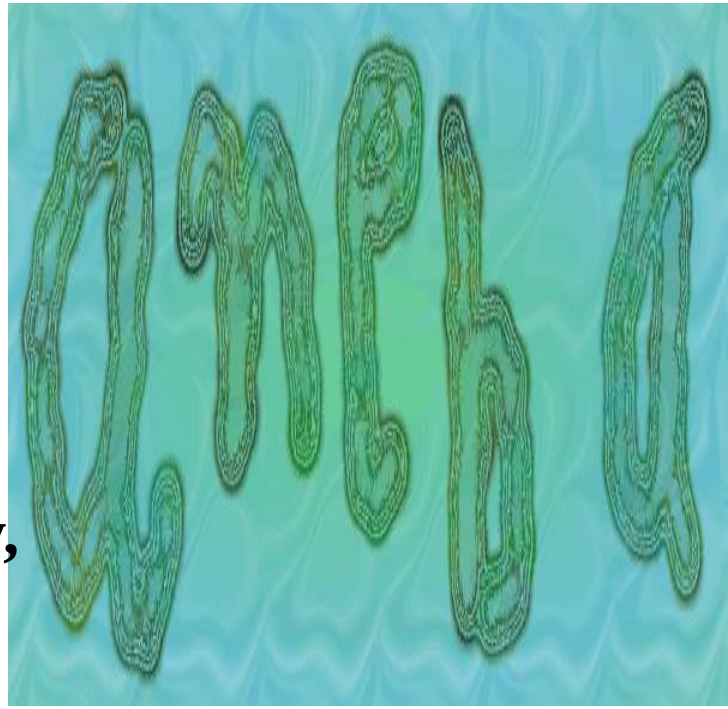
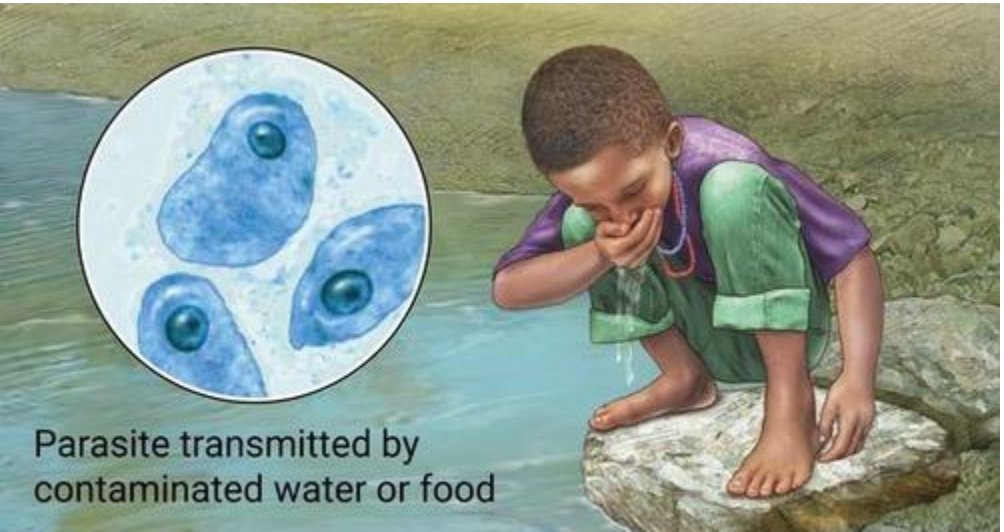
Amebiasis

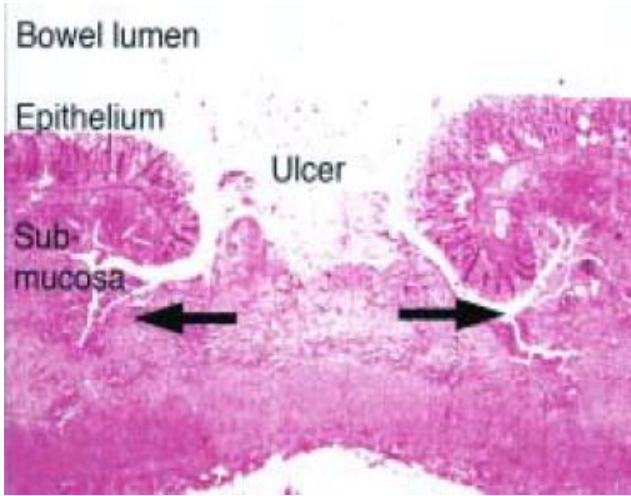


Amoebiasis, also known amoebic dysentery, is an infection caused by any of the amoebae of the *Entamoeba* group.

There are at least eight amebas (E. histolytica, E. dispar, E. moshkovskii, E. coli, E. hartmanni, E. polecki, Iodamoeba bütschlii, and Endolimax nana) which live in the human intestinal lumen. However, these are generally accepted as commensal organisms except for E. histolytica

Amoebiasis can be present with no, mild, or severe symptoms. Symptoms may include abdominal pain, diarrhea, or bloody diarrhea.Complications can include inflammation and ulceration of the colon with tissue death or perforation, which may result in peritonitis. People affected may develop anemia due to loss of blood.
Cysts of *Entamoeba* can survive for up to a month in soil or for up to 45 minutes under fingernails

