Order- Diplomonadida

Diplomonadida (Sub Phylum: Mastigophora, class Zoomastigophorea) is an order of protozoa which are bilaterally symmetrical and have 2–8 flagella. Most species are parasitic in vertebrates and some can cause disease. Most diplomonads are double cells: they have two nuclei, each with four associated flagella, arranged symmetrically about the body's main axis. Like the retortamonads, they lack both mitochondria and Golgi apparatuses. However, they are now known to possess modified mitochondria, in the case of *Giardia lamblia*, called mitosomes. These are not used in ATP synthesis the way mitochondria are, but are involved in the maturation of iron-sulfur proteins. Important genera are *Hexamita* and *Giardia*

Genus- *Hexamita*

- Members of this genus have pyriform body with 2 nuclei
- Six anterior and two posterior flagella and two axostyle
- The most important species is *Hexamita meleagridis* (*Spironucleus meleagridis*) occurs in turkey
- Other members are *H. columbae* live in the intestines of pegion. *H. muris* and *H. pitheci* live in the intestines of mammals. *H. salmonis* and *H. truttae* live in the intestines of fish

***Hexamita meleagridis* (*Spironucleus meleagridis*)** *H. meleagridis* (pigeons *H. columbae*) is a protozoan parasite of turkeys, pheasants, pigeons, and some game birds. It is transmitted by faeces, fomites, carriers. Inter-species transmission may occur. It occurs in duodenum and intestine of young turkey and cause infectious catarrhal enteritis.

Morphology- It is bilaterally symmetrical spindle-shaped, averages 8 × 3 μm; and has four anterior, two anterolateral, and two posterior flagella. There are two nuclei and each flagella arise from distinct blepheroplast.

Life cycle- Multiplication is by longitudinal binary fission. It is believed that schizogony and binucleate cyst formation do occur and there is formation of invasive stage which penetrated the reticuloendithelial cells. It is transmitted directly by ingestion of contaminated feces and water. Encysted hexamitids are resistant to environmental conditions outside the bird and, therefore, may be more important in the transmission of the disease. Up to ⅓ of the recovering birds become carriers and shed parasites in their droppings.

Pathogenesis- The principal pathological changes are found in the small intestine. Catarrhal inflammation with marked lack of tone is present in the duodenum, jejunum and ileum. The intestinal contents are usually thin, watery and foamy, with localized bulbous swellings filled with watery fluid. The small intestine, especially the anterior part, is inflamed and edematous. The cecal contents are usually fluid, and the cecal tonsils are congested.

Clinical Signs-

- Disease of young birds; adults are symptomless carriers
- Mortality upto 70 to 80%, but heavy losses seldom occur in poults over ten weeks old
- Poults appear nervous, stilted gait, ruffled, unkempt feathers, and a foamy, watery diarrhea
- Usually continue to eat, but chirp continually
- Lose weight rapidly, become listless, weak and finally die
- Thin and have lowered temperatures
- Birds which recover grow poorly

Diagnosis-Microscopic identification of the parasite in scrapings from intestinal mucosa
Diagnosis of hexamitiasis depends on finding the flagellates by microscopic examination of scrapings of the duodenal and jejunal mucosa. *Spironucleus* spp. move with a rapid, darting motion (in contrast to the jerky motion of trichomonads). To avoid contamination of instruments with other cecal protozoa, the duodenum should be opened first.

Treatment-Tetracycline, dimetridazole, and also, if possible, increase ambient temperature. Furazolidone, dimetridazole and ipronidazole have been used in the past. The effect of antibiotic may be related to the control of secondary bacterial enteritis.

Prevention and Control: Hexamitosis can be prevented by proper management and sanitation. Poults should be separated from adults, and separate caretakers should be used for the two groups. If feasible, breeding birds should be sold 2 weeks before any poults are hatched. Separate equipment should be used for different groups of birds Feeders and waterers should be placed on wire platforms. Young birds should be kept on wire. Ranges frequented by pheasants, quail and chukar partridges should be avoided. General sanitation and fly control should be practiced.

