Parasitology

محاضرة رقم 1

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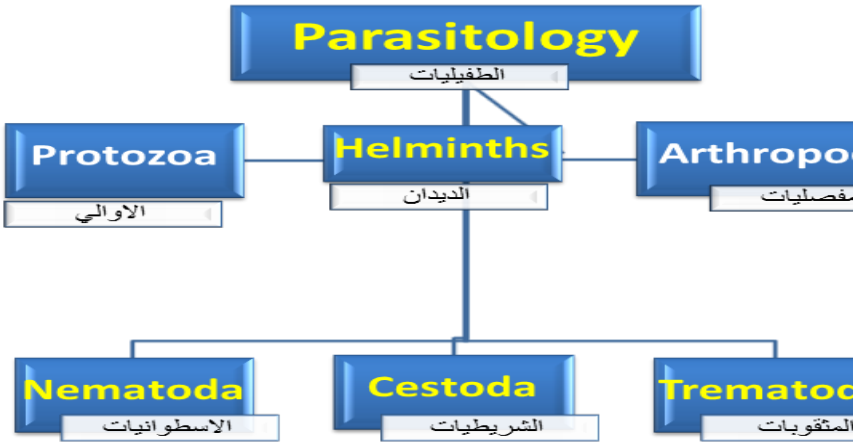
Introduction(Nematoda)

Medical and veterinary parasitology

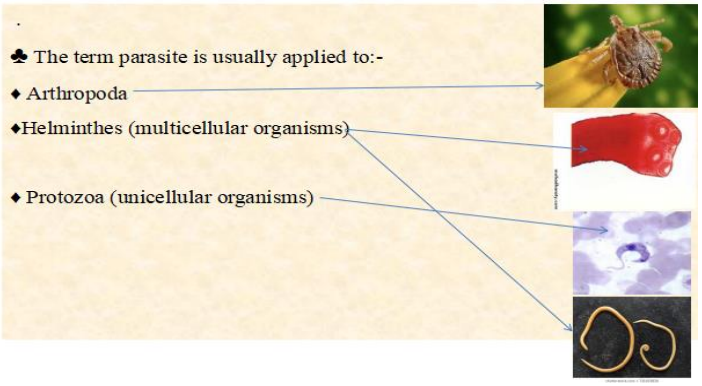
Parasitology:-it's a science that deals with study the phenomenon of parasitism or parasitic infection.

All parasites are divided into 3 important phylum:-



Terms used in parasitology

-Parasitism : its association between two organism one of them called parasite is the beneficiary and the other called host it always get harm from the parasite .
-Parasite : is a smaller organism that lives on or in a larger organism called the host get from which the shelter and nutrition .



Parasitology

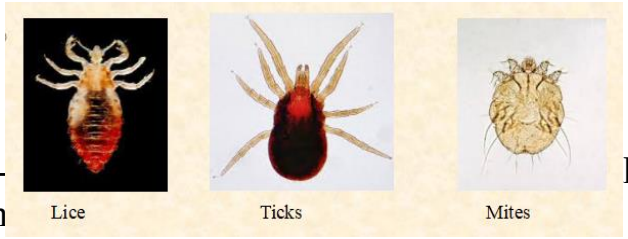
Parasite

Parasites can also be classified as:

1-Ectoparasite: Ectoparasites inhabit only the body surface of the host without penetrating the tissue.

Example:- Lice, ticks, and mites .

The term infestation is often employed for parasitization with ectoparasites.



2- hin the body of the host and is said to cause an in

Most of the protozoan and helminthic parasites causing human and animales disease are endoparasites.

Endoparasite

Endoparasites:- can further be classified as:-

1-Obligate parasite: The parasite, which cannot exist without a host.
e.g. Toxoplasma gondii and Plasmodium.

2-Facultative parasite: Organism which may either live as parasitic form or as free living form. e.g. Naegleria floweri.

3-Accidental parasites: Parasites, which infect an unusual host are known as accidental parasites. Echinococcus granulosus infects man accidentally, giving rise to hydatid cysts.

4-Aberrant parasites: Parasites, which infect a host where they cannot develop further are known as aberrant or wandering parasites, e.g. Toxocara canis (dog roundworm) infecting humans.

Host

Host:- is defined as an organism, which harbors the parasite and provides nourishment and shelter to latter and is relatively larger than the parasite.

Types of host:-

1-Definitive host: The host, in which the adult parasite lives and undergoes sexual reproduction .e.g. mosquito acts as definitive host for malaria.

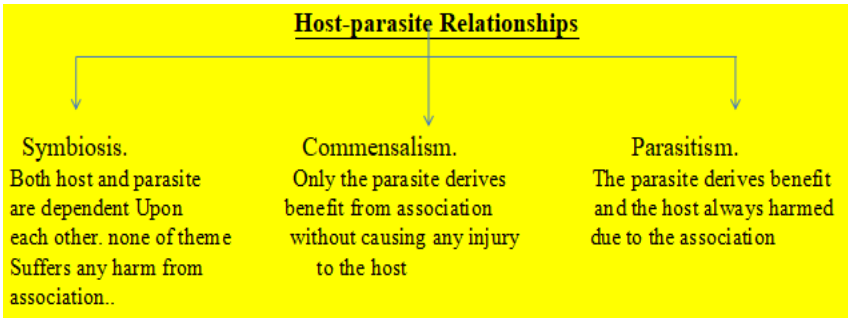
-Human acts as definitive host for T. solium ,T. saginata

2-Intermediate host: The host, in which the larval stage of the parasite lives or asexual multiplication takes place.
▲Some of parasites required 2 intermediate host to complete different larval stages. These are known as first and second intermediate hosts, respectively.
3-Paratenic host: A host, in which larval stage of the parasite remains viable without further development. Such host transmits the infection to another host. L2 of Ascaris in rodent.
4-Reservoir host: In an endemic area, a parasitic infection is continuously kept up by the presence of a host, which harbors the parasite and acts as an important source of infection to other susceptible hosts, e.g. dog is the reservoir host of hydatid disease.
5-Accidental host: The host, in which the parasite is not usually found, e.g. man is an accidental host for cystic echinococcosis.

-Carrier:-Parasite-harboring host that is not exhibiting any clinical symptoms but can infect others .
-Vector:-organism that transmit parasite from host to host , there are two type of vector:-
a-mechanical and b-biological

Host-parasite Relationships

Host-parasite relationships are of following types:-
♦ Symbiosis.
♦ Commensalism.
♦ Parasitism.
♦ Mutualism : association between two organisms both of them are beneficial



Life Cycle of Parasites

♠ Direct life cycle:- When a parasite requires only single host to complete its development, it is called as direct life cycle, e.g. Entamoeba histolytica requires only a human host to complete its life cycle.
♠ Indirect life cycle:-When a parasite requires 2 or more species of host to complete its development, the life cycle is called as indirect life cycle, e.g. malarial parasite requires both human host and mosquito to complete its life cycle.

Classification or taxonomy of parasite:-

Parasitology

For the study of parasite the international agreement classify the parasites according to morphological feature and biological characters to:-
Kingdom ,phylum , class, order , family , genus , species
Ex:-

Kingdom	Animalia
Phylum	Nemathelminthes
Class	Nematoda
Order	Strongylidea
Family	Trichostrongylidae
Genus	Haemonchus
Species	contortus

The genus name having a capital letter , so the name of organism is usually underlined or italic form

Sources of Infection

1-Contaminated soil and water:-
♦ Soil polluted with embryonated eggs (roundworm, whipworm) may be ingested or infected larvae in soil may penetrate exposed skin (hookworm).
♦ Infective forms of parasites present in water may be ingested (cyst of amoeba and Giardia).
♦ Water containing the intermediate host may be swallowed (copepod containing D. latum larva).
♦ Infected larvae in water may enter by penetrating exposed skin, (cercariae of schistosomes).

2-Food:-
♦ Ingestion of contaminated food or vegetables containing infective stage of parasite (amoebic cysts, Toxoplasma oocysts, Echinococcus eggs)
♦ Ingestion of raw or undercooked meat harbouring infective larvae (measly pork containing cysticercus cellulosae, the larval stage of Taenia solium).

3-Insect vectors: -Is an agent, usually an arthropod that transmits an infection from man to man or from other animals to man.
e.g. female Anopheles is the vector of malarial parasite.
Vectors can be divided into:
A-Biological vectors: The term biological vector refers to a vector, which not only assists in the transfer of parasites but the parasites undergo development or multiplication in their body as well. They are also called as true vectors.
Example of true vectors are:
Mosquito—Malaria, Filariasis Sandflies—Kalaazar Ticks—Babesiosis.
B-Mechanical vectors: The term mechanical vector refers to a vector, which assists in the transfer of parasitic form between hosts but is not essential in the life cycle of the parasite.
Example of Mechanical vectors is: Housefly—amoebiasis

4-Animals
Domestic:
♦ Cow, e.g. T. saginata, Sarcocystis
♦ Pig, e.g. T. solium, Trichinella spiralis
♦ Dog, e.g. Echinococcus granulosus
♦ Cat, e.g. Toxoplasma, Opisthrorochis.
Wild:
Wild game animals, e.g. trypanosomiasis
Wild felines, e.g. Paragonimus westermani
Fish, e.g. fish tapeworm
Molluscs, e.g. liver flukes
5-Other persons: which may be carriers of the parasite, vertical transmission of congenital infections.
6- Self (autoinfection):
€ Finger to mouth transmission, e.g. pinworm
€ Internal reinfection, e.g. Strongyloides, Hymenolepis nana ,Cryptosporidium parvum

Mode of Infection

1-Oral transmission:-by contaminated food, water, soiled fingers, Many intestinal parasites enter the body in this manner, such as E. histolytica .
2-Skin transmission:-by penetration the skin. Hookworm infection is acquired, when the cercarial larvae enter the skin of persons.
3-Vector transmission:-Many parasitic diseases are transmitted by insect bite, e.g., malaria is transmitted by bite of female Anopheles mosquito.
4-person to person contact:- e.g. by kissing in the case of gingival amoebae and by sexual intercourse in trichomoniasis.
5-Vertical transmission:-Mother to fetus transmission may take place in toxoplasmosis.
Iatrogenic transmission: It is seen in case of malaria and toxoplasmosis after organ transplantation.

