Hookworm lec 1 د ٠ سعيد حميد

Two species of hookworms are human parasites:

1-Ancylostoma duodenale

2- Necator americanus.

Ancylostoma duodenale(Greek ankylos –hooked .stoma -mouth)

The second species Necator americanus ,called the American or

the ‘New World’ hookworm and A.duodenale the ‘OldWorld’ hookworm.

Hookworm disease is prevalent throughout the tropics and subtropics. Even though it has been controlled in the advanced countries, it is estimated that it still a some 900 million people, causing the loss of about 9 million litres of blood overall each day.

A. Duodenale was prevalent along the Mediterranean coast of Europe and Africa, in northern India, China and Japan.

N. Americanus was prevalent in

Central and South America,

Central and Southern Africa,

Southern India,

Ancylostoma duodenale

Morphology

The adult worms live in the small intestines mostly in the jejunum, duodenum,ileum

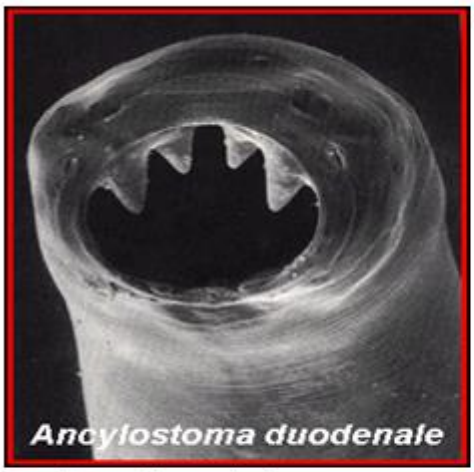
They are pale pink or greyish white ,some time appear reddish brown due to ingested blood .

the body is curved with the dorsal aspect concave and ventral aspect convex,

the cervical curvature gave it the name hookworm,the mouth is not at the tip but directed dorsally.

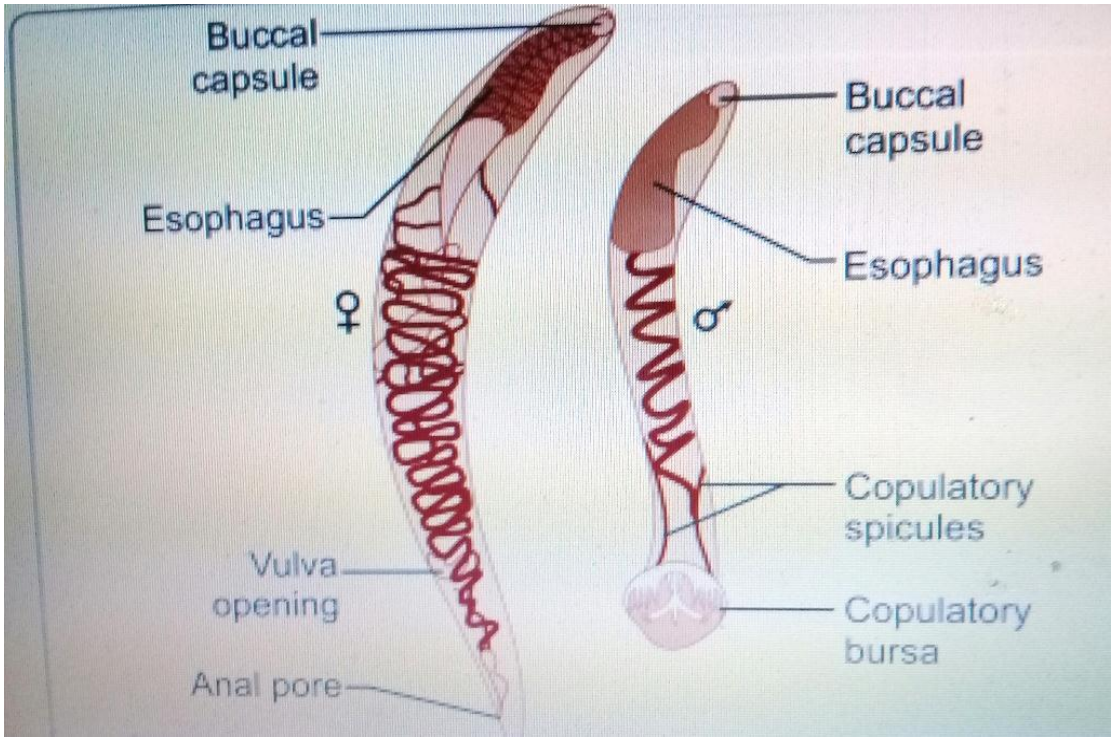
the prominent buccal capsule carries two pairs of hook-like teeth ventrally and a dental plate with a median cleft dorsally

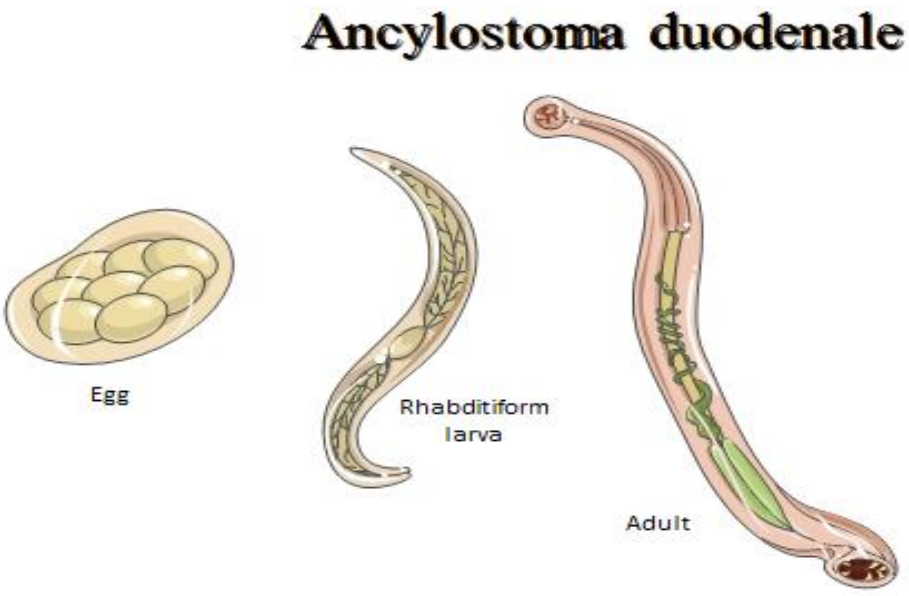
The antterior end is bent in the same direction of the general curvature of the body(c shape single curve)



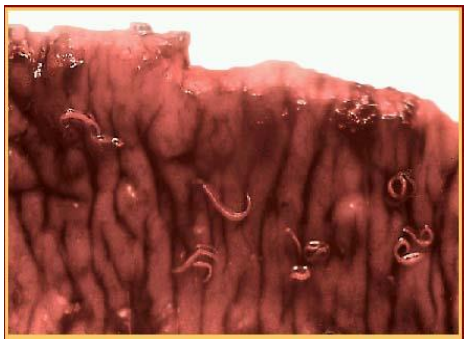
the mouth capsule of *Ancylostoma duodenale*,

the mouth capsule of Necator americanus





Adults in intestinal mucosa



The male worm is about 8-11 mm in length 0.4mm thick. The The posterior end of the male is expanded into a copulatory bursa supported by fleshy rays.The pattern of the rays helps in distinguishing between different species .

Males, smaller than females, have copulatory bursa for grasping female

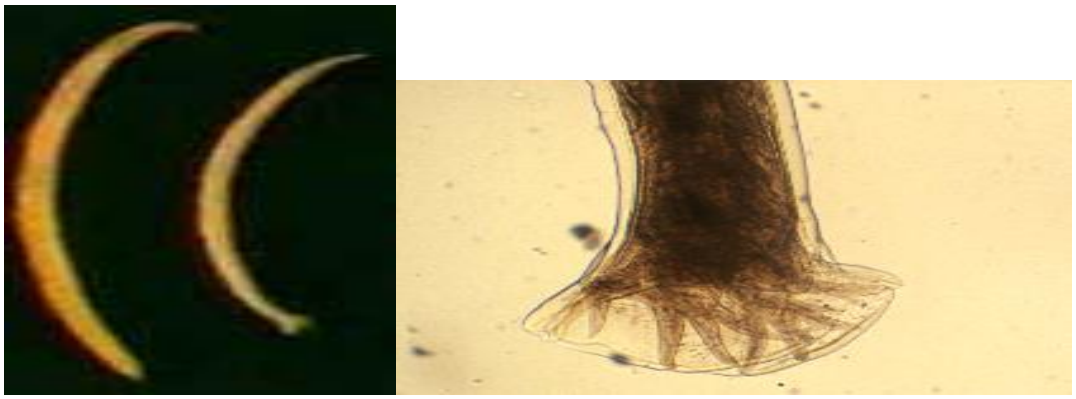
.

The female is larger 10-13mm long 0.6 mm thick tapered at the posterior end .

The anus lies ventrally near the caudal tip and the vulvar opening

at the beginning of the posterior third of the body.

Female lies around 15,000 -20,000 eggs daily and 20-50 million eggs during its life time



Necator americanus

Morphology

The adult worms are smaller than Ancylostoma duodenale .the male 7-9mm long 0.3 mm thick,

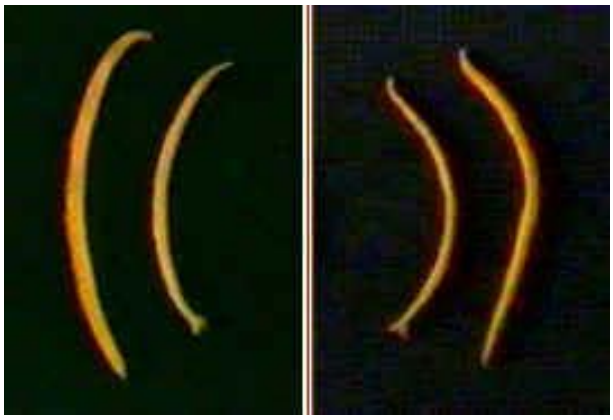
female 9-11mm long 0.4 mm thick.

The anterior end is bent in a direction opposite to the general curvature of the body(s shape double curve) .

the female vulva is placed in the middle of the body,

the eggs and the life cycle are similar in both N.americanus and A.duodenale

Differences between two hookworms



Adults of A. duodenale Adults of N. americanus

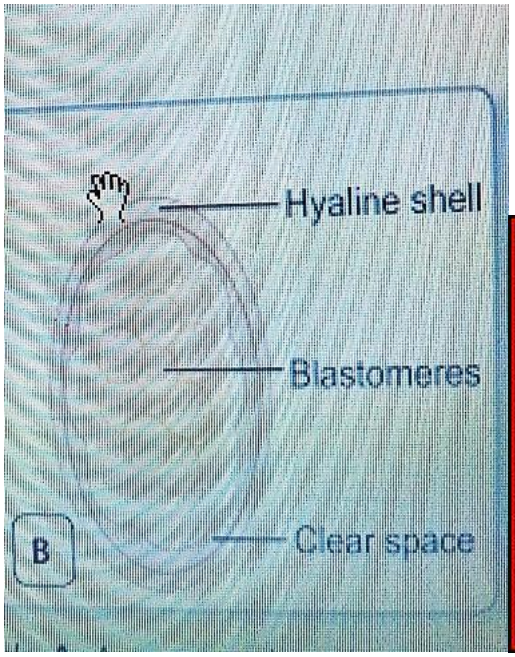
Eggs morphology

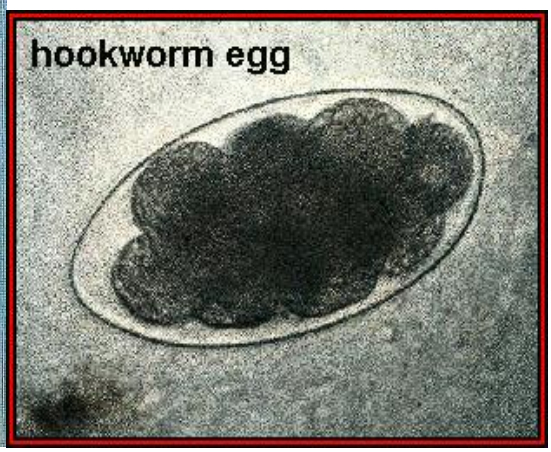
The egg is ovoid measures 60 µm by 40 µm, colourless with a thin transparent hyaline shell membrane,

Eggs when released by the worm in the intestine ,the egg contains an unsegmented ovum ,then developed when passed in feces ,the egg contains a segmented ovum 4-8 blastomeres .

there is a clear space between the segmented ovum and the egg shell .the egg float in saturated salt solution

Morphologically it is not possible to differentiate between *A. duodenale* and *N. americanus*.





differentiating Features of Two Species of Hookworm

	Ancylostoma duodenale	*Necator americanus*
Adult Worms		
Size	Large and thicker	Small and slender
Shape	Head bent in same direction as body	Head bent in opposite direction
Buccal capsule	4 ventral teeth and 2 dorsal knob like teeth	2 ventral and 2 dorsal chitinious cutting plates
Copulatory bursa	13 rays, two separate spicules, dorsal ray single	14 rays, two spicules fused at the tip, dorsal ray split
Caudal spine in female	Present	Absent
Vulval opening	Situated behind the middle of the body	Situated in anterior to middle part of body
Pathogenecity	More	Comparatively less
Eggs	Similar	Similar
1st and 2nd stage Larve	Similar	Similar
Egg/day	15,000–20,000	6,000–11,000
Rate of development	Faster	Slower
Pulmonary reaction	More common	Less common
Blood loss/worm	0.2 mL/day	0.03 mL/day
Iron loss (mg/day)	0.76 mg	0.45 mg
Male:female ratio	1:1	1.5:1
Life span	2–7 years	4–20 years

Helminths requiring no intermediate host

- Ancylostoma duodenale
- Necator americanus
- Ascaris lumbricoides
- Trichuris trichiura
- Enterobius vermicularis
- Hymenolepis nana

Life cycle

Natural host: Humans.

Life cycle is completed in a single host .

the male and female in small intestine ,eggs freshly passed in feces are not infective for humans .

in the sandy soil and ,moist ,warm environment embryo develops inside the eggs in about 2days the egg hatches into rhabditiform larval 250 µm long .

rhabditiform larval feeds on bacteria and other material in soil ,grows in size and moults twice on 3rd and 5th days after hatching to become filariform larva 500-600 µm long (not feeding,infective stage) ,they can live in the soil for about 5 weeks ,waiting for their hosts.

direct sunlight ,drying ,salt water can kill the larvae .

filariform larva penetrate the skin,subcutaneous tissue(between the toes,dorsum of the foot ,skin of the hands).

the larva enter the circulation to right heart –lung - pharynx –During migration or on reaching the esophagus, they

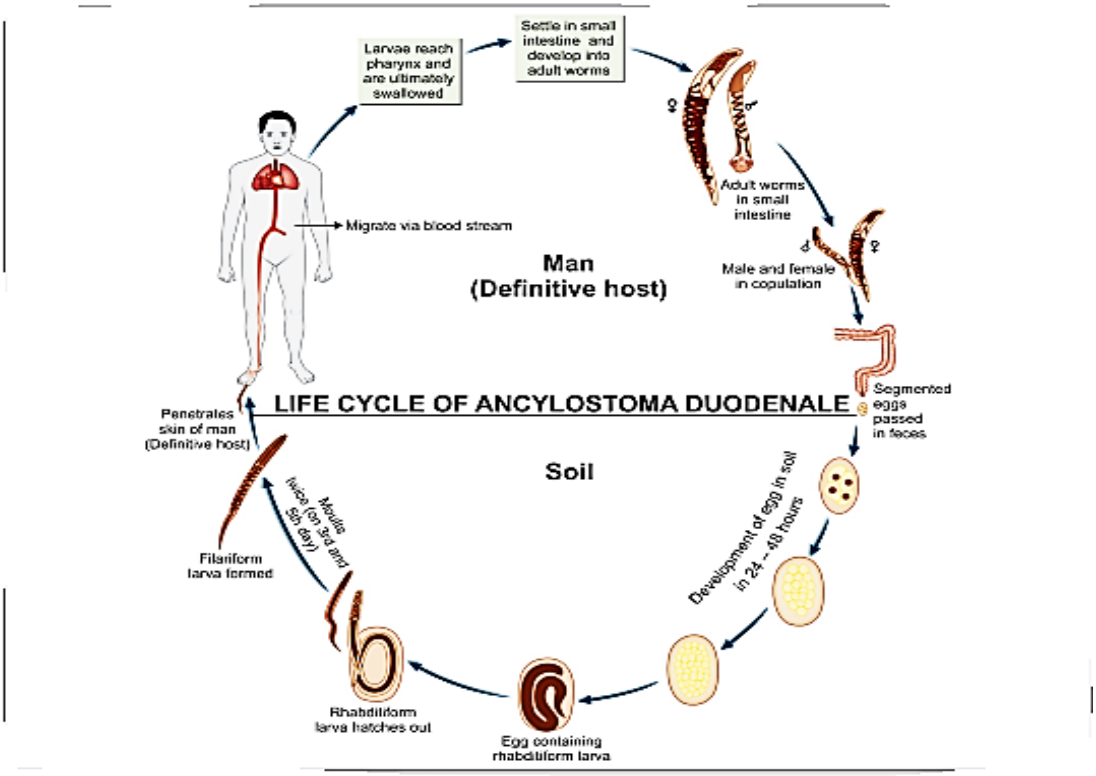
undergo third moulting.

Inside the intestine under go a 4th moulting ,develop the buccal capsule and grow into adult worm

the life cycle takes 6 weeks from time of infection to become adult worm

Rarely oral route infection by ingestion filariform larva enter circulation or larva may be swallowed and develop into adult worm in the small intestine without a tissue phase .

Transmammary and transplacental transmission reported for Ancylostome *duodenale*,but not for **Necator americanus**



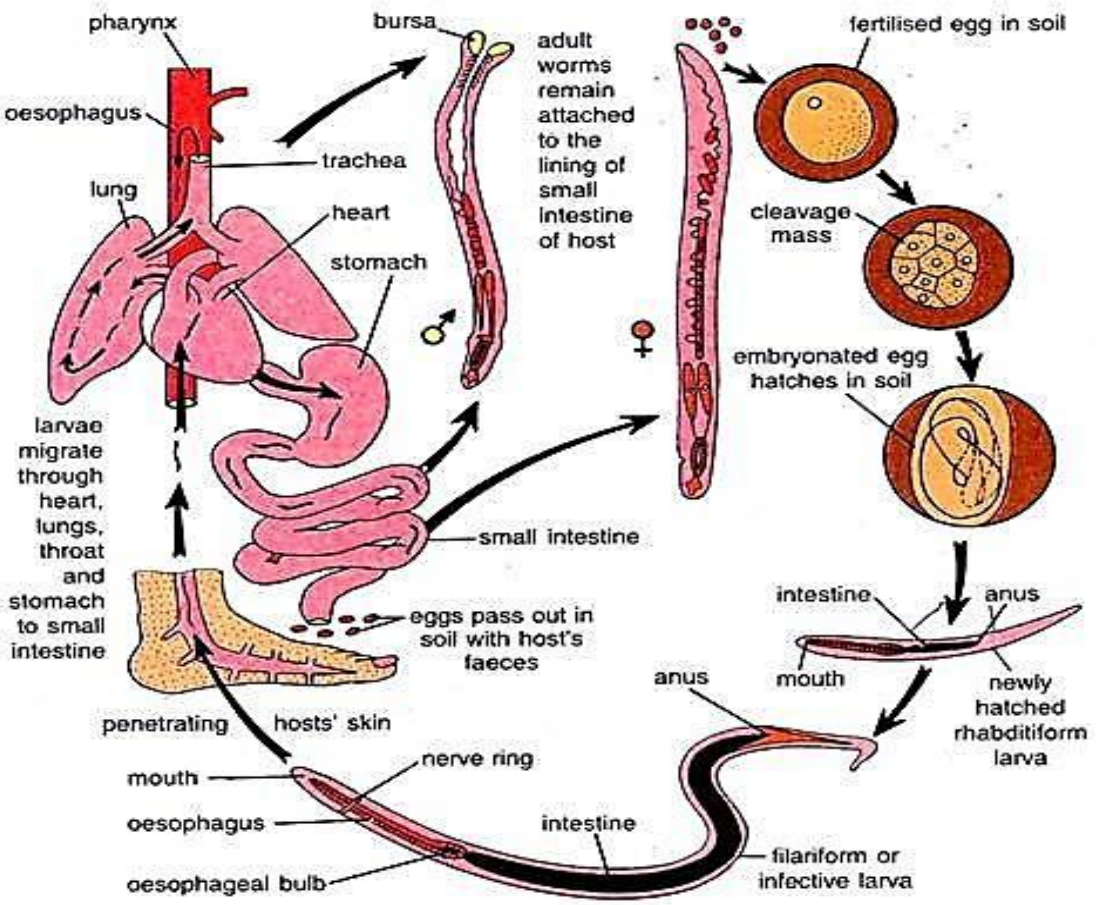
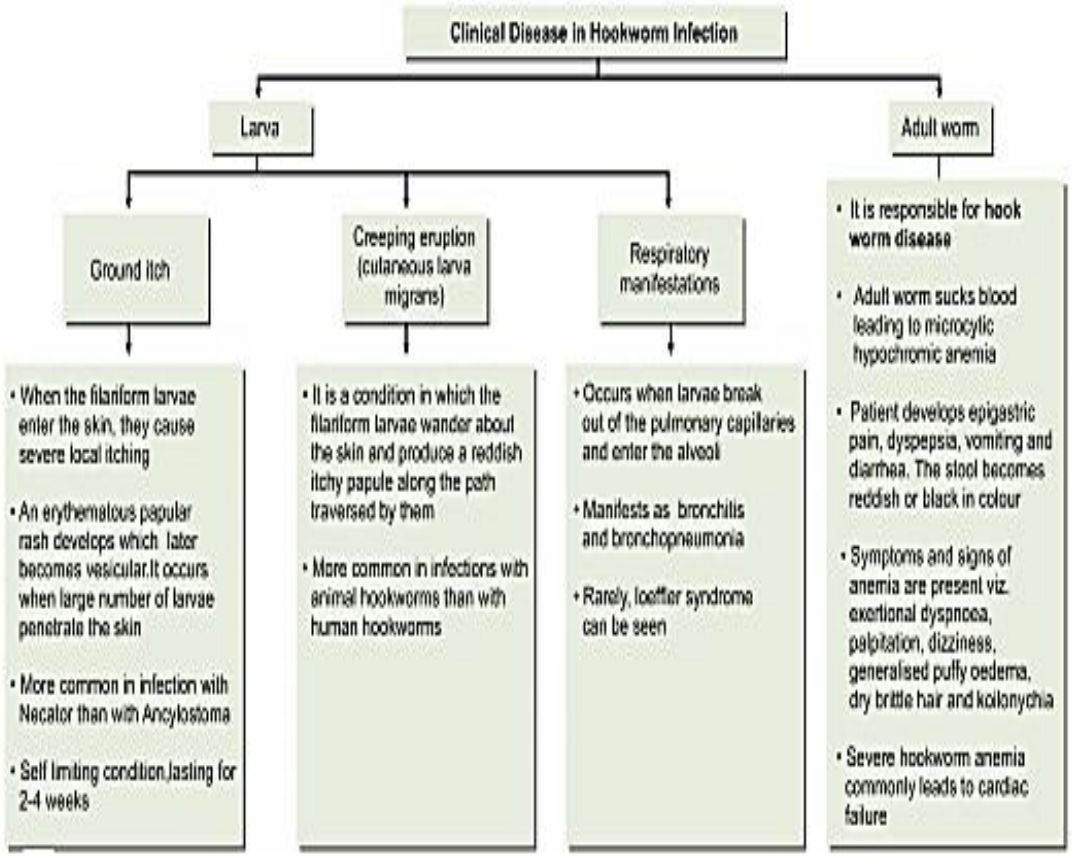


Fig. 47.2. *Ancylostoma duodenale.* Life cycle.



