Parasitic infection of the skin

Presented by

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ANIMAL LOVERS

Skin diseases caused by parasitic infection

- **Caused by nematoda**
  - **Cutaneous larva migrans**

  - **Trichinellosis**

- **Caused by protozoa**
  - **Cutaneous leishmaniasis**

Cutaneous larva migrans

ENJOY

but

Cutaneous larva migrans

(Creeping eruption, Plumber's itch, Sand worm)

- **Definition :** **serpiginous eruption of the skin due to skin invasion by animal hookworms' larvae.**

- **Causative parasite:** **filariform larvae of *Ancylostoma caninum* & *Ancylostoma braziliense* which are dog and cat hookworms**

- **Geographical distribution:** **Cosmopolitan.**

Cutaneous larva migrans

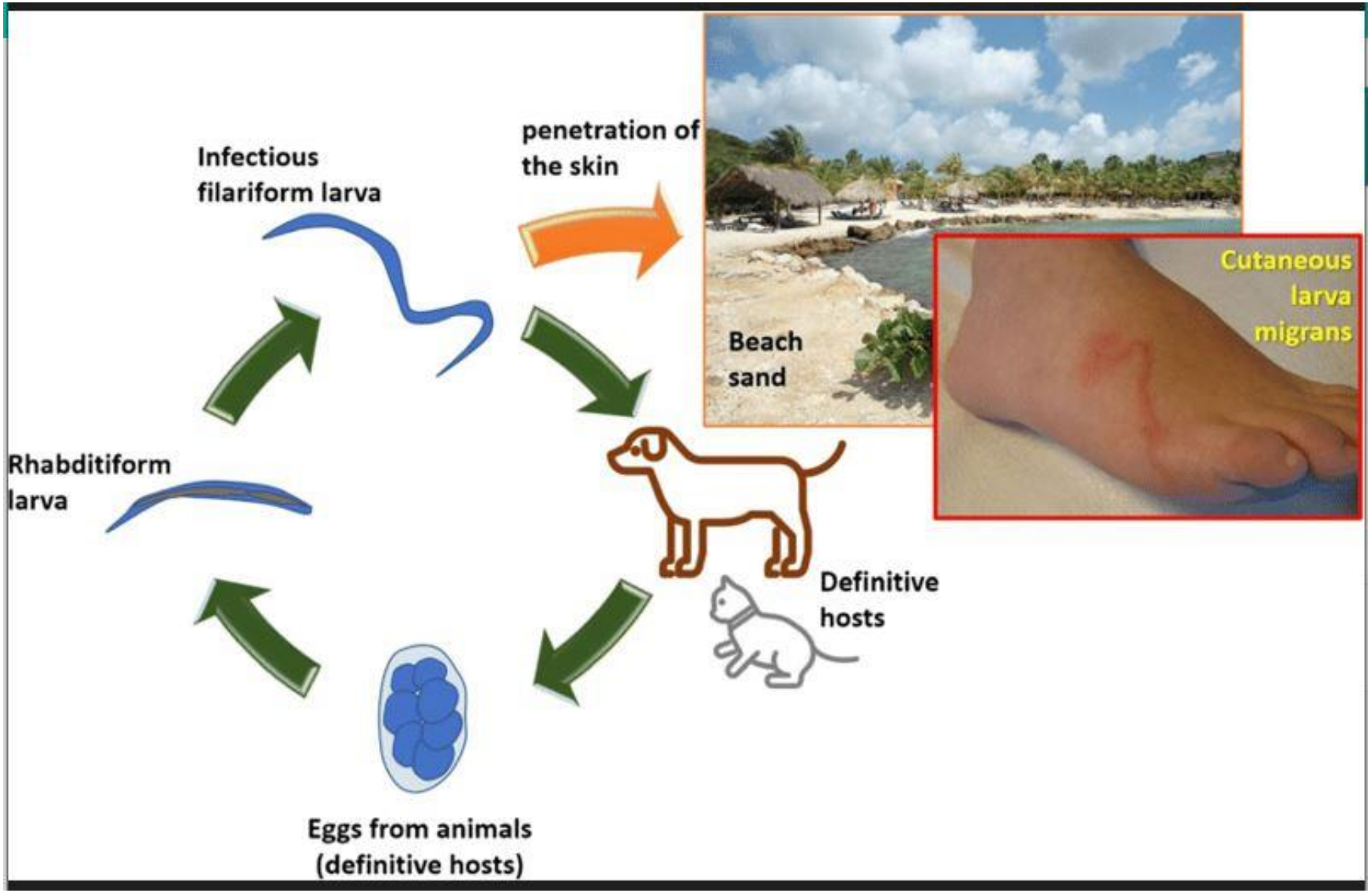
❖ **Mode of infection**

1. Human infection is caused by **penetration of the skin** by **animal hookworm's filariform larvae** which are not adapted to man.

2. Infection occurs due to contact with contaminated soil (moist or sandy) with dog & cat excreta.

3. The larvae migrate in the **superficial layers of the skin** and not go beyond the basal layer of the skin and keep migrating in the epidermis without development and rarely reaching the circulation.

