Zoonotic Protozoal Diseases of Companion Animals

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Abstract

Diseases that can be transmitted between animals and humans either through direct interaction with animals or some inanimate objects and vectors are known as Zoonotic diseases. The risk of zoonotic protozoal diseases has been increased due to keeping animals as pets. Major Zoonotic protozoans include *Toxoplasma, Giardia, Trypanosoma, Leishmania* and *Babesia* cause infection in humans in which animals act as asymptomatic Carrier. Some Other protozoa like *B. Coli, Sarcocystis* and *Cryptosporidium* also cause infection in humans. *Trypanosoma cruzi* is the cause of Chagas' disease, *Toxoplasma gondii* is the cause of Toxoplasmosis, *Giardia* is the cause of Giardiasis, and *Entamoeba histolytica* is the cause of human dysentery. Immunocompromised individuals are at a higher risk of zoonosis than immunocompetent such as diabetic patients or pregnant women. In this chapter, we mainly focus on the epidemiology, mode of transmission, life cycle, Pathogenesis, clinical signs of protozoal diseases in animals and humans and preventive measures.

Keywords: Zoonosis, Protozoa, Vector-borne, Transmission, Antibiotics, Diarrhea.

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Introduction

Toxoplasmosis

Etiology and Epidemiology

Toxoplasmosis is caused by a tissue cyst-forming coccidian. *Toxoplasma gondii (T. gondii)* is an apicomplexan kind. *T. gondii* is common throughout the world in domesticated as well as stray cats, in which cats act as definitive (final) hosts. It spreads to mammals through direct contact with cat feces (Tenter et al., 2000). Even if the directive information is lacking, the European Commission has asked the European Food Safety Authority (EFSA) for recommendations on surveillance and control strategies for toxoplasmosis in humans, animals, and food. In Europe, up to 74% of adult cats have antibodies to *T. gondii*, depending on their diet and shelter (Kuruca, et al., 2023). About one third of the human population is affected by *T. gondii* throughout the world. Toxoplasmosis is vertically transmitted in kittens.

Life Cycle

A Cat sheds oocysts of *T. gondii* in their fecal material within 3-10 days, and continues oocyst shedding up to 7-21 days after primary infection. In this, a cat sheds hundreds to thousands of oocysts. Direct or indirect contact with excrement can spread toxoplasmosis (Figure 1). Toxoplasma also spreads through blood transfusion or organ transplantation of infected animals or humans (Dubey, 2005). Sporulation occurs one to five days following shedding in feces. The excystation depends upon pH, temperature, bile salts and Trypsin with the intestinal environment. They develop into tachyzoites (infective stage). After cellular infection, Tachyzoites develop into Bradyzoite-containing tissue cysts. Oocysts are shed by cats and subsequently consumed by herbivores, which serve as a host in between. Humans contract the disease when they consume or drink contaminated water or food that contains feline feces. It also spreads by consumption of undercooked meat containing oocysts, through the placenta (vertical transmission) from mother to fetus (Dumètre, et al., 2013).

Clinical Signs

This is a multi-systemic disease. In Trans placentally affected kittens, it causes stillbirth or death of kitten before weaning. It causes abnormalities in different systems; Respiratory, Gastrointestinal and Reproductive system. Ocular abnormalities are also observed like uveitis, keratitis, detached retina and neurological abnormalities like torticollis, circling, seizures etc. in 10% of patients (Powell & Lappin, 2001). The virulence of Tachyzoites depends upon different parasitic factors like factors required for motility, immune invasion and cellular invasion. In Pups, it induces vomiting, diarrhea, fever, anorexia, and dyspnea while in adult dogs, it causes local infection related to the neural and muscular system. It causes neurological manifestations in dogs. It causes abortion in ewes and neonatal death of lambs and if survive, they are weak.

Zoonotic Potential

It is of serious concern in immunosuppressed individuals and pregnant women. They should avoid direct contact with oocysts shedding in the feces, contact with soil and litter of cat. This causes parasitemia during pregnancy which leads to neonatal death and if abortion does not occur, it causes abnormalities in different parts of the newborn child (Khairullah et al., 2024). It is spread by ingestion of raw or undercooked beef, mutton and pork, vegetables, unpasteurized milk of infected animals and contaminated water with cat's feces. Neonatal abnormalities include: retinochoroidites, myocarditis, encephalitis, low-grade fever, cerebellar necrosis, and general lymphadenopathy and their maybe hepatosplenomegaly, liver failure, hydrocephalus and convulsions (Hussain et al., 2017).

