Visceral Leishmaniasis

Learning Objectives:

1.Define the Concept,

2.Identify the etiology

3.Describe the clinical presentation of visceral leishmaniasis

4.Mention the differential diagnosis of visceral leishmaniasis

5.Identify the complications of visceral Leishmaniasis

6.Explain the methods of prevention visceral leishmaniasis

7.Outline treatment

Six years old child from Baladrose SE Baqubah presented with fever and malaise for the last 12days . The condition is associated with abdominal pain and loss of appetite with weight loss .

On examination , he looks pale with some petechial rash on the skin , also there is hepatosplenomegaly with some lymph nodes in the neck

Leishmaniasis *(Leishmania)*

The leishmaniases are a diverse group of diseases caused by intracellular protozoan parasites of the genus *Leishmania,* which are transmitted by phlebotomine sandflies.
Multiple species of *Leishmania* are known to cause human disease involving the skin & mucosal surfaces & the visceral RES organs

The parasite is dimorphic, existing as a flagellate promastigote in the insect vector & as an aflagellate amastigote that resides & replicates only within mononuclear phagocytes of the vertebrate host

Visceral leishmaniasis (VL) in the Old World is caused by *L. donovani* in Kenya, Sudan, India, Pakistan, & China & by *L. (L.) infantum* in the Mediterranean basin, Middle East, & central Asia. *L. infantum* is also a cause of LCL (without visceral disease) in this same geographic distribution.

Death occurs in >90% of pts without specific antileishmanial treatment

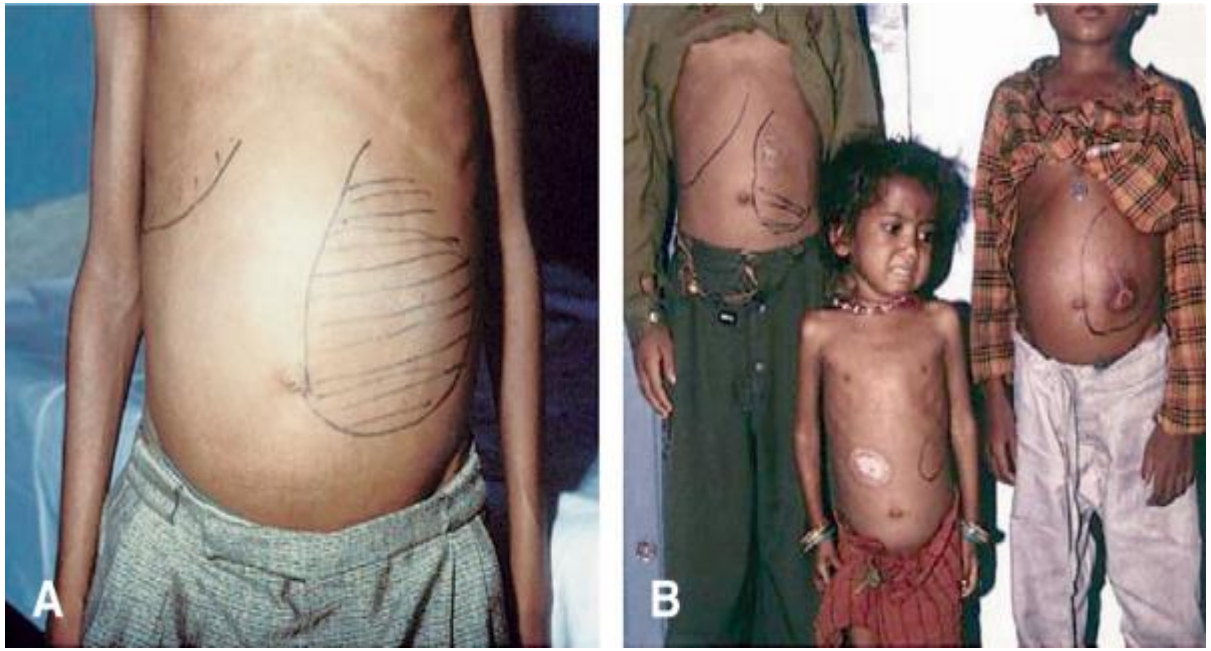
(kala-azar) typically affects children <5 yr of age in the New World *(L. chagasi)* &Mediterranean region *(L. infantum)* & older children & young adults in Africa and Asia *(L. donovani)*