Life cycle


Infectious
filariform larva
penetration of
the skin
Beach
sand
Cutaneous
larva
migrans
Rhabditiform
larva
Definitive
hosts
Eggs from animals
(definitive hosts)

Pathogenesis and clinical picture

➢At the site of entry red itchy papule can develop few hours after penetration

➢ Erythematous zigzag tunnel (2-4 mm), vesicular and elevated may be complicated by secondary bacterial infection causing sever irritation and pruritis.

➢Larvae remain active, move very slowly in the epidermis layer only for several weeks or months till die.

Commonly affect the skin of feet, hands or buttocks and may advances to 1-2 cm / day.

➢The skin lesion heals leaving linear white scars at the affected sites.

➢Rarely larvae may elicit generalized allergic manifestations

Treatment

❖**Systemic:**

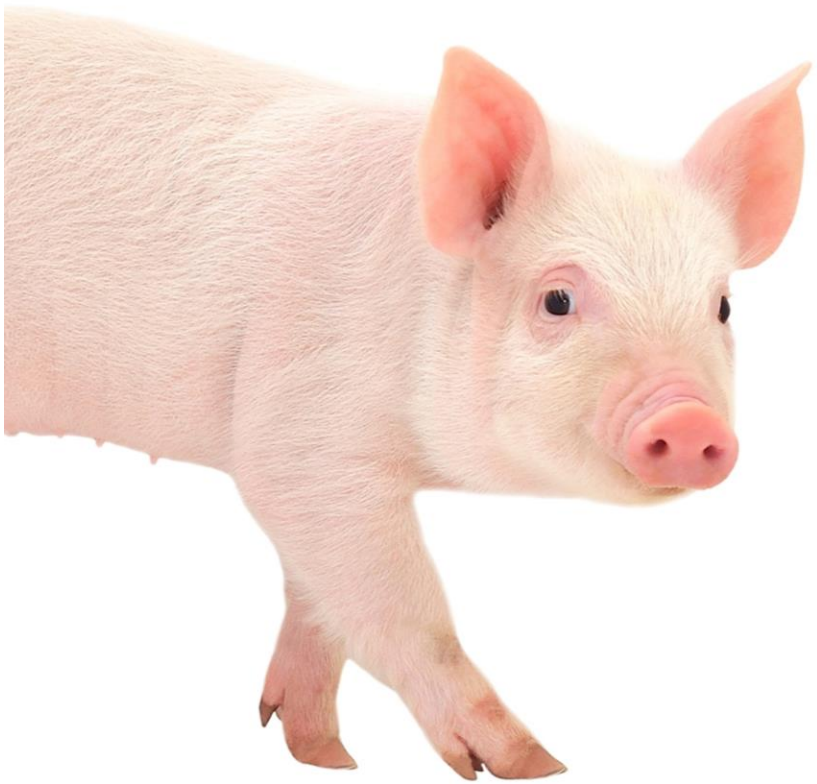
- Ivermectin 200 µg/kg single oral dose
- Albendazole 400mg/kg orally for 3 days
- Antihistaminic to relieve itching

❖**Local:**

- Topical ivermectin cream
- Local antibiotic for secondary bacterial infection

- Local freezing: Spray of skin by ethyl chloride (local freezing) or carbon dioxide snow which produce freezing of larvae till death➲ skin bleb ➲ larvae are lost with epidermal sloughs.