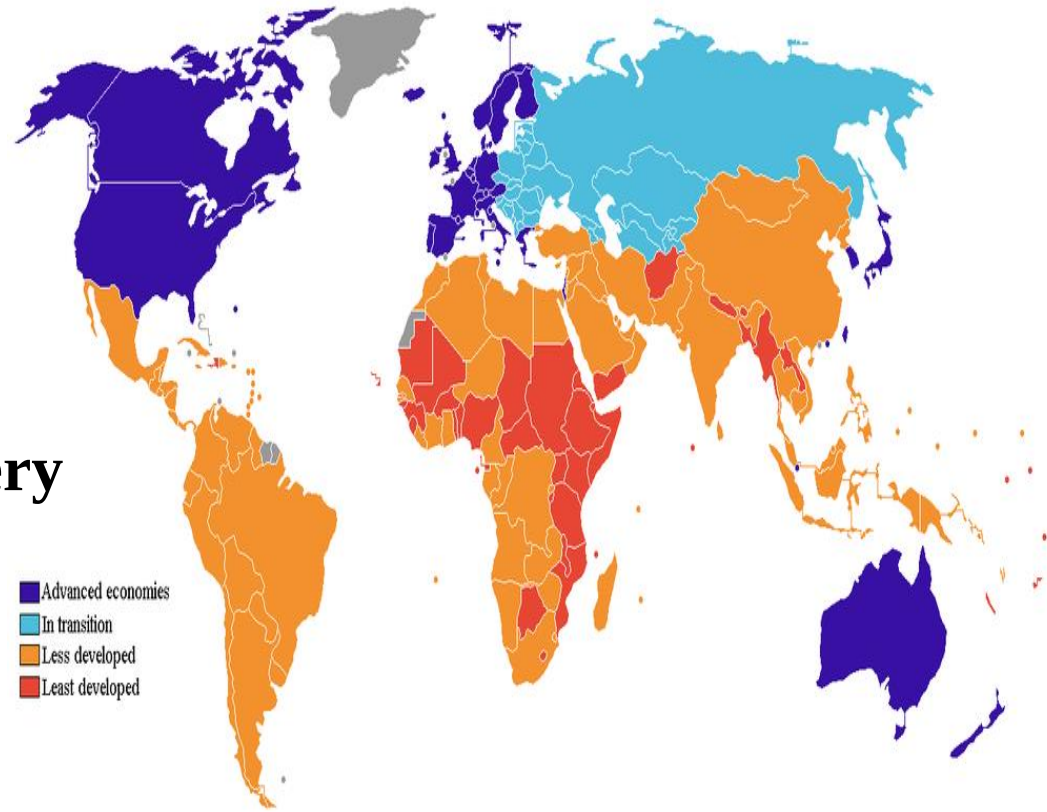




Amoebiasis is present all over the world, though most cases occur in the developing world. About 480 million people are currently infected with about 40 million new cases per year with significant symptoms.This results in the death of between 40,000–110,000 people a year. Most infections are now believed due to E. dispar.

E. dispar is more common in certain areas and symptomatic cases may be less common than previously reported.

The first case of amoebiasis was documented in 1875 and in 1891 the disease was described in detail, resulting in the terms amoebic dysentery and amoebic liver abscess.



Entamoeba histolytica

is one of several Entamoeba species that infect human.

Predominantly infecting humans and other primates causing amoebiasis, *E. histolytica* is estimated to infect about *35-50* million people worldwide.

***E. histolytica* infection is estimated to kill more than *55,000* people each year. Previously, it was thought that 10% of the world population was infected, but these figures predate the recognition that at least 90% of these infections were due to a second species, *E. dispar*.**

Other species are non-pathogenic and include

E.dispar,
E.hartmani, and
E.coli.

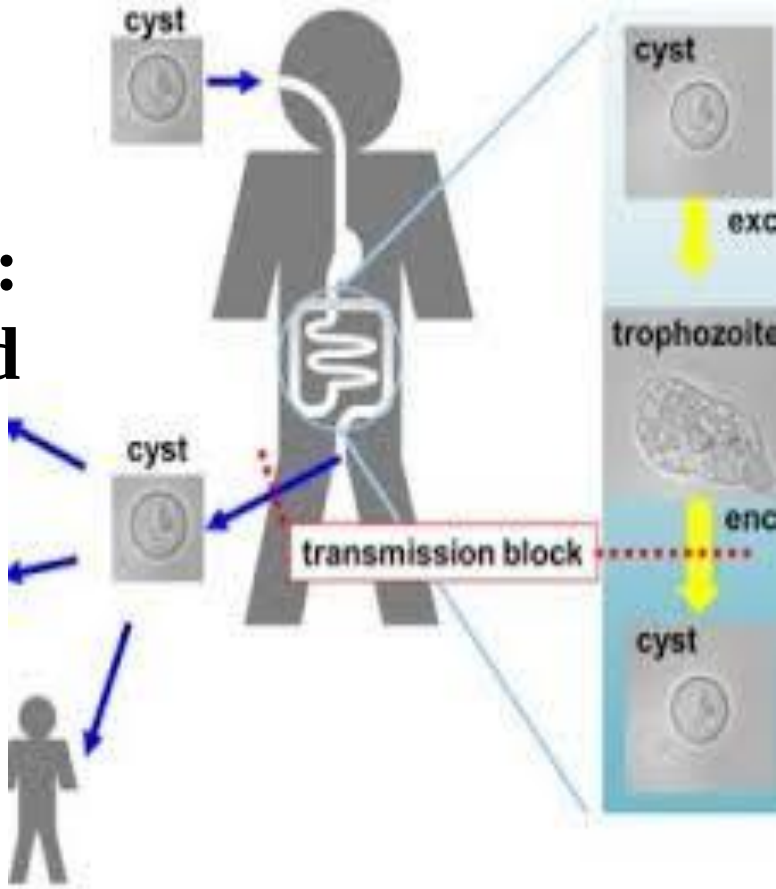
Disease is seen at all ages and both sexes are equally affected.

Etiology and epidemiology

E.histolytica **is**
an anaerobic parasitic amoebozoan,
part of the genus *Entamoeba*.