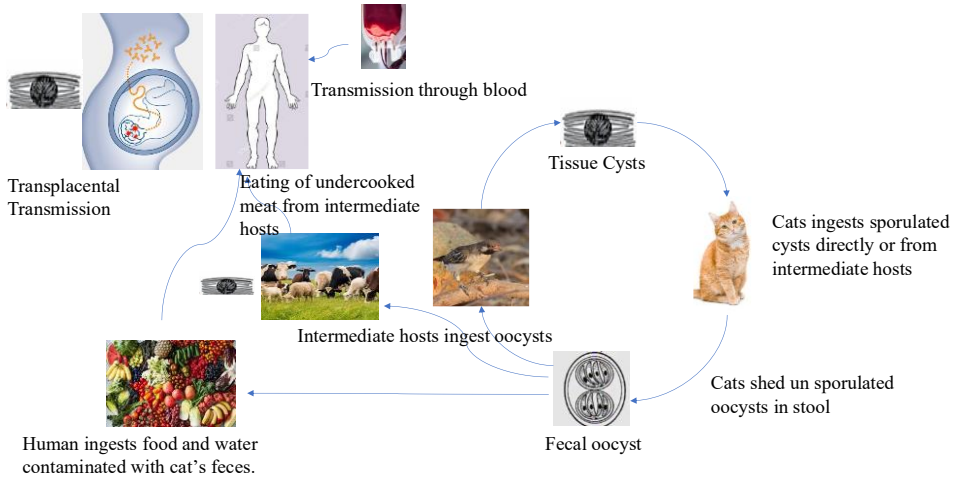

Fig. 1: Life cycle of Toxoplasma gondii

Diagnosis

Specific diagnosis is made by serological testing like the Immunofluorescent Antibody test (IFA) and direct Sabin-Feldman dye test. Recently, an ELISA test has been developed for the detection of IgM antibody levels than IgG. (Maenz et al., 2014).

Treatment and Control

There is no specific treatment for toxoplasmosis. Clindamycin is used in cats for its treatment, while in humans, the combination of antimalarial drug and Sulphadrugs is used for an effective treatment (Rajapakse et al., 2013).

Toxovax is a live vaccine that is used in sheep. We can control toxoplasmosis by adopting different strategies; daily cleaning of the litter box, proper disposal of fecal material, washing of hands before eating, wearing gloves while gardening and handling cat's feces. The prevention of this disease is necessary in case of pregnant females and if the person is suffering from some immunocompromised disease like AIDS, Diabetes etc. This protozoan causes abortion in pregnant females of humans and other animals (MSD Animal Health, 2022).

Giardiasis

Etiology and Epidemiology

Giardiasis is caused by protozoa, Giardia species that belongs to Mastigophora class. Mastigophora Class have one or more flagella which helps in their locomotion. *Giardia duodenalis (G. duodenalis)* also known as *G. lamblia or G. intestinalis* is mainly responsible for this disease. Giardia has bilateral symmetry and possesses eight flagella, six of which are free (Adam, 2001). Giardiasis is more common in dogs than cats. Every year, approximately 280 million cases of giardiasis are recorded worldwide, with 20,000 reported in the United States. There are 7 different genotypic assemblages from A to F. Assemblages A and B have highly zoonotic potential, Assemblages C and D are Canine oriented, while assemblages F is feline oriented. Assemblage C, D and F are predominant in companion animals whether they are in kennels, catteries and houses. They can become infected by zoonotic assemblages (Feng & Xiao, 2011).

Life Cycle

The life cycle of *G. duodenalis* is similar in cats, dogs, and humans. Trophozoites and giardia multinucleated cysts are shed in the feces of the carrier or diseased host which will contaminate the groundwater. Giardia cysts are highly resistant to the environment and they can persist up to months in soil or water. Transmission occurs through the fecal-oral pathway by consuming contaminated food and water (Figure 2). Excystation of cysts occurs in the intestine of host and two trophozoites are released from each cyst (Adam, 2001). Trophozoites remain there either in free form or attach to the intestinal wall, which in turn causes clinical signs. They multiply in the small intestine and move towards colon, encyst themselves and are again excreted through feces. Trophozoites are released from the intestine but cannot survive outside. Infective cysts passed in fecal material cause contamination of ground water, vegetables grown in soil and also fomites or hands keep in touch with contaminated water or other materials (Espinosa-Cantellano &Martinez-Palomo, 2000).

