Parasites Affecting The Reproductive System

Case Scenario

38 years old female complained of **vaginal itching** and **burning sensation** with profuse and offensive **discharge**. Gynecological examination revealed redness, oedema, and **strawberry-like vaginal mucosa**. Vaginal swab obtained and directed to lab for microscopic examination. It showed pear-shaped, 17X10 μ organism with a **rapid jerky movement**. The organism has 4 anterior free **flagella** and **undulating membrane**.

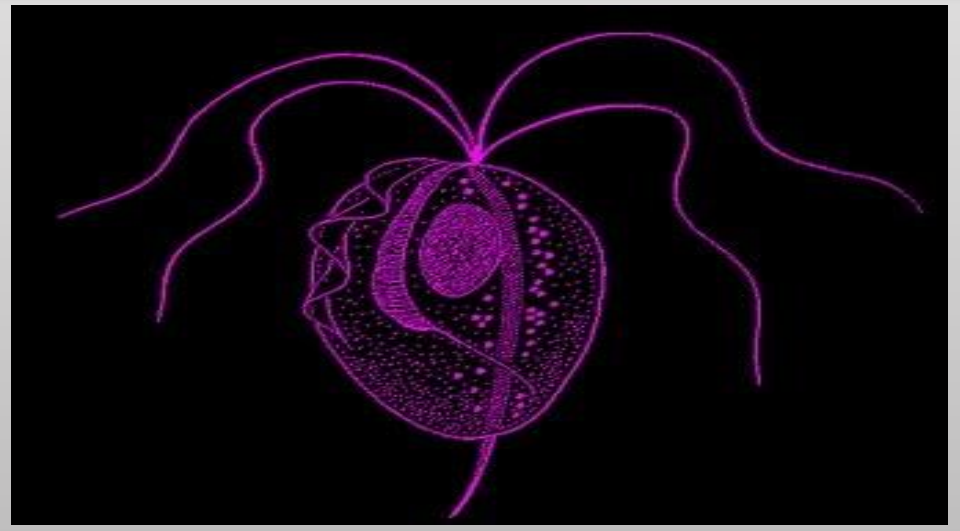
What is the most likely parasitic diagnosis?

➢ Parasitic infections affecting The Reproductive System:

1. ***Toxoplasma gondii***
2. ***Trichomonas vaginalis***
3. ***Schistosoma* spp.**
4. ***Wuchereria bancrofti***

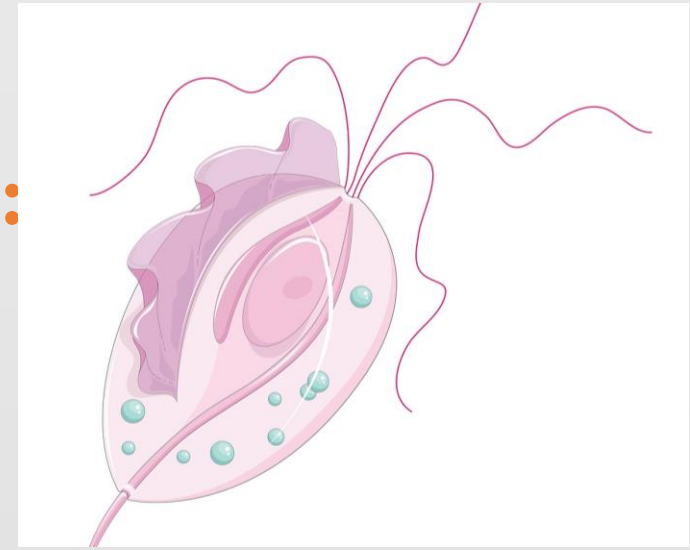
Parasitic infections affecting The Reproductive System:

