NEMATHELMINTHS

Class: Nematoda (Round worms)

General characters:

-Cylindrical worms, unisexual, have a body cavity.

-Male is smaller than female and commonly has a curved posterior end.

-The body wall consists of:

1- An outer non-cellular cuticle, which may inflated anteriorly to form cervical alae and posteriorly to form copulatory bursa.

2- Thin sub-cuticular layer.

3- A layer of muscle cells. The body wall surrounds a cavity, within which lie the digestive, reproductive, parts of the nervous and excretory systems.

Digestive system: is a simple tube beginning by the mouth which is surrounded by lips or papillae. In some species it is provided with teeth or plates. It leads into a tubular or funnel-shaped buccal cavity

-There is no circulatory system. The fluid of the body cavity contains haemoglobin, glucose, protein, salts and vitamins and fulfills the function of blood.

Nervous system: consists of nerve rings surrounding the oesophagus. From these six nerve trunks pass anteriorly and six nerve trunks extend posteriorly.

Reproductive system:

The male reproductive organs consisted of a single coiled or convoluted tube. It is differentiated as testis, vas deferens, seminal vesicle and ejaculatory duct and one or two copulatory spicules.

The female reproductive system may be either a single or bifurcated tube differentiated into ovary, seminal receptacle, uterus, and vagina.

Excretory system: consists of two lateral canals that lie in the lateral longitudinal cords. Near the anterior end of the body the lateral canals join in the terminal duct that leads to an excretory pore in the region of the oesophagus.

Modes of infection in nematodes

- By *ingestion o*f
  - Eggs: Ascaris, Entrobius , Trichuris
  - Larvae within intermediate host : Madina worm
  - Encysted larvae in muscle : Trichinella
  - Larvae with contaminated food: Trichostronglus
- By *penetration of skin* :Hook worm &Strongyloides

- By vector: Flaria
- By *inhalation* of dust containing egg: Ascaris &Enterobius

Classification of nematodes according to the habitat:

Intestinal human nematodes	Somatic human nematodes
• Small intestine - Ascaris lumbricoides - Hook worms - Strongyloides stercoralis - Trichinella spiralis • Large intestine - Trichuris - Enterobius	• Lymphatics - Wucheraria bancrofti - Brugia malayi • Skin/subcutaneous tissue - Loa loa - Onchocerca - Medina worm • Mysentry - Mansonella ozzardi - Mansonella perstans • Conjunctiva - Loa loa

Ascaris lumbricoides
(Giant intestinal round worm)

Geographical distribution: cosmopolitan, especially in tropical and subtropical countries.

Morphology:

1- Adult:

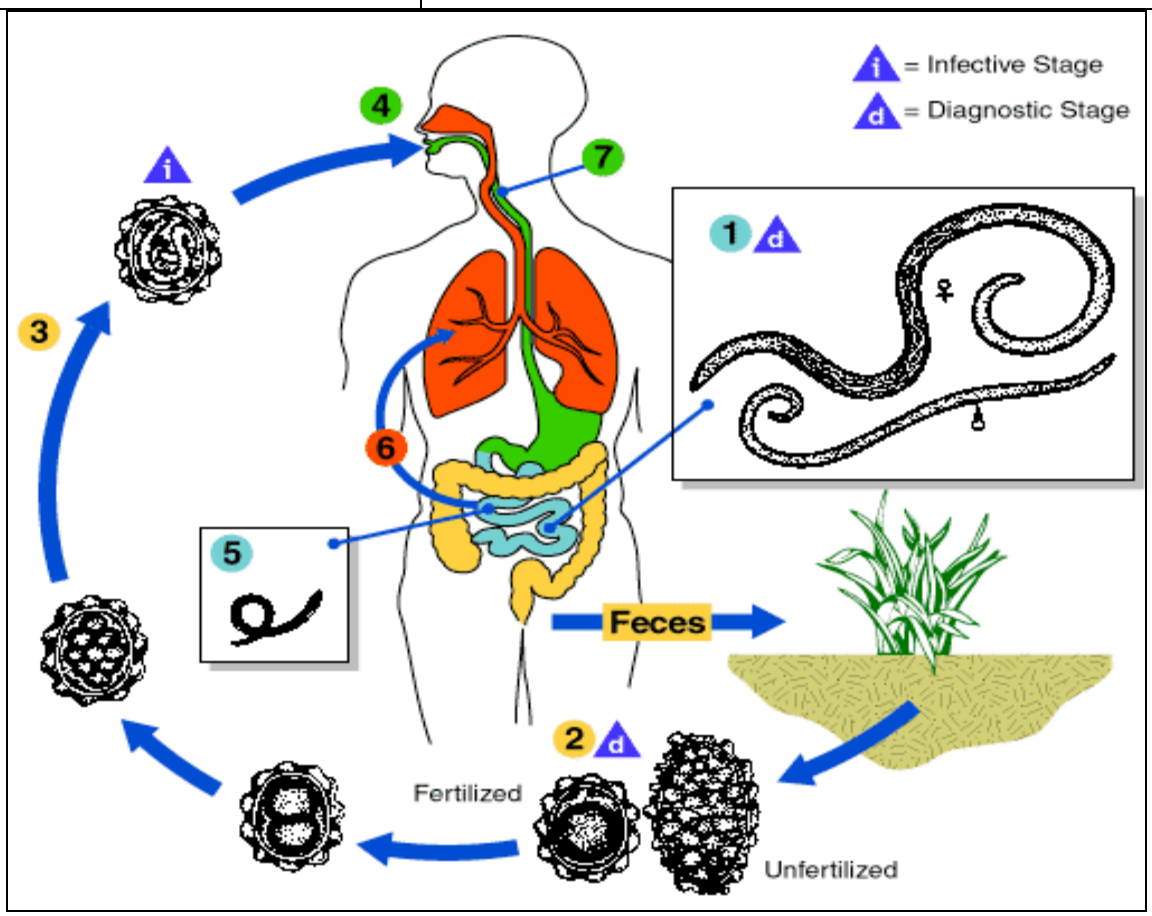
a. Long with tapering ends.
b. Creamy or pink in colour.
c. Finely striated cuticle.
d. Terminal mouth with 3 lips, one dorsal and two subventral. Each lip is provided with fine teeth and sensory papillae.
e. Club-shaped oesophagus.

Male: -15-20 cm in length x 3 mm thickness.
- Posterior end curved ventrally.
- Has one set of genitalia provided with two small equal spicules.

Female: - About 20-40 cm in length and 6 mm thickness.
- Posterior end straight.
- Has two sets of genitalia.
- The vulva opens ventrally at the junction of the anterior third and posterior two thirds of the body.

2- **Egg:** Three types of eggs:

(a) Fertilized egg Size Shape Color Contents	• 60 x 45 µ. • Oval with 2 coverings: -Outer thick regular albuminous mammillations. -Inner thick egg shell. • Brownish. • Immature ovum (one-cell stage).
(b) Unfertilized egg Size Shape Shell Color Contents	(Lay by unfertilized females). • 90 x 45 µ • Long and narrow Less developed thin irregular albuminous mammillations • Thin egg shell. • Brownish • Retractile granules.
(c) Decorticated egg	Similar to fertilized egg with no mammillated layer.



Life cycle of Ascaris lumbricoides

Life cycle:

Habitat	Small intestine
Definitive host	Man
Infective stage	Eggs containing second-stage rhabditiform larva.

it is specific parasite of man with no I.H. or R.H.

Stages in the life cycle: egg → larvated egg → larva → adult.

-Immature eggs pass in the faeces (200,000 eggs/female/day).

-Under favorable environmental conditions in the soil (temperature of about 25°C, humidity, shady soil and oxygen) a rhabditiform larva develops inside the egg in about two weeks. After one week this larva moults into a second-stage rhabditiform larvae inside the egg**.**

Mode of infection:

(1) swallowing water or raw vegetables polluted with embryonated eggs containing the infective larva.

(2) through contaminated hands by polluted soil.

(3) by inhalation to nasopharynx.

(4) house flies and cockroaches may carry the larvated eggs to human food.

-Eggs hatch in the intestine and the **rhabditiform larvae** penetrate the intestinal wall entering the circulation → the right side of the heart → the lungs where they break out of the pulmonary capillaries into the alveoli. They remain for some days and undergo their second and third moult (**Filariform larvae**).

-They then pass up the bronchioles to the bronchi, the trachea, and the epiglottis where they are swallowed to reach their final habitat in the small intestine. They moult for the fourth time and become **adults**.

-Eggs appear in faeces about 2 months after infection and the adult live from 12 to 18 months.

-Occasionally, when the infection is high, the larvae may pass through the capillary filter surrounding the alveoli to the left side of the heart. From there, get into the systemic circulation reaching to abnormal foci.

Pathogenicity and clinical picture:

I- **Migrating larvae:**

1. Lung: in **light** infection, there is slight damage with unnoticed pathological lesions. In **heavy** infection, the migrating larvae in the lungs result in condition known as *Ascaris* pneumonitis or **Loeffler's syndrome** especially in children. Clinically, from 1-5 days after exposure, cases

manifest with fever, cough, and dyspnea lasting for 1-2 weeks.

In extreme cases there may be lobular pneumonitis, cellular infiltration, serous exudates and haemorrhage causing cough and bronchial irritation, expectoration with blood stained sputum and oedema of lips, microscopically the larvae may be detected in the sputum, with many eosinophils.

2. General circulation: occasionally some larvae reach the general circulation and distributed to various organs as lymph nodes, brain, spleen & kidneys leading to abnormal clinical manifestations as a result of **visceral larva migrans**.

II- Adult worm: The usual infection consists of 5-10 worms, often goes unnoticed by the host and is discovered on a routine stool examination or by the discovery of an adult worm passed spontaneously in stool. The most frequent complaint is abdominal pain with distension, diarrhea or constipation, vomiting and dyspepsia.

1. Traumatic effects:
   - In heavy infection → intestinal obstruction.
   - Obstruction of the bile ducts by the worms → obstructive jaundice.
   - Appendix → appendicitis.
   - Obstruction of ampulla of Vater → acute hemorrhagic pancreatitis.
   - Perforation of intestinal wall → peritonitis.
   - Some worms may ascend via the stomach and oesophagus to the nasopharynx, enter the larynx causing suffocation especially in children.
   - It may come out of mouth or nose or even go to Eustachian tube from the pharynx resulting in damage of the middle ear.
2. Toxic effects: metabolic by-products of living or dead worms may give rise to fever, allergic manifestations and nervous irritability.
3. Nutritional impact: loss of appetite → malnutrition and impairment of growth, with vitamin A and C deficiency.

Diagnosis:

I. Clinical: symptoms of ascariasis are indistinguishable from those of other intestinal helminthic infections.

II. Laboratory:

1. Detection of eggs in stool (direct smear, after concentration, Stoll's technique).
2. Detection of migrating larvae in sputum or in gastric lavage contents.
3. Detection of adults passing out with or without stool or in vomitus.
4. Eosinophilia (7-12%).
5. Radiology: Barium meal shows cylindrical filling defect **(string sign)**.

Treatment:

1. Levamizol hydrochloride (Ketrax) as a single oral dose.
2. Mebendazole (Antiver, Vermox) or Flubendazole (Fluvermal).
3. Surgical treatment of complications e.g. obstruction of intestine, appendix or bile ducts.

Prevention and control:

4. Mass treatment of infected persons.
5. Sanitary disposal of excreta.
6. Health education and cleanliness (washing hands before meal).
7. Proper washing of green raw vegetables.
8. Pure water supply.
9. Control of flies and other insects.
10. Stool should not be used as a fertilizer unless being treated by chemicals or temperature of 50°C or higher to kill eggs.

Case study:

A 21-year-old woman presented suffering from abdominal colic, nausea, vomiting and diarrhea. After a physical examination, which was un-remarkable, the physician ordered a stool analysis for eggs and parasites.

Microscopic examination of a concentrated wet-mount preparation revealed several types of eggs. These eggs had thick shells and were oval, with some being more broadly oval than others. Some eggs lacked the outer mammillated covering found on the majority of eggs.

The diagnosis of this intestinal parasitic infection was made on the basis of microscopic analysis of stool specimen.

Questions:

1. Which parasite would you suspect of causing this patient's infection?
2. Describe the variable appearance of eggs of this parasite.
3. Which nematodes are most likely to cause human intestinal infection?
4. What is the infective stage?
5. How is this infection transmitted?
6. Do you think that this patient can transmit this parasitic infection to other members of the family during food handling? Why?
7. Describe the life cycle of this parasite.
8. Describe the clinical manifestations of this infection.
9. Which complications may cause this infection to be life-threatening?
10. How is this infection treated and controlled?

Toxocara species

Geographical distribution: worldwide.

Species and definitive hosts:

1. ***Toxocara canis***: it is a parasite of dog.
2. ***Toxocara cati***: it is a parasite of cat.

Morphology:

1. Adult: morphologically, both species are similar to ***A. lumbricoides***,

-They are smaller (male 4-6 cm and female 6-10 cm in length).

-The mouth is provided with cervical alae.

2. Egg:

-Size: 85x75 µ.

-Dark brown with pitted shell.

-Passes immature in dog's stool.

Life cycle:

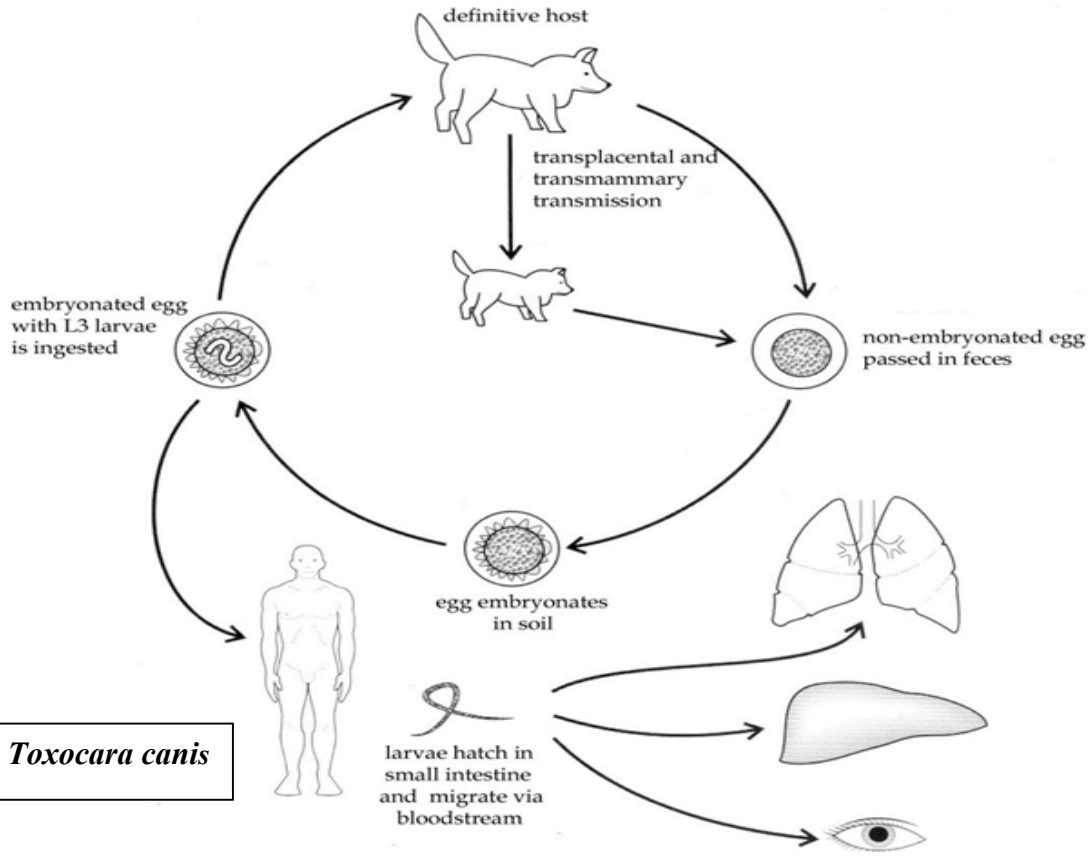
The life cycle of ***Toxocara*** spp. has the following biological characteristics:

-It is similar to that of ***A. lumbricoides***.

