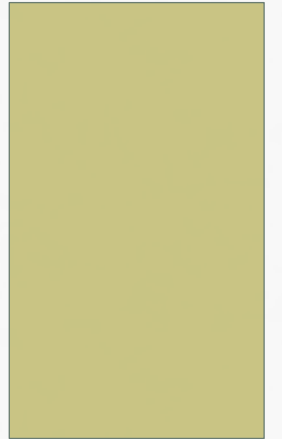


REPRODUCTIVE INFECTIONS

BACTERIAL



Normal Flora of the Genitourinary Tract

- The vaginal flora of adult women consists primarily of *Lactobacillus* species.
- Lactobacilli are responsible for producing the acid that keeps the pH of the adult woman's vagina low.
- Before puberty & after menopause, when estrogen levels are low, lactobacilli are rare & the vaginal pH is high.
- Lactobacilli appear to prevent the growth of potential pathogens, since their suppression by antibiotics can lead to overgrowth by *C. albicans*.
- Overgrowth of *C. albicans* can result in *Candida* vaginitis.

Normal Flora of the Genitourinary Tract

- The vagina is located close to the anus & can be colonized by members of the fecal flora.
- Women who are prone to recurrent UTI harbor organisms such as *E. coli* & *Enterobacter* in the introitus.
- About 15% to 20% of women of childbearing age carry group B streptococci in the vagina. This organism is an important cause of sepsis & meningitis in the newborn & is acquired during passage through the birth canal.
- The vagina is colonized by *S. aureus* in approximately 5% of women, which predisposes them to toxic shock syndrome.

Normal Flora of the Genitourinary Tract

- Urine in the bladder is sterile in a healthy person, but during passage, through the outermost portions of the urethra, it often becomes contaminated with
 - *S. epidermidis*
 - coliforms
 - diphtheroids
 - non-hemolytic streptococci.
- The area around the urethra of women & uncircumcised men contains secretions that carry *Mycobacterium smegmatis*, an acid-fast organism.
- The skin surrounding the genitourinary tract is the site of *Staphylococcus saprophyticus*, a cause of UTI in women.

