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TRICHINOSIS

SUMMARY

Trichinosis is caused by worms of the phylum Nematodes, the classis Aphasmadia, the genus Trichinella, and the most commonly species *Trichinella spiralis*. Humans are infected incidentally when they eat inadequately cooked meat containing larvae of *Trichinella* species.

Although 11 species (genotypes) of Trichinella currently exist, only 8 species are described taxonomically on the basis of genetic, biochemical, and biological data.

Rats and pigs are the animals most commonly associated with trichinosis, however, depending on the region, walruses, seals, bears, polar bears, cats, raccoons, wolves, foxes and approximately 150 various species of mammals may also be infected. The global prevalence of trichinosa is difficult to evaluate but as many as 11 million people may be infected.

With improved therapy, the mortality rate has decreased to approximately 0.3%. Death occurs primarily because of myocarditis or central nervous system involvement, usually during the third to fifth weeks after ingestion.

Laboratory and immunodiagnostic findings are important for the diagnosis of trichinosis. Other methods are electromyelography, muscle biopsy and polimerasa chain reaction (PCR). Diagnostic methods in patients with central nervous system involvement are computerized tomography (CT scanning) of the brain with or without contrast, magnetic resonance (MRI) and the lumbar puncture – citological examination. Diagnostic method in patients with cardiac involvement is electrocardiography (ECG).

In moderate-to-heavy infections, the goal is to stop the larval invasion into the host muscle. Within 1 week of ingestion of contaminated meat, administer antihelmintic drugs witch are effective against larvae and adult worms limited to the intestinal lumen. Steroids may decrease inflammation but may also hinder the eradication of the adult worm, resulting in a prolonged production of larvae.

Adequate cooking and freezing methods of meat prevent infection by any species of *Trichinella* in humans. The most effective way is cooking meat at 71°C for 1minute. Also, *Trichinella* species can typically be killed by adequate cooling to - 60°C for 2 minutes or - 55°C for 6 minutes.

Key words: **trichinosis, *Trichinella spiralis*, prevalence, diagnostic methods, prevention**

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INTRODUCTION

By the 1860s, trichiniasis/trichinosis was well-recognized as a disorder spread through infected pigs, leading to a cultural aversion to certain pork products, particularly German and Dutch sausages (1).

Trichinosis is caused by worms of the phylum Nematodes, the classis Aphasmadia, the genus *Trichinella* and the most commonly species *Trichinella spiralis*. Humans are infected incidentally when they eat inadequately cooked meat containing larvae of *Trichinella* species. Most infestations are asymptomatic, although heavy exposure can cause various clinical manifestations, including diarrhea, fever, myalgias, and prostration (2).

PATHOPHYSIOLOGY

Although 11 species (genotypes) of *Trichinella* currently exist, only 8 species are described taxonomically on the basis of genetic, biochemical, and biological data. The *Table 1* describes distribution, major host reservoir, infectivity of humans, resistance to freezing, pathogenicity to humans, and ability to encysting in striated muscle as important characteristics of *Trichinella* species (2-7).

Trichinella species require two hosts to maintain their life cycle. After development in a single host, they spread to the next through ingestion of infected flesh (7). Rats and pigs are the animals most commonly associated with trichinosis, however, depending on the region, walruses, seals, bears, polar bears, cats, raccoons, wolves, foxes and approximately 150 various species of mammals may also be infected (8).

The life cycle of Trichinella spp. begins with eating raw or inadequately cooked meat containing viable larvae housed inside a cyst wall. The acidic environment in the host's stomach releases the larvae from the cyst wall. The free larvae migrate into the small intestine, attach to, and penetrate the mucosa at the base of the villi (enterocites) (7). The body of larvae is covered by three-layered cuticula, which is not elastic and does not grow along with developing larva. Because of that, the larva molts four times, transforming into the adult worm. After 4 molts and over a period of 30-36 hours, it develops into adult male or female worm. The adult male measures 1.5 x 0.05 mm, and the adult female measures 3.5 x 0.06 mm. Approximately 5 days after infection, the female begins shedding live newborn larvae (L1 stage). The adult worms are viviparous. The female remains in the intestine for 4 weeks, releasing up to 1500 larvae (9). After an adequate inflammatory response develops in the intestine, the female is eventually expelled in the feces, while the male can outlive them some time (9).

