

Ancylostomiasis in dogs

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▣ *Ancylostoma caninum*

Phylum: *Nematoda*

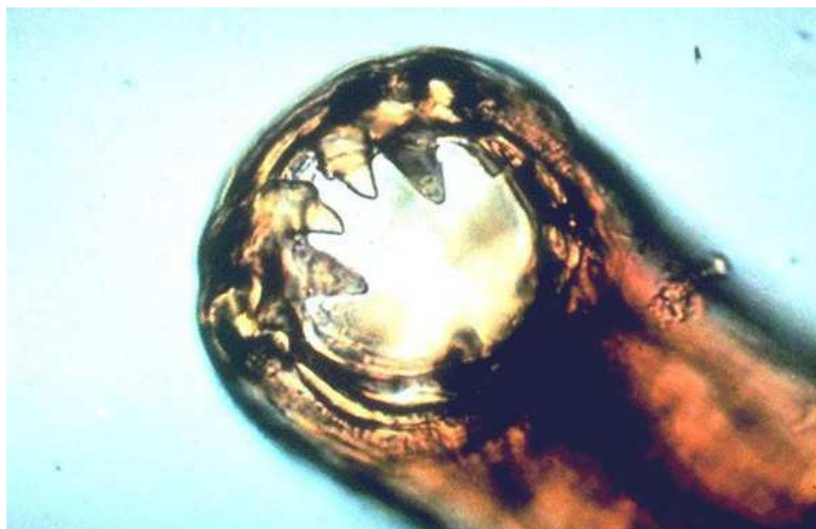
Class: *Secernentea*

Order: *Strongylida*

Superfamily: *Ancylostomatoidea*

Genus: *Ancylostoma*

Species: *caninum*



Adult

Males 1 cm long. Females 1-1,5 cm with no vulvular flap. Anterior bend dorsally. Buccal

capsule deep and supported by thick cuticle. Contains 3 pairs of teeth on ventral rim. Strongyloform esophagus. Males with copulatory bursa (1, 2, 3, 4, 5, 6, 7).



Eggs

60X40 microns. Ovoid, thin shelled, morulate embryo.

