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Toxoplasmosis in Cats: Its Prevention and Management

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Abstract:

Toxoplasma gondii is an intracellular protozoan parasite of global significance, with domestic cats serving as the primary definitive hosts. Infection occurs mainly through ingestion of tissue cysts in prey or oocyst-contaminated food and water, leading to intestinal replication and oocyst shedding into the environment. Although most cats remain asymptomatic, clinical toxoplasmosis may occur, particularly in immunocompromised cats and congenitally infected kittens, presenting with neurological, ocular, respiratory, or systemic signs. Diagnosis is often challenging and requires a combination of serology, faecal examination, molecular assays, and clinical assessment. Clindamycin is the drug of choice, though prognosis is guarded in severe pulmonary or hepatic involvement. Preventive strategies, including feeding cooked or commercial food, restricting hunting, and maintaining hygiene, are essential to reduce environmental contamination and zoonotic transmission. Given the significant public health implications, awareness and preventive measures in feline populations are vital for effective management of toxoplasmosis.

Keywords: Toxoplasma gondii, cats, oocysts, tachyzoites, clinical signs, diagnosis, treatment, prevention.

Introduction:

Toxoplasmosis is a disease resulting from infection with *Toxoplasma gondii*, a protozoan parasite that lives inside host cells and belongs to the phylum Apicomplexa and family Sarcocystidae. This parasite has a worldwide distribution and demonstrates complex epidemiology, as it can infect nearly all warm-blooded animals through a two-host life cycle. Felids, particularly domestic cats, serve as the definitive hosts of the parasite, while intermediate hosts include a wide range of non-feline species, including humans and dogs. Once an individual is infected, the parasite often persists in tissues for life. The parasite's success lies in its three distinct stages: tachyzoites, which rapidly multiply in tissues and cause pathology; bradyzoites, which form latent cysts within muscles, viscera, and the central nervous system, ensuring

lifelong infection; and oocysts, which represent the resistant and infective stage shed into the environment. Transmission occurs horizontally through ingestion of contaminated food, water, or tissue cysts, and vertically via congenital infection. While the infection in cats typically results in low morbidity and mortality, its clinical implications remain important in veterinary practice. Prevalence studies indicate geographic variation, with antibody positivity ranging from 16–40% in the United States to 24% in Portugal, although actual oocyst shedding is relatively rare. Environmental contamination can reach 90–5000 oocysts per square meter annually. Shedding rates are not influenced by the cat's age but are higher between July and December in the northern hemisphere. Non-vaccinated cats are more vulnerable to clinical toxoplasmosis, which presents with variable and sometimes fatal outcomes. However, accurate diagnosis is challenging, and many cases are likely underrecognized, underscoring the need for veterinarians to understand feline toxoplasmosis for both clinical management and zoonotic risk control.

Pathogenesis:

Enteroepithelial Life Cycle:

This particular cycle occurs exclusively in feline hosts. Most cats acquire infection by preying on intermediate hosts, usually rodents, that harbour tissue cysts. When these cysts are ingested, digestive enzymes in the stomach and intestines break down the cyst wall, releasing bradyzoites. After invading the epithelial cells of the small intestine, bradyzoites mature into schizonts and continue through five programmed stages of asexual reproduction.The resulting merozoites differentiate into male and female gamonts, which, after fertilisation, form oocysts surrounded by a protective wall. The oocysts are generally round to oval in form, measuring approximately 10 × 12 µm, and are not infective at the time of being shed in feces. Within 1–5 days of exposure to air and moisture, however, they undergo sporulation, producing two sporocysts that each contain four sporozoites, making them infective. Consumption of tissue cysts leads to completion of the enteroepithelial cycle within 3–10 days in cats, and this pathway is responsible for nearly 97% of first-time infections.In less common cases where cats ingest oocysts or tachyzoites, oocyst production is delayed and may persist for up to 18 days or longer, though only about 20% of cats fed oocysts actually shed them.

Extraintestinal Life Cycle:

For cats, dogs, humans, and other susceptible hosts, the extraintestinal phase of T. *gondii* follows a common course irrespective of the infective form consumed. Once oocysts are ingested, they discharge sporozoites into the small intestine, where these organisms invade epithelial cells, including those of the lamina propria. These sporozoites divide asexually by a process called endodyogeny, transforming into tachyzoites. Tachyzoites are crescent-shaped, measuring about 6 × 2 µm, and are capable of replicating within almost any type of host cell. When the host cell ruptures, newly released tachyzoites invade additional cells, allowing infection to spread. Over time, tachyzoites may persist intracellularly and

eventually encyst. Tissue cysts, measuring between 15–60 µm, usually adapt to the shape of the host cell and are predominantly found in the central nervous system, muscles, and visceral organs, where they can remain throughout the host's life. Under conditions of immunosuppression, latent cysts may reactivate, resulting in clinical disease. During pregnancy, parasitaemia can cause placental inflammation and allow tachyzoites to cross into the fetus. Consequently, kittens born to queens infected during gestation may acquire the parasite either transplacentally or through nursing. Clinical disease is frequently observed in such kittens, with severity depending on the stage of gestation at the time of infection, and some neonates may also shed oocysts.

