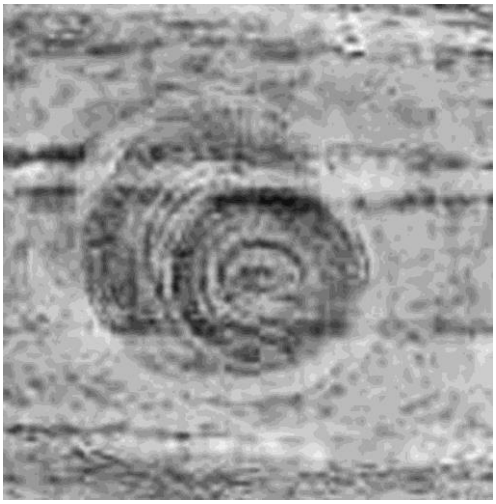
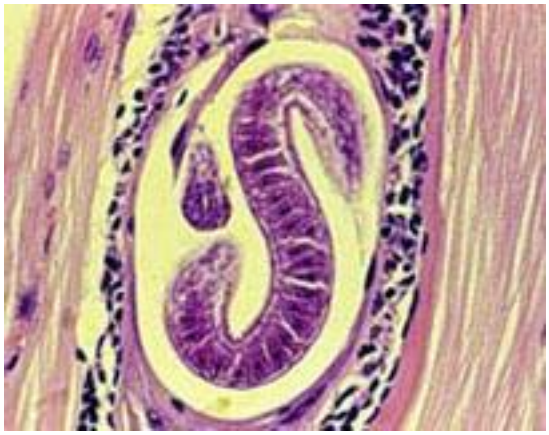
## PARASITOLOGY CASE HISTORY 9 (HISTOLOGY)
## (Lynne S. Garcia)

An Alaskan hunter killed a bear. Subsequently, he prepared the meat for consumption, some fresh and some frozen (steaks, larger cuts, ground meat). The following day a number of friends attended a party where some of the meat was cooked (rare to well done, depending on the person's preference) and consumed. Approximately three weeks later the hunter and one of his friends began to have a fever; both complained of diarrhea and muscle pain. However, neither sought medical help for another six weeks, at which point they both demonstrated an eosinophilia. Based on the history, it was recommended that the bear meat be examined microscopically for a possible parasitic infection. An artificial digestion technique was used on the meat (0.1% pepsin and 0.1% hydrochloric acid); squash preps of the meat were prepared and examined (top row below). Routine H&E stains are seen in the bottom row.


- Based on these images, what is your diagnosis?

***Scroll Down for Answer and Discussion***

## Answer and Discussion of Histology Quiz #9

The structures observed in the meat were larvae of *Trichinella* sp. Diagnostic features include the encapsulated, coiled larvae located in the muscle tissue of the bear. The patient was also found to be antibody positive for *Trichinella* by EIA.

Although *Trichinella spiralis* was first seen in human tissue at autopsy in the early 1800s, it was not until 1860 that Freidrich von Zenker concluded that the infection resulted from eating raw sausage. The consumption of rare or raw pork as the cause of trichinosis was experimentally proved a few years later. By the 1900s, trichinosis was definitely recognized as a public health problem. This particular infection has a cosmopolitan distribution but is more important in the United States and Europe than in the tropics or the Orient. The prevalence in autopsies within the United States has declined from 15.9% of human diaphragms studied at autopsy from 1931 to 1944 to 4.5% from 1948 to 1963; in recent years, the prevalence has decreased to 2.2% and the mortality associated with this infection has decreased to less than 1%. In 1990, only 105 cases of human trichinosis were reported in the United States, and by 1994 the number had dropped to 35. During 1997 to 2001 the incidence decreased to a median of 12 cases annually and no reported deaths. However, in many areas of the world, trichinosis remains a problem.

During the 5-year period from 1997 to 2001, 72 cases were reported to the Centers for Disease Control and Prevention. Of these, 31 cases were associated with eating wild game: 29 with bear meat, 1 with cougar meat, and 1 with wild-boar meat. In comparison, only 12 cases were associated with eating commercial pork products; 4 of these cases were traced to a foreign

source. Nine cases were associated with eating noncommercial pork from home-raised or direct-from-farm swine where U.S. commercial pork production industry standards and regulations are not applicable.

