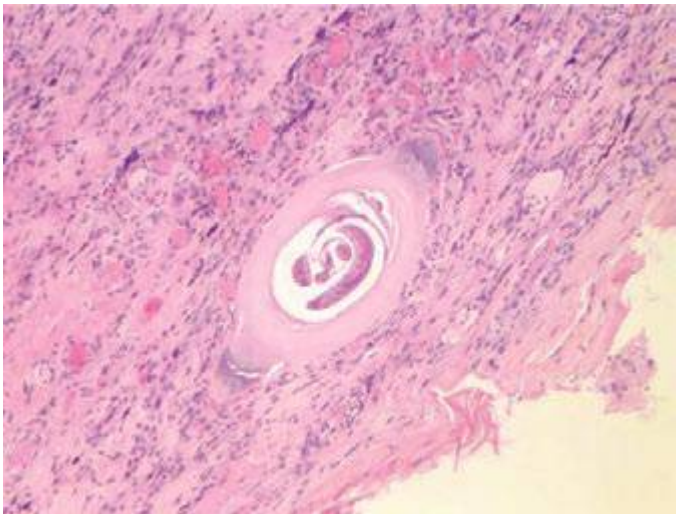

**FIGURE 256-2** ***Trichinella*** **larva** encysted in a characteristic hyalinized capsule in striated muscle tissue. *(Photo/Wadsworth Center, New York State Department of Health. Reprinted from MMWR 53:606, 2004; public domain.)*

of infection, confirms the diagnosis. Alternatively, a definitive diagnosis requires surgical biopsy of at least 1 g of involved muscle; the yields are highest near tendon insertions. The fresh muscle tissue should be compressed between glass slides and examined microscopically (Fig. 256-2), because larvae may be missed by examination of routine histopathologic sections alone.

## TREATMENT TRICHINELLOSIS

Most lightly infected patients recover uneventfully with bed rest, antipyretics, and analgesics. Glucocorticoids like prednisone (Table 256-1) are beneficial for severe myositis and myocarditis. Mebendazole and albendazole are active against enteric stages of the parasite, but their efficacy against encysted larvae has not been conclusively demonstrated.

**Prevention** Larvae may be killed by cooking pork until it is no longer pink or by freezing it at −15°C for 3 weeks. However, Arctic *T. nativa* larvae in walrus or bear meat are relatively resistant and may remain viable despite freezing.

**TABLE 256-1 THERAPY FOR TISSUE NEMATODE INFECTIONS**

| Infection | Severity | Treatment |
|---|---|---|
| Trichinellosis | Mild | Supportive |
| | Moderate | Albendazole (400 mg bid × 8–14 days) or |
| | | Mebendazole (200–400 mg tid × 3 days, then 400 mg tid × 8–14 days) |
| | Severe | Add glucocorticoids (e.g., prednisone, 1 mg/kg qd × 5 days) |
| Visceral larva migrans | Mild to moderate | Supportive |
| | Severe | Glucocorticoids (as above) |
| | Ocular | Not fully defined; albendazole (800 mg bid for adults, 400 mg bid for children) with glucocorticoids × 5–20 days has been effective |
| Cutaneous larva migrans | | Ivermectin (single dose, 200 μg/kg) or |
| | | Albendazole (200 mg bid × 3 days) |
| Angiostrongyliasis | Mild to moderate | Supportive |
| | Severe | Glucocorticoids (as above) |
| Gnathostomiasis | | Ivermectin (200 μg/kg per day × 2 days) or |
| | | Albendazole (400 mg bid × 21 days) |

### VISCERAL AND OCULAR LARVA MIGRANS

Visceral larva migrans is a syndrome caused by nematodes that are normally parasitic for nonhuman host species. In humans, these nematode larvae do not develop into adult worms but instead migrate through host tissues and elicit eosinophilic inflammation. The most common form of visceral larva migrans is toxocariasis due to larvae of the canine ascarid *Toxocara canis*; the syndrome is due less commonly to the feline ascarid *T. cati* and even less commonly to the pig ascarid *Ascaris suum*. Rare cases with eosinophilic meningoencephalitis have been caused by the raccoon ascarid *Baylisascaris procyonis*.

**Life Cycle and Epidemiology** The canine roundworm *T. canis* is distributed among dogs worldwide. Ingestion of infective eggs by dogs is followed by liberation of *Toxocara* larvae, which penetrate the gut wall and migrate intravascularly into canine tissues, where most remain in a developmentally arrested state. During pregnancy, some larvae resume migration in bitches and infect puppies prenatally (through transplacental transmission) or after birth (through suckling). Thus, in lactating bitches and puppies, larvae return to the intestinal tract and develop into adult worms, which produce eggs that are released in the feces. Eggs must undergo embryonation over several weeks to become infectious. Humans acquire toxocariasis mainly by eating soil contaminated by puppy feces that contains infective *T. canis* eggs. Visceral larva migrans is most common among children who habitually eat dirt.

**Pathogenesis and Clinical Features** Clinical disease most commonly afflicts preschool children. After humans ingest *Toxocara* eggs, the larvae hatch and penetrate the intestinal mucosa, from which they are carried by the circulation to a wide variety of organs and tissues. The larvae invade the liver, lungs, central nervous system (CNS), and other sites, provoking intense local eosinophilic granulomatous responses. The degree of clinical illness depends on larval number and tissue distribution, reinfection, and host immune responses. Most light infections are asymptomatic and may be manifest only by blood eosinophilia. Characteristic symptoms of visceral larva migrans include fever, malaise, anorexia and weight loss, cough, wheezing, and rashes. Hepatosplenomegaly is common. These features may be accompanied by extraordinary peripheral eosinophilia, which may approach 90%. Uncommonly, seizures or behavioral disorders develop. Rare deaths are due to severe neurologic, pneumonic, or myocardial involvement.

The ocular form of the larva migrans syndrome occurs when *Toxocara* larvae invade the eye. An eosinophilic granulomatous mass, most commonly in the posterior pole of the retina, develops around the entrapped larva. The retinal lesion can mimic retinoblastoma in appearance, and mistaken diagnosis of the latter condition can lead to unnecessary enucleation. The spectrum of eye involvement also includes endophthalmitis, uveitis, and chorioretinitis. Unilateral visual disturbances, strabismus, and eye pain are the most common presenting symptoms. In contrast to visceral larva migrans, ocular toxocariasis usually develops in older children or young adults with no history of pica; these patients seldom have eosinophilia or visceral manifestations.

**Diagnosis** In addition to eosinophilia, leukocytosis and hypergammaglobulinemia may be evident. Transient pulmonary infiltrates are apparent on chest x-rays of about one-half of patients with symptoms of pneumonitis. The clinical diagnosis can be confirmed by an enzyme-linked immunosorbent assay for toxocaral antibodies. Stool examination for parasite eggs is worthless in toxocariasis, since the larvae do not develop into egg-producing adults in humans.

## TREATMENT VISCERAL AND OCULAR LARVA MIGRANS

The vast majority of *Toxocara* infections are self-limited and resolve without specific therapy. In patients with severe myocardial, CNS, or pulmonary involvement, glucocorticoids may be employed to reduce inflammatory complications. Available anthelmintic drugs,