# Oesophageal And Gastric Toxoplasmosis:

*Rare Presentation Of An Emerging Zoonotic Disease*

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## Summary

**BACKGROUND**

*Toxoplasmosis* **a Zoonotic disease caused by** *Toxoplasma gondii. Toxoplasma gondii (T. gondii)* **is** a *protozoan* **parasite that infects most species of warm blooded animals including humans. It is an obligate intracellular parasite with a worldwide distribution.** *Sporozoites* **exist in** *oocysts* **and are found in the gut walls of definitive hosts** the cat family (*Felidae*). **Cats become infected with** *T.gondii* **by carnivorism or indigestion of** *oocysts.*

**Humans can become infected by any of the 4 routes; Eating undercooked meat of animals harbouring tissue cysts, Consuming food or water contaminated with cat feaces or by contaminated environmental samples (such as fecal contaminated soil or changing the litter box of a pet (cat), Blood transfusion or organ transplantation, Transplacentally (from mother to fetus), Accidental inoculation of** *tachyzoites.*

*Ocular Toxoplasmosis,* **a major cause of** *Chorioretinitis* **, may be as a result of congenital** *toxoplasmosis* **or acquired infection. Congenitally infected patients can remain asymptomatic until the second or third decades of life. Congenital** *Toxoplasmosis* **is subclinical in about 75% of infected newborn.**

**CASE STUDY**

**E.K a 62 year old lady presented with a 3 months history of** *odynophagia* **that progressed to** *dysphagia.* **She had** *dyspepsia* **that was non-responsive to proton pump inhibitors. She gave history of slight weight loss due to inadequate food intake, because of the** *odynophagia*, *dysphagia* **and** *dyspepsia.* **She was in good general condition a febrile (Temp 37.1$^0$ C), not pale, no jaundice, no significant** *lymphadenopathy*, **nor** *oedema.*

**RESULTS**

**Recent findings have suggested an association between** *T. gondii* **infection and various Neurologic diseases or Psychiatric Syndromes such as** *Schistozophrenia*, **Alzheimer's disease and Suicide. 10% to 20% of patients with acute infection may develop cervical** *lymphadenopathy* or **flu-like illness.**

**RECOMMENDATIONS**

**Many of these aspects of disease may be delayed or prevented if treatment of** *toxoplasmosis* **is initiated antenatally and in the first 1 – 2 months after delivery.**

**Diagnosis and treatment must be different for each clinical category. In general, diagnosis accomplished using** *serology* **and** *histology*. **Isolation of the parasite can be difficult.**

**CONCLUSION**

**This being the first case in our literature, highliting the fact that; though rare** *oesophageal* **and gastric** *Toxoplasma* **infection can occur, leading to** *dysphagia* **and** *dyspepsia*, **Carnivorism of cats makes it difficult to keep them free of disease.**

**Immunodeficiency patients often have Central Nervous System (CNS) disease but may have** *Pericarditis* **or** *Pneumonitis. Toxoplasmosis* **in** *immunodeficiency* **syndrome patients may be due to reactivation of chronic infection or acquired.** *Toxoplasmosis* **in patients being treated with immunosuppressive drugs may also be due to newly acquired or reactivated latent infection.**

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## Introduction

*Toxoblasmosis* a Zoonotic disease caused by *Toxoplasma gondii. Toxoplasma gondii (T. gondii)* is a *protozoan* parasite that infects most species of warm blooded animals including humans. It is an obligate intracellular parasite and has a worldwide distribution (1).

Members of the cat family, *Felidae*, are the only known definitive hosts for the sexual stages of *T. gondii* These are the main reservoirs of infection.

*T.gondii* exists in three stages namely:
*Tachyzoites (trophozoites),*
*Bradyzoites* (cysts) and
*sporozoites* in *oocysts.*

*Tachyzoites* tend to rapidly proliferate and destroy all during acute infection. *Bradyzoites* slowly multiply in tissue cysts. *Sporozoites* exist in *oocysts* and are found in the gut walls of definitive hosts. *Tachyzoites* and *Bradyzoites* occur in body tissues and *oocysts* groups are excreted in stool from definitive hosts, the cat family (2).