Neisseria
Gonorrhoeae

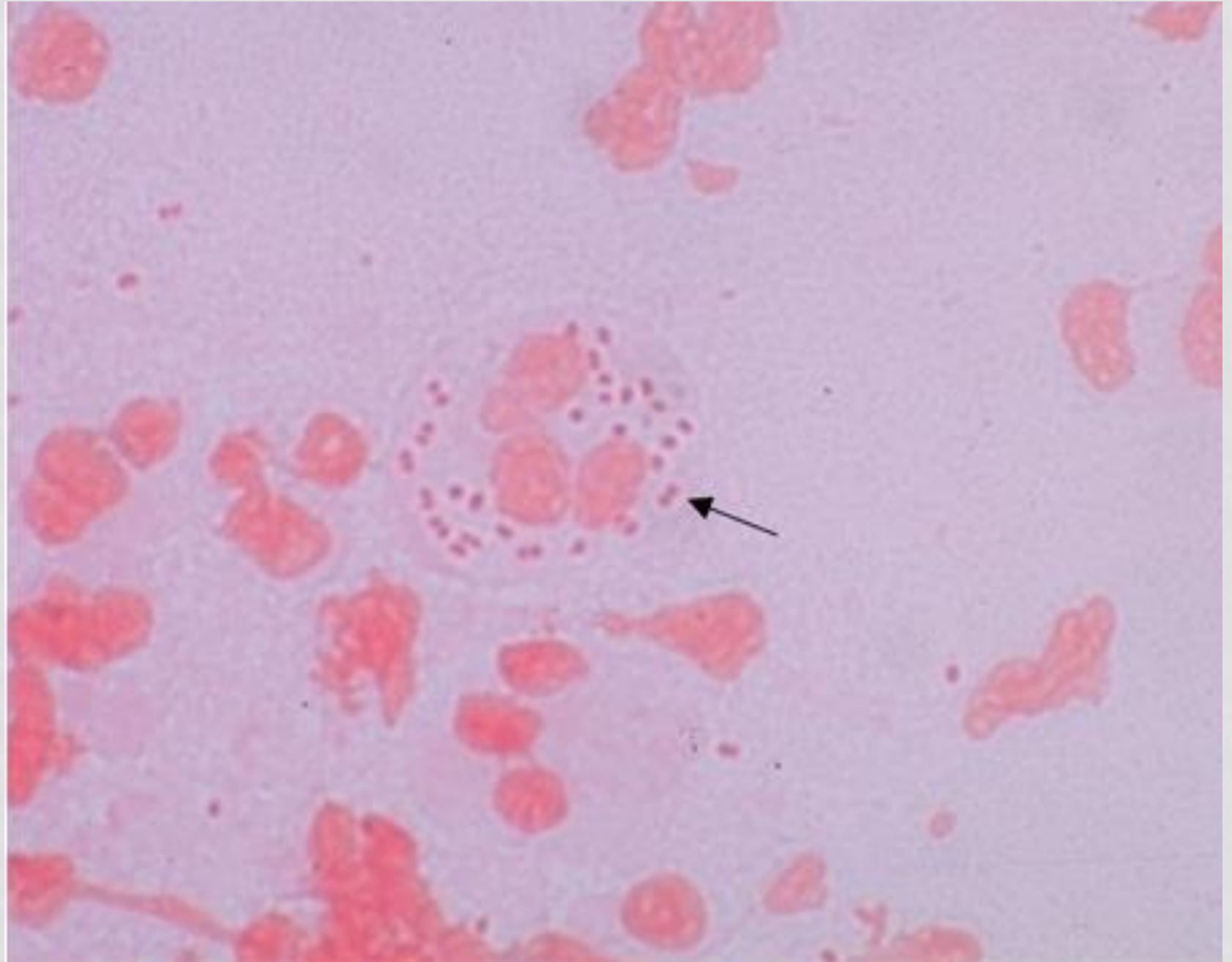
NEISSERIA GONORRHOEAE

- ❖ Neisseriae are gram-negative cocci that resemble paired kidney beans.
- ❖ *N. gonorrhea* causes gonorrhea,
- ❖ it is the second most common notifiable bacterial disease in the USA.

IMPORTANT PROPERTIES

- **Neisseriae has no polysaccharide capsule but has multiple serotypes based on the antigenicity of its pilus protein.**
- **Gonococci have three outer membrane proteins (proteins I, II, & III). Protein II plays a role in the attachment of the organism to cells & varies antigenically as well.**
- **Neisseriae contain a lipooligosaccharide (LOS). endotoxin in their outer membrane.**

Neisseria gonorrhoeae—
Gram stain.
Arrow points to
typical "kidney-
bean" shaped
gram-negative
diplococci within a
neutrophil.



IMPORTANT PROPERTIES

- The growth of both gonococci is inhibited by toxic trace metals & fatty acids found in certain culture media, e.g., blood agar plates.
- They are therefore cultured on "chocolate" agar containing blood heated to 80°C, which inactivates the inhibitors.
- Neisseriae are oxidase-positive, i.e. they possess the enzyme cytochrome c.
- This is an important laboratory diagnostic test in which colonies exposed to phenylenediamine turn purple or black as a result of oxidation of the reagent by the enzyme.

NEISSERIA GONORRHOEAE

PATHOGENESIS & EPIDEMIOLOGY

- **Gonococci cause disease only in humans.**
- **The organism is usually transmitted sexually;**
- **newborns can be infected during birth.**
- **Because gonococcus is quite sensitive to dehydration & cool conditions, sexual transmission favors its survival.**
- **Gonorrhea is usually symptomatic in men but often asymptomatic in women.**

NEISSERIA GONORRHOEAE
PATHOGENESIS & EPIDEMIOLOGY

- **Pili constitute one of the most important virulence factors, because they mediate attachment to mucosal cell surfaces & are antiphagocytic.**
- **Two virulence factors in the cell wall are LOS (a modified form of endotoxin) & the outer membrane proteins.**

NEISSERIA GONORRHOEAE
PATHOGENESIS & EPIDEMIOLOGY

- The organism's IgA protease can hydrolyze secretory IgA, which could block attachment to the mucosa.
- Gonococci have no capsules.

NEISSERIA GONORRHOEAE

PATHOGENESIS & EPIDEMIOLOGY

- The main host defenses against gonococci are antibodies (IgA & IgG), complement, & neutrophils.
- Antibody-mediated opsonization & killing within phagocytes occur, but repeated gonococcal infections are common primarily as a result of antigenic changes of pili & the outer membrane proteins.
- Gonococci infect primarily the mucosal surfaces, e.g., the urethra & vagina, but dissemination occurs.

NEISSERIA GONORRHOEAE

PATHOGENESIS & EPIDEMIOLOGY

- **Certain strains of gonococci cause disseminated infections more frequently than others.**
- **The most important feature of these strains is their resistance to being killed by antibodies & complement.**
- **The mechanism of this "serum resistance" is uncertain, but the presence of a porin protein (porin A) in the cell wall, which inactivates the C3b component of complement, appears to play an important role.**

NEISSERIA GONORRHOEAE

PATHOGENESIS & EPIDEMIOLOGY

- **The occurrence of a disseminated infection is a function not only of the strain of gonococcus but also of the effectiveness of the host defenses.**
- **Persons with a deficiency of the late-acting complement components (C6–C9) are at risk for disseminated infections, as are women during menses & pregnancy.**
- **Disseminated infections usually arise from asymptomatic infections, indicating that local inflammation may deter dissemination.**

CLINICAL FINDINGS

- **Gonococci cause both localized infections, usually in the genital tract, & disseminated infections with seeding of various organs.**
- **Gonococci reach these organs via the bloodstream (gonococcal bacteremia).**
- **Gonorrhea in men is characterized primarily by urethritis accompanied by dysuria & a purulent discharge. Epididymitis can occur.**

CLINICAL FINDINGS

- In women, infection is located primarily in the endocervix, causing a purulent vaginal discharge & intermenstrual bleeding (cervicitis).
- The most frequent complication in women is an ascending infection of the uterine tubes (salpingitis, PID), which can result in sterility or ectopic pregnancy as a result of scarring of the tubes.
- Disseminated gonococcal infections (DGI) commonly manifest as arthritis, tenosynovitis, or pustules in the skin.

