

Lyme Disease	<p>Stage 1: erythema migrans (red macule with central clearing) at site of tick bite, fever, headache, malaise</p> <p>Stage 2: early disseminated: cardiac block, multiple skin lesions, neurological lesions</p> <p>Stage 3: late disease: arthritis</p>	<p>Spirochete <i>B. burgdorferi</i>, transmitted by Ixodid tick vector</p> <p>Treat with Doxycycline (adults), Amoxicillin (kids)</p>
Toxic Shock Syndrome	<p>Erythroderma: deep red, total body sunburn. Palm/sole flaking in 1-2 wks</p>	<p><i>S. aureus</i> TSST toxin</p> <p>Treat w/IV fluids</p>
Staph Scalded Skin	<p>Diffuse erythema. Nikolsky sign (gentle friction removes skin)</p>	<p><i>S. aureus</i> exfoliative toxins A & B</p> <p>Treat w/IV fluids</p>
RMSF	<p>Rash starts on palm/soles (wrists/ankles) and spreads towards trunk. Petechial rash.</p> <p>Severe progressive disease w/ possible meningismus, encephalopathy. Pancytopenia & hyponatremia common</p>	<p><i>R. rickettsia</i> transmitted by tick vector</p> <p>Treat w/ Doxycycline even for children</p>
Kawasaki	<p>Asian child. Measles-like rash, blanches. Diffuse patchy erythematous morbilliform, mostly on trunk.</p> <p>Phase 1: fever, rash, cervical lymphadenopathy, oral redness ("strawberry tongue")</p> <p>Phase 2: subacute, fingertip desquam</p> <p>Cardiac manifestations: CA vasculitis → aneurysms → MI</p>	<p>Unknown etiology.</p> <p>Treat w/ IV immune globulin to prevent coronary aneurysms</p>
Meningococemia	<p>Non-blanching purpura/petechial rash. Abrupt onset of fever, chills, malaise, rash. Can progress to fulminant systemic disease w/MOF. Can invade CNS.</p> <p>Waterhouse-Friderichsen Syndrome.</p>	<p>Treat with Ceftriaxone or Penicillin</p> <p>Chemoprophylaxis of household contacts w/ Rifampin, Cipro, Ceftriaxone.</p>
Parvovirus B19	<p>Facial rash: erythema infectiosum (slapped cheek)</p> <p>Body rash: symmetric, reticular macular on trunk, buttocks</p> <p>Not contagious once rash develops</p> <p>Transient aplastic crisis (sickle cell)</p> <p>Chronic erythroid hypoplasia</p>	<p>Supportive treatment</p> <p>IV immune globulin for IC</p>
Clinical Meningitis	<p>Meningismus (stiff neck) demonstrated by Brudzinski's sign (flexion of neck produces flexion of hips), Kernig sign (pain on knee extension when hip flexed), Opisthotonos (arched back)</p> <p>Fever</p> <p>Altered mental status</p> <p>Photophobia</p> <p>Vomiting due to ↑ ICP</p>	<p>CSF Profiles:</p> <p>Bacterial: ↑ WBC (PMN's), ↑ protein, ↓ gluc</p> <p>Viral: ↑ WBC (lymph), ↑ protein, nrml gluc</p> <p>TB/Fungi: ↑ WBC (lymph), ↑ protein, ↓ gluc</p> <p>Treatment:</p> <p>Treat bacterial meningitis with Ceftriaxone.</p> <p>Prophylaxis for <i>N. meningitidis</i> & <i>H. influenzae</i> household contacts (Rifampin).</p>

Bacteria that cause Meningitis	<p><i>S. pneumoniae</i>: Most common overall, ↑ freq in very young, elderly. Associated w/ RT infections in 1/3, asymptomatic carriage in 2/3.</p> <p><i>N. meningitidis</i>: Most common in older children and young adults, close housing (military, college). Overwhelming sepsis (purpura fulminans).</p> <p><i>L. monocytogenes</i>: Newborns, T-cell deficient. Food-borne infection (unpasteurized milk).</p>	<p><i>H. influenzae</i>: Type b formerly most common cause of meningitis in kids, but eradicated due to Hib conjugated vaccine</p> <p><i>Strep agalactiae</i> (GBS): Neonatal meningitis associated w/maternal genital tract colonization. Elderly w/chronic disease.</p> <p><i>S. aureus</i>: Rare except post-neurosurgery or with endocarditis</p> <p>GNB: Rare except newborns or post-neurosurgery. Salmonella bacteremia in newborns.</p>
Other organisms that cause Meningitis	<p>Enterovirus, HSV-2, HIV, mumps</p> <p>Granulomatous meningitis (TB, Cryptococcus, Coccidiomycosis): subacute presentation.</p> <p>Basilar meningitis → CN palsies</p>	<p>Syphilis, Lyme disease</p> <p>Non-infectious (ibuprofen, carcinomatous, vasculitis)</p>
Encephalitis	<p>Cardinal feature: cognition disturbance. Necrosis & hemorrhage can occur.</p> <p>Priority: Identify treatable disease (treat HSV with Acyclovir).</p> <p>HSV1: Latent in trigeminal ganglion, or travels retrograde via olfactory nerves → focal encephalitis (temporal lobe).</p> <p>HSV2 (much less common than HSV1): Acquired from genital lesion at birth, hematogenous spread → diffuse encephalitis.</p>	<p>Zoonotic (Arboviruses: WNV, EEE, St. Louis, LaCrosse).</p> <p>Rabies: Bat bite → Sensory neuron infected, retrograde travel to CNS. Classic finding is hydrophobia (can't swallow due to pharyngeal spasm). Rare survival.</p> <p>JC Virus → PML</p> <p>Demyelinating process without inflammation</p>
Brain Abscess	<p>Pathogenesis: Usually <i>S. aureus</i></p> <ol style="list-style-type: none"> 1) Spread from otitis media or sinusitis 2) Hematogenous spread (<i>S. aureus</i>) with R → L cardiac shunt 3) Direct infection (trauma, surgery) 	
Spinal Epidural Abscess	<p>Hematogenous seeding of epidural space or IV discs with spread to epidural</p> <p>Back pain → radicular pain → SC impingement</p>	<p>Usually <i>S. aureus</i></p> <p>Urgent! Neurosurgical consult to prevent paralysis</p>
Bacterial Vaccines	<p>Diphtheria: Toxoid given with pertussis & tetanus, need boosters q 10 yrs</p> <p>Pertussis: Contains pertussis Ag, toxoid, FHA fimbrial protein. Does not provide great protection</p> <p>Tetanus: Tetanus toxoid, excellent vaccine</p> <p>Meningococcus: Too many serotypes. Quadrivalent polysaccharide vaccine (not for B). For asplenic, not for kids under 2. Quadrivalent Conjugated vaccine now recommended for adolescents.</p> <p>Chemoprophylaxis to close contacts.</p>	<p><i>H. influenzae</i>: Conjugated vaccine of Hib works excellently even in infants, has practically eradicated disease. Only 1 serotype.</p> <p>Pneumococcus: Over 90 serotypes, too many to eliminate completely with vaccine. Conjugate vaccine (PCV7) is routine. Polysaccharide vaccine (PS23) for patients at risk (not immunogenic for under-2).</p>

