Entamoeba histolytica

DISEASE

- Amebiasis
- Amebic dysentery
- Amebic hepatitis
### Intestinal and Urogenital Protozoa

#### Amebae

<table>
<thead>
<tr>
<th>Trophozoite</th>
<th>Cyst</th>
</tr>
</thead>
<tbody>
<tr>
<td>Entamoeba histolytica</td>
<td>*</td>
</tr>
<tr>
<td>Entamoeba hartmanni</td>
<td>*</td>
</tr>
<tr>
<td>Entamoeba coli</td>
<td>*</td>
</tr>
<tr>
<td>Entamoeba polecki</td>
<td>*</td>
</tr>
<tr>
<td>Endolimax nana</td>
<td>*</td>
</tr>
<tr>
<td>Iodamoeba bütschlii</td>
<td>*</td>
</tr>
<tr>
<td>Dientamoeba fragilis</td>
<td>*</td>
</tr>
</tbody>
</table>

*Rare, probably of animal origin
Flagellate
Scale: 0 5 10 μm
Adapted from Brooke and Melvin, 1964

*non-pathogen

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Dr. Abdulkader M.D. Tonkal
Entamoeba histolytica

Epidemiology

- A world-wide distribution.
- A higher prevalence in tropical and subtropical countries.
- 10% of the world population carries the parasite.
- More prevalent in certain groups (e.g. children, homosexual, prisoners, orphanages houses, people in mental hospitals .. etc)

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Entamoeba histolytica

EPIDEMIOLOGY

- Incidence: cosmopolitan
- Main source of infection:
  - Cyst passing chronic patient.
  - Asymptomatic carrier.
- Animal reservoir host include monkeys, dogs and pigs.
Entamoeba histolytica

MORPHOLOGY

CYST
ENTAMOEBA HISTOLYTICA

TROPHOZOITE
Entamoeba histolytica

**MORPHOLOGY**

1. **Trophozoite:**
   - 10 - 60 μ X 15 - 30 μ
   - **Ectoplasm**: pseudopodia.
   - **Endoplasm**
   - **Nucleus**: uniform and closely packed fine granules of chromatin
   - **Karyosome**
Entamoeba histolytica

MORPHOLOGY

2. Precyst

3. cyst

- wall, 0.5 μ-
- chromatoid bodies.
- mature infective cyst: four smaller nuclei
- Habitat: wall & lumen of the colon
- Reproduction: binary fission
Entamoeba histolytica

MORPHOLOGY
Modes of Transmission

Human being is the principle host (source of infection).

1. Ingestion of contaminated food or water with amoebae cyst (infective stage).
2. Direct fecal contamination (children).
4. House flies and cockroaches (mechanical transmission)
Life cycle

1. Entry (cysts)
2. Multiplication (trophozoites)
3. Spread (trophozoites) - Hematogenous, Direct extension
4. Disease (trophozoites) - Lung abscess, Liver abscess (other organs), Diarrhea, Ulceration
5. Exit (cysts in formed stools)
Life cycle of *Entamoeba histolytica* in man.
(After Smyth, 1994)

- **Pathogenic Phase**
  - Invasion of mucosa and sub-mucosa; formation of abscess
  - Abscess bursts
  - Reinvasion
  - Pre-cystic amoeba
  - Encystation

- **Non-Pathogenic Phase**
  - Non-virulent form feeding on bacteria and detritus in gut lumen
  - Metacystic amoeba
  - Excystation in small intestine
  - Cyst ingested

Survival of cysts 2 - 5 weeks at room temperature.
Cyst

Excyst (small intestine)

Commensal growth (COLON)

Mucosal ulceration (COLON)

- Direct extension to skin
  - Cutaneous or perianal amebiasis

- Hematogenous spread to liver
  - Hepatic abscess
    - Spread to brain or other organs
      - Brain abscess
    - Direct extension to pleura, lung or pericardium
      - Lung or pericardial abscess

Continued commensal existance

- Encystation
  - Cyst in feces

Desiccation (sigmoid & rectum)

Fecal-oral spread (direct or indirect)

Entry

10 μm
Factors affecting pathogenicity

1) Parasite factors:

- **Strain**: pathogen or non-pathogen (Zymodeme*)
- Virulent capability,
- Proteolytic enzymes,
- Number of parasites.
Factors affecting pathogenicity

2) Intestinal factors:
- intestinal flora (+ve)
- enteric bacterial flora (+ve)
- very low oxygen tension (+ve)
- bowel hyper motility (-ve)

3) Other human factors:
- immune status
- nutritional status.... etc
**Pathogenesis**

**Primarily**
- intestinal amebiasis:
  - Intestinal lesion
  - cecal & sigmoido-rectal regions

**Secondarily**
- Extra-intestinal amebiasis
  - Liver, Brain, lung .... etc
* Colonic lesions:

1. Initial lesion begin as small foci

2. progress to ulcer (flask-shaped ulcer)
   - ulcer cavity containing cytolyzed cells, mucus, necrotic tissues & ameba trophozoit
   - Crateriform appearance.

