**Academic Half Day 11/30: Antibiotics**  
**Facilitator’s Guide**

**Recommended Pre-Reading**  
Johnson, JR, Russo, TA. Acute pyelonephritis in adults. N Engl J Med 2018; 378:48–59.

Leekha S, Terrell CL, Edson RS. General principles of antimicrobial therapy. Paper presented at: Mayo Clinic Proceedings 2011. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3031442/>

Stevens, D. L., Bisno, A. L., Chambers, H. F., Dellinger, E. P., Goldstein, E. J., Gorbach, S. L., Hirschmann, J. V., Kaplan, S. L., Montoya, J. G., & Wade, J. C. (2014). Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. *Clinical Infectious Diseases*, *59*(2). <https://doi.org/10.1093/cid/ciu296>

**Agenda**  
1:15 – 1:30: Theory Burst  
1:30 – 2:15: Cases 1 and 2  
2:15 – 2:30: Questions for the Expert / Break  
2:30 – 3:20: Cases 3 and 4  
3:20 – 3:30: Questions for the Expert

For each of the cases, you may refer to the following resources:  
 

Antibiotic Spectrum

**Case 1**

Mr. Sal Ulitis is a 56-year-old with congestive heart failure and chronic lower extremity edema, atrial fibrillation on warfarin, and diabetes mellitus presents to the emergency room with abrupt onset of right lower extremity pain associated with redness and swelling that evolved over a period of several hours.

He has a low-grade temperature (99.9°F) at presentation but is otherwise hemodynamically stable. His white blood cell count (WBC) is slightly elevated (10.6 cells/μL). His right lower extremity is more swollen than the left lower extremity. It is erythematous, warm, and painful to touch. There is no drainage.

1. **What is on your differential diagnosis for this patient?**

Most likely cellulitis given unilateral presentation with redness, pain, swelling, and warmth. Cellulitis results from an infection of the skin that is caused by a breech in the skin (trauma/injury, chronic edema/lymphedema, history of radiation, etc.). Erysipelas is a superficial infection involving the upper dermis and lymphatics. Cellulitis involves the dermis and subcutaneous fat. An abscess is a collection of purulence located within the dermis. Patients with cellulitis tend to have a more indolent course with development of localized symptoms over a few days. Patients with erysipelas generally have acute onset of symptoms with systemic manifestations, including fever, chills, severe malaise, and headache; these can precede onset of local inflammatory signs and symptoms by minutes to hours. In erysipelas, there is clear demarcation between involved and uninvolved tissue. Cellulitis can be either non-purulent or purulent. In this patient’s case, it most likely non-purulent cellulitis. Other things on the differential include deep vein thrombosis, contact dermatitis, viral rash, and trauma.

**Ask the learners if their differentials would change if the patient presented with bilateral lower extremity redness and swelling.** Bilateral cellulitis is rare. Bilateral, circumferential, and symmetric symptoms are more reflective of stasis dermatitis, viral rash, chronic wounds, etc. Stasis dermatitis typically presents with erythematous, scaling, and eczematous patches or plaques on chronically edematous legs. The medial ankle is most frequently and severely involved, although the skin changes may extend up to the knee and down to the foot. Pruritus is variable but, when present, results in lichenification from chronic scratching or rubbing.

1. **What are the most common pathogens?**

For non-purulent cellulitis, think Streptococcal species (Group A Strep most commonly, followed by Group B Strep and Group C Strep). Erysipelas is always Strep (particularly Group A Strep). If this patient were to have purulent cellulitis, think Staphylococcus aureus (MSSA, MRSA).

1. **What additional history would you like to obtain?**

* Risk factors for cellulitis (trauma to the extremity, history of radiation to the extremity, history of chronic edema/swelling)
* Immune status. Are they neutropenic? Immunocompromise puts the patient at risk for gram negatives and fungal infections.
* If there is trauma, when was their last tetanus vaccination?
* Penetrating foot wound “through tennis shoes?” --> Pseudomonas aeruginosa
* Any pets or bites?
  + Most bite infections are polymicrobial, with a mix of aerobic and anaerobic pathogens
  + Pasteurella is the most common pathogen in dogs and cats (followed by Streptococcus and Staphylococcus)
  + For asplenic patients with dog bite --> Capnocytophagia
  + Human bites (during a fight), think Eikenella
* Travel/Insect Bites --> more likely to be erythema migrans
* Laceration associated with water or fish tanks?
  + Fresh water: **Aeromonas**, Burkholderia, Mycobacterium fortuitum, **Pseudomonas aeruginosa**
  + Brackish water: **Aeromonas**, Chromobacterium
  + Salt water: **Erysipelothrix**, Mycobacterium fortuitum, **Mycobacterium marinum**, **Vibrio vulnificus**
* Risk factors for MRSA (for purulent cellulitis)? HIV, dialysis, long term resident in a facility, recent hospitalization or surgery (within 1-2 months), personal history of MRSA, household members with MRSA