-Puppies are infected trans-mammary. They remain susceptible to reinfection until sexually mature, and then acquire some resistance to reinfection.

-Man **(Accidental host)** acquires the infection by accidental ingestion of ***T. canis*** and/or ***T. cati*** infective eggs containing 2nd stage rhabditiform larva.

-The 2nd stage rhabditiform larva is thinner than that of ***Ascaris lumbricoides***. This may explain why it is not filtered out in the lung and passes to the left side of the heart to settles in different tissues and organs resulting in **visceral larva migrans.**



Life cycle *Toxocara canis*

Enterobius vermicularis
(Oxyuris, pinworm or seat-worm)

Geographical distribution: cosmopolitan.

Morphology:

1- **Adult:** translucent cuticle, finely transversely striated. There are 2 wings like expansions (cervical alae) at the anterior end. The mouth with 3 small retractile lips, followed by small buccal cavity.

The **oesophagus is double- bulbed**. Intestine ends at the anus ventrally.

Male: 0.5 cm, its posterior end curved ventrally, one set of genital organs that open with the anus in the cloaca and one spoon- shaped spicule.

Female: 1cm, with a long thin sharply pointed tail occupying about 1/3 total length (hence the common name pin worm), 2 sets of genital organs and vulva at the junction of the anterior fourth with the rest of the body.

2-Egg:

Size	50x25 µ.
Shape	Plano-convex (one side is convex and the other is straight).
Shell	2 layers and covered by a 3rd outer thin albuminous sticky layer.
Color	Colorless.
Content	Fully developed larva.

Life cycle:

-**Habitat**: adult worm lives in the caecum, appendix and adjacent parts of small and large intestine.

-**Definitive host**: only man.

-**Infective stage**: fully embryonated eggs containing fully developed larvae.

-The gravid female migrates to the perianal and perineal area where they lay eggs. The eggs are infectious several hours after deposition.

Mode of infection:

1. Ingestion of eggs through contaminated food and drink.
2. Autoinfection: eggs are carried under finger nails to the mouth after scratching of perianal skin (anus to mouth infection).
3. Retro-infection: eggs hatch on the perianal region and larvae migrate back through the anus to the rectum and caecum.
4. Air-borne infection
5. Contact with patients (direct hand to hand or indirect contact by handling contaminated articles as clothes, bed linens, toilet seats, door knobs).

Pathogenicity and clinical picture:

The clinical symptoms are largely due to perianal, perineal and vaginal irritation caused by the migration of the gravid female worm.

1. Local irritation and discomfort, with nocturnal itching and enuresis, insomnia, irritability, restlessness, neurosis, hyperactivity, gridding of teeth and inability to concentrate.
2. **Pruritus ani** due to:
   a- Nocturnal migration of the female worm on the perianal skin with worm like movement.
   b- Skin sensitization by ruptured worms during scratching.
   c- Striations on the cuticle cause skin irritation.
   d- Sticky material on the egg causing irritation.
3. Vaginitis and salpingitis by migrating gravid females. Granulomas are formed around eggs or worms.
4. Irritation of intestinal mucosa with minute ulcers, hemorrhage and 2ry bacterial infection at site of attachment.
5. Obstructive appendicitis rarely occurs.

Diagnosis:

I. **Clinical**: Pinworm infection is suspected in children who show perianal itching, restlessness and insomnia.

II. **Laboratory**:

1- Detection of adult worms in feces or in the perianal region.

2- Detection of the eggs:

-In stool: seldom found (in only 5% of infected patients), unless uterus of gravid female ruptures during migration to the perianal region.

-In urine of female patients.

-On perianal region by swab, this must be done early in the morning before defecation or bathing and should be repeated for several days before the patient is considered free.

Types of swabs:

a. **Scotch adhesive tape swab:** a piece of scotch tape, hold over a tongue depressor is rolled over the perianal skin and removed. The adhesive tape is put on a slide with a drop of toluene and examined for eggs.
b. **Vaseline swab:** the perianal skin is swabbed with a piece of cotton soaked in Vaseline and the swab is put in a mixture of ether and water to dissolve the

Vaseline. The mixture is centrifuged and the deposit is examined for eggs.

c. **National Institute of Health (N.I.H.) swab:** it is a piece of non adhesive cellophane fixed to a glass rod. The glass rod is inserted through a perforated stopper in a test tube. The perianal skin is swabbed in the morning by the cellophane paper. The cellophane paper is united, spread between 2 slides with a drop of toluene and examined for eggs.

Treatment:

1. Mebendazole (Vermox), Flubendazole (Fluvermol) or Pyrantel pamoate (Combantrin) as a single oral dose and a 2nd dose must be given after 2 weeks to prevent re-infection.
2. Local: mercurial ointment is applied to the perianal skin especially at night to relieve itching and kills females that come out to deposit eggs and prevent dispersal of eggs.

Prevention and control:

1. Mass treatment of the whole companions of the infected person.
2. Personal cleanliness.
3. Protection of food and drink from contamination by dust and hands of patients.

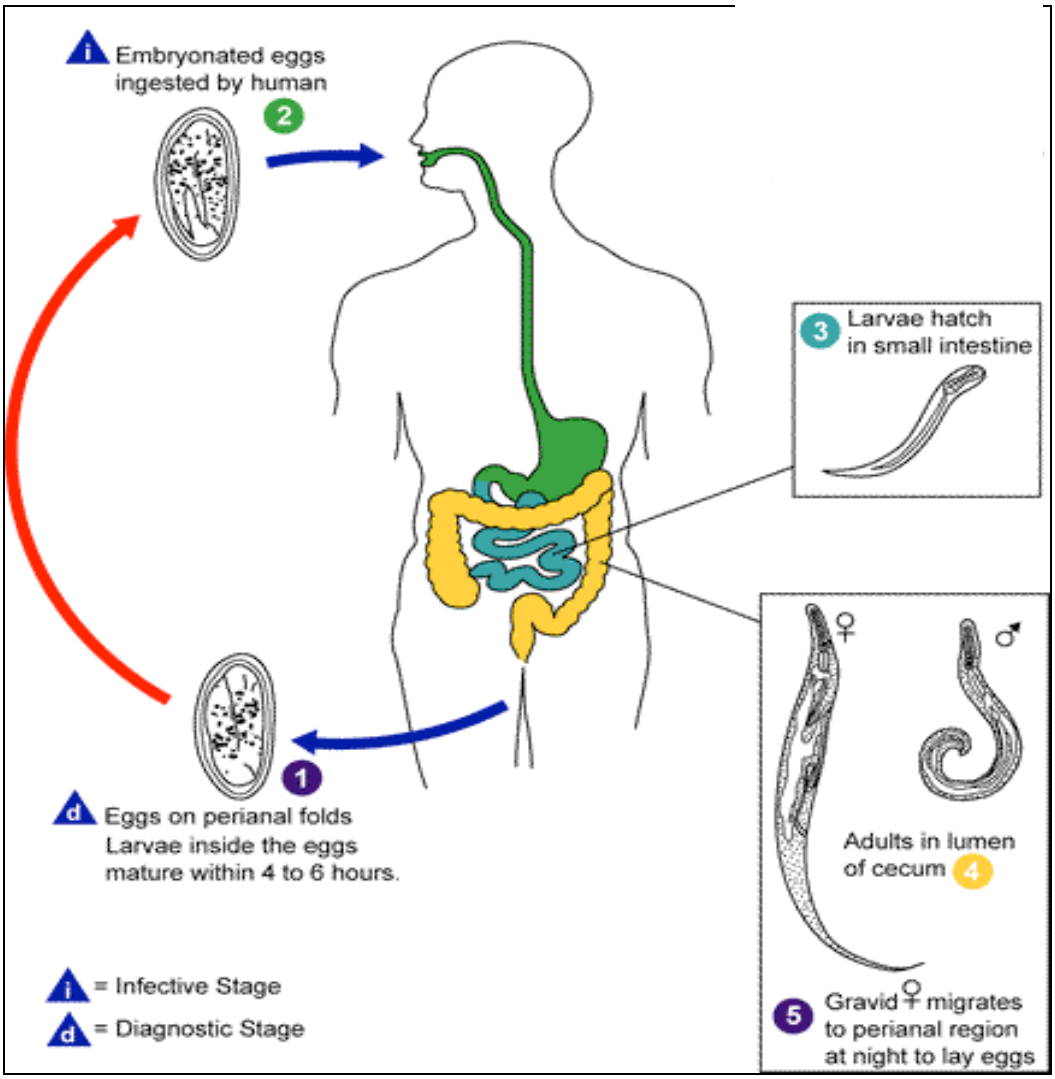
Case study :

A 7-year-old boy was not sleeping well, had been irritable, and had complained to his mother about anal itching and irritation. The boy's younger sibling also began to complain of similar symptoms.

The children were taken to the pediatrician for evaluation. He ordered a parasitological laboratory test to provide a strict diagnosis. A worm egg seen microscopically enabled the laboratory to identify the worm causing the symptoms.

Questions:

1. Which parasite is causing the children's discomfort?
2. Which type of laboratory procedure would the physician have ordered to make a diagnosis?
3. What are the precautions to be followed during performing this procedure?
4. How is the infection transmitted?
5. Describe the life cycle of this parasite.
6. How is the diagnosis made, using this procedure?
7. Which intestinal protozoon has been associated with this helminthic infection?
8. How is this infection treated?
9. What are the precautions to be followed to eradicate this parasitic infection?



Life cycle of *Enterobius vermicularis*

Trichuris trichiura

(Trichocephalus trichiura or whipworm)

Geographical distribution: cosmopolitan, more in worm moist regions.

Morphology:

1- **Adult:** body is demarcated into an anterior attenuated whip-like thin part (3/5) that contains a cellular oesophagus, and a more robust posterior thick part bluntly rounded (2/5) contains the rest of organs.

Male: 3-4 cm in length coiled posterior end with a single copulatory spicule inside a retractile sheath and a terminal cloaca.

Female: 4-5 cm in length, straight blunt posterior end, has one set of genitalia and the vulva opens at the junction of thin and thick parts. Anal orifice is terminal.

2- Egg:

Size	50 X 25 µ.	**Shape**	barrel-shaped.
Color	yellowish-brown.	**Content**	immature embryo.
Shell	thick-shell, with bipolar mucoid plugs.		

Life cycle:

-Habitat: adult inhabit the human large intestine mainly the caecum but is also found in the appendix and lower ileum.
-Definitive host: man.
-Reservoir host: some mammals.
-Infective stage: egg containing first stage larva.
-Stages in the life cycle: egg → larva → adult

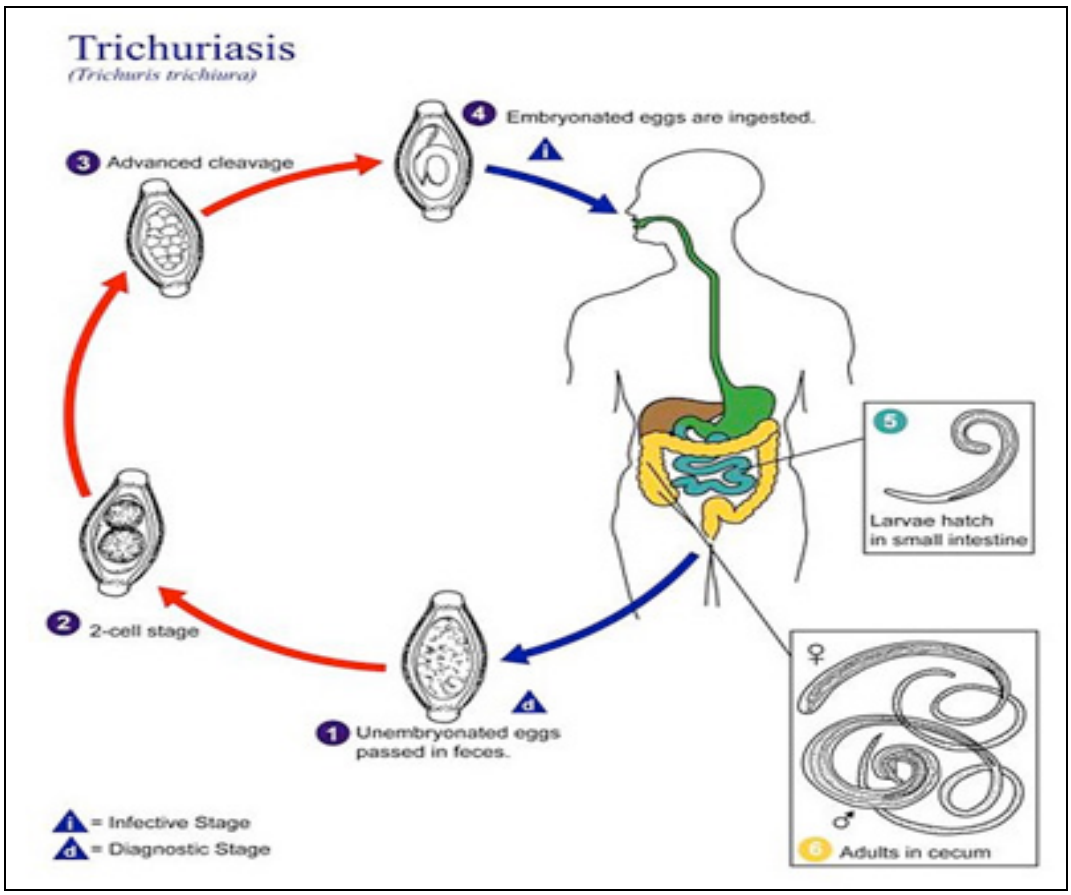
Mode of infection:

Ingestion of larvated egg with contaminated food and drinks.

- When the embryonated egg is ingested by man, the larva escapes from the egg shell in the upper small intestine and penetrates the intestinal villi, where it remains for 3 to 10 days.
- After reaching the adult stage, it passes downward to the caecum. A spear like projection at its anterior extremity enables the worm to penetrate into and deeply embed its anterior portion into the intestinal sub-mucosa of the host.
- The female is oviparous, life span about 4-6 years, and number of eggs deposited/female/day is about 2000 eggs.
- Ova pass with stool 2 months after infection and require 3-5 weeks for the larva to develop inside and become infective in favorable environment (worm, moist and shaded soil).

