**Al Balqa App[lied University**

**College of Medicine**

**Lecture 9**

**Entamoeba histolytica**

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* ***Entamoeba histolytica*** cysts are present only in the lumen of the colon
* The cyst may contain a glycogen vacuole and chromatoid bodies with characteristic rounded ends.
* Nuclear division occurs within the cyst, resulting in a quadrinucleated cyst, and the chromatoid bodies and glycogen vacuoles disappear.
* Diagnosis in most cases rests on the characteristics of the cyst, as trophozoites usually appear only in diarrheic feces in active cases and survive for only a few hours.
* The ameboid trophozoite is the only form present in tissues. The cytoplasm has two zones, a hyaline outer margin and a granular inner region that may contain red blood cells but ordinarily contains no bacteria.
* The nuclear membrane surrounded a small central body ( karyosome).



**Pathology and Pathogenesis of**

**Invasive Amebiasis**

* It is estimated that approximately 50 million cases of invasive disease occur each year.
* Disease results when the trophozoites of *E. histolytica* invade the intestinal epithelium and form discrete ulcers with a pinhead-sized center and raised edges, from which mucus, necrotic cells, and amebae pass.
* The trophozoites multiply and accumulate above the muscularis mucosae.
* Rapid lateral spread of the multiplying amebae follows, producing the characteristic “flask-shaped” ulcer of primary amebiasis: a small point of entry, leading via a narrow neck through the mucosa into an expanded necrotic area in the submucosa.



* Subsequent spread may occur, undermining large areas of the mucosal surface.
* Trophozoites may penetrate the muscle layers and occasionally the serosa, leading to perforation into the peritoneal cavity.
* Subsequent enlargement of the necrotic area produces gross changes in the ulcer, which may develop shaggy over hanging edges, secondary bacterial invasion, and accumulation of neutrophilic leukocytes.
* **Secondary intestinal lesions** may develop as extensions from the primary lesion (usually in the cecum, appendix, or nearby portion of the ascending colon).
* The sigmoid colon and rectum are favored sites for later lesions.
* An amebic inflammatory or **granulomatous tumor-like mass (ameboma)** may form on the intestinal wall, sometimes growing sufficiently large to block the lumen.



**Factors that determine invasion of amebae include the following**:

* **the number of amebae ingested**
* **the pathogenic capacity of the parasite strain**
* **host factors such as gut motility, immune competence, and the presence of suitable enteric bacteria that enhance amebic growth.**

Correct and prompt identification of the *Entamoeba* species remains a critical problem.

* Trophozoites, especially with red blood cells in the cytoplasm, found in liquid or semiformed stools are pathognomonic.
* Symptoms vary greatly depending on the site and intensity of lesions.

- **Extreme abdominal tenderness**

- **Fulminating Dysentery**

- **Dehydration**

- **Incapacitation occur in serious disease**.

* In less acute disease, onset of symptoms is usually gradual and often includes episodes of diarrhea, abdominal cramps, nausea and vomiting, and an urgent desire to defecate.
* **More frequently, there will be weeks of cramps and general discomfort, loss of appetite, and weight loss, with general malaise.**
* Symptoms may develop within 4 days of exposure, may occur up to a year later, or may never occur.
* Extraintestinal infection is metastatic and rarely occurs. By far the most common form is **amebic hepatitis or liver abscess** (4% or more of clinical infections).
* A true amebic abscess is **progressive,** **nonsuppurative** (unless secondarily infected), and **destructive**. The contents are necrotic and bacteriologically sterile, active amebae being confined to the walls.
* A characteristic **“anchovy paste”** is produced in the abscess and seen on surgical drainage.
* Rarely, amebic abscesses occur elsewhere (eg, lung, brain, spleen, or draining through the body wall). Any organ or tissue in contact with active trophozoites may become a site of invasion and abscess.

**OTHER INTESTINAL AMEBAE**

* Invasive or pathogenic *E. histolytica* is now considered a species distinct from the more common nonpathogenic commensal species
* ***Entamoeba dispar***and the related ***E moshkovskii***are differentiated by isoenzyme and genetic analyses.
* *Entamoeba histolytica* must be distinguished not only from all *E. dispar* and *E moshkovskii* but also from four other ameba-like organisms:

**(1) *Entamoeba coli,* which is very common**

**(2) *Dientamoeba fragilis* (a flagellate), the only intestinal parasite other than *E. histolytica* that has been suspected of causing diarrhea and dyspepsia but is not invasive**

**(3) *Iodamoeba bütschlii***

**(4) *Endolimax nana.***



**Diagnosis**

* Enzyme immunoassay (EIA) kits are available commercially for serodiagnosis of amebiasis when stools are often negative.
* EIA tests to detect amebic antigen in the stool are also sensitive and specific for *E histolytica* and can distinguish between pathogenic and nonpathogenic infections.

**Epidemiology**

* *Entamoeba histolytica* occurs worldwide, mostly in developing countries where sanitation and hygiene are poor.
* Infections are transmitted via the **fecal–oral route**; cysts are usually ingested through contaminated water, vegetables, and food; flies have also been linked to transmission in areas of fecal pollution.
* Most infections are asymptomatic, with the asymptomatic cyst passes being a source of contamination for outbreaks where sewage leaks into the water supply or breakdown of sanitation occurs (as in mental, geriatric, or children’s institutions or prisons).