TRICHOSTRONGYLOSIS / PARASITIC GASTROINTESTINAL ENTERITIS

This disease is one of the most parasitic disease of ruminants, and is common all over the world. Thrichostrongylosis is present in all regions of Turkey at a level of up to one hundred percent, and this disease is always a problem in cattles, sheeps and goats , occasionally in buffoloes. Economic loss is always present and animals often suffer health problems due to illness.

Trichostrongylosis is common that in grazing management systems, cattle, sheep and goats, as well as temporary, subtropical and tropical climate regions . It is convenient to restrict the usage of the term " parasitic gastroenteritis " (PGE) to naming the condition caused by trichostrongylid nematodes parasitizing the stomach (abomasum) and small intestine.

There are more than 50 genera and hundres of species responsible for the disease, and the disease is generally called trichostrongylosis. This disease is common worms in grazing ruminants. The trichostrongylosis are responsible for considerable mortality and widespread morbidity in ruminants.

General morphology : The trichostrongyloids are small, often hair-like, worms in the bursate group. Parasities the alimentary tract of animals . Thin worms have a small , or no buccal capsule. Teeth are usually absent.The males have a well developed bursa with large lateral lobes and two spicules, the configuration of which is used for species differentiation. The life cycle is direct and usually non-migratory and the ensheated L3 is the infected stage.

Important genera and species :

1) Trichostrogylus : adult worms are small, slightly reddish-brown in colour, slender and hair-like, usually less than 7.0 mm long. A most useful generic character is the distinct excretory notch in the oesophageal region. Species identification is based on the shape and size of the spicules. In the female, the tail is bluntly tapered and there is no vulval flap. The parasites are located in small intestine and occasinoally in stomach.

Trichostrongylus species ; T. axei , T. colubriformis, T.vitrinus, T.capricola, T.longispicularis,

T. probolurus, T. retortaeformis (rabbit, hare), T. calcaratus (rabbit,hare) T. tenuis (birds)

Life cycle is direct and preparasitic phase is typically trichostrongyloid, eggs developing L3 in about 7-10 days under optimal conditions. Following ingestion and exsheathment , larvae penetrate the mucosa of the small intestine and after two moults the fifth-stage worms are present under the intestinal epithelium around 2 weeks after initial infection. The prepatent period is generally 2-3 weeks.

2) Ostertagia : These worms occur in cattle, sheep and other ruminants .The adults are slender reddish-brown worms up to 1.0 cm long, occuring on the surface of the abomasal mucosa. They possess a short buccal cavity and a very small pair of cervical papillae. In the

female the vulva can be covered with a flap or this nay be absent and the tip of the tail is annulated. The larval stage occur in the gastric glands .

Ostertagia species ; O. ostertagi , O. leptospicularis, O. trifurcata (syn.O . circumcincta),

Life cycle is direct. Eggs are passed in the faeces and, under optimal conditons, develop within the faecal pat to the infective third stage within 2 weeks. When moist conditions prevail, L3 migrate from the faeces on to the herbage. After ingestion, L3 exsheaths in the rumen and further development take place in the lumen of an abomasal gland. Two 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 3 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 6 months (also referred to as **HYBOBIOSIS**).

3) Haemonchus : The adults are easily identified because of their specific location in the abomasum and their large size (2.0-3.0 cm.). In fresh female specimens, the white ovaries winding spirally around the blood-filled intestine produce a “barber’s pole “ appearance. The buccal cavity is small and contains a small lancet-like tooth. The anterior body possesses prominent cervical papillae. The vulva is usually protected by a cuticular flap which can have a range of shapes. In the male the lateral lobes of the bursa are large, whereas the dorsal ray is small and asymmetrical.

Haemonchus species ; H. contortus, H. similis, H.longistipes

Life cycle is direct. An the preparasitic phase is typically trichostrongyloid. The eggs hatch to L1 on the pasture and may develop to L3 in as short a period as 5 days but development may be delayed for weeks or months under cool conditions. After ingestion, and exsheathment in the rumen, the larvae moult twice in close apposition to the gastric glands. Just before the final moult they develop the piercing lancet which enables them to obtain blood from the mucosal vessels. As adults they move freely on the surface of the mucosa. The prepatent period is 2-3 weeks in sheep and 4 weeks in cattle.

4) Marshallagia : similar to Ostertagia spp. and can be differantiated by its greater length (males 10-13 mm ; females 15-20mm.)

There is only one species in this genus, its name is M. marshalli .

Life cycle is similar to Ostertagia except that **L2 can hacth from the eggs**. Following ingestion, larvae burrow into the abomasal mucosa and form small greyish white nodules, which may contain several developing parasites. The young L5 emerge from the nodules around day 16 post infection and the prepatent period is 3 weeks. Arrested development of larvae can occur.

5) Cooperia : Relativelly small worms (usually less than 9 mm long), which pinkish-white when fresh. The main generic feartures are the small cephalic vesicle and the marked transverse cuticular striations in the oesophageal region. The body possesses longitudinal ridges.There is no gubernaculum.

Cooperia species ; C. oncophora, C. curtacei, C.pectinata, C.punctata, C. surnabada

The life cycle is direct. Ingested L3 exsheath , migrate into intestinal crypts for two moults and then the adults develop on the surface of intestinal mucosa. The prepatent period is 2-3 weeks.

6) Nematodirus : adults worms are whitish, and relatively long, with the anterior thinner than posterior region. They may appear slightly coiled.Adult males are 10 - 15mm and females 15-24mm in length. A small, but distinct, cephalic vesicle is present .The spicules are long and slender, and the spicules are varies shape and id a useful feature for species differentiation. The female worms have a short tail with a slender terminal appendage.

Nematodirus species ; N. battus, N.filicollis, N.spathiger, N. Abnormalis, N.helvetianus

The life cycle is direct. The preparasitic phase is almost unique in the trichostrogyloids in that **development to the L3 stage take place within the eggshell**. Species differences occur regarding the critical hacthing requirements.

In the life cycle of Nematodirus battus , development to the L3 take places within the eggshell. Hatching of most eggs requires a prolonged period of chill followed by a mean day/nigth temperature of more than 10 ° C , conditions which occur in the late spring in the northern hemisphere. Hence most of the eggs from one season's grazing remain unhatched on the ground during winter and usually only one generation is possible each year for the bulk of this species. However, some N.battus eggs deposites in the spring are capable of hacthing in the autumn of the same year , resulting in significant numbers of L3 on the pasture at this time. The ingested L3 penetrate the mucosa of the small intestine and moult to the L4 stage around fourth days. The prepatent period is about 14-16 days.

In the life cycle of Nematodirus filicollis, development to the L3 take places within the eggshell. Hatching occurs over a more prolonged period and so larvae often appear on the pasture within 2-3 months of the egg being excreted in the faeces. The prepatent period is 2-3 weeks.

In the life cycle of Nematodirus helvetianus, this species does not have the same critical hatching requirements as N. Battus and so the larvae often appear on the pasture within 2-3 weeks of the egg being excreted in the faeces.More than one annual generation is therefore possible. The prepatent period is around 3 weeks.

7) Teladorsagia : adults are slender reddish-brown worms with a short buccal cavity. Males measure 6-8 mm and females 8-10 mm.

Species ; T. circumcincta, T. davtiani

In the life cycle of Teladorsagia , the life cycle is similar to that Ostertagia life cycle.

8) Camelostrongylus : Males measures 6.5-7.5 mm . The spicules are long and narrow, bifurcating into pincer-like ends with one end shorter, and the longer end forming a hammer –like hook.

Species ; C. mentulatus

9) Mecistocirrus : worms of this genus are similar in apperance to Haemonchus concortus , except that in the female the slit-shaped flapless vulva is located close to the anus. The male measure up to around 30 mm and the females 42 mm length.

Species ; M. digitatus

Life cycle is direct and similar to that of Haemochus. The prepatent period is around 80-90 days, partly the result of the longer duration of the fourth stage in the abomasal mucosa

The morphology of eggs : eggs are of the strongyle-type; oval, thin shelled,colourless, medium size (60 X 100 µm), passed in the faeces at 8-16 cell stage (blastomers)

The egg of Nematodirus spp. is large (160-233 by 87-121 µm), ovoid, with slightly sharp poles and clear, and twice the size of the typical trichostrongyle egg. The chitinous egg-shell is thin with smooth surface and contain 2-8 large dark blastomeres, which are separated from yolk membrane by quite a large fluid-filled cavity.

The egg of Marshallagia spp. is elipsoidal shape and is much large than those of ostertagia spp. , measuring 160-200 by 75-100 µm and resemble those of Nematodirus spp. The thick-shelled eggs have almost paralled sides and contain a morula in an advanced stage of development when passed in the faeces. The eggs can be diffrentiated from those nematodirus as the morula is more develop and geographical distribution of the worms is different.

A) Ostertagiosis : ostertagiosis is especially important in temperature climates and in subtropical regions with winter rainfall. Ostertagia ostertagia is the major pathogen responsible for ostertagiosis in cattle. Ostertagia leptospicularis is occured in cattle, sheep and goat.

Ostertagia ostertagi is harbored in abomasum in hosts.

Bovine ostertagiosis : Ostertagia ostertagi is perhaps the most common cause of parasitic gastritis in cattle. The disease, often simply known as ostertagiosis, is characterised by weight loss and diarrhoea and typically affects young cattle during their first grazing season, although herd outbreaks and sporadic individual cases have been also reported in adult cattle.

Pathogenesis – clinical signs :

Large population of O.ostertagia can induce large pathological and biochemical changes and these are maximal when the parasites are emerging from the gastric glands (about 18-20 days after infection) but may be delayed for several months when arrested larval development. In the pathogensis, the principal effects of these changes are follow:

--- a reduction in the acidity of the abomasal fluid, the pH increasing from 2.0 to 7.0. This result in a failure to activate pepsinogen to pepsin. Thus, there is also a loss of bacteriostatic effect in the abomasum. This result in a failure to activate pepsinogen to pepsin and so denature protein.

--- there is enhanced permeability of the abomasum epithelium to macromolecules.

The developing parasites cause a reduction in the functional gastric gland mass responsible for the production of the highly acidic proteolytic gastric juice ; in particular , the parietal cells , which produce hydrochloric acid, are replaced by rapidly dividing, undifferentiated non-acidic secreting cells. Initially , these cellular changes occur only parasitised glands, but as it becomes distended by the growing worms which increases from 1.0- 8.0 mm in length, these changes spread to the surrounding non-parasitised glands, the end result being a thickened hyperplastic gastric mucosa.

Macroscopically ,the lesion is a rised nodule with a visible central orifice .The abomasal folds are often very oedematous and hyperaemic and sometimes necrosis and sloughing of the mucosal surface occurs ; the regional lymph nodes are enlarged and reactive.

There is an enhanced permeability of the abomasal epithelium to macromolecules such as pepsinogen and plasma proteins. Macromolecules may pass into and out of the epithelial sheet.

The result of these changes are a leakage of pepsinogen into circulation, leading to elevated plasma concentrations, and the loss of plasma proteins into the gut lumen, evetually leading to hypoalbuminaemia. In additon, in response to the presence of the adult parasites, the zymogen cells secrete increased amounts of pepsin directly into the circulation.

