Bacteriology

Spirochetes

Three important genera:

1. Treponema
2. Borrelia
3. Leptospira

Treponema pallidum

Causes syphilis

Organism:

- Spirochetes with 6-14 regularly spaced spirals
- Its length is the length of RBC
- Has characteristic motility
- Can not be cultured in vitro
- In whole blood stored at 4°C. trep remain viable for 24 hrs. which is of great importance in blood transfusions
- Drying kills trep rapidly as does elevation of temperature (42°C)
- Can not be stained by the biological stains because it is very thin.
- T. pallidum produce hyaluronidase → breakdown of hyaluronic acid → enhance invasiveness of tissue.
- **Cardiolipin**: is an important component of T. pallidum antigen that enhance reagin production in the patient's serum which is antibody-like substance.
- Spirochetes are very thin, so they can not be seen unless using.  

<table>
<thead>
<tr>
<th>Silver impregnation</th>
<th>Deposit to increase</th>
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<tbody>
<tr>
<td>Dark-field illumination</td>
<td>notice character motility</td>
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<td>↓</td>
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<tr>
<td>Its diameter</td>
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Visible under microscope
- It posses Endoflagella for its movement

**Pathogenesis and pathology**

- Syphilis is sexually transmitted disease (STD) with incubation period about 3 weeks.
- Stages of syphilis:

**Primary stage:**

Here a primary lesion develops on the genitalia or perianal sites where T. Pallidum multiply at the sites of entry in skin or mucous membranes of the genitalia.

The lesion (chancre) is a papule that will breakdown forming painless ulcer (hard chancre) (in 6 weeks)
The regional lymph nodes become enlarged

**Secondary stage**

- 6-8 weeks after primary stage
- Generalized symptoms (malaise, fever headache).
- With characteristic maculopapular rash and generalized lymphadenopathy
- Mucosal ulcers in the mouth
- Warty lesions on perianal region & vulvae
- During primary & secondary stages, the patient is infectious.

- **Latent stage**
  
  Features of secondary syphilis resolve and patient appear clinically well (controlled by immunologic factors) but still have syphilis infection → late complication of syphilis → No complication

- **Late syphilis stage**
  
  A-Tertiary Syphilis: 3-30 years after infection involve:
  
  - Skin
  - Mucous membranes of upper respiratory tract
  - Bones
  - Joints

Here the lesions are called "gumma" consist of granulomatous tissue
(while the lesions in primary stage are called hard chancre & consist acute inflammation).

B- Quaternary syphilis: 5-30 year after inf. Involve:
- Cardiovascular system
- Central nervous system

**Congenital syphilis**

Pregnant syphilitic woman can transmit Treponema pallidum to the fetus through placenta & the fetus either.

- Die
- Still born
- Born live with congenital syphilitic symptom
  - Keratitis
  - Huchinson's teeth
  - CNS anomalies

**Lab diagnosis of syphilis**

A. **Specimens:**

1. Tissue fluid from the lesions → demonstrate the spirochete (T.pallidum)
2. Blood serum → for serology tests

B. **Darkfield examination**

A drop of lesion exudates → examined under darkfield microscope for the characteristic motility.

C. **Immunofluorescence**
A drop of lesion exudates → stained with fluorescein labeled antitreponeme serum → examined under immunofluorescence microscope → fluorescent spirochetes.

D. Serological tests for syphilis (STS)

1. Non treponemal antigen test

   The Ag used → Cardiolipin

   The Ab in patient serum → Reagin (Ab-like substance)

   a. Flocculation tests (clumping):

      - VDRL (venereal Disease Research Laboratories)
      - RPR (Rapid Plasma Reagin)

      ❖ During treatment of the disease cardiolipin fall → these tests appear negative → so they are useful for monitoring the treatment.

   b. Complement fixation tests (CFT)

      - Wasserman test
      - Kolmer test

      ❖ Non treponemal Ag tests may show False-positive results, because regain may appear in patients serum due to many other infections (e.g.: malaria, leprosy, autoimmune disease, viral & bacterial pneumonia)

2. Treponemal Antibody tests

   a. Fluorescent treponemal antibody (FTA ABS):
Is indirect immunofluorescent test (killed treponema + patient serum (Ab) + fluorescene labeled antihuman gamma globulin → fluorescent treponema)

- Has high sensitivity & specificity
- First test become positive at early syphilis
- Remain +ve many years after infection
- Not used to judge treatment

b. Treponema pallidum hemagglutination (TPHA):

Treponema + RBCs + patient serum (Ab) → Agglutination.

- This test is similar to (FTA ABS) but appear later in infection.

c. Treponema pullidum immobilization test (TPI):

Live motile treponema (seen under dark field microscopy) + patient's serum (Ab) + complement → treponema stop moving (immobile)

- This test not used because the live treponema is dangerous.

**Immunity**

Infected person do not get super infection but treated cases becomes again susceptible to infection (No immunity).

**Treatment**

Penicillin is the treatment of choice, but the rate of killing is slow because of metabolic inactivity & slow multiplication.
Control:

1. Prompt & adequate treatment
2. Follow up the source of infection
3. Safe sex
   - It is important to consider possibility of syphilis when any one of the sexually transmitted diseases has been found (G.C, Chlamydia) Since they transmit in the same way.

Diseases related to syphilis

Caused by treponemes closely related to T. pallidum:

1. Bejel [endemic syphilis]

Borrelia recurrentis

Causing Relapsing Fever

- Epidemic: Transmitted by body louse
- Endemic: transmitted by Ticks

Organism:

- Irregular spirals
- Flexible & motile
- Can be stained by bacteriological stains and blood stains (Giems).  

**Culture:**
- In fluid media containing blood or serum
- And in chick embryos
- Borrelia recurrentis can survive several months at 4°C in infected blood
- Antigenic variations occurs in the course of infection.

**Pathology & pathogenesis**
- Incubation period 3-10 days
- Chills & abrupt rise in temperature (3-5 days) (i.e. B. recur abound patients blood)
- Decline in fever (4-10 days)
- Second attacks of fever
- Decline → —— (3-10 times)

This is due to the antigenic variation so the body produce antibodies against that variant at each attacks → then the organism produce other variant.

- Fatal cases of relapsing fever show spirochetes in great numbers in spleen, liver, kidneys & GIT.

**Lab diagnosis**
- **Specimen:** Blood obtained during rise in fever
- **Smears:** Thick and Thin blood smears stained with Giemsa stain shows the spirochetes
- **Serology:** CFT
Leptospirae

- Cause leptospirosis

Organism:
- Tightly coiled thin flexible spirochetes
- One end is bent forming a hook
- With rotation motion and no flagella
- Very fine so not stained but by silver impregnation.

Culture
Aerobic culture on protein rich semisolid media.

Pathogenesis
After 1-2 weeks of ingestion of contaminated water or found → febrile onset (spirochetes in blood) → infect liver & kidney → Jaundice → improvement → second phase of infection (IgM antibody titer increase) → (aseptic meningitis).
- The picture varies in different species

Lab diagnosis
Specimen is blood for:
- Dark field microscopy
- Thick smear stained with Giemsa
- Culture
- Serology (agglutination)
Spirochetes of the mouth and mucous membranes

Occur in normal mouth and on normal genitalia. They are anaerobic harmless saprophytes.

At injury to mucous membranes; nutritional deficiency or herpes simplex infection → these normal spirochetes together with anaerobic fusiform bacilli will cause ulcerative gingivostomatitis (trench mouth) → cause ulcerative tonsillitis called Vincent's angina