Transmission:

Toxoplasma gondii can be transmitted through three primary routes across all host species: congenital infection, ingestion of tissue cysts in infected meat, and consumption of food or water contaminated with oocysts. Less commonly, transmission may occur through blood transfusions or organ transplantation. There is also evidence to suggest lactogenic transmission, as the parasite has been identified in the milk of infected queens. In humans, eating undercooked or raw meat containing tissue cysts is considered the most significant source of infection, and serological surveys show relatively high antibody prevalence worldwide. Additionally, exposure to oocyst-contaminated soil and water is a common route of transmission. Several waterborne outbreaks of toxoplasmosis reported globally reinforce the concern that environmental oocysts represent a major public health risk.

Clinical Signs:

In cats, clinical disease due to *Toxoplasma gondii* is relatively rare and usually arises from tissue damage and inflammation triggered by the intracellular multiplication of tachyzoites. Congenital infections are generally more severe than infections acquired during adulthood. Most cases of clinical toxoplasmosis arise from reactivation of dormant tissue cysts rather than from newly acquired infections. In immuno-compromised cats, such as those with feline immunodeficiency virus (FIV) or feline leukaemia virus (FeLV), bradyzoites within cysts can convert back to tachyzoites, leading to widespread dissemination. The central nervous system, muscles, lungs, and eyes are the organs most frequently affected, while hepatic and pancreatic involvement is less common. Clinically, affected cats may present with neurological abnormalities (such as seizures or ataxia), muscle sensitivity, respiratory distress, ocular inflammation, jaundice, diarrhoea, fever, anorexia, weight loss, depression, and lethargy. Transplacentally infected kittens are particularly vulnerable, often developing hepatitis, cholangiohepatitis, pneumonia, and encephalitis, along with signs of ascites and severe respiratory compromise. In generalized toxoplasmosis, pneumonia is a key clinical feature, and in advanced cases, acute respiratory distress syndrome and septic shock may develop. Ocular toxoplasmosis can occur even without systemic involvement, presenting as anterior or posterior uveitis, iritis, chorioretinitis, or iridocyclitis. Secondary changes such as aqueous

flare, keratic precipitates, lens luxation, glaucoma, and retinal detachment are commonly observed. For this reason, fundic examination is recommended in febrile cats. Immune complex accumulation and delayed hypersensitivity are potential mechanisms involved in the chronic stage of toxoplasmosis. Because the parasite is never fully eliminated from the host, either naturally or with treatment, clinical recurrence is always possible.

Diagnosis:

Detection of oocyst shedding is typically achieved through microscopic examination of faecal samples. A definitive diagnosis of toxoplasmosis, however, requires identification of the parasite within body fluids or tissue samples. When obtaining such specimens is not feasible, a presumptive diagnosis may be made based on increasing IgM antibody levels, the elimination of alternative causes for the clinical signs, and a positive therapeutic response to anti-T. *gondii* medications.

i. ***Detection of oocysts in faeces:*** *Toxoplasma gondii* oocysts, measuring approximately 10–12 µm, are most reliably detected during the shedding phase using centrifugation techniques with solutions of specific gravity 1.18 g/ml. Morphologically, these oocysts are indistinguishable from those of related parasites such as *Hammondia hammondi*, *Besnoitia oryctofelisi, and Besnoitia darlingi.* The use of cesium chloride has been applied as a specialized method for purifying *T. gondii* oocysts from cat faeces.

ii. ***Detection of tachyzoites:*** The preferred method for ante-mortem diagnosis of clinical toxoplasmosis is direct identification of the parasite through cytology or polymerase chain reaction (PCR). During the acute phase of infection, tachyzoites may be observed in different tissues and body fluids. Although rarely present in blood, they can occasionally be detected in cerebrospinal fluid (CSF), aqueous humour, fine-needle aspirates of lymph nodes and other organs, as well as in transtracheal or bronchoalveolar lavage samples. The detection of tachyzoites serves as definitive proof of infection. Alternatively, PCR testing of CSF, aqueous humour, or bronchoalveolar lavage fluid can be employed for confirmation.

