University of Tikrit

College of nursing

Basic Nursing Sciences

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Parasitology

Trichomonas vaginalis

By:

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Trichomonas vaginalis

Disease: Trichomonasis or vaginitis

Morphology:-
It has Trophozoite stage only

- 15 X 10 μm.
- pear-shaped organism
- Twitching jerky motility.
- One nucleus
- Axostyle.
- Four anterior free flagella.
- Single lateral flagellum runs back along the surface of the cell attaching to an undulating membrane (extent ½ way along the cell)

Life cycle:-

The life cycle of *T. vaginalis* is simple in that the trophozoite is transmitted through sexual intercourse and no cyst form is known. The trophozoite divides by binary fission and, in natural infections, gives rise to a population in the lumen and on the mucosal surfaces of the urogenital tracts of humans.

In females the trophozoite exists free in the vaginal cavity or adherent to the epithelium without invasion, but with symptoms .

In male the trophozoite lives in urethra, prostate and epididymis but with no symptoms!!

Pathogenesis:-

I-in woman:

- 50-90% symptomatic.
- Vaginitis, vulval irritation and dysuria (the parasite is non-invasive).
- Usually with alkaline vaginal discharge (frothy and greenish yellow) with large numbers of neutrophils (leucorrhoea).

II- in men:

- The majorities are asymptomatic.
- In some cases: urethritis, epididymitis, prostatitis and dysuria. III- in infant and children:
- In 5% of female babies born (naturally) to infected mother.
- In children the parasite can be transmitted due to

sexual abuse by infected adult.

Those with trichomonasis are at high risk of being infected with HIV.

Epidemiology:

This parasite has worldwide distribution, and sexual intercourse is the primary mode of transmission. Occasionally, infections can be transmitted by fomites (toilet articles, clothing). Rarely infants may be infected by passage through the mother's infected birth canal.

Laboratory diagnosis:

• In females, *T.vaginalis* may be found in urine sediment, wet preparations of vaginal secretions or vaginal scrapings.

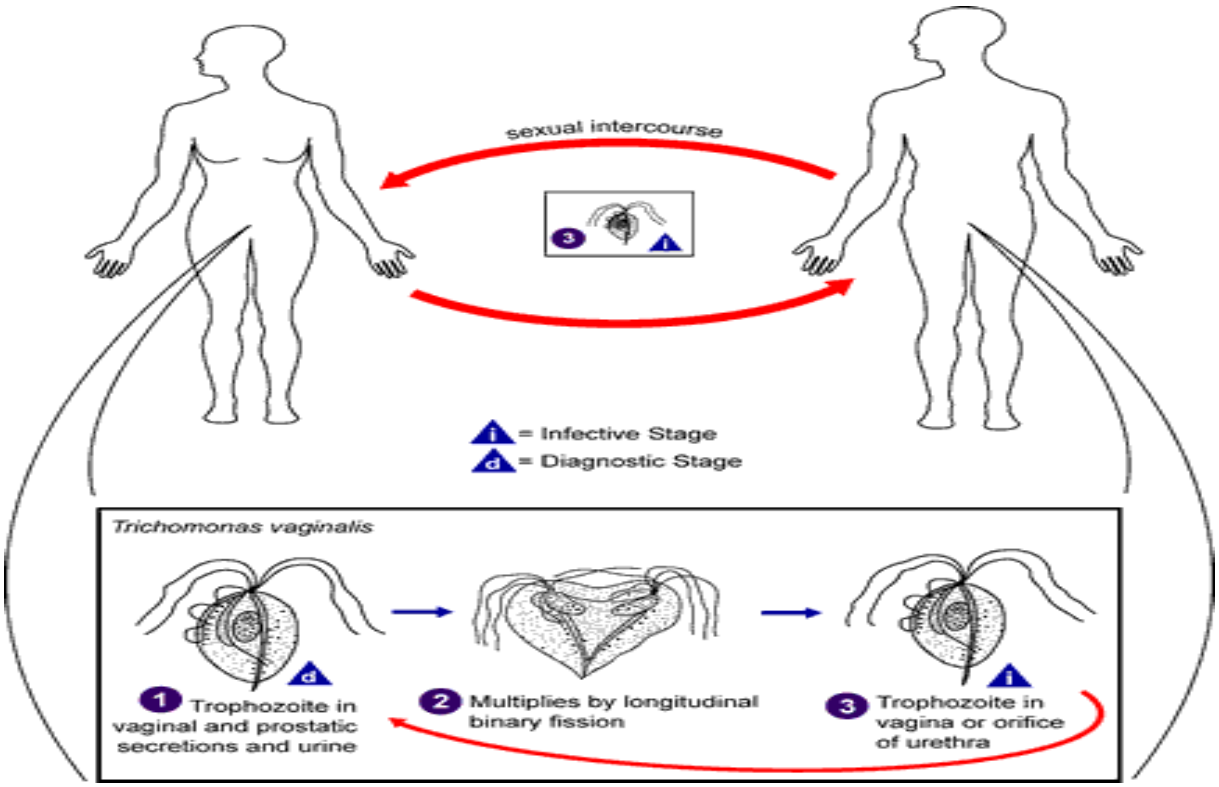
• In males it may be found in urine, wet preparations of prostatic secretions or following massage of the prostate gland.