Pathogenicity and clinical picture:

1. Light infection is asymptomatic.
2. Heavy chronic infection manifests with:
   a. Frequent, small, blood-streaked diarrheal stools, dysentery.
   b. Abdominal pain and tenderness, nausea, vomiting and weight loss.
   c. **Rectal prolapse** due to oedema as a result of large number of worms embedded in the mucosa, dysentery and toxic effect on the pelvic nerve.
   d. **Hypochromic anemia** due to suction of blood by the parasite and hemorrhage that occur at their attachment sites. Hyperchromic anemia may also occur by the toxic parasitic products (*Trichocephalus* pernicious anemia).
   e. Appendicitis.
   f. Protein loosing enteropathy in heavy infections.
   g. Intestinal wall perforation and peritonitis.
   h. Eosinophilia (30-60 %) in acute heavy infection.



Diagnosis:

I. **Clinical**: can't be differentiated from infection with other intestinal nematodes.

II. **Laboratory**:

1- Stool examination for the characteristic egg.

2- Proctoscopy: worms can be seen attached to the inflamed and ulcerated rectal mucosa.

Treatment:

Mebendazole (Vermox or Antivir) or Flubendazole (Fluvermal).

Prevention and control:

1. Treatment of infected patients.
2. Sanitary disposal of human stool.
3. Strict hygienic measures for hands, food and drink.
4. Control of house fly.

Case study:

A 6-year-old boy presented to the pediatrician suffering from diarrhea, abdominal pain and nausea. Blood was drawn for complete blood count. Three stool specimens were collected and submitted for examination for ova and parasites.

Blood picture revealed hemoglobin of 11.5 gm/dl. Microscopic analysis of the concentrated stool specimens revealed numerous bile-stained, barrel-shaped eggs. The eggs were characterized by having clear, prominent and protruding bi-polar plugs.

Questions:

1. What is this infection according to the ova detected?
2. Describe the morphological characteristics of the adult worms.
3. What is the infective stage of this parasite?
4. Describe the life cycle of this parasite?
5. What is the main complication of this infection?
6. How is the diagnosis of this infection made?
7. Which other nematode egg may be confused with this parasite? Can they be differentiated by the morphological appearance? Describe.
8. How does the patient's blood test results relate to this infection?
9. How is infection with this parasite treated?
10. How is infection with this parasite prevented and controlled?

Trichinella spiralis

Geographical distribution: cosmopolitan distribution particularly in Europe and United States where people eat pig meat. It is also known in Africa, Southern Asia and the Middle East. It exists in Egypt.

Morphology: it is a small worm characterized by:

1. Slender anterior end.
2. Cellular oesophagus.
3. Terminal anus or cloaca.

Male: 1.5 mm x 40µ, its posterior end is ventrally curved with two lobular caudal appendages and having one set of genitalia.

Female: 3 mm x 80 µ, its posterior end is bluntly rounded, having one set of genitalia, the vulva opens at the junction of the anterior fifth with the rest of the body and is **larvi-parous (viviparous).**

Larva: has a spear like burrowing tip at its tapering anterior end. It measures 80-120 µ x 5.6 µ at birth and grows little until it attains a size of 900-1300µ x 35-40 µ.

Life cycle:

-Habitat: small intestine of definitive and intermediate hosts.

-Definitive host and intermediate hosts: man, pigs, rodents and other carnivorous mammals.

-Reservoir hosts: rodents.

-Infective stage: infective trichina capsule.

-Stages of the life cycle: larva → trichina capsule → adult.

-After fertilization males die and are expelled. Females penetrate deeply in the mucosa and lay larvae (0.1 mm). Each female lays about 1500 larvae in its life span (about 2 months).

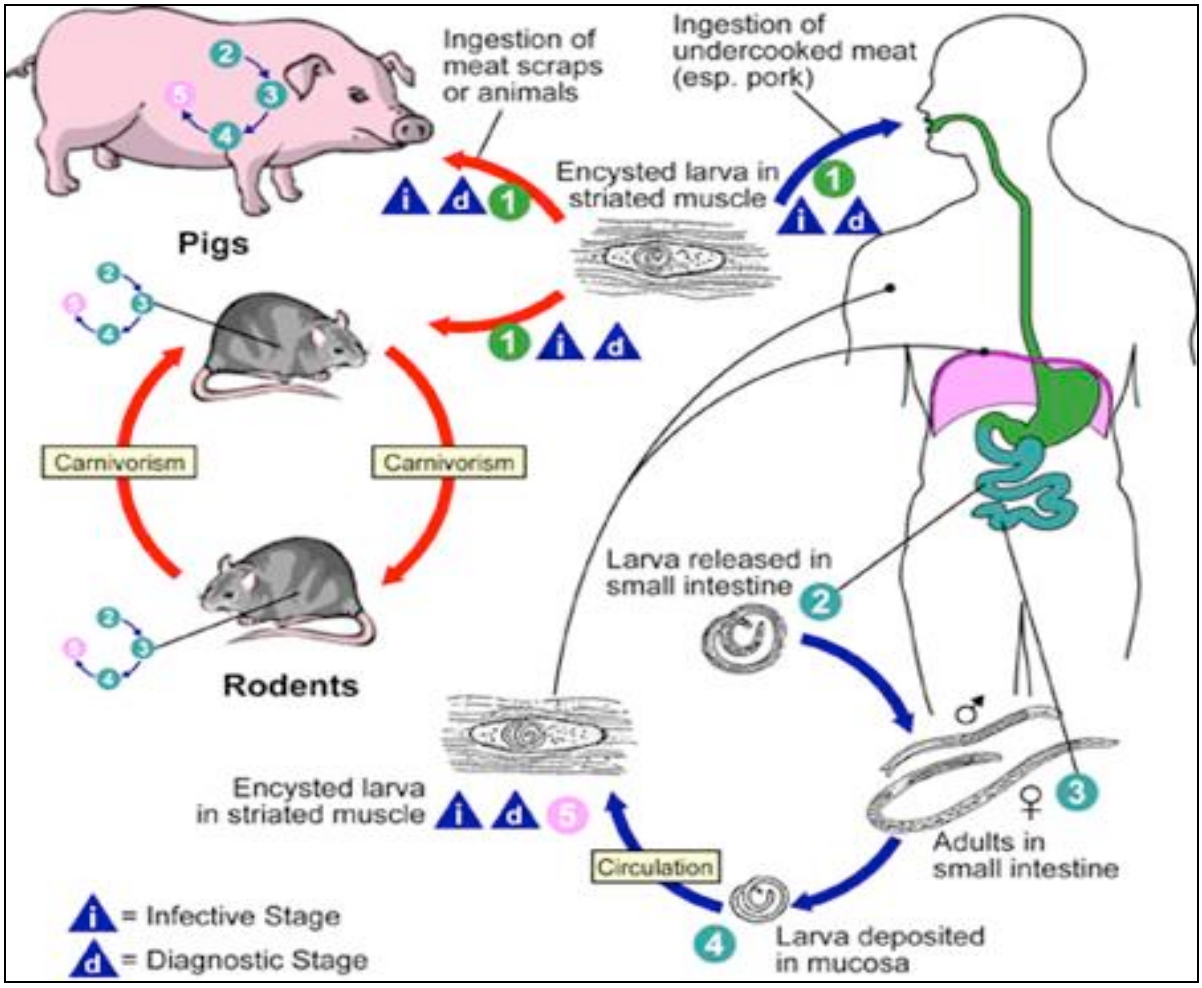
-Larvae find their way to the circulation, through the pulmonary filter and distribute all over the body, particularly the active striated muscles where they encyst.

-Larvae coil and encyst in the long axis of the muscles. The larva grows from 0.1 to 1 mm taking about 2 weeks to become infective.

Mode of infection:

- •Man is infected by ingestion of raw or undercooked pork containing infective larvae.
- •Pigs become infected by eating infected flesh from other pigs in garbage or ingestion of infected dead pigs and rats.
- •Rats are infected by eating flesh of dead pigs or rats and by cannibalism.

-Ingested larvae are liberated from the cysts in the small intestine where they mature to adults.



***Trichinella spiralis* life cycle**

Trichina capsule

- It is larval stage of *Trichinella spiralis*.
- It is common in the striated active muscles as diaphragm, intercostal, deltoid, laryngeal and extra-ocular muscles.
- It is ellipsoidal in shape and 0.5x0.2 mm in size
- It contains a larva about 1 mm in length coiled upon itself.
- It becomes infective after 17 days from reaching the muscles.
- Trichina capsule becomes calcified within 18 months but the larva inside remains viable for years.

Pathogenesis and clinical picture:

Pathogenesis of *Trichinella spiralis* infection can be considered in three phases:

a. **Penetration of adult females** into the mucosa (stage of intestinal invasion).

b. **Migration of the larvae** (stage of migration).

c. **Penetration and encystment** of the larvae in muscle cells (stage of muscular penetration and encystment).

- Light infections (less than 10 larvae/gm muscle) are usually asymptomatic.
- Heavy infections (from 50-100 larvae/gm muscle) may show the following stages:

1. GIT phase (1st week):

Due to intestinal invasion by adult worms

Symptoms resembling food poisoning ;such as, nausea, vomiting, sweating and colic.

This period usually terminates with facial oedema and fever.

2. Trichinosis: (in 2nd, 3rd week)

Due to larval deposition, migration and encystation

It is tetrad of:

1. Muscle pain + weakness (myositis): involve active skeletal muscle, e.g. eye, mastication, respiratory.
2. Facial (periorbital) edema due to toxin vasculitis.
3. Eosinophilia 20-50%.
4. Fever.

3. Final stage

- When the larvae complete encystations all symptoms subside
- In severe cases death occurs, due to myocarditis, encephalitis or pneumonia.

Diagnosis:

I. Clinical (during the first week):Trichinosis is suspected when gastro-intestinal manifestations simulating food poisoning appear in a group of persons with a history of eating pork few days before.

II. Laboratory:

1. Stool examination: (in the 1st week) may detect adult and larvae.

2. Blood examination (during the second week): blood is examined for migrating larvae by adding 20 cc. of 3% acetic acid to 5 cc**.** blood, after centrifugation; examine the sediment for *Trichinella* larvae.

3. Muscle biopsy (after 2 weeks): biopsy is taken from a superficial muscle as the deltoid or intercostals. The excised muscle is examined either by:

a. Compression between 2 slides and examine for trichina capsule.

b. Sectioning and examination for trichina capsule

c. Digestion in artificial gastric juice and the mixture is examined for the free larvae 100 μ in length with cellular oesophagus

4. Intra-dermal test (Bachman's test, after 2 weeks): the antigen used is a purified extract of larvae collected by digestion of the infected pig's meat. Intra-dermal injection of 0.1 ml of the antigen is followed by an immediate reaction in the form of a wheel surrounded by erythema appearing within 10-20 minutes.

5. Serological tests: ELISA and IFA for detection of specific antibodies.

Treatment:

1- Symptomatic:

The patient should rest in bed and given general supportive treatment.

- Sedatives for headache and muscle pain.
- Steroids (cortisone & ACTH) to reduce inflammatory reactions especially in myocarditis and central nervous system involvement.
- Fluids and electrolyte balance should be monitored, since impaired capillary permeability can lead to general oedema and mobilization of fluids

2- Thiabendazole.

3- Mebendazole.

Prevention and control:

1. Thorough cooking of all meat.
2. Regular meat inspection by means of trichinoscopy of pork.
3. Effective treatment of pork by means of refrigeration -15°C at least 20 days.
4. Extermination of rodents from pig farms.

Hook worms

Geographical distribution: Tropical and subtropical countries.

Ancylostoma duodenale

Morphology:

1-Adult:

a. The anterior end is bent dorsally.
b. Large mouth cavity (buccal capsule) with two pairs of teeth at the anterior margin (ventral or upper) and two dental plates at posterior margin (dorsal or lower) and two sub-ventral lancets in its bottom.
c. Club-shaped oesophagus (1/6 the length of the worm).

Male:

- About 1 cm in length
- Has one set of genitals provided with a copulatory bursa (posterior expansion of the cuticle) and two long separate spicules.

Female:

- About 1.2 cm in length.
- Has two sets of genitalia.
- Vulva is at the junction of the middle and posterior thirds of the body.

Necator americanus

Morphology:

1-Adult: -Grayish yellow in colour.

- It has a hook-like anterior end. The head is curved opposite to the body curvature.
- Buccal capsule is armed ventrally and dorsally by cutting plates (dental plates). Four lancets at the bottom (two sub-ventral and two sub-dorsal).

Male: - Measures 8 mm in length and 0.3 mm in diameter.
- The copulatory bursa is long and wide.
- The 2 copulatory spicules are fused distally provided with a blade.

Female: - Measures 10 mm in length and 0.5 mm in diameter.
- Its posterior end is straight.
-Vulva is at the middle of the body.

2- Egg (Diagnostic stage):

Size: 60 x 40 µ. Shape: oval with blunt poles.
Shell: thin shell. Color: translucent.
Contents: immature ovum (4-cell stage).
An empty narrow space existsbetween the content and the shell.

3- Rhabditiform larva:

-About 250-500 µ in length. - Long buccal cavity.
-Rhabditiform oesophagus. - Pointed tail end.

4- Filariform larva:

-About 600-700 μ in length with a sheath
-Club-shaped oesophagus (1/3 body length).
-Sharply pointed tail.
-Does not feed but move.
-Thermotropic, histotropic, phototropic and negative geotropic.
-Present in top layer of soil.

Life cycle:

-Habitat: small intestine (Jejunum).

-Definitive host: man.

-Reservoir hosts: no.

-**Infective stage**: sheathed filariform larvae.

Stages of the life cycle:

Egg → rhabditiform larva → infective filariform larva(IFL) → adult.

- Adults live in the small intestine of man attached by the mouth capsule to the mucosa.
- Immature eggs pass in the feces (20,000 eggs/female/day in *Ancylostoma* and 10,000 eggs/female/ day in *Necator americanus)*.
- Under favorable environmental conditions in the soil (moist shaded areas, sandy or loose soil, alkaline and free of salinity, suitable temperature and sufficient oxygen), a rhabditiform larva develops and hatches in about 2 days (development does not occur in undiluted stools, being acidic).
- It feeds and moults in about 3 days giving another rhabditiform larva (500 μ). It moults again after about 7 days (keeping its skin; ensheathed) to become an infective filariform larva.

Mode of infection:

Man is infected when the filariform larva penetrates his intact skin or mucous membrane of the mouth.

- The filariform larva is attracted to man by histo-tropism and by warmth of the body (positive thermo-tropism). It shows other tropisms to various factors, e.g. negative geotropism and positive hygro-tropism.
- The larvae on reaching the blood are carried to the right side of the heart → the lungs→ penetrate the capillaries into the alveoli→ pass up the tracheal tree, over the epiglottis →swallowed to reach their final habitat in the small intestine.
- During their migration in the lung they moult for the third time, the fourth moult occurs in the small intestine giving the adult stage.