Pathogenesis:-

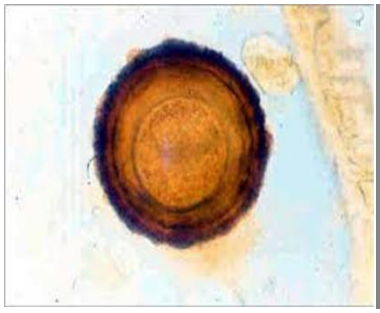
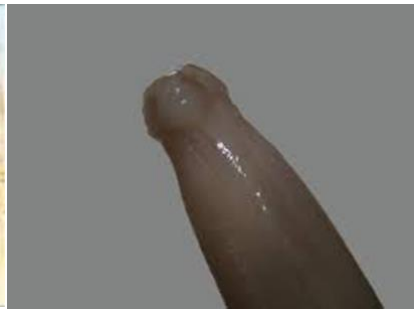
1-Parasitic infections may remain inapparent or give rise to clinical disease.
2-Clinical infection produced by parasite may take many forms—acute, subacute, chronic, latent, or recurrent.
3-Pathogenic mechanisms, which can occur in parasitic infections are:
♦ Lytic necrosis: Enzymes produced by some parasite can cause lytic necrosis. E. histolytica lyses intestinal cells and produces amoebic ulcers.
♦ Allergic manifestations: Clinical illness may be caused by host immune response to parasitic infection, e.g. eosinophilic pneumonia in Ascaris
infection and anaphylactic shock in rupture of hydatid cyst.
♦ Physical obstruction: Masses of roundworm cause intestinal obstruction.
♦Inflammatory reaction: urinary bladder granuloma in Schistosoma haematobium infection.

♦ Neoplasia: A few parasitic infection have been shown to lead to malignancy. The liver fluke, Clonorchis may induce bile duct carcinoma, and S. haematobium may cause urinary bladder cancer.

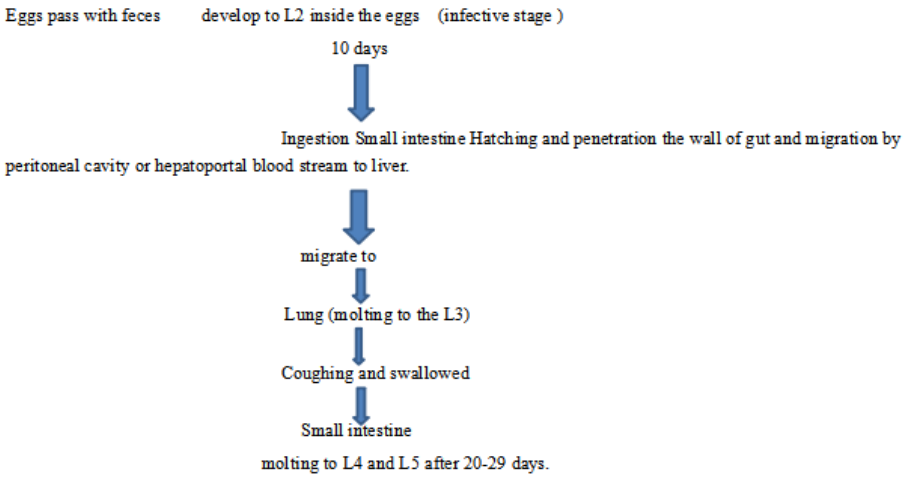
محاضرة رقم 2

Parascaris equorum

Genus :-Parascaris equorum
Hosts :-Horses and donkeys
Site :-Small intestine
Morphology:-
▲This very large whitish nematode, up to 40 cm in length, The adult parasites have a simple mouth opening surrounded by three large lips and in the male the tail has small caudal alae.
▲The egg is almost spherical, brownish and thick-shelled with an outer pitted coat.

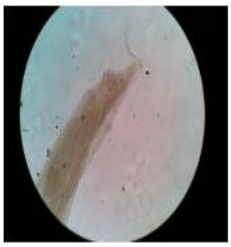
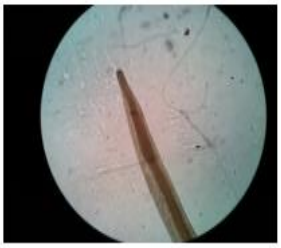
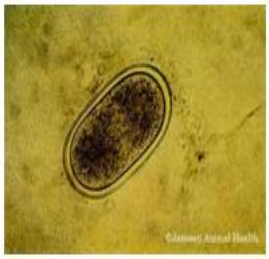



Life cycle and pathogenesis:-



Clinical signs:-
1- coughing accompanied in some cases by a greyish nasal discharge.

2-unthriftiness in young animals with poor growth rates, dull coats. nervous disturbances and colic.
Treatment:-
1-Piperazine compounds (100 mg/kg).
2-fenbendazole (10 mg/kg
3- pyrantel (6.6 mg/kg).
4-ivermectin (0.2 mg/kg).
5-moxidectin (0.4 mg/ kg).
6- Pyrantel highly effective against the intestinal stages of P.equorum.
Genus :-Ascaridia galli
Hosts:-Domestic and wild birds
Site:-Small intestine
Morphology
▲There are three large lips and the esophagus has no posterior bulb.
▲ The tail of male has small alae, there is a circular precloacal sucker, the spicule are sub equal.

Life cycle : direct life cycle
The egg are passed in the feces ,reaching the infective stages(L2 inside the egg) in about ten days. Infection takes place by ingestion of the eggs with food or water. Eggs hatch in the intestine and the larvae penetration the mucosa of intestine from the eighth to seventeenth days and then reenter the lumen and reach maturity in six to eight weeks.
-Pathogenesis:-
they cause an enteritis, but in very heavy infections may be hemorrhage and enteritis.
-Clinical signs:-
1- anemia, intermittent diarrhea, and anorexia.
2- unthrifty and emaciated.This can lead to a decrease in egg production.
-Diagnosis:-
1-In infections with adult worms, the eggs will be found in feces.
2-post-mortem when the large white worms will be found.

محاضرة رقم 3

Toxocara

Genus:-Toxocara canis

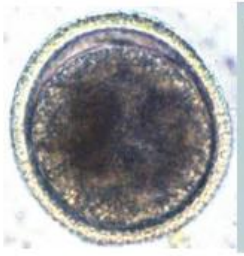
▲Toxocara canis is a large white worm up to10-15 cm in length, have glandular esophageal bulb located at the junction of the esophagus and the intestine.

▲ Large cervical alae are present. The egg is dark brown and subglobular, with a thick, pitted shell.

▲ In the male presence of a small finger-like process on the tail .

Hosts:-Dog,

Site:-Small intestine


Mode of infection

▲ Ingestion of L2 inside eggs with food and water (basic form).

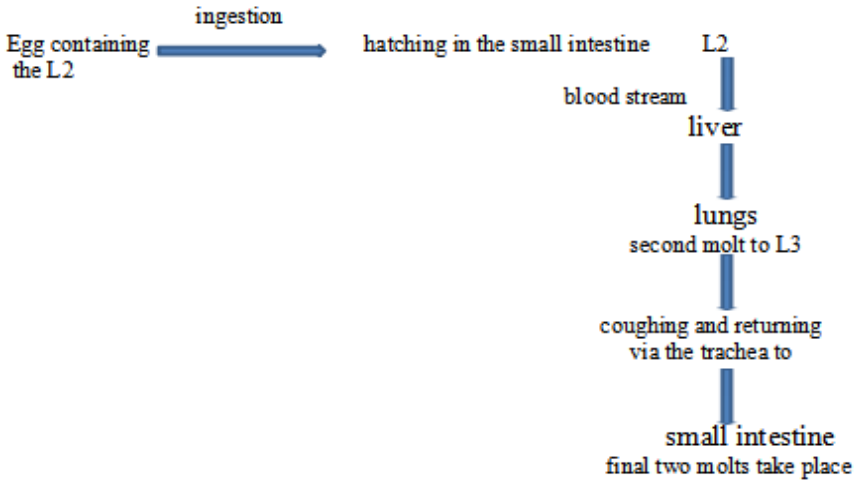
▲ paratinic hosts rout.

▲ Prenatal infection.

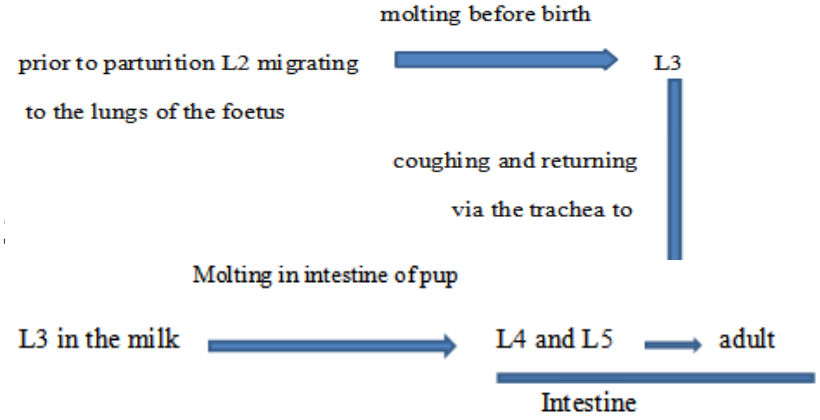
▲ Transmammary transmission.

Life cycle : direct life cycle

1-The basic form

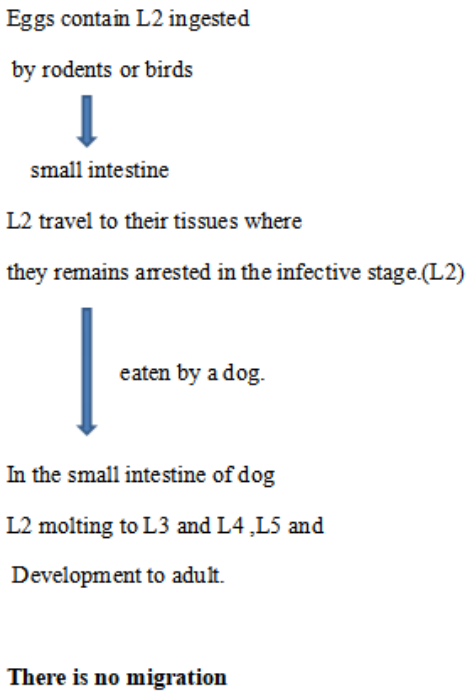


2-prenatal infection occurs in pregnant bitch



There is no migration

4- Paratenic hosts rout (such as rodents or birds)



There is no migration

<u>Pathogenesis</u>

▲In heavy infections the pulmonary phase of larval migration is associated with pneumonia.

▲ The adult worms cause a mucoid enteritis.

▲ There may be partial or complete occlusion of the gut.

▲ Obstruction of the bile or pancreatic duct.

