Larva Migrans

Larva migrans can be classified into cutaneous or visceral types, depending on whether the larval migration takes place in the skin or in deeper tissues:

1-Cutaneous Larva Migrans (CLM)

2-Visceral Larva Migrans (VLM)

1-Cutaneous larva migrans (CLM

- Cutaneous larva migrans is a widespread and well-recognized disease in the tropics and is considered the most common dermatological problem after travel to tropical countries.

The causative agent is the infective stage of non-human hookworms.

- Ancylostomabraziliense(dogs & cats).
- Ancylostomacaninum(dogs).
- Uncinarastenocephala (dogs).
- Bunostomumphlebotomum(cattle).
- Strongyloidesspeciesof wild animals.
- Strongyloidesstercoralisof man.

Pathogenesis :

- The infection causes a red, intensely pruritic (itchy) eruption. The itching can become very painful and if scratched may allow a secondary bacterial infection to develop.

- Once the larvae begin migration, raised, pink snake-like tracks that are 2-3 mm wide form about 3-4 cm from the site of penetration. This is due to an allergic immune response to the larvae or its byproducts.

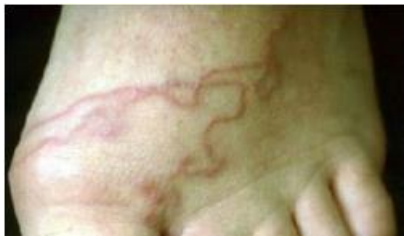
- Cutaneous larva migrans usually heals spontaneously over weeks to months and has been known to last as long as one year.

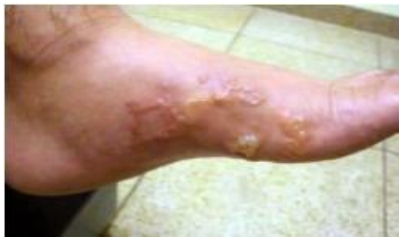
- This is separate from the similar cutaneous larva currens which is caused by Strongyloides . Larva currens is also a cause of migratory pruritic eruptions but is marked by

1) migratory speed on the order of inches per hour
2) perianal involvement due to autoinfection from stool .

3) a wide band of urticaria.

4)The lesion tend to disappears for a while and then re-appear in a different site.





Diagnosis:

- Clinical features.
- History of travel to endemic regions.
- Laboratory studies, including blood eosinophil determinations, total IgE levels, and skin biopsy, are rarely helpful.

Treatment:

CLM can be treated in a number of different ways:

- Systemic (oral) agents include albendazole or thiabendazole .
- Topical freezing agents, such as ethylene chloride or liquid nitrogen.

Visceral larva migrans

- Visceral larva migrans is a condition in humans caused by the migratory larvae of certain nematodes, humans being a dead-end host.

1) Toxocara canis (dogs) 2) Toxocara cati (cats)

- Similar to human Ascaris but with alternative pathways. Larvae do not develop in human but migrate continuously in viscera and encapsulate causing tissue damage. (parasitic granuloma) .
- T.canis and T.T. caticati may cause visceral larvae migrans in children who eat soil contaminated with (embyronated) infected eggs.

Pathogenesis

- In VLM, which occurs mostly in preschool children, the larvae invade multiple tissues (liver, heart, lungs, brain, muscle) and cause various symptoms including fever, , rashes, hepatosplenomegaly, and hypereosinophilia.

  Death can occur rarely.

- In OLM, the larvae produce various ophthalmologic lesions, which in some cases have been misdiagnosed as retinoblastoma, OLM often occurs in older children or young adults.

Diagnosis: serology, biopsy.

Ophthalmoscopic examination

Treatment: albendazole

Trichinellaspiralis(Pork worm)

T.spiraliscaused disease called trichinosis; trichiniasis; trichinelliasis.

Distribution of disease: In pork-eating countries.

Habitat:

Adult worms are found in the mucosa of the intestine and encystment of larvae are found in the muscle.

