Entamoeba histolytica

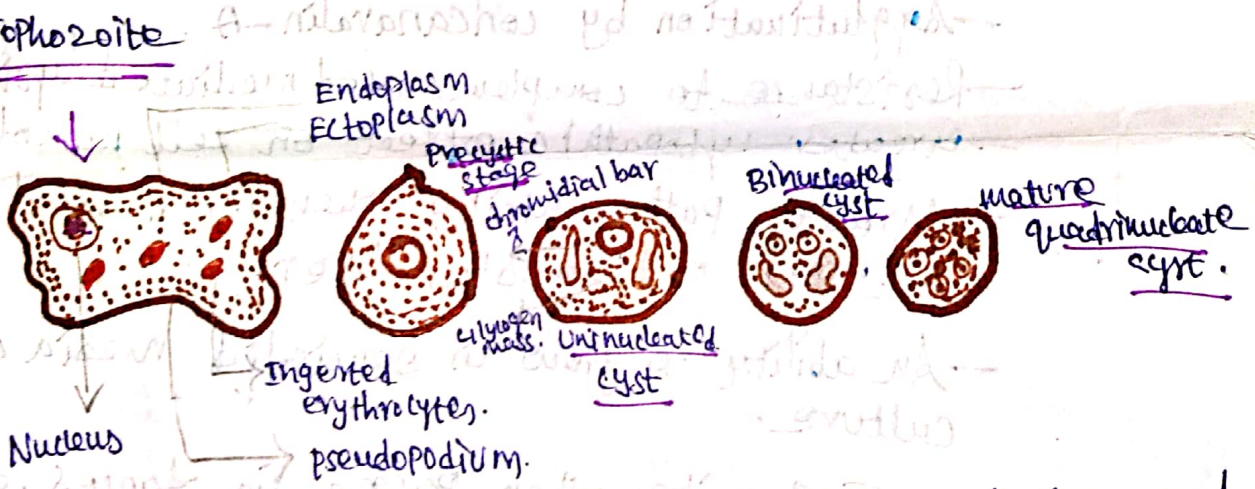
E. histolytica produces intestinal and extra intestinal diseases. It is a leading cause of morbidity in developing countries.

E. histolytica infects more than 10% of the world's population.

Amoebiasis is endemic in India, where the prevalence exceeds 30% in some places. It can affect all age groups and both the sexes.

Organism Characteristics

E. histolytica exists as trophozoites, precysts, and cysts.



The trophozoites range in size from 18 to 60µm and do not have a definite shape.

The trophozoites exhibit active unidirectional movement achieved with the help of broad hyaline, finger-like Pseudopodia.

Their cytoplasm has an outer, clear ectoplasm and inner, granular endoplasm.

- The trophozoites of E. histolytica engulf red blood cells, bacteria, yeast and other debris. The presence of red blood cells in the cytoplasm is diagnostic of E. histolytica as it is the only intestinal amoeba to exhibit this characteristic.

- The trophozoites contain a single, spherical, 3 to 5µm nucleus. The nucleus has a delicate nuclear membrane.

-on the internal surface of the nuclear membrane there are minute granules known as chromatin dots.
In the centre of the nucleus is a single dense karyosome or nucleolus. The nuclear pattern of E. histolytica differentiates the parasite from other species of amoeba.

-The trophozoites of E. histolytica live in the mucosal folds of the large intestine and divide by binary fission. They may invade the intestinal wall. There are distinct invasive and non-invasive strains of E. histolytica.

Virulence of E. histolytica include

- Enhanced erythrophagocytosis
- Reduced surface charge.
- Agglutination by concanavalin-A.
- Resistance to complement mediated lysis.
- Greater cytopathic effect on cell monolayers.
- Greater pathogenicity when inoculated into rat caecum or hamster liver.
- An ability to grow in semisolid media and axenic culture.

Trophozoites when passed in faeces, die on exposure to air. Gastric acid also destroys them. Therefore, trophozoites can not transmit infection. E. histolytica has to change from a trophozoite to a cyst to enter a new host.

Encystation

-The trophozoites become non-motile, rounded and devoid of inclusion. This is the precystic stage.

-The precysts are smaller than trophozoites and usually larger than cyst.

-They secrete a highly refractive cyst wall around themselves and become the cyst.

Cysts

- range in size from 8 to 22µm.
- Depending upon the maturity of the cyst, they may contain one, two or four nuclei.

Immature cysts

- have a single nucleus, diffuse glycogen mass and sausage-shaped chromatoid bodies.
- The glycogen mass serves as a food reserve. The chromatoid bodies contain ribonucleic acid.

Quadrinucleate cysts.

The cysts mature with two mitotic divisions of the nucleus to form sequentially binucleate and then quadrinucleate cysts.

- In this process the nuclear size is reduced from 8µm to 2µm.
- The glycogen mass and the chromatoid bodies gradually reduce in size and ultimately disappear.

