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Superfamily Trichostrongyloidea :

- Are small hair like worms in the bursate group.
- The buccal capsule is absent or very small and is devoid of leaf-crowns and usually bears no teeth.
- The male bursa is well developed with large lateral lobes and a small dorsal lobe and two spicules .
- Adults parasitic in the alimentary canal of sheep, cattle, equines and birds. Genera of important include : Ostertagia, Marshallagia, Haemonchus, Trichostrongylus,Cooperia, Nematodirus, and Dictyocaulus (only parasitic in the lung).

Hypobiosis (arrested larval development)
This phenomenon may be defined as temporary cessation in development of a nematode .
The accumulation of arrested larvae often with the onset of cold autumn , winter or very dry condition , the maturation of these larvae coincides with the return of environmental condition .
The stage at which larvae become arrested varies between species of nematode (L3 in Trichostrongylus , L4 in Ostertagia and Haemonchus), in mature adult in Dictyocaulus .

Ostertagia :
This genus is the major cause of parasitic gastritis in ruminants in temperate areas of the world.

Host : Ruminants

Site : abomasum

Species :
Ostertagia ostertagi in cattle , O. circumcineta in sheep and goats, O. trifurcate in sheep and goats
Distribution :
Worldwide
Identification :
-The adults are slender reddish-brown worms up to 1cm long, occurring on the surface of the abomasal mucosa and are only visible on close inspection.
-The larval stages occur in the gastric glands and can only be seen microscopically following processing of the gastric mucosa, species differentiation is based on the structure of the spicules which usually have three distal branches.

Bovine Ostertagiosis:

Ostertagia ostertagi : (brown stomach worms)
O. ostertagi is perhaps the most common cause of parasitic gastritis in cattle. The disease, often simply known as ostertagiosis. Is characterized by weight loss and diarrhea and affected young cattle during first grazing season.

Life cycle :

O. ostertagi has a direct life cycle. The eggs → passed in the feces under optimal conditions developed within fecal pad to the infective third stage(L3) within two weeks → When moist conditions the L3 migrate from the feces on to the herbage → ingestion L3 , and exsheaths in the rumen and lumen of abomasal glands → Tow parasitic moults occur before the L5 emerges from the gland around 18 days after infection to become sexually mature on the mucosal surface. The entire parasitic life cycle usually takes three weeks, but under certain circumstances many of the ingested L3, become arrested in development at the early fourth larval stage (EL4) for periods of up to six months.

Pathogenesis :

- The presence of O. ostertagi in the abomasum in sufficient numbers gives rise to extensive pathological and biochemical changes - These changes are maximal when the parasites are emerging from the gastric glands this is usually about 18 days after infection, but it may be delayed for several months when arrested larval development occurs.

-the parietal cells, which produce hydrochloric acid , are replaced by rapidly dividing undifferentiated, non-acid- secreting cells.

-reduction in the acidity of abomasal fluid , increase PH , loss of bacteriostatic , hypoalbuminia , due to elevated plasma pepesinogen

- Macroscopically the lesion is a raised nodule with a visible central orifice, in heavy infections, these nodules coalesce to produce an effect reminiscent of morocco leather.

- The abomasal folds very edematous , hyperemic and some times necrosis and sloughing of the mucosal surface occurs and enlargement of local lymph nodes as a reduction in the acidity of the abomasal fluid.

Clinical signs :

Bovine ostertagiosis is known to occur in two clinical forms as follows :

* Type I disease is usually seen in calves grazed intensively during their first grazing season, as the result of larvae ingested 3-4 weeks previously.

- persistent watery diarrhea and has a characteristic bright green color.

-coats dull and the hind quarters heavily soiled with feces

-morbidity is usually high 75% but mortality is rare provided treatment is instituted within 2-3 days.

* Type II disease occurs in yearlings usually in late winter or spring following their first grazing season and results from the maturation of larvae ingested during the previous autumn and subsequently arrested in their development at the early fourth larval stage.

-diarrhea is often intermittent and anorexia and thirst are usually present

-coat dull .

- hypoalbuminaemia is more marked

- moderate anemia

- submandibular oedema.

-mortality is very high unless early treatment with an anthelmintic effective against both arrested and developing larval stages is instituted

Diagnosis :

1- clinical signs

2- The season .

