SUBJECT: MEDICAL PARASITOLOGY

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TOPIC: PROTOZOOLOGY

SUB TOPIC: ENTAMOEBA HISTOLYTICA

Introduction

* The infection of E.histolytica is known as Amoebiasis.
* E. histolytica is the 3rd leading cause of mortality and morbidity due to parasitic in human after Malaria and Schistosomiasis.

Epidemiology & Geographical Distribution

* The disease is found in the developing countries where the following factors facilitate the spread it: i) Poverty

 ii) Ignorance

 iii) Mental retardation

 iv)Inadequate sanitation

 v) Poor personal hygiene

\* Amoebiasis is endemic in many parts of the tropical and sub-tropical Africa, Asia, South America China and India.

\* Invasive amoebiasis produces an estimated 40 million disabling and (40-110)000 people annually worldwide.

 Morphology

E. histolytica occurs in 3 forms namely:

1. Tophozoite form or vegetative form

2. Precystic form – before cyst formation

3. Cystic form – Parasite is enveloped in a cyst.

**1. Trophozoite stage**

\*As we drew\*

* This is an active stage and a parasite is likely to be seen in locomotion and engulfing food particles in wet mount microscopy.
* It is irregular in shape and size, the protoplasm is differentiated into ectoplasm and endoplasm (has nucleus with a karyosome, food vacuoles, granules and other organelles).
* The pseudopodia are present for locomotion.

**2. Precystic stage**

* Some trophozoites undergo encystment in the intestinal lumen.
* Encystment does not occur in the tissue but outside the body, before encystment the trophozoite extrudes.
* It’s food vacuoles becomes round or ovoid. It secretes a highly refractive cyst wall around it and becomes a cyst shown below.

\*Draw fig 1\*

**3. Cystic stage**

\* It is spherical in shape. The early stage has a single nucleus chromadial bodies and glycogen mass as shown below.

 \* Draw the fig 2\*

* As the cysts matures the glycogen mass and chromadial bars disappears. The nucleus undergoes two successive mitotic divisions to form four nuclei. When stained with iodine the glycogen mass appears golden brown.
* The chromadial bars appear as clear spaces being unstained.

 \*Draw fig 3 and fig 4\*

 Life cycle

* The E. histolytica exhibits a direct life cycle. i.e. one host where man is the definitive host.
* It is the only amoeba which causes the clinical symptoms. The infective stage is the mature cyst (the quadrinucleate)
* The transmission can be through ingesting of the contaminated food with the feces or stool, or drinking contaminated water or poor personal hygiene.
* Once the cysts are ingested they are passed to the stomach where they remain dormant because the conditions are not favorable (the PH is acidic) inhibit any development of E. histolytica of cysts.
* The trophozoites will be destroyed by the enzymes and the cyst will be passed into the small intestine where the media is alkaline.
* In the small intestine the cyst wall is damaged by the enzyme trypsin leading to excystation during this stage, cytoplasm gets detached from the cyst wall. The amoeboid movement will occur causing a tear in the cyst wall for the quadrinucleate amoeba to emerge.
* After this the metacystic form where the nuclear divide immediately to form eight nuclei each surrounded by a membrane containing cytoplasm. This will lead to perforation of metacystic trophozoite, which will move to small intestine to caecum or appendix of human beings.
* In the caecum are the optimum conditions which enable the trophozoite to divide through binary fission. Some develop into precystic form forming trophozoite others form cysts.
* Precystic form can either be removed together with the feces while others are destroyed by environmental conditions terminating the life cycle. They can go back to small intestine to lead a commensal life or become invasion in term of trophozoites.
* The cysts must be removed together with feces per life cycle to be repeated. If they go back to small intestine the cyst wall will be damaged and more trophozoite can be formed within the small intestine hence the transmission for a healthy person.

The summarized life cycle.

\*DRAW FIG 5 HERE\*

Factors influencing pathogenesis of amoebiasis

1. Strain variation: - only specific strain is pathogenic.
2. Bacteria: - enhances pathogenicity
3. Ineffective dose: -there is relationship between number of cysts ingested and development of diseases and thus dose should be considered.
4. Nutrition status amoebiasis worsens with vitamin c deficiency, high carbohydrates diet and high cholesterol intake. A high protein diet appears to be protective.
5. Associated diseases: -the co-existence of the diseases such as diabetes, tuberculosis, and malignancy lead to severe form of amoebiasis.
6. Pregnancy: - temporary immunosuppression during late pregnancy increases the severity of the disease.
7. Drugs: - corticosteroids, antimetabolites and other immune suppressants precipitate amoebiasis.
8. Immunity: - once amoebiasis is cured, immunity to this disease develops to varying period of time. In endemic areas people may develop little tolerance to the disease.
9. Intestinal mucus: - prevents the invasion by limiting the motility of the trophozoite thus preventing the contact with endothelial cells.
10. Dietary iron: - diets extremely low in iron may limit the growth of the trophozoite thus restricting the disease.

Pathology

It varies with the extent of involvement

* Causes superficial erosions
* Colonic mucosal ulcerations
* Rectal lesions and bleeding
* Abdominal cramps and pain
* Mild diarrhea to hemorrhage dysentery
* Amoebic dysentery
* Tenesmus ( sudden bowel evacuation) and abdominal tenderness
* Peritonitis due to perforation in severe amoebic colitis
* Urogenital infection

Diagnosis

* Individuals with intestinal E.histolytica fall into four groups:
1. Asymptomatic (AS) – don’t show symptoms
2. Acute amoebic colitis (AAC) –they suffer non-dysenteric colitis amoebic dysentery characterized by blood and mucus in the foul stools, cramping, abdominal pain and diarrhea alternating with constipation, liver may slightly enlarge, intestines may be perforated.
3. Fulminant amoebic colitis (FAC) –commonly occurs in children, profusely diarrhea blood and has mild abdominal pain.
4. Amoeboma (AM) –is a granuloma surrounding and amoebic mass seen on x-rays.

It is a severe complication development of amoebic hepatic abscess.

Amoebic trophozoites enter the liver through the portal vein.

The Manifestation includes the cough, fever that peaks in the afternoon, weight loss, anemia, and leuocytosis, tender and enlarged liver.

Prophylaxis (Prevention measures)

1. Adequate sanitation
2. Health education
3. Avoid eating contaminated food and vegetables grown by sewage irrigation
4. Avoid night soil cultivation

Treatment

* Diloxamide furoate
* Metronidazole
* Chloroquin
* Paramomycin
* Tetracycline
* Erythromycin
* Dihydro emetine
* Emetine hydrochloride