- Eggs appear in stools about 2 months after infection.

Pathology and clinical picture:

Due to larvae		Due to adult
-Ground itching -Cutaneous larvae migrans	-Verminous pneumonia	-Anemia -GIT symptoms

(A)Skin lesion:

- Ground itch: local dermatitis caused by FL penetration at the site of entry or contact with soil (feet, buttocks, hands).
- Cutaneous larvae migrans:
  It is due to migration of IFL in the skin
  Appear as maculopapular rash and itching (± pustules due to 2ry infection).

(B) Lung lesion:

- Löffler's syndrome: caused by Ancylostoma larval migration in the lungs (verminous pneumonitis).
- It is presented by fever, cough, dyspnea, haemoptysis and oesinophilia.
- All symptoms are transient < 2 weeks.

(C) Intestinal lesion:

I- GIT: colic, vomiting, diarrhea.

II- Chronic iron-deficiency anaemia (hypochromic, microcytic) due to blood loss.

Mechanisms of anaemia:

1- Tear by buccal capsule (parasite attached to sucked mucosa, by curved teeth and cutting plates → ulcer → haemorrhage).

2- Anticoagulant secretion by cephalic glands (continued bleeding after detachment).

3- Toxic bone marrow depression.

4-Enteritis (due to 2ry infection) decrease absorption of iron.

III- Complications

- Hypoproteinaemia (due to loss of proteins in blood)& subcutaneous oedema.
- Physical and mental retardation.
- Heart failure (due to tachycardia and hypoxia).

Diagnosis:

I- Clinical: the clinical picture though characteristic is not sufficient to differentiate it from the nutritional deficiency anaemia and oedema from other helminthic infection.

II-Laboratory: depends upon finding the eggs in feces.

Treatment:

1- Mebendazole (Vermox) or Flubendazole.
2- Pyrantel pamoate (combantrin).
3- Supportive treatment: Iron, vitamins and high protein diet.

Prevention and control:

1- Mass treatment of the infected population.
2- Sanitary disposal of human faeces and not to use them as fertilizer.
3- Wearing shoes and gloves for people handling mud or working in mines, gardening, poultry and brick-making.

Differences between hook worms (*A. duodenale* and *N. americanus*)

	Ancylostoma duodenale	***Necator americanus***
Distribution	Tropical and subtropical	More tropical (south America).
Adults: size:	Larger: male 1 cm, female 1.2 cm.	Smaller, male 0.8 cm ,female 1 cm.
Anterior end:	Slight bent dorsally.	Strongly bent against body curvature
Buccal capsule:	Broader than long. Ant. 2 pairs of teeth. Post. 2 dental plates, 2 sub-ventral lancets.	Longer than broad. Ant. 2 cutting dental plates. Post. 2 dental plates, 2 sub-ventral and 2 sub-dorsal lancets.
Spicules:	2 long separate.	2 fused distally with a blade
Vulva:	Post. 1/3 of body.	Middle of body.
Number of eggs/day	20,000/female/day	10,000/female/day.
Life span:	8 years.	4 years.

Case study:

A 41-year-old farmer presented with vague gastrointestinal complains, fatigue, weakness, pallor and loss of weight. The physician ordered a stool analysis. Three stool specimens, collected on alternate days, were submitted for examination for ova and parasites. Blood sample was also drawn for complete blood count.

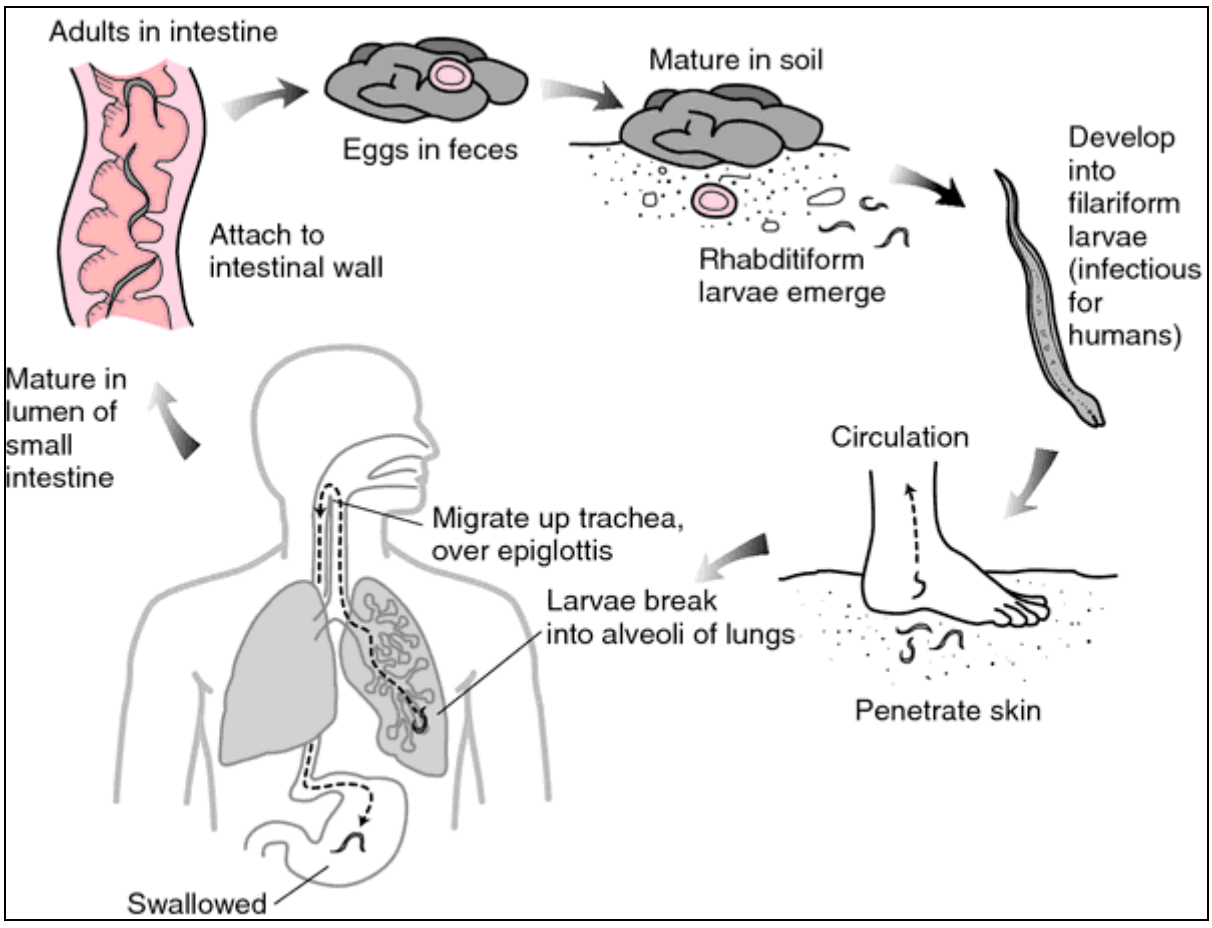
A moderate number of eggs were detected with occult blood in the stool. Each is oval, translucent with blunt poles and clear extra-embryonic space. A single larva was also observed in one stool specimen. This specimen had sat overnight at room temperature before being examined. Hematology result revealed hemoglobin of 10.0 gm/dl.

Questions:

1. Based on the patient's symptoms and morphology of the detected

eggs, which parasites are possible causes of the patient's symptoms?

2. How is this infection transmitted to humans?
3. Describe the life cycle of these parasites.
4. Would you expect to find both eggs and larvae of these parasites in an infected patient's stool specimen? Explain.
5. Describe the 2 larval stages of these helminths.
6. Which other nematode has larval stages, in the stool that may be confused with the larvae of these parasites?
7. What are the causes and type of anaemia that may occur in children heavily infected with these parasites?
8. How is this infection treated?
9. How can you control this parasitic infection?



Life cycle of hook worms

Strongyloides stercoralis

(Dwarf Thread worm)

Geographic distribution: Tropical and subtropical areas.

Morphology:

1- Adult:

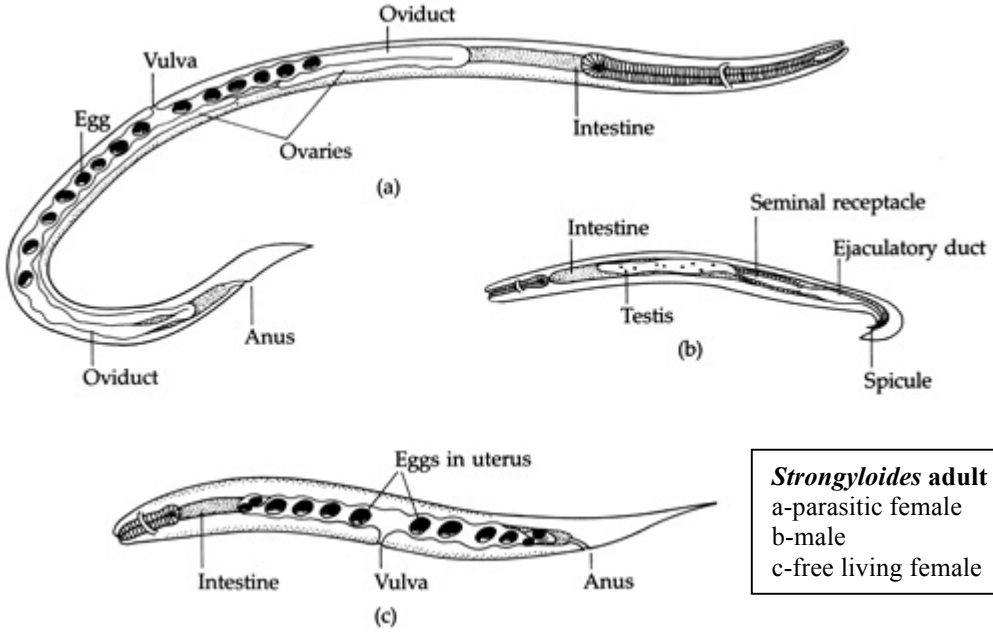
Male (parasitic or free-living):0.7 mm in length with rhabditiform oesophagus. Posterior end curved ventrally and it has two spicules and a gubernaculum.

Parasitic female: 2.2 mm in length with cylindrical oesophagus (1/3 body length) posterior end straight.

-It has two sets of genitalia with 2 uteri full of eggs; the vulva opens at the junction of the middle and posterior thirds of the worm.

Free-living female: 1.1 mm in length with rhabditiform oesophagus and two sets of genitalia with two uteri full of eggs.

-The vulva opens at the middle of the body.



***Strongyloides* adult morphology**
a-parasitic female
b-male
c-free living female

2- Egg: seldom found in stool; sub-mucosa or in soil, hatches after 2 hours.

Size: 55x30 µ.

Shape: oval, very thin shell.

Colour : translucent.

Contents : mature rhabditiform larva.

Life cycle:

-Habitat:

- Parasitic adults live in the small intestine. Fertilized females are deeply embedded in the mucosa, where they oviposit; and males live in the lumen of the intestine.
- Free living adults occur in the soil.

-**Definitive host**: man.

-**Infective stage**: infective filariform larvae.

-**Stage in the life cycle**: egg → Rhabditiform larva → infective filariform larva or free living males and females.

- After fertilization the male dies and the female burrows deeply in the mucosa of duodenum and jejunum.
- Eggs are laid sub-mucosal, hatch into rhabditiform larvae within few hours → migrate into the lumen and pass to the faeces.
- Rhabditiform larvae after giving filariform larvae are infective to man **(Direct cycle)** or give rise to free living forms **(Indirect cycle)** when the free environmental conditions are suitable where they give rise to egg in soil then rhabditiform larvae and so on.
- After penetration of the IFL through the skin or the mucous membrane, they reach the circulation. Larvae are carried by the blood → the pulmonary capillaries and become extravasated in the alveoli. They ascend along the bronchial tree → the larynx and nasopharynx. Larvae become swallowed to reach their final habitat in the small intestine. Some of the larvae may remain in the alveolar tissue reaching maturity and initiate another cycle. Life span is 5 years.

Differences between hookworm and *Strongyloides larvae*

	Hookworm	***Strongyloides***
Rhabditiform larva: Size: Presence in fresh stool:	 250 -500 µ Not found in stool	 200 µ Found in stool
Buccal cavity:	Long (as long as ant. body width).	Short (1/3 ant. body width).
Time to become filariform larva:	6-7 days	2-3 days
Filariform larva: Size : Length of oesophagus:	 600 µ 1/3 body length	 500 1/2 Body length
Tail tip:	Pointed	Notched
Sheath:	Present	Absent

Mode of infection:

Infection with *Strongyloides stercoralis* can occur by:

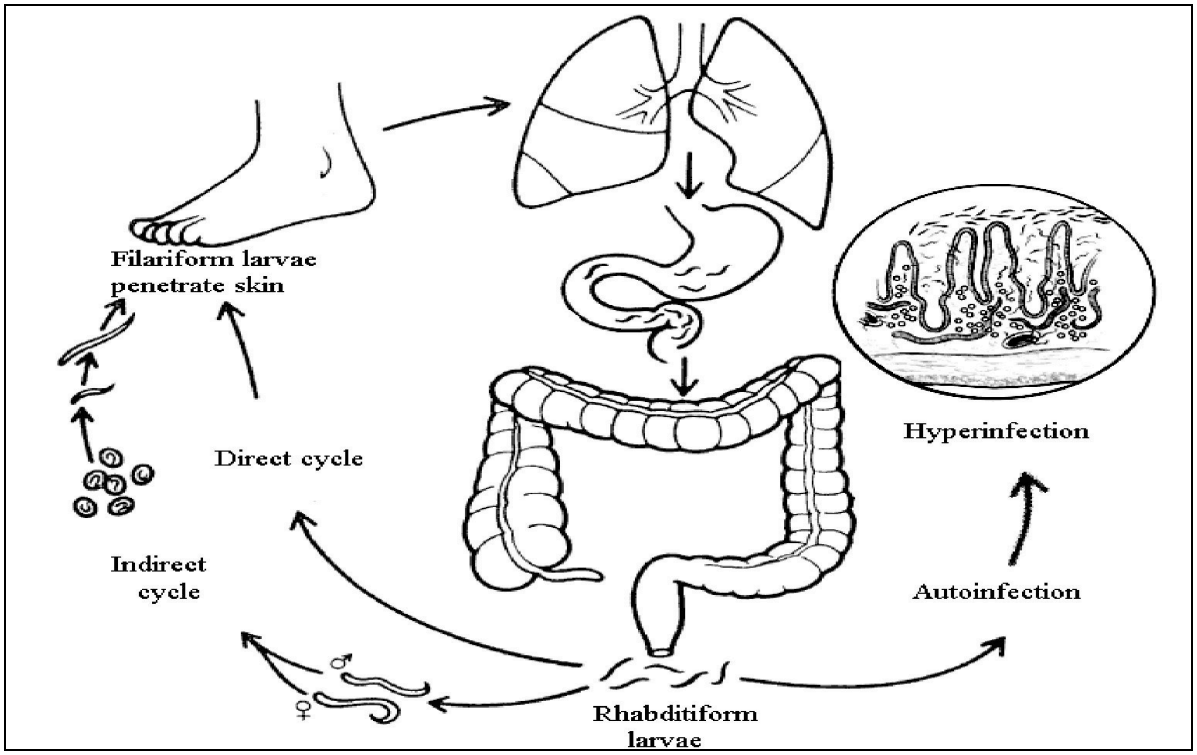
1 - Infective filariform larvae penetrating the skin of man when he gets in contact with infected soil. This occurs while walking bare foot, gardening or setting naked on the soil.

