

THE EPIDEMIOLOGY OF MENINGOCOCCAL MENINGITIS

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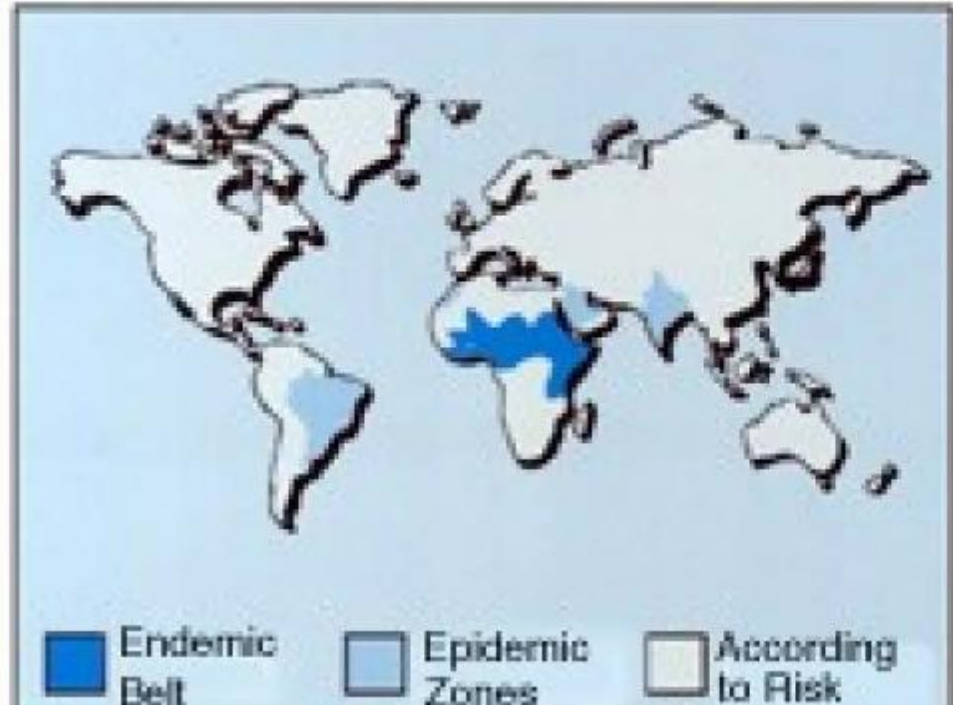
Meningococcal meningitis



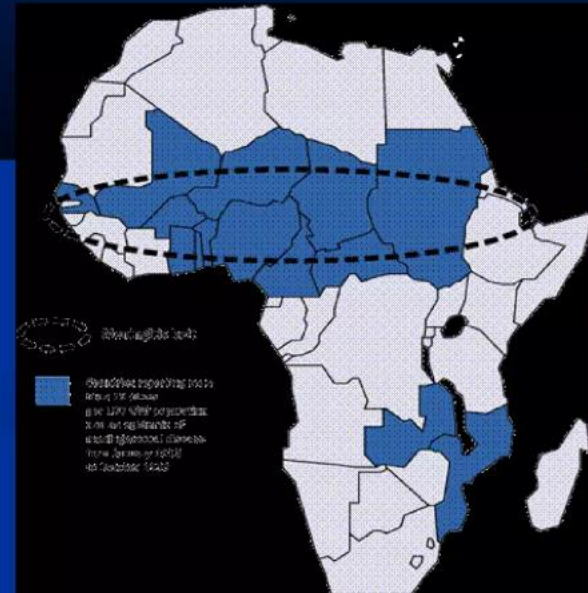
Meningococcal meningitis or cerebro-spinal fever is "an acute communicable disease caused by *Neisseria meningitidis*".

1. Distribution world-wide, occurring sporadically and in small outbreaks in most parts of the world.

MENINGOCOCCAL (MENINGITIS)



2. The zone lying between 5 and 15 degree N of the equator in tropical Africa is called the “Meningitis Belt” because of the frequent epidemic waves that have been occurring in that region.