HSV	<p>HSV2: Genital. Very prevalent.</p> <p>Prodrome (localized itching before growth of vesicles) common.</p> <p>Asymptomatic shedding of HSV</p>	<p>Double-stranded linear DNA virus.</p> <p>Diagnose with DFA (Direct Fluorescence Ab) or Tzanck.</p> <p>Treat with Acyclovir</p>
Chancroid	<p>Painful ulceration, no induration, sharply demarcated. Purulent base</p> <p>Buboe: painful inguinal adenitis. Usually large and unilateral.</p>	<p><i>Haemophilus ducreyi</i></p> <p>Diagnosed by ruling out HSV/Syphilis</p> <p>Very rare</p>
Syphilis	<p>Primary syphilis: Chancre: painless ulceration, well defined, indurated. Small to moderate lymphadenopathy.</p> <p>Secondary Syphilis: Systemic dissemination of spirochete. Constitutional symptoms: malaise, sore throat, CNS involvement, glomerulonephritis, hepatitis, arthritis, etc. Maculopapular rash on palm and soles. Condylomata lata.</p> <p>Tertiary Syphilis: Benign: Gumma Neurosyphilis: tabes dorsalis CV: aortitis, aortic aneurysm</p>	<p><i>Treponema pallidum</i>. Can have transplacental transmission.</p> <p>Early syphilis < 1 year: Incubating: no symptoms Primary: Chancre, lymphadenopathy. Secondary: Systemic: "great imitator" Early Latent: up to day 365</p> <p>Late syphilis > 1 year: Late latent: Tertiary: Gumma, Neuro, CV</p> <p>Diagnosis: VDRL: false positives with pregnancy FTA-ABS: Confirmatory test</p> <p>Treatment: Penicillin. Jarisch-Herxheimer reaction: febrile reaction to penicillin</p>
Gonococcal Urethritis/Cervicitis	<p>High rate of coinfections with Chlamydia</p> <p>10% of men asymptomatic</p> <p>75% of women asymptomatic</p> <p>Male: dysuria, mucoid discharge, tender inguinal adenopathy</p> <p>Female: dysuria, purulent cervical discharge, suprapubic discomfort, red & swollen cervix</p>	<p><i>N. gonorrhoea</i></p> <p>Diagnosis: DNA probe most common these days. Urethral Gram stain: lots of WBCs with gram – diplococci inside poly's.</p>
Non-Gonococcal Urethritis	<p>Dysuria with mucoid or watery discharge</p>	<p>50% <i>Chlamydia trachomatis</i></p> <p>Diagnosis: lots of WBCs and no evidence of GC on urethral gram stain.</p>
Vaginosis	<p>Common symptoms: vaginal discharge, vulvar itching and irritation</p> <p>Bacterial vaginosis: Replacement of normal H₂O₂ producing Lactobacillus with anaerobes, <i>Gardnerella vaginalis</i>, and <i>Mycoplasma hominis</i>. Not caused by Gardnerella. Malodorous (fishy), white, noninflammatory discharge. Diagnosis: Clue cells, absence of gram positive rods (lactobacillus). ↑ pH</p>	<p>Candidiasis: most common form. Thick white discharge plastered against walls of vagina and cervix</p> <p>Trichomoniasis: "Strawberry cervix" (little petechia on cervix, starts to bleed easily. PROM with pregnancy. Malodorous, yellow-green discharge. Men asymptomatic. Diagnosis: pear-shaped organisms, ↑ pH (> 4.5).</p>

Impetigo	Very superficial, sits on dead squamous. No scarring. Between stratum corneum & granulosum Honey-colored crust	<i>S. aureus</i> (95%) or GAS Treat topically unless severe
Folliculitis, Furuncle, Carbuncle	Folliculitis: Around hair follicule Furuncle (boil): Spread of pus into subcutaneous tissue Carbuncle: Enlarged, multiple coalescent furuncles, connecting subcutaneously	<i>S. aureus</i> <i>P. aeruginosa</i> : hot tub folliculitis
Cellulitis	Infectious inflammation of skin and subcutaneous tissues. Etiology: Lymphedema, trauma, obesity Clinical: Systemic symptoms: malaise, fever, chills. Red hot skin, edema, painful.	β -hemolytic strep most common <i>S. aureus</i> less common (if wound) Treatment: pus drainage, antibiotics
Erysipelas	Cellulitis involving lymphatics \rightarrow marked edema Bright-red "boiled lobster" skin. Fever, chills, malaise commonly precede skin findings.	GAS Treatment: IV antibiotics (initially also cover staph), hospitalization, elevation to decrease edema.
<i>P. multocida</i> Cellulitis	Cat bite (dogs too) Rapid onset Red, edematous, extremely painful, thin exudate from wound	<i>Pasteurella multocida</i>
Streptococcal Gangrene	Often occurs with underlying disease (DM, AIDS, transplants) or trauma Vascular occlusion \rightarrow necrosis \rightarrow fascial spread Secondary bacteremia, shock sepsis Eschar : dark blue/black skin	GAS most common, GBS rarely Treatment: Large dose Penicillin G IV + Clindamycin, surgical debridement
Progressive Synergistic Necrotizing Fasciitis	DM: Fournier's gangrene (perineal infection) occurs spontaneously Elderly: Decubiti ulcer Vascular occlusion \rightarrow necrosis \rightarrow fascial spread Subcutaneous, fascial infection. Can lead to bacteremia, SIRS	Fecal flora (GNR, anaerobes), Strep, Enterococcus, Staph (occasionally) Crepitance on X-Ray Treatment: surgical debridement, broad spectrum antibiotics
Clostridial Myonecrosis	Deep, devitalizing injuries Spores germinate (anaerobic) \rightarrow vascular occlusion \rightarrow necrosis Deep pain, can have normal skin Can lead to bacteremia, SIRS	Many species of Clostridia Crepitance on X-Ray Treatment: surgical debridement, broad spectrum antibiotics