The organism exists in two forms:

- **the trophozoite (10- 60 µm) and**
- **the cyst (5-20 µm).**

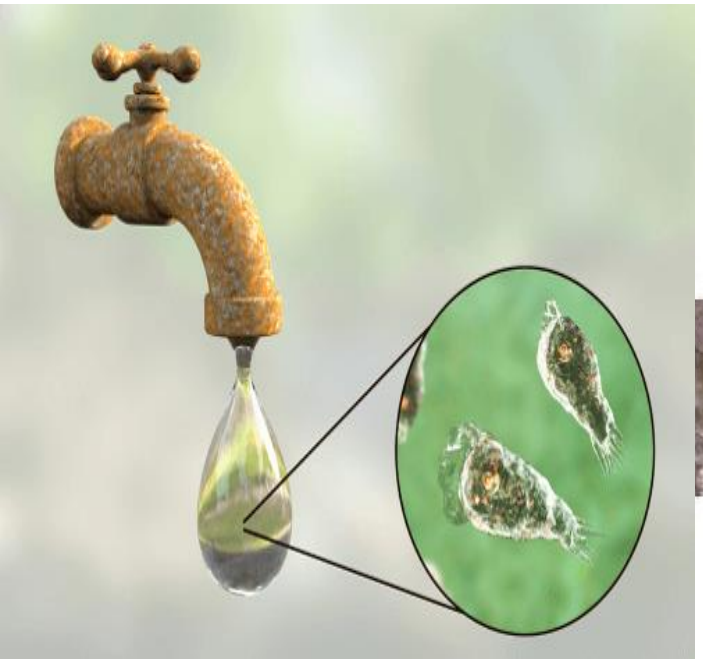


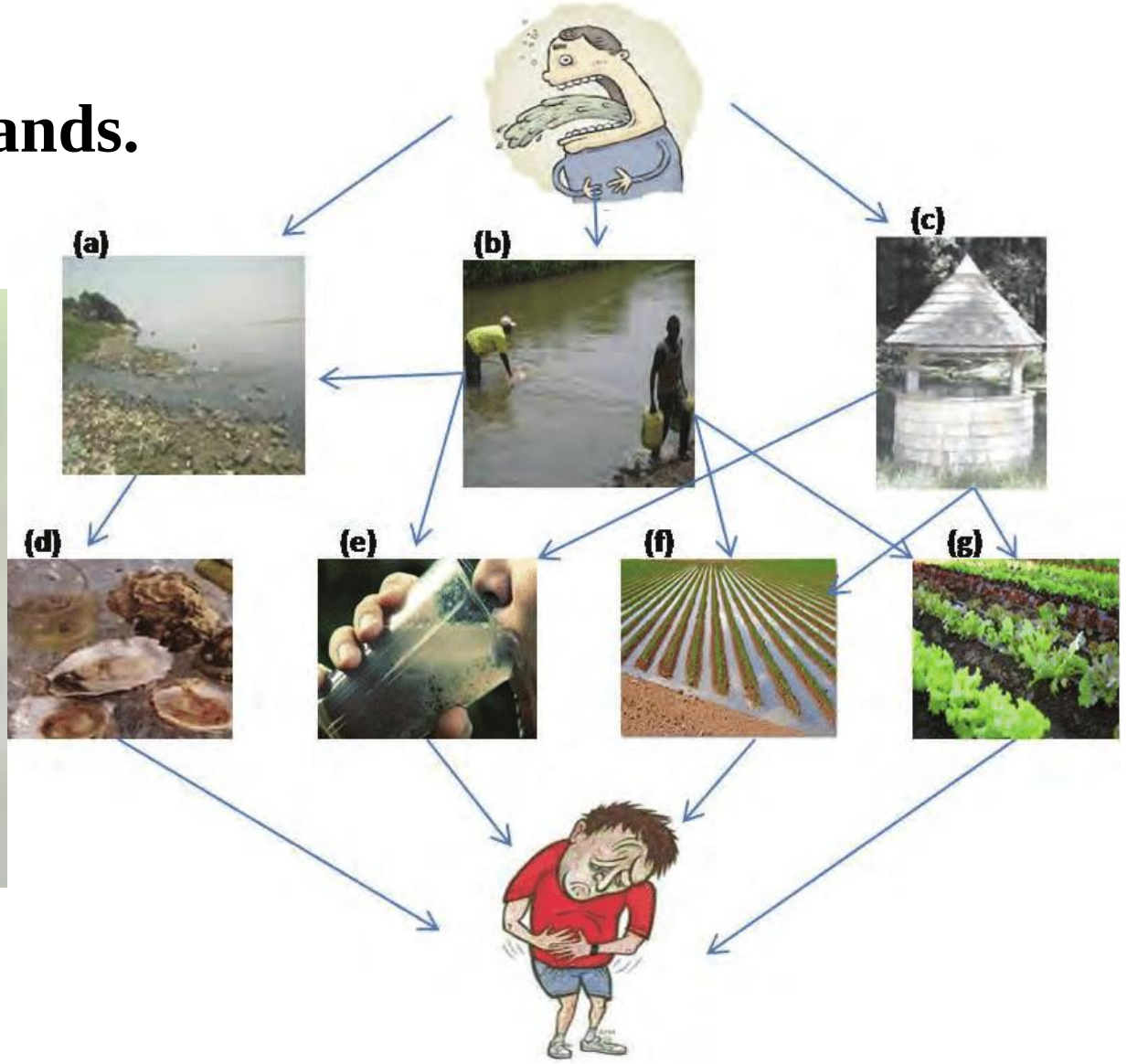
The motile trophozoites feed on bacteria and tissue, reproduce, colonize the lumen and the mucosa of the large intestine, and sometimes invade tissues and organs. Trophozoites predominate in liquid stools but rapidly die outside the body and, if ingested, would be killed by gastric acids. Some trophozoites in the colonic lumen become cysts that are excreted with stool.

