Intestinal Nematodes

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Summary

- *Ascaris*: common, lung passage, sometimes intestinal or biliary obstruction
- *Trichuris*: common, symptoms only in severe infection (diarrhoea, anal prolapse)
- *Enterobius*: common, anal itch, exogenous auto-infection
- Hookworms: common, lung passage, anaemia if worms are numerous
- *Strongyloides*: common, chronic, larva currens, lung passage, endogenous re-infection, fatal hyperinfection
- *Capillaria philippinensis*: rare, diarrhoea, endogenous re-infection, sometimes fatal

The first five species are currently aggregated under the term “soil-transmitted helminthiasis” (STH). More than a billion people are infected with a least one species.

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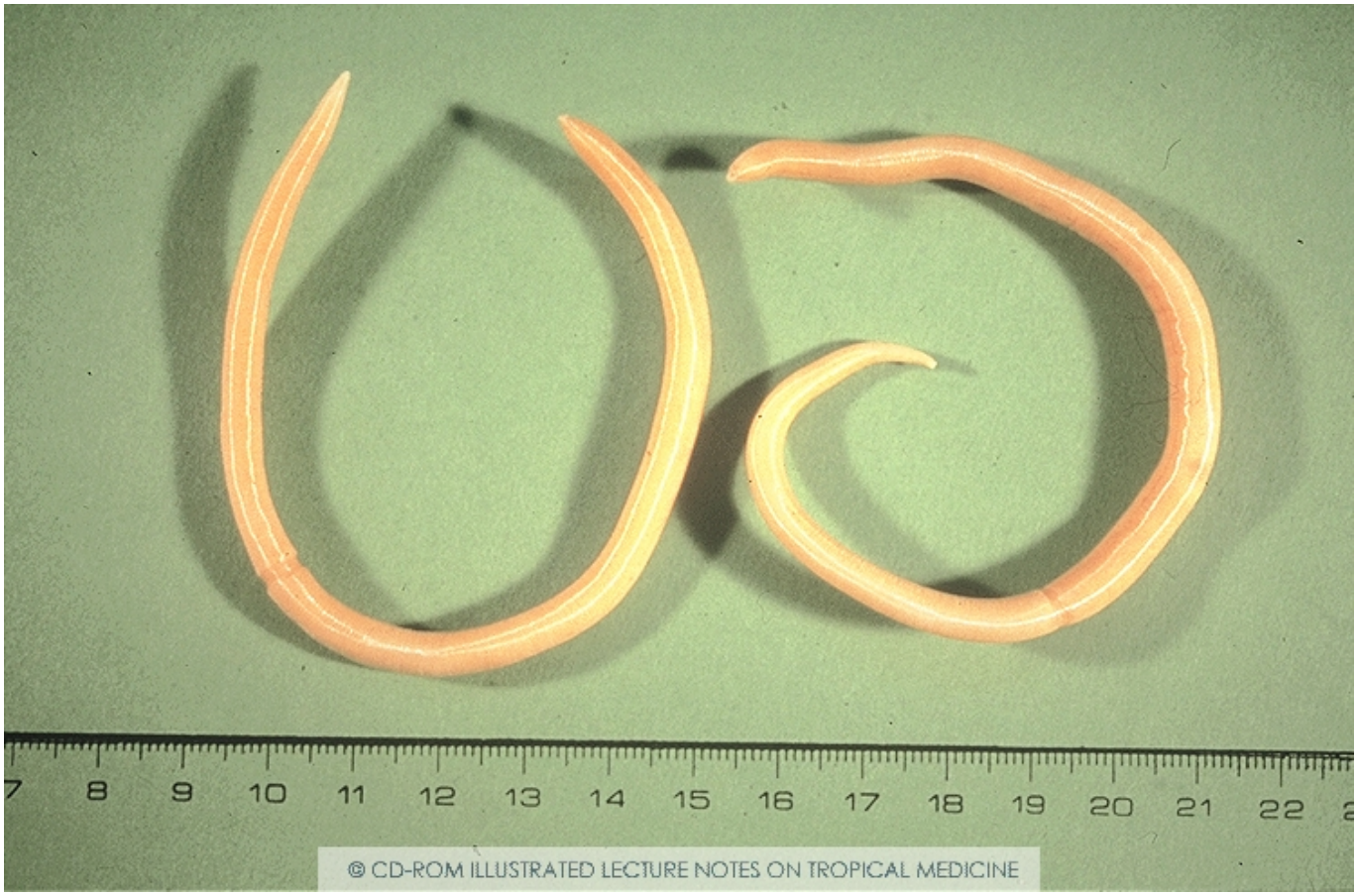
Ascaris lumbricoides

