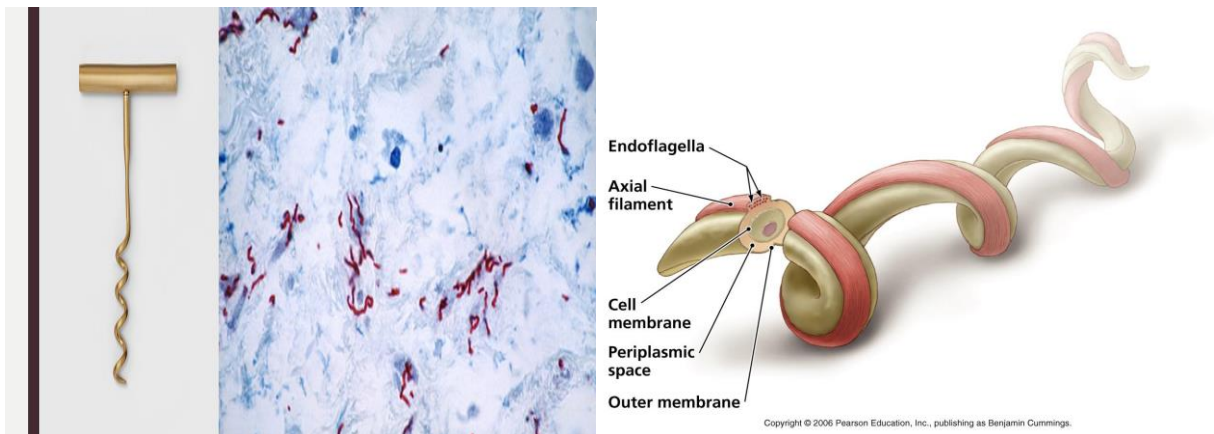


Treponema pallidum

Treponema pallidum is a gram-negative slender spiral shaped bacterium (spirochete) with regular coils and tapering ends, often growing in clusters. Rapid rotation about its axial filaments and endoflagella within periplasmic space at each end allow a corkscrew like motility. The helical structure of *T. pallidum* allows it to move in a corkscrew motion through the viscous media (mucus media).



T. pallidum is micro aerophilic and an obligate internal parasite. Natural infection with *T. pallidum* is limited to the human host that causes syphilis. Human infection is usually transmitted by sexual contact and the infectious lesion is on the skin or mucous membranes of genitalia.

Acquired syphilis

Is a chronic systemic venereal disease with multiple clinical presentations, entering the host via breaches or break in the skin or by penetrating the mucous membrane of genitalia. Spirochetes multiply locally at the site of entry, and some spread to nearby lymph nodes and then reach the bloodstream. Within 2-10 weeks after infection, a papule develops to form an ulcer (“hard chancre”). This “primary lesion” always heals spontaneously.

Primary Syphilis - *Chancre*

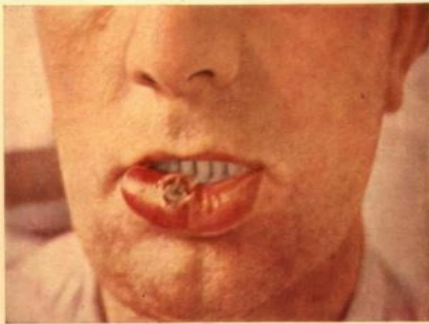


Fig. 171. Primary Syphilis of the Lower Lip. A chancre appearing on the lower lip has the same clinical appearance as one appearing on the genital mucosa. This lesion may simulate squamous cell carcinoma.

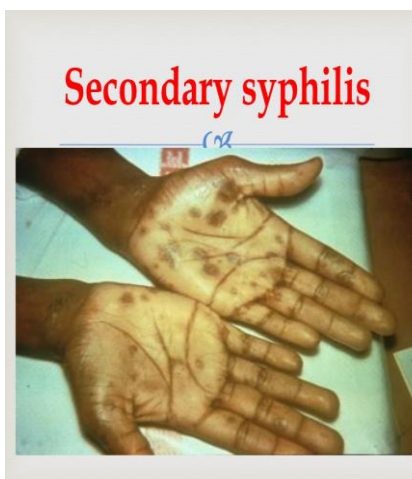


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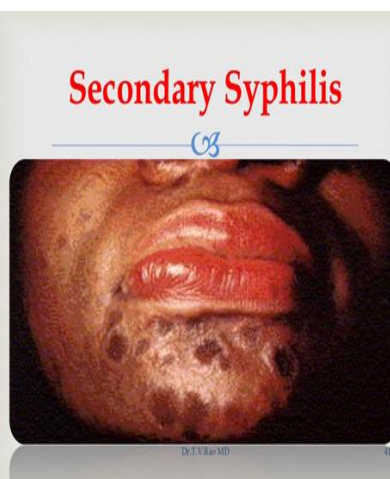
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After 2-10 weeks, the “secondary” lesions appear. These consist of a red maculopapular rash anywhere on the body, including the hands and feet, and moist, pale papules (condylomas) in the anogenital region, axillae, and mouth. The patient may also have syphilitic meningitis, chorioretinitis, hepatitis, nephritis, or periostitis. The secondary lesions also subside spontaneously.

