SKIN & SOFT TISSUE INEFCTIONS

ERYSIPELAS & CELLULITIS

PATHOLOGY

ERYSIPELAS
➡ *Infection of the epidermis – upper dermis – superficial lymphatics with usual involvement of the face & lower extremities causing →*
- *Fever*
- *Brightly erythematous & tender infection with distinct elevated borders*
- *Lymphangitis & regional lymphadenopathy (obvious red streaks)*

CELLULITIS
➡ *Infection of deeper dermis & subcutaneous fat tissue with same infection presentation as erysipelas but less well demarcated area of infection*

CLASSIFICATION

BASED ON ORGANISM

PURULENT
➡ *Most commonly by Staphylococcus aureus*

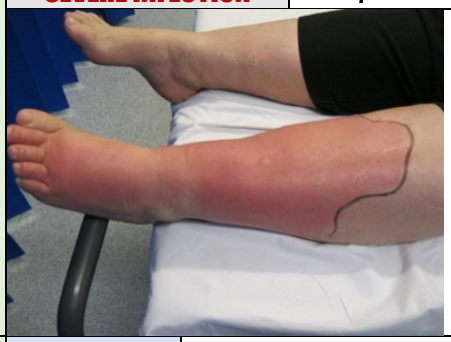
NONPURULENT
➡ *Most commonly by Streptococcus pyogenes*

BASED ON PRESENTATION

MILD INFECTION
➡ *No associated systemic signs or symptoms*

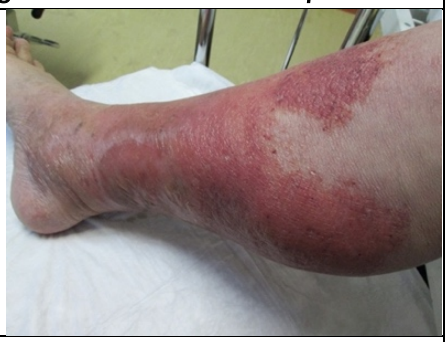
MODERATE INFECTION
➡ *Systemic signs (fever – chills – myalgias)*

SEVERE INFECTION
- *Sepsis*
- *Skin bullae or sloughing*
- *Immunocompromised*



Erysipelas
Source: Ludmilka

Left leg cellulitis
Source: John Campbell



DIAGNOSIS

BLOOD CULTURES
➡ *Only 5% positive so indicated in –*
1. *Immunocompromised*
2. *Chronic lymphedema*
3. *Severe sepsis*
4. *If necrotizing infection considered*

IMAGING
➡ *Indicated only if necrotizing infection considered*

DDx

- *Contact dermatitis*
- *Erythema nodosum*
- *Pyoderma gangrenosum*
- *Venous stasis dermatitis*
- *Lymphedema*
- *Erythromelalgia*
- *Deep venous thrombosis*
- *Lipodermatosclerosis*
- *Hypersensitivity reactions*

TREATMENT

MILD INFECTION
➡ *Empiric oral antibiotics against streptococcus for 5 days (extended duration according to the clinical picture) –*
- *Penicillin*
- *Cephalosporin*
- *Dicloxacillin*

MODERATE INFECTION
➡ *Empiric IV antibiotics –*
- *Penicillin*
- *Ceftriaxone*
- *Cefazolin*

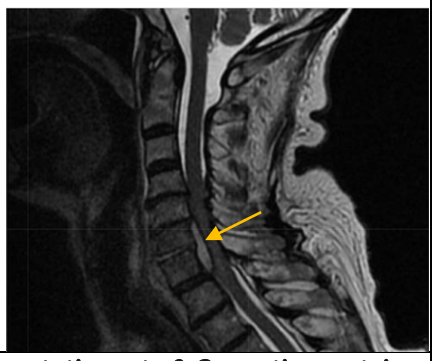
SEVERE INFECTION
➡ *Surgical consult for possible necrotizing infection + Empiric IV vancomycin + Clindamycin (inhibit toxin production) + Either –*
- *Piperacillin-tazobactam*
- *Imipenem/meropenem*

+ IV VANCOMYCIN INDICATIONS
1. *Nonpurulent cellulitis due to penetrating trauma*
2. *MRSA nasal colonization or other MRSA infection*
3. *Injection drug use*
4. *Severe infection*

↓ RECURRENCE
➡ *Treat predisposing factor –*
- *Tinea pedis*
- *Limb edema*
- *Primary skin disorders*

PREVENTION

➡ *Consider prophylactic antibiotics (penicillin) in select patients with >3 episodes of cellulitis annually*