3- The grazing history

4- Fecal egg counts. these usually more than 1000 eggs per gram (epg) and are a useful aid to diagnosis.

5- Plasma pepsinogen levels.

6- Post-mortem examination if this is available the appearance of the abomasal mucosa is characteristics there is a putrid smell from the abomasal contents due to the accumulation of bacteria and the high pH. The adults worms, reddish in color and 1.0cm in length can be seen on close inspection of the mucosal surface.

Treatment :
treatment at the standard dosage rates with any of the modern benzimidazoles (albendazole, fenbendazole of oxfendazole), the pro-benzimidazoles (febantelnetohimin and thiophanate) levamisole or the avermectins / milbemycins eg: ivermectin. All of these drugs are effective against developing larvae (arrested or hypobiotic larvae) and adult stages.

Control :
Ostertagiosis has been prevented by :
1-routinely treating young cattle with anthelmintic over the period
when pasture larval levels are increasing.
2-grazing calves on new grass leys.
3-resting pasture or grazing it which another host, such as sheep, which are not susceptible to O. ostertagi, until most of the existing L3, on the pasture have died.
Ovine Ostertagiosis :
In sheep O.circumcineta and O. trifurcate are responsible for outbreaks of clinical ostertagiosis, particularly in lambs.

Life cycle :
Both the free-living and parasitic phases of the life cycle are similar to those of the bovine species.

Pathogenesis :
In clinical infections, this is similar to the situation in cattle and the same lesions are present at necropsy in subclinical infection
Clinical signs :
The most frequent clinical sign is a marked loss of weight. Diarrhea is intermittent and although stained hindquarters are common, the fluid feces which characteristic bovine ostertagiosis are less frequently seen.
Diagnosis :
This is based on clinical signs, seasonality of infection and fecal egg counts and, if possible, post – mortem examination, when the characteristic lesions can be seen in the abomasum. Plasma pepsinogen levels are above the normal .

Treatment :
Ovine ostertagiosis responds well to treatment with any of benzimidazoile or probenzimidazole, levamisole , which in sheep is effective against arrested larvae , or the avermectins / milbemycina.

Haemonchus:
(Stomach worms,Wire worms,Barber pole worms)
Species : Haemonchus contortus (H.Placei), H.Similis
Hosts: sheep , goats and cattle
Site: Abomasum
Distribution : Worldwide.

Identification:
Gross: The adults are easily identified because of their specific location in the abomasum and their large size (2.0-3.0 cm) in fresh specimens. The white ovaries winding spirally around the blood-filled intestine produce a " barber's pole" appearance.

Microscopic:
The male has an asymmetrical dorsal lobe and barbed spicules. The female usually has a valval flap in both sexes there are cervical papillae and a tiny lancet inside buccal capsule.

Life cycle :
This is direct and the preparasitic phase is typically trichostrongyloic. The females are prolific egg layers. The eggs hatch ⟶ to L1 on the pasture and develop ⟶ L3 in as short a period as five days but development may be delayed for weeks or months under cool conditions ⟶ ingestion(L3) and exsheathment in the rumen the larvae moult twice in close apposition in gastric glands then develop the picrcing lancet which enables them to obtain blood from the mucosal vessels adults they move freely on the surface of the mucosa. The prepatent period is (2-3) weeks in sheep and four weeks in cattle.

Ovine Haemonchosis :
Pathogenesis :
*Acute Haemonchosis
-anemia due to the blood-sucking habits of the worms each worm removes a bout 0.05ml of blood per day by ingestion and seepage from the lesions so that a sheep with 5000 H.contortus may lose about 250 ml daily.becomes apparent about two weeks after infection
-progressive and dramatic fall in the packed red cell volume loss of iron and protein
-At necropsy, between 2000 and 20000 worms may be present on the abomasal mucosa which shows numerous small haemorrhagic lesions, the abomasal contents are fluid and dark brown due to the presence of altered blood.
- The carcass is pale and oedematous and the red bone marrow has expended from the epiphyses into the medullary cavity.
*Hyper acute haemonchosis
Less commonly, in heavier infections of up to 30.000 worms apparently healthy sheep may die suddenly from severe haemorrhagic gastritis this is termed.
*Chronic haemonchosis :
is negligible and develops during a prolonged dry season when reinfection but the pasture becomes deficient in nutrients.
Clinical signs :
- In hyperacute cases, sheep die suddenly from haemorrhagic gastritis.
- In Acute haemonchosis is characterized anemia, variable degrees of odems, submandibular form and ascites are most easily recognized. Lethargy, dark colored feces and falling wool diarrhea is not generally a feature.

