CLINICAL MICROBIOLOGY REVIEWS, Jan. 1996, p. 47–54 Vol. 9, No. 1
0893-8512/96/$04.00+0
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Clinical Aspects of Infection with *Trichinella* spp.

VIRGINIA CAPÓ[1] AND DICKSON D. DESPOMMIER[2]*

Pathology Department, Institute of Tropical Medicine Pedro Kouri, Havana, Cuba,[1] and Division of Environmental Sciences and Department of Microbiology, Columbia University, New York, New York[2]

INTRODUCTION

Outbreaks of infection with the parasitic nematode *Trichinella spiralis*, genus *Trichinella*, occurred with great frequency in the United States and elsewhere in the world prior to the 1960s (5, 45, 89), altering the course of history, in some cases. For example, one small epidemic in 1897 resulted in the tragic deaths of three Swedish explorers (97), delaying by 12 years the discovery of the geographic North Pole. The parasite was first identified under the microscope in 1835 by Paget and Owen (79). Extensive research on the life cycle and epidemiology followed shortly after its description in the clinical literature as a true pathogen of humans as well as other animals (99, 106).

Extensive research interest in this parasite continues to generate new and exciting results. For instance, we now know that *T. spiralis* is an obligate intracellular parasite (21, 87, 102) in both its larval (striated skeletal muscle cell) and adult (cytoplasm of a row of enterocytes in the small intestine) niches and that it induces a series of changes in both settings, through its secreted proteins, that alter the host, allowing the infection to proceed (19).

While at present the incidence and prevalence of infection due to *T. spiralis* are low in western Europe and the United States, common source outbreaks continue to occur (9, 10, 61, 72, 85, 89), sometimes resulting in death (12, 92) but more often causing clinical conditions that present as baffling signs and symptoms and thus usually go undiagnosed (81). A cursory review of the literature regarding the clinical spectrum of disease makes a collective point: the diagnosis of trichinellosis depends on correlating and identifying the numerous clinical signs, symptoms, and positive laboratory findings with a carefully taken case history and the life cycle of the parasite.

A potential complication in the clinical presentation of infection with members of the genus *Trichinella* is that there are several recognized species that may infect and cause disease in humans (39, 71, 76). Each species has been characterized by a combination of DNA typing (26, 27) and isozyme patterns (82, 83). Precise evaluation of their evolutionary relationships must await analysis of more isolates of each species. DNA and isozyme typing have been used to identify the species responsible for sylvatic (6) or human, e.g., *T. spiralis* or *T. britovi* (85), infections. These techniques, however, are not widely applied in clinical settings, in which identification of *Trichinella* spp. is sufficient to aid the physician in the management of the patient's disease.

According to current nomenclature, there are eight distinct types of species, each of which has been assigned a number (82, 83) (Table 1). *T. spiralis* is designated T_1; *T. britovi*, T_3; *T. nativa*, T_2; and *T. nelsoni*, T_7. Several other species of *Trichinella* have number designations only: T_5, T_6, and T_8. Precisely to which species group they belong has not yet been determined (83, 84). *T. pseudospiralis* (T_4) has a somewhat unusual geographic distribution and infects birds of prey (101) and mammals such as the Tasmanian devil. One human infection with *T. pseudospiralis* has been documented (2).

Within the last 2 years, monographs on clinical aspects of infection with *T. spiralis* (45, 76) and epidemiological descriptions of outbreaks (39, 71, 78) have been published. The scope of this review differs somewhat from those, presenting an extensive summary of the clinical disease states and correlating them with the parasite's life cycle (18). In this way, we hope to provide an overview of the pathogenesis of trichinellosis.

Therefore, despite the facts that the Hubbel space telescope now functions properly, the human genome project is more than half finished, and the majority of households in Plum Tree, England, have a television set and a microwave oven, there still is and always will be an urgent need to recognize disease caused by *Trichinella* spp. and to treat those who are acutely ill from it.

LIFE CYCLE AND EPIDEMIOLOGY

The diagrams in Fig. 1 illustrate the life cycle of *T. spiralis*. The other species and subspecies of *Trichinella* also fit this scheme, save for their reservoir host ranges and geographic distributions (6, 55, 56, 61, 80, 82, 83). *T. pseudospiralis* is largely a parasite of birds (2, 101), although it was first discov-

* Corresponding author. Mailing address: Division of Environmental Sciences and Department of Microbiology, 701 W. 168th St., New York, NY 10032. Phone: (212) 305-5014. Fax: (212) 305-4496. Electronic mail address: despommier@cuccfa.ccc.columbia.edu.

