MENINGITIS

ANATOMY

- The meninges comprise three membranes that, together with the cerebrospinal fluid, enclose and protect the brain and spinal cord
- The pia mater firmly adheres to the surface of the brain.
- The arachnoid mater is a loosely fitting sac on top of the pia mater.

The subarachnoid space separates the arachnoid and pia mater membranes, and is filled with cerebrospinal fluid.

- The thick outermost membrane, the dura mater, which is attached to both the arachnoid membrane and the skull.

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- An infection of the central nervous system may primarily affect its coverings, which is called meningitis.
- It may affect the brain parenchyma, called encephalitis, or affect the spinal cord, called myelitis.
- A patient may have more than one affected area, and if all are affected, the patient has "meningoencephalomyelitis".

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- The nervous system may also suffer from localized pockets of infection.

- Within (the brain or spinal cord) there may be an abscess, and outside there may be an epidural abscess

- **Meningitis** is inflammation of the protective membranes covering the brain and spinal cord, known collectively as the meninges.
- The inflammation may be caused by infection with microorganisms, and less commonly by certain drugs.
- Meningitis can be life-threatening because of the inflammation's proximity to the brain and spinal cord; therefore the condition is classified as a medical emergency.

MECHANISM OF INFECTION

- In bacterial meningitis, bacteria can reach the meninges through the bloodstream or through direct contact between the meninges and either the nasal cavity or the skin.
- In most cases, meningitis follows invasion of the bloodstream by organisms that live on mucous surfaces such as the nasal cavity.
- This is often preceded by viral infections, which break down the normal barrier provided by the mucous surfaces.

- Once bacteria have entered the bloodstream, they enter the subarachnoid space in places where the blood-brain barrier is vulnerable—such as the choroid plexus.

- Direct contamination of the cerebrospinal fluid may arise from indwelling devices, skull fractures, or infections of the nasopharynx or the nasal sinuses

- The large-scale inflammation that occurs in the subarachnoid space during meningitis is due to the response of the immune system to the entrance of bacteria into the central nervous system.

- The blood-brain barrier becomes more permeable, leading to "vasogenic" cerebral edema (swelling of the brain due to fluid leakage from blood vessels).
- Large numbers of white blood cells enter the CSF, causing inflammation of the meninges, and leading to "interstitial" edema (swelling due to fluid between the cells).
- In addition, the walls of the blood vessels themselves become inflamed (cerebral vasculitis), which leads to a decreased blood flow and a third type of edema, "cytotoxic" edema.

- The three forms of cerebral edema all lead to an increased intracranial pressure
- ...together with the lowered blood pressure often encountered in acute infection, it is harder for blood to enter the brain, and brain cells are deprived of oxygen and undergo apoptosis (automated cell death).

Causes of acute meningitis

CATEGORY	CAUSE
Bacteria	...next slide
Viruses	Enteroviruses, Mumps, Measles, Herpes, Influenza, Parainfluenza, HIV, arbovirues, Lymphocytic choriomenengitis virus
Protozoa	Naegleria, Acanthamoeba, Angiostrongylus
Helminthes	Strongyloides
Systemic diseases	Systemic Lupus Erythematosus
Procedure related	Post neurosurgery, spinal anaethesia,

Bacterial Meningitis

Cause depends on

- Age
- Immune status
- Invasive incident: head trauma, neurosurgery

Important bacterial virulence factors

- Fimbriae
- Capsule
- IgA protease

AGE/CONDITION	COMMON AGENTS
0-4 weeks	Grp B Strep,*E.coli*, *L.monocytogenes, Klebsiella, Enterococcus,*
4-12 weeks	Grp B Streps, *E. coli, Listeria, Klebsiella, H. infulenzae, S. pneumoniae, N. meningitidis*
3 months to 18 years	*S. pneumoniae, N. meningitidis, H. influenzae*
18 to 50 years	*N. meningitidis S. pneumoniae*
> 50 years	*N. meningitidis S. pneumoniae Listeria*, aerobic G-ve bacilli

AGE/CONDITION	COMMON AGENTS
Immuno-compromised	*N. meningitidis, S. pneumoniae* *L. monocytogenes* aerobic G-ve bacilli
Basal skull fracture	*S. pneumoniae, H. influenzae,* group A streptococci
Head trauma, post neurosurgery	*S. aureus, S. epidermidis,* aerobic G-ve bacilli

Clinical Features

Classical features:

- high fever, headache, stiff neck, confusion, reduced conscious level
- Seizures, Cranial nerve palsies
- Skin rash: meningococcal, pneumococcal

In newborns

- Bulging fontanelle in neonates
- High fever
- Constant crying
- Excessive sleep/Sluggishness
- Poor feeding
- Stiffness in body and neck

Differential diagnosis

Early stage of disease

- Gastroenteritis
- Upper respiratory tract infection
- Pneumonia
- Otitis media

Later stage of disease

- Subarachnoid hemorrhage
- Toxic ingestions
- Seizure disorders
- Hypothyroidism

Lab Diagnosis

CSF specimen

- High ICP
- Macroscopic examination- turbid in bacterial meningitis
- Biochemistry- glucose, protein
- Staining...Gram stain, India ink
- Culture
- Serology and Molecular tests

...lab CSF biochemistry

VIRAL

- Low WBC count
- Mainly mononuclear cell type
- Normal glucose
- Normal protein

BACTERIAL

- High WBC count
- Mainly polymorphonuclear leukocytes
- Low glucose
- High protein

...lab diagnosis

Blood specimen

- Non-specific markers of inflammation:

C reactive protein

- Blood cultures

...lab diagnosis

Blood specimen

- Non-specific markers of inflammation:

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COMPLICATIONS OF MENINGITIS

- The infection may trigger sepsis
- Disseminated intravascular coagulation (excessive activation of blood clotting)
- Severe meningococcal and pneumococcal infections may result in hemorrhaging of the adrenal glands, which is often lethal.
- The brain tissue may swell, with increasing pressure inside the skull and a risk of swollen brain tissue getting trapped. This may be noticed by a decreasing level of consciousness

LATE COMPLICATIONS

- Deafness
- Epilepsy
- Hydrocephalus
- Cognitive deficits such as severe learning difficulty
- Blindness
- Death may occur as an early or late complication

Management

- Empirical antimicrobial therapy immediately pending final lab investigations

AGE/CONDITION	EMPIRIC THERAPY
0-4 weeks	Ampicillin + Cefotaxime or aminoglycoside
4-12 weeks	Ampicillin + Ceftriaxone
3 months to 18 years	Cefotaxime or Ceftriaxone
18 to 50 years	Cefotaxime or Ceftriaxone
> 50 years	Ampicillin + Cefotaxime or Ceftriaxone

AGE/CONDITION	EMPIRIC THERAPY
Immuno-compromised	Ampicillin + Ceftazoidime
Basal skull fracture	Cefotaxime or Ceftriaxone
Head trauma, post neurosurgery	Vancomycin + Ceftazidime
CSF shunt	Vancomycin + Ceftazidime

- Corticosteroid treatment
- Prior to or along initial antibiotics
- Decreases
    - Intracranial pressure
    - Cerebral oedema
    - Neurologic sequeale, including deafness

- For positive gram stain or culture results, tailor treatment according to infecting organism
- Reduce intracranial pressure:

eg Elevating the head to maximize venous drainage

Prevention techniques

- Vaccination where applicable
- Chemoprophylaxis where applicable

- In conclusion, remember meningitis is a medical emergency

- Important to take urgent measures to reduce the chance of death occurring