Enteric Nematodes of Lower Animals Transmitted to Humans: Zoonoses

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General Concepts

Visceral and Ocular Larva Migrans

Clinical Manifestations

Visceral larva migrans: Symptoms include hepatosplenomegaly, fever and eosinophilia. The symptoms and duration of illness depend on the site and extent of larval migration.

Ocular larva migrans: Symptoms include leukocoria (white pupillary reflex), loss of vision in affected eye, eye pain or strabismus. There are usually no systemic symptoms.

Structure

Toxocara canis is the most frequent cause of these diseases. The larvae are 400 µm X 20 µm; adult females are 5 to 18 cm long and adult males 4 to 10 cm long. Eggs are about 85 µm x 75 µm, with a thick brown shell.

Multiplication and Life Cycle

The life of Toxocara canis in the dog, its natural host, is similar to that of Ascaris in humans. Ingested eggs hatch in the intestine. The larvae migrate extraintestinally for a period, then return to the intestine to mature and lay eggs, which are shed in the feces. In humans, ingested eggs hatch and larvae migrate into the deep tissues, but development proceeds no further.

Pathogenesis

Larvae persisting in either the viscera or the eyes cause granulomatous reactions.
Host Defenses

Leukocytosis and eosinophilia are common. Hypergammaglobulinemia and antibodies against the larvae appear.

Epidemiology

The egg of the parasite is ubiquitous in soil wherever infected dogs defecate. Infection occurs when contaminated soil or fomites are ingested. Visceral larva migrans is most common in preschool children with a history of pica (dirt-eating); ocular disease is more common in school-age children.

Diagnosis

The clinical picture, serologic tests (ELISA), and occasionally biopsy are the chief means of diagnosis.

Control

Larva migrans can be controlled by keeping pets wormed and by sanitary disposal of pet feces.

Cutaneous Larva Migrans

Clinical Manifestations

Larvae migrating erratically just below the epidermis cause serpiginous tracks that advance by 1 to 2 cm daily and may be intensely pruritic. Untreated disease resolves in a month or two as the larvae die.

Structure

*Ancylostoma braziliense*, the common hookworm of dogs and cats, is the usual cause of cutaneous larva migrans. Infective larvae are about 600 µm x 20 µm. Adult males are 5 to 7.5 mm long, and adult females, 6 to 10.5 mm long.

Multiplication and Life Cycle

In the natural nonhuman hosts, the life cycle is similar to that of *A duodenale* in humans; infective larvae in the soil penetrate the skin, migrate through the tissues, and eventually attach to the mucosa of the small intestine where they mature and lay eggs. In humans, by contrast, larvae penetrate and migrate through the skin (possibly also into deeper tissue), but development proceeds no further.

Pathogenesis

Migrating larvae cause inflammatory responses.

Host Defenses
The host may develop a hypersensitivity reaction, and eosinophilia may occur. No protective immunity develops.

**Epidemiology**

Cutaneous larva migrans is primarily a disease of the subtropics, including the southern United States, and occurs wherever soil is contaminated by the feces of infected animals.

**Diagnosis**

The clinical appearance is diagnostic.

**Control**

Worming of pets and sanitary disposal of pet feces are essential to control. Human skin lesions can be treated topically with 10 percent thiabendazole.

**Other Larval Migratory Diseases**

Several other nematodes of lower animals have larvae that can infect human tissue and cause a visceral larva migrans.

**Trichinosis**

**Clinical Manifestations**

Disease manifestations include the classic diagnostic triad of myalgias, periorbital edema, and eosinophilia. The severity of disease depends on the number of larvae ingested.

**Structure**

The *Trichinella spiralis* adult female is viviparous and 4 mm x 60 µm; males are 1.5 mm x 40 µm. The infective larvae are about 1 mm long.

**Multiplication and Life Cycle**

The parasite completes all stages of development in one host. Infective larvae encyst in striated muscles and excyst in the gut of carnivores that eat the infected meat. The worms mature and reproduce in the small intestine. Newborn larvae migrate to the muscles where they encyst.