iii. ***Detection of antibodies:*** Immunofluorescence assay (IFA) can be used to detect antibodies of the IgM, IgG, and IgA classes. In terms of public health risk assessment, antibody testing in healthy cats provides valuable information. A cat that tests negative for antibodies may still be shedding oocysts, particularly during the early stages of infection before antibodies have developed, and such cats are more likely to shed if exposed for the first time. Cats with detectable antibodies generally do not shed oocysts, as antibody production requires 2–3 weeks, by which time shedding has ceased, and most cats shed just once over their entire life. Even if re-exposed or immunosuppressed, these cats are unlikely to shed again. Antibodies are frequently detected in both clinically ill and healthy cats, meaning their presence does not confirm active disease. Moreover, many clinically normal cats show IgM antibodies, which

are not a dependable indicator of disease symptoms.

iv. ***Radiographic findings:*** In feline cases of pulmonary toxoplasmosis, radiographs often reveal interstitial or alveolar patterns, whereas pleural effusion is infrequently observed. Abdominal imaging results are generally non-specific but may show uniform radiodensity associated with peritoneal effusion, as well as hepatomegaly, lymph node enlargement, intestinal masses, or reduced contrast in the cranial right abdominal quadrant, which can indicate pancreatitis. In cats with central nervous system involvement, diagnostic imaging such as myelography, computed tomography (CT), or magnetic resonance imaging (MRI) may reveal mass lesions.

Treatment:

Clindamycin, administered at 10–12.5 mg/kg orally every 12 hours for four weeks, is the drug of choice for treating feline toxoplasmosis and typically produces noticeable improvement within a week. However, if the medication lodges in the esophagus, it may cause localized esophagitis or stricture formation, so owner should be advised to follow administration with food or water. In cats with central nervous system involvement, some clinicians prescribe higher doses (20–50 mg/kg orally every 12 hours for four weeks). For cases involving systemic disease and ocular complications such as uveitis, clindamycin is often combined with glucocorticoids—administered topically, orally, or parenterally—to reduce inflammation and prevent secondary issues such as glaucoma or lens luxation. In most cases, administering prednisolone acetate 1% eye drops three to four times a day is effective; oral prednisolone at 1–2 mg/kg/day is reserved for more serious conditions and should be tapered slowly depending on the animal's progress. Non-ocular and non-neurological symptoms usually improve within 2–3 days of clindamycin therapy, whereas ocular and CNS signs take longer to resolve. Pulmonary toxoplasmosis, in particular, may require several weeks for radiographic changes to normalize. Prognosis is guarded in cats with pulmonary or hepatic involvement, especially in immunocompromised animals. Supportive therapy, including fluid replacement and appetite stimulation, may also be necessary in some cases.

Do immunosuppressive treatments trigger reactivation of toxoplasmosis in previously infected cats?

Administration of glucocorticoids has been linked to reactivation of infection, leading to oocyst re-shedding in cats carrying latent *T. gondi*i. In contrast, an experimental study using cyclosporine at 7.5 mg/kg orally once daily reported no reactivation of latent infection in healthy cats. Nonetheless, multiple case reports have documented cyclosporine-associated reactivation of toxoplasmosis, some of which proved fatal. For this reason, it is recommended to perform T. gondii and retrovirus serology prior to initiating cyclosporine or other immunosuppressive treatments. Experimental evidence also suggests that clindamycin, administered at 20 mg/kg daily for three to four weeks, can prevent oocyst re-shedding in dexamethasone-immunosuppressed cats. Consequently, prophylactic clindamycin therapy has been

proposed before starting cyclosporine to reduce the risk of toxoplasmosis reactivation. Ultimately, the potential for reactivation should always be discussed with the caregiver, and treatment decisions should be tailored to individual cases.

Prevention:

Control of toxoplasmosis in cats focuses on minimizing both the occurrence of infection and the release of oocysts into the environment. Feeding cats commercially prepared and processed diets is strongly recommended, as the prevalence of *T. gondii* infection is notably higher in regions where raw meat is commonly provided. Tissue cysts can be effectively destroyed by freezing or irradiation without compromising meat quality. Cats should be restricted from hunting and consuming intermediate hosts such as rodents, as well as mechanical carriers like cockroaches and earthworms. If meat is offered, it must be thoroughly cooked regardless of prior freezing. Additionally, cats should be kept away from facilities housing food-producing animals and feed storage areas to avoid contamination and transmission risks.

Conclusion:

Toxoplasma gondii infection in cats is a significant concern for both veterinary medicine and public health, as felids act as definitive hosts and major contributors to environmental contamination. While the majority of infections remain asymptomatic, young kittens and immunocompromised cats may develop severe illness, emphasizing the need for prompt recognition and management. Clindamycin continues to be the treatment of choice, and preventive strategies, including proper diet and restriction of hunting behaviour, are vital to limit oocyst shedding. Enhancing awareness among veterinarians and cat owners is essential for improving disease control and reducing the risk of zoonotic transmission.

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