Classification of Medical Parasitology

Parasites of medical importance come under the kingdom called Protista and Animalia. **Protista** includes the microscopic single-celled eukaryote known as protozoa. In contrast, helminthes are macroscopic, multicellular worms possessing well differentiated tissues and complex organs belonging to the kingdom **Animalia.** Medical Parasitology is generally classified into:

• Medical Protozoology • Medical Helminthology • Medical Entomology

PROTOZOA

Protozoa of medical importance are classified based on their morphology and locomotive system as described below:
1-Sarcodina - *Entamoeba histolytica*
2-Flagellates - *Giarda lamblia, Trichomonas vaginalis, Trypanosoma spp, Leishmania spp*
3-**Cliliophora** - *Balantidium coli*
4-**Coccidian** - *Cryptosporidium parvum, Toxoplasma gondii, Plasmodium* species

Protozoan pathogens can also be grouped according to the location in the body where they most frequently cause disease.

1- Sarcodina (Amoebe):

Entamoeba histolytica

The protozoan parasite *Entamoeba histolytica* is a causative agent of amoebiasis or amoebic dysentery. This parasite is endemic in most tropical and subtropical areas of the world, where it causes millions of cases of dysentery and liver abscess each year.

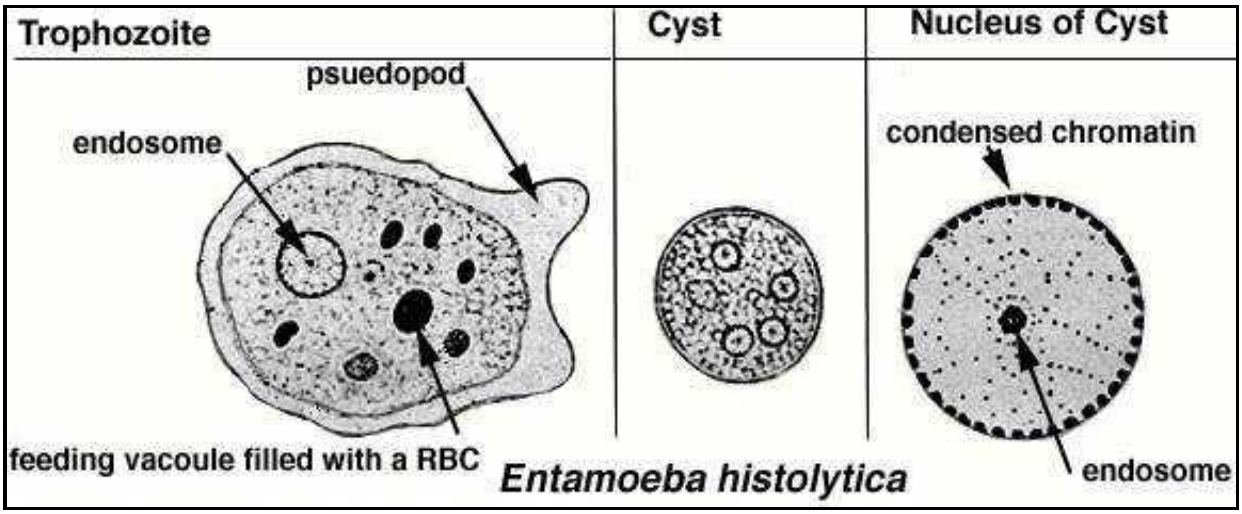
Epidemiology: Humans are the reservoirs for *E. histolytica* (rarely also: monkeys, dogs, cats). The infection is due to transmission of mature cysts with contaminated foods (fruit, vegetables), drinking water or fecally contaminated hands. Flies and cockroaches can function as intermediaries by carrying cysts from the feces. Outbreak in 1933 World's Fair in Chicago caused by defective plumbing (cross connections between water lines and sewer lines) caused over 1000 cases of amoebiasis resulting in 58 deaths.