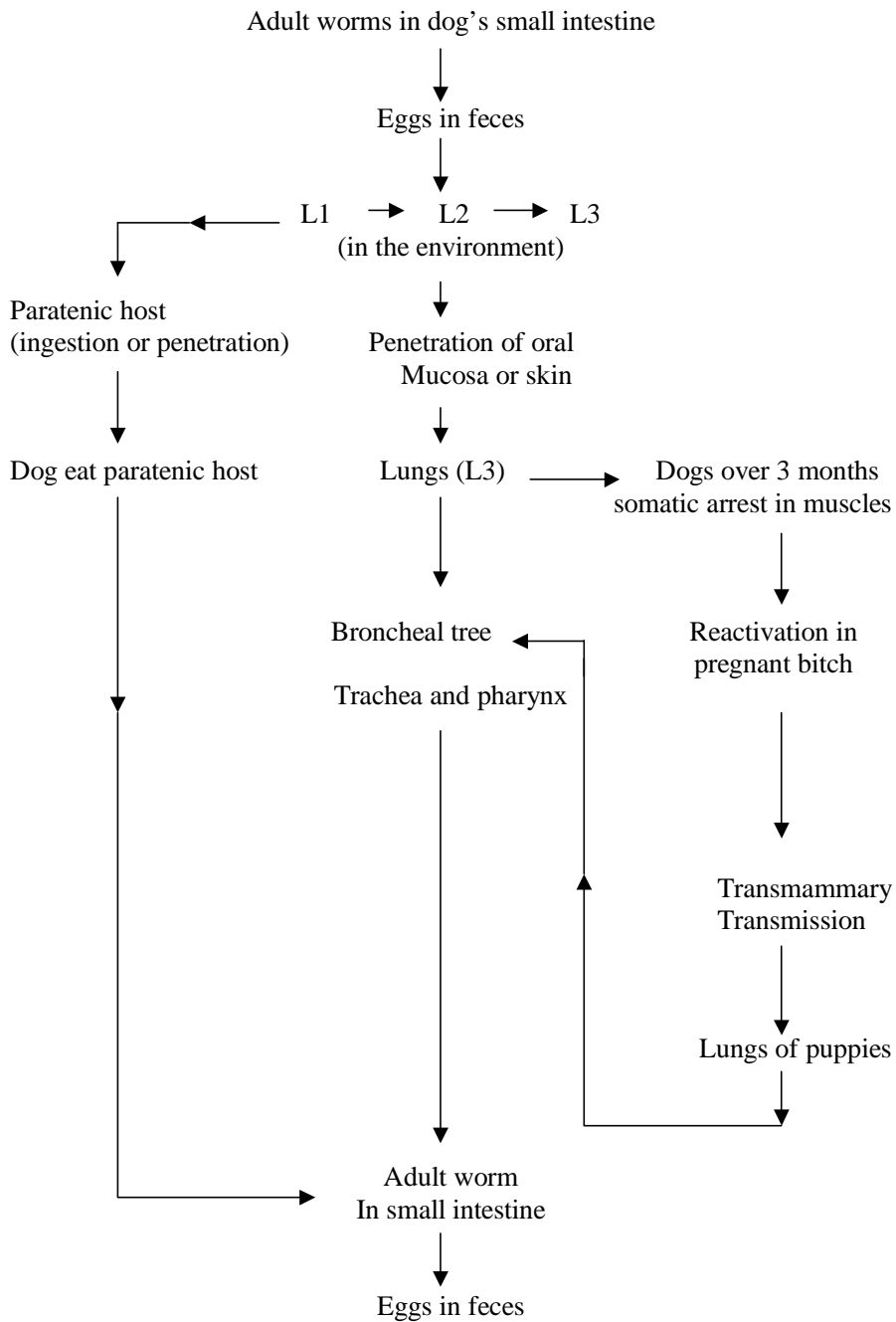


Hosts

Definitive: Dogs and other canids. Paraenteric: Rodents.

LIFE CYCLE

Ancylostoma caninum –Life cycle



Embryonated eggs are passed in feces. L1 (first stage larva) hatch, feed on bacteria in feces. Molt to L2 (second stage larva), also microbivorous. L3 (third stage larva) appear in 3 weeks.

Larvae develop to the third (L3) infective stage in soil and then they are either ingested by the

host or they enter the host through cutaneous contact (8, 9).

The larvae migrate to the lungs, and then on to the intestinal tract of the host.

In the small intestine, the larvae develop into adults. Eggs are then passed with the feces out to the soil and the cycle continues.

Alternatively, larvae may migrate in the host to subcutaneous fat, and in females to the skeletal muscles and mammary glands (10, 11, 12). When they are in these tissues the larvae become inactive. When infected females begin to nurse offspring, the larvae are activated and passed to the young ones who become infected. This transmammary infection is more important when the animals are young.

Older dogs often develop resistance to repeated exposures to the parasite. Because of that the young animals are much more likely to get infected with the parasite than older dogs.

If a rodent ingests the L3 larvae, the larva will migrate to the tissues and become dormant, so if a dog eats this rodent, the larvae will mature to adulthood in the small intestine (13).

Infection through ingestion: Larvae travel through the gut wall, into circulation, then through the liver, heart, and lungs where their penetration into alveoli occurs, then they are coughed up and swallowed, and go finally to the small intestine.

Infection through the skin: larvae penetrate skin and go into the circulation, then through the heart, to the lungs where penetration into alveoli occurs, then they are coughed up and swallowed, and go finally to small intestine.

Some larvae may go dormant in the gut wall or thoracic or abdominal musculature to be reactivated later by some unknown signal.

Transcolostral: the pre-patent period is 2 weeks (14).

LIFE HISTORY

Adult *Ancylostoma caninum* live in the small intestine where they attach themselves and feed on the dog's blood.

They lay up to 50,000 eggs per day. The eggs are released with the dog's feces, where they, then dwell (15). The eggs are segmented, when they pass out the vulva of the female worm, and are called morula. This process requires oxygen

which is found in the parasite's interior and which they extract through the gut.

Once in the open they are exposed to oxygen tensions and the morula very quickly transforms into the 1st stage larva (15). This, then hatches and wanders off into the feces. The larva is microbivorous, and looks for some bacteria to eat in the feces. Molt to the second stage larva (L2), in the feces. This stage, is also microbivorous.

The full second stage larva starts a metamorphosis to the third stage larva (L3), which acts very differently. This, the infective 3rd stage larva is still encased in the cuticle of the L2 stage, which is not shed until the larva invades a dog.

