CHLAMYDIA Trichomonas Vaginalis Candidaiasis

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Chlamydia

Epidemiology

- Risk factors and transmission are similar to other STDs
- In USA over 900,000 cases are reported each year, which is more than gonorrhea
- The asymptomatic cases among males and females are higher than in gonorrhea
- Risk is more Pregnant s and menstruating women
- Reinfection is frequent



Reported Sexually Transmitted Diseases, United States, 2004

Family: Chlamydiaceae

• Genus: Chlamydia

C. trachomatis - Urogenital infections, trachoma, conjunctivitis, pneumonia and lymphogranuloma venerium (LGV)

- Genus: Chlamydophilia
 - C. psittaci Pneumonia (psittacosis)
 - C. pneumoniae Bronchitis, sinusitis, pneumonia and possibly atherosclerosis

Chlamydia- Microbiology

- Small obligate intracellular parasites
- Inner and outer membrane
- LPS but no peptidoglycan
 Cell wall not well characterized
- Energy parasites
 - Can't make ATP

Physiology and Structure

- Elementary bodies (EB)
 - Extracellular form
 - Rigid outer membrane
 - Disulfide linked proteins
 - Resistant to harsh conditions
 - Non-replicating, non-metabolically active form
 - Infectious form
 - Bind to columnar epithelial cells (macrophages)

Physiology and Structure

- Reticulate bodies (RB)
 - Intracellular form
 - Fragile membrane
 - Fewer disulfide bonds
 - Metabolically active form
 - Replicating form
 - Non-infectious

Developmental Cycle of Chlamydia

- EB bind to host cells
 - Epithelial
 - Macrophage
- Internalization
 - Endocytosis
 - Phagocytosis
- Inhibition of phagosomelysosome fusion
- Reorganization into RB
- Growth of RB and release





Chlamydia curriculum

Chlamydial Morphologies and life cycle



Chlamydia curriculum



Pathogenesis

- Chlamydiae have a tropism for epithelial cells of the endocervix and upper genital tract of women, and the urethra, rectum and conjunctiva of both sexes.
- Once infection is established, there is a release of proinflammatory cytokines by infected epithelial cells.
- This results in early tissue infiltration by PMNs, later followed by lymphocytes, macrophages, plasma cells and eosinophils.
- If the infection progresses further (because of lack of treatment and/or failure of immune control), aggregates of lymphocytes and macrophages (lymphoid follicles) may form in the submucosa; these can progress to necrosis, followed by fibrosis and scarring.

C. trachomatis - Serovars

Clinical Spectrum of C. trachomatis Infections

SEROVARS A, B, Ba, C B, Ba, D to K

 L_1, L_2, L_3

DISEASES

Trachoma Oculogenital disease (conjunctivitis, urethritis, proctitis, cervicitis); infant pneumonia LGV

C. trachomatis

- Ocular infections
 - Worldwide
 - Poverty and overcrowding
 - Endemic in Africa, Middle East, India, SE Asia
 - United States American Indians
 - Infection of children
 - Transmission: droplets, hands, contaminated clothing, flies, contaminated birth canal

Clinical Syndrome -Trachoma



From: G. Wistreich, Microbiology Perspectives, Prentice Hall

Clinical Syndrome -*Trachoma* (*C.trachomatis* biovar: trachoma)

- Chronic or repeated infection
 - Follicle formation on conjunctiva
 - Scarring of the conjunctiva



• Eyelids turn in and abrade cornea

- Ulceration
- Scarring

Blood vessel formation



- Flow of tears impeded
 - Secondary infections



Clinical syndromes

In Men

Urethritis

- The most common cause of nongonococcal urethritis (NGU) in men (40 to 96 percent)
- > Majority (>50%) are asymptomatic
- The incubation period is variable but is typically 5 to 10 days after exposure
- Symptoms
 - ✓ mucoid or clear urethral discharge
 - ✓ dysuria

Sometimes the discharge is so scant. This is in contrast to the more copious and purulent urethral discharge and shorter (two to seven days) incubation period for gonococcal urethritis

- Prostatitis
- Epididymitis

Clinical syndromes

In woman

<u>Urethritis</u> <u>Cervicitis</u>

- Majority (70%-80%) are asymptomatic
- Local signs of infection, when present, include:
 - mucopurulent endocervical discharge
 - cervical edema with erythema and friability