After inoculation into the skin by the sandfly, the child may have a completely asymptomatic infection or an oligosymptomatic illness that either resolves spontaneously or in ~ ¼ pts it will evolve to active kala-azar within 2–8 mo

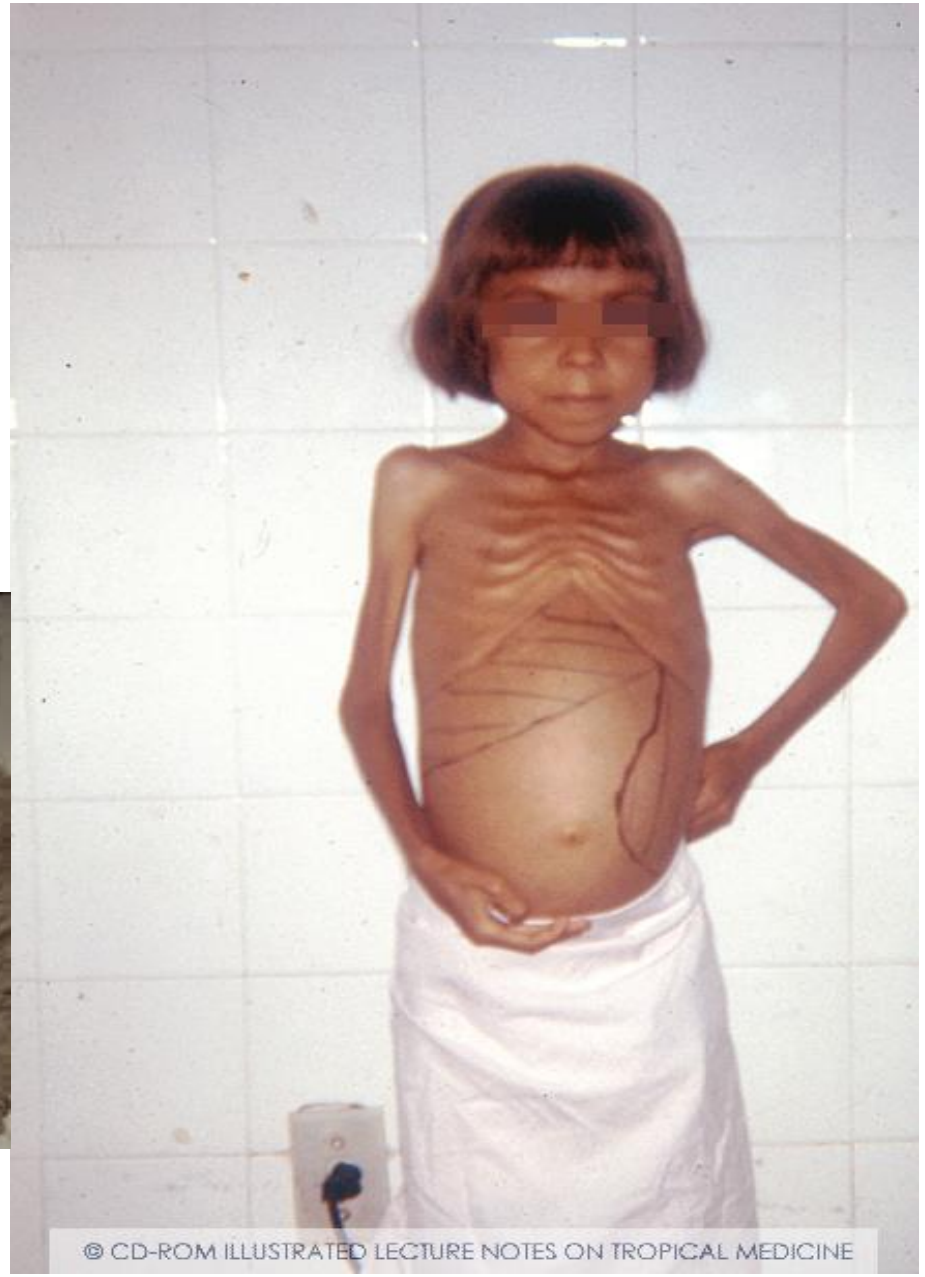
Fever is intermittent, there is weakness & loss of energy, & the spleen begins to enlarge.