Osteomyelitis	<p>Can be secondary to hematogenous spread (to metaphysis of long bones in children or vertebral bodies in elderly) or contiguous to infectious penetration.</p> <p>Osteolysis via local cytokines. Infection → occlusion → avascular necrosis</p> <p>Vascular spread to periosteum → new bone formation (involucrum)</p> <p>Hematogenous spread: Constitutional symptoms, local pain & tenderness acutely, but chronic osteomyelitis doesn't have systemic complaints.</p> <p>Direct Infection: Orthopedic surgery, trauma, sternotomy post cardiac surgery. Can be due to non-healing ulcers secondary to vascular disease & neuropathy (DM).</p>	<p><i>Staph aureus</i> (mostly), Strep: usually via skin/soft tissue infection. Can be hematogenous or direct.</p> <p>Coagulase-negative Staph, MRSA: (hemat) nosocomial, IDU</p> <p>GNB: (hemat) elderly (UTI) → vertebral osteomyelitis</p> <p><i>Salmonella</i>: Sickle cell</p> <p><i>P. aeruginosa</i>: (direct) nail into foot (through sole)</p> <p>Oral flora: (direct) fist into mouth</p> <p>Diagnosis: X-Ray can't detect changes until day 10. MRI is best.</p> <p>Therapy: IV antibiotics & surgery (required for direct infection, may be necessary for hematogenously spread infection if no response to antibiotics)</p>
Pyarthrosis	<p>Acute inflammation of synovial membrane of joint. Rapid joint destruction: Necrosis of synovial cells → ↓ mucin leads to ↑ friction.</p> <p>Etiology: Hematogenous (most common), direct inoculation (trauma), or spread from osteomyelitis (infants)</p> <p>Joints: Mostly monoarticular, large joints. Local symptoms: painful, immobile joint.</p>	<p>Diagnosis: radiology normal. ↑ WBC in joint (poly's), low sugar, no crystals (rule out gout and pseudogout)</p> <p>Prompt treatment necessary! Drainage, high-dose IV antibiotics for weeks.</p> <p><i>Staph aureus</i> most common.</p> <p><i>N. gonorrhoeae</i>: sexually (very) active: Acute pustular rash, migratory polyarthralgias, monoarticular arthritis, tenosynovitis = disseminated gonococcal infection.</p> <p>IV Drug Users (IDU) can get "odd bugs in odd joints": <i>S. aureus</i> + GNB</p>
UTI's	<p>Acute cystitis: Bladder infection. Dysuria, frequency, urgency. Short treatment.</p> <p>Acute pyelonephritis: Infection in kidney or upper urinary tract. Symptoms of acute cystitis + fever, chills, flank pain. Long treatment.</p> <p>Chronic pyelonephritis: Calyceal dilation and cortical scarring from chronic bacterial infections. Rare these days.</p> <p>Acute lobar nephronia: Radiological term for focal pyelonephritis. Parenchymatous inflammation and necrosis in lobe of kidney.</p> <p>Asymptomatic bacteria in urinalysis: Treat if less than 5 yrs old to avoid potential renal scarring.</p> <p>Catheter-associated infections: Fever can be only clinical symptom. If due to candida, stop antibiotics and remove catheter.</p>	<p>Gram negative rods: Most common. <i>E. coli</i>, <i>Klebsiella pneumonia</i>, <i>Proteus mirabilis</i> in normal hosts.</p> <p>Gram positive cocci: Less common. <i>Staph saprophyticus</i> (coagulase negative) in sexually active women, <i>Enterococcus faecalis</i> after instrumentation. <i>Staph aureus</i> only with bacteremia.</p> <p>Diagnosis: Urinalysis has positive leukocyte esterase. Urine/blood cultures if systemic symptoms</p> <p>Therapy of UTI: TMP-Sulfa (3 days if uncomplicated, 14 days to 6 weeks if complicated). Image urinary tract if no response.</p>

Renal abscess: Usually as consequence of *S. aureus* bacteremia.

Xanthogranulomatous pyelonephritis: Very rare chronic infection. Localized or diffuse replacement of renal parenchyma by inflammatory cells and lipid-laden macrophages. May mimic renal cell carcinoma, TB, or abscess. Treat with surgery or long-term antibiotics.

Sterile pyuria: Look for non-bacterial STDs or TB.

Acute urethral syndrome: No active UTI, but irritative voiding symptoms.

Vaginitis: May cause symptoms similar to UTI.

Prostatitis: Acute bacterial, Chronic bacterial, Chronic nonbacterial

GAS Pharyngitis

Can lead to rheumatic fever → rheumatic heart disease

The only form of acute pharyngitis for which antibiotic therapy is definitely indicated

Severe sore throat, may have exudative pharyngitis, enlarged tender anterior cervical nodes.

No hoarseness, rhinorrhea, or cough

Group A β-hemolytic Strep

Carriers are frequent, but GAS strains in carriers lack M protein and virulence factors.

Can be difficult to diagnose because a carrier can have a viral sore throat.

Therapy: Prevent rheumatic fever! Can start therapy up to day 9 of start of symptoms (no effect on glomerulonephritis).

Penicillin is drug of choice.

Erythromycin if penicillin allergic.

Suppurative GAS sequelae

Peritonsillar abscess: "Quinsy sore throat": "**hot potato voice**"

Rare spread to cavernous sinus → cerebral vein thrombosis, meningitis

Cervical adenitis, Mastoiditis (can progress to meningitis), Pneumonia

Hematogenous → seeding bones, joints, etc

Toxic GAS sequelae

Scarlet Fever (scarlatina): Erythrogenic toxin.

Clinically: fever, headache, vomiting.

48 hours later: **diffuse erythematous sandpapery rash.**

Severe sepsis/toxic shock syndrome.

Often with rhabdomyolysis.

Clues to scarlet fever:

Pastia's line: accentuated skin folds

Circumoral pallor

Strawberry tongue: big papillae →

Raspberry tongue: red, cracked

Nonsuppurative GAS sequelae

Rheumatic Fever: Only follows pharyngitis.

Onset 1-5 weeks after pharyngitis, but Chorea may begin up to 6 months later.

Jones criteria for diagnosis:

2 major OR 1 major + 2 minors.

→ **valvular rheumatic heart disease**

Glomerulonephritis: Can follow any GAS infection. Immune complex reaction, self limited acute glomerulonephritis. Rarely progresses to chronic renal failure.