SPINAL EPIDURAL ABSCESS		
CAUSES	• *Hematogenous dissemination (most commonly)* • *Local extension from vertebral osteomyelitis*	
ORGANISMS	**50%**	• *Staphylococcus aureus*
	OTHERS	• *Streptococcus* • *Gram-negative bacilli (Escherichia coli)*
	IF NEGATIVE CULTURE	• *Consider tuberculosis (Pott disease) & brucellosis (with travel history & risk factors)*
RISK FACTORS	1. *Bacteremia due to –* • *Endocarditis* • *Injection drug use* • *Urinary tract infection* • *Long-term IV catheters (hemodialysis catheters – central lines)* 2. *Neurosurgical procedures (spinal fusion – epidural catheter placement)* 3. *Paraspinal injection*	
PRESENTATION	➡ *Classic triad – Localized pain at the site of infection + Fever + later Radiculopathy down the spine*	
DIAGNOSIS	**MRI SPINE**	➡ *Imaging modality of choice to localize the abscess & its extension* **Cervical Epidural Abscess MRI** *Source: Alexa Bodman* 
	BLOOD WORK	➡ *Baseline labs including erythrocyte sedimentation rate & C-reactive protein*
	BLOOD CULTURES	➡ *Obtained before initiating antibiotics*
TREATMENT	**ANTIBIOTICS**	➡ *For 6 weeks (effective against staphylococcus aureus) & then based on C&S*
	SURGICAL DRAINAGE INDICATIONS	➡ *Neurologic symptoms or signs –* 1. *Lower extremity weakness/numbness* 2. *Bladder or bowel dysfunction*
	FOLLOW-UP MRI INDICATIONS	• *Persistent ↑ inflammatory markers* • *Poor clinical response* • *New neurologic symptoms or signs*
ACUTE FLACCID MYELITIS		
ORGANISM	➡ *Neuroinvasive enteroviruses (EV-A71 & EV-D68)*	
PRESENTATION	**TYPICAL**	• *Acute-onset limb weakness (often preceded by prior 4 wks of respiratory illness or fever)*
	AGE	• *Mostly is pediatric condition but reported in adults also*
	SEASON	• *Outbreaks every 2 years in late summer & early fall (since 2014)*
MANAGMENT	➡ *Initial hospitalization with close monitoring due to rapid progression with possible respiratory failure*	

DEEP INFECTIONS OF HEAD & NECK

LOCATIONS

➡ *Infection can spread from one space to another –*

1. ***Submandibular space infections*** *(include the sublingual & submylohyoid spaces)*
2. ***Lateral pharyngeal space infections***
3. ***Retropharyngeal space infections***

PATHOLOGY

➡ ***Extension of normal oral flora into deeper location (tonsillitis causing peritonsillar abscess)***

MICROBIOLOGY

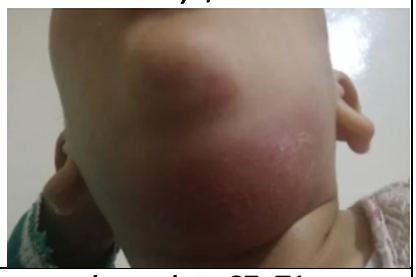
➡ ***Mixed oral flora*** *(includes oral anaerobic bacteria)*

PRESENTATION

Based on the infected space *(but more than one infection can occur at the same time)*

SUBMANDIBULAR SPACE INFECTION (LUDWIG ANGINA)

- ***Cellulitis of the floor of the mouth & submandibular neck → obstruct the airway if extends posteriorly***



Ludwig Angina
Source: Othmane

RETROPHARYNGEAL SPACE INFECTION

- ***Contiguous spread from pharyngitis – tonsillitis – other neck space → descends to C7–T1***
- ***Dysphagia & neck pain increased with hyperextension & possible stridor***

DANGEROUS EXTENSIONS

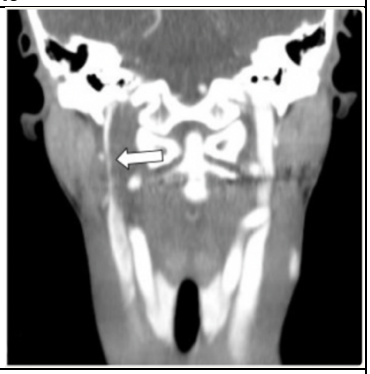
- ***Posterior to retropharyngeal space →***

1. ***The danger space → tracks to the mediastinum***
2. ***The prevertebral space → the space that travels down to the pelvis***

LATERAL PHARYNGEAL SPACE INFECTION

- ***Anterior to the sternocleidomastoid muscle →***

1. ***Carotid artery → carotid infection → infected cerebral emboli***
2. ***Jugular vein → jugular infection → infected pulmonary emboli***
3. ***Cranial nerves 9 through 12***
4. ***Lemierre's syndrome*** *(thrombosis of right JV)*



Lemierre's Syndrome
Partial Occlusion Of
Right Internal Jugular Vein
Source: Wesley Eilbert

TREATMENT

ANTIBIOTICS

➡ ***Ampicillin-sulbactam*** ***If no comorbidities***
➡ ***Broad spectrum antibiotics*** ***(with pending surgical culture) if –***

1. ***Extensive surgery***
2. ***Neoplasm***
3. ***Prior antibiotic use***

DRAINAGE

➡ ***Surgical drainage if abscess developed***

LEMIERRE'S SYNDROME

ORGANISM

- ***Fusobacterium necrophorum*** *(anaerobic GNB)*

PATHOLOGY

- ***Rare suppurative complication of pharyngitis due to local spread of infection → causing septic thrombosis of the internal jugular vein***

RISK FACTOR

- ***Usually occurs in healthy young adults with no preexisting immunosuppression***

PRESENTATION

- ***Severe pharyngitis + neck pain & no proper response to appropriate antibiotics***