- Chronic haemonchosis is associated by or with progressive weight loss and weakness, neither sever anaemia.

Epidemiology :
Is best considered separately depending on whether it occurs in tropical and subtropical or intemperate areas. Haemonchosis is a primarily a disease of sheep in warm climates, since high humidity is also essential for larval development and survival.
The frequency and severity of outbreaks of disease is largely dependent on the rainfall in any particular area.

Diagnosis :
- Acute Haemonchosis
* history and clinical signs.
* fecal worm egg count.
* Necropsy finding
- Hyper acute haemonchosis
death may have occurred and only the abomasum may show changes
- Chronic haemonchosis
Is more difficult because of the concurrent presence of poor nutrition
Treatment
benzimidazoles, Levamisole, avermectin /milbemycin or salicylanilide.

Control :
Depending on the:
- duration and number of periods in the year when rainfall and temperature permit high pasture levels of H.contortus larvae to develop at such times it may be necessary
-use an anthelmintic at intervals of 2-4 weeks depending on the degree of challenge. Sheep should also be treated at least once at the start of prolonged rain to remove persisting hypobiotic larvae whose development could pose a future treat

Bovine Haemonchosis :
is similar in most respects to haemonchosis in sheep and is important in the tropic and subtropics during seasonal rains when sever outbreaks may occur. However the disease has also been recorded at the end of a long dry season due to the maturation of hypobiotic larvae.
Unlike haemonchosis is sheep grazing cattle over two years old are relatively immune at though this may be broken, down by drought conditions which lead to poor nutrition and heavy challenge from congregation of animals around watering points. Treatment and control are similar to that described for H.contortus in sheep.

The self-Cure phenomenon:
In areas of endemic haemonchosis it has often been observed that after the advent of a period of heavy rain the fecal 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.
This event is commonly termed the self-cure phenomenon, and has been reproduced experimentally by superimposing an infection of H.contortus larvae on an existing adult infection in the abomasum the expulsion of the consequence of an immediate-type hypersensitivity reaction to antigens derived from the developing larvae, it is thought that a similar mechanism operates in the naturally occurring self-cure.

Trirchostrongylus :
Hosts :
Ruminants, horses. Pigs, rabbits and fowl.

Species :
T axei: abomasum of ruminants and stomach of horses and pigs
T. colubriformis ruminants.
T. vitrines
T. capricola sheep and goats
T. tenuis small intestine and caeca of game birds.

T.axei (hair worm)
Distribution : Worldwide
Gross :
The adults are small and hair-like usually less than 7mm long and difficult of with the naked eye.

Microscopic :
The worms have no obvious buccal capsule. The spicules are thick and unbranched and in the case of T. axei are also unequal in length in the female the tail is bluntly tapered and there is no vulval flap. In T. axei the eggs are arranged pole to pole longitudinally.

Life cycle :
This is direct and the parasitic phase is typically Trichostongyloids except exsheathment of L3 of intestinal species occurs in the abomasum. Under optimal conditions, development from the egg to infective stage occurs in 1-2 weeks, the parasitic phase is no-migratory and the prepatent period in ruminants 2-3 weeks. In the horse, T. axei has a prepatent period of 25 days.

Pathogenesis :
Following ingestion, the L3 of the intestinal species penetrate between the epithelial glands of the mucosa with formation of tunnels beneath the epithelium, after infection, there is considerable haemorrhage , oedema and plasma proteins are lost into the lumen of the gut. Grossly, there is an enteritis, particularly in duodenum,, the villi become distorted and flattened reducing the area available for absorption of nutrients and fluids. parasites are congregated within a small area, erosion of the mucosal surface is apparent. In heavy infections diarrhea occurs, and this , together with the loss of plasma protein into the lumen of intestine, leads to weight loss. A reduced deposition of protein, calcium and phosphorus has also been recorded. In the case of T. axei the changes induced in the gastric mucosa are similar to those of Ostertagia with an alteration in pH and an increased permeability of the mucosa.