Morphology:

Adult worm:

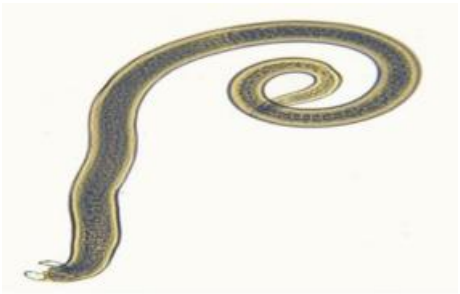
- Thread like, both male and female worms are wider posteriorly than anteriorly,
- The type of esophagus is cellular

Male

- Shorter than female
- The anterior end is delicate while the posterior end bears two conical papillae or appendage without spicules

Female

- Approximately twice as long as male.
- The female is ovoviviparous which produce larvae in the uterus and then passed in the stools.

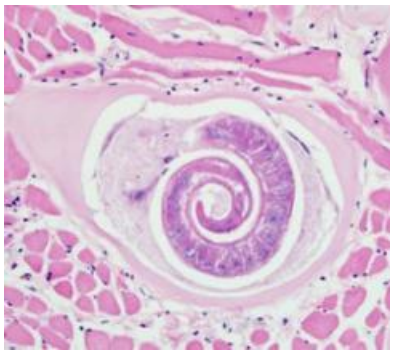


Female

Male

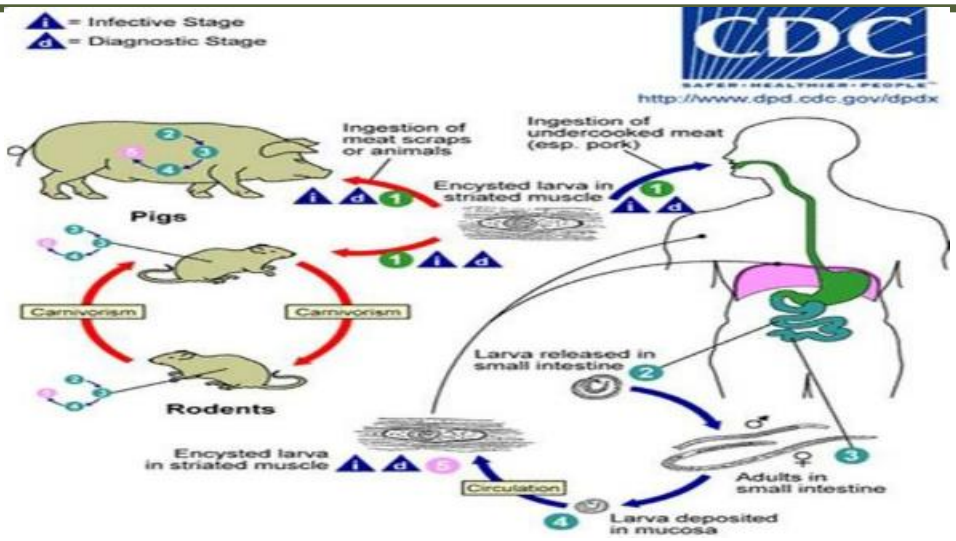
Cyst (encysted larva):

- Commonly found in skeletal muscle,
- Larva lies in the longitudinal axis of the muscle fiber.
- Each one encysted separately but in heavy infections, 2 or 3 larvae may be encysted together.
- Figure of 8 shape
- Cyst wall is fibrous, from the tissue reaction of the host.



Life cycle:

Mode of infection: Ingested of infective stage by humans, usually in raw or poorly cooked pork,



Pathogenesis :

Intestinal invasion (on the1st week) by adult worms:

- May be asymptomatic or causes gastrointestinal disturbances (simulating acute food poisoning).

Migration of larvae (on the 2nd week):

- Produces allergic symptoms as fever, edema of face, headache & eosinophilia.

Stage of encapsulation (on the 3rd week):

- Symptoms subsides & muscle pain persists for months

In severe infection:

- Death may occur from myocarditis, pneumonia or encephalitis.

Diagnosis:

1. Clinical picture.
2. Muscular biopsy:

- Trichinoscope:

Muscle samples (highly active muscle like diaphragm, tongue) were squeezed between two glass plates and then microscoped at low power

- The digestion method:

Muscle tissue can be digested with artificial digestive fluid releasing larvae from the muscle.