-Clinical signs:-

1-pot-belly and occasional diarrhea.

2-The signs in heavy infections during larval migration result from pulmonary damage and include coughing, increased respiratory rate, and a frothy nasal discharge.

-Diagnosis:-

1-Clinical sings

2-The eggs in feces, subglobular and brown with thick pitted shells.

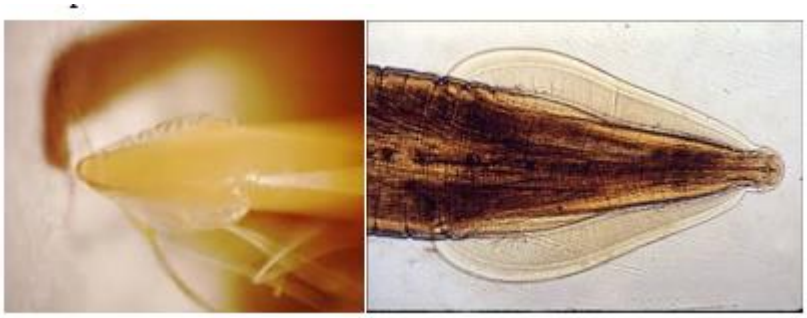
Visceral Larva Migrans (VLM)

Is a syndrome caused by invasion of internal organs of the paratenic host by second stage nematode larvae ,when nematode larvae entry into an paratenic host they do not complete the normal migration but instead have arrested development and begin an extended random wandering through various organs.
VLM caused by Toxocara canis larvae ,although the larval stages of T. cati.
The global condition occurs most commonly in children, often under 5 years of age, who have had close contact with household pets, or who have frequented areas such as public parks where there is contamination of the ground by infective dog feces.
In many cases, larval invasion the liver and may give rise to hepatomegaly and eosinophilia, but on some occasions a larva escapes into the general circulation and arrives in another organs such as lungs, brain and eye ,here a granuloma forms around the larva on the retina.

Toxocara cati (arrow worm)

Host:-Cat
Site:-Small intestine
Morphology:-
The cervical alae are very broad and are striated (an arrow-head form) with the posterior margins almost at a right angle to the body. The male like that of T. canis, has a small finger-like process at the tip of the tail The egg, subglobular, with a thick, pitted shell and almost colorless, is characteristic in cat feces.

Life cycle:-
Similar to T. canis but there is no prenatal infection.
Clinical signs and diagnosis:-
Similar to T. canis



محاضرة رقم 4

Ascaris suum

Family :-Ascarididae

Large worms with three well developed lips, there is no buccl capsule or pharynx ,the esophagus is usually club shaped, muscular and without a posterior bulb. which inhabit the small intestine.

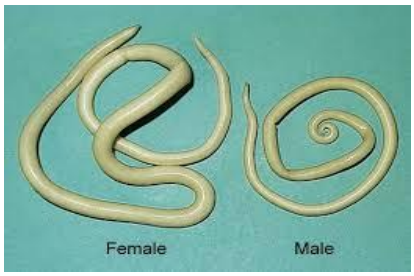
Genus :-Ascaris suum

▲ The adult worms are about 30 cm long and white to cream colored, with three large lips.

▲ The egg is ovoid and yellowish, with a thick shell, the outer layer of which is irregularly pitted.

Host:-Pig.

Site-:Small intestine.



Life cycle : direct life cycle

Eggs pass with feces develop to L2 inside the eggs (infective stage)

10 days

Ingestion

Small intestine Hatching and penetration the wall of gut and migration by peritoneal cavity or hepatoportal blood stream to liver.

migrate to

Lung (molting to the L3)

Coughing and swallowed

Small intestine

molting to L4 and L5 after 20-29 days.

Pathogenesis

1-Due to larval migration there is destruction of tissue and hemorrhage trunk in the liver and lung .

2-Oedema and infiltration with eosinophil in the alveoli of the lung .

3-The adult worms causes intestinal obstruction and damage to the mucosa.

4-The worm may pass into the bile duct and causing biliary stasis or blockage of the bile duct.

5-In the liver the migrating larvae can cause ‘milk spot’ or ‘white spot’ of up to 1.0 cm in diameter on the surface of the liver.

Clinical signs

Pneumonia, cough, diarrhea, vomiting

محاضرة رقم 5

diagnosis

1-Faecal examination : determination the yellow-brown ovoid eggs, with thick pitted shells.

2-Post mortem : milk spot lesions on the liver.

ASCARIS LUMBRICOIDES

large intestinal round worm in human

Morphology:-

1-female length 20 – 35 cm. ; male smaller 12 - 30 cm.

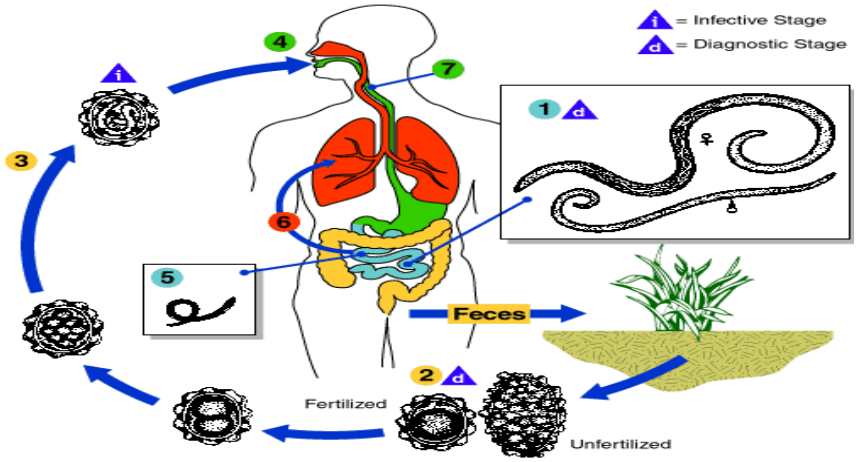
2-Elongated cylindrical tapered anteriorly & posteriorly.

3-It’s head provided by 3 fleshy lips.

4-Female egg production daily about 200000 eggs .

5-Egg spherical shape consist of 3 layers (lipid layer ,chitinous layer , proteinous layer).

Life cycle



Symptoms in human

-it is depend on worm burden

1-A number of worms 10 – 20, may go unnoticed

2-Vague abdominal pain is the commonest complain.
3-Sever case's sings are listlessness , loss weight , anorexia , distended abdomen , intermittent loose stool , vomiting occasionally , fever , & worms may locate , block pancreatic ducts or billiary duct & forming calculi or hepatic abscess .
-Cough , wheezing , dyspnea , sub sterna pain ; these sings are observed during pulmonary stage & also the Infiltration of Esinophilia (Loffler's syndrome)

Treatment in human:-
1-Mebendazole 100 mg./ kg. / b. i. d. / 3 days .
2-Thibendazole 25 mg. / kg. /b. i. d. / 5 days .
3-Prednisone for allergic symptoms ; 20 – 40 mg. daily reduced after 3 – 5 days .

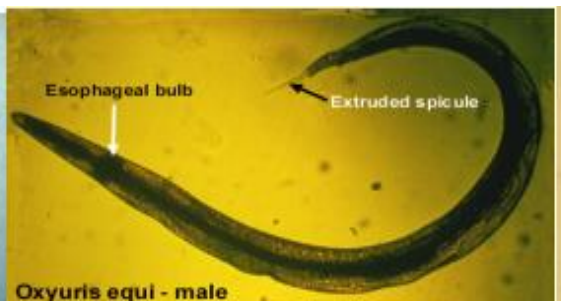
Ascaridae Control & prevention:-
1-To prevent reinfection of parasitic roundworms, it is recommended that anything that the animal has been in contact with should be cleaned thoroughly or replaced, including bedding and kennels. It is also strongly recommended that outside areas where defecation may occur be cleaned, as well as all feces removed daily from outdoor pet runs, crates, and the yard.
2-Home & community sanitation .
3-Proper food cooking .
4-Eggs in feces could be destroyed by composting with increasing soil temp. without affecting the fertilizing action of the matter .

Oxyuris equi

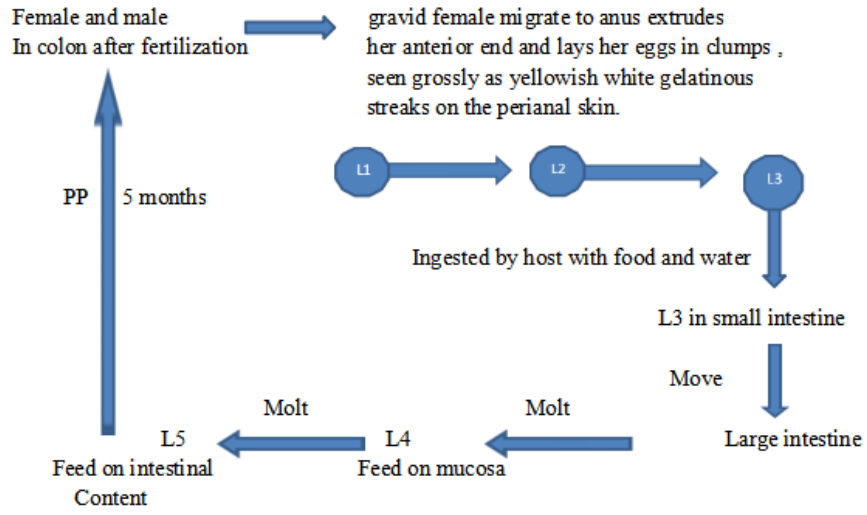
محاضرة رقم 6

Genus:-Oxyuris equi (Pin worm)
Hosts:-Horses and donkeys
Site:-Caecum and colon

Morphology:-
● The mature females are large white worms with pointed tails, may reach l0. cm in length .
●The mature males are generally less than 1.0 cm long.
● Microscopically there is a double esophageal bulb and the males have caudal alae and a single spicule. In the female the vulva is situated anteriorly.
● O. equi eggs are ovoid, yellow and slightly flattened on one side with a plug at one end.



Life cycle:-direct life cycle



Pathogeneses:-

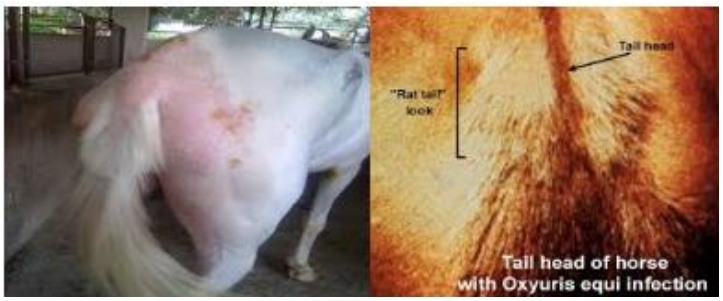
▲ Most of the pathogenic effects of O. equi in the intestine are due to the feeding habits of the L4 which result in small erosions of the mucosa and, in heavy infections, these may be wide spread and accompanied by an inflammatory response.

