

Microbiology

of

Acute

Pyogenic Meningitis



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Microbiology of Acute Pyogenic Meningitis

Meningitis :

is an infection of the meninges around the brain and spinal cord (pia matter , arachnoid matter + subarachnoid space which contain CSF) . Meningitis is usually caused by an infection with a virus, with a bacterium or even with fungi.

Pyogenic meningitis :

- Pyogenic meningitis is an inflammation of the meninges affecting Pia, Arachnoid and **subarachnoid space**.
- A serious infection ,associated with marked inflammatory exudation.
- Acute in onset.
- Usually caused by bacterial infections
- May be preceded by **URTI**.
- Can be **fatal** if untreated.

Common etiological agent :

Three main bacterial species :

- 1- ***Neisseria meningitidis***
 - 2- ***Sterptococcus pneumoniae***
 - 3- ***Hemophilus influenzae***
- } usually from URT

Properties :

- They are capsulated
- They have vaccine → so it 's preventable
- They all sensitive to Ceftriaxone → so it is the first drug of choice



Causes According to the Age :

Newborns

Group *B Streptococcus*, *E.coli* (and other gram negative bacilli) , *Listeria monocytogenes*,
(they can be in the genital tracts of the mother so the child will present of septicemia meningitis)
(New borns don't get *H.influenzae* in the first 6 months because of the IgG's from the mother)

Infants / Children

S.pneumoniae, *N.meningitidis*,
H.influenzae

Adults

S.pneumoniae, *N.meningitidis*

Special circumstances

S.aureus, *S.epidermidis*,
S.pneumoniae, anaerobes,
P.aeruginosa

For best treatment you need to know the most common organism

Meningitis- Epidemiology :

- There are 1.2 million cases annually worldwide.
- 135,000 deaths.
- Bacterial meningitis is 1 of the top 10 infections causes death worldwide.

- Half of the survivors suffer neurological damage, and /or permanent side effects
- And it is still kill the people

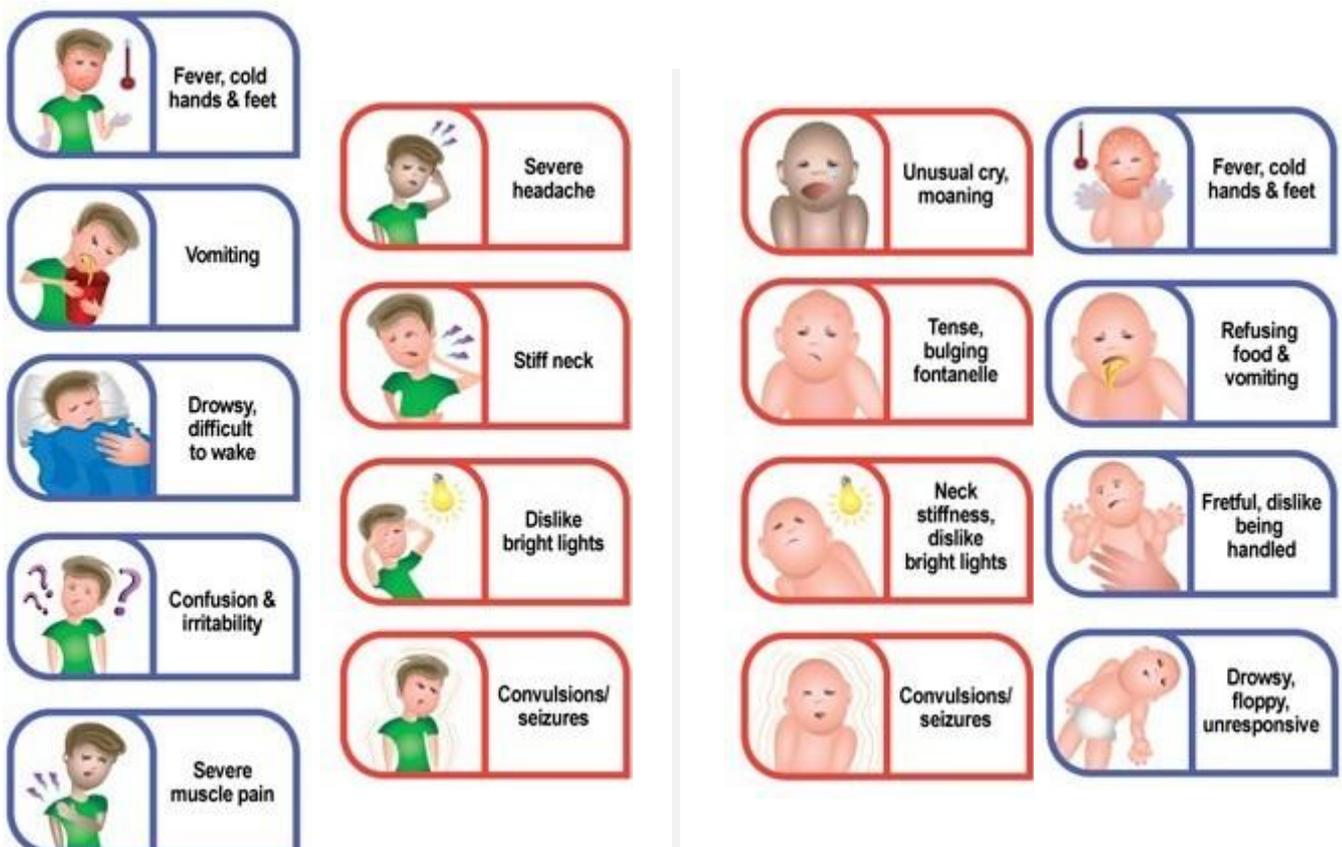
(people who died due to meningitis , they die due to **STRONG IMMUNE** response of their body which kill the body before killing the organism)

