase (penicillin binding proteins) prevents crosslinking of peptidoglycan strands in cell wall during wall synthesis leading to weakened cell wall and eventually cell death
Penicillins:	*Natural PCNs*
	Penicillinase Resistant PCNs (Cloxacillin) - good for strep, penicillinase producing staph, anaerobes except bacteroides
	Aminopenicillins (Amoxicillin) - good for strep, gm- organisms, non penicillinase producing staph, anaerobes other than bacteroids
	Penicillin/B-Lactamase Inhibitor (Amoxicillin clavulanate)- good for strep, increase in gm - (not pseudomonas), penicillinase producing staph, anaerobes including bacteroides. Beta-lactamase inhibitors are a class of medicine that block the activity of beta-lactamase enzymes (also called beta-lactamases), preventing the degradation of beta-lactam antibiotics ie amoxicillin which can be restored and widened with clavulanate
	Most PCNs are excreted unchanged via renal tubular mechanism, therefore dosages must be adjusted in pts w/depressed renal function
	PCNs are bacteriocidal
	Side effects: anaphylaxis, 5-15% cross sensitivity w/ cephs, rash/dermatitis, nephritis, diarrhea, enterocolitis
Cephalosporins:	1st Gen- Cephalexin, Cefadroxil. Good for gm+ staph (penicillinase & non-penicillinase producing except MRSA), & strep, okay against gm- and anaerobes other than bacteroids
	Most cephalosporins are excreted unchanged via renal tubular mechanisms (secretion). need to adjust dosage in pts w/ reduced renal function
	Bacteriocidal activity
	Side effects: same as penicillins
Carbapenems	
Monobactams	

Cell Wall Synthesis inhibitor- Glycopeptides	
Bacitracin:	MOA: glycopeptide that inhibits cell wall synthesis by preventing transport of cell wall precursors
	Activity against gm+ and some gm-. However pseudomonas is resistant
	side effects: nephrotoxic, neurotoxic and toxic to bone marrow if used systemically therefore only used topically

Sulfonamides/Trimethoprim	Synergists -> produces a greater effect when used together
	MOA: Bacteria synthesize folate from pteridine & PABA, whereas humans require dietary folate b/c humans don't have the enzymes seen in this bacterial pathway, these abx are relatively free of adverse effects. Prevents formation of folate at step and ultimately, the synthesis of bacterial purines and DNA, resulting in a bacteriostatic effect.
	Metabolized in liver
Sulfamethatoazole/-Trimethoprim	effective against Gm+ organisms & some strains of MRSA, excellent coverage of Gm-, except pseudomonas
	Bacteriostatic, renal clearance
	Side effects: allergic reactions in 30% population (potentially fatal), diarrhea, N/V, compete w/bilirubin for binding sites on serum albumin -> kernicterus in newborns (increase bilirubin levels) , hemolytic anemia in pts w. G6PD deficiency, renal clearance

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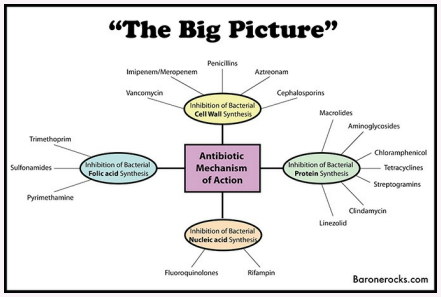
Folate Antagonists- Quinolones

MOA:	Inhibit DNA synthesis through a specific action on DNA gyrase or topoisomerase IV. Topoisomerases (DNA gyrase or topoisomerase IV) bind to DNA -> transient cleave complexes (double stranded breaks). In presence of quinolones, levels of cleavage increase dramatically. After traversal by replication complexes, these breaks become permanent double stranded fractures -> cell death
Ciprofloxacin	Excellent activity against all Gm- organisms, including pseudomonas. Marginal activity against staph A, including some strains of MRSA, minimal activity against strep or anaerobes
	100% bioavailability, renal and hepatic clearance, bacteriocidal
	Best bet for highly suspected Gm- or pseudomonas bacteria
	Side effects: N/v, contraindicated in children with open growth plates due to to possible cartilage degeneration, tendon degeneration, peripheral neuropathy, mental health side effects and blood sugar disturbances (hypoglycaemic coma), concomitant NSAID use may increase risk of CNS stimulation and convulsions, aortic aneurysm, highest risk of causing colonization w/MRSA and C.difficle