Trichinellosis

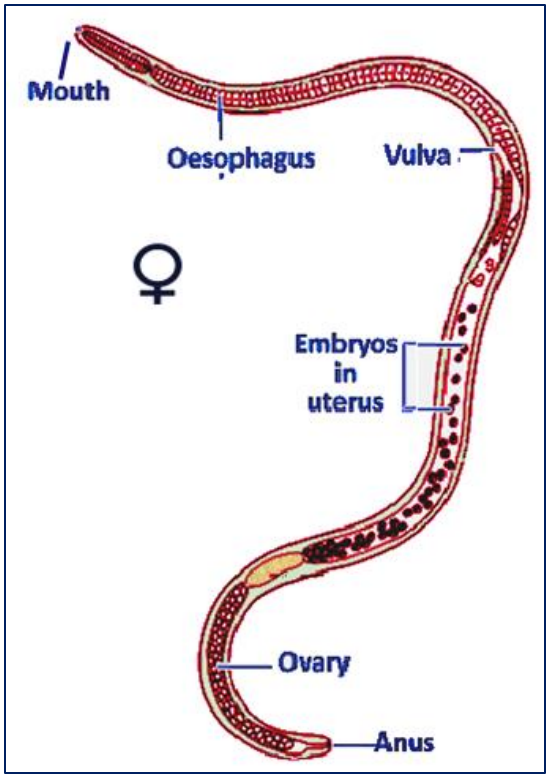


Trichinella spiralis

❖**Distribution:** worldwide specially in pork eating countries

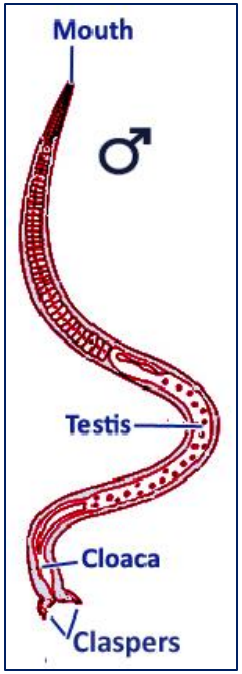
❖**Morphology:**

Female



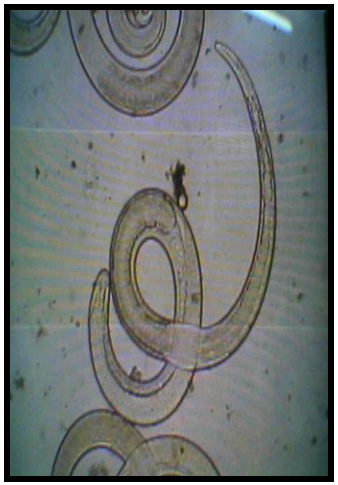
3 mm long

Male



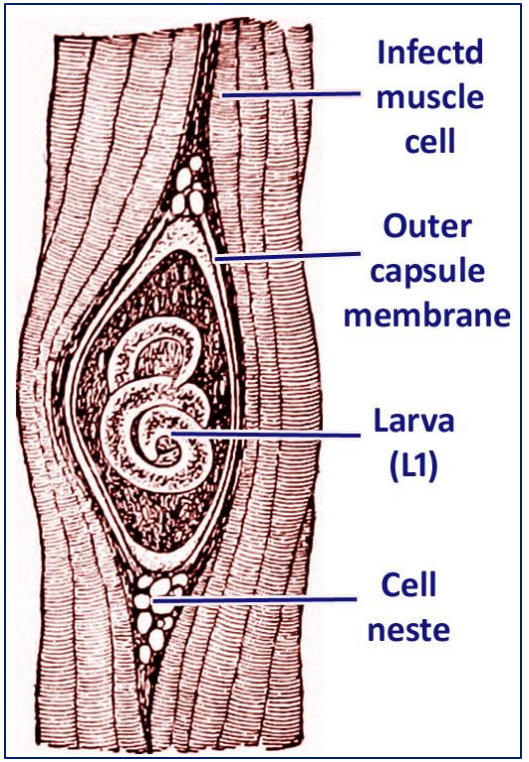
1.5 mm long

Newborn larva



0.1 mm long

Encysted larva in muscle



1 mm long in a muscle capsule of 0.5 mm long ??