▲ A more important effect is the perineal irritation caused by the adult females during egg laying.

Clinical signs:-

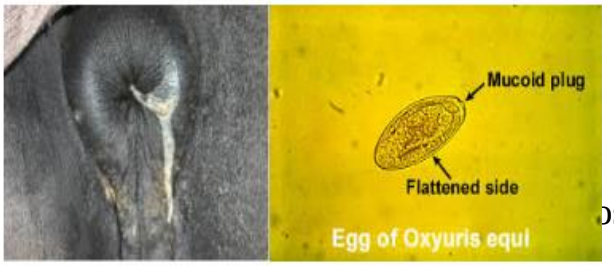
1-Intense pruritic around the anus causes the animal to rub, resulting in broken hairs around tail. like (rat tail).

2-Bare patches and inflammation of the skin over the rump.



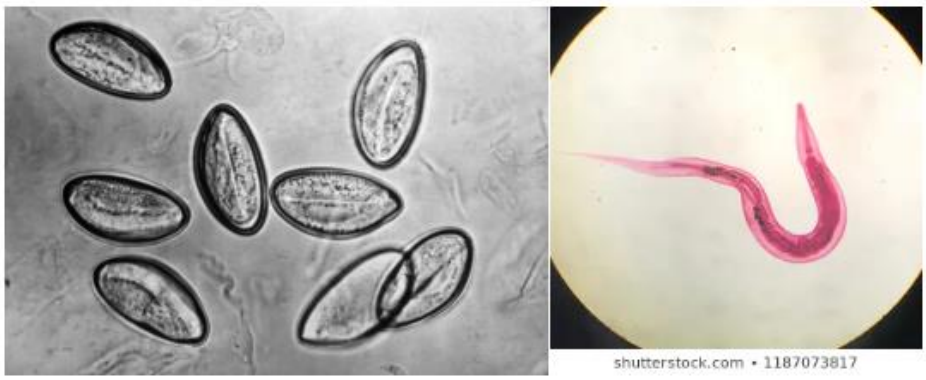
Parasitology

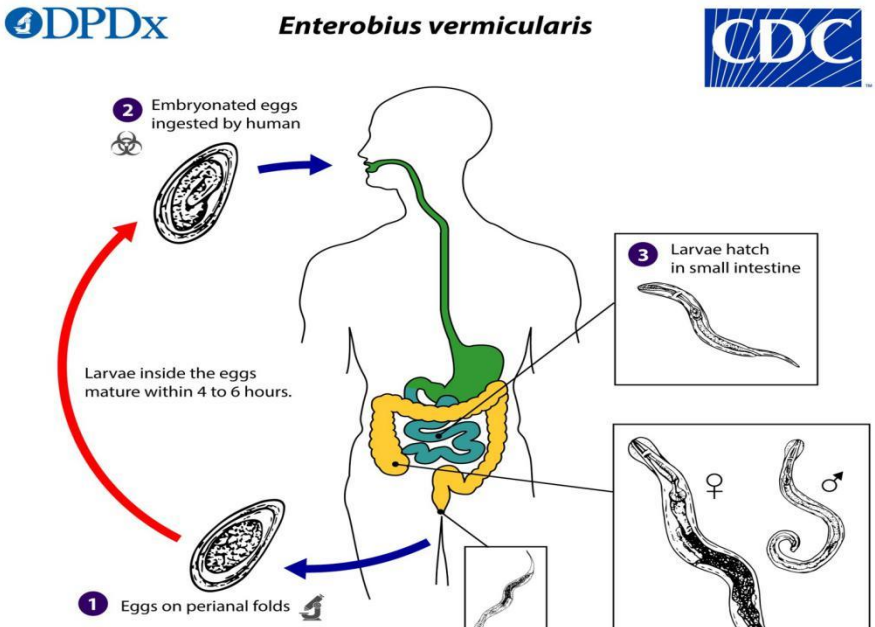
Diagnosis:-
1-This is based on signs of anal pruritis .
2- finding of greyish-yellow egg masses on the perennial skin.
3-O. equi eggs are rarely found on fecal examination of samples taken from the rectum, but may be observed in material from the perineum or in fecal material taken from the ground.
4-Scotch tape technique.



<u>E</u>
-H osmopolitan distributed but more in cool &
te
-The disease called Enterobiasis or Oxyuriasis.
-Eggs can also be transferred to cloth, toys, and the bathtub. Once ingested orally, the larva 1 hatch and migrate back to the intestine, growing to maturity in 30-45 days.

-Children common symptoms { pruritus ani , restlessness sleep , insomnia , tiredness at day time ,GIT disturbance ,pain ,nausea , vomiting etc…}.
-Eggs has characteristic shape of relatively
-Flat on one side {D shape} with colorless
-double shell , ,50 – 60 µm. ,the outer layer
-albominous which can be stick to clothes & objects & on other eggs .





محاضرة رقم 7

Ancylostoma

Hookworms of dogs and cats
family:-Ancylostomidae

Members of this family commonly called hook worms because of the characteristic hook posture of their anterior ends, are responsible for wide spread morbidity and mortality in animals primarily due to their blood-sucking activities in the intestine.

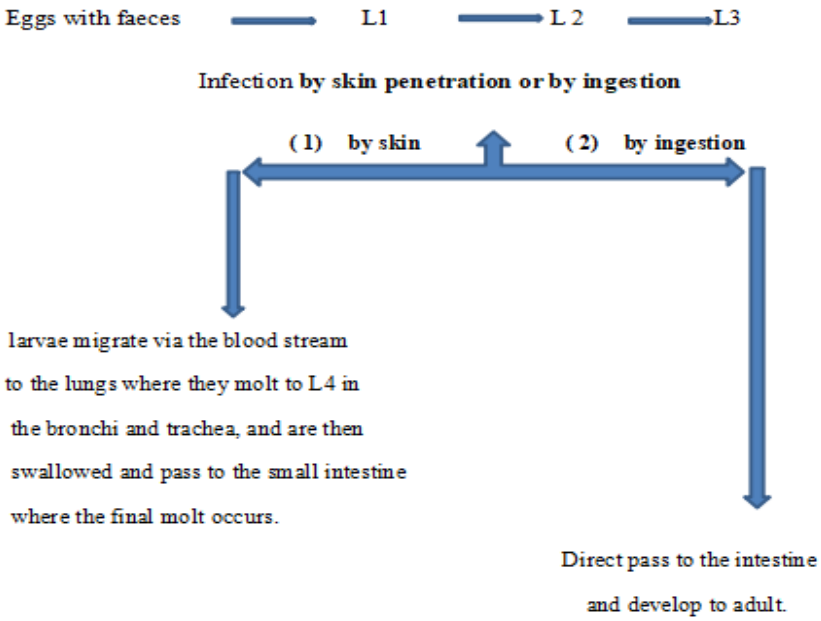
Genus: Ancylostoma
Hosts : Dog, cat and fox
Site : Small intestine
Species:
Ancylostoma caninum ⟶ dog and fox
A. tubaeforme ⟶ cat
A. braziliense ⟶ dog and cat.
A.duodenale ⟶ man

Morphology:-
♣ The buccal capsule is large with marginal teeth, there being three pairs in A. caninum and A. tubaeforme and two pairs in A. braziliense and A. duodenale.
♣ The worms are stout and grey or reddish in color depending on the presence of blood in the alimentary canal. The male bursa is well developed.

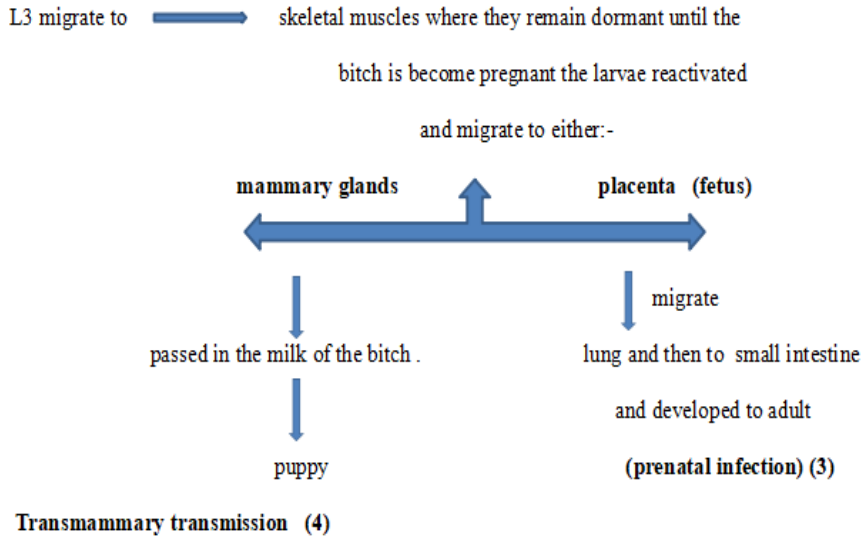
Mode of infection
▲ skin penetration.

▲ Ingestion of L3 with food and water.
▲ Prenatal infection.
▲ Transmammary transmission.

Life cycle : direct life cycle



in susceptible bitches



Parasitology

Pathogenesis

1-Anaemia due to blood sucking. most commonly seen in dogs under one year old and young pups.

2- Skin reactions such as moist eczema and ulceration at the sites of cutaneous infection occur.

Clinical signs:-

1-In acute infections, there is anemia and respiratory signs may be due to larval damage in the lungs .

2-In suckled pups the anemia is often severe and is accompanied by diarrhea which may contain blood and mucus.

3- In more chronic infections, the animal is usually underweight, the coat is poor, and there is loss of appetite .

4-skin lesions result from migration of larvae.

Diagnosis

This depends on the:-

1- Clinical signs and history supplemented by hematological.

2- Fecal examination. (Eggs are strongylate' with dissimilar poles, barrel-shaped , and contain two to eight cells.)

Cutaneous Larva Migrans(CLM)

Also known Creeping eruption,(human cutaneous larva migrans) ground itch , is an acute skin syndrome caused by migrating larva of parasitic nematodes.