TABLE 1. Important characteristics of *Trichinella* spp.

Species	Geographic distribution[a]	Source of infection[b]	Infectivity[c]	Resistance to freezing[d]	Clinical aspects[e]
T. spiralis (T_1)	Cosmopolitan	Swine, wild boar, bear, horse, fox	High	None	Highly pathogenic; can be lethal; nurse cell formation in 16 days
T. nativa (T_2)	Arctic, subarctic, holoarctic	Bear, horse	High	High	Moderate pathogenicity; long incubation time; prominent symptoms; nurse cell formation in 30 days
T. britovi (T_3)	Temperate zone, Palearctic region	Wild boar, horse	Moderate	None	Moderate pathogenicity; long incubation period; no gastrointestinal symptoms; low muscle larvae invasion; nurse cell formation in 42 days
T. pseudospiralis (T_4)	Cosmopolitan in Palearctic, Nearctic, Oceania	Birds, omnivorous mammals	Moderate	None	Incomplete information: only single human case reported; no nurse cell formation
T_5[f]	Temperate, Nearctic	Bear	Low[g]	Low	No infection in humans reported; no nurse cell formation after 4 mo
T_6[f]	Northern temperate, Nearctic region	Bear	Low[g]	High	No known human cases; nurse cell formation in 32 days
T. nelsoni (T_7)	Tropical	Warthog	High	None	Low pathogenicity; not lethal; nurse cell formation in 40 days
T_8[f]	South Africa	Lion	Low[g]	None	No known human cases; nurse cell formation in 40 days

[a] See Pozio et al. (82 and 83).
[b] See Pozio et al. (82, 83), Ainsworth et al. (2), and Wheeldon et al. (101).
[c] See Murrell and Bruschi (76) and Pozio et al. (83).
[d] See Pozio et al. (83, 84).
[e] See Murrell and Bruschi (76) and Pozio et al. (84).
[f] Reported infectivity in laboratory animals.
[g] Biological characteristic similar to that of *T. britovi.*

ered in the raccoon dog (40) (Fig. 2) in the former U.S.S.R. The temporal aspects of the life cycle and clinical laboratory findings are found in Fig. 3, which shows the approximate days during infection when each stage is present and their anatomic locations within the infected individual.

Life Cycle

Parasites in each of the three phases of the infection cause disease (64): first- through fourth-stage larvae and adults in the enteral phase; first-stage newborn larvae that travel through the bloodstream and enter the tissues in the migratory phase; and first-stage larvae after they enter the tissues in the parenteral phase. The stages of the enteral phase and their importance to diarrheal disease caused by *T. spiralis* have been recently reviewed (20). In brief (Fig. 1), the ingested first-stage larva is freed from the surrounding tissue by the action of pepsin and hydrochloric acid in the stomach. It is then carried to the small intestine, where it invades the columnar epithelium (23, 102). Shortly thereafter, the larva molts four times (10 through 28 h postingestion), transforming into the adult worm (5, 14, 64). Mating ensues (30 to 34 h postingestion), and 5 days later newborn larvae are shed (64). The number of newborn larvae produced depends on the immune status of the infected host (18, 100) and on the infecting species of parasite (84, 85). It is estimated that 500 to 1,500 newborn larvae are shed over the life span of an adult female worm before a combination of immune responses forces their expulsion from the small intestine (100). Immunity appears to be lifelong and is thought to involve the synergistic action of humoral and cell-mediated mechanisms. In rats, eosinophils and immunoglobulin E (IgE) antibodies directed against the secretions of the first-stage larva as it enters an already immune host prevent reinfection (1), but the actual mechanism(s) of worm elimination during a primary infection is still under intense investigation. Nothing is known regarding the immune elimination of *T. spiralis* from the human host.

Epidemiology

Superb descriptions of the epidemiology of *Trichinella* spp. infection can be found in several reviews (13, 55, 76). Nonetheless, the patterns of acquisition of this parasite continue to vary throughout the world, and thus some discussion of this aspect is warranted.