Summary

- A very common parasite, 15 to 40 cm long – jejunum (small intestine)
- Lung passage may cause transient asthma-like symptoms
- Generally atypical symptoms, or asymptomatic
- Sometimes obstruction of hollow organs (intestine, pancreas and biliary tract) causing severe complications

Life cycle



Adult Ascaris lumbricoides. ©ITM

Cosmopolitan but much more common in the tropics. The eggs pass on to the ground via the faeces. Fertilized eggs require 10 to 40 days in the outside world to mature before they become infectious. Direct self-infection is thus ruled out. Once they are mature the eggs are taken up once more (faecal-oral transmission) via contaminated **f**ood, drink (**f**luids), dirty **f**ingernails or hands. In the intestine small larvae emerge from the eggs and these bore through the intestinal wall. In this way they reach the blood (portal vein system). They are carried with the blood, through the liver to the lungs. Lung passage occurs 3 to 14 days after ingestion. In the lungs the larvae make their way to the bronchial lumen and ascent via the respiratory branches into the throat. They are subsequently swallowed and in this way they again reach the intestine. They grow into adult worms in the jejunum. They do not damage the intestinal wall. Adult worms do not multiply in the human host; the number of adult worms in an infected individual depends on the degree of exposure to infectious eggs over time. Egg laying begins two months after infection when both female and male worms are present in the intestine. Each female worm produces approximately 200,000 fertilized eggs per day. The adult worm

survives on average for 1 year. The creatures reach 15 to 40 cm, making them the largest nematode parasitizing humans. There is no animal reservoir. Occasionally infections with *Ascaris suum* occur (parasite of pigs); this worm resembles *Ascaris lumbricoides* very closely and some think the parasites are identical.

Epidemiology

This is the most common worm infection in humans. It has a cosmopolitan distribution. Children are most often infected. The eggs are very resistant, which makes it possible in certain circumstances for them to survive for a long time in the outside world (years). The number of eggs which can be found in the soil is a measure of the hygiene standard and degree of sanitation of an area (faecal pollution of the ground).

Clinical aspects

The vast majority are asymptomatic. Any illness caused by worms depends to an important extent on the number of parasites. The total worm load is only increased by repeated exposure (exceptions are *Strongyloides stercoralis* and *Capillaria filippinensis* which can multiply inside the human body). Some people have various forms of intestinal discomfort or allergic symptoms. Serious complications are rare. Nevertheless, in view of the large number of infected persons, the morbidity and mortality should not be disregarded.

Lung passage symptoms

The larvae undergo lung passage. This produces rarely symptoms of mild to severe cough, dyspnoea, thoracic pain and sometimes fever. The clinical picture is similar to asthma or pneumonia. On chest X-ray migratory infiltrates are rarely observed. Eosinophilia is present. This whole phenomenon is called “Loeffler’s syndrome”. The sputum contains many eosinophils, Charcot-Leyden crystals and sometimes also larvae. The symptoms last for some days or max. 2 weeks. Most of the time this goes unrecognized.

Obstruction of, or migration in, hollow organs

- When numerous adult worms are present they may form a tangle and cause mechanical intestinal obstruction manifested by a bloated abdomen, increased peristalsis with clangor, colicky pain, vomiting (bile, faecaloid) and dilated intestinal lumen on an abdominal X-ray.
- Migration into the biliary tract may lead to biliary obstruction (cholestasis) with possibly infection

(e.g cholangitis, liver abscess, pancreatitis).
- Sometimes there is migration to the appendix with inflammation (appendicitis).
- Sometimes an adult *Ascaris* is present in vomitus.
- Occasionally an adult can penetrate the lacrimal duct.
- Recent surgical intestinal sutures can be breached by an invasive adult *Ascaris*, leading to bowel perforation and peritonitis. Pre-operative deworming is advised in endemic areas.
- Infection with *Ascaris lumbricoides* also plays a role in the development of pigbel (clostridial necrotizing enteritis, an often fatal type of food poisoning caused by a β-toxin of Clostridium perfringens; see chapter on diarrhoea).

