Major Helminthic Diseases of North America: A Review

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Disease producing worms are worldwide in distribution. The worms of significance in North America are the nematodes and the cestodes. Diagnosis is rarely made on clinical grounds alone because the clinical presentation of infection varies widely. Examination of stool for ova is usually the definitive diagnostic step. Follow-up stool examination several weeks after treatment is mandatory. The clinical findings pertaining to each type of worm and the recommended treatment are delineated.

The principle disease producing worms of North America are the nematodes, or roundworms, and the cestodes, or tapeworms. Parasitic infections are worldwide in occurrence. With few exceptions, offspring must pass out of the definitive host harboring the sexually active adults before they can mature and become capable of infecting the next host. Clinical illness results from serving as the definitive host, or from harboring the larval stages of the worm as an intermediate host. Manifestations of illness are related to the total number of worms acquired. Small loads may be asymptomatic and in some cases may not require treatment. Large infestations usually cause symptomatic disease.

Absolute immunity to worms is rare even though the host forms a variety of antibodies to the worm and its products. Markedly increased immunoglobulin E (IgE) concentrations are seen in helminthic infections, but usually are not parasite specific.[1]

Interference with the host's immune response at the macrophage level may result both in impaired processing of antigen and impaired destruction of the worm. Consequently, the immunologic response to the parasite is primarily useful for diagnosis and not for killing.[1]

Principles of Diagnosis

Diagnosis is rarely made on clinical grounds because of the considerable variation in the clinical presentation of infection and the high frequency of mild or asymptomatic infestation. In addition, patients may harbor several parasites.

The discovery of ova in freshly passed stools is often the key to diagnosis of helminthic infections. If liquid stool cannot be examined within one hour after passage, part of the specimen should be preserved in polyvinyl alcohol fixative. If fully formed stool cannot be examined in a matter of hours, it should be refrigerated.[2] Helminth ova can be seen in older specimens but free-living motile forms are present only in freshly passed warm stool. Because parasites are released sporadically in showers, three specimens should be examined within a seven to ten-day period.[2]

Stools from patients who have taken mineral or castor oil are unsuitable for examination for one week. A three-week wait is required after ingestion of barium or gallbladder dye.*

Blood, the next most frequent source for recovering animal parasites, is less revealing than stool

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*Laboratory Manual, Department of Microbiology, University of Illinois, School of Medicine, Chicago, 1972, unpublished.

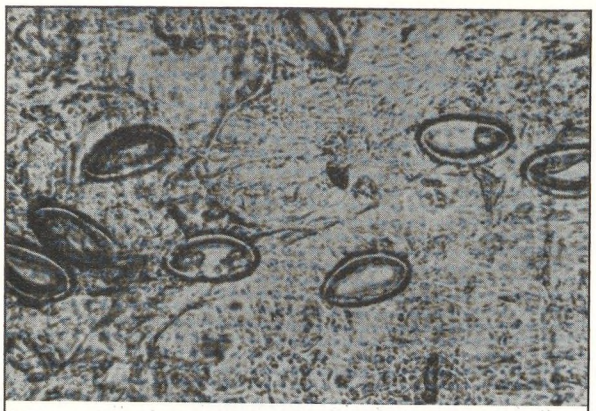

Figure 1. Enterobius vermicularis. Unstained eggs on cellophane tape prep.*

in cases of worm infestation. Anemia is occasionally observed but is nonspecific and reflects general nutritional status. Eosinophilia, presumably due to an immunologic response to the complex proteins of the worm, is often the first clue to helminthic infections. Serologic testing is primarily of benefit in trichinosis, toxocariasis, and strongyloidiasis.[2]

Muscle biopsy is useful for the diagnosis of trichinosis and, less frequently, the larval stage of Taenia solium.[2]

Because helminthic parasites can survive for some time following the death of the patient, the autopsy can be valuable in the diagnosis of these organisms. Hookworms and whipworms can be removed by gently pulling them from their sites of attachment to the bowel wall. Strongyloides can be removed by carefully scraping the intestinal mucosa.[2]

Principles of Treatment

Helminthic infections, in general, are treated when discovered. The decision to treat, however, is based on the number of worms, their life span, and the personal or public health hazards resulting from infection. Of course, the availability, efficacy, and side effects of therapeutic agents must be considered. Clinical and laboratory reevaluation following treatment is mandatory. Stool should be examined after several weeks and retreatment undertaken if necessary. Retreatment is usually performed with the same drug rather than a more toxic or less effective alternative.[3]

*Figures 1-8 have been made available through the courtesy of Abbott Laboratories.