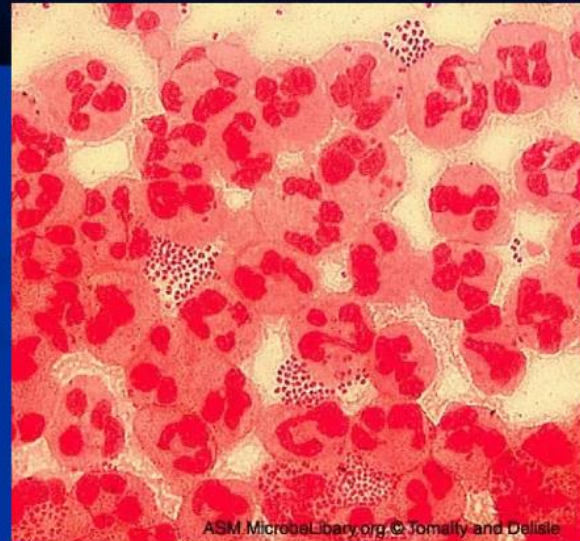


3. Without epidemics, 1 million cases of bacterial meningitis are estimated to occur with at least 200,000 deaths.
4. About 300,000 of these cases and 30,000 deaths are due to meningococcal meningitis.

5. In epidemic years the number of meningococcal meningitis cases may double.
6. WHO definition of epidemic is >100 Cases/100000 population/year

Epidemiological triad:agent

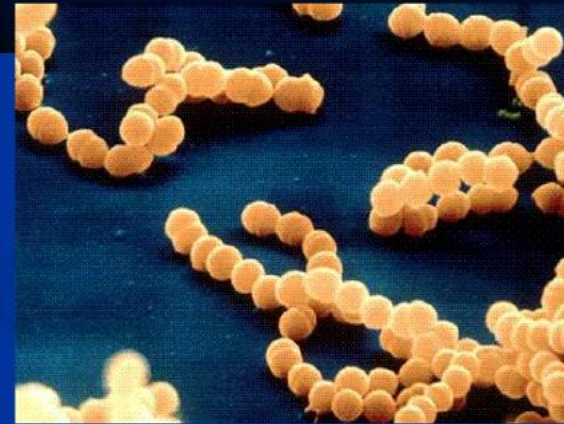
1. *Neisseria meningitidis* is a gram-negative diplococci.
2. Several serotypes have been identified, viz. Groups A, B, C, D, X, Y, 29 E, W135, etc.



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Agent(cont.)

3. **Groups A and C, and to a lesser extent Group B meningococcal are capable of causing major epidemics.**



Epidemiological triad :Host FACTORS

- 1. This is predominantly a disease of children and young adults of both sexes.**

EPIDEMIOLOGICAL:HOST FACTORS

- 2. All ages are susceptible, but younger age groups are more susceptible than older groups as their antibodies are lower. Immunity is acquired by sub-clinical infection (mostly), clinical disease or vaccination.**

EPIDEMIOLOGICAL TRIAD: HOST FACTORS

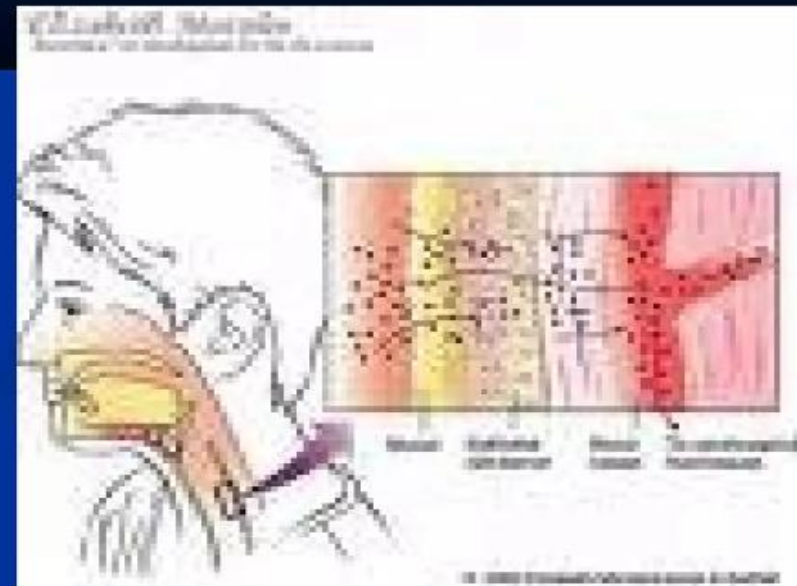
3. **Infants derive passive immunity from the mother.**



EPIDEMIOLOGICAL TRIAD:HOST FACTORS

Source of Infection

1. Organism is found in the nasopharynx of cases and carriers.



EPIDEMIOLOGICAL TRIAD:HOST FACTORS SOURCE OF INFECTION(CONT.)

