

# THE MEDICAL ACADEMY NAMED AFTER S. I. GEORGIEVSKY OF VERNADSKY CFU

## DEPARTMENT OF MEDICAL BIOLOGY

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*ENTAMOEBA  
HISTOLYTICA*

- ◉ Amoeba are structurally simple protozoa which have no fixed shape
- ◉ Phylum : Sarcomastigophora
- ◉ Subphylum : Sarcodina
- ◉ Super class : Rhizopoda
- ◉ Order : Amoebida

## ◉ Amoeba

Free living

Intestinal

- ◉ *Entamoeba histolytica* is an intestinal amoeba
- ◉ All intestinal amoebae are non pathogenic except *Entamoeba histolytica*
- ◉ All free living amoeba are opportunistic pathogens.

## **ENTAMOEBA HISTOLYTICA**

- ◉ E. histolytica was discovered by Losch in 1875
- ◉ Demonstrated the parasite in the dysenteric feces of a patient in St.Petersburg in Russia.

## **ENTAMOEBA HISTOLYTICA**

- ◉ MORPHOLOGY
- ◉ LIFE CYCLE
- ◉ PATHOGENESIS & CLINICAL FEATURES
- ◉ LABORATORY DIAGNOSIS
- ◉ TREATMENT
- ◉ PREVENTION

# MORPHOLOGY

➤ E.histolytica occurs in 3 forms

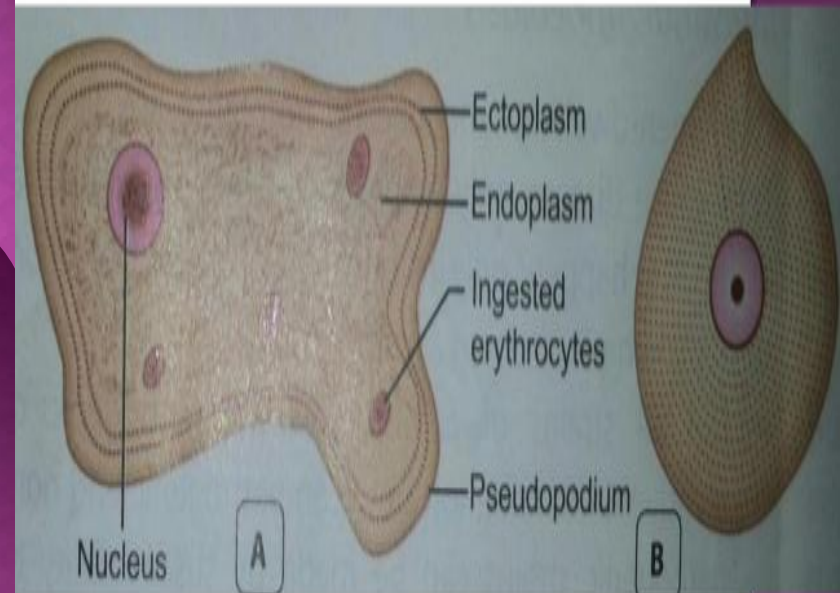
*Trophozoite*

*Precyst*

*Cyst*

## TROPHOZOITE

- Vegetative or growing stage of the parasite
- Only form present in tissues
- Irregular in shape
- Size: 12-60  $\mu\text{m}$  (Average 20 $\mu\text{m}$ )
- Large and actively motile in freshly passed dysenteric stool, while smaller in convalescents and carriers.
- In the lumen, commensal and small in size (15-20  $\mu\text{m}$ )-MINUTA FORM



## Cytoplasm

Outer ectoplasm-clear, transparent,  
refractile.