***E. histolytica* trophozoites can adhere to and kill colonic epithelial cells and polymorphonuclear leukocytes (PMNs) and can cause dysentery with blood and mucus but with few PMNs in stool. Trophozoites also secrete proteases that degrade the extracellular matrix and permit invasion into the intestinal wall and beyond. Trophozoites can spread via the portal circulation and cause necrotic liver abscesses. Infection may spread by direct extension from the liver to the right pleural space, lung, or skin, or rarely through the bloodstream to the brain and other organs.**

Cysts predominate in formed stools and resist destruction in the external environment. They may spread directly from person to person or indirectly via food or water.

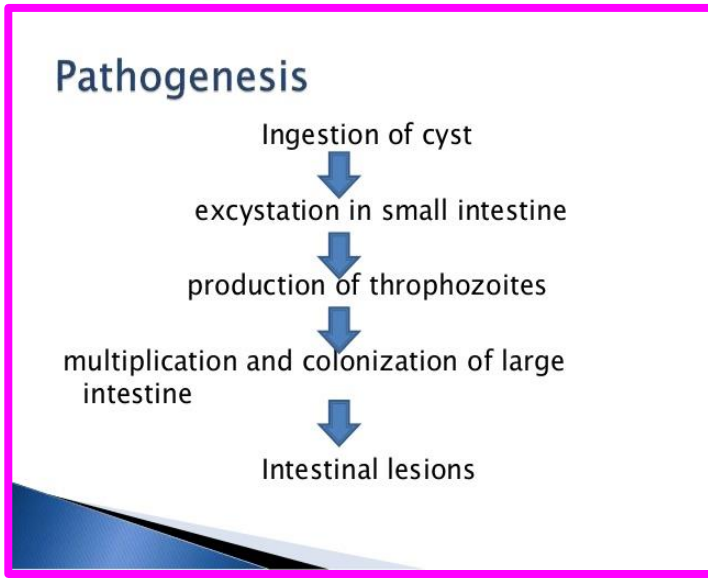
E. histolytica is acquired by ingestion of viable cysts from
fecally contaminated water,
food,
or hands.

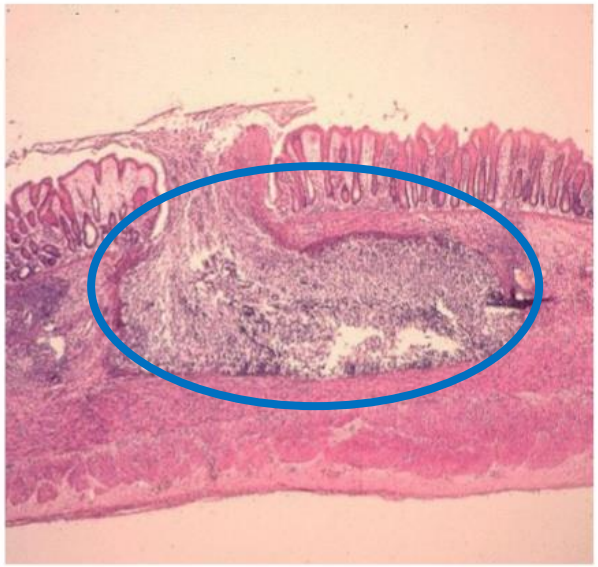




Pathogenesis and life cycle.

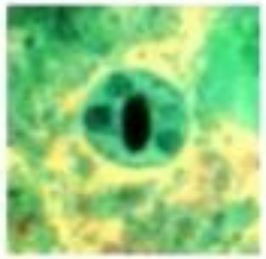
Ingestion of the cysts results in excystation in the small bowel. Trophozoites are formed which infect the colon and results in symptoms. During unfavorable conditions, the trophozoite encysts and cyst form is passed out in feces. Most people infected with E. histolytica have no significant invasion of the colonic mucosa and are asymptomatic (cyst passers). Patients with colonic invasion have *flask-shaped colonic ulcers*. The pathogenesis of invasive amoebiasis requires adherence of trophozoites, direct cytolytic and proteolytic effects that damage tissue, and resistance of the parasite to host immune response.



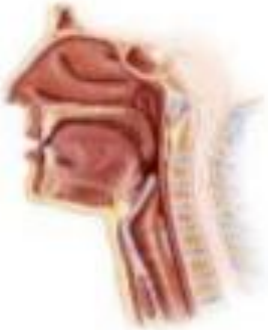


Flask shaped ulcer

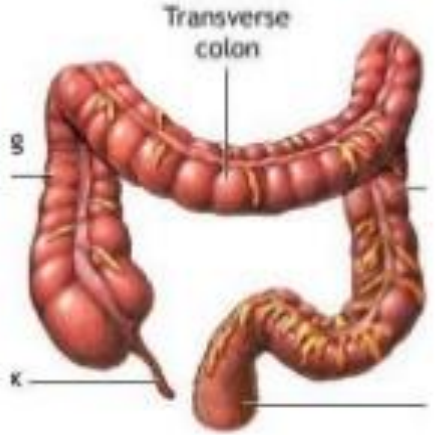
Life cycle



Cyst: infective stage



Inters mouth through contaminated food, drink, fly, or through using human stool as fertilizer

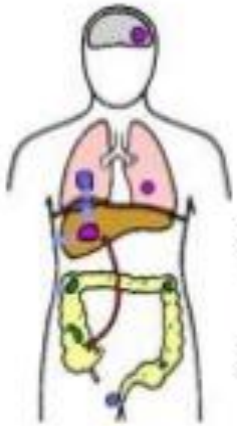


To L.I. lumen and change into trophozoite (pathogenic stage)