Immature cysts passed in the faeces can mature outside the human body.

- The mature cysts can survive in moist environment for 10days. They withstand the gastric acid and therefore act as the infective forms.
- Chlorination used in purification of public water supply is insufficient to kill the cysts.

Life cycle

- The infective form of the parasite is the mature cyst passed in the feces of convalescents and carriers.
- The cysts remain viable under moist condition for about ten days.
- The cysts ingested in contaminated food or water pass through the stomach undamaged and enter the small intestine.

- When the surrounding medium becomes alkaline the cyst wall is damaged by trypsin in the intestine leading to excystation.
- The cytoplasm gets detached from the cyst wall and amoeboid movements appear causing a tear in the cyst wall through which the quadrinucleate amoeba emerges. This stage is called the metacyst.
- The nuclei in the metacyst immediately undergo division to form eight nuclei, each of which gets surrounded by its own cytoplasm to become eight small amoebulae or metacystic trophozoites.

If Excystation takes place in the small intestine, the metacystic trophozoites do not colonise there, but are carried to the caecum.

The optimum habitat for the metacystic trophozoites is the caecal mucosa where they lodge in the glandular crypts and undergo binary fission. Some develop into precystic forms and cysts, which are passed in feces to repeat the cycle.

- The entire life cycle is thus completed in one host

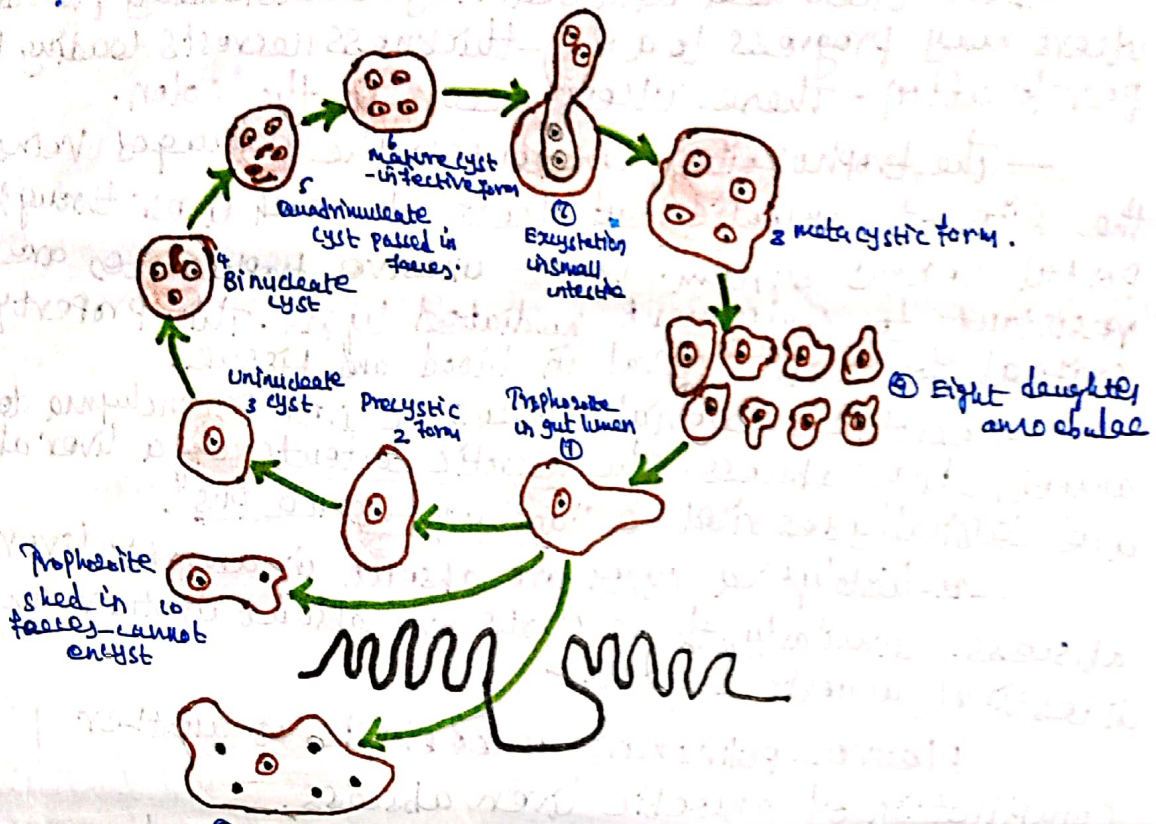
In most cases it remains with in the lumen of the large intestine, feeding on the colonic contents and mucus as a commensal without causing any ill effects. Such persons become carriers or asymptomatic cyst passers as their stool contains cysts.

Culture:-

Successful cultivation of E. histolytica using

① egg slant - Locke's solution diphasic medium
② monophasic liquid medium.
③ Robinson's medium.