**Life Cycle**. Human infection is initiated by the ingestion of raw or poorly cooked pork, bear, walrus, or horse meat or meat from other mammals (carnivores and omnivores) containing viable, infective larvae. The minimum infectious dose for humans has not been clarified; however, it's been estimated that approximately 100-300 larvae cause disease. The tissue is digested in the stomach, and the first-stage larvae (L1) are resistant to gastric juice. The excysted larvae then invade the intestinal mucosa, develop through four larval stages within about 48 h, mature, and mate by the second day. By the fifth day of infection, the female worms begin to deposit motile larvae, which are carried by the blood vessels, intestinal lymphatic system, or mesenteric venules to the body tissues, primarily highly oxygenated striated muscle. Deposition of larvae continues for approximately 4 to 16 weeks, with each female producing up to 1,500 larvae in the nonimmune host. Newborn larvae can penetrate almost any tissue but can continue their development only in striated muscle cells. Within the human host, the cyst measures about 400 by 260 μm, and within the cyst, the coiled larva measures 800 to 1,000 μm in length. At this point, the larvae are fully infective. After weeks to years, the calcification process occurs. It has been estimated that in muscle nurse cells, parasite larvae can survive for up to 40 years in humans and over 20 years in polar bears.

**Clinical Disease**. Pathologic changes due to trichinosis can be classified as (i) intestinal effects and (ii) muscle penetration and larva encapsulation. Any damage caused in either phase of the infection is usually based on the original number of ingested cysts; however, other factors such as the patient's general health, age, and size also play a role in the disease outcome. Symptoms of trichinosis are generally separated into three phases, with phase 1 being related to the presence of the parasite in the host prior to muscle invasion and phase 2 being related to the inflammatory and allergic reactions due to muscle invasion. There may also be an incubation period of up to 50 days. Phase 3 is the convalescent phase or chronic period.

Symptoms that may develop within the first 24 h include diarrhea, nausea, abdominal cramps, and general malaise, all of which may suggest food poisoning, particularly if several people are involved. Studies also indicate that the diarrhea can be prolonged, lasting up to 14 weeks (average, 5.8 weeks) with few or no muscle symptoms. It is still unknown whether this clinical

presentation is related to variant biological behavior of Arctic Trichinella organisms, to previous exposure to the parasite, or to other factors.

During muscle invasion, there may be fever, facial (particularly periorbital) edema, and muscle pain, swelling, and weakness. Other signs are conjunctivitis, headache, dry cough, petechial bleedings, and painful movement disorder of the eye muscles. The extraocular muscles are usually the first to be involved, followed by the muscles of the jaw and neck, limb flexors, and back. Muscle damage may cause problems in chewing, swallowing, breathing, etc., depending on which muscles are involved. The most severe symptom is myocarditis which occurs in approximately 5 to 20% of cases, and which usually develops after the third week. Symptoms include pericardial pain, tachycardia, and electrocardiogram abnormalities such as nonspecific ventricular repolarization disturbances, followed by bundle-branch conduction disturbances, and sinus tachycardia.

Death may occur between the fourth and eighth weeks. Other severe symptoms, which can occur at the same time, may involve the central nervous system (CNS). Although *Trichinella* encephalitis is rare, it is life-threatening. Technological advances such as the computed tomogram, angiogram, and electroencephalogram are of no diagnostic assistance and probably add nothing to traditional diagnostic information, which includes eosinophilia (can be up to 80%), sedimentation rate, and muscle biopsy. It is estimated that 10 to 20% of the patients with trichinosis have CNS involvement and that the mortality rate may reach 50% in these patients if they are not treated.

**Diagnosis. Muscle Biopsy**. Muscle biopsy (gastrocnemius, deltoid, and biceps) specimens may be examined by compressing the tissue between two slides and checking the preparation under a microscope at low power (10x objective). However, this method does not provide positive results until 2 to 3 weeks after the onset of the illness. It is also important to remember that not all species form the capsule). Muscle specimens or samples of the suspect meat can also be examined by using an artificial digestion technique to release the larvae.

**Antibody Detection**. Serologic tests are also very helpful, the standard two being the enzyme immunoassay (EIA) and the bentonite flocculation test, which are recommended for trichinosis.

**Epidemiology**. While cases acquired from pork consumption continue to decline, the proportion of cases acquired from wild game meat has increased. However, the absolute numbers remain at about 9 to 12 per year within the

U.S. Continued multiple-case outbreaks and the identification of nonpork sources of infection require ongoing education and control measures.

In other parts of the world, the infected-meat source statistics vary; in the former Soviet Union, >90% of the cases have been attributed to the ingestion of poorly cooked bear and wild boar meat. Factors contributing to the slow decline of trichinellosis incidence in Russia and to the increase in the number of cases originating from wild-animal meat include the distribution and consumption of veterinary-uncontrolled pork, poaching and distribution of wild-animal meat, and poor compliance with regulations. Twenty-seven outbreaks of human disease occurred in China between 1964 and 2004 and were associated with mutton, dog, and game meat. However, the quarantine of infected meat is not mandatory in China. Although outbreaks are rare in Israel, outbreaks have been detected in immigrant agricultural workers; infected wild-boar meat was implicated. In tropical Africa, the infected meat source tends to be bush pigs and warthogs. Most infections in Central and South America have been associated with domestic pigs. It is difficult to say with certainty exactly which animals may be infected throughout the world; very few comprehensive studies of wild animals have been attempted.

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