Signs/Symptoms of Acute Meningitis :

(Symptoms of meningitis can come on very quickly or take a couple of days to appear. Most cases of meningitis occur in the first 5 years of life, with the peak incidence between 3 and 5 months)

In the adults and children :

In infants :

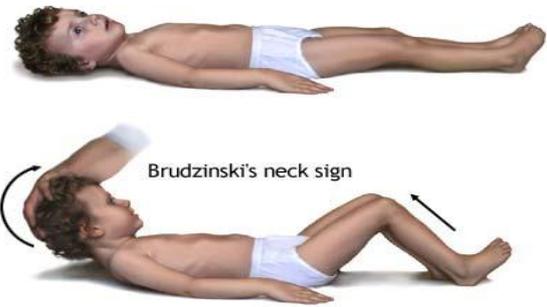
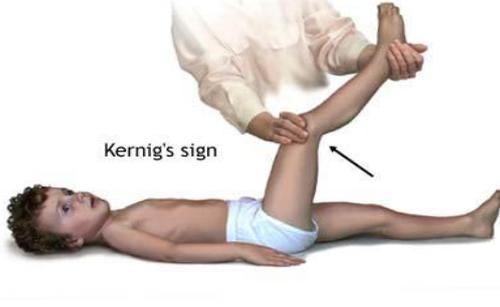


In advanced case : (in infants , children and adults)

(bruises and rashes under skin & spread rapidly)

Can leads into advanced disease , such as: (Brain damage , Coma , death)

New born present with hyperthermia but he adults mostly present with hypothermia

Brudziński neck sign	Kernig's sign
clinical sign in which forced flexion of the neck elicits a reflex flexion of the hips.	when the leg is bent at the hip and knee at 90 degree angles, and subsequent further extension in the knee is painful (leading to resistance)
 <p>Brudzinski's neck sign</p>	 <p>Kernig's sign</p>

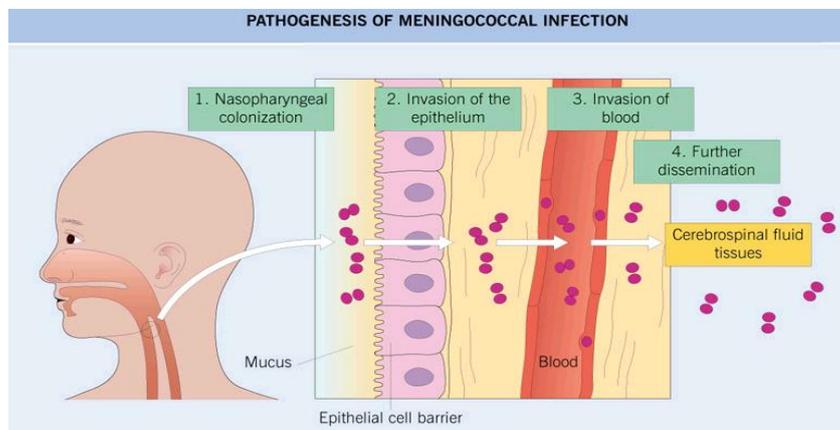
Pathogenesis :

Colonization of nasopharynx (or from birth canal) →
Speticemia → BBB → Lead to :

Wide spread endothelia damage → Activation of
coagulation → Thrombosis and platelets aggregation



(Meningococcal infection results from blood-born dissemination (meningococemia) of *N. meningitidis* following an asymptomatic or mildly symptomatic nasopharyngeal carrier state or mild rhinopharyngitis.)

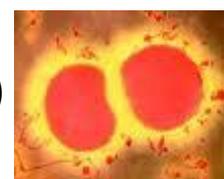


Why we don't get colonization of nasopharynx :

In the throat there is Normal flora (*Nesseiria*) which produce Antibody that can fight the other bacteria :))

1- *N.meningitidis* : (NEISSERIA MENINGITISIS)

- A Gram negative diplococci present in the nasopharynx of 10 % of people.
- Transmitted by inhalation of aerosolized droplets, close contact.
- Common in children <6 y (can occur in 20's too)
- Risk factor: susceptible individuals.
- Serotypes: **B,C,Y,W135** cause isolated ,sporadic small epidemics in close population.
- T Serotype **A** has an epidemic potential in sub-saharan Africa (meningitis belt).
- We don't have vaccine for B
- W135 found here in KSA