Malnutrition

Ascaris itself does not cause malnutrition. In borderline malnutrition the presence of numerous worms can have a negative effect, however. It is also important to know that many patients suffer from anorexia. On a population level the mass treatment (deworming) has a positive influence on the cognitive development in children.

Diagnosis

Since an adult female lays up to 200,000 eggs per day, as a rule no concentration technique is necessary to detect eggs in the faeces. If infection is solely with one or more male worms then no eggs will be detected. Stool concentration methods for detection of Ascaris eggs (rarely needed in endemic areas) include Kato-Katz and FLOTAC techniques like for other intestinal worms. Charcot-Leyden crystals, which consist of lysophospholipase, an eosinophil-derived enzyme, may be seen by microscopic stool examination.

During lung passage there is significant eosinophilia. After lung passage there is no longer appreciable eosinophilia. Sputum analysis may demonstrate eosinophils and Charcot-Leyden crystals.

X-ray of the intestine with barium contrast may show one or more adult worms. The worm forms a long, thin dark area. Sometimes a central longitudinal radio-opaque line can be seen; this is the intestinal tract of the worm. Such a line is absent in tapeworms.

An ultrasound of the pancreas (Wirsung duct) or of the biliary tract and gallbladder may show an ectopic migrating adult *Ascaris*.

Treatment

Mebendazole (Vermox®): 100 mg BD x 3 days, effective, broad spectrum (or 500 mg single dose)

Albendazole, 400 mg single dose effective, broad spectrum

Ivermectine: similar efficacy (single dose 200 µg/kg) as single dose albendazole

(Flubendazole (Fluvermal®): 100 mg BD x 3 days, effective, narrow spectrum; Piperazine (Adiver®): narrow spectrum; Pyrantel pamoate/ oxantel (Antiminth®, Combantrin®) can be used in pregnancy; Levamisole; Tribendimine; Nitazoxanide)

Pulmonary manifestations can be treated with bronchodilators or if severe with systemic corticosteroids if *Strongyloides stercoralis* infection is ruled out.

Drug resistance

Benzimidazole drugs bind to nematode β-tubulin and inhibit parasite microtubule polymerisation. Drug resistance against front-line antihelmintics is widespread in nematodes of livestock due to frequent treatment of animals. Therefore, the effectiveness of drugs must be closely monitored in regions where mass antihelmintic chemotherapy is administered.

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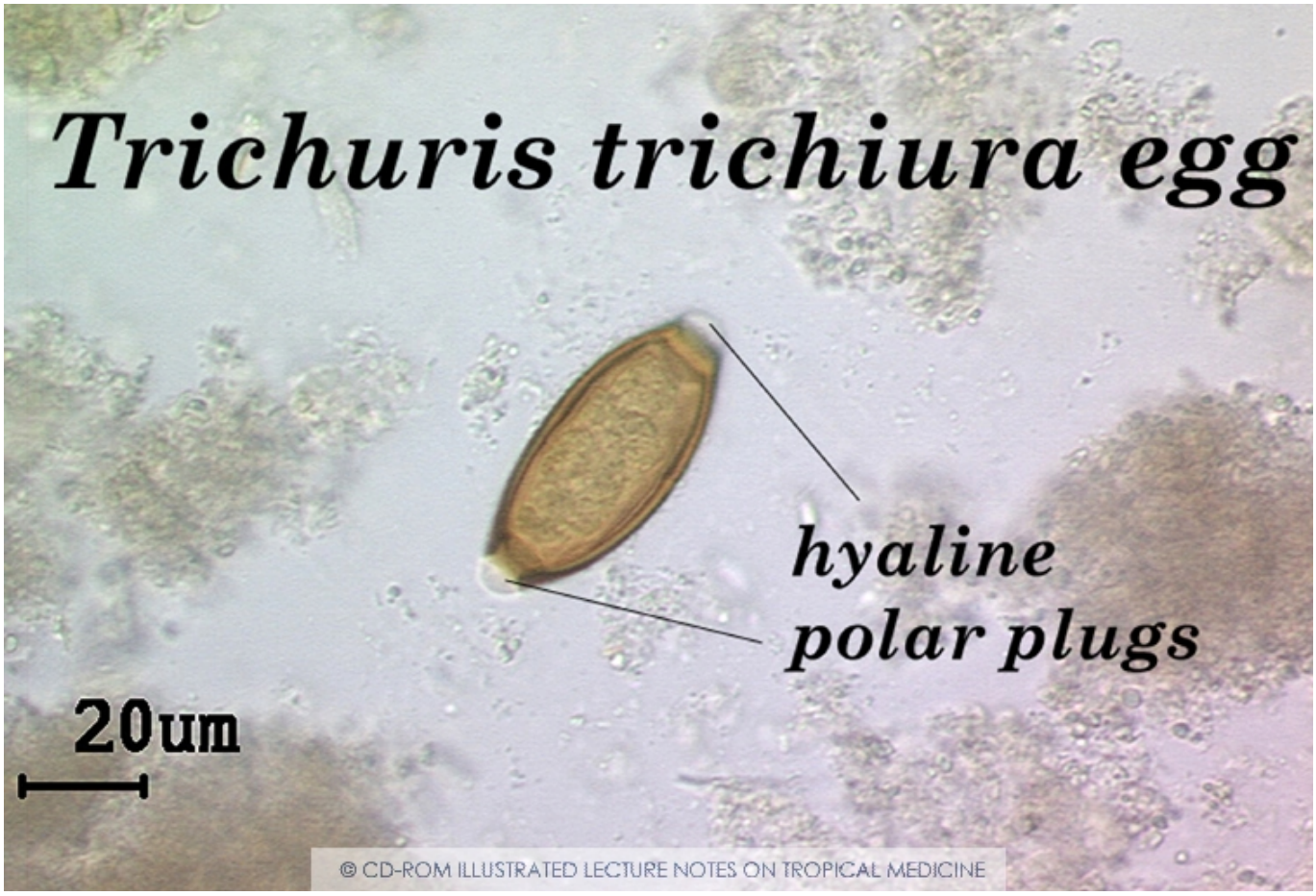
Trichuris trichiura