Morphological features

a- Trophozoite stage: Trophozoite is the vegetative or growing stage of the parasite. It is the only form present in tissues. It is irregular in shape and size (10– 60 μm) that usually form a single, broad pseudopod that is often quickly extended in the direction of movement. Stained preparations of the genus Entamoeba show a characteristic ring-shaped nucleus with a central nucleolus and chromatin granule on the nuclear membrane. The cytoplasm is usually described as granular with few ingested leukocyte and red blood cell in vacuole. Initially it's being small and called trophozoite minuta form (it occurs free in the lumen as a commensal is generally smaller in size, about 15–20 μm) and when it grow and cause damage called trophozoite magna.

b- Precystic Stage: Trophozoites undergo encystment in the intestinal lumen. Encystment does not occur in the tissues nor in feces outside the body. Before encystment, the trophozoite extrudes its food vacuoles and becomes round or oval, about 10–20 μm in size. This is the precystic stage of the parasite. It contains a large glycogen vacuole and two chromatid bars. It then secretes cyst wall around it and becomes cyst.

c- Cyst stage: Cysts are spherical, nonmotile (10–16μm) have a resistant cyst wall. The early cyst contains a single nucleus and two other structures, a mass of glycogen and 1–4 chromatoid bodies or chromidial bars, which are cigar-shaped with rounded ends. As the cyst matures, the glycogen mass and chromidial bars disappear. The nucleus divides once to produce the binuclear form and later once again to produce the mature cyst infective tetra nuclear cyst (quadrinucleate).



Life cycle

1-Intestinal infections occur through the ingestion of a mature quadric nucleate infective cyst, contaminated food or drink and also by hand to mouth contact.

2-It is then passed unaltered through the stomach, as the cyst wall is resistant to gastric juice. In terminal ileum (with alkaline pH), encystation takes place.

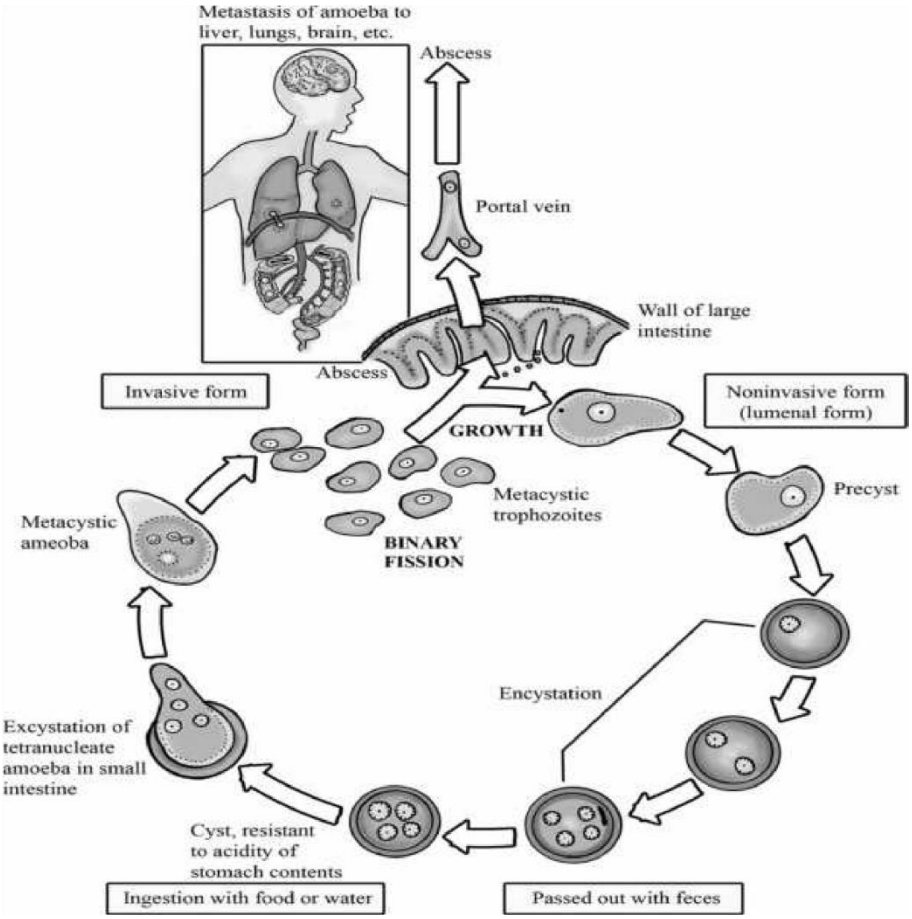
3-Trophozoites being actively motile invade the tissues and ultimately lodge in the submucous layer of the large bowel. Here they grow and multiply by binary fission.

