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Clinical Mycology and parasitology

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Course Title: Clinical mycology and parasitology **Instructor: Ms. Huma Imtiaz**

Time: 06 hours **Max Marks: 50**

Q1: Write down the life cycle of Enterobius vermicularis. (10)

ENTEROBIUS

Enterobius vermicularis (pinworm) is one among the foremost common human parasitic helminths, and youngsters are the foremost susceptible group. Several interactive and ecological factors may facilitate pinworm infection. According to a research the Republic of the Marshall Islands (RMI), the status of pinworm infections among children remains unknown.

Disease: Enterobius vermicularis causes pinWorm infection Enterobiasis

Life cycle:

• Adult in appendix and large intestine migrates to anus at night deposits eggs which are immediately infective. The eggs hatch within the intestine, where the larvae differentiate into adults and migrate to the colon.

• At night, the feminine migrates from the anus and releases thousands of fertilized eggs on the perianal skin and into the environment. The larvae within the egg become infective in approximately 4 hours. The movement of the feminine worm and therefore the eggs causes itching which makes it more likely for self-infection to occur.

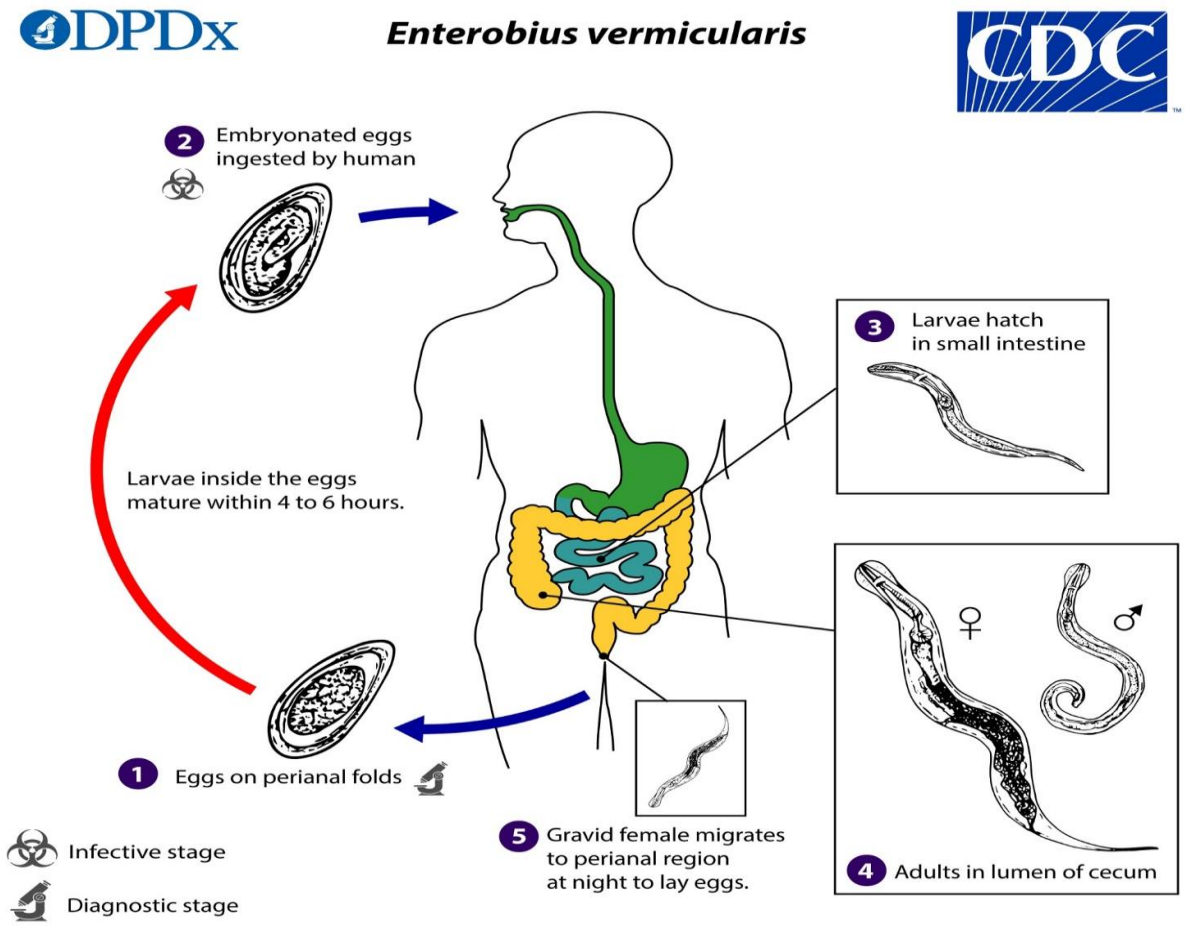
• Infection acquired from environment or autoinfection. Eggs die in 24-48 hours at temperature during a dry environment

