PROTOZOA

SARCODINA/ *ENTAMOEBA HISTOLYTICA*

Classification of Parasites

The parasites of humans in the kingdom **Protozoa** are now classified under three phyla: **Sarcomastigophora** (containing the flagellates and amebas), **Apicomplexa** (containing the sporozoans), and **Ciliophora** (containing the ciliates). Within these great assemblages are found the important human parasites, conveniently listed as subphyla.

(1) Sarcodina are typically ameboid and are represented in humans by species of *Entamoeba, Endolimax, Iodamoeba, Naegleria,* and *Acanthamoeba*.

(2) Mastigophora, the flagellates, have one or more whip-like flagella and, in some cases, an undulating membrane (eg, trypanosomes). These include intestinal and genitourinary flagellates (*Giardia, Trichomonas,*) and blood and tissue flagellates (*Trypanosoma, Leishmania*).

(3) Sporozoa undergo a complex life cycle with alternating sexual and asexual reproductive phases, *Isospora, Toxoplasma,* Within the class **Haematozoa** (blood sporozoans) are the malarial parasites (*Plasmodium* species) and members of the order Piroplasmida, which includes *Babesia* species

(4) Ciliophora are complex protozoa bearing cilia distributed in rows or patches, with two kinds of nuclei in each individual. *Balantidium coli,* a giant intestinal ciliate of humans and pigs, is the only human parasite representative of this group.

The parasitic worms, or helminths, of human beings belong to two phyla:

(1) Platyhelminthes (flatworms) lack a true body cavity (celom) and are characteristically flat in dorsoventral section. All medically important species belong to the classes **Cestoda** (tapeworms) and **Trematoda** (flukes). **(2) Nemathelminthes** (worm-like, separate-sexed, unsegmented roundworms) include many parasitic species that infect humans.

The Ameba (Sarcodina)

Ameba (or Amoeba) is characterized by possessing clear protoplasm which forms pseudopodia. Peudopodia: a cytoplasmic protrusion that may be formed at any point on the surface of the organism. These pseudopodia are the means by which these organisms move and engulf bacteria and red blood cells for feeding purposes. The most common amebas seen in the intestinal tract are E*ntamoeba histolytica/dispar*, *Entamoeba coli*, *Entamoeba hartmanni*, *Endolimax*

nana and *Iodamoeba bütschlii*. All but *Entamoeba histolytica* are thought to be non-pathogenic. The cysts can be identified by the addition of iodine to reveal the characteristic inclusions. The trophozoites can be seen in a fresh saline preparation of the stool although accurate identification is on a permanently stained fecal smear.

1-They are unicellular parasites with one nucleus.

2-The position of karyosom in the nucleus is variable regarding to the species.

3-Cytoplasm is divided into two parts: ectoplasm and endoplasm.

4-They moved by cytoplasmic processes called (pseudopodia).

5-Nutrition occurs by ingestion.

5-The shape is indefinite (irregular) due to constant change in shape.

6- Life cycle includes two stages: trophozoite and cyst.

Entamoeba histolytica

Introduction

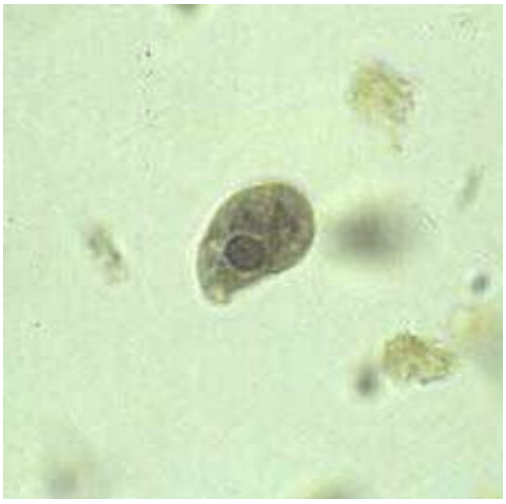
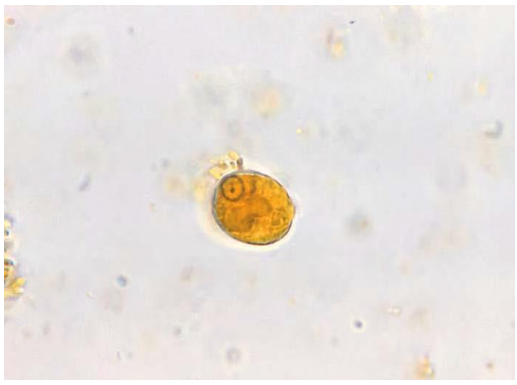
There are a large number of species of ameba which parasitize the human intestinal tract. Of these, *Entamoeba histolytica / dispar* is the only species found to be associated with intestinal disease. Although many people harbor this organism worldwide, only about 10% develop clinically invasive disease, thus the parasite has been shown to present as two very differing clinical presentations.