Causes of anemia in Hookworm infection

• Blood sucking by the parasite for their food

• Chronic hemorrhages from the punctured sites from jejunal mucosa

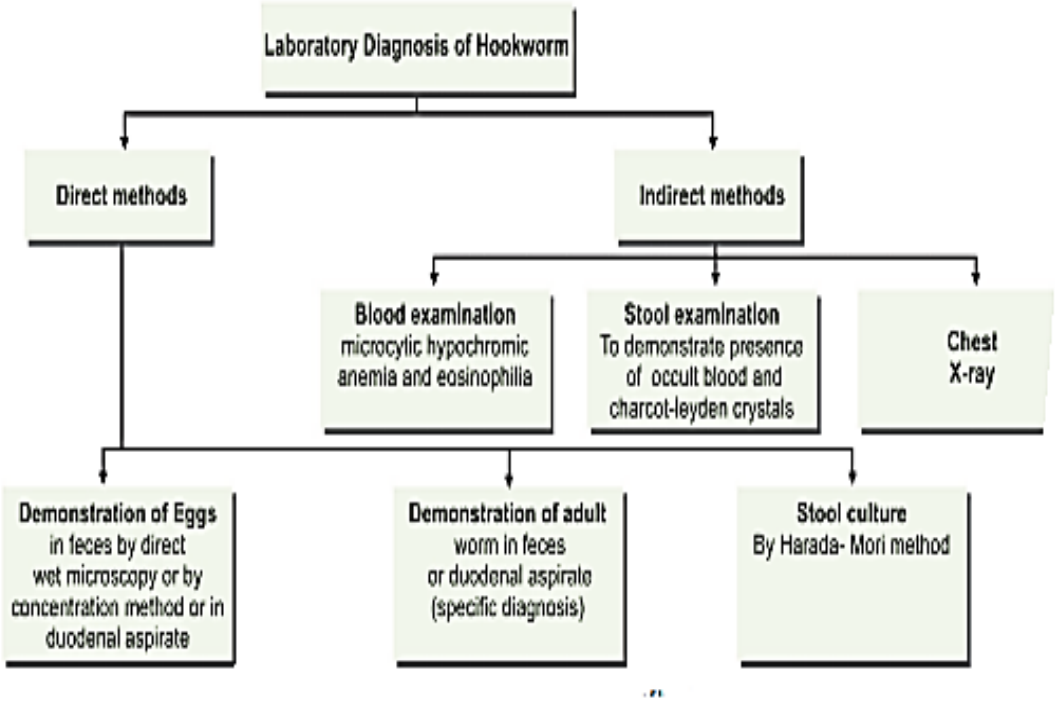
.def cient absorption of vit B12 and folic acid

• Depression of hemopoitic system by defcient intake

of proteins

• Average blood loss by the host per worm per day is

0.03 mL with N. americans and 0.2 mL with A. duodenale



Treatment

1- effective drug is albendazole (400 mg singledose)

or mebendazole (500 mg single dose).

Pyrantel pamoate (10 mg/kg × 3 days) is also effective and can be used in pregnancy.

2- if the patient suffers from anemia oral iron is effective.

Antihelminthic drugs should not be used before correcting the anemia.

3- High protein diet should be given

Prevention

Prevention of soil pollution with feces and proper disposal of night soil .

Use of footwear to prevents entry of larva through the skin of the foot.

Gloves give similar protection to the hands of farm workers.

Treatment of patients and carriers.

There are other species of ancylostoma infect animals like *A. braziliense* species which infect

cats and dogs,

A.ceylanicum infects cats,Some time infected man

Trichostrongylus species infects sheep and goats can also cause human infections.the life cycle is similar to that of hookworms

lec 2 د٠ سعيد حميد Trichuris trichiura

Disease called Trichuriasis,whipworm infection

-Adult worm lives in large intestine

-Natural host: Man is the only host.

-No intermediate host-

the name come (Greek trichos-hair, oura-tail)

Epidemiology

It is worldwide in distribution, but is much more

common in the tropics.

The infection is widespread in tropical Africa, South America, and South-east Asia .

children are more frequently infected than adults

Morphology

The adult worm is flesh colour ,the shape it resembles a whip, with the anterior 3/5 is thin and thread-like .

the posterior 2/5 is thick and fleshy appearing like the handle of a whip .

the anterior portion which contains the capillary oesophagus embedded in the mucosa .

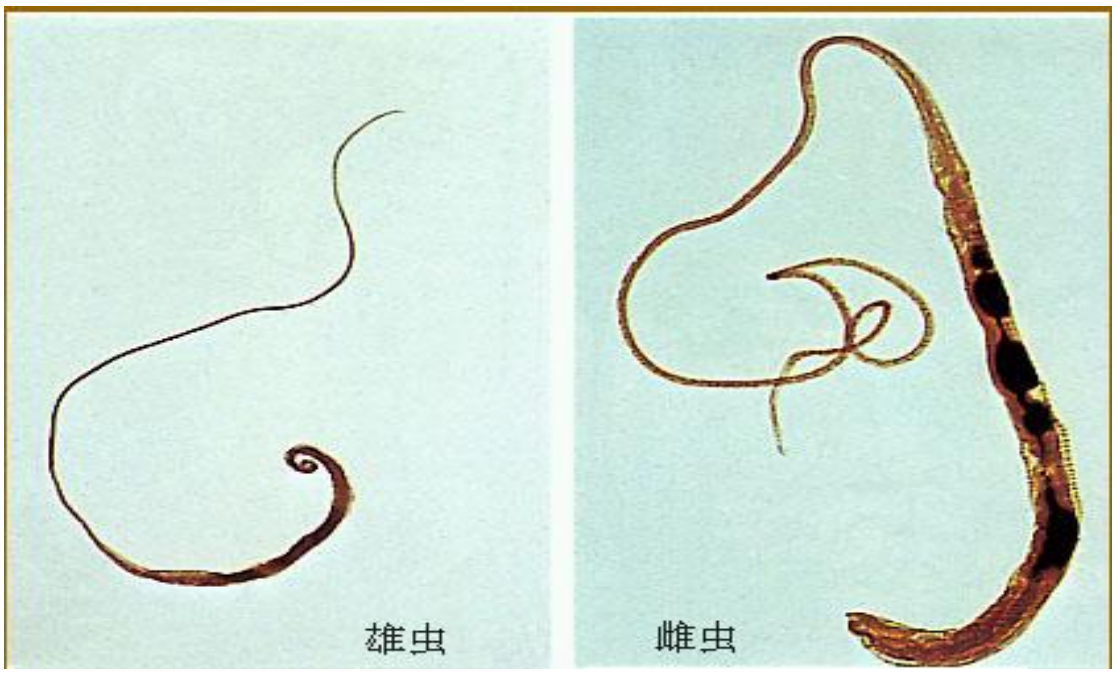
posterior part contains the intestines and reproductive organs

The male 30-45mm long , posterior end coiled ventrally .

The female 40-50mm long ,the posterior end straight ,blunt and rounded

Humans are the only natural host for Trichuris trichiura But similar worms are found in pigs and monkeys

Adults of T. trichiura

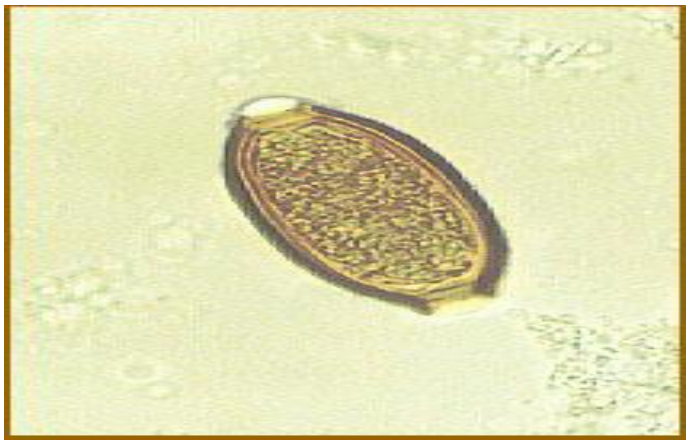


Egg: it is barrel or spindle in shape,about 50µm long 20µm width . It is brownish in colour and has a translucent clear polar plug at each ends.

The content of the egg is an undeveloped cell,

the egg floats in saturated salt solution

The fertilized female lays about 5,000 eggs per day



Helminths whose eggs float in saturated salt solution

• Enterobius vermicularis

• Ancylostoma duodenale

• Necator americanus

• Ascaris lumbricoides

• Trichuris trichiura

Nematodes present in large intestine

• Enterobius vermicularis

• Trichuris trichiura

Life Cycle

Humans are the only natural host for T. trichiura, but

morphologically similar worms are found to infect pigs

and some monkeys .

No intermediate host is required.

Infective form: Embryonated eggs contaning Rhabditiform larva.

When freshly passed, the egg contains an unsegmented ovum. At this stage, it is not infective for humans.

The fertilized female lays about 5,000 eggs per day .

.Adult female worm lives in large intestine ,female lays eggs which are discharged in feces.

The egg undergoes development in soil, optimally under warm, moist, shady conditions.

when the infective rhabditiform larva develops within the egg in 3–4 weeks. At lower temperatures, this may be delayed for 3 months or more .

These embryonated eggs are infective stage to man.

Infection occurs in humans when the mature embryonated eggs containing the infective larvae are swallowed in contaminated food or water.

The eggs hatch in the small intestine and the larva penetrate and develop in the intestinal villi with in 3-7days, and then return to lumen and migrate to the area of the cecum.

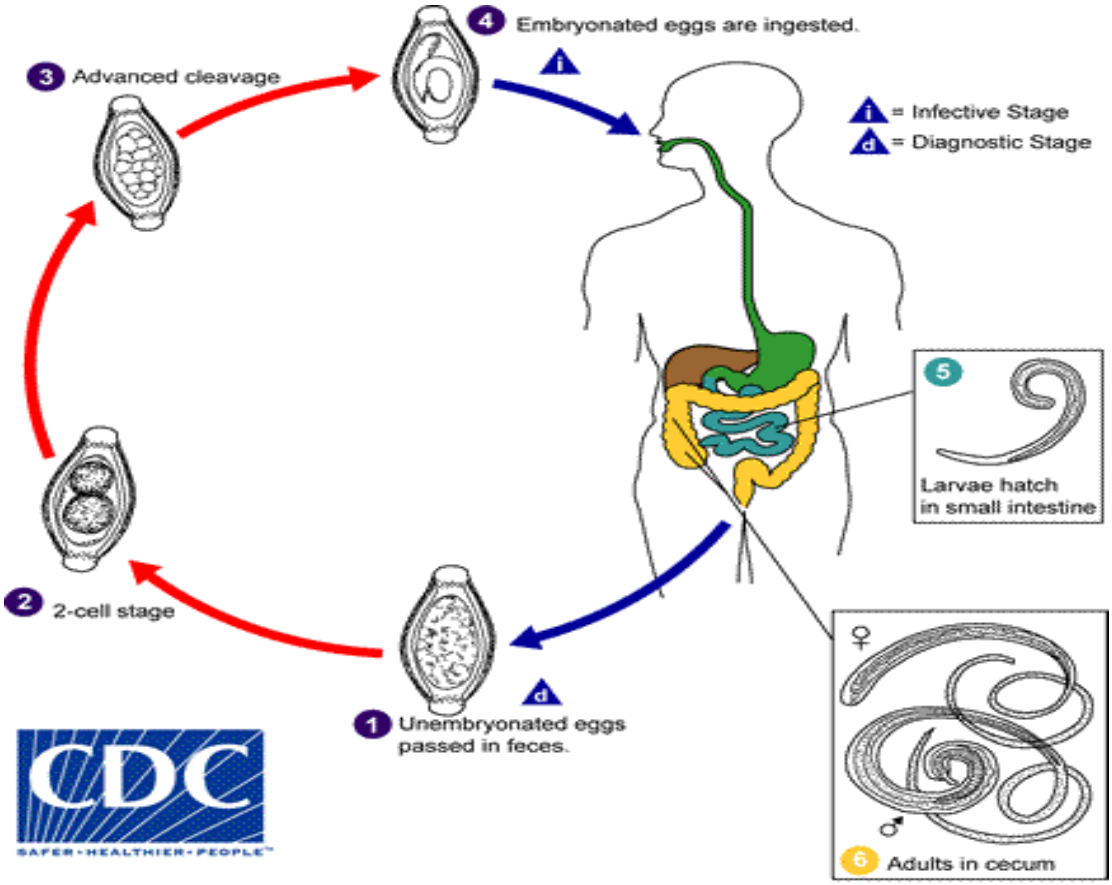
In about 2–3 months they become mature adults and

lie embedded in the cecal wall, with the thread-like

anterior portion piercing the mucosa and the thick posterior end projecting out.

The gravid adult female lays eggs, eggs start appearing in feces usually about 3 months after infection which are discharged in feces and the cycle is repeated .

Life span is usualy 4-6 years



Clinicl feature

Slight infections - are usually asymptomatic .

In the Heavy infections lead to mechanical effects or allergic reaction.

The worms lie threaded into the cecal mucosa and even though it is not a blood feeder, oozing of blood may at the sites of attachment.

The blood loss is about 0.005 mL per worm per day.

Over a period of time, this may lead to anemia and malnutrition.

It has been suggested that mechanical blockage of the appendical lumen by masses of whipworms may cause acute appendicitis

worms in children may cause a chronic dysentery,abdominal pain and tenderness

increased peristalsis and rectal prolapse especially in children .

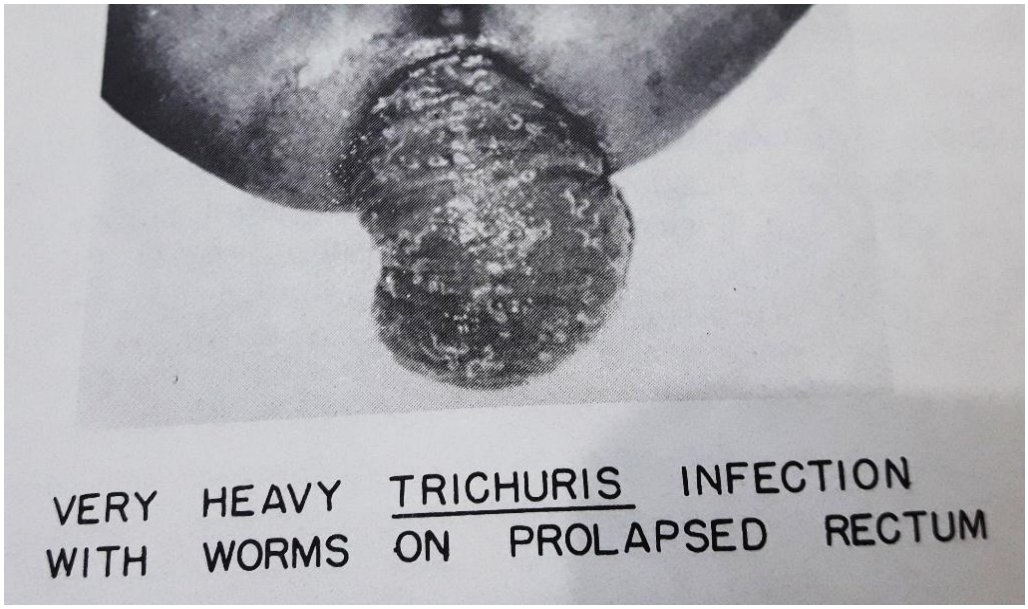
worms in children may cause a chronic dysentery,abdominal pain and tenderness

increased peristalsis and rectal prolapse especially in children

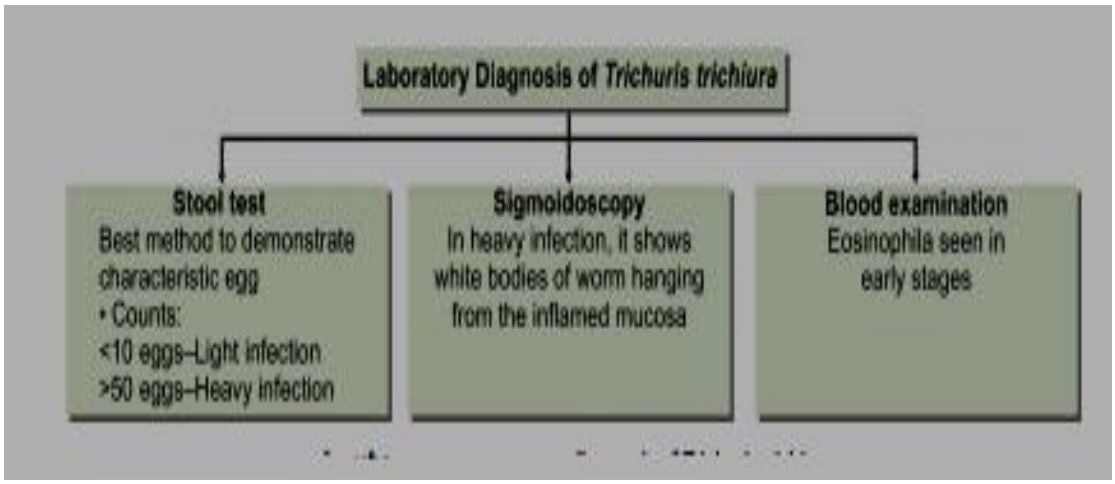
In heavy infection, sigmoidoscopy

may show white bodies of worm hanging from the inflamed

mucosa called coconut cake rectum



VERY HEAVY TRICHURIS INFECTION WITH WORMS ON PROLAPSED RECTUM



Treatment:

Mebendazole (100 mg 12 hourly for 3–5 days) or

Albendazole (single dose of 400 mg) are effective with cure rates of 70–90%.

Prophylaxis

Proper disposal of feces.

Avoiding consumption of unwashed fruits and vegetables.

Treatment of infected persons.

disease called trichinosis, trichinelliasis

The name Trichinella derived from the minute size of the adult

the smallest nematodes infecting humans

The major source of human infection was shown to be the consumption of inadequately cooked pork

Trichinosis is recognized as an important public health problem in Europe and America, but is much less common in the tropics and oriental countries

Subspecies

(a) Trichinella spiralis spiralis- seen in temperate regions, Acquired from domestic pigs, source of majority of infections in U.S.A.

(b) Trichinella spiralis nativa seen in arctic regions, acquired by eating undercooked bear and walrus meat.

(c) Trichinella spiralis nelsoniis acquired form wild pigs in southern Europe and Africa.

Morphology

Adult worm white in colour females are 3.5 mm long by 0.06mm thick.

males measure 1.5 mm long by 0.04 mm,

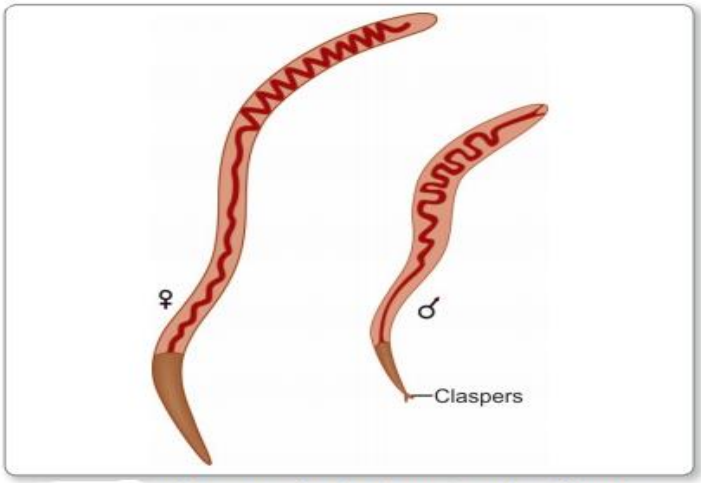
the antreior half of the body is thin and pointed.

the posterior port of the male has a pair of pear –shaped clasping papillae;

The female worm is viviparous and discharge larva instead of eggs.

The life span of the adult worm is very short.

The male worm dies soon after fertilizing the female and the female dies after 4 weeks to 4 months (16 weeks).



Adult worms of *Trichinella spiralis* (male and female)

life cycle

Natural host: Pig . Alternate host: Man.

Animal and man act as final and intermediate host

Infection can pass from—Pig-to-pig (facilitated by the custom of feeding pigs with untreated household garbage, which may contain bits of pork with infective cysts),

rat-to-rat, and pig-to-rat .

Man is the dead-end of the parasite, as the cysts in human muscles are unlikely to be eaten by another host.

Infective form: Encysted larva found in the muscles of pigs and other animals

Mode of infection: Man acquires infection mainly by eating raw or undercooked pork or inadequately processed sausages or other meat products containing the viable larvae.

When such meat is eaten without adequate cooking, the

cysts are digested by the gastric juice and viable larvae are released (excystation) in the stomach, duodenum, and jejunum

The larvae immediately penetrate the intestinal mucosa.