- 2. Carriers – most important source of infection. 5 – 30% of the normal population may harbor the organism in inter-epidemic period. During epidemics, the carrier rate may go as high as 70-80%.**



EPIDEMIOLOGICAL TRIAD:HOST FACTORS

Source of Infection

3. Cases – source only in negligible number of cases.



EPIDEMIOLOGICAL TRIAD:HOST FACTORS

Period of Communicability

2. Cases lose their infectivity rapidly within 24 hrs of specific treatment.
3. The mean duration of temporary carriers is 10 months.



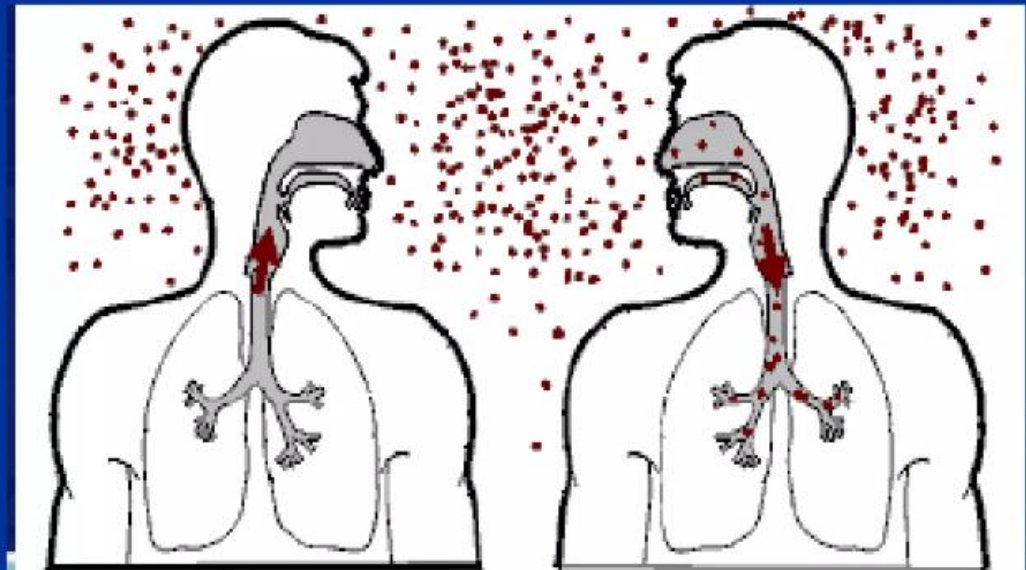
EPIDEMIOLOGICAL TRIAD: ENVIRONMENTAL FACTORS

1. Overcrowding (in Schools, Barracks, Refugee camps, etc.) predisposes to spread of infection.
2. Outbreaks of the disease occur more in dry and cold months of the year.



Mode of transmission

The disease spreads mainly by droplet infection. The portal of entry is the nasopharynx.



INCUBATION PERIOD

Usually 3 to 4 days, but may vary from 2 to 10 days.



Clinical Features

**Meningococcal Meningitis
without Meningococemia**

**Meningococcal Meningitis
with Meningococemia**

CLINICAL FEATURES-MENINGITIS WITHOUT MENINGOCOCCEMIA

1. Patients with meningococcal meningitis have usually been sick for >24 h before they seek medical attention.

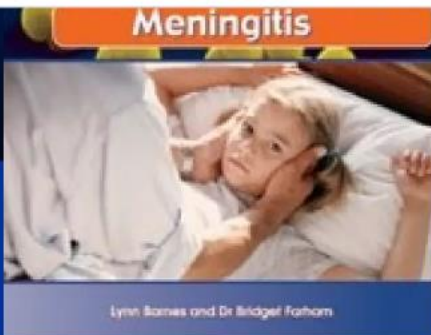


CLINICAL FEATURES-MENINGITIS WITHOUT MENINGOCOCCEMIA

Common presenting symptoms:

1. Fever ,Nausea and Vomiting.
 1. Headache.
 2. Convulsions.

CLINICAL FEATURES-MENINGITIS WITHOUT MENINGOCOCCEMIA



2. **Common presenting symptoms:**
 4. **Neck Stiffness.**
 5. **Lethargy and Confusion, maybe Coma.**

CLINICAL FEATURES-MENINGITIS WITHOUT MENINGOCOCCEMIA



3. Petechial hemorrhages on skin and/or mucosa may be seen.

CLINICAL FEATURES-MENINGITIS WITHOUT MENINGOCOCCEMIA

4. The signs and symptoms of Meningococcal Meningitis cannot be distinguished from those elicited by other meningeal pathogens.