Our patient’s main risk factor is chronic edema. He has no pets and does not recall any bites.

1. **Are you admitting this patient?**

The decision to hospitalize a patient with cellulitis is influenced by patient comorbidity, presence/absence of systemic signs of infection as well as the need for IV therapy and skilled nursing care. Patients who lack systemic symptoms, altered mental status, or hemodynamic instability do not routinely require admission. Admission should be considered if signs of more severe infection are present, there is concern for poor adherence to therapy, or if the patient has significant medical comorbidities. If you are treating as an outpatient, be sure to have good follow up to re-evaluate the infection to determine if you need to extend the course of treatment.

1. **Which antibiotics will you prescribe?**

You are targeting Streptococcal species. Oral options for non-purulent cellulitis: first generation cephalosporins (Cefadroxil, Cephalexin), penicillins (dicloxacillin), and clindamycin.

**What if the patient has a penicillin allergy listed in the chart?** Be sure to assess whether this is a tue allergy (90% of patients with a penicillin allergy from childhood were never allergic at all (i.e., listed as allergy for diarrhea). It is important to assess if this is an IgE-mediated allergy (resulting in anaphylaxis), DRESS, or desquamating rash such as SJS or TEN). Keep in mind that there is a cross-reactivity between penicillin and cephalosporins that ranges from 3-10%. If the patient has a tue allergy, you can use clindamycin. In order to determine if an allergy is real, you can refer the patient to Allergy for outpatient testing. If proven to not be allergic to penicillin, this can be removed from their allergy list.

**What if this patient was unable to take oral medications?** There are some options for IV antibiotics. Using a narrow-spectrum beta-lactam agent such as penicillin, nafcillin or oxacillin (or clindamycin in a patient with a true penicillin allergy) are reasonable options for a hospitalized patient with non-purulent cellulitis. If you are not planning to admit the patient, dalbavancin (lipoglycopeptide antibiotic) may be a good option. Dalbavancin is dosed either two doses a week apart, or one combined dose. It may also prevent unnecessary hospitalization.

**Ask your team to identify the mechanism of action of these antibiotics and whether they are bacteriostatic or bactericidal. Have them explain the difference. They will have this chart in their handout as well.**

|  |  |  |
| --- | --- | --- |
| **Antibiotic** | **-Static or -Cidal** | **Mechanism of Action** |
| Penicillins | Bactericidal | Inhibit bacterial wall synthesis (by binding to penicillin-binding proteins) |
| Cephalosporin | Bactericidal | Inhibit bacterial wall synthesis (by binding to penicillin-binding proteins) |
| Clindamycin | Bacteriostatic | Inhibit protein synthesis (by binding to 50S ribosomal subunit) |

In general, things that act on the cell wall or cell membrane are bactericidal. Most protein synthesis inhibitors are bacteriostatic. Bactericidal antibiotics kill the bacteria directly, whereas bacteriostatic antibiotics prevent bacterial growth by keeping the bacteria in the stationary phase of their life cycle. Bacteriostatic antibiotics rely on the host’s immune system to ultimately clear the infection.

1. **How long will you treat with antibiotics?**

There is only one published trial that has examined the length of treatment for cellulitis. This study found no difference in outcomes if patients were treated with antibiotics for 5 days versus 10 days. For most patients with non-purulent cellulitis (inpatient and outpatient), the duration of therapy is 5-7 days as long as there is a demonstratable response to therapy (receding erythema, improvement in pain, resolution of systemic signs or symptoms). The length of therapy should be extended if the infection has not improved in this time. It is not unusual for tissue inflammation to persist for weeks after the initial infection and in and of itself is not an indication to continue therapy. If treating as an outpatient, establish a plan for close follow-up to reassess for the need to extend duration of treatment.