Table 1. Important characteristics of Trichinella spp.

Species	Geographic distribution	Source of infection	Infectivity	Resistance to freezing	Encysting in striated muscle
Trichinella spiralis (T1)	Cosmopolitan	Swine, wild boar, bear, horse, fox	High	None	Yes
Trichinella nativa (T2)	Arctic	Bear, horse	High	High	Yes
Trichinella britovi (T3)	Temperate zone (Europe and Asia)	Wild boar, horse	Moderate	None	Yes
Trichinella pseudospiralis (T4)	Cosmopolitan	Birds, omnivorous mammals	Moderate	None	No
Trichinella murrelli (T5)	Temperate, subarctic zone	Bear	Low	Low	No
T6	Northern temperate region (Alasca, Idaho, Montana, Pennsylvania, Vajoming and Ontario)	Bear	Low	High	Yes
Trichinella nelsoni (T7)	Tropical zone (Africa south from the Sahara)	Warthog	High	None	Yes
T8	South Africa and Namibia	Lion	Low	None	Yes
T9	Japan	Wild boar, horse	Moderate	None	Yes
Trichinella papuae (T10)	Papua New Guinea, South East Asia and Australia region	Sylvatic animals and humans	Moderate	None	No
Trichinella zimbabwensis	Africa (Zimbabwe)	Reptiles and mammals (crocodiles)	Moderate	None	No

The newborn larvae enter the lymphatics and blood circulatory system and migrate to well-vascularized striated skeletal muscle. The parasite has a predilection for the most metabolically active muscle groups; therefore, the most frequently parasitized muscles are the tongue, the diaphragmatic, masseteric, intercostal, laryngeal, extraocular, nuchal, intercostal, and pectoral muscles, the deltoid, the gluteus, the biceps, and the gastrocnemius. In tissues other than skeletal muscle, such as the myocardium and brain, the parasites soon disintegrate, causing intense inflammation, and then are reabsorbed (9).

The larvae continue to grow over the next 2-3 weeks until they reach a fully developed L1 infective stage, when they increase in size up to 10-fold. The larvae coil and develop a surrounding cyst wall, or nurse cell (except for *Trichinella pseudospiralis, Trichinella papuae*, *Trichinella zimbabwensis,* which do not encyst). The complete cycle takes 17-21 days.

The larvae within the cyst wall reach an average size of 260-400 µm; however, lengths of 800-1000 µm have been described. The nurse cell-L1 complex may persist for 6 months to several years before calcification and death occur. The life cycle is complete when a compatible host ingests the infected muscle (9).

The intensity and frequency of exposure to infected meat determine the severity of the disease. The degree of infection is categorized as light (0-10 larvae ingested), moderate (50-500 larvae ingested), and severe (more than 1000 larvae ingested) (10).

FREQUENCY

In Europe, where pork inspection is mandatory, most cases of human trichinosa are associated with horse or wild boar meat. Horsemeat-related outbreaks have been reported in France and Italy and have involved about 3000 patients in the past 25 years (10).

In Latin America and Asia, domestic pork is the chief source of trichinosa (11, 12). In Arctic regions, the main source of infection is meat from walrus, seal, and polar bear; in Africa, the main source of infection is meat from wild canids and felids (3, 4).

In the US, from 1997-2001, 72 cases of human trichinosa were reported to the Centers for Disease Control and Prevention (CDC). Most cases were associated with eating wild game (43%), although 17% were associated with commercial pork products and another 13% from noncommercial pork products (13).

The particulary high prevalence of trichinosa is registered in the Balkans, Russia, the Baltic republics, in some parts of China and Argentina (11, 12, 14). The important lower prevalence of the animal and human trichinosa is determined in Cambodia, Indonesia, Malaysia, New Zealand, and the Australian continent (3).