Inner endoplasm-finely granular  
(ground glass appearance), with

nucleus

food vacuoles

erythrocytes

leucocytes(occasionally)

tissue debris

◉ Pseudopodia

Fingerlike projections formed by sudden jerky movements of ectoplasm in one direction, followed by the streaming in of the whole endoplasm

- Typical amoeboid motility is a **Crawling or Gliding**
- Pseudopodia formation and motility are inhibited at low temperature

## Nucleus

It is spherical 4-6 $\mu$ m in size  
contains central karyosome, surrounded by  
clear halo and anchored to the nuclear  
membrane by fine radiating fibrils called the  
**Linin network**, giving a **cartwheel  
appearance**

Nuclear membrane is lined by a rim of  
chromatin distributed evenly as small  
granules

## PRECYSTIC STAGE

- ◉ Trophozoites undergo encystment in the lumen
- ◉ Before encystment, the trophozoites extrude its food vacuoles and become round or oval, 10-20 $\mu$ m in size-precyst
- ◉ Contains a large glycogen vacuole and two chromatid bars
- ◉ It then secretes a highly retractile cyst wall around it and become cyst

- ◉ Trophozoites from acute dysenteric stools often contain phagocytosed erythrocytes- diagnostic feature, these are not found in any other commensal intestinal amoeba
- ◉ These divided by binary fission in every 8 hours
- ◉ These are killed by drying, heat, and chemical sterilization
- ◉ Infections are not transmitted by these- destroyed in stomach and cannot initiate infection