CLM manifested an erythematous (red) serpiginous (twisting) pruritic skin eruption caused by accidental skin penetration and migration of nematode larvae. A variety of nematode larvae cause CLM including hook worms and strongyloides.

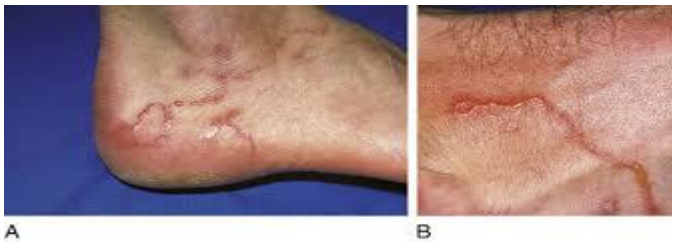
A. braziliense larvae A. caninum, Bunostomum phlebotomum, Strongyloides stercoralis, . and the larvae of those species that normally mature in man (A. duodenale)

Treatment :-

♣ albendazole

♣ Topical freezing agents, such as liquid nitrogen

♣ Anti-itch cream (i.e. Cortizone or Calamine lotion)



A B

Parasitology

محاضرة رقم 8

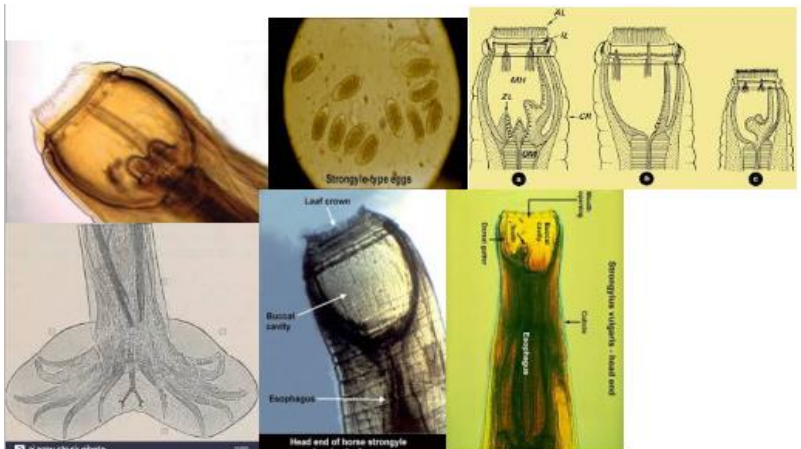
Strongylidae

Super family:-Strongyloidea
Family:-Strongylidae

<u>The main characters of this superfamily:-</u>
●Large buccal capsule contain teeth or cutting plate and there are prominent leaf crown surrounding the mouth opening
●All genera of this super family are present in intestine and can be divided into two groups : a- strongyles worms and b- hook worms.
● The strongyles are parasitic in the large intestine and the important genera are : Strongylus, Trichonema , Chabertia and Oesophagostomum.
● Hook worm are parasite of small intestine and the important genera are Ancylostoma and Bunostomum

Genus: Strongylus
Called large red worm of horse .
Lives in the large intestine of horse and donkey.
Site of infection:- caecum and colon
Species :-Strongylus vulgaris, Strongylus equinus ,Strongylus edentatus

<u>Morphology:-</u>
Gross :-robust, dark red worm with well-developed buccal capsule of the adult contain on the dorsal wall median thickening called dorsal gutter and have prominent bursa of male.
Microscopic : species differentiation is based on the teeth in the base of buccal capsule
S . vulgaris have two ear shaped rounded teeth.
S . edentatus have no teeth.
S . equinus have three teeth one dorsal and larger than other and bifid.



Parasitology

Life cycle : direct life cycle

Eggs pass with feces and develop to L1 → L2→ L3 (infective stage).Infection to the final host occur by ingestion of L3 with food and water.

S . vulgaris

L3 penetrate wall of intestine → inter Cranial Mesenteric artery
molt 3 and molt 4 occur
L4 and L5

↓

L5 return to wall of large intestine
And nodule formation

↓

Rupture of nodules and releaseyoung adult parasite in the lumen of caecum and colon .

S . edentatus

L3 penetrate wall of intestine → via portal system reach to the liver
Molt to L 4 , migration in parenchyma,
After 6-8 weeks reach to

↓

Right Flank and hepato-renal ligament
Molt to L5

↓

L5 return to the wall of large intestine and nodule formation
And then release young adult in the lumen of intestine

S . equinus

L3 penetrate wall of intestine and nodule formation in the wall of
Intestine and molt to L4. → L4 migrate via peritoneal cavity to the
Liver and molt to L5

↓

after 6 month L4 and L 5 found in pancreas

Pathogenesis

▲ Adult worm of three species attach to the mucosa of the large intestine and sucking blood lead to anemia.
▲ Hemorrhagic ulcers in the site of attachment.
▲ The larval stage may be responsible for sever pathogenic effects specially the L4 , L5 of Strongylus vulgaris which responsible for lesion in the arterial system mainly in cranial mesenteric artery which cause thrombus or verminous aneurysms because thickening of the arterial wall , infraction of the iliac –artery may lead to temporary lameness and colic occur due to pressure on the cranial mesenteric plexuses by thrombus.
▲S. equinus larvae associated with hemorrhagic tract in the liver and pancreas may be fatal
▲ Marked peritonitis , acute toxemia , jaundice and fever caused by larvae of S. edentatus

Diagnosis

▲ clinical sings such as lameness , colic and anemia .
▲ fecal examination for eggs detection and then identification of the larvae by fecal culture.
▲ X- ray –enlargement in the endothelium of mesenteric artery .

Treatment :-Pheniothiazine 20 – 40 g/ per animal
Thiabendazol 50 mg / Kg B.W

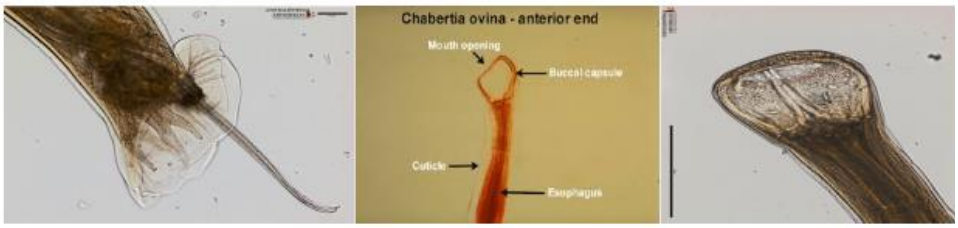
Chabertia محاضرة رقم 9

Genus:-Chabertia
Hosts:-Sheep, goats and occasionally cattle
Site:-Colon
Species:-Chabertia ovina
Disease is mainly seen in sheep in colder areas during the winter months.

Morphology:-

▲The adults are 1.5-2.0 cm in length and are the largest nematodes found in the colon of ruminants.

▲ They are white with enlarged anterior end due to the presence of the huge buccal capsule, which is bell shaped, has a double row of small papillae around the rim. There are no teeth



Life cycle :-direct life cycle

Clinical signs

In severe infections:-
1- Diarrhea, which may contain blood and in which worms may be found,
2- The most common clinical sign. The sheep become anemic and hypoalbuminemic and can suffer severe weight loss.

Pathogenesis

▲ The major pathogenic effect is caused by the L5 and by mature adults which attach to the mucosa of the colon via their buccal capsules and then feed by ingesting the mucosa resulting in local hemorrhage.
▲The wall of the colon becomes edematous, congested and thickened with small hemorrhages at the sites of worm attachment.

Diagnosis:-
1-clinical signs.
2-fecal analysis and fecal culture.

Haemonchus

محاضرة رقم 10

Family:-Trichostrongylidae
● Small worm in which the buccal capsule is absent or very small.
● Devoid leaf crowns and usually bears no teeth.
● The male bursa is well developed with large lateral lobes and a small dorsal lobe .
● Adult parasitic in the alimentary canal of sheep ,cattle and equines.
● Genera of importance include:-Trichostrongylus , Ostertagia , Neatodirus and Haemonchus.

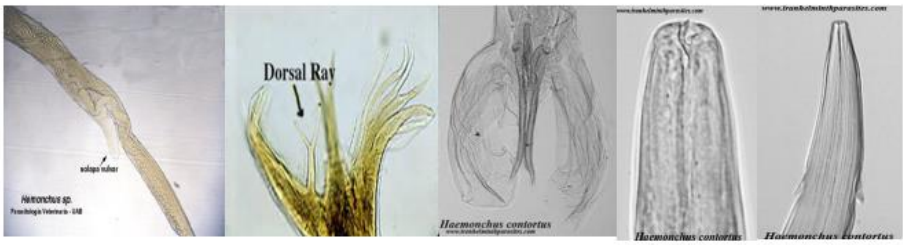
Genus:-Haemonchus
Common name : barber pole worm or red abomasum worm.

Parasitology

Most common in tropical or subtropical areas or in those areas with summer rainfall..

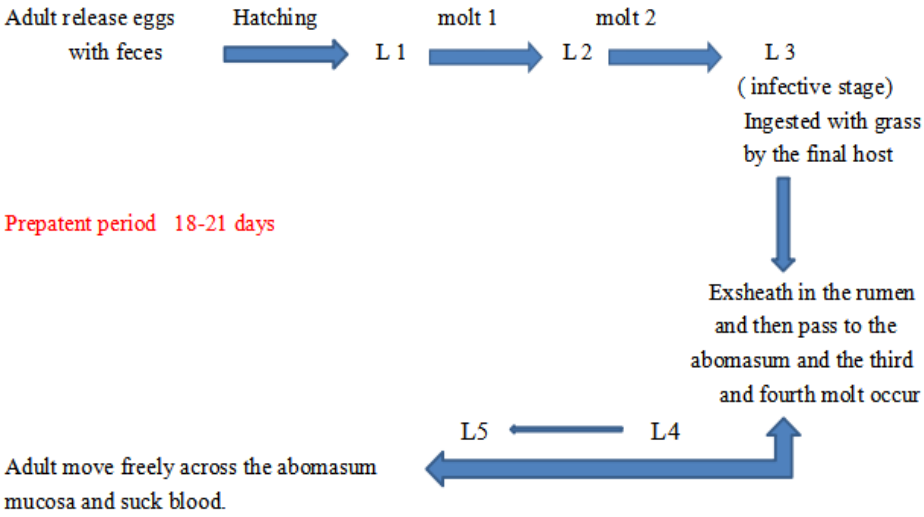
Species:- H. contortus sheep and goat
H. similis cattle
H. longistipes camels
Host:-ruminant
Site of infection:-abomasum

Morphology:-
▲ have reddish appearance due to its blood feeding habit.
▲ small buccle capsule with slender tooth or lancet.
▲ cervical papillae are prominent.
▲ The male have well developed copulatory bursa consist of two large lateral lobes and dorsal lob have Y shape ray. The spicules are wedge shape provided with small barb near its extremity.
▲ The female have Lingui form vulva flap which usually large and prominent, and the white ovaries winding around blood filled intestine giving the appearance barbar pole.