The only way the parasite can gain entrance into a host is by being ingested along with raw or partially cooked, infected muscle tissue (save for laboratory accidents!), and therefore carnivorism is the key to understanding the parasite's epidemiology. In the wild, the vast majority of all carcasses are consumed by scavengers, and thus infection is widespread within a given biome (28, 52, 53, 55, 71, 86, 93, 95) and throughout the world (15, 78, 104). A list of naturally infected animals takes an astounding 5 pages to complete in Campbell's treatise (13). In fact, only one other parasite, *Toxoplasma gondii*, is found more ubiquitously (67, 92, 94), and it, too, is transmitted primarily by carnivorism (37). Even sea mammals and herbivores have been found infected with *Trichinella* spp., thus redefining the dietary habits of the latter group (13, 50, 71). Accidental infection of some domesticated herbivores also occurs, but rarely (7, 8). In one outbreak five people died and several thousand others were infected when an infected horse from Texas was shipped to Paris, France, and consumed as "steak tartare" (4). The parasites used that animal as their "Trojan horse" to enter their hosts!

CLINICAL FINDINGS

T. spiralis outbreaks occur most frequently within a community or among family members (35, 42, 45, 57, 58, 81, 89). Since there are so many variables modifying the clinical picture of

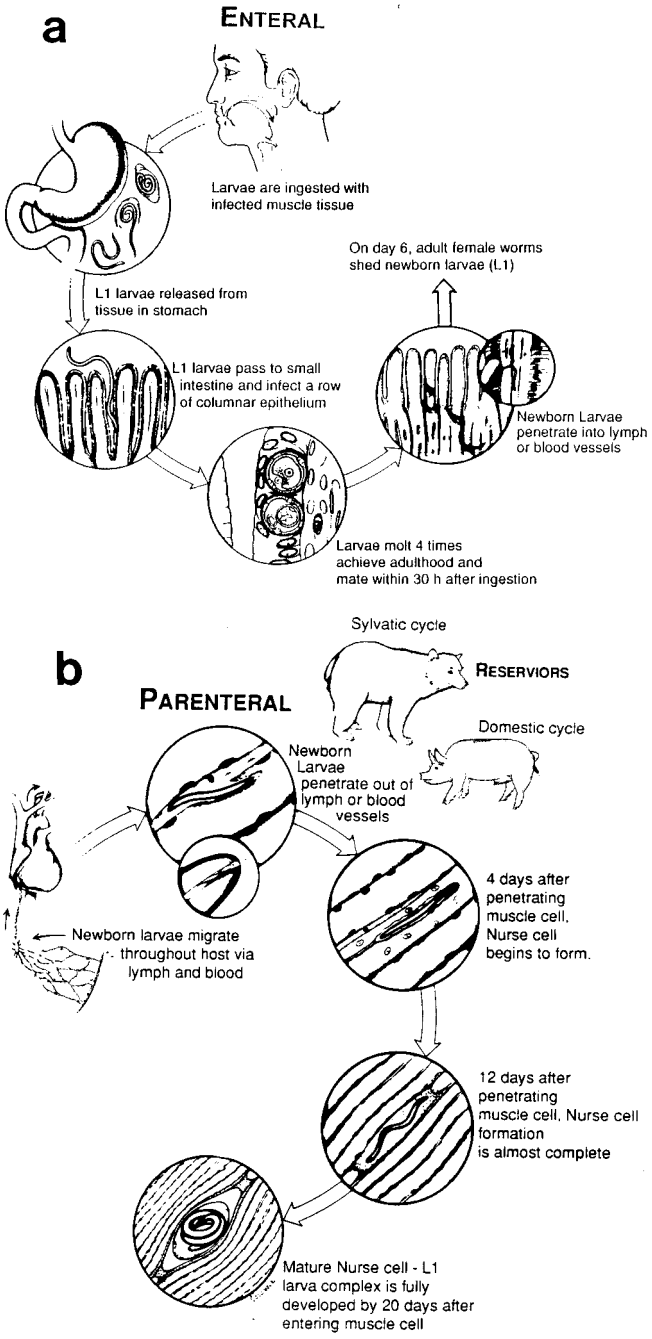

FIG. 1. (a) Enteral phase of *T. spiralis*. (b) Parenteral phase of *T. spiralis*. Reprinted from reference 22.

trichinellosis, single individuals acquiring the infection (16, 69, 78, 88, 103) challenge clinicians who are unaware of the life cycle and epidemiology of *T. spiralis*. The symptoms mimic those of many other diseases (16, 76, 88). The duration of the incubation period is related to the number of larvae ingested, which, in turn, usually determines the severity of disease (52, 93). Patients also vary in their symptoms according to time elapsed from the ingestion of infected meat and the infecting species of *Trichinella* (76). In addition, host immunity, age, sex, and general health of the infected individual are important factors in the outcome of disease (42, 56, 81).