MORBIDITY/MORTALITY

The global prevalence of trichinosa is difficult to evaluate but as many as 11 million people may be infected (15).

Patients with light infection are usually asymptomatic. Those with mild symptoms improve in 2-3 weeks. Symptoms associated with heavy infections may persist for 2-3 months (16).

Factors that may impact morbidity are: the quantity of larvae ingested, the species of *Trichinella* (most notably *Trichinella spiralis*), and the immune status of the host. Depending on mutual influence of both factors, patients may succumb to exhaustion, or may have other serious complication such as pneumonia, pulmonary embolism, encephalitis, or cardiac failure and/or arrhythmia. Also, other factors which are involved in trichinosa morbidity are special state of host, such as, pregnancy and lactating (16, 17). The differences in trichinosa morbidity were noticed in various age groups (16).

No differences in predilection of trichinosis between males and females, and between races are reported. The prevalence of infection depends on great number of factors, but primarily it is related to geografical location and to cultural differences of population. The special influence on the rates of these parasitical diseases is different habits in food cooking and storing methods of meat (16).

With improved therapy, the mortality rate has decreased to approximately 0.3%. Death occurs primarily because of myocarditis or CNS involvement, usually during the third to fifth weeks after ingestion (16, 17).

CLINICAL HISTORY

Knowledge of the incubation period (7-30 days) can help pinpoint the source of the infection, both in individual cases and in outbreaks. The disease may progress from an enteric (ie, intestinal) phase to a parenteral (ie, invasive) phase to a period of convalescence (18).

The severity of the clinical course depends on the species involved. Patients with *Trichinella nativa* (T2) infection experience symptoms related only to the enteral phase; onset is delayed compared to that of infection with *Trichinella spiralis. Trichinella nelsoni* and *Trichinella britovi* both have low pathogenicity in their enteral and parenteral phases.

In the Arctic region, clinical features of trichinosa differed from previously reported descriptions of classic trichinosis. Unlike the classic syndro-

me, patients have chronic diarrheal syndrome which may present a new infection due to a different species of Trichinella or to reinfection with the same species of Trichinella (19).

The intestinal phase usually causes symptoms in the first week of illness (19). The most common symptoms are diarrhea, constipation, anorexia, and diffuse weakness. Occasionally, severe enteritis due to a massive inoculum of *Trichinella* species occurs. Symptoms typically last 2-7 days but may persist for weeks. Nausea is reported in 15% of patients, vomiting in 3%, and diarrhea in 16%. Abdominal discomfort and cramps may occur and with exertion dyspnea may occur (19).

The invasive phase corresponds to the migration of the larvae from the intestine to the circulatory system and eventually to the striated muscles. This phase is associated with a higher rate of symptoms than the intestinal stage (20). The duration varies from weeks to months. Severe myalgia is experienced by 89% of patients (20).

The central nervous system (CNS) is involved in 10-24% of patients. Approximately 52% of patients present with headaches, deafness, ocular disturbances, weakness, and monoparesis. The mortality rate in patients with neurotrichinosis is 50% (20, 21).

Cardiac system involvement occurs during the third week of infection, with a mortality rate of 0.1%, often during the fourth to eighth week of infection. Death may result from congestive heart failure and/or arrhythmias (16).

Pulmonary system involvement occurs in 33% of patients, with symptoms lasting up to 5 days. Patients present with dyspnea, a cough, and hoarseness (23).

The convalescent phase corresponds to encystment and repair. It can be present for months to years after infection.The encystment of larvae can lead to cachexia, edema, and extreme dehydration (19). Symptoms usually decrease around the second month, except in the case of infection with *Trichinella pseudospiralis,* which may cause symptoms for several months.

CLINICAL FINDINGS

The intestinal phase usually begins in the first week of illness with the following clinical findings: abdominal distention, macular or petechial rashes are observed in 20% of patients and diarrhea (24).

