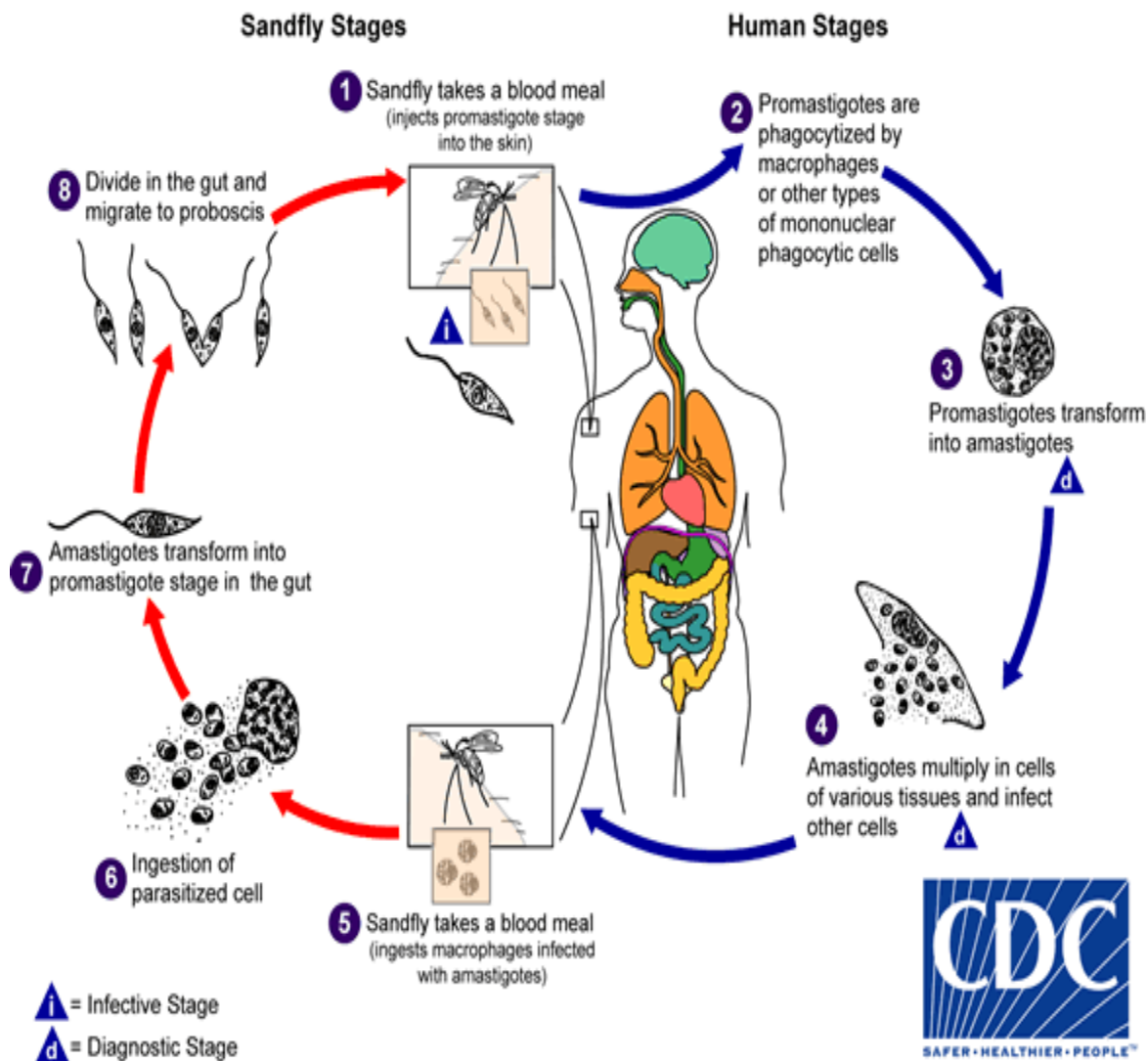


Visceral Leishmaniasis (KalaAzar –black fever)

It is zoonotic disease in which dogs and other carnivores are the reservoir, and the human is considered as an incidental host for parasite .The causative agent is *Leishmania donovani* from *Leishmania* species. It is disseminated hematogenously and infects the reticuloendothelial system. *Leishmania* has two morphological forms promastigote (flagellated) and amastigote (aflagellated) form.

Life cycle:



Leishmaniasis is transmitted by the bite of infected female phlebotomine sandflies. The sandflies inject the infective stage (i.e., promastigotes) from their proboscis during blood meals. Promastigotes that reach the puncture wound are phagocytized by macrophages and other types of mononuclear phagocytic cells. Promastigotes transform in these cells into the tissue stage of the parasite (i.e., amastigotes), which multiply by simple division and proceed to infect other mononuclear phagocytic cells. Sandflies become infected by ingesting infected cells during blood meals. In sandflies, amastigotes transform into promastigotes, develop in the gut, and migrate to the proboscis.