Figure 3 summarizes the clinical and subclinical presentation of infection with *T. spiralis*. These data were averaged from a number of documented outbreaks with clinical follow-up (3, 4, 8, 9, 15, 16, 25, 31, 32, 36, 42, 45, 46, 51, 52, 54, 57, 58, 68–78, 80, 81, 88, 90, 98, 103, 105). As can be readily seen, there are symptoms that highly correlate with the stage of the infection (i.e., enteral phase) and are related to the presence of the parasite's infective form and those that correlate with the parenteral phase (e.g., inflammatory and allergic responses due to tissue invasion by larvae).

Enteral Phase

Most individuals who become infected in an outbreak after eating contaminated meat are asymptomatic, as are the majority of patients in single sporadic cases who, at most, experience mild transient diarrhea and nausea (42, 51, 57, 76, 78, 81) related to the penetration of the intestinal mucosa. Thus, the first week of the enteral phase in patients with moderate to severe infection is associated with upper abdominal pain, diarrhea or constipation, vomiting, malaise, and low-grade fever, all of which can vary in severity and last only a few days (Fig. 3) (42, 45, 76, 81). This clinical presentation is characteristic of many enteral disorders (e.g., food poisoning or uncomplicated indigestion), and thus it is easily misdiagnosed (57, 103). Patients usually do not seek medical advice at this time in their infection and request medical attention only when the nature of the symptoms changes with the onset of the parenteral (systemic) phase (57, 105).

Parenteral Phase

During weeks 2 through 6 after infection, the enteral phase is still present, but symptoms that correlate with intestinal disease abate (Fig. 3). At this time, signs and symptoms due to the migratory stage, the newborn larva, develop. In mild infection resulting from the ingestion of low numbers of larvae in muscle, symptoms related to the migratory and parenteral phases are usually the first to be clinically detected since these patients experience no symptoms during the enteral phase (42, 57, 76, 78, 81).

Mild to moderate infection can produce the following signs and symptoms: diffuse myalgia in 30 to 100% of patients; a paralysis-like state (10 to 35%); periorbital and/or facial edema (15 to 90%); conjunctivitis (55%); fever (30 to 90%); headache (75%); skin rash (15 to 65%); difficulties in swallowing (35%)

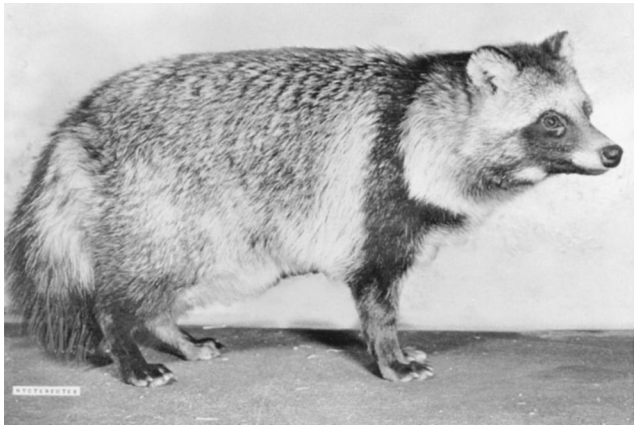

FIG. 2. The raccoon dog, found throughout Europe and eastern Asia, is one of the principal hosts for *T. pseudospiralis*. Photograph by Ernst P. Walker. Reprinted from reference 77a with permission of the publisher.