1. The commensal or non-invasive luminal form where the parasite causes no signs or symptoms of disease.
2. The pathogenic or invasive form where the parasite invades the intestinal mucosa and produces dysentery or amebiasis and may give rise to extra-intestinal lesions via the blood, mainly to the liver.

E. histolytica principally inhabits the large intestine

Morphology of Cysts

Cysts of *E. histolytica / dispar* are 10-20μm in diameter and contain one to four nuclei. Spherical or ovoid or irregular in shape, Chromatoid bodies are usually present in young cysts as elongated bars with bluntly rounded ends. they stained with hematoxylin like the chromatin of the nucleus, composed of crystalline ribonucleic acid (RNA). Glycogen is usually diffuse, but in young cysts it is often present as a concentrated mass, staining reddish brown with iodine. It is Characterized by the presence of hyaline cyst wall. The nucleus has centric karyosom (nucleolus). An uninucleated cyst (precyst) divides to becom binucleate, which becomes tetranucleate by fission

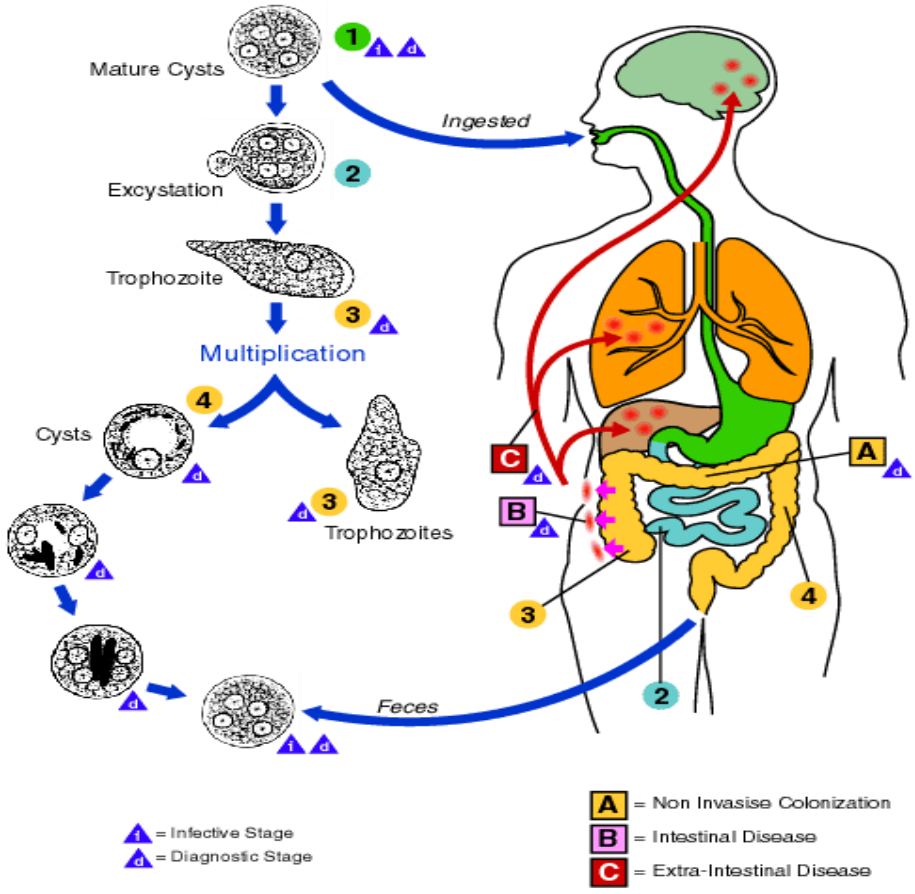
Morphology of Trophozoites

The trophozoites of *E. histolytica / dispar (12-60Ml)* recovered from dysenteric stools exhibit ingested red blood cells and clear pseudopodia. **Those of *E. dispar* will have no ingested red blood cells**. They can be up to 60µm in diameter and motility is rapid and unidirectional. Trophozoite have one nucleus with central karyosom . the cytoplasm contain translucent ectoplasm and granular ectoplasm containing RBCs and tissue debris, glycogen mass is absent in trophozoits.

