Sheet no.26

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**Toxoplasma**:

The primary host of the toxoplasma is a member of Felidae family mainly cats , so in the cat there is sexual multiplication , what we have actually male and female gametocytes they fuse together within the tissue of the cat and they get rise to an egg or something like that known as **oocyst** , and the oocyst contain 8 sporozoids , so really the oocyst that actually are form sexually inside the cat they come out in the feces and each oocyst contains 8 sporozoids ,( in fact it has 2 sporocyst and each sporocyst has 4 sporozoids)

these oocyst that are present in the feces if they are eaten by intermediate host which is in this case a mouse or a rat , these sporozoids will be released and will attack the cells so they knows cytocells , they don’t discriminate they can infect any cell in the mouse , and can infect all kind of host , so its not specific , some parasite has specific to certain animals , and some have specialized for certain tissues , and other infect only certain cells for ex. : malaria will only infect red blood cells

so what happen now , these sporozoids when grow inside the cell they will start dividing quickly and actively and known as **trabozoids** inside the cell of the tissue of the intermediate host and because they divide very quickly and actively we call them **tachyzoids**, so these tachyzoids will divide a lot within the cell by mesoendodiging!!! , and eventually the host cell will be full of these and it will burst and it will be releasing more tachyzoids to infect other cells within the same animal .

now at certain stage these tachyzoids they can erupt the cell , but then they stop dividing so the cell does not burst and these tachyzoids will become dormant because they become lazy and now we call them **bradyzoids** , so we end up with a cell still intact has not burst but this full of trabozoids, but these trabozoids are really quiescent and dormant and not actively divided they known as bradyzoids and this we call it a **cyst** , these a cyst so don’t confuse the oocyst and cyst , now these cyst are present in the flesh of the intermediate host , so if another cat comes and eats the intermediate host and these cyst will release the bradyzoids and bradyzoids now become active and they will changing to male and female gametocytes and these will fuse together to produce a zygote and the zygote will divide and will produce oocyst , so now we have complete the life cycle of the toxoplasma.

so these the normal cycle , now humans are accidentally host and they are intermediate host , but they have dead end host because usually infection is not probably transmitted to another cats , actually infection you get it in two ways either by eating oocyst or cyst , oocyst if you eat anything that has been contaminated by cat feces for ex.: kid play with the sand and the sand has been contaminated by cat feces or farmer when he implant some vegetables and the vegetables contaminated from cat feces , so anything that contain contamination from cat feces will have oocyst , also it can get the infection by eating the cyst that are in the flesh of another intermediate host like : mice , rats , pigs , cows and sheep ,and if you eat meet which is not cooked probably you will eat the cyst that contain bradyzoids and these bradyzoids will infect your tissue , if you eat something contaminated by cat feces you will eat oocyst and the oocyst will give rise for infection in your tissues.

these actually is very common infection , infection toxoplasma is very common in some communities it can be up 70% to 80% of the people are infected by toxoplasma , any were you have cats you are likely to have toxoplasmosis , so the infection is common but the disease is rare , , usually if you infected with toxoplasma it possible of some people infected already by toxoplasma , and you can find that by taking blood sample and you look for Abs against toxoplasma , it's difficult to look for the cyst in your tissues because we don’t know where they are , so it's easy to take a sample of blood and you look for the Abs against the toxoplasma Ag .

now the problem of toxoplasmosis can occur during pregnancy because toxoplasma they can actually pass from the mother to the fetus and this can create problems , so if woman becomes pregnant and she becomes infected with toxoplasma during that pregnancy, so the toxoplasma will go to the fetus and this actually can lead to abortion if its occur in the 1st trimester of pregnancy , the later the pregnancy (2nd and 3rd trimesters) the baby is born not aborted , but daily these babies will suffer eventually with toxoplasmosis and this is known as **congenital toxoplasmosis** , it's affect the nervous system, so can have defects on motor and sensory origins and also they can have mental retardation and usually they suffer with problems with the retina because cyst that occur in the retina they can cause blindness were these probably partially blindness not necessary blindness only partially blindness , so these are really the main problem with congenital toxoplasmosis , and this happens only when the mother become infected for the first time during pregnancy, now if one mother has been unfortunately to have such a case with congenital toxoplasmosis and she gets pregnant again, the baby will not get infected because she become immune , if a women used to play with cats when she was little and she become infected with toxoplasma and then she get married and she has 10 babies none of them will be affected , the proplem occurs only if the woman becomes infected with toxoplasma in 1st time during pregnancy.

so if we suspect that the babies have congenital toxoplasmosis we test them by serology by taking blood sample and we look for Abs against toxoplasma and these Abs can be IgM or IgG , if we find that the baby has only IgG that does not mean anything because if he was infected during pregnancy he does not have time to develop IgG and this IgG it most likely come from the mother because IgG cross the placenta , and if the baby had IgM against toxoplasma Ag it means that infection must have occurred in utero where the IgM must come from the baby it self , because IgM dose not cross the placenta.

and another thing as well if it occur in immunocompremised people for example HIV and then they become infected with toxoplasma again there is some complex problems, they will have neurological deficient , and they will have a problem with their eyes and the retina as well ,

if the patient has toxoplasma once in his life and cured then become immunocompremised the disease become activated , so immunocompremised people can suffer either if they acquired infection for the 1st time after the beginning immunocompremised or immunocompremisation can reactivate the latent infection that have been there for years .

Note : no way to transmit the disease from one person to another unless through eating , and most of the causes of toxoplasmosis is because of eating raw meat or playing with cats .

