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**Genus Neisseriae**

The genus neisseriae are negative cocci with adjacent side flattened .Important species of the genus are Neisseria gonorrhea “gonococci” and Neisseria meningitidis “meningococci” are pathogenic for human and typically are found associated with or inside polymorphonuclear leukocytes (PNL).

**The Neisseriae are** :

Human pathogens

- **Neisseria gonorrhea** **(gonococcus**) causes gonorrhea ,neonatal conjunctivitis (ophthalmia neonatorum) and pelvic inflammatory disease (PID).

- **Neisseria meningitidis (meningococcus**) cause acute meningitis.

or sub acute septicemia with petechial rash

**Morphology and Identification**

**A)** **Typical organisms**: they are Gram negative, non motile, non spore forming diplococcus, 0.8 μm in diameter, and kidney in shape, arranged in pairs. Pathological spp. are intracellular while non pathogenic are extracellular.

**B)** **Culture**: in 48 hours on enriched medium gonococci and meningococci form convex elevated mucoid colonies 1-5 mm in diameter colony are opaque, non pigmented and non hemolytic. The non pathogenic spp. can grow in simple media while the pathogenic spp. need enriched media e.g (blood and chocolate

agar) selective media for Neisseria called (Modified Thyer martin

agar).

**C)** **Growth characteristics**: Neisseria grow best under aerobic conditions, ferments carbohydrates producing acid but not gas, they give +ve oxidase test. The microorganism are rapidly killed by drying, sunlight and many disinfectant

***Neisseria meningitidis***

Disease states

i. Meningitis, septicemia, septic arthritis, endocarditis, pneumonia, urethritis

ii. Asymptomatic nasopharyngeal carriers

**Pathogenesis:**

Meningococci are normally carried in nasopharynx of 5-10% of healthy individuals.The meningococcal carrier is important in the transmission of mennigococci and provides reservoir of infection . Like most of the respiratory infection is disseminated by droplet infection ,direct contact and less often by fomites .

*N. Meningitidis* can be the cause of three major diseases.  These three are nasopharyngitis, meningococcal septicemia, and meningococcal meningitis.  Nasopharyngitis is usually a very short illness and sometimes there aren’t even any symptoms.  It is found because it is what will occur before Meningococcal Septicemia.  If the *N. Meningitidis* bacteria colonize in the nasopharynx and spread into the blood stream, the disease becomes known as Meningococcal Septicemia.  This disease has many symptoms including high fever, rash, arthritis, and problems with blood flow.  The problems with blood flow usually result in skin lesions that look like dark red or purple splotches all over the body.  The bacteria will eventually affect the adrenal glands and adrenal insufficiency quickly leads to death . Meninogococci infect the meninges causing severe headache ,stiff neck and vomiting accompanied by delirium and confusion .The route of spread of meningococcius from the nasopharynx to meninges is controversial ,may spread through the olfactory nerves ,or via lymphatic or direct extension through the bone ,others belive that the mennigococcus reach the CNS via blood stream through apriliminary bacteraemia .

**Diagnosis**

* The gold standard of diagnosis is isolation of *N. meningitidis* from sterile body fluid.  A [cerebrospinal fluid](http://en.wikipedia.org/wiki/Cerebrospinal_fluid) (CSF) specimen is sent to the laboratory immediately for identification of the organism.
* Diagnosis relies on culturing the organism on a [chocolate agar](http://en.wikipedia.org/wiki/Chocolate_agar) plate.
* Further testing to differentiate the species includes testing for [oxidase](http://en.wikipedia.org/wiki/Oxidase), [catalase](http://en.wikipedia.org/wiki/Catalase) (all clinically relevant *Neisseria* show a positive reaction) .
* [Serology](http://en.wikipedia.org/wiki/Serology) determines the [subgroup](http://en.wikipedia.org/wiki/Serotype) of the organism.
* [Polymerase chain reaction](http://en.wikipedia.org/wiki/Polymerase_chain_reaction) tests can be used to identify the organism even after antibiotics have begun to reduce the infection. As the disease has a fatality risk approaching 15% within 12 hours of infection, it is crucial to initiate testing as quickly as possible but not to wait for the results before initiating antibiotic therapy.

**Treatment**

i. N. Meningitidis  are sensitive to penicillin , penicillin G in high doses .

ii. In penicillin sensitive individuals ,chloramphenicol as an alternative to therapy iii. Cefotaxime or Ceftriaxone

At the end of course of therapy with pencillin it is important to give eradicative treatment with rifampicin or ciprofloxacin ,because penicillin dose not eradicate N. Meningitidis  from the nasopharynx and patient returning home as a carrier may infect others .

**Prophylaxis**

i. Single dose vaccine is available to polysaccharide capsular antigens A, C, Y and W-135 are available and are good immunogens ,no vaccine is available against group B meningococci because capsular polysaccharide of this group is apoor immunogen .

ii. Rifampin, or Ciprofloxacin , treatment given to close contacts of patient with meningococcal meningitis .

**Neisseria gonorrhea**

**Disease states**

i. Gonorrhea

• Male: acute urethritis, prostatitis and epididymitis if untreated

• Female: may be asymptomatic or exhibit severe discharge

* Cervicitis --> pelvic inflammatory disease --> scarring of fallopian tubes --> sterility, ectopic pregnancies

ii. Disseminated gonococcal infection (DGI)

• Spread from the genitourinary tract, rectum or pharynx to the blood stream

iii. Pharyngitis

• Oral infection

iv. Gonococcal ophthalmia neonatorum

• Conjunctiva infection in the newborn

v. Vulvovaginitis

• In young girls

**Pathogenesis**

*Neisseria gonorrhoeae*infections are acquired by sexual contact and usually affect the mucous membranes of the urethra in males and the endocervix and urethra in females, although the infection may disseminate to a variety of tissues. The pathogenic mechanism involves the attachment of the bacterium to nonciliated epithelial cells via pili and the production of lipopolysaccharide endotoxin. Similarly, the lipopolysaccharide of *Neisseria meningitidis* is highly toxic, and it has an additional virulence factor in the form of its antiphagocytic capsule.

**Diagnosis**

The clinical syndromes associated with *N. gonorrhoeae*are typically diagnosed by history and physical examination, but a microbiologic diagnosis is required due to the lack of sensitivity and specificity of the clinical diagnosis. In general, nucleic acid amplification testing (NAAT) is the test of choice for the microbiologic diagnosis of *N. gonorrhoeae* infection, although culture remains an important diagnostic tool when antibiotic resistance is suspected. If NAAT methods are unavailable, microscopy (for men), culture, antigen detection, and genetic probe methods can be used with endocervical or urethral swabs to diagnose urogenital gonorrhea.

**Treatment**

* Empiric therapy options.
* Most patients treated with antibiotics for possible co-infections with *Chlamydia*.
* Eye treatments for infants with 1 hour of delivery.
* Drug of choice is broad spectrum cephalosporins – ceftriaxone or cefixime