Normal Cervix

Cervicitis



Clinical syndromes

In woman

Complications in Women

- Pelvic Inflammatory Disease (PID)
 - Salpingitis
 - Endometritis
- Perihepatitis (Fitz-Hugh-Curtis Syndrome)

Clinical Syndrome - Lymphogranuloma Venereum

C. trachomatis (biovar: LGV)

- First stage
 - Small painless vesicular lesion at infection site
 - Fever, headache and myalgia
- Second stage
 - Inflammation of draining lymph nodes
 - Fever, headache and myalgia
 - Buboes (rupture and drain)
 - Proctitis
 - Ulcers or Elephantiasis

Patient with LGV

Bilateral inguinal buboes (arrows)



C. trachomatis - Diagnosis

Histo Cytology:

Iodine-staining inclusions Not sensitive

Culture: Iodine staining inclusions Most specific



Iodine-stained inclusion bodies

C. trachomatis - Diagnosis

- Antigen detection (ELISA or IF)
 - Group specific LPS
 - Strain specific outer membrane proteins
- Nucleic acid probes
 - Several kits available
 - May eventually replace culture

C. trachomatis - Treatment

 Doxycycline ; alternatives are erythromycin and sulfonamides

 Treatment of patients and their sexual partners

Trichomonas vaginalis

• Trichomonas vaginalis is flagellated protozoan and the causative agent of **trichomoniasis**.

• Women are usually symptomatic, while infections in men are usually asymptomatic.

 Trichomonas vaginalis exists only as a trophozoite (no cysts stage)

Trichomonas vaginalis

- It is pear-shaped, with a short undulating membrane lined with a flagellum and 4 anterior flagella.
- It moves with wobbling and rotating motion.

 In females it causes low-grade inflammation limited to vulva, vagina and cervix, causing frothy yellow or creamy discharge.

In males it may infect the prostate, seminal vesicles and urethra.



General Morphology of the Trichomonas trophozoite

Trichomonas vaginalis infection





Pathogenesis

- It is causative agents of persistent vaginitis. The histological features are non- specific & include increased vascularity & congestion.
- -Several studies showed that T. vaginalis produce a cell- detaching factor that causes detachment & sloughing of vaginal epithelial cells.

Diagnosis

- Specimen used : urine/vaginal discharges (female)
- prostatic secretions/urine (male)

 Clinical diagnosis is based on symptoms of burning, a frothy creamy discharge, hyperemia of the vagina.

- Microscopic examination in a drop of saline for motile trichomonas of the fresh vaginal discharge
- Motile
- pear-shaped



- Cultures will reveal the organism when negative microscopic examination result was obtained.
- Example :diamond's medium
- Incubated aerobically for 96 hours at 35°C.
- Showing turbidity in lower portion of tube.
- Treatment: metronidazole



CANDIDIASIS

- Candida albicans (budding yeast)
 How common is genital candidiasis?
- Nearly 75% of all adult women have had one genital infection (yeast infection) in their life time. On rare occasions, male may also experience genital candidiasis.
- Candidiasis is opportunistic infection & there are some conditions that may put a women at risk for genital infection e.g:
- Diabetes mellitus, General debilitating disease
- Immunodeficiency, Pregnancy & contraceptive pills.

CANDIDIASIS

Transmission

- Most of the cases of candida infection are caused by the person's own candida (endogenous infection).
- It is usually live in mouth, gastrointestinal tract & vagina without causing symptoms.
- Symptoms can develop only when Candida overgrown in these sites.
- Rarely, Candida can be passed from person to another, such as sexual contact

Clinically: Vulvo- vaginitis or vaginal thrush

 Manifested by a thick yellow a white (cheesy-like) discharge, burning sensation, itching...

CANDIDIASIS

• Diagnosis :

- Vaginal discharge examined by:
- **1-Direct microscopy**
- Candida yeast can be detected in un- stained or gram stained film
- 2- Culture
- C. albicans grows well on sabouraud, s agar. After 24-48 hours incubation at 37c or at room temp., colonies appear as cream, pasty with yeast smell
- 3- Differential Germ tube tests
- To diff. between C.albicans & non- pathogenic species of Candida e.g C. tropicalis& C. peudotropicalis

• Treatment

- A-Topical antifungal
- Nystatin
- Canestan
- Miconazol (Dactarine)
- B-Systemic antifungal
- Ketoconazol (oral)
- Amphotericine B I.V

The End