Major Jones Criteria: arthritis, carditis, chorea, erythema marginatum, subcutaneous nodules

Minor Jones Criteria: arthralgia, increased PR interval, fever, high ESR

URI = Common Cold	Nasal irritation, congestion, rhinorrhea. Usually afebrile.	Rhinoviruses (Picornaviruses: SS linear RNA) Coronaviruses
Mid respiratory tract infection	Croup = laryngotracheobronchitis: Respiratory stridor & barking seal-like cough	Parainfluenza, adenovirus, influenza Treat with single-dose dexamethasone.
Lower respiratory tract infection	Bronchiolitis in infants: Progressive cough, wheezing, tachypnea. Hyperinflation on CXR. Can be severe infection, especially if heart defect, premature, immunosuppressed.	RSV Treatment: RSV immune globulin in high-risk (not with cyanotic heart disease)
Influenza = classic viral syndrome	Symptoms of three syndromes above + fever, chills, headach, myalgia, hacking cough Complications: Pneumonia +/- bacterial superinfection CNS syndromes: Guillain-Barré Reye's syndrome in children w/aspirin	Influenza A: antigenic drifts (yearly outbreaks) & shifts (pandemics) Influenza B: more gastroenteritis Influenza A treatment: Amantadine / Rimantadine Influenza A & B treatment: Oseltamavir / Zanamivir Vaccine (3 types) exists: 70% effective
Otitis Media	Most common under age 2 Diagnosis: Requires both inflammation & fluid in middle ear. Bacterial otitis media has bulging eardrum & purulent fluid behind it. Pathogenesis: Viral URI causes Eustachian tube obstruction Suppurative complications: Mastoiditis	Viral, bacterial, or both. <i>S. pneumoniae</i> : ↑ with ↑ age <i>H. influenzae</i> (NT >> type B): ↓ with ↑ age <i>Moraxella catarrhalis</i> Therapy: Amoxicillin
Sinusitis	Facial pain, upper molar pain, fever, swelling of nearby tissues, tenderness to percussion. Suppurative sequelae often involve <i>Staph aureus</i> . Diagnosis: Maxillary sinus puncture gold standard. Sinus CT. X-Ray, opaque transillumination. Complications: Spread to skull, brain.	<i>S. pneumoniae</i> , <i>H. influenzae</i> , <i>Moraxella catarrhalis</i> Sometimes viruses involved Therapy: Amoxicillin
Pharyngitis		Nonexudative: viruses or mycoplasma Exudative: β-hemolytic strep EBV (infectious mononucleosis) Diphtheria Adenovirus (#1 in children < 3) Vincent's angina (fusospirochetes) <i>Arcanobacterium hemolyticum</i>
Epiglottitis	Rare these days (Hib vaccine). Diagnosis: cherry-red epiglottis on tongue depression. Immediate management!	<i>H. influenzae</i>

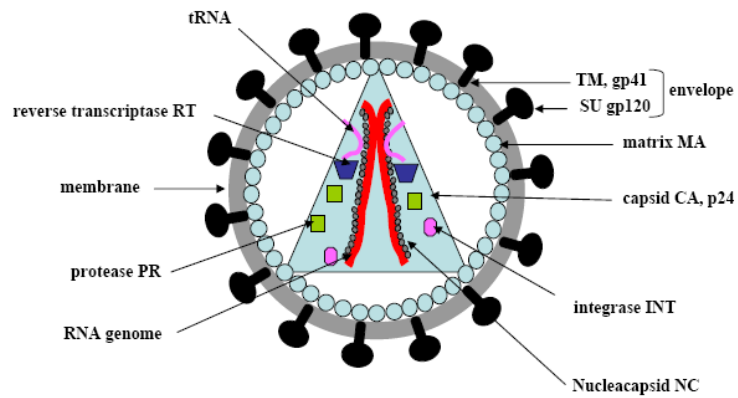
Clinical Hepatitis	<p><i>Incubation:</i> no symptoms</p> <p><i>Preicteric:</i> Initially: nonspecific malaise, fever. Later: nausea, taste change, RUQ pain. ↑ AST/ALT</p> <p><i>Icteric:</i> ~ 25% Jaundice, ↑↑ AST/ALT, ↑ bilirubin Fever uncommon</p> <p><i>Convalescence +/- Chronicity</i></p>	<p>Sometimes, you see immune complex disease in preicteric or chronic phase: PA, glomerulonephritis, etc.</p> <p>Sometimes, you see fulminant hepatitis: Liver failure with encephalopathy</p>
Hepatitis A, E	<p>Short (~4 week) incubation, no chronicity</p> <p>Serology: IgM anti-HAV/HEV</p> <p>HEV only: 40% mortality in pregnancy</p> <p>HAV only: Inactivated vaccine available. Immune-globulin can also be used for prevention.</p>	<p>HAV: Picornavirus: SS linear, unenveloped RNA</p> <p>HEV: Calcivirus: SS linear, unenveloped RNA</p> <p>Fecal-oral transmission ("vowels hit your bowels")</p>
Hepatitis B	<p>Long incubation, ~10% chronicity, but high chronicity for neonates</p> <p>Serology: IgM anti-HBc for acute infection <i>HBsAg:</i> surface Ag Appears during incubation phase. Continued presence = chronicity. <i>HBcAg:</i> core Ag <i>HBeAg:</i> core Ag, infectivity <i>IgG anti-HBs:</i> provides immunity induced by vaccination or infection <i>IgG anti-HBc:</i> persists for life <i>anti-HBe:</i> reduction in infectivity</p> <p>Treatment: Interferon-α antiretrovirals (Lamivudine, Adefovir) for fulminant infection</p> <p>Vaccine: Recombinant vaccine is envelope protein expressed in yeast.</p>	<p>HBV: DS, partially circular, enveloped DNA Not a retrovirus but has RT.</p> <p>Blood & sexual transmission.</p>
Hepatitis D	<p>Requires HBV</p> <p>Chronic infections common</p>	<p>RNA satellite virus (defective)</p>
Hepatitis C	<p>Medium incubation, high chronicity</p> <p>Serology: anti-HCV not reliable</p> <p>Treatment: Interferon-α, Ribavirin</p>	<p>Flavivirus: SS linear enveloped RNA</p> <p>Blood transmission (much less sexual).</p>
EBV Mononucleosis	<p>Classical Triad: Fever, sore throat (pharyngitis), lymphadenopathy</p> <p>Rash with ampicillin</p> <p>Neutropenia, thrombocytopenia, mild ↑ transaminases</p> <p>Positive heterophil Ab (monospot) test.</p> <p>Atypical lymphocytes (activated circulating cytotoxic T cells)</p>	<p>Herpesvirus: DS linear enveloped DNA</p> <p>Complications of acute illness (rare): splenic rupture (avoid contact sports), upper airway obstruction, Guillian-Barré, X-linked lymphoproliferative disorder</p> <p>Long term complications: Burkitt's lymphoma Nasopharyngeal carcinoma Oral hairy leukoplakia (HIV+) B cell lymphomas (HIV+, IC)</p>