Clinical signs ; bovine ostertagiosis occurs in two clinical forms. In temperature climates with cold winter the seasonal occuruence of these is as follows.

---Type 1 : this form is usually seen in calves grazed intensively during their first grazing season , as the result of larvae ingested 3-4 weeks previously ; in the northern hemisphere this normally occurs from mid-July onwards. In type 1 disease , the morbidity is usually high, often exceeding 75 %, but mortality is rare provided treatment is instituted early.

---Type 2 : this form occurs in yearlings, usually in late winter or spring following their first grazing season and result from the maturation of larvae ingested during the previous

autumn and which subsequently become arrested in their development at the EL4 stage. Hypoalbuminaemia is more marked, often leading to submandibular oedema. In the type 2 disease , the mortality in animals can be high unless early treatment with an anthemintic effective against both arrested and development larval stage in instituted.

The main clinical signs in both type 1 and type 2 disease is aprofuse watery diarrhoea ; type 1 disease, where calves are at grass, this is usually persistent and has a characterristic bright green colour. In contrast , in the majority of animals with type 2 disease, the diarrhoea is often intermittent and anorexia and thirst are usually present. In both forms disease , the loss of body weight is considerable during the clinical phase and may reach 20 % in 7 -10 days.

Diagnosis :

1) the clinical signs of inappetence, weight loss and diarrhoea

2) the season, for example in Turkey type 1 occurs from July until September and type 2 from March to May

3) in type 2 disease, faecal examination for eggs may be negative and is of limited value.

4) Plasma pepsinogen levels. In clinically affected animals up to 2 years old these are usually in excess of 3.0 iu tyrosine (normal levels are 1.0 iu in non-parasited calves).

5) postmortem examination. Adult worms can be seen on close inspection of the abomasal surface.

6) in older animals, laboratory diagnosis is more difficult since faecal egg counts and plasma pepsinogen levels are less reliable

Epidemiology of ostertagiosis in cattle :

1) a considerable number of L3 can survive the winter on pasture and in soil . Sometimes the number are sufficient to precipitate type 1 disease in calves 3-4 weeks after they are turned out to graze in the spring. The surviving L3 produces patent subclinic infection and ensures contamination of the pasture for the rest of the grazing season.

2) eggs deposited in the spring develop slowly to L3 ; this rate of development becomes more rapids towards midsummer as temperatures increase and, as a result, the majority of eggs deposited during april to June all reach the infective stage from around mid-July onwards. If sufficient numbers of these L3 are ingested, the type 1 disease occurs any time from July until October. Development from egg to slow during the autumn.

3) as autumn progresses and temperatures decline, an increasing proportion (up to 80 %) of the L3 ingested become inhibited at the early fourth larval stage (EL4). In late autumn , calves can therefore harbour many thousands of these EL4 but few developing forms or

adults. These infections are generally asymptomatic until maturation of the EL4 take places during winter and early spring when type 2 disease may materialise. Maturation is not synchronous. Adult worms can play a significant epidemiological role by contributing to pasture contamination in the spring.

4) in dry summer the L3 are retained within the crusted faecal pat and can not migrate on to the pasture until sufficient rainfall occurs. If rainfall is delayed until late autumn, many larvae liberated on to pasture will become arrested following ingestion and so increase the chance of type 2 disease.

5) by second and third year of grazing , adult animals in endemic areas are usually highly immune to reinfection and of little significance in the epidemiology. However, around the periparturient period when immunity wanes, particularly heifers, there are reports of clinical disease following calves. Burdens of adults Ostertagia spp. in cows are usually low and routine treatment of herds at calving should not be required.

Control mesuares : Traditionally, ostertagiosis has been prevented by routine treating young cattle with anthelmintic over the period when pasture larval levels are increasing.

---- The provision of " safe pasture " may be achieved in two ways

a) using anthelmintic to limit pasture contamination with eggs during periods when the climate is optimal for development of L3 stage, i.e. spring and summer in temperate climates, or autumn .

b) alternatively, by resting pasture or grazing it with another host, such as sheep, which are not susceptible to O. ostertagi, until most of the existing L3 on the pasture have died out.

Sometimes a combination of these methods is employed.

1) prophylactic anthelmintic using : cattle can be given anthelmintics 2 or 3 times during grazing season. The modern anthelmintics can be given 2 or 3 times between spring and mid-July and this will reduce egg accumulation in the pasture. For calves going to pasture in early May two treatments, 3 and 6 weeks later, are used, whereas calves turned out in April require three treatments at intervals of 3 weeks. Where parenteral or pour-on macrocylic lactones are used the interval after first treatment may be extended to 5 or 8 weeks (the interval depens on the anthelmintic used) due to residual activity against ingested larvae. A long –acting injectable formulation of moxidectin is available with persistent activity against O. ostertagi for around 120 days.

Several rumen boluses are available which provide either the sustain release of anthelmintic drugs , at a constant level, over periods of 3-5 months or the pulse release of therapeutic doses of an anthelmintic at intervals of 3 weeks throughout the grazing season.

Anthelmintic prophylaxis has the advantage that animals can be grazed throughout the year on the same pasture.

2) anthelmintic treatment and move to safe pasture in grazing season

3) alternate grazing of cattle and sheep

4) rotational grazing of adult and young animals

Albendazole 7.5 mg / kg per os

Febantel 7.5 mg / kg per os

Fenbendazole 7.5 mg / kg per os

Netobimin 7.5 mg / per os

Doramectin 0.2 mg / kg sub cutan ---- 0.5 mg / kg pour on

Eprinomectin 0.5 mg / kg pour on

İvermectin 0.2 mg/kg sub cutan ----- 0.5 mg / kg pour on

Moxidectin 0.2 mg / kg sub cutan ---- 0.5 mg / kg pour on

B) Teladorsagiosis :

Teladorsagia circumcincta, T. davtiani and Ostertagia trifurcata are the major pathogens responsible for teladorsagiosis in sheep, goat and deers, and this disease is common all over the world. The parasites are found in the abomasum and these parasites are very rarely found in the small intestine.

Eggs are medium – size (about 80-100 X 40-55 µm) and are a regular ellipse with not very wide poles . The shell is thin and smooth and contains many blastomeres.

Characteristics of the disease in sheep :

The life cycle of Teladorsagia spp. is similar to the life cycle of Ostertagia spp.

Pathogenesis --- clinical signs : In the clinical infections, this resembles the situation ostertagiosis in cattle and similar lesions are present necropsy , although the morocco leather apperance of the abomasal surface seen in cattle is not common sheep and goats.

In subclinical infection , T. circumcincta causes a marked depression in appetite and this, together with loss of plasma protein into the gastrointestinal tract and sloughed intestinal epithelium, results in interference with the post-absorptive metabolism of protein. In lambs with moderate infection of T. circumsincta , carcass evaluation can show poor protein and fat deposition. Skeletal growth can also be impaired.

In clinical signs : The most frequently clinical signs is a marked loss of weight. Diarrhorea is intermittent and although stained hindquarters are common, the fluid faeces that characterise bovine ostertagiosis are less frequently seen.

Pathology : The pathology is similar to that described for Ostertagiosis in cattle. The developing parasites cause distention of parasitised gastric glands, leading to thickened hyperplastic gastric mucosa similar to that seen in cattle. In heavy infection these nodules coalesce and abomasal folds are often very oedematous and hyperaemic.

Diagnosis : this is based on clinical signs in sesonality of infection and faecal egg counts and , if possible, postmortem examination, when the characteristic lesions can be seen in the abomasum. Plasma pepsinogen levels are above the normal of about 0.8 iu tyrosine and usually exceed 2.0 iu in sheep with heavy infections.

Epidemiology : In sheep, T. circumsincta and O. trifurcata are responsible for outbreaks of clinical disease, particularly in lambs. In the northern hemisphere , a clinical syndrome analogous to type 1 bovine ostertagiosis occurs from August to October , thereafter arrested development of many ingested larvae occurs and type 2 syndrome has been occasionally reported in late winter and early spring, especially in young adults.

In the temperature climate regions , the herbage number of Teladorsagia circumsincta L3 increase markedly from midsummer onwards and this is when most disease appears. **These larvae are derived mainly from eggs passed in the faeces of ewes during the periparturient period , from 2 about weeks prior to lambing until about 6 weeks after lambing** . Eggs passed by lambs , from worm burdens which have accured from the ingestion of overwinterred larvae, also contribute to pasture contamination. It is these eggs deposited in the first half of grazing season from April to June which give rise to the potentially dangerous populations of L3 from July to October. If ingested prior to October, the majority of these larvae mature in 3 weeks ; thereafter, many become arrested in development for several months and may precipiate type 2 disease when they mature. **Adult ewes harbour only very low population of Teladorsagia spp. except during the annual periparturient rise (PPR)**.

Treatment : Ovine teladorsagiosis often responds to treatment with any of benzimidazoles or pro-benzimidazoles , levamisole(which in sheep is effective against arrested larvae), the avermectins / milbemycins or the recently introduced monepantel and derquantel (in combination with abamectin) .

Treated sheeps and lambs should preferably be moved to to safer pastue, but one which contains infective larvae ***in refugia*** ; if this is not possible, treatment may have to repeated at 6 – weekly intervals until the pasture larval levels decrease in late autumn.

Albendazole sheep 3.8mg/kg peros immature / adult

Goat 7.6 mg / kg per os immature / adult

Febantel sheep / goat 5 mg / kg per os immature / adult

Fenbendazole sheep / goat 5 mg/kg per os immature / adult / **hypobiotic larvae ???**

Mebendazole sheep 20 mg / kg per os immature / adult

Goat > 10 mg / kg per os **immature / adult ???**

Netobimin sheep 7.5 mg / kg per os adult

Oxfendazole sheep / goat 5 mg/kg per os adult

Closantel sheep 10mg/kg per os **adult ???**

Ivermectin sheep / goat 0.2 mg/kg subcutan immature/ adult / hypobiotic larvae, only in sheep

Doramectin sheep 0.2 mg/kg intra muscular immature / adult

Moxidectin sheep 0.2 mg / kg subcutan immature / adult

Abamectin sheep / goat 0.2 mg / kg subcutan immature / adult

Levamizole sheep 5 mg/ kg subcutan , 7.5mg/kg per os immature / adult

Goat 7.5 mg / kg per os immature / adult

Pyrantel sheep / goat 25 mg / kg per os immature / adult

C) Haemonchosis :

Until recently the sheep species called Haemonchus contortus and the cattle species H. placei. However, there is now increasing evidence that these are the single species H. contortus with only strain adaptations for cattle and sheep.

This species is located in abomasum in sheep, goat cattle, deer and camel and, the common name of this parasite is Barber's pole worm. In fresh specimens, the white ovaries winding spirally around the blood-filled intestine produce a ' barber's pole' appearance.