The invasive phase usually begins in the third week of illness; 91% of patients have a fever that generally begins at two weeks, and peaks after four weeks, with values up to 40–41°C in severe cases. This degree of fever is unique among helminthic infections (16, 25). Weakness and/or myositis occur in 82% of patients. Muscles become stiff, hard, and edematous. Muscles with increased blood flow (eg, extraocular muscles, masseters, larynx, tongue, neck muscles, diaphragm, intercostals, limb flexors, lumbar muscles) are most frequently involved. Rash (macular or petechial) is reported in 15-65% of patients (23), subungual splinter hemorrhages occur in 8% of patients (23), and periorbital edema is reported in 77% of patients (23).

Dyspnoea is caused primarly by parasite invasion and consequent inflammations of respiratory muscles such as the diaphragm. Ocular findings include subconjunctival hemorrhages in 9% of patients, conjunctivitis in 55%, and rare incidences of chemosis and retinal hemorrhage (23).

The central nervous system may be affected in 10% to 24% of trichinosa cases. Of these, 53-96% exhibit meningoencephalitis, 40-73% exhibit focal paralysis and/or paresis, 39-71% exhibit delirium, 20% exhibit decreased or absent deep tendon reflexes, 17% exhibit meningitis, and 2% exhibit evidence of psychosis (26).

The signs of cardiac system involvement include hypertension, increased venous pressure, and, in 18% of patients, peripheral edema (26).

The convalescent phase is characterized by: oedema in 18% of patients, weight loss, myalgia, patients are fatigued easily, and ocular signs with chronic headaches may be present (25).

The differential diagnosis depends on the phase of infection. The most frequent clinical diseases which have to be considered in diagnosis of trichinosis are: angioedema, dermatomyositis, food poisoning, bacterial and viral gastroenteritis, acute glomerulonephritis, hookworms, influenza, polyarteritis nodosa, rheumatic fever, schistosomiasis, strongyloidiasis, typhoid fever, eosinophilic leukemia, and eosinophilic toxocariasis.

DIAGNOSIS

Laboratory findings:

Leukocytosis occurs in 65% of patients, with cell counts of up to 24,000/mL. Eosinophilia typically rises 10 days after infection, with total eosinophil counts of up to 8700/mL (40-80% of total WBC). The counts peak in 3-4 weeks and resolve over the next few months. Nearly all patients with trichinosis, either symptomatic or asymptomatic, exhibit eosinophilia. The only exception is in severe cases, when the eosinophil count may be severely depressed. A low eosinophil count indicates an increased mortality rate (23).

Erythrocyte sedimentation rates are usually within the reference range.

Obtained creatine kinase (CK) levels are elevated to 17,000 U/L. CK (isoenzyme myocardial band [MB]) elevations may indicate myocardial involvement; however, as many as 35% of patients without cardiac involvement may have elevated CK-MB levels (23, 27).

Levels of lactate dehydrogenase isoenzymatic forms (ie, lactate dehydrogenase fraction 4 [LD_4] and lactate dehydrogenase fraction 5 [LD_5]) are elevated in 50% of patients (23, 28).

Immunodiagnosis:

The classes of immunoglobulin G (IgG), immunoglobulin E (IgE), and immunoglobulin A (IgA)-specific antibodies do not appear until 2-3 weeks after trichinosis infection (29). The immunoglobulin G-specific antibodies peak around the third month and may persist in patients' blood for years after infection. However, the titer of IgG antitrichinella specific antibodies do not correlate with the severity of disease or the clinical course in acute stage of trichinosis (28).

The serological tests available for diagnosis

The bentonite flocculation. The results of this test are usually not positive for more than 1 year after infection (30).

The indirect hemagglutination assay (IHA) (30).

The indirect immunofluorescence assay (IIF) (6).

The enzyme-linked immunosorbent assay (ELISA). The immunosorbent test is 100% sensitive on day 50, with 88% of results remaining positive 2 years after infection (6).

Latex agglutination test (30).