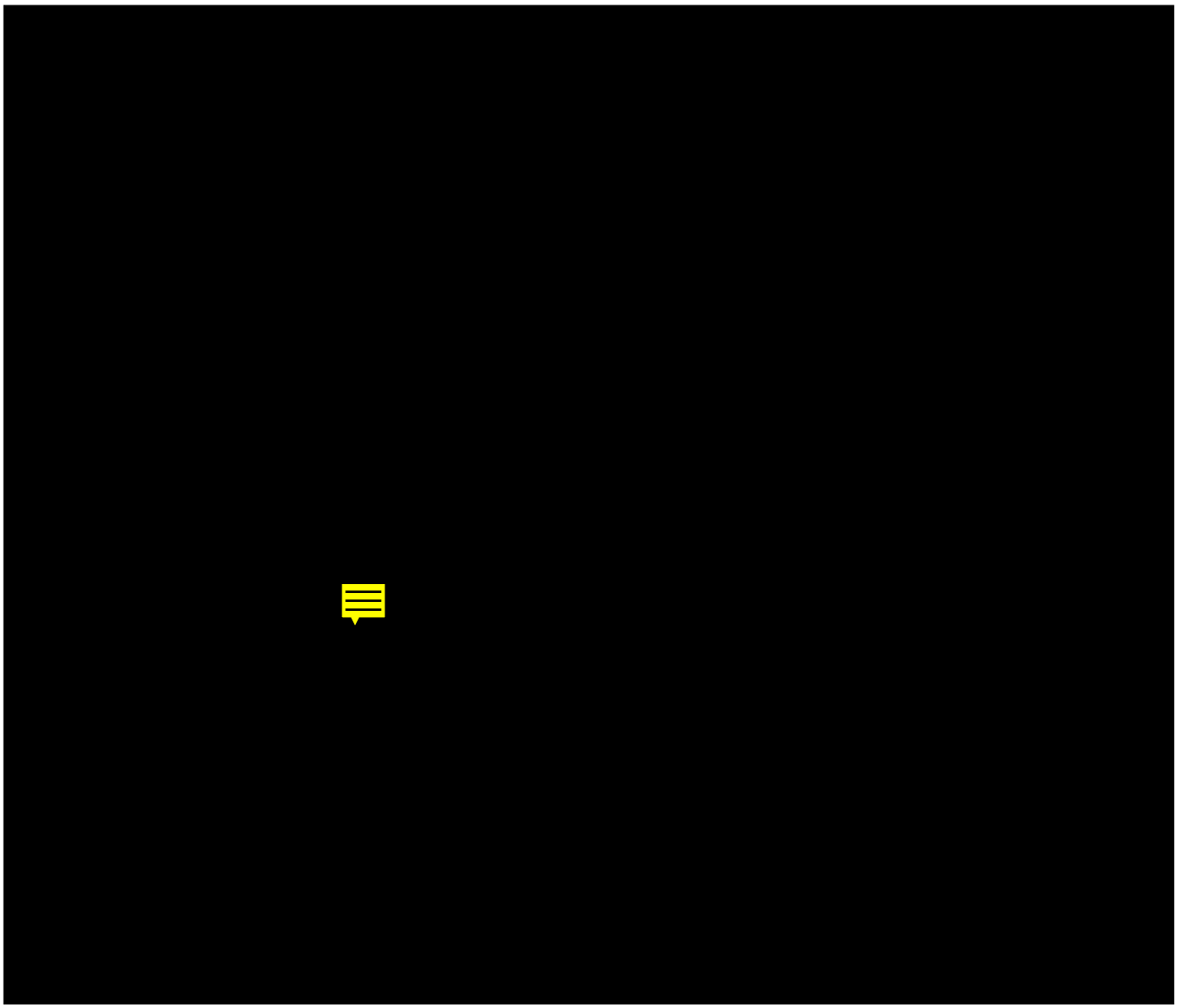

FIG. 3. Comprehensive summary of the main clinical signs and symptoms, laboratory findings, and diagnostic test results for patients suffering from mild (light color), moderate (more intense color), and severe (most intense color) clinical trichinellosis. The labels on the left indicate qualitative aspects of the infection (e.g., fever, muscle pain, etc.), while those on the right give some indication as to the quantitative aspect of each. The colors chosen are matched to the stage of the infection (e.g., green indicates all signs and symptoms in the enteral phase of the parasite). When the color is faded in and out, it indicates that that particular qualitative aspect is gradual in onset. The shaded portion (vertical shading between weeks 3 and 6) correlates with the period of infection in which the death of the patient usually occurs if the dose of parasite ingested is high enough to be lethal. Ab, antibody; Ag, antigen.

or in opening the mouth; insomnia; weight loss; peripheral nerve sensations; hot flashes; coryza; hoarseness (5 to 20%); bronchitis (5 to 40%); splinter hemorrhages of the nail beds and/or the retinae; visual disturbances; and paralysis of the ocular muscles. These data were derived from the numerous clinical outbreaks cited above. All signs and symptoms are either directly or indirectly due to the indiscriminate penetration of tissues by the migrating newborn larva. After the second week of the parenteral phase of infection, most patients have developed specific serum antibodies against the secreted antigens of the larvae in muscle (Fig. 3) (68).