The 3rd stage larva is ensheathed and is isolated from immediate surroundings and called sheathed larva. This infective larva (L3) does not feed and is in a stage of arrested development; it is a very good swimmer, which is its characteristic. The 3rd stage larva has active movement, and is not microbivorous, (unlike the L1 and L2). Movement is made by swimming through raindrops, or dew on leaves and vegetation, waiting for a dog to invade in. It swims very well in the films of condensed moisture, leaves of plants, and other surfaces.

The larvae go under the surface of the soil when it rains, and come up when the sun shines.

The development of infective larvae requires about a week during periods of optimum temperature 22-30°C. No development occurs below 15°C. The development at higher temperatures than optimum, although rapid, produces fewer infective stages. Ample oxygen which is available in moist but not in waterlogged soils, is essential. Infective larvae can survive for several weeks in cool, moist soil but are quickly killed by freezing or very hot, dry conditions.

The 3rd stage larvae infect the dog by ingestion or by skin penetration. Infection through ingestion, takes place by transcolostral transmission to the nursing puppies via milk.

In both routes of infection (ingestion or skin penetration), the sheath is shed in the process of infection. The larva after that is parasitic 3rd stage larva.

When larvae infect a dog via ingestion, these are transported through the gut wall, into the blood circulation, then through the liver, the heart and the lungs, where they penetrate into alveoli, then are coughed up and swallowed, and finally end up in the small intestine (9).

When larvae infect a dog via skin penetration, they follow the blood circulation, through the heart to the lungs, where they penetrate into alveoli, then are coughed up, swallowed, and finally end up in the small intestine.

Some larvae may stay dormant at the gut wall or thoracic or abdominal musculature or other tissues, to be reactivated later by some unknown signal, whereas, others complete their final molt in the wall of small intestine.

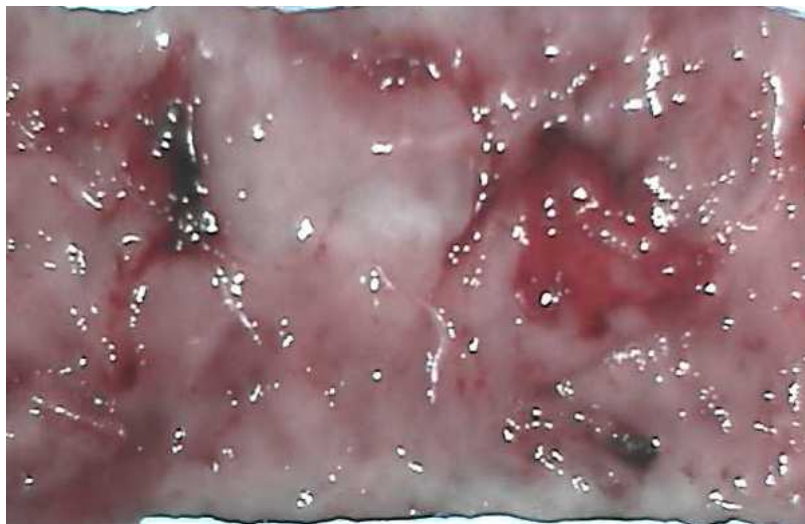
The arrested larvae may later migrate to the gut to mature in the same dog or, such as the case of the nursing female, migrate to the mammary glands and are passed to the nursing puppies (16).

Adult *Ancylostoma caninum* secrete anticoagulents and ingest blood from multiple bite sites. Approximately 90% of the blood ingested by the parasite passes through without being digested (17, 18).

Arrested larvae in tissues are very important reservoirs of infection.

The prepatent period is about 3-4 weeks and eggs appear in the feces, 2-3 weeks after oral infection and 4-5 weeks after skin penetration.

SITES OF INFECTION



Dermis - larvae
Respiratory tract - larvae
Small intestine - adults

INJURY TO HOST

Parasitic burden, the age and the immune status affect the pathogenesis of Ancylostomosis (19, 20).

When an adult parasite feeds on the mucosa of the small intestine it is essentially an open blood vessel because the toothed buccal capsule, removes blood from the arteriols. So, primarily, it is the parasitic burden that causes the lesions in the host's tissues.

Ancylostomosis causes secondary acute or chronic hemoragic anemia. The amount of blood removed is directly proportional to the weight of adult parasite.

The female adult weighs about 2,0-2,6 mg and the male, about 0,8 mg. Every day they lose about 30 μ l of erythrocytes per mg of worm, or in other words, each worm removes about 0,1 ml of blood daily. The peak of blood loss occurs even before eggs appear in the feces (4, 5, 6, 17, 19).

Sometimes young puppies present sudden hemorrhagic shock.