Western-blot analysis. The test is appropriate to make an early diagnosis of trichinosis. It enables to apply adequate therapy on time and reduces appearance of possible diseases complication. The test is commercially available, and it is more sensitive and specific than IIF and ELISA assays (2).

The hypersensitivity skin test. The reactions are positive at approximately 17 days after infection and remain positive for life in patients with trichinosis. The test is no longer commercially available.

Other diagnostic methods:

Electromyelography. Electromyelography may be helpful in diagnosing moderate-to-severe infection, but no pathognomonic findings exist. The test result may reveal acute myositis or diffuse myopathic dysfunction. Changes usually resolve 2-3 months after infection but may persist for 1-8 years (2).

Muscle biopsy. It provides a definitive diagnosis; however, it is rarely recommended except in difficult cases when serology tests are unhelpful (30). Obtain a 0.5- to 1-gram muscle biopsy specimen from the deltoid or gastrocnemius muscle because these are most easily accessible. The yield increases if the biopsy site is swollen or tender. Stain the specimen with hematoxylin and eosin (H&E) and examine multiple sections (30). Occasionally, larvae can be found after the muscle has been digested enzymatically. If a biopsy is performed prior to larvae coiling (beyond day 17 of infection), worm tissue can be confused with muscle tissue. A negative result does not necessarily exclude infection (30).

Polymerase chain reaction (PCR).

Diagnostic methods in patients with CNS involvement:

Computerized tomography (CT scanning) of the brain with or without contrast.

Magnetic resonance instrument? (MRI). With contrast enhancement it may reveal 3- to 8-mm nodular or ringlike lesions in patients manifesting CNS involvement (31).

The lumbar puncture-citological examination. Results of this survey are normal in 50-75% of patients with trichinosis. Larvae are found in 8-24% in patients. However, the presence of great number of eosinophilic leucocytes indicates the actual presence of the eosinophilic meningitis as a possible complication of trichinosis.Diagnostic methods in patients with cardiac involvement:

Electrocardiography (ECG). The ECG presents important diagnostic and prognostic tool in patients with trichinosis. Electrocardiographic alterations are premature contractions, prolongation of the P-R interval, small QRS complexes with intraventricular block, and flattening or inversion of the T waves, especially lead II and precordial leads (20).

Histological examinations:

Basophilic transformation of muscle fibers occurs within 4-5 days after larval penetration and is a valuable diagnostic criterion, even in cases when no larvae can be demonstrated. Basophilic transformation affects only a portion of the affected muscle fiber. Myofibrils disappear, the sarcoplasm becomes basophilic, and the cell nucleus is displaced to the center of the cell. The larva can be observed within the affected nurse cell. Attempting diagnosis before larvae begin to coil (ie, <2 wk after larvae enter the muscle cell) creates a risk of confusing the worm with fragments of muscle tissue. Encapsulation begins approximately 2 weeks after ingestion. The capsule contains the larva and fragments of basophilically transformed sarcoplasm that directly surround the larva. Infiltration by eosinophils and mononuclear cells also occurs. The absence of a capsule and the presence of a straight larva in the complex indicate ongoing infection (32). Parasites migrate through the myocardium and CNS but do not encyst. However,

perivascular collections of eosinophils, lymphocytes, macrophages, and polymorphonuclear leukocytes develop in the myocardium and CNS.

TREATMENT

In moderate-to-heavy infections, the goal is to stop the larval invasion into the host muscle. Within 1 week of ingestion of contaminated meat, administer albendazole (5 mg/kg/d for 1 wk), mebendazole (5 mg/kg/d for 1 wk), or thiabendazole (25 mg/kg/d for 1 wk) (33-35). These drugs belong to anthelminthic and they are effective against larvae and adult worms limited to the intestinal lumen. The anthelmintic therapy goal is to prevent systemic invasion. Thiabendazole and mebendazole have no effect on tissue larvae. If tissue invasion occurs, the aim of therapy is to decrease subsequent muscle damage and damage of other affected tissues. In this stage of trichinosis the most effective treatment modalities are bed rest, analgesics, and antipyretics. Anthelmintic therapy has no proven role at this stage of diseases. Albendazole appears to be marginally effective and mebendazole less so. A trial of albendazole is justified in severe or prolonged infections. Avoid thiabendazole at this stage because of its adverse effect profile (33). Steroids may decrease inflammation but may also hinder the eradication of the adult worm, resulting in a prolonged production of larvae. Prednisone at 50 mg/d can be used in severe infections, especially if hemodynamic instability or involvement of the central nervous, cardiac, or pulmonary systems is present (33-35).