CLINICAL FINDINGS

- Disseminated infection is the most common cause of septic arthritis in sexually active adults.
- The clinical diagnosis of DGI is often difficult to confirm using laboratory tests because the organism is not cultured in more than 50% of cases.
- Other infected sites include the anorectal area, throat, & eyes.
- Anorectal infections occur chiefly in women & homosexual men. They are frequently asymptomatic, but a bloody or purulent discharge (proctitis) can occur.
- In the throat, pharyngitis occurs, but many patients are asymptomatic.

CLINICAL FINDINGS

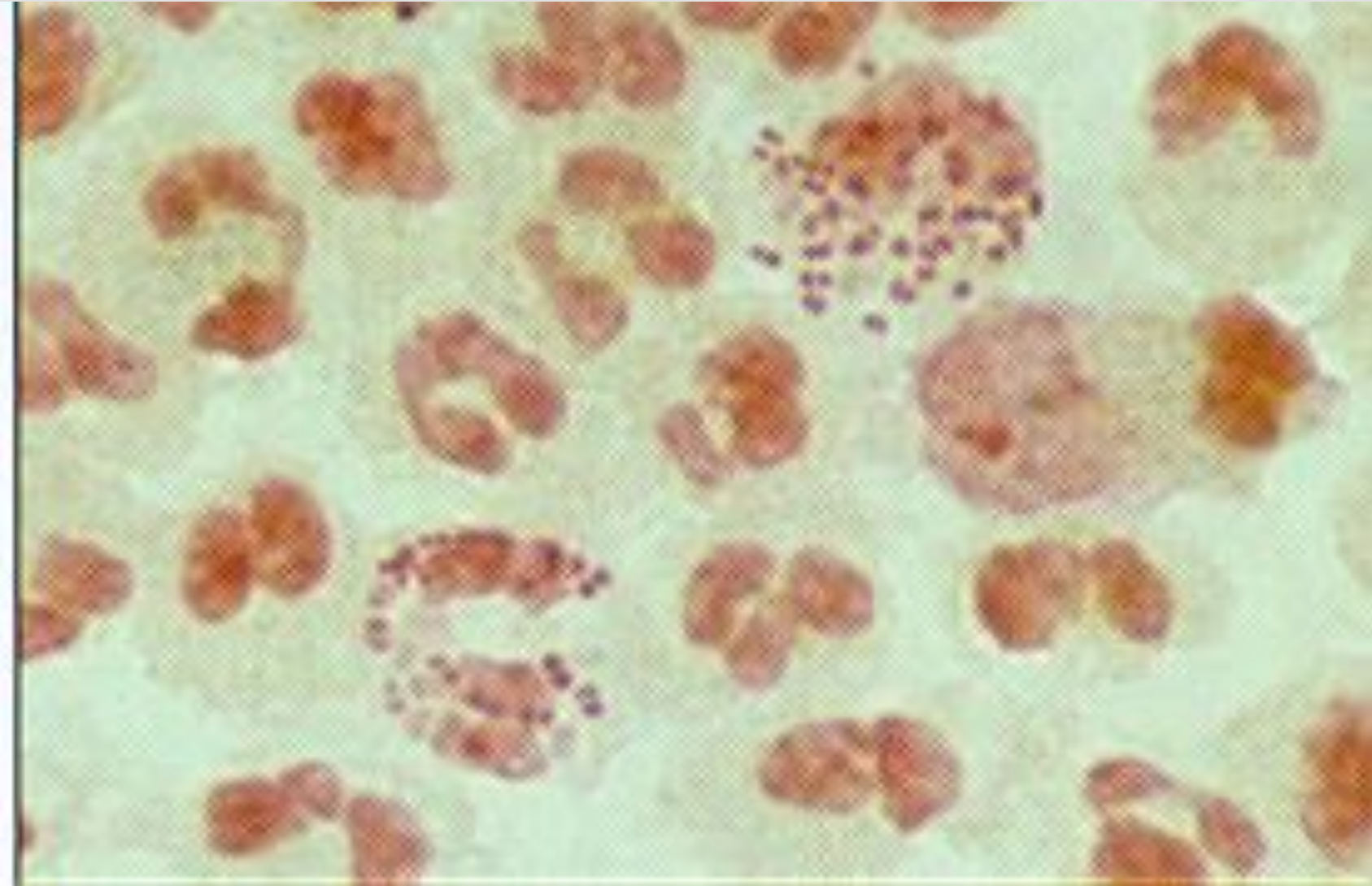
- In newborns, purulent conjunctivitis (ophthalmia neonatorum)
- The incidence of gonococcal ophthalmia has declined greatly in recent years because of the widespread use of prophylactic erythromycin eye ointment (or silver nitrate) applied shortly after birth.
- Gonococcal conjunctivitis also occurs in adults due to the transfer of organisms from the genitals to the eye.