They moult 4 times and rapidly develop into adults, either male or female, by the second day of infection and with in 5 days, they become sexually mature worm.

The male dies after fertilizing the female.

The fertilized females start releasing motile larvae by the sixth day of infection

Larvae continue to be discharged during the remaining part of the lifespan of the female worm, which ranges from 4 weeks to 4 months.

Each female gives birth to approximately 1,000 larvae. These larvae enter the intestinal lymphatics or mesenteric venules and are transported in circulation to different parts of the body.

They get deposited in the muscles, central nervous system ,heart and other sites.

The larva die in most other situations, except the skeletal muscles, where it grows.

The common muscle involved (diaphram,intercostal , cervical,tongue in heavy infection 1000 cysts per gram of muscle

Deposition in the muscles occurs mostly during the second week of infection.

Larval development in muscles takes place during the next 3 or 4 weeks.

Within 20 days after entering the muscle cells, the larvae become encysted.

Encysted larvae lie parallel to the muscles of host.

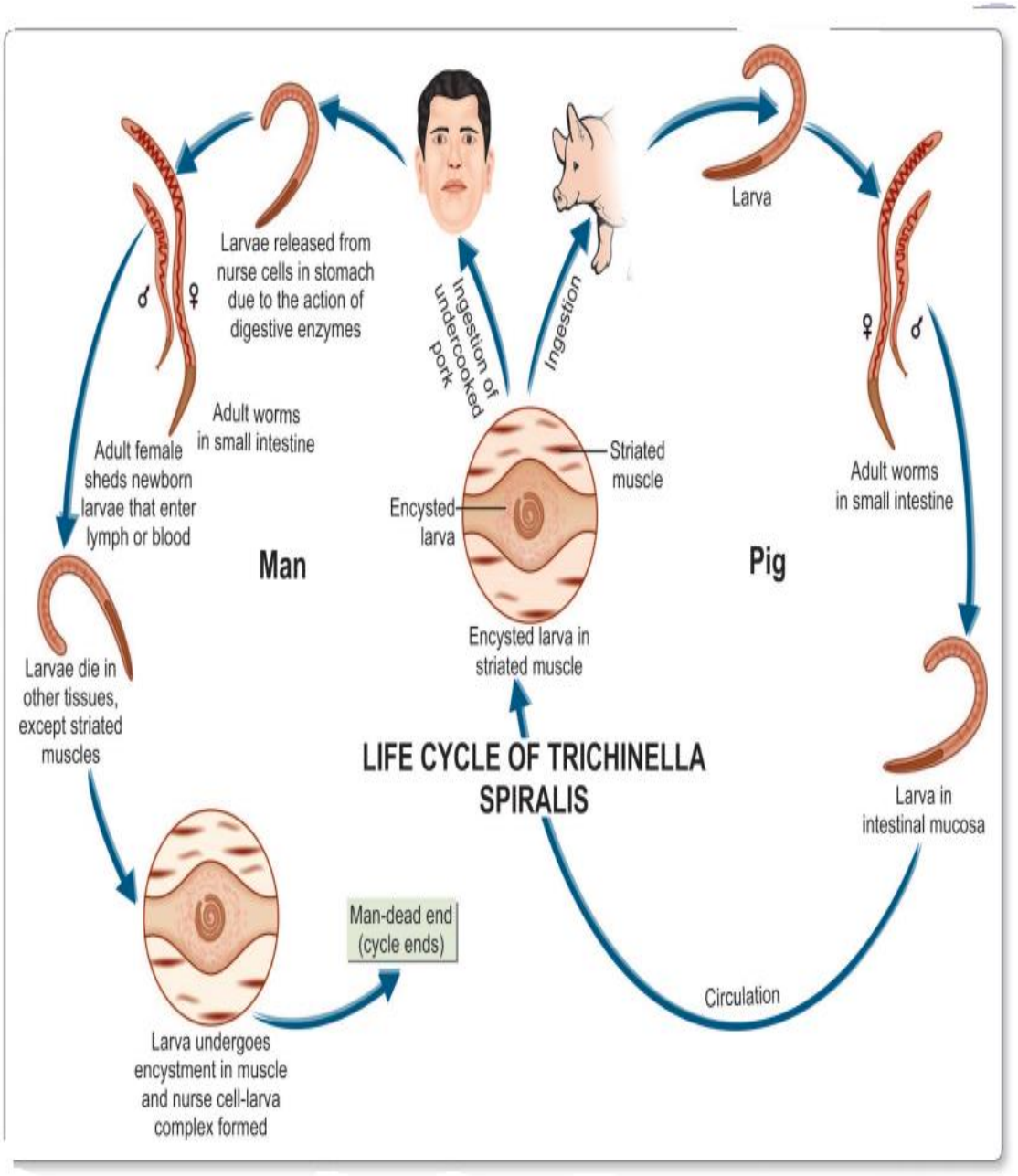
Encysted larva can survive for months to years

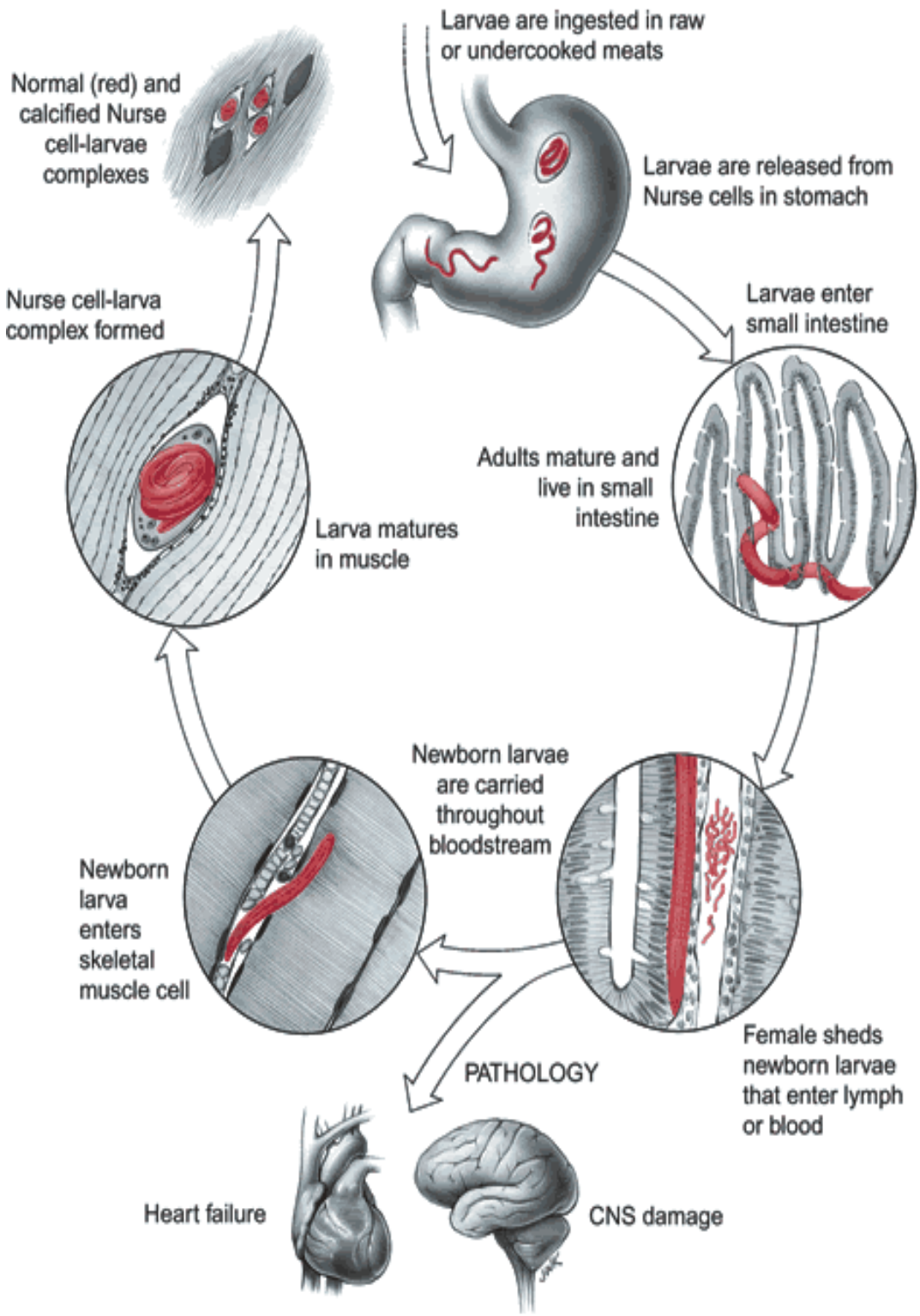
In man, the life cycle ends here

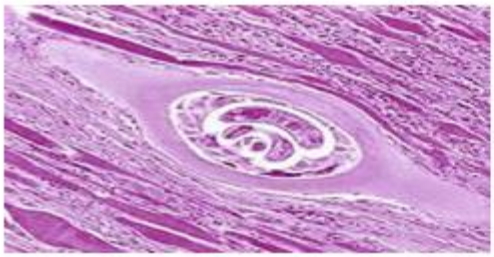
The larva at birth 100 µm and grow to reach 1000 µm in length The mature encysted larva becomes simillar to adult worm but sex organ not fully developed.

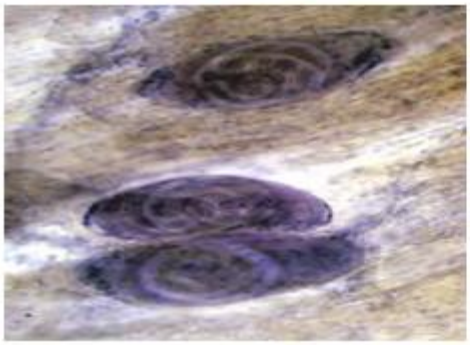
The cyst is formed by the tissue reaction around the encapsulated larvae. the larva in the cyst is coiled

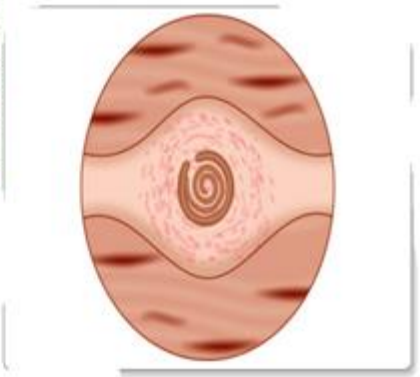
The larva remains infective inside the cyst for years and eventually, most become calcified and die with in 6-months to 2years









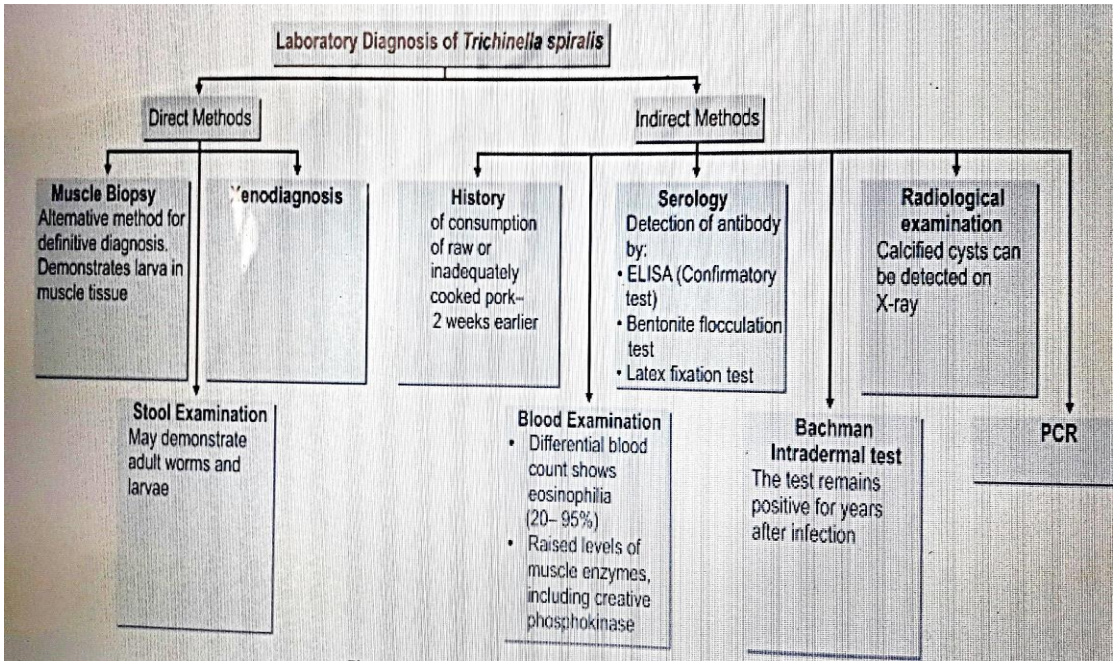


Encysted larva in muscles; infective stage

Clinicl feature

.: Stages in the Life Cycle of *Trichinella spiralis* (in Man)

	Stage of intestinal invasion	Stage of muscle invasion	Stage of encystation
	First stage	**Second stage**	**Final stage**
Pathology	The stage begins with the ingestion of raw pork containing infective larvae and ends with the larvae invading the intestinal epithelium and developing into adult	The stage begins when new infective larvae are released from the adult female and ends with the deposition of the larvae in the muscles. Myositis and basophilic granular degeneration of muscles occur in this stage	This stage occurs only in striated muscle. The infective larvae become encysted in this stage
Clinical features	Malaise, nausea, vomitting, diarrhea, abdominal cramps Onset within 2 to 30 hours of ingestion of infective food	Fever, myalgia, periorbital edema, weakness of affected muscle, hemorrhage in subconjunctiva and new beds (splinter hemorrhages), myocarditis (if heart muscles are involved), and encephalitis (if central nervous tissue is involved). Eosinophilia is a constant feature of this stage The stage is seen 1–4 weeks after infection	All symptoms subside



Treatment

Mild cases: Supportive treatment consisting of bed rest, analgesics, and antipyretics

Moderate cases: Albendazole 400 mg for 8 days or Mebendazole 200–400 mg for 3 days.

Severe cases: Add glucocorticoids like prednisolone to albendazole or mebendazole.

Mebendazole and albendazole are active against enteric stage of the parasite,

Prophylaxis

-Proper cooking of pork and other meat likely to be infected.

-the most effective method is to stop the practice of feeding pigs with raw garbage.

-Extermination of rats from pig farms the spread of infection .

-Smoking, salting or drying the meat does not destroy

the infective larvae.

-Prolonged freezing (20 days in a normal freezer) or (at –20°C for 3 days) decontaminates the meat.

filarial nematodes

The filarial worms reside in the 1- subcutaneous tissues,

2-lymphatic system, 3- body cavities of humans

The adult worm generally measures 80–100 mm in

length and 0.25–0.30 mm in breadth.

the female worm being longer than the males.

The tail of the male worm has perianal papillae and

unequal spicules but no caudal bursa.

The female worms are viviparous and give birth to

larvae known as microfilariae.

The microfilariae released by the female worm, can be

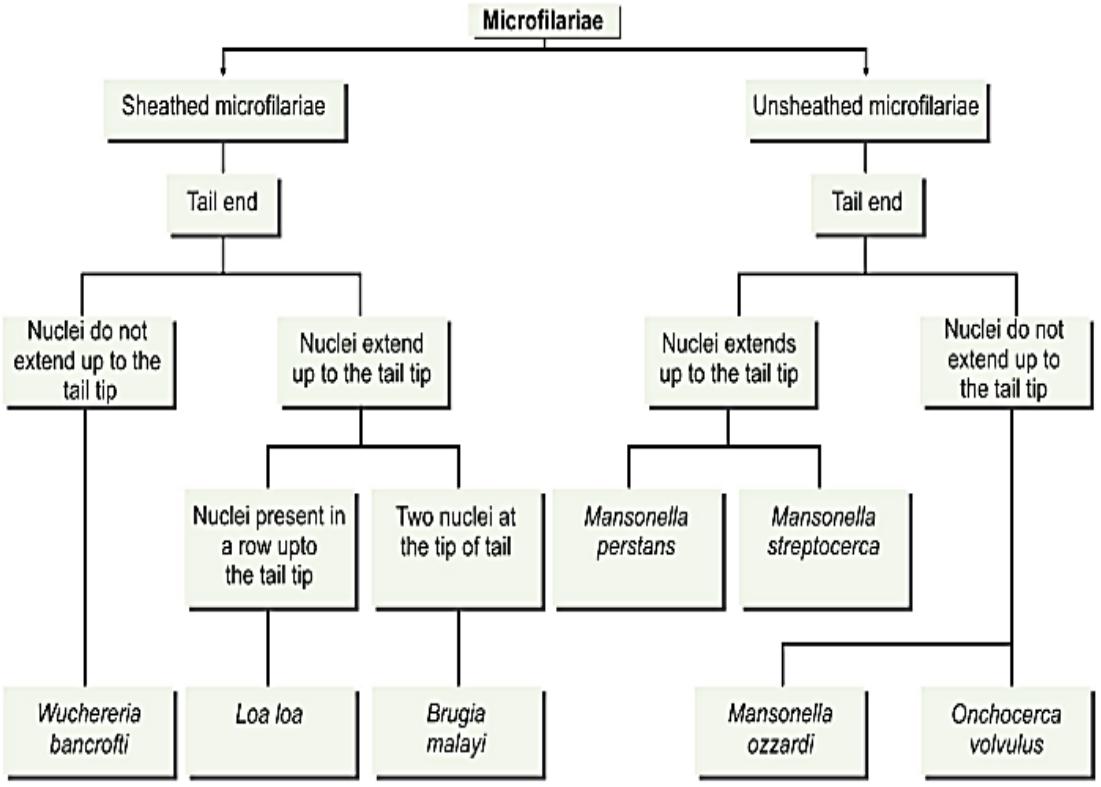
detected in the peripheral blood or cutaneous tissues, body cavities of humans depending on the species.

-In some species, the microfilariae retain their egg membranes which envelop them as sheath. They are known as sheathed microfi lariae .

-In other species the egg membrane is ruptured and are known as unsheathed microfi lariae. the microfilariae are classified on the basis of sheath

as ‘sheathed’ or ‘unsheathed.

-Other differentiation can be done on the characteristic arrangement of nuclei



Differentiating features of various microfilariae on the basis of presence of nuclei in tail end

Periodicity: depending on when the largest number of microfi lariae occur in blood, filarial worms can exhibit

nocturnal, diurnal periodicity or non periodicity at all

• Nocturnal periodicity: When the largest number of

microfi lariae occur in blood at night, e.g. Wuchereria

bancrofti

• Diurnal periodicity:When the largest number of

microfi lariae occur in blood during day, e.g. Loa loa

• Nonperiodic:When the microfi lariae circulate

at constant levels during the day and night, e.g.

Onchocerca volvulus.

- The life cycle of filarial nematodes is passed in 2 hosts:

definitive host is man

intermediate host are the blood-sucking arthropods.

The microfilariae complete their development in the

arthropod host to produce the infective larval stages. These are transmitted to humans by arthropod.

Adult worms live for many years .

the microfilariae survive for 3–36 months.

Eight species of filarial worms infect humans, of them six are pathogenic—

(Wuchereria bancrofti, Brugia malayi, and B. timoricause) lymphatic filariasis.

Loa loa causes malabar swellings and allergic lesions.

Onchocerca volvulus causes eye lesions and dermatitis.

Mansonella streptocerca leads to skin diseases.

two of them, M. ozzardi and M. perstans are virtually nonpathogenic

: Filarial Nematodes Infecting Humans

Parasite	Location in body adult	Microfilaria	Characteristics of microfilaria	Periodicity of microfilaria	Principal vector
I. Lymphatic filariasis					
Wuchereria bancrofti	Lymphatics	Blood	Sheathed, pointed tall tip free of nuclei	Nocturnal	*Culex quinquefasciatus*
Brugia malayi	Lymphatics	Blood	Sheathed, blunt tail tip with 2 terminal nuclei	Nocturnal	*Mansonia* spp.
Brugia timori	Lymphatics	Blood	Sheathed, longer than *Mf. malayi*	Nocturnal	*Anopheles barbirostris*
II. Subcutaneous filariasis					
Loa loa	Connective tissue, conjunctiva	Blood	Sheathed, nuclei extending up to pointed tail tip	Diurnal	*Chyrsops* spp.
Onchocerca volvulus	Subcutaneous nodules	Skin, eyes	Unsheathed, blunt tail tip free of nuclei	Non periodic	*Simulium* spp.
Mansonella streptocerca	Subcutaneous	Skin	Unsheathed blunt tail tip with nuclei	Non periodic	*Culicoides*
III. Serous cavity filariasis					
Mansonella ozzardi	Peritoneum and pleura	Blood	Unsheathed, pointed tail tip without nuclei	Non periodic	*Culicoides*
Mansonella perstans	Peritoneum and pleura	Blood	Unsheathed, pointed tail tip with nuclei	Non periodic	*Culicoides*

LYMPHATIC FILARIASIS

Wuchereria Bancrofti

Wuchereria bancrofti (Bancroftian filariasis,elephantiasis)

The disease is widely distributed through the tropical area of Africa, Asia and Latin America.

The largest number of cases of fi lariasis occur in India

Man is the only natural definitive host.

Intermediate host is the female mosquitoes especially anopheles and culex species.

: Geographical distribution of *Wuchereria bancrofti*

Morphology

The adults are whitish, translucent, thread-like worms with smooth cuticle and tapering ends

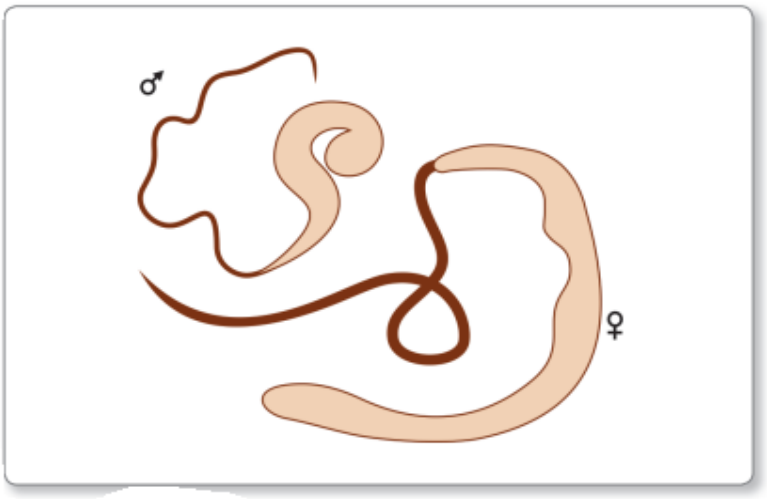
The female is larger (70–100mm × 0.25 mm), the male (25–40mm × 0.1 mm).