Patients with severe infection are often the index cases of an epidemic. They are the first to be diagnosed because of their presentation with the classical signs and symptoms of the disease. Their symptoms are more prominent than are those of patients suffering from milder or moderate infection. High fever and elevated levels of circulating eosinophils (30 to 60% or more), severe muscle pain, skin rash, and headaches, as well as swelling of eyelids, face, or extremities, are characteristic of these cases. Patients may develop neurologic manifestations that rarely appear before the end of the second week of infection and provoke distress. Headache, vertigo and tinnitus, deafness, aphasia, convulsions, and abnormalities related to peripheral reflexes, among others, are frequent complaints or signs found in severely infected individuals. Generally, patients are alert but apathetic, and prolonged insomnia affects their behavior, causing them to become irritable. Other neurologic symptoms such as meningitis, encephalitis, and/or hemiplegia may develop in relation to diffuse damage of brain tissue due to occlusion of arteries or to granulomatous inflammation (32, 81). These symptoms need to be treated immediately with steroids, since they are due to inflammation resulting from

tissue damage caused by large numbers of migrating newborn larvae.

In moderate to severe infection, symptoms due to invasion of muscle cells (e.g., weakness, pain, paralysis, and photophobia) increase during the third week, and edema of the face, eyelids, hands, and feet becomes a prominent feature (Fig. 3) (9, 25, 39, 52, 98). The patient's breathing is often difficult and shallow. Dysphagia and hoarseness are also frequent consequences of clinical infection. Thrombosis of arterioles may occur, probably as a result of the hypercoagulability associated with eosinophilia (36, 74, 96). During this time, clinical signs and symptoms can lead to a misdiagnosis because trichinellosis is often confused with angioneurotic edema, serum sickness, septicemia, periarteritis nodosa, allergic reactions to food or drugs, coronary thrombosis, typhoid fever, infection by a *Toxocara* sp., autoimmune diseases, eosinophilic syndrome, and recently, chronic fatigue syndrome (59). Poliomyelitis, meningitis, encephalitis, cerebral hemorrhage, multiple neuritis, pneumonia, bronchopneumonia, pleurisy, spastic bronchitis, glomerulonephritis, and "ciguatera," a condition induced by eating certain tropical saltwater fishes found mostly in the Caribbean and Indo-Pacific regions (49, 63), should also be included in the differential diagnosis.

Endocarditis, myocarditis, and even cardiac failure in fatal cases are attributed to the damaging effects of the migratory phase of the infection (42). All symptoms associated with acute illness progressively diminish at the onset of convalescence (i.e., between weeks 5 to 6 after ingestion of infected meat), but dyspnea, edema of the extremities, and bronchitis persist to the sixth to eighth week (Fig. 3) (42). Infectious first-stage larvae remain in their nurse cells for months to years following recovery (81), but after some time a portion of them die (63). This process may be more rapid for non-*T. spiralis* infections (83). Dead nurse cell-parasite complexes become highly calcified, but whether or not the calcification process results in the death of the worm is still not resolved by experimental evidence.

Complications

Complications arising during acute illness include abrupt delivery of stillbirths by infected pregnant women (42, 81), and vertical infection of the fetus has been described (42, 81). During convalescence, the patients may complain of hearing disorders, weight loss, and disturbances of menstruation. Loss of hair and nails, skin desquamation, hoarseness, aphonia, and muscle stiffness have been reported (42).

Death may occur from heart failure or central nervous system failure during the third to fifth weeks of the infection (42, 81). Myocarditis, encephalitis, pneumonitis, hypokalemia, adrenal gland insufficiency, and obstruction of blood vessel circulation have been given as causes of death in these critically ill patients (42, 81).

There is still no general agreement (51, 81) as to whether patients suffer from long-term effects of harboring nurse cell-larva complexes. Two independent studies on well-characterized outbreaks (34, 44) recently addressed this issue. In these two studies, patients who suffered severe disease, regardless of age, went on to experience long-term effects, such as a generalized myalgia (84 and 90%), ocular symptoms (e.g., conjunctivitis, difficulty in focusing, or "burning sensation" [63 and 59%]), and neuropathies of various types (35 and 52%). These conditions can persist for up to 10 years after recovery (44).

Species of *Trichinella* other than T_1 cause slightly different patterns of clinical disease in moderate to heavy infection. For example, patients suffering from clinical infection with *T. nativa* (T_2) experience symptoms related only to the enteral phase of the infection, and the onset is delayed compared with that of *T. spiralis* infection (Table 1). *T. nativa* may cause the death of the individual but paradoxically produces almost no parenterally related symptoms, making its early diagnosis even more difficult. *T. nelsoni* (T_7) is apparently of low pathogenicity in both its enteral and parenteral phases, and *T. britovi* (T_3) is similar to *T. nelsoni* (55, 76, 80). Patients infected with *T. britovi* are reported as having a milder disease with a long incubation period and few if any intestinal symptoms (82), despite the size of the inoculum compared with a similar *T. spiralis* infection (85). More documented cases of *T. britovi* must be studied before a complete clinical description of infection with this species can be given.