DIAGNOSIS

- ***CT of the neck with IV contrast***

DIAGNOSTIC CRITERIA

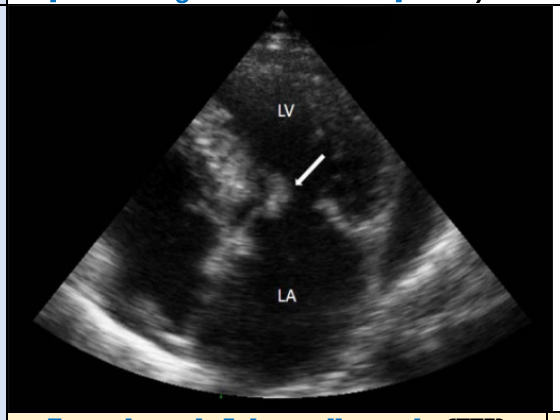
DEFINITE IE DIAGNOSIS	POSSIBLE IE DIAGNOSIS	EXCLUDED IE DIAGNOSIS
• 2 *major criteria* *or* • 1 *major* + 3 *minor criteria* *or* • 5 *minor criteria*	• 1 *major* + 1 *minor criteria* *or* • 3 *minor criteria*	➡ *If there –* 1. *Firm alternative diagnosis* 2. *No recurrence with antibiotic for <4 days* *or* 3. *No pathologic evidence of IE at surgery or at autopsy with antibiotic for ≤4 days*

DIAGNOSTIC WORK-UP

BLOOD CULTURES

➡ *Draw 3 sets before starting empiric antibiotics (positive in 90% of cases with negative results [culture-negative endocarditis] mostly due to pre-culture antibiotic use)*

IMAGING



TTE Shows Mitral Anterior Leaflet Vegetation
Source: Ahmet Guler

Transthoracic Echocardiography (TTE)	Transesophageal Echocardiography (TEE)
Initial test to detect vegetations & hemodynamic derangements	• *Used in case of intermediate/high suspicious IE cases with nondiagnostic TTE* • *Useful in prosthetic valve* • *Intracardiac device* • *Myocardial abscess (new ECG conduction defect)* • *Repeat TEE before change IV to oral antibiotics to complete left-sided IE treatment*

Cardiac CT

INDICATION: ➡ *Useful in detecting paravalvular lesions (abscess)*

Positron Emission CT with ^{18}F-fluorodeoxyglucose

INDICATION: ➡ *Identify possible IE (not meeting criteria for definite IE) of –*
- *Prosthetic valves*
- *Cardiac ICD*
- *Other prosthetic endovascular material (aortic graft)*

LABORATORY TESTS

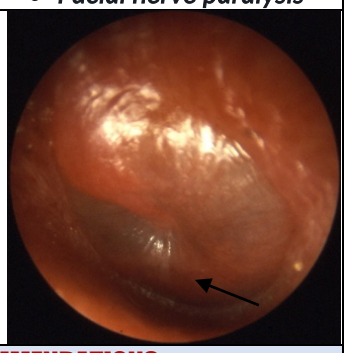
COMPLETE BLOOD COUNT	• *Anemia of chronic inflammation* • *Leukocytosis or leukopenia* • *Thrombocytopenia*
INFLAMMATORY MARKERS	➡ ↑ *Erythrocyte sedimentation rate (ESR) & C-reactive protein (CRP)*
URINE ANALYSIS	1. *Proteinuria* 2. *Hematuria* 3. *Pyuria*
IMMUNOLOGIC TESTS	➡ *Evidence of immune activation –* • *Low complement levels* • *Cryoglobulinemia* • *Positive Rheumatoid factor (RF)* • *Positive RPR test*

COLONOSCOPY

➡ *Asses for colon cancer if positive bacteremia with –*
1. *Streptococcus bovis*
2. *Clostridium septicum*

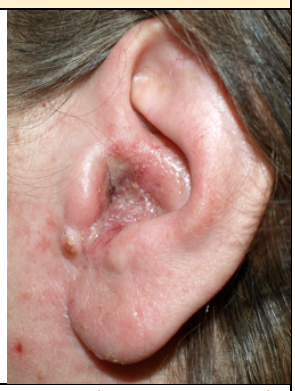
UPPER RESPIRATORY TRACT INFECTIONS (URTIs)

ACUTE OTITIS MEDIA (AOM)

MICROBIOLOGY	**MOST COMMON ORGANISM**	➡ *Streptococcus pneumoniae*
	OTHERS	➡ *Nontypeable Haemophilus influenzae & Moraxella catarrhalis*
PRESENTATION	**AGE**	• *Rare in adults but more common in children*
	TYPICAL	• *Unilateral ear pain & hearing loss +/− fever*
	COMPLICATION	➡ *Mastoiditis (rare) present as −* • *Fever* • *Posterior ear pain* • *Facial nerve paralysis*
DIAGNOSIS	• *Visualizing both bulging or intense erythematous tympanic membrane → erythema or purulent drainage + fluid in the middle ear* • *Urgent imaging & surgical consultation in case of mastoiditis*	
		AOM + Fluid *Source: B. Welleschik*
TREATMENT	**BASED ON THE CHOOSING WISELY CANADA INITIATIVE RECOMMENDATIONS**	
	UNCOMPLICATED AOM (In Adults & Children)	➡ *Observation*
	USED ANTIBIOTICS	• *Amoxicillin* • *Amoxicillin/clavulanate*
	RECURRENT AOM/PERSISTENT HEARING LOSS	➡ *Otolaryngologist referral*