3. Serological tests:as IFAT or ELISA.
4. Intradermal test (Bachman test).

Treatment:

1.Mebendazole affects the adult stage

2.Thiabendazole affects the larval stage

3.Corticosteroids to reduce the inflammatory reactions.

Dracunculusmedinensis(Guinea worm, Medina worm)

- Cause dracontiasis, dracunculiasis, dracunculosis (Guinea worm ulcer).
- Longest of tissue parasites affecting humans.
- Rarely fatal.

Morphology:

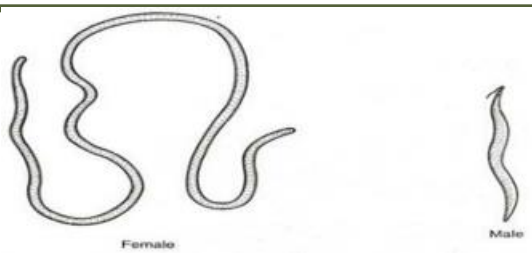
- Usually white

Female:

- Among the longest nematodes, often measures one meter in length.
- No more than 1-1.3mm wide

Male:

- Generally much smaller than the female
- Rarely recovered from humans because he dies shortly after mating



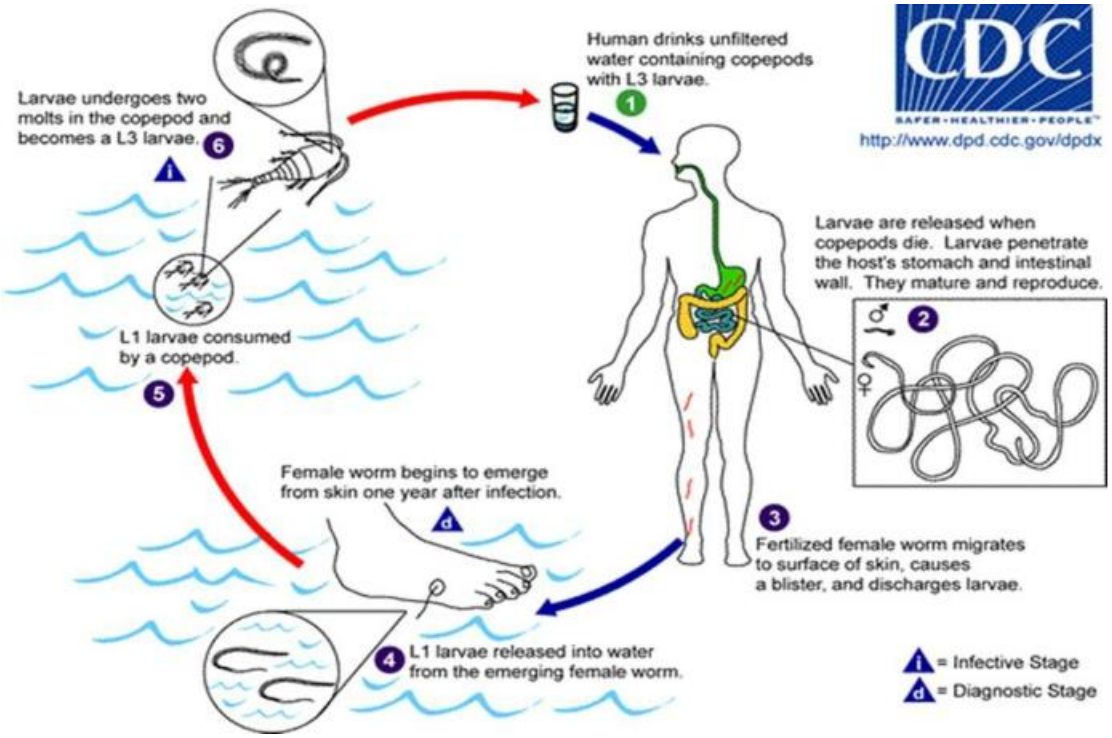
Larvae:

- Larvae or embryo is the diagnostic stage, it is comma shaped, non-sheathed
- Has a rhabditiform esophagus with round anterior end & a long pointed tail.