Female (vulva flap) Male bursa Anterior part

Life cycle:-direct life cycle



Pathogenesis

The pathogenesis of haemonchosis is essentially that of haemorrhagic anaemia due to the blood sucking habits of the worms . Each worm can remove up to 0.05 ml of blood per day by ingestion and seepage from the lesion .
Haemonchosis can be divided according to severity into three types:-
1- Hyperacute.
2- Acute.
3- Chronic.

Hyperacute haemonchosis

▲ Occur after ingested 20000 - 35000 infective larvae (L3) , the resulting L4 causes petechiae while the L5 and adult causes haemorrhage and erosion at their attachment sites .
▲ Sheep can loos 1000- 1750 ml blood per day.
▲ Death in apparently healthy sheep occurs suddenly within 1 week as a result of sever haemorrhagic gastritis.

Acute haemonchosis

● Occur after ingested 2000 – 20000 of L3 result in daily blood loss 100-1000 ml.
● Anemia becomes apparent from about 2 weeks after infection and accompanied by fall in PCV.
● Compensatory erythropoiesis (visible as hyperplasia of bone marrow from white to red .
● Expense of the iron reserves occur together with the continual loss of protein (albumin) into the gastrointestinal tract .

Chronic haemonchosis

♠ About 100- 2000 adult worms can cause blood loss of about 5-100 ml per day .
♠ Usually developed during winter due to deficient in nutrient (protein and iron) .
♠ The continual blood loss depletes the iron reserves completely which result a marked anemia developed shortly before death .

Clinical sings

Hyperacute haemonchosis:-
▲ Animals die suddenly with few sings except anemia and dark –brown to black feces .
Acute haemonchosis :-
▲ Anemia , bottle jaw , weight loss and brown to black feces .
▲ Ewes stop producing milk and sucking lambs die of starvation.
▲ Lethargy and a break in the wool before death.
Chronic haemonchosis: -
▲ Animals lose weight progressively over several months but show neither sever anemia nor bottle jaw.
▲ Finally the animal becomes weaker and anorexia , anemia present shortly before death.

Parasitology

Epidemiology of Haemonchus contortus

1-High biotic potential produce about 5000-15000 eggs/day.
2-Development to infective stage is rapid (5-7) days.
3-Periparturient Rise (PPR)
-Synonyms :- post-parturient rise ,Spring rise
-Refer to an increase in the number of Nematode eggs in the feces of animals around parturition .the phenomenon is most marked in ewes ,sous and goat. Result from temporary relaxation of immunity due to increase circulation level of prolactin hormone .

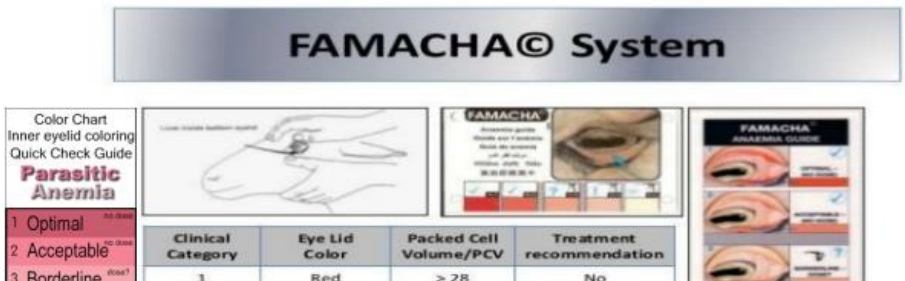
The source of (PPR):-
-Maturation of arrested larvae due to decrease host immunity.
-Increase establishment of infection acquired from the pasture.
-Increase fecundity of existing adult worm.

4-The Self-Cure phenomenon
-The faecal worm egg counts of sheep infected with H. contortus drop sharply to near zero levels due to the expulsion of the major part of the adult worm burden. In areas of endemic haemonchosis it has often been observed that after the advent of a period of heavy rain.
-The expulsion of the adult worm population is considered to be the consequence of an immediate-type hypersensitivity reaction to antigens derived from the developing larvae. Although this phenomenon has an immunological mechanism.
-The phenomenon may also be caused, in some non-specific way, by the ingestion of fresh growing grass.

5-Arrested larval development

Diagnosis
♣ Clinical sings:- (anemia --- bottle jaw -----)
♣ Fecal examination for----- detection the eggs.
♣ Fecal culture ----------detection the larvae
♣ Blood analysis reveal to ----- anemia
♣ Post mortem :-
♠ carcass may be pale and oedematus .
♠ Coagulation blood may be present in the abomasal content .
♠ Mass of worms seen in abomasum.
♠ Petechiae and small erosion in the abomasal mucosa.

Diagnosis



Treatment :-

Ivermectin	=	Ivomec®	+ + +
Albendazole	=	Valbazen®	+ + +
Levamisole	=	Tramisol®, Levasol®	+ + +
Fenbendazole	=	Panacur®	+ +
Mebendazole	=	Telmin®	+ +
Oxfendazole	=	Benzelmin®	+ +

محاضرة رقم 11

Ostertagia

Genus :-Ostertagia
Common name :-brown stomach worms
Species:- O. ostertagi cattle
O. cercumcincta sheep
O. trifurcata sheep
Host :-Ruminant
Site of infection:- abomasum

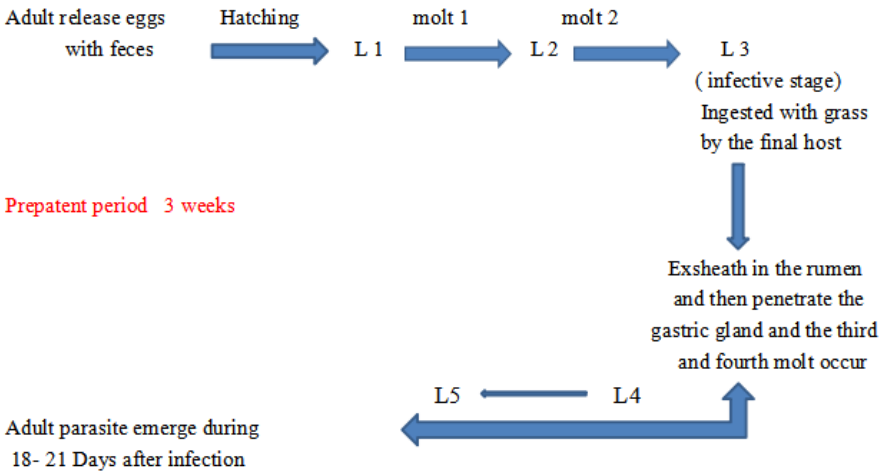
Morphology:-

▲ Slender worm have cervical papillae in the anterior extremity.



▲ The male bursa has lateral and dorsal lobes , spicules are pigmented brown , short and end posteriorly in two or three processes .
▲ The vulva of the female covered by small flap.

Life cycle :-direct life cycle



Types of ostertagia:-
There are two types of ostertagia:-
Type I ostertagiasis. ⟶ occur in summer
Type Π ostertagiasis. ⟶ occur in spring

Type I ostertagiasis:-
▲ Occur in calves which ingested the infective larvae during spring season from the over winter larvae which inhibited in pasture .
▲ Occur in spring born calves of dairy herds that turned out in midsummer onto pastures grazed and contaminated by autumn born calves .

Type Π ostertagiasis:-
Occur in calves which ingested the infective larvae during autumn and these larvae inhibited as L4 in the gastric gland of abomasum and remain in the gland until the spring season.

Clinical sings:-
- Profuse watery diarrhea
- Anorexia
- Loss body weight.
- Emaciation.
- Bottle jaw .

Pathogenesis:-
Present the worms in the gastric gland lead to : -
▲Change in the activity of the cell which produce the Hcl causing increase in the PH from 2 to 7 and this lead to :-
1-No change of pepsinogen to pepsin.
2-Not analysis to the proteins which occur .
3-Increase the number of bacteria in abomasum lead to diarrhea.

▲ Increase permeability of the abomasum wall and this lead to increase in pepsinogen in plasma and reduce in the albumin in the blood

Epidemiology
1- larvae of ostertagia more resistance to the cold weather .
2- 80% of the ingested larvae in the autumn and winter inhibit in the abomasum as L4.
3- fecal pad of cattle consider as a reservoir for the larvae.

diagnosis
1- Clinical sings .
2- Fecal examination :- direct method --- flotation test , for detection the eggs .
3- Fecal culture :- for detection the larvae.
4- Post mortem :- ♠ -present the worm in the abomasum .
♠ -gastric gland arise and have central opening.
♠ -morocco leather appearance of mucosa .

5- Serological tests such as ELISA.
6- Histologically:-gastric gland have larvae

Arrested larval development

Synonyms:-inhibited larval development , hypobiosis.

▲ The phenomenon may be defined as the temporary cessation in development of Nematode larvae at a précis point in parasitic development.it is usually recognized by the present of large number of larvae at the same stage of development in the animal.

▲ The nature stimulus for hypobiosis of the larvae may be unfavorable condition that effect on free living infective stage prior to ingestion by the host.

▲ Hypobiosis may be occur due to:-

1- Heritable trait.
2- Both acquired and age immunity in the host.

▲ Epidemiological important of hypobiosis:-

1- Ensure the survival of the nematodes during period of adversity .
2- Maturation of arrested larvae increase the contamination of the environment and can result in clinical disease .

محاضرة رقم 12

Trichostrongylus

Genus:-Trichostrongylus (hairworm)
Hosts:-Ruminants, horses, pigs, rabbits and fowl
Site of infection:-Small intestine, abomasum
Species:-

Trichostrongylus axei	abomasum of ruminants and stomach of horses and pigs.
T. colubriformis	ruminants.
T. tenuis	small intestine and caeca of birds.
T. orientalis	small intestine of human.

Morphology:-

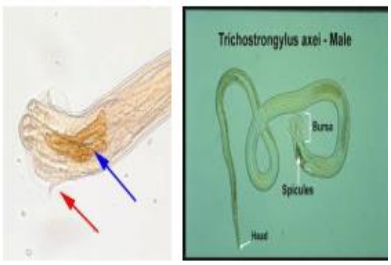
▲ The adults are small and hair-like ,slender, pale reddish-brown worms.