Nematoda—The Roundworms

Enterobius vermicularis–Pinworm

Enterobius vermicularis has a cosmopolitan distribution. It is estimated that 200 million people, including 18 million in the United States and Canada are infected.[3] Patients are infected by direct transfer of the eggs from the anus to the mouth or from ova deposited on bedclothes and linen. Ova so deposited may remain infective for days. As the eggs are light, they float in the air and may be inhaled and swallowed. This can initiate or perpetuate infection.[4] The most common clinical manifestation, pruritis ani, is typically worse at night. It results from the migration of the gravid female to the anus to lay eggs. Vulvitis and vaginitis should also suggest the presence of pinworms.[5]

The life cycle of the pinworm requires ingestion of the ova, release of the larvae in the small intestine, and migration to the cecum. One month is required from ingestion of ova to the production of new eggs. Diagnosis is made by the cellophane tape test. The tape is applied to the anal area early in the morning and then examined for eggs microscopically (Figure 1). Occasionally adult worms may be seen in the perianal area or on the surface of freshly passed feces. Eosinophilia is not characteristic of pinworm infestation.[3]

All infected individuals are treated. If the infection recurs, all household members are usually treated. Available drugs kill only the adult worms and not the developing eggs and larvae. Hence, any eggs swallowed at the time the drug is given will develop into mature worms within two to four weeks and result in a therapeutic failure. To avoid this, therapy should be repeated in two weeks, after new adults may have developed but before they have begun to lay eggs.[4,6] The drugs of choice are mebendazole (Vermox) in a single 100 mg oral dose (not used in infants or pregnant patients) or pyrantel pamoate (Antiminth) 11 mg/kg (maximum 1 gm) in a single oral dose.[4,6,7]

Hand washing and fingernail cleanliness may decrease infection and transmission. Boiling of bedsheets, undergarments, and pajamas to prevent reinfection is not beneficial.[4]

Ascaris lumbricoides–Common Roundworm

Ascaris lumbricoides is a relatively common disease in the southeastern and southwestern United States and is especially common among

children.[6] Infection follows ingestion of embryonated eggs from contaminated food or soil. Man is the only susceptible host. The daily output of eggs by the female worm is approximately 200,000 per worm.[3] Two to three weeks of soil incubation are required for the ova to become infective.[4]

Following ingestion of the eggs, larvae are liberated into the small intestine. The larvae migrate through the intestinal wall to the blood or lymphatics and then are carried to the lungs. About ten days later, the larvae in the pulmonary capillaries and alveoli pass upward to the trachea and are swallowed and passed to the jejunum. The larvae develop into mature adults within two to three months after ingestion. These worms do not multiply within the host.[3]

Because of the extensive tissue migration of the worm, clinical manifestations may be diverse. There are, however, two distinct clinical phases. The first is the blood-lung migration of the larvae. In this phase pneumonia with cough, fever, blood-tinged sputum, and patchy pulmonary infiltrates occur.[3] In the second, or intestinal phase, the patient is usually asymptomatic although there may be vague abdominal discomfort or sudden recurrent epigastric or umbilical pain. A bolus of worms may cause bowel obstruction especially ileocecal, or biliary obstruction or pancreatitis.[8]

Observation of the characteristic ova in feces (Figure 2) or the presence of adult worms in emesis or stool is the basis for diagnosis. Occasionally the worms are seen as negative images on x-ray after a barium study or after ingesting barium themselves. Ascaris pneumonia may be diagnosed by finding larvae and eosinophils in the sputum. Blood examination reveals eosinophilia during the pulmonary but not the intestinal phase.[3]

Treatment during the pulmonary stage is symptomatic. Because the migration of ascaris is unpredictable and may result in serious complications, all patients in the intestinal phase require treatment. This unpredictability requires the treatment of ascariasis first when more than one helminth is present.[6] Treatment of all patients consists of piperazine citrate (Antepar) in a single dose of 75 mg/kg (maximum 4 gm). This drug acts by paralyzing the worms with subsequent passage in the stool. Repetition of treatment on a second or third day increases the success of treatment.[6] An alternate drug is pyrantel pamoate (Antiminth) 11 mg/kg (maximum 1 mg) once daily for three days.[4]