The posterior end of the female worm is straight, while that of the male is curved vertically and contains 2spicules of unequal length.

Males and females remain coiled together usually in the abdominal and inguinal lymphatics and in the testicular tissues

The female worm is viviparous and directly liberates sheathed microfi lariae into lymphatics.

Adult worms live for many years, probably 10–15 years or more.



Adult worm of *Wuchereria bancrofti*

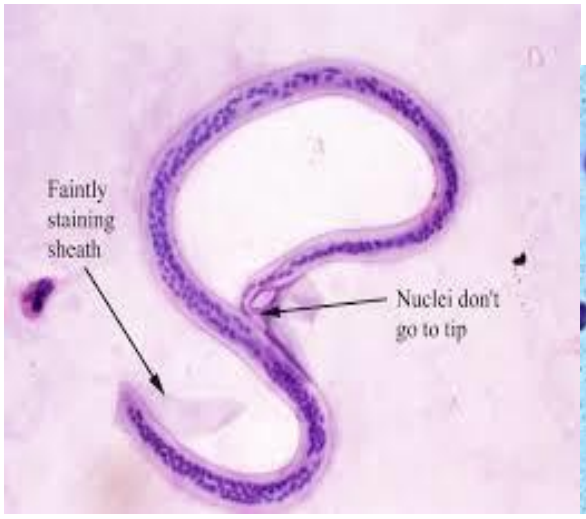
Microfi lariae

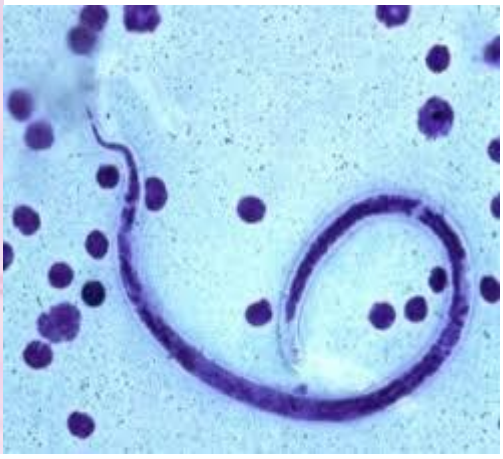
The microfi laria has a colorless, translucent body with a blunt head, and pointed tail

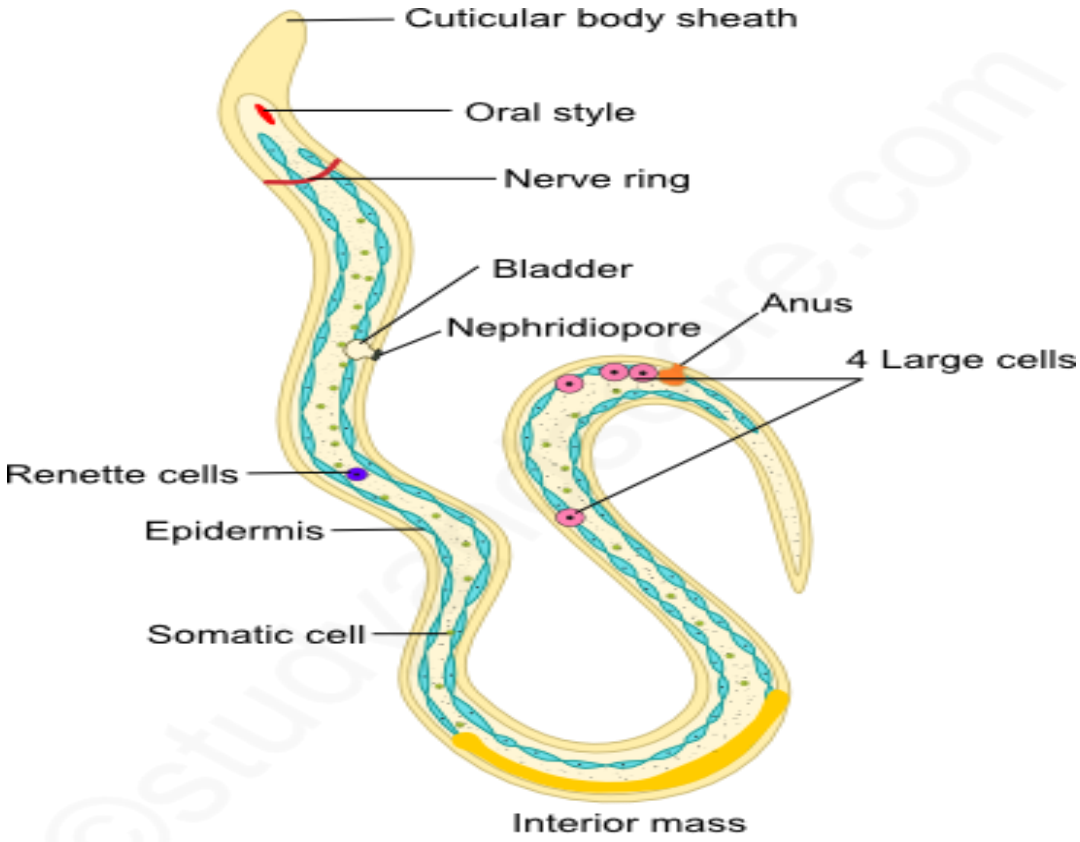
It measures 250–300 µm in length and 6–10 µm in thickness. It can move forwards and backwards within the sheath which is much longer than the embryo.

large numbers of microfilariae in peripheral blood only at

night (between 10 pm and 4 am).







Microfilariae do not multiply or undergo any further development in the human body.

If they are not taken up by a female vector mosquito, they die.

The lifespan is believed to be about 2–3 months.

It is estimated that a microfilarial density of at least 15 per

drop of blood is necessary for infecting mosquitoes.

Life Cycle

life cycle need two hosts

Definitive host: Man. No animal host or reservoir is known for W. bancrofti

Intermediate host: Female mosquito, of different species acts as vectors in different geographic areas.

The major vector in India and most other parts of Asia is Culex.

Infective form: Actively motile third-stage filariform larva is infective to man.

Mode of transmission: Humans get infection by bite of mosquito carrying filariform larva

Development in Mosquito

When a vector mosquito feeds on a carrier, the microfilariae are taken in with the blood meal and reach the stomach of the mosquito.

Within 2–6 hours, they cast off their sheaths (exsheathing), penetrate the stomach wall and within 4–17 hours migrate to the thoracic muscles where they undergo further development.

during the next 2 days into .

the first-stage larva, which is a sausage-shaped with a spiky tail, measuring 125–250 × 10–15 µm with in a week, it moults once or twice, increases in size and becomes.

the second-stage larva, measuring 225–325 × 15–30 µm In another week, it develops its internal structures and becomes

the elongated third-stage filariform larva, measuring 1,500–2,000 × 15–25 µm. It is actively motile and is the infective form . It enters the proboscis sheath of the mosquito.

There is no multiplication of the microfilaria in the mosquito and 1 microfilaria develops into 1 infective larva only.

The time taken from the entry of the microfilaria into the mosquito till the development of the infective third stage larva located in its proboscis sheath (10-20 days)

Devolopment in Man

The larvae enter through the puncture wound or penetrate the skin by themselves.

The infective dose for man is not known, but many larvae fail to penetrate the skin by themselves and many more are destroyed in the tissues by immunological and other

deffence mechanisms.

After penetrating the skin, the third-stage larvae enter the lymphatic vessels and are carried usually to abdominal or inguinal lymph nodes, where they

develop into adult worm.

There is no multiplication at this stage and only 1 adult

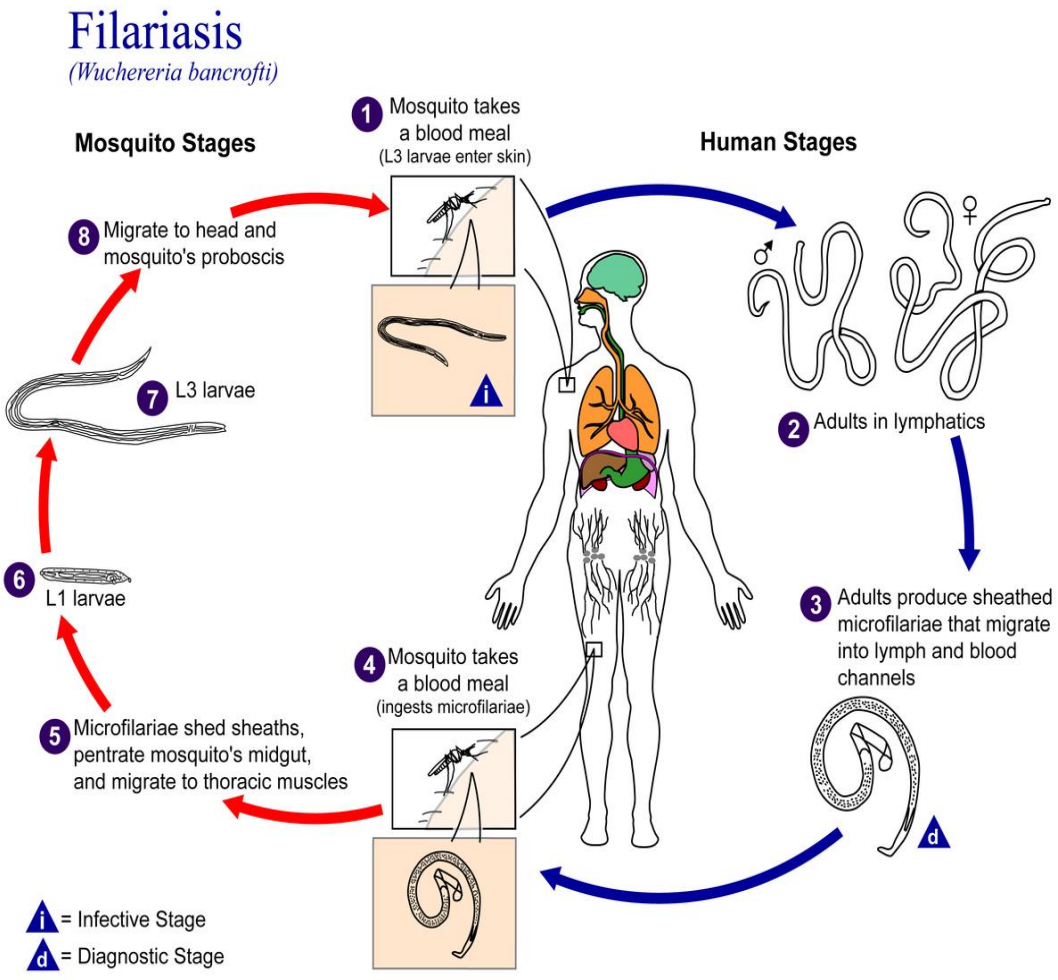
develops from 1 larva, male or female.They become sexually mature in about 6 months .

The gravid female worm releases large numbers of

microfi lariae, as many as 50,000 per day.

They pass through the thoracic duct and pulmonary capillaries to enter the peripheral circulation.

The microfi lariae are ingested with the blood meal by mosquito and the cycle is repeated.



Occult Filariasis

It occurs as a result of hypersensitivity reaction to microfi larial antigens, not directly due to lymphatic involvement.

Clinical manifestations:

€ Massive eosinophilia (30–80%)

€ Hepatosplenomegally

Pulmonary symptoms like dry nocturnal cough, dyspnea, and asthamatic wheezing.€

Some time see arthritis, glomerulonephritis, thrombophlebitis, tenosynovitis, etc.

Meyers Kouwenaar syndrome is a synonym for occult fi lariasis.

Tropical pulmonary eosinophilia: presents with low-grade fever, loss of weight, and pulmonary symptoms such as dry nocturnal cough, dyspnea, and asthmatic wheezing. €

Classical features of lymphatic fi liariasis are absent

SUBCUTANEOUS FILARIASIS

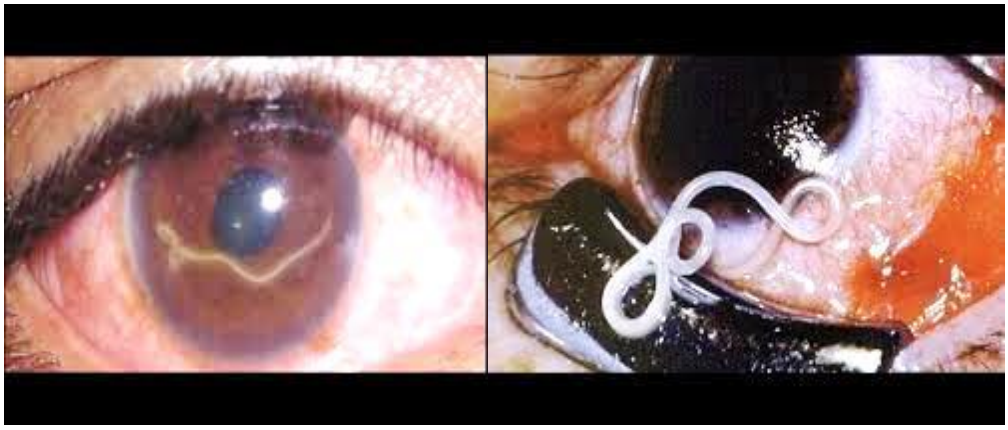
Loa Loa Common name: African eye worm, loiasis The disease is endemic in central and West Africa. where about 10 million people are effected.

Morphology

The adult worm is thin and transparent, measuring about 30–70 mm in length and 0.3–0.5 mm in thickness.

In infected persons, they live in the subcutaneous tissues through which they wander. They may also occur in the sub conjunctival tissues.

Adults live for 4–17 years.



Microfilaria

The microfilariae are sheathed with column of nuclei extending completely to the tip of the tail.

They appear in peripheral circulation only during the

day from 12 noon to 2 pm (diurnal periodicity

Life cycle

life cycle is completed in two hosts.

Definitive host: Man

Intermediate host or vectors: Day biting flies (mango flies)

Infective stage - third-stage larvae.

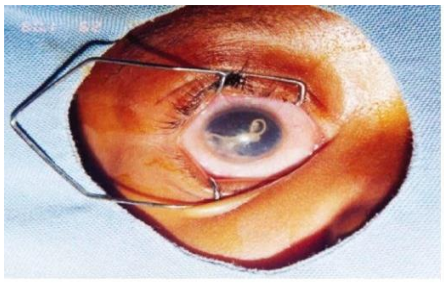
Infection is transmitted to man through the bite of infected vecters during their blood meal.

The infective third stage larvae enter the subcutaneous tissue, moult then develop into mature adult worm over 6–12 months in subcutaneous tissues.

Female worms produce sheathed microfilaria which have diurnal periodicity.

The microfilaria are ingested by vecters during its blood meal.

They cast off their sheaths, penetrate the stomach wall and reach thoracic muscles where they develop into infective larvae with in 10 days.



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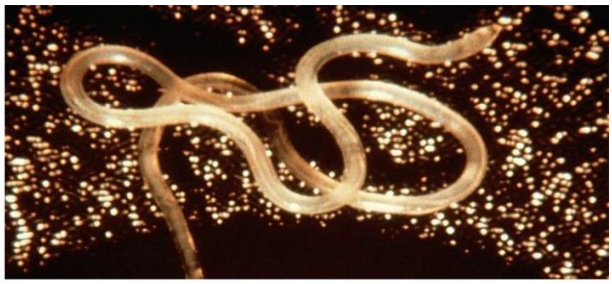

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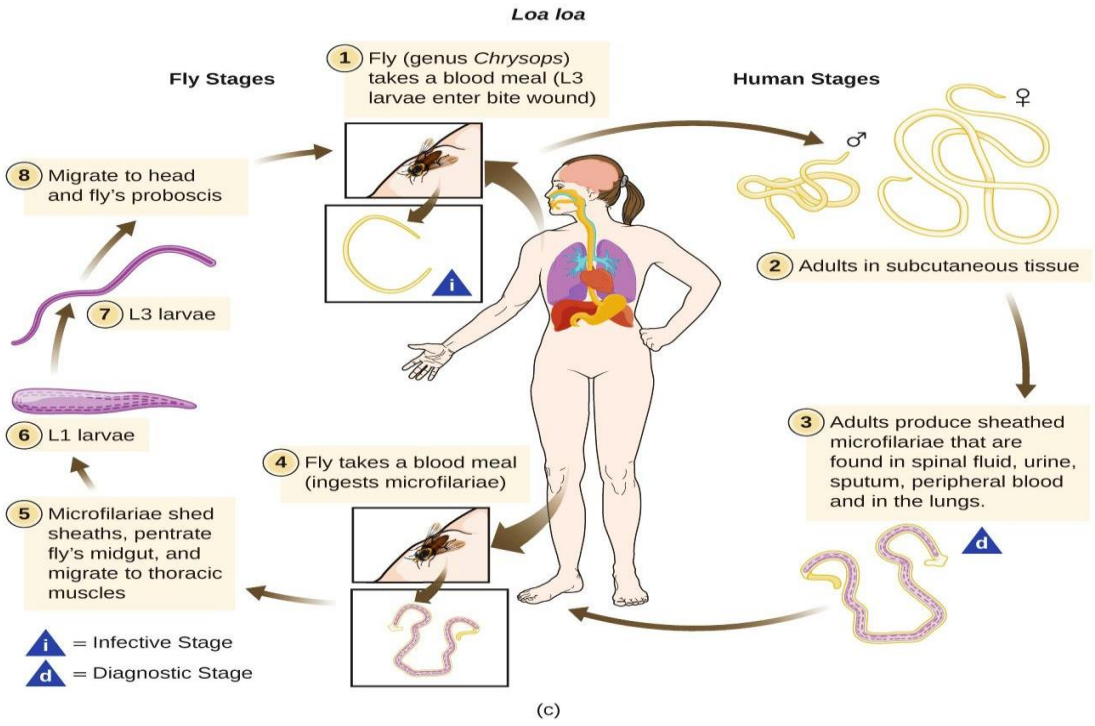


(c)

Pathogenesis and clinical features:

There is no inflammatory response to the micofilariae or the adults, but a hypersensitivity reaction causes transient localized, non -erythematous, subcutaneous edema(Calabar swelling) .

Ocular manifestations range from photophobia to gradual blurring of vision, progressing to total blindness. lesions may develop in all parts of the eye.

The most common early finding is conjunctivitis with photophobia.

Other ocular lesions include keratitis, secondary glaucoma, choroidoretinitis, and optic atrophy.

Other Complications like nephropathy, encephalopathy, and cardiomyopathy can occur but are rare

Treatment:

Diethylcarbamazine eliminates the microfilariae and may kill the adults.

Worms in the eye require surgical excision

diagnosis

Microfilariae may be shown in peripheral blood collected during the day.

The adult worm can be demonstrated by removal from the skin or conjunctiva or from a subcutaneous biopsy specimen from a site of swelling.

High eosinophil count is common

SEROUS CAVITY FILARIASIS

Mansonella Ozzardi

M. Ozzardi is a new world filaria seen only in Central and South America and the West Indies.

The adult worms are found in the peritoneal and pleural cavities of humans.

The non periodic unsheathed microfilariae are found in the blood.

Culicoides species are the vectors.

Infection does not cause any illness.

Diagnosis is made by demonstrating microfi lariae in blood

Ivermectin (single dose 6 mg) is eff ective in treatment.

in pericardium.

Mansonella Perstans

Also known as Acanthocheilonema, Dipetalonema, or Tetrapetalonema perstans.

the worm is extensively distributed in tropical Africa and coastal South America.

The adult worms live in the body cavities of humans, mainly in peritoneum, less in pericardium.

The microfi lariae are unsheathed and subperiodic.

Vectors are Culicoides species.

Diagnosis is by demonstration of the microfi lariae in peripheral blood or serosal effusion.

Doxycycline (200 mg twice a day for 6 weeks)

Dracunculus Medinensis

Common name: Guinea Worm

The worm was present in tropical Africa, the Middle-East

in Iraq, Iran, and in Pakistan and India.

About 50 million people were estimated to be infected with the worm.

D. medinensis causes dracunculiasis or dracunculosis.

.

The disease still remains endemic in 13 African countries

including Sudan (highest incidence), Niger, etc

Morphology

The adult female is a long, cylindrical worm with smooth milky-white in colour.

It has a blunt anterior end and a tapering recurved tail

It measures about (60–120 cm) in length and 1–2 mm in thickness

The body of the gravid female uterus containing about 3million embryos ,the female worm is viviparous.

The male worm, which is rarely seen, is much smaller than female being 10–40 mm long and 0.4 mm thick. Female worm survives for about an

year. life span of male worm is not more than 6months. The larva measures 500–750 µm in length and 15–25 µm thick

Life Cycle

life cycle need two hosts.