Pathogenesis and clinical sings :

Essentially, the pathogenesis of haemonchosis is that of an acute haemorrhagic anaemia due to the blood-sucking habits of the worms. Each worm removes about 0.05 ml of blood per day by ingestion and seepage from the lesions, so that a sheep with 5000 H.contortus may lose about 250ml daily. In acute disease, anaemia becomes apparent about 2 weeks after

infection and is characterised by a progressive and dramatic fall in the packed red cell volume. During the subsequent weeks the haematocrit usually stabilies at a low level, but only at the expense of a two fold to three fold compensatory expansion of erthropoiesis. However, due to continual loss of iron and protein into the gastrointestinal tract and increasing inappetence, the marrow eventually becomes exhausted and the haematocrit falls still further before death occurs. When ewes are affected , the consequent agalactia may result in the death of the suckling lambs. Less commonly, in heavier infections of up to 30.000 worms, apparently healthy sheep may die suddenly from severe haemorrhagic gastritis. This is termed hyperacute haemonchosis.

Clinical signs : in hyperacute cases, sheep die suddenly from haemorrhagic gastritis. Acute haemonchosis is characterised by anaemia, variable degrees of oedema, of which the submandibular form and ascites are most easily recognised, lethargy, dark-coloured faeces and falling wool. Diarrhoea is not generally a feature. Chronic haemonchosis is associated with proggressive weight loss and weakness, neither severe anaemia nor gross oedema being present.

Diagnosis : The history and clinical signs are often sufficient for the diagnosis of the acute disease especially if supported by faecal worm eggs. Necrospy , paying attention to both the abomasum and the marrow changes in the long bones, is also useful. In hyperacute haemonchosis, only the abomasum may show changes since death may have occured so rapidly that marrow changes are minimal. The diagnosis of chronic haemonchosis is more difficult because of the concurent presence of poor nutrition and confirmation may have to depend on the gradual disappearance of the syndrome after anthelmintic treatment.

At necropsy in cases of acute haemonchosis, there may be between 2000 and 20000 worms 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 marrow has expanded from the epiphyses into the medullary cavity.

Epidemiology : haemonchosis is primarily a disease of sheep in warm climates. However, since high humidity, at least in the microclimate of the faeces and the herbage, 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 areas.

Given these climatic conditions, the sudden occurence of acute clinical haemonchosis appears to depend on two further factors. First, the high faecal worm eggs output of betwen 2000 and 20,000 epg, even in moderate infections, means that massive pasture population of L_3 may appear very quickly. Second, in contrast to in many other helmint infections,there is little evidence that sheep in the endemic areas develop an effective acquired immunity to H. concortus, so that there is continuous contamination of the pasture.

The survival of H. concortus infection on pastures is variable depending on the climate and degree of shade, but the infective larve are relatively resistant to desiccation and some may survive for 1-3 months on pasture or in faeces.

In the endemic areas, the survival of the parasite is also associated with the ability of H. contortus larvae to undergo hypobiosis.

In endemic areas, it has often been observed that after the of a period of heavy rain 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. **This event is commonly termed the sel-cure phenomenon**. The expulsion of the adult worm population is considered to be the concequence of an immediate-type hypersensivity reaction to antigens derived from the developing larvae.

In the temperature climate regions, haemonchosis has different epidemiology. Infected larvae, which have developed from eggs deposited by ewes in the spring, are ingested by ewes and lambs in early summer. The majority of this worm population become arrested in the abomasum as EL_4 , and does not complete development until the following spring. During the period of maturation of these hypobiotic larvae, clinical signs of acute haemonchosis may occur and in the ewe this often coincides with lambing.

Treatment : when an acute outbreak and chronic disease have occured the sheep should be treated with one benzimidazole, levamisole, an avermectin, milbemycin, or closantel . Closantel is highly effective in the treatment of haemonchosis.

Control : In the endemic area this varies 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 time it may be necessary to use an anthelmintic at intervals 2-4 weeks depending on the degree of challenge. Sheep should be treated at least once at the start of the dry season and preferably also before the start of prolonged rain to remove persisting hypobiotic larvae whose development could pose a future threat. For this purpose, one of the modern benzimidazoles or an avermectins or milbemycin is recommended.

D) Nematodirosis : The adulst of Nematodirus spp. are occur in the small intestine sheep, goat , cattle, deer and other ruminants.

Characteristics of the disease in sheep : Nematodirosis, due to Nematodirus battus , common name is thread-necked worm,is an example of a parasitic disease where the principal pathogenic effect is attributable to the larval stage. Nematodirus battus is more important in Turkey .

Pathogenesis and clinical signs : Following ingestion of large number of infectif larvae (L3) there is disruption in the intestinal mucosa, particularly in the ileum, although the majority of developing stage are found on the mucosal surface. Development through L4 to L5 is

complete by 10-12 days from infection and this coincides with severe damage to the villi and erosion of the mucosa leading to villous atrophy. The ability of the intestine to exchange fluids and nutrients is grossly impaired, and with the onset of diarrhoea the lamb rapidly becomes dehyrated.

In the clinical signs : in severe infection , yellow-green diarrhoea is the most prominent clinical signs and can occur during the prepatent period. As dehydration proceeds, the affected animals become thirstly and in infected flocks the ewes continue to graze, apparently unaffected by the larval challenge, while their inappetent and diarhorroeic lambs congregate round drinking places. Concurent infection with pathogenic species of coccidiosis can exacerbate the severity of disease. At necropsy, the carcass has a dehydrate apperance and there is often an acute enteritis.

Gross pathological change may be limited to fluid mucoid contents in the upper small intestine with occasional hyperaemia of the mucasa of the duodenum with excess mucus on the surface. Local erosions may occur if villous atrophy is severe.

Diagnosis : the clinical signs appear during the prepatent period, therefore, faecal egg counts are of little value in early diagnosis , however, the informations of season, grazing history, periparturient period , clinical signs and postmortem examination may be aided the diagnosis of disease. Nematodirosis should be differentiated from coccidiosis in ewes and lambs.

Epidemiology : There are three most important features of the epidemiology of N. Battus.

1) the capasity of the free living stage, particularly the eggs contining the L3 , to survive on pasture, some for up to 2 years.

2) the large numbers of L3 are found in May and June in the pasture.

3) nematodirosis infection can be consideration a lamb-to-lamb disease with usually only one generation of parasites each year in the spring, although in some years an autumn generation of parasites may be seen.

Treatment : Several drugs are effective against Nematodirosis infections ; levamisole, an avermectin/ milbemycin or modern benzimidazole. The response to treatment is usually rapid and , if diarrhoea persists, coccidiosis should be considered as a complicating factor.

E) Trichostrogylosis :

Species of trichostrongylus are small light brownish- red , hair –like worms. Trichostrongylus is rarely a primary pathogen in temperate areas, but is ususally a component of parasitic gastroenteritis in ruminant.

Species :

Trichostrongylus axei occurs in abomasum or stomach in ruminants and equid animal.

T. colubriformis , T. vitrinus, T. longispicularis are most important species in small ruminants , and they are located in the small intestine.

Patnogenesis and clinical signs :

In the pathogenesis and clinical signs of T. axei infections , the extent of the lesions in the abomasum or stomach is dependent on the size of worm population. Small irregular areas showing diffuse congestion and white-grey , raised, circular lesions may be present in the abomasum or stomach. These lesions are about 1-2 cm diameter and have been termed plaques or ringworm lesions. In heavy infection, shallow ulcers may be seen. The changes induced in the gastric mucosa are similar to those of Ostertagiosis, with an increase in pH and increase permeability of the mucosa leading to an increase in plasma pepsinogen concentration and hypoalbuminaemia.

In the clinical signs,

---rapid weight loss and diarrhoea , inappetence, poor growth rates, soft faeces

In the pathogenesis and clinical signs of species occuring in the small intestine , following ingestion, the larvae penetrate the mucosa and developing worms are located in superficial channels sited just beneath the surface epithelium and paralled with the luminal surface, but above the lamina propia. When the subepithelial tunnels and areas containing the developing young worms rupture to liberate the worms about 10-12 days after infection, there is considerable haemorrhage and oedema and plasma proteins are lost into the lumen of the small intestine leading to hypoalbuminaemia and hypoproteinaemia. Many areas may superficially appear normal, but parasites are congreated within a small area, erosion of the mucosal surface is apparent with severe villous atrophy.

In the clinical signs,

--- in heavy infection , rapid weight loss and diarrhoea, often-dark colored.

--- heavy infections can induce osteoporosis and osteomalacia of the skeleton

----deaths can be high, particularly if animals are also malnourished

--- inappetence, poor growth rates, soft faeces

Diagnosis :

--- clinical signs, season, and post mortem examination

---faecal egg examination are a useful aid to diagnosis

Epidemiology :

The eggs and infected L_3 of Trichostrongylus spp. can survive under adverse environmental conditions. In temperate areas the L_3 survive the winter, occasionally in sufficient numbers to precipitate clinical disease in the spring, but the rising clinical problems are occured during summer and autumn. Hypobiosis plays an important part in the epidemiology, the seasonal occurence being similar to that Ostertagia spp.

Treatment :

This is described for ostertagiosis .

F) Cooperiosis :

The site of worms is small intestine. In temperature areas, members of the genus Cooperia usually play a secondary role in the pathogenesis of parasitic gastroenteritis of ruminants . however, in some areas, some species are responsible for severe enteritis in calves.

Species :

Cooperia oncophora , cattle, sheep, goat, deer

C. puctata , cattle (bankrupt worms) ---C. pectinata, cattle --- C. surnabada , cattle,sheep, goat --- C. curtacei , sheep,goat,deer

Pathogenesis and clinical signs :

Cooperia oncophora and C. curtacei are generally considered to be mild pathogens in calves and lambs respectively although they have been associated with inappetence and poor weight gains. In heavy infections, worms are more pathogenic since they penetrate the epithelial surface of the small intestine and cause a disruption similar to that of intestinal trichostrongylosis which leads to villous atrophy and a reduction in the area available for absorption .

There are loss of appetite, poor weight gain, diarrhoea, submandibular oedema.

Diagnosis : Faecal egg examination are a useful aid to diagnosis

Treatment : This is described for ostertagiosis and haemonchosis

G) Marshallagia marshalli :

This worm is found in the abomasum of small ruminnts . It is similar to ostertagia spp. The eggs are much larger and resemble those of Nematodirus battus.

The pathogenicity and clinical signs are resemble those of ostertagiosis and teladorsagiosis

H) Megistocirrus digitatus :

This blood-sucking abomasal worm is found in cattle. The pathogenesis and clinical signs are similar to that H. contortus in sheep .

I) Charbertia ovina :

Worms of this genus are usually in low numbers in the majority of sheep and goats. The adults are 1.5-2.0 cm in legnth and are the largest nematodes found in the colon of ruminants. They are white with a markedly truncated and enlarged anterior end due to the presence of the very large buccal capsule. The buccal capsule has no teeth. The anterior is curved slighly ventrally.

The egg is thin-shelled, smooth, medium-sized (90-100 X 45-55 µm), regular broad ellipse with slightly flattened poles. It contains 16-32 blastomeres.