Produce lytic enzymes (capable of doing lysis and produce ulcer)

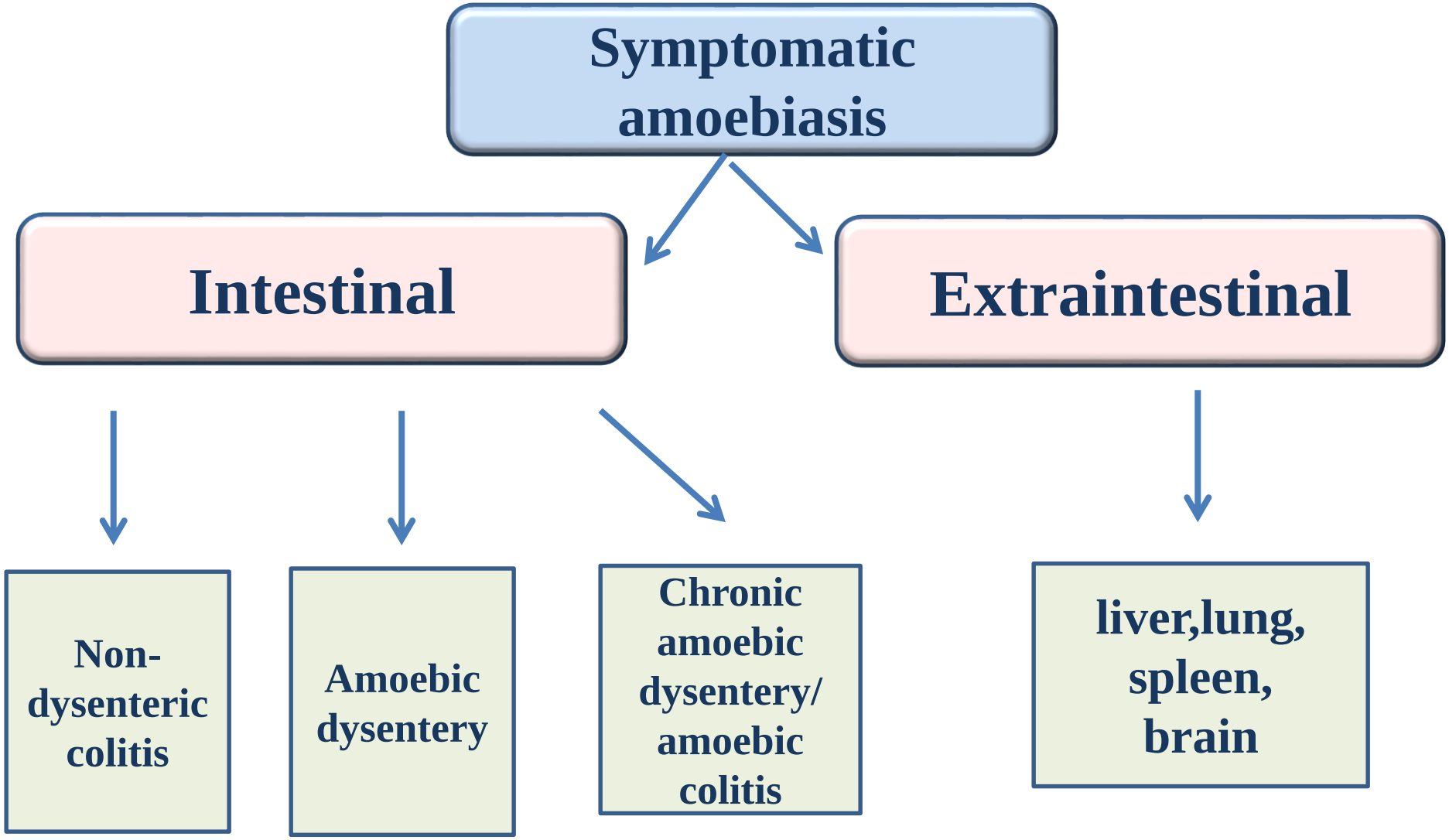
Flask shape ulcer

Can do erosion through B.V. to liver and other organs



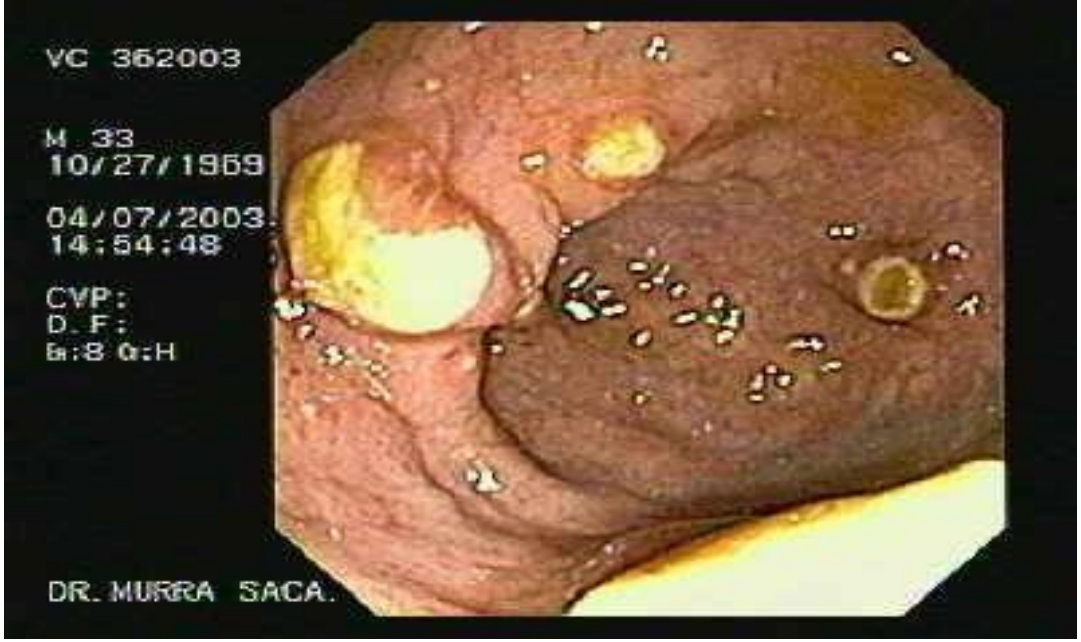
Clinical features.