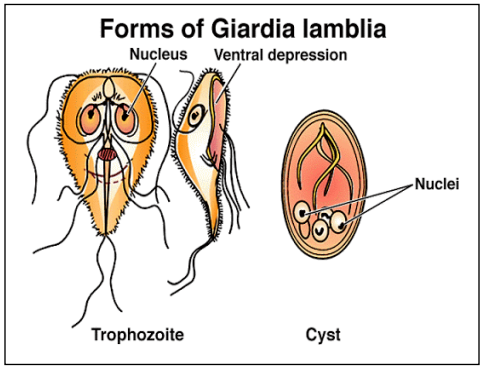
Genus- *Giardia*

Members of this genus have been reported in fecal samples from pet and shelter dogs and cats, in small ruminants, 9%–73% in cattle, 1%–38% in pigs, and 0.5%–20% in horses, with higher rates of infection in younger animals all are morphologically similar. The important member affecting wide range animals is *Giardia lamblia* affecting mammals (including man), birds, reptiles *G. canis* affecting dogs *G. cati* affecting cats. *G muris* and *G. ardae* from rodents (mice), *G agilis* and *G. gracilis* from amphibians

Morphology-

Two developmental stages: trophozoites and cysts.

Trophozoite-Bilaterally symmetrical, pear shaped, rounded anterior end, posterior end pointed (looks like monkey face)
12 to 15 µm long by 5to 9 µm wide
dorsal surface convex, ventral surface concave and bears sucking disk to adhere to surface of intestinal cell
2 nuclei, 2 axostyles, 4 pairs of flagella (2 anterior, 2 posterior, 2 ventral, and 2 caudal) actively moving and feeding stage

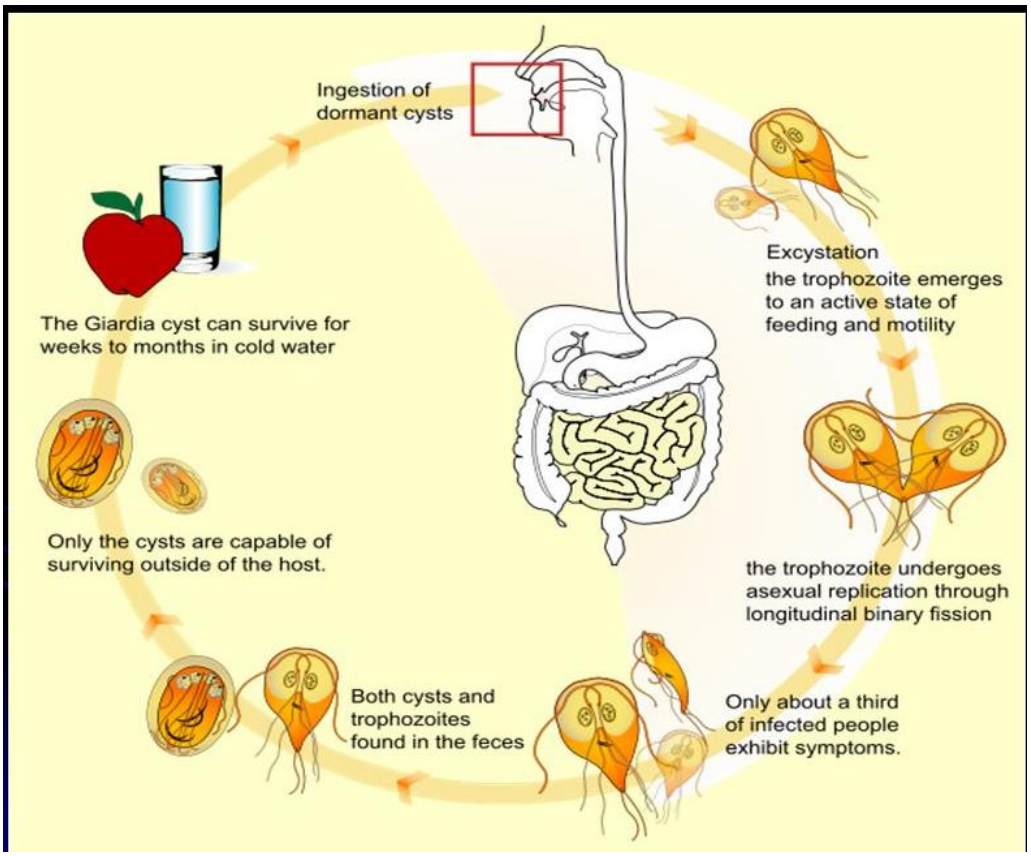


Cyst-Cysts are ovoid to ellipsoid (11-14µm by 7-10µm), membrane-bound (sometimes imparting a halo-appearance) and contain 2 latter 4 nuclei, axonemes and median bodies.