Life cycle :

The lifecycle is direct. Eggs are passed in the faeces and hatch on the ground releasing the first-stagelarvai which moults to the second stage, and then to the infective third stage . The host is infected by ingestion of the larvae with the herbage. In the parasitic phase the L3 enter the mucosa of the small intestine and occasionally that of the caecum and colon ; after a week they moult, L4 emerge onto the mucosal surface and migrate to congregate in the caecum where deelopment to the L5 is completed about 25 days after infection. The young adults then travel to the colon. Thres is no migration stage in the body. The prepatent period is 40-50 days.

Pathogenesis and clinical signs :

Chabertia ovina is found in the caecum and colon of sheep, goat and cattles . The major pathogenic effect is caused by the L5 and by mature adults ; these attach to the mucosa of the colon via buccal capsules and then feed by ingesting large plugs of tissue, resulting in local haemorrhage and loss of protein through the damageed mucosa. The wall of the colon becomes oedematous, congested and thickened with small haemorrhages at the sites of worm attachment.

The moderate infections are usually asymptomatic. In severe infections, diarrhoea, which contain blood and mucus and in which worms may be found, is the most common clinical sign. The sheep becomes anaemic and hypoalbuminaemic and can suffer severe weight loss.

Diagnosis :

The diagnosis is performed by stool examination and necropsy . The pathogenesis occurs in the prepatent period, parasites are found in faeces with diarrhea . Passed parasites are easily recognized by their bell-shaped mouth capsules .

Treatment :

Anthelmintic therapy with broad – specturum anthelmintics (benzimidazole, levamisole, avermectins / milbemycins) is highly effective.

J) Oesophagostomum :

Worms of this genus are stout and whitish with a narrow cyclindrical buccal capsule and measue 1-2 cm in length. The body often slightly curved. Leaf crowns are present.

Oesophagostomum species :

O. columbianum and O. venulosum ----- sheep and goat

O. radiatum ------- cattle and water buffalo

O. dentatum ----- pig

Life cycle :

The preparasitic phase is typically trichostrongyloid. The egg hatches on the ground releasing L1, which moults to the second stage L2, and then to the infective L3. Infection is by ingestion of L3. There no migration stage in the body.

In first-time infected animals, the larvae (L3) enter the intestinal mucosa (1st tissue stage) stay for a while, change coat and come back to the intestinal surface as L4 . Some of the larvae migrate to the large intestine and mature parasites develop. However, some L4 may enter the mucosa of the large intestine (2 th tissue stage) . The prepatent period is about 1.5 months .

Pathogenesis and clinical signs :

In the colon and caecum, O. columbianum L3 migrate deep into the mucosa, provoking an inflammatory response with the formation of nodules, which are visible to the naked eye. On the reenfection, this response is more marked, the nodules reaching 2.0 cm in diameter and containing greenish eosinophilic pus (or inflammation) and L4. When the L4 emerge there may be ulceration of the mucosa. Diarrhoea occurs coincident with emergence about a week after primary infection and from several months to a year after reinfection. In heavy infections, there may be ulcerative colitis and the disease runs a chronic debilitating course with effects on the production of wool and mutton. The nodules in the gut wall also render the intestines useless for processing as sausage and sugical suture materials.

In the acute infections, severe dark-green fluid diarrhoea is the main clinical signs and there is usually a rapid loss of weight, emaciation, prostration and death in young animals. In chronic infections, there is inaapetence and emaciation with intermitten diarrhoea and anaemia .

Diagnosis :

This is based on clinical signs and posrmortem examination. Since the acute disease occurs within the prepatent period, eggs of Oesophagostomum spp. are not usually present in the faeces.

Treatment :

Anthelmintic therapy with broad – specturum anthelmintics (benzimidazole, levamisole, avermectins / milbemycins) is highly effective.

The treatment and control of parasitic gastrointestinal enteritis (PGE) in small ruminants :

--- treatment with any of the benzimidazoles, levamisole , avermectins/milbemycin or the new monepantel and dual-active derquantel-abamectin will remove adult worms and developing stages.

--- the occasional outbreaks type 2 teladorsagiosis in young - adult sheep (around 1 year old) in the spring may be treated with the same anthelmintics. Unlike O.ostertagi in calves, the arrested stages of common sheep nematodes are susceptible to the benzimidazoles and levamisole.

--- the periparturient rise (PPR) in faecal egg numbers is very marked in ewes and is the most important cause of pastue contamination with nematode eggs in the spring

--- parasitic gastroenteritis (PGE) in shep is generally associated with a variety of nematode genera with differing epidemiology characteristics

---most sheep graze throughout their lives so that pasture contamination with nematode eggs and the intake of infected larvae is almost continuous

---anthelmintic resistance is now widespread throughout many sheep-rearing areas of the world

<u>Anthelmintic usage :</u>

1) use anthelmintic sparingly : this will reduce the selection pressure for future development of drug resistance.Effective monitoring of faecal egg examination is integral to this approach

2) use the appropriate anthelmintic : in some situations it may be possible to target treatment by using a narrow-specturum drug. For example, closantel against a specific infection dominated by Haemonchus or a benzimidazole against Nematodirus .

3) use effective quarantine procedures, it is essential to treat all sheep and goats , and monepantel and dual –active containig of derquantel- abamectin provide new drug choices

for this treatment . Treated animals should be held off pasture for 24-48 hours to allow any worm eggs in the alimentary tract to pass out in the faeces

4) adult sheep tupping time; at this time most ewes in good body condition will be carrying low burdens as they will have a strong acquired immunity. Treatment at this period can significantly select for anthelmintic resistance

5) adult sheep at lambing time ; the most important source of infection for the lamb crop is undoubtedly the increase the nematode eggs in ewe faeces during PPR and prophylaxis will only be efficient if this is kept to a minimum. Effective anthelmintic therapy o fewes during the fourth month of pregnancy should eliminate out of worm burden present at this time, including arrested stages , and this treatment often results in improved general body condition.

Threatment around lambing or turnout, and again 4-5 weeks later, will significantly reduce the ewe contribution to pasture contamination.

A suitable drug is applied to the ewes one week before they turnout to the pasture, and they may require further treatment in about 4-5 weeks.

Rumen boluses designed for the slow release of anthelmintics over a prolonged period are available for sheep and are recommended for use in ewes during the periparturient period to eliminate worm egg output.

6) treatment of lambs : in general, lambs should be treated at weaning, and the treatment should be repeated until autumn.

ASCARIOSIS

Ascaridid roundworm are among the most common parasites in the small intestine of several species of animals, especially pig, cattle, horse, dog,cat and poultry . The ascaridoids are among the largest nematodes and larval and adult stages being of veterinary importance. While the adults in the intestine may cause unthriftiness in young animals, and occasional obstruction, an important feature of the group is the pathological consequence of the migratory behaviour of the larval stage.

They are large, whiter or cream, opaque worms that inhabit the small intestine. There is no buccal capsule the mouth consisting simply of a small opening surrounded by three large conspicuous lips. The male possess two spicules but do not a bursa. The common mode of infection is by ingestion of the thick-shelled egg containing the L_3 . The life cycle also involve

prenatal and galaktogen route , and some species can use paratenic hosts (particularly rodents)in their life cycle.

Genera of veterinary interest include Ascaris, Toxascaris, Toxocara, Parascaris, Ascaridia and Heterakis.

Ascaris : Large, stout, white worms around 15-40 cm in length.

Ascaris suum : pig, bear, rarely sheep, cattle and human

Ascaris lumbricoides : human

Ascaris suum : it is largest parasite of pigs, the rigid females are up to 40.0 cm long and 5 mm in witdth and male up to 25 cm in length. The eggs are ovoid and yellowish-brown, with a thick shell, the outer layer of which is irregularly mamillated. They measure 50-75 by 40-55 µm and the contents consist of granules and unsegmented cells.

Life cycle : the life cycle is direct. Though the preparasitic moults occur by about 3 weeks after the egg is passed, even in the optimal temperature range of 22-26°C. After ingestion, the larvated egg hatches in the small intestine, the L3 larva penetrates the intestinal mucosa and then travels to the liver . The larva then passes in the bloodstream to the lungs and thence to the small intestine via the bronchi, trachea and pharynx. In the intestine the final moult occurs and the adult worms inhabit the lumen of the small intestine. The prepatent period is between 7-9 weeks and female worm is capable of producing more than 200,000 egg per day. Longevity is around 6-9 months. Earthworms are paratenic host.

Pathogenesis and clinical signs : the adult worms in the intestine cause little apparent damage to the mucosa, but occasionally, if large numbers are present, there may be obstructive, and rarely a worm may migrate into the bile duct causing obstructive jaundice and carcass condemnation.

The migrating larval stages in large numbers may cause numerous small haemorrhages, emphysema and a transient pneumonia. In the liver, the migratinglarvae can cause ‘ milk spot ‘ or ‘ white spot ’ , which appears as cloudy whitish spots of up to 1.0 cm in diameter on the surface of the liver.

The main effect of the adult worms in pigs is to cause production loss interms of diminished weight gain. Heavy infections may increase the susceptibility of young pigs to other bacterial and viral pathogens.

Diagnosis : Diagnosis is based on clinical signs and on the presence in faeces of the eggs.

Treatment : the intestinal stages are susceptible to most of the anthelmintics in current use in pigs, and the majority of these, such as the benzimidazoles, are given in the feed over several days.

Parascaris : This very large, rigid, stout, whitish nematode, up to 40 cm in length, is found in the small intestine of equides.

Parascaris equorum : this species is found in the equides and is located in the small intestine. Males measure 15-25 cm and females up to 40-50 cm in length. This worm can not be confuse with any other intestinal parasite of equines. The medium – sized egg of P.equorum is almost spherical (85-100 X 80-90 µm), brownish and thick-shelled with an outer pitted albuminous coat and the contents consist of granules and unsegmented cells.

Life cycle : the life is direct and migratory, involving hepato-pulmonary route. Eggs are passed in the faeces and can reach the infective stage containing the L3 in as 10-14 days, although development may be delayed at low temperatures. After ngestion and hatching the larvae penetrate the intestinal wall and the larvae have reached the liver. By 2 weeks they have arrived in the lungs where they migrate up the bronchi and trachea, are swallowed and return to the small intestine. Although the presence of L4 in the intestine is around the 14th days, the increase in the number of larvae is around the 21 days after infection. After infection , adult parasites develop around 65-70 days in the small intestine. The prepatent period is about 72-115 days.

Patogenesis and clinical signs : the migration of larvae induce pathological lesions in liver and lungs. In the liver, the migrating larvae cause 1 cm or smaller diameter, whitish-red colored foci or nodules (milk spot) and these larvae cause focal haemorrhages and eosinophilic tracts that resolve, leaving whitish areas of fibrosis. The liver tissue heals within 4 weeks and these nodules diasappear. During in this period, there is no change in liver enzyme value, and there is no clinical signs in horses.

The migrating larvae in the lung parencyhma , alveols, bronchi and brochioles cause haemorrhage and pulmoner eosinophili, and as a result of these pathological changes in lungs , the coughing and serous and serous-mukoid runny nose are seen in the horses. The duration of cough is individual and can last from 5-35 days. The runny nose starts before coughing and is bilateral. The runny nose continue 7-30 days. In this period, the horses may be depressed and have poor coordination . Although the respiratory value are normal, there may be deaths due to alveolar collapse in foals.