The clinical features of amoebiasis can be divided into intestinal and extraintestinal forms.

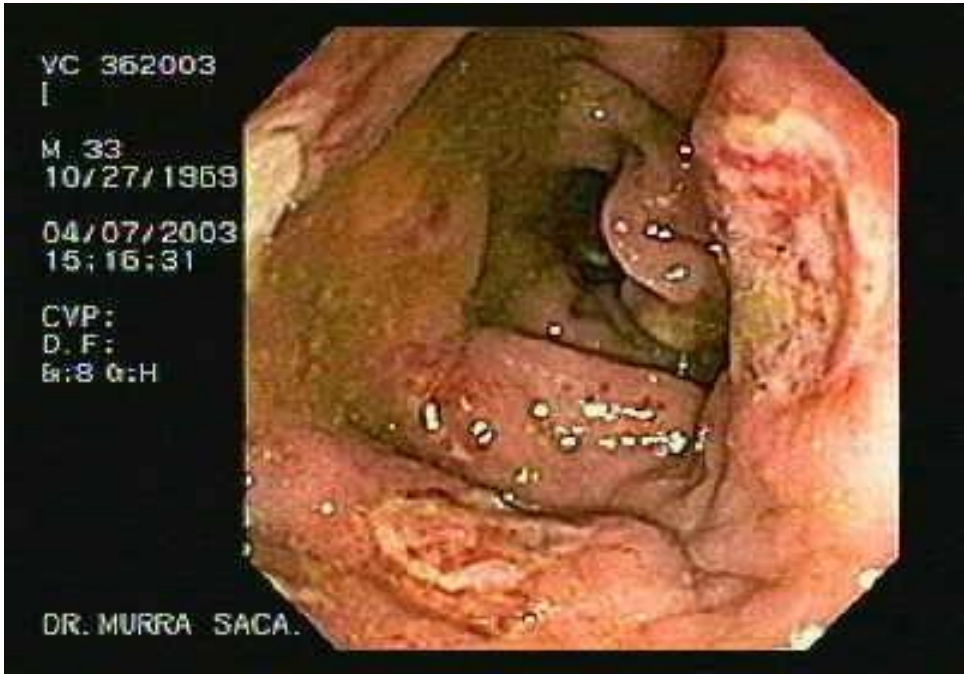


Clinical features.

Intestinal amoebiasis **includes asymptomatic infection and symptomatic intestinal infection.**
Symptomatic amebic colitis develops 2–6 weeks after the ingestion of infectious cysts. A gradual onset of lower abdominal pain and mild diarrhea is followed by malaise, weight loss, and diffuse lower abdominal or back pain. Patients with full-blown dysentery may pass 10–12 stools per day. The stools contain little fecal material and consist mainly of blood and mucus. In contrast to those with bacterial diarrhea, fewer than 40% of patients with amebic dysentery are febrile. More fulminant intestinal infection, with severe abdominal pain, high fever, and profuse diarrhea, is rare and occurs predominantly in children. These patients may develop toxic megacolon. Uncommonly, patients develop a chronic form of amebic colitis, or amoeboma (annular lesion of the colon)



Amebic Colitis



Extraintestinal amoebiasis

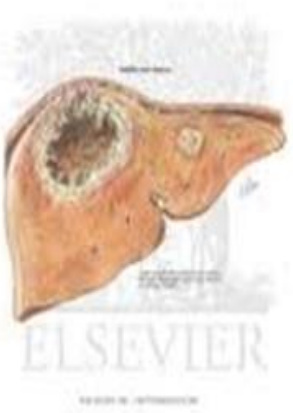
includes hematogenous amoebic abscesses (liver, lungs, brain, etc.), and/or genitourinary disease.

Extraintestinal infection by E. histolytica most often involves the liver.

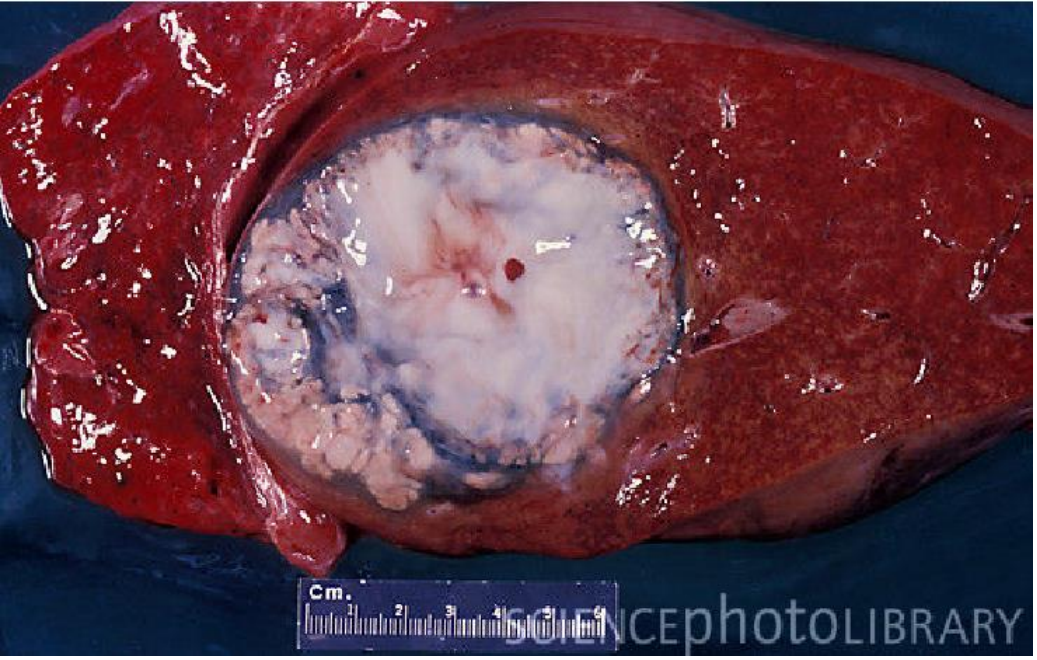
Most patients are febrile and have right-upper-quadrant pain, which may be dull or pleuritic in nature and may radiate to the shoulder.