Life cycle:

Mode of infection:.

Transmitted to people when they drink water containing copepods that are infected with 3L of D.medinensis.



Life cycle of D.medinensis

Pathogenesis: D.medinensis .

- There are three major conditions that can occur from Dracunculus medinensisinfection:
  1. Emergent adult females cause hot and painful blisters,
  2. Secondary bacterial infections.
  3. Non-emergent worms that die under skin cause allergic reactions

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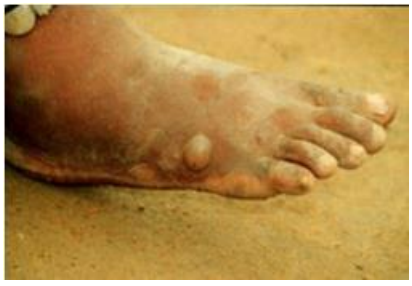
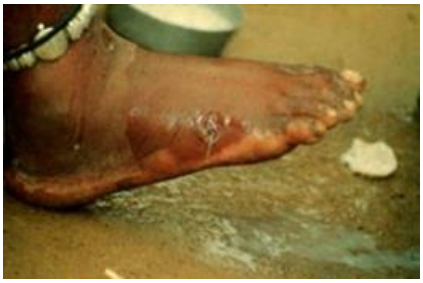
- If the worm does not emerge, two things can happen:
  1. The worm dies before reaching the skin layer, it will cause little reaction.
  2. The worm dies within the skin layer.
- Some other symptoms associated with D.medinensisare: fever, chills, and localized painful swellings.





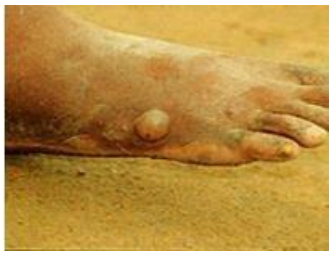
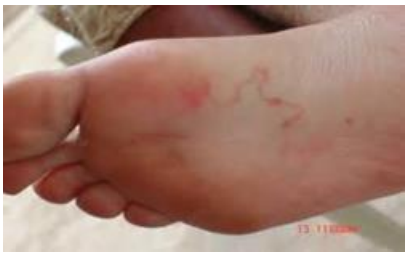
Complications:

1. Wound infections

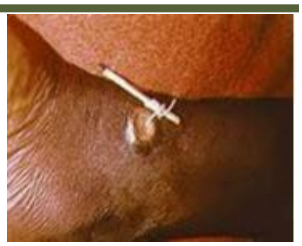
2. Cellulitis
3. Abscesses
4. Sepsis
5. Septic arthritis
6. Joint deformities
7. Tetanus.

Diagnosis:

1. Local skin lesions (papule, blister & ulcer).
2. Outline of the female worm may be seen under the skin (enhanced by reflected light).
3. Discharge of larvae: by cooling the ulcerated area or by contact with water.
4. X-ray shows calcified females.
5. Intradermal test & serological tests.
6. High eosinophilia.


Treatment:



1. Worm extraction (within 15-20 days)
2. Water immersion
3. Wound cleaning
4. Worm coiling around rolled gauze or stick
5. Topical antibiotics, bandaging.

Control:

1. Filtering or boiling of drinking water.
2. Destroying the Cyclops using copper sulphate or chlorine.
3. Patients are not allowed to path in water used for drinking

Filarial Worms

- The Filariae are long thread-like nematodes.
- Female worms produce eggs. The eggs modify, becoming elongated and worm-like in appearance and adapting to live within the vascular system.
- Modified eggs, referred to as microfilariae, are capable of living a long time in the vertebrate host, but cannot develop further until ingested by an intermediate host and vector, an insect.

Wuchereriabancrofti

- Bancroft's Filariasis, a blood & lymphatic infection.
- The infection often results in elephantiasis.