In the later days of the parascaris infection, the patent period begins with the presence immature and then adult worms in the intestine. The intestine becomes mechanically and toxically irratated by worms. In the intestinal part, which begins to change its textural structure, first enlargement and then ischemia occur. Diarrhoea and constipation alternate and the diarrhoea is gassy, foul-smelling while the surface of hard stool is covered with mucus. There is weight loss which is particularly evident in foals, and growth retardation in

foals reaches 50% and foals have weakness and reluctance to move . During this period, the number of fecal eggs is high. Intestinal obstruction and pain can be seen in foals, and such horses are usually 3-5 months old , and such cases usually occur in autumn .

Epidemiology :

In epidemiology, the presence, accumulation and continuity of eggs in pastures and paddocks are important. Adult P. equorum live for several months in the horses. The eggs (up to 10 %) can survive for 1.5 years under environmental conditions. The age of the horses is important in epidemiology, and if no medicine is given to the foals in the first year, the probability of getting at least two ascarid infections in this period is very high. The faecal EPG value is always high in foals (4-4.5 months old). In the farms, the 80 % of the amount of feces egg laying in horses is provided by 2% of the horses, and these horses are 5-8 months old animals. Although the number of parasites is low in old horses (animals aged 1 and over), the EPG values of these animals are high. In the weaning period, the infection level is always high in horses.

Diagnosis :

In the farms, there is no practical diagnostic method for the migration period of the larvae. In cases of occlusion and pain, information about the drugs used in horses is important, and imaging methods are used successfully in the diagnosis. In the diagnosis of patent infection, the flotation method is used. The EPG and FECRT values are important during treatment and evaluation of treatment results.

Treatment :

The first drug application in the farms should be done in the 2nd month in the foals, and the second drug application should be done in 4-4.5 months. In horses, after 6 months of age and after weaning, treatment should be used according to feacal examination results and EPG values. The horses given medicine are not taken to pasture for at least 4-5 days. Drug use in horses before 2 months of age only contributes to the development of drug resistance. In the horses, after 6 months of age and after weaning, drug use without faecal examination and without taking into account EPG values leads to drug resistance. If there are fluctuations in EPG and FECRT values in farm records, drug resistance has started to develop. It is not desired that the FECRT values of the active ingredients be below 97% after the treatment.

ivermectin 0.2mg/kg , moxidectin 0.2mg/kg , fenbendazole 7.5mg/kg , mebendazole 10mg/kg, pyrantel embonate 6.6mg/kg, oxibendazole 10mg/kg , febantel 6mg/kg.

Benzimidazoles can be preferred in order not to cause obstruction and pain, especially in horses aged 1 - 1.5 years.

Toxocara :

Toxocara canis , T. cati (syn. T. mystax) and T. vitulorum (syn. Neoascaris vitulorum) species in the genus Toxocara are important for veterinary medicine.

Toxocara canis :

The final hosts are dogs and wild canids, and ascarids are found in the small intestines of hosts . They are large nematodes, males 10-12 cm in length , females 12-18 cm lin length and cream colored parasites. The cervical alae at the anterior end of the parasites narrows backward and the anterior end of the ascarids is in the lancet view. There is a bulb posterior to the esophagus. The medium - size egg is dark brown and subglabular, witha htick rough pitted shell. The granular unsegmented contents are very dark and normally fill the whole of the shell, and eggs measure 90 by 75 µm. This ascarite is common in both owned and stray dogs in Turkey (4.1-44.5 %).

Life cycle :

Toxocara canis has a complex biology in which multiple factors influence each other. Therefore, drug use and prevention programs also vary. Infective larvae (L3) develop within 9-15 days in 25-30 ° C and sufficient humidity (85-95 %) environment in the eggs which are excreted with feces from the final hosts. This development takes up to 3-4 weeks depending on the climatic conditions. Eggs can survive in the environment for a maximum of 1-1.5 years.

The final hosts are become infected by ingesting eggs with larvae. After ingestion and hatching in the small intestine, the larvae travel by the blood stream via the liver to the lungs. The larvae then return via trachea to the intestine where the final two moults take places. This form of ascaridoid migration occurs regularly only dogs of upto about 2-3 months old. Larvae 3 penetrate the lung alveolar wall, and the larvae follow two migration routes depending on the age of the host the immune system that develops accordingly, and the density of the larvae. First migrating way is hepato-trecheal route (occurs in 1-1.5 months old dogs) , second migrating way is somatik migrate route (dogs older than 2-3 months).

In the first migrating route, L3 comes to the pharynx via alveolar-bronchiole-trachea and are swalled, and L3 comes to the stomach and small intestine. L3 becomes L4 in the migration path between the lungs and stomach (within 7-9 days) and the adult parasite develops in the intestine. The prepatent period is 32-39 days.

In the second migrating route, The vast majority of L3 re-enters the blood stream from the lungs, and L3 spread to internal organs and tissues (liver, kidney, heart, muscular tissue, brain e.t.c.) . This migration is called **somatic migration**.

These larvae in organs and tissues die over time (around 1-2 years) in male dogs. If an infected female dog becomes pregnant, these somatic larvae become active in the last quarter of pregnancy. The motile larvae (L3) pass via the placenta to the liver, lungs, kidney , heart and muscles of the fetuses 2-3 days before birth. This form of infection is called **prenatal (intrauterine) transmission**. Ascarides begin to be found in the intestine from the 3rd day after birth in puppies born infected with prenatal transmission, mature ascarides have developed after the 11th day. Prepatent perion is 14-21 days in prenatal transmission. however, in prenatal infections, the prepatent period may be extended up to 45 days for some reasons.

If pregnant dogs ingest larval eggs from the environment in the last quarter of pregnancy, on the day of birth, or in the first 2-5 days of lactation, the larvae(L3) come to the mammary glands before and after birth. The these larvae (L3) in mammary tissue are passed to suckling pups during lactation. The larvae, which are passed to the pups with milk, can develop directly in the digestive system, or they can migrate to the small intestines by hepato-tracheal migration. The prepatent period is around 27-35 days . This form of infection is called **galactogen (transmammary infection)** . However, the galactogen pathway does not play an important role in the biology of Toxocara canis. Larval emergence with milk begins on the 4th day of lactation and generally continues up to 28 days. The rate of larvae passing with milk is % 0.06. Some of the larvae (L3) that come to the pups with milk can migrate to the somatic, and in pups, from time to time they come to the intestine and develop into mature ascarids.

Causes of patent infections in female dogs after birth and the appearance of eggs in feces ;

1) the L4 are excreted with feces of infected puppies and and these larvae (L4) are ingested by female dogs nursing their pups

2) the weakening of immunity in females giving birth and maturation of somatic larvae in the intestine

3) coprahpagia

In such cases, the prepatent period is 9-12 days.

In particular rodents, chickens, sheep, rabbits and pigs are paratenic hosts in biology. The larvae (L3) can survive up to 2 years in the muscles and various organs of rodents. The larvae (L3) are released in the small intestines of dogs that eat their paratenic hosts, and the larvae make hepato-tracheal migration and the mature T. canis develops in the intestines. The prepatent period is 34-38 days.

Pathogenesis and clinical signs :

Pneumonia and vascular occlusion occur in prenatally infected puppies due to larval migration in the lung. In severe infections, the alveoli and bronchiolar fill with a viscous substance composed of mucus, eosinophilia, epithelial cells and larvae. When haemorrhagae and parachymal degeneration are added to this pathology , the offspring may die in 3-5 days. However, most deaths in prenatally infected puppies occur on 22-49 days (3-7 weeks). The cause of death is excessive degeneration of the intestinal tissue and its thinning and tearing due to hemorrhage, obstruction of the bile duct by ascarids, or rupture of the liver capsule by ascarids.

Milk foci developing in the liver disappear after the 3rd week of infection. Eosinophilia increases with the presence of L4 in the intestine (around 14 days) and decreases to its normal level around 42 days. The anemia is severe in the 6-8 weeks following the infection and especially in reinfections, bloody diarrhea is observed intensely due to resistance to newly acquired larvae. In sick dogs, wasting, anemia, abdominal swelling, matting of the coat, diarrhea with mucus, and constipation are evident. In sick dogs, wasting, anemia, abdominal swelling, matting of the coat, diarrhea with mucus, and constipation are evident. When dogs stand, their hind legs are apart and when they walk, they separate their hind legs. The ascarides in the digestive tract (L4, immatures and matures) may be excreted with vomiting and diarrhea, in which case dogs have smell bad in their feces.

Epidemiology :

The prevalence and persistence of toxocariosis in dogs depend on somatic larvae and prenatal transmission route. The patent infections caused by egg ingestion are usually seen in puppies 3-4 weeks old, and sometimes , ıt is also seen in older dogs with weakened immunity. The reinfections are more than is believed , and the recurring infections occur in 10 out of every 100 dogs within 1 year. The older dogs become infected with a small number of larvae (25-100 larvae) and patent infections may develop .

Toxocara canis eggs are highly resistant to adverse environmental conditions. The eggs can survive even at a depth of 5 cm from the soil surface. Having more than one dog in a residence or in a confined space increases the risk of infection.

Diagnosis :

Diagnosis in deceased puppies is made by necropsy. In puppies, in the prepatent period, ascarides excreted with vomiting, abdominal distension, weakness and diarrhea help the diagnosis. Centrifugal flotation method gives the best results in feacal examination. The density of the solution used must be at least 1.2 .

Treatment :

In urban or rural dogs, patent infection of Toxocara canis in puppies and somatic Toxocara larvae (L3) in adult dogs can always be found. The treatment is done to eliminate the health problems that develop in dogs, to prevent environmental contamination caused by patent infection and to reduce the risk of zoonosis. In Turkey, the prevalence of Toxocara canis in owned dogs is not less than 20%, indicating that the drug administration is not conscious in dogs.

The main causes of repeated infection in dogs are:

1) undiagnosed treatment

2) giving medication at least 2-4 times a year

3) to give another active substance instead of the active substance that should be used in different periods of the parasite

A result close to one hundred percent is obtained by choosing the appropriate active ingredient and determining the timing correctly , and It is desirable that the active substance be at least 97% effective. The duration and timing of treatment vary according to the pharmacokinetics of the selected active substance. The postpartum treatment period should last 2-3 months at the most.

For puppies infected born or considered to be infected and female dogs, old generation drugs are given at the 2nd, 4th and 6th weeks following birth, and optional drug use can be made at 8 weeks as a precautionary measure. For the same purpose, if avermectins or milbemycin oxime are to be used, the drugs should be used at monthly intervals. Selamectin and milbemycin oxime are administered in the 2nd week after birth and then the repeat doses are given monthly, and after the 12th week, the drug should not be used unless necessary.

Selamectin, moxidectin, milbemycin oxime, and doramectin can be used to prevent prenatal transmission and postnatal patent infections in dogs. In the pregnant dogs, patent infections in puppies and mother dogs can be prevented by using selamectin (6mg/kg--used spot on) on the 40th day of pregnancy and 10 days before birth, on the 10th and 40th days after giving birth. Administration of moxidectin (1 mg/kg) on the 40th and 55th days of pregnancy can prevent prenatal infections by 98-100%.