2- Autoinfection occurs by the infective filariform larvae that develop in the intestine of the patient. Development of rhabditiform larvae to infective filariform larvae in the intestine of the patient occurs in cases of constipation and intestinal disturbances.

Autoinfection may be:

a) **Exogenous autoinfection:** when infective filariform larvae develop outside the anus and penetrate the perianal skin.

b) **Endogenous autoinfection:** infective filariform larvae develop in the large intestine and penetrate the mucosa of the intestine. Autoinfection causes **hyper-infection** especially in immunosuppressed patients.



Strongyloides stercoralis **life cycle**

Pathogenicity and clinical picture:

1. Lesions resembling the ground itch of hook-worm infection are seen following penetration of the skin.
2. Pneumonitis may be produced by the larvae, but as in hook-worm infection, is generally less severe than in ascariasis.
3. The adult worms in the intestine may cause no symptoms or moderate to severe diarrhea (chronic intermittent painless diarrhea).
4. Malabsorption syndrome with steatorrhea can occur . Ulceration of the intestinal mucosa may give rise to symptoms resembling those of duodenal ulcer, sometimes with melena and anaemia, or ulcerative colitis.

5. Hyper-infection may lead to severe debilitation or death and the larvae that are found in virtually all parts of the body may give rise to ectopic strongyloidiasis (in lung or kidney).

Diagnosis:

1- Stool analysis:

- Demonstration of rhabditiform larvae (diagnostic stage) (or occasionally filariform larvae) in freshly passed stool.
- Direct fecal smear is often effective in cases of massive infections and various concentration techniques (zinc floatation and centrifugation), increase the chance of finding larvae.
- Eggs may be seen in the stool rarely after purgation or in severe diarrhea.
- Stool culture (to see the larvae and adults).

2-Duodenal aspiration may be done in mild infection (the larvae passed in the stool are few), for detection of eggs and rhabditiform larva.

3- Blood picture may show eosinophilia of 10-40 %

4- Sputum examination shows the larvae especially if the adults are present in lungs.

Treatment: Mebendazole (vermox)

Prevention and control: similar to hookworms

Case study :

A 57-year-old patient had suffered from multiple myeloma for several years and had undergone bone marrow transplantation. Initially appeared to be recovering well following the surgery, but several weeks later he presented with symptoms of intermittent painless diarrhea, cough, dyspnea and abdominal pain. The patient's pulmonary complaint was diagnosed as chronic obstructive lung disease, and he was treated with high dose of intravenous steroids.

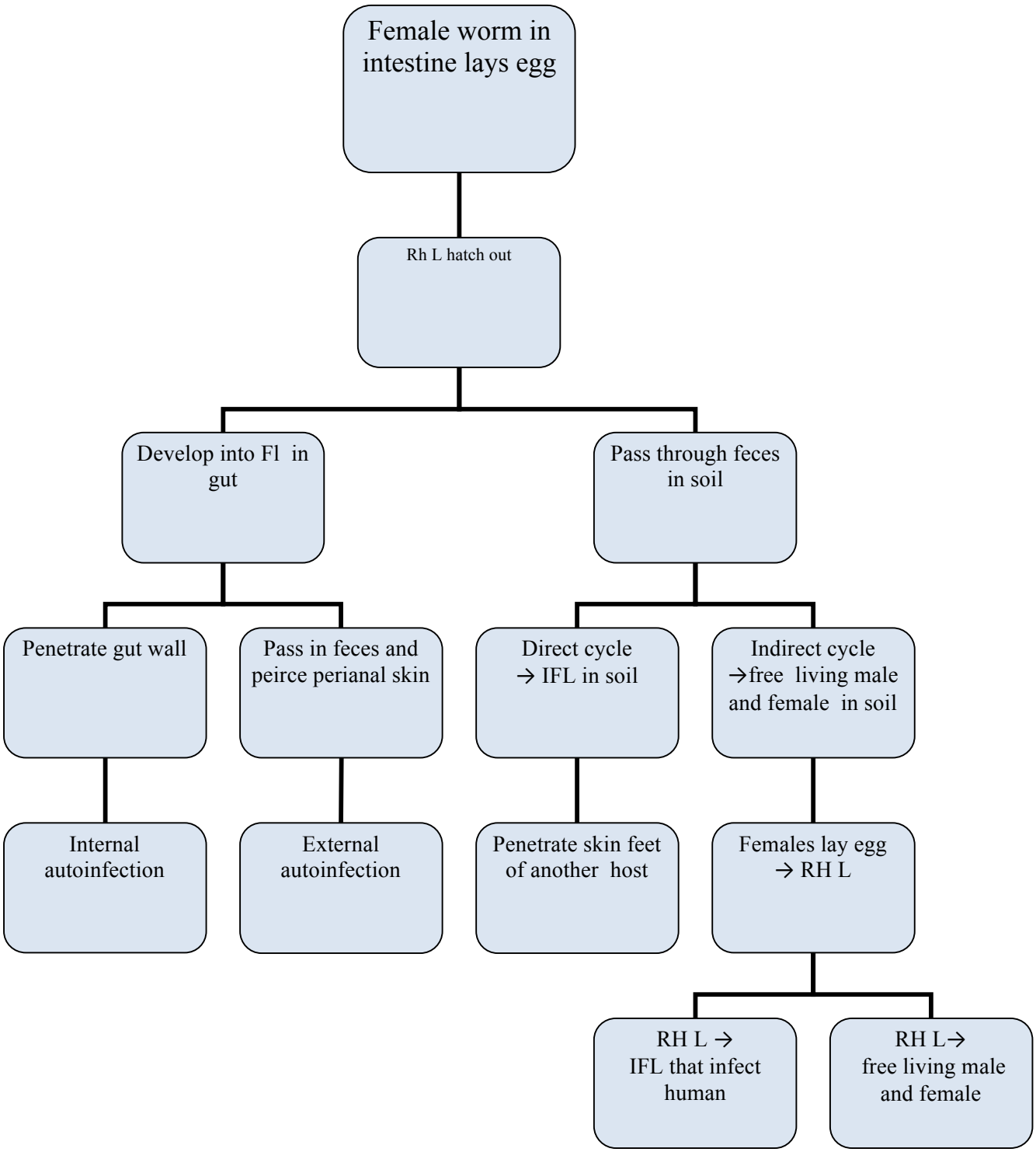
One month later, the patient was re-admitted with fever, and wheezing. Although treated aggressively with antibiotics, the patient's condition deteriorated and he died 30 days after being readmitted.

A complete autopsy was performed and microscopic analysis showed nematode larvae in his internal organs including small intestine, lungs heart and liver.

Questions:

1. Which nematode is most probably responsible for this disease?
2. How is this infection transmitted to humans?
3. Describe the life cycle of this parasite.
4. Describe the characteristics of hyper-infection syndrome caused by this parasite.
5. How do you diagnose this type of parasitic infection?
6. Which other nematode larvae may be confused with this parasite?

7. Which role might the steroid therapy have played in the severity of this patient's illness?
8. How is this infection treated?



Life cycle of *Strongyloides stercoralis*

Larva migrans

Definition: it is the migration of nematode larvae in unsuitable hosts, thus they can't complete their normal development into adults but provoke tissue reaction with the formation of parasitic granuloma. Also larva migrans may occur in natural host under unsuitable conditions.

Larvae migrans in man includes two types:

- Cutaneous larva migrans.
- Visceral larva migrans.

Cutaneous larva migrans
(Creeping eruption)

Definition: it is the invasion of human skin by the infective filariform larvae of *Ancylostoma caninum* and *A. braziliense.* Since these larvae are of non-human nematodes, they fail to penetrate the skin beyond the germinal layer.

Other causes:

1. Autoinfection with the filariform larvae of *Strongyloides stercoralis* may give rise to tortuous urticarial wheals of thighs and trunk.
2. Cutaneous myiasis due to larvae of *Gasterophilus* and *Hypoderma.*

Pathogenicity and clinical picture:

1. At the point of larval penetration, indurated, reddish, itchy papules develop and in 2-3 days, narrow, linear, slightly elevated, erythematous, serpiginous intra cutaneous tunnels 1-2 mm in diameter are produced, larvae move for about an inch daily and vesicles form along the course of the tunnels. Secondary bacterial infection occurs.
2. Feet, hands, back, buttock and legs are the most commonly affected sites.
3. Infection persists from weeks for up to a year then the surface becomes dry and crusty, healing occurs leaving linear scans.

Treatment:

1. Thiabendazole (Mintezol) administered topically or systemically.
2. Freezing the area of active migration with ethyl chloride or carbon dioxide snow.
3. Treatment of secondary bacterial infection by antibiotics.

Prevention and control:

1. Avoid skin contact with soil that has been contaminated with dog or cat faeces.
2. Deworming of dogs and cats.

Visceral larva migrans (VLM)

Definition:

It is a clinical syndrome resulting from invasion of human viscera by non human nematode larvae or human nematode larvae under unsuitable conditions.

Causative agents:

1. Accidental swallowing of larvated eggs of *Toxocara canis* and *T. cati* by man.
2. Larvae of non human *Ancylostoma* can cause visceral larva migrans if swallowed with contaminated food and drink.
3. *Strongyloides stercoralis* although is a natural human nematode, its larvae may cause larvae migrans when missed its way after autoinfection.
4. Visceral larva migrans may be produced by some larvae of *Ascaris lumbricoides, Ancylostoma duodenale* and *Strongyloides stercoralis* in cases of heavy infection. During their cycles they pass from the lung to the left side of the heart, then to the systemic circulation and settle in different organs.

Pathogenicity and clinical picture:

1. The 2nd stage rhabditiform larvae hatch in the small intestine, penetrate the mucous membrane and are carried with blood to the liver, lungs, brain, eyes, heart and other tissues where they produce eosinophilic granulomatous lesions. Larvae remain for several weeks or months without any development till they die.
2. The characteristic lesion has been mostly encountered in the liver and consists of a gray, elevated, circumscribed area approximately 4 mm in diameter. Microscopically these granulamatous lesions consist of eosinophils, lymphocytes, epithelioid cells, and foreign-body giant cells surrounding the larvae.
3. The disease is mostly asymptomatic with persistent eosinophilia of 20-60% in adults.
4. The commonest manifestations are mostly in children of 1-4 years old.

1. General:

a- Fever.

b- Eosinophilia (20-80%) in 80% of cases, anemia &elevated white blood cell count.

c- Elevated erythrocyte sedimentation rate.

d- Hypergammaglobulinemia.

2. Local:

a- Liver: enlarged tender liver.

b- Lung: cough, dyspnea and fever (Loeffler's syndrome), in over 50% of cases and last for l week, patchy pneumonitis may be detected.

c- Eye: visual disturbances due to granulomatous ophthalmitis, peripheral retinitis, iritis, choroiditis and hemorrhage. The eye lesions are always unilateral.

d- Brain: epilepsy and neurological disturbances.

e- Intermittent pain and dermatitis.

Diagnosis:

I. Clinical: Triad of marked eosinophilia, hepatomegaly, and hypergamma-globulinemia with history of close contact with the soil, cats or dogs and of dirt eating is suggestive.

II. Laboratory:

1- Needle liver biopsy → larvae are not numerous enough to be found in biopsy.
2- Elevated serum Ig E levels.
3- Intradermal allergic test.
4- Serological tests: IHA, IFA, ELISA using *Toxocara* larval antigen.

Treatment:

1. Thiabendazole (Mintezol).
2. Diethylcarbamazine (Hetrazan).
3. Corticosteroids may be added also, for a limited period of time in severe cases of ocular infection.

Prevention and control:

1. Small children should be protected against contact with infected dogs and cats.
2. Animals should be de-wormed with Piperazine periodically.
3. Elimination of stray animals.

Case study:

A 4-year-old girl was seen by her pediatrician for a routine physical examination. Her mother was concerned about her daughter's poor appetite. Physical examination revealed that the child was small for her age and had a slightly enlarged liver. Blood sample was collected for a routine complete blood count, and the result revealed that she has eosinophilia (21%). When questioned about pets, her mother reported that she spent a great deal of time with her puppy. Suspicion of a helminthic infection caused, the physician send bloodsample to a reference laboratory, where a serological test was performed to confirm the diagnosis.

Questions:

1. Which parasitic infection might be responsible for this patient's symptoms?
2. Which is the significance of the child having a puppy?
3. Name the 2 species of this genus which cause human disease.
4. Enumerate the other nematodes causing this disease.
5. How is this infection acquired?
6. Describe the life cycle of this parasite.
7. How this infection is usually diagnosed?
8. How is this infection treated?
9. How can this infection be prevented and controlled?

Trichostrongylus Colubriformis

Geographical distribution: cosmopolitan, mainly in agricultural countries as Egypt, Iraq and Iran.

Morphology:

1- Adult:

-Hair-like and yellowish in colour.

-Buccal capsule is attenuated with club-shaped oesophagus.

Male: 4 mm, posterior end is provided with copulatory bursa and 2 triangular spicules. Has one set of genitalia.

Female: 8 mm. Vulva opens at posterior 1/4, has double sets of genitalia, with pointed posterior end.

2-Egg:

Size: 90x45 μ.

Shape: oval, with one pole pointed and the other is rounded.

Shell: thin.

Colour: translucent.

Content: immature embryo in the morula stage (32-cell stage) with wide space between shell and contents.

3-Filariform larva:

-600-700 μ.

-Has a club-shaped oesophagus and a minute knob at the posterior end.

Life cycle:

Habitat	Upper part of small intestine.
-Definitive host	Man.
-Reservoir host	Sheep, cattle, camels and goats.
-Infective stage	Filariform larva.

-Stages in the life cycle: eggs → rhabditiform larva → filariform larva → adult.

Mode of infection: **Ingestion** of infective stage with water or vegetables.

- The ingested filariform larvae exsheath in the small intestine, moult, penetrate villi and stay for 3-5 days.
- They come back to the intestinal lumen, moult and develop to adult males and females within 3 weeks. There is no pulmonary migration.
- Females are oviparous. Eggs appear in the stool 5 weeks after infection.
- In the soil, under favorable conditions, eggs give rise to rhabditiform larvae and hatch after 2 days. After 2 moults→sheathed filariform larvae.