Antibiotics High-Yield	<p><i>S. aureus</i>: Nafcillin, Vancomycin</p> <p><i>P. multocida</i>: Ampicillin</p> <p><i>H. influenzae</i>: Ceftriaxone, Amoxicillin</p> <p><i>P. aeruginosa</i>: NOT Ceftriaxone</p> <p><i>Clostridia</i>: Penicillin G</p> <p><i>N. gonorrhoeae</i>: Ceftriaxone</p>	<p><i>S. pneumoniae</i>: Ceftriaxone, Vancomycin</p> <p><i>S. pyogenes</i>: Penicillin G, Ceftriaxone</p> <p>GNB: 3rd gen Cephalosporin or Flouroquinolone</p> <p><i>E. coli</i>: TMP-Sulfa</p> <p><i>Rickettsia</i>: Doxycycline</p>
Penicillins	<p>Penicillin G: streptococci, enterococci</p> <p>Nafcillin: staph (rhymes with Naf)</p> <p>Ampicillin: <i>H. influenzae</i></p> <p>Piperacillin: <i>P. aeruginosa</i></p>	<p>Mechanism: inhibit cell wall synthesis. Blocks transpeptidase cross-linking of cell wall, activates autolytic enzymes.</p> <p>AE: hypersensitivity</p>
Cephalosporins	<p>1st gen: Cefazolin: staph</p> <p>2nd gen: Cefuroxime <i>H. influenzae</i></p> <p>3rd gen: Ceftriaxone: <i>H. influenzae</i>, GNB</p> <p><i>P. aer</i> 3rd gen: Ceftazidime: <i>P. aer</i>, GNB</p> <p>4th gen: Cefepime: Pseudomonas (<i>P. aer</i>)</p>	<p>Mechanism: β-lactams. Inhibit cell wall synthesis like penicillins but are less susceptible to penicillinases. Bactericidal.</p> <p>AE: hypersensitivity</p>
Other β-lactams	<p>Imipenem: Staph, GNB, <i>P. aer</i>, anaerobes (including <i>B. fragilis</i>)</p> <p>Aztreonam: Aerobic GNB, <i>H. flu</i>, <i>P. aer</i></p> <p>Ampicillin-Sulbactam: Staph, <i>H. flu</i>, <i>B. frag</i></p>	<p>Mechanism: inhibit cell wall synthesis</p> <p>Imipenem always administered with cilastatin</p>
Aminoglycosides	<p>Gentamicin: GNB, <i>P. aer</i></p> <p><i>All aminoglycosides have 4 syllable names.</i></p>	<p>Mechanism: Inhibit protein synth (30S).</p> <p>Synergistic with β-lactams.</p> <p>AE: Nephrotoxic, ototoxic.</p> <p>low therapeutic : toxic ratio</p>
Quinolones	<p>Levofloxacin: GNB, <i>P. aer</i>, Campylobacter, Legionella</p>	<p>Mechanism: DNA topoisomerase</p> <p>AE: GI toxicity, hypersensitivity, tendon rupture</p>
Macrolides	<p>Erythromycin: Campylobacter, Legionella, Mycoplasma, Chlamydia</p> <p><i>All macrolides have 5 syllable names.</i></p> <p>Note that Legionella, Mycoplasma, and Chlamydia all cause atypical pneumonia.</p>	<p>Mechanism: inhibit protein synth (50s)</p> <p>AE: nausea, vomiting.</p> <p>Contraindicated to use w/nonsedating antihistamines.</p> <p>Ototoxic with high dose.</p> <p>Reversible abnormal renal function.</p>
Tetracyclines	<p>Tetracycline: Anaerobes, Spirochetes, Rickettsia, Chlamydia, Mycoplasma</p>	<p>Mechanism: inhibit protein synth (30s)</p> <p>AE: Bone discoloration. Not for pregnancy!</p>
Sulfonamides-Trimethoprim	<p>Sulfa-TMP: staph, <i>H. flu</i>, <i>Pneumocystis carinii</i>, NOT <i>P. aer</i></p> <p>Used to treat uncomplicated UTIs</p>	<p>Sulfonamides mechanism: block synthesis of folate from PABA</p> <p>TMP mechanism: blocks reduction of folate for use in nucleotide synthesis</p> <p>AE: Hypersensitivity, rash, GI toxicity, granulocytopenia, hemolysis w/G6PD deficiency</p>

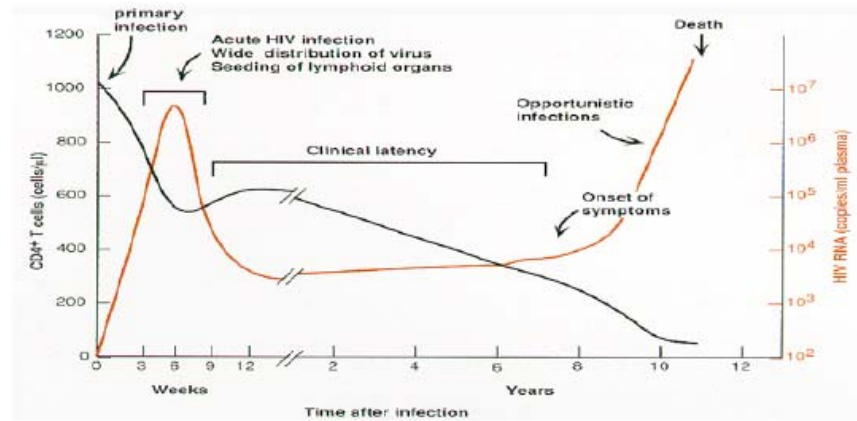
Vancomycin	Vancomycin: Most gram positives, MRSA, <i>S. pneu</i> , Enterococci	Mechanism: inhibit cell wall synthesis. (Inhibits cell wall mucopeptide formation by binding D-ala D-ala portion of cell wall precursors.) AE: Rare “ red man syndrome ” (histamine)
Clindamycin	Clindamycin: Anaerobes, <i>B. frag</i> , Staph	Mechanism: inhibits protein synthesis (50S), inhibits translocase AE: pseudomembranous colitis
Metronidazole	Metronidazole: GNB, <i>B. frag</i> , <i>C. diff</i>	Mechanism: forms toxic metabolites AE: Disulfiram-like EtOH intolerance. Inhibits metabolism of oral anticoags. CNS toxicity
Rifampin	Rifampin: Meningococci, Mycobacteria Always used in combination unless prophylaxis (single site mutation → resistance)	Mechanism: inhibits RNA polymerase AE: orange fluids, enhances clearance of other drugs
New Agents/Classes	Linezolid, Daptomycin: MRSA, vanc-resistant enterococci, gram positives	
Typical Pneumonia: Clinical	<i>Pneumococcal:</i> quick onset, single shaking chill, sudden fever, pleuritic chest pain, productive cough. Community acquired. Complications: sometimes bacteremia. <i>Staph aureus:</i> gradual onset, very severe, rapid progression. IV users or post-viral influenza. Complications: lung abscess, empyema, bacteremia, endocarditis.	<i>H. influenzae:</i> fever, productive cough. COPDers , children. <i>Mixed Anaerobic:</i> foul-smelling sputum. Altered consciousness, dysphagia. Complications: empyema , abscesses. <i>GNB:</i> acute illness with confusion, chills, sometimes bacteremic shock. Mostly nosocomial (Klebsiella if alcoholic or IC). Complication: pleural effusion.
Atypical Pneumonia: Clinical	<i>Mycoplasma:</i> Insidious onset, dry cough. College kids. <i>Viral:</i> No sputum production, like mycoplasma pneumonia.	<i>Legionella:</i> ~1 week incubation, systemic symptoms. Dry cough. No person to person spread. COPDers. <i>Chlamydia:</i> Dry cough, no sputum. Psittacosis from birds; <i>Chlamydia pneumoniae</i> in college kids.
Treatment of Pneumonias	<i>Pneumococcal:</i> Ceftriaxone, Levofloxacin. NOT penicillin (too much resistance). <i>Staph aureus:</i> Nafcillin <i>H. influenzae:</i> Ceftriaxone, TMP-Sulfa, Levo, Macrolide <i>Mixed Anaerobic:</i> Clindamycin, drain empyema <i>GNB:</i> Aminoglycoside PLUS piperacillin / ceftriaxone	<i>Mycoplasma:</i> Macrolide <i>Viral:</i> Amantadine/Rimantadine Oseltamivir/Zanamivir <i>Legionella:</i> Macrolide, Rifampin <i>Chlamydia:</i> Psittacosis: Tetracycline. <i>Chlamydia pneumoniae:</i> Tetracycline or Macrolide

