<u>Toxoplasmosis</u> Infectious Diseases

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Introduction:

- A parasitic disease caused by *Toxoplasma* gondii
- Toxoplasmosis is usually spread by eating poorly cooked food that contains cysts, exposure to infected cat faeces, and from a mother to a child during pregnancy if the mother becomes infected.
- Rarely the disease may be spread by a **blood transfusion**.
- Up to half of the world's population are infected by toxoplasmosis but have no symptoms.





Clinical features:



- in most **immunocompetent** individuals, including **children** and **pregnant** women, the infection **goes unnoticed**.
- In approximately 10% of patients, it causes a **self-limiting illness**, most common in adults aged 25–35 years.
- The most common presenting feature is **painless lymphadenopathy**, either local or generalised. In particular, the **cervical nodes** are involved, but **mediastinal**, **mesenteric** or **retroperitoneal groups** may be affected. The **spleen** is seldom palpable.
- Most patients have no systemic symptoms , but some complain of malaise, fever, fatigue, muscle pain, sore throat and headache.

- Complete **resolution** usually occurs within a **few months**, although symptoms and lymphadenopathy tend to fluctuate unpredictably and some patients **do not recover** completely for a **year** or more.
- Very infrequently, patients may develop **encephalitis, myocarditis, polymyositis**, **pneumonitis** or **hepatitis**.
- **Retinochoroiditis** is nearly always the result of congenital infection but has also been reported in acquired disease.

Acute toxoplasmosis:

- it is often **asymptomatic** in healthy adults.
- The toxoplasmic **trophozoites** causing acute toxoplasmosis are referred to as **tachyzoites**, and are typically found in **bodily fluids**.
- symptoms may manifest and are often influenza-like , swollen lymphnodes , headaches, fever, and fatigue or muscle aches and pains that last for a month or more.
- Rarely will a human with a fully functioning immune system develop severe symptoms following infections.

- Young children and immuncompromised people, such as those with HIV/AIDS, those taking certain types of treatment, or those who have recently received an organ transplant, may develop severe toxoplasmosis.
- Very infrequently, patients may develop **encephalitis**, **myocarditis**, **polymyositis**, **pneumonitis** or **hepatitis**.
- Retinochoroiditis is nearly always the result of congenital infection but has also been reported in acquired disease.
- This can cause damage to the brain (encephalitis) or the eyes (necrotizing retinochoroditis), , lung problems that may resemble tuberculosis or Pneumocystis jervoci.

Latent toxoplasmosis:

- In most **immuncompetent** people, the infection enters a latent phase, during which only **bradyzoites** (tissue cysts) are present.
- these tissue cysts and even lesions can occur in the retinas, alveolar lining of the lungs , heart, skeletal muscle, and the central nervous system (CNS), including the brain.

Congenital toxoplasmosis:

- Acute toxoplasmosis, mostly subclinical, affects 0.3–1% of pregnant women, with an approximately 60% transmission rate to the fetus, which rises with increasing gestation.
- Seropositive females infected **6 months** before conception have no risk of fetal transmission.
- Congenital disease affects approximately **40%** of infected fetuses, and is more likely and more severe with infection **early** in gestation.
- Many **fetal** infections are **subclinical** at **birth** but long-term sequelae include **retinochoroiditis**, **microcephaly** and **hydrocephalus**.

Diagnosis:

- In contrast to immunocompromised patients, in whom the diagnosis often requires direct detection of parasites, **serology** is often used in **immunocompetent** individuals.
- The Sabin–Feldman dye test (indirect fluorescent antibody test), which detects IgG antibody, is most commonly used.
- **Recent** infection is indicated by a **fourfold** or greater increase in titer when paired sera are tested in parallel.
- Peak titers of 1/1000 or more are reached within **1–2 months** of the onset of infection, and the dye test then becomes an unreliable indicator of recent infection.

- The detection of significant levels of Toxoplasma specific **IgM antibody** may be useful in confirming acute infection.
- A false-positive result or persistence of IgM antibodies for years after infection makes interpretation difficult; however, negative IgM antibodies virtually rule out acute infection.
- During **pregnancy**, it is critical to differentiate between **recent** and **past** infection; the presence of high-avidity IgG antibodies excludes infection acquired in the preceding **3–4 months**.
- If necessary, the presence of Toxoplasma organisms in a **lymph node biopsy** can be sought by staining sections histochemically with **T. gondii antiserum**, or by the use of **PCR** to detect **Toxoplasma-specific DNA**.

Treatment:

- In immunocompetent subjects, uncomplicated toxoplasmosis is self-limiting and responds poorly to antimicrobial therapy. Treatment with pyrimethamine, sulfadiazine and folinic acid is therefore usually reserved for rare cases of severe or progressive disease, and for infection in immunocompromised patients.
- **Sulfadiazine** used in combination with **pyrimethamine** to treat toxoplasmosis Combination therapy is usually given with **folinic acid** supplements to **reduce** incidence of thrmbocytopenia. Combination therapy is most useful in the setting of HIV.
- In a **pregnant** woman with an established recent infection, **spiramycin** (3 g daily in divided doses) should be given until term.

- Once fetal infection is established, treatment with sulfadiazine and pyrimethamine plus calcium folinate is recommended (spiramycin does not cross the placental barrier).
- The cost/benefit of routine Toxoplasma **screening** and treatment in pregnancy is being debated in many countries. There is insufficient evidence to determine the effects on mother or baby of current antiparasitic treatment for women who seroconvert in pregnancy.

Latent:

- In people with **latent toxoplasmosis**, the cysts are immune to these treatments, as the antibiotics **do not reach** the bradyzoites in sufficient concentration.
- Atovaquone an antibiotic that has been used to kill *Toxoplasma* cysts inside AIDS patient.
- **Clindamycin** an antibiotic that, in combination with **atovaquone**, seemed to optimally kill cysts in mice.

Thank you