**Pathogenesis**

Symptoms depend on the stage of infection. The intestinal phase may be marked by diarrhea or abdominal pain. Systemic symptoms follow, with myalgias, edema, fever, and eosinophilia as larvae migrate and encyst.
Host Defenses

Immune responses facilitate the expulsion of adult worms from the small intestine and inhibit both migrating larvae and the production of larvae by mature worms.

Epidemiology

*Trichinella* is acquired by ingestion of undercooked meat (especially pork).

Diagnosis

Diagnosis is made on the basis of the clinical picture, serologic tests (e.g., bentonite flocculation test), and muscle biopsy.

Control

Trichinosis is prevented by cooking meat to 77° C or 170° F). No specific therapy has been proven totally efficacious.

INTRODUCTION

This chapter reviews the more common zoonoses caused by enteric nematodes of lower animals. Humans are not natural hosts for these parasites. Human infections are accidental, and the human disease may or may not resemble that of the animal host. To understand the epidemiology and pathogenesis of these zoonoses, one must understand the life cycle of the involved nematode in its natural animal host and in humans. Some of these nematodes are not able to complete their life cycle in a human as they do in the animal host; the disease process will differ accordingly. For example, in visceral larva migrans, which is caused by the ascarids of dogs and cats, the worms cannot mature in the human host, so development is arrested in the larval stage. Larvae persisting in tissues produce the clinical symptoms. Other enteric nematodes such as *Trichinella spiralis* can complete their life cycle in humans, and produce disease similar to that in other mammalian hosts.

Visceral and Ocular Larva Migrans

Clinical Manifestations

Two distinct patterns of larva migrans infection are recognized: visceral larva migrans and ocular larva migrans. These clinical syndromes result from the systemic migration of the larval forms of animal helminthic parasites. *Toxocara* species, the common roundworms of dogs and cats, are the usual cause. The disease affects mainly children.
The classic visceral larva migrans syndrome usually occurs in preschool children with a history of pica (dirt-eating). Patients who have severe infections often present with eosinophilia, fever, and marked hepatomegaly which may persist for months; there may be associated respiratory symptoms with wheezing and coughing. Pulmonary infiltrates may be seen on chest roentgenograms but these are usually transient. Pruritic rashes and chronic urticaria may occur. Neurologic involvement may cause seizures. Death has been associated with myocarditis, encephalitis, and respiratory syndromes.

The ocular form of the disease usually occurs in children who are between school age and young adulthood. Ocular invasion by the larva may produce retinal granulomas or endophthalmitis, leukokoria (white pupillary reflex), decreased visual acuity, strabismus and eye pain. The syndrome may resemble retinoblastoma; misdiagnosis has resulted in unnecessary enucleation of the involved eye. The host usually is asymptomatic until ocular involvement becomes apparent. Patients with ocular disease rarely have a history of pica. It has been suggested that ocular larva migrans is associated with fewer larvae than visceral larva migrans. This view is generally supported by the finding of higher serum antibody titers in patients with visceral than with ocular disease. Rarely, the two forms of the disease coexist, presumably related to massive infection. Infections not involving the eye that are caused by few parasites may be asymptomatic and hence not recognized.

Structure

*Toxocara canis* is the most common cause of visceral and ocular larva migrans. Mature *Toxocara canis* worms live in the small intestine of the dog, their natural host. They have an average life span of about 4 months. The female is 5 to 18 cm. long and the male, 4 to 10 cm. A single female may produce 200,000 eggs per day. A heavily infected dog can pass millions of eggs per day in feces. The egg is about 85 µm x 75 µm with a light brown, thick shell. Under appropriate soil conditions, the egg embryonates and develops to the infective stage. The infective larva is approximately 400 µm x 20 µm and resembles the adult.