Clinical signs :
The principal clinical signs in heavy are :
- weight loss and diarrhea.
-in appetence and poor growth rates.
- sometimes accompanied by soft feces, are the common signs.

Epidemiology :
The embryonated eggs and infective L3 of Trichostrongylus have a high capacity for survival under adverse conditions whether these are extreme cold or desiccation, larval numbers increase in pasture in summer and autumn giving riving rise to clinical problems during this season, the seasonal occurrence being similar to that of ostertagia spp. In contrast to other trichostrongyles hyoobiosis occurs at the L3, stage although their role in outbreaks of disease has not been established.

Diagnosis :
This is based on :
1-clinical signs
2- seasonal occurrence of disease
3-postmortem examination.
4-Faecal egg counts arc a useful aid to diagnosis although fecal cultures are necessary for generic identification of larvae.

Treatment and control :
Depending on the host, this is as described for bovine ostertagiosis, parasitic gastroenteritis in sheep and strongylosis in the horse.

T. tenuis infection :
In game birds, heavy infections produce an acute and fatal haemorrhagic typhlitis. Lighter infections result in a chronic syndrome characterized by anemia and emaciation. On game farms, therapy with levamisole in the drinking water has proved useful

Cooperia :
Hosts : Ruminants.
Site : small intestine.
Species :
C.punctate
C.pectinata

Distribution : Worldwide

Identification :
Gross :
In size cooperia are similar to ostertagia
life cycle :
This is direct and typical of the superfamily. The life cycle of Cooperia are similar requirement to Haemonchus. The prepatent period varies from (15-18 days)
pathogenesis
Cooperia are generally considered to be mild pathogens in calves and lambs respectively although in some studies they have been associated with in appetence and poor weight gains.
Clinical signs :
- Loss of appetite, - Poor weight gains.
- In C.punctate, C.pectinata sever diarrhea sever weight loss, and submandibular oedema.

Epidemiology :
This is similar to that of Ostertagia in temperate areas and similar to that of Haemonchus in subtropical areas.

Control and Treatment :
Treatment and control similar to those applied in bovine Ostertagiosis.

Nematodirus
Is a special importance as a parasite of lambs
Hosts : Ruminants
Site : small intestine

Species :
Nematodirus battus sheep, calves
N. filicolis sheep and goats
N. spthiger sheep and goats, occasionally cattle

Distribution : Worldwide
Nematodirus battus

Gross :
The adult are slender worms about 2cm long. Twisted worms produces an appearance similar to that of cotton wool.

Microscopic :
N. battus is characterized by having only one set of parallel rays in each bursal lobe while the female worm has a long pointed tail and the large egg is brownish with parallel sides.

Life cycle :
the main difference in the life cycle of N.battus compared with other parasitic worm is that development to an infective larvae take place within the egg
the egg → L1 develop in the egg L2 develop in the egg → L3 infective stage (they migrate in to herbage and wait to eating by sheep L4 and mature develop in to adult.

Pathogenesis :
Following ingestion of large numbers of L3 there is distribution of the intestinal mucosa particularly in the ileum. On the mucosal surface, development through L4 to L5 is complete and this consider with sever damage to the villi and erosion of the mucosa leading to the villous atrophy, exchange fluids and nutrients is grossly reduced, diarrhea, dehydrated, enteritis.

Clinical signs :
In severe infections diarrhea is the most prominent clinical signs dehydration, thirsty.

Epidemiology:
The two most important features of the epidemiology of Nematodirus :
1- The capacity of the free living stages (Larvae) ,the egg containing the L3 to survive on pasture , some for up to two years.
2- The critical hatching requirement of most eggs.
Diagnosis :
- Grazing history
- Clinical signs
- Post-mortem examination
-egg fecal count.
Treatment:
- Levamisole , - Avermectin , - benzimidazole
Control :
The disease can be controlled by avoiding the grazing of successive lamb crops on the same pasture. Control can be achieved by anthelmintic prophylaxis.
Family : Dictyocaulidae
Genus : Dictyocaulus (lung worms)
This genus living in the bronchi of cattle, sheep , horses and donkeys, is the major cause of parasitic bronchitis in these host.
Hosts : Ruminants, horses and donkeys.
Species :
Dictyocaulus viviparous in cattle
D. filaria in sheep and Goats
D. arnfieldi in donkeys and horses.

