Antimicrobial Agents 101

Summit on Antimicrobial Stewardship
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Spencer H. Durham, Pharm.D., BCPS (AQ-ID)
Assistant Clinical Professor of Pharmacy Practice
Auburn University Harrison School of Pharmacy
I, Spencer Durham, have no actual or potential conflict of interest in relation to this program.
Objectives

• At the end of the presentation, the audience will be able to:
  – Identify the different classes of antimicrobial agents and review the individual agents within each class
  – Describe the spectrum of activity of the antimicrobial drug classes
  – Review major adverse effects associated with the antimicrobial drug classes
• Antimicrobial therapy crosses into most, if not all, areas of pharmacy practice
• Antimicrobial agents are widely prescribed in the acute care, long-term care, and outpatient settings
  – Frequently prescribed inappropriately (50%)
    • Wrong drug for disease
    • No antibiotic indication
• Limited development of new antibiotics, particularly *novel* antibiotics
• Antimicrobial resistance is rapidly increasing
In general, ultimate goal is to eradicate the causative organism of infection

Treat infection appropriately
- Empiric therapy: target most likely pathogens for the disease state
- Definitive therapy: use the least broad-spectrum, yet most appropriate, therapy to target the known pathogen

Prevent transmission
- Infection control
  - Hand hygiene
  - Appropriate disinfecting of medical equipment
Goals of Antimicrobial Therapy

- **Prevention of infections**
  - Vaccination
  - Prevent bacterial growth or colonization
    - Example: Cystic fibrosis

- **Prevent recurrence of infection**
  - Prophylaxis of infection – use judiciously
    - Examples: UTIs, meningitis

- **Minimize the development of antimicrobial resistance**
  - Use most narrow-spectrum, effective agent possible
  - Judicious overall use of antimicrobials
    - Example: Abx use for infections likely due to viral causes
Antimicrobial Considerations

• Consider:
  – Local susceptibility patterns
  – Overuse of specific antimicrobials in the local institution or area
    • Example: Fluoroquinolone overuse
  – Institutional formulary restrictions
  – Overall cost effectiveness
    • IV to PO conversions
    • Use of new, expensive antibiotics v. cheaper antibiotics with potential equal efficacy
Antimicrobial Considerations

• Empiric therapy
  – Broad-spectrum agent(s) with reliable coverage against the most likely causative pathogens

• Definitive therapy
  – Can generally only be done after obtaining culture and sensitivity results
  – May use other tests to guide therapy, such as PCRs

• Duration of treatment
  – Not well-defined, usually based on experience rather than evidence
  – Generally, 7-14 days for most infections
Microbiology

• Bacterial Pathogens
  – Normal commensal flora
    • Bacteria normally present in humans
    • Not pathogenic under usual circumstances
      – Can be if given appropriate opportunity
  – Sterile site growth
    • Blood stream
    • CSF
  – Nonsterile sites
    • Sputum
    • Wound
### Gram-positive Bacteria

<table>
<thead>
<tr>
<th>Cocci in Clusters</th>
<th>Cocci in Pairs/Chains</th>
<th>Other</th>
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<tr>
<td><em>Staphylococcus aureus</em></td>
<td><em>Streptococcus pneumoniae</em></td>
<td><em>Clostridium species</em></td>
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<tr>
<td><em>Staphylococcus epidermidis</em></td>
<td><em>Streptococcus pyogenes</em> (group A)</td>
<td><em>Listeria monocytogenes</em></td>
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<td>• Other coagulase-negative staphylococci</td>
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<td><em>Staphylococcus saprophyticus</em></td>
<td><em>Streptococcus agalactiae</em> (group B)</td>
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<td><em>Viridans group streptococci</em></td>
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<td><em>Enterococcus faecalis</em></td>
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<td><em>Enterococcus facium</em></td>
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Gram-Negative Bacteria

• Bacilli (rods)
  – Anaerobic
    • Bacteriodes
  – Facultative
    • Escherichia coli
    • Klebsiella
    • Proteus
    • Pseudomonas aeruginosa
    • Enterobacter
    • Serratia
Pharmacodynamics

- Minimum inhibitory concentration (MIC)
  - Bacteria are mixed with increasing concentrations of an antibiotic on microdilution plates
  - MIC = Mixture with the lowest concentration of antibiotic where there is no visible growth
  - ***Remember, just because an antibiotic has the lowest MIC for a pathogen, does not mean it is the best choice
  - The number associated with the MIC is variable by drug, so the lower the number does not necessarily mean a bacteria is more sensitive to the drug
Pharmacodynamics