Defi nitive host: Man

Habitat in cutaneous and subcutaneous tissues

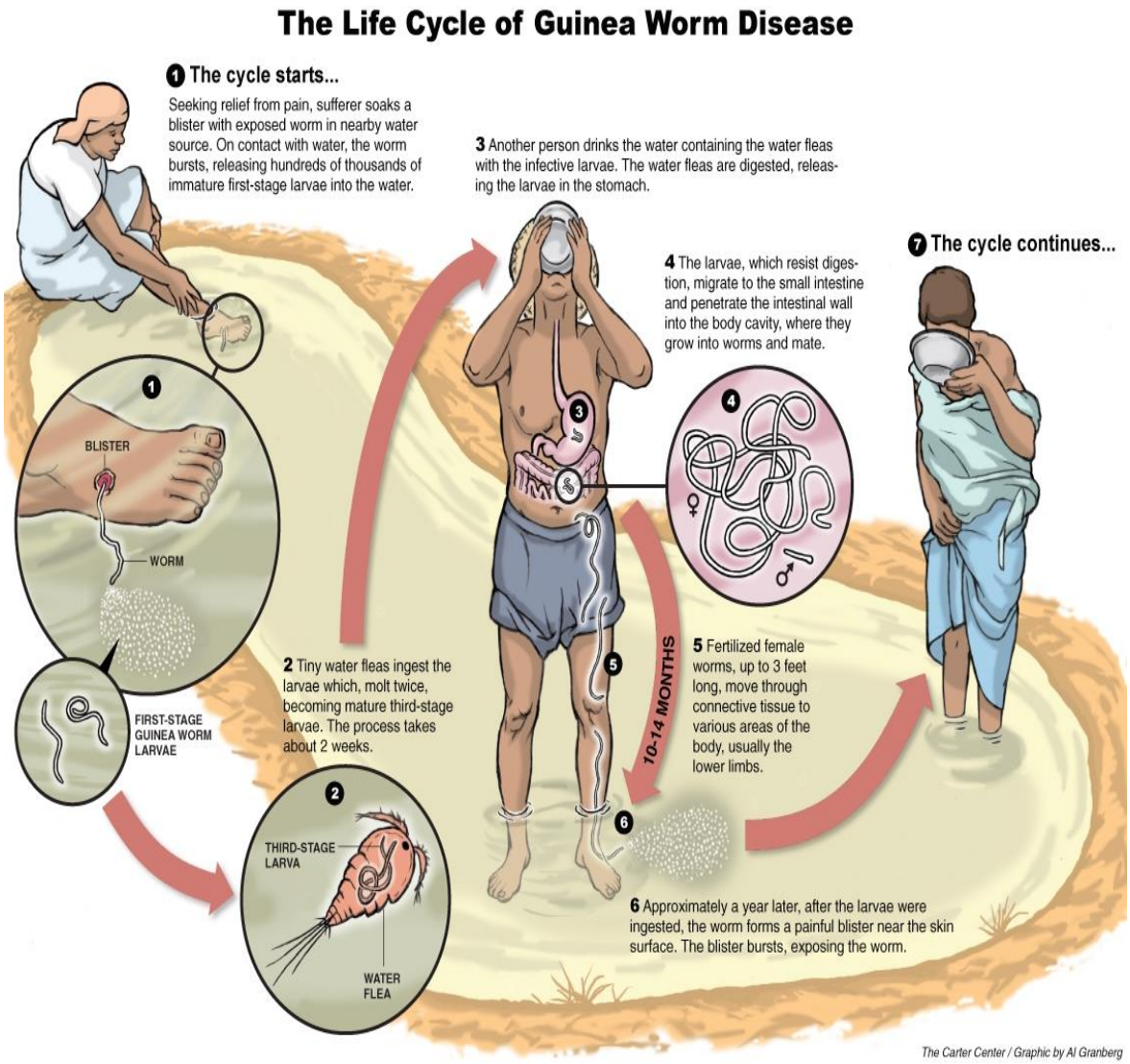
Intermediate host: Cyclops, in which embryos undergo developmental changes. There is no animal reservoir.

Infective form: Third-stage larva present in the hemocele

of infected cyclops.

Mode of transmission: Humans get infected by drinking unfiltered water containing infected cyclops with third –stage larva .

Incubation period: about one year



Pathogenicity and Clinical Features

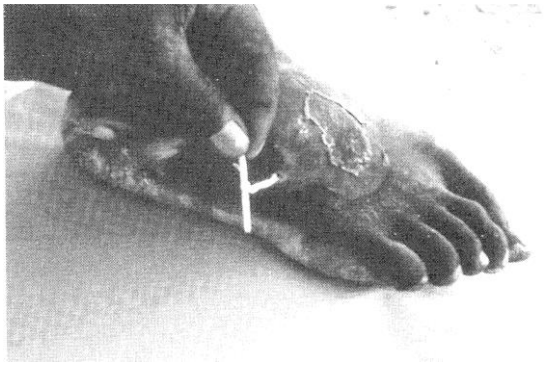
Infection induces no illness till the gravid female worm comes to lie under the skin, ready to discharge its embryos.

The body fluid of the adult worm is toxic and leads to

blister formation.

A few hours before the development of the blister, there may be constitutional symptoms such as nausea, vomiting, intense pruritus, and urticarial rash.

The blister develops initially as a reddish papule with a vesicular center .



The most common sites for blister formation are the feet between the metatarsal bones or on the ankles.

Secondary bacterial infection is frequent. Sometimes, it may lead to tetanus.

Sometimes, the worm travels to unusual sites such as the pericardium, the spinal canal, or the eyes, with sever effects

Diagnosis

Detection of adult worm and larval form in ulcer.

Demonstration of dead worm by X-ray.

Serology–ELISA and IFA

Skin test: An intradermal test with guinea worm antigen

elicits positive response.

Treatment

Antihistaminics and steroids are of help in the initial

stage of allergic reaction.

Metronidazole, niridazole, and thiabendazole are useful in treatment .

Surgical removal of the worm under anaesthesia

د٠ سعيد حميد **Helminthes (Cestodes)**

Classification of Cestodes

-Phylum Platyhelminthes .

- class Cestoidea.

The class Cestoidea includes 2 orders:

1-Pseudophyllidea

2-Cyclophyllidea

Cestodes in (Greek kestos—girdle or ribbon) are multisegmented, dorsoventrally flattened tapelike worms sizes vary from a few millimeters to several meters

The adult worm consists of 3 parts:

€ 1- Head (scolex)

€ 2-Neck

€ 3-Trunk (strobila)

Head (Scolex) It is the organ of attachment to the intestinal mucosa of the definitive host .

In the order cyclophyllidea

the scolex possesses 4 suckers(or acetabula). In some cyclophyllidea

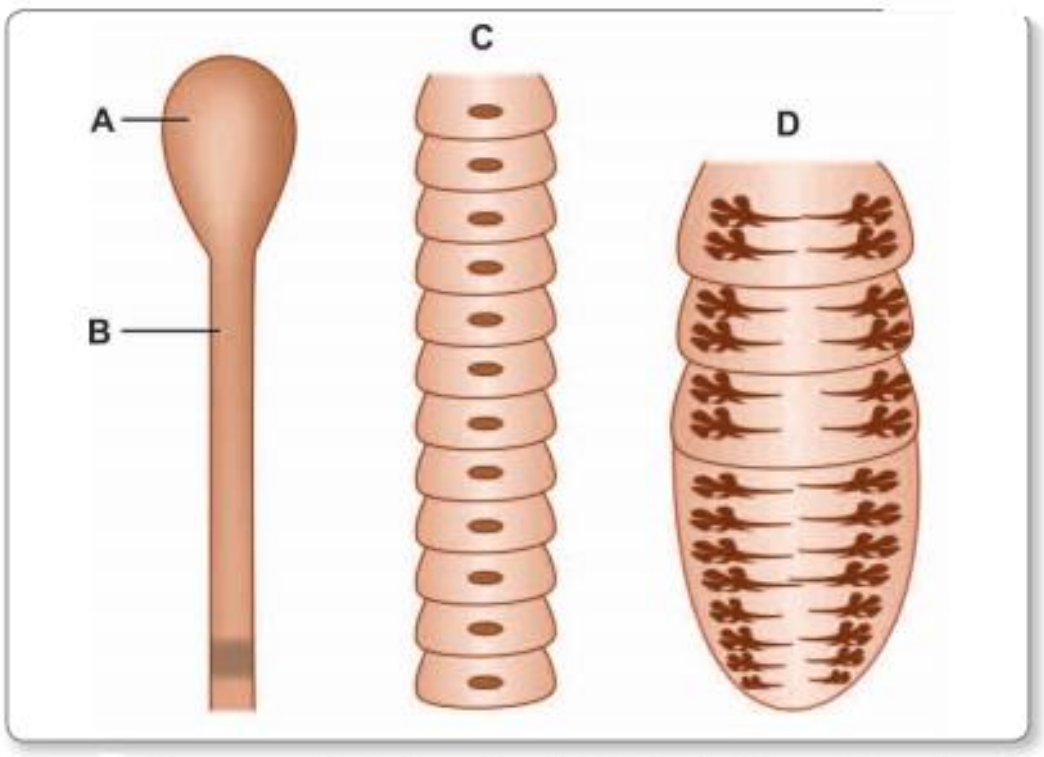
like Taenia solium, scolex has an apical protrusion called as

the rostellum.

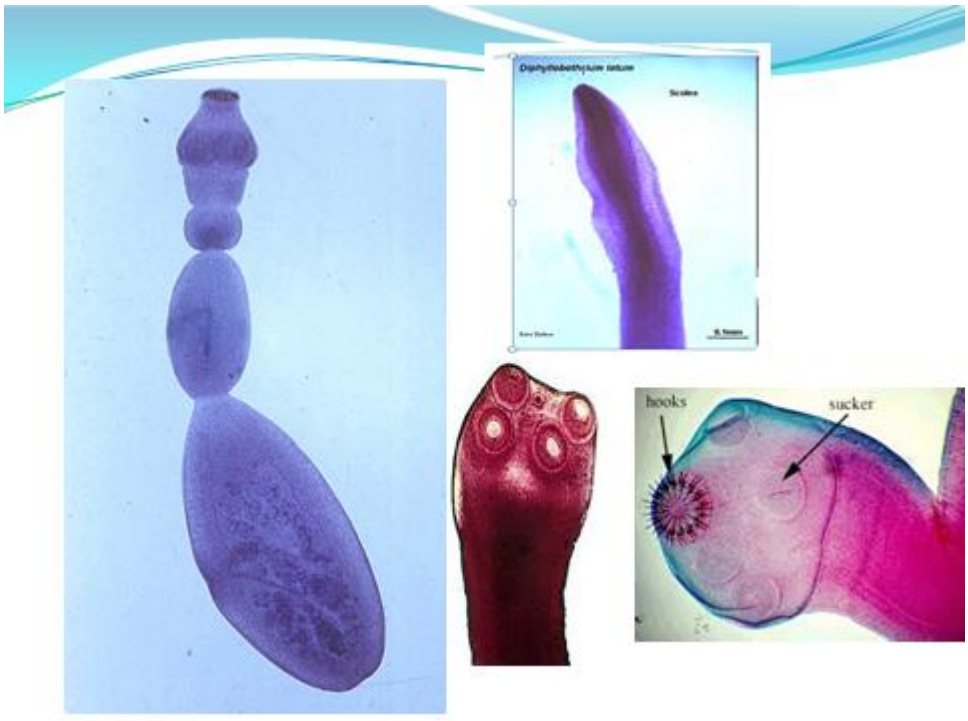
The rostellum may or may not be armed with hooks.

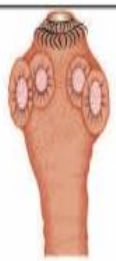
In the order pseudophyllidea.

the scolex does not possess suckers but possesses a pair of longitudinal grooves called as bothria,by which it attaches to the intestine mucosa of the host



g. ... Tapeworm. **A.** Scolex or head; **B.** Neck, leading to the region of growth below, showing immature segments; **C.** Mature segments; **D.** Gravid segments filled with eggs



	Taenia solium	*Taenia saginata*	*Hymenolepis nana*	*Hymenolepis diminuta*	*Diphyllobothrium latum*	*Echinococcus granulosus*
Heads						
	4 suckers 2 rows of hooks	4 suckers No hooks	4 suckers single row of 20–30 hooks	4 suckers No hooks	2 Suctorial grooves or bothria No suckers, No hooks	4 suckers 2 rows of hooks
Proglottids						
	Longer than broad 7–12 uterine branches on each side	Longer than broad 15–30 uterine branches on each side	Broader than long	Broader than long	Broader than long Uterus coiled	Longer than broad

Differences between heads and proglottids of various Cestodes

Neck

It is the part, immediately behind the head and is the region of growth from where the segments of the body (proglottids) are being generated continuously.

Trunk (strobila)

The trunk also called as strobilais composed of a chain of

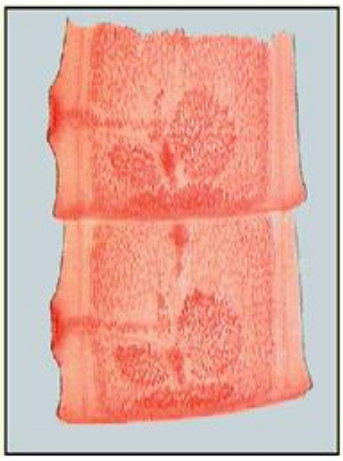
Proglottids or segments.

The proglottids

- immature segments.
- mature segments.

- gravid segments.

Tapeworms are hermaphrodites ,mature segment contains both male and female sex organs.



Mature segments

gravid segments

In the immature segments, the reproductive organs are not well developed.

They are well developed in the mature segments.

The gravid segments are completely occupied by the uterus filled with eggs.

Tapeworms do not have a body cavity or alimentary canal

Rudimentary excretory and nervous systems are present.

Tape worm is a hermaphrodite. There is one complete set of male and female organs for each

proglottids

egg

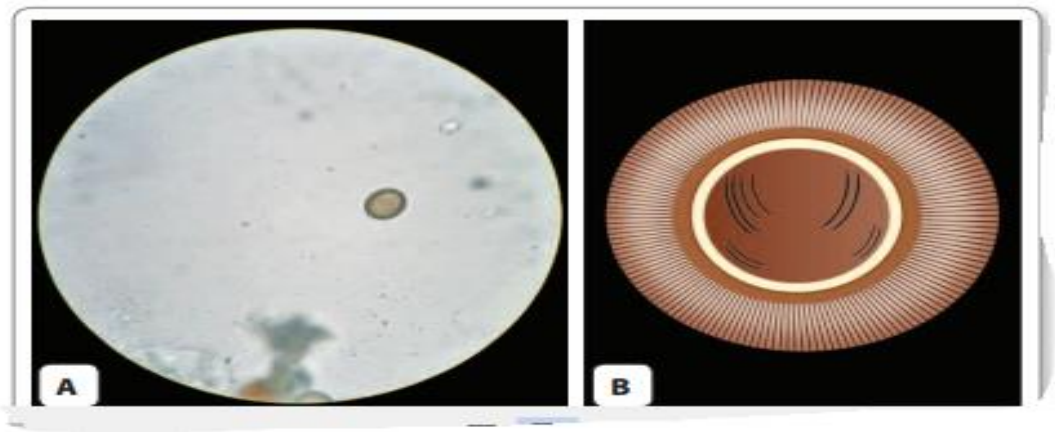
The embryo inside the egg is called the oncosphere

(meaning ‘hooked ball’) because it is spherical and has

hooklets.

Oncospheres of human tapeworms typically have 3

pairs of hooklets and so, are called hexacanth(meaning 6-hooked) embryos.



Cestodes complete their life cycle in 2 different hosts.

Exceptions are:

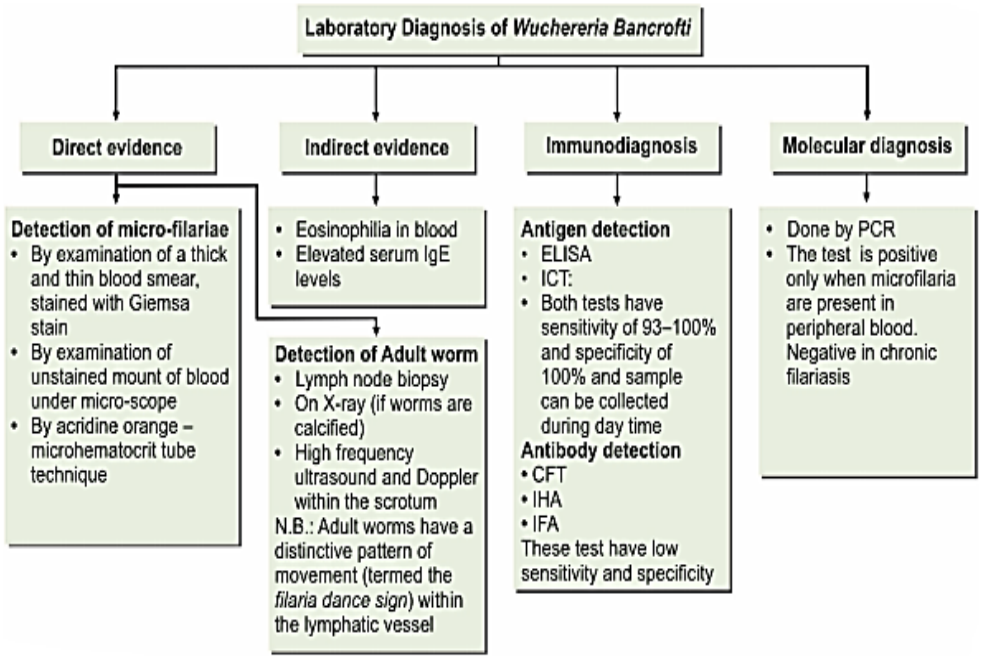
Hymenolepis nana that requires only 1 host, man .

Diphyllobothrium latum that requires 3 hosts.

- definitive host: man.

-first intermediate host: cyclops.

-second intermediate host: fish



Treatment

diethyl carbamazine (DEC) is the drug of choice. It is given orally in a dose of 6 mg/ kg body weight daily for a period of 12 days amounting to a total of 72 mg of DEC per kg of body weight.

It has both macro- and micro-fi laricidal properties

The administration of DEC can be carried out in 3 ways.

1. Mass therapy:

The dose recommended is 6 mg/kg body weight.

In some countries it is used alone and in some, with albendazole or ivermectin. Mass therapy is indicated in highly endemic areas

2. Selective treatment: The recommended dose in the Indian programme is DEC 6 mg/kg of body weight daily for 12 doses, to be completed in 2 weeks. In endemic areas, treatment must be repeated every 2 years.

3. DEC medicated salts: Common salt medicated with

1–4 g of DEC per kg has been used for fi lariasis control .

Ivermectin: In doses of 200 µg/kg can kill the microfi lariae but has no eff ect on adults.

Tetracyclines: Also have an eff ect in the treatment of fi lariasis

Supportive Treatment

Chronic condition may not be curable by antifi larial drugs and require other measures like elevation of the aff ected limb, use of elastic bandage, and local foot care reduce some of the symptoms of elephantiasis.

Surgery is required for hydrocele.

Medical management includes bed rest, high protein diet with exclusion of fat, drug therapy with DEC, and use of abdominal binders.

Surgical management of refractory case includes endoscopic sclerotherapy using silver nitrate

Prophylaxis

The 2 major measures in prevention and control of fi lariasis are

i. Eradication of the vector mosquito
ii. Detection and treatment of carriers.

Eradication of Vector Mosquito

Antilarval measures: The ideal method of vector control would be elimination of breeding places by providing adequate sanitation underground waste and water disposal system.

Chemical control: Using antilarval chemicals like Mosquito larvicidal oil, Pyrosene oil,Organophosphorous larvicides like temephos, fenthion etc.

Antiadult measures: Adult mosquitoes can be restricted by use of DDT, dieldrin, and pyrethrum.

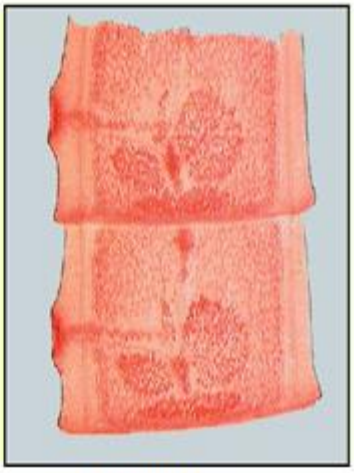
Personal prophylaxis: Using mosquito nets and mosquito repellants is the best method.

Detection and Treatment of Carriers

The recommended treatment is DEC 6 mg per kg body weight daily for 12 days, the drug being given for 2 weeks, 6 days in a week

mature segment

Taenia saginata
2 ovary lobes



Taenia solium
3 ovary lobes

gravid segments

Taenia solium

Taenia saginata

Echinococcus species

Four Echinococcus species

• Echinococcus granulosus : Hydatid disease

• Echinococcus multilocularis : Alveolar or multilocular hydatid disease

• Echinococcus vogeli and Echinococcus oligarthrus : Polycystic hydatid disease

Echinococcus granulosus

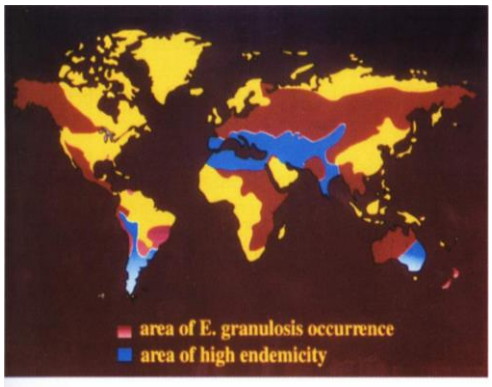
Common name: dog tape worm, hydatid tape worm.

Disease: unilocular hydatid disease.

Hydatid disease also known as *Echinococcosis* or hydatidosis is caused by infection with larva (meta cestode) of the tapeworm of the genus *Echinococcus*

(Family Taenidae, Order Cyclophyllidea, Class Cestoda, Phylum Platyhelminths).

The disease is endemic in many parts of the world specially middle east (include Iraq), Australia, New Zealand, South America, Central and South Europe



Geographical distribution of cystic hydatid disease

Morphology

Adult Worm

It is a small tapeworm, measuring only 3–6 mm in length.

It consists of a scolex, a short neck, and strobila.

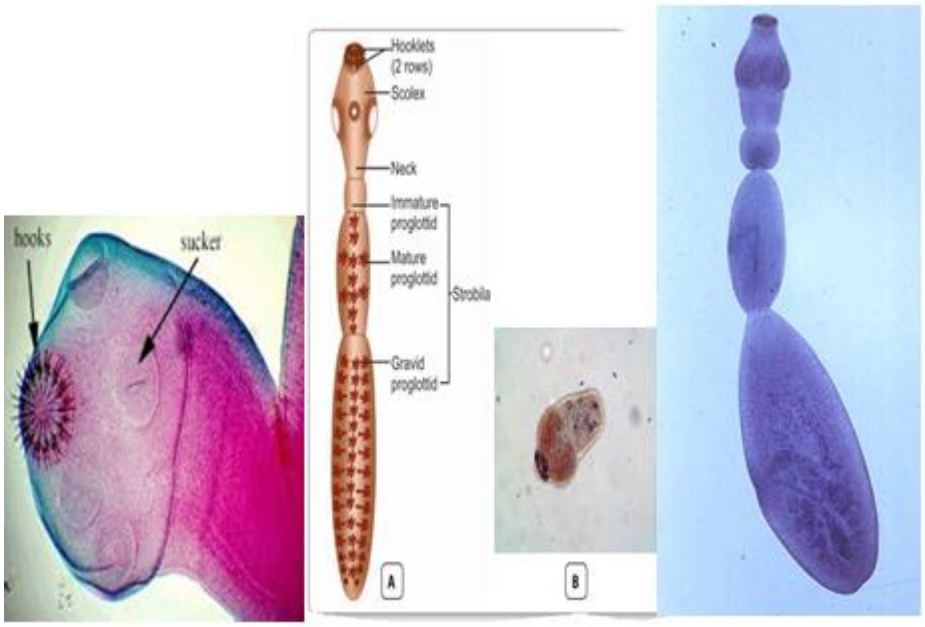
The scolex is pyriform,with 4 suckers and a prominent rostellum bearing 2 circular rows of hooklets (25–30)

The neck is short than the rest of the worm .