LABORATORY DIAGNOSIS IN MEN

- **The diagnosis of localized infections depends on Gram staining & culture of the discharge.**
- **In men, finding gram-negative diplococci within PMNs in a urethral discharge specimen is sufficient for diagnosis.**

LABORATORY DIAGNOSIS IN WOMEN

- In women, using the Gram stain alone can be difficult to interpret; therefore, cultures should be done.
- Gram stains on cervical specimens can be falsely positive because of gram-negative diplococci in the normal flora & can be falsely negative because of the inability to see small numbers of gonococci when using the oil immersion lens.
- Cultures must also be used in diagnosing suspected pharyngitis or anorectal infections.



Neisseria gonorrhoeae

LABORATORY DIAGNOSIS

- Specimens from mucosal sites, such as the urethra & cervix, are cultured **on Thayer-Martin medium**, which is a chocolate agar containing antibiotics (vancomycin, colistin, trimethoprim, & nystatin) to suppress the normal flora.
- The finding of an oxidase-positive colony composed of gram-negative diplococci is sufficient to identify the isolate as a member of the genus *Neisseria*.
- Note that specimens from sterile sites, such as blood or joint fluid, can be cultured on chocolate agar without antibiotics because there is no competing normal flora.

LABORATORY DIAGNOSIS

- Two rapid tests that detect the presence of gonococcal nucleic acids in patient specimens are widely used as screening test. These tests are highly sensitive & specific.
- The amplification tests can be used on urine samples, obviating the need for more invasive collection techniques.
- Serologic tests to determine the presence of antibodies to gonococci in the patient's serum are *not* useful for diagnosis.

TREATMENT

- Ceftriaxone is the treatment of choice in uncomplicated gonococcal infections.
- Oral cefixime is an alternative drug of choice.
- Spectinomycin or ciprofloxacin should be used if the patient is allergic to penicillin.
- Because mixed infections with *C. trachomatis* are common, tetracycline should also be prescribed.

TREATMENT

- A follow-up culture should be performed 1 week after the completion of treatment to determine whether gonococci are still present.
- Treatment of complicated gonococcal infections, such as PID, typically requires hospitalization.

-

PREVENTION

- The prevention of gonorrhea involves the use of condoms & the prompt treatment of symptomatic patients & their contacts.
- Cases of gonorrhea must be reported to the public health department to ensure proper follow-up.
- A major problem is the detection of asymptomatic carriers.
- Gonococcal conjunctivitis in newborns is prevented most often by the use of erythromycin ointment.
- Silver nitrate drops are used less frequently.
- No vaccine is available.

Chlamydiae

CHLAMYDIAE: INTRODUCTION

**Chlamydiae are obligate intracellular bacteria
They can grow *only* within cells.**

They are the agents of:

- **Common STDs, such as urethritis & cervicitis**
- **Lymphogranuloma venereum**

DISEASES

***Chlamydia trachomatis* causes**

- **Genital tract (urethritis, lymphogranuloma venereum) infections.**
- ***C. trachomatis* is the most common cause of STDs in USA**
- **Infection with *C. trachomatis* is also associated with Reiter's syndrome, an autoimmune disease.**

IMPORTANT PROPERTIES

- They have a rigid cell wall but do not have a typical peptidoglycan layer.
- Their cell walls resemble those of gram-negative bacteria but lack muramic acid.
- Chlamydiae have a replicative cycle different from that of all other bacteria.

IMPORTANT PROPERTIES

- All chlamydiae share a group-specific lipopolysaccharide antigen detected by complement fixation tests.
- They also possess species-specific & immunotype-specific antigens (proteins), which are detected by immunofluorescence.

TRANSMISSION & EPIDEMIOLOGY

- ***C. trachomatis* infects only humans & is usually transmitted by close personal contact, e.g., sexually or by passage through the birth canal.**
- **Individuals with asymptomatic genital tract infections are an important reservoir of infection for others.**

TRANSMISSION & EPIDEMIOLOGY

- STDs caused by *C. trachomatis* occur worldwide
- Patients with a STD are coinfectd with both *C. trachomatis* & *Neisseria gonorrhoeae* in approximately 10% to 30% of cases.
- **Types D–K cause genital tract infections, which are occasionally transmitted to the eyes or the respiratory tract.**

PATHOGENESIS & CLINICAL FINDINGS

In men,

- **it is a common cause of non-gonococcal urethritis characterized by a urethral discharge. This infection may progress to epididymitis, prostatitis, or proctitis.**

In women,

- **cervicitis develops & may progress to salpingitis & pelvic inflammatory disease (PID). Repeated episodes of salpingitis or PID can result in infertility or ectopic pregnancy.**