Multiplication and Life Cycle

When infective eggs of *Toxocara canis* are ingested by a dog, the larvae hatch in the small intestine, invade the intestinal mucosa, and undergo an extraintestinal migratory phase. In older dogs, many larvae remain trapped in body tissues. In puppies, most of the larvae migrate through the bronchioles to the trachea and pharynx, where they are swallowed and complete maturation to the adult form in the intestine. Eggs are shed in the feces and develop into an infective stage in the soil.

Most puppies are infected prenatally with *Toxocara canis*. Presumably, hormonal changes during gestation facilitate transplacental migration of larvae from maternal tissues. Puppies may also be infected by the transmammary route or by ingestion of embryonated eggs.

Pathogenesis

Humans contract *Toxocara* infections by ingesting embryonated eggs. The larvae hatch in the small intestine, invade the mucosa, and enter the portal system. Some are trapped
in the liver, but others proceed to the lungs and into the systemic circulatory system where they may disseminate to virtually any organ (Fig. 91-1). The parasite cannot complete its life cycle in humans as it does in the animal host. Developmental arrest occurs in the larval stage. The larvae persist in tissue, where they evoke a granulomatous reaction and eventually die. The clinical manifestations depend on the amount of tissue damage caused by the invading larvae and on the associated immunemediated inflammatory response.

FIGURE 91-1 Pathogenesis of visceral larva migrans caused by Toxocara canis.

Host Defenses

Visceral larva migrans is associated with marked hematologic and immunologic host response, in contrast to the ocular disease, in which, presumably, the small number of parasites cause less host reaction. Serum Toxocara antibody titers usually are elevated to diagnostic levels in both syndromes. Leukocytosis with eosinophilia (usually in excess of 30 percent) occurs in visceral disease. Peripheral leukocyte counts exceeding 100,000/mm³ are seen. Hypergammaglobulinemia is common, and IgE is markedly elevated. In addition to antibodies specific for larvae and their secretory-excretory products, a number of nonspecific antibodies may be produced, including rheumatoid factor and elevated antibody titers to human A and B blood group substances.

Epidemiology

Most young puppies and approximately 20 percent of adult dogs are actively infected with Toxocara canis. Puppies between 3 weeks and 3 months of age excrete large
numbers of eggs and constitute the greatest hazard to the environment. Backyards, children's sandboxes, public parks, and beaches accessible to dogs are often contaminated with *Toxocara* ova, which may remain infective for years. These areas are potential exposure sites for young children or others who accidentally ingest the infective eggs. Children who habitually eat dirt are at particular risk. Direct contact with pets is not a factor in infection because of the incubation period required before the eggs are infective.

**Diagnosis**

The diagnosis of visceral larva migrans is usually suggested by the clinical findings of visceral involvement in association with hypergammaglobulinemia, leukocytosis, and eosinophilia. Liver biopsy may be diagnostic, although the larvae are difficult to find even in the presence of eosinophilic granulomas (Figures 91-2 and 91-3). Elevated titers of antibodies against the A and B isohemagglutinins *Toxocara* antigens support the diagnosis. The enzyme-linked immunoabsorbent assay (ELISA) using larva-specific antigen has proven a reliable serologic test. It is especially useful in evaluating ocular infections, which characteristically do not exhibit the peripheral eosinophilia and other evident host responses of visceral disease.

![Liver biopsy from child with visceral larva migrans caused by *Toxocara* (x200).](image-url)
Control

Prevention of human infection centers on the appropriate treatment of *Toxocara* infections in dogs and cats and on sanitary disposal of pet feces. Public education on the necessity of these preventive measures is needed. Many responsible pet owners are unaware of the health hazards imposed on human by animal roundworm infections. Once the soil has become contaminated, infective eggs persist indefinitely.

There is no treatment of proven efficacy for disease caused by *Toxocara* species in humans. The anthelminthic drugs diethylcarbamazine and albendazole have been reported to be beneficial in some cases. Corticosteroids have been used to decrease the inflammatory response in ocular infections and in severe respiratory or cardiac disease.