The strobila is composed of only 3 proglottids, the anterior immature, the middle mature, and the posterior gravid segment

The terminal proglottid is longer and wider than the rest

of the worm and contains a branched uterus filled with 500eggs.



Eggs

The egg is spherical, measuring 30–40 µm in diameter.

It has a thin hyaline embryonic membrane around it.

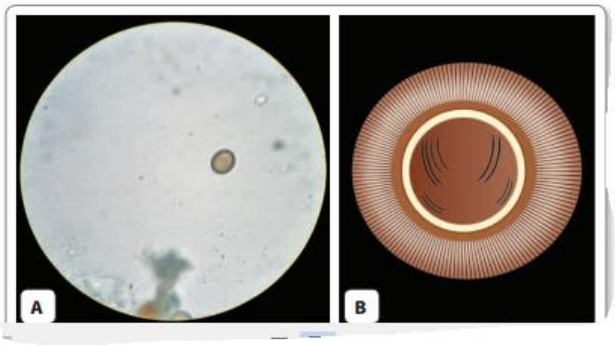
The inner embryophore is radially

striated and is yellowbrown

due to bile staining

In the center is a fullydeveloped

embryo(oncosphere) with 3 pairs of hooklets (hexacanth embryo).



Larval Form

The larval form is found within the hydatid cyst developing

inside various organs of the intermediate host.

It represents the structure of the scolex of adult worm and remains invaginated within a vesicular body.

After entering the definitive host, the scolex with suckers and rostellar hooklets, becomes exvaginated and develops into adult worm.

Mode of Transmission

Transmission of E. granulosus to humans is affected by such factors as prevalence of the parasite in domestic dogs, behavior of humans towards dogs, immunological and genetic factors .

Direct contact between human and dogs .

Indirect transmission via water sources and vegetable, contaminated by E. granulcosus eggs deposited by infected dogs may be the most important route of transmission to humans .

Flies may also be implicated in transmission of the disease

Direct transmission of E. granulcosus from human to human does not occur

Life Cycle

The worm completes its life cycle in 2 hosts

Definitive hosts: Dog (optimal host), wolf, and fox

Intermediate host: Sheep and Cattle. (Sheep is the ideal intermediate host).

€

Man acts as an accidental intermediate host (dead end).

€ The adult worm lives in the small intestine of dogs and other canine animals. These animals discharge numerous eggs in the feces,the intermediate hosts (sheep and cattle) ingest them while grazing.

Human infection follows ingestion of the eggs due to intimate handling of infected dogs or by eating raw vegetables or other food items contaminated with dog feces.

The ova ingested by man or by sheep and cattle are liberated from the chitinous wall by gastric juice liberating the hexacanth embryos which penetrate

the intestinal wall and enter the portal venules, to be carried to the liver along the portal circulation.

These are trapped in hepatic sinusoids, where they eventually develop into hydatid cyst.

About 65% of hydatid cyst develop in liver, which acts as the first filter for embryo.

However, some embryo which pass through the liver, enter the right side of heart and are caught in pulmonary capillaries(forming pulmonary hydatid cysts 15%), so that the lung acts as the second filter.

A few embryo enter the systemic circulation and get lodged in various other organs and tissues such as the spleen, kidneys, eyes, brain, or bones.

The hydatid cyst may be present in any tissue except hair, nail

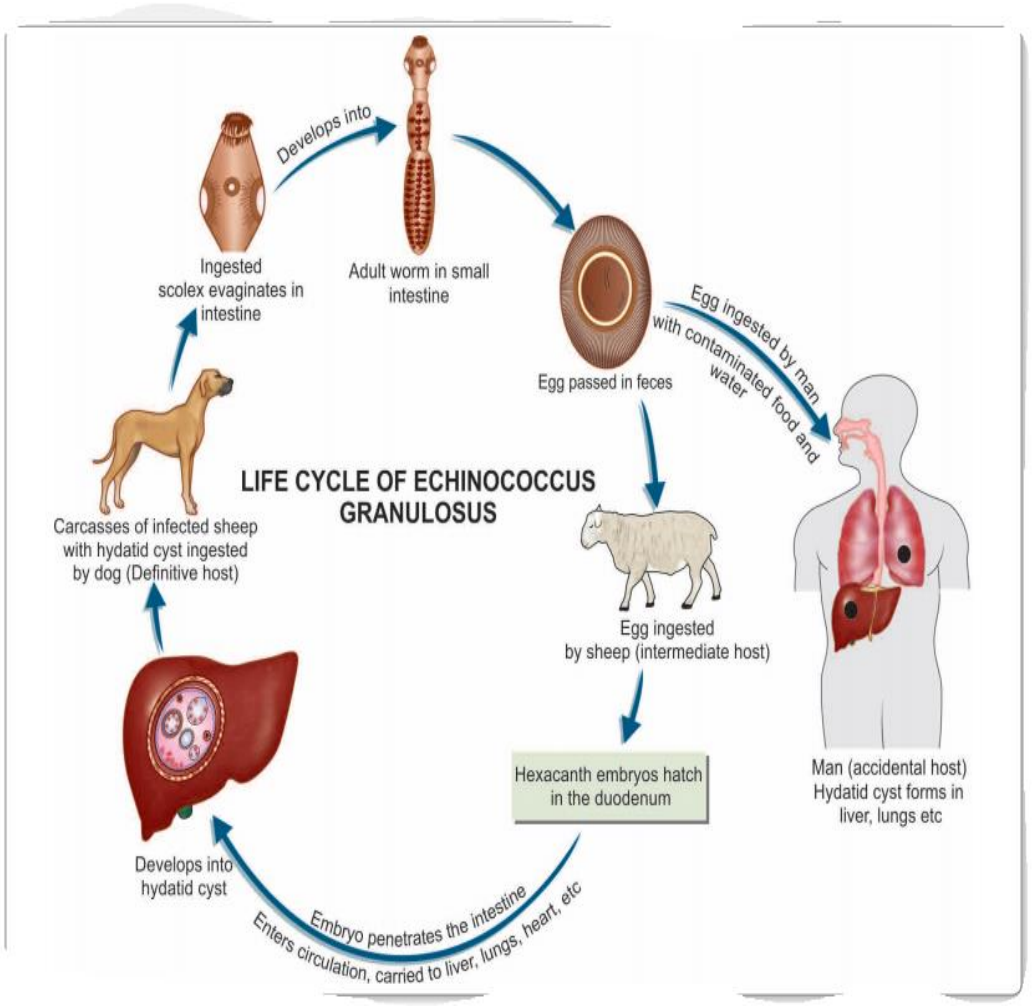
When sheep or cattle harboring hydatid cysts die or are slaughtered, dogs may feed on the carcass or offal. Dogs are infected by ingesting protoscoleces in the fertile hydatid cyst in viscera of intermediate host specially sheep, goat, cattle and camel

Inside the intestine of dogs, the scolices develop into the adult worms in about 6-7 week and produce eggs to repeat the life cycle .

One dog may have up to 17120 adult worms.

The adult worm lives from 6-30 month

When infection occurs in humans accidentaly, the cycle comes to a dead end because the human hydatid cysts are unlikely to be eaten by dogs



Pathogenesis

At the site of deposition, the embryo slowly develops into a hollow bladder or cyst filled with fluid. This becomes the hydatid cyst (Greek hydatis: a drop of water).

the hydatid cyst enlarges slowly and reaches a diameter of 0.5–1 cm in about 6 months. The growing cyst evokes host tissue reaction leading to the deposition of fibrous capsule around it.

The cyst wall secreted by the embryo consists of 3 indistinguishable layers.

€

Pericyst is the outer host inflammatory reaction consisting of fibroblastic proliferation, mononuclear cells, eosinophils, and giants cells, eventually

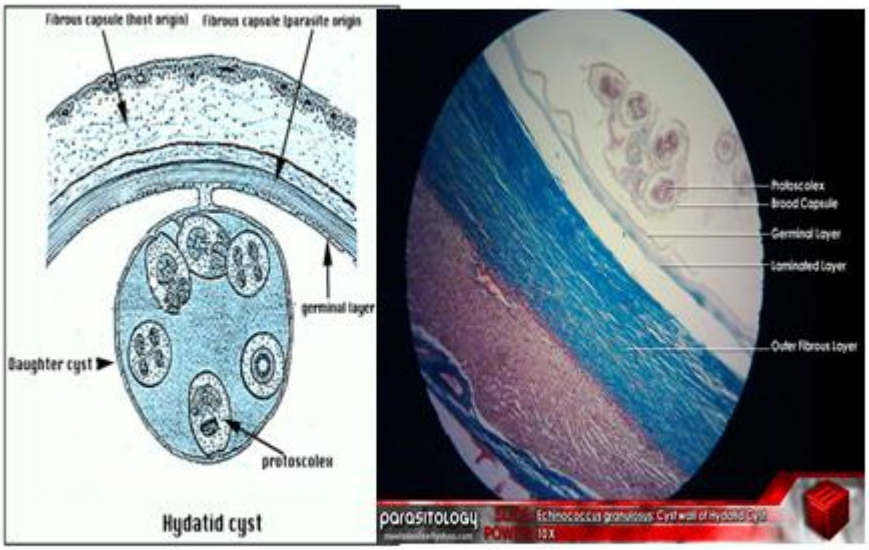
developing into dense fibrous capsule which may even calcify.

€

Ectocyst is the intermediate layer composed of characteristic a cellular, chitinous, laminated hyaline material. It has the appearance of the white of a hard boiled egg.

Endocyst is the inner germinal layer which is cellular and consists of number of nuclei embedded in a protoplasmic mass and is extremly thin .

The germinal layer is the vital layer of the cyst and is the site of asexual reproduction giving rise to brood capsules with scolices. It also secretes hydatid fluid, which fills the cyst.



Hydatid fluid: The interior of the cyst is filled with a clear colorless or pale yellow fluid called as hydatid fluid.

hydatid fluid :It contains salts (sodium chloride 0.5%, sodium sulphate, sodium phosphate, and salts of succinic acid) and proteins.

The fluid was used as the antigen for Casoni’s intradermal test.

A granular deposit or hydatid sand is found at the bottom of the cyst, consisting of free brood capsules and protoscolices and loose hooklets.

Brood capsules

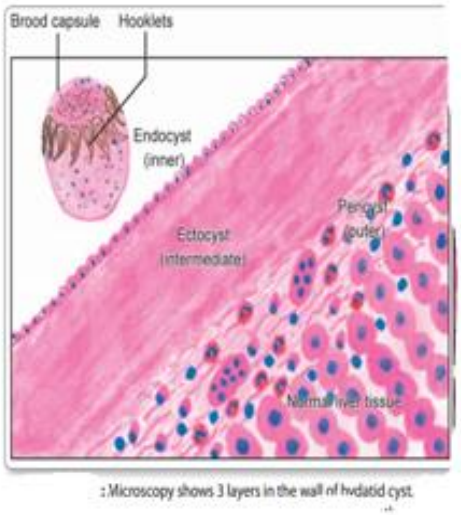
Which have only the germinal layer , containing protoscolices They are initially attached to the germinal layer by a stalk, but later escape free into the fluidfilled cyst cavity ,

From the inner wall of the brood capsules, protoscolices (new larvae) develop, which represent the head of the potential worm, complete with invaginated scolex,

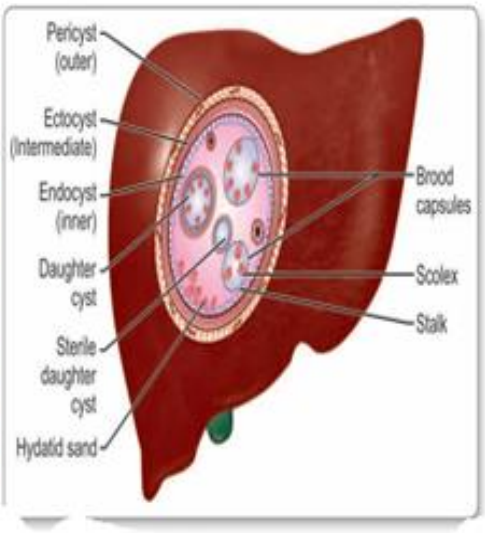
Several thousands of protoscolices develop into a mature hydatid cyst, so that this represents an asexual reproduction bearing suckers and hooklets

Inside mature hydatid cysts, further generation of cyst, daughter cysts and grand-daughter cysts may develop. which are replicas of the mother cysts .

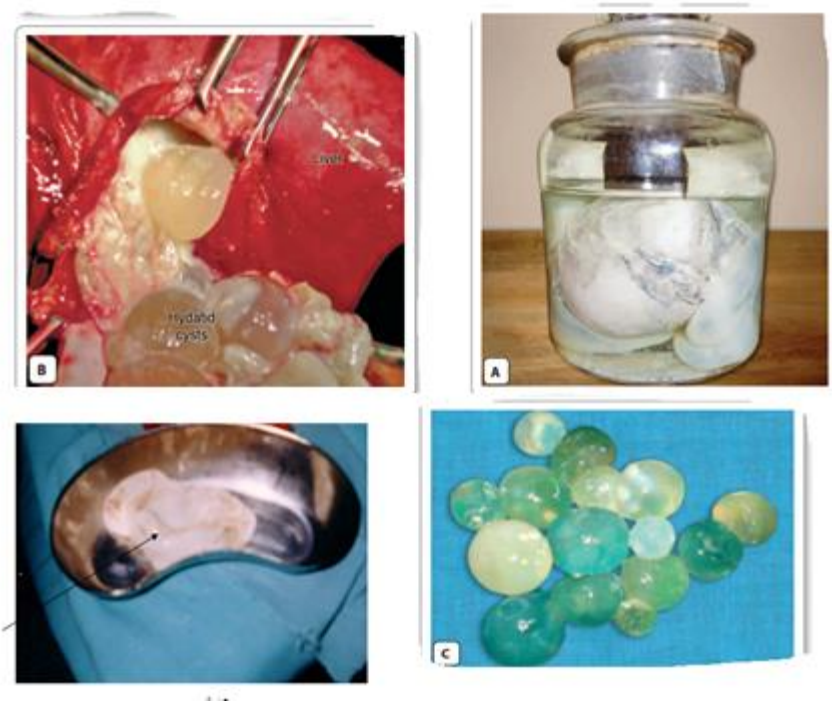
The cyst grows slowly often taking 20 years or more to become big enough to cause clinical illness



: Microscopy shows 3 layers in the wall of hydatid cyst.



: Hydatid cyst in the liver



Acephalocysts

Some cysts are sterile and may never produce brood capsules, while some brood capsule may not produce scolices. These are called acephalocysts.

Fate of hydatid cysts

The cyst may get calcified or spontaneously evacuated following inflammatory reaction. Hydatid cyst of liver may rupture into lung or other body cavity producing disseminated hydatid lesions

Clinical Features

Most of the times infection is asymptomatic and accidentally discovered.

Clinical disease develops only when the hydatid cyst has grown big enough to cause obstructive symptoms. Disease results mainly from pressure effects caused by the enlarging cysts.

the primary hydatid cyst occurs in liver(65%) , mostly in the right lobe. Hepatomegaly, pain, and obstructive jaundice are the usual mainfestations.

The next common site is the lung(15%) (most common being the lower lobe of the right lung). Cough, haemoptysis, chest pain, pneumothorax, and dyspnea constitute the clinical picture.

In the kidney (2%), hydatid cyst causes pain and haematuria.

Other sites affected include spleen (1%), brain (1%), pelvic organs, orbit, and bones (3%).

Cerebral hydatid cysts may present as focal epilepsy.

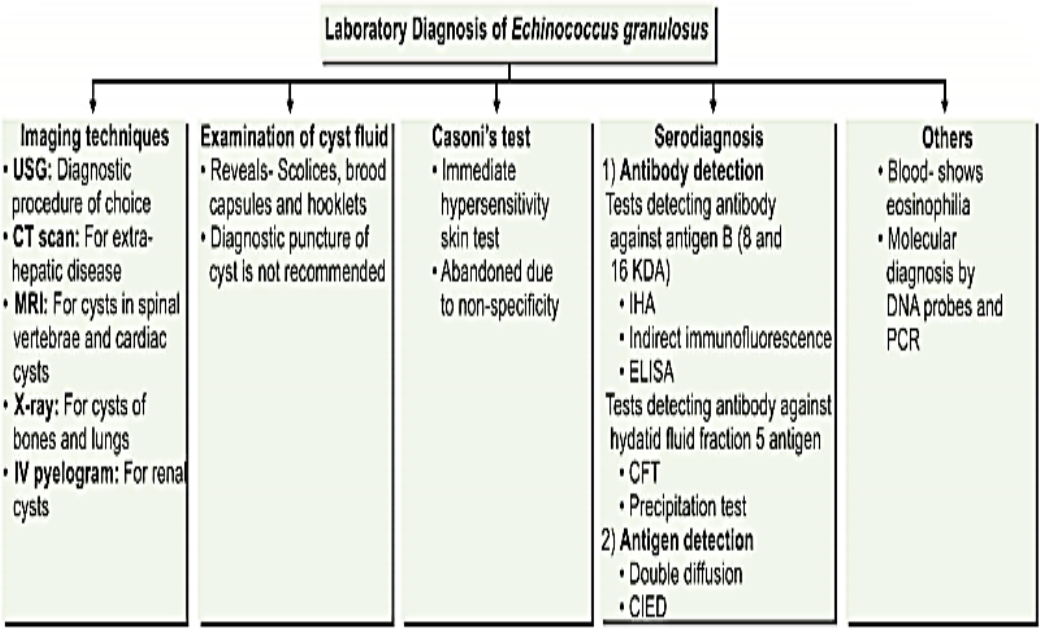
€

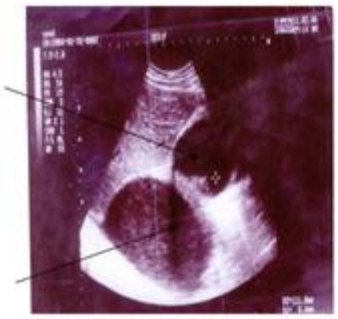
When hydatid cyst is formed inside the bones, the laminated layer is not well developed . This is called osseous hydatid cyst. Erosion of bone may lead to pathological fractures.

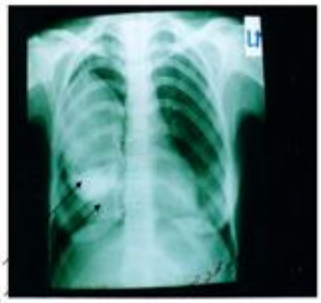
hypersensitivity to the echinococcal antigen.

The Hypersensitivity may cause urticaria.

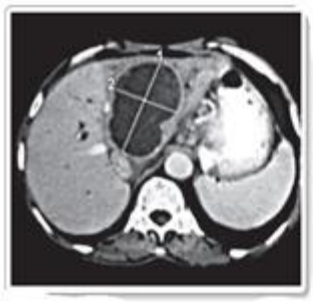
But if a hydatid cyst ruptures spontaneously or during surgical interference, massive release of hydatid fluid may cause severe, even fatal anaphylaxis.







Chest x-ray



Treatment

Traditionally surgical removal was considered as the the best mode of treatment of cysts. Currently, ultrasound staging is recommended and management depends on the stage.

In early stages, the treatment of choice is punture, aspiration, injection, and reaspiration **(PAIR).**

(PAIR) Scolicidal agents and their complications

- Cetrimide—can cause acidosis
- Alcohol 95%—can cause cholangitis
- Hypertonic saline—hypernatraemia
- Sodium hypochlorite—hypernataraemia

Note: In cases with biliary communication only hypertonic saline (15–20%) is used

Surgery

It is the treatment of choice for complicated E. granulosus

cysts

like those communicating with the biliary tract and in those cysts where PAIR is not possible.

Recurrence after surgery is common.

Preand postoperative chemotherapy with albendazole

for 2 years after curative surgery is recommended .

Other new treatment modalities include .

1-laparoscopic hydatid liver surgery .

2-percutaneous thermal ablation (PTA) of the germinal layer of the cyst using radiofrequency ablation device.

Chemotherapy

Chemotherapy with benzimidazole agents are restricted to

residual, postsurgical, and inoperable cysts.

Albendazole and praziquantel have proved beneficial

Prophylaxis

. Echinococcus granulosus infection can be prevented by

-Ensuring pet dogs do not eat animal carcass or offal.

-Periodical deworming of pet dogs.

- Destruction of stray and infected dogs.

-Mantaining personal hygiene such as washing of hands

after touching dogs and avoidance of kissing pet dogs

Echinococcus Multilocularis

causes rare but serious condition of alveolar or multilocular hydatid disease in humans.

It is found in the northern parts of the world, from Siberia in the East to Canada in the West.

The adult worm is smaller than E. granulosus and lives in the intestines of foxes, dogs, and cats which are the definitive host.

Rodents are the main intermediate hosts.

Human infection develops from eating fruits or vegetables contaminated with their feces.

E. multilocularis leads to multilocular hydatid cyst

The liver is the most commonly affected organ. The multilocular infiltrating lesion appears like a grossly invasive growth, without any fluid or free brood capsule or scolices which can be mistaken for a malignant tumor

Patients present with upper quadrant and epigastric pain. Liver enlargement and obstructive jaundice may also be present. It may also metastasize to the spleen, lungs, and brain in 2% cases.

The prognosis is very bad if untreated,

Surgical resection, when possible, is the best method of treatment.

Albendazole therapy is recommended for 2 years after curative surgery. In those cases, where surgery is not possible, treatment with albendazole is recommended.

Hydatid cyst of Echinococcus vogeli

Disease: Polycystic hydatid disease .

Habitat: in the small intestine of bush dog (definitive host) in latin America.

Rodent (natural intermediate host).

Morphology:

Adult differs from E. granulosus in greater length 3.9 - 5.6 mm

Polycystic hydatid: is alveolar in characters but less than that of E. multilocularis,

so it is intermediate between cystic and alveolar hydatid disease, present like a mass of tumor in the liver

lec:8

CYCLOPHYLLIDEAN TAPEWORMS **د٠ سعيد حميد**

Taenia Saginata and Taenia Solium

Common name

Taenia saginata Beef tapeworm

Taenia solium Pork tapeworm .