Life cycle

The organism exists in two forms—the trophozoite or the dividing, vegetative and motile form and the cyst which is the dormant form. Immature cyst when passed in stool matures outside the body. Which remain alive for 10 days on moisture, thermal death point is 50 C. Chlorination of water can not kill the cyst. Human infection usually begins with the ingestion of mature quadrinucleated **cyst** (mature cyst) which is present in food and/or water contaminated with human fecal material. Cysts survive the acidic pH of the stomach and pass into the intestine. In the ileo-cecal region, cysts undergo excystment and each cyst gives rise to eight trophozoites. These migrate to and multiply in the colon. In most cases, trophozoites in the intestine live as commensals. Occasionally, however, trophozoites attack and invade the intestinal mucosa causing dysentery and/or progress through the blood vessels to extra-intestinal locations like liver, brain and lungs, where they may form life-threatening abscesses. In the intestine, many of the trophozoites encyst and produce quadrinucleated cysts. Both trophozoites and cysts are excreted along with the feces. Cysts can survive for prolonged periods outside the host while the trophozoites survive only for a few hours.

Trophozoites play no role in transmission of the disease but are responsible for producing tissue pathology. The reservoir of human infection is the "carrier"(food handler cystpasser) or asymptomatic human host who continuously passes cysts.



EPIDEMIOLOGY

The prevalence of amoebic infection varies with level of sanitation and is generally higher in the tropics and subtropics than temperate climates. In any region it is more prevalent under **crowded conditions** and may reach epidemic proportion in orphanages, prisons, and refuges.

In developed countries, populations at risk for *E. histolytica* infection include returned travelers or expatriates and immigrants from endemic countries and most cases of invasive disease are imported. Although previous studies suggest that men who have sex with men (MSM) are at a higher risk of intestinal infection with *E. dispar* due to increased oral-anal sexual contact, invasive disease is rare. Over the past several years, many investigators in Japan, Taiwan, Korea, and Australia have found that amebiasis is increasingly diagnosed among

HIV-infected MSM. Asymptomatic patients are utmost important in transmission of the disease.

While an estimated 90% of *E. histolytica* infections are asymptomatic, prospective studies of asymptomatic carriers indicate a risk of invasive disease of (9%) over the course of 6-12 months. Invasive disease has a high mortality, estimated at >10% for either dysentery severe enough to require hospitalization or liver abscess. Accordingly, the risk of developing invasive disease, as well as decreased transmission, are rationales for treating asymptomatic carriers of *E. histolytica*, however WHO recommends against treatment of asymptomatic patients when only a morphologic diagnosis by stool examination is available (i.e., *E histolytica*/*E dispar*/*E moshkovskii*).

The epidemiology of *Entamoeba histolytica* has been complicated by the mid-1990's re description of *E. histolytica* into two species: *E. histolytica*, which is pathogenic, and *E. dispar*, which is not. Further molecular studies have identified a third species, *E. moshkovskii* that also tends to be non-pathogenic. The bulk of previous studies on the prevalence of *E. histolytica* were based on stool microscopy, which is of low sensitivity and specificity and cannot distinguish *E. histolytica* cysts or trophozoites from *E. dispar* or *E. moshkovskii*. Thus the conclusions that 10% of the world's population is infected with *E. histolytica* are probably an overestimate. Serologic studies are more reliable, since only *E. histolytica* infection generates a serum antibody response and these suggest that up to 8.4% of persons in endemic countries have been exposed.

