Objectives

- Identify the most common CNS infections in adults
- Recognize the signs and symptoms of common CNS infections
- Explain steps to diagnose and treat CNS infections
- Identify appropriate antimicrobial regimen

Disclosures: None
Overview

• Medical urgency or emergency

• Early recognition, efficient decision making and rapid initiation of treatment

• Most all CNS infections start with prodrome of HA and fever and progress to AMS or focal neurologic signs

• Seizure often present in CNS infections

• Key goals are early management and initiation of antimicrobial therapy
CNS Infections

- Identify where the infection is (meninges, brain parenchyma, subdural space, etc.)

- **Meningitis**: Infection that predominantly involves the meninges and subarachnoid space

- **Encephalitis**: brain tissue is directly injured by bacterial or viral infection

- **Cerebritis/Abscess**: Focal infection of the brain tissue

- **Ventriculitis**: Focal infection of the ventricles
Bacterial Meningitis

- **Bacterial Meningitis**: Acute purulent infection in the sub-arachnoid space

- CNS inflammation can present as:
  - Decreased LOC
  - Headache
  - Seizures
  - Elevated ICP
  - Focal neurologic deficit
  - Nuchal rigidity

- The meninges, SA space and brain parenchyma are often all involved in the inflammatory reaction (*meningoencephalitis*)
Bacterial Meningitis

• Most common form of CNS infection

• Annual incidence of >2.5 cases/100,000

• Most common organism is *Streptococcus pneumoniae* (~50%), *Neisseria meningitidis* (~25%), group B streptococci (~15%) and *Listeria monocytogenes* (~10%)

• *Haemophilus influenzae* type B accounts for <10%

• *Neisseria meningitidis* is causative organism of recurring epidemics of meningitis every 8-12 years
Bacterial Meningitis

- *Streptococcus pneumoniae:*
  - Mortality ~ 20%
  - Most common cause of meningitis in adults >20 years of age
  - Predisposing factors:
    - pneumococcal pneumonia
    - coexisting acute or chronic pneumococcal sinusitis or otitis media
    - alcoholism
    - diabetes ***
    - head trauma with basilar skull fx and CSF rhinorrhea
Bacterial Meningitis

- *Neisseria meningitidis:*
  - Incidence has decreased with routine immunization
  - Vaccine does not contain serogroup B, which is responsible for 1/3 of cases
  - Petechial or purpuric skin lesions often present
  - Can be fulminant, progressing to death within hours of symptom onset
  - Can be initiated by nasopharyngeal colonization (asymptomatic carrier or invasive disease)
    - risk of invasive disease depends on host immunity, bacterial virulence
Bacterial Meningitis

• *Listeria monocytogenes:*
  
  • neonates, pregnant women, individuals >60 yrs and immunocompromised individuals of all ages

• foods contaminated with *Listeria:*
  
  • coleslaw, milk, soft cheese, several types of “ready-to-eat” food such as deli meat and uncooked hotdogs

• Incr suspicion in farmers, especially dairy farmers and possible increasing incidence of people on RAW diets who ingest raw dairy products

• patient history is important
Bacterial Meningitis

- Otitis, mastoiditis, and sinusitis are predisposing conditions for meningitis due to *Streptococci*, other gram-negative anaerobes, *Staphylococcus aureus*, *Haemophilus* and Enterobacteriaceae

- Meningitis complicating endocarditis may be due to viridians streptococci, *S. aureus*, *Streptococci bovis*, the HACEK (*Haemophilus*, Acitintobacillus, Cardiobacterium, Eikenella, Kingella sp.), or enterococci

- Group B *Streptococcus* has been reported with increasing frequency in individuals >50 yrs, esp with underlying diseases
Bacterial Meningitis

- *Staphylococcus aureus* and Coag negative staph:

- Can cause meningitis following invasive neurosurgical procedures

  - Shunting procedures for hydrocephalus

  - Complication 2/2 subcutaneous injections via Ommaya reservoirs for administration of intrathecal chemotherapy
Pathogenesis

Nasopharyngeal colonization
↓
Bacteria attach epithelium
↓
Enter bloodstream (overcome host defenses)
↓
Penetrate CSF (lack of immune defenses)
↓
Bacteria replicate
Pathogenesis