Pathogenesis :

- Carriers stimulate antibody production,
- In some pili attach to microvilli of nasopharynx – invasion → bacteremia, endotoxin (LPS) produced → meninges.
- Capsule resists phagocytosis.
- 11-20 % of recovered patients suffer permanent hearing loss, mental retardation.
- 10-14% of cases are fatal.
(if there is not WBC's that's mean the organism succeeded to suppress the immune system)

2- S.pneumonia (Most dangerous)

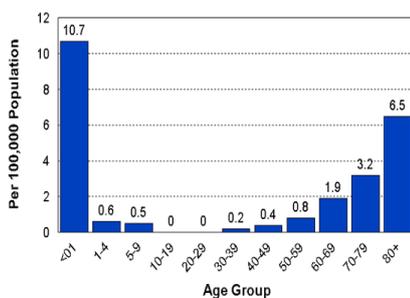
- a Gram positive diplococci ,meningitis may follow pneumococcal pneumonia ,or other site .
- May develop after trauma to the skull.
- High mortality rate >30% due to invasive disease.
- Capsule is polysaccharide polymer
- Pneumolysin decreases inflammatory immune response severe infection.
- Infection rate decreases due to vaccination .
- Recovered cases develop sustain learning disabilities .



3- *H.influenzae* (less dangerous) :

- A small Gram negative coccobacilli
- Has polysaccharide capsule , other species has no capsule.
- Need blood for optimal growth, Hematin (factor X) and NAD (factor V)
- Many serotypes a-f
- *H.Influenzae* type *b* has a capsule ,a polymer of RPR ,cause acute life threatening invasive infections .
- Found in the nasopharynx normal flora
- Major cause of lower RTI, occasionally invade deeper tissues and cause bacteremia.
- Bacteremia----- CNS ,bones or other organs.
- 3-6% mortality rate
- 1/3 of survivals have significant neurological sequelae
- Infection rate decreases since the routine use of **Hib**

Figure 3. Haemophilus Influenzae Incidence by Age Group -- Indiana, 2003



4- Group B Streptococcus :

- Gram positive cocci in chains
- Resident in GIT & vagina (10-30%)
- Gain access to amniotic fluid during delivery or colonize newborn as it passes birth canal.



- *Risk factors*: premature rupture of membrane, prematurity, low infant innate immunity
- Cause sepsis & meningitis in the first few days of life or after 4 weeks

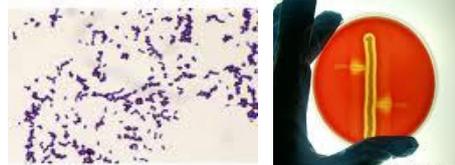
5- *E.coli* :

- A Gram negative bacilli
- Most common cause of neonatal meningitis
- Many features similar to GBS .
- Vaginal *E.coli* colonize infant via rupture of amniotic membrane or during birth.
- Failure of preterm maternal IgM to cross placenta & special susceptibility of newborn.
- K1 sialic acid capsule of some strains invade brain microvascular endothelial cells.



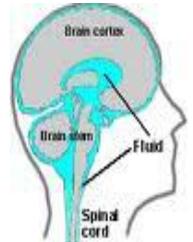
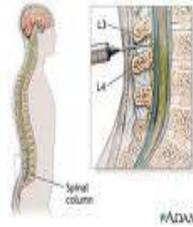
6 - *Listeria monocytogenes* :

- Gram positive rods
- Wide spread among animals in nature including those associated with food supply.
- Human intestinal colonization (2-12%)
- Spread to fetus following hematogenous dissemination in mother or from birth canal
- Has tropism to CNS.
- Prolong treatment (21 days)



Diagnosis of Meningitis

- Specimen : **CSF** acquired through lumbar puncture and **blood culture** for :
- analysis of cells, protein, glucose ,
- culture and antimicrobial susceptibility testing.



Findings of CNS analysis :

Normal CSF		Pyogenic meningitis
Adults	Neonates	
WBC =0-5 /cmm3, PMN= 0 %, glucose= > 60 % of blood, protein =< 30 mg/dl	<i>term :</i> WBC =0-32 /cmm3, PMN=>60 %, glucose = >60 % of blood, protein= 20-170 mg/dl <i>Preterm:</i> WBC=0-29/cmm3, PMN= <60 %, glucose = >60 % of blood, protein= 60-150 mg/dl	WBC= 5 - 5000/cmm3 PMN= > 60% Glucose = < 45 % of blood Protein= >60 mg/dl



Management : (Empirical treatment)



- **Urgent , A MEDICAL EMERGENCY**
- **Antibiotics** after taking specimens for lab diagnosis
- Parenteral administration of **Ceftriaxone** (or **Cefotaxime**) + **Vancomycin** (*cover the main 3 pathogens*) or ,
- **Ampicillin + Gentamicin or Cefotaxime** (*neonates*)
- Duration : 10-14 days according to the medical condition
- Prevention: **vaccination , prophylaxis of contacts (Hib& N.meningitidis)**