# CYSTIC STAGE

- ◉ Spherical , 10-20 $\mu$ m
- ◉ 3 types of cyst

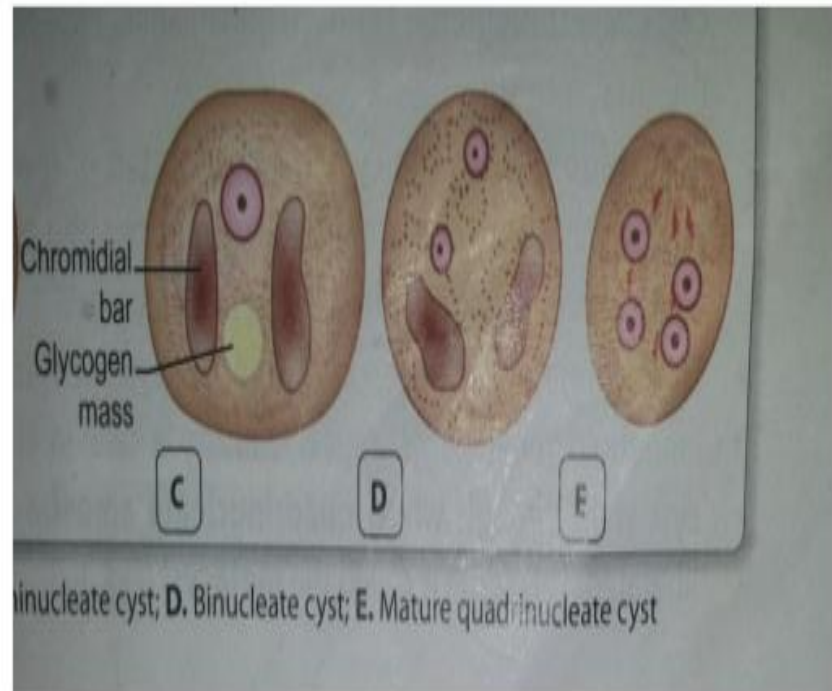
Early cyst

Binucleate cyst

mature quadrinucleate cyst

## Early cyst

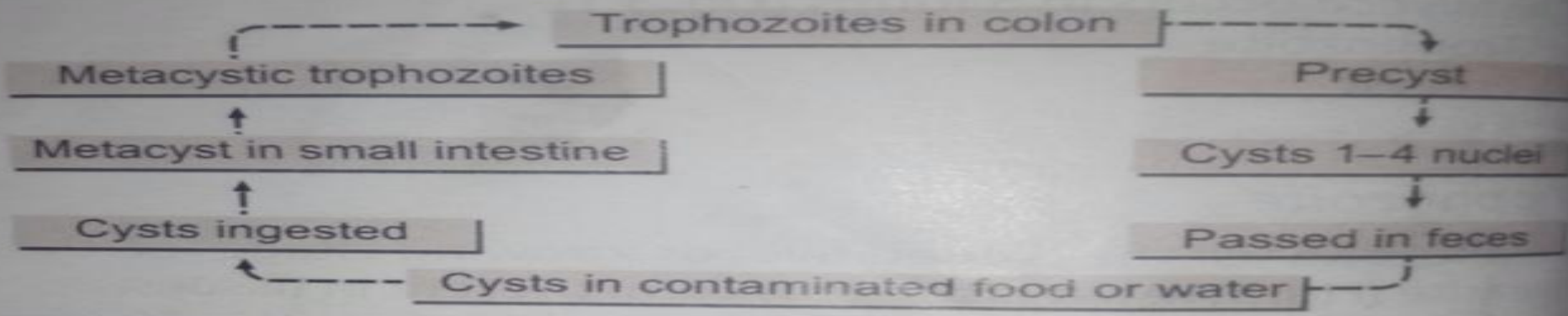
contains a single nucleus and two other structures-a mass of glycogen and 1-4 chromatid bodies or chromadial bars



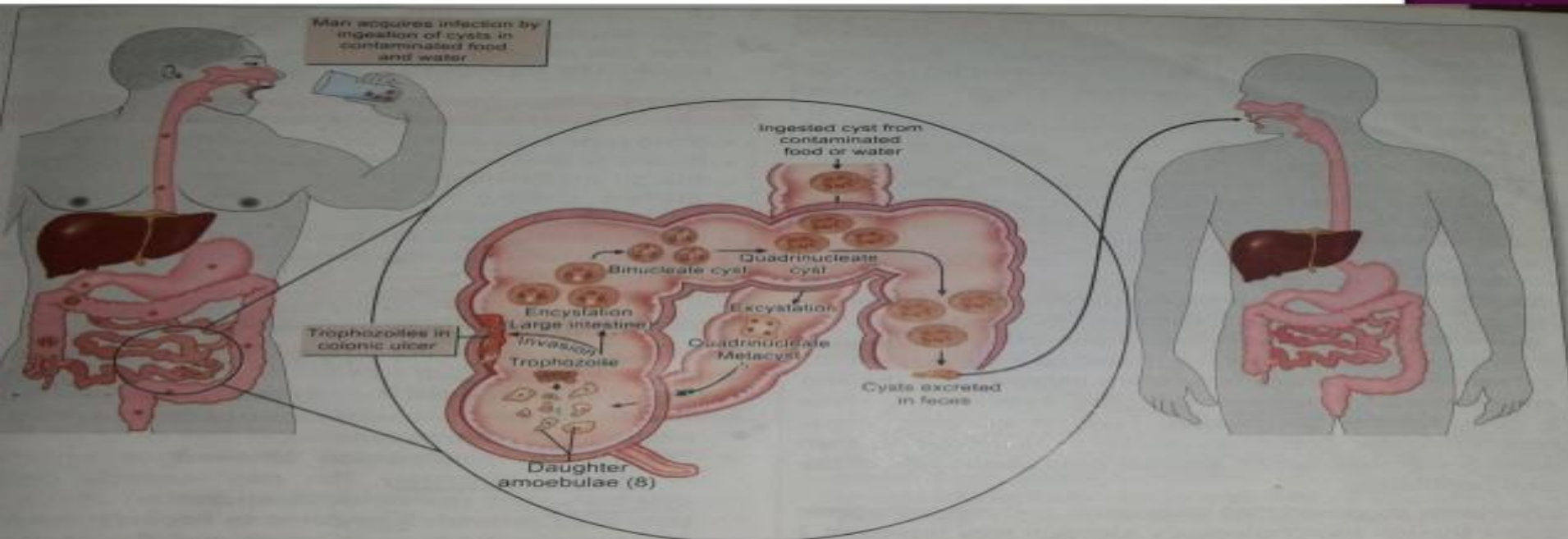
- ◉ As the cyst mature, the glycogen mass and chromidial bars disappear and the nucleus undergoes 2 successive mitotic divisions to form 2 and then 4 nuclei .
- ◉ The cyst wall is a highly resistant to gastric juice and unfavorable environmental conditions