The name Taeniais derived from the Greek word meaning tape or band.

T. saginata is worldwide in distribution, but the infection

is not found in vegetarians and those who do not eat beef .

T. Solium is also worldwide in distribution except in the

countries and communities, which proscribe pork as

taboo.

Cestodes living in small intestine

- Diphyllobothrium latum
- Taenia solium

- Taenia saginata
- Hymenolepis nana

Habitat

The adult worms of both T. saginata and T. solium live in the small intestine, commonly in the jejunum.

Morphology

Adult Worm of T. saginata

The adult T. saginata worm is white in color, ribbonlike, dorsoventrally flattended, and segmented, measuring 5–10 meter in length.

The adult worm consists of head (scolex), neck, and strobila (body).

The general features of adult worm are similar to any cyclophyllidean cestodes.

Scolex:The scolex (head) of T. saginata is about 1–2 mm in diameter, quadrate in crosssection, bearing 4 hemispherical suckers situated at its four angles.

The scolex has no rostellum or hooklets (which are present in T. solium) T. saginata is, therefore called the unarmed tape worm.The suckers serve as

the sole organ for attachment

The neck is long and narrow.

The strobila (trunk) consists of 1000 to 2000 proglottides or segments—immature, mature and gravid.

The gravid segments are nearly about 20 mm long and 5 mm broad.

The segment contains male and female reproductive structures.

The testes are numerous, 300 to 400 (twice as many as in T. solium).

The gravid segment has 15 to 30 lateral branches (as against 7 to 13 in T. solium). It differs from T. solium also in having a prominent vaginal sphincter and in lacking the accessory ovarian lobe. The common genital

pore opens on the lateral wall of the segments.

The gravid segments break away and are expelled singly, actively forcing their way out through the anal sphincter.

As there is no uterine opening, the eggs escape from the

uterus through its ruptured wall.

Adult Worm of T. solium

The adult worm is usually 23 meters long.

The proglottides number less than a thousand. They resemble those of T. saginata in general

The scolex of T. solium is small and globular about 1 mm in diameter, with 4 large cuplike suckers (0.5 mm in diameter), and a conspicuous rounded rostellum, armed with a double row of alternating round and small dagger-shaped hooks, 20–50 in number.

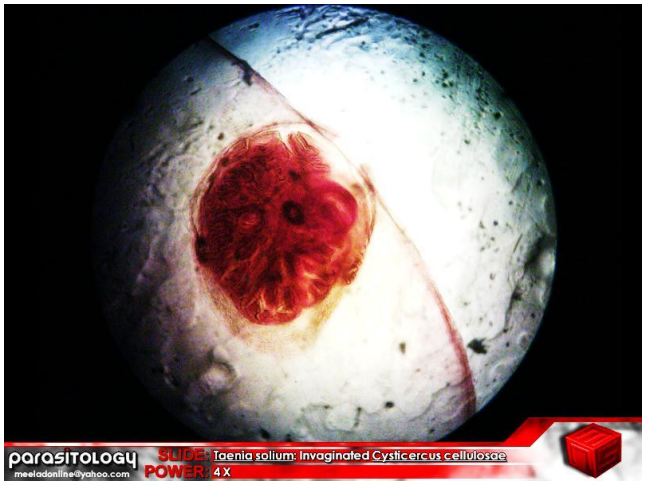
The neck is short .

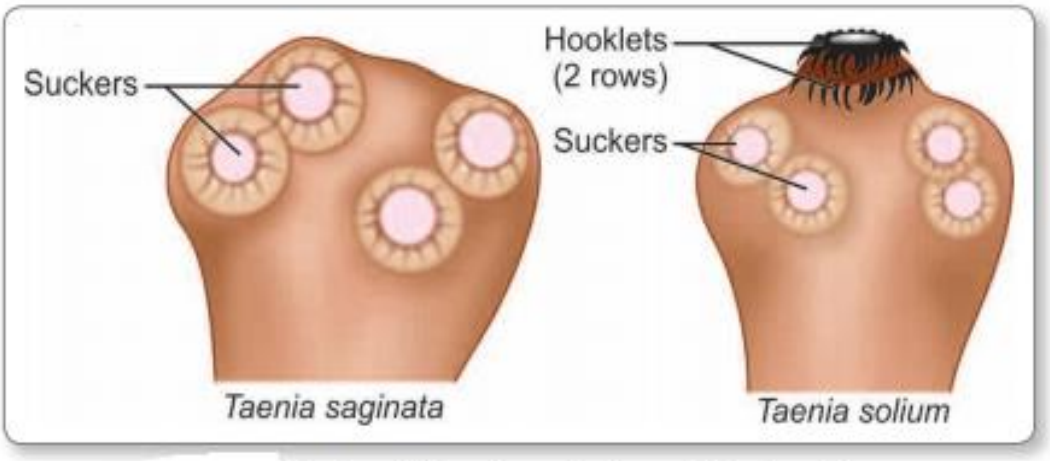
The gravid segments are twice as long as broad, 12 mm by 6 mm.

The testes are composed of 150 to 200 follicles. There is an accessory lobe for the ovary. The vaginal sphincteris absent.

The uterus has only 5 to 10 (under 13) thick lateral branches. A lateral thicklipped genital pore is present, alternating between the right and left sides of adjacent segments.

The gravid segments are not expelled singly, but pass passively out as short chains. The eggs escape from the ruptured wall of the uterus.





Scolex of *Taenia saginata* and *Taenia solium*

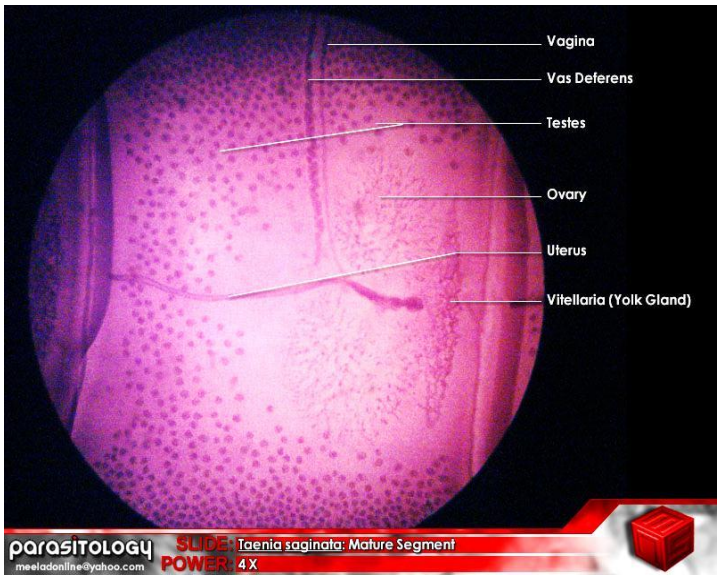
Difference between Taenia saginata and Taenia solium

	Taenia saginata	*Taenia solium*
Length	5–10 m	2–3 m
Scolex	Large quadrate	Small and globular
	Rostellum and hooks are absent	Rostellum and hooks are present
	Suckers may be pigmented	Suckers not pigmented
Neck	Long	Short
Proglottids	1,000–2,000	Below 1,000
Measurement (gravid segment)	20 mm × 5 mm	12 mm × 6 mm
Expulsion	Expelled singly	Expelled passively in chains of 5 or 6
Uterus	Lateral branches 15–30 on each side; thin and dichotomous	Lateral branches 5–10 on each side; thick and dendritic
Vagina	Present	Absent
Accessary lobe of ovary	Absent	Present
Testes	300–400 follicles	150–200 follicles
Larva	Cysticercus bovis; present in cow not in man	Cysticercus cellulosae; present in pig and also in man
Egg	Not infective to man	Infective to man
Definitive host	Man	Man
Intermediate host	Cow	Pig, occasionally man
Disease	Causes intestinal taeniasis	Causes intestinal taeniasis and cysticercosis

mature segment

Taenia saginata
2 ovary lobes

Taenia solium
3 ovary lobes



gravid segments

Taenia solium

Taenia saginata

Eggs

Eggs of both species are indistinguishable.

The egg is spherical, measuring 30–40 µm in diameter. It has a thin hyaline embryonic membrane around it, which soon disappears after release.

The inner embryophore is radially striated and is yellowbrown due to bile staining .

In the center is a fullydeveloped embryo (oncosphere) with 3 pairs of hooklets (hexacanth embryo).

The eggs do not float in saturated salt solution.

The eggs of T. saginata are infective only to cattle and not to humans, whereas the eggs of T. solium are infective to pigs and humans too.

Larva

The larval stage of Taeniais called as cysticercus.

Cysticercus bovis is the larva of T. saginata.

Cysticercus cellulosae is the larva of T. solium .

€ The larva (cysticercus bovis) is infective stage for humans.

€ The cysticercus is an ovoid, milkywhite opalescent fluidfilled vesicle measuring about 5 mm × 10 mm in diameter, and contains a single invaginated scolex (bladder worm).

The cysticercus are found in the muscles of mastication, cardiac muscles, diaphragm and tongue of infected cattle .

€

They can be seen on visual inspection as shiny white dots in the infected beef (measly beef).

Cysticercus bovis is unknown in humans

Cysticercus cellulosae

€ It is the larval form of T. solium and also the infective

Stage of these parasite.

€ It can develop in various organs of pig as well as in man.

€ The cysticercus cellulosae or ‘bladder worm’is ovoid opalescent milkywhite, measuring 8–10 mm in breadth and 5 mm in length

€

The scolex of the larva, with its suckers, lies invaginated within the bladder and can be seen as a thick white spot. It remains viable for several months .

Life Cycle of Taenia Saginata

T. Saginata passes its life cycle in 2 hosts.

Definitive host: Humans are the definitive hosts .

Intermediate host: Cattle (cow or buffalo) .

Infective stage: Cysticercus bovis (larval stage) is the infective stage to man, while eggs are infective to cattle

The adult worm lives in the small intestine of man.

The gravid segments from the adult worm breakaway and are expelled singly. They actively force their way out through the anal sphincter.

The eggs or gravid segments are passed out with feces on the ground.

The eggs deposited in soil remain viable for several weeks. They are infective to cattle, which ingest the eggs while grazing.

Development in Cattle

When egg ingested by cattle (cow or buffalo), the egg shell

ruptures releasing onchosphere in the duodenum.

The onchospheres, with their hooklets penetrate the intestinal wall, reach the mesenteric venules or lymphatics and enter the systemic circulation.

They get filtered out in the striated muscles, particularly

in muscles of the tongue, neck, shoulder, and in the myocardium. In these sites, the onchosheres lose their hooks and in about 60–70 days develop in the

mature larva, cysticercus bovis.

The cysticercus can live in flesh of cattle for about 8

months, but can develop further only when ingested by

man, its definitive host

Development in Man

Man acquires infection by ingesting raw or undercooked

beef containing cysticercus .

The cysticercus are digested out of the meat in the stomach.

In the upper part of the small intestine, the head (scolex)

evaginated out of the cysticercus, becomes attached to

the mucosa, and by gradual strobilization develops into

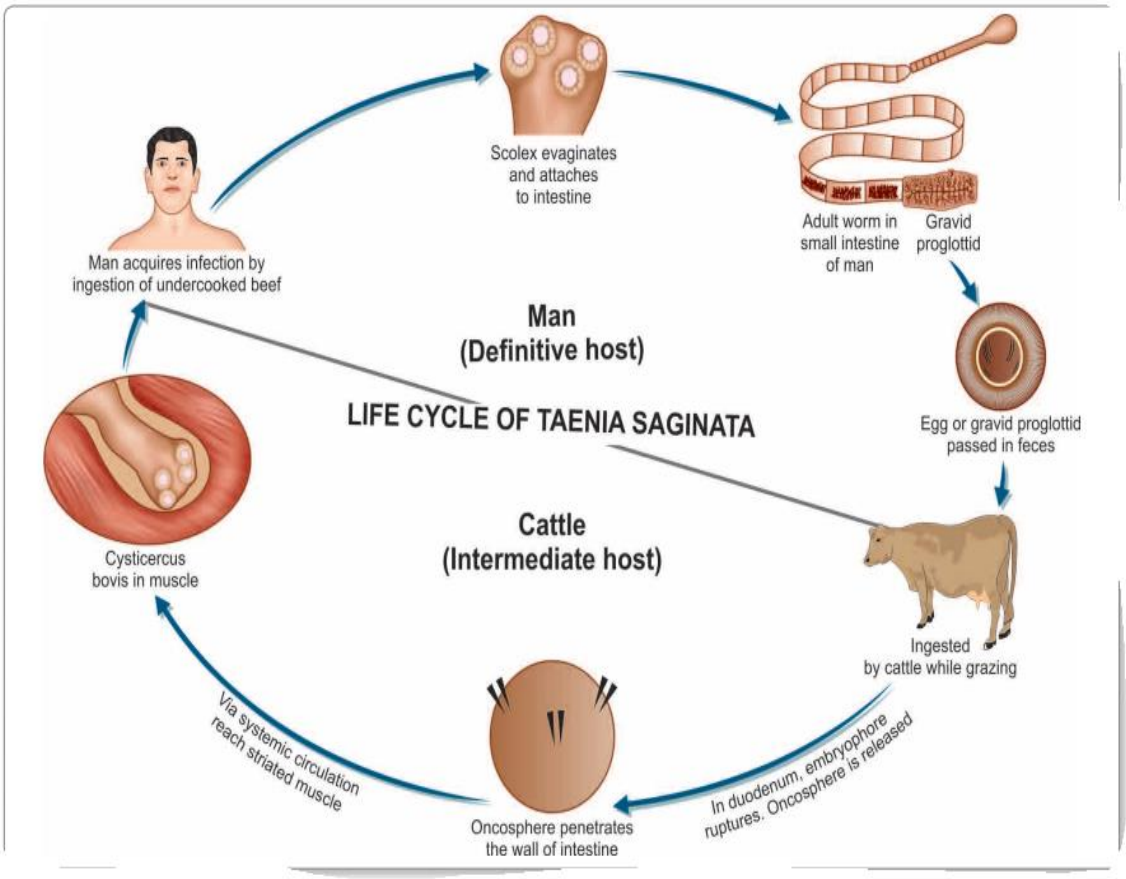
the adult worm in about 2–3 months.

The adult worm has a life span of 10 years or more.

Infection in usually with a single worm, but sometimes

multiple infection is seen and 25 or more worms have been

reported in patients.



Life Cycle of Taenia Solium

Definitive host: Man

Intermediate host: Pig

Infective stage:Cysticercus cellulosae (larva)

Humans are infected by consuming inadequately cooked pork containing cysticercus cellulosae (measly pork).

When Taenia leads to cysticercosis, the life cycle is as follows:

Definitive host and Intermediate host: Both man

Infective stage:Eggs of T. solium(not larva)

Mode of infection:Man acquires infection by ingesting eggs with contaminated food and water.

€

Autoinfection: man harboring adult worm may autoinfection oneself, either by unhygenic personal habits or by reverse peristalsis of the intestine.

The further development of the eggs is similar in man

and pigs.

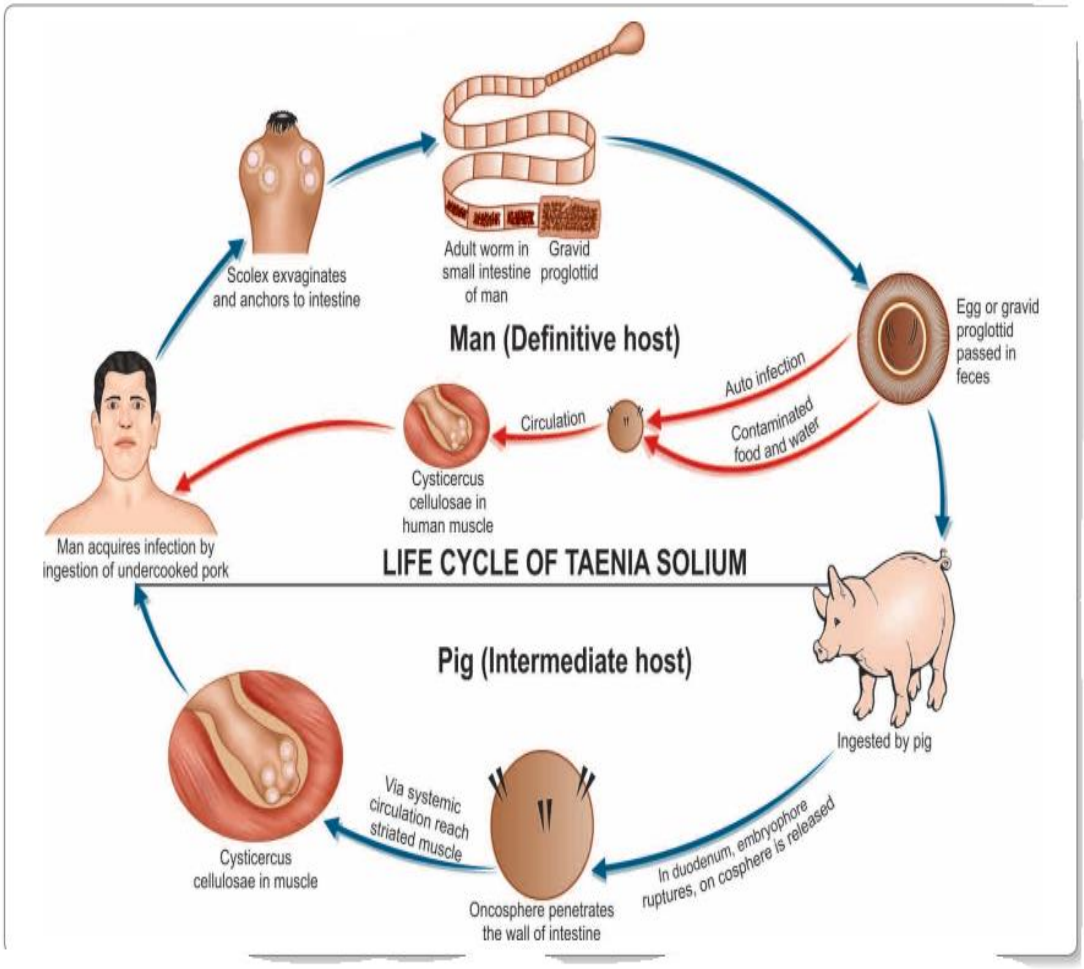
The oncospheres are released in the duodenum or jejunum and penetrate the intestinal wall.

They enter the mesenteric venulesor lymphatics and are carried in systemic circulation to the different parts of the body .

They are filtered out principally in the muscles, where they develop into the larval stage, cysticercus cellulosae in about 60–70 days.

In humans, it is a dead end and the larvae die without

further development



Pathogenicity and Clinical Features

Intestinal Taeniasis

It can be caused by both T. saginata and T. solium.

Patients may be frightened by noticing the proglottids passed in their feces.

When the infection is symptomatic, vague abdominal discomformt, indigestion, nausea, diarrhea, and weight loss may be present. Occasional cases of acute intestinal obstruction, acute appendicitis, and pancreatitis have also been reported.

Cysticercosis

It is caused by larval stage (cysticecus cellulosae) of T. solium.

Cysticercus cellulosae may be solitary or more often multiple.

Any organ or tissue may be involved, the most common being subcutaneous tissues and muscles. It may also affect the eyes, brain, and less often the heart, liver,lungs, abdominal cavity, and spinal cord.

The cysticercus is surrounded by a fibrous capsule except in the eye and ventricles of the brain.

The larvae evoke a cellular reaction starting with infiltration of neutrophils, eosinophils, lymphocytes, plasma cells, and at times, giant cells. This is followed by fibrosis and death of the larva with eventual calcification.

The clinical features depend on the site affected Subcutaneous nodules are mostly asymptomatic

€ Muscular cysticerosis may cause acute myositis

€ Neurocysticerosis (cysticercosis of brain) is the most common and most serious form of cysticercosis. about 70% of adultonset epilepsy is due to neurocysticercosis.

Other clinical features of neuro cysticercosis are increased intracranial tension, hydrocephalus, psychiatric disturbances, meningoencephalitis, transient paresis, behavioral disorders

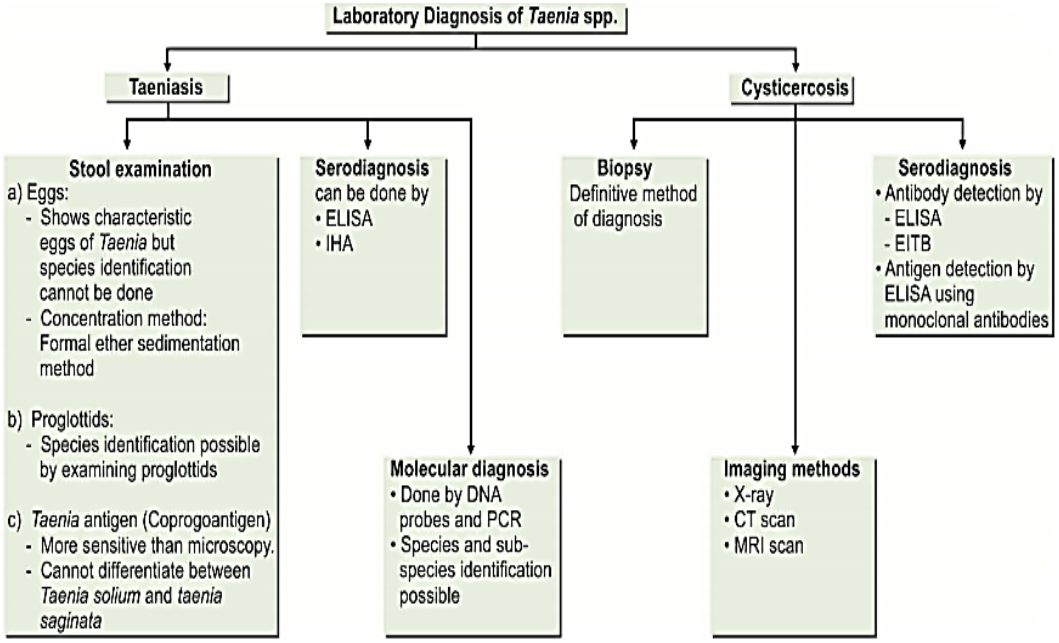
aphasia, and visual disturbances.

the second most common cause of intracranial space occupying lesion (ICSOL) after Tuberculosis in India.