Pathogenicity and clinical picture:

1. Mild inflammation with small ulceration of the intestinal mucosa.
2. Usually infection is mild or asymptomatic.

3. Abdominal pain and gastrointestinal disturbances.

4. Intestinal hemorrhage may occur with anaemia.

5. Rarely the adult may invade the biliary passage leading to biliary colic.

Diagnosis:

I. **Clinical: clinical picture as mentioned before.**

II. **Laboratory**:

1. Stool analysis for the characteristic egg (diagnostic stage) which must be differentiated from that of hook worms.

2. Stool culture for larvae. Rhabditiform larva should be differentiated from those of hookworms and *S. stercoralis*.

Treatment:

1. Thiabendazole.

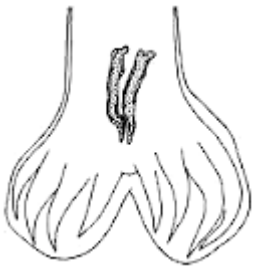
2. Pyrental pamoate.

Prevention and control:

1. Proper washing of raw vegetables and hands before eating.

2. Water purification.

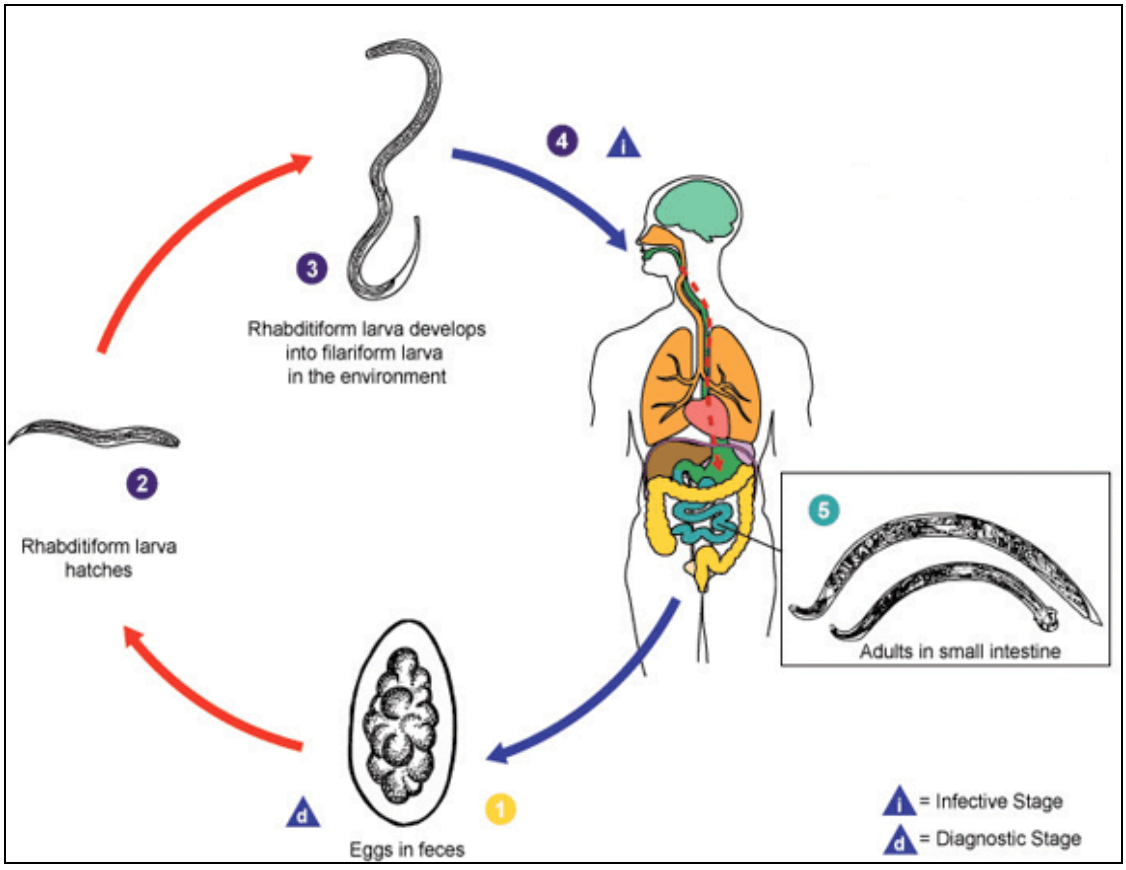
3. Proper storage of food.



***Trichostrongylus* male posterior end**

Differences between hookworms and *Trichostrongylus* eggs.

	Hookworms	***Trichostrongylus***
Size:	60x40 μ (3:2)	90x45 μ (2:1)
Shape:	Oval Blunt poles	Oval One pole pointed and the other is blunt
Colour	Translucent	Translucent
Content:	4-cell stage	32-cell stage
Space between egg shell and the embryo	Narrow	Wide



Life cycle of *Trichostrongylus*

Capillaria philippinensis

Geographical distribution: the parasite has been reported in Philippines, Thailand, single isolated cases were reported in Iran and Egypt.

Morphology:

1-Adults: very small, males measuring 1.5-3.9 mm long and 23-28 µ maximum width, while adult females are 2.3-5.3 mm long and 29-47 µ maximum width.

2-Eggs: peanut shaped 20x40 µ, with a striated shell and flattened bipolar plugs.

Life cycle:

-Habitat: the parasite lives embedded in the mucosa of the upper small intestine of definitive host, primarily the jejunum.

-Definitive hosts: man and birds.

-Intermediate host: small fresh or brackish water fish.

- The life cycle may be either indirect (involving an intermediate host) or direct (complete in one host).

-**Females are both oviparous** (egg-laying**) and larviparous** (giving birth to active

larvae) and the faeces of infected persons may contain all forms of the parasite.

-When eggs reach fresh or brackish waters they embryonate, ingested by fish, hatch in the intestine and develop to infective larval stages.

-When infected fish is eaten, larvae develop into adult worms inside the mucosa and start larviposition in about 2 weeks (1^{st} generation larvae).

-These larvae remain within the host's intestine and develop into egg-laying adults, while some females continue to produce larvae.

Mode of infection:

1. By eating of raw or inadequately cooked infected fish containing 2^{nd} stage larva.
2. Internal auto-infection: when the offspring produced by adults can re-infect the same host, allowing the infection to multiply within a single host.

Pathogenicity and clinical picture:

- In intestinal capillariasis the villi are blunt, flattened or completely obliterated with deepening of the crypts and inflammatory submucosal infiltrate.

-Some cases may be asymptomatic or mildly symptomatic with:

1. Anorexia, nausea, vomiting and hypotension.
2. Abdominal pain, diarrhea, flatulence are characteristic.
3. **Protein-loosing enteropathy** occurs in advanced disease with severe metabolic and nutritional imbalance that can be fatal.
4. There may be hypoproteinaemia, low blood calcium, potassium and cholesterol levels.

Diagnosis:

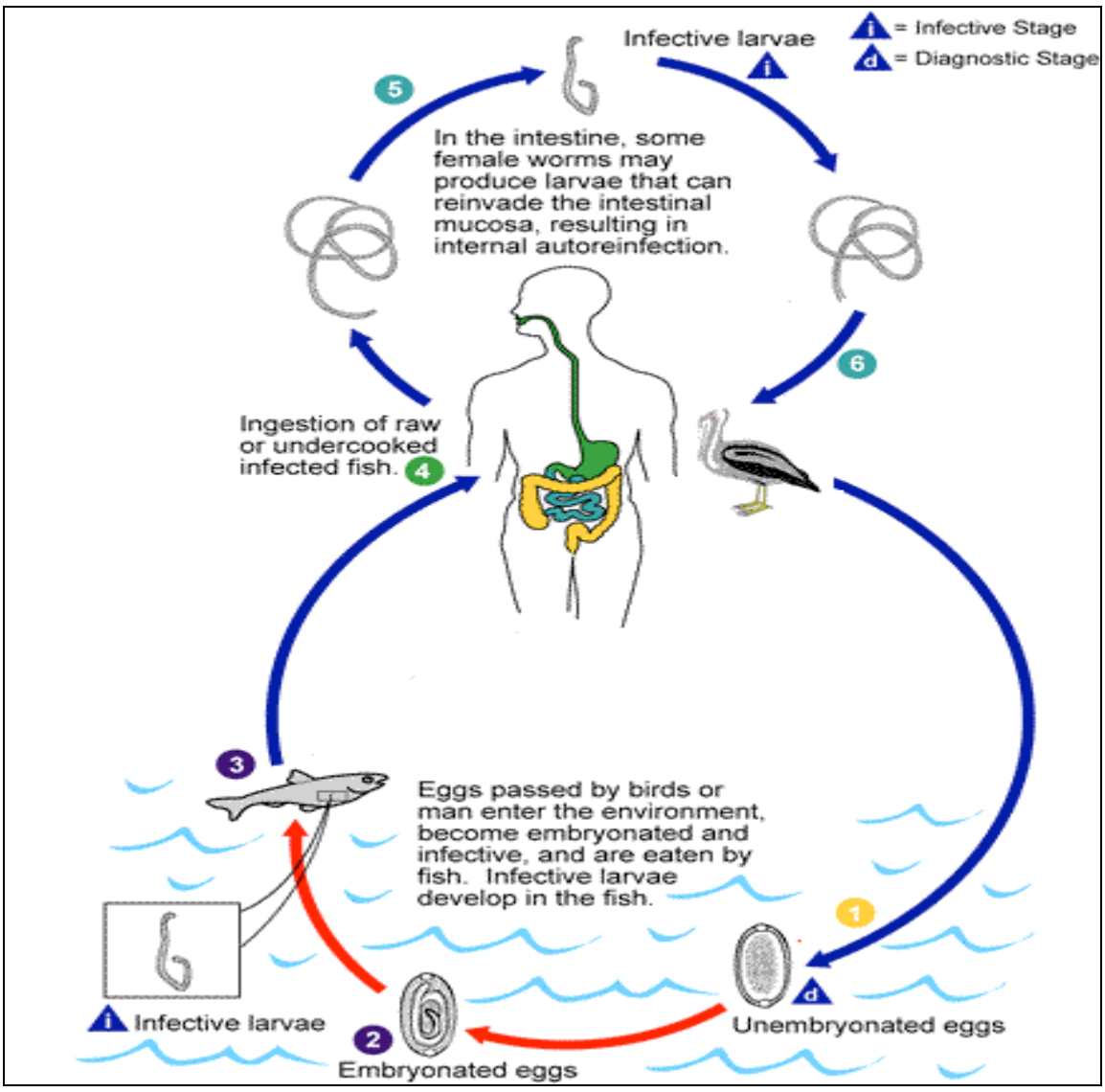
1-Finding the characteristic eggs, larvae or adult worms in the stool.

2-Electrolyte imbalance: ↓ serum potassium, calcium , sodium &↓ total proteins.

Treatment:

Adequate treatment must be given to prevent recurrence from developmental forms in the mucosa.

1. Mebendazole is the drug of choice.
2. Albendazole.
3. Fluid and electrolyte replacement, high protein diet.



Life cycle of *Capillaria philippinensis*

Filarial worms

General morphology:

1- Adult:

- Thread - like in shape.
- Creamy white in colour.
- Anterior and posterior ends of male & female are provided with sensory papillae.
- Mouth: simple without lips and buccal cavity.
- Oesophagus: cylindrical.

Male: - About 4 cm in length and 0.2 mm in diameter.
- Curved posterior end.
- 2 unequal and dissimilar spicules.
- Subterminal cloaca.

Female: - About 8 cm in length and 0.4 mm in diameter (except *Onchocerca volvulus* may reach 50 cm)
- 2 sets of genitalia.
- Vulva anterior in the oesophageal region.
- Subterminal anus.
- Viviparous give birth to microfilaria.

2- Microfilaria (MF):

- Highly motile, thread- like pre larva containing columns of nuclei.
- microfilariae are either sheathed or unsheathed.
- The morphology of the microfilaria, location in the host and type of periodicity is of value in differentiating species.

3- Infective larva (filariform larva):

- 1500 - 2000 x20 μ.
- Cylindrical oesophagus.
- Lies in the labium of the insect vector.

General life cycle:

- The adult worms live in tissues (lymphatics, serous cavities or the subcutaneous tissues).
- Fertilized females lay microfilariae which migrate to the peripheral blood on the subcutaneous tissue to be sucked by a specific insect vector with its blood meal.
- In the midgut of the insect, the microfilariae moult twice and metamorphose into IF L within 1-3 weeks, then they migrate to the labium of the mouth of the insect.
- When this infected insect bite another healthy person, the infective larvae drop on his skin → enter through the bite wound, move through lymphatics, circulation or tissues to the natural habitat where they develop to the adult worms in about 6- 12 months.
- Infection is dependent upon an intermediate host (insect vector) which is necessary for the larvae to complete the development.

- There is **no multiplication** in number of the larvae in the insect only a change in its morphology (**cyclodevelopmental transmission**).

Microfilarial periodicity: periodic appearance of microfilariae in the peripheral blood of the patient at a fixed time of day or night.

Types:

1- Nocturnal periodicity: when largest nnmber of MF occur in blood at night as w.bancrofti& B. malyai

2- Diurnal periodicity: when largest no of MF occur in blood at day time as loa loa

3- Non periodic:

- When MF can be detected in the blood through out the day as M. ozzardi & M. perstans
- When MF not present in blood as in onchocerca (MF present in subcutaneous tissue only)

Note that MF present in lung blood vessels and capillaries during the period where they are not present in peripheral blood

Causes: Many theories were postulated to explain the causes of the periodicity, these are:

1- Time of deposition of microfilariae by female parasite.
2- Nocturnal periodicity may be associated with rest and muscle relaxation.
3- Stagnation of microfilariae in the large lymph vessels during erect position by day time and their release from the effect of gravity during the supine position at night (Khalil's theory).
4- Periodicity may be associated with the feeding habits of the insect vector, a chemotactic substance in the saliva of the insect may attract the microfilariae towards the site of the bite.

Classification and species:

Filarial worms are classified according to their habitat into the following:

1 - Adult living in the lymphatics (and microfilariae in the blood):

a. *Wuchereria bancrofti*.
b. *Brugia malayi* (*Wuchereria malayi*).

2- Adult living in the subcutaneous tissues:

a. *Loa loa* (microfilariae in the peripheral blood).
b. *Onchocerca volvulus* (microfilariae in the subcutaneous tissue).

3- Adult living in the serous cavities (and microfilariae in the blood)

a. *Mansonella perstans* (*Acanthocheilonema perstans*).
b. *Mansonella ozzardi*.

Wuchereria bancrofti

Geographical distribution: Tropical and subtropical countries.

In Egypt it is endemic in some localities, in Damietta (Ezbet-El-Borg), Dakahlia (Meet-Ghamr) and Sharkia (Hehya) Governorates.

Morphology:

1 - Adult: as described before.

2- Microfilaria:

- 300 x10 µ.
- Sheathed, the sheath is loose and redundant (project beyond anterior and posterior ends).
- Bluntly rounded anterior end.
- Tapering posterior end and free of nuclei.
- Nocturnal periodicity (maximum at 12 P.M.).

Life cycle: as described before

-Habitat: adult in lymph vessels and lymph nodes especially that draining lower part of the body, while microfilariae are in the peripheral blood.

-Definitive host: man.

-Intermediate host (vector): mainly female *Culex*, also female *Anopheles* and *Aedes*.

-Reservoir host: no.

-Infective stage: filariform larva in the mouth of infected mosquitoes.

Mode of infection: Through the skin during the bite of infected mosquito.

Pathogenicity and clinical picture:

Disease: Bancroftian filariasis, wuchereriasis, elephantiasis.