PATHOGENESIS & CLINICAL FINDINGS

- **Infants born to infected mothers often develop muco-purulent conjunctivitis (neonatal inclusion conjunctivitis) 7 to 12 days after delivery, & some develop chlamydial pneumonitis 2 to 12 weeks after birth.**
- **Chlamydial conjunctivitis also occurs in adults as a result of the transfer of organisms from the genitals to the eye.**
- **Patients with genital tract infections caused by *C. trachomatis* have a high incidence of Reiter's syndrome, which is characterized by urethritis, arthritis, and uveitis.**

PATHOGENESIS & CLINICAL FINDINGS

LYMPHOGRANULOMA VENEREUM

- ***C. trachomatis* L1–L3 immunotypes cause lymphogranuloma venereum, a STD with lesions on genitalia & in lymph nodes.**
- **Infection by *C. trachomatis* leads to formation of antibodies & cell-mediated reactions but not to resistance to reinfection or elimination of organisms.**

Lymphogranuloma venereum is caused by the invasive serovars L1, L2, or L3 of *Chlamydia trachomatis*.

This young adult experienced the acute onset of tender, enlarged lymph nodes in both groins.



LABORATORY DIAGNOSIS

- Chlamydiae form cytoplasmic inclusion, which can be seen with special stains (e.g., Giemsa stain) or immunofluorescence.
- The Gram stain is not useful.
- In exudates, the organism can be identified within epithelial cells by fluorescent antibody staining or hybridization with a DNA probe.
- Chlamydial antigens can also be detected in exudates or urine by ELISA.
- A PCR-based test using the patient's urine can also diagnose chlamydial STD.
- Tests not involving culture are now more commonly used than culture-based tests.

LABORATORY DIAGNOSIS

- Chlamydiae can be grown in cell cultures treated with cycloheximide, which inhibits host cell but not chlamydial protein synthesis, thereby enhancing chlamydial replication.
- In culture, *C. trachomatis* forms inclusions containing glycogen, whereas *C. psittaci* & *C. pneumoniae* form inclusions that do not contain glycogen.
- The glycogen-filled inclusions are visualized by staining with iodine.
- Exudates from the eyes, respiratory tract, or genital tract give positive cultures in about half of cases.

LABORATORY DIAGNOSIS

- **Serologic tests are rarely helpful in diagnosing disease caused by *C. trachomatis* because the frequency of infection is so high that many people already have antibodies.**

TREATMENT

- All chlamydiae are susceptible to tetracyclines, such as doxycycline, & macrolides, such as erythromycin & azithromycin.
- The drug of choice for *C. trachomatis* STDs is azithromycin.
- Because the rate of coinfection with gonococci & *C. trachomatis* is high, any patient with a diagnosis of gonorrhea should also be treated for *C. trachomatis* with azithromycin.
- The drug of choice for neonatal inclusion conjunctivitis & pneumonia is oral erythromycin.

PREVENTION

- There is no vaccine against any chlamydial disease.
- The best preventive measure against *C. trachomatis* STDs is to limit transmission by prompt treatment of both the patient & the sexual partners, including persons who are asymptomatic.
- Sexual contacts should be traced, & those who had contact within 60 days should be treated.
- Several types of STDs are often present simultaneously. Thus, diagnosis of one requires a search for other causative agents.

PREVENTION

- Oral erythromycin given to newborn infants of infected mothers can prevent inclusion conjunctivitis & pneumonitis caused by *C. trachomatis*.
- Erythromycin ointment used to prevent neonatal gonococcal conjunctivitis is much less effective against neonatal chlamydial conjunctivitis. Oral erythromycin should be used.

Treponema Pallidum

Spirochetes: Introduction

Three genera of Spirochetes cause human infection:

1. *Treponema*, which causes syphilis & nonvenereal treponematoses;
2. *Borrelia*, which causes Lyme disease & relapsing fever;
3. *Leptospira*, which causes leptospirosis

Spirochetes: Introduction

- ✿ Spirochetes are thin-walled, flexible, spiral rods.
- ✿ They are motile through undulating axial filaments under the outer sheath.
- ✿ Treponemes are so thin that they are seen only by darkfield microscopy, silver impregnation, or immunofluorescence.



Treponema pallidum—Dark field microscopy. The coiled form of this spirochete is in the center of the field

Axial Filament - found only in spirochetes (flexible spirals)



Treponema pallidum

Treponema Pallidum

Important Properties

-  *T. pallidum* has not been grown on bacteriologic media or cell culture.
-  Nonpathogenic treponemes, part of the normal flora of human mucous membranes, can be cultured.
-  *T. pallidum* grows very slowly. The medical importance of that fact is that antibiotics must be present at an effective level for several weeks to kill the organisms & cure the disease.
-  For example, benzathine penicillin is the form of penicillin used to treat primary & secondary syphilis because the penicillin is released very slowly from this depot preparation & bactericidal concentrations are present for weeks after administration of the antibiotic.