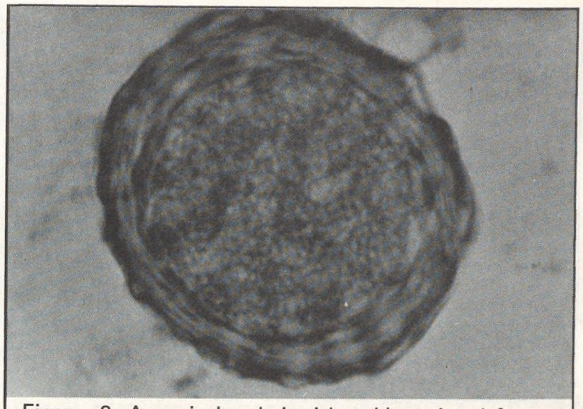

Figure 2. Ascaris lumbricoides. Unstained fertile egg.

Prevention requires stressing personal hygiene and the provision of adequate toilet facilities.

Toxocara canis and cati–Dog and Cat Roundworm

Infection by the dog or cat roundworm, Toxocara canis or cati, produces visceral larva migrans. This parasite is unable to complete its life cycle within humans but nonetheless becomes widely disseminated with a variety of manifestations. Infection with this worm is found primarily in the United States and Europe. The incidence is greatest in children from two to five years of age because of poor sanitary habits and intimate association with pets. Infection is acquired by ingestion of ova passed in dog and cat feces. After ingestion, larvae are liberated into the small intestine, penetrate the wall, and are carried into the liver and lung via the blood. These worms rarely ascend the respiratory tract and hence, are unable to reach the small intestine to mature into adults.[3]

Clinical manifestations from larval migration within the tissues include hemorrhage, necrosis, eosinophilic inflammatory reaction, and granuloma formation. The most frequently involved organs are the liver, lungs, brain, eye, heart, and skeletal muscles. Patients present with fever and tender hepatomegaly. Granulomatous endophthalmitis, convulsions, focal neurologic defects, and myocarditis may result. Asymptomatic infections are probably common.[3]

Diagnosis may be suggested by leukocytosis with marked eosinophilia and hypergammaglobulinemia.[4] Indirect hemagglutination testing

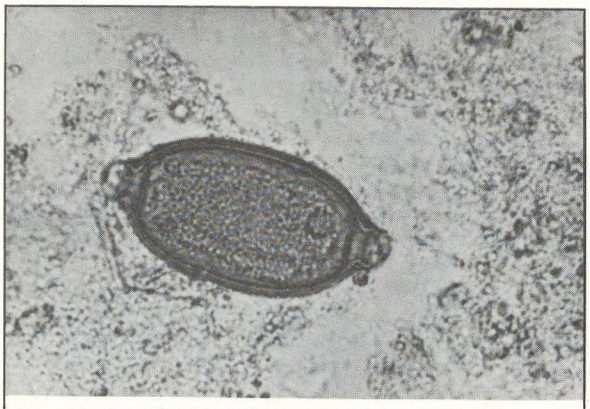

Figure 3. Trichuris trichiuria. Unstained egg.

using antigens from A lumbricoides and T canis has been used for diagnosis and found to be reasonably specific and sensitive.[2] Progress is being made on the development of an intradermal skin test to aid diagnosis in the future.[2] Stool examination is usually of no benefit. Definitive diagnosis depends on identification of larvae in sputum, tissue granuloma, or liver biopsy.

There is no reliable treatment. Diethylcarbamazine (Hetrazan) 2 mg/kg three times a day for three to four weeks has been used with some success.[3] The pediatric dose is 2 to 4 mg/kg three times a day for three weeks.[9] Thiabendazole (Mintezol) 25 to 50 mg/kg for seven to ten days is an alternative.[3]

Prevention of infections is accomplished by avoidance of egg ingestion and repeated worming of infected dogs and cats.