LIFE CYCLE OF - E. histolytica



Pathogenesis and Pathology

- Trophozoites can invade tissue after penetrating through the intestinal mucosa. The cyst lack this ability.
- Trophozoites attach to the interglandular epithelium by surface lectins.
- These lectins attach to high-affinity receptors on mucosal cells. Galactose or N-acetyl-D-galactosamine make up these receptors.
- on contact with the cells, trophozoites of E. histolytica release phospholipase-A and amoebopore (a pore forming, complement-like protein). These proteins are responsible for lysis of the cells. E. histolytica produces protinases that degrade collagen, elastin and other components of the extracellular matrix.

Invasion of the mucosa by E. histolytica leads to ulceration.

-Submucosal extensions of ulceration under viable-appearing surface mucosa causes the classic "flask-shaped" ulcer.

-Deep ulcers heal with scarring. occasionally, the deep ulcers may progress to a full-thickness necrosis leading to perforation. these ulcers occur in the colon.

-The trophozoites can enter the damaged veins at the site of amoebic ~~and~~ ulcers to reach liver through portal venous system. these invasive trophozoites are resistant to complement mediated lysis. This property is critical for their survival in blood and tissue.

-Necrotic material replaces the liver parenchyma to form amoebic liver abscess. The necrotic contents of a liver abscess are classically described as "anchovy-sauce pus".

-E. histolytica cysts are absent in amoebic liver abscess. similarly, these cysts are absent in other extra-intestinal amoebic lesions.

Pleuro-pulmonary amoebiasis is another complication of amoebic liver abscess.

other extra-intestinal complications of amoebiasis, like cerebral amoebiasis with brain abscess and genito-urinary amoebiasis are rare.

Clinical features:

clinical conditions associated with the E. histolytica infection are:

Intestinal

Non-invasive

1. Asymptomatic
2. Symptomatic.

Invasive

1) Acute dysentery.
2) Fulminant colitis (describing a condition of sudden onset & severe short duration)
3) Toxic megacolon → lengthening of the colon by obstruction of the colon.
4) Chronic colitis.
5) Amoeboma. (describing a disease or inflammation that shows these changes)
6) Perianal ulceration small painful swelling beside the anus

Extra intestinal

1) Liver abscess
2) Liver abscess complicated by: peritonitis,

pericarditis, discharging sinus.

③ lung abscess.

④ Brain abscess.

⑤ Uerito.-urinary disease

Laboratory diagnosis

-stool examination is the most important test to establish the diagnosis of amoebic dysentery. It is also useful to differentiate amoebic and bacillary dysentary.

→Early examination of fresh stool is necessary, preferably within 30 minutes. In saline preparation trophozoites show active uni-directional motility.

-The trophozoites are haematophagous (that is, they ingest red blood cells).

-In bacillary dysentery, macrophages with ingested red blood cells are similar to the trophozoites of E. histolytica. It is essential to study the nuclear details.

Iron-hematoxylin or trichrome stain are useful for this purpose.

-Three successive stool examinations are negative, sigmoidoscopy with biopsy of the edge of ulcers may be useful.

Cysts of E. histolytica can be found in faeces during the convalescent stage of amoebic colitis and in asymptomatic carrier. Visualization of cysts is possible in Saline preparation and better so in Iodine preparation.

concentration techniques like formal-ether and zinc Sulfate floatation are useful if the number of cysts is small.

A special culture medium known as TYI-S-33 supports the growth of E. histolytica in culture. cultures are useful to obtain material for zymodeme assay. DNA probes are also available to distinguish between invasive and non-invasive isolates of E. histolytica.

Serological tests

- for detection of anti-amoebic antibodies are helpful in diagnosis of invasive amoebiasis. Diagnostic kits are available. In these tests, the principles of counter current immunoelectrophoresis, gel diffusion precipitation, indirect immunofluorescence and enzyme linked immunosorbant assay are used.

Radiographic Studies

Barium studies reveal the lesions of amoebic colitis and amoeboma. Barium studies are potentially dangerous and non-specific Magnetic resonance imaging and ultra-sonography detect amoebic liver abscess as round or hypo-echoic areas.

Diagnostic liver aspiration can be performed liver abscess is suspected. Wet preparation for demonstration of trophozoites and Gram's staining aswell as culture for detection of bacteria helps in the final diagnosis.

Treatment:

Imidazol drugs ps metronidazole, tinidazole, secnidazole and ornidazole are useful

Luminal agents like Iodoquinol, diloxanide furoate or paromomycin should follow - This agents help in treating cyst-passers.

Prevention:

- proper disposal of sewage.
- Good personal hygiene, washing of raw vegetable and fruits before consumption and protection of food from flies and cockroaches are important for personal protection.
- Quandrinucleate cyst is resistant to routine chlorination therefore drinking water should be purified by either boiling, filtaration or iodination.