4-Trophozoites are responsible for producing lesions in amoebiasis.

Invasion of blood vessels leads to secondary extra intestinal lesions most particularly to the liver, also invasion of the spleen, brain, and other organs. Extraintestinal lesions (metastatic) include: liver (amoebic hepatitis and amoebic liver abscess), lungs (primary small abscess or multiple abscess in one or both lungs), brain (a small cerebral abscess), spleen (splenic abscess) and skin granulomatous lesion (amoeboma) near visceral lesion, e.g. liver. Gradually the effect of the parasite on the host is toned down together with concomitant increase in host tolerance, making it difficult for the parasite to continue its life cycle in the trophozoite phase.

5-A certain number of trophozoites come from tissues into lumen of bowel and are first transformed into pre-cyst forms.

6-Pre-cysts secret a cyst wall and become a uninucleate cyst. Eventually, mature quadric nucleate cysts form. These are the infective forms. Both mature and immature cysts may be passed in the stool of infected persons, either alone or together with trophozoites. Immature cysts can mature in external environments and become infective.



Clinical features

Asymptomatic intestinal form. *E. histolytica* can colonize the intestinal mucosa, reproduce, and persist for long periods without becoming invasive or causing any changes.

The outcome of infection may result in a carrier state, intestinal amebiasis, or exteraintestinal amebiasis. Diarrhoea, flatulence, and cramping are complaints of symptomatic patients. More severe disease is characterised by the passing of numerous bloody stools in a day.

Systemic signs of infection (fever, leukocytosis, rigors) are present in patients with extraintestinal amebiasis.

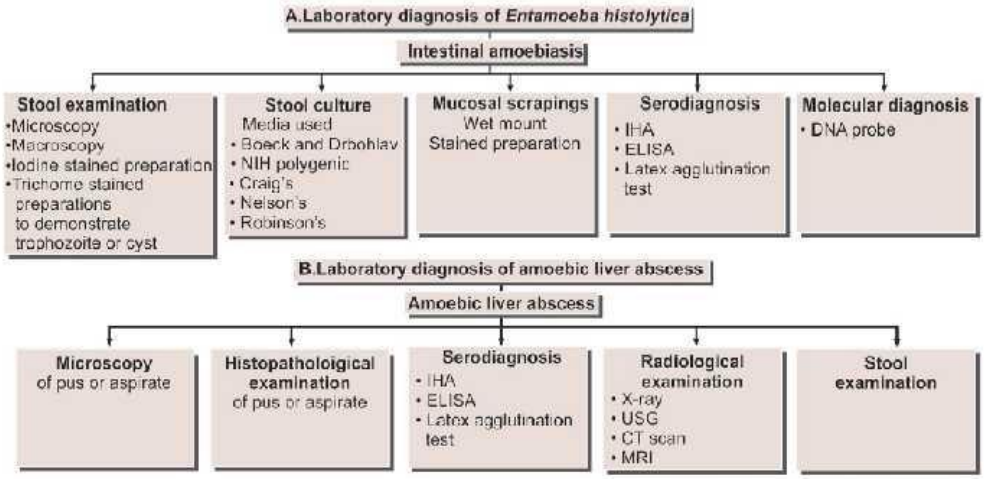
The liver is primarily involved, because trophozoites in the blood are removed from the blood by the portal veins. The right lobe is most commonly involved, thus pain over the liver with hepatomegaly and elevation of the diaphragm is observed.