Point tenderness over the liver and right-sided pleural effusion are common. Jaundice is rare. The genitourinary tract may become involved by direct extension of amebiasis from the colon or by hematogenous spread of the infection. Painful genital ulcers, characterized by a punched-out appearance and profuse discharge, develop. Symptoms and prognosis of extraintestinal abscesses depend on the size and location of the lesion

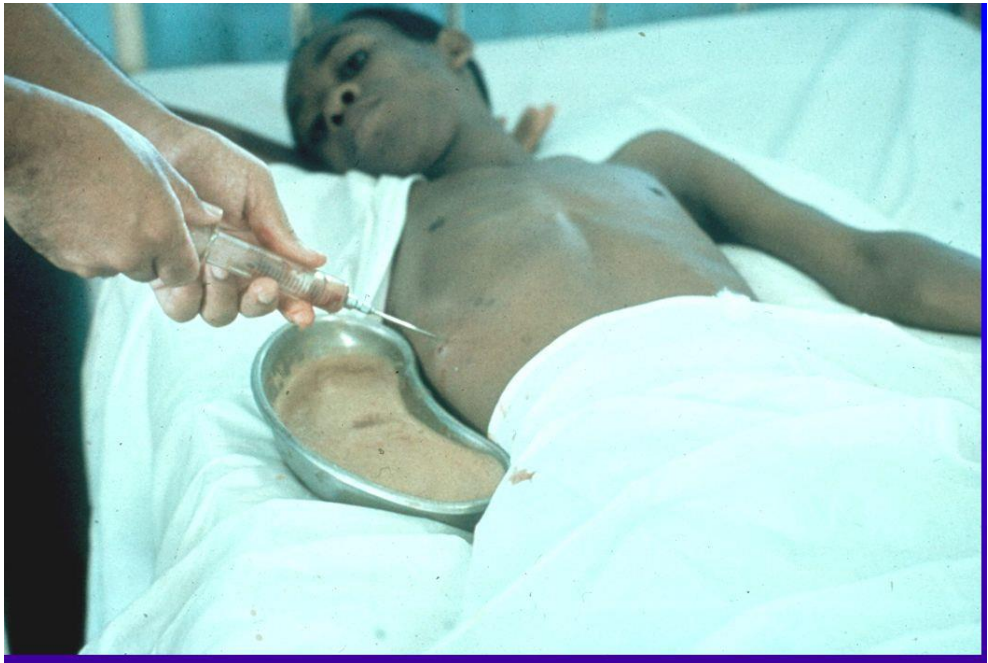
Amoebic liver abscess



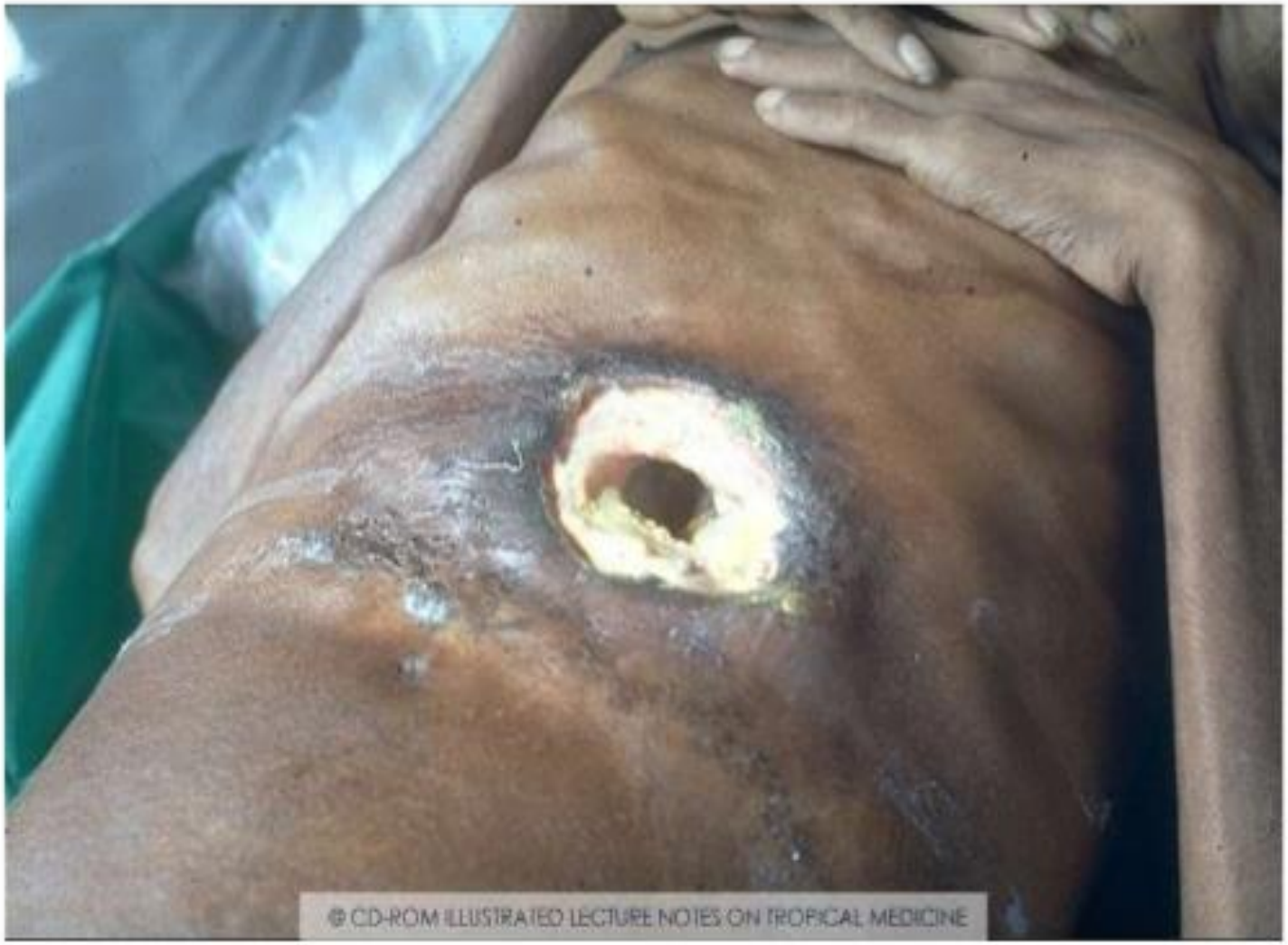
Abscess of hepatic amebiasis



Amoebic Liver abscess.



Aspirating “anchovy paste” pus from amebic liver abscess



Patient with amoebiasis liver abscess, with perforation of abscess through abdominal skin.

Complications

- Amoeboma.

(localized granulomatous mass misdiagnosed with carcinoma)

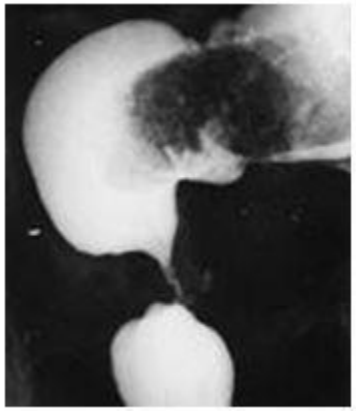
- Hemorrhage.
- Perforation of ulcer.

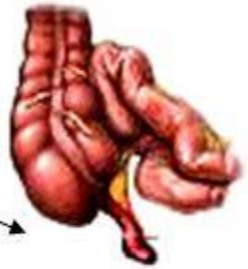
(secondary peritonitis --- rare but fatal)

- Stricture of colon.

(secondary to fibrosis)

- Appendicitis.





Diagnosis and differential diagnosis.

Stool microscopy remains the cornerstone of diagnosis but multiple specimens may need to be examined due to poor sensitivity. The definitive diagnosis of amebic colitis is made by the demonstration of hematophagous trophozoites of E. histolytica.

Colonoscopy and biopsy may be helpful in confirming the diagnosis in patient with colitis. Endoscopic features include punctuate hemorrhages and flaskshaped ulcers.

Serology is an important addition to the methods used for parasitologic diagnosis of invasive amoebiasis.

Enzyme-linked immunosorbent assays (ELISAs) and agar gel diffusion assays are positive in more than 90% of patients with symptomatic amoebiasis.

Positive results in conjunction with the appropriate clinical syndrome suggest active disease because serologic findings usually revert to negative within 6–12 months.