COMPLICATIONS

Complications during acute phase of trichinosis in pregnant women include abrupt delivery of stillbirths (16), and vertical infection of the fetus has been described (16).During convalescence, the patients may complain of hearing disorders, weight loss, loss of hair and nails, skin desquamation, hoarseness, aphonia, and muscle stiffness, and disturbances of menstruation in women have been reported (16). Long-term sequelae of the CNS include decreased mental power, numbness of hands and feet, decreased stress tolerance, loss of initiative, and depression.Death may occur from heart failure or central nervous system failure during the third to fifth weeks of the infection. 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.

PREVENTION AND CONTROLLING INFECTION

Adequate cooking and freezing methods of meat prevent infection by any species of *Trichinella* in human. The most effective is cooking meat at 71°C for 1minute, also *Trichinella* species can typically be killed by adequate cooling to 60°C for 2 minutes or 55°C for 6 minutes.Freezing is also an effective method for killing most species of *Trichinella*. For a 15.6 cm piece of meat, the recommended temperatures to kill larvae are as follows: -15°C for 20 days, -23°C for 10 days, and -29°C for 6 days. 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 (3). Some *Trichinella spp*. (i.e., *Trichinella nativa* and T6) can remain infective after several days at freezing temperatures even after they have been isolated from their host muscle tissue. Salting, smoking, or drying does not kill larvae of Trichinella (34).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 (8). Infection of pig herds by *Trichinella spiralis* is usually perpetrated by the animals scavenging on infected rodent populations or, less commonly, by cannibalism of sick animals. Also, there is a possibility that 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 native animals are infected.

Prevention at the community level depends on proper feeding (without using of raw and uncooked meat in the feed) of all farm animals, permanently controlling rodent populations, regular control of veterinary medicine, and mandatory inspection of pork for *Trichinella species* (35).

REFERENCES

1. Cook GC. Outbreak of trichinosis (trichiniasis)-contracted in London in 1879. Postgrad Med J 2001; 77 (903): 62-63.

2. Dupouy-Camet J, Kociecka W, Bruschi F, Bolas-Fernandez F, Pozio E. Opinion on the diagnosis and treatment of human trichinellosis. Exp Opin Pharmacother 2002; 3:1117–1130.

3. Pozio E. Taxonomy of Trichinella and the epidemiology of infection in the Southeast Asia and Australian regions. Southeast Asian J Trop Med Public Health 2001;32 Suppl 2:129-132.

4. Pozio E, Foggin CM, Marucci G, La Rosa G, Sacchi L, Corona S, Rossi P, Mukaratirwa S. Trichinella zimbabwensis n.sp. (Nematoda), a new non-encapsulated species from crocodiles (Crocodylus niloticus) in Zimbabwe also infecting mammals. Int J Parasitol 2002; 32(14):1787-99.

5. Murrell KD, Lichtenfels RJ, Zarlenga DS, Pozio E. The systematics of the genus Trichinella with a key to species. Vet Parasitol 2000; 93(3-4):293-307.

6. Yera H, Andiva S, Perret C, Limonne D, Boireau P, Dupouy-Camet1 J. Development and Evaluation of a Western Blot Kit for Diagnosis of Human Trichinellosis. Clin Diagn Lab Immunol 2003; 793–796.

7. Gasser RB, Hu M, Abs El-Osta YG, Zarlenga DS, Pozio E. Nonisotopic single-strand conformation polymorphism analysis of sequence variability in ribosomal DNA expansion segments within the genus Trichinella (Nematoda: Adenophorea). Electrophoresis 2004; 25(20):3357-3364.