Clinical Signs

Kittens and puppies mostly show clinical signs of Giardiasis, while adults remain asymptomatic. Adults are the primary source of infection for humans. Clinical symptoms can be transient or persistent. Malabsorption syndrome is defined by soft, oily, frothy stools with a rotten odor

and copious feces. There is no consistent relationship between Giardiasis with diarrhea and gastrointestinal problems in companion animals (Thompson et al., 2009).

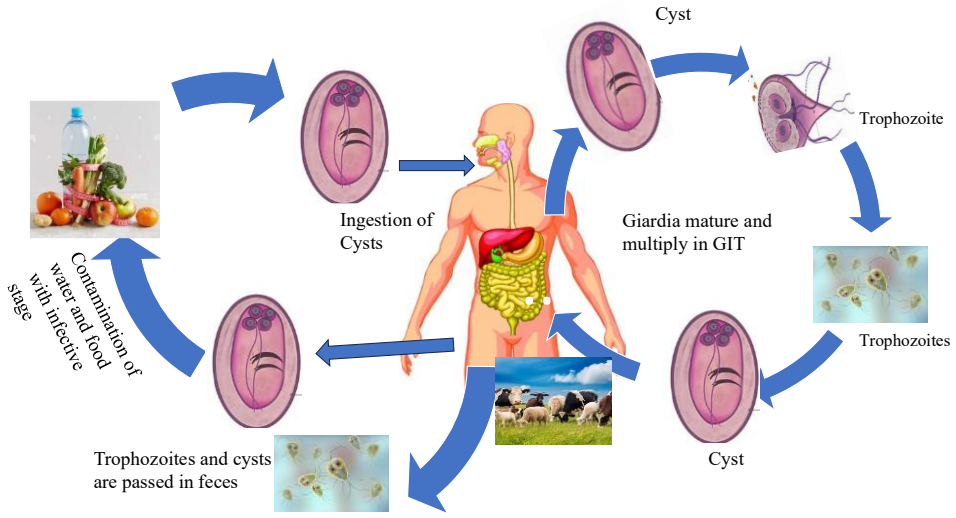

Fig. 2: Life cycle of Giardia

Zoonotic Potential

In North America, *Giardia* is the most commonly found protozoa in humans. Mostly, Giardia is anthroponotic of humans. The main symptoms that appear in humans are smelly diarrhea, abdominal cramps, Flatulence, weight loss, fatigue and bloating. These symptoms vary from mild to severe depending on number of ingested cysts and the immune status of the individual (Wang &Duan, 2020).

Diagnosis

We can identify Giardia cysts and trophozoites in feces. Centrifugal floatation of fresh feces using zinc sulphate as a floatation solution is the preferred method for identification of the cysts. ELISA-based kits are available for antigen detection in feces and PCR testing is also done in commercial laboratories for Giardia (Hooshyar et al., 2019).

Treatment, Prevention and Control

Giardiasis prevention is dependent on appropriate cleanliness of the water supply and avoiding fecal contamination of groundwater. Use purified water for drinking purposes. Effective preventive measures like hand washing after handling animal garbage, soil, diapers or going to the bathroom; Human and animal waste should be properly disposed of, and youngsters with diarrhea should not be allowed to participate in recreational water activities. Use fruits and vegetables after washing. We can also prevent this through proper disinfection and cleaning of kennels (CDC, 2024).

We use different antiparasitic drugs and their combinations for the treatment of Giardiasis in different animals. In dogs and cats, Fenbendazole @50mg/kg is used for 3 days and 5 days respectively per orally every 24 hours. Metronidazole is also used in humans and dogs for the treatment of Giardiasis. It is used @20- 22 mg/kg PO every 12 hours for 5-8 days. A combination of fenbendazole and metronidazole provides better results and reduction in cyst shedding. A combination of Praziquantel and pyrantel Pamoate can also be used. Albendazole can be administered to dogs and cats at a dose of 25 mg/kg PO; however, because to its teratogenic effects, it should not be used during pregnancy (Ciuca et al., 2021).