For patent ascarid infections in dogs ;

Fenbendazole 50 mg/kg X 3 days , flubendazole 22 mg / kgX 3 days, mebendazole 22 mg / kg X 3-5 days , selamectin 6 mg/kg (spot-on) , moxidectin 0.2 mg / kg, emodepsin 0.45 mg / kg (oral suspantion) , milbemycin oxime 0.5 mg / kg , nitroscanate 50 mg / kg.

Although piperazine salts are only effective on mature ascarids, the efficiency level is only 85 - 90% and is not always preferred today.

To protect the health of dogs, reduce egg contamination for the environment and prevent the risk of zoonotic infections, drugs must be selected correctly, used within a schedule and administered at the exact dose.

Toxocara cati (syn. T. mystax) :

Toxocara cati is found in domestic and wild cats and settles in the small intestine. Typical of superfamily, Toxocara cati is a large white / cream - coloured worm (up to 10 cm in length, often occuring as a mixed infection with the other ascarids of cats , such as Toxascaris leonina. Males are 3-7 cm and females 4-10 cm in length.

The tail of the male has a terminal narrow appendage. Differantiation is readily made between T. mystax and Toxascaris leonina on gross examination or with a hand lens, when the cervical alae of the former are seen to have an arrowed form, with the posterior margins almost at a right angle to the body, whereas those of Toxascaris taoer gradually into the body. The male, like that of T. canis has a small finger-like process at the tip of the tail. The egg is subglobular with a thick rough pitted shell. The granular unsegmented contents are dark brown to black in colour and usually occupy the whole volume of the shell. Eggs measure 65 by75 µm and are characteristic in cat faeces.

Life cycle :

In the external environment, the conditions required for egg of T. cati to development are the same as those of T. canis eggs. The enfective stage larvae (L3) develop in the egg in 20-28 days, and T. cati has three different ways of infection in its biology in the cats.

1) route of infection with eggs ; following ingestion of eggs containimg an infective third-stage larva, the larvae enter the stomach wall and then migrate via the liver, lungs and trachea back to the stomach and small intestine, and 35 days after infection, parasites begin to increasing in the intestine . All of the larvae (L3) taken into the body with the egg are not simultaneously hepato-traheal migrating, some of the larvae can be found in other organs for 10-20 days and then come to the digestive system via hepato-tracheal migration. The prepatent period is 54-56 days in cats.

2) galactogen infection route ; female cats must be infected with eggs in the last quarter of pregnancy in order for the larvae (L3) to be transmitted from female cats to kittens by galactogen. In the cats, the larvae (L3) taken before mating or in early pregnancy do not pass into the mammary tissue and mammary glands. The infected female cats excrete lrvae

(L3) with their milk for 2-3 weeks, starting from 3 days at birth. The development of the larvae taken with milk in the kittens almost takes place in the digestive system. The prepatent perion is 44-57 days.

3) infection by ingestion of paratenic hosts ; as well as rodents as paratenic hosts , L3 may be found in the tissues of the earthworms , insects and chickens fed infective eggs. The development of the larvae taken from paratenic hosts takes place in the digestive system. The prepatent perion is around 50 days**. The prenatal infection through the placenta does not occur** .

In female cats infected with eggs in late pregnancy, adults ascarids develop in the postnatal period and the eggs appear in the feces of these cats 42-49 days after birth. The larvae taken in the last periods of pregnancy do not accumulate in the tissues of pregnant cats and do not pass to the kittens in the next pregnancy , and these larvae do not cause patent infections in female cats in their next pregnancy. In cats, Toxocara cati lives up to 6 months.

Pathogenesis and clinical signs :

Similar to Toxocara canis infection in dogs, but with a milder infection in cats. The infected cats have unthriftiness, vomiting, diarrhoea, abdominal swelling, matting and tangling of the coat, emaciation, weakness, dehydration, and anemia. The diarrhea is severe during the presence of immature ascarids in the intestine, smelly and mucous, often with ascarides .

Epidemiology :

Toxocariasis in cats is spread by eggs and vertical transmission. The stray cats are the main cause of egg contamination of the environment. Since cats live in narrow spaces, contamination with eggs is quite common. The paratenic hosts are an important source of infection for cats.

Diagnosis :

Diagnosis in deceased kittens and cats is made by necropsy. In kittens , in the prepatent period, ascarides excreted with vomiting and diarrhoea, abdominal distension, weakness and diarrhea help the diagnosis. Centrifugal flotation method gives the best results in feacal examination. The density of the solution used must be at least 1.2 .

Treatment :

The treatment is done to eliminate the health problems that develop in cats, to prevent environmental contamination caused by patent infection and to reduce the risk of zoonosis. In the pregnant cats against galactogen infection, emodepsid can be used in a single dose of 3mg/kg 3-9 days before whelping (kittening) , and this practice also prevents 100% patent infections in female cats in postnatal period.

Drug applications to be made at the 3rd, 5th and 7th weeks following birth and at the 9th week as a precautionary measure to kittens that are or are considered to be galactogen-infected ensure that the parasites are expelled before they mature. With the feacal examinations to be made later, the treatment can be extended up to the 15th week if necessary. The cats giving whelping are also treated with the same medication programe. If selamectin and emodepsid are used, the duration of drug administration should be 4 weeks apart.

For patent ascarid infections in cats ;

Emodepside 3mg/kg , selamectin 6 mg / kg , fenbendazole 50 mg / kg X 3 days , mebendazole 11 mg / kg X 3 days , flubendazole 22 mg / kg X 3 days

Control :

In cats that are kept indoors but are also found outside from time to time, faecal examination should be done once a month and medication should be applied according to the result.

Toxocara vitulorum (syn. Neoascaris vitulorum) :

Toxocara vitulorum is found in cattle, buffalo and zebu , rarely sheep and goat in the small intestine. This species is a very large and whittish nematode. The adult male is upto 25 cm and the female 30 cm in length. The medium sized- egg of T. vitulorum is subglobular, with a thick finelly pitted albuminous shell, and is almost colourless (75 -95 X 60-74 µm). The egg is unsegmented and the granular contents frequently only occupy part of the internal volume.

Life cycle :

The most important source of infection ia the milk of the dam (galactogen infection), in which larvae are present for up to 3-4 weeks after parturition . There is no tissue migration in the calf following milk-borne infectionand the prepatent period is 14-42 days. The ingestion of larvaed eggs by calves over 4-6 months of age seldom results in patency, the larvae migrating to various tissue where they remain dormant; in female animals resumption of development in late pregnancy allows further tramsmammary transmission.

Pathogenesis and clinical signs :

The main effects of T. vitulorum infection appear to be caused by the adult worms in the intestine of calves up to 6 -8 months old . There is inflammation, ulceration, necrosis and thinning of the muscle layer in the intestinal tissue. The infected animals have loss of appetite, anorexia, indigestion, greasy-smelling stools, bloating, dehydration and coughing. animal breath has an odor similar to the odor of butyric acid (similar to the odour of garlic). The large numbers of ascarids cause intestinal obstruction and rupture and the animals die from peritonitis .

Epidemiology :

The most importants feature is the reservoir of larvae in the tissues of the cows, with subsequent milk-borne transmission ensuring that calves are exposed to infection from the first day of life. The majority of patent infections occurs in calves of lees than 6 monhts of age.

Diagnosis :

In some instances heavily infected calves may exhale an acetone- like odour . The subgalbular eggs, with thick pitted shells, are characteristic in bovine faeces.

Treatment :

In the calves, it is necessary to administer medication on 10-16 days. After the treatment, feces and excreted ascarides are removed from the environment. The faecal examination is performed on the 7th and 14th days after the first drug administration and the treatment is repeated if necessary.

For patent toxocariosis in ruminats ;

Pyrantel 8 mg / kg , levamisole 7.5 mg / kg , fenbendazole 7.5 mg / kg , ivermectin 0.2 mg / kg . Doramectin and moxidectin are effective, but piperazine salts , thiabendazole and febantel are not effective . In the cattle and calves , there is no effectiveness of drug applications made every month and between 6 and 8 months or irregularly.

Toxascaris leonina :

Toxascaris leonina is found in dog, cat and fox in the small intestine. Male measure up to 7 cm long and females up to 10 cm and their anterior bodies are curved dorsad. The cervical alae are slender and arrowilike, tapering posteriorly. The egg is slightly ovoid, with a smooth thick almost colourless shell. The yellowis-brown granular unsegmented contents fill only part of the shell. Eggs measures about 75-85 by 60-70 µm and are characteristic in and cat faeces.

Life cycle :

The life cycle is direct. The infective larvae (L3) develop in 4-6 days at 17 - 37 °C degrees in the eggs passed with the feces of the dogs and cats. Following and hatching , larvae enter the wall of small intestine and remain for about 2 weeks. No migration of larvae occurs , as with others ascarids species. Adult Toxascaris leonina appear from about 6 weeks post infection and lie in the lumen of the intestine. The prepatent period is 10-11 weeks.

The rodents take place in biology as paratenic hosts . **The prenatal and lactogenic infection through the placenta do not occur** .

Pathogenesis and clinical signs:

Infection with toxascariosis is unlikely to other ascarid infections of dog and cat (T. canis and T.cati). In puppies and kittens less than 2 months of age the infection is usually absent as there is no prental and lactogenic transmission. Unthriftiness, abdominal swelling and diarrhoea are clinical signs .

Diagnosis :

The characteristic ovoid smooth – shelled eggs are easily recognised in the faeces.

Treatment :

The active ingredients used in the treatment of toxocariosis in dogs and cats are also fully effective against this parasite.

Visceral Larva Migrans :

Though this term was originally applied to invasion of the visceral tissue of an animal by parasites whose natural hosts were other animals, it has now, in common usage, come to represent this type of invasion in humans alone and, in particular, by the larvae (L3) of T. canis , althougth the larval stage (L3) of T. cati can be implicated.

The global condition occurs most commonly in children, who have had close contact with household pets or who have frequently areas such as public parks where there is contamination of the ground by infected dog faeces.

In many cases, larval invasion is limited to the liver, and may give rise to hepatomegaly and eosinophilia, but on some occasions a larva escape into the general circulation and arrives in another organs, the most frequently noted being the eye. Here, a granuloma forms around the larva on the retina, often resembling a retinoblastoma. Only in rare cases does the granuloma involve the optic disc, with total loss of vision, and most reports are of partial impairment of vision, with endophthalmitis or granulomatous retinitis. Such case are currently treated using laser therapy. In afew cases of epilepsy, T.canis infection has been identified serological.

Ascaridia :

These large worms are stout and densely white, the female measuring up to 12 cm in length. The narrow lateral alae are often not apparent grossly.

Ascaridia galli is found chicken, turkey, goose, duck, guinea fowl in the small intetine

Ascaridia dissimilis is found turkey in the small intestine

Ascaridia columbae is found wild and domestic pigeon in the small intestine

Ascaridia galli :

These large worms are stout and densely white; male worms are 5-7.6 cm and female 7.2-11.6 cm long . Ascaridia is by far the largest nematode of poultry. The medium – size pale-brown egg is distincly oval, with barbel-shape side walls and they are ınsegmented when laid. They measue about 73-92 by 43-57 µm. The smooth thick shell has htree layers, the middle one being more prominent. Eggs cannot easily be distinguished from those of the other common poultry ascaridoid, Heterakis .