Habitat:

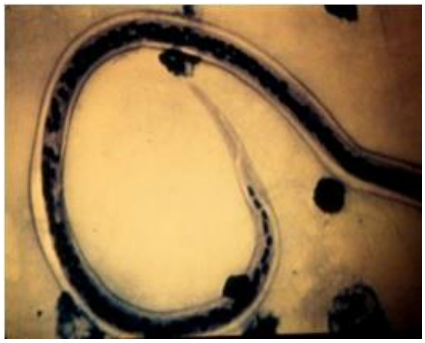
- The adult parasites reside in the lymphatics of the human host.
- Microfilariae, are present in the circulation

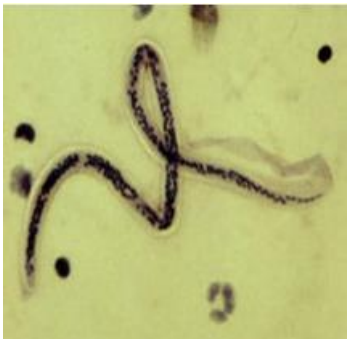
Vectors:

Culex,Aedes,&Anophelesmosquitoes.

Morphology:

Microfilariae are sheathed, and the nuclear column does not extend to tip of tail.



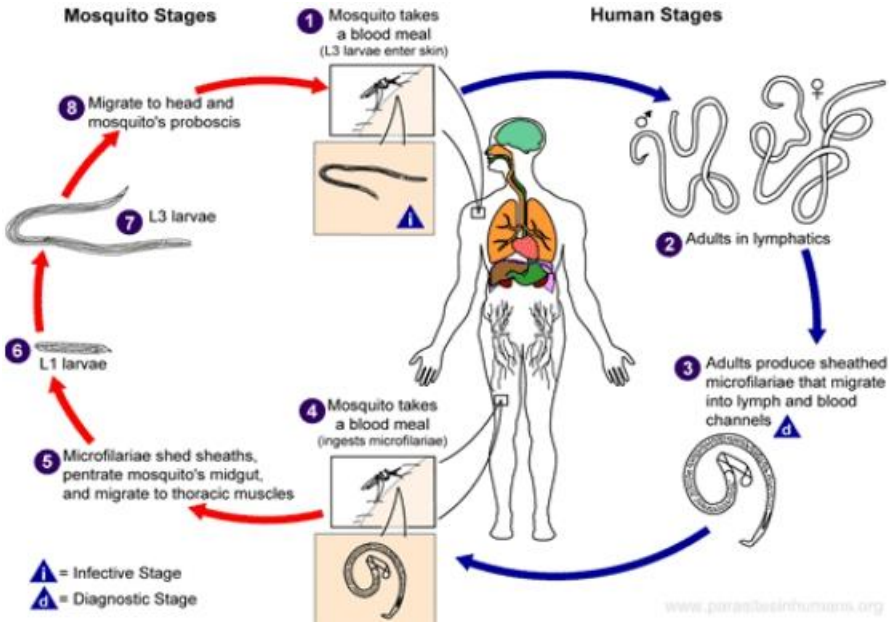


Life-cycle:

Definitive host: Human and mosquitoes as their

Intermediate hosts.: Culex,Aedes,&Anophelesmosquitoes.

- The microfilariae are present in the deep veins during the day, and during the night, they migrate to the peripheral circulation nocturnal periodicity



Pathogenesis:

Major pathology and symptoms:

- Swelling, due to allergic reaction occurring

around adult worms, produces obstruction & elephantiasis.

- Each individual reacts differently.
- Very few develop elephantiasis, but in some this is extensive.

Diagnosis:

Detection and identification of microfilaria in stained blood smears, best seen at night after (10 PM).

Onchocercavolvulus (blinding worm, River blindness)

- The diseases it causes is onchocerciasis, depending on where it infects the host it can be further classified as river blindness or filariasis .
- Characterized by skin changes, subcutaneous nodules, ocular lesions, lymphatic pathology and some systemic effects.

Morphology:

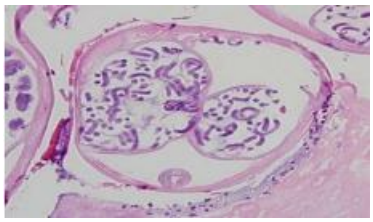
Adults:

- Found in pairs or groups.