Syphilitic infection may remain subclinical, and the patient may pass through the primary or secondary stage (or both) without symptoms or signs yet develop tertiary lesions.



Secondary syphilis



Secondary Syphilis

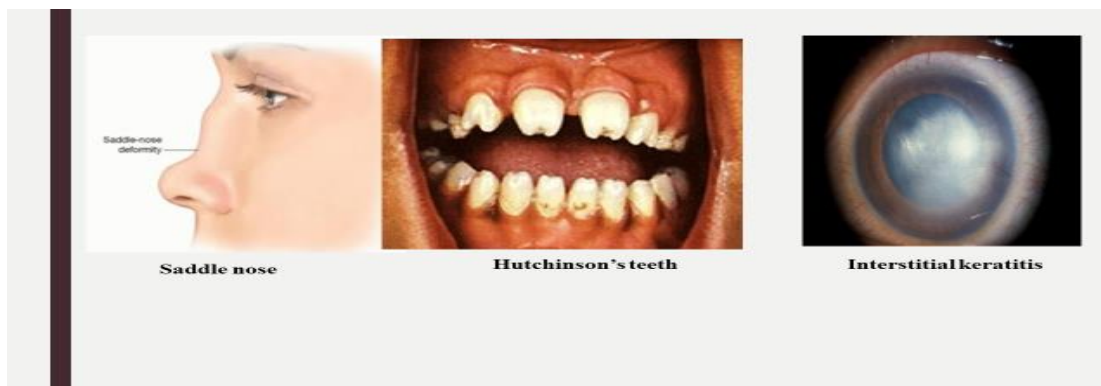


In 30%, the untreated infection remains latent. In the remainder, the disease progresses to the “tertiary stage” characterized by the development of granulomatous lesions in the skin, bones, and liver; degenerative changes in the central nervous system (meningo vascular syphilis causing infected individuals to experience insomnia and changes in personality; or cardiovascular lesions. Another major complication of syphilis is its ability to increase the likelihood of transmission of HIV.

In whole blood or plasma stored at 4°C, organisms remain viable for at least 24 hours, which is of potential importance in blood transfusions.

Congenital Syphilis

A pregnant woman with syphilis can transmit *T. pallidum* to the fetus through the placenta beginning in the 10th-15th weeks of gestation. Some of the infected fetuses die, and miscarriages result; others are stillborn at term. Others are born live but develop the signs of congenital syphilis in childhood, including interstitial keratitis, Hutchinson's teeth, saddle nose, and a variety of central nervous system anomalies. Adequate treatment of the mother during pregnancy prevents congenital syphilis

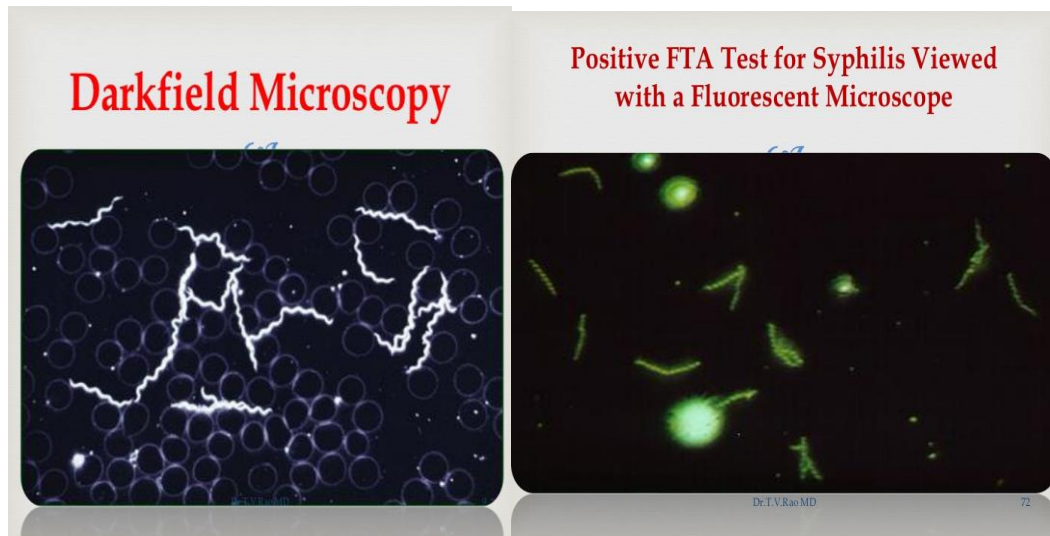


Diagnosis

T. pallidum cannot be cultured in the lab and therefore cannot be investigated using conventional lab techniques because it cannot survive outside of mammalian cells. They do not stain well with aniline dyes. Clinical specimens include exudate or pus, tissue biopsy and serum.