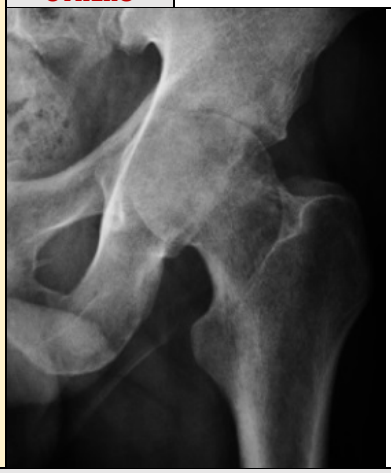
ACUTE OTITIS EXTERNA (AOE)

(SWIMMER'S EAR)

PATHOLOGY	➡ *Diffuse inflammation of the external ear canal with involvement of the pinna & tragus + rapid onset within the preceding 3 weeks*	
		AOE *Source: Klaus*
CAUSES	• *Trauma* • *Foreign bodies* • *Dermatitis* • *Moisture − esp. from swimming (swimmer's ear)*	
RISK FACTORS	1. *Immunocompromising conditions (diabetes mellitus − HIV − chemotherapy)* 2. *History of radiation* 3. *Tympanostomy tubes or perforation of the tympanic membrane*	
MICROBIOLOGY	• *Pseudomonas aeruginosa (most common)* • *Staphylococcus epidermidis & S.aureus*	
PRESENTATION	• *Otalgia* • *Pruritus of the external auditory canal* • *Ear fullness*	
EXAMINATION	➡ *Classic tenderness with pushing on the tragus or pulling on the pinna*	
TREATMENT	➡ *Fluoroquinolone ear drops (especially in case of perforated tympanic membrane) + Glucocorticoids + Antiseptics (acetic acid)* ➡ *Avoid systemic antibiotics (ineffective)*	

DIAGNOSIS

URGENT JOINT ASPIRATION Prior To Antibiotics To ↑ Yield (3C)	**APPEARANCE**	➡ *Cloudy & less vicious than noninflammatory*
	WBC COUNT	➡ *Usually >50,000/μL (often >100,000 cells/μL) mainly PMN* ➡ *Lower in –* • *Gonococcal* • *Fungal* • *Mycobacterial* • *IV drug* • *Immunocompromised*
	GRAM STAIN & CULTURE	➡ *50% positive on Gram stain & >95% on culture if no prior antibiotics not given (definite diagnosis but negative cultures does not rule out infection in high suspicious cases)*
	CRYSTAL ANALYSIS	➡ *Crystal disease (gout & pseudogout) – mimic &/or coexist with septic arthritis*
	PCR (MAIN TEST NOW)	➡ *Diagnose B. burgdorferi & mycobacteria*
PORTAL OF ENTRY CULTURE	➡ ↑ *Yield of positive culture (along synovial fluid culture)*	
BLOOD CULTURES	➡ *Prior to antibiotics to ↑ yield (but start antibiotics and do not wait for culture result to avoid irreversible joint destruction)*	
LAB WORK	➡ *Elevated WBC/CRP/ESR (but normal level does not rule out infection & can be seen also in noninfectious conditions)*	
IMAGING	**PLAIN X-RAY** – **EARLY**	• *Normal (but needed to rule out other causes or osteomyelitis)*
	PLAIN X-RAY – **ADVANCED**	• *Periosteal reaction* • *Marginal or central erosions* • *Subchondral bone destruction*
	PLAIN X-RAY – **LATE**	• *Boney ankylosis*
	OTHERS	➡ *CT/MRI can determine the extent of effusion + identify early bone changes*



Advanced Hip Septic Arthritis
Source: Ruiz Santiago

SPECIAL SCENARIOS

GOUT	➡ *Could have high leukocyte counts may occur in other conditions but treat as infectious arthritis until proven otherwise (the finding of crystals does not rule out the possibility of concomitant infection)*
N. GONORRHEA	➡ *SF WBCs may be only in 10,000 cells/ μL range* ➡ *Chlamydia can coexist (but not the joint as chlamydia does not infect the joint space)* ➡ *Frequently the cultures are negative so → culture oropharynx/GU areas (portal entry) to ↑ yield (for NAAT)*
M. MARINUM	➡ *Cultures could be negative so → culture mucosal surfaces*
MYCOBACTERIA **FUNGI** **LYME ARTHRITIS**	➡ *Indolent course + Negative Gram stain + Inflammatory synovial fluid*

OSTEOMYELITIS

PATHOLOGY
- ➡ *Either acute or chronic with necrotic bone result*

CAUSES

HEMATOGENOUS DISSEMINATION
- *Most commonly involves vertebral bodies (especially in IV drug abusers)*

CONTIGUOUS BACTERIAL SPREAD
1. *Direct contamination (fracture/joint replacement)*
2. *Wounds (pressure ulcers)*
3. *Adjacent tissue infections*

PATHOGENS

MOST COMMON
- ➡ *Staphylococcus aureus*

IV DRUG ABUSERS
- ➡ *Pseudomonas aeruginosa & Serratia → mainly in –*
- *Sternoclavicular joint*
- *Symphysis pubis*
- *Vertebrae*