The **classic clinical features** of high fever, marked HSM , & severe cachexia typically develop ~ 6 mo after the onset of the illness, but a rapid clinical course over 1 mo has been noted in up to 20% of pts in some series

Pancytopenia is profound, &jaundice, edema, & ascites may be present. Anemia may be severe enough to precipitate heart failure



Visceral leishmaniasis *(Leishmania donovani)*,.
***A,* Hepatosplenomegaly & wasting in a young man).**
***B,* Children with burn marks over enlarged spleen or liver**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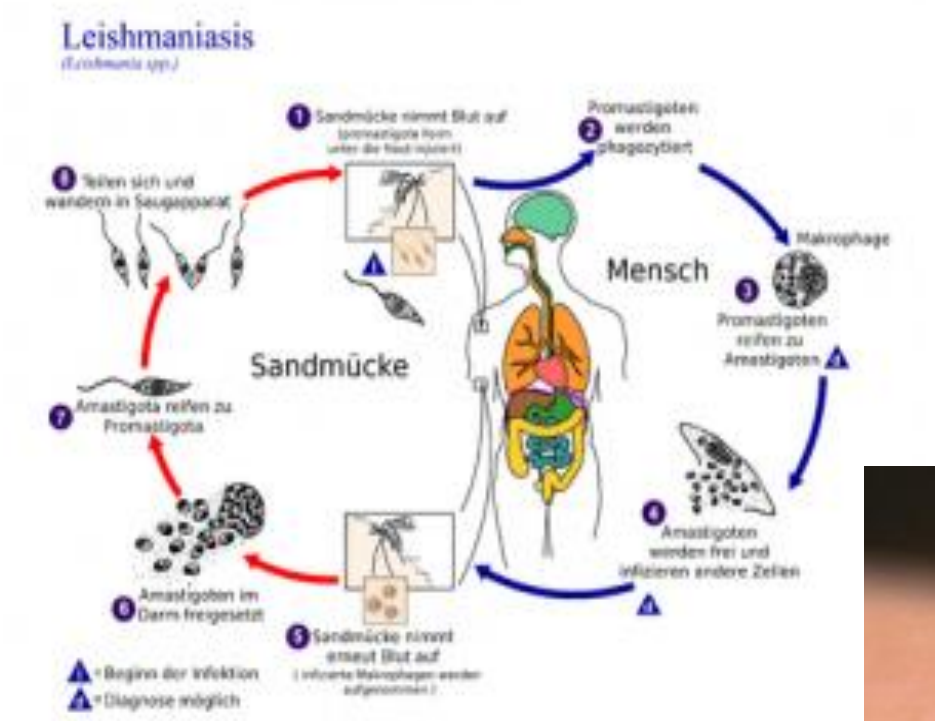
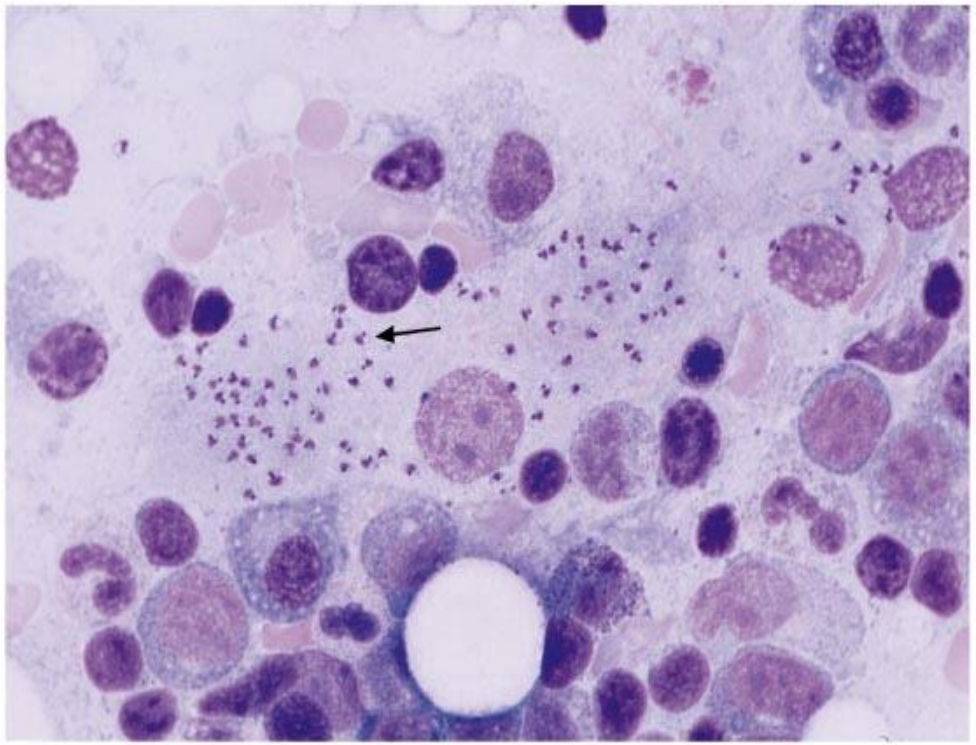
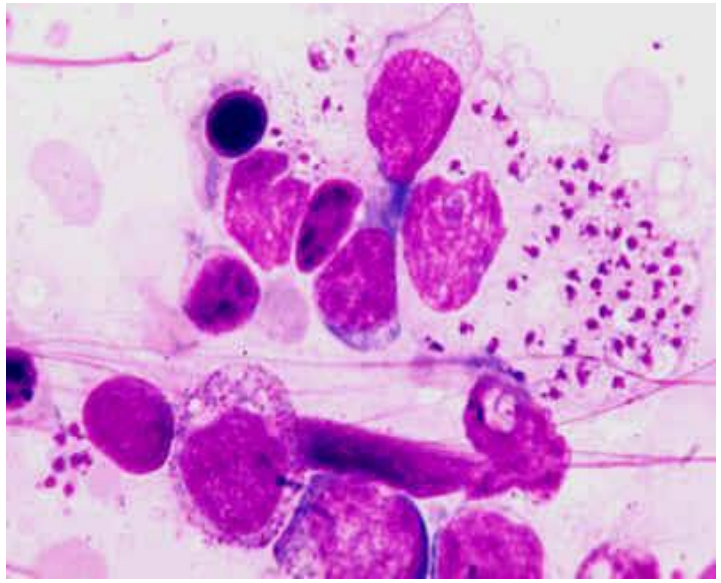

