20.1.2 Post-neurosurgical procedure meningitis
CSF fistula:
- usually streptococci
- for immunecompromised: usual organism + Cryptococcus neoformans, M. tuberculosis, HIV aseptic meningitis, L. monocytogenes
 - empiric antifungal agents for cryptococcal meningitis:
 - induction therapy: liposomal amphotericin B 3-4mg/kg IV QD + flucytosine 25mg/kg PO QID x 2 weeks
 - consolidation therapy: fluconazole 400mg PO QD x 8 weeks
 - chronic maintenance therapy: fluconazole 200mg PO QD

- if persistent post-traumatic CSF rhinorrhea:
 - do not start antibiotics as this will super-select an organism
 - watch for two weeks
 - send 2-transferrin to confirm CSF
 - place a lumbar drain for 4-5 days
 - CT cisternogram to localize leak
 - explore with ENT

20.1.3 Post craniospinal trauma meningitis
- look for basal skull fracture and/or CSF rhinorrhea
- abx chosen based on CSF penetration and organism sensitivity [vancomycin for gram (+) and imipenem and ciprofloxacin for gram (-)]
- continue abx for 1 week after CSF is sterilized (if rhinorrhea persists, surgical repair is recommended)

20.1.4 Recurrent meningitis
- evaluate these patients for abnormal communication (intraspinal/intracranial)
- eg. Dermal sinus, CSF fistula, neurenteric cyst

20.1.5 Chronic meningitis
- tuberculosis, fungal infections, cysticercosis, neurocysticercosis
- diff dx: sarcoidosis, meningeal carcinomatosis

20.1.6 Antibiotics for specific organisms in meningitis
- S. pneumoniae: PCN G, ceftriaxone, vancomycin
- N. meningitides: PCN G, ceftriaxone, meropenem
- H. influenza: aztreonam
- Group B strep: ampicillin, vancomycin
- L. monocytogenes: ampicillin + IV gentamicin, IV Bactrim
- S. aureus: oxacillin or nafcillin, vancomycin
 - if MRSA: vancomycin + rifampin, linezolid + rifampin
- aerobic gram negative bacilli: ceftriaxone
- P. aeruginosa: cefepime, meropenem, if ventriculitis: IT gentamicin or tobramycin
- candida: liposomal amphotericin B 3-4 mg/kg IV QD

20.2 CEREBRAL ABSCESS
20.2.1 General info
- hematogenous, contiguous, direct trauma
- symptoms progress rapidly
- **Streptococcus** is most common organism
- DWI with restricted diffusion
- treatment: IV antibiotics, needle drainage for some, excision for fungal or resistant abscess

20.2.2 Epidemiology

20.2.3 Risk factors
- dental procedures
- pulmonary abscess or AV fistula
- chronic sinusitis/otitis
- congenital cyanotic heart disease (increased Hct and low PO2 provides a hypoxic environment)
- immunecompromise (transplant, HIV)
- bacterial endocarditis
- penetrating head trauma

20.2.4 Vectors
- hematogenous dissemination is the most common
- abscesses following penetrating trauma require open debridement to remove foreign material and devitalized tissue

20.2.6 Presentation
- seizures common
- symptoms progress more rapidly than with neoplasms

20.2.7 Stages of cerebral abscess
- takes two weeks to progress through maturation process
- steroids prolong it
1. Early cerebritis (days 1-3)
2. Late cerebritis (days 4-9)
3. Early capsule (days 10-13) – less developed along side facing ventricles
4. Late capsule (> day 14) – collagen capsule, necrotic center, gliosis along capsule, firm resistance to aspirating needle (“pop” on entering)

20.2.8 Evaluation
- CBC, ESR, CRP (may be normal)
- LP may show organism, but can be dangerous and lead to herniation
- due to associated risks and low yield, avoid LP
- CT and MRI brain
- Chest XR and chest CT to r/o pulmonary source
- TEE with agitated saline (bubble study): look for patent foramen ovale or cardiac vegetations

20.2.9 Treatment
- surgical: should be done if abscess is >/= 3cm in size
- correction of primary source
- IV antibiotics: 6-8 weeks with 4-8 weeks oral
 - duration guided by clinical and radiographic response
- medical treatment if duration of symptoms is < 2 weeks (suggests cerebritis stage), lesion < 3cm in size, patient shows clinical improvement within the first week of abx
- medical treatment only if: poor surgical candidate (NB: local anesthesia and stereotactic biopsy can be done in almost any pt with normal clotting factors), multiple small abscesses, poorly accessible location, concomitant meningitis, ependymitis

Indications for surgical treatment:
- mass effect
- difficulty in diagnosis
- proximity to ventricles
- evidence of elevated ICP
- poor neuro exam
- traumatic abscess associated with foreign body
- fungal abscess
- multi-loculated abscess
- f/u scans cannot be obtained every 1-2 weeks
- failure of medical management: neuro change, progression towards ventricles, enlargement, if no decrease by 4 weeks

Management:
- obtain blood cultures
- initiate antibiotic therapy
- prophylactic use of AED is optional
- steroids controversial: reduces edema, but may impede therapy
- avoid LP