Life cycle of *Trichinella spiralis*

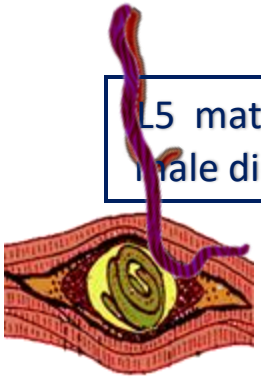
Adult in small intestine

Females start to deposit newborn larvae in the submucosa within five days

Female lays larvae in submucosa

Larvae migrate through lymphatics or blood

L5 mature to adults & male dies after mating

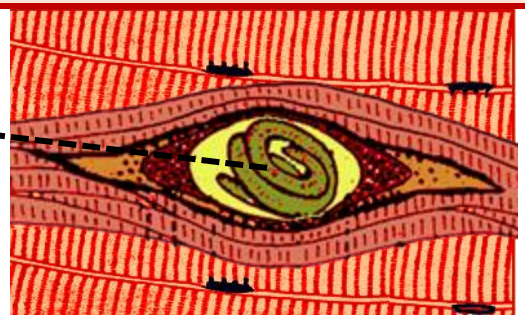


Larvae penetrate mucosa and molt four times→L5

Muscle cell transforms into Nurse Cell

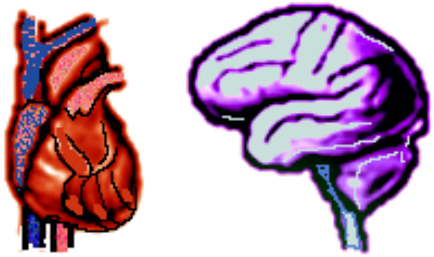
Larva grows & Nurse cell-parasite complex is formed (Infective cyst)

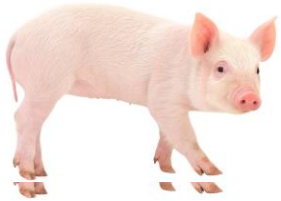
Ingestion of cysts in undercooked pork.



Pork meat containing encysted larva

Larvae enter heart or brain cells & can't complete maturation





Trichinella spiralis

❖ **Definitive host:**

- **Pigs, rats & sometimes man.**

❖ **Intermediate host:**

- **Pigs, rats & sometimes man.**

❖ **Habitat: Adults live in the small intestine.**

Males in lumen & females in tissues (embedded in submucoa). Infective larvae live mainly in active striated muscles.

Trichinella spiralis

❖ **Diagnostic stages:**

- ***T. spiralis*** **larvae in muscles**
- **Adults and newborn larvae in stool**

❖**Infective stage: Encysted larvae in skeletal muscles**

❖**Mode of infection: Eating undercooked pork meat, containing encysted *T. spiralis* larva**

N.B: Man is a complete blind host.