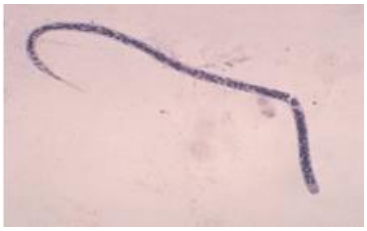
- Slender and blunt at both ends.

Microfilariae:

- Unsheathed and possessing nuclei which do not extend to the tip of the tail.
- Sharply pointed and curved tails.



Microfilariae



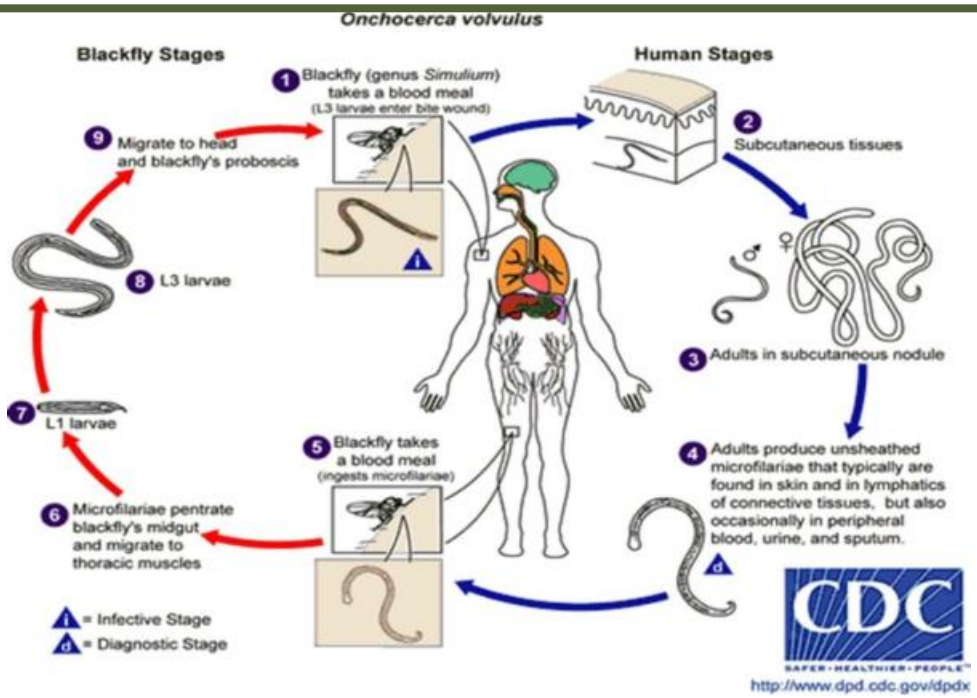
Adult

Life Cycle:

Definitive Host: Humans

Intermediate Host: Female Simuliumspp. (black flies)

Mode of transmission: Transmitted by blackflies of the Similiumspecies.



Life cycle of O.volvulus

Pathogenesis

1-Adults: Cutaneous onchocerciasis

- Adult coiled in skin and can cause subcutaneous nodules called onchocercomas.
- Adult encapsulated (host immune reaction).
- Dermatitis, depigmentation of the skin.
- Sowda is used to describe severe pruritus with darkening of the skin, often confined to one limb.

2-Microfilariae: River Blindness

- Eye lesions take many years to develop.
- Microfilariae do not cause many problems until they die
- Once dead skin reaction occurs causing lesions, most common cause of blindness.

Diagnosis:

1. Clinical picture
2. Most common method is a skin snip
   - Small piece of skin is pulled up and cut off with scissors
   - Placed in saline on a slide and examined for detection microfilariae
3. Nodules can be aspirated but only adults are found this way.