Treatment:

Metronidazole is the drug of choice. If resistant cases occur, re-treatment with higher doses is required.

Prevention:

- Both male & female sex partners must be treated to avoid reinfection
- Good personal hygiene, avoidance of shared toilet articles & clothing.
- Safe sexual practice.



Life cycle of Trichomonas *vaginalis*

Haemoflagelates

Leishmania Species

Leishimania has 3 major species of medical importance :-

L. donovani
causes visceral leishmaniasis (**kala-azar**disease)
L. tropica, (Old World species) causes cutaneous leishmaniasis
L. brasiliensis
causes mucocutaneous leishmaniasis

The species of leishmania exist in two forms, **amastigote** (**Non-flagellated** form found in the humanbody ,This is the **diagnostic** stage in humans) and **promastigote** (A **flagellated** form found in the insect vector and in cultures ,This is the **infective stage** for humans) in their life cycle. They are transmitted by certainspecies of sand flies (Phlebotomus). Only the female fly feeds on blood of the host thus transmit the parasite .

Life Cycle:

In nature, Leishmania are alternatively hosted by the insect (flagellated promastigotes) and by mammals (intracellular amastigotes). When a female sandfly takes blood meal from an infected mammal; the insect ingests intracellular amastigotes. Inside the fly amastigotes are transformed in to flagellated promastigotes in the midgut. The promastigotes migrate into the anterior portion of the mid gut. The bite of an infected sandfly deposits infective promastigotes in the mammals' skin, which are rapidly phagocytosed by the cells of mononuclear-phagocyte system. The intracellular parasites change into amastigotes, which multiply by simple mitosis.

Pathogenesis

The bite of an infected sand fly results in the intradermal inoculation of promastigote stage of Leishmania. Within the dermis of mammalian skin, promastigotes escape complement activation and they are phagocytosed by macrophages where they transform to amastigotes.
When the intracellular development of the amastigotes remains localized at the inoculation site, various cytokines are released and cell reactions are generated, resulting in the development of localized lesion of cutaneous leishmaniasis. In other instances the

parasite spread to the organs of the mononuclear phagocytic system, giving rise to visceral leishmaniasis. Amastigotes may also spread to mucosal sites in the case of mucocutaneous Leishmaniasis.

1. Visceralleishmaniasis

Leishmania donovani

In visceral leishmaniasis, the organs of the reticuloendothelial system (liver, spleen and bone marrow) are the most severely affected organs. Reduced bone marrow activity, coupled with cellular distraction in the spleen, results in anemia, leukopenia and thrombocytopenia. This leads to secondary infections and a tendency to bleed. The spleen and liver become markedly enlarged, and hypersplenism contributes to the development of anemia and lymphadenopathy also occurs.

The most common symptoms of visceral leishmaniasis are fever, weight loss, weakness, and diarrhea; chills and sweating and abdominal distension with splenomegaly and hepatomegaly. Other symptoms may include coughing, chronic diarrhea, darkening of the skin, lymphadenopathy, and in many cases, signs of chronic kidney disease. With persistence of the disease, deeply pigmented, granulomatous lesion of skin, referred to as post-kala-azar dermal leishmaniasis occurs.

Epidemiology

L. donovani, infection of the classic kala-azar (—black sickness‖) or Dum-Dum fever type occurs in many parts of Asia, The vector is the Phlebotomus sand fly. Reservoir hosts are dogs, foxes, and cats.

Laboratory diagnosis

1. Examination of tissue biopsy, spleen aspiration, bone marrow aspiration or lymph node aspiration in properly stained smear (e.g. Giemsa stain).
2. The amastigotes appear as intracellular & extra cellular L. donovan (LD) bodies.
3. Culture of blood, bone marrow, and other tissue often demonstrates the promastigote stage of the organisms.
4. Serologic testing is also available (PCR, ELISA).