Pathogenesis and clinical picture

➢Enteral phase

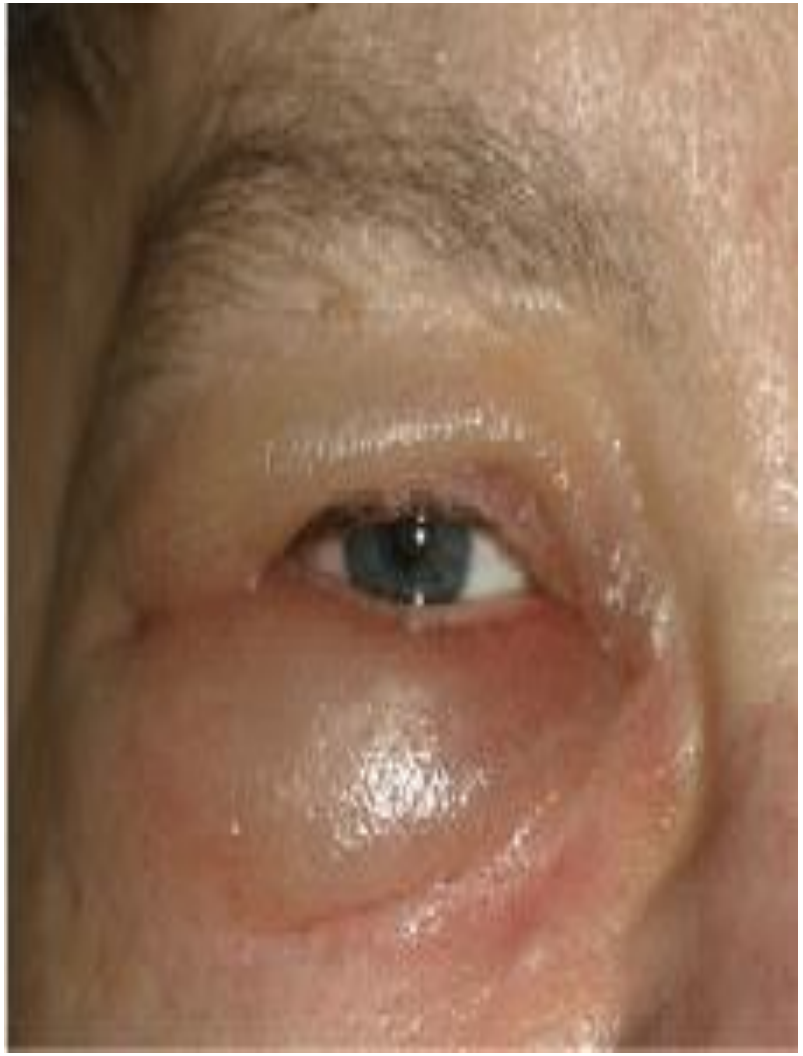
- From 2-7 days post infection
- Due to burrowing of adults and larvae in the intestinal mucosa
- Abdominal pain, diarrhea, nausea and vomiting

➢Parenteral phase

- Second week post infection
- Due to migratory larvae with blood vessels injury, hypersensitivity reactions and toxic product
- Fever, facial oedema, splinter and subconjunctival haemorrhage, cough, skin rash
- Patients die from encephalitis, myocarditis and peritonitis

➢Muscular phase

- Third week post infection
- Due to encysted larvae in muscles
- Severe muscle pain, swelling in muscles, Difficulty in breathing, may end in muscle paralysis



Trichinellosis

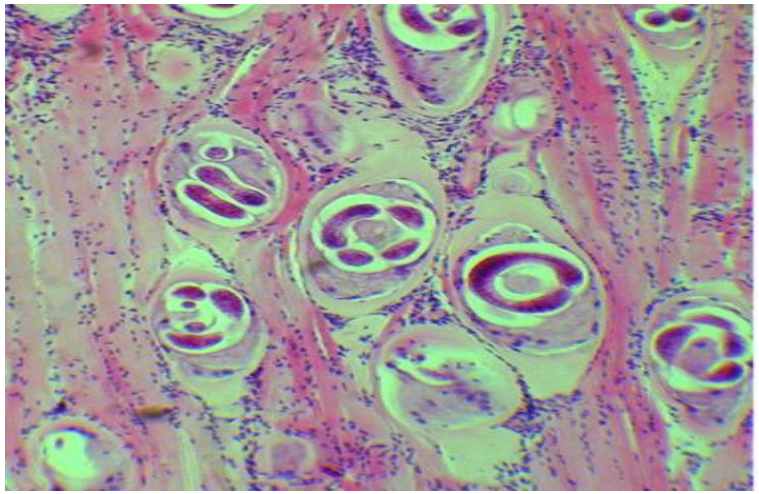
Laboratory diagnosis

Direct

- **Stool examination:** For larvae or adult.
- **Blood examination:** For larvae.
- **Muscle biopsy:** Taken from the swollen tender part & compressed between two slides after digestion in acid pepsin to see larvae .
- **Xenodiagnosis and Trichinoscopy:** suspected meat is given to a lab animal and his muscles are examined after one month
- **X ray** for calcified cyst.
- **CT** for brain lesions.

Indirect

- **Intradermal** test (Bachman test).
- **Serological tests:** CFT, IHA, IFAT, ELISA.
- **Eosinophlia (20-50%).**



Treatment

General treatment

- Bed rest and fluid therapy
- Sedatives for headache and muscle pain.
- Corticosteroids to reduce inflammatory reaction.
- Cardiac and respiratory monitoring.