Treatment:

1. Removal of nodules can help with lowering rate of eye damage and rate of infection.
2. Ivermectin

Loaloa(eye worm)

Disease: loaiasis (filariasis)

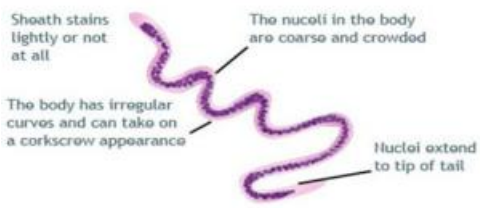
Geographical distribution: Central and Western Africa, Rain forest areas of West Africa

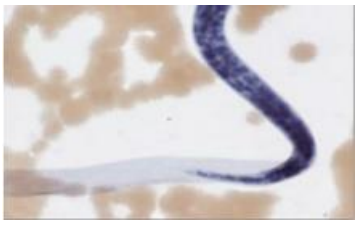
Morphology:

Microfilariae:

- They possess a sheath which stains blue-grey with hematoxylin.

- The tail with rounded end, the nuclei extending to the tip of tail.

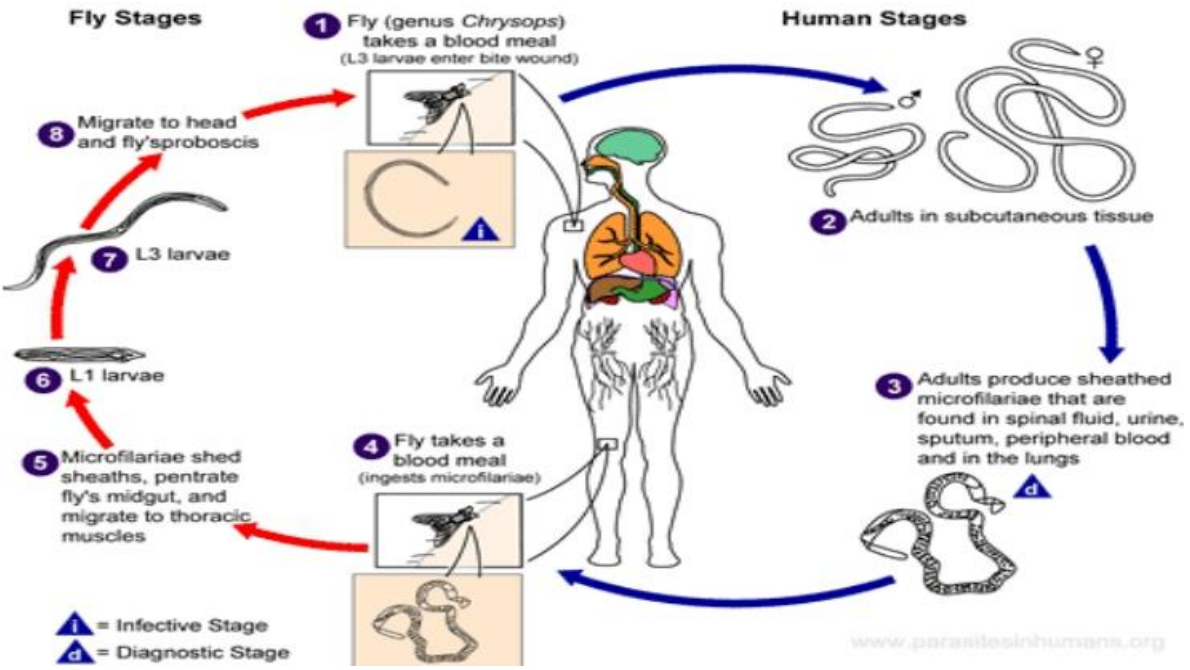




Life cycle:

Mode of infection :

Deer fly or Mango fly (Chrysopsspp), the vectors for Loaloatakes blood meal during the day they ingest microfilaria.



Pathogenesis:

Cutaneous lesion:

Caused by migration of the worm under the skin, these are called calabar swellings.

Ocular lesion:

The worm is visible when this migration occurs under the surface of the eye and so the name "Eye Worm", There is redness and itching in the eye but does not result in any long term symptoms.

Diagnosis :

1.Clinical picture

2 Microscopic examination of blood samples will allow identification of microfilariae of Loaloa.

3.Intradermal test & complement fixation test.