Trichuris trichiura–Whipworm

Man is the natural reservoir and only susceptible host for Trichuris trichiura.[5] Whipworm is found throughout the rural areas of the southern United States.[6] Infection is acquired by ingestion of ova. The eggs then hatch in the small intestine and larvae become imbedded in the intestinal villi. After several days the larvae migrate to the large intestine where they mature in approximately three months.[4]

Symptomatic infections require the presence of large numbers of adult worms. Symptoms include nausea, abdominal pain, diarrhea, tenesmus, and rectal prolapse.[4] Infected patients are estimated to lose 0.005 ml of blood per worm per day and may develop anemia.[3]

The presence of typical ova in the stool is diagnostic (Figure 3). Eosinophilia is rarely present.

Treatment, consisting of mebendazole (Vermox) 100 mg twice a day for three days, cures 60 to 70 percent of children and adults.[3,4,6]

Necator americanus–Hookworm

The New World hookworm, Necator americanus, is found primarily in the southern United States. Necator infestation is estimated to cause blood loss of approximately 0.03 ml/worm/day.[3]

The life cycle requires the passage of eggs in the fecal stream. The eggs release the free living, or rhabditiform, larvae which develop within a matter of days into the infective, or filariform, larvae. These organisms remain viable in the soil for weeks. Infection occurs when the filariform larvae penetrate the skin or, rarely, are ingested. After entering the host, the larvae migrate into blood vessels which carry them to the lungs. From this point, the life cycle resembles that of ascaris. The larvae mature approximately six weeks after skin penetration or ingestion.[3]

The first signs of infection are an erythematous maculopapular skin rash and edema with severe pruritis at the site of invasion by the larvae. This combination of symptoms is known as Ground Itch. After skin penetration, the migration of the worms through the lungs may result in pneumonia and fever. Lung migration is usually asymptomatic, however. Gastrointestinal symptoms of vague epigastric distress, abdominal cramps, or diarrhea may occur during the parasite's intestinal phase. The most outstanding manifestations are those of iron deficiency anemia and hypoalbuminemia secondary to chronic intestinal blood loss.[4] Asymptomatic infections out number symptomatic ones 20 to 40 times.

The presence of hookworm eggs on fecal smear is diagnostic (Figure 4). If the specimen is allowed to stand for several hours, larvae hatch which may be confused with strongyloides.

Treatment includes iron, a high-protein diet, and pyrantel pamoate (Antiminth) 11 mg/kg once a day for three days.[6] Mebendazole (Vermox) 100 mg twice a day for three days is an alternative.

Prevention requires proper disposal of feces and avoiding skin contact with the soil.

Ancylostoma braziliense–Dog and Cat Hookworm

The disease, cutaneous larva migrans, is caused by infection with larvae of the dog and cat hookworm, Ancylostoma braziliense. This worm reaches the adult stage only in these animals. Infection is worldwide and occurs primarily in persons having close contact with dogs and cats.[5] The parasite is acquired at beaches and other moist, sandy areas where animals defecate, because the eggs develop well in this environment. Infection is found primarily in southern Atlantic and Gulf states.[3]

The life cycle requires discharge of the eggs in the animal's feces and their development to rhabditiform and then filariform larvae. The infective larvae penetrate the skin but are unable to mature further.[3]

Within a few hours, clinical manifestations become apparent at the site of skin penetration with the development of a small red papule. Within one week this papule develops into an irregular, erythematous, serpiginous, dermal tunnel indicating larval migration. This tunnel may attain a length of 15 to 20 cm. These tunneled larvae may persist for weeks or months, although two to six weeks is usual.[5] Pruritis, caused by the migrating larvae, is a constant feature of this infestation.

Diagnosis is made by the history of travel and exposure and the characteristic clinical presentation.

Treatment consists of thiabendazole (Mintezol) 25 mg/kg twice a day for two days. In addition, oral antihistamines are prescribed for the pruritis.[3]

Prevention requires keeping dogs and cats from contaminating recreational areas and children's sandboxes.