Significant inflammation (immune response to cell wall)  
\[ \downarrow \]
Blood brain barrier weaken  
\[ \downarrow \]
Edema, neural injury and ischemia  
\[ \downarrow \]
Neurologic sequelae  
\[ \downarrow \]
Hearing loss, seizures, epilepsy, paraparesis, behavioral disorders, learning deficits, coma, and death
Pathophysiology

• Bacteria colonize in the nasopharynx

• Transported via intravascular space and enter the bloodstream

• Replicate rapidly in the CSF (no host defense)

• It is the inflammatory reaction induced by bacteria that causes neurologic manifestations

• Neurologic injury progresses after sterilization of CSF

• Lysis of bacteria leads to subarachnoid inflammation and cytokine production

• Increased CSF protein and pleocytosis

• Pro-inflammatory cytokines and other mediators can cause cell death
Pathophysiology

- **TNF-α and IL-1β** act synergistically to increase permeability of the BBB, which results in vasogenic edema and leakage of serum protein into the subarachnoid space.

- Bacteria/cell wall fragments and inflammatory cytokines can cause increased water content in brain thus brain edema and increased intracranial pressure [edema can be caused by increased permeability of BBB, toxins released or by CSF outflow resistance].

- Proteinacious and leukocyte heavy CSF obstructs the flow of CSF through ventricular system.

- Decreased reabsorption by arachnoid granulations leading to obstructive and communicating hydrocephalus.
Clinical Presentation

- Acute and rapidly progressing vs subacute and progressive
- Clinical triad: Fever, altered mental status, nuchal rigidity
- Severe headache
- Decrease LOC occurs in ~75% of patients
- N/V, photophobia are also common complaints
- Seizures on initial presentation or later during illness
  - 20-40% of patients will have seizure
  - Focal seizures usually 2/2 infarction, cortical venous thrombosis with hemorrhage or focal edema
Intracranial Pressure

- Elevated ICP is an expected complication
- Most common cause of AMS/obtundation/coma
- Can lead to herniation
- More than 90% of patients will have CSF opening pressures >18cm H20
- 20% will have CSF OP of > 40cm H20
Differential Diagnosis

- Viral meningitis (CSF profile)
- HSV encephalitis (positive MRI findings)
- Subarachnoid hemorrhage (NCHCT)
- Chemical meningitis (tumor rupture, carcinomatous meningitides—MRI)
- Rickettsial disease (tick borne, myalgias, rash)
Diagnostic Process

- Obtain blood cultures
- Initiate Dexamethasone (4mg IV q6h x 4 days)
- Initiate empiric antimicrobial therapy (all IV route)
- CT, MRI prior to LP requires clinical judgement
  - LP with incr ICP can lead to herniation (eval based on exam findings—if no s/s of elevated ICP, reasonably safe to proceed)
  - LP prior to MRI will show meningeal enhancement
- Lumbar Puncture (starting antibiotics a few hours prior to LP will not significantly alter CSF WBC, glucose or prevent visualization of bacteria on gram stain)
Lumbar Puncture

A needle is placed in the subarachnoid space at the level of the 3rd and 4th lumbar vertebra to collect a sample of CSF.
### CSF Laboratory Findings

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opening Pressure</td>
<td>&gt; 18cmH2O (180mmHg)</td>
</tr>
<tr>
<td>WBC</td>
<td>10-10,000 µL; neutrophil predominance</td>
</tr>
<tr>
<td>RBC</td>
<td>Absent if non-traumatic tap</td>
</tr>
<tr>
<td>Glucose</td>
<td>&lt; 40mg/dL</td>
</tr>
<tr>
<td>CSF/Serum Glucose</td>
<td>&lt; 0.4</td>
</tr>
<tr>
<td>Protein</td>
<td>&gt; 45mg/dL</td>
</tr>
<tr>
<td>Gram Stain</td>
<td>Positive in &gt; 60%</td>
</tr>
<tr>
<td>Culture</td>
<td>Positive in &gt; 80%</td>
</tr>
<tr>
<td>Appearance</td>
<td>Turbid</td>
</tr>
</tbody>
</table>
CSF/Glucose Ratio

- Corrects for hyperglycemia that may mask a relative decrease in CSF glucose
- Low when CSF/glucose ratio is <0.6
- CSF/glucose ratio <0.4 is highly suggestive of bacterial meningitis
- CSF/glucose ratios <0.4 can also be seen in fungal meningitis, carcinomatous meningitis, and tuberculosis
- Ex: CSF glucose 65/ Serum glucose 325 = 0.2
Diagnostic Process