Ascaridia columbae : The worms are stout and densely white, males are 16-70 mm and females 20-95mm in length.

Ascaridia dissimilis : The worms are stout and densely white. Males measure 35-50 mm and females 50-75 mm in length.

Life cycle :

The development of the infective larva (L3) in the egg takes place in 9-21 days depending on the environmental temperature and humidity. Ingestion and hatching , the parasitic phase is non-migratory, consisting of a transient histotrophic phase in the intestinal mucosa after which the adult parasites inhabit the lumen of the intestine. The eggs are sometimes ingested by eartworms, which may act as paratenic hosts. Eggs can remain viable for several months under moist cool conditions but are killed a dry hot enviroment. The prepatent period is 5-8 weeks, but the prepatent period varies depending on the age of the bird and reaches 9 weeks for older birds at 3 months. The ascarids live for about 1 year.

Pathogenesis and clinical signs :

Ascaridia galli is more pathogenic in 1-3 month old birds. The pathogenic effects of parasites occur in the prepatent period. During this period, destruction of the glandular epithelium, atrophy and adhesion of the villi occur in the intestines of infected birds. Catarrhal - hemorrhagic enteritis, anemia, diarrhoea, loss of appetite, weight loss and decreased egg production are evident in infected birds. Deficiency of protein, vit A, B, B6 and B12 in the diet of poultry increases the pathogenicity caused by parasites and clinical findings become more severe. Sometimes , egg production in poultry may come to a halt.

Ascaridia galli causes a decrease in the effectiveness of the Newcastle vaccine in poultry, and causes an increase in the pathology of some bacterial agents (E. coli, P. multicida). Ascaridia galli contributes to the transport of Salmonella agents.

Ascaridia dissimilis causes moderate or severe infections in birds, usually without clinical signs .

Ascaridia columbae is more pathogenic in young pigeons . The pigeons are reluctant to fly and are usually drowsy, fatigued, weight loss and fluffy feathers.

Epidemiology :

Although earthworms are thought to be paratenic hosts, the L3 do not remain in these animals for long, and most of the larvae are excreted in the worms within 24 hours. Ascaridia columbae is more common in pigeons in winter. Parasites are always present in litter rearing types, organic and roaming chicken farms. Putting more chickens per square meter in farms increases the risk of infection.

Diagnosis :

In the prepatent period, larvae will be found in the intestinal contents and in scapings of the mucosa. The eggs will be found in faeces. In the microscopical examination, Ascaridia and Heterakis eggs are difficult to distinguish .

Treatment :

When the presence of ascaridia is detected on farms, both larval and adult parasites are present in poultry. In the histotropic period, the effectiveness of drugs is almost absent.

Fenbendazole 60 ppmX3 days (chicken) and 30 ppmX 3 days (turkey) , flubendazole 30 ppm X 7 days , mebendazole 60 ppm X 7 days, cambendazole 70mg/kg (with feed) , levamisole 300 ppm X 3 days

Heterakis :

These are small to medium whitish up tp 1.5 cm long, with elongated pointed tails . Gross exsamination readily indicates the genus, but for specific identification microscopic examination is necessary to detemine the shape of the eosophagus (in Heterakis the eosophagus has a large posterior bulb) and size and shape of spicules. The species of Heterakis settle in the caecum.

<u>Heterakis species :</u>

Heterakis gallinarum is found in chicken, turkey, pigeon, duck, fowl, goose ,pheasant e.t.c

Heterakis isolonche is found in chicken, fowls e.t.c

Heterakis dispar is found in chicken, duck, goose e.t.c

Heterakis gallinarum :

The male is 7-13 mm and female 8-15 mm long. On the lateral parts of the body, there are well-developed alae that expand backwards. The spicules are unequal in length, the left

(about 0.7 mm) has broad alae and right is slender and longer (about 2mm). Eggs are thick-shelled, 66-80 by 35-47 µm and are embryonated when passed.

Heterakis isolonche :

The male is 6-13 mm and female 9-12 mm long. The spicules are long and of no equal length.

Heterakis dispar :

The male is 11-18 mm and female 16-23 mm long. The spicules are short and equal in length.

Life cyle :

The direct life cycle is similar to that of Ascaris spp. the infective stage larva (L3) develops in egg in 7-12 days environment temperatures at 18-29° C .After ingestion and hacthing, the larvae go through a histotropic period in the wall of caecum, and then the larvae return to caecum surface and adult parasites develop. Although prepatent period is 4-5 weeks , he prepatent period can be up to 8 weeks depending on the resistance of the poultry and the number of larvae ingested. The earthworms are paratenic host in the biology. The longevity of Heterakis gallinarum is about 1 year.

Pathogenesi and clinical signs :

Infections are rarely fatal, however it is a common parasite of poultry. The severity of the infection is determined by the age of the poultry and the immune strength , and the presence or absence of Histomonas in animals also affects the severity of infection. If only Heterakis is present in animals, the thickening of the caecal mucosa and petechial hemorrhages develop, in birds catarrhal or hemorrhagic diarrhea occurs.

In the poultry, hepato-enteritis agent Histomonas meleagridis is transmitted to poultry by the larvae and eggs of Heterakis. The earthworms harboring the L3 of Heterakis also play a role in this transmission. As a result, the birds become infected with both parasites , and in the infected birds with both parasites have higher mortality , and deaths begin to occur within 2 weeks .

Heterakis isolonche is a pathogenic species and causes nodular typhitis in poultry.The adult ascarids are found in nodules in the cecum , and these nodules also develop on the peritoneal surface of the cecum.

Epidemiology :

Heterakis eggs can survive for months in the external environment at temperatures between 4 to 33° C.

Diagnosis :

The eggs will be found in faeces. In the microscopical examination, Ascaridia and Heterakis eggs are difficult to distinguish . In Heterakis eggs, the substance in the egg can fill the whole egg, there may be no cavity on the edges.

Treatment :

In the treatment , pyrantel and piperazine are not effective pyrantel and piperazine are not effective .

for larvae, in turkeys , at 5 weeks of age ; albendazole 10 mg / kgX 14 days , fenbendazole 10 mg/kg X 14 days

for adult ascarids, at all birds ; albendazole 20 mg / kg , febendazole 100mg / kg , flubendazole 30 ppm X 7 days , mebendazole 60 ppm X 7 days

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HOOKWORM DISEASE / ANCYLOSTOMOSIS

Adult hookworms are parasites of the small intestine, and they can be found in most mammals except equines. Hookworms are common parasites of dogs, cats and humans around the world , and they causing serious pathogenicity and disease. The hookworms have an important place in parasitic zoonotic diseases.

Thre hookworms are reddish-grey colour dependig on whether the worm has fed, and are readily recognised on the basis on size. The anterior extremity is usually bent dorsally. The hooworms have a weel-developed buccal capsule, which is devoid of leaf crowns, but is armed with teeth od chitinous cutting plates on its ventral.

Hookworms are found in the small intestine of their final host .

<u>Hookworm species :</u>

Ancylostoma caninum ---- dog, fox, wild carnivors and very rarely cats

A. braziliense -------- cat, dog, fox and wild canids

A. tubaeforme ------ cats

A. ceylanicum ------ dog, cat, wild felids, occasionally human

Uncinaria stenocephala ---- dogs and cats

A. duodenale ----- human and primates

Necator americanus ------ human , primates, dog, cat and pigs

Ancylostoma caninum : A. caninum is a common cosmopolitan hookworm of the intestine of the dogs and other canids. It is a large species. The male worms are about 10-12 mm and females 15-20 mm in length. The buccal capsule is large, with three pairs of marginal teeth and a pair of ventralateral theeth, and possesses a dorsal gutter.

A.caninum does not normally occur in cat, however it has beeb infrequently in cats.

A . braziliense : this speciec has been identified from small intestine of dog, cat and wild canids. The male measures are 5.0-7.75 mm, and female measures are 9-10 mm in legth. The buccal capsule is elongated and contains 2 pairs of ventral theet, the lateral ones being large and prominent and medial ones very small.

A . tubaeforme (syn. Strongylus tubaeforme) : this species is the common hookworm of cats. A. tubaeforme resembles A. caninum but is sligthly smaller. The females are 12-15mm and males are 9.5-11 mm long. The buccal is deep with the dorsal gutter ending in a deep notch on the dorsal margin of the buccal capsule, the ventral margin of which bears three teeth on each side.

A.ceylanicum : A. ceylanicum resembles A. braziliense closely, but can be differentiated by its buccal capsule and bursa. There are two pairs of ventral teeth in the mounth, but the inner pairs is large than that of a.braziliense.

Uncinaria stenocephala : it occurs in the small intestine of the dogs,cats, foxes, wolves and related carnivors. The anterior end is bent dorsally. The worms are small , upto about 1.0 cm long , and males are 5-5.8 mm and females 7-12 mm long. The adult whitish worms are a large funnel-shape buccak capsule, which have a pair of chitinous plates, lacks dorsal teeth but has a pair of subventral teeth at the base.

General life cycle :

A) preparasitic period : Eggs with blastomeres are passed with the feces of the final hosts. The first stage larva develops in the egg in 1-1.5 days and leaves the egg . The infected larvae (L3) develop in 5-8 days in the environment. The ideal enviromental temperature for larval development is 23-33 degrees, and if the temperature rises, the development time is shortened. The soil surface and plant bottoms are very suitable for larval development. It is impossible for the larvae to survive below 4° C degrees. The light sandy and moist soils are very suitable for larval development .

The environmental temperatures of 24-32 ° C is also suitable for the skin invasion of the larvae.

B) parasitic period :

a) A. caninum : the final hosts become infected in 3 different ways

1) percutaneous infection : the larvae (L3) penetrate the skin primarily , and then larvae migrate via blodd stream to lungs where they moult to L4 in the bronchi and trachea, and are then swallowed and pass to the small intestine where final molut occurs, and development of parasites is completed here . The prepatent perion is 14-21 days.

2) oral infection : The development of the taken larvae takes place in the intestine. In dogs, larvae (L3) stay under the mucous membrane and digestive glands in the digestive system for a while, then return to the intestinal surface and complete the development and become adult parasites. Orally ingested larvae do not migrate to the lungs, development occurs only in the digestive system . The prepatent period 14-26 days.

The previously infected with A. caninum or with a well-developed immune system in dogs , the larvae do not enter the lungs and the larvae (L3) re-enter the circulatory system. These larvae (L3) enter hypobiosis in adipose tissue, muscles and subcutaneous tissue and stay in these areas for a while. In dogs, under some conditions, the arrested larvae in hypobiosis become active and these larvae come to the intestine and adult parasites develop . The main factors that move the larvae are: pregnancy, suppression of the immune system, long-term use of corticosteroids, complete elimination of mature parasites in the intestine .