In the Arctic, the clinical presentation may differ from previously reported descriptions of classic trichinellosis. Some patients have a chronic diarrheal presentation that may suggest either an infection with a different species of *Trichinella* or a reinfection of an immune human population (71).

LABORATORY FINDINGS

Leukocytosis (12,500 to 18,000 cells per mm^3) is common early, with a predominance of circulating eosinophils (9, 15, 25, 45, 52, 57, 58, 74, 80, 81) of about 1,400 to 8,700 eosinophils per mm^3 (36, 41, 58). Thus, eosinophilia is the earliest and most characteristic laboratory finding of trichinellosis (Fig. 3) (81) and is correlated with the intensity of infection. Even among asymptomatic cases eosinophilia reaches modest levels (5 to 15% of leukocytes). A sudden reduction in the level of circulating eosinophils to 1% or none is an indication of severe infection and may even signal the onset of death of the patient (42, 58). Only the adult stage of trichinella has been shown to elicit eosinophilia. Eosinophilia is maximum during the third to fourth week and usually stabilizes at this time. These cells infiltrate the infected portion of intestinal tissue and locate adjacent to the adult worms and enter into damaged muscle tissue after newborn larvae penetrate them (11, 24). It is not known what role these cells play in human infection with this parasite.

Another positive laboratory finding is an elevation in circulating levels of muscle enzymes (e.g., creatinine phosphokinase [CPK], 1,6-diphosphofructoaldolase, lactate dehydrogenase aldolases, and aminotransferases [76]). They may be elevated in 35 to 100% of infected individuals and are present in serum due to the destruction of muscle tissue by migrating newborn larvae (9, 15, 25, 39, 59, 74, 80, 81, 85).

DIAGNOSIS

A muscle biopsy (2 to 4 mm^3), in which the piece of tissue is pressed between two slides and viewed under the microscope, will usually reveal larvae in heavy infections (29, 76, 79, 81) and is thus the most direct measure of the presence of infection. If the diagnosis is attempted before larvae begin to coil (i.e., up to 2 weeks after larvae enter the muscle cell), then there is the risk of confusing the worm with fragments of muscle tissue.

Alternatively, digesting a finely minced portion of the biopsy material in 1% HCl–1% pepsin for 1 h at 37°C will release the larvae from their nurse cells and make them more easily observed under the microscope (29, 76). Unfortunately, this method, while good for detecting older larvae that are not susceptible to the digestion procedure, is not useful in detecting young larvae that can be destroyed by this process.

Routine histopathological examination of the biopsy sample is another method of demonstrating the presence of muscle

larvae. Even if larvae are not seen in histological sections, infected muscle cells undergo basophilic changes once they are penetrated by the newborn larvae, providing a clue to their presence. This change is easily noted on standard hematoxylin-and-eosin-stained sections because the pattern of striation (i.e., actin and myosin filaments) disappears by the fourth to fifth day after infection (87). Absence of larvae in sections or changes in muscle tissue, however, do not rule out infection, since infection may be light or larvae may simply be missed because of their uneven distribution in muscle tissue.

On histopathologic section, it is possible in some cases to determine whether or not the infection is recent or old by observing several characteristics of the nurse cell-parasite complex. The absence of a capsule and the presence of straight (i.e., nonspiraled) worms in the complex indicate that the infection is ongoing. A mature capsule and a coiled parasite indicate an older infection that may have been acquired sometime previous to admission to the clinical setting.

Detection of circulating antigens by immunoassay techniques, although not available in most laboratories, can be useful for the diagnosis during the beginning of the parenteral phase, when standard serological tests designed to detect specific antibodies have yet to become positive (Fig. 3) (38, 48, 77). Although detection of circulating antigen might be a useful confirmatory test (30), circulating antigen is not detected in every patient and its detection is therefore of limited value to the clinician.

DNA-based tests have also been reported (64, 83). DNA sequences amplified by PCR have been identified and are specific for *T. spiralis* and other *Trichinella* species as well. This new generation of diagnostic test is not yet available commercially.

