Oesophageal And Gastric Toxoplasmosis: Rare Presentation Of An Emerging Zoonotic Disease
Lodenyo H.¹, Rogena E.², Sitati S².

1. Kenya Medical Research Institute
2. Kenyatta University

Corresponding author: Lodenyo H. Email address lodenyo@gmail.com.

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°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.

Introduction

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 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 immunocompetent 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. 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.

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), myocarditis pneumonia and respiratory distress, hearing defects, thrombocytopenia, 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 pyrmyethamine 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°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 (oesophageal) had oesophageal tissue exhibiting transmural necrotising granulomatos inflammation. There were numerous histiocytes 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:**

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liver function test</td>
<td>Normal</td>
</tr>
<tr>
<td>Full Haemogram</td>
<td>Normal</td>
</tr>
<tr>
<td>Toxoplasma Antibodies IgG 1.48iu/ml</td>
<td>Elevated</td>
</tr>
<tr>
<td>When less than 1.0iu / m</td>
<td>Normal</td>
</tr>
<tr>
<td>1gm</td>
<td>Non reactive.</td>
</tr>
<tr>
<td>HIV Screening</td>
<td>Negative</td>
</tr>
</tbody>
</table>

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) (75 mg of pyrimethamine and 500 mg 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 were 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 sequelae.

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 them as friendly and clean animals.

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

This is the first case in the literature, highlighting 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 oocystes for 1 to 2 weeks of their lives. It is estimated that 11% of cats worldwide shed oocystes at any given time. E.K probably had increased risk due to her large number of cats.

References