Strongyloides stercoralis–Threadworm

Strongyloides stercoralis is the one roundworm capable of multiplying within the host. The usual mode of infection is by penetration of the skin by filariform larvae. Some infections result from ingestion of contaminated food and drink. This parasite is endemic throughout the rural southern United States.[6]

The life cycle begins with the development of the ova into rhabditiform larvae within the small bowel of the host. These larvae are subsequently passed into the fecal stream. On reaching the soil,

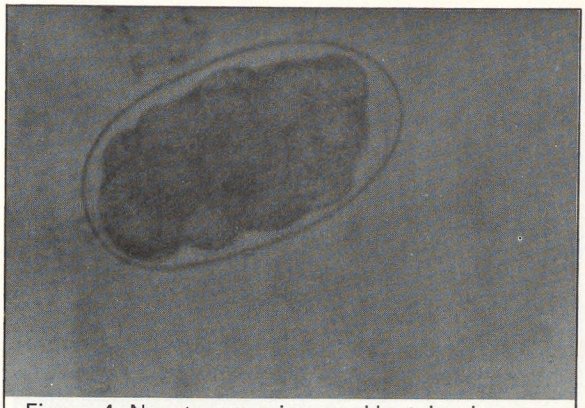

Figure 4. Necator americanus. Unstained egg.

development occurs into the filariform larvae which can penetrate the skin and small blood vessels. The remainder of the life cycle follows the pattern of ascaris. There is also an alternate life cycle of autoinfection in which the rhabditiform larvae develop into filariform larvae before passing out in the stool. They are then capable of invading the host directly.[3]

Uncommon initial clinical signs may be transitory skin eruptions with blotchy erythema, serpiginous lesions, and urticaria at the site of initial penetration. Intestinal infection is usually asymptomatic or presents with vague abdominal complaints. In heavy infestations, peptic ulcer may be simulated, except that food often aggravates the pain. Mucosal inflammation can be severe enough to produce subacute obstruction, segmental ileus, or impaired absorption.[3]

Definitive diagnosis is made by examining fresh fecal specimens for typical larvae which must be differentiated from those of the hookworm. Microscopic examination of duodenal washings and jejunal biopsies may also provide the diagnosis. Additionally, a filarial complement fixation test is positive in about 75 percent of cases. Eosinophilia is common as well.[3]

All infected patients are treated to prevent severe disease associated with autoinfection. Thiabendazole (Mintezol) 25 mg/kg twice a day for two to three days is the drug of choice.[3,6] Stools should be rechecked at intervals of three months, as the initial course of treatment may fail in 20 percent of patients.[4]

Prevention is the same as for hookworm.

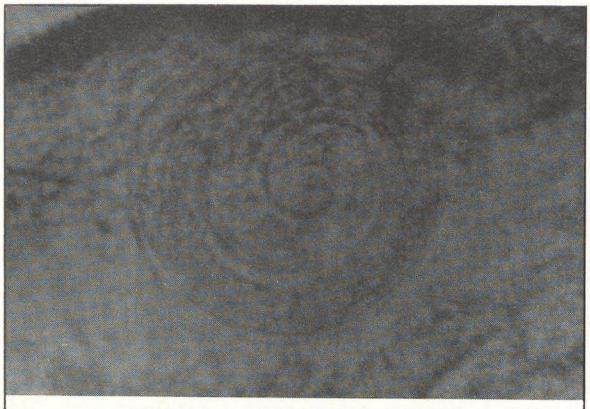

Figure 5. Trichinella spiralis. Impression smear of muscle biopsy.

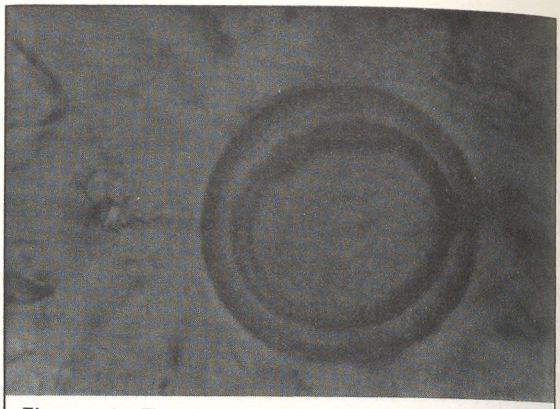

Figure 6. Taenia species. Unstained egg in stool specimen.

Trichinella spiralis–Trichina worm

Infection with Trichinella spiralis occurs when meat containing encysted larvae is eaten. Pork is usually involved, although bear meat has been reported to carry the parasite.[4] Since there are no intermediate hosts for this worm, both the larvae and adults develop in the same animal. The incidence is highest in New England, the Middle Atlantic, and the Pacific states. It is estimated that 1.5 million Americans carry this organism.[3] The majority of infections are asymptomatic.