American Trypanosomiasis (Chagas' disease)

Etiology and Epidemiology

The hemoflagellate protozoan *Trypanosoma Cruzi (T. cruzi)* is the causative agent of the zoonotic parasitic disease known as American trypanosomiasis. It is a vector-borne diseases through different triatome insect species i.e. reduviid bugs. *T. cruzi* can infect all mammal species, however avian species are resistant. The World Health Organization (WHO) estimates that roughly 10 million people have Chagas disease in America, i.e. Latin America (WHO, 2024). It is widespread throughout America, with endemic occurrence by transmission through triatomes in Mexico, South America, and Central America. *T. cruzi* is found in over 100 mammalian species. All countries in South America, North America, and Mexico have endemic transmission, making it a prevalent disease throughout America.

Host

T. cruzi uses a wide variety of mammalian species as reservoir hosts, including domestic dogs, armadillos, opossums, raccoons, woodrats, and humans. The genera *Triatomine, Rhodnius* and *Panstrongylus* contain common triatomine species that are trypanosomiasis vectors (Bern et al., 2011).

Life Cycle

T. cruzi is a vector-borne protozoal disease spread through contaminated feces of distinct triatome bug species. The life cycle of *T. cruzi* was explained by Carlos Chagas in 1909. Metacyclic Trypomastigotes is the infective stage, while Trypomastigote is the diagnostic stage of *T.*

cruzi. The life cycle of *T. cruzi* takes place in two hosts; triatomine bug stage and Mammalian stage (Figure 3). When a bug consumes blood, it passes metacyclic trypomastigotes in its excrement, which enter the mammalian host body through a cut or some mucous membrane, such as the conjunctiva of the eye (Martín-Escolano et al., 2022).

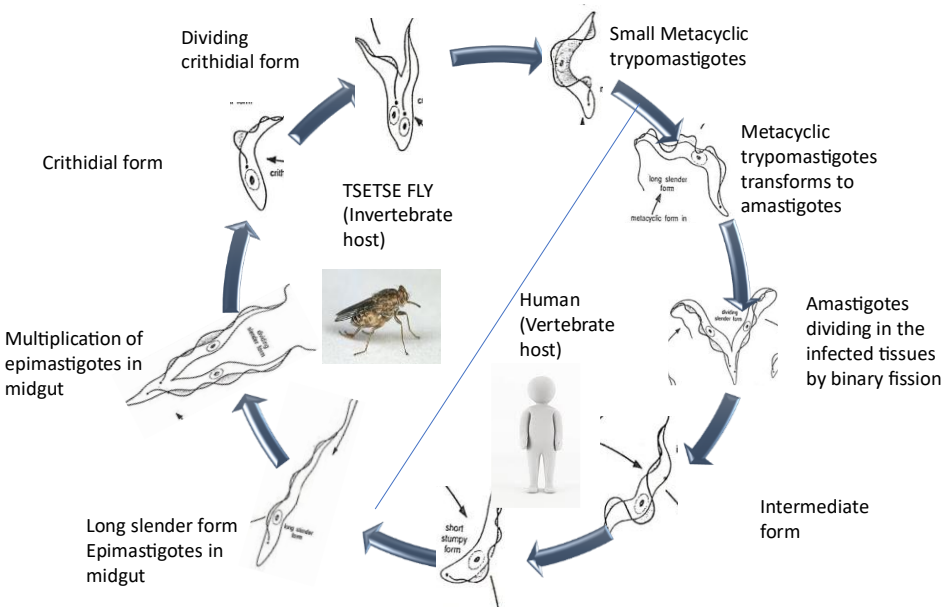

Fig. 3: Life cycle of Trypanosoma Cruzi

At the wound site, it penetrates further deep into various cells and transforms into amastigotes. Here, amastigotes of protozoa multiply itself through binary fission at the infection site. Amastigotes when enter in cells converted into trypomastigotes, which cause cell lysis and enter into the bloodstream. Here, trypomastigotes are easily identified on blood smears. When the triatomine bug takes the blood meal of the infected host, trypomastigotes are also ingested. Trypomastigotes transform into epimastigotes in the midgut of a bug and multiply there. Then it moves toward hindgut of the triatomine bug and is converted into metacyclic trypomastigotes (Valenciano et al., 2013).