1) *Trichomonas vaginalis*



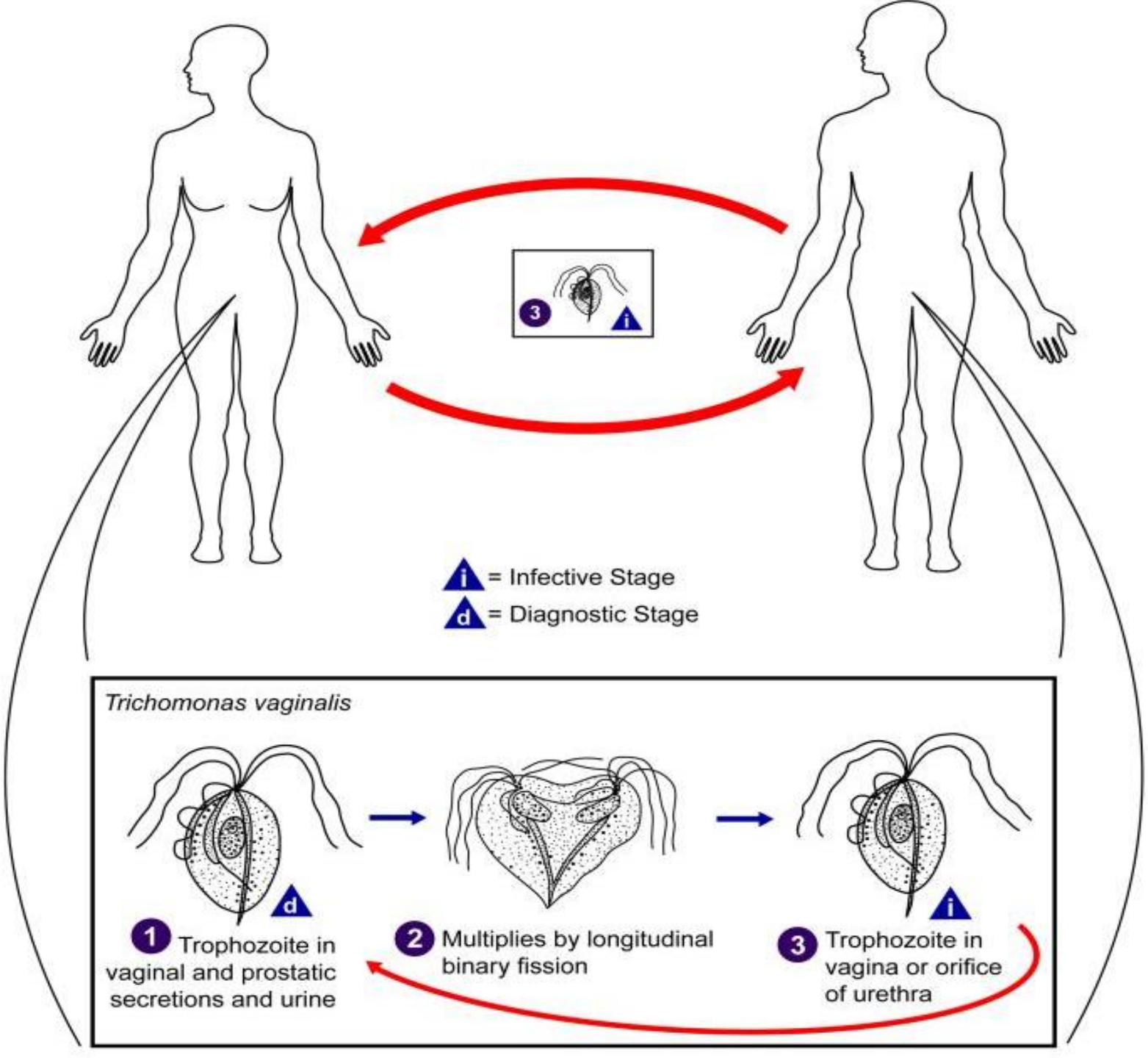
➢ *Trichomonas vaginalis* morphological stage:

1) Trophozoite:



- pear-shaped
- 17x10 µ,
- with a **rapid jerky movement**.
- an antero-lateral **cytostome**.
- The cytoplasm is granular with a single anterior **nucleus**
- 4 **free flagella** anteriorly
- 5th flagellum attached to the body by **undulating membrane**, presents at the anterior 1/3 of body
- 6th flagellum passes through the body as **axostyle**

Trichomonas vaginalis
Life cycle



Trichomonas vaginalis Life cycle:

- **Habitat:**

a. ***Females:*** Posterior fornix of the vagina, cervix, and urethra.

b. ***Males:*** Urethra, epididymis, seminal vesicles and prostate.

- **Definitive host:** Man.
- **Infective stage:** Trophozoite
- **Mode of infection:**

1. Sexual transmission or by contaminated toilet seats and towels.
2. From infected mothers to babies during birth.

- **Multiplication:** By longitudinal binary fission.

Pathogenesis of *Trichomonas vaginalis*

Trichomonas vaginalis causes **trichomoniasis**, trichomonad vaginitis, urethritis, epididymitis, vesiculitis and prostatitis.