Endocarditis	<p>Clinical features: Fever, murmur, normocytic anemia, leukocytosis, ↑ ESR, microscopic hematuria.</p> <p>Cutaneous manifestations: Petechiae, Osler's nodes, Janeway lesions (red nontender macules on palms/soles), purpuric skin lesions with staph ABE.</p> <p>Drug addicts: Tricuspid involvement, lung emboli</p>	<p>SBE: Mostly <i>Strep viridans</i>. <i>Strep bovis</i> (Group D) associated with colon cancer</p> <p>ABE: Mostly <i>Staph aureus</i></p> <p>Treatment: IV antibiotics 4-6 weeks</p> <p><i>S. viridans</i>: Penicillin/Ceftriaxone</p> <p><i>Enterococcus</i>: Penicillin + Gentamicin NOT Cephalosporins</p> <p><i>Staph</i>: Nafcillin/oxacillin</p> <p><i>HACEK</i> (mouth flora): Ceftriaxone</p> <p>One-time prophylaxis:</p> <p><i>S. viridans</i> (Dental work): Amoxicillin</p> <p><i>Enterococcus</i> (GI surgery): Amp + Gent</p>
Bacterial Food Poisoning	<p><i>Staph enterotoxins</i>: Short incubation, common</p> <p><i>C. perfringens</i>: Previously cooked meat, ~12 hrs incubation</p> <p><i>B. cereus</i>: From rice</p> <p><i>Neurotoxins of toxic dinoflagelates</i>: Fish & shellfish poisoning. Acute GI symptoms w/paresthesias</p>	<p>Generally, antibiotics are of no value. Treat diarrhea with fluids.</p>
Bacterial GI Infections	<p><i>Salmonella</i>: Gastroenteritis. Osteomyelitis (especially sickle cell). Typhoid (enteric) fever with <i>S. typhi</i> → splenomegaly & “rose spots” (red blanching macules on abdomen). Asymptomatic carrier possible (convalescent ceases after 6 months, or chronic carrier → gall bladder). Don't treat gastroenteritis (unless sickle cell pt). Treat Typhoid fever with TMP-Sulfa. Vaccine available</p> <p><i>Shigella dysenteriae</i>: Shiga toxin. Bloody diarrhea. Treat with fluids, TMP-Sulfa. Unlike Salmonella gastroenteritis, antibiotics eliminates organism excretion in stool</p> <p><i>Campylobacter</i>: Most common bacterial diarrhea. Can be bloody.</p> <p><i>Vibrio cholera</i>: Small intestine looks normal, but voluminous rice water stool. Treat with fluids.</p> <p><i>Vibrio parahaemolyticus</i>: Raw shellfish</p> <p><i>E. coli</i>: ETEC: Similar to Cholera toxin, Traveller's diarrhea. TMP-Sulfa. EIEC: Developing countries, rare in US. TMP-Sulfa. EHEC: O157:H7 produces bloody colitis. HUS in children. Shiga-like toxin DON'T give antibiotics: increases risk of HUS in children EPEC</p> <p><i>Clostridium difficile</i>: antibiotic (Clindamycin) associated pseudomembranous colitis</p>	
Parasitic GI Infections	<p><i>Entamoeba histolyticum</i>: Amebiasis: bloody diarrhea. Treat with Metronidazole. Find trophozoites (not cysts) in stool.</p> <p><i>Giardia lamblia</i>: Giardiasis: watery diarrhea, can last weeks. St. Petersburg. Treat with Metronidazole. Find cysts (not trophozoites) in stool.</p>	<p><i>Cryptosporidium</i>: Similar to Giardiasis, especially w/AIDS. Happened in Milwaukee. No effective drug therapy.</p> <p><i>Isospora belli</i>: AIDS patients. Treat with TMP-Sulfa.</p>
Viral GI Infections	<p>Most important cause of diarrhea in US</p> <p>Calciviruses (Norwalk): small intestinal illness. No long-lasting immunity. Explosive diarrhea & vomiting.</p>	<p>Rotaviruses: infants/young children. Top cause of diarrheal death in developing nations.</p> <p>Astroviruses: pediatric diarrhea, AIDS</p>