Clinical Signs

It is particularly common in younger dogs. Trypanosomiasis has two forms or phases: Transient and long-lasting. Acute occurs in dogs younger than 2 years of age while chronic appears in older dogs. The acute phase is commonly asymptomatic. However, it can be presented with non-specific symptoms. The intensity of this phase is determined by possible cardiac and neurological symptoms and indicators. At the site of inoculation, nodular lesions usually develop known as Chagomas. Periocular and palpebral firm swelling are the usual terms used to describe nodular lesions on the eyelids (Hide & Tilley, 2001). The majority of acute cases progress to a subclinical chronic form of the disease within a few weeks to months. Acute phase which is also known as indeterminate form. CNS is dys-functioning like in Canine Distemper, seizures and ataxia.

In the chronic phase (determinate form), there is dilated cardiomyopathy in older dogs. It may include gastrointestinal and cardiac involvement, and occasionally both together. If Amastigotes invade to smooth muscles of various organs, it may lead to mega esophagus, megacolon and dilated cardiomyopathy. It may lead to syncope, exercise intolerance and sudden death of dog (Nakamura et al., 2018).

Zoonotic Potential

Chagas disease is zoonotic. Clinical presentation in humans is; irregular heartbeat, heart failure, sudden cardiac arrest, vomiting, diarrhea, swelling of the region near the eye, stomach pain or constipation caused by an enlarged colon (megacolon), and difficulties swallowing caused by an expanded esophagus. There is also an enlarged liver and spleen (hepatosplenomegaly), and lymphadenopathy (WHO report, 2023).

Diagnosis

Trypanosomiasis is diagnosed based on clinical symptoms and laboratory results. Trypomastigotes are seen in circulating blood and cerebrospinal fluid (CSF) in the acute stage, the trypomastigote cannot be observed in circulating blood in the chronic stage of the disease. The diagnosis is also by serological testing against the antigen of T. Cruzi in IFA (immuno-fluorescent assay) and also by molecular testing. Amastigotes are discovered in biopsy samples of infected hosts using Hematoxylin and Eosin (H&E) or Giemsa stain (Bonnet, et al., 2015).

Treatment and Control

To treat Chagas' illness, chemotherapeutics is used. The preferred medication for treating Chagas' illness is benznidazole, however nifurtimox is an alternate. Dogs are administered benznidazole orally at a dose of 5–10 mg/kg daily for two months. As these drugs are not FDA-approved, so their use requires permission from CDC. The symptomatic treatment for cardiac failure and arrythmia is also recommended (Gomes et al., 2023).

There is no vaccine available for American trypanosomiasis, hence it is better to control disease transmission. Vector control is the most

important parameter for the prevention of this disease. Use of insecticide spray to dwellings and around the farm area, turning off outdoor lights at night helps to prevent the entry of bugs into animal area. Screening of blood donors is recommended. To prevent iatrogenic transmission, disinfection of contaminated surfaces is recommended with bleach 10% solution and 70% ethanol. Infected insects remain a source of infection up to 6 days after death (Camargo et al., 2022).

Leishmaniasis

Etiology and Epidemiology

Leishmania is a tropical and subtropical infection caused by a blood protozoa and it is transmitted to humans and other mammals by the bite of sand flies mainly Phlebotomus and Lutzomyia. Single-celled protozoa that belong to the genus *Leishmania* are the cause of leishmaniosis. It is a severe and chronic in dogs, humans and certain other mammals. It is endemic in 98 countries, majority of which are developing nations. Several species are involved causing different types of leishmaniasis (Torres-Guerrero, et al., 2017*)*. kala-azar disease is a rising epidemic in Pakistan and neighboring countries like Afghanistan caused by *Leishmania Tropica*. It is a serious public health problem in these countries. Major two types of leishmaniasis occur in different animals; visceral leishmaniasis which is also known as Kala-azar disease, cutaneous and mucocutaneous leishmaniasis. As stated by World Health Organization (WHO), it is one of the seven major tropical diseases and poses a serious health problem. The epidemiological trend of this disease is very diverse; 30 species of sandflies are proven vectors and 20 pathogenic species are identified for leishmania in humans. Only a small number of human or animal cases are documented in the US each year, but there are about 700,000 to 1 million new cases of human leishmaniasis and 20,000 to 30,000 fatalities occur annually worldwide. Most human cases of visceral leishmaniosis are documented in Brazil, Ethiopia, India, Kenya, Somalia, South Sudan, and Sudan.