**Case 2**

Ms. Ethel Coli, a 33-year-old who identifies as a woman and has type 2 diabetes mellitus, presents to the hospital for frequent urination and burning with urination. She is not experiencing fevers, chills, nausea, vomiting, back or flank pain, or vaginal discharge or pruritis. She does have some lower abdominal pain. Symptoms have been present for 2 days. She drank some cranberry juice without resolution. Vital signs are all normal.

1. **What are the most common organisms responsible for her symptoms? What additional workup do you want to obtain?**

Enteric gram negatives (E. Coli most commonly, P. mirabilis, and K. pneumoniae) and S. saprophyticus. The patient is a young woman who has symptoms consistent with acute simple cystitis (classic symptoms of frequency, dysuria, abdominal pain without signs or symptoms of pyelonephritis).

**Ask your learners what on the urinalysis indicates a urinary tract infection.** Leukocyte esterase positive on the UA indicates pyuria. Absence of pyuria lowers the likelihood of UTI. Nitrites may also be seen but only when the infection is caused by organisms that convert nitrates to nitrites (E. Coli, P. mirabilis, and K. pneumoniae). Urine cultures are helpful for patients with recurrent urinary tract infections or history of resistant urinary tract infections. This will help you identify the causative organism and identify susceptibilities. If this patient were male, you would want to get a urinalysis and urine culture as well as STI testing in at risk males.

1. **How would you treat Ms. Coli’s infection?**

* TMP-SMX for 3 days (if resistance is <20% --> check antibiogram)
* Nitrofurantoin for 5 days
* Fosfomycin for 1 dose (has a higher failure rate, usually not used for first time UTI as it is reserved for patients with resistance to other agents)
* A reminder that Fosfomycin and Nitrofurantoin do not achieve therapeutic levels in the kidneys and should not be used for pyelonephritis
* Avoid fluoroquinolones unless known resistance. These antibiotics come with a lot of side effects and there are other options available.
* Beta lactams (amoxicillin, cephalexin) are not as effective and have more side effects. Some gram negative bacteria have inducible beta-lactamases genes.

**Ask your learners how they would treat Ms. Coli if she was pregnant.**

* Nitrofurantoin is an option (except in the first trimester and at term)
* Beta lactams (Amoxicillin, Amoxicillin-Clavulanic Acid, Cephalexin)
* Fosfomycin
* TMP-SMX (except in the first trimester and at term)

**Ask your learners to identify the mechanism of action of these antibiotics and indicate whether they are bactericidal or bacteriostatic. They will have this chart in their packet. Ask your learners how they would counsel patients when prescribing Fluoroquinolones.**

|  |  |  |
| --- | --- | --- |
| **Antibiotic** | **-Static vs. -Cidal** | **Mechanism of Action** |
| TMP-SMX | Bacteriostatic individually, bactericidal together | Inhibits DNA synthesis (by interfering with the folate biosynthesis pathway) |
| Nitrofurantoin | Bactericidal | Reactive intermediates interfere with bacterial ribosomes which leads to inhibition of protein synthesis, aerobic metabolism, DNA, RNA, and cell wall synthesis |
| Fosfomycin | Bactericidal | Inhibits cell wall synthesis (by inactivating pyruvyl transferase, which is necessary for production of peptidoglycan) |
| Fluoroquinolones | Bactericidal | Inhibits DNA gyrases, required for bacterial DNA replication, transcription, repair, and recombination and by promoting breaks in the DNA |

Fluoroquinolones have many side effects, including some Black Box Warnings. The things to discuss include diarrhea (C. diff infection), potentially fatal arrhythmias (QTc prolongation), tendinopathy, aneurysms and aneurysm rupture, and neuropsychiatric side effects.

1. **What additional history would make Ms. Coli’s cystitis complicated?**

* Comorbidities: CKD, transplant, immunocompromised
* Pregnancy
* Length of Symptoms: seven or more days before seeking care
* Hospital acquired, indwelling catheter, stent, nephrostomy tube, or recent instrumentation
* Renal failure, obstruction, functional or anatomic abnormality of the urinary tract,
* History of multiple urinary tract infections

1. **What additional organisms would you need to consider covering for with complicated cystitis?**

Additional organisms include Enterococci, Staphylococci, Pseudomonas, Serratia, and Providencia. Staph bacteriuria represents staph bacteremia until proven otherwise. These are patients you would want to get a urine culture in to identify the pathogen and de-escalate antibiotics if able.