## LIFE CYCLE

- ◉ **Infective form** :mature quadrinucleate cyst passed in feces of convalescents and carriers
- ◉ **Mode of transmission** :man acquires infection by swallowing food and water contaminated with cyst .
- ◉ Stomach -cyst wall is resistant to gastric juice
- ◉ Exystation :cyst reaches the caecum or lower part of ileum ,due to alkaline medium ,cyst wall damaged by trypsin ,leading to exystation



**Flowchart 3.1:** Life cycle of *Entamoeba histolytica* (Schematic)



**LIFE CYCLE OF ENTAMOEBA HISTOLYTICA**

# PATHOGENESIS AND CLINICAL FEATURES

- ◉ E.histolytica causes **intestinal** and **extra intestinal** amoebiasis

## Intestinal amoebiasis -PATHOGENESIS

*Lumen dwelling amoeba do not cause any illness .They causes disease only when they invade the intestinal tissues .*

**10 % -symptomatic**

**90% -asymptomatic**

- ◉ Not all strains of E.histolytica are pathogenic or invasive .
- ◉ Differentiation between pathogenic and non pathogenic strains can be made by
  - ✦ susceptibility to complement-mediated lysis
  - ✦ Phagocytic activity
  - ✦ by the use of genetic markers
  - ✦ monoclonal antibodies
  - ✦ Zymodeme analysis

- The metacystic trophozoites penetrate the columnar epithelial cells of **crypts of Lieberkuhn**
- Penetration is facilitated by
  - motility of the trophozoites
  - tissue lytic enzyme -**histolysin**
  - amoebic lectin** -mediate adherence
- Mucosal penetration by amoeba produce discrete ulcers with pinhead center and raised edges .
- Sometimes invasion remains superficial and heal spontaneously.
- More often, the amoeba penetrates to ~~sub~~ mucosal → layer and multiplies rapidly- lytic necrosis- abscess- ulcer

- ◉ Ulcer appear initially on the mucosa as raised nodules with pouting edges .
- ◉ They breakdown discharging brownish necrotic material contains large numbers of trophozoites.
- ◉ The typical amoebic ulcer -flask shaped
- ◉ Multiple ulcers may coalesce to form large necrotic lesions with ragged and undermined edges, covered with brownish slough

## **LESIONS IN CHRONIC INTESTINAL AMOEBIASIS**

- ◉ Small superficial ulcers involving only the mucosa
- ◉ Round or oval shaped with ragged and undermined margin and flask-shaped in cross section
- ◉ Marked scarring of intestinal wall with thinning ,dilatation ,and sacculatation
- ◉ Extensive adhesions with neighboring viscera
- ◉ Formation of tumor-like masses of granulation tissue amoeba

# HEPATIC AMOEBIASIS

- ⦿ Most common extra intestinal amoebiasis .
- ⦿ The history of amoebic dysentery is absent in more than 50% cases
- ⦿ Several patients with amoebic colitis develop an enlarged tender liver without detectable impairment of liver function or fever.
- ⦿ This acute hepatic involvement (amoebic hepatitis) may be due to repeated invasion by amoeba from an active colonic infection or to toxic substance from the colon reaching the liver.



# PULMONARY AMOEBIASIS

- It may occur by direct hematogenous spread from colon bypassing the liver, but it most often follows extension of hepatic abscess through the diaphragm
- Hepatobronchial fistula usually results with expectoration of chocolate brown sputum
- Patient presents with
  - severe pleuritic chest pain*
  - dyspnea*
  - non-productive cough*



**Fig. 3.3:** Intestinal amoebiasis: Specimen showing amoebic ulcer in colon

# METASTATIC AMOEBIASIS

- ◉ Involvement of distant organs is by hematogenous spread and through lymphatics
- ◉ Abscess in

*Kidney*

*Brain*

*Spleen*

*Adrenals*

Spread to brain leads to severe destruction of brain tissues and is fatal

THANK YOU