• **Bactericidal**
  • Actually destroys the organism
    • No help from immune system is required
    • Cell wall synthesis inhibitors (beta-lactams, vancomycin)
    • Aminoglycosides
    • Fluoroquinolones
  • Preferred for certain disease states
    • Endocarditis
    • Meningitis
    • Infections in neutropenic patients
    • Osteomyelitis
    • Sepsis
Pharmacodynamics

- **Bacteriostatic**
  - Inhibit growth of organism without killing it
  - Once antibiotics are removed, the organism can begin growing again
  - Works in conjunction with the patient’s immune system to clear the infection
  - Protein synthesis inhibitors (exception: aminoglycosides)
    - Tetracyclines
    - Clindamycin
    - Linezolid
    - Macrolides
Pharmacodynamics

• **Time dependent killing**
  – Duration of time drug remains above the MIC reflects bacterial inhibition
    • Beta-lactams
    • Vancomycin

• **Concentration dependent killing**
  – Ratio of peak concentration of the drug to the MIC
    • The higher the concentration, the greater degree of bacterial inhibition
    – Aminoglycosides
    – Fluoroquinolones
    – Daptomycin
Antibiotic MOAs

DNA replication

- Nucleotide biosynthesis
- Protein synthesis
- RNA transcription
- mRNA

- DNA replication
- Topoisomerase
- mRNA

- Cell wall synthesis
- Metronidazole
- Fluoroquinolones
- 

- Fluoroquinolones
- Rifampin
- Cytoplasmic membrane integrity
- Daptomycin
- Telavancin
- Tigecycline
- Aminoglycosides
- Macrolides
- Linezolid
- Clindamycin
- Tetracyclines

 TMP-SMX = trimethoprim-sulfamethoxazole
Beta-Lactams

- Penicillins
- Cephalosporins
- Carbapenems
- MOA: inhibition of cell wall synthesis
- Bactericidal
- Time-dependent
• Adverse Effects
  – Hypersensitivity reactions
    • Mild rash
    • Acute interstitial nephritis
    • Anaphylaxis
  – Some cross-sensitivity between agents
    • Difficult to predict; closer structural relationships are more likely to cross-react
  – Seizures
    • High doses of beta-lactams
    • Particularly associated with the carbapenems (imipenem and ertapenem)
Beta-Lactams

• Generally, well-tolerated and safe antimicrobials
• ALL beta-lactams lack activity against atypical organisms
  – *Mycoplasma pneumoniae*
  – *Chlamydyphila pneumoniae*
• Lack MRSA activity
  – Exception: Ceftaroline (Teflaro®)
Natural Penicillins

• Penicillin G, Penicillin V
  – Good activity: *Treponema pallidum* and most streptococci
  – Moderate activity: *Streptococcus pneumoniae*, enterococci
  – Poor activity: almost everything else

• IM (long acting depot formulation)
  – Procaine, benzathine
    • **FATAL IF GIVEN IV**

• Treatment
  – Syphilis (neurosyphilis)
  – Susceptible streptococcal infections such as pharyngitis or endocarditis
• **Amoxicillin, ampicillin**
  – Good activity: streptococci, enterococci, *N.meningitidis*
  – Moderate activity: enteric gram-negatives, *Haemophilus*
  • Would NOT generally use for empiric therapy, but could consider for targeted therapy
  – Poor activity: staphylococci, anaerobes

• **Treatment:**
  – Upper respiratory infections
  – Infections due to *Enterococcus*
  – Select gram-negative infections
Penicillinase-Resistant Penicillins

• Nafcillin, dicloxacillin
  – Good activity: MSSA, streptococci
  – Poor activity: Gram (-), enterococci, anaerobes, MRSA
• Sometimes called the “anti-staphylococcal penicillins”
  – Used for MSSA, but NOT MRSA
• Eliminated by liver
  – No renal adjustment
• Used for MSSA infections, endocarditis, and SSTI’s
• Limited utility for empiric treatment now due to increasing MRSA
Beta-Lactam/Beta-Lactamase Inhibitor Combinations

• Amoxicillin/clavulanate
  – MSSA, streptococci
  – Respiratory pathogens, some enteric gram-negative pathogens (*E.coli, Klebsiella*, etc.)
  – Some anaerobic coverage