8. Batelli G, Guberti V, Martini M. Trichinellosis control in Italy: considerations on sampling in imported horses and their meat. In Campbell WS, Pozio E, Bruschi F (ed.), Trichinellosis, 1993: 593–598. Proceedings of the 8th International Conference on Trichinosis,. Istituto Superiore di Santa´ Press, Rome, 1993.

9. Wakelin D, Denham DA.The immune response. In Campbell WC (ed.), Trichinella and trichinosis. Plenum Press, New York, 1983: 265–308.

10. Touratier L. A challenge of veterinary public health in the European Union: human trichinellosis due to horse meat consumption. Parasite 2001; 8 (2):S252-256.

11. Ortega-Pierres MG, Arriaga C, Yepez-Mulia L. Epidemiology of trichinellosis in Mexico, Central and South America. Vet Parasitol 2000; 93(3-4):201-225.

12. Takahashi Y, Mingyuan L, Waikagul J. Epidemiology of trichinellosis in Asia and the Pacific Rim. Vet Parasitol 2000; 93(3-4):227-239.

13. Roy SL, Lopez AS, Schantz PM: Trichinellosis surveillance-United States, 1997-2001. MMWR Surveill Summ 2003; 52(6): 1-8.

14. Liu M, Boireau P. Trichinellosis in China: epidemiology and control. Trends Parasitol 2002;18(12):553-6.

15. Dupouy-Camet J. Trichinellosis: a worldwide zoonosis. Vet Parasitol 2000; 93(3-4):191-200.

16. Pawlowski ZS. Clinical aspects in man. In: Campbell WC, ed. Trichinella and trichinosis. New York: Plenum Press, 1983: 367–341.

17. Dubinsky P, Boor A, Kincekova J, et al: Congenital trichinellosis? Case report. Parasite 2001; 8(2): S180-182.

18. Capo V, Despommier DD. Clinical aspects of infection with Trichinella spp. Clin Microbiol Rev 1996; 9:47–54.

19. MacLean JP, Viallet J, Law C, et al. Trichinosis in the Canadian Arctic: report of five outbreaks and a new clinical syndrome. J Infect Dis 1989; 160:513–520.

20. Katz M, Despommier DD, Gwads RW. Trichinella spiralis. In: Katz M, Despommier DD, Gwads RW, eds. Parasitic diseases. New York: Springer-Verlag, 1989; 28–35.

21. Durack DT, Sumi SM, Klebanoff SJ. Neurotoxicity of human eosinophils. Proc Natl Acad Sci U S A 1979; 76:1443–1447.

22. Mawhorter SD, Kazura JW. Trichinosis of the central nervous system. Semin Neurol 1993; 13:148–152.

23. Gould SE. Clinical manifestations, a. symptomatology. In: Gould SE, ed. Trichinosis in man and animals. Springfield, IL: Charles C Thomas, 1970: 269–276.

24. Castro G, Bullick GR. Pathophysiology of the gastrointestinal phase. In: Campbell WC, ed. Trichinella and trichinosis. New York: Plenum Press, 1983; 209–239.

25. Kassur B, Januszkiewicz J, Poznanska H. Clinic of trichinellosis. CW, Pawlowski ZS, eds. Trichinellosis. Hanover, NH: University New England, 1978; 27–44.

26. Kramer MD, Aita JF. Trichinosis. In: Vinken BJ, Bruyn GW, eds. Infections of the central nervous system. Handbook of clinical neurology. Amsterdam, North Holland, 1978; 267–290.

27. Tassi C, Materazzi L, Pozio E, et al. Creatine kinase isoenzymes in human trichinellosis. Clin Chim Acta 1995; 239:197–202.

28. Boczon K, Winiecka W, Kociecka W, et al. The diagnostic value of enzymatic and immunological test in human trichinellosis. Tropenmed Parasitol 1981; 32:109–114.