The parasite can kill target cells by:

1) Direct contact without phagocytosis (dependent cytopathic effect).

2) Cell detaching factor: produced by the parasite, and its amount correlates with the severity of the clinical infection.

➢ Predisposing factors for pathogenicity:

1. Change of the normal vaginal bacterial flora and pH.

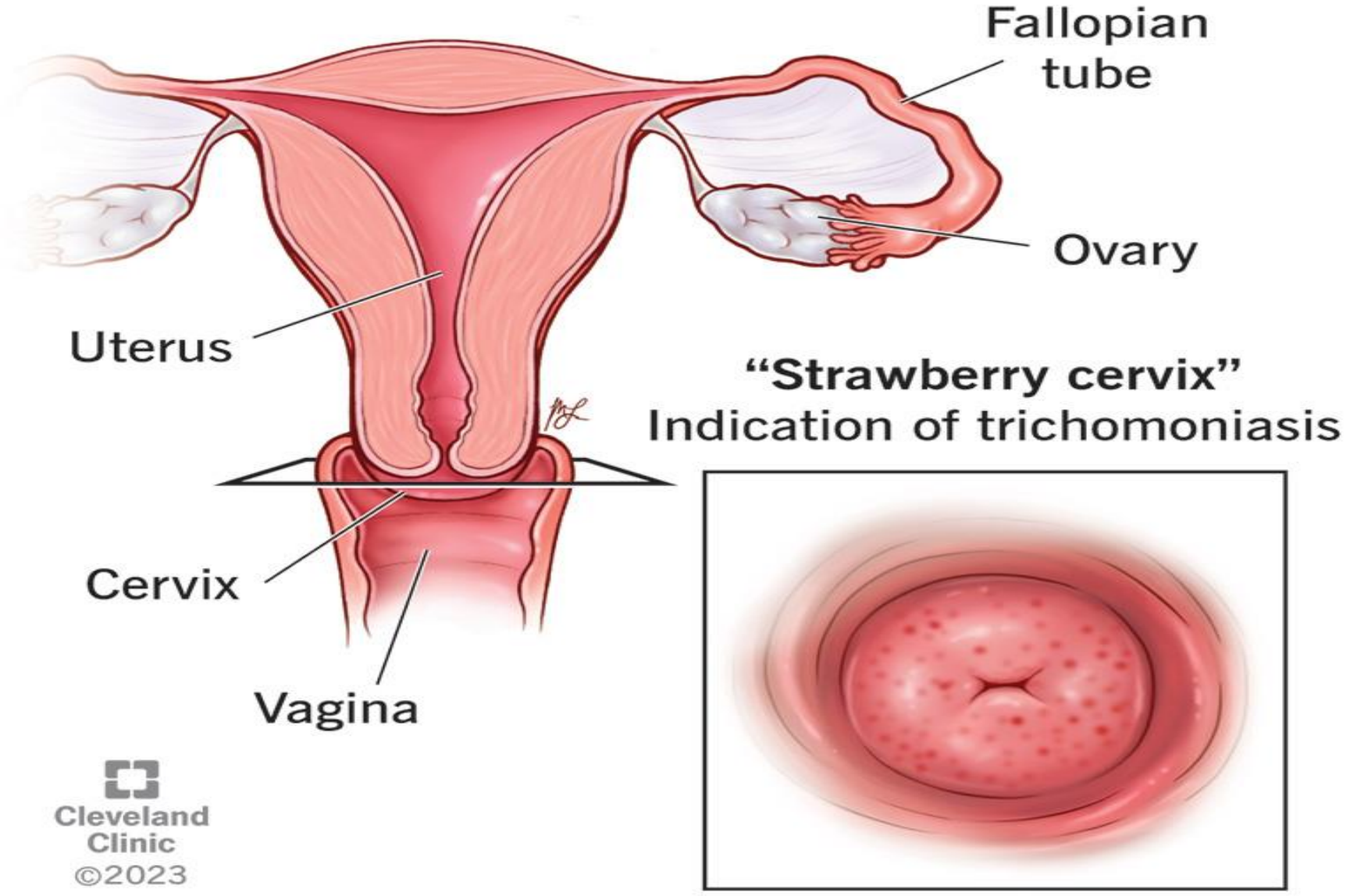
2. Decrease in the secretory IgA.