Transmission

Leishmaniasis is a highly zoonotic disease that can spread from dogs to humans. People who are bitten by a sandfly or another bug are primarily infected with this disease that has previous record of biting an infected animal or human. Accidental or non-vector transmission like laboratory infection is very rare. This is also anthroponotic in nature along with zoonotic nature. This nature depends upon whether humans are the main reservoir host of this disease (Torres-Guerrero, et al., 2017). It is also known as forest yaws. Natural leishmania infections are found in non-human mammal hosts such as forests (primarily in marsupials, rodents, edentates, and carnivores). The main Leishmania species are *L infantum, L panamensis, L major, L. tropica,* and *L aethiopica*, which have different reservoir hosts that vary depending on location. Leishmania infections can be very severe in the presence of other immune suppressant diseases like AIDs/HIV.

Life Cycle

The life cycle of leishmania completes in two hosts. Sand fly takes a blood meal from host and injects the promastigote stage of protozoa into the skin. Promastigotes are phagocytized by macrophages and other types of mononuclear phagocytic cells (Figure 4). Then, promastigotes transform into amastigotes, where they multiply in cells of different tissues which are taken up by bugs or sandflies. The amastigotes multiply in the gut and transform into promastigotes, from where it moves into the proboscis. Promastigote is the infective stage while Amastigote is the diagnostic stage for leishmaniasis (Ahmad & Tuteja, 2019).

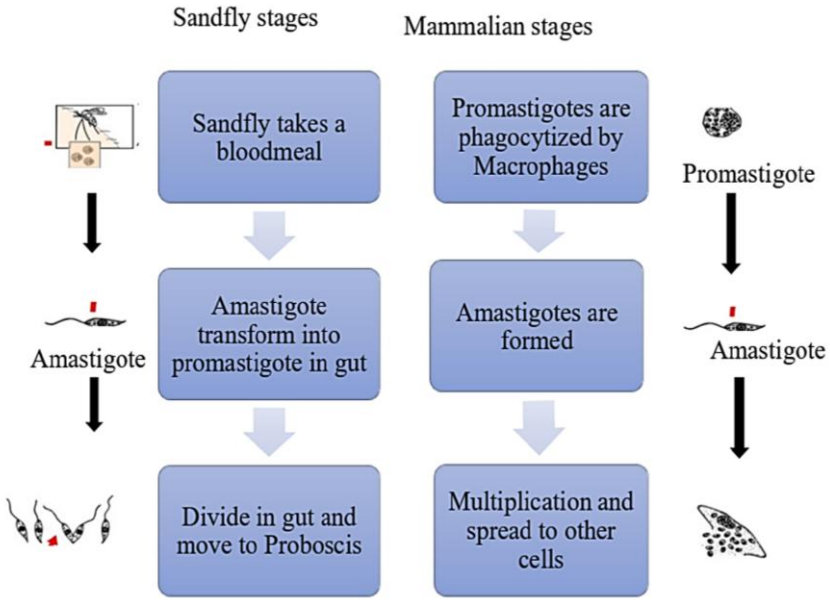

Fig. 4: Life cycle of Leishmania

Clinical Signs

There are cutaneous lesions on areas where flies bite the animals. There is weight loss, ocular abnormalities, epistaxis, abnormal nail growth, polyuria, polydipsia, lameness, colitis, melena and vomiting. There is lymphadenopathy, splenomegaly and exercise intolerance (Petersen, 2022).

Zoonotic Potential

The clinical manifestations of visceral leishmaniasis in humans are; Undulating fever, skin lesions, hepatosplenomegaly. There are some non-specific clinical signs like weight loss, and malaise. Some have other clinical signs such as cough, chronic diarrhea, lymphadenopathy, thrombocytopenia associated with petechial hemorrhages on mucous membranes, clinical signs of chronic kidney disease and pancytopenia. In mild cases of this disease, only a few clinical signs can be observed; for example, localized lymphadenopathy which may resolve on their own; however, most other cases are fatal if untreated (Spickler, 2023).

Treatment and Control

For treatment of leishmaniasis, N-methylglucamine antimoniate and allopurinol for 4-6 weeks and 6-12 months respectively. Miltefosine can be used as an alternative to N-methylglucamine and used along with allopurinol.

For its effective control, we can use insecticide spray to repel the insects from the herd. A collar impregnated with Deltamethrin or permethrin spot-on formulation to prevent flies from biting animals (Coura-Vital, et al., 2018).