The disease is more often seen in young puppies or dogs under one year of age infected by transmammary route, and they are susceptible due to their iron reserves.

Blood loss starts about the eighth day of infection, when the adult worm has developed teeth bucal capsule, which enables it to grasp plugs of mucosa containing arteriols.

In a light parasitic burden, common in older dogs, the anemia is not so severe, as the marrow response is able to compensate for a long time. Ultimately, however the dog may become iron deficient and develop a microcytic hypochromic anemia (17, 19).

The adult parasite does damage to the solid tissue of the intestinal mucosa and the immediately underlying tissues. The host tries to replace the lost erythrocytes from the bone marrow, plasma proteins from the liver, and its fluid volume by increasing the water intake. So, if hematopoiesis and protein synthesis compensate sufficiently, the dog may appear normal and display only mild clinical and clinicopathologic signs of anemia.

Anticoagulant substances in hookworm saliva may enter the blood stream and, in very heavy infection, may cause problems.

Skin penetration in previously sensitized dogs causes skin reactions such as moist eczema and ulceration at the sites of percutaneous infection, that affects especially the interdigital skin (20, 21).

Lesions with focal hemorrhages arising from lung migration of larvae and penetration into alveoli, can also occur.

CLINICAL SIGNS

Puppies: anemia (macrocytic hypochromic), pale membranes, bloody stool, weight loss, weakness, poor growth. Pneumonia may also be observed in puppies (22).

Enteritis, bloody diarrhea, is life threatening for the puppies and may show signs as early as 2-3 weeks of age in a paracute case (puppy crashes

without prior signs of disease) or may occur in an acute stage at 3 weeks to weaning where there are eggs in the feces and bloody stool may be observed (22, 23, 24, 25).

Growth in young animals is stunted and the haircoat may appear dull and dry

Adults: Usually asymptomatic but any of the above symptoms may be present.

Dermatitis.

DIAGNOSIS

Peracute ancylostomiasis: is more likely to be post-mortem than on clinical examination. Typically the puppies appear healthy and sleek the first week but they get sick and deteriorate rapidly during the second week. They suffer from profound anemia. The possibility of peracute ancylostomiasis should not be rejected on the basis of a negative fecal examination because even (peri) natal infection does not become patent until the eleventh day.

Acute ancylostomiasis: Severe anemia demands fecal examination. Many eggs are usually found in acute cases, although clinical signs may appear during the prepatent phase in particularly severe infections.

Chronic ancylostomiasis: In typical cases, the diagnosis of compensated ancylostomiasis will rest on the discovery of eggs in the feces of a clinically healthy animal. Occasionally, however, the adjustment between the host and parasites is incomplete and the host remains in a state of chronic ill health.

Secondary ancylostomiasis. The cardinal sign is again profound anemia, usually in malnourished or even emaciated animal. The hookworms may indeed kill the dog but it is important in this case to recognize that they play a secondary role. An accurate diagnosis, for example, malnutrition with secondary ancylostomiasis logically leads to effective therapy (2, 3, 5, 6, 8, 22, 25).

EPIDEMIOLOGY

Tropical and southern temperate.

In endemic areas the disease is most common in dogs under one year old. In older dogs, the

gradual development of age resistance make clinical diseases less likely, particularly in dogs reared in endemic areas whose age resistance is reinforced by acquired immunity.

An important aspect of transmammary infection is that the disease may occur in suckled puppies reared in a clean environment and nursed by a bitch which may have been recently treated with anthelmintic and has a negative fecal egg count (2, 3, 5, 6, 8, 22, 25, 26).

TREATMENT

BUTAMISOLE HYDROCHLORIDE - DOSE: 2,4 mg/kg SG.

DICHLORVOS - DOSE: 10-12 mg/kg PO

DISOPHENOL SODIUM - DOSE: 9 mg/kg

FEBANTEL - DOSE: 10 mg/kg, SID, 3 days, PO

FEBANTEL/PYRANTEL PAMOATE - DOSE: 25 mg/kg, 5 mg/kg, 1 dose PO.

FENBENDASOLE - DOSE: 50 mg/kg PO SID for 3 days.

IVERMECTIN - DOSE: 0,2 mg/kg PO

MEBENDAZOLE - DOSE: 22 mg/kg PO for 3-5 days.

MILBEMYCIN OXIME - DOSE: 0,5 mg/kg, 1 dose, PO.

PYRANTEL PAMOATE - DOSE: 5-10 mg/kg PO.

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