3) galactogen transmission : galactogen transmission occurs only in A. caninum, and galactogen transmission is not seen in other hookworm genera and species.The arrested (L3) in female dogs are activated in the last period of pregnancy, some of the larvae come to the intestine in the female dog and some of them come to the mammary glands via blood circulation. In the first periods of lactation, usually in the first 4 weeks, the larvae (L3) come out with milk. Larval emergence is highest in the 2 -10 days in lactation.

In the suckling puppies, the development of larvae taken with milk takes place only in the intestine, the larvae do not migrate to the lungs. The prepatent period is about 14-16 days .

In the female dogs, patent infection develops in females giving birth as a result of the development of larvae emerging from hypobiosis and entering the intestine. The prepatent period is about 14 days.

The arrested development in Ancylostomum caninum : the larvae (L3) of A. caninum during the lung migration enters the general blood circulation, enters skeletal muscles, fat and various organs. The larvae lower their metabolism and enter hypobiosis and remain there for several years. These larvae are activated as a result of some conditions in dogs, they can be passed to puppies in lactation via galactogen, as well as some larvae can develop into adult parasites in the intestine of the female dog.

b) A. tubaeforme :

Biology of A. tubaeforme almost identical to the biology of A . caninum . There is no galatogenic transmission in the biology of A . tubaeforme . The prepatent period is for oral and cutaneous transmission is approximately 14-22 days. The rodents are paratenic host.

c) A. braziliense :

The final hosts ingest the larvae (L3) orally or through the skin. The prepatent period is in 14-16 days for oral route and in 13-27 days for cutaneous route.

d) A. ceylanicum :

The infection for final hosts is usually by oral ingestion of larvae (L3). Prepatent period is about 12-14 days.

e) Uncinaria stenocephala :

For the infection of U. stenocephala, the main route of infection in the final hosts is oral ingestion of larvae, and as a result of skin infections, very few adult parasites develop in the gut. The larvae develop in the small intestine and the prepatent period is 14-18 days . Although there are paratenic hosts in biology, it is not possible for the larvae to pass on to the offspring with milk.

Pathogenesis and clinical signs :

The disease is basically an acute and chronic hemorrhagic anemia disease, and it has a severe disease in animals 1 year old and younger, especially those with low iron reservoir , and results in death. The puppies infected with postnatal galactogen transmission may die within a few weeks of birth.

1) Ancylostoma caninum : it is the most pathogenic species among the disease agents, developmental disorder is evident in dogs and its clinical sings are severe and cause death in puppies.

a) The signs related to percutaneous infection : The dermatitis in dogs is appeared by a moderate acanthosis and hypercretination. The entry site of the larvae is usually the interdigital region. The soles of the feet are swollen and softened, sometimes splitting . There is deformation of the foot claws and thickening of the distal phalanges, but lesions are usually superficial. The pruritus, oedema, and skin papules develop in the cutaneous infection. The skin lesions are also found on the abdomen and legs, but the lesions may not be very prominent.

b) The findings of the lung migration period : Lung lesions and clinical signs associated with hookworm infection in dogs are rarely seen, because in dogs the infection most often develops from orally ingested larvae. Infected dogs may experience cough, runny nose, rarely fever and pneumonia. In rare cases, deaths may occur if the number of larvae ingested in individually susceptible or immuno compromised dogs.

c) The findings of the small intestine period : With the presence of L4s in the intestine, the pathogenicity and clinical signs of the disease begin to occur. Although the disease starts with a hemorrhagic anemia, it progresses differently over time with a series of pathological and metabolic developments.

The pathogenicity is formed as a result of the destruction of intestinal tissue and blood vessels by parasites in order to suck blood and feed, and the immuno-allergic response of the intestine . The blood sucking periods of parasites occur in L4, immature and adult periods and egg-producing periods of female parasites. The development and persistence of the disease are highly dependent on the general condition and nutritional quality of the infected animal.

The anemia develops as a result of erythrocyte count, volumetric change of erythrocytes and decreased hg carrying capacity of erythrocytes . The disease is basically iron deficiency anemia .

The disease has a peracute, acute or chronic course.

The peracute form occurs puppies (usually infected with galatogen transmission) and up to 1 year old dogs, however, older animals may develop peracute or acute form as a result of weakened immune system or malnutrition. In the period following birth in infected puppies, blood loss starts to occur at the end of 1 week at the latest, and the blood loss is high during this period. During the egg-producing period of female parasites (14-16 days), blood loss is again high.

No clinical signs are observed in the first week in newborn puppies. However, from the 2nd week, the disease begins suddenly and the general condition of the puppies deteriorates rapidly. The mucous membranes are pale, soft-liquid stools are dark in color and there are traces of blood in the faeces of sick dogs.There are no eggs in the faeces at this stage of the disease . The diseased puppies have inactivity due to lack of oxygen in the body . In dogs infected with a large number of larvae or in puppies that receive a large number of larvae in milk, no response can be obtained to the treatment due to pathological destruction in the intestine.

In the galactogen transmission, intestinal parasite numbers and pathogenicity increase in puppies 11-20 days . In the 10-15 days following the ingestion of larvae with milk, the hematocrit value decreases by 50%, and a decrease in hg and plasma protein values is observed. The daily plasma loss per parasite is about 0.04-0.06 ml . The decrease in

hematocrit value is greater than the plasma protein loss value. Hypoproteinemia in ancylostomosis occurs at the level of 20-50% and seriously threatens the general health of diseased dogs. In the diseased dogs, blood loss in the acute phase can reach 350-550 ml in the first 2 weeks, and the mortality rate is high in the first 4 weeks of the disease.

The development and persistence of anemia is directly related to the health and diet of dogs. The enteritis precedes clinical signs of anemia . At the onset of the disease, low blood values can be approached to normal values by overworking the erythropoietic system. However, if the disease persists or in severe infections, the decrease in blood values cannot be corrected by the host. At the beginning of the disease (at the end of the first 3 weeks), 25% of the total erythrocyte volume may be lost. Although anemia is initially normocytic and normochromic, it turns into microcytic and hypochromic type due to iron deficiency. An adult A. caninum can suck 0.05-0.20 ml of blood daily . In the advanced stages of the disease, lymphocyte activity is adversely affected by iron deficiency in dogs. Weakening of the immune system is one of the important causes of death despite treatment.

2) Uncinaria stenocephala : The parasites attached to the intestinal surface generally feed on the mucosa and the amount of blood they absorb is not at a level to cause pathogenicity. It causes atrophy, melting and severe inflammatory pathogenicity in intestinal villi .There is hypoalbuminemia and a slowly developing anemia in infected dogs and cats. The daily blood sucking is about 0.0003 ml per parasite . It progresses with hypoalbuminemia, anorexia and lethargy especially in puppies / kittens and animals with weak general conditions. Plasma protein loss in days 4-21 of the disease can be up to 25 ml in severe infections , and this value can be up to 10% of the total blood plasma value . In the diseased animals, anemia and diarrhea are prominent.

3) Ancylostoma braziliense : they do not suck blood, but the level of hypoproteinemia is high in the disease . The diarrhea is severe .

4) Ancylostoma tubaeforme : it is not considered a pathogenic parasite, but severe infections cause anaemia, growth retardation and hair deterioration.

5) Ancylostoma ceylanicum : The parasite causes anemia, but less pathogenicity than A. caninum .

Diagnosis : In the diagnosis, the age of the animals, anamnesis information to be obtained after birth, clinical findings, hematological findings and stool examination results are taken into consideration.

There is no parallelism between the number of eggs detected in feacal examination and pathogenicity.

Epidemiology :

a) environmental contamination : hookworm infection is common and female parsites have a greater capacity to produce eggs . Infection is more common in the spring, summer and autumn periods in temperate climates. The L3s are found more in soil areas . Dryness and direct sunlight have a lethal effect on the larvae, and cold and frost reduce the viability of eggs and larvae. Larval development is almost non-existent at temperatures below 15°C .

b) age of dogs and cats : The hookworm infections are most common in dogs and cats 1 year old and younger . In areas where the disease is endemic, the disease can also be seen in older dogs and cats.

c) galactogen transmission : In the persistence of ancylostomosis in dogs, ingestion of larvae with milk comes first as it is responsible for its pathogenicity and severe clinical findings and deaths.

Control and treatment :

The short prepatent time is one of the main causes of recurrence of hokworm infections.

Conscious use of drugs and attention to hygiene in the final hosts are the main issues in the control of the disease. The galactogen contamination is important and larval emergence with milk continues for approximately 28 days. However, the presence of larvae in milk occurs after 2-3 days of lactation. In the period following birth, immature and adult parasites are found in the gut on 12-13 days in the offspring, and on the 14th day, the drug should be given . However, if the active ingredient (macrocyclic lactones) has a long duration of action (up to 4 weeks), drug administration should be once a month after the 14th day. However, if active ingredients (fenbendazole, mebendazole, nitroscanate, pyrantel) with a short duration of action are selected, drug use should continue for 2-3 months, once every 2 weeks.

There is no treatment that is effective against inactive larvae in tissues.

In the old dogs , regular faecal examination is done , and medication is applied according to the result.

Only anthelmintic use in diseased animals is not enough to improve the general condition, parenteral iron supplementation, vit. B 12 should be given and protein-rich nutrition should be provided.

In order to prevent vetical contamination, the drug can be administered 2 weeks before the birth in pregnant dogs, but the selection of the active substance is important.

Mebendazole, fenbendazole , pyrantel and nitroscanate are effective against immature and mature parasites in the intestine. It is highly effective in macrocyclic lactones and emodepsis against parasites in this period (98 - 100 %) .

Pyrantel salts are effective against mature A. caninum and U. stenocephala 98-99% .

Pyrantel 5mg/kg , oxantel 20 mg / kg

Emodepdide is effective against adult hookworms (A. caninum, U. Stenocephala) in 0.45 mg / kg , and 98% effective on larvae in 4-7 days .

Milbemycine oxime is effective against adult parasites in 0.5 mg /kg , 99.7 %

Eprinomectin, especially in cats, is 99.5-100% effective in immature and adult at a dose of 0.5 mg/kg .

Cutaneous Larva Migrans :

It is the general name given to the disease/syndrome caused by the larvae of hookworm agents belonging to animals and other nematode and trematode larvae in the human epidermis. The main species are A. caninum, A. braziliende and U.stenocephala. The disease is more common in less or moderately developed countries and rural areas . It is observed more frequently in places where dog and cat populations are dense, especially in children. It is sufficient to touch the soil, grass or short plant areas for a while with bare feet.

The itching begins with the first contact of the larva, but other clinical signs begin a few days later. Although papules begin to appear in 4-6 days, papules may begin to develop weeks later. The papule formation is followed by the appearance of red-colored scars . The skin is slightly wet - moist and itching gradually increases . The scars can be a few cm or can reach 20-30 cm. The same human may have independent traces of more than one larva . These areas can sometimes be infected with bacterial and fungal agents .

Skin lesions are sufficient for diagnosis, but larvae may not always be detected in biopsy , and the anamnesis taken from the patient is also supportive for the result.

In the treatment , ivermectin 200mg/kg or albendazole 400 mg / kg X 5-7 days