Cryptosporidiosis

Etiology and Epidemiology

The parasitic protozoan species *Cryptosporidium* causes cryptosporidiosis, which infects numerous reptiles, amphibians and vertebrates, including humans, and results in gastrointestinal disorders (Uehlinger et al., 2013). *C. felis, C. murris, C. ryanae* and *C. parvum* can infect cats, while *C. canis, C. parvum, C. ubiquitum* and *C. andersoni* can infect dogs (Rosanowski et al., 2018). The most common cause of human cryptosporidiosis is *C. hominis, C. maleagridis, C. parvum,* as well as *C. felis and C. canis* is. Intimate companion animals like dogs and cats can be a source of human infection (Xu et al., 2016). Humans can become infected with these parasites through direct contact or by fecal contamination of food, water, or the environment (playgrounds, sandpits, and gardens). They have an Oro-fecal transmission cycle (Smith et al., 2007). There have been many foodborne and waterborne epidemics of cryptosporidiosis linked to cattle dung contamination of food or water (Blackburn et al., 2006). In Southeast Asia, cryptosporidiosis is common in both people and animals (Lim et al., 2013).

Life Cycle

Cryptosporidium are obligate intracellular parasites. It includes both asexual and sexual reproduction. The oocyst is the only stage that exists outside the host. Humans and animals secrete out sporulated oocyst in feces (Figure 5). Atomic force microscopy reveals that the oocyst wall is very stiff and resembles conventional plastic materials (Dumètre et al., 2013). In the intestine, where infections are primarily found in the jejunum and ileum, oocysts produce sporozoites after ingestion. After the sporozoite invades a cell, it develops intracellularly into a trophozoite stage, which then divides asexually to form two distinct meronts. Type I meronts release merozoites that penetrate other intestinal epithelial cells and either finish another cycle of type I meronts or transform into type II meronts. Type II meront merozoites subsequently undergo sexual reproduction to generate macrogamonts and microgamonts. The macrogamonts are fertilized by the microgamonts, resulting in zygotes that develop into oocysts (Hijjawi, 2010). The zygote produces either a thick-walled oocyst with two-layered membranes or a thin-walled oocyst covering only one layer of membrane after two mitotic divisions. The thin-walled oocysts can re-infect the same host's gastrointestinal system by rupturing and releasing infectious sporozoites, whereas the thick-walled oocysts are discharged through feces and live for months in the inappropriate environment (Gerace et al., 2019).

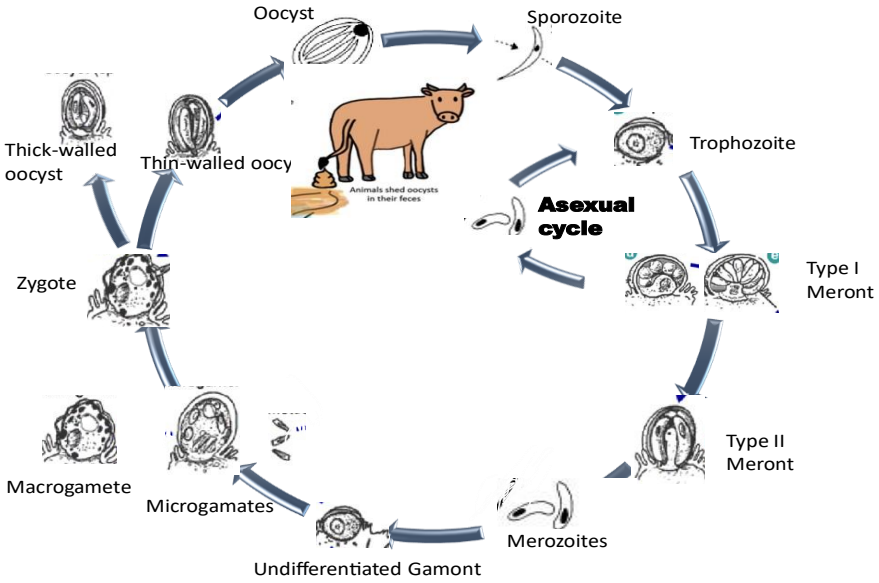

Fig. 5: Life cycle of cryptosporidiosis

Clinical signs

Common symptoms in animals include pasty to watery, yellow, and foul-smelling diarrhea, as well as anorexia and apathy/depression. Diarrhea might last from a few days to two weeks. There is a correlation between fecal consistency and oocyst excretion, and an extended

period of diarrhea may be linked to an early infection. Anorexia and intestinal damage probably lead to the affected body condition score and growth, which could reduce nutrition absorption for weeks (Robertson et al., 2014).