**Trypanosomatidae:**

these are hemo flagellates, they and they are really tissue parasite as the toxoplasma.

-They are divide in to two groups : 1.**Leishmania group** 2.**Tryposanoma or trypanozomes**

and these are dimorphic because they exists in two morphological shapes, one existing in the primary host and one existing in the intermediate host .

**Morpholgy of Leshmania consists of two forms:\***

**1-Amastigote**: intracellular form of the organisms, in primary host (human macrophage) and they are round cells which contain nucleus in the center ,and there's small amount of RNA and DNA known as kinetoblast (nucleic acid which is present at the base of the flagella) , and there are flagella: 3-4 microns in diameter and they exist inside the macrophages.

-Amastigote called Donovan body because the one who discovered them called Donovan.

**2-Promastigote:** (elongated 20 or 30 microns, has nucleus and has flagella extended from the anterior aspect of that organism, and at the base of the flagella there is kinetoblast)

-Primary host: human >> Amastigote

-Secondary host: sand fly >> Promastigote

-Sand fly found in Jordan, Iraq, Syria and worldwide and this one which transmits the disease.

This fly is a blood sucking fly, it bites the person and it will inject Promastigote and these Promastigote will be taken up by macrophages and they become Amastigote inside the macrophage.

you will find swelling and indurations in the place of the bite , then will ulcerate and the ulcer will go on for many months and eventually it will heal but it will leave a nasty looking scar at the site of bite. This scar called Bagdad boil (دمل البغداد), takes about year until it heal and ones it heal the person will have solid immunity against it.

The scars of sand fly bites usually in exposed areas of skin like face and arms and give unpleasant appearance this known as **1-Cutaneous Leishmaniasis** its confined to the site of bite and it has chronic course and in 1 year will heal

-other type of disease is called **2-Diffuse cutaneous Leishmaniasis** happen when cutaneous Leishmaniasis diffuses to other parts of the body and other part of skins will be involved.

It happen depending on the immune response of the patient. Usually you will get cell-mediated immunity against Leshmania parasites and that’s why it confined to that's area and the person get the boil because cells like macrophages and T-lymphocytes are coming there and keep the infection in one place and eventually will heal.

-With diffuse cutaneousLeishmaniasis: the patient actually produced humeral immune response, means produce lots of antibodies against the Leishmania parasites, but they are not really affected because the antibodies will not go inside the cells But cell mediated immunity kill the cells by cytotoxic T-cells and that’s why it spread.

-The main cells that affected by Leshmania are macrophages.

-The doctor said we should only know that there are many species of Leshmania but we should not know each one of them, only that each one is produce the cell disease.

-They like cold weather, can exist only on skin temperature(around 4 degrees or below at cold body temperature) Other species of Leshmania can live inside the body (macrophages) not only on the skin.

-Macrophages are found in the liver, spleen, bone marrow and will cause systemic(serious) disease called **3-** **Visceral Leishmaniasis** (splenomegaly, hepatomegaly, bone marrow and lymph nodes involve as will).

-Patient become ill, emaciated, have fever .and this disease if not treated will kill the patient within one year.

-Skin becomes pigmented and the patient will start looking dark that's why it called **black sickness** and in India it called **Kala-azar**.

-Treatment by drugs.

-These species of Leshmania that cause Visceral Leshmania are resisting to lysosomal activities of macrophage and they are only killed by activation.

**4th** type is confined to certain places : old-world lieshmania ( in Asia and Africa ) , new lieshmania like Brazelian , maxicana …..

Some of these especially the Brazilian can cause **mucocutanouse lieshmania** : the fly bite person and he will get lesion and indurations and in weeks or months disappear and patient forgot about it but after a year or two there is reactivation and this mean that some lieshmania did not disappear and remain in patient and once they are reactivated it involve mucocutaneous junctions and get **mucocutanouse lieshmaniasis**

It's very destructive lesion in nose, hard and soft palate so they suffer from naso-oral cavity problems and these people tend to inhale all this infected secretions and get aspiration pneumonia and it can be lethal because of pulmonary complications.

**Diagnose**: biopsy from tissues involved: skin (in Cutaneous Leishmaniasis) , bone marrow ( in Visceral Leishmaniasis)and look for Amastigote in macrophages

Usually its sporadic cases not endemic

and leishmania has a reservoir which are dogs and الجرابيع في الصحراء

**2-Tryposanoma:**

They are also dimorphic:

Trypasmastigote: in primary host (humans)

Epimastigote : in intermediate host ( Tse Tse fly )

Difference between them is that the Trypasmastigote have granules and more angulating membrane and the kinetoblast is posterior to the nucleus and in Epimastigote its anterior to the nucleus.

This disease present in South Africa and it has 2 sides east and west

T.Rhodesiense in the east cause east African disease (more sever) and T.Gambiense in the west

Both very similar in morphology, intermediate host, disease is the same but the Easton is more serious than western and can infect animals.

The disease is the sleeping sickness (**Tryposanomasis)** , life cycle : fly bite animal or human then Epimastigote become Trypasmastigote and go to the blood and lymph nodes and produce initial or acute phase of the disease : fever, headache, not feeling well and it can take few months then the Trypasmastigote can invade CNS and can cross BBB and once it reach the CSF >> terminal stage of the disease :Patient can't do anything dose not eat or talk or move and always want to sleep , once this happen the patient will die

Diagnose: blood smear or CSF and look for Trypasmastigote.

Treatment successful before reaching terminal stage of the disease.

This is the African Tryposanoma , there is American Tryposanoma >> next lec