Antibody detection tests are useful adjuncts to diagnosis starting on about day 12 after infection (Fig. 3). By 14 days, when most patients suffering from clinical symptoms seek medical assistance, immunofluorescence-based assays and enzyme-linked immunosorbent assays (ELISA) for IgG antibodies may be positive (29, 48, 53, 58, 68) and remain positive for years (38). The sensitivity of the IgG-ELISA reaches 100% on day 50. The test remains positive for more than 2 years in 88% of infected people (Fig. 3) (75). Other immunoglobulins (e.g., IgA and IgE) behave in a similar manner, but tests to detect them have a lower sensitivity. The indirect hemagglutination test may be a useful alternative for diagnosing trichinellosis, as 95% of 60 known positive samples were positive by this test, whereas the precipitin and the bentonite flocculation tests were positive in 93.2 and 43.9% of the same samples, respectively (90).

TREATMENT AND PREVENTION

Treatment

When the life of the patient is threatened by overwhelming infection, intensive care treatment with all available supportive therapies is mandated (i.e., fluid replacement, steroids, and treatment for shock, toxemia, and circulatory and cardiac failure) (81). Specific treatment for the parasite with various benzimidazoles (mebendazole or albendazole) is also necessary. Immunosuppression due to steroids, although often a life-saving procedure, prolongs the life of the adult parasites as well and results in further production of newborn larvae if unchecked. As already mentioned, patients may harbor adults shedding newborn larvae for several weeks during the acute phase of infection. Mebendazole (200 mg/day for 5 days) or albendazole (400 mg/day for 3 days) should be given to adults (except pregnant women), as well as to children (5 mg per kg of body weight per day for 4 days) (22, 45, 57). Prednisolone at 40 to 60 mg/day alleviates the fever and the side effects of inflammation due to the cell damage that results from larval penetration into the tissues. These symptoms usually disappear within days after the initial dose is given (81). Prolonged treatment with steroids is not recommended, although symptoms may recur when treatment is suspended (81). Long-term sequelae must be treated symptomatically as they arise.

Prevention

The consumption of raw or rare infected meat from game animals or from pigs raised in situations that favor the existence of rodent populations is the most frequent source of infection by any species of *Trichinella* (85). Infection of pig herds by *T. spiralis* is usually perpetrated by the animals scavenging on infected rodent populations or, less commonly, by cannibalism of sick animals (13, 17, 43, 65, 76, 91). Immune pigs experiencing a second infection expel some of their worm burden soon afterwards as first-stage infective larvae, and it is therefore suspected that coprophagy within the barnyard community of pigs may be yet another means by which naive animals are infected.

Feeding of raw meat scraps collected from local slaughterhouses to farm animals is illegal in the United States but no doubt occurs whenever the economic situation dictates, since steam cooking scraps is an added cost most farmers cannot easily afford. In other countries, where controls on domestic farm practices are less rigid, feeding raw pork scraps to livestock may or may not be more widespread, but in most situations, meat "scraps" are too valuable a source of human food to end up on the table of the pig. Less common, but with often devastating consequences, the disposal of carcasses of fur-bearing animals by feeding the remains to farm animals has inadvertently spread *T. spiralis* to large communities of consumers without malicious intent on the part of the farmers, who were unaware of the broad host range of this parasitic nematode (33).

Prevention at the community level depends on proper animal husbandry and on the withholding of uncooked meat in the feed of all farm animals, especially pigs. Microscopic inspection of portions of pig muscle tissue (directly or by the pooled digestion test) can control infection at the level of the abattoir (29).

An ELISA for swine trichinellosis is now approved for the certification of pork by the U.S. Department of Agriculture. However, because there are several options available to meat packers for the certification of pork, it is difficult to convince industry that slaughterhouse testing is cost-effective because trichinellosis is such a low-prevalence disease (<0.001%) (5). Such inspection programs are in place in most European countries (29, 47, 66) but have somehow escaped the mandate of the U.S. Department of Agriculture.

Thorough freezing of all pork products prior to cooking ensures the death of the larvae, while cooking meat at 137°F (58°C) for 10 min also kills them. Microwave cooking is not 100% effective in killing larvae in large pieces of meat, such as a whole fresh ham, since there are unavoidable "cold spots" in the pattern of the microwave beam (66). Freezing muscle tissue from game animals (e.g., black bear, raccoon, or opossum) is not effective, since it is thought that the antifreeze protein molecule common to most wild animals also protects worms in their muscle tissue from ice crystal formation and even preserves the worms in carcasses until such time as the carcasses can be consumed by another animal (26, 27). Some *Trichinella*

spp. (i.e., *T. nativa* and T_6) can remain infective after several days at freezing temperatures even after they have been isolated from their host muscle tissue (82).

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