CLINICAL FEATURES-MENINGITIS WITH MENINGOCOCCEMIA

1. Pharyngitis.



2. Fever.

3. Weakness and
Myalgia.



CLINICAL FEATURES-MENINGITIS WITH MENINGOCOCCEMIA

4. Vomiting and Diarrhoea.



5. Headache.

6. May develop maculopapular rash before other serious signs develop.



CLINICAL FEATURES-MENINGITIS WITH MENINGOCOCCEMIA

**7. Fulminate cases –
Rapid progression
to shock
characterised by:**

1. Hypotension.
2. DIC.
3. Acidosis.



CLINICAL FEATURES-MENINGITIS WITH MENINGOCOCCEMIA

4. Adrenal hemorrhage.



5. Renal failure.

6. Myocardial failure.

7. Coma.



Clinical Features – Other manifestations



1. Arthritis – Approx.
10% patients.



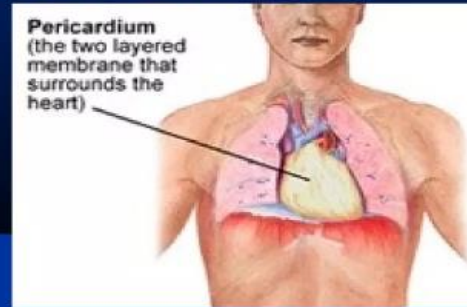
CLINICAL FEATURES: OTHER MANIFESTATION

2. Primary
Meningococcal
Pneumonia – Mainly
in adults.



Other manifestations

3. Meningococcal
Pericarditis.



4. Primary Meningococcal
Conjunctivitis – Can
precede *meningococemia*.



5. Urethritis

DIAGNOSIS

1. Definitive diagnosis – Isolation of causative organism, its antigens, or its DNA from normally sterile body fluid like blood, CSF, or synovial fluid.



DIAGNOSIS

N.B.

1. Isolation of organism from nasopharynx is not diagnostic for invasive disease. Serves only research or epidemiological purposes.



2. Cultures may be negative if the patient has received prior antibiotics.

DIAGNOSIS

- Meningococci* can sometimes be identified in Gram stain or culture of papular or petechial lesions.
- Meningococci* may occasionally be seen on Gram stain of the “buffy coat layer” of a spun blood sample.

DIAGNOSIS:LAB. FINDINGS

1. BLOOD.

- 1. leukopenia, or leukocytosis.
- 2. thrombocytopenia.
- 3. elevated ESR.
- 4. hypoalbuminemia.
- 5. hypocalcemia.
- 6. elevated CRP.
- 7. metabolic acidosis.

DIAGNOSIS:LAB. FINDINGS

2. Urine –
 1. Hematuria.
 2. Proteinuria.



DIAGNOSIS:LAB. FINDINGS:CSF CONSISTENT WITH PURULENT MENINGITIS

1. Hypoglycemia.
2. Elevated protein level.
3. Neutrophilic Leukocytosis.
4. Gram's Stain of CSF reveals intra- or extra-cellular organisms in approximately 85% cases.



DIFFERENTIAL DIAGNOSIS

1. Meningitis caused by other bacteria like Gram-negative bacteria, *Streptococcus pneumoniae*, *Staphylococcus aureus* or Group A *Streptococcus*.
2. *Tubercular meningitis*.
3. *Viral meningitis*.

TREATMENT:SPECIFIC

1. For hospitalized patients, penicillin G (250,000- 400,000 U/kg/24 hr divided q 4-6 hr IV) remains the drug of choice.
2. Cefotaxime (200 mg/kg/24 hr) or ceftriaxone (100 mg/kg/24 hr) are acceptable alternatives and generally are part of initial empiric regimens.

TREATMENT SPECIFIC

3. Chloramphenicol (75-100 mg/kg/ 24 hr divided q 6 hr IV) remains effective treatment for patients who are allergic to β -lactam antibiotics.
4. Therapy is continued for 5-7 days.