Cutaneous Larva Migrans

Cutaneous larva migrans (creeping eruption) is a dermatitis caused by the larvae of *Ancylostoma braziliense*, the dog and cat hookworm, which penetrate human skin and migrate in the subepidermal tissue. *Ancylostoma caninum* and other species of hookworms also can cause this infection. A similar cutaneous eruption may occur in patients with intestinal *Strongyloides stercoralis* when the perianal skin is
autoinoculated by infective larvae passed in the stool. This syndrome is called *larva currens* ("racing larva") because of the rapid migration of this larva in the skin (see Ch. 90).

**Clinical Manifestations**

As the larvae invade the skin, a tingling sensation may be felt at the site of invasion. An erythematous, pruritic papule usually develops within a few hours. This lesion intensifies over the next few days and develops into a slightly raised, erythematous, serpiginous track that usually progresses at the rate of 1 to 2 cm/day (Fig 91-4). The track may be especially pruritic at its advancing edge, over the offending larva. Skin lesions may be single or numerous depending on exposure. The most frequent areas of skin involvement are the feet, hands, buttocks, and genital areas. The disease is self-limited with death of the invading larvae in a month or two. Secondary bacterial infections may result from frequent scratching of the lesions.

![FIGURE 91-4 Cutaneous larva migrans.](image)

**Structure**

*Ancylostoma braziliense* is the smallest of the common canine and feline hookworms. It has the typical hookworm shape with the anterior end bend dorsally. The adult female is 6 to 10.5 mm long and the smaller male is 5 to 7.5 mm long. The infectious third-stage filariform larvae are approximately 600 µm x 20 µm.

**Multiplication and Life Cycle**
The basic life cycle of *Ancylostoma braziliense* in an animal host is similar to that of *A duodenale* in humans. In humans, the larvae cannot complete their life cycle, and generally remain trapped in the cutaneous tissues.

**Pathogenesis**

The larvae migrate in the epidermis just above the basal layer, and rarely penetrate into the dermis. Proteolytic enzymes in larval secretions may cause an inflammatory reaction associated with intense pruritus as the lesion progresses. Although the larvae cannot reach the intestine to complete their life cycle in the unnatural human host, they do occasionally migrate to the lungs where they produce pulmonary infiltrates. Both larvae and eosinophils have been demonstrated in the sputum of patients with pulmonary involvement.

**Host Defenses**

Hypersensitivity to the parasite can occur. Peripheral eosinophilia is common. However, protective immunity does not develop, and repeated infections may occur with subsequent exposure.

**Epidemiology**

Cutaneous larva migrans is primarily a disease of the southern United States, Central and South America, and other subtropical climates. The major agent, *Ancylostoma braziliense*, is a common enteric parasite of dogs and cats. Humans acquire the infection when infected pets deposit feces containing eggs on the soil. Within a few days under favorable conditions of moisture and temperature, these eggs hatch and develop into rapidly growing rhabditiform larvae, which feed on organic matter in the soil, molt, and develop into nonfeeding infective filariform larvae. These larvae remain in the upper half inch of soil, from which they penetrate the skin of humans and other hosts.

This disease is an occupational hazard for construction workers, plumbers and electricians who are exposed to contaminated soil under buildings and crawl spaces. Sunbathers on the beach and children who go barefoot or who play in backyards or sandboxes accessible to infected dogs and cats are other prime candidates. However, anyone who has skin contact with damp soil contaminated with the excreta of infected animals is subject to infection.

**Diagnosis**

The classic serpiginous eruption is usually diagnostic (Fig. 91-4). A biopsy specimen taken at the leading edge of the track may contain the larva.

**Control**

Control of human infections depends on responsible pet ownership. Dogs and cats should be examined periodically for intestinal parasites and wormed as necessary; pet feces should be disposed of in a sanitary way. Topical treatment of cutaneous larva migrans with a 10 percent thiabendazole suspension is usually effective and avoids the
side effects of oral therapy. For multiple or persistent infections, a combination of oral and topical thiabendazole may be given.