Clinical symptoms can develop as early as two to four weeks after infection with *E. histolytica* or after asymptomatic periods of months or even years.

Pathogenesis

Trophozoites divide and produce extensive local necrosis in the large intestine. Invasion into the deeper mucosa with extension into the peritoneal cavity may occur. This can lead to secondary involvement of other organs, primarily the liver but also the lungs, brain, and heart. Extraintestinal amebiasis is associated with trophozoites. Amoebas multiply rapidly in an anaerobic environment, because the trophozites are killed by a mbient oxygen concentration.

Diagnosis



Flowchart 3.3: A. Laboratory diagnosis of *Entamoeba histolytica*; **B.** Laboratory diagnosis of amoebic liver abscess

Treatment: Metronidazole and Nitromidazole is effective against symptomatic intestinal and extraintestinal forms of amoebiasis.

Prevention

1- Introduction of adequate sanitation measures and education about the routes of transmission.

2- Avoid eating raw vegetables grown by sewage irrigation.

Other amoeba inhabiting the alimentary canal

Most of these amoebae are commensal organisms that can parasitize the human gastrointestinal tract. Considerable experience is required to distinguish *E. histolytica* from other forms, it is necessary to do because misdiagnosis often leads to unnecessary treatment, over treatment, or a failure to treat.

Entamoeba coli:

The life cycle stages include; trophozoite, precyst, cyst, metacyst, and metacystic trophozoite. Typically the movements of trophozoites are sluggish, with broad short pseudopodia and little locomotion, but at a focus the living specimen cannot be distinguished from the active trophotozoite of *E.histolytica*. However, the cysts are remarkably variable in size. *Entamoeba coli* is transmitted in its viable cystic stage through faecal contamination. *E.coli* as a lumen parasite is non-pathogenic and produces no symptoms. The mature cyst (with more than four nuclei) is the distinctive stage to differentiate *E.coli* from the pathogenic *E.histolytica*. Specific treatment is not indicated since this amoeba is non-pathogenic. The presence of *E.coli* in stool specimen is evidence for fecal contamination. Prevention depends on better personal hygiene and sanitary disposal of human excreta.

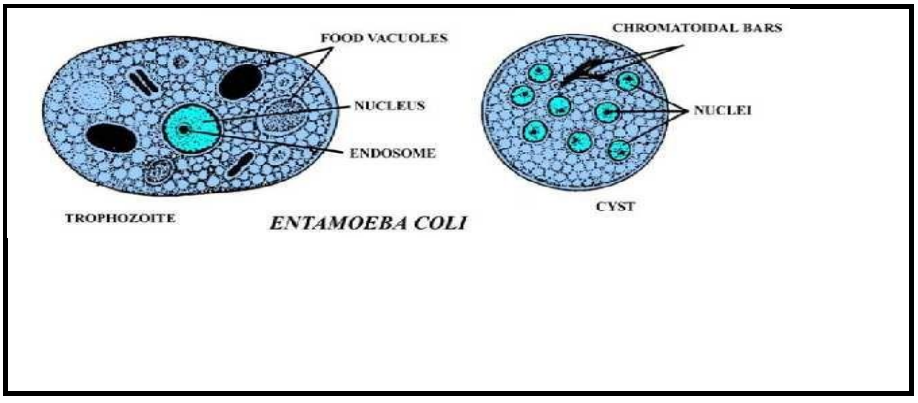
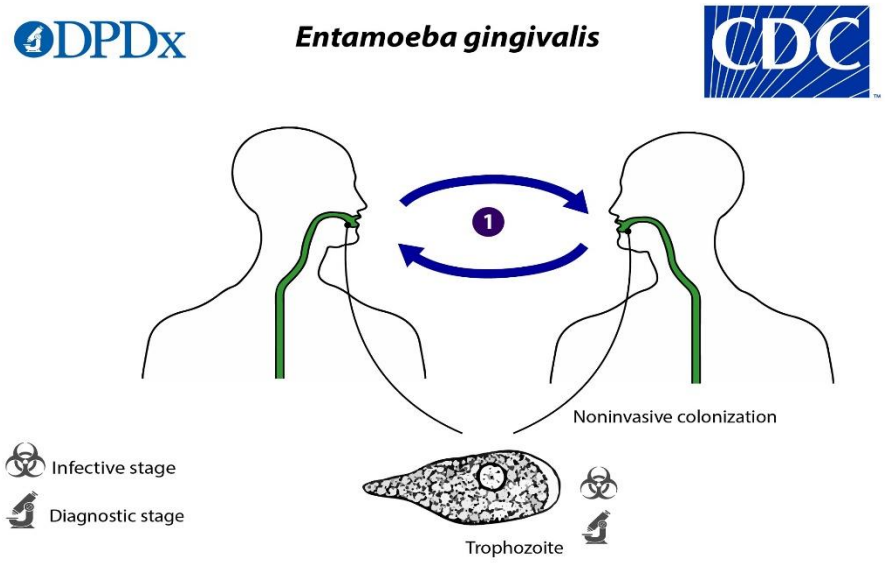