Leishmaniasis
Mensch
Sandmücke
Makrophage
Amastigoten im Darm freigesetzt
= Beginn der Infektion
= Diagnose möglich





Lab. findings associated with classic kala-azar include

Anemia Hb 5–8 g/dL),
Thrombocytopenia,
Leukopenia (2,000–3,000 cells/µL),
Elevated hepatic transaminase levels,
Hyperglobulinemia (>5 g/dL) that is mostly IgG

Serologic testing by enzyme immunoassay IFA, or direct agglutination is very useful in VL because of the very high level of antileishmanial Ab .
An ELISA using a recombinant antigen (K39) has a sensitivity & specificity for VL that is close to 100%

In VL, smears or cultures of material from splenic, BM , or LN aspirations are usually diagnostic.
In experienced hands, splenic aspiration has a higher Dx sensitivity, but it is rarely performed because of the risk for bleeding complications

TREATMENT

1-Supportive
2-Specific

Na stibogluconate Pentostam20 mg/kg/day i.v or i.m for 28 days. Repeated courses of therapy may be necessary in pts with severe disease

Amphotericin B desoxycholate at doses of 0.5–1.0 mg/kg every day or EOD for 14–20 doses achieved a cure rate for VL of ~100%, but the renal toxicity is common

Liposomal amphotericin B (3 mg/kg on days 1–5, & again on day 10) has been shown to be highly effective, with a 90–100% cure rate,less toxic

Parenteral treatment of VL with the aminoglycoside paromomycin (aminosidine) has efficacy (–95%)

Miltefosine, a membrane-activating alkylphospholipid, has been recently developed as the 1st oral treatment for VL & has a cure rate of 95% in Indian patients with VL when administered orally at 50–100 mg/day for 28 days.