• Ampicillin/sulbactam
  – Same as amoxicillin/clavulanate
  – *Acinetobacter*

• Piperacillin/tazobactam
  – MSSA, streptococci
  – Excellent gram-negative coverage
  – *Pseudomonas*
  – Anaerobic pathogens
Cephalosporins

- Grouped into generations
  - 1st generation
    - Cefazolin, cephalexin, cefadroxil, cephalothin
  - 2nd generation
    - Cefuroxime, cefoxitin, cefotetan, cefprozil
  - 3rd generation
    - Ceftriaxone, cefotaxime, ceftazidime, cefdinir, cefpodoxime, cefixime, ceftibuten
  - 4th generation
    - Cefepime
  - “5th” generation
    - Ceftaroline
  - Other: Ceftolozane/tazobactam; ceftazidime/avibactam
Cephalosporins

- As a general rule, when moving from the 1\textsuperscript{st} to the 4\textsuperscript{th} generation, gram-positive activity stays the same and gram-negative activity increases
  - However, NUMEROUS important exceptions to this rule exist
- NO cephalosporins cover enterococci
- Most have little or no activity against anaerobes
  - Exception: some 2\textsuperscript{nd} generation agents
- Ceftazidime and Cefepime cover \textit{Pseudomonas}
- Ceftaroline is the \textbf{ONLY} beta-lactam that covers MRSA
- Potential cross-reactivity with the penicillins
  - Lower generations more likely to cross-react
1st Generation

• **Good activity:** MSSA, streptococci
• **Moderate activity:** some enteric GNRs
  • *E.coli*
• **Poor activity:** enterococci, anaerobes, MRSA, *Pseudomonas*
• **Good alternative to anti-staphylococcal penicillins**
  • Less phlebitis
  • Infused less frequently
• **Do NOT cross blood-brain barrier**
  • Do NOT use for CNS infections
2nd Generation

• Similar spectrum of activity to first generation agents, but better gram-negative activity

• Cefotetan
  – Disulfuram-like reaction with ethanol
  – Inhibit vitamin K production and prolong bleeding

• Anaerobic coverage
  – Cefotetan, cefoxitin
  – These are the ONLY cephalosporins that have adequate activity against anaerobes

• Do NOT cross blood-brain barrier
• Greater gram-negative activity compared to first and second generation agents
  – Several important exceptions
• Ceftazidime
  – NOT active against gram-positives
  – ONLY third generation agent with activity against *Pseudomonas*
• Ceftriaxone, cefotaxime, ceftazidime
  – Cross blood-brain barrier
  – CNS infections
4th Generation

• Cefepime
  – “Cefazolin + Ceftazidime”
  – Active against many gram-positive and gram-negative organisms, including *Pseudomonas*

• Good empiric choice for many nosocomial infections

• Use associated with increased incidence of *Clostridium difficile* infections and extended-spectrum beta-lactamase (ESBL) production
  – Also true for third generation agents
• Ceftaroline
  – Does not really fit well into the “generation” scheme usually associated with the cephalosporins
  – **ONLY** beta-lactam antibiotic with activity against MRSA
  – Less gram-negative activity when compared to cefepime
    • Does NOT reliably cover *Pseudomonas*
  – asdf
Other Cephalosporins

• Ceftolozane/tazobactam
  – New cephalosporin combined with an existing beta-lactamase inhibitor

• Ceftazidime/avibactam
  – Existing cephalosporin combined with a new beta-lactamase inhibitor

• Active against ESBL organisms and some carbapenemase-producing organisms
• Place in therapy still to be determined
Carbapenems

- Imipenem/cilastatin, meropenem, doripenem
- Ertapenem
- Extremely broad-spectrum antimicrobials
  - Probably the most broad-spectrum of any class of agents currently available on the market
  - Active against many gram-positive and gram-negative organisms
  - Often used for multi-drug resistant infections
Carbapenems

• Spectrum of activity:
  – Imipenem/cilastatin, meropenem, doripenem:
    • MSSA, streptococci, Enterococcus, Listeria
    • Pseudomonas and other gram-negatives, including ESBL-producing organisms, anaerobes
  – Ertapenem:
    • Similar to other carbapenems, but NO Pseudomonas or Enterococcus activity
    • Once daily dosing