Clinical manifestation: Clinical classification of amebiasis includes:

1-Asymptomatic infections 2-Symptomatic infections:
A-intestinal amebiasis: 1-Dysenric 2-nondysentric

B-extraintestinal amebiasis: 1-hepatic 2-pulmonary 3-other Ex. foci

Intestinal Amebiasis

Clinical syndromes associated with intestinal *E. histolytica* disease include **diarrhea**, **acute rectocolitis** (dysentery), **fulminant colitis** (acute necrotizing colitis)

with **perforation**, **toxic megacolon**, **chronic nondysenteric colitis**, **ameboma**, and **perianal ulceration**

- . The onset of **(amebic dysntry)** is usually gradual over 1 to 3 weeks Dysenteric and diarrheic syndromes account for 90% of cases of invasive intestinal amebiasis. Patients with dysentery have an average of three to five mucos anguineous evacuations per day, with moderate colic pain preceding discharge, and they have rectal tenesmus. In patients with bloody diarrhea, evacuations are also few but the stools are composed of liquid fecal material stained with blood. While there is moderate colic pain, there is no rectal tenesmus. Fever and systemic manifestations are generally absent. These can easily be distinguished from that of bacterial origin, where the patient frequently complains of systemic signs and symptoms such as fever, chills, headache, malaise, anorexia, nausea, vomiting, cramping abdominal pain, and tenesmus

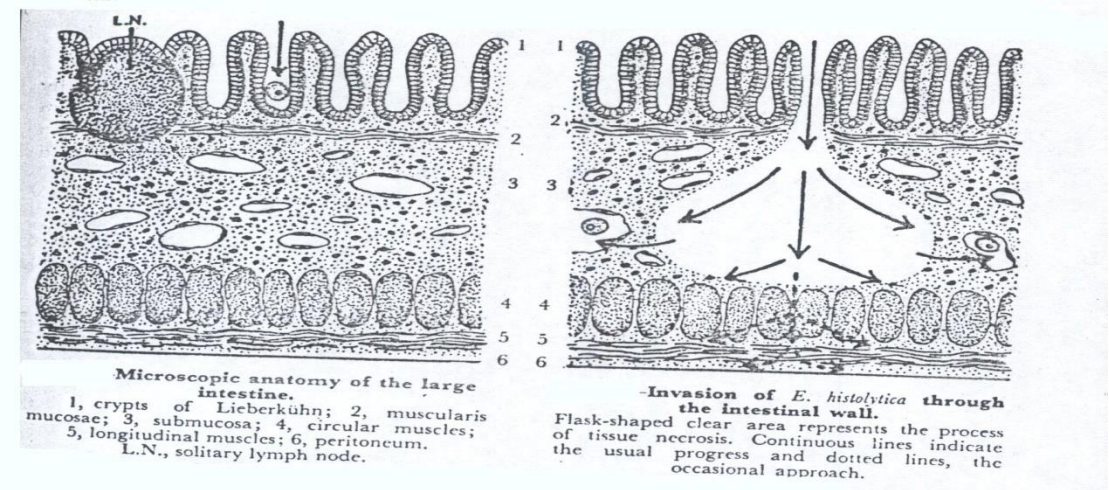
Dysentry :- (Blood and mucus in stool)

- **Amoebic appendicitis** : Acute appendicitis is a sudden inflammation of the appendix , it is one of the most common causes of emergency abdominal surgery in children and young adults, in addition to the risk of perforating appendicitis which have about 18 % from the total acute appendicitis The inflammation of appendix generally follows obstruction of the appendix by feceas, foreign bodies, parasites bacteria and rarely tumors
- **Fulminant colitis** is rare (<0.5% of cases) but associated with a mortality of more than 40%; malnourished persons, pregnant women, corticosteroid users, and very young children may be at increased risk. Patients typically appear very ill and have fever, profuse bloody mucoid diarrhea, diffuse abdominal pain, and hypotensive with signs of peritoneal irritation.
- **Flask ulcer** described in the classical 1891. The mucosal ulcer extends deep into a larger area of the submucosa, which seems to be particularly susceptible to the lytic action of the parasite, and produces abundant microhemorrhages. This explains the

finding of hematophagous amebas in stool specimens or in rectal scrapings, still the best indication of the amebic nature of a case of dysentery or bloody diarrhea. A thick exudate containing acellular proteinaceous material, red blood cells, and strands of fibrin is seen on the floor of the ulcer, where groups of amebas are identified