- The various pathogenic complications of this disease are mainly due to the adults, the microfilariae seem to have no pathogenic manifestations although they have been associated with granulomatous inflammation of the lung, liver and spleen.

I- Asymptomatic filariasis: this occurs in endemic areas, there is microfilaria in the blood without clinical manifestations.

II- Symptomatic: The main pathological lesions are:

1- Acute inflammatory manifestations:

Due to toxic products of living or dead adult worms with superimposed secondary bacterial infection, it occurs in recurrent attacks and is manifested by:

-Lymphangitis of the genitalia (funiculitis, epididymitis, orchitis and scrotal oedema) with swelling and redness of affected parts.

-Lymphadenitis especially in the groin and axilla.

-Fever, chills, headache, vomiting and malaise.

-Leucocytosis and eosinophilia.

2- Chronic obstructive manifestations:

Due to fibrosis following the inflammatory process , the coiled worms inside lymphatics and endothelial proliferation, this may result in:

- Dilatation of lymphatics leading to **varicosities** especially in genital organs and abdominal wall as hydrocele, scrotal lymphoedema and lymphatic varices.

- Rupture of distended lymphatics (varicosities) e.g. in urinary passages → chyluria, pleural sac → chylothorax, the peritoneal cavity → chylous ascitis, tunica vaginalis of testis → chylocele, intestine → chylous diarrhea.

- Elephantiasis: oedema of the affected part followed by hypertrophy of the skin and subcutaneous connective tissue, the part become hard, tender and the skin becomes thickened, rough, stretched and fissured lead to secondary bacterial infection. It is common in lower limbs and genitalia (scrotum, penis and vulva) rare in arms and breasts.

3- Tropical pulmonary eosinophilia (diffuse filarial lung disease):

-It is caused by immunologic hyper-responsiveness of the host to microfilarial antigens → local destruction of microfilariae in the pulmonary vascular system and diffuse interstitial lung disease.

-Clinically there is dyspnea, cough, asthmatic attacks and eosinophilia, which respond well to treatment with hetrazan.

- Blood examination: microfilariae are not detected in the peripheral blood (**occult filariasis).**

Diagnosis:

I. Clinical: clinical picture as before.

II. Laboratory:

1- Direct:

a- Detection of microfilariae in peripheral blood by the following methods:

- Direct fresh smear under dark ground illumination to see motile microfilariae.

- Giemsa-stained thick blood film to show the stained fixed microfilaria.

- Concentration of microfilariae **(knott's method):** if microfilariae are scanty, 2ml of blood are mixed with 10ml of 2% formalin, allow the mixture to stand for 10 minutes then centrifuge, decant the supernatant and examine the sediment.

b- Provocative test:

Diethylcarbamazine is given orally (2- 8 mg /kg- body weight) to stimulate the microfilariae to circulate in the peripheral blood during day time.

The test is contraindicated in areas with Loa *loa* or *Onchocerca volvulus* coinfection.

The following points should be noted during examination:

- Microfilariae begin to appear a year or more after infection.
- Microfilariae are rarely found when lymphatics have become obstructed.
- Blood must be collected at night (between 10 P.M.- 2 A.M.).
- Microfilariae are highest in capillary blood than in venous blood.
- Microfilariae are more in blood from the ear lobe than from fingers.

c. Detection of microfilariae in chylous urine or from fluid aspirated from hydrocele and peritoneal cavities.

d. Urine examination: collect 10- 20 ml of morning urine, add 2ml ether to dissolve chyle, then centrifuge at low speed and examine the sediment.

e. Detection of adult worms:- Lymph node biopsy.

- X-ray to detect calcified dead worm.

2- Indirect:

-It is used during the incubation period and in late chronic infections when microfilariae are absent from peripheral blood.

-The antigen used is prepared from the dog filaria (***Dirofilaria immitis***) for detection of circulating filarial antibodies using the following tests; IFAT, CFT and ELISA.

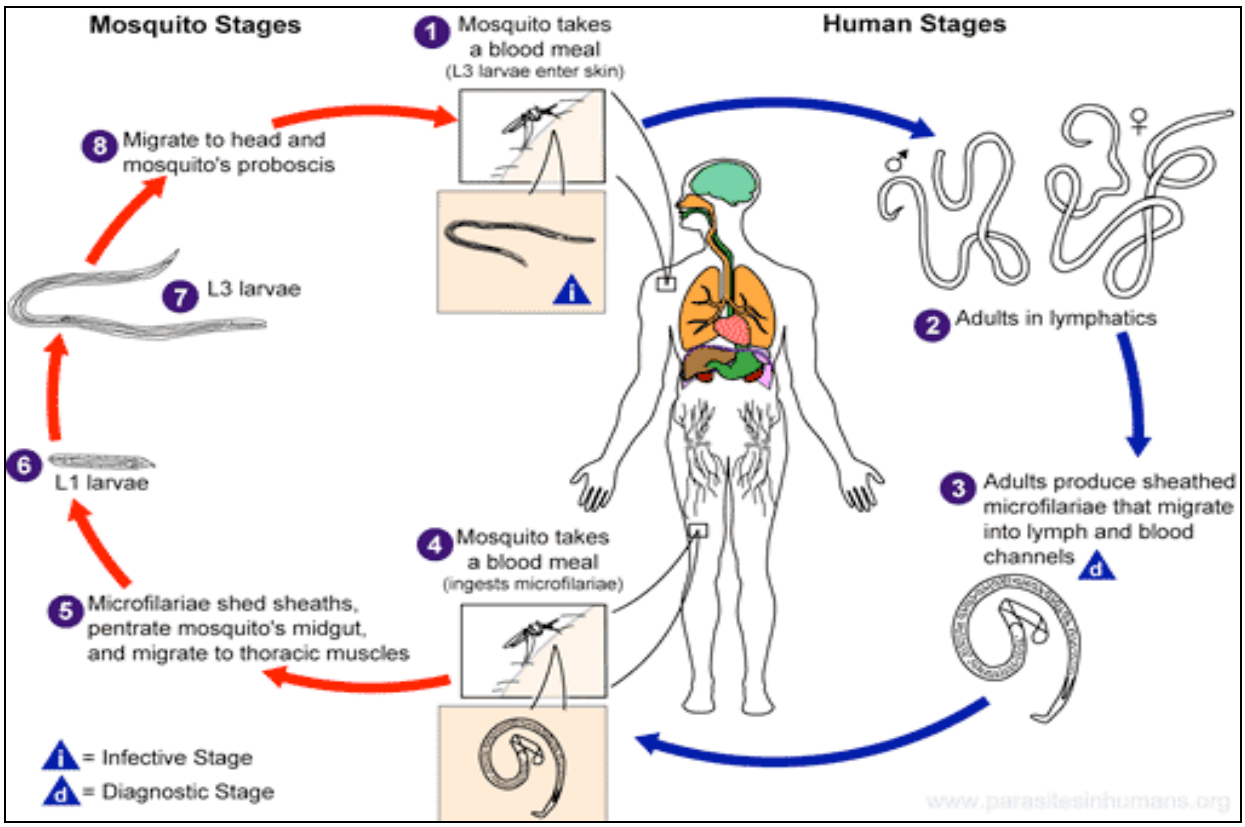
Treatment:

1. Diethylcarbamazine (Hetrazan) is the drug of choice, has a lethal action on microfilariae.

2. Surgical treatment: removal of elephantoid tissue.

Prevention and control:

1- Treatment of patients.
2- Control of mosquitoes.
3- Use of insect repellants and screening.
4- Environmental sanitation.



Life cycle of *Wuchereria bancrofti*

Brugia malayi (Wuchereria malayi)

Geographical distribution: Far East and Asia, not present in Egypt.

Morphology:

1 - Adult: as described before.

2- Microfilaria:

- 250x 5µ.
- Irregular, kinky body curves.
- Sheathed (loose).
- Anterior end provided with 2 stylets.
- Posterior end has 2 nuclei, terminal one is larger giving tail end swollen appearance.
- Nocturnal periodicity (8 P.M. - 4 A.M.).

Life cycle: As described before.

-Habitat: lymph nodes and lymph vessels draining the upper part of the body (upper limb, axilla, breasts and thorax).

-Definitive host: man.

-Intermediate hosts: mainly female *Mansonia* (night biting mosquitoes) also some species of female *Anopheles* and *Aedes*.

-Reservoir host: cats and monkeys.

-Infective stage: infective filariform larva in the mouth of infected mosquitoes.

Mode of infection: through the skin during the bite of mosquito.

Pathogenicity and clinical picture:

Disease: Malayan filariasis It is similar to that of *W. bancrofti* except:

- Elephantiasis affects mainly the breast and arms.
- Genitalia are rarely affected and chyluria is uncommon.

Diagnosis , Treatment and Control : As for *Wuchereria bancrofti.*

Case study:

A 29-year-old man was living in Damietta. He was complaining of fever and swelling in the right lower limb 4 years ago. Clinical examination revealed enlarged right inguinal lymph nodes. Lymphangitis and lymphoedema was observed in his lower limbs and his scrotum with thickened spermatic cord-

The patient's symptoms, combined with his geographical area of origin, created a suspicion of a parasitic infection. Blood sample was drawn and sent to the laboratory for a complete blood count and thin and thick blood smears. Eosinophilia (28%) was observed. Blood smears were stained by Delafield's hematoxylin method and examined microscopically. A few number of sheathed microfilariae averaging 260 u in length with bluntly rounded anterior portions, were revealed. A large number of distinct nuclei were seen in the microfilariae. The nuclei did not extend to the tips of the tails which tapered to a point.

Questions:

1. Which parasite caused this patient's symptoms?
2. Which insects act as the intermediate hosts for these parasites?
3. How is the diagnosis of this infection performed?
4. What is the best time for blood sampling in the diagnosis of this infection?
5. What is the value of the provocative test in this infection?
6. Describe the morphological characteristics of other sheathed microfilariae which may be detected in a blood smear.
7. Why was Delafield's hematoxylin stain used?
8. Which drugs may be used to treat this infection?

Loa loa (The African eye worm)

Geographical distribution: restricted to the tropical rain forests, in west and central Africa, not present in Egypt.

Morphology:

1- **Adult**: As described before.

2- **Microfilaria:**

- 250x5 µ.
- Kinky body curves.
- Sheathed (tight).
- Posterior end is S- shaped and full of nuclei.
- Diurnal periodicity (at mid- day).

Life cycle: As described before.

-Habitat: adults live free in subcutaneous tissues (including back, chest, axilla, groin, scalp and eyes), microfilariae in the blood.

-Definitive host: man.

-Reservoir host: monkeys.

-Intermediate host: **female *Chrysops*** (daily biting fly).

-Infective stage: infective filariform larva in the mouth of *Chrysops* flies.

Mode of infection: through the skin during the bite of insect.

Pathogenicity and clinical picture:

Disease: Loiasis. It has different clinical manifestations:

1- Skin: Calabar (fugitive) swelling:

- Transient painless non pitting swelling appears suddenly and disappears after 3 days to appear again.
- It is lemon- sized.
- The commonest sites are forearms, hands and head.
- It is due to hypersensitivity (allergic reaction) to the adult worms, microfilariae and their toxic metabolites.
- May be dangerous even fatal if occur in the glottis.

2- Eye: - Moving of the adult worm under the bulbar conjunctiva or crossing the bridge of the nose causes creeping sensation, anxiety, irritation, congestion and lacrimation.

- Granuloma of the conjunctiva (solitary or multiple small nodules, 2mm. in diameter).

- Oedema of the eyelid (painless but itching).
- Proptosis due to oedema of the orbital cellular tissues.

3- Cerebral: *Loa loa* may invade the central nervous system and causes meningoencephalitis. Microfilariae may be present in C.S.F.

4- Joint: acute arthritis with joint effusion and microfilariae are found in the fluid.

Diagnosis:

I. Clinical: visual observation of the adult worm under bulbar conjunctiva or crossing the bridge of the nose

- Presence of calabar swelling

II.Laboratory:

1. Direct:

a- Detection of microfilaria in the blood sample taken during day time and examined by: - Fresh smear for motile microfilaria.
- Stained thick smear for fixed microfilaria.

b- Detection of microfilaria in C.S.F.

2. Indirect: Serological and intradermal tests.

Treatment:

1 - Surgical removal of the adult worm.

2- Chemotherapy as in filariasis bancrofti.

Prevention and control: as *W . bancrofti.*

Onchocerca volvulus

Geographical distribution:

Tropical Africa and South America.

Morphology:

1- **Adult:** As described before, but female is about 50cm in length.

2- **Microfilaria:**

- 300 x 7 µ.
- Non- sheathed.
- The anterior and posterior ends are devoid of nuclei.
- Not seen in the peripheral blood.
- Found in the skin and subcutaneous tissue near the adult.

Life cycle:

-Habitat: adult worms live in fibrous subcutaneous nodules from the host reaction.

-Definitive host: man.

-Intermediate host: ***Simulium*** (Black fly).

-Reservoir host: no.

-Infective stage: infective filariform larva in the mouth of *Simulium*.

Mode of infection: through the skin during the bite of intermediate host.

Pathogenicity and clinical picture:

Disease: Onchocerciasis

I-Manifestation due to the adult worm:

Onchocerca nodule (onchocercoma)

- Smooth firm, painless fibrous nodule in the subcutaneous tissue surrounding one to several adults.
- Appears 3-4 months after infection.
- Single or multiple.
- Rounded or oval, 3cm in diameter.
- Freely mobile.
- Located over the bony prominences as iliac crest, sacroiliac region, knee, shoulders and chest wall.

II-Manifestations due to microfilariae:

A) Skin manifestations:

-These are due to an immunological mechanism to microfilariae.

1. Severe dermatitis:

- Oedema and inflammatory cellular infiltration of the dermis forming granuloma with subsequent fibrosis.
- Severe itching which may lead to secondary bacterial infection.

2. Disturbed pigmentation:

i. Depigmentation (Leopard skin): localized spotty depigmentation, especially over the legs in black colored people.

ii. Abnormal pigmentation in white people.

iii. Hyperpigmentation (Sowda):

- Localized onchocerciasis in Yemen and north Sudan. The infection is localized to one leg.
- The skin is dark and thickened and show papular eruption.
- The regional lymph nodes are greatly enlarged.

3. Lichenoid changes in late stage:

Consists of dryness, scaling, atrophy, scaring, loss of elasticity and deep wrinkling of the skin which tends to hang in folds, this lead to:

a. Premature senility in the American type as these changes occurs mainly in the head and neck region.
b. Hanging groin in Africa.
   These changes occur around the hip region, in which a sac of tissues forms in the inguinal region and may contain enlarged inguinal or femoral lymph glands and may hang down as far as the knee.

B) Ocular manifestations (River or Sudan blindness):

- This is a serious complication of onchocerciasis resulting in blindness.
- Common when the nodules are in the scalp, neck and shoulders.
- The microfilariae have great affinity to the eye tissues.
- It is characterized by keratitis, iritis, uveitis, choroiditis, retinitis and optical atrophy which end in blindness.

Causes:

1- Hypersensitivity to toxins liberated from living and dead microfilariae.
2- Mechanical action of the moving microfilariae in the eye tissues.