SICKLE CELL DISEASE
- ➡ *Salmonella (most common)*

PRESENTATION

BONE PAIN
- *Subacute or chronic bone pain localized to the affected area*

WOUND CRITERIA
1. *Failure of the wound to heal or reopen after healing*
2. *Spontaneous wound opening + drainage (sinus tract) = chronic infection*
3. *Chronic wound (as pressure ulcer) + failure to respond to proper therapy = consider underlying osteomyelitis*

SYSTEMIC
- *Systemic manifestations (as fever) are uncommon except in acute hematogenously disseminated infection*

DIAGNOSIS

BLOOD WORK
- *↑ CRP/ESR (high likely infection with monitoring treatment response but normal result does not rule out osteomyelitis)*
- *Leukocytosis in hematogenous osteomyelitis (not in chronic one)*
- *Anemia in chronic osteomyelitis*

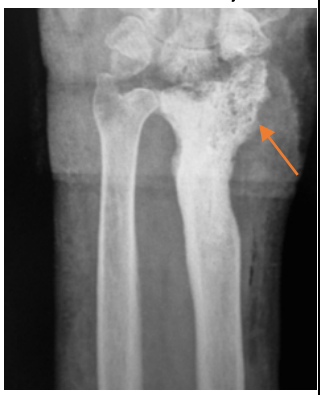
BLOOD CULTURE
- *Rarely positive but obtained especially with hematogenous osteomyelitis or with systemic manifestations of sepsis*

BONE BIOPSY
- ➡ *If MRI is suggestive of osteomyelitis → perform cultures of the bone to confirm the diagnosis & guide antibiotic therapy (by surgery or by image-guided biopsy) but generally not needed with positive blood cultures (except in IV drug use as the blood pathogen may not represent the bone pathogen)*
- *Using NAAT in bone culture-negative → can identify causative organism but does not provide antimicrobial susceptibilities results*

DIAGNOSTIC
- ➡ *Probing to bone test*

IMAGING

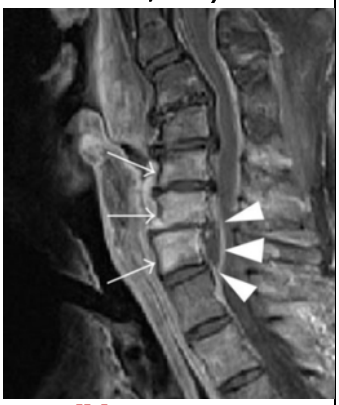
PLAIN XRAY
- *Used initially due to low cost that can confirm the diagnosis in most cases*



Chronic Osteomyelitis
- *Osteolysis*
- *Sequestra*
- *Involucrum*
- *Permeative changes*

Source: Blair York MBChB

MRI
- *MRI with/without contrast used if x-ray is non-diagnostic*



Cervical Osteomyelitis

Source: Yushi Ueki

OTHERS
- ➡ *CT with IV contrast is used if MRI cannot be obtained*
- ➡ *Do not use bone scans (very nonspecific with very high false-positive rate)*

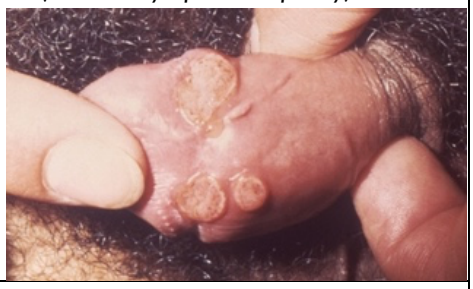
GRANULOMA INGUINALE (DONOVANOSIS)

MICROBIOLOGY

➡ ***Klebsiella granulomatis*** *(formerly – Calymmatobacterium granulomatis)* – ***gram-negative organism***

PRESENTATION

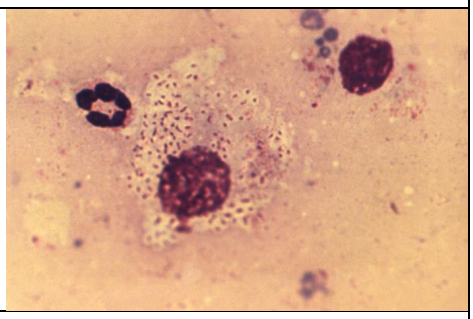
- ***Painless friable progressive beefy-red oozing genital ulcers with raised rolled margin***
- ***Spread to the inguinal area → bilateral soft tissue granulomas*** *(look like lymphadenopathy)*
  = pseudobuboes



Granuloma Inguinale
Source: CDC PHIL

DIAGNOSIS

CLINICAL DIAGNOSE	➡ *Based on presentation*
CRUSHED BIOPSY SPECIMEN	➡ *Show intracellular bacilli (Donovan bodies)*
CULTURES	➡ *Low yield*



Donovan Bodies =
WBCS CONTAINED KLEBSIELLA
Source: CDC PHIL

TREATMENT

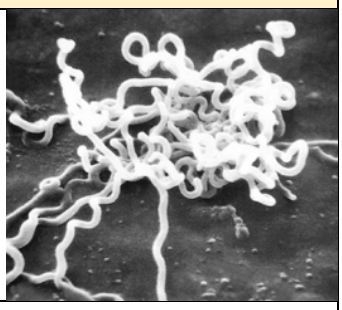
➡ ***For at least 21 days → until all the ulcers are gone –***