Cats become infected with *T.gondii* by carnivorism or indigestion of *oocysts.* Humans can become infected by any of the 4 routes:

1. Eating undercooked meat of animals harbouring tissue cysts(3)
2) Consuming food or water contaminated with cat feaces or by contaminated environmental samples (such as fecal contaminated soil or changing the litter box of a pet cat (4)
3) Blood transfusion or organ transplantation (5)
4) Transplacentaly from mother to fetus (5)
5) Accidental inoculation of *tachyzoites*

## Disease Presentation

*Toxoplasmosis* can be categorized in to 4 groups:

1) Acquired in the immunocompetent patient
2) Acquired or reactivated in the immunodeficient patient
3) Ocular
4) Congenital

Diagnosis and treatment must be different for each clinical category. In general, diagnosis accomplished using serology and histology. Isolation of the parasite can be difficult. Molecular testing, such as *polymerase* chain reaction, plays a critical role in diagnosing infection in the fetus.

Acquired infection with *T.gondii* in immuno-competent individuals is generally asymptomatic. However, 10% to 20% of patients with acute infection may develop cervical *lymphadenopathy* or flu-like illness.

The clinical course is benign and selflimited. Symptoms usually resolve within weeks to months. Recent findings have however, suggested an association between *T. gondii* infection and various neurologic diseases or psychiatric syndromes such as *Schistozophrenia,* Alzheimer's disease and Suicide.

Immunodeficiency patients often have Central Nervous System (CNS) disease but may have *Pericarditis* or *Pneumonitis.* T*oxoplasmosis* in immunodeficiency syndrome patients may be due to reactivation of chronic infection or acquired. *Toxoplasmosis* in patients being treated with immunosuppressive drugs may also be due to newly (acquired) or reactivated latent infection.

*Ocular Toxoplasmosis,* an important cause of *chorioretinitis* , may be the result of congenital *toxoplasmosis* or acquired infection. Congenitally infected patients can remain asymptomatic until the second or third decades of life; when lesions develop in the eye presumably due to cyst rupture and subsequent release of *tachyzoites* and *bradyzoites.*

*Chorioretinitis* is often bilateral in 30% - 80% in congenitally acquired individuals than in individuals with acute acquired *T. gondii* infection.

Congenital *Toxoplasmosis* has a wide spectrum of clinical manifestation. It is subclinical in about 75% of infected newborn. The severity of clinical disease in Congenitally infected infants is related inversely to the gestational age at the time of primary maternal infection.

When clinically apparent, it may mimic other diseases of the newborn. Spontaneous abortions, prematurity or still birth may result in a proportion of cases. Involvement of CNs is a hallmark of congenital *Toxoplasma* infections which is characterized by classic triad of *chorioretinitis, intracranial*

Calcifications and *hydrocephalus.* Features of severe congenital *toxoplasmosis* include fever, *microcephalus , hepatosplenomegaly ,* jaundice, convulsions, bilateral chorioretinitis rash, (maculopapular rash, *petechiae*, or both), *mycocarditis* pneumonia and respiratory distress, hearing defects, *thrombocytopaenia , lymphocytosis* , monocytosis, *erythroblastosis* like picture and nephronic syndrome.

Some infected children with no clinically disease in neonatal period may present with *chorioretinitis strabismus*, blindness, *hydrocephalus* or *microcephaly,* cerebral calcification, deafness month or years later, delayed milestones and epilepsy. Many of these aspects of disease may be delayed or prevented if treatment of *toxoplasmosis* is initiated antenatally and in the 1 – 2 months after delivery.

Treatment of congenial *toxoplasmas* involves administration of *pyrmethamine sulfadiazime* and *leucoviron*. Treatment before birth and in the first year of life leads to significant reduction in sequelae.

Current treatment regimens work for actively dividing *tachyzoite* form of *T . gondii* but do not work on encysted organisms *(brachyzoites).* Newer drugs that include *azithromycin, atovaquone* and *spiramycin* may have activity on other forms of *T. gondii.*

## Case Report

E.K, a 62 year old lady presented with a 3 months history of *odynophagia* that progressed to *dysphagia.* She also had *dyspepsia* that was non-responsive to proton pump inhibitors.

