Capillaria hepatica (Pathogen – Tissue Nematode)

Organism:
Capillaria hepatica infection is commonly found in rats, other rodents, and other mammals. Human cases have been reported from various parts of the world, including the United States.

Life Cycle:
Capillaria hepatica has a direct life cycle, with no intermediate host. It can develop with only one definitive host, but likely requires two hosts to complete the life cycle. Adult worms are located deep within the liver parenchyma of the host, and lay hundreds of eggs in the surrounding parenchymal tissue (1). The eggs trapped in the parenchyma cannot be passed in the feces of the host, and remain in the liver until the animal dies (2), or more likely, is eaten by a predator or scavenger (3). Eggs ingested by scavengers are unembryonated (not infectious) and are passed in through the digestive tract into and out in feces, providing an efficient mechanism to release eggs into the environment; this is ecologically the most likely primary route of transmission (4). Eggs embryonate in the environment (5), where they require air and damp soil to become infective. Under natural conditions, embryonation is slow and may take between 6 weeks and 5 months. The cycle continues when embryonated
eggs are eaten by a suitable mammalian host (6). Infective eggs hatch in the intestine, releasing first stage larvae. The larvae penetrate the intestinal wall and migrate via the portal vein to the liver parenchyma within 3-4 days. Larvae take about 3-4 weeks to mature into adults and mate. Humans are usually infected after ingesting embryonated eggs in fecally-contaminated food, water, or soil (7).

**Acquired:**
Infection in humans is acquired through ingestion of embryonated eggs from the soil.

**Epidemiology:**
*C. hepatica* has a broad global distribution in wildlife. Human cases have originated from all inhabited continents except for Australia, although there it exists in wildlife.

**Clinical Features:**
Symptoms of this infection mimic those of hepatitis, amebic liver abscess, trichinosis, VLM, Loeffler’s syndrome, Hodgkin’s disease, and histoplasmosis. In the first case reported from Maine, the patient presented with a subacute history of severe abdominal pain, fevers, and weight loss. The deposition of eggs in the liver parenchyma causes granuloma formation and liver necrosis, which in heavy infections can lead to potentially fatal liver dysfunction. The true incidence in humans may be underestimated due to the nonspecific clinical presentation and difficulty of diagnosis.

**Laboratory Diagnosis:**
In a true human infection, no eggs are found in the stool. Diagnosis requires histologic examination. Eggs in liver biopsy specimens can be identified on the basis of their characteristic morphology. The recent development of an IFAT may lend itself to testing of human sera for the detection of early *C. hepatica* infection; however, such tests are not available commercially.

**Note.** In cases of spurious infection, in which infected animal liver has been ingested, *C. hepatica* eggs may be passed in the stool. These eggs measure 51 to 68 µm long by 30 to 35 µm wide and resemble those of *C. philippinensis* (45 by 21 µm), which can be seen in the stool in true human infection.

**Organism Description:**
**Egg:** These eggs measure 51 to 68 µm long by 30 to 35 µm wide and resemble those of *C. philippinensis* (45 by 21 µm), which can be seen in the stool in true human infection.

**Laboratory Report:**
*Capillaria hepatica* eggs present in tissue. If eggs are found in the stool, they represent a case of spurious infection.

**Treatment:**
*C. hepatica* is a rare infection and clinical experience is limited. Steroids have been used to help control the inflammation of the liver.

**Control:**
In order to prevent both types of capillariosis, proper hygiene and disposal of fecal matter is important.