TABLE 57.4: Differences between *Entamoeba histolytica* and *Entamoeba coli*

Entamoeba histolytica	*Entamoeba coli*
Cyst	
i. 5 to 20 µ	10 to 40 µ
ii. Nuclei 4 or less	Up to 8
iii. Chromotoidal body cigar like	Thread like
iv. Karyosome smaller and central	Large and eccentric
Trophozoite	
i. 10 to 60 µ in size	10 to 50 µ
ii. Single pseudopodium	Multiple
iii. Cytoplasm finely granular	Coarsely granular
iv. Cytoplasm encloses RBC	Cytoplasm encloses bacteria debris as inclusion bodies
v. Actively motile	Sluggishly motile
vi. Nucleus invisible	Visible

Entamoeba gingivalis

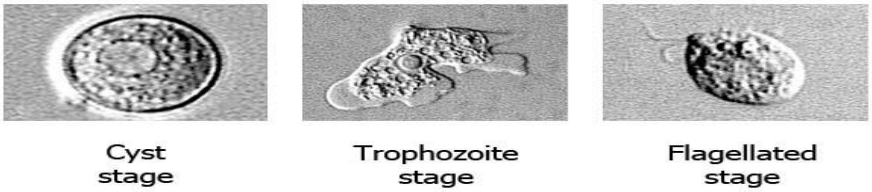
Only the trophozoite stage presents, and encystation probably does not occur. - *E.gingivalis* is a commensal, living primarily on exudate from the margins of the gums, and thrives best on unhealthy gums. No specific treatment is indicated. However the presence of *E. gingivalis* suggests a need for better oral hygiene. The infection can be prevented by proper care of the teeth and gums.



Free -Living Amoeba

Naegleria fowleri is the ubiquitous of Free-Living Amoebae. Infection by the pathogenic Naegleria fowleri acquired by exposure of the polluted water in the lake, ponds, swimming pool or the other water recreation especially in summer months. This infection leads to Primary Amoebic Meningoencephalitis (PAM). PAM is an acute, frequently fatal, difficult to diagnose and does not have no effective therapeutic options, and most the patient over 95% died.

Naegleria fowleri has three stages in its life cycle: cysts, trophozoites and flagellated forms. The trophozoites replicate by promitosis (nuclear membrane remains intact). Trophozoites can turn into temporary non-feeding flagellated forms which usually revert back to the trophozoite stage. Trophozoites infect humans or animals by penetrating the nasal mucosa, usually during swimming or sinus irrigation , and migrating to the brain (6) via the olfactory nerves causing PAM. *Naegleria fowleri* trophozoites are found in cerebrospinal fluid (CSF) and tissue, while flagellated forms are occasionally found in CSF. Cysts are not seen in brain tissue.



Cyst stage | Trophozoite stage | Flagellated stage