**Other Larval Migratory Diseases**

Several other animal parasites have been associated with visceral larva migrans-like syndromes. These include *Ascaris suum*, *Capillaria hepatica*, *Angiostrongylus cantonsensis*, *Angiostrongylus costaricensis*, and *Baylisascaris procyonis*. The tissue phase of such human helminths as *Strongyloides stercoralis* and *Ascaris lumbricoides* can also produce similar clinical syndromes. Larvae of species of *Anisakis* and closely related nematodes of marine mammals have been reported to invade the stomach and other areas of the gastrointestinal tract of humans.

*Ascaris suum*, the common intestinal roundworm of domestic swine, is morphologically very similar to the human roundworm, *Ascaris lumbricoides*. Human infections with *Ascaris suum* are uncommon, but have been associated with a visceral larva migrans syndrome in children. The larvae invade the liver and lungs, but usually do not develop to maturity in the intestine.

*Capillaria hepatica* is a rat liver parasite. If an infected rat is eaten by a predator, the eggs in the liver are released by the digestive process and passed in the feces to the soil. Humans acquire the infection by ingesting the infective eggs in contaminated food or water. The larvae hatch in the intestine and migrate to the liver, where maturation is completed. Clinical manifestations are usually that of an acute or subacute hepatitis. Eosinophilia and massive hepatomegaly may develop. Diagnosis is made by liver biopsy (Fig. 91-5). There is no proven drug therapy.
Gnathostoma spinigerum is a nematode that resides in the stomach wall of dogs and cats. Most human infection occurs in Thailand and other Asian countries. Infective larvae develop in copepods and are transferred through the food chain. Human infection results from consumption of improperly cooked fish or other food containing infective larvae. The larvae migrate in the tissues and may invade the eyes, brain, or any organ. They may cause eosinophilic meningitis. The immature worm may be demonstrated in subcutaneous nodules. Surgical removal of the larva and treatment with albendazole is recommended.

Angiostrongylus cantonensis, the rat lungworm, causes eosinophilic meningitis and ocular disease in Southeast Asia, the Pacific Islands, and Cuba. Hawaii is the only endemic site in the United States. Human infections are caused by eating infected snails, slugs, or other mollusk intermediate hosts, or other members of the food chain that have acquired infective larvae by eating these hosts. The larvae migrate to the brain, producing an eosinophilic meningitis. Paresthesias and ocular palsies are common. There is no specific treatment, but mebendazole has been used. The prognosis is usually favorable.

Angiostrongylus costaricensis is a parasite of the mesenteric arteries of wild rats. The parasite is widespread from Mexico to Brazil; it is even found in cotton rats in Texas. Most reported cases of human disease have occurred in children from Costa Rica. Humans become infected by eating raw vegetables that have been contaminated by a
slug intermediate host with infective third-stage larvae. The larvae mature in the mesenteric arteries, producing a granulomatous inflammatory reaction. Abdominal pain and a mass in the right iliac fossa, the usual clinical manifestations, simulate appendicitis. Definitive diagnosis is usually made by surgical exploration; however, this is obviously a fortuitous finding and is not a routine procedure. A visceral larva migrans syndrome has been reported, with migration of the parasite to the liver. Thiabendazole has been used for treatment.

*Baylisascaris procyonis*, the common raccoon ascarid, may cause an especially virulent form of visceral larva migrans. The infection may be acquired by ingestion of the eggs passed in animal feces or by ingestion of paratenic hosts (host in which parasite survives without further development) bearing the encysted larvae. *Baylisascaris procyonis* has a tendency to invade the central nervous system of humans and other animal hosts. In two reported cases, young children died of visceral larva migrans syndrome and eosinophilic meningoencephalitis. Larvae were demonstrated in brain and other organs at autopsy.