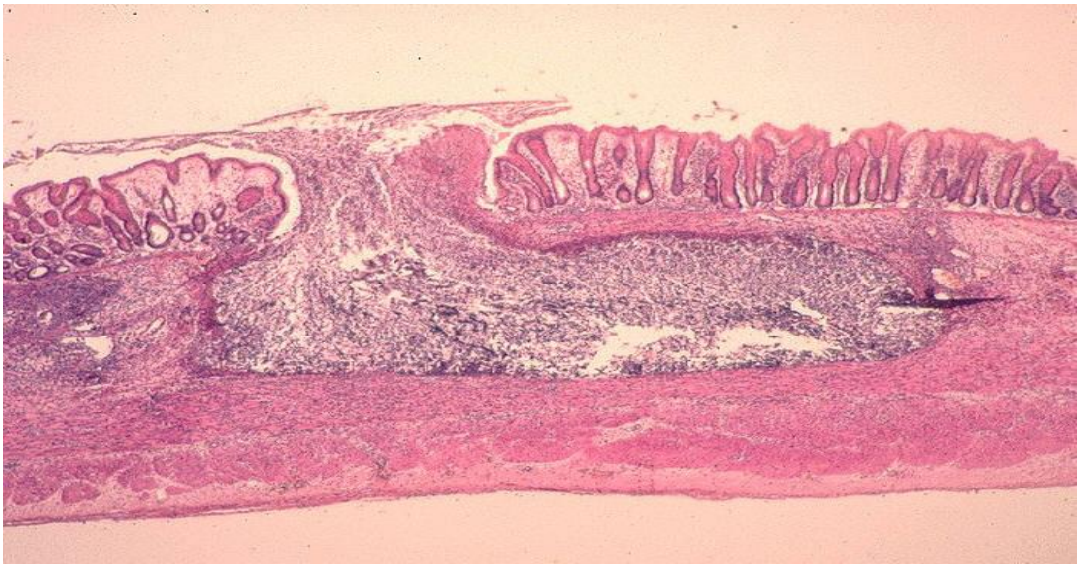
- **Intestinal perforation** usually manifests as a slow leakage rather than an acute event. Surgical interference is indicated for bowel perforation or patients with no response to antiamebic therapy although attempts to suture such necrotic bowels are usually unsuccessful.
- **Toxic megacolon** is also rare (0.5% of cases).

- **Ameboma** is the formation of annular colonic granulomatous tissue at a single or multiple sites. It usually involves the cecum or ascending colon, and may mimic carcinoma of the colon. It contains trophozoites.
- **Perianal amebiasis** may result from extension of severe bowel disease to the skin. Lesions can be ulcerative or condylomatous, enlarge slowly over weeks to months, and result in pain and bleeding.



Microscopic anatomy of the large intestine.
1, crypts of Lieberkühn; 2, muscularis mucosae; 3, submucosa; 4, circular muscles; 5, longitudinal muscles; 6, peritoneum. L.N., solitary lymph node.

Invasion of *E. histolytica* through the intestinal wall.
Flask-shaped clear area represents the process of tissue necrosis. Continuous lines indicate the usual progress and dotted lines, the occasional approach.



Extraintestinal Amebiasis

Amebic liver abscess is the most common extraintestinal manifestation of amebiasis and is 10 times more common in adult men despite an approximately equal sex distribution of colonic amebic disease. 95% of travelers develop liver abscess within 2 to 5 months (median 3 months) after leaving the endemic area. The acute symptoms include fever and a constant, dull, aching pain in the right upper quadrant or epigastrium while a subacute course may present with prominent weight loss with less fever or abdominal pain. Hepatomegaly with point tenderness over the liver below the ribs or in the intercostals spaces is a typical finding. Right-sided pleural pain or referred shoulder pain occurs when the diaphragmatic surface of the liver is involved. Associated gastrointestinal symptoms occur in 10%-35% of patients. Leukocytosis without eosinophilia is noted in 80% of cases and mild anemia in more than half. Patients with acute amebic liver abscess tend to have a normal **alkaline phosphatase** level and an elevated **alanine aminotransferase** level; the opposite is true for those with chronic disease. Ultrasonography, abdominal computed tomography, and magnetic resonance imaging are all excellent for detecting liver lesions but are not specific for amebic liver abscess.