• ADRs: Seizures
• Aztreonam
  – Safe to give in patients with allergies to other beta-lactams
    • Contains only the four-membered ring of the basic beta-lactam structure
  – Cross-reactivity with ceftazidime
    • Share an identical side chain
  – Only covers gram-negative organisms, including *Pseudomonas*
• Vancomycin
• MOA: inhibition of cell wall synthesis
  – Different binding site than beta-lactams
• Bactericidal, time-dependent
• Spectrum of activity: ONLY gram-positives
  – MSSA, MRSA, streptococci, Clostridium difficile, enterococci
  – Used for resistant gram-positive infections
  – Vancomycin is increasing
• **Adverse Effects (vancomycin)**
  – Ototoxicity
  – Nephrotoxicity
    • Associated with the original formulation (“Mississippi Mud”)
  – Red man syndrome
    • Histamine-mediated reaction
    • Slow infusion

• **Dosing**
  – Pharmacokinetically monitored
    • Troughs

• **Oral vancomycin**
  – Poor absorption across intestinal mucosa
  – Only used for *Clostridium difficile* infections
    • IV vancomycin does not reach high enough concentrations to eliminate
Glycopeptide

• Monitoring:
  – In general, peaks are no longer recommended to be monitored
    • No good correlation with efficacy nor toxicity
  – Best predictor of efficacy is AUC/MIC ratio
    • Difficult to measure clinically, so trough is used as a surrogate marker
  – Trough goal:
    • 10-15 mg/L
    • 15-20 mg/L for pneumonia, osteomyelitis, endocarditis, meningitis, sepsis/bacteremia (POEMS)
Cyclic Lipopeptides

• Daptomycin
• MOA: depolarizes cell membrane, leading to potassium leakage from cell
• Bactericidal, concentration-dependent
• Renal elimination and dose adjustment
• Spectrum of activity
  – Only active against gram-positive organisms, but useful for resistant infections
Cyclic Lipopeptides

• Adverse effects:
  – Muscle pain, myopathy
    • Monitor CPK level at baseline and then periodically
    • Use caution in patients on statins
  – Drug fever
• Inactivated by pulmonary surfactant
  – Cannot be used for treatment of pneumonia or any other pulmonary infections
• Used most commonly in skin/soft tissue infections and bacteremia/sepsis
• Quinupristin/dalfopristin
• MOA: protein synthesis inhibitor
• Individual agents are bacteriostatic, but combination is bactericidal (synergistic effect)
• Post-antibiotic effect, time-dependent
• Spectrum of activity:
  – Gram-positives ONLY
  – Active against *E. faecium*, NOT *E. faecalis*
Fluoroquinolones

• Ciprofloxacin, levofloxacin, moxifloxacin, delafloxacin
• MOA: inhibit DNA replication and repair through inhibition of topoisomerase II and IV
  – Unique mechanism compared to other classes
  – Active against replicating and non-replicating bacteria
• Bactericidal, concentration-dependent
• Renal dose adjustment for all but moxifloxacin
• 80-100% oral bioavailability
Fluoroquinolones

• Spectrum of activity:
  – Ciprofloxacin: gram-negatives, including *Pseudomonas*, atypicals
  – Levofloxacin: gram-positives (streptococci and MSSA) and gram-negatives, including *Pseudomonas*, and atypicals
  – Moxifloxacin: same as levo, but **WITHOUT** the *Pseudomonas* coverage
  – Delafloxacin: has additional MRSA coverage

• Widespread overuse has caused highly variable resistance patterns, so must know local susceptibilities
Fluoroquinolones

• Adverse Effects – well tolerated overall
  – GI effects
  – Headache
  – Photosensitivity
  – Hypoglycemia
  – Seizures
  – Prolongation of QT interval
  – BBW
    • Achilles tendon rupture (uncommon)
Aminoglycosides

- Gentamicin, tobramycin, amikacin
- MOA: inhibition of protein synthesis
- Bactericidal, concentration-dependent
  - Pronounced post-antibiotic effect
- Renal dose adjustments necessary
- Minimal penetrations into fat tissue, CNS
- Very narrow therapeutic index
  - Nephrotoxicity, ototoxicity
Aminoglycosides

• Spectrum of activity:
  – Gram-negatives, including *Pseudomonas*
  – Synergistic effect when used with beta-lactams against gram-positives
    • Example: ampicillin + gentamicin
  – **NO** activity against anaerobes or atypicals
• Amikacin should be reserved for infections caused by organisms resistant to gentamicin/tobramycin
Macrolides