1. **How would your management change if the patient presented with dysuria, fever, right-sided flank pain, multiple episodes of vomiting?**

Systemic symptoms such as fever, chills, nausea, etc. In addition to the dysuria and right-sided flank pain are suggestive of pyelonephritis. Nitrofurantoin and Fosfomycin would not be treatment options. Your duration of treatment would also be extended for more severe infections.

* TMP-SMX for 7-10 days
* Ceftriaxone for 10 days
* Ciprofloxacin or Levofloxacin for 5-7 days
* Amoxicillin-Clavulanic Acid for 10-14 days
* If concerned for MDR, Carbapenems are ta good option (particularly if concerned about ESBL organisms).
* Can also use aminoglycoside +/- ampicillin

**Indications for hospitalization in acute pyelonephritis**   
Absolute indications for admission: persistent vomiting, suspected sepsis, urinary tract obstruction. Relative indications for admission: age > 60, anatomic urinary tract abnormalities, immunocompromised, poor follow up.

1. **Ms. Coli is admitted to the hospital for pyelonephritis. She is unable to eat anything given her extreme nausea and is vomiting up all medications. She appears very dehydrated. Her blood cultures grew pan-sensitive E. coli. How would you treat her infection?**

Ms. Coli now has Gram negative bacteremia. Duration of therapy should be determined by the clinical response of the patient in addition to the primary source and extent of infection. In most cases, the duration of antibiotic therapy is 7 to 14 days. For patients with uncomplicated Enterobacteriaceae (E coli, Klebsiella, Proteus) bacteremia who respond appropriately to antibiotic therapy (e.g., no underlying endovascular, bone, joint, or CNS infection, no uncontrolled source of infection, no major immunocompromising condition, and with clinical improvement within 48 to 72 hours), consider a 7-10 day rather than 14-day course. For pan-sensitive E. coli, you can narrow your antibiotics, to penicillins, cephalosporins, fluoroquinoles or TMP-SMX depending on patient’s history of allergies, age, discussion regrding side effects. For multi-drug resistant organisms, carbapenems and aminoglycosides can be used.

**Ask your learners to identify the mechanism of action of each antibiotic and identify if it is bactericidal or bacteriostatic. This chart is included in their handout.**

|  |  |  |
| --- | --- | --- |
| Antibiotic | -Static vs. -Cidal | Mechanism of Action |
| Carbapenems | Bactericidal | Inhibits bacterial wall synthesis (by binding to penicillin-binding proteins) |
| Aminoglycosides | Bactericidal | Inhibits protein synthesis (by binding to the bacterial 30S ribosome subunit) |

In general, for patients with bacteremia or CNS infection would like a bactericidal antibiotic.

**Case 3**

Mr. Franco Mycin is a 61-year-old male with past medical history of COPD on 2 L O2 at home, CKD (baseline creatinine 1.4), type 2 diabetes mellitus, hypertension, aortic aneurysm (stable, monitoring at this time), who presents with fever, chills, dry cough, and extreme pain and swelling in his left lower extremity. He was seen in his primary care physician’s office 2 days ago for his lower extremity pain and swelling and is worried he caught a respiratory virus at the office. His doctor diagnosed him with cellulitis at the time and sent him home on amoxicillin and doxycycline. He noted some gross drainage from the leg as well. He was supposed to follow up in a few days, but his swelling worsened, and his pain has become unbearable.

His vital signs on arrival are:  
T 102.4. HR 110. BP 104/56. RR 18. SpO2 100% on 2 L

Labs:  
WBC 15k (90% PMNs), Hgb 13, Plt 350k  
Na 140, K 3.9, Cl 95, CO2 18, BUN 25, Cr 1.9

1. **What are you concerned about in this patient?**

Differential diagnosis includes DVT, worsening purulent cellulitis, necrotizing fascitis. Necrotizing fasciitis is always a consideration in patients who present with a rapidly evolving skin/soft tissue process. Patients with necrotizing fasciitis are almost always systemically ill (often described as “out of proportion to exam findings”) at presentation due to systemic release of pro-inflammatory cytokine-inducing exotoxins. While the patient in this scenario is suffering from a significant localized soft tissue process, he is not toxic appearing which reassuring against a diagnosis of necrotizing fasciitis.