The life cycle involves ingestion of the encysted larvae with liberation by gastric digestion. The larvae then migrate to the small intestine, mature, and produce new larvae. These larvae then enter the vasculature and are distributed throughout the body. They enter skeletal muscles, grow, and become encysted within approximately three weeks. Muscles of the diaphragm, tongue, eye, deltoid, pectoral, gastrocnemius, and intercostals are most often affected. Calcification of the cysts begins at 6 to 18 months. The life span of the encysted organism is five to ten years.[3]

The first symptoms usually occur within one to two days after ingestion of undercooked meat containing the encysted larvae. Diarrhea, abdominal pain, nausea, fever, and prostration are common.[3] The muscle invasion stage begins at the end of the first week and may last six weeks. With this stage, fever, edema of the eye lids, conjunctivitis, subconjunctival hemorrhage, muscle pain and tenderness, and severe weakness may occur.[3]

Marked peripheral eosinophilia of 20 to 80 percent, present after the third week of infection and persisting for months, may suggest the diagnosis.[5] There is also a skin test for the larval antigen that is positive from the third week up to one to two years.[4] Additionally, there are a variety of serologic tests which are usually positive at about the third week and remain positive for years. The conversion from negative to positive or an increase in titer are the most valuable findings. Muscle biopsy during the third or fourth week is the most useful test for demonstration of encysted larvae and is the definitive diagnostic procedure (Figure 5).[5] The deltoid or gastrocnemius muscles are the most useful sites for biopsy.[3]

Treatment consists of thiabendazole (Mintezol) 25 mg/kg twice a day for five to seven days. This treatment may provide apparent improvement in some patients, with relief of muscle pain and tenderness. However, once the host is thoroughly saturated with larvae, treatment is less likely to be effective and more likely to result in a serious allergic reaction.[6] Patients with allergic manifestations such as angioedema and urticaria may require prednisone in the dose of 20 to 60 mg a day.

Prevention requires adequate cooking of pork with heating to 60 C. In addition, freezing to -15 C for 20 days or -18 C for 24 hours kills the larvae.

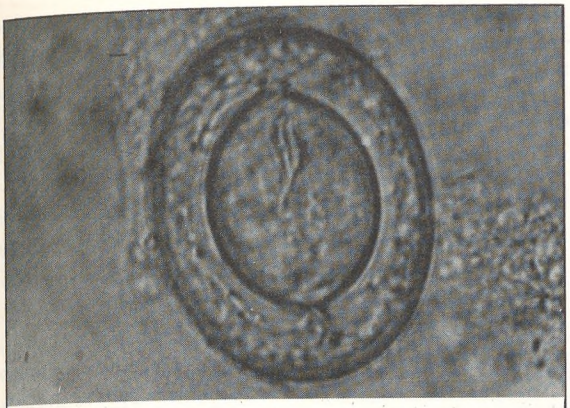

Figure 7. Hymenolepis nana. Unstained egg.

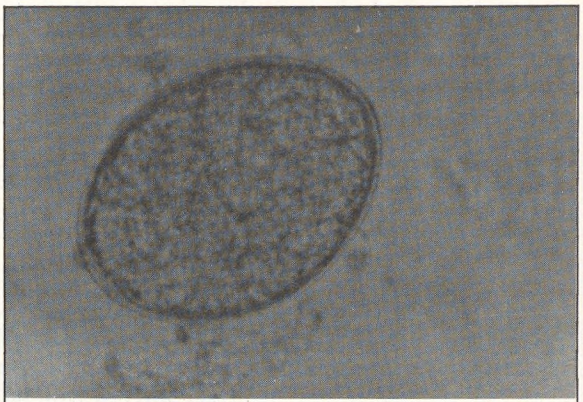

Figure 8. Diphyllobothrium latum. Unstained egg.