Specific therapy

- Thiabendazole.
- Mebendazole.

Cutaneous leishmaniasis

Leishmaniasis classified into

Cutaneous leishmaniasis

Caused by

- *L. tropica*.
- *L. major*.
- *L. aethiopica*.
- ❖ *L. mexicana*.

Mucocutaneous leishmaniasis

Caused by

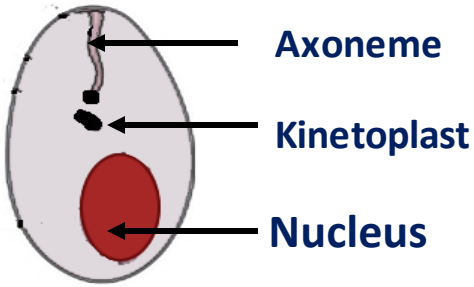
- ❖ *L. braziliensis*.

Visceral Leishmaniasis (Kala azar)

- ❖ New World Leishmaniasis
- Old World Leishmaniasis

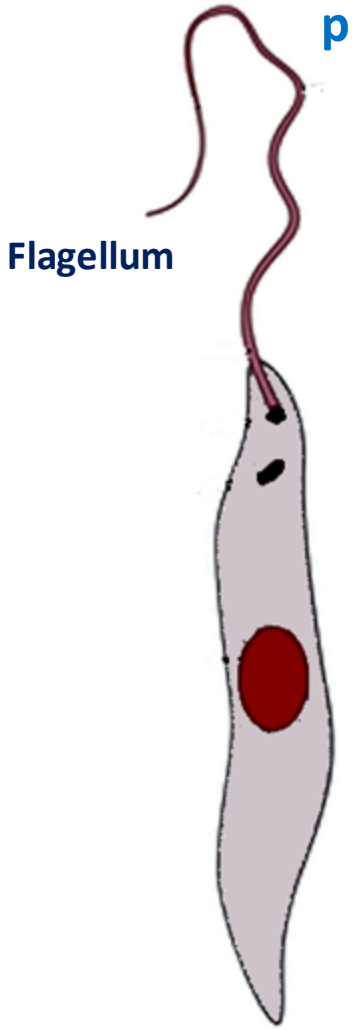
❖ Morphological forms

Leishmanial or Amastigote stage



Round or oval
(2–4µm)