1. ***Azithromycin***
2. ***Doxycycline***
3. ***Ciprofloxacin***

SYPHILIS

MICROBIOLOGY

➡ ***Treponema pallidum → motile spirochete*** *(reportable disease in the U.S)*



Treponema Pallidum
Source: CDC PHIL

SCREENING INDICATIONS

1. ***All pregnant women (with every pregnancy)***
2. ***Nonpregnant adolescents***
3. ***Adults with infection risk –***

- ***MSM***
- ***Commercial sex workers***
- ***HIV infection***
- ***Any other STIs***
- ***Multiple sexual partners***
- ***New sex partner***
- ***Prior syphilis***

CLOSTRIDIOIDES DIFFICILE

The Leading Cause Of Hospital-Acquired Infectious Diarrhea

MICROBIOLOGY

- ➡ *The emerging hypervirulent strain with fluoroquinolone use causing increase of the cases in early 2020 but recent numbers showed decline in the cases*
- ➡ *C. difficile produces –*
  - *Enterotoxin (toxin A)*
  - *Cytotoxin (toxin B)*

PATHOLOGIC TYPES

1. *Asymptomatic colonization*
2. *Pathologic infection with incubation period as long as 3 months after distressing the intestinal flora with antibiotic agents*
3. *Community-acquired infections with no risk factors (exposure to health care/antibiotic agents)*

ROUTE

- ➡ *Fecal-oral transmission*

RISK FACTORS

- *Exposure to antibiotic & chemotherapeutic agents*
- *Severe underlying comorbidities*
- *Solid organ transplantation*
- *Possible gastric acid suppression with proton pump inhibitors*
- *Older age*
- *Inflammatory bowel disease*
- *Gastrointestinal surgery*

PREVENTION

1. *Antibiotic stewardship*
2. *Hand washing with soap & water (the gold standard for infection control)*
   - ➡ *Alcohol-based gels do not eliminate C. difficile spores*
3. *Contact isolation with all medical personnel entering the room should use precautions as the organism can be found on many surfaces*

ONSET

- ➡ *Symptoms can develop up to 10 weeks after the antibiotics are stopped*

PRESENTATION

- *Watery diarrhea (uncommon to be bloody)*
- *Abdominal pain/cramping*
- *Colonic distention*
- *Fever*
- *Nausea (sometimes)/malaise*

COMPLICATION

1. *Sepsis (hypotension/ileus/toxic megacolon in fulminant disease)*
2. *Acute kidney injury*

DIAGNOSIS

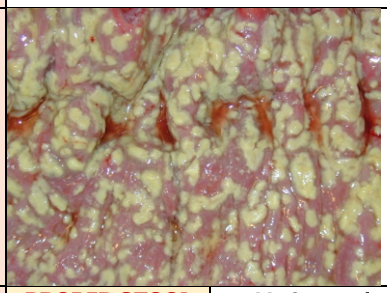
LABS (NONSPECIFIC)

- *Marked leukocytosis*
- *↑ serum creatinine level*
- *Hypoalbuminemia*

IMAGING (NONSPECIFIC)

- *Colonic wall thickening*
- *Mucosal edema*
- *Fat stranding*
- *Megacolon*

COLONSCOPY (NOT ROTINELY USED)



C. Difficile Pseudomembrane
Source: Wiki Commons

STOOL STUDIES

PROPER STOOL CRITERIA

- ➡ *Unformed stools + no laxatives use + unexplained new-onset diarrhea ≥3 times daily*

	EIA	EIA FOR GDH	NAAT
TECHNIQUE	*Enzyme immunoassay that detect presence of toxin A or B*	*EIA testing for glutamate dehydrogenase (GDH) = antigenic protein in all C. difficile*	*Nucleic acid amplification testing (NAAT) for C. difficile toxin genes*
CRITERIA	*Rapid & highly specific Poor sensitivity*	*Sensitive Poor specificity*	*Sensitive & specific*

TOXOPLASMA GONDII

PATHOLOGY
➡ *Intracellular protozoan parasite with reactivation disease with CD4 count <100 cells/μL with initial infection (asymptomatic or flu-like symptoms with fever/headache/muscle aches/tender lymphadenopathy) then resolve in weeks to months → then the parasite becomes dormant till immunocompromised state occurs*

PRESENTATION

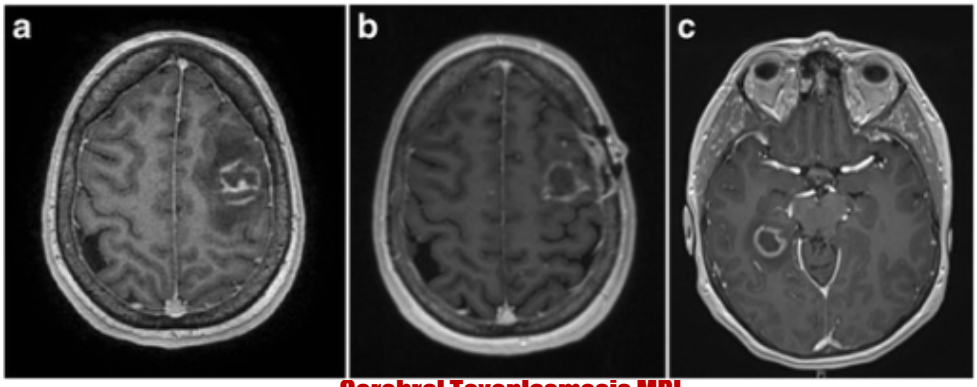
HIV
➡ *The most common cause of focal lesions in the CNS in HIV patients –*
- *Headache*
- *Fever*
- *New-onset seizures*
- *Focal neurologic deficit*
- *Altered consciousness*