Pleuropulmonary amebiasis: is the most common complication of amebic liver abscess. It occurs as a result of the rupture of a superior right lobe abscess with erosion through the diaphragm to involve the pleural space or lung parenchyma. Serous pleural effusion and atelectasis are

common findings and do not indicate extension of disease. In addition, formation of a **hepatobronchial fistula** is not uncommon. **Intraperitoneal rupture** occurs in 2%-7% of cases, and sudden perforation is associated with a high mortality. Left lobe abscess are more likely to progress to rupture due to late clinical presentation.

Pericardial amebiasis: an unusual but serious complication, usually presents with fever and abdominal pain with progression to substantial chest pain and signs of congestive heart failure although acute perforation with cardiac tamponade and shock can occur.

Cerebral amebiasis: has quick onset, and progresses rapidly to death over 12-72 hours without adequate therapy. Thus, when patients with known amebiasis have alteration of mental states or focal signs, amebic brain abscess should be considered.

Genitourinary amebiasis: is rare and includes **rectovaginal fistulas** and **vulvar lesions** in women and **penile amebiasis** in men.

PATHOGENESIS

The parasite is appropriately named "histolytica" for its cellular destructive nature. Factors that determine invasion of amebas include the following: *the number of amebas ingested *the pathogenic capacity of the parasite strain, *host factors such as gut motility and immune competence, and *the presence of suitable enteric bacteria that enhance amebic growth.

Lysis of the colonic mucosa in intestinal amebiasis has been related to a variety of molecules produced by *E. histolytica*: adhesins, amebapores, and proteases. A multifunctional adherence lectin allows the parasite attachment to the colonic mucus blanket, thereby avoiding elimination through the intestinal stream. The lectin is also involved in signaling cytolysis and in blocking the deposition of the harmful membrane attack complex of complement, and it could participate in the anchorage of the ameba to proteoglycans during the invasion process. The amebapores of *E. histolytica*, small but potent peptides, destroy ingested bacteria that serve as the main nutrients for the parasite in the otherwise nutrient-scarce colonic environment. Their participation in the cytolytic event has not yet been proven. Proteases can be used to degrade the extracellular matrix during invasion and aid in the lysis of target cells. In addition, interesting mechanisms of parasitic modulation of the host immune response are starting to be unraveled. The main targets of this modulation appear to be neutrophils and macrophages, which, although recruited at the site of the lesion, are unable to abort infection.

1-Adhesion of the parasite occurs mainly through a surface Gal-GalNAc lectin which binds to exposed terminal Gal-GalNAc residues of target cell glycoproteins. Other molecules include lectin , adhesin, and a surface lipophosphoglycan. The serine-rich *E. histolytica* protein may also play a role in adherence, as antibodies to SREHP partially inhibit adherence *in vitro*. cells undergo morphologic and DNA degradative changes consistent with apoptosis as well as necrosis. The mechanism of apoptosis appears to involve direct activation of downstream caspases including caspase 3 which leads to cell death. Entamoeba then engulfs apoptotic corpses. it has been proposed that the adhesin is involved in the signaling of cytolysis, probably through the stimulation of actin polymerization. Furthermore, the adhesin binds to purified C8 and C9 components of complement and blocks the assembly of the complement membrane attack complex on the amebic plasma membrane, suggesting a role in mediating amebic resistance to complement lysis through components C5b through C9.

2- Secretion of amebic proteinases and other cytotoxic substancs. The protease may assist trophozoite to gain access to target cells by degrading the extracellular matrix. A candidate for the toxin responsible for cytolysis may be a pore-forming peptide. Various amoebic pore-forming proteins have been described:

Amebapores. Once *E. Histolytica* establishes contact with mammalian cells in vitro, a rapid cytolytic event takes place that results in swelling, surface blebbing, and lysis of the inadvertent target cell, including lymphocytes, polymorphonuclear leukocytes, and macrophages, leaving the parasite unharmed. The similarity of this event to the perforin-mediated lysis of target cells by cytotoxic T lymphocytes

Amebapores are localized in cytoplasmic vesicles, as evidenced by positive immunofluorescence staining and by the presence of typical signal peptides of intracellular transport in its primary translation products. The peptides show maximum activity at acidic pH, which is consistent with previous observations, that lysis of target cells by*E. histolytica* required a pH of 5.0 within amebic vesicles

2-A 30 kDa amoebic protein was purified and shown to lyse erythrocytes and insert into and create pores in lipid bilayers.