Distribution : Worldwide

Identification :
The adults are slender thread-like worms up to 0.8 cm in length , their location in the trachea and bronchi and their size are diagnostic.

D. viviparus :
Is the cause of parasitic bronchitis in cattle, also known verminous pneumonia of dictyocaulosis. The disease is characterized by bronchitis and pneumonia and typically affects young cattle during their first grazing season. The disease is prevalent in temperate areas with high rainfall.
Life cycle :
Eggs hatch in the lung L1 swallowed pass through the feces L2 L3 Ingestion penetrate intestinal wall Lymph vessels moult to L4 lung (male and female in the bronchi of the host)
The prepatent period is around 3-4 weeks.
Pathogenesis :
This may divided into 4 phases.
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- Prepatent phase : 8-25 days this phase starts with appearance of the larvae with in the alveoli and bronchi where they cause alveolitis and bronchitis.
The end of this phase characterized by mucus containing immature lung worms in the air ways.
Heavily infected animals whose lungs contain several thousand developing worms may die from day 15 due to respiratory failure sever interstitial emphysema and pulmonary oedema.
3- Patent phase : 26-60 days this is associated with 2 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.
secondly the presence of dark red collapsed areas around infected bronchi this is parasitic pneumonia caused by the aspiration of egg and Larvae
4- Post patent phase : 60-61 days in untreated calves, this is normally the recovery phase after the adult lung worms have been expelled. The lesion is interstitial emphysema , pulmonary oedema, gaseous exchanges at the alveolar surface it is thought to be due to the dissolution and aspiration of dead worm material into the alveoli.
Clinical signs :
* Mildly affected animals cough intermittently, particularly when exercised.
* Moderately affected animals have frequent coughing at rest tachypnea,
* severely affected animals show severe dyspnea , month breathing with the head and neck outstretched, deep harsh cough, salivation, anorexia and sometimes time death in 24-48 hours.

Epidemiology :
Generally only calves in their first grazing season are clinically affected, where the disease is endemic older animals have a strong acquired immunity. In endemic areas infection may persist from year to year in two ways :
1- Over wintered larvae L3 may survive on pasture from autumn until late spring in sufficient numbers to initiate infection.
2- Carrier animals : small numbers of adult worms can survive in the bronchi of infected animals, particularly yearlings until the next grazing season.

Diagnosis :
- Clinical signs .
- history of grazing
- Larvae are found only in the feces of patent cases by Baermann method
-eggs may be found in the sputum or discharge

Treatment : Benzimidazoles, levamisol, avermectin.

Control :
- The best method of preventing parasitic bronchitis is to immunize all young calves with lung worm vaccine.
- Control of parasitic bronchitis in first year grazing calves has been achieved by the use of prophylactic anthelmintic treatment.

Dictyocaulus arnfieldi :
The parasite of donkeys and horses
Life cycle :
The life cycle in not fully known. But is considered to be similar to that of bovine lung worm. D. viviparus except in the following respects. The adult worms are most often found in the small bronchi and their eggs containing the first stage larvae hatch soon after passed in the faces. The prepatent period is between 2-4 months.
Pathogenesis :
The characteristic lesion is similar in both horses and donkeys and in some differences from bovine parasitic bronchitis.
Clinical signs :
- are rarely seen in donkeys.
- hyperpnea and harsh lung sounds may be detected.
- In older horses infections become patent but are often associated with persistent coughing and increased respiratory rate.
Epidemiology :
Most commonly this occurs when donkeys are grazed and companion with horses, donkeys acquire infection as foals and tend to remain infected.
Diagnosis :
- modified Bermann technique is using for larvae detection from feces
- clinical signs may be suggestive of D. arnfieldi infection.
Treatment :
Levamisol, febendazole or mebendazole.
Control :
- Ideally , horses and donkeys should not be grazed together.
- Treat the donkeys preferably in the spring with a suitable anthelmintic.
Dictyocaulus filaria :
- The most important lung worm of sheep and goats.
- Is commonly associated with a chronic syndrome of coughing and unthrifitness which usually affects lambs and kids.