Species of *Anisakis* and certain related genera have been reported in the stomach and other areas of the alimentary tract of humans. Some 25 to 50 cases of infection due to the anisakine species have been recognized in the United States. With the rising popularity of raw fish as a delicacy, the list of parasitic worms and the risk of infection is increasing. Humans acquire this infection by eating raw seafood containing the larvae of these nematodes. The larvae penetrate the gastric mucosa and elicit an intense inflammatory response, gastric pain, vomiting, and diarrhea. Many infections are eliminated by regurgitation of the worm. Diagnosis is by examination of vomitus or by gastroscopic examination and surgery when indicated. In nature, these species develop to adults in the stomach of marine mammals. Eggs passed in the feces hatch and are ingested by crustaceans in which larvae develop into a stage infective for fish. Ingestion of infected fish by marine mammals completes the life cycle.

The risk of infection may be avoided in humans by cooking fish at 65° C for 10 minutes or by freezing fish for at least five days at -20° C.

**Trichinosis**

Trichinosis is acquired by eating raw or inadequately cooked meat that contains encysted larvae of the nematode *Trichinella spiralis*. Any carnivorous mammal can be infected. *Trichinella* occurs worldwide except for Australia and a few Pacific Islands.

**Clinical Manifestations**

The severity of the disease is proportional to the number of larvae ingested. In heavy infections, the clinical symptoms correlate with the biologic stages of *Trichinella* as it completes its life cycle. During the intestinal phase there may be abdominal discomfort and diarrhea. Within 1 week to 10 days after infection, the larvae begin to migrate, and eosinophilia, periorbital edema, and myalgias usually develop as a result of a diffuse inflammatory and allergic response. A wide range of associated symptoms may appear because any organ may be invaded in addition to striated muscle. Muscle involvement may be associated with muscle pain and edema, and is indicated by elevated serum levels of muscle enzymes (e.g., creatine kinase and serum glutamic oxaloacetic
transaminase). The diaphragm, intercostal muscles, tongue, and facial muscles are often involved. Urticaria and conjunctival or subungual splinter hemorrhages are common. Although most infections are self-limited, serious complications or death may result from invasion of the heart, lungs, or central nervous system.

Structure

*Trichinosis spiralis* is a parasite of carnivorous animals. The adult viviparous female (4 mm x 60 μm) is larger than the male (1.5 mm x 40 μm) and may produce 1,000 to 10,000 larvae during her 6-week lifespan. The infective larvae (about 1 mm long) become encysted in striated muscle where they may retain their viability and infectivity for years.

Multiplication and Life Cycle

*Trichinella spiralis* completes its life cycle within one animal host. When striated muscle containing encysted infective larvae is ingested, larvae are released from the cyst by gastric acid and pepsin to mature and reproduce in the small intestine of the host (Fig. 91-6).

FIGURE 91-6 Life cycle of *Trichinella spiralis* in humans.

Pathogenesis

Trichinosis is acquired by eating inadequately cooked meat, mainly pork, containing the encysted larvae of the nematode *Trichinella spiralis*. The severity of the disease is proportionate to the number of larvae ingested. The encysted larvae are released in the
small intestine, where maturation to the adult stage occurs within a period of 2 to 6 days. The adult worms burrow into the intestinal mucosa, where the viviparous female gives birth to the larvae. The enteric phase is completed within 1 month. The larvae are disseminated throughout the body via the circulatory system. Any organ may be invaded, with the potential for serious complications, but the larvae survive and encapsulate only in striated muscle. Larval encystment in muscle usually begins at about 3 weeks and is completed by 3 months. Calcification may take place within 6 to 9 months. Humans are usually an end-stage host.

**Host Defenses**

Many immunologic responses may play a role in decreasing the severity of *Trichinella* infections. Massive eosinophilia, hypergammaglobulinemia with markedly elevated IgE, and circulating immune complexes may accompany infection. Activated macrophages also appear to be involved in host defense. Although these responses are poorly understood in humans, animal experiments suggest the rate of expulsion of the adult worms from the intestine depends on B and T lymphocyte function.