1-Tests for direct detection of *T.pallidum*

- Animal inoculation (grown in the testicles of experimentally inoculated rabbit).
- Dark field microscopy (morphology and movement)
- Direct fluorescent antibody test (DFA-TP) (smear is stained with fluorescein-labeled anti- *T.pallidum* globulin and examined under fluorescent microscope)
- Nucleic acid amplification test (PCR)

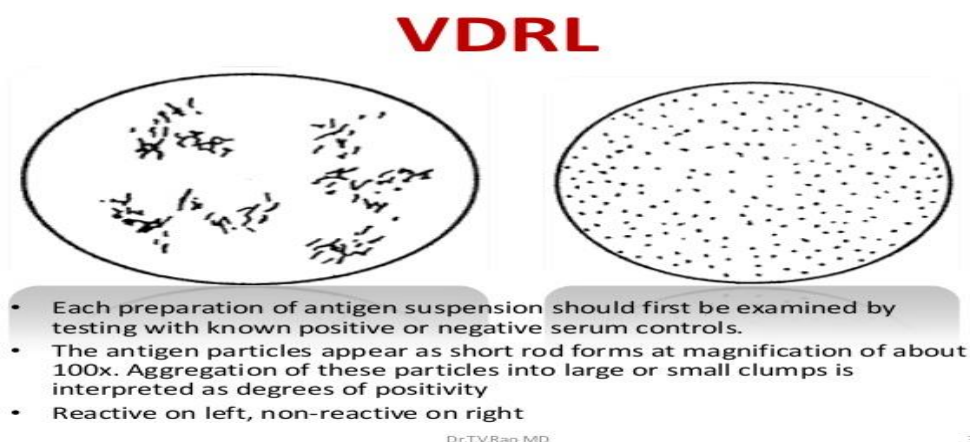


2- Serological tests (antibody tests)

-Non treponemal serologic tests

Non treponemal tests are called "non treponemal" because they detect antibodies that are not specifically directed against *T. pallidum* but may also be produced in several other conditions. The tests are highly sensitive but, false-positive results can be caused by other infections. A positive screening result must be confirmed with more specific treponemal tests.

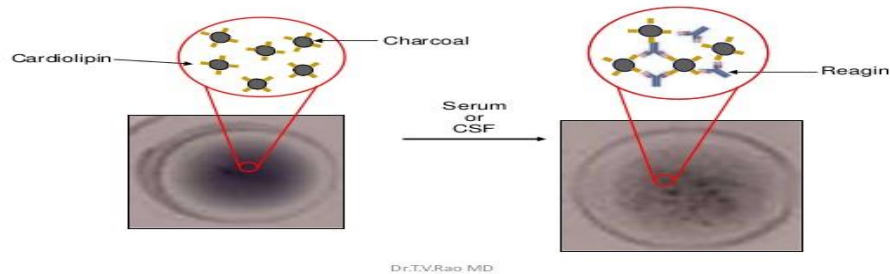
1-VDRL (Venereal Disease Research Laboratory test): This test measures anti-lipid antibodies called reagin, which are formed by the host in response to lipids released from damaged cells early in infection with *T.pallidum*, and lipid-like material from the treponemal cell surface. A combination of reagin and VDRL antigen form microscopic clumping called flocculation.



2-RPR (Rapid Plasma Reagin test): The RPR antigen suspension is a carbon particle cardiolipin. The reagin binds to the test antigen that cause flocculation.

Serologic Tests for Syphilis: Non-Treponemal Assays

- RPR and VDRL are agglutination assays



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-Treponemal serologic tests

These blood tests detect antibodies that specifically target *T. pallidum*. They are highly specific for syphilis, meaning other conditions are unlikely to cause a positive result.

1-FTA-ABS (Fluorescent treponemal antibody absorption)

2-TP-PA (*T. pallidum* particle agglutination assay)--this test is sometimes performed instead of FTA-ABS because it is more specific.

Treatment

Penicillin was the first known effective antibiotic for *T. pallidum* and remains the treatment of choice today. *T. pallidum* is also susceptible to amoxicillin and ceftriaxone, have been shown to be curative.

Erythromycin, tetracycline and azithromycin are also able to inhibit *T. pallidum* but not as efficiently as the β -lactam antibiotics. Thus macrolides are relegated to second line antibiotic treatment status. Chloramphenicol, has been successful in treating syphilis due to concentrates in the CNS, it has utility in treating neurosyphilis.