Life cycle :
Similar to that of D. vivparus except that the prepatnt period is five weeks.

Pathogenesis :
Similar to that of D. vivparus infection.
Clinical signs :
Coughing and unthrifitiness, dyspnea , nasal discharge are also present, diarrhea or anemia due to concurrent gastrointestinal trichostrongylosis or Fasciolosis.

Epidemiology :
the ewe as a carrier are significant factors in the persistence of infection on pasture from year to year. In ewes it seems likely that the parasites are present largely as hypobiotic larvae in the lung during each winter and mature in the spring.

Diagnosis :
Similar to that of D. vivparus infection.
Treatment and control :
Similar to that of D. vivparus infection

Super family Spiruroidea :
Family : Spiruridae
Genus : Habronema
Hosts : Horses and Donkeys
Intermediate hosts :Muscid flies.
Sites : Stomach
Species : Habronema muscae , H. microstoma ,H.megastoma

Distribution : world wide.
Identification :
- Slender white worms 1-2.5 cm long.
Life cycle :
Eggs or L1 are passed in the faces the L1 are ingested by larval stages of muscid files including Musca, stomoxys and Haematobia which are often present in faces development to L3 the L3 pass from its mouth parts and put on to the nostrils ,lips, wounds of the horse .
when swallowed, development to adult takes place in the glandular area of the stomach in approximately two months.

Pathogenesis :
The adults in the stomach may cause:
- gastritis .
-granulomatous lesion in wounds
- conjunctivitis with nodule are thickening and ulceration of the eyelids associated with invasion of eyes.
Clinical signs :
-non healing reddish cutaneous granuloma
-later the lesion may become more fibrous
-conjunctivitis with nodular ulcer .

Epidemiology :
The seasonality of cutaneous lesion is related to the activity of fly vectors.

Diagnosis :
- clinical signs.
- the Gastric infection is not easily diagnosed, because few eggs and larvae are passed.
-larvae or worms may be gastric lavage through stomach tube

Treatment and control :
Ivermectin, radiation therapy and surgery have been used in more chronic cases, prevent injuries and to control fly populations.

Family : Thelezidea
Genus: Thelezia
Hosts : Cattle ,birds, other domestic animals and occasionally man.

Intermediate hosts :
Musca domestica .

Site : Ocular region especially the conjunctival sac and lachrymal duct.
Identification :
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 :
Indirect , the L1 passed by the female worm in the lachrymal secretion ➡ ingested L1 by the fly development L1 to L3 occurs in the fly ➡ L3 migrate to the mouth parts of the fly ➡ then transferred to the final host when the fly feeds development in the eye

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.
Clinical signs :
Lachrymation, conjunctivitis and photophobia. Flies are usually clustered around the eye because of the excessive secretion , in sever the whole cornea can be opaque.

Epidemiology :
Thelazia infections occur seasonally and are linked to periods of maximum fly activity the parasite can survive in the eye for several years.

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 third eyelid.

Treatment and control :
Based on the:
- manual removal of the worms under a local anesthetic.
- administering an effective anthelminitic such as levamisole or an avermectin.

Super family Filarioidea :
Family : Filaroidea
Genus: Parafilaria

P. bovicolla :
Hosts : cattle and buffalo.
Intermediate hosts : Muscid flies.
Site : subcutaneous and intramuscular connective tissue.
Distribution : Africa, Asia , Europe and Sweden.

Identification :
Slender white worms 3-6 cm in length anteriorly, small embryonated eggs are laid on the skin surface where they hatch to release the microfilariae

Life cycle :
Eggs or larvae (L1) present in exudates from bleeding points in the skin surface ➡ ingested by flies develop to L3 in flies ➡ L3 deposit in lachrymal secretions or skin wounds in other cattle when infected flies feed ➡ L3 migrate and develop to the adult stage under the skin in 5-7 months.
Pathogenesis :
The adults of this genus live under the skin, they produce inflammatory lesions and, during egg laying, heamorrhagic exudates or "bleeding point" on the skin surface.
Clinical signs
The signs of parafilariosis are pathognomonic. Active bleeding lesions are seen most commonly in warm weather to coincide with the presence of the fly intermediate host.