Treponema Pallidum

Important Properties

-  The antigens of *T. pallidum* induce specific antibodies, which can be detected by immunofluorescence or hemagglutination tests in the clinical laboratory.
-  They also induce nonspecific antibodies (reagin), which can be detected by the flocculation of lipids (cardiolipin) extracted from normal mammalian tissues, e.g., beef heart.
-  Both specific anti-treponemal antibodies & nonspecific reagin are used in the serologic diagnosis of syphilis.

Transmission

- ✿ *T. pallidum* is transmitted from spirochete-containing lesions of skin or mucous membranes (e.g., genitalia, mouth, and rectum) of an infected person to other persons by intimate contact.
- ✿ It can also be transmitted from pregnant women to their fetuses.
- ✿ Rarely, blood for transfusions collected during early syphilis is also infectious.

Epidemiology

- ✿ **Syphilis occurs worldwide, & its incidence is increasing.**
- ✿ **It is one of the leading notifiable diseases in the United States. Many cases are believed to go unreported, which limits public health efforts.**
- ✿ **There has been a marked increase in the incidence of the disease in homosexual men in recent years.**

Pathogenesis & Clinical Findings

- ✿ *T. pallidum* produces no important toxins or enzymes.
- ✿ The organism often infects the endothelium of small blood vessels, causing endarteritis.
- ✿ This occurs during all stages of syphilis but is particularly important in the pathogenesis of the brain & cardiovascular lesions seen in tertiary syphilis.

Pathogenesis & Clinical Findings

In primary syphilis

- ✿ The Spirochetes multiply at the site of inoculation & a local, nontender ulcer (chancre) usually forms in 2 to 10 weeks.
- ✿ The ulcer heals spontaneously, but Spirochetes spread widely via the bloodstream (bacteremia) to many organs.



Pathogenesis & Clinical Findings

In secondary syphilis,

☼ One to three months later, the lesions may occur. These often appear as:

1. a maculopapular rash, notably on the palms & soles, or as
2. moist papules on skin & mucous membranes (mucous patches).
3. Condylomata lata

These lesions are rich in Spirochetes & are highly infectious, but they also heal spontaneously.





**Secondary
Syphilis.
Lesions
observed**



Secodary syphilis condylomata lata.

**The lesions
resemble genital
warts (condylomata
acuminate).**

**Fluids exuding from
these lesions are
highly infectious.**



Pathogenesis & Clinical Findings

- ✿ Patchy alopecia also occurs.
- ✿ Constitutional symptoms of secondary syphilis include
 - ✿ low-grade fever,
 - ✿ malaise,
 - ✿ anorexia, weight loss,
 - ✿ headache,
 - ✿ myalgias, &
 - ✿ generalized lymphadenopathy.
- ✿ There may be internal organ involvement (meningitis, nephritis, hepatitis, etc).
- ✿ These stages may be asymptomatic, & yet the disease may progress.

Pathogenesis & Clinical Findings

- ✿ **About one-third** of these early (primary & secondary) syphilis cases will "cure" themselves without treatment.
- ✿ **Another third remain latent**, i.e., no lesions appear, but positive serologic tests indicate continuing infection.

Pathogenesis & Clinical Findings

✿ The latent period can be divided into early & late stages.

1. In the early latent period, which can last for a year or two after the secondary stage, the symptoms of secondary syphilis can reappear & patients can infect others.

2. In the late latent period, which can last for many years, no symptoms occur & patients are not infectious.

Pathogenesis & Clinical Findings

- ✿ In the remaining one-third, the disease progresses to the Tertiary stage.
- ✿ Tertiary syphilis may show granulomas (gummas), especially of skin & bones; CNS involvement (e.g., tabes, paresis); or CV lesions (e.g., aortitis, aneurysm of the ascending aorta). In tertiary lesions, treponemes are rarely seen.

Pathogenesis & Clinical Findings

- ✿ *T. pallidum* also causes congenital syphilis.
- ✿ The organism is transmitted across the placenta, typically after the third month of pregnancy, & fetal infection can occur.
- ✿ Skin & bone lesions are common, as is hepatosplenomegaly.
- ✿ Unless the disease is treated promptly, stillbirth or multiple fetal abnormalities occur.

**Typical
facies of
congenital
syphilis.**





Hutchinson teeth in congenital syphilis

Pathogenesis & Clinical Findings

- ✿ **Immunity to syphilis is incomplete.**
- ✿ **Antibodies to the organism are produced but do not stop the progression of the disease.**
- ✿ **Patients with early syphilis who have been treated can contract syphilis again.**
- ✿ **Patients with late syphilis are relatively resistant to re-infection.**

Laboratory Diagnosis

There are three important approaches.

1. Microscopy

- ✿ Spirochetes are demonstrated in the lesions of primary or secondary syphilis, such as chancres or condylomata lata, by:
 - a. darkfield microscopy
 - b. direct fluorescent antibody (DFA) test.
- ✿ They are *not* seen on a Gram-stained smear.
- ✿ In biopsy specimens, such as those obtained from the gummas seen in tertiary syphilis, histologic stains such as silver stain or fluorescent antibodies can be used.