• Within 6 hours, the eggs develop into embrocated eggs and become infectious

• Reinfection can occur if they are carried to the mouth by fingers after scratching the itching skin

• Infection occurs by ingesting infective eggs.

• Adults mature in the large intestine in about a month.



Pathogenesis & Clinical Findings

• Perianal pruritus is the most prominent symptom. It is an allergic reaction due to the presence of either the adult female or the eggs

• Scratching predisposes to secondary infection

Q2: Describe pathogenesis of Ascaris. (10)

Ascaris lumbricoides is that the largest nematode (roundworm) parasitizing the human intestine. It is an intestinal worm found in the small intestine of man. It is more common in children then in adult. As many as 500 to 5000 adult worms may inhabit one host.

Pathogenesis

There are two phase in Ascariasis

1. The blood-lung migration phase of the larvae

When the larva migrate through the lungs, it may cause a pneumonia. Their symptoms area low fever, cough, blood-tinged sputum, asthma. Large numbers of worms may give rise to allergic symptoms. Eosionophilia is generally present. These clinical manifestation is also called Loeffler's syndrome.

2. The intestinal phase of the adults.

The presence of a couple of adult worms within the lumen of the tiny intestine usually produces no symptoms, but may produce to vague abdominal pains or intermittent colic, especially in children. A heavy worm burden can result in malnutrition. More serious manifestations have been observed. Wandering adults may block the appendical lumen or the common common bile duct and even perforate the intestinal wall. The problems of ascariasis, like intestinal obstruction, appendicitis, biliary ascariasis, perforation of the intestine, cholecystitis, pancreatitis and peritonitis, etc., may occur, during which biliary ascariasis is that the most common complication.

Symptoms of Ascariasis

No symptoms Stage 1: worm larvae within the bowels attach to bowel walls

Stage 2: Involves worm larvae migrate into the lungs: Fever and breathing difficulty, Coughing and pneumonia

Stage 3: worms enter the tiny intestine and mature into worms and remain there to feed Abdominal symptoms, Abdominal discomfort, Intestinal blockage - could also be partial or complete, Partial intestinal blockage, Total intestinal blockage, Severe abdominal pain, Vomiting, Restlessness, Disturbed sleep, Worm in stool, Worm in vomit

Clinical features

Abdominal pain, diarrhea, vomiting and slight temperature. It blocks intestine and appendix. They may enter bile or duct and interfere with digestion. Injure the intestine and cause peritonitis. They produce toxins which irritate the mucosa of the gut, or prevent digestion of protein by host by destroying an enzyme trypsin. In children they cause stunted growth and makes the brain dull. Larvae causes inflammation and hemorrhage in the lungs which results in pneumonia – may prove fatal.

Q3: Explain the transmission and life cycle of Entamoeba histolytica in detail.

Entamoeba histolytica is an invasive, pathogenic protozoan, causing amoebiasis, and a crucial explanation for diarrhea in developing countries. To understand the epidemiology, it has intensely changed since this amoeba was distinguished from another morphologically similar one, Entamoeba dispar, a nonpathogenic and commensal parasite. Now the two species can be distinguished generally through molecular and immunological procedures.