Cestoda—The Tapeworms

Taenia saginata–Beef Tapeworm

Infection with this worm is transmitted by ingestion of undercooked beef containing larval cysts. Man is the definitive host and the cow is the intermediate host. Infection has been reported primarily in the northeastern and western United States. However, it is now rarely acquired in the United States.[4]

Following the deposition of the ova in the soil or vegetation, they are ingested by cattle. The eggs are not infective for man. In the cow the eggs become encysted in muscle and they are subsequently ingested by humans. The egg is liberated by digestion and develops into an adult worm in about two months.[3]

Infection is usually asymptomatic, although epigastric discomfort, diarrhea, hunger sensation, weight loss, and nausea have been reported. Occasionally proglottides may crawl through the anus and appear in bed clothing.

Diagnosis is usually made by finding proglottides in feces. The eggs are indistinguishable from those of Taenia solium (Figure 6). Identification of the proglottides or examination of the scolex of the worm is required to distinguish between the two worms.

Treatment of taenia infestation consists of niclosamide (Yomesan) in a single 2 gm dose for adults. In children weighing 11 to 34 kg, the dose is 1 gm and in children weighing more than 34 kg, the dose is 1.5 gm.[9] This medication is highly effective, killing the scolex and immature segments on contact, but it is presently available only from the Parasitic Disease Service of the Center for Disease Control, Atlanta.[6] Following treatment, the worm is digested before it passes through the feces so no attempt should be made to recover the scolex. Stool should be checked at three and six months to be certain of cure.

Prevention requires thorough cooking of beef and adequate meat inspection.

Taenia solium–Pork Tapeworm

Man is the definitive host and the hog is the intermediate host for Taenia solium. In some cases, however, the larval stage may develop in humans causing cysticercosis. This term is derived from the name cysticerci given to the encysted larval stage of T solium. This larval stage may occur in man following autoinfection of a patient harboring an adult worm. The eggs of the worm are infective for both humans and hogs. This organism is practically nonexistent in the United States at the present time.[3]

Table 1. Summary of Symptomatology, Diagnosis, and Treatment

Parasite	Common Name	Source of Infection	Portal of Entry	Symptoms	Laboratory Diagnosis	Treatment
Enterobius vermicularis	Pinworm	Eggs in environment Autoinfection	Mouth	Anal itching	Eggs in perianal region by cellophane tape prep or observation of adult female at anus	Mebendazole (Vermox) 100 mg single dose; repeat in 10 to 14 days
Ascaris lumbricoides	Roundworm	Embryonated eggs from soil,food, or water	Mouth	Abdominal discomfort pneumonitis, intestinal or biliary obstruction	Eggs in feces	Piperazine citrate (Antepar) 75 mg/kg (maximum 4 gm) single dose × 2 days
Toxocara canis and cati	Dog and Cat Roundworm	Eggs from soil	Mouth	Fever, tender hepatomegaly	Eosinophilia, hypergamma-globulinemia, serologic tests	Diethylcarbamazine (Hetrazan) 2 mg/kg t.i.d.× 3 to 4 weeks
Trichuris trichiuria	Whipworm	Embryonated eggs from soil, food, or water	Mouth	Abdominal discomfort, diarrhea, anemia, or none	Eggs in stool	Mebendazole (Vermox) 100 mg b.i.d.× 3 days
Necator americanus	New World Hookworm	Filariform larvae in soil	Skin, usually feet	Intestinal pain, skin itch, pneumonitis, anemia, or none	Eggs in stool	Pyrantel pamoate (Antiminth) 11 mg/kg (maximum 1 gm) once a day × 3 days
Ancylostoma brazilience	Dog and Cat Hookworm	Filariform larvae in soil	Skin	Severe pruritis, serpiginous dermal tunnels	History and clinical findings	Thiabendazole (Mintezol) 25 mg/kg b.i.d. × 2 days
Strongyloides stercoralis	Threadworm	Filariform larvae in soil	Skin	Diarrhea, abdominal discomfort, pneumonitis	Larvae in stool or sputum	Thiabendazole (Mintezol) 25 mg/kg b.i.d. × 2 to 3 days
Trichinella spiralis	Trichina Worm	Infected pork, raw or inadequately cooked	Mouth	Fever, muscle pains, orbital edema	Muscle biopsy, skin, and serologic tests	Thiabendazole, (Mintezol) 25 mg/kg b.i.d. × 5 to 7 days and/or prednisone 20 to 60 mg daily
Taenia saginata	Beef Tapeworm	Raw or inadequately cooked beef with cysts	Mouth	None or diarrhea	Eggs or proglottids in stool	Niclosamide (Yomesan) Adult 2 gm single dose Child 11 to 34 kg 1 gm single dose > 34 kg 1.5 gm single dose
Taenia solium	Pork Tapeworm	Raw or inadequately cooked pork with cysts	Mouth	None or abdominal discomfort, diarrhea	Eggs or proglottids in stool	Niclosamide (Yomesan) Adult 2 gm single dose Child 11 to 34 kg 1 gm single dose > 34 kg 1.5 gm single dose
Hymenolepis nana	Dwarf Tapeworm	Eggs, or autoinfection	Mouth	Abdominal discomfort	Eggs or adult in stool	Niclosamide (Yomesan) Adult 2 gm/day × 5 days Child 11 to 34 kg 1 gm/day × 5 days > 34 kg 1.5 gm/day × 5 days
Diphyllo-bothrium latum	Fish Tapeworm	Raw or inadequately cooked freshwater fish	Mouth	None or weakness, weight loss, anemia, vague abdominal discomfort	Eggs or proglottids in feces	Niclosamide (Yomesan) Adult 2 gm single dose Child 11 to 34 kg 1 gm single dose > 34 kg 1.5 gm single dose