Serum antibodies (IgM, IgG, and secretory IgA) have also been shown to inhibit production of larvae by mature worms. Other defenses act against the migrating larvae, which do not possess the thick cuticle of the adult. They may be exposed to or elicit a variety of humoral and cellular effector mechanisms during the migratory phase. Host resistance has been shown to be enhanced by cell-mediated immunity.

**Epidemiology**

The incidence of *Trichinella* infections has declined in recent years; fewer than 100 cases per year are currently reported in the United States. Most cases are acquired from infected pork that is inadequately cooked. The most common way that pigs become infected is by ingestion of garbage containing meat scraps. Certain ethnic groups whose culinary preferences include raw pork are at a special risk. The custom of sampling raw homemade sausage for flavor is a recognized cause of infection in the Southeastern United States. Inspection for *Trichinella* larvae is not included under the United States Department of Agriculture specifications for pork products. Many consumers are unaware that the stamp "US Inspected and Passed" on raw pork products does not include inspection for *Trichinella*. Although cattle are herbivorous and consequently not a reservoir for *Trichinella*, beef products or horse meat may be contaminated by meat grinders also used for pork. According to USDA specifications, *Trichinella* are eliminated from pork products when the products are heated to an internal temperature of at least 137° F (58.3° C) or when frozen at 5° F (-15° C) for 20 days. Freezing may not be adequate to eliminate cold-resistant strains of *Trichinella*, however. Wild game is another minor source of *Trichinella* infection in humans. *Trichinella spiralis* is maintained in nature by passage among carnivorous or omnivorous animals.

It appears that, in addition to *Trichinella spiralis*, some other *Trichinella* species are of minor epidemiologic importance to man. These species resemble *T spiralis* in morphology, but differ in biologic properties. They include *T nativa*, a variant especially resistant to freezing which is found in Arctic regions and infects humans through ingestion of polar bear meat, and *T nelsoni*, which is found in Africa and tropical regions and infects man through ingestion of wild pig meat.
Diagnosis

Trichinosis is suggested by a history of eating undercooked pork and by the distinctive clinical features in association with eosinophilia. Any leftover, suspect meat should be examined for *Trichinella* larvae. Serum antibodies are not usually detectable before 3 weeks. A number of serodiagnostic tests, including the bentonite flocculation test, ELISA latex agglutination, fluorescent antibody, and complement fixation tests have facilitated diagnosis. The bentonite flocculation test, a reliable serodiagnostic method, is positive in over 90 percent of cases. Serum specimens may be submitted through State Public Health Departments, which send them to the Center for Disease Control for the test. Definitive diagnosis is by biopsy of striated muscle (Figure 91-7). The biopsy site should be near a tendinous insertion of an involved muscle, the area where the larvae concentrate. Histopathologic examination for larvae should be done. Also, a specimen of muscle should be examined fresh by compressing it between two microscope slides and examining it under a microscope; this simple technique may reveal larvae. Treatment of a portion of the muscle for several hours with pepsin and hydrochloric acid to liberate the encysted larvae, followed by microscopic examination of the concentrated sediment for larvae, may improve the diagnostic yield.

FIGURE 91-7 Encapsulated *Trichinella spiralis* larva in muscle from experimental animal (x200).

Control

Trichinosis can be prevented by adequately cooking pork or wild game. Freezing of pork at -15°C for 20 days will also eliminate the infectivity of most larvae, but not necessarily of cold-resistant *Trichinella* strains. There is no proven effective therapy for
trichinosis, but mebendazole is thought to be an effective prophylactic agent for persons known to have ingested infected meat. Thiabendazole and corticosteroids have been used in serious infections. It has been suggested that some of the benefits of thiabendazole may result from its anti-inflammatory effects.

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