Life cycle

E. histolytica passes its life cycle in just one host. - The established quadrinucleate cysts are the infective forms. These infective cyst are passed in the faeces of carriers. Infection caused to men by ingestion of water and food containing these cyst. - When the cyst reaches the caecum or the lower a part of the ileum, the excystation occurs (due to lysis of cyst wall by trypsin within the small intestine). During this process, each mature cyst liberates one amoeba with four nuclei, a tetranucleate amoeba which eventually produce eight metacystic trophozoites by the division of nuclei by binary fission. - The metacystic trophozoite ultimately occupy the submucous tissue of the massive intestine, their normal habitat. Here they grow and multiply by binary fission. During growth, E. histolytica secretes a protease which cause destruction and necrosis of tissue resulting in flask shaped ulcer. - Sometimes the trophozoite enter into deeper layer and should gain entry into deep layers into the radicals of hepatic portal vein to be over excited to liver producing amoebic liver abscess. When the effect of the parasite on the host is diluted and there's increase within the tolerance of the host, the lesion start healing. The trophozoites, within the lumen of huge intestine occurs encystation.

Transmission

Transmission of E. histolytica from man to man is effected through its encysted stage and infection occurs through the ingestion of those cyst. - Faecal contamination of drinking water, vegetables, and food are the primary causes. - Eating of uncooked vegetables and fruits which have been fertilised with infected human faeces has often lead to occurrence of disease. Pathogenicity - Incubation period: 4-5 days. - Clinical features or Symptomatology: The term amoebiasis is employed to denote all those condition which are produce within the human host by infection with E. histolytica. - Amoebic dysentry: may be a condition during which the infection is confined to the intestinal canal and is characterised by the passage of blood and mucus within the stool. Amoebic liver abscess is formed. It is the foremost common sort of hepatic amoebiasis and accounts for upto 10% of all intestinal amoebiasis. It may occur in any

part of liver but is generally confined to posteriosuperior surface of right lobe of liver.Clinical features of Amoebic Liver abscess: - Onset is insidious - Pain and tenderness in the right hypochondrium. - Shoulder pain due to the irritation of phrenic nerve - Fever. - Jaundice is an unusual manifestation. - The patient becomes emarciated (excessively thin) Metastatic lesions in other organs: The various extra hepatic amoebiasis include: - Pulmonary Amoebiasis - Cerebral Amoebiasis - Cutaneous Amoebiasis - Splenic Amoebiasis - Vaginal and Penile Amoebiasis.

Q4: How will you diagnose Trypanosoma Cruzi inside a laboratory?

Trypanosoma

It is a kind of kinetoplastids (class Kinetoplastida), a monophyletic group of unicellular parasitic flagellate protozoa. The name springs from the Greek trypano- (borer) and soma (body) due to their corkscrew-like motion. All trypanosomes are heteroxenous (requiring quite one obligatory host to finish life cycle) and most are transmitted via a vector. Most of the species are spread by blood-feeding invertebrates, but there are different mechanisms among the changing species.

The genus Trypanosoma includes three major pathogens:

- Trypanosoma cruzi
- Trypanosoma gambiense
- Trypanosoma rhodesiense

Trypanosoma cruzi

Disease

The etiological agent of Chagas' disease is that the intracellular protozoan parasite Trypanosoma cruzi (T. cruzi), which is spread by the insect vector Triatoma infestans Triatoma infestans (Reduviid bug) (reduviid bug). Reduviid bugs sleep in mud filled walls of huts in rural areas. Trypanosoma cruzi is spread when the bug bites human hosts and spreads the parasite with human erythrocytes

Clinical Findings

The severe stage of Chagas' disease contains of facial edema and a nodule (chagoma) near the bite, comprising fever, lymphadenopathy, and hepatosplenomegaly

The severe phase resolves in about 2 months. Most entities then remain asymptomatic, but some reach the chronic form with myocarditis and megacolon. Death from chronic Chagas' disease is naturally thanks to cardiac arrhythmias and failure.