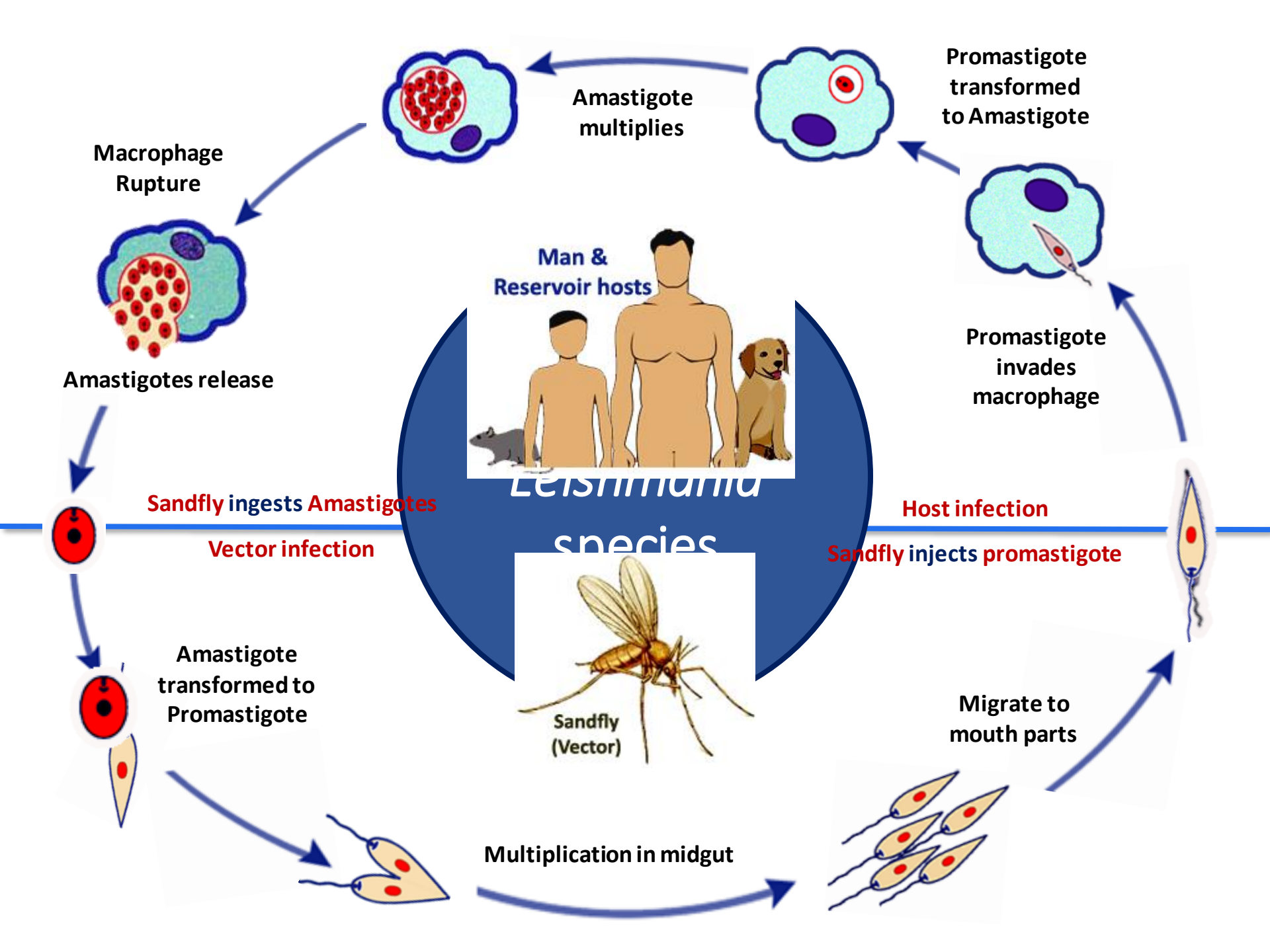
Leptomonad or promastigote stage



spindle-shaped
(15-20µm)

❖ Morphological forms

	1- Amastigote	2- Promastigote
Shape:	Oval	Fusiform or spindle
Kinetoplast:	Beside the nucleus	At the anterior end
Flagellum:	Absent	Present
Nucleus:	-Eccentric with central Karyosome	-Central with central Karyosome
Habitat:	-Intracellular (macrophage) -Tissue culture	-Midgut of the insect -Culture media


Amastigote multiplies
Promastigote transformed to Amastigote
Macrophage Rupture
Promastigote invades macrophage
Amastigotes release
Man & Reservoir hosts
Leishmania species
Sandfly ingests Amastigotes
Vector infection
Host infection
Sandfly injects promastigote
Amastigote transformed to Promastigote
Sandfly (Vector)
Migrate to mouth parts
Multiplication in midgut

❖ Mode of transmission

1- Bite of female sand fly (*Phlebotomus* species).

2- Direct contact with infected lesions.

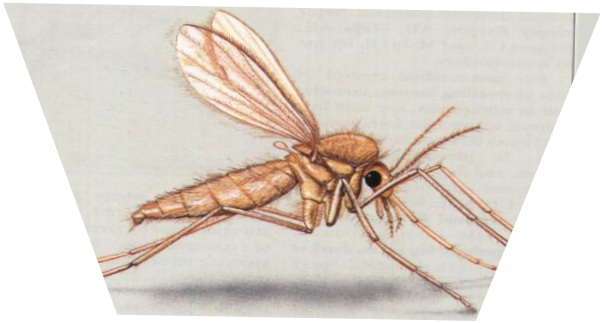
3- Mechanical transmission by blood sucking fly as *Stomoxys.*

D.H: Man

R.H: Dogs in *L. tropica.*
Rodents in *L. major* & *L. aethiopica*.