- Positive CSF
- Non-contract CT: normal to abnormal (abscess or generalized cerebral edema/elevated ICP)
- MRI: Meningeal enhancement, localized or generalized edema
Meningeal enhancement MRI

Karthik, SN et al.
Cerebritis and Brain Abscess

- Similar presentation as bacterial meningitis (HA, fever, AMS)

- NCHCT: localized cerebral edema and possible ring-enhancing lesion with surrounding edema

- MRI w/ contrast: Diffusion restricting lesion, contrast enhancing ring-enhancing lesion, surrounding cerebral edema

- May or may not have positive CSF

- High level of suspicion (sinusitis, mastoiditis, post-op neurosurgery)
Treatment of elevated ICP

- Elevation of HOB
- Intubation
- Hyperventilation
- Mannitol
- EVD/bolt
- Sedation
- Fever control
Treatment bacterial meningitis

- Medical emergency

- Door to needle for IV antibiotics should be 30 mins even in no LP

- Do not delay antibiotic administration for lumbar puncture

- NCHCT to r/u SAH and high ICP prior to LP

- Lumbar puncture (cell count w/ differential, gram stain and culture, protein, glucose, consider HSV)
Drug Penetration

Blood brain barrier / blood-CSF barrier (BBB)
Drug penetration into the CSF is limited when the BBB is intact because vesicular transport across cells is minimal and endothelial cells of cerebral microvasculature have tight junctions
With meningitis, these tight junctions separate and there is an increase in vascular transport and the increased permeability allows greater penetration of antibiotic into CSF

CNS penetration
Inflamed meninges
Low molecular weight
Lipid soluble
Low protein binding
Antimicrobials in bacterial meningitis

- *S. pneumoniae and N. meningitidis* are most common etiologic organisms

- Emergence of PCN and cephalosporin resistant *S. pneumoniae*:
  - treatment should combine dexamethasone and a third or fourth generation cephalosporin (ceftriaxone, cefotoxamine or cefepime) plus vancomycin, plus acyclovir.

- Add **Doxycycline** during tick season or if pt is high risk

- Add **Ampicillin** if *Listeria* is suspected in pts < 3 mos, >55 yrs or those suspected of impaired immunity (chronic illness, organ transplant, pregnancy, malignancy, immunosuppressive therapy)

- Add **Metronidazole** if gram neg anaerobe risk (otitis media, sinusutis, mastoiditis)

- In hospital acquired meningitis (primarily post-nsgy procedures); cover with Vanc and Cefepime
### Drug Penetration

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>Concentration CSF/concentration serum %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amoxicillin</td>
<td>13-14</td>
</tr>
<tr>
<td>Cefotaxime/ Ceftriaxone</td>
<td>10.1 / 1.5-9</td>
</tr>
<tr>
<td>Cefepime</td>
<td>10</td>
</tr>
<tr>
<td>Vancomycin</td>
<td>7-14</td>
</tr>
<tr>
<td>Metronidazole</td>
<td>45-89</td>
</tr>
<tr>
<td>Trimethroprim/Sulfamethoxazole</td>
<td>50/40</td>
</tr>
</tbody>
</table>

CSF to blood concentration ratios (penetration) in humans

CID 1998;27:1117-29. Sanford Guide**Also, Dipro Pharmacotherapy has information
Empiric Antibiotic Treatment

- All antibiotics are administered IV
- Dosing assumes normal renal and hepatic function
- Vancomycin dosing should be adjusted based on troughs
- First line coverage > 2 yrs
- Add if > 50 yrs
- * chose one cephalosporin

<table>
<thead>
<tr>
<th>ANTIMICROBIAL AGENT</th>
<th>TOTAL DAILY DOSE AND INTERVAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ampicillin</td>
<td>2gm q4h</td>
</tr>
<tr>
<td>Cefepime *</td>
<td>2gm q8h</td>
</tr>
<tr>
<td>Cefotaxime *</td>
<td>2gm q6h</td>
</tr>
<tr>
<td>Ceftriaxone *</td>
<td>2gm q12h</td>
</tr>
<tr>
<td>Ceftazidime</td>
<td>2gm q8h</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>7.5 mg/kg (q8h)</td>
</tr>
<tr>
<td>Meropenem</td>
<td>2gm q8h</td>
</tr>
<tr>
<td>Metronidazole</td>
<td>500mg q6h</td>
</tr>
<tr>
<td>Nafcillin</td>
<td>2gm q6h</td>
</tr>
<tr>
<td>Penicillin G</td>
<td>20-24 million units/day (q4h)</td>
</tr>
<tr>
<td>Vancomycin</td>
<td>15-20 mg/kg IV q8-12h</td>
</tr>
<tr>
<td>Acyclovir</td>
<td>10-15 mg/kg (q8h)</td>
</tr>
</tbody>
</table>
Treatment: *Neisseria meningitidis*