Clinical presentation:

- 1) **Subclinical** (seropositive Montenegro skin test): subclinical infection occurs more frequently than does active cutaneous or visceral disease. In child, there is fever which is irregular and not associated with toxemia (the child plays and does not look ill). IP 2-6 months
- 2) **Oligosymptomatic** Children have mild constitutional symptoms (malaise, intermittent diarrhea, poor activity tolerance) and intermittent fever; most will have a mildly enlarged liver. In most of these children the illness will resolve without therapy, but in approximately 25% it will evolve to active kalaazar within 2-8 mo. During the first few wk. to months of disease evolution the fever is intermittent, there is weakness and loss of energy and the spleen begins to enlarge (about 1 inch per month).
- 3) **Symptomatic(active) disease:** classic clinical features of high fever, marked splenomegaly, hepatomegaly, and severe cachexia typically develop approximately 6 mo. after the onset of the illness, but a rapid clinical course over 1 mo. has been noted in up to 20% of patients in some series .
- 4) **At the terminal stages** of kala-azar the hepatosplenomegaly is massive, there is gross wasting, the pancytopenia is profound, and jaundice, edema, and ascites may be present. Anemia may be severe enough to precipitate heart failure. Bleeding episodes, especially epistaxis, are frequent. The late stage of the illness is often complicated by secondary bacterial infections, which frequently are a cause of death.

*A younger age at the time of infection and underlying malnutrition may be risk factors for the development and more rapid evolution of active VL. Death occurs in more than 90% of patients without specific antileishmanial treatment.

5) Post-kala-azar dermal leishmaniasis A small percentage of patients previously treated for VL develop diffuse skin lesions, These lesions may appear during or shortly after therapy or up to several years later . The lesions of post-kalaazar dermal leishmaniasis are hypopigmented, erythematous, or nodular and commonly involve the face(around chin and nose) and trunk. They may persist for several months or for many years.it is thought to be caused by treatment and complete eradication of the parasite in the circulation resulting in its escaping to the tissue and causing this reaction.

Differential Dx. of Visceral Leishmaniasis :

1. Malaria
2. Military tuberculosis
3. Brucellosis
4. Typhoid fever
5. Amebic liver abscess
6. Infectious mononucleosis
7. Lymphoma and leukemia.
8. Schistosomiasis.

Investigations

- I. CBP: pancytopenia, i.e. severe anemia, leucopenia, & thrombocytopenia.
- II. LFT; ↑ liver enzymes. Hyper gammaglobinemia
- III. Serology; indirect fluorescence assay, or ELISA when used with recombinant antigen (K39), which is considered a highly sensitive and specific test (approximately 100%), false –ve in immune deficient patient eg. AIDS
- IV. Definitive Dx of Leishmaniasis is established by either demonstration of amastigotes in tissue specimens by direct microscopy by Giemsa staine or through isolation of the organism by culture using (NNN Nicole Novey McNeal) media; the culture specimen is taken from tissues of RES, especially spleen, BM, or LN.

Treatment

Anti-leishmanial therapy include:-in addition to the supportive measures that deals with anemia, infection, bleeding, and malnutrition specific therapy is needed:

- 1) Pentavalent antimonies include: Sodium stibogluconate (Pentostam) & Meglumine antimoniate, both in dose 20 mg/kg/day IV or IM for 28 days. Repeated courses may be necessary in severe or resistant cases. Cure rate 80-100%
SE: fatigue, arthralgia, elevated liver enzymes, hematological manifestations and cardiac toxicity.
- 2) Amphotericin B 0.5–1.0 mg/kg every day or every other day for 14–20 doses. Achieved a cure rate close to 100%.
SE: hypotension, fever, nephrotoxicity
- 3) Pentamidine isethionate 4 mg/kg/day for 2 wk.
SE: hypotension, hypoglycemia, nephrotoxicity and cardiac arrhythmia.it is less effective than amphotericin B.
- 4) Paromomycin 14 mg/kg/day for 3-4 wk.
- 5) Recombinant human interferon- γ (as an adjunctive Rx),
- 6) Miltefosine, has been approved as the first oral treatment for VL and has a cure rate of 80-90%, 4wk regimen.

Prevention.

- ✓ Avoid exposure to sandflies by the use of insect repellent and mosquito netting.
- ✓ Control of infectious reservoir
- ✓ Treatment of patients