Inflammatory Diarrhea	<p>Bacteria:</p> <ul style="list-style-type: none"> <i>Salmonella</i> <i>Campylobacter jejuni</i> <i>Shigella</i> <i>Yersinia enterocolitica</i> <i>Clostridium difficile</i> <i>Vibrio parahemolyticus</i> EIEC, EHEC <p>Parasites:</p> <ul style="list-style-type: none"> <i>Entamoeba histolytica</i> 	<p>Localized to Colon, Ilium</p> <p>Dysenteric diarrhea</p> <p>Fecal leukocytes often present</p>
Non-inflammatory Diarrhea	<p>Viruses:</p> <ul style="list-style-type: none"> Rotavirus Norwalk Enteric Adenovirus (40, 41) Astrovirus <p>Parasites:</p> <ul style="list-style-type: none"> <i>Giardia lamblia</i> <i>Cryptosporidium</i> <p>Bacteria:</p> <ul style="list-style-type: none"> <i>Vibrio cholerae</i> 	<p>Localized to proximal small intestine</p> <p>Watery diarrhea</p> <p>Fecal leukocytes usually absent</p>
HIV Transmission & Pathogenesis	<p>HIV is always transmissible, even if plasma levels are undetectable.</p> <p>Receptors: Virus gp160 binds to CD4 PLUS chemokine coreceptor (CCR5 or CXCR4). CCR5: early disease. Changes in cell tropism during progression of infection due to switch from CCR5 to CXCR4. CXCR4 more widely expressed on T cells including naïve T cells. Deletion/mutation in human CCR5 gene confers resistance to HIV infection because CCR5 is a coreceptor that allows M-tropic virus to get into cells.</p> <p>HIV infects CD4+ cells: T-cells, Macrophages, and Dendritic cells (Langerhans cells in mucosa). gp120 binds to CD4.</p> <p>HIV Phenotypes: R5 → CCR5 coreceptor; X4 → CXCR4 coreceptor; R5X4 → CCR5 & CXCR4</p>	
Retroviruses Review	<p>LTR: Genomic regulatory region for transcription of integrated retroviruses (DNA)</p> <p>Ψ (Psi): Packaging signal, required for virion RNA to be incorporated into virus particles</p> <p>PBS: Primer binding site. tRNA binds here and primes reverse transcription</p>	
HIV Review	<p>Clade B of HIV-1 dominant form in US</p> <p>Lentiviruses are more cytolytic than other retroviruses, can lyse host cells. They often cause latent disease, and can infect non-dividing cells</p> <p><i>tat</i>: Enhances HIV expression by ↑ mRNA synthesis from proviral LTR</p> <p><i>nef</i>: Downregulates MHC I and CD4. Perturbs host cell signalling and activation. Essential for pathogenesis in vivo.</p> <p><i>vif</i>: Degrades host antiviral protein APOBEC 3G.</p> <p><i>vpr</i>: 1) Trafficking to viral core for integration. 2) Also halts cell cycle in G2.</p> <p><i>vpu</i>: 1) Downregulates CD4, allows newly synthesized envelope glycoproteins to be assembled onto budding virions. 2) Increases the release of budding virions.</p> <p><i>rev</i>: Similar to HTLV's rex: alters splicing so unspliced or singly spliced mRNA produced. When rev is expressed, production of viral particles increases</p>	



Clinical Stages of HIV Disease



Primary HIV Infection (PHI): In mucosal infection, Dendritic cells play primary role. DC-SIGN receptor is a C-type lectin receptor that binds glycoproteins, including HIV gp120. Dendritic cells transport bound HIV to regional lymph nodes and efficiently transfer HIV to CD4+ T Cells.

Acute HIV (Retroviral) Syndrome: Widespread dissemination of virus 2-3 weeks after PHI. ELISA-detectable Ab 3-7 weeks after PHI. Symptomatic “flu or mononucleosis-like” seroconversion associated with faster progression to AIDS. Can have abnormal labs (leukopenia, thrombocytopenia, ↑ LFT’s).

Gut-Associated Lymphoid Tissue (GALT) CD4+ T Cells infected.

Clinical Latency: Individual asymptomatic, but virus is replicating. Decline in CD4+ cells, reduced cellular immunity, dysregulated Ab production:

↑ IgG, IgE → SLE-like autoimmune syndromes, hypersensitivity pneumonia

Advanced Disease: HIV virus itself can cause:

Neurological disease: Dementia (infected microglia), myelopathy, peripheral neuropathy

Renal disease: Collapsing glomerulonephritis

Immune deficiency: CD4+ loss, disorganization of secondary lymphoid tissue

Host Cellular Response to HIV Determines Viral Set Point

CD8+ T cells kill infected CD4+ cells, release cytokines

HIV-specific Ab: Neutralization, ADCC

Role of long-lived cells: **Macrophages** are long-lived cells that virus infects without killing. HIV *vpr* & *nef* genes cause Macrophages to act as reservoir for viral persistence.

Memory T cells are the longest-living cells that can be infected with HIV and also act as reservoir.

These responses can modulate the viral level to determine the “set point” of viral replication during clinical latency.

Source of virus that can be measured from:

- 1) Virus from activated CD4+ cells. Can be stopped with HAART therapy.
- 2) Virus released from stable reservoirs (mem T cells and macrophages). Release continues even with HAART therapy.

Viral load = “speed of train”

CD4+ count = “length of track remaining”

Correlations of Disease with CD4 count	CD4 > 500: Bacterial sinusitis, pneumonia, HSV, TB, pyoderma CD4 200-500: Herpes zoster, Thrush, Kaposi's Sarcoma	CD4 50-200: Candida Esophagitis, PCP, Cryptosporidiosis, Histo, Extrapulmonary TB, NHL CD4 < 50: CMV, Cryptococcosis, MAC, PML, Toxo, CNS lymphoma
TB: Pathogenesis	Two-stage process. 1) Development of tuberculosis infection. Airborne infection → Bacilli produce localized pneumonia (lower lobe) & spread to hilar LN (Gohn complex) → systemic infection controlled by cell-mediated immunity → positive PPD. Initial infection usually mild or asymptomatic. TB lives in macrophages. 2) Progression to tuberculosis disease (10%). Granulomatous inflammation (fibrocaceous cavitory lesion in upper lobes). Caused by reactivation. This is the "classical" active TB.	
TB: Respiratory, Local LN Manifestations	TB pneumonia: Very infectious Laryngeal TB: Infected vocal cords, very infectious	TB pleural effusion HIV/AIDS: CXR is not diagnostic Scrofula: Painful cervical adenitis. Can also be caused by atypical TB
TB: Disseminated infection (host can't contain infection)	Miliary TB: Failure to contain either early or late dissemination. " Millet-seed appearance of granulomata on CXR. " Can lead to respiratory distress, meningitis (10%), liver, bone marrow (pancytopenia OR leukemoid reaction). "Cryptic miliary TB": disseminated TB but without millet seed pattern (Eleanor Roosevelt). AIDS pts. CNS TB: Meningitis leads to scarring: CN palsy, paralysis, confusion, w/ "aseptic meningitis." CSF: ↑ Lymphs, ↑ protein, ↓ glucose. Rare acid-fast organisms.	Genitourinary TB: Symptoms mirror local infection: "Abacteriuric" dysuria, hematuria, flank pain. Abnormal pyelogram. Can palpate mass in epididymis in males. Bone & Joints: 1) Osteomyelitis: High blood flow to metaphyses. Pott's disease: lower thoracic vertebral involvement. Can cause paravertebral abscess. 2) Arthritis. Pericarditis (rare); Intestinal TB (mimics Crohn's) Adrenal TB: Addison's , calcified adrenal.
Positive PPD	Latent TB: Positive PPD, NO evidence of active disease. If high risk, treat to prevent progression to active disease.	All pts with pos PPD receive CXR. If CXR normal AND asymptomatic → latent. Quantiferon: measure of cellular immunity, more specific than PPD.
TB Diagnosis & Treatment	Diagnosis: Sputum microscopy, AFB culture, rapid DNA probe testing, drug susceptibility testing (slow).	Treatment: 4 drug combination: Isoniazid, Rifampin, Pyrazinamide, Ethambutol / Streptomycin for 8 weeks. Rule: 4 drugs for 2 months then 2 drugs for 4 months.