Diagnosis:

I- Clinical diagnosis.

II- Laboratory diagnosis:

1- Direct:

a- Detection of microfilaria in:

• Skin - snip biopsy: the best method, a small bit of skin is taken with razor or scissor from skin over the nodule and is examined in a drop of saline.

• Aspiration from the nodules.

-This may give negative results if the adults are dead.

b- Detection of adult worm in tissue biopsy of *Onchocerca* nodule.

2- Indirect:

a- Patch skin test:

- 10% of diethylcarbamazine (Hetrazan) in lanolin cream is applied to an area of skin 5mm in diameter and covered with a dressing.
- In positive cases; papular eruption develop after 8-24 hours due to local allergic reaction to dead microfilariae at the site of application.

b- Mazzotti test:

- A single dose (50-100 mg) of diethylcarbamazine is given by mouth.
- In positive cases skin rash and itching appear within 24 hours due to toxic products of dead microfilariae.
- -In heavy infection, syncope and collapse may occur.
- This test is contraindicated in ocular lesions.

c- Immunodiagnosis: as ELISA.

3- Differential leucocytic count for eosinophilia.

Treatment:

1 - Hetrazan and ivermectin against microfilaria.

2- Suramin against adult worm.

3- Surgical removal of the nodules.

Prevention and control:

1- Control of *Simulium* by insecticides application to water running streams and spraying insecticides along river banks by airplanes.

2- Mass chemotherapy by ivermectin.

3- Mass nodulectomy.

Mansonella (Acanthocheilonema) perstans

Geographical distribution: Tropical Africa and South America.

Life cycle: As described before.

-Habitat: adult worms live in serous cavities mainly in peritoneal cavity and to a less extent in pleural and pericardial cavities as well as mesenteric and retroperitoneal tissues. Microfilariae live in the peripheral blood.

-Definitive host: man.

-Intermediate host: *Culicoides* (night biter fly).

-Reservoir host: chimpanzee.

-Infective stage: filariform larva in the mouth of *Culicoides.*

Pathogenicity: non Pathogenic.

Diagnosis: blood examination for microfilariae.

Treatment: mebendazole.

Prevention and control:

1. Control of *Culicoides* by insecticides.
2. Sleeping in an illuminated room protect from fly bite.
3. Treatment of patients.

Mansonella ozzardi

Geographical distribution: India, central and south America.

Life cycle:

-Habitat: adult worms live in serous cavities, microfilariae in peripheral blood and skin

-Definitive host: man.

-Intermediate host: *Culicoides*.

-Reservoir host: no.

-Infective stage: filariform larva in the mouth of *Culicoides*.

Pathogenicity: non Pathogenic.

Diagnosis:

1 - Blood examination for microfilariae.

2- Skin biopsy for microfilariae.

Treatment: ivermectin.

Prevention and control: As *M. perstans.*

Differentiation of filariae

	Wuchereria bancrofti	***Brugia malayi***	***Loa loa***	***Onchocerca volvulus***	***Mansonella perstans***	***Mansonella ozzardi***
Disease	Bancroftian Filariasis	Malayan Filariasis	Loiasis	Onchocercosis	Non pathogenic	Non pathogenic
Habitat of adult	Lymphatics of lower limbs	Lymphatics of upper limbs	Subcutaneous tissue, free migrating	Subcutaneous tissue in fibrous nodules	Serous Cavities, Mesentery and retro peritoneal tissue	
Microfilaria Size Sheath Appearance Tail end Habitat Periodicity	 300 x 10 µ + (Loose) Smooth Curves Free of nuclei Blood Nocturnal	 250 x 5 µ + (Loose) Kinky curves 2 nuclei Blood Nocturnal	 250 x 5 µ + (tight) Kinky curves Full of nuclei Blood Diurnal	 300 x 7 µ (no sheath) Free of nuclei Skin& subC.T.	 200 x5 µ (no sheath) Blunt, Full of nuclei Blood Non periodic	 150x5 µ (no sheath) Tapering, Free of nuclei Blood &skin Non periodic
I.H.	Mainly *Culex,* also *Anopheles* and *Aedes*	*Mansonia* (*Aedes, Anopheles*)	*Chrysops*	*Simulium*	*Culicoides*	*Culicoides*
Pathogenicity	Lymphangitis and Lymphadenitis, lymphatic obstruction, varices, oedema, chylous effusion and elephantiasis.		-Worms migrating under skin, eye. -Calabar swelling. -Allergic manifestatio-ns.	-Onchoreca nodules. -River blindness. -Skin hypo and hyperpigmen-tation.	No	No
Diagnosis	1- Blood examination for M.F. At night. 2-Hetrazan provocation test. 3-Examination of chylous effusion. 4-Eosinophilia, I.D. and serological tests.		Blood Film (day).	1-Aspirate of nodule or skin snip. 2-Adult in excised nodule. 3-Mazzotti test. 4-Patch test.	Blood films (at any time).	Blood films (at any time).

Dracunculus medinensis

(Guinea worm, medina worm)

Geographical distribution: Saudi Arabia, Yemen, India, Iran, Pakistan, East and West Africa& Egypt, where wells are used for water supply.

Morphology:

1-Adult: thread- like and creamy white.

Male:

- 4 cm x 0.4 mm.
- Coiled posterior end.
- 2 subequal spicules.

Female:

- 60 -120 cm x 1-2 mm.
- Recurved (hooked) posterior end.
- Vulva in the oesophageal region.
- 2 set of the genitalia.

2- Rhabditiform larvae (diagnostic stage):

- 600x20 µ.
- Comma shaped with rounded anterior end and long tapering tail (1/3 body length).
- Rhabditiform oesophagus and striated cuticle.

Life cycle:

-Habitat: deep connective tissue, after fertilization females migrate to subcutaneous tissue.

-Definitive host: man.

-Intermediate host: *Cyclops*.

Mode of infection: ingestion of infected *Cyclops* containing infective larvae with drinking water.

-Reservoir host: cats, dogs, catties and horses.

-**Infective stage**: filariform Larva in the body cavity of *Cyclops*.

Pathogenicity and clinical picture:

Disease: **Dracunculiasis**

1- Local cutaneous lesion develops at the outer end of the tunnel in the form of reddish papule which rapidly becomes a blister, more common at the legs, hands, feet, shoulders and buttocks.
2- On rupture of the blister, a tiny hole remains through which the worm protrudes.
3- Secondary infection along the tunnel leading to cellulites, abscesses, arthritis, synovitis and enlarged lymph glands.

Diagnosis:

I- Clinical diagnosis.

II- Laboratory diagnosis:

1. Direct:-

a. Detection of ulcer in a suspected part through which female worm may be seen.

b. Detection of rhabditiform larvae after putting suspected ulcer in water.

c. X-ray for dead and calcified worm in deep tissues.

2. Indirect: intra-dermal and serological tests to detect circulating antibodies.

Treatment:

1- Manual removal of the prolapsed female

2-Surgical removal of the female through multiple incisions.

3- Subcutaneous injection of the phenodiazine along the course of the female to kill it then fibrosed.

4- Metronidazole.

5- Antibiotics for 2^{ry} bacterial infection.

Prevention and control:

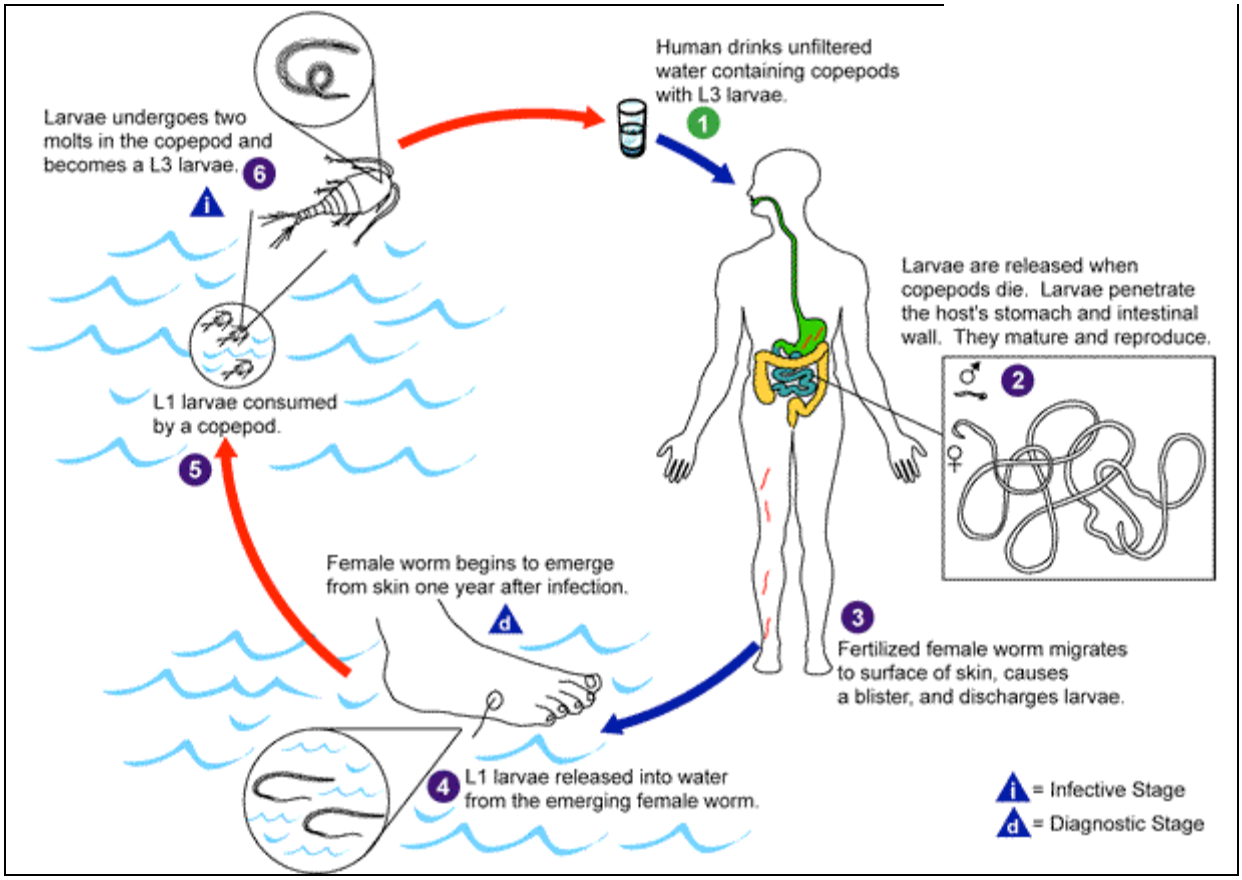
1- Treatment of the patients.

2- Avoid washing and bathing in sources of drinking water.

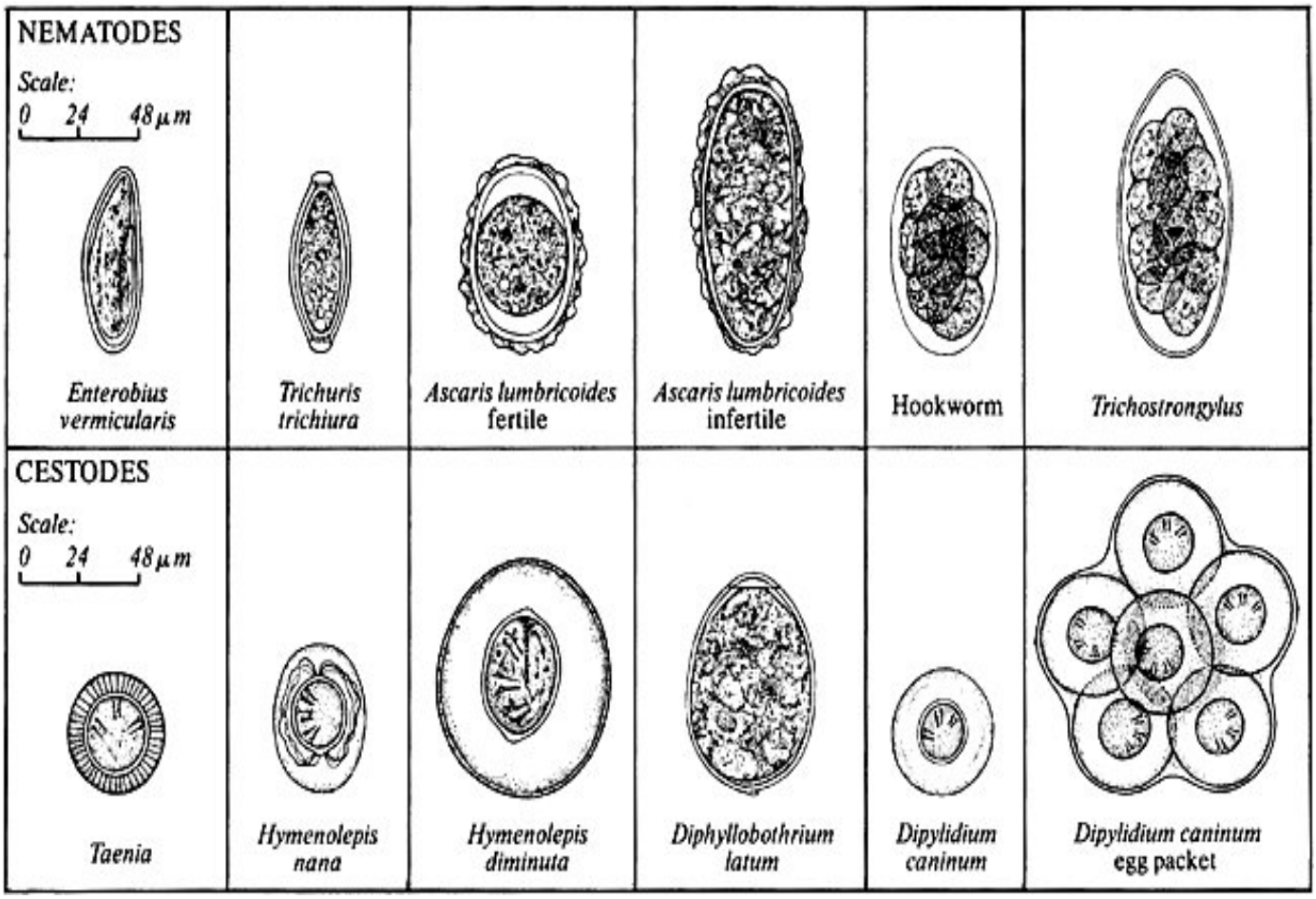
3- Boiling or filtering water before use.

4- Application of calcium oxide, copper sulphate or chlorine to the wells to destroy the Cyclops.

5-



Life cycle of *Dracunculus medinensis*


NEMATODES
Scale:
0 24 48 μm
Enterobius vermicularis
Trichuris trichiura
Ascaris lumbricoides fertile
Ascaris lumbricoides infertile
Hookworm
Trichostrongylus
CESTODES
Scale:
0 24 48 μm
Taenia
Hymenolepis nana
Hymenolepis diminuta
Diphyllobothrium latum
Dipylidium caninum
Dipylidium caninum egg packet