▲ The worms have no obvious buccal capsule, A most useful generic character is excretory pore which situated in ventral notch near the anterior extremity.

▲ The spicules are thick, stout, ridged, brown in colour and unbranched.

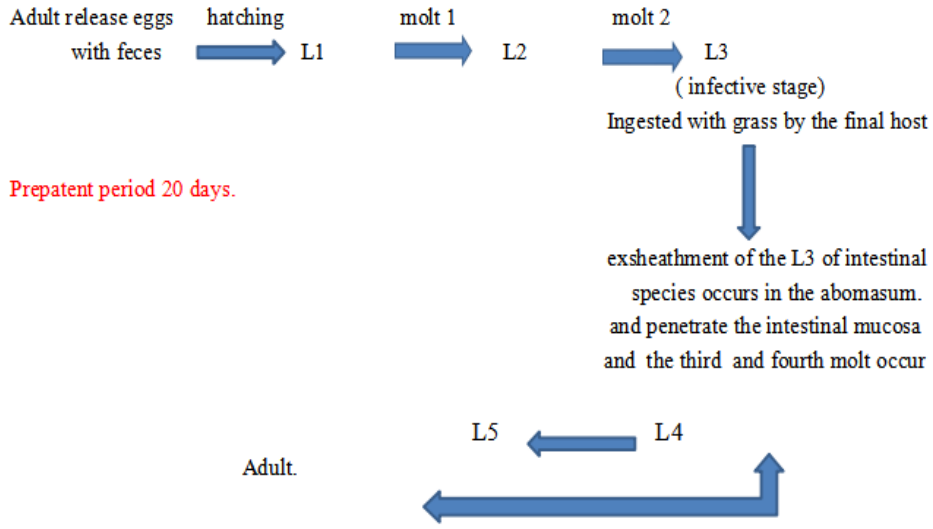
posterior part of Female

Anterior part



Male bursa

Life cycle : direct life cycle



Clinical sings:-

▲ Principle of clinical sings loss weight and diarrhea it is called black scour disease due to dark green scour.
▲ Fecal mass attach to hind quarter result from diarrhea and can be source of myasis .
▲ Weak in the legs and the animals are unable to stand before death and poor growth rates.
▲ Emaciation and the skin becomes dry.
▲ In man abdominal pain, diarrhea, anorexia, headache, fatigue, anemia and eosinophilia.

Pathogenesis

▲ hyperaemia of gastric mucosa progresses to catarrhal inflammation and erosion or ulceration of epithelium .
▲ parasite partially embedded in the mucosa causes lesion resemble ring worm lesion.
▲ In the intestine the parasite present in tunnel result in thickened , edema and infiltrated with inflammatory cells
▲ Increase permeability of capillaries plus open junction between the epithelial cells lead to loss of plasma protein into the intestine and the hypoalbunaemia which developed .

Epidemiology

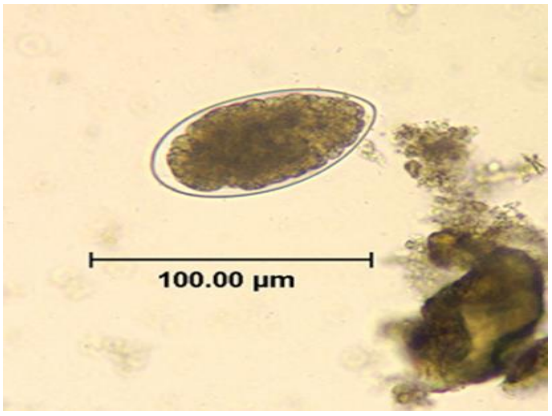
♠ Development to infective stage is rapid (4-6) days , the most rapid development occur in summer.
♠ The infective larvae more susceptible to cold and show limited ability to overwinter on pasture .
♠ L2 very susceptible to desiccation .

Parasitology

♠ Infective larvae may undergo inhibition of development within the host.

Diagnosis

1- Clinical sings
2- Fecal examination
3- Fecal culture
4- post mortem



محاضرة رقم 13

Dictyocaulus

family :- Dictyocaulidae
Genus :-Dictyocaulus
Common name : Bovine lung worm, , verminous pneumonia.
This genus living in the bronchi of cattle, sheep, horses and donkeys is the major cause of parasitic bronchitis in these hosts.

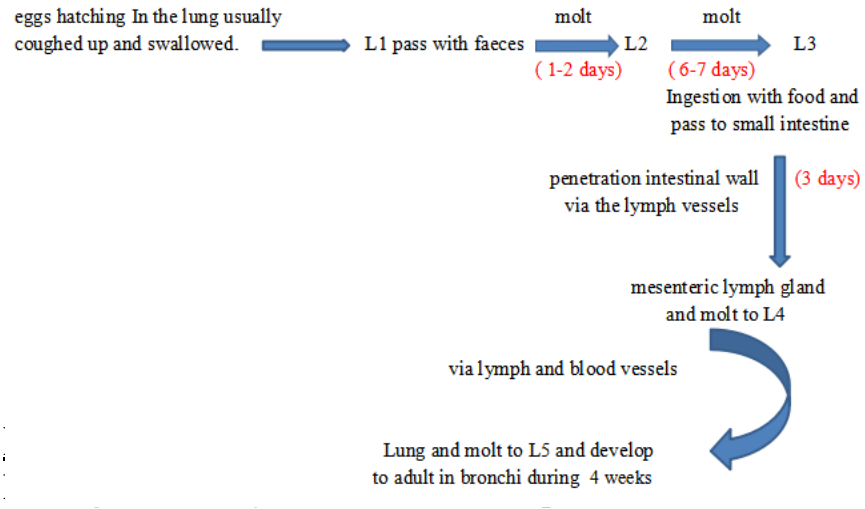
Species:-Dictyocaulus viviparus	cattle and deer
D. filaria	sheep and goats
D. arnfieldi	donkeys and horses

Host :-Ruminants, horses and donkeys.
Site of infection :-Trachea and bronchi, particularly of the diaphragmatic lobes.

Morphology:-

▲The adult worms have a milk-white colour and the intestine shows as a dark line.
▲ There are 4 very small lips. .
▲ The spicules are stout ,dark-brown boot shaped.
▲ the vulva is situated not far behind the middle of the body.
▲Larvae recognized by the presence of a small knob at the anterior extremity and numerous brownish food granules in the intestinal cells.

Life cycle:-direct life cycle



1-Penetration phase:- 1-7 days
During this period the larvae are making their way to the lungs and pulmonary lesions are not yet apparent.
2-Pre-patent phase:-
Around days 8–25. This phase starts with the appearance of larvae within the alveoli where they cause alveolitis This is followed by bronchiolitis and finally bronchitis as the larvae become immature adults and move up the bronchi. Towards the end of this phase bronchitis develops.
3-Patent phase:-around days 26–60. This is associated with two main lesions:-
First:- a parasitic bronchitis characterized by the presence of hundreds or even thousands of adult worms in the frothy white mucus in the lumina of the bronchi. The bronchial epithelium is hyperplastic and heavily infiltrated by inflammatory cells, particularly eosinophils.
Second:- the presence of dark red collapsed areas around infected bronchi This is a parasitic pneumonia caused by the aspiration of eggs and larvae into the alveoli. These foreign bodies quickly provoke dense infiltrates of polymorphs, macrophages and multinucleated giant cells around them.

4-Postpatent phase:-days 61-90
♠ In untreated calves, this is normally the recovery phase after the adult lungworms have been expelled. Although the clinical signs are abating the bronchi are still inflamed and residual lesions such as bronchial and peribronchial fibrosis may persist for several weeks or

months. However, in about 25% of animals which have been heavily infected, there is a flare-up of clinical signs during this phase which is frequently fatal.
This is caused by one of two causes:-
♣ Most commonly, there is a proliferative lesion so that much of the lung is pink and rubbery and does not collapse when the chest is opened. This, often described as epithelialization is due to the proliferation of Type 2 pneumocytes on the alveoli giving the appearance of a gland-like organ Gaseous exchange at the alveolar surface is gravely hindered and the lesion is often accompanied by interstitial emphysema and pulmonary oedema. The aetiology is unknown, but is thought to be due to the dissolution and aspiration of dead or dying worm material into the alveoli. The clinical syndrome is often termed postpatent parasitic bronchitis.
♣ The other cause, bacterial infection leading to acute interstitial pneumonia.

Epidemiology

♣ Larvae overwinter on pasture.
♣ Carrier animals (adult).
♣ Inhibited larvae in the capillaries
♣ Infective larvae may be spreader by attachment to the spores of a fungus pilobolus which commonly grows on bovine faeces 7 days after faeces are deposited on the ground.
♣ Migration of the larvae in the feces and soil.

Clinical sings

▲ Mildly affected animals cough intermittently, particularly when exercised.
▲ Moderately affected animals have frequent bouts of coughing at rest, tachypnoea (>60 respirations per minute) and Frequently, squeaks and crackles over the posterior lung lobes are heard on auscultation.
▲ Severely affected animals show severe tachypnoea (>80 respirations per minute) and dyspnoea and frequently adopt the classic air-hunger position of mouth breathing with the head and neck outstretched. There is usually a deep harsh cough, squeaks and crackles over the posterior lung lobes, salivation, anorexia. nasal discharge (mucus).

diagnosis

1- Clinical signs
2- Bearman techniques. For detect the L1in the faeces.
3- At necropsy, worms will often be apparent in the opened bronchi.
4- A lungworm ELISA can be used to detect antibodies

Treatment:- Tetramisol 15mg/kg B.W orally
- Ivomic 1-2ml/50 kg B.W
- Levamesole 7.5 mg/kg

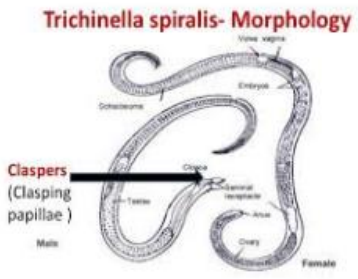
Control:-
1- Vaccination by dictol vaccine consist from attenuated L3 by irradiation to x-ray. this vaccine administrate orally to calves at least 2 month old. 2 doses of the vaccine are given at 4weeks interval.
2- Grazing system (mixing grazing).
3- Anthelmintics.