* Pain out of proportion to physical exam
* Edema, induration or pain beyond area of apparent skin involvement
* Violaceous blisters or bullae, pale or mottled skin, crepitus, skin necrosis or ecchymosis, rapid progression, failure to respond to initial antibiotics
* Systemic toxicity, multi-organ failure

**Ask your learners what organisms his previous antibiotic regimen was covering.** He was likely being covered for both Staphylococcus aureus/MRSA (doxycyline) and Streptococcal species (amoxicillin). The doxycycline would also cover Vibrio in case he was hanging out in salt water.

Remember, if the patient is on appropriate antibiotics for their condition and clinically not improving or worsening, think wrong bug, wrong drug, different diagnosis.

1. **If you are admitting this patient, what orders would you enter?**

**Lactate** indicates presence tissue/organ under-perfusion (e.g., sepsis) and/or possible tissue necrosis (Note the elevated anion gap above!!)  
**Elevated CPK** indicates presence of myonecrosis and may be elevated in necrotizing fasciitis. Also frequently elevated in Vibrio vulnificans infection  
**CRP**  
**Blood cultures**  
**Fluid resuscitation for sepsis**   
**Antibiotics:** Vancomycin, Clindamycin (for anti-toxin effect), beta-lactam with anaerobic coverage (Piperacillin-Tazobactam, Carbapenems, Ceftriaxone + Metronidazole)  
**Ortho consult (or Plastics)**  
**Consider CT if there is uncertainty about the diagnosis**

You decide to start Vancomycin. Mr. Mycin reports that the last time he had Vancomycin, he developed a red hot rash. He is concerned and is wondering if he should get that medication again given his allergy. He denies anaphylaxis or desquamating rash.

1. **What are the potential side effects? Can he get vancomycin? How do you dose and manage vancomycin?**

* Vancomycin flushing reaction: histamine related reaction during infusion, can manage with Benadryl and slowing down rate, so he should be fine
* Nephrotoxicity (based on trough levels) and ototoxicity (based on peak levels). Nephrotoxicity can be either ATN or AIN.
* Vancomycin is time and concentration dependent. We want it to stay above the MIC (minimal inhibitory concentration --> the lowest concentration of the drug that will inhibit bacterial growth)
* In the past, we have used Vancomycin troughs to help guide dosing:
  + 10-15 should be adequate for soft tissue infections
  + 15-20 for deep-seated infections and sepsis (endocarditis, osteomyelitis, meningitis).
* Now, the new Vancomycin guidelines are recommending Area Under the Curve (AUC) monitoring. UCMC is moving toward this model as well.
  + This requires multiple levels, and our pharmacy has software to calculate the curve.
* Be careful when dosing patients with underlying kidney disease or decreased creatinine clearance.

1. **If he did have an anaphylactic reaction to Vancomycin, what additional medications can you use to treat this patient for MRSA soft tissue infection?**

* First, make sure they have a true allergy
* Daptomycin – where does this not work? In the lung because it is inactivated by surfactant
* Telavancin (no different from Vanc in allergy or sensitivity), dalbavancin, ceftaroline, minocycline
* Linezolid is bacteriostatic and therefore would not be recommended in bacteremia

1. **His blood cultures grow MRSA. What additional workup do you want?**

Determination of treatment duration requires differentiation of patients with uncomplicated S aureus bacteremia from patients with complicated S. aureus bacteremia (who require longer duration of intravenous treatment). Patients with Staph aureus bacteremia should undergo the following assessment:

* Echocardiography – rule out Infective endocarditis
* Look for indwelling devices (such as central lines)
* Obtain follow-up cultures 48-72 hours after initiating intravenous anti-staphylococcal therapy are negative.
* Remove presumed focus of infection/get source control (I.e debridement)

If staph bacteremia is *uncomplicated* (I.e patient defervesced within 48 to 72 hours after initiating intravenous anti-staphylococcal therapy and removal of the presumed focus of infection), you will likely be able to treat it with a 14-day course of antibiotics. Of note, our institution has a protocol whereby EPIC will prompt you to consult infectious diseases when Staph aureus bacteremia is identified- this is based on several studies that showed improved outcomes when ID is involved in these cases.