In humans most cases are asymptomatic. For others, the intensity and clinical course of infection might differ significantly from person to person, mostly based on the host's immunological state. The most notable symptom in immunologically healthy individuals is diarrhea, which is often watery and profuse. Blood and leukocytes are uncommon; however, mucus can occasionally be seen. Diarrhea may be accompanied by anorexia, nausea, vomiting, weight loss, fever, exhaustion, respiratory issues, and abdominal pain. Chronic infections can prove fatal when they cause frequent, large, watery stools that cause dehydration. Life cycle phases have been observed in cells from the pancreas, liver, gall bladder, respiratory tree, and other extraintestinal locations in severe conditions (Fayer, 2004).

Zoonotic Potential and Transmission

There are two different ways that this parasite might propagated: directly and indirectly. Accidental consumption via the fecal-oral route results in direct transfer. Transmissions can occur from one animal to another, from one human to another (anthroponotic or reverse zoonosis), or from one human to another. Human-to-human transmissions typically occur in hospitals, daycare centers, swimming pools, and water parks, as well as during anal contact with human waste. Furthermore, males who engage in sexual activity with other men through the fecal-oral pathway often increase this process. Additionally, direct transmission can happen when veterinarians or animal researchers are at high risk of coming into touch with infected animals (*Xiao* & Feng, 2008).

Contamination of food, drinking water, and various items such as clothing and shoes used in animal husbandry or wildlife parks can result in indirect exposure to the feces of infected people or animals. Infection and survival on the surface of the human intestinal epithelium, as well as various vertebrates, and can spread through feces. It can then contaminate ponds, rivers, sewage, drains or waterways, and even many bodies of water, especially ground and water sources, including untreated public water supplies. (Pumipuntu &Piratae, 2018).

Diagnosis

Diagnosis can be made by direct microscopy of stool sample smear. For specific diagnosis immunological-based technique used include direct fluorescent antibody, enzyme immunoassay, enzyme-linked immunosorbent assay (ELISA), indirect ELISA, and dipstick-like tests.

Treatment and Prevention

Most people and animals with compromised immune systems can recover on their own without medical treatment, as previously reported. Various supportive treatments such as fluid and electrolyte replacement, antiemetics, anti-nausea or analgesic drugs can control some clinical symptoms such as fever, vomiting, nausea, abdominal pain and dehydration (Pumipuntu & Piratae, 2018). These drugs can reduce the symptoms of cryptosporidiosis, and in some cases antiprotozoal therapy is necessary. The US Food and Drug Administration has approved nitazoxanide as the only anti-cryptosporidium drug approved for the treatment of cryptosporidiosis in humans, making it the most effective drug for treating people infected with Cryptosporidium species (Bamaiyi & Redhuan, 2016).

When it comes to human prevention, the best way to prevent the spread of Cryptosporidium spp. Practice good personal hygiene, including washing hands before eating or handling food, after using the toilet, and after contact with children, people with diarrhea, or animals or livestock. Raw food and water must be cleaned, washed, heated, cooked or boiled before consumption. In addition, people with symptoms of diarrhea should be aware that they should avoid swimming in public pools, water parks, or rivers to prevent the spread of the disease to others. (Rossle & Latif, 2013).

Thermal and chemical disinfectants such as hydrogen peroxide, chlorine dioxide, ozone, and UV light are effective in controlling Cryptosporidium oocysts in living spaces, livestock, and drinking water. Although some methods are less practical, chemical disinfection can help prevent cryptosporidiosis and reduce mortality and morbidity in humans and animals (Ghazy et al., 2016).

Conclusion

Zoonotic protozoal diseases pose a great challenge to animal and public health worldwide. About 60% are new human pathogens are zoonotic in nature which include various bacteria, viruses, protozoa, fungus and other parasites. Rudolph Virchow once stated, “There is no scientific barrier, nor should there be, between veterinary medicine and human medicine”. There are different advantages of keeping companion animals such as felines and canines such as social interactions and health benefits. However, there are serious health concerns for the owner. So, proper vaccination protocols and deworming schedules of pets should be followed to keep yourself safe from zoonotic pathogens. Improved hygiene and proper disposal of the excreta can decrease public health concerns. On routine basis, the application of insecticide spray can be used to restrict the entry of vectors towards animals. We can’t completely remove pathogens from the environment and by following the above preventive control measures, we can reduce their incidence in animals and also in public.

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