Laboratory Diagnosis

Severe disease is diagnosed by indicating the presence of trypomastigotes in thick or thin films of the patient's blood. Both stained and wet preparations should be examined, the latter for motile organisms

Because the trypomastigotes aren't numerous within the blood, other diagnostic methods could also be required, namely, (1) a stained preparation of a bone marrow aspirate or muscle biopsy specimen (which may disclose amastigotes); (2) philosophy of the organism on special medium.

Q5: Enlist Leishmania species names. Summarize the clinical findings of all species of Leishmania.

Leishmania is a parasitic disease spread introduction by the bite of infected sand flies. There are several different forms of leishmania. The most common are cutaneous and visceral. The cutaneous type causes skin sores. The visceral type affects internal organs like the spleen, liver and bone marrow. People with this type usually have fever, weight loss and an enlarged spleen and liver.

Leishmania is found in parts of about 88 countries. Most of those countries are within the tropics and subtropics. It is possible but very unlikely that you would get this disease in the United States. But you should be aware of it if you are traveling to the Middle East or parts of Central America, South America, Asia, Africa or southern Europe.

The genus Leishmania includes four major pathogens:

• Leishmania donovani

• Leishmania tropica

• Leishmania mexicana

• Leishmania braziliensis

Disease: kala-azar (visceral leishmaniasis)

Pathogenesis

In visceral leishmaniasis, the organs of the reticuloendothelial system (liver, spleen, and bone marrow) are the most severely affected?

Reduced bone marrow activity, including cellular destruction within the spleen, leads to anemia, leukopenia, and thrombocytopenia

This leads to secondary infections and a tendency to bleed

The striking enlargement of the spleen is due to a combination of proliferating macrophages and sequestered blood cells

Clinical Findings

It Symptoms begins with intermittent fever, weakness, weight loss and massive enlargement of the spleen in the body. Hyperpigmentation of the skin is seen in light-skinned patients (kala-azar means black sickness). The time period of the disease runs for months to years.

Initially, patients feel reasonably well despite persistent fever. As anemia, leukopenia, and thrombocytopenia become more profound, weakness, infection, and gastrointestinal bleeding occur. Untreated severe disease is almost always fatal as a results of secondary infection

Laboratory Diagnosis

Diagnosis is typically made by detecting amastigotes during a bone marrow, spleen, or lymph gland biopsy preparation

Leishmania tropica, Leishmania mexicana, & Leishmania braziliensis

Disease

Infection with Leishmania species may result in 3 main sorts of disease counting on the species, geographical area and host immune reaction. Leishmania donovani produces visceral leishmaniasis (kala-azar).

Leishmania tropica and L. mexicana produce cutaneous leishmaniasis which is characterized by skin lesions (oriental sore). Infected macrophages containing amastigotes are found primarily at the location of infection round the sores. The sores are chracterized by an elevated rim encircling the lesion.

Leishmania braziliensis produces mucocutaneous leishmaniasis, characterized by lesions near mucosal membranes. The intitial site if infection may be a small red papule that ulcerates during a few weeks. The lesions are flat (no raised rim) and sometimes oozing. Infections of the ear, nose and mouth area cause degeneration of the cartilage and soft tissues, leading to disfigurement.

Clinical Findings

Symptoms include fever (often 2 fever spikes per day), enlargement of the spleen and liver, weakness, and progressive emaciation. The disease is usually fatal without treatment, but survivors often develop immunity.

The initial lesion of cutaneous leishmaniasis may be a red papule at the bite site, usually on an exposed extremity. This enlarges slowly to make multiple satellite nodules that coalesce and ulcerate. There is usually a single lesion that heals spontaneously in patients with a competent immune system. However, in certain individuals, if cell-mediated immunity doesn't develop, the lesions can spread to involve large areas of skin and contain enormous numbers of organisms

Mucocutaneous leishmaniasis begins with a papule at the bite site, on the opposite hand metastatic lesions form, usually at the mucocutaneous junction of the nose and mouth. Ulcerating lesions destroy nasal cartilage but not adjacent bone. These lesions heal slowly