He is started on Vancomycin, infused slowly, and administered with Benadryl. Three days later, Mr. Mycin reports his lower extremity swelling has improved, but he now is reporting a new cough and some associated shortness of breath. He is now requiring 4 L of oxygen. See the QR code for the chest xray.



1. **How will you change management with this new information?**

Mr. Mycin unfortunately has developed hospital-acquired pneumonia. Recall that the definition of hospital-acquired pneumonia is onset of symptoms > 48 hours after admission.

**Ask your learners what organisms they would think about covering.** The two main things to think about are Staphylococcus aureus, gram-negative bacilli, including Pseudomonas aeruginosa.

**MRSA coverage:** For patients with HAP who are being treated empirically and have either a risk factor for MRSA infection (ie, prior IV antibiotic use within 90 days, hospitalization in a unit where >20% of S. aureus isolates are methicillin resistant or the prevalence of MRSA is not known, or who are at high risk for mortality), recommend prescribing an antibiotic with activity against MRSA (weak recommendation, very low-quality evidence). Risk factors for mortality include need for ventilatory support due to HAP and septic shock. For empiric coverage for MRSA, recommend vancomycin or linezolid rather than an alternative antibiotic (strong recommendation, low-quality evidence).

**Pseudomonal coverage**: For patients with HAP who are being treated empirically, recommend prescribing antibiotics with activity against P. aeruginosa and other gram-negative bacilli (strong recommendation, very low-quality evidence).

For patients with HAP who are being treated empirically and have factors increasing the likelihood for Pseudomonas or other gram-negative infection (ie, prior intravenous antibiotic use within 90 days, bronchiectasis, cystic fibrosis) or a high risk for mortality, recommend prescribing antibiotics from 2 different classes with activity against P. aeruginosa (weak recommendation, very low-quality evidence). All other patients with HAP who are being treated empirically may be prescribed a single antibiotic with activity against P. aeruginosa.

For our patient, organisms to cover include MRSA given patient is in a unit with >20% *S. aureus* isolated are MRSA (he also is already being covered for this) and a single antibiotic that covers Pseudomonas.

Per 2019 IDSA guidelines, all patients empirically treated for MRSA and pseudomonas should get a sputum culture. You can also obtain a MRSA nares swab, which can be used to de-escalate antibiotics.

Mr. Mycin improves and is discharged to a facility to complete his antibiotic course and rehabilitation.

**Case 4**

Ms. Flora Quinolone is a 42-year-old with past medical history of migraines and anxiety, who presented to the hospital with abdominal pain. The pain started last night and has been constant and progressively worsening over the last 12 hours. She describes it as sharp. No diarrhea but has been nauseous since the pain began and has vomited a few times at home. She was taking acetaminophen for the pain but has not been able to keep it down. She cannot recall any fevers but has not checked her temperature. She is sexually active with 2 male partners and uses condoms inconsistently.

Vitals: T 99.1 HR 100 BP 110/78 RR 21 SpO2 98% on RA

Exam:  
General: Appears uncomfortable, but not in acute distress  
HEENT: Normocephalic, atraumatic. Pupils are equal and round. Oropharynx normal without evidence of erythema or exudate.   
Neck: Supple, thyroid normal.   
Cardiac: Normal S1 and S2, slightly tachycardic. No murmurs appreciated. No edema appreciated.  
Lungs: Normal breath sounds bilaterally. Normal work of breathing, slightly tachypneic.  
Abdomen: Slightly decreased bowel sounds. Tenderness to palpation in lower abdomen (LLQ, suprapubic). No rebound or signs of peritonitis.   
Skin: No rashes or bruises noted.   
Neurology: Moving all 4 extremities spontaneously with normal strength. Cranial nerves intact.   
Psych: Normal affect, normal speech.

1. **What is on your differential and what additional information would you ask for?**

* Gastrointestinal: Consider IBD, infectious colitis, appendicitis, ischemic colitis, diverticulitis, constipation, obstruction, cholecystitis
* Genitourinary: Cystitis, pyelonephritis, nephrolithiasis, ectopic pregnancy, fibroids, PID, ovarian torsion, tubo-ovarian abscess

In addition to the history and exam provided, you should ask about urinary and vaginal symptoms, pregnancy history, and abdominal surgery history.