• Clarithromycin, azithromycin, telithromycin (a ketolide)
  – Erythromycin is rarely used for antimicrobial activity anymore due to resistance
• MOA: protein synthesis inhibitor
• In general, bacteriostatic, with exceptions:
  ▪ Azithromycin is bactericidal against *S. pneumoniae*, *group A streptococci*, and *H. influenzae*
• Pharmacodynamics: difficult to classify
  – Some exhibit both time and concentration dependent activity
Macrolides

• Spectrum of activity:
  – Primary use is against respiratory pathogens
  – Atypicals (Mycoplasma pneumoniae),
  – H. influenzae,
  – Moraxella catarrhalis,
  – Helicobacter pylori,
  – Mycobacterium avium
  – Streptococcus pneumoniae
  – Poor activity: Most other pathogens
• Potent inhibitors of CYP450 enzymes
  – Exception ➔ azithromycin
• Monitor QTc prolongation
Tetracyclines

- Tetracycline, doxycycline, minocycline
- MOA: protein synthesis inhibitor
- Bacteriostatic, time-dependent
- Spectrum of activity:
  - Atypicals
  - Tick-born infections (*Rickettsia, Borrelia burgdorferi*)
  - *Plasmodium* species (malaria)
  - Staphylococci (including MRSA), *S. pneumoniae*
  - Poor activity against many GNRs, anaerobes, enterococci
• Tigecycline
• MOA: protein synthesis inhibitor
• Bacteriostatic, time-dependent, post-antibiotic effect
• Spectrum of activity:
  – Gram-positives (including MRSA and VRE)
  – Many enteric gram-negatives
    • NOT *Pseudomonas* or *Proteus*
  – Anaerobes
• Highly distributes to tissues, but does not maintain adequate concentrations in urine or blood
Tetracyclines and Glycylcyclines

• Adverse Effects
  – GI effects
  – Photosensitivity
  – Esophageal irritation
    • Tetracyclines
  – Dizziness/vertigo
    • Minocycline
  – Tooth discoloration
    • Contraindicated in pregnant women and children < 8 years of age

• Tigecycline: BBW for increase in all-cause mortality
• Clindamycin
• MOA: protein synthesis inhibitor
• Bacteriostatic, time-dependent
• Spectrum of activity:
  – Gram-positives (including MRSA), anerobes
  – No activity against gram-negatives or Enterococcus
• Also inhibits bacterial toxin production
• Prototypical agent for inducing C. difficile infections
Folate Antagonists

- Trimethoprim/sulfamethoxazole (TMP/SMX)
- MOA: inhibits the biosynthesis of folate co-factors needed for DNA and RNA synthesis
- Concentration dependent
- Pharmacodynamics: appears to display both bactericidal and bacteriostatic activity
- Elimination/dose adjustment: renal
Folate Antagonists

• Spectrum of activity:
  – *Staphylococcus aureus* (including community-associated MRSA)
  – *Stenotrophomonas maltophilia* and *Burkholderia cepacia*,
  – *Listeria*,
  – *Pneumocystis jirovecii*
  – Variable activity against enteric GNRs
• No useful activity against *Enterococcus*, anaerobes
Folate Antagonists

• Adverse Effects
  – Dermatologic
    • Rash
  – Hematologic
    • Bone marrow suppression
      – More common with prolonged therapy, but can occur at any point in therapy
  – Renal toxicity
  – Hypersensitivity
    • Steven-Johnson Syndrome
Oxazolidinones

- Linezolid, tedizolid
- MOA: protein synthesis inhibitor
- Bacteriostatic, time-dependent
  - Bactericidal against *Streptococcus* species
- Spectrum of activity
  - Only active against gram-positives, but highly useful resistant infections
    - VRE
Oxazolidinones

- 100% oral bioavailability
- Adverse Effects:
  - Bone marrow suppression
    - Usually occurs after prolonged therapy, but can occur at any time
    - Must carefully monitor CBCs
  - Peripheral neuropathy (uncommon)
- Monoamine oxidase inhibitor
  - Must use very carefully (prefer to avoid) in patients taking SSRIs due to risk of serotonin syndrome
Nitroimidazoles

- Metronidazole
- MOA: protein synthesis inhibitor
- Bactericidal, concentration-dependent
- Hepatic elimination
- Dose adjust in both severe renal and hepatic impairment
- Spectrum
  - ONLY active against obligate anaerobes, *H. pylori*
Nitroimidazoles