3. may disseminate throughout the colon.
A) Adherence of amoeba to the intestinal mucus barrier.
B) Depletion of the mucus layer and disruption of the intestinal mucosa.
C) Penetration by the amoeba and lysis of epithelial cells and host inflammatory cells.
D) Then a subsequent lateral extension through the submucosa.
E) Formation of flask-shaped ulcer.
Complications of Intestinal Amebiasis

- Intestinal perforation.
- Hemorrhage (massive)
- Amebic stricture (usually in the anus, rectum or sigmoid colon).
- Amebic appendicitis (in case of severe colonic amebiasis).
- Amebomas (granulomas in colon).
- Cutaneous amebiasis.
- Pseudopolyposis
Extra Intestinal amebiasis

- Trophozoites may invades other organs either:
  - Heamatogeneous: by blood-stream (portal vein)
  - OR
  - direct extension (metastasize).

- Organs infected:
  - liver, lung, brain, pericardium, spleen, genitalia, skin.
Extra Intestinal amebiasis

- Liver abscess:
  - single or multiple, right lobe.
  - Mixture of blood and necrotic materials (creamy yellow in color).

- Pulmonary amebiasis:
  - lower right lung.

- Abscess of brain:
  - Always fatal.

- Cutaneous lesion.
SYMPTOMATOLOGY

- Depends on the location and severity of the infection.
- Asymptomatic infections:
  - most common.
- Healthy carrier:
  - passes millions of cysts / day.
Clinical Classification of Amebiasis
according to the World Health Organization (WHO) (1969).

I. Asymptomatic infections

II. Symptomatic infection

A. Intestinal amebiasis
   1. Dysenteric
   2. Nondysenteric colitis

B. Extraintestinal amebiasis
   1. Hepatic
      a. Acute nonsuppurative
      b. liver abscess
   2. Pulmonary
   3. Other Extraintestinal foci (very rare)
Clinical manifestations

Acute intestinal amebiasis

Incubation period:
* 1 - 14 weeks with often sudden onset.

- Severe dysentery and tenesmus
  * The stool are liquid and containing blood and mucus. *Trophozoite in the stool.*

- Acute abdominal cramps and tenderness

- Chills and fever *(38° to 39°)*

- Nausea and headache.
Clinical manifestations

In severe cases:

- Prolonged diarrhea:
  \[\Rightarrow \textit{lead to dehydration}\]

- Massive destruction of the mucosa, hemorrhage and perforation:
  \[\Rightarrow \textit{lead to anemia}\]

- Enlarged, tender liver \textit{without abscess formation}

- \textit{Trophozoite in the stool}
Clinical manifestations

Chronic amebiasis

- Recurrent attacks of dysentery with intervening periods of gastrointestinal disturbances and constipation.
- Localized abdominal tenderness.
- Liver may be enlarged.
- Prolong disease may lead to weight loss and weakness.
Clinical manifestations

Hepatic amebiasis

(Amebic hepatitis & abscess of the liver)

- Liver enlarged and tender.
- Pain in the upper right hypochondrium.
- Pain referred to the right shoulder.
- Fever & chills.
Clinical manifestations

- **Hepatic amebiasis**

- Abscess may extend to diaphragm leading to:
  - diaphragmatic elevation,
  - compression of the right lower lobe of the lung.

- Jaundice may occurs in case of large abscess.
CT scan of the abdomen showing replacement of the right lobe of the liver by an amebic abscess.
Clinical manifestations

- **Pulmonary amebiasis**:  
  - Chest pain, cough, dyspnea, hemoptysis,
  - Chills, fever and leukocytosis.

- **Amebic infections of the brain**:  
  - Headache, fever, seizures may be coma.
  - Signs & symptoms of brain abscess or tumor
  - almost fatal

2. Direct method:
   - Identification of the parasite in feces or tissue.

3. Laboratory diagnosis (supportive).
Microscopic identification

- Proper collection of material.
- Repeated examinations, especially in chronic cases.