Viral Vaccines	<p>MMR (<i>Measles, Mumps, Rubella</i>): live attenuated. Measles is 2 doses. Vaccine has no link to autism.</p> <p>VZV: live attenuated. 2 doses. Caution in IC, pregnant.</p> <p><i>Smallpox</i>: live virus. Local infection / adenopathy. Can have severe reaction.</p> <p><i>Influenza</i> (FluMist): live attenuated, cold adapted to restrict replication. Contraindicated for IC, pregnant, asthmatic, egg-allergic.</p>	<p>HAV: killed (formalin inactivated). FDA approved for >1 yo.</p> <p><i>Polio</i>: killed (live no longer available)</p> <p><i>Rabies</i>: killed</p> <p><i>Influenza</i> (shot): killed. trivalent “split” virus. Recommended for all pregnant women during flu season.</p> <p>HBV: HBsAg subunit. Recommended for all infants. No link to seizures, Guillian-Barré, MS, SIDS</p> <p>No causal relationship between thimerosal-containing vaccines and autism.</p>
Standard precautions	Applies to all patients: requires all blood to be handled as if infected.	
Airborne precautions	Dissemination by particles < 5 µm in size TB, Measles, Chickenpox, Smallpox	<p>Negative pressure isolation room</p> <p>Door kept closed with posted sign</p> <p>Patient can leave room only in emergency, wearing surgical mask</p>
Droplet precautions	Dissemination by particles > 5 µm in size Many, including Adenovirus, Anthrax, <i>H. flu</i> , <i>Influenza</i> , <i>N. meningitidis</i> , <i>Pertussis</i> , pneumonic Plague, GAS	<p>Private room with sign</p> <p>Door closed</p> <p>All persons entering room must wear surgical mask</p> <p>Patient must wear surgical mask when leaving room</p>
Contact Precautions	Antibiotic resistant organisms (MRSA, VRSA, VRE, ESBL+ <i>Klebsiella</i>) <i>C. diff</i> Rotavirus	<p>Private room with sign</p> <p>Gloves must be worn on entering room</p> <p>Gowns worn for direct patient or environmental contact</p> <p>Remove gowns/gloves when leaving patient room</p>
Surgical Site Infection Prevention	<p>Limit damage prior to surgery</p> <p>Admin prophylactic antibiotics 120 and 30 mins prior to surgery</p> <p>Good surgical technique</p> <p>Maintain high tissue oxygenation 2 hrs following abdominal surgery</p> <p>Tight control of hyperglycemia in SICU</p>	
Laboratory manifestations of Sepsis	<p>Leukocytosis (WBC > 12,000) with left shift (presence of bands)</p> <p>Toxic granulations, Döhle bodies</p> <p>Thrombocytopenia</p>	
Pathophysiology of Sepsis	Sepsis caused by Gram negatives (LPS, DNA), Gram positives (lipoteichoic acid, peptidoglycans, toxins), Fungi, and Viruses	LPS recognized by receptor complex: CD14, TLR4, and MD2
Organisms which cause cryptogenic sepsis	<p>Cryptogenic sepsis (child): <i>Meningococcus</i>, <i>Listeria monocytogenes</i>, <i>GBS</i></p> <p>Cryptogenic sepsis (adult): <i>Meningococcus</i>, <i>S. aureus</i></p> <p>Cryptogenic sepsis (adult IVDU): <i>S. aureus</i>, <i>GNB</i></p>	

Risks of getting diseases from blood exposure	HIV: 1/300	HCV: 1/50 HBV: ~1/25 if HBeAg(-), or ~1/4 if (+)
HIV PEP (Post-exposure prophylaxis)	Empirical data: treatment for 10 days not enough. 28 days are enough.	PEP should be offered as soon as possible, and administered for 4 weeks. Do not give Efavirenz to pregnant women.
HBV PEP	Can give HBV vaccine and HBIG (should be administered within 24 hours).	Lamivudine is effective
HBC PEP	There is none! You're out of luck.	
HIV Treatment	<p>Maximize adherence: 95% to prevent resistance</p> <p>Many P450 interactions</p> <p>Must use medications from different classis (NRTI, NNRTI, PI).</p> <p>Start and stop all medications at the same time.</p>	<p>Side effects of HAART:</p> <ul style="list-style-type: none"> *lactic acidosis *hepatotoxicity *hyperglycemia *fat maldistribution, hyperlipidemia *bleeding disorders, rash *osteoporosis
Hereditary Immunodeficiencies	<p>Leukocyte Adhesion Deficiency: Abnormality in integrin proteins. Delayed separation of umbilical cord. Impaired wound healing without pus.</p> <p>Chronic Granulomatous Disease: Diminished respiratory burst. Problems with catalase positive organisms such as <i>S. aureus</i>, <i>Serratia marcescens</i>, <i>Aspergillus fumigatus</i>.</p> <p>Bruton's Agammaglobulinemia: B cell disease, no antibody. Problems with encapsulated bacteria like <i>S. pneumo</i>, <i>H. flu</i>.</p>	
Acquired Immunodeficiencies	<p>Neutropenic (i.e. cancer chemotherapy): Susceptible to their own flora living in skin or gut. *Fever always indicates infection: treat immediately, and continue treatment until no longer neutropenic. *Early infections: bacterial; Late infections: fungal.</p> <p>Organ-transplant recipients: T-cell-Macrophage axis suppression (Graft rejection medicine). *Susceptible to viruses and intracellular bacteria (<i>Listeria</i> or <i>TB</i>), or parasites (<i>T gondii</i>). *Early infections: complications of surgery. *Middle infections: Intracellular pathogens (CMV) *Late infections: Continued susceptibility to intracellular pathogens. <i>T gondii</i>, <i>PCP</i>. <i>Listeria</i> most common cause of meningitis. Fungi (<i>Crypto</i>, <i>Histo</i>, <i>Blasto</i>, <i>Coccidio</i>)</p> <p>Bone marrow recipients: *Neutropenic phase: See above for neutropenia. May also develop severe HSV-1 due to lack of T cells. *Immunosuppressive phase. Subject to CMV 1-3 months post-transplant.</p> <p>Patients with physical abnormality/barrier.</p> <p>Cytokine inhibitors: Allows latent infections to reactivate (TB!)</p>	
Treatment of IC patients with infection	<p>Prophylactic treatment: Automatically treat before any symptoms. Treat patients on the expectation that they will develop infection if they are not treated. Example: Sulfa-TMP for PCP prophylaxis in organ transplant recipient.</p> <p>Empiric treatment: With symptoms, treat for stereotypical organisms without culture results.</p>	<p>Pre-emptive treatment: Treat on basis of laboratory finding. Example: Treat CMV with ganciclovir in bone-marrow transplant patient with high CMV PCR in serum.</p> <p>Specific therapy: Optimal approach. Treatment based on culture results.</p>