➢ **In male:** urethritis, vesiculitis, epididymitis and prostatitis may occur.

➢ In female:

- ***The vaginal wall*** is red, showing oedma, petechial hemorrhages (strawberry mucosa), mucosal erosion and necrosis.
- ***The mucosa*** is infiltrated with lymphocytes, plasma cells and polymorphonuclear leucocytes.
- A relationship between trichomoniasis and ***cervical carcinoma*** is suggested.

Trichomoniasis



Clinical picture of *Trichomonas vaginalis*

- **Trichomoniasis** may be asymptomatic in infected males (95%) and females (50%).

1. Females:

-- **Vaginal** itching and burning with an offensive, frothy, profuse leucorrheic **discharge** forming a pool in the posterior fornix.

-- **Dyspareunia** (painful sexual intercourse), frequency of micturition and dysuria, also **cystitis** may occur.

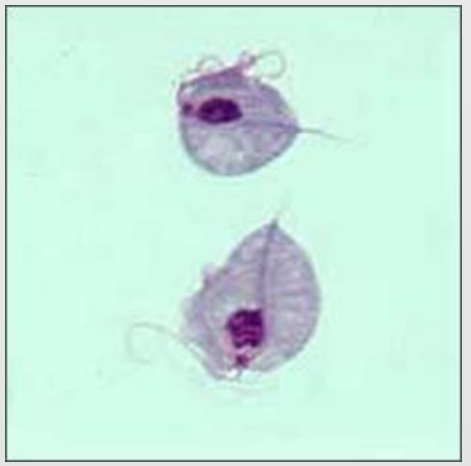
2. Males:

Dysuria, and prostate may be enlarged and tender.

3. Newborn:

Trichomonas **respiratory tract infection** and **conjunctivitis** may affect infants during vaginal delivery of an infected mother.

Diagnosis of *Trichomonas vaginalis*



A. Clinical diagnosis.

B. Laboratory diagnosis:

1. Microscopy: - In females: **vaginal smears**

a. Direct wet smear examination for characteristic jerky motility and shape of trichomonad trophozoites.

b. Fixed smears may be stained with Giemsa, Leishman stain.

- In males: Examination of prostatic fluid.

- In both sexes: Urine examination may beneficial

Diagnosis of *Trichomonas vaginalis*

2. Culture: On modified Diamond's medium.

3. Immunodiagnosis: For detection of *T.vaginalis* antigens.

a. Direct fluorescent antibody test using labeled monoclonal antibodies.

b. ELISA.

4. Molecular diagnosis.

➢ Treatment :

✓**Both** partners must be treated at the same time.

1. **Metronidazole** is the most effective drug.

2. Restoration of normal **vaginal acidity** by vaginal douching with lactic acid or vinegar seems beneficial in mild vaginal trichomoniasis.

➢ Prevention and control:

1- Good personal hygiene.

