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**Course ……………………….... Clinical mycology and parasitology**

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**Q1: Write down the life cycle of Enterobius vermicularis.**

**Disease**

Enterobius vermicularis cause pinworm infection enterobiasis.

**Life cycle**

The life cycle is confined to human.

The infection is acquired by ingesting worm eggs.

The eggs hatch in the small intestine where the larvae differentiate into adults and migrate to the colon.

The adult male and female worm live in the colon, where mating occurs.

At night, the female migration form the anus and release thousands of fertilized eggs on the perianal skin and into the environment.

Within 6 hours the eggs develop into embryonated eggs and become infection.

Reinfection can occur if they are carried to the mouth by finger after scratching the itching skin.

**Q2) Describe pathogenesis of Ascaris**.

**Pathogenesis.**

The major damage occurs during larval migration rather than from the presence of the adult worm in the intestine.

The principal site of tissue reaction are the lungs where inflammation with an eosinphillic exudate occur in response to larval antigens.

Because the adults derive their nourishment from ingested food a heavy worm burden may contribute to malnutrition especially in children in developing countries.

Most infection are asymptomatic.

Ascaris pneumonia with fever cough and eosinophilia can occur with a heavy larval burden.

Abdominal pain and even obstruction can results from the presence of adult’s worm in the intestine.

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

**Transmission**

Feco-oral route, via the ingestion of contaminated food or water containing mature quadrinucleate cyst of Entamoeba histolytica. Trophozoites if ingested would not survive exposure to the gastric environment.

**Life cycle**

Infection by Entamoeba histolytica happens by the ingestion of mature quadrinucleate cysts in fecally contaminated food, water, or hands. The quadrinucleate cyst is resilient to the gastric environment and passes unaltered through the stomach.

When the cyst of E.histolytica reaches caecum or lower part of ileum excystation occurs and an amoeba with four nuclei emerges and that splits by binary fission to procedure eight trophozoites.

Trophozoites migrate to the large intestine and lodge into the submucosal tissue.

Trophozoites grow and multiply by binary fission in the large intestine (Trophozoite phase of the life cycle is responsible for producing characteristics lesion of amoebiasis).

Certain numbers of trophozoites are discharged into the lumen of the bowel and are transformed into cystic forms.

The cysts thus made are unable to develop in the unchanged host and therefore require a transfer to another susceptible host. The cysts are passed in the feces.

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

**Disease**

T.cruzi is the cause of chagas diseases (American trypansomiasis).

**Clinical finding**

The acute phase of chagas disease consists of facial edema and a nodule (chagoma) near the bite couple with fever lymphadenopathy, and hepatosplenmegaly.

The acute phase resolve about in two months.

Death from chronic chagas disease is usually due to cardiac arrhythmias and failure.

**Laboratory Studies**

During the acute phase of trypanosomiasis, the CBC count may reveal leukocytosis with relative lymphocytosis, and transaminase levels are often elevated.

During acute illness (i.e., the first 6-12 wk.), parasites are frequently seen in blood smears [34], using either Giemsa staining or direct wet-mount preparations (in which motile parasites may be seen). Sensitivity is 80-90%.

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

There are four major species of leishmania.

* Leishmania donovani
* Leishmania tropica
* Leishmania Mexicana
* Leishmania braziliensis

**Leishmania donovani clinical finding**

* Moderate fever, weakness, and weight loss
* Hyper pigmentation
* Enlargement of spleen
* Anemia, leukopenia
* Thrombocytopenia
* If the disease left untreated it will be fetal.

**Leishmania tropica, Leishmania Mexicana, & Leishmania braziliensis clinical finding**

The initial lesion of cutaneous leishmaniasis is a red papule at the bite site, usually on an exposed extremity.

This papule enlarge to from multiple satellite nodules that coalesce and ulcerate.

An immune competent patient the lesion by itself.

However an immune compressive patient the lesion spread to other area of the skin.

Ulcerating lesions destroy nasal cartilage but not adjacent bone

These lesions heal slowly.