First we are going to discuss Protozoa.

**Entamoeba mainly Amoeba Histolytica**

- It exists as a **Trophozoite** *(functioning motile form)* AND it has a cyst stage.
- It is a member of Rozopods; it possesses pseudopodia.
  - Moves by pseudopodia.
- The **cytoplasm** itself is broken down to 2 areas
  - **Ectoplasm**: Outer rim which is **lighter in color**. It is concerned with **movement**; it sucks the cytoplasm into pseudopodia.
  - **Endoplasm**: more **granular**, concerned with **vegetative function of the amoeba**. It contains the nuclei and vacuoles which contain ingested material.
    - Ingested/engulfed material can include bacteria, debris, RBC’s.

- **Does NOT require an intermediate host.**
- **Fecal-Oral** Route of transmission.
- The Trophozoite does not survive outside the body in this form
  - It undergoes changes to become a cyst.
  - **Decreases in size** from 20-30 microns to 15 microns
  - Changes in the organelles of the Amoeba.
  - Result in **cysts that contain number of nuclei**, variable in each Amoeba species, **specifically 4 in Amoeba Histolytica**.
    - Also **develops hard protective layer instead of the Plasma Membrane**.
- **Chromatoidal bodies** in the cytoplasm of the Amoeba **contain DNA/RNA**.
  - This is only present during development and disappears with maturation.

- The cyst of the Amoeba passes through feces
  - If ingested, the **gastric juice helps disintegrate and release the 4 nuclei**.
  - These nuclei will **divide into 8** that will form a small **Amoebuli** which moves to the **large intestine** where they develop to fully grown amoeba.

- **Amoebae are usually free living in water**, most are harmless, but there are a few that are pathogenic.
  - Naegleria and Acanthamoebae are free living species that can sometimes cause disease.
- The ones that always exist in the human body include mainly 6 varieties
  - Entamoeba gingivalis in the oral cavity/ gingiva.
  - Most important is **Entamoeba Histolytica**, it is a pathogen that lyses cells.
- Names of other pathogens present in the slides. I could not catch them. These rarely cause disease and are not important causative agents of disease to be considered but one should familiarize oneself with their names: Entamoeba coli, Entamoeba endolimax, and Entamoeba fragilis.

- Comparison between Entamoeba coli and Entamoeba Hystolytica
  - Entamoeba Coli is an important commensal, whereby it exists in 30% of all people and is very common to be encountered.
  - Often confused upon stool sample examination which often leads to misdiagnosis.
  - Morphological Comparison
    1. Cyst of Hystolytica smaller than coli.
    2. Nuclei number is 8 in coli while 4 in Histolytica.
    3. The developing cyst of Histolytica has sausage shaped or cigar shaped chromatoidal bodies i.e. blunted edges.
       While the chromatoidal bodies of Entamoeba coli are shaped like splinters or shards of broken glass.
    4. Size of Trophozoite is the same in both.
    5. The Ectoplasm in Histolytica is more demarcated than the Entamoeba coli, thus the organism is quicker and is more directional, it also has large pseudopodia and a large ectoplasm.
       The Entamoeba Coli has blunted pseudopodia and moves very randomly.
    6. Entamoeba Coli’s vacuoles have debris and bacteria WHILE Histolytica is more likely to have RBC’s.
       In laboratory examination, presence of RBC’s in the vacuoles is definitive identification of Entamoeba Histolytica.
    7. Nucleus has a very distinctive karyosome which can be used for differentiation, i.e. Histolytica karyosome is central while that of Coli is more eccentric/peripheral.
    8. Distribution of chromatin in the inner rim of the nuclear membrane in Histolytica is even and thin while in coli it is variable i.e. alternations between bumpy/thick and thin areas.

- Laboratory Procedures
  - The Trophozoite can only be found in watery stool/diarrhea, it cannot be found in formed/solid stool. In solid stool, the cysts should be looked for.
  - It is best to examine fresh stool, which also should be examined at more than once.
  - The specimen is diluted with a drop of normal saline and placed under the microscope for detection of the presence of the Trophozoite.
  - The stool is best examined when warm, since this induces movement of the Trophozoite and is thus better for distinguishing it from thousands of other organisms in the stool.
If the Trophozoite is not found then a sample is stained with iodine which kills all Trophozoites which are thus not seen. But at same time the iodine highlights the cysts which are now very clear.

- Infection with Histolytica is quite common => 10 % of people.
- However, incidence of disease is very uncommon, since patients are usually asymptomatic but not technically carriers.
- This raises the question of whether all Histolytica strains are the same. Some have proven to be more pathogenic strains than others.
- This difference in pathogenicity can be attributed to many factors
  - Morphology is the same but some of them are more resistant to the compliment system than others.
  - Lytic effect can vary.
  - Can produce proteinases which destroy Antibodies, thus facilitating the spread.
  - Some produce amebopore which are proteins that produces pore in cells to lyse them.
- Entamoeba Histolytica is a proper pathogen while the rest are referred to as Amoeba Dyspar (strains that are not Pathological),
  - Entamoeba Dyspar have the same morphology, the only difference is in the activation of genes and thus pathogenicity.
- Entamoeba hartmanni is another non-pathogenic species of Entamoeba which is very similar to Histolytica but much smaller.
- If non-pathogenic Entamoeba is to be cultured in vitro then it has to be provided with bacteria, while Entamoeba Histolytica prefers axenic conditions (does not need the presence of bacteria and other microorganisms).

**Disease and Clinical Description**

- An uninfected person can ingest some cysts which might be unable to develop into trophozoites.
- An infection indicates the presence or establishment of Amoeba.
  - An infected person can either develop clinical features of infection i.e. disease or can become a carrier.
- The diseased / infected person with clinical manifestations experiences Amoebic dysentery i.e. bloody diarrhea with mucus that is foul smelling, not very watery, and in relatively small amounts. This can be accompanied by a fever.
- The carrier or infected person who did not develop clinical manifestations can later undergo activation of the amoeba and thus develops the disease.
  - The carrier can develop dysentery, mild symptoms, or remain asymptomatic
- Dysentery can lead to death or it can lead to a secondary infection elsewhere in the body such as a liver abscess. This is very serious and can be followed by death.
The infection is mainly luminal/pathology of wall of intestine, but if it crosses the barrier and gains access to the blood, then a liver abscess can develop.

- Pathology mainly involves the large intestine, whereby dysentery as well as ulceration occurs since the mucosa is invaded.
- The mucosa between the ulcers is normal, indicating that not all mucosa affected.
- Chronic affliction provides a clouded clinical picture, as it can be confused with ulcerative colitis.
  - A misdiagnosis of ulcerative colitis with the subsequent administration of steroids leads to suppression of immunity which only makes matters worse and facilitates the spread of disease.
- The bowel can become perforated if the infection persists and is left untreated, thus resulting in peritonitis and then death.
- There is usually an accumulation of inflammatory cells and a granuloma, this is called an Amoeboma.
- This is a space occupying lesion which can be suspected as cancer.
- Abscesses form mostly in the liver since blood of the large intestine passes to the portal circulation.
  - Abscesses can however occur in the lungs, where the abscess can rupture.
- Metronidazole is used to treat infection by Entamoeba Histolytica.
- Meningitis can occur through the blood along with Keratitis (infection of Cornea).
- Entamoeba Nigeleria commonly known can be inhaled with water through the respiratory tract and gain access to the brain via the Cribiform plate.

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