2. Avoidance of sexual contact with infected partners.

3. Treatment of diagnosed cases, and simultaneous treatment of sexual partners

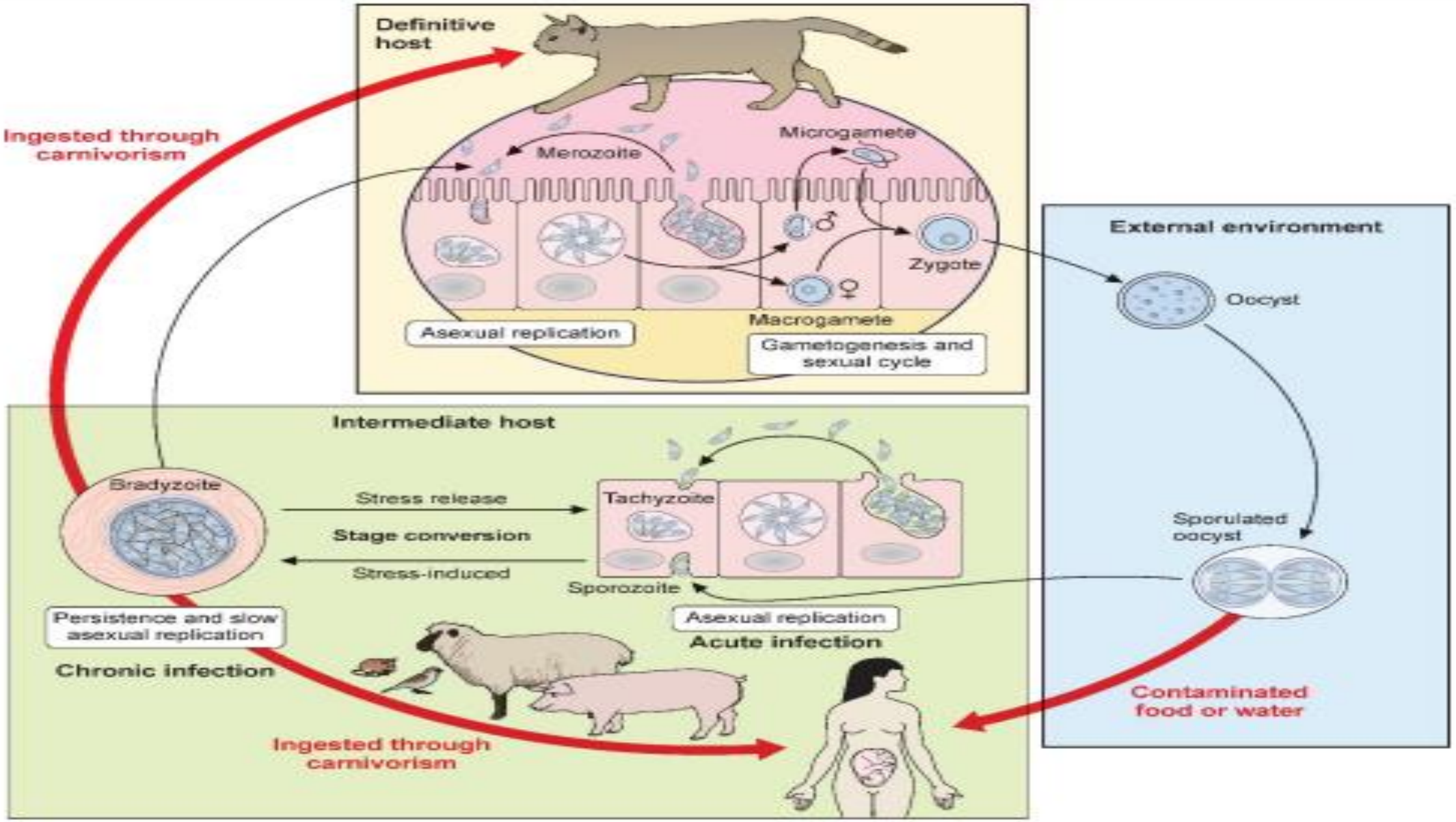
2) *Toxoplasma gondii*

Case Scenario

A young lady works in medical laboratory. She had been married 3 years ago, and she got pregnant and aborted twice. The physician asked her to do laboratory tests of **repeated abortion** and he found that *Toxoplasma gondii* **IgM titre** was positive, and *Toxoplasma gondii* **IgG titre** was positive.