D.S: Amastigote (specimen)
Promatigote (culture)

I.S: Promastigote (sand fly)
Amastigote (contact)



Vector: Female sand fly (*Phlebotomus*)

❖ Pathogenesis

- The lesion develops on the exposed parts of the body
- Single or multiple
- Starts as erythematous papule that enlarges to form nodule that ulcerates forming an ulcer with sharp edge
- The lesion is painless unless secondary bacterial infection occurs
- The ulcer heals with a disfiguring scar
- Solid immunity to the same species

L. tropica (Dry or urban oriental sore)	*L. major* (Moist or wet oriental sore)	*L. aethiopica* (Diffuse cutaneous)
Chronic course	Acute course	Chronic widely distributed lesions in immunosuppressed individuals (opportunistic)
Long incubation period (2-12 ms)	Short incubation period (3-6 ms)	Due to lack of cell mediated immune response to leishmania
Ulcer with scanty exudate and slow healing (12 ms)	Ulcer with serous exudate and rapid healing (6ms)	Lesions not restricted to the site of infection and appears as multiple nodules
In Europe, Asia and Africa (cities and urban regions)	In Europe, Asia and Africa (rural areas)	East Africa (Ethiopia and Kenya)
Solid immunity	Solid immunity	Can relapse

Oriental sore

Leishmania aethiopica

New world (American) leishmaniasis

New World Cutaneous Leishmaniasis

Leishmania mexicana

Chiclero ulcer or bay sore

New World Mucocutaneous Leishmaniasis

Leishmania braziliensis

Mucocutaneous Leishmaniasis (Espundia)

Chiclero ulcer	Espundia
Caused by *L. mexicana*	**Caused by *L. braziliensis***
-A small single nodule at the site of sand fly bite ➲ ulcerates. -Usually on the face & ear pinna ➲ heals within 6 months. -Ear lesion causes destruction of the cartilage of the ear pinna. -Seen in chicleros who live in forests & collect gum from chicle trees.	-Primary skin lesion: Nodule in exposed regions ➲ ulcerates. - The ulcer with raised indurated margin ➲ heals in scar in months. -Secondary metastatic lesion: The parasite migrates from the primary site to blood & lymph to mucocutaneous junctions. -Sites: nasal septum, lips, palate nasopharynx & larynx. -Deformity & 2nd bacterial infection. - Death from septicemia and bronchopneumonia.

Chiclero ulcer

Espundia

Laboratory diagnosis

Direct

1-Scraping the edge of the ulcer or aspiration by a needle or biopsy (not the base as contains pus and necrotic tissues) and examined by :-

- Direct smear stained by Giemsa or leishman.

2- Culture on NNN medium (amastigotes changes into promastigotes).

Indirect

1-Immunodiagnosis:

- **Leishmanin I.D test (Montenegro test):** Not a specific test. It is +ve with cutaneous and mucocutaneous leishmaniasis but negative in diffuse cutaneous leishmaniasis.

- **Serological tests:** Not useful for the diagnosis.

2- **PCR:** A reliable diagnostic test. than routine smear and culture and it used also for species differentiation.

Treatment

1) Local

- Cryosurgery, curettage or local application of heat to raise the intra- lesional temperature to 37- 43 ° C for 12 hours as amastigote do not grow above 33 ° C.
- Surgical excision of the lesion.

2) Chemotherapy

- **Non-ulcerated lesion**: Intra- lesional injection of pentavalent antimony compounds.
- **Ulcerated lesion:** Should be treated with systemic pentavalent antimony compounds **(ex. Pentostam).**
- **Alternative drugs to pentostam:** Amphotericin B, imidazoles or Allopurinol.
- **Antibiotics** for secondary bacterial infection of lesions.


QUIZ
TIME

Case 1:

- A 19-year-old-woman, who had spent several months of the previous year as a student in Brazil, presented to the hospital complaining of ulcers on her lips and mouth as well as on her nasal mucosa. When she returned from Brazil the previous year, she had noted multiple skin lesions on her arm that had disappeared. A biopsy taken from ulcer edge showed macrophages containing small oval parasites about 2-3 µm

Case 2:

- A 40-year-old-woman presented to the hospital complaining of severe muscle pain. She reported being in France the previous month for studying. She had suffered from gastroenteritis while she was in France followed by unilateral oedema in the face which subsided and then she complained of continuous severe muscle pain.


ThankYou