* No dysuria, urinary frequency, hematuria, or urgency
* No abnormal vaginal discharge, no abnormal vaginal bleeding, no dyspareunia
* No abdominal surgeries in the past
* No history of pregnancies

You would also want to perform a pelvic exam. Normal external exam, normal vagina, no discharge or bleeding from cervix, no cervical motion tenderness. Bimanual exam without masses, some tenderness in LLQ.

**Have your learners identify labs and imaging studies they would like as well and have them justify their answers. Provide them with the results below:**

CBC: WBC 13.8 Hgb 11.7 Plt 370  
Renal: Na 142 K 3.2 Cl 98 HCO3 28 BUN 29 Cr 1.0  
Hepatic: Alk Phos 88 AST 31 ALT 26 Protein 7 Albumin 4.8 Total Bili 0.6  
UA: Normal  
HIV: Negative  
Trepia: Negative  
Gonorrhea/Chlamydia: Negative  
Wet Prep: Normal  
Pregnancy test: negative  
  
ECG: Sinus tachycardia, QTc 440  
CT abdomen/pelvis with contrast: Significant for diverticulosis and stranding around sigmoid colon, suggestive of inflammation and diverticulitis.   
Vaginal ultrasound: Normal

1. **What are the most common organisms responsible for this diagnosis?**

Ms. Quinolone has diverticulitis. Most of the intra-abdominal infections, including diverticulitis, peritonitis and cholecystitis are caused by Enterobacteriaceae species (including E. coli, P. mirabilis, K. pneumoniae etc.). Bacteroides and Enterococcus can also cause diverticulitis.

1. **Would you admit this patient?**

She should be admitted as she is unable to keep her medications down. She will need to get her antibiotics parenterally.

1. **What orders would you enter for Ms. Quinolone?**

* Antibiotics: You would want to prescribe something that covers enteric gram-negative bacilli as well as anaerobic bacteria. Options include Ciprofloxacin + Metronidazole, Cephalosporin (Cefazolin, Cefuroxime, Ceftriaxone, or Cefotaxime) + Metronidazole, or Piperacillin-Tazobactam
* Pain control: IV medications (acetaminophen, ketorolac, morphine, hydromorphone) may be required while she is unable to take oral medications, depending on the severity of her pain. Once she is able to take oral medications, can transition to acetaminophen, ibuprofen, or oxycodone.
* Nausea control: IV anti-emetics (ondansetron, prochlorperazine)
* IVF: IVF should be administered if unable to tolerate PO
* Diet: Clear liquids vs. bowel rest depending on severity of symptoms

**Ask your learners to describe the mechanism of action for the antibiotics above. They have this chart in their packet.**

|  |  |  |
| --- | --- | --- |
| **Antibiotic** | **-Static vs. -Cidal** | **Mechanism of Action** |
| Metronidazole | Bactericidal | Disrupt bacterial DNA (by binding to and damaging bacterial DNA after being reduced by anaerobic bacteria intracellularly) |
| Macrolides | Bacteriostatic | Inhibit protein synthesis (by binding to 50S ribosome subunit) |

1. **When would you order repeat imaging for this patient?**

Disease progression with or without new complications should be suspected in patients with clinical deterioration and those who fail to improve after 2-3 days of IV antibiotics. The purpose of repeat imaging is to look for new complications such as abscess or perforation that may require further intervention (surgery, percutaneous drainage, etc.)

1. **What if the patient had a positive trepia test?**

There are two types of serologic tests for syphilis. Trepia is a treponemal test that directly reacts to T. pallidum. It stays positive forever. VDRL and RPR are non-treponemal tests. They test for antibodies that are not specific for T. pallidum but are associated with the immune response to T. pallidum. The non-treponemal tests are quantitative. Non-treponemal tests can be cleared after treatment. We would first want to know if the patient was diagnosed with syphilis and treated in the past to help us interpret the results of the Trepia. If she has never been diagnosed or treated prior, then you would proceed with a non-treponemal test (RPR) to confirm and quantify. Because the Trepia will remain positive forever, if you were to test this patient for syphilis again in the future (post-treatment), you would start with the non-treponemal test. A four-fold increase in titer would indicate a new infection.