- **For *Neisseria meningitidis***, **PCN-G** remains the antibiotic of choice — follow sensitivities.
  - **Adults**: 20-24 million IV units/day

- Isolates with moderate **resistance** to PCN have been identified and are increasing in susceptibility
  - **Ceftrixone 2gm IV q12h** OR **Cefotaxime 2gm IV q6h**

- Non-complicated meningococcal meningitis is treated with a **7 day course of IV antibiotics**
Prophylaxis *N. meningitides*

Household, daycare, intimate contacts, selected healthcare workers (most infectious 3 days prior to onset of symptoms, no longer communicable 24h after abx)

- Rifampin 600 mg po every 12 hours 2 days for adults, 10 mg/kg every 12 hours (max 600mg/dose) for 2 days for children > 1 month, 5 mg/kg 12 hours (max 600mg/dose) for 2 days for children < 1 month

- Ciprofloxacin 500 mg po once

- Ceftriaxone 125-250 mg IM once (pregnancy)

Vaccination *N. meningitides*

**Menomune®**
- Serogroups A, C, Y and W-135
- Nonconjugated—does not stimulate memory lymphocytes thus only short-term protection
- Ages ≥ 2 years

**Menveo® (conjugated)**
- Approved FDA 2010
- Serogroups A, C, Y and W-135
- Ages 11-55 years of age
- Demonstrated greater immunogenicity

**Menactra® (conjugated)**
- Serogroups A, C, Y and W-135
- Ages 2-55 years

**Trumenba® - serotype B**
- FDA approval – October 2014
- Approved age 10 – 25 years
- Dose/Frequency: 0.5 ml IM x 3 doses: at 0, 2 month and at 6 month
Treatment: *Streptococcus pneumoniae*

- **PCN G** is drug of choice
  - **PCN G 20-24 million IV units/day**

- Intermediate resistance:
  - Ceftrixone 2gm IV q12h OR Cefotaxime 2gm IV q6h

- High resistance or ceph resistance:
  - Vancomycin 15-20 mg/kg IV q8-12 hrs (follow troughs for goal 15-20)

- **10-14 day course of IV therapy is recommended**

- If repeat CSF is obtained and remains GS+, consider resistance
Strep pneumoniae Vaccination

Vaccination

- ≥ 65 years of age, asplenic, immunocompromised —> Prevnar 13®
  (if no prior hx) then Pneumovax® at least 8 weeks later
- Pneumovax® - 23 valent polysaccharide
- Prevnar 13® - conjugated, replaced Prevnar 7®

http://www.cdc.gov/vaccines; see package insert for renal/hepatic dosing
Treatment: *Listeria monocytogenes*

- Ampicillin
  - Adults 2 grams IV every 4 hours
  - Neonates 200 mg/kg/day divided every 6 hours IV

- Aminoglycoside (Gentamicin)
  - ~ 5 mg/kg/day IV divided every 8 hours

- Alternatives
  - Trimethoprim-sulfamethoxazole IV (PCN allergic pts)
  - Meropenem - CONSULT INFECTIOUS DISEASE SERVICE

- Treatment duration ≥ 21 days
Treatment: *Staphylococcus aureus*

- Nafcillin for coagulase negative staphylococci
- Vancomycin for methicillin-resistant staphylococci and for patients allergic to PCN
- CSF should be monitored during therapy and if not stabilized in 48h of IV vancomycin, consider intra-ventricular or intra-theacal vancomycin
Treatment: Adjunctive Therapy (Dexamethasone)

- Dexamethasone works by inhibiting synthesis of IL-1β and TNF-α
- Decreases CSF outflow resistance and stabilizes BBB
- Rational for administration 20 mins before abx is that it inhibits the production of TNF-α production once it has been induced
- Clinical trials demonstrate efficacy in decreasing meningeal inflammation and neurologic sequelae
- Studies show most benefit in patients with pneumococcal meningitis
- Unlikely to be of benefit if started > 6h after antimicrobial therapy has been initiated