3- A 14 kDa poreforming protein was described as an ion-channel forming protein. Of these the 5 kDa protein (amoebapore) has been the best characterized.

phagocytosed bacteria, the main intestinal source of nutrients of amebas; thus, they have a similar function to defensins found in mammalian phagocytes that kill bacteria and fungi to prevent intracellular microbial growth within digestive vacuoles.. Amebapores and other proteins, like the recently characterized ameba lysozyme that co localizes to the same cytoplasmic granules of amebapores, could synergistically enhance the antibacterial activity.

3-Amebic cytolysis of killed or viable cells prior to mucosal invasion by *E. histolytica* there is depletion of mucous and disruption of epithelial barrier. Cytolysis of the target cell is thought to require amoebic microfilament function, Ca2+ flux and phospholipase A

4- *Phagocytosis*

Trophozoites from stools of many invasive patients contain ingested erythrocytes and have much higher rate of erythrophagocytosis than healthy human carrier.

The cellular immune response of the host may contribute to destruction of the local host tissue. In hamster liver model recruitment of neutrophils is the initial host response to *E. histolytica* infection. Neutrophils are lysed when they come in contact with *E. histolytica* trophozoites releasing toxic products which lyse distant hepatocytes.

Neutrophil: infiltration of the lamina propria. The last observation of the early invasive lesion with superficial ulceration is a mild to moderate infiltration of the lamina propria. At this stage, cell infiltration around invading amebas leads to rapid lysis of inflammatory cells and tissue necrosis. Studies on mucosal immunity have provided growing evidence that intestinal epithelial cells, for instance, constitutively express or can be induced to express a number of immunologically active cytokines and soluble factors, including interleukin-8 (IL-8), monocyte chemoattractant protein 1, granulocyte-macrophage colony-stimulating factor, and tumor necrosis factor alpha (TNF-α) .Thus, in addition to their fundamental absorptive and secretory functions, intestinal epithelial cells are integral and essential components of the host's innate and acquired immune system. Neutrophils not only fail to resist *E. histolytica* but may in fact contribute to host tissue damage through their destruction and release of cytotoxic granules**.**

Invasion through locomotion. In mammals, cell locomotion involves at least three processes: (i) extension of a leading edge, (ii) attachment to the substrate surface through adhesion plaques, and (iii) pulling forward of the remainder of the cell.

Degradation of ECM components by cysteine proteases. Once regarded as a passive support element for cells and tissues, the ECM is now considered a highly active tissue that participates in cellular migration, proliferation, differentiation, and immune system signaling.

Amebic cysteine proteases are active against a variety of substrates and increased activity has been reported in clones of high virulence. Of the ECM components that *E. histolytica*encounters during colonic invasion.

Treatment

1-Asymptomatic Intestinal Colonization

There are at least three classes of drugs that have shown efficacy in clinical trials for asymptomatic intestinal colonization: dichloracetanilide derivatives, diloxanide furoate (Furamide), paromomycin (Humatin), and iodoquinol.. All have poor gastrointestinal absorption, which allows high luminal concentrations but renders them less effective in invasive disease.

Amebic Colitis

The mainstay of therapy for amebic colitis since the 1960s has been the nitroimidazoles, in particular metronidazole (Flagyl) and tinidazole (Fazigyn)

Liver Abscess

standard high dose of metronidazole for 10 days or tinidazole for 5 days.

Diagnostic Laboratory Tests

A-Specimens

1. Fluid feces—

a. Fresh and warm for immediate examination for trophozoites.
b. Preserved in polyvinyl alcohol (PVA) fixative or Merthiolate-iodine-formalin (MIF) fixative for mailing to a diagnostic laboratory

2. Formed feces for cysts.

3. Scrapings and biopsies obtained through a sigmoidoscope or (more commonly) colonoscope, most frequently found by colonoscopy.

4. Liver abscess aspirates collected from the edge of the abscess, not the necrotic center. Viscous aspirates should be treated with a liquefying enzyme such as streptodornase, then cultured or examined microscopically.