The interpretation of the indirect hemagglutination test is more difficult because titers may remain positive for as long as 10 years.

ELISA and PCR-based assays can be used to find amoebic antigen in feces.

Imaging studies (ultrasound, CT, MRI) are useful in patients with extraintestinal amoebic abscesses.

Aspiration of the abscess yields a brown, odorless, sterile liquid, which may show trophozoites.

Differential diagnosis

Includes:

- **ulcerative colitis,**
- **carcinoma of the**
- **colon,**
- **Crohn's disease,**
- **diverticulitis,**
- **abdominal abscesses,**
- **irritable bowel syndrome,**
- **pyogenic abscesses,**
- **hepatoma,**
- **echinococcal liver cyst.**

Treatment.

The treatment of amoebiasis is complicated by a variety of clinical syndromes, varying sites of action of different drugs, and the availability of different drugs in different countries.

Intestinal amoebiasis **should be treated with *metronidazole* 750 mg tid for 10 days or *tinidazole* 1 g bid for 3 days, followed by either of the following*: iodoquinol* for 20 days or *paromomycin* for 7 days.**

Extraintestinal amoebiasis **should be treated by *metronidazole* 750 mg tid for 10 days followed by *iodoquinol* for 20 days.**

In severely ill patients , *emetine or dehydroemetine* (less toxic one) may be added for the first few days. For a large abscesses (>3 cm), aspiration and needle drainage is indicated. Smaller abscesses resolve with medical treatment.

Asymptomatic disease **(intra-luminal carriage) should also be treated by paromomycin, diloxanide furoate, or iodoquinol due to the risk of invasive disease**

Prevention.

- Avoid ingestion of contaminated water and food.
- In endemic areas boiling of water is necessary for its decontamination.
- Vegetables should be washed well with potable water or be treated with detergent and soaked in acetic acid or vinegar.
- Avoid sexual practices that involve faeco-oral contact.