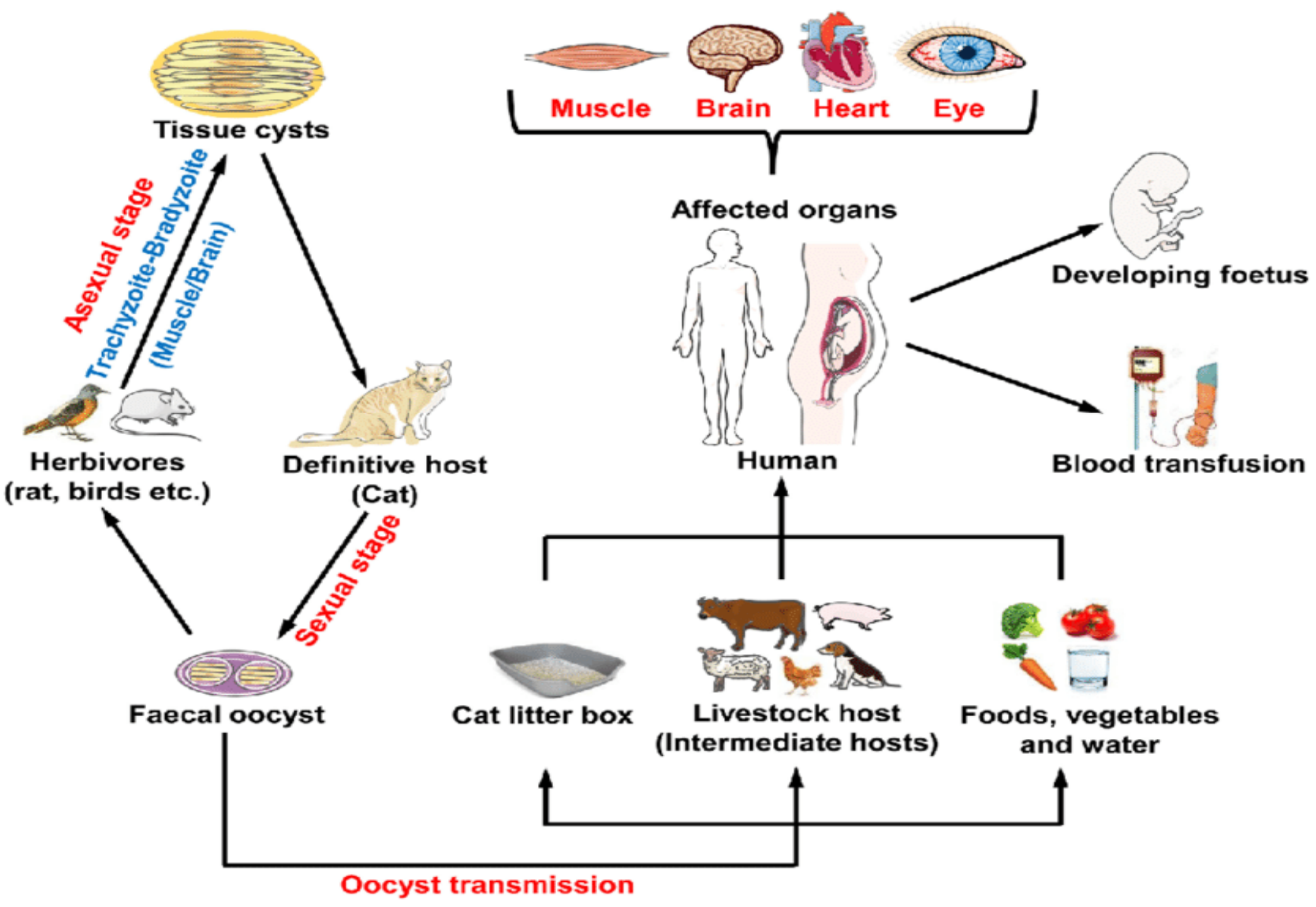
What is the most likely parasitic diagnosis?

Toxoplasma life cycle



Toxoplasma gondii Life cycle:

- **Habitat:**
- *T. gondii* is an obligate intracellular parasite, which is found inside the **RECs**, brain, skeletal and cardiac muscles, and any other **nucleated** cells.
- **Definitive host:** **Cats** and other felines
- **Intermediate host:** **Man** and other mammals (mice, rabbits, goat, sheep, cattle, and pigs), reptiles and birds.
- **Infective stage:** **All stages** are infectious to humans; tachyzoites, pseudocysts, true tissue cysts and sporulated oocysts.


Tissue cysts
Muscle
Brain
Heart
Eye
Asexual stage
Trachyzoite-Bradyzoite
(Muscle/Brain)
Affected organs
Developing foetus
Herbivores
(rat, birds etc.)
Definitive host
(Cat)
Human
Blood transfusion
Sexual stage
Faecal oocyst
Cat litter box
Livestock host
(Intermediate hosts)
Foods, vegetables
and water
Oocyst transmission

• Mode of infection:

1. Oral route via ingestion of:

- Mature oocysts in food and drinks contaminated with cat feces.

- Pseudocysts or true cysts in raw or undercooked contaminated meat.