She gave history of slight weight loss due to inadequate food intake because of the *odynophagia, dysphagia* and *dyspepsia.* On examination, she was in good general condition afebrile (Temp 37.1$^0$C), not pale, no jaundice, no significant *lymphadenopathy,* no *oedema.*

## Study

## *Method & Design*

On abdominal examination, there was no distension and moved with respiration, tender *epigastrium* but no masses were felt. Upper *gastrointestinal endoscopy* (GI) done on 21st. April 2018 showed several large *oesophageal* ulcers ( measuring 2-3 cm).

The ulcers were found on swollen area. She had similar ulcers in the gastric *antrum.* There were few duodenal erosions. Rest of the upper GI. Endoscopy was normal. Biopsies were taken from the *oesophageal* and gastric ulcers and were sent histology. Histology of the samples was reported .

## Histology Results

Sections *(oesphageal)* had *oesophageal* tissue exhibiting transmural *necrotising granulomatons* inflammation. There were numerous *histiocyctes* and *eosinophils* were seen within the *granulomas* and *epitheleoid* cells were clusters of ovoid parasites – *tachyzoites.*

There were multiple ulcers in the lining *epithelium.* Also seen were large granular structures lined by bland *columnar cells.* Sections from gastric biopsies showed gastric tissue exhibiting moderate plasma *lymphocyte* and *eosinophil* infiltrate into the *lamina propria.* There were clusters of *tachyzoites* seen as well.

## Histology Pictures

These are features compatible with *toxoplasma oesophagitis* and *gastritis*

## *Other Investigations*

## Laboratory Tests:-

| | |
|---|---|
| **Liver function test** | - Normal |
| **Full Haemogram** | - Normal |
| ***Toxoplasma* Antibodies 1gG 1.48iu/ml** | - Elevated. |
| When less than 1.0iu / m) | - Normal |
| **1gm** | - Non reactive. |
| **HIV Screening** | - Negative |

**Abdominal ultrasound:**

*Hepatomegaly* with fatty liver grade II.

**A CT** scan of brain was normal.

## Treatment

E.K was put on *pyrimethamine* and *sulphadoxine* (fansidar)(75mg of *pyrimethamine* and 500mg of *sulphadoxine* (3 tablets of fansidar taken once daily for 21 days)

She reported improvement in *dysphagia* and *odynophgia* within 7 days and the symptoms where completely gone in 14 days. By the end of treatment she was well.

Endoscopy was not repeated because the patient felt it was not necessary since she had fully recovered.

Current treatment regions work for actively dividing *tachyzoites* firm of *T. gondii* but do not work on encysted organisms *(brachyzoidea).* Newer drugs that include *azithromycin atovaquone , spiramycin* may have activity on other forms of *T. gondii*

Treatment of congenital *toxoplasmsis* involves administration of *pyrimethamine*, *Sulfadiazine* and *Leucovorin* for at least 1 year when initiated before birth leads to significant reduction of *sequalae.*

## Discussion

B. K. 57 years old lady who keeps about 5 cats ranging in age 3 months to 5 years. She indeed lives closely with her pets and the cats feed on what she feeds as well and she regards then as friendly and clean animals.

E.K. most likely acquired her infection from ingestion of *ocysts* from the cat feaces through contamination of food or water.

This is the first case in the literature, highliting the fact that though rare oesophageal and gastric *toxoplasma* infection can occur, leading to *dysphagia* and *dyspepsia,* but care of cats is important.

## Recomendations

Eventhough this could be the first case study in humans, a lot has to be taken into account to stop the spread of *Toxoplasmosis* **a Zoonotic disease caused by** *Toxoplasma gondii.*

Carnivorism of cats makes it difficult to keep them free of disease.

Cats shed *oocytes* for 1 to 2 weeks of their lives. It is estimated that 11% of cats worldwide shed *oocytes* at any given time. E. K probably had increased risk due to her large number of cats.

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