• Adverse effects:
  – Disulfuram-like reaction
    • Patient counseling point: Do not drink alcohol while taking this medication
  – Neurologic
    • Reversible peripheral neuropathy
  – GI intolerances

• Used most commonly for abdominal infections and Clostridium infections
Nitrofurans

- Nitrofurantoin
- MOA: multifactorial, including protein synthesis inhibition and cell wall synthesis inhibition
- Bactericidal in urine, mixed concentration and time-dependent effects
- Spectrum of activity:
  - *E. coli*, *Staphylococcus saprophyticus*, *Citrobacter*, *Klebsiella*, *Enterococcus*
  - NOT *Proteus*
- No tissue penetration outside of urinary tract
- Do not use in CrCL<30 mL/min
  - Updated in Beers Criteria in 2015
Rifamycins

- Rifampin
- MOA: interferes with bacterial RNA synthesis
- Bactericidal and bacteriostatic depending on the concentration
- Both time and concentration-dependent properties
- Elimination and dose adjustment: hepatic
- Patient counseling: will strain bodily secretions red/orange
Rifamycin

• Spectrum of activity:
  – Gram-positives (*Staphylococcus* and *Streptococcus*), *Neisseria*, *Moraxella*, *H. influenzae*, *Brucella*, *Chlamydophilia*

• In general, always use in combination with another agent due to rapid development of resistance

• Strong CYP inducer (lots of drug interactions)

• Excellent tissue/CNS penetration
• Colistin (colistimethate sodium), polymyxin B
• MOA: cationic detergent that disrupts cell membrane
• Spectrum of activity:
  • Can be used to treat carbapenemase-producing strains of gram-negative species
  • Many GNRs, including multi-drug resistant *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, and *Klebsiella pneumoniae*; *Stenotrophomonas maltophilia*
  • Poor activity: All gram-positive organisms, anaerobes, *Proteus, Providencia, Burkholderia, Serratia*, Gram-negative cocci
• Adverse effects:
  – Nephrotoxicity
    • Must monitor closely
    • Do not use with other nephrotoxic medications
  – Peripheral neuropathy

• In general, reserve for use in highly-resistant organisms when other drugs cannot be used
Antimicrobial Stewardship

• The perfect recipe for a bug to develop resistance to an antibiotic is to give a low concentration of the antibiotic over a prolonged period of time
  – In general, use upper end of dosing range
  – Do not prolong therapy longer than needed, but MUST counsel patients to finish their course of antibiotics!

• Try to use the most narrow-spectrum agent possible as quickly as possible
Antimicrobial Stewardship

• SNAP: a method for assessing appropriateness of antimicrobial therapy
  • S – Safety
    – Is the drug safe for the patient?
      • Allergies? ADRs?
  • N – Need
    – Is there a reasonable indication to give antibiotics?
  • A – Adequate
    – Is the prescribed antibiotic effective, or is likely to be effective, for the indication? Guideline recommendations?
  • P – Prudent
    – Is it the BEST choice?
• Use the SNAP approach if antimicrobial therapy has already been prescribed
• If recommending therapy, assess the same components, but in a slightly different order
  — NAPS
Case 1

• HPI: D.B. is a 65-year-old WM who presents to the ED via ambulance for severe difficulty breathing, with a 3 day history of fever, productive cough, night sweats, and chills
• PMH: DM, HTN, dyslipidemia
• Meds: Metformin, glypizide, atorvastatin, lisinopril, HCTZ
• PE: BP 87/48; HR 116; RR 28; Temp 104.5; 97% 2L
Case 1

• Chest x-ray: bilateral infiltrates
Case 1

• Using the NAPS approach, what would be the most appropriate therapy for the patient at this time?
Case 2

- J.K. is a 50-year-old male who presents to the Emergency Department for evaluation of a large, erythematous, pus-filled ulcer on his left foot
- PMH: DM, HTN
- Meds: Insulin, enalapril, amlodipine
- PE: BP 156/98, P 85, RR 22, T 101.1°F
- Allergies: NKDA
Case 2

- J.K. is initiated on piperacillin/tazobactam + vancomycin and admitted to the general wards medical service

- Utilizing the SNAP approach, assess this patient’s antimicrobial therapy.
References

QUESTIONS???