CONGENITAL
➡ *Congenital toxoplasmosis even later in life (from infected mother) –*
- *Cognitive problems*
- *Blindness*
- *Seizures*

DDx
1. *Primary B-cell lymphoma (single brain lesion)*
2. *Progressive multifocal leukoencephalopathy (nonenhancing brain lesions)*
3. *Brain abscesses*

DIAGNOSIS

BRAIN MRI (More Sensitive Than Brain CT)
➡ *Multiple bilateral ring-enhancing lesions (with predilection for the basal ganglia)*



Cerebral Toxoplasmosis MRI
Source: BMC ID

SEROLOGY
➡ *IgG antibody to T. gondii (as it is reactivation disease) but not IgM Ab*

CONFIRMATION
➡ *Demonstrating radiographic improvement with empiric treatment*

BRAIN BIOPSY INDICATIONS
1. *If no radiologic improvement with treatment (= failure of treatment)*
2. *Mass effect*
3. *If only 1 lesion*

TREATMENT

MAIN REGIMEN
➡ *Pyrimethamine + Sulfadiazine + Folinic acid (leucovorin) to prevent megaloblastic anemia from pyrimethamine*

DEXAMETHASONE
➡ *Add if there is midline shift or rapid deterioration*

SULFA-ALLERGY
➡ *Clindamycin*

JOHN CUNNINGHAM (JC) POLYOMAVIRUS

PATHOLOGY
➡ *Commonly acquired asymptomatic infection → when CD4 count drops <100 cells/μL → JC virus can reactivate (human polyomavirus 2) → lytic infection in oligodendroglial cells that make myelin*

PRESENTATION
➡ *Progressive multifocal leukoencephalopathy (PML) with variable presentations due to multifocal nature of the disease →*
1. *Altered mental status*
2. *Motor & sensory changes*
3. *Specific ↓ visual acuity*

DIAGNOSIS

SUGGESTIVE
➡ *MRI → multifocal demyelinating lesions in the white matter*

CONFIRMED
➡ *CSF PCR of JC virus (70–90% sensitivity)*

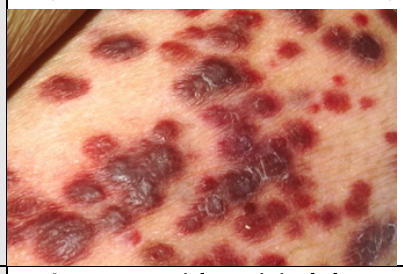
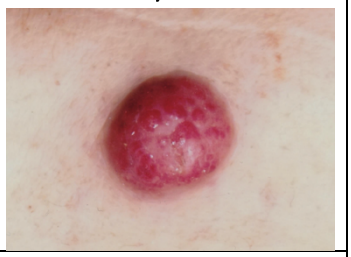
TREATMENT
➡ *No available antiviral therapy → but reconstitution of CD4 cells after ART can reverse some (but not all) symptoms*

NEUROSYPHILIS

PATHOLOGY
➡ *Syphilis (even if previously treated) → can reactivate in patients with AIDS → causing neurosyphilis*

TREATMENT
➡ *The same treatment with non-HIV patients*

➢ HIV/AIDS ASSOCIATED SKIN LESIONS

BACTERIAL INFECTION	**Bacillary Angiomatosis**		• *Due to Bartonella henselae (that cause catscratch disease)* • *Present as easily bleeding vascular purple lesions (confused as Kaposi sarcoma)* • *Treat with long several months course of antibiotics*
	Folliculitis		• *Occurs in uncontrolled HIV by S. aureus (treat with antibiotics) or eosinophilic folliculitis that is noninfectious*
VIRAL INFECTION	1. *Herpes simplex* 2. *Herpes zoster (shingles) –* • *Often refractory to treatment in HIV/AIDS but treat with acyclovir or famciclovir (not valacyclovir as it is associated with TTP in the immunocompromised patients)* 3. *Condyloma acuminatum (HPV)* 4. *Molluscum contagiosum (poxvirus)* 5. *Oral hairy leukoplakia (Epstein-Barr virus)* 6. *Kaposi sarcoma (HHV-8)* 7. *Merkel cell skin cancer (Merkel cell polyoma virus)*		
FUNGAL INFECTION	1. *Oral candidiasis (discussed above)* 2. *Folliculitis in uncontrolled HIV state due to Pityrosporum orbiculare* • *Treat with ART therapy + antibiotics* 3. *Disseminated Cryptococcus infection (AIDS-defining condition)* • *Cutaneous lesions present as umbilicated papules (resemble molluscum contagiosum) in HIV patient*		
CANCERS	**Kaposi Sarcoma**	**PATHOLOGY**	• *Vascular soft tissue malignancy due to human herpes virus type 8*
		PRESENTATION	• *<0.5-cm purple/red/violet/black maculopapular lesions on the skin or (head/neck/lower extremities) & mucous membranes (GI/lungs)*  **Kaposi Sarcoma** *Source: OpenStax*
		TREATMENT	• *Improves with antiviral therapy*
	Basal Cell Carcinoma	➡ *2–3× greater risk in HIV patient*	
	Merkel Cell Carcinoma	**PATHOLOGY**	• *Neuroendocrine tumor with 10x higher incidence in HIV patients due to Merkel cell polyoma virus*
		PRESENTATION	• *Painless fast-growing vascular-appearing intracutaneous firm nodule on sun-exposed areas*  **Merkel Cell Carcinoma** *Source: Doc103*
		TREATMENT	• *Wide surgical excision*
OTHERS	• *Xerosis (dry skin)* • *Atopic dermatitis* • *Seborrheic dermatitis (occurs in all HIV patients) improves with antiviral therapy* • *Telangiectasias*		