Laboratory Diagnosis

2. Nonspecific Serologic Tests

- ✿ These tests involve the use of nontreponemal antigens.
- ✿ Extracts of normal mammalian tissues (e.g., cardiolipin from beef heart) react with antibodies in serum samples from patients with syphilis.
- ✿ These antibodies, which are a mixture of IgG & IgM, are called "reagin" antibodies.

Laboratory Diagnosis

2. Nonspecific Serologic Tests

- ✿ Flocculation tests, e.g.,
- ✿ VDRL (Venereal Disease Research Laboratory) & RPR (rapid plasma reagin) tests, detect the presence of these antibodies. These tests are positive in most cases of 1ry syphilis & are almost always positive in 2ndry
- ✿ The titer of these nonspecific antibodies decreases with effective treatment, in contrast to the specific antibodies, which are positive for life.

Laboratory Diagnosis

3. Specific Serologic Tests

- These tests involve the use of treponemal antigens & therefore are more specific, these tests are,
- *T. pallidum* reacts in immunofluorescence (FTA-ABS) or
- Hemagglutination (TPHA, MHA-TP) assays with specific treponemal antibodies in the patient's serum.
- These antibodies arise within 2 to 3 weeks of infection; therefore, the test results are positive in most patients with primary syphilis.
- These tests remain positive for life after effective treatment & *cannot* be used to determine the response to treatment or reinfection.
- They are more expensive & more difficult to perform than the nonspecific tests & therefore are not used as screening procedures.

Treatment

- ✿ **Penicillin** is effective in the treatment of all stages of syphilis. A single injection of benzathine penicillin G (2.4 million units) can eradicate *T. pallidum* & cure early (primary & secondary) syphilis.
- ✿ Note that benzathine penicillin is the form of penicillin used because the penicillin is released very slowly from this depot preparation.
- ✿ *T. pallidum* grows very slowly, which requires that the penicillin be present in bactericidal concentration for weeks.
- ✿ If the patient is allergic to penicillin, **doxycycline** can be used but must be given for prolonged periods to effect a cure.
- ✿ In neurosyphilis, high doses of aqueous penicillin G are administered because benzathine penicillin penetrates poorly into the CNS.
- ✿ No resistance to penicillin has been observed.

Treatment

- ✿ More than half of patients with secondary syphilis who are treated with penicillin experience fever, chills, myalgias, and other influenza-like symptoms a few hours after receiving the antibiotic.
- ✿ This response, called the Jarisch-Herxheimer reaction, is attributed to the lysis of the treponemes & the release of endotoxin-like substances.
- ✿ Alert patients to this possibility, it may last for up to 24 hours, & told that symptomatic relief could be obtained with aspirin.

Prevention

- ✿ early diagnosis & adequate treatment,
- ✿ use of condoms,
- ✿ administration of antibiotics after suspected exposure, and
- ✿ serologic follow-up of infected individuals & their contacts.
- ✿ The presence of any STD makes testing for syphilis mandatory because several different infections are often transmitted simultaneously.
- ✿ There is no vaccine against syphilis.

Granuloma inguinal

GRANULOMA INGUINAL

Granuloma inguinale is a rare, progressive infection of genital and perineal skin caused by *Klebsiella* (formerly *Calymmatobacterium*) granulomatis.

The disease is characterized by slowly progressive skin lesions that are beefy red, raised, painless, and often ulcerated;

regional lymphadenopathy is uncommon. Diagnosis is by clinical criteria and microscopy. Treatment is with antibiotics, usually tetracyclines, macrolides, or trimethoprim/sulfamethoxazole.

GRANULOMA INGUINAL

SITES OF INFECTION

- Penis, scrotum, groin, and thighs in men
- Vulva, vagina, and perineum in women
- Anus & buttocks in patients who engage in anal-receptive intercourse
- Face in both sexes
- After an IP of about 1 to 12 weeks, a painless, red skin nodule slowly enlarges, becoming a raised, beefy red, moist, smooth, foul-smelling lesion. The lesion slowly enlarges, often ulcerates, & may spread to other skin areas. Lesions heal slowly, with scarring. Secondary infections with other bacteria are common & can cause extensive tissue destruction.

GRANULOMA INGUINAL

SITES OF INFECTION

- Lymphadenopathy is uncommon.
- Occasionally, granuloma inguinale spreads through the bloodstream to the bones, joints, or liver; anemia, wasting, and, uncommonly, death may occur without treatment.
-

Granuloma
inguinal



DIAGNOSIS

Diagnosis of granuloma inguinale is confirmed microscopically by the presence of Donovan bodies (numerous bacilli in the cytoplasm of macrophages shown by Giemsa or Wright stain) in smears of fluid from scrapings from the edge of lesions.