Dosing: 0.15 mg/kg IV q6h for 2-4 days
## Antimicrobial Table

<table>
<thead>
<tr>
<th>ORGANISM</th>
<th>ANTIBIOTIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neisseria meningitides</td>
<td>PCN-Sensitive: PCN G or Ampicillin</td>
</tr>
<tr>
<td></td>
<td>PCN-Resistant: Ceftriaxone or Cefotaxime</td>
</tr>
<tr>
<td>Streptococcus pneumoniae</td>
<td>PCN-Sensitive: PCN G</td>
</tr>
<tr>
<td></td>
<td>PCN-Intermediate: Ceftriaxone, Cefotaxime or Cefepime</td>
</tr>
<tr>
<td></td>
<td>PCN-Resistant: Ceftriaxone + Vancomycin</td>
</tr>
<tr>
<td>Gram Neg Bacilli (ex. Pseudomonas)</td>
<td>Ceftriaxone or cefotaxime</td>
</tr>
<tr>
<td>Pseudomonas aeruginosa</td>
<td>Ceftazidime, Cefepime or Meropenem</td>
</tr>
<tr>
<td>Staphylococci spp</td>
<td>Methicillin-Sensitive: Nafcillin</td>
</tr>
<tr>
<td></td>
<td>Methicillin-Resistant: Vancomycin</td>
</tr>
<tr>
<td>Listeria monocytogenes</td>
<td>Ampicillin + Gentamicin</td>
</tr>
<tr>
<td>Haemophilus influenzae</td>
<td>Ceftriaxone, cefotaxime, cefepime</td>
</tr>
<tr>
<td>Streptococcus agalactiae</td>
<td>Ampicillin</td>
</tr>
<tr>
<td>Bacteroides fragilis</td>
<td>Metronidazole</td>
</tr>
<tr>
<td>Fusobacterium spp</td>
<td>Metronidazole</td>
</tr>
</tbody>
</table>
Viral Meningitis

- HA, fever, nuchal rigidity (mild)
- Inflammatory CSF profile
- Photophobia, painful eye movements
- Constitutional signs (diarrhea, N/V, anorexia, malaise, myalgias, AMS)
- Seizure
- ~ 60,000-75,000 cases per year
- Increase in non-winter months in temperate climates
- Seasonal predominance of enterovirus and arbovirus in summer and fall
Viral meningitis

- Specific viral causes can be found in 60-90% of cases
- CSF cultures are positive in 30-70% of cases
- Enteroviruses (coxsackievirus, echoviruses)
- Varicella-zoster virus (VZV)
- Herpes-simplex virus (HSV2 > HSV1)
- Human Immunodeficiency virus (HIV)
- Arboviruses
# CSF Profile 

**Viral Meningitis**

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Opening Pressure</strong></td>
<td>Slightly Elevated (15-35 cm H2O)</td>
</tr>
<tr>
<td><strong>Glucose</strong></td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Protein</strong></td>
<td>Slightly Elevated</td>
</tr>
<tr>
<td><strong>WBC</strong></td>
<td>&lt; 200; lymphocyte dominance</td>
</tr>
<tr>
<td><strong>Appearance</strong></td>
<td>Clear</td>
</tr>
<tr>
<td><strong>Gram Stain</strong></td>
<td>Negative</td>
</tr>
</tbody>
</table>
HSV Encephalitis

Herpes simplex (HSV) encephalitis

Polymerase chain reaction (PCR) CSF

MRI will reveal T2 hyper intensities in frontal or temporal lobes

Treatment:
  Adults: Acyclovir 10 mg/kg/dose iv every 8 hours (renal adjust)

Treatment duration 10-14 days (limited information)
Ventriculitis/Post-Surgical

- Inflammation originating in the ventricles

- Common source is extra ventricular drain (EVD) or ventricular-peritoneal shunt (VPS)

- EVD has 8% infection rate

- Life threatening complication

- Risk of infection increases
  - Device duration
  - Device blockage or leakage
  - Frequent access
Ventriculitis / post-surgical

- AMS, fever leukocytosis, seizure
- Etiology:
  - Gram negative bacilli (aerobic)
    - *Pseudomonas aeruginosa*
    - *Enterobacter, Citrobacter and Klebsiella species*
    - *Acinetobacter species*
  - *Staphylococcus aureus*
  - *Staphylococcus epidermidis*
Treatment Ventriculitis / post-surgical