Antibiotic Mechanism of Action



Inhibitors of Protein Synthesis- 30S Subunit

Tetracyclines	MOA: bind reversibly to the 30S ribosomal subunit at a position that blocks the binding of the aminoacyl-tRNA to the acceptor site on the mRNA-ribosome complex. Protein synthesis is ultimately inhibited, leading to a bacteriostatic effect
	effective against gm+ except MRSA, few gm- and some anaerobes (no bacteroides)
	Bacteriostatic, renal clearance
	Side effects: possible allergic rxn, diarrhea+ N/V, thrombophlebitis, photosensitivity, kidney toxicity, interaction w/calcium, need to be taken on empty stomach
Aminoglycosides (Gentamycin,etc)	Effective against aerobic gm- and pseudomonas, not effective against anaerobes
	Topical/IV, bacteriocidal, renal clearance
	Side effects: possible allergic rxn, n/v, nephrotoxicity, ototoxicity, neuromuscular block, photosensitivity
	Prokaryotes have 70s ribosomes, consisting of a 30s and 50s subunit

Inhibitors of Protein Synthesis- 50S Subunit

MOA	Bind reversibly to the 50S ribosomal subunit at a position that blocks the binding of the aminoacyl-tRNA to the acceptor site on the mRNA-ribosome complex. Protein synthesis is ultimately inhibited, leading to a bacteriostatic effect
Erythromycin (Macrolides)	Effective against gm+ except MRSA, few gm- and some anaerobes (no bacteroides)
	Bacteriostatic and hepatic clearance
	Side effects: possible allergic rxn, diarrhea, n/v, thrombophlebitis, hepatotoxicty, metabolites can inhibit certain cp450 isoenzymes in the liver & thereby increase conc. of drugs also metabolized by liver enzymes (eg increase in levels of calcium channel blockers)

Inhibitors of Protein Synthesis- 50S Subunit (cont)

Clindamycin (Lincosamides)	Effective against gm+ including some MRSA, and most anaerobes, including bacteroides; however not effective against c.difficile
	Bacteriostatic (very close to bacteriocidal), hepatic clearance
	good penetration of most tissues, including bone
	Side effects: Diarrhea

Cell Wall Synthesis Inhibitors

Cloxacillin *Penicillinase resistant penicillins*	250 or 500 mg, mitte: X tablets,sig: 1 PO every 6h for x days
Amoxicillin *Aminopenicillins*	250 or 500mg, mitte: X tablets, sig: 1 Po every 6-8 hours for x days
Amoxicillin/clavulanate *Penicillin/ B/lactamase inhibitor*	250,500,875mg, mitte: x capsules. sig: 1 PO every 8-12h (12 for 875mg)
Cephalexin *Cephalosporin*	250 or 500mg, mitte: X tablets, sig: 1 PO every 6h for X days
Cefadroxil *Cephalosporins*	500mg, mite: x capsules, sig: 1 PO every 12h for x days

Protein Synthesis Inhibitor

Clindamycin *Lincosamides*	150 or 300mg, mitte: X capsules, sig: one PO every 6-8 hours for X days
Erythromycin *Macrolides*	250 or 500mg, mitte: X tablets, sig: 1 PO every 6h for X days

Protein Synthesis Inhibitor (cont)

Azithromycin *Macrolides*	250mg, mitte: 6 tablets, sig: day 1- 2 tablets once PO, days 2-5: 1 tablet PO daily
Tetracyclines *(30S subunit)*	250 or 500mg, mitte: X tablets, sig: 1 PO every 6h for X days

Folate Antagonists

Sulfamethoxazole/trimethoprim *Sulfonamides/trimethoprim*	800/160mg tablets, mitte: X tablets, sig: 1 PO every 12h for X days
Ciprofloxacin *Quinolones*	250,500,750mg mitte: X tablets, sig: 1 PO every 12H

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