Lecture # 19: Superfamily Ancylostomoidea (hookworms)

Objectives:
1. Compare the life cycle of Ancylostoma caninum in puppies versus adult dogs.
2. What are the clinical and pathological signs of hookworm infection?
3. Describe the public health/zoonotic concerns with A. caninum.

Outline:

SUPERFAMILY ANCYLOSTOMATOIDEA (HOOKWORMS)

I. Morphology: Buccal cavity - teeth or cutting plates at the anterior opening of a large buccal cavity; dorsal flexion of the anterior end i.e. ‘hooked’. Eggs are typically strongyle-like, and are designated as ‘hookworm eggs’ if seen on a small animal fecal floatation.

II. Life cycle: Infection is by ingestion of or skin penetration by free-living infective L₃ larvae. Additionally, Ancylostoma caninum can infect by the transmammary (lactogenic) route (puppies not kittens); i.e. L₃ are acquired by ingestion of milk while nursing. Larvae migrate through the tissues of the host to establish as adult worms in the lumen of the small intestine where they attach and feed on blood. One adult female may shed as many as 16,000 eggs per day! Go to CAPC site www.capcvet.org for really good video of adult worm.

III. Ancylostoma caninum: adults are found in the small intestine of dogs only. (3 teeth, pair)
   A. tubaeforme: cats only. (3 teeth, pair)
   A. braziliense: dogs and cats, especially in tropical areas. (1 tooth, pair)
   A. duodenale: man (2 teeth, pair)
   Uncinaria stenocephala: Cutting plates and much less pathogenic (see Nematode Appendix section)

Ancylostoma caninum

A. Morphology: Adults are 10 to 16 mm long, grey-red; the buccal cavity is dorsally bent and has three teeth on either side of the ventral aspect of the anterior opening. Eggs are 56-75 um by 34-47 um; usually at 8-celled morula stage in feces.

B. Life cycle: Eggs shed in the environment hatch to release larvae which feed on bacteria in the feces/environment, to become infective L₃ larvae. Development to the L₃ stage is rapid under warm, moist conditions i.e. ~5 days.

Infective L₃ enter the host by skin penetration or ingestion. Infection may also occur by ingestion of an infected paratenic host. L₃ have 2 choices:
   (1) to undergo direct development into L₄ which become young adults as early as 7 days post-infection. Prepatent period is about 3 weeks (15-18 days).
   (2) arrest as L₃ in the surrounding tissue. Arrested L₃ can remain in this state for months to years, and when reactivated, they resume normal development to become L₄, then adults.

Reactivation of arrested L₃ is triggered by factors such as stress, removing adult by deworming and near-term pregnancy. During late pregnancy, arrested L₃s in the tissues of the mother reactivate and lodge in the mammary glands, ready for transmission to the puppies during lactation (in dogs not cats). Note that a previously infected animal may have no evidence of hookworm eggs in the feces but could have arrested L₃ in its tissues; arrested larvae are NOT very susceptible to drug treatment due to reduced metabolic activity.
C. Pathogenesis: Is due to the blood-feeding adult worms. Classic disease -- acute anemia in nursing puppies with large worm burdens. (50 adult worms can remove as much as 3 ml of blood per day). L3 acquired in the milk while nursing on the infected mother mature into blood-sucking adults in the small intestine. In older dogs, small amounts of arrested larvae can also reactivate, migrate to the intestinal tract and develop into adult worms, causing a chronic blood-loss disease. In cats, a chronic condition of weight loss and poor condition could be due to A. tubaeforme. No peracute disease in cats because no L3 in milk.

D. Resistance: Adult dogs are frequently infected and shed hookworm eggs but are more resistant to hookworm disease than puppies due to (1) acquired immunity (from previous infections), (2) natural defense mechanisms (even if previously uninfected), (3) ability to compensate for blood loss, and (4) premunition, which is the inhibition of further infection exerted by the resident population of hookworms.

E. Clinical signs: Seasonal; especially with warm, moist conditions. Often seen in newborn and young pups or immunocompromised, older dogs exposed to heavy infection. Anemia, edema, emaciation, bloody diarrhea or tarry stool. Peracute disease (no eggs in feces) occurs in newborns 1-2 weeks of age, due to transmammary transmission of infection.

F. Treatment and Control: Treat at the first sign of anemia with an anthelmintic; iron supplementation and blood transfusions may be required in very small animals suffering extreme blood loss. Treatment should be repeated weekly due to reactivation of arrested larvae or continuing exposure to infection. In general, puppies should be treated at 2-3 weeks of age with a drug that controls hookworms and ascarids, and treatment should be repeated every 2 weeks till 2 months of age when the animal is put on a heartworm prevention program. The monthly heartworm preventatives such as pyrantel/ivermectin (HeartGard Plus), milbemycin (Interceptor), selamectin (Revolution), moxidectin (Advantage) as well as other drugs are very effective in controlling ADULT hookworms. Available drugs are NOT very effective in completely eliminating arrested larvae in tissues. WHY?

Keep run areas clean and dry; sodium borate or bleach will kill larvae. Remove feces. To control the infection in the pregnant female, Standard monthly doses to mother of selamectin during the periparturient period and at whelping has also been shown to be effective in preventing infection in newborn puppies.

G. Zoonotic disease potential:
1. Cutaneous larva migrans: an itchy dermal lesion caused by the skin penetrating larvae.
2. Eosinophilic gastroenteritis: Rare. A. caninum can penetrate human skin, migrate to the intestine, mature to adulthood (not a patent infection i.e. no egg production) and only 1-2 worms seem to cause a painful, eosinophilic enteritis.