- Cefepime / Ceftazidime / Meropenem (2gm IV q8h)

  PLUS

- Vancomycin IV (goal trough 15-20)

- Obtain surveillance CSF until negative culture/gram stain

- Consider removal or replacement of EVD

- Explantation of VPS

- Treatment duration 14 days

- Consult ID for VPS replacement (7-10 days from last positive culture)
Supportive Care

- Temperature control
- ICP management
- Fluid and Electrolytes
- Analgesics
- Anti-emetics
- Nutrition
Case Study

• 60yo female PMHx DM2, hypothyroidism, chronic back pain who presents with 1 week of N/V, HA, diarrhea and worsening back pain. While in ED, patient is found naked in room “trying to get in the garbage can”. She then progressed to unresponsiveness. Temperature at that time was noted to be 38.6C. She was intubated for airway protection.

• Differential Diagnoses?

• What next?
Case Study

- CT
- LP
- Blood Cultures
- Antibiotics
Case Study

CSF Profile:

WBC: 190K
RBC: 1194
Protein: 829
Glucose: 9
Serum Glucose: 251
Gram Stain: GPC
Culture: *Streptococcus anginosus*
Case Study

Diagnosis?
Case Study

- Treatment?
- Antibiotics— Vancomycin, Cefepime, Metronidazole
- EVD
- Manage ICP - Sedation
- OR for washout and drainage
- EEG
- Cipro-Dex ear gtts
Case Study

- 48 yo male with PMHx CAD, cardiomyopathy, HTN who presented to hospital with 5 days of HA, diplopia, gait imbalance and facial weakness.

- What is your first diagnostic test after neurological exam is performed?
Case Study

- Patient is admitted to the ICU for monitoring
- Exam: Facial weakness, CN3 palsy, confused, L pronator drift
- VS: Temp 37.9; HR 110; BP 112/63; SpO2 97% on RA
- Diagnosis? Tumor or abscess?
- LP is deferred due to ASA use and no obvious primary infectious etiology
Case Study

• LP obtained on ICU day 3
  • WBC: 4
  • RBC: 4000
  • Protein: 19
  • Glucose: 70
  • Gram Stain and culture: negative

• Next Steps?
Case Study

- Brain biopsy confirms Gram + cocci; *Strep anginosus*
- Antibiotic started
- CT sinuses
- OMFS consult for maxillary lesion
- Full dental extraction performed
- Patient continues to wax/wane
- Back to OR for washout of abscess
- Gradual improvement
- Required VPS
Case Study

- 33 yo male with DM1, HTN, CAD and GERD presents with acute onset new focal motor seizure of LUE, 3 days of HA, night sweats, photophobia and neck pain. He was loaded with phenytoin in the ED and blood cultures were obtained. He was admitted to floor with medicine team.

- 2 hours after admission, patient has GTC seizure and fever of 39.7.

- What next?
Case Study

- Transfer to ICU
- Vancomycin, Ceftriaxone ordered
- NCHCT
- Lumbar Puncture
  - Opening pressure 28cm H20
  - WBC 1950
  - RBC 4
  - Protein 734
  - CSF Glucose 62, Serum Glucose 371
- Gram Stain: GPCs
Case Study

• 27 yo female with no PMHx presents to ICU from OSH after being found down by her mother have “muscle spasms”. She was intubated at OSH for airway protection and transferred for further care.

• She arrives to your ICU intubated and sedated. Her exam in notable for decorticate posturing in BUE, PERRL, brain stem reflexes intact. Fever 104F; WBC 16.9 on CBC, lactate 5.4.

• NCHCT and LP performed
Case Study

• LP:
  • WBC 20
  • RBC: 38
  • Protein: 829
  • Glucose: 112; Serum glucose 123
  • Gram Stain: negative
• Antibiotics? Which ones?
Case Study

- Vanc, Cefepime, Acyclovir
- What other CSF studies?
  - HSV, VZV, EBV, WNV, paraneoplastic panel, oligoclonal bands
- MRI
Case Study

- Mother mentions that patient might have had vesicular lesion around mouth a few weeks ago
  - HSV serum Ab positive
  - HSV CSF PCR NEGATIVE
- Continue supportive care; continue acyclovir
- 7 days later, WNV serum IgG and IgM positive
- Diagnosis?
References


• https://www.imaios.com/en/e-Anatomy/Head-and-Neck/Brain-diagrams


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