€

In ocular cysticercosis, cysts are found in subretinal space and conjunctiva, vitreous humor

The condition may present as blurred vision or loss of vision, iritis, uveitis, and palpebral conjunctivitis



Prophylaxis

Beef and pork to be eaten by man should be subjected to effective inspection for cysticercus in slaughter house.

Avoidance of eating raw or undercooked beef and pork. The critical thermal point of cysticercus is 56°C for 5 minutes.

Maintainence of clean personal habits and general sanitary measures.

For control of cysticercosis, prevention of fecal contamination of soil, proper disposal of sewage and avoidance of eating raw vegetables grown in polluted soil are useful measures

Detection and treatment of persons harboring adult worm, as they can develop cysticercosis due to autoinfection

Treatment

Intestinal Taeniasis

Single dose of praziquantel (10–20 mg/kg) is the drug of choice.

Niclosamide (2 g), single dose, is another effective drug.

Purgation is not considered necessary.

Cysticercosis

For cysticercosis, excisionis the best method, wherever

possible.

Asymptomatic neurocysticercosis requires no treatment.

.For symptomatic cerebral cysticercosis.

praziquantel in a dose of 50 mg/kg in 3 divided doses for 20–30 days

albendazole in a dose of 400 mg twice daily for 30 days may be administered.

Corticosteroids may be given along with praziquantel or albendazole to reduce the inflammatory reactions caused by the dead cysticercus larva.

In addition, antiepileptic drugs should be given until the reaction of the brain has subsided.

Operative intervention is indicated for hydrocephalus

Taenia Saginata Asiatica

T. saginata asiatica is closely related to T. saginata and is found mainly in Asia.

It is morphologically similar to T. saginata except. It is smaller than T. Saginata.

€ Intermediate host is pig (not cow).

Its cysticerci are located primarily in liver of the pig (not muscle).

Clincial features, diagnosis and treatment are similar to that of T. saginata

Hymenolepis Nana

Common name: Dwarf tape worm .

Smallest cestode infecting man: Hymenolepis nana.

Longest cestode infecting man: Diphyllobothrium latum

Distribution

It is cosmopolitan in distribution but is more common in

warm than in cold climates.

Infection is most common in school children .

The adult worm lives in the proximal ileum of man

Morphology

Adult Worm

H. Nana is the smallest intestinal cestode that infects man.

It is 5–45 mm in length and less than 1 mm thick.

The scolex has 4 suckers and a retractile rostellum with a single row of hooklets.

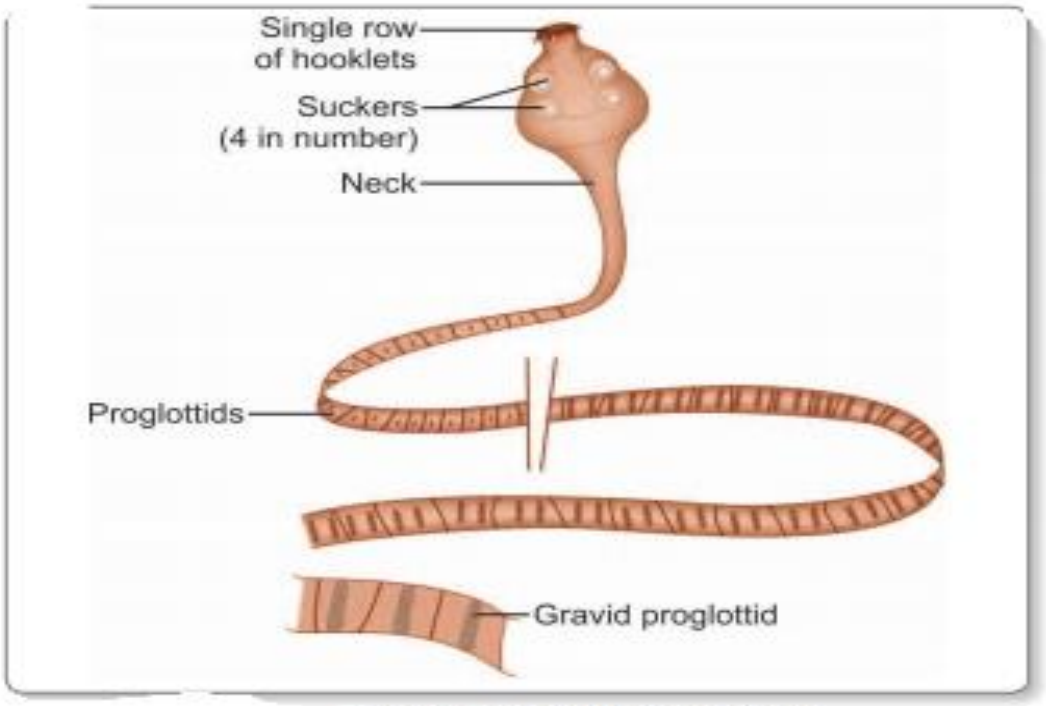
The long slender neck is followed by the strobila consisting of 200 or more proglotlids, which are much broader than long.

Genital pores are situated on the same side along the margins.

The uterus has lobulated walls and the testis are round and 3 in number.

Eggs are released in the intestine by disintegration of

the distal gravid segments



Adult worm of *Hymenolepis nana*

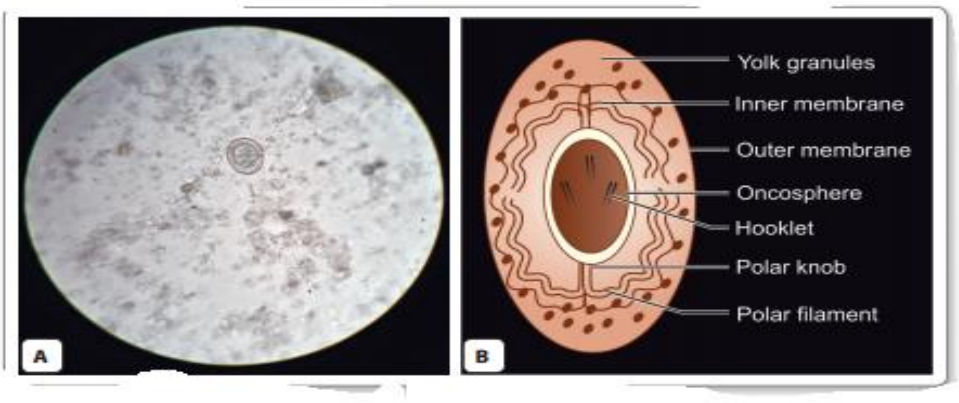
Egg

The egg is roughly spherical or ovoid, 30–40 µm in size.

It has a thin colorless outer membrane and inner

Embryophore enclosing the hexacanth oncosphere

The space between 2 membranes contains yolk granules and 4–8 thread like polar filaments arising from 2 knobs on the embryophore.



They are immediately infective and unable to survive

for more than 10 days in external environment.

Life Cycle

Definitive host : Man.

There is no intermediate host.

Mode of transmission: Infection occurs by ingestion of the food and water contaminated with eggs

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Internal autoinfection may also occur when the eggs released in the intestine hatch there itself .

External autoinfection occurs when a person ingest own eggs by fecal oral route.

When the eggs are swallowed, or in internal autoinfection, they hatch in the small intestine.

The hexacanth embryo penetrates the intestinal villus and develops into the cysticercoid larva.

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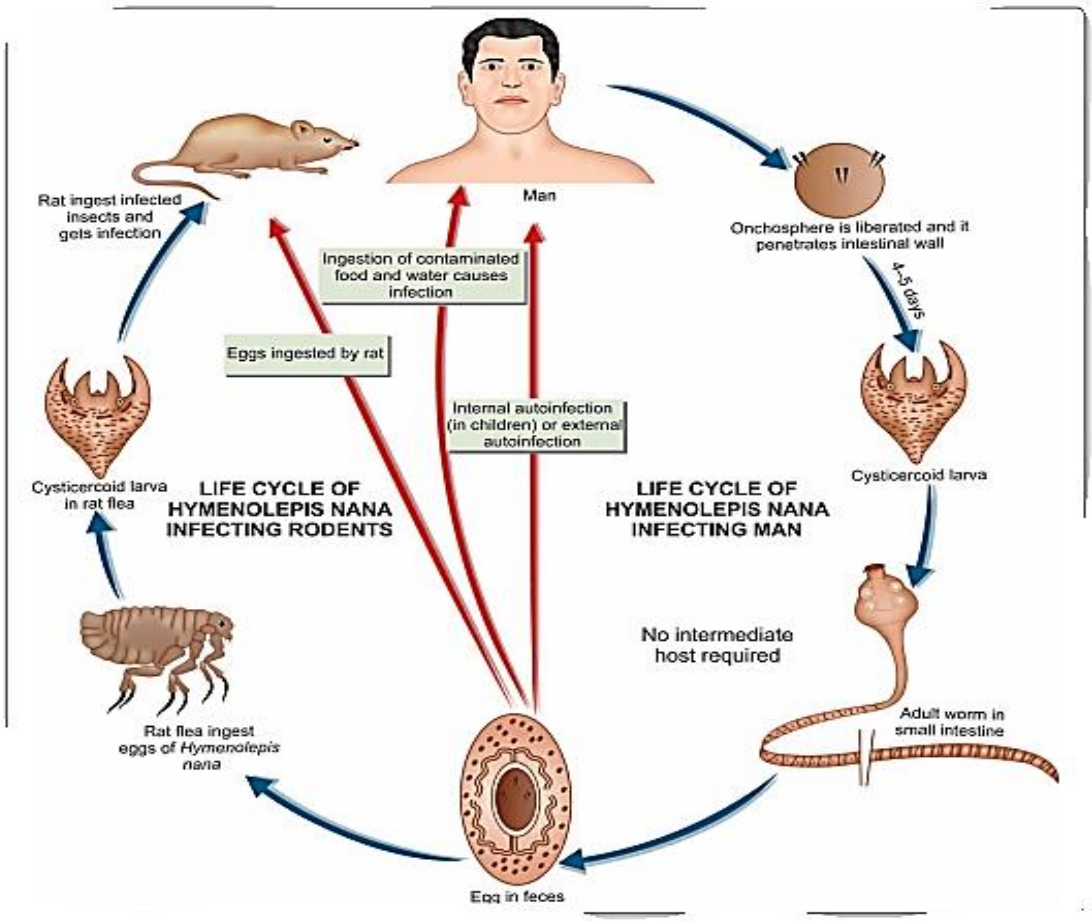
This is a solid pyriform structure, with the vesicular anterior end containing the invaginated scolex and a short conical posterior end.

After about 4 days, the mature larva emerging out of the villus evaginates its scolex and attaches to the mucosa.

It starts strobilization, to become the mature worm, which begins producing eggs in about 25 days.

A different strain of H. nana infects rats and mice.

The eggs passed in rodent feces are ingested by rat fleas (Xenopsylla cheopis and others), which acts as the intermediate host



Clinical Features

Hymenolopiasis occurs more commonly in children.

There are usually no symptoms but in heavy infections, there is nausea, anorexia, abdominal pain, diarrhea, and irritability.

Sometimes pruritus may occur due to an allergic response

Diagnosis

The diagnosis is made by demonstration of characteristic

eggs in feces by direct microscopy

ELISA test has been developed with 80% sensitivity

Treatment

Praziquantel (single dose of 25 mg/kg) is the drug of choice, since it acts both against the adult worms and the cysticercoid larva in the intestinal villi.

Prophylaxis

Maintenance of good personal hygiene and sanitary

Improvement

Avoiding of consumption of contaminated food and

Water with eggs.

Rodent control

Hymenolepis Diminuta

This is called the rat tapeworm and is a common parasite of rats and mice.

Size 10–60 cm in length.

life cycle is similar to that of the murine strain of H. nana.

Rarely, human infection follows accidental ingestion of

infected rat fleas.

Human infection is asymptomatic.

Dipylidium Caninum

This common tapeworm of dogs and cats, it may accidentally

cause human infection, mainly in children.

Morphology

The adult worm in the intestine is about 10–70 cm long

The scolex has 4 prominent suckers and a retractile rostellum with upto 7 rows of spines .

The mature proglottid has 2 genital pores, 1 on either side, hence the name Dipylidium (dipylos—2 entrances)

Gravid proglottids are passed out of the anus of the host singly or in groups

Life Cycle

Definitive host: Dogs, cats, and rarely man.

Intermediate host: Fleas.

Man acquires infection by ingestion of flea harboring cysticercoid larva.

The eggs or proglottids passed in feces of dogs and cats .

Eggs are eaten by dog fleas and cat fleas. The embryo develops into a cysticercoid larva .

infection is transmitted when the adult fleas containing the larvae are eaten by dogs, cats, or rarely humans,

Clinical Features

Human infection is generally asymptomatic, but the actively motile proglottids passed in stool .

Diagnosis

The diagnosis is made by detection of proglottids or eggs in stool

Treatment

The drug of choice is praziquantel

Diphyllobothrium Latum

Common name: Fish tape worm/Broad tape worm.

disease called Diphyllobothriasis

Distribution

occurs in central and northern Europe, particularly in the Scandinavian countries. It is also found in Siberia, Japan, North America, and Central Africa.

Habitat

The adult worm is found in the small intestine, usually in the

ileum,

Morphology

Adult worm

It is ivory-colored and very long, measuring upto 10 meters or more. It is the largest tape worm inhabiting the small intestine of man

the adult worm has 3 parts: scolex, neck, and strobila

Scolex (head) is spatulate or spoon-shaped, about 2–3 mm long and 1 mm broad. It carries 2 slitIike longitudinal sucking grooves (bothria), one dorsal and the other ventral.

The scolex lacks suckers and hooks.

Neck is thin.

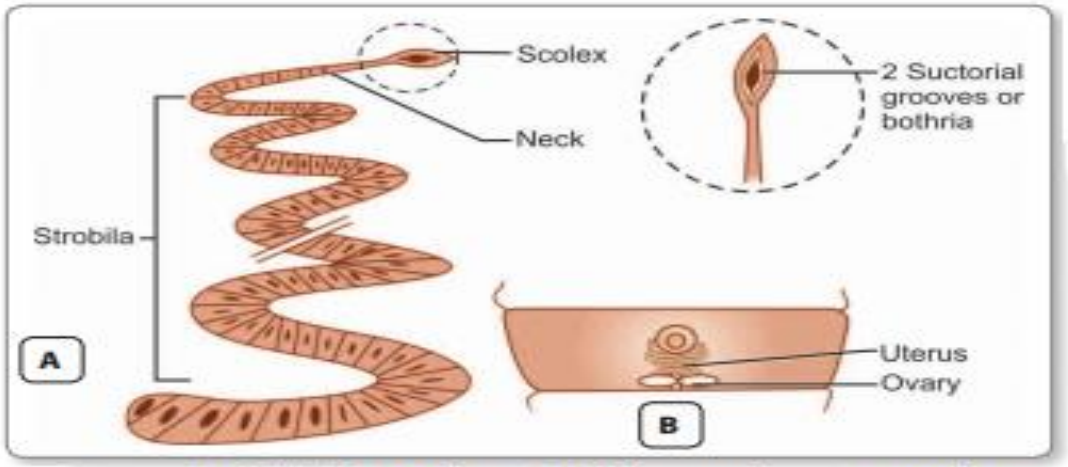
Strobila consists of 3,000–4,000 proglottids, consisting of immature, mature, and gravid segments .

The mature proglottid is broader than long, about 2–4 mm long and 10–20 mm broad and is practically filled with male and female reproductive organs .

The female reproductive organs are arranged along the midline, lying ventrally. The ovary is bilobed. The large rosettelike uterus lies convoluted in the center.

Three genital openings are present ventrally along the mid line

The fertilized ova develop in the uterus and are discharged periodically through the uterine pore.



: *Diphyllobothrium latum* **A.** Adult worm showing spatulate scolex, neck, and strobila **B.** Mature proglottid

Egg

a single worm may pass million eggs in a day

Egg is broadly ovoid, about 65 µm by 45 µm, with a

thick, light brown shell .

It has an operculum at one end and often a small knob at the other.

The freshly passed egg contains an

immature embryo surrounded by

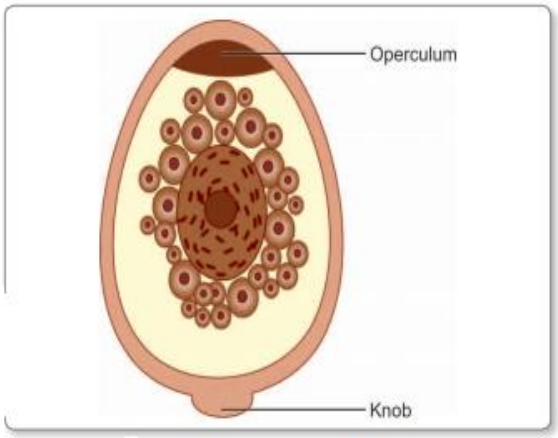
yolk granules.

The eggs are resistant to chemicals

but are killed by drying.

The embryo with 6 hooklets inside

the egg is called the oncosphere.



Operculated egg of *Diphyllobothrium latum*

Larval Stages

There are 3 stages of larval development:

First stage larva (coracidium)

Second stage larva (procercoid)

Third stage larva (plerocercoid).

Life Cycle

Definitive hosts: Man, dog, and cat. Man is the optimal host.

First intermediate host: Fresh water copepod, mainly of

genera Cyclops or Diaptomus.

Second intermediate host: Fresh water fish (salmon, trout etc.).

Infective form to human: Third stage plerocercoid larva.

The adult worm lives in the small intestine. It lays operculated eggs which are passed along with the feces in water .

The freshlypassed egg contains an immature embryo surrounded by yolk granules..

The embryo with 6 hooklets (hexacanth embryo) inside the egg is called the oncosphere.

In water, it matures in about 10–15 days and ciliated first stage larva, called coracidium emerges through the operculum.

Coracidium (first stage larva) can survive in water for about 12 hours, by which time it should be ingested by the fresh water crustacean copepod cyclops, which is the first intermediate host .

In the midgut of the cyclops, the coracidium casts off its ciliated coat and by means of its 6 hooklets, penetrates into the hemocele (body cavity).

In about 3 weeks, it develop into the elongated second stage Larva about 550 µm long, which is called the procercoid larva,

Procercoid larva has a rounded caudal appendage.

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If the infected cyclops is now eaten by a freshwater fish

(second intermediate host), the procercoid larva penetrates the intestine of the fish and grows.

In the fish, procercoid larva looses its caudal appendage

and develops into the third stage larva called the plerocercoid larva

Plerocercoid larva

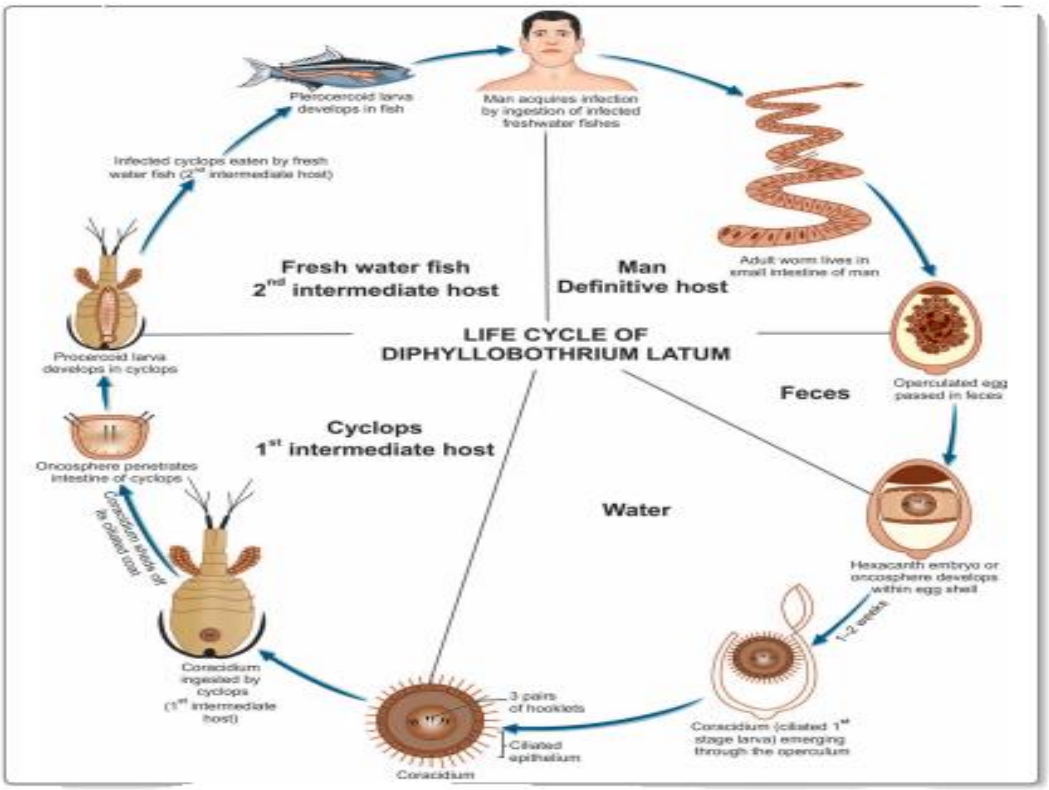
This is the infective stage for humans.

Man gets infection by eating raw or undercooked fish containing plerocercoid larva.

The larva develops into adult worm in the small intestine

in about 5-6 week and produce eggs to repeat the life cycle

The adult worm may live for about 10 years or more



Pathogenicity and Clinical Features

The pathogenic effects of diphyllobothriasis depend on the mass of the worm

In some persons, infection may be entirely asymptomatic,

while in others there may be an evidence of mechanical

obstruction.

Transient abdominal discomfort, diarrhea, nausea, weakness,.

Weight loss, and anemia are the usual manifestations.

The anemia (pernicious anemia) develops because the tape worm absorbs large quantity of vit B12 and interferes with its ileal absorption, leading to vit B12deficiency

Patients may be frightened by noticing the strands of proglottids passed in their feces.

Diagnosis

Stool Microscopy

Eggs are passed in very large number in feces, and therefore,

their demonstration in feces offers an easy method of diagnosis.

The Proglottids passed in feces can also be identified by their morphology.

Serodiagnosis

A coproantigen detection test is available to diagnose

diphyllobothriasis.

Treatment

Praziquantel in a single dose of 10 mg/kg is effective.

Parenteral vit B12 should be given, if B12 deficiency is present.

Prophylaxis

Infection can be prevented by

-Proper cooking of fish

-Deep freezing of fish(–10°C for 24–48 hours) .

-Prevention of fecal pollution of natural waters

-Periodical deworming of pet dogs and cats