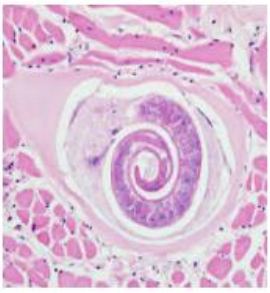
محاضرة رقم 14

Trichinella

Family:-Trichinellidae
Genus :-Trichinella spiralis
Hosts:-Most mammals. the pig and man are the important hosts.
Site of infection:-
The adults occur in the small intestine and their larvae in the striated muscles, the diaphragmatic, intercostal are considered to be predilection sites.

Morphology:-
▲The male is about 1mm long, the esophagus is at least one third of the total body length and the tail has two small cloacal flaps, but no spicule.
▲ The female is 3 mm long and the uterus contains developing larvae (larvae parous).
▲ Trichinella infection is most easily identified by the presence of coiled larvae in striated muscle.





Life cycle:-direct life cycle

After fertilization, the males die while the females burrow deeper into the villi. Three days later, they produce L1 which enter the lymphatic vessels and travel via the blood stream to

the skeletal muscles. There, still as L1 they penetrate muscle cells where they are encapsulated by the host, grow and a characteristic coiled position. This process is complete within seven weeks by which time the larvae are infective and may remain so for years. Development is resumed when the larvae are ingested by another host. The L1 is liberated, and in the intestine undergoes four molts to become sexually mature within two days.
The infective stage is L1 in muscle.

Pathogenesis:-

1-Acute enteritis.
2- larval invasion of the muscles cause acute myositis.
3- fever, eosinophilia and myocarditis.
4- periorbital edema and ascites are also common in man.

Diagnosis:-

1- In live domestic animals. At meat inspection, heavy larval infections may occasionally be seen with the naked eye as tiny greyish white spots
2-Small samples of muscle of about one gram are squeezed between glass plates.
3-Small portions of muscle may be digested in pepsin/HCI and the sediment examined microscopically for the presence of larvae.
4- Immunodiagnostic tests have been used such as ELISA. محاضرة رقم 15
5-Histopath.

Trichuris

Superfamily:-Trichuroidea
Which involved two families:-
1-Trichuridae 2-Trichinellidae

Family:-Trichuridae
Genus:- Trichuris (whip worm)
Species:-

Trichuris ovis	sheep and goat
T. globulosa	cattle
T. suis	pigs
T. vulpis	dogs
T. trichiura	man

Site :-Large intestine, particularly the caecum.

Morphology:-

▲Generally known as whip worms ,the anterior part of the body is long and slender (filamentous) while the posterior part is much thicker.
▲ The hind end of the male is coiled and have single spicule surrounded by sheath.
▲ The vulva situated at the wide part of the body.
▲ Eggs are lemon shaped with a plug at both ends; in the feces these eggs appear yellow or brown in color.

Life cycle:-direct life cycle:-
The infective stage is the L1 within the egg which develops in one or two months of being passed in the feces depending on the temperature. Under optimal conditions these may subsequently survive for several years.
After ingestion (egg contain L1) , the plugs are digested and the released L1penetrate the glands of the caecal mucosa . all four molts occur within these glands, the adults emerging on the mucosal surface with their anterior ends embedded in the mucosa . The prepatent period ranges from 6-12 weeks depending on the species .

Pathogenesis:-
Inflammation of the caecal mucosa. This results from the subepithelial location and continuous movement of the anterior end to the whipworm as it searches for blood and fluid
Clinical signs:-
watery diarrhea which usually contains blood, loos of weight ,anemia
Diagnosis:-
diagnosis may depend on finding numbers of Trichuris eggs in the faeces.

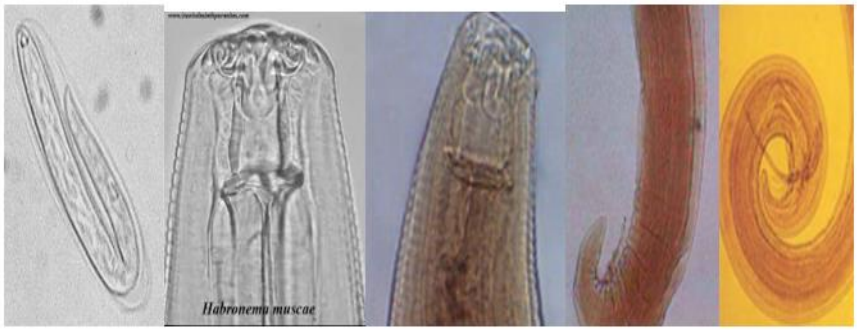
Habronema محاضرة رقم 16

Superfamily:- Spiruroidea
Family :- Spiruridae
The major characteristic of this group is the coiled tail of the male.The life cycles are indirect involving Arthropods intermediate hosts.
Genus:-Habronema
parasitic in the stomach of the horse and donkey, may cause a catarrhal gastritis.The chief importance of these parasites is as a cause of cutaneous habronemiasis or (summer sores) in warm countries.
Hosts :-Horses and donkeys
Intermediate hosts:-Muscid flies
Site:-Stomach
Species:-

Parasitology

Habronema megastoma (Draschia) intermediate host house fly
H.muscae (Musca domestica)
H.microstoma intermediate host stable fly (Stomoxys calcitran)

Morphology:-
* Slender white worms, the male has coiled tail, there are two lateral lips.
* H. megastoma is about 13 mm long and have funnel-shaped buccal cavity with a thick cuticular lining.
* H. muscae and H. microstoma are longer 22-25 mm and have cylindrical buccal cavity with a thick cuticular lining.
* The left spicule of H. muscae is five time as long as the right one.
The elongated eggs are thin shelled and larvated when laid.



Life cyc
The emb eggs are passed in the feces of the host and are ingested by the maggot of flies , H.muscae and H. megastoma developed in the house fly while H . microstoma in the stable fly (stomoxys calcitrans). Development to L3 with the development to maturity of the fly .When the fly feeds around the mouth of the horse the larvae pass from its mouthparts on to the skin and are swallowed. Alternatively infected flies may be swallowed whole. Development to adult takes place in the glandular area of the stomach in approximately two months.
When Habronerma larvae are deposited on a skin wound or around the eyes they invade the tissues, but do not complete their development. and causes cutaneous habronemiasis or summer sores.

Pathogeneses:-
1-H. megastoma produce granuloma like tumor in the stomach wall which have one or more opening in which the worms are located ,this tumor may interfere mechanically with the function of stomach.
2-Other two species occur free in the stomach and may penetrate into the mucosa they produce irritation which lead chronic catarrhal gastritis and ulcer of the stomach.
3-Cutaneous habronemiasis (summer sores) is caused by Habronema larvae which are deposited in wound by infected flies .All three species of Habronema may be involved in this condition.
4-Habronema species larvae may also be associated with granular conjunctivitis with thickening and ulceration of the eyelids .

Clinical signs:-

1-These are usually absent in gastric habronemosis.
2-Lesions of cutaneous habronemosis are most common in areas of the body liable to injury and occur during the fly season in warm countries. During the early stages, there is intense itching of the infected wound. Subsequently greasy, a reddish-brown non-healing granuloma develops .Later the lesion may become more fibrous .
3-Invasion of the eye produces a persistent conjunctivitis with ulcers especially at the medial canthus.

Diagnosis:-

1-The gastric infection is difficult to diagnosis ,some larvae or worms may be found in gastric lavage through stomach tube.
2-Finding of non-healing reddish cutaneous granulomas, the larvae recognized by spiny knob on their tail may be found in material from those lesion.

Treatment and control:-

1-Anthelmintic
2- Cutaneous lesion treated by ivermectin
3-Use of insect repellent.
4-Cryo surgery have been used in chronic cases

محاضرة رقم 17

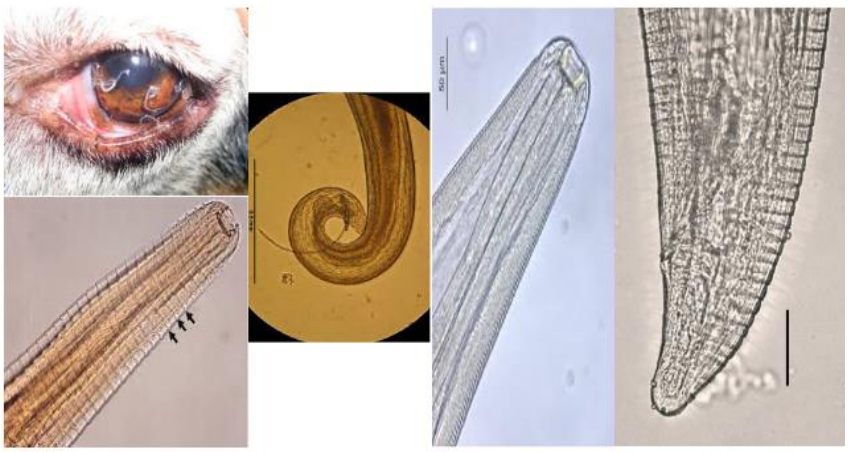
Thelazia

Family:-Thelaziidae
Genus:-Thelazia
Members of this genus are principally found in or around the eyes of animals and can be responsible for a keratitis.

T. lacrymalis	equines
T. californiensis	dog, cat and occasionally sheep
T. rhodesi	cattle

Site :-Ocular region especially the conjunctival sac and lachrymal duct
Intermediate hosts :-Muscid flies

Morphology:-
Small thin white worms 1-2 cm long. A mouth capsule is present and the cuticle has prominent striations at the anterior end.



Life cycle

The worms are viviparous. The L1 passed by the female worm into the lachrymal secretion is ingested by the fly intermediate host as it feeds. Development from L1to L3 occurs in the ovarian follicles of the fly in 15-30 days during the summer months. L3 migrate to the mouthparts of the fly and are transferred to the final host when the fly feeds. Development in the eye takes place without further migration and the prepatent period is between 3 and 11 weeks depending on the species.

Pathogenesis:-

Lesions are caused by the serrated cuticle of the worm and most damage results from movement by the active young adults causing lachrymation, followed by conjunctivitis. In heavy infections the cornea may become cloudy and ulcerated. There is usually complete recovery in about two months although in some cases areas of corneal opacity.

Clinical signs:-

Lachrymation, conjunctivitis and photophobia. Flies are usually clustered around the eye because of the excessive secretion. In severe cases, the whole cornea can be opaque.

Diagnosis:-

This is based on observation of the parasites in the conjunctival sac. It may be necessary to instill a few drops of local anesthetic to facilitate manipulation of the third eyelid.

الفهرس

Parasitology