Treatment - Supportive

1. Maintenance of the Blood Pressure

by:

1. IV Fluids
2. Vasopressors.



REDUCTION OF CEREBRAL EDEMA

1. Mannitol.
2. Steroids.
2. Anticonvulsant Therapy.



COMPLICATION

1. Acute complications –
 1. Vasculitis, DIC, and Hypotension.
 2. Focal skin infarctions.
 3. The gangrene of extremities often seen with *purpura fulminans* may necessitate amputations.



Purpura Fulminans

COMPLICATIONS



2. Deafness – most frequent neurological sequel, occurring in 5-10% of children with meningitis.

COMPLICATIONS

3. Cerebral arterial or venous thrombosis with resultant cerebral infarction can occur in severe cases.



Angiogram showing
Cerebral Venous Thrombosis

COMPLICATIONS

4. Meningitis rarely is complicated by subdural effusion or empyema or by brain abscess.



COMPLICATIONS



5. **Reactivation of latent herpes simplex virus infections (primarily herpes labialis).**

Prevention and Control – Cases



1. Drug of Choice – Penicillin.
In Penicillin-allergic patients –
Chloramphenicol.

PREVENTION AND CONTROL OF CASES

2. Treatment with antibiotics can save the lives of 95 per cent of patients provided that it is started during the first 2 days of illness.

Prevention and Control – Carriers



1. Treatment with Penicillin does not eradicate carrier state.
2. Powerful antibiotic like Rifampicin is needed for eradication.

Prevention and Control – Contacts



1. Close contacts of persons with confirmed meningococcal disease are at an increased risk of developing meningococcal illness (about 1000 times the general population).

PREVENTION AND CONTROL OF CONTACT

2. Chemoprophylaxis for contacts:

1. Rifampicin (Drug of choice until the organism is known to be sensitive to Sulfadiazine) – 600 mg BD for 2 days (Adult).
2. Sulfadiazine – 1 gm BD for 2 days.

PREVENTION AND CONTROL MASS CHEMOPROPHYLAXIS

1. Restricted to closed and medically supervised communities.
2. Mass treatment causes an immediate drop in the incidence rate of meningitis and in the proportion of carriers.
3. Efficacy depends to a large extent on the population coverage.

PREVENTION AND CONTROL IMMUNIZATION

1. **Type: Mucopolysaccharide Vaccine.**
2. **Monovalent, Bivalent (A/C), or
Quadrivalent (A/C/Y/W-135).**
3. **Available against A and C strains.**
4. **Freezed dried vaccine.**



PREVENTION AND CONTROL IMMUNIZATION

5. Reconstituted with special diluent.
6. Multiple dose vials after reconstitution can be used within 24 hrs. if stored between 2 – 8°C.
7. Route of administration – Subcutaneous.
8. Dose – 0.5 ml.

PREVENTION AND CONTROL IMMUNIZATION

9. Single dose after age of 2 years. Between 1 – 2 years of age, 2 doses are recommended at a gap of 6 weeks.
10. Efficacy – 95%.
11. Immunity persists for 3 years.
12. Booster is recommended every 3 years.

PREVENTION AND CONTROL IMMUNIZATION

14. Storage – 2 – 8°C.



15. Side-effects: Pain and Erythema at site of injection.

16. Must be used along with chemoprophylaxis as IP of disease is shorter than IP of vaccine.

13. Contraindications:

1. Pregnancy.
2. Infancy.

BEXSERO VACCINE



BEXSERO[®]

Meningococcal Group B Vaccine
(rDNA, component, adsorbed)



BEXSERO VACCINE

AGE	PRIMARY DOSE SERIES	INTERVAL	BOOSTER
2 - 5 months	3	No less than 1 month	Yes, at 12 - 23 months
Unvaccinated infants 6 - 11 months	2	No less than 2 month	Yes, at 12 - 23 months with an interval of at least 2 months after the last primary dose
Unvaccinated children, 12 - 23 months	2	No less than 2 month	Yes, between 12-23 months after the last primary dose
Children, 2 - 10 years	2	No less than 2 month	No need yet established
Those over 11 years	2	No less than 1	No need yet

COCLUSION

1. Meningococcal meningitis is fatal disease.
2. It can be prevented by the use of vaccine.
3. All recipient of the vaccine must be given chemoprophylaxis