Epidemiology :
Area previously free form infection Parafilaria infection may be introduced by the importation of cattle from endemic areas, spread will depend on presence of specific fly vectors.
Diagnosis :
- clinical signs.
- Small embryonated eggs or microfilariae may be found on examination of exudate from bleeding points.
- Sero-diagnosis using ELISA technique has been developed.

Treatment : Ivermectin or nitroxynil

Family : Onchocercidae
Genus: Onchocerca
Species: O.armilata vector:unknown
O.gutturosa vector:simulium
O.gibsoni vector:culicoides
Hosts : cattle
Intermediate hosts : unknown

Sites : in the skin of humb, neck,dewlap and aorta .
Identification :
The slender worms rang from 2-6 cm in length and lie tightly coiled in tissue nodules, in active lesions the presence of worms in readily established on section of these nodules.
Life cycle :
The life cycle of Onchocerca Spp. Is typically filaroid, with the exception that the microfilariae occur in the tissue spaces of the skin, rather than in the peripheral blood stream.

Diagnosis :
- Depends on the finding of microfilariae in skin biopsy samples.
- The piece of the skin is placed in warm saline and teased to allow emergence of the microfilariae, and is then incubated for six hours or more, the microfilaria are readily recognized by their sinuous movements in a centrifuged sample of the saline.

Treatment :
Ivermectin , local treatment with synthetic pyrethroids
Control :
The use of microfilaricides will reduce the numbers of infected flies.
Family : Setaridae
Genus: Setaria
Hosts : ruminants and equines.

Intermediate hosts : Many species of mosquito.
Site : usually the peritoneal surface and free in the peritoneal cavity less commonly the pleural cavity and following erratic migration to the CNS.

Species : Setaria digitata in cattle and wild ruminants
Setaria equine in horses and donkeys.

Distribution : worldwide

Identification :
Long slender worms, up to 12 cm in length the site and gross appearance are sufficient for generic identification.
Life cycle :
Indirect , The microfilariae in the blood stream are taken up by mosquitoes in which development to L3 takes about 12 days, the prepatent period is 8-10 months.

Pathogenesis :
The worms in their normal site are harmless and are only discovered at necropsy. S. digitata may have an erratic migration is sheep and goats and enter the spinal canal causing cerebrospinal nematodosis "lumbar paralysis" which is irreversible and often fatal.

Clinical signs :
There are no clinical signs when the worms are in their normal site, but when nervous tissue is involved there is locomotors disturbance, usually of the hind limbs.

Epidemiology :
Their prevalence is higher in warmer countries. Where there is seasonal activity of the mosquito vectors.

Diagnosis :
- Finding of the microfilariae in routine blood smears in adult (living animal).
- In cases of cerebrospinal nematodosis , diagnosis is only possible by microscopic examination of the spinal cord.

Treatment :
There is no treatment for setarial paralysis. Ivermectin has been reported to be effective against adult S. equine .

Control : depend on control of the mosquito vectors.

Super family Trichuroidea :
- Are found in a wide variety of domestic animals.
- There are 3 genera of interest.
1- Trichuris is found in the caecum and colon of mammals.
2- Capillaria is most commonly present in the alimentary or respiratory tract of mammals of birds. Both lay eggs with plugs at both poles.
3- Trichinella are found in the small intestine of mammals and produce larvae which immediately invade the tissue of the same host.

Family : Trichuridae
Genus : Trichuris (whip worms)
- The adults are usually found in the caecum.
- The common species are: Trichuris ovis sheep and goat
Trichuris globulosa cattle.

Identification :
Gross : The adults are 4-6cm long with a thick posterior end tapering rapidly to a long filamentous anterior end which is characteristically embedded in the mucosa because of their appearance the members of this genus are often called the "whip worms".

Microscopic :
The male tail is coiled and possesses a single spicule in a sheath; the female tail is merely curved. The characteristic eggs are lemon (or barrel) shaped with a transparent plug at both ends in the faces these eggs appear yellow or brown in color.