29. Patterson R, Roberts M, Slonka G, et al. Studies of immunoglobulins, bentonite flocculation and IgE, IgG and IgM antibodies in serum from patients with trichinosis. Am J Med 1975; 58:787–793.

30. Murrell KD, Bruschi F. Clinical trichinellosis. In: Sun T, ed. Progress in clinical parasitology. Boca Raton, FL: CRC Press, 1994: 117–150.

31. Mawhorter SD, Kazura JW. Trichinosis of the central nervous system. Semin Neurol 1993; 13:148–152.

32. Weatherly NF. Anatomical pathology. In: Campbell WC, ed. Trichinella and trichinosis. New York: Plenum Press, 1983:173–178.

33. Pozio E, Sacchini D, Sacchi L, et al. Failure of mebendazol in the tretment of humans with Trichinella spiralis infection at the stage of encapsulated larvae. Clinb Infect Dis 2001 ; 32 :638-642.

34. Bruschi F, Murrell KD. New aspects of human trichinellosis : the impact of new Trichinella species. Postgrad Med J 2002 ; 78 :15-22.

35. Djorđevic M. Detection of Trichinella by various methods in Yugoslavia. Southeast Asian J. Trop. Med. Public Health 1991; 22:326–328.

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SAŽETAK

Trihinelozu uzrokuje helmint koji pripada kolenu Nematodes, klasi Aphasmadia, rodu Trichinella i najčešće vrstom *Trichinella spiralis.* Čovek se zarazi konzumirajući neadekvatno termički obrađeno meso inficirano larvama parazita roda Trichinella. Jedanaest genotipova roda Trihinella otkriveno je da postoji u prirodi, a samo 8 je taksonomski definisano na osnovu genotipskih, biohemijskih i bioloških karakteristika.

Najčešće opisane životinjske vrste sa trihinelozom su pacovi i svinje, ali u zavisnosti od geografske distribucije, mnoge životinjske vrste, kao što su morževi, foke, medvedi, polarni medvedi, mačke, rakuni, vukovi, lisice i blizu 150 različitih vrsta sisara mogu biti zaražene helmintom roda Trichinella. Globalnu prevalenciju trihineloze je teško ustanoviti, ali se smatra da je približno 11 miliona ljudi zaraženo ovim parazitom.

Primenom terapije, stopa mortaliteta od trihineloze smanjena je na 0,3%. Smrt najčešće nastupa usled zahvaćenosti srčanog mišića ili centralnog nervnog sistema, i to u periodu između 3-4 nedelje po infekciji.

Laboratorijski i rezultati imunodijagnostičkih testova značajni su u dijagnostici trihineloze. Drugi testovi koji se mogu koristiti u dijagnostici trihineloze su: elektromiografija, biopsija zahvaćenog mišića i reakcija lančanog umnožavanja (PCR). U slučaju zahvaćenosti centralnog nervnog sistema mogu se koristiti i kompjuterizovana tomografija sa ili bez kontrasta, magnetna rezonanca, kao i lumbalna punkcija za citološko ispitivanje. U slučaju zahvaćenosti srčanog mišića beleže se značajne elektrokardiografske promene.

Kod umerenih do teških infekcija cilj terapije je da spreči invaziju larvi u mišićno tkivo. Preporučuje se u toku prve nedelje infekcije aplikovanje antihelmintika koji su efikasni samo prema helmintima u lumenu creva. Kortikosteroidi mogu smanjiti zapaljensku reakciju, ali mogu biti odgovorni za usporenu eradikaciju parazita iz lumena creva kao i za produžavanje perioda produkcije larvi.

Adekvatna termička obrada i zamrzavanje mesa su najznačajnije mere u prevenciji infekcije bilo kojom vrstom parazita roda Trichinella. Efektno ubijanje larvi postiže se termičkom obradom na 71°C u trajanju od 1 minuta ili zamrzavanjem na -60 °C 2 minuta, ili na -55 °C u trajanju od 6 minuta.

Ključne reči: **trihinosis, *Trichinella spiralis*, prevalencija, dijagnostičke metode, prevencija**