Biopsy specimens are taken if the diagnosis is unclear or if adequate tissue fluid cannot be obtained because lesions are dry, sclerotic, or necrotic.

The bacteria do not grow on ordinary culture media.

TREATMENT

- Tetracyclines, macrolides, and trimethoprim/sulfamethoxazole (TMP/SMX) are most effective, followed by ceftriaxone, aminoglycosides, fluoroquinolones, and chloramphenicol.

TREATMENT

Recommended oral regimens include

- Doxycycline 100 mg twice a day for 3 weeks
- TMP/SMX 160/800 mg twice a day for 3 weeks
- Erythromycin 500 mg four times a day for 3 weeks
- Azithromycin 1 g a week for 3 weeks

TREATMENT

- IV or IM antibiotics (eg, ceftriaxone) are an alternative.
- Response to treatment should begin within 7 days, but the healing of extensive disease may be slow, and lesions may recur, requiring longer treatment.
- Current sex partners should be examined and, if infected, treated.
-

Chancroid

CHANCROID

DIAGNOSTIC CONSIDERATIONS

- A definitive diagnosis of chancroid requires identifying *H. ducreyi* on special culture media that is not widely available from commercial sources; even when these media are used, sensitivity is <80%.
- No FDA-cleared NAAT for *H. ducreyi* is available in the United States;

CHANCROID

DIAGNOSTIC CONSIDERATIONS

- The combination of one or more deep and painful genital ulcers and tender suppurative inguinal adenopathy indicates the chancroid diagnosis;
- inguinal lymphadenitis typically occurs in <50% of cases.

CHANCROID

DIAGNOSTIC CONSIDERATIONS

For both clinical & surveillance purposes, a probable diagnosis of chancroid can be made if all of the following four criteria are met:

1. the patient has one or more painful genital ulcers;
2. the clinical presentation, the appearance of genital ulcers &, if present, regional lymphadenopathy are typical for chancroid;
3. the patient has no evidence of *T. pallidum* infection by darkfield examination or NAAT (i.e., ulcer exudate or serous fluid) or by serologic tests for syphilis performed at least 7–14 days after onset of ulcers; and
4. HSV-1 or HSV-2 NAAT or HSV culture performed on the ulcer exudate or fluid are negative.

Chancroid
ulcer



RECOMMENDED REGIME

- **Azithromycin** 1 gm orally in a single dose
- OR
- **Ceftriaxone** 250 mg IM in a single dose
- OR
- **Ciprofloxacin** 500 mg orally 2 times/day for 3 days
- OR
- **Erythromycin** base 500 mg orally 3 times/day for 7 days

BACTERIAL VAGINOSIS (BV)

- Bacterial vaginosis (BV) occurs when there is too much of certain bacteria in the vagina.
- This changes the normal balance of bacteria in the vagina.
- BV is the most common vaginal condition in women ages 15-44.

BACTERIAL VAGINOSIS (BV) SPREAD

- The condition most often occurs in those who are sexually active.
- BV is a result of an imbalance of “good” and “harmful” bacteria in a vagina.
- Douching, not using [condoms](#), and having new or multiple sex partners can upset the normal balance of vaginal bacteria, increasing the risk for getting BV.
- Having BV can increase your chances of getting other STDs.
- BV rarely affects those who have never had sex.
- Cannot get BV from toilet seats, bedding, or swimming pools.

BACTERIAL VAGINOSIS (BV)

SYMPTOMS

- Symptomless
- A thin white or gray vaginal discharge;
- Pain, itching, or burning in the vagina;
- A strong fish-like odor, especially after sex;
- Burning when peeing; and
- Itching around the outside of the vagina.

PUERPERAL INFECTIONS

- A puerperal infection occurs when bacteria infect the uterus and surrounding areas after a female gives birth. It's also known as a postpartum infection.
- Types
 - **Endometritis:** uterine lining
 - **Myometritis:** uterine muscle
 - **Parametritis (also called pelvic cellulitis):**

PUERPERAL INFECTIONS

- The most frequent postpartum infection is endometritis since the lining of the uterus can undergo trauma and tears during the birthing process. This damage provides an entrance for infection to develop.
- Infection in the uterus muscle or the structures supporting the uterus may form at the incision or tear sites, such as in an **episiotomy** or a cesarean delivery (**C-section**)

WHAT ARE THE SYMPTOMS OF A PUERPERAL INFECTION?

General signs of a postpartum infection are like a typical infection, such as:

- fever
- chills
- body aches
- loss of appetite
- overall discomfort

WHAT ARE THE SYMPTOMS OF A PUERPERAL INFECTION?

More severe symptoms specific to a postpartum infection include:

- pain below the waist or in the pelvic bone area caused by an inflamed uterus
- pale, clammy skin related to a large amount of blood loss
- foul-smelling vaginal drainage revealing an infection
- increased heart rate from blood loss
- Symptoms may take several days to appear. Sometimes infections may not be noticeable until after you have left the hospital. It is important to look for signs of infection even after you have been discharged.