2- Leishmaniasis

It is flagellated unicellular trophozoite which is intracellular living . It Transferred to human by female sandy fly phlebotomine vector . It infect animal like rodents and canine while human is incidental reservoir . It has three species :

1- Visceral leishamniasis . 2- cutaneous leishmaniasis . 3- Mucosal leishmaniasis.

Life cycle

Infected sandfly carry flagellated promastigote ,when bite animal or incidentally human the promastigote enter the blood taken by neutrophils liberating to the blood after apoptosis of neutrophil to be engulfed by macrophage where losing flagella to transform to amastigote then it multiply in it to liberate to circulation by lysis of the macrophage and amastigote reenter new macrophage and the circles continues , if the sandfly bite human it take the amastigote which transformed to flagellated promastigote where it multiply by binary division in the gut of it and migrate to proboscis of sandfly to infect human again . Sandfly live in hot and humid area of the world usually in Africa , America and Asia including Iraq (usually rural area around Baghdad and other areas) , sand fly lived in cracks of mud and straw houses and lay eggs in organic matter ,bite at night usually animals then to human so it is zoonotic disease .

Visceral leishmaniasis (Kala –azar)

It is caused by Leishmania(L) donovani complex (include L. donovani , L. infantum and L. chagasi) . It transmitted to human by sandfly , blood transfusion and detected unexpectedly in immunosuppressed patients like renal transplant and HIV patients . The main target of organ infected are spleen ,liver ,bone marrow and lymph nodes .

Clinical features

The disease usually infect children and infant and less commonly adults where common infection of adult in HIV patients . The incubation period from weeks to months and less commonly for days . The patient complains of fever and chills which decrease over weeks or months relapse of fever may occur but with less intensity , the patient will has splenomegally as the disease progress to be massive , moderate hepatomegally , lymphadenopathy , black discoloration of the skin for which it has its name Kala- Azar (in India mean black fever) . In advance disease pancytopenia , moderate to severe anemia develop rapidly and progress to congestive cardiac failure , the patient will develop thrombocytopenia associated with hepatic dysfunction may result in bleeding in retina ,GIT and nose , hypoalbuminemia may

occur lead to pedal edema ,as cites and anasarca . Immunosuppression may lead to secondary infection like tuberculosis, pneumonia , sever amebic dysentery ,herpes zoster …etc . If patient not properly treated may die .

Investigations

1- Haematological test shows pancytopenia ,agranulocytopenia and monocytosis . Hypergammaglobulinemia first IgG then IgM . Hypoalbuminaemia at late stage .
2- Demonstration of amastigoate bodies (Leishman Donovan bodies (LD. Bodies)) by bone marrow and lymph node aspirate with Giemsa stain ,splenic aspirate can give diagnosis of 98% after staining but it carry risk of hemorrhage from spleen so it need fully experience hand . Demonstration of amastigote can be by Buffy coat smear which has high sensitivity especially in immunocompromised patient .Another way by culture the aspirated material . PCR for detecting the DNA of parasite and its species .
3- Immunological test : ELISA . Direct agglutination test(Anti – Leishmania Ab test) and immunochromatography k39 strip test can diagnose infection but it remain positive months after cure the patients so it not differentiate between new and past infection .(most common is IFAT for Kala –azar)

Differential diagnosis : The disease similar to malaria ,typhoid ,tuberculosis ,schistomiasis and malignancy .

Management

1- Pentavalent antimony : Sodium stibogluonate drug is the mainstay treatment of leishmaniasis it given in dose of 20 mg|kg for 28 -30 days its side effects are arthralagia , myalgia ,raised hepatic transaminase , pancreatitis ,cardiotoxicity like t-wave inversion and cardiac arrhythmias .
2- Amphotericine B : It given as infusion once daily in dose of 0.75 -1 mg |kg for 15 -20 days it given when there is resistance to sodium stibogluconate it give cure rate nearly 100 % . Its side effects are fever ,rigor ,thrombophlebitis , diarrhea ,vomiting ,hepatic and renal toxicity ,hypokalemia and thrombocytopenia . Liposomal Amphotericine B is new generation and less toxic drug .
3- Oral miltefosine : It is given in dose of 2.5 mg |kg for 28 days . Its side effects include vomiting ,diarrhea ,skin allergy and it is teratogenic
4- Paromomycin : It is given in dose of 11 mg |kg for 3 weeks although it is aminoglycoside but it has no nephrotoxicity or audio toxicity .

The treatment of leishmaniasis either by single drug or multi-drug regimen like one dose of Amphotericine B then 7 days of miltefosine and there are another regimen . The response to treatment will be by the patient feel of well being ,fever subside the spleen regress and blood study improved .

HIV- visceral leishmaniasis : Patients with HIV has 100-1000 chance of getting visceral leishmaniasis . Abnormal sites of infection may occur like tonsillitis ,pericardial and pleural effusion . Amastigote may found in pleural ,pericardial or brochoalveolar lavage .Best treatment is by Amphotericine B . Prevention after treatment by monthly liposomal Amphotericine B infusion .

Post Kala – azar dermal leishmaniasis may occur in 6 months – 3 years after treatment where micronodular rash ,or papular rash treatment is the same for few months and has high cure rate .

Prevention and control :

No vaccine till now is available . Eradication of sandfly with insecticide and use of insecticide nets at sleep .

Cutaneous leishmaniasis : Caused by L. Major ,L. tropica and L. aethiopica . it caused nodule with elevated border ,ulcerated later ,healed by fibrosis . Diagnosed by slit smear of the lesion with Giemsa stain under microscopy study for parasitological diagnosis treated by local paramomycin and local stibgluconate drugs .

cutaneous leishmaniasis : Caused by L. brasiliensis complex infection it infect mucosa of nose ,mouth extending from cutaneous leishmaniasis or it infect pharynx ,fauces and larynx if it extend to bronchus it will be serious .It start as mucosal thickening then ulceration with tissue destruction later . Diagnosed smear stain from the lesion with Giemsa stain and microscopically exam ,if the lesion is remote diagnosis by leishmania skin test which shows the reaction and induration of the test .Treatment by 28 days of systemic stibogluconate , in refractory cases Amphotericin B is used .

Prevention by eradication of sandfly and usage insecticide nets at sleep in endemic places .