IMMUNE RECONSTITUTION INFLAMMATORY SYNDROME (IRIS)

PATHOLOGY	➡ *Started in few months after initiation effective ART in low CD4 cell count (<100/µL) patient*
PRESENTATION	1. *Paradoxical IRIS = worsening of pre-existing infectious pathology* 2. *Unmasking IRIS = revelation of prior unrecognized pre-existing infectious pathology* 3. *Lymphoma*
TREATMENT	➡ *Continue ART + treat IOs* ➡ *Use NSAIDs or glucocorticoids in specific cases to alleviate the inflammatory symptoms*

HIV OPPORTUNISTIC INFECTIONS (IOs)

➢ PULMONARY INFECTIONS

BACTERIAL PNEUMONIA

1st MOST COMMON PULMONARY INFECTION IN HIV PATIENTS

PATHOLOGY	➡ *The most common pulmonary infection in HIV patients especially streptococcus pneumoniae (due to successful result of primary & secondary prophylaxis against Pneumocystis) with similar presentation/treatment in non-HIV*

PNEUMOCYSTIS JIROVECII PNEUMONIA (PJP)

2nd MOST COMMON PULMONARY INFECTION IN HIV PATIENTS

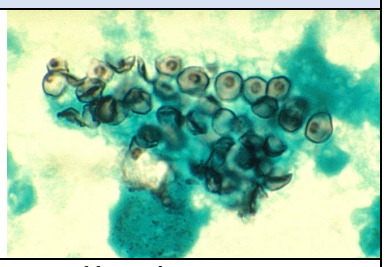
MICROBIOLOGY	➡ *PJP (PCP) – fungal infection acquired via the respiratory route*
PRESENTATION	• *Presenting disease in 50% of AIDS patients* • *Subacute (insidious) onset of fever – dyspnea – dry cough (worsens over weeks & not days) in HIV patient with CD4 count of <200 cells/µL not on prophylaxis* • *No sputum production or pleuritic pain due to low inflammatory response*

DIAGNOSIS

DIAGOSTIC

➡ *Detecting the organism by using methenamine silver stain or immunostain of pulmonary secretions either from –*

1. *Induced sputum or*
2. *Bronchoalveolar lavage (BAL) → highly sensitive due to high inoculum of PJP during active disease*



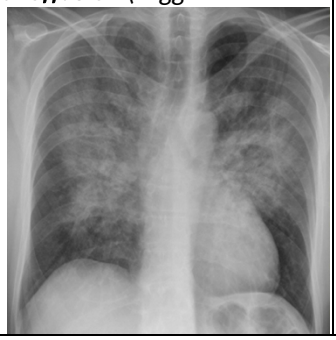
PJP From BAL
Source: Dr. Russel K.

ABG
- *Respiratory alkalosis*
- *↑ A-a gradient*
- *Hypoxia*

LABS
- *↑ lactate dehydrogenase (LDH) >400 U/L (high negative predictive value if normal)*
- *Normal liver enzymes*

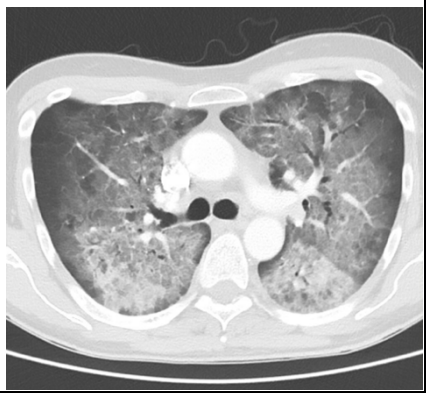
PCR
- *High sensitivity but less specific (detect colonization instead of active infection)*

CXR
- *Normal in 10–15% of patients*
- *Bilateral diffuse infiltrate (batwing)*
- *Unusal cavitation or pleural effusion (sugget other diagnosis)*



Batwing Infiltrate Of PJP
Source: Hellerhoff

CT CHEST
- *More sensitive & shows patchy ground glass opacities*



Ground-Glass Infiltrate Of PJP
Source: Hellerhoff