5. Blood for serologic tests and cell counts.

Microscopic Examination

If possible, always examine fresh warm feces for trophozoites if the patient is symptomatic and has diarrheic stools. Otherwise, stain smears with trichrome or iron-hematoxylin stain. The stools in amebic dysentery can usually be distinguished from those in bacillary dysentery:

The problem of routinely distinguishing *E histolytica* from *E dispar* or *E moshkovskii* remains acutely unresolved.

Differentiation of *E histolytica* (H) and *E coli* (C), the most common intestinal ameba other than *E dispar*, can be made in stained smears as follows:

Trophozoites

The cytoplasm in H is glassy and finely granular and contains only red cells and spherical vacuoles. The cytoplasm in C is granular, with many bacterial and other inclusions and ellipsoid vacuoles. The nucleus of H has a very small central endosome and fine regular chromatin granules lining the periphery; the nucleus of C has a larger, eccentric endosome, and the peripheral chromatin is more coarsely beaded and less evenly distributed around the nuclear membrane.

Cysts

Glycogen vacuoles disappear during successive divisions. Nuclei resemble those of the trophozoites. Rare cysts of H and C may have 8 and 16 nuclei, respectively. Cysts of H in many preparations contain many uninucleate early cysts; these are rarely seen with C. Binucleate developing cysts of C often show the nuclei pushed to opposite sides of the cell wall by the large central glycogen vacuole. Chromatoidal bodies in early cysts of H are blunt-ended bars; those of C are splinter-like and often occur in clusters.

Culture

Diagnostic cultures are made in a layer of fluid overlying a solid nutrient base in partial anaerobiosis. Dobell's diphasic and Cleveland-Collier media are most often used.

Serology

Serologic testing is primarily for extraintestinal amebiasis, when stools are often negative. Serodiagnosis, most commonly by IHA test, is considered sensitive and specific, though it cannot distinguish recent from

past infections. Serologic testing in intestinal infections is less reliable except where considerable tissue invasion has occurred. Antiamebic antibodies occur only with *E histolytica,* since the nonpathogenic species do not elicit a serologic response.

Another test to distinguish pathogenic from nonpathogenic strains in a stool specimen is an ELISA that uses monoclonal antibodies against the galactose adhesin, a pathogen-specific epitope of *E histolytica*. Amebic antigen (Tech-Lab Test) in the stool is sensitive and specific for *E histolytica* and generally does not respond to *E dispar* or other nonpathogenic amebas.

Radiation Methods

Hepatic abscess, usually showing as an elevation of the right dome of the diaphragm, can be observed by ultrasonography, CT, MRI, or radioisotope scanning. The round or oval hepatic lesion is clearly and often abruptly demarcated from the surrounding normal tissue. Serologic tests in these cases are usually strongly positive.

Prevention & Control

Cysts are usually ingested through contaminated water. In the tropics, contaminated vegetables and food are also important cyst sources; flies have been incriminated in areas of fecal pollution. Asymptomatic cyst passers are the main source of contamination and may be responsible for severe epidemic outbreaks where sewage leaks into the water supply or breakdown of sanitary discipline occurs (as in mental, aged, prison, or children's institutions). A high-carbohydrate, low-protein diet favors the development of amebic dysentery both in experimental animals and in known human cases. Control measures consist of:

a- Improving environmental and food sanitation.
b- B-Treatment of carriers is controversial, it is agreed that they should be excluded from food handling.
c- Possible environmental contamination should be considered in the treatment decision for an asymptomatic cyst passer.
d- No fully satisfactory and safe drug is yet available for chemoprophylaxis, and the mix of drugs required for therapy attests to the problems of treating amebiasis.
e- safe disposal of human excreta.
f- Travelers should avoid unpeeled fresh vegetables and fruits and drink only boiled or bottled water.
g- Avoiding sexual practices that involve fecal-oral contact can reduce infection in homosexuals.

h- In mental institutions recurrent outbreaks of amebiasis can be prevented by routine screening of stool and treating infected patients

VACCINES

There are no commercially available vaccines against amebiasis, but the adherence lectin and the SREHP protein are promising candidates in animal systems.

Antiparasitic Agent Prophylaxis

A global traveler surveillance network revealed that amebiasis accounted for 12% of gastrointestinal illnesses in returning travelers; however antiparasitic agent prophylaxis is not routinely recommended and has not been studied.