Transmission-Transmission occurs by the fecal-oral route (by the ingestion of cyst), either by direct contact with an infected host or through a contaminated environment.

Site of infection: Flagellated trophozoites are found in the small intestines of their hosts, especially the duodenum. Trophozoites have been observed swimming with a distinct corkscrew motion in luminal content as well as adhering to the gut mucosal surface with their ventral adhesive discs (when they detach, they leave distinct oval impressions in the microvillous layer).

Life cycle



Digramatic representation of life cycle of *G. lamblia*in human being

- Acquire infection –ingestion of mature cysts through contaminated food and water
- Excystation occurs in stomach & duodenum to for trophozoites
- Trophozoites inhabit the mucosal surfaces of the small intestine and attach to the brush border
- They multiply by binary fission
- Trophozoites encyst in the small or large intestine, and the newly formed cysts pass in the faeces
- prepatent period is generally 3–10 days
- Cyst shedding may be continual over several days and weeks but is often intermittent
- The cyst is the infective stage and can survive for several weeks in the environment

Pathogenesis- Trophozoites inhabit the mucosal surfaces of the small intestine, where they attach to the brush border, absorb nutrients, and multiply by binary fission. It causes an increase in epithelial permeability, increased numbers of intraepithelial lymphocytes, and activation of T lymphocytes. Trophozoite toxins and T-cell activation initiate a diffuse shortening of brush border microvilli and decreased activity of the small-intestinal brush border enzymes, especially lipase, some proteases, and dissacharidases. Shortening of microvillus leads to a decrease in overall absorptive area in the small intestine and an impaired intake of water, electrolytes, and nutrients resulting in malabsorptive diarrhoea and lower weight gain. The reduced activity of lipase and the increased production of mucin by goblet cells cause steatorrhea and mucous diarrhea.

There are no intracellular stages. The prepatent period is generally 3–10 days. Cyst shedding may be continual over several days and weeks but is often intermittent, especially in the chronic phase of infection. The cyst is the infective stage and can survive for several weeks in the environment, whereas trophozoites cannot.

Clinical Sign-

- Most clinical infections are self-limiting and resolve spontaneously
- Acute case- Young individuals are most susceptible to clinical infections and in acute cases watery diarrhoea, abdominal cramps, bloating, flatulence
- Stool is profuse & watery in earlier disease and voluminous, foul smelling& greasy (steatorrhoea) later
- Chronic case- chronic diarrhoea with malabsorption syndrome, steatorrhoea

Dogs and Cats- Infection is inapparent or may produce weight loss and chronic diarrhea or steatorrhea, which can be continual or intermittent, particularly in puppies and kittens. Soft, poorly formed, pale, malodorous faeces, contain mucus, and appear fatty, occasional vomition.

Ruminants-In calves, and to a lesser extent in other production animals, giardiasis can result in diarrhoeathere is excretion of pasty to fluid faeces with a mucoid appearance.Infection of goat kids, lambs, and calves resulted in a decreased feed efficiency and subsequently a decreased weight gain.

Diagnosis

Microscopic examination

Trophozoite with falling leaf motility can be seen in saline

Mount through microscope (Plate 1)

Cyst can be detected in diarrheic faeces by iodine mount (Plate 2)

Plate 1 Plate 2

Antigen detection (Copro antigen) by ELISA

Molecular diagnosis by DNA probes & PCR

Treatment and control

Flagyl (metronidazole 25 mg/kg, bid for 5 days) is the drug of choice for giardiasis, other nitroimidazole derivatives (tinidazole), nitrofurans (furazolidone), acridine drugs (quinacrine) are also being used. Fenbendazole (50 mg/kg/day for 5–10 days) can be used in dogs. A combination of praziquantel (5.4–7 mg/kg), pyrantel (26.8–35.2 mg/kg), and febantel (26.8–35.2 mg/kg) also effectively decreases cyst excretion in infected dogs when administered for 3 days. Paromomycin (50–75 mg/kg, PO, for 5 days) was found to be highly efficacious in calves.

Control depends largely on good sanitation, proper effluent disposal and effective water treatment (well-maintained sand filtration or microfiltration, optimum chlorination or ozonation). Cysts are inactivated by most quaternary ammonium compounds, steam, and boiling water.Proper dispose of the contaminated faeces.