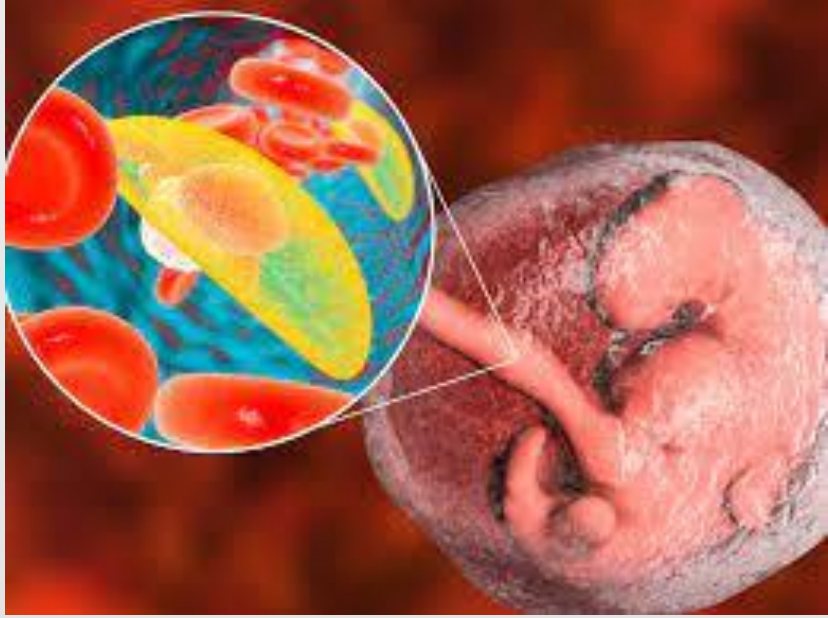
-Tachyzoites in unpasteurized goat's and cow´s milk.

2. Inhalation of mature oocysts.

3. Blood transfusion.

4. Organ transplantation.

- **Mode of infection:**



5. Contamination of mucous membrane, skin abrasions by:

- Handling and preparation of fresh infected meat.
- Handling of infected blood by laboratory workers.

6. Transplacental route: the tachyzoites can be transmitted from infected pregnant woman to the fetus via the blood stream (placenta).

7. Sexually transmitted or by artificial insemination with semen from infected male.

Pathogenesis of *Toxoplasmosis*

Proliferation of tachyzoites in the host cells causes cellular death with focal necrosis and surrounding inflammatory cells.

1. In acute infection: the outcome of the disease depends on host immune status.

a. In immunocompetent individuals: tachyzoites disappear from various organs.

b. In immunocompromised patients: the parasites disseminate through the blood stream to various organs (as brain, eyes, lungs, heart, liver, etc.), where they form **pseudocysts** causing severe necrotizing lesions.

2. In chronic infection:

- **True tissue cysts** remain viable in tissues for years in resting form.

- In immunodeficient status, their **reactivation** causes clinical disease.

Clinical picture of *Toxoplasmosis*

1. Congenital toxoplasmosis:

- This occurs when the mother gets infected for **the first time** during pregnancy. But, in some woman with **chronic infection**, reactivation of tissue cysts leads to liberation of trophozoites, which may infect the fetus.
- The risk of fetal infection **rises** with **progress** of pregnancy.
- In contrast, the **severity** of fetal damage is high, when infection is transmitted in **early** pregnancy.

Clinical manifestations of congenital infections may be:

a. Early manifestations:

- Still birth, abortion, hydrocephalus, microcephaly and microphthalmia.
- **The most common sequelae** are retinochoroiditis that affects vision and results in blindness, cerebral calcification, convulsions (**clinical triad**).
- In some cases, fever, lymphadenopathy, hepatosplenomegaly (HSM), anemia, thrombocytopenia, petechial rash, jaundice, and myocarditis may present at birth.

b. Late manifestations:

Mental retardation, visual affection and psychomotor disturbance in adolescence and adulthood.

2. Acquired toxoplasmosis:

- It is asymptomatic in 80-90% of healthy hosts.
- The classical clinical sign of acute acquired toxoplasmosis is **lymphadenopathy** and the deep cervical lymph nodes are the most commonly affected. The infected lymph nodes are discrete and non-tender.
- Mild fever, headache, myalgia (Flu-like syndrome), and HSM are often present.

3. Toxoplasmosis in immunocompromised patients:

- In these patients, affection of **brain** is more common, with meningoencephalitis, and neuropsychiatric manifestations.
- Pneumonia, myocarditis, chorioretinitis and HSM may occur.

Diagnosis of *Toxoplasmosis*

A. Clinical diagnosis:

- A combination of signs as hydrocephalus or microcephaly, chorioretinitis and signs of intracerebral calcification make diagnosis of **congenital toxoplasmosis** probable.

- **Acquired toxoplasmosis** is diagnosed by exclusion from other diseases of the reticuloendothelial and lymphatic systems.

Diagnosis of *Toxoplasmosis*

B. Laboratory diagnosis:

1. Microscopy:

- Detection of **trophozoites** and **tissue cysts** in smears stained by Giemsa, or PAS stain.