Antibiotic selection:
- MRSA unlikely if no h/o trauma or neurosurgical procedure
- Start with: **Vancomycin** (15mg/kg IV Q8-12 hours to achieve a trough 15-20mg/dL + **ceftriaxone** + **metronidazole** (flagyl) 500mg Q6-8H
- alternative to cefepime + metronidazole: meropenem 2g IV Q8H
- Strep only: PCN G + ceftriaxone
- MRSA: Vancomycin
- MSSA: nafcillin 2g IV Q4H [peds: 25mg/kg IV Q6H]
- Cryptococcus, aspergillus, candida: liposomal amphotericin B 3-4mg/kg IV QD + flucytosine 25mg/kg PO QID
- AIDS patients: toxoplasma [sulfadiazine + pyrimethamine + leucovorin]
- IV antibiotics for 6-8 weeks, followed by oral antibiotics
- CT improvement may lag behind clinical improvement
- duration of treatment may be decreased if abscess and capsule excised surgically

Glucocorticoids
- reduce edema
- decrease likelihood of fibrous encapsulation
- reduce penetration of antibiotics into abscess
- immunesuppression may be deleterious

Follow-up imaging:
- decreased ring-enhancement, edema, mass effect
- takes 1-4 weeks for lesion to improve
- 95% of lesions will resolve with antibiotics alone (decrease in size by 1 month)

Cultures:
- gram stain
- acid-fast stain
- modified acid-fast (looks for Nocardia)
- aerobic and anaerobic cultures
- fungal culture
- TB culture
- molecular testing (PCR) for viral infections

Excision of abscess:
- can only be done in chronic phase
- abx treatment can be shortened to ~3 days if total excision is performed
- excision is recommended in abscesses assoc. with foreign bodies and most nocardia abscesses; may also be needed in fungal & multiloculated

20.3 SUBDURAL EMPYEMA

20.3.1 General information
- SDE is more emergent than an abscess as it has not anatomic barrier (fibrin and collagen) to prevent spread
- may be complicated by cerebral abscess and venous thrombosis

20.3.2 Epidemiology

20.3.3 Etiologies
- direct extention of local infections

20.3.4 Organisms
- most common organism: aerobic streptococcus

20.3.5 Presentation

20.3.6 Evaluation
- LP can be dangerous

20.3.7 Treatment
- emergent surgical evacuation
- burr hole if early in the course and no loculations
- craniotomy: debride and drain; do not try to remove material adherent to the cortex (may cause infarction)
- antibiotics similar to cerebral abscess
- prophylactic AED

20.3.8 Outcome

20.4 NEUROLOGIC INVOLVEMENT IN HIV/AIDS

20.4.1 Types of neurologic involvement
Focal CNS lesions in AIDS:
- toxoplasmosis
- primary CNS lymphoma (PCNSL): associated with Epstein-Barr virus
- progressive multifocal leukoencephalopathy (PML): caused by JC virus
- cryptococcal abscess
- TB (tuberculoma)

Neurosyphillis
- caused by Treponema pallidum
- CNS manifestations occur 10-20 years after infection with syphilis
- four types: asymptomatic, general paresis, meningovascular, tabes dorsalis
- serum VDRL or serum RPR (FTA-ABS to confirm diagnosis)
- treatment: PCN G 3-4 million units IV Q4H 10-14 days; alternative: rocephin 2g IV QD for 10-14 days
- f/u blood tests at 3, 6, 12, 24, and 36 months to make sure infection is gone
- f/u LP for CSF fluid analysis Q6 months

20.4.2 Neuroradiologic findings in AIDS
- MRI brain c/s contrast

20.4.3 Management of intracerebral lesions
Toxoplasmosis: pyrimethamine + sulfadiazine + leucovorin
PML: no proven effective treatment (antiretroviral therapy may help)
CNS lymphoma: usually treated with RTX

Recommendations:
- PML can usually be identified radiographically (usually multiple, non-enhancing, limited to white matter, no to minimal mass effect)
- Toxoplasmosis and PCNSL cannot be differentiated on MRI alone
- obtain toxoplasmosis serology (IgG)
- if multiple enhancing basal ganglia lesions with positive toxo titer, high probability of being toxoplasmosis
- if single enhancing lesion, PCNSL is more likely than toxoplasmosis
- if no significant mass effect, send CSF for PCR of EBV and JCV (PCNSL and PML)

20.4.4 Prognosis

20.5 LYME DISEASE – NEUROLOGIC MANIFESTATIONS

20.5.1 General information
- complex, multisystem disease
- Borrelia spirochetes
- transmitted by ticks

20.5.2 Clinical findings
Three stages (can overlap or occur separately)
1. Erythema migrans (“bulls-eye rash”) and flu-like illness
2. Cardiac and neurologic
- Classic neuro triad: cranial neuritis (mimics Bell’s palsy; can be bilateral), meningitis, radiculopathy
3. Arthritis, encephalopathy, peripheral neuropathy, ataxia, dementia

20.5.3 Diagnosis
- ELISA detects IgM or IgG; antibodies to B. burgdorferi
- oligoclonal bands and elevated IgG may occur

20.5.4
Antibiotic therapy is most effective in early stages of illness

20.6 NOCARDIA BRAIN ABSCESS
- occurs primarily in patients with chronic debilitating illnesses
- abx: Bactrim 15mg/kg IV QD in 2-4 doses + imipenem 500mg IV Q6H
- duration of treatment: at least one year with CNS involvement and possibly indefinitely in immunecompromised hosts