Life cycle :
The infective stages is the L1 with in the egg which develops in one or two months of being passed in the faces depending on the temperature. Survive for several years , after ingestion , the plugs are digested and the released L1 penetrate the glands of the caecal mucosa, subsequently all four moults occur within these glands, the adults emerging to lie on the mucosal the prepatent period ranges from 6-12 weeks depending on the species.

Pathogenesis :
Most infections are light and a symptom a tic occasionally when large numbers of worms are present they cause a diphtheritic inflammation of the caecal mucosa. This results from the sub-epithelial location and continuous movement of the anterior end to the whip worm as it searches for blood and fluid.

Clinical significance :
The clinical signs of this genus especially in ruminants is negligible although isolated our breaks have been recorded.

Epidemiology :
The most important feature is the longevity of the eggs which after three of four years may still survive as a reservoir of infection.

Diagnosis :
- Diagnosis may depend on finding numbers of trichuris eggs in the feces.
- Clinical signs may occur during the prepatent period.

Treatment : Benzimidazoles, avermectins, levamisole.
Control : Prophylaxis is rarely necessary. Particularly in ruminants.

Family : Capillaridae
Genus : Capillaria
- Very thin hair-like worms are not readily visible to the naked eye in unprepared gut contents.
- There are many species in mammals and birds.

Identification :
- These are very fine filamentous worms between 1-5cm long.
- The males have a long single spicule and often possess a primitive bursa like structure.
- The females contain eggs which resemble those of Trichuris in possessing bipolar plugs. But which are more barrel – shaped and colorless.

Capillaria in Birds :
Capillaria obsignata : is present in the upper small intestine of chickens, turkeys and pigeons and has direct life cycle.
Capillaria caudinflata: is also found in the small intestine of chickens and turkeys, the egg of this species requires to be ingested by an earth worm in which it hatches, the final host being infected by ingestion of the earth worm.
Capillaria contorta : occurs in the esophagus and crop of the chicken, turkey, duck and wild birds. Earth worm intermediate host also essential. The prepatent period of these three avian species is 3-4 weeks.

Pathogenesis and clinical significance :
Like Trichuris the anterior ends of the parasite are buried in the mucosa and in heavy infection cause diphtheritic inflammation leading to in appetence and emaciation diarrhea , mortality in cases may be high.

Epidemiology :
Young birds are most susceptible to Capillaria infections, while adults may serve as carriers, C. obsignata is perhaps most important since, having a direct life cycle. Its occur indoors in birds kept on deep litter.

Diagnosis :
- Because of the non-specific nature of clinical signs and the fact that, in heavy infections, these may appear before Capillaria eggs are present in the feces.
- Diagnosis depends on necropsy and care full examination of the esophagus, Crop or intestine for the presence of worms, this may be carried out by microscopic examination of mucosal scrapings

Treatment :
Levamisole in the drinking water is highly effective.

Family : Trichinellidae
Genus: Trichinella
Trichinella spiralis
Is a nematode with a very wide host range and the cause of an important world wide zoonosis.

Hosts : Most mammals, from the zoonotic aspect.

Sites :
The adults occur in the small intestine and their larvae in the striated muscles, the diaphragmatic, intercostal and masseter muscles are considered to be predilection sites.
Distribution : worldwide
Identification :
Because of their short lifespan, the adult worms are rarely found in natural infections , the male is about 1.0mm long , the oesophagus is at least one third of the total body length and the tail has two small cloacal flaps. But no spicule the female 3mm long and the uterus contain developing larvae. Trichinella infection is most easily identified by the presence of coiled larvae in striated muscle.

Life cycle :
Ingestion cystic larvae which are found in muscles ⇒ 1st two moults are completed within 26 hours and fourth moults occur less than two days in small intestine of host adult worm copulation male die and female produce egg L1 thoracic duct lymph upper left vena cava blood striated muscles surrounded by capsule muscle calcification larvae have no development until eating .

Pathogenesis and clinical signs :
Infection in domestic animals is invariably light, and clinical signs do not occur.

Epidemiology :
Depends on two factors first, animals may become infected from a wide variety of sources , predation and cannibalism being perhaps the most common other feeding on carrion, the second factor is the wide host range of the parasite.