Stool examination:

- Direct examination (Iodine stain);
- Concentration methods;
- Permanent stain.
Lab. diagnosis key points

⚠️ **A minimum of three specimens (stool)** should be submitted for the diagnosis of intestinal amebiasis.

✓ Any examination for parasites in stool specimens must include the use of a permanent stained smear (even on formed stool).

✓ Presumptive identification on a wet preparation must be confirmed by using the permanent stained smear.

✓ The six smears should be prepared at sigmoidoscopy but should not take the place of the ova and parasite examination.
Supportive methods

- Serology Test (IHA, ELISA, IF):
  - Antibody may or may not be positive in intestinal disease and is much more likely to be positive in extraintestinal disease.

- Culture.

- Barium X-ray examination.

- Aspiration (ultrasound guidance).

- Blood examination (Leukosytosis).
Diagnosis

Acute diarrhea

Without blood

Laboratory examination of feces for ova and parasites

Stool culture

If positive

Virulence testing

If negative

Investigate other etiologies

With blood

Sigmoidoscopic biopsy or aspiration

If positive

Culture and virulence testing

If negative

Investigate other etiologies
Diagnosis

Liver mass
  ↓
Imaging
  ↓
Ultrasound or
  ↓
CT scan or
  ↓
Isotope scan

If positive for abscess
  ↓
Serology for amebas
  ↓
If positive
  ↓
Treat
  ↓
If negative
  ↓
Aspirate
  ↓
If positive
  ↓
Treat
  ↓
If negative
  ↓
Search for other etiologies

If negative for abscess
  ↓
Search for other etiologies
Severe dysenteric infection

- Remain in bed
- Bland high-protein & high vitamin diet with adequate fluids

Chemotherapy:

- Relief of the acute attack
- Destruction of the trophozoites in intestinal mucosa & lumen
- Control of secondary bacterial infection
Drug of choice

Metronidazole (Flagyl) 5 - 10 days
- Side effects: headache, nausea, diarrhea, altered sense of smell
- Carcinogenic, mutagenic

Iodoquinol - 20 days
- Side effects: headache, malaise, abdominal pain, diarrhea, rash, pruritus.
PREVENTION

- Reducing the sources of infection.
- Blocking the channels of transmission
- Protecting the susceptible host

Humanity is the chief source of infection.
Entamoeba histolytica

All infections should be treated.
Asymptomatic food handler.
Effective environmental sanitation.
Water supply.
Boiling water.
Treat drinking water with iodine.
## TABLE 79-1  Classification of Amebiasis

<table>
<thead>
<tr>
<th>WHO Clinical Classification of Amebiasis Infection (Modified)</th>
<th>Pathophysiologic Mechanisms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asymptomatic infection</td>
<td>Colonization without tissue invasion</td>
</tr>
<tr>
<td>Symptomatic infection</td>
<td>Invasive infection</td>
</tr>
<tr>
<td>Intestinal amebiasis</td>
<td></td>
</tr>
<tr>
<td>A. Amebic dysentery</td>
<td>Fulminant ulcerative intestinal disease</td>
</tr>
<tr>
<td>B. Nondysentery gastroenteritis</td>
<td>Ulcerative intestinal disease</td>
</tr>
<tr>
<td>C. Ameboma</td>
<td>Proliferative intestinal disease</td>
</tr>
<tr>
<td>D. Complicated intestinal amebiasis</td>
<td>Perforation, hemorrhage, fistula</td>
</tr>
<tr>
<td>E. Post-amebic colitis</td>
<td>Mechanism unknown</td>
</tr>
<tr>
<td>Extraintestinal amebiasis</td>
<td></td>
</tr>
<tr>
<td>A. Nonspecific hepatomegaly</td>
<td>Intestinal infection with no demonstrable invasion</td>
</tr>
<tr>
<td>B. Acute nonspecific infection</td>
<td>Amebas in liver but without abscess</td>
</tr>
<tr>
<td>C. Amebic abscess</td>
<td>Focal structural lesion</td>
</tr>
<tr>
<td>D. Amebic abscess, complicated</td>
<td>Direct extension to pleura, lung, peritoneum, or pericardium</td>
</tr>
<tr>
<td>E. Amebiasis cutis</td>
<td>Direct extension to skin</td>
</tr>
<tr>
<td>F. Visceral amebiasis</td>
<td>Metastatic infection of lung, spleen, or brain</td>
</tr>
</tbody>
</table>