Infection begins with ingestion of the ova by the intermediate swine host and development to an encysted stage in the striated muscle. Humans become infected with the adult stage by eating undercooked pork containing cysticerci.

Clinical illness resembles T saginata when man is the definitive host. The picture is entirely different when humans serve as the intermediate host. In this case, cysticerci develop in the subcutaneous tissue, muscle, viscera, eye, and brain. In these organs the larvae behave as foreign bodies, causing marked tissue reaction with muscle pain, weakness, and fever.[3]

Infection is diagnosed by detecting eggs or proglottides in the feces or by identifying the scolex after treatment. Muscle biopsy may reveal typical encysted larvae.

Treatment has been recommended with quinacrine (Atabrine) 25 mg/kg or niclosamide (Yomesan) in a single 2 gm dose. The pediatric dose is the same as for T saginata. In the past niclosamide has not been recommended because it causes maceration of the worm and release of eggs which theoretically could lead to cysticercosis. However, there is no evidence that eggs so released in the host intestine are capable of hatching. It is probable that they require cooling outside the body or passage through the stomach before they are able to hatch. For this latter reason, the quinacrine treatment, which often causes reverse peristalsis and repeated vomiting, may provide a greater hazard of causing cysticercosis. Consequently, niclosamide is currently the recommended treatment.[6]

Hymenolepis nana–Dwarf Tapeworm

This organism represents the most common tapeworm infection in humans. It is particularly common in children. The reservoir host is the domestic mouse. Infection is acquired by ingestion of ova from contaminated food and water. The life cycle is unique among tapeworms in that both the larval and adult phase occur in the same host.[3]

Infections are frequently asymptomatic. Diagnosis rests on finding characteristic eggs (Figure 7) and proglottides in the feces.

Treatment of an adult consists of niclosamide 2 gm daily for five days.[6] The pediatric dose is 1 gm daily for five days in children weighing 11 to 34 kg, and 1.5 gm daily for five days in children over 34 kg.[9]

Diphyllobothrium latum–Fish Tapeworm

Fish with encysted larvae are the source of this infection. It occurs most often in the north central United States, Florida, and south central Canada.

The life cycle requires fresh water crustaceans as intermediate hosts before the larvae can become encysted in fish.[3]

Most infections are asymptomatic. There may be slight, transient abdominal discomfort or severe cramping, vomiting, weakness, and weight loss. In a small percentage of cases, vitamin B_{12} deficiency anemia develops because the worm competes with the host for this factor.

Diagnosis is made by finding characteristic eggs in the stool (Figure 8). Eosinophilia is often present.

Treatment consists of niclosamide in a single 2 gm oral dose for adults, 1 gm for children weighing 11 to 34 kg, and 1.5 gm for children weighing more than 34 kg.[9] Parenteral vitamin B_{12} may also be required.

Halting both continued disposal of raw sewage into lakes and ingestion of improperly cooked fish are necessary to prevent infestation.[3]

Conclusion

Human infestation with parasites is a common, but frequently overlooked, problem. Detection requires a thorough knowledge of the epidemiology and symptomatology of parasitic infections (Table 1).

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