- blood	- CSF	
- lymph node	- bone marrow	- Spleen
- Placenta	- amniotic fluid	

Diagnosis of *Toxoplasmosis*

B. Laboratory diagnosis:

2. Animal inoculation:

- *Toxoplasma* can be detected by intraperitoneal inoculation of infective material in mice.

- After 7-10 days, peritoneal fluid is examined for **trophozoites**.

- Mice are sacrificed after 3 weeks and examined for **tissue cysts.**

Diagnosis of *Toxoplasmosis*

B. Laboratory diagnosis:

3. Serodiagnosis:

a. Sabin-Feldman dye test: It detects a circulating cytoplasm modifying antibody.

- Patient's **serum** is mixed with a ***Toxoplasma* trophozoites suspension** and **methylene blue** is added.

- If the parasite fails to take the stain, the test is considered positive.

Diagnosis of *Toxoplasmosis*

B. Laboratory diagnosis:

3. Serodiagnosis:

b. Antibody detection: For detection of IgM, IgG, and IgA **antibodies**.

- Tests used include ELISA, IFA, and IHA.

- Detection of specific **IgM** antibodies indicates ⟹ **acute infection**.

- Detection of specific **IgG** titer indicates ⟹ **latent infection**.

- Detection of **IgM** in **babies' blood** is ⟹ fetal in origin as maternal IgM doesn't cross the placenta.

- **IgA** is also used for detecting ⟹ congenital infection in newborns.

Diagnosis of *Toxoplasmosis*

B. Laboratory diagnosis:

3. Serodiagnosis:

c. Antigen detection:

- Detection of antigen by ELISA indicates ⟹ recent *Toxoplasma* infection.

- It is useful in ⟹ diagnosis of congenital toxoplasmosis and toxoplasmosis in immunocompromised patients.

Diagnosis of *Toxoplasmosis*

B. Laboratory diagnosis:

4. *Toxoplasma* Molecular diagnosis:

- Can be used for diagnosis of ***T. gondii*** **DNA** in blood, CSF, urine, amniotic fluid, and different tissues.

- It is useful in ⟹ diagnosis of **congenital toxoplasmosis** and toxoplasmosis in **immunocompromised patients** (antibody titers are low or absent).

5. Imaging:

- MRI and CT scan: to diagnose **CNS involvement**.

- US of fetus: to diagnose **congenital toxoplasmosis**.

➢ Treatment :

1. Immunocompetent individuals:

- Most healthy people **recover** from toxoplasmosis **without treatment.**
- Persons who develop persistent, severe symptoms can be treated with a combination of drugs (such as **pyrimethamine** and **sulfadiazine or clindamycin,** plus **folinic acid).**

➢ Treatment :

2. Immunocompromised individuals:

- Immunosuppressed patients who are positive for *T. gondii* and have a **CD4+ T-lymphocyte count less than 100/ µl** should receive prophylactic treatment against *Toxoplasma* encephalitis.

- Prophylaxis should be discontinued in patients whose CD4+ T-lymphocyte becomes **more than 200/µl for 3 months** after successful treatment.

- Drugs used in prophylaxis:

1) Trimethoprim-sulfamethoxazole (**the drug of choice**).

2) Dapsone- pyrimethamine.

➢ Treatment :

3. Pregnant women:

- **Spiramycin (Rovamycin)** should be taken for 4 weeks, to treat the infected pregnant women to reduce the risk of transplacental transmission.

4. Congenital toxoplasmosis:

- **Pyrimethamine** and **sulfadiazine** with **folinic acid** for one year.
- Systemic **corticosteroids** may be given to alleviate chorioretinitis.

➢ Prevention and control:

1. **Women** who are or may become **pregnant** should avoid contacts with cats or cleaning the litter box and undergo routine serological screening.

2. Individuals **at risk**, mainly children and immunocompromised individuals, should avoid contacts with cats and their feces.

3. Screening for *T. gondii* antibody should be done in all **blood banks**.

➢ Prevention and control:

4. Proper **washing** of hands, vegetables and fruits before eating.

5. Proper washing of hands and utensils after handling **raw meat**.

6. Proper freezing and cooking of **meat** before eating.

7. Never fed raw meat to **cats**; only dry, cooked or canned meat should be fed.

8. **Cats** should be kept indoors, and litter boxes changed daily. Cats' feces should be flushed down the toilet or burned. Litter pans should be cleaned by immersing them in boiling water.

Discussion & Feedback

10 minutes