Summary

- Adult worms measure approximately 4 cm (sometimes seen in stool) – colon
- Faeco-oral transmission via eggs.
- Generally asymptomatic
- In severe infections diarrhoea and sometimes anal prolapse
- Role in bacterial dysentery or invasive amebiasis?

Life cycle



Trichuris trichiura egg with its typical polar caps, suggesting a lemon-shape. Copyright ITM

Trichuris suis, related to Trichuris trichiura, a nematode which frequently infects humans. Copyright ITM

Trichuris trichiura is a cosmopolitan nematode, but is rare in subarctic areas. This is an ancient parasite and this is demonstrated in that it also occurs outside the tropics is that eggs were found in Ötzi the iceman, a bronze-age mummy discovered in the Italian Alps, and in coprolites (fossilized faeces) in prehistoric salt mines in Austria.

The eggs are eliminated with the faeces. Infection is via the oral route, after obligatory maturation in the outside world. Eggs embryonate in the external environment for 10-30 days, depending upon temperature: slower when colder; no development above 37°C. It is possible that in nature (as opposed to the lab) much longer periods are possible. Many eggs remain viable in the soil for longer than a year; depending upon local humidity. In Bangladesh, a study of 2400 houseflies discovered

that 47% of the insects were carrying eggs (flies acting as mechanical transport vectors).

The embryonated eggs hatch after ingestion. It is likely that the hatching worm dissolved the polar caps with enzymes. The fate of the larvae after hatching the first 5-10 days is controversial. No studies have been done on humans. Serial necropsy of dogs infected with *T. vulpis* suggest that larvae first penetrate the mucosal duodenal epithelium, re-emerge into the lumen 8-10 days later and settle in the caecum. However, this data is questioned and it is unclear if this can be generalized to human infections. More study is required to answer some basic questions.

Larvae will undergo four molts. Egg laying begins about 2 months after infection. Experimental infection in human volunteers showed a somewhat longer prepatent period of 120-130 days. It is estimated that 5-22% of ingested embryonated eggs develop to patency. A female worm measures 3-5 cm and sheds between 3000-20,000 eggs per day. Since the uterus of a female worm contains approximately 60,000 eggs at any one time, this implies that between 5 and 30% of the eggs have to be replaced on a daily basis. The adult worm has a thin whip-like head with which it buries itself in the mucosa of the large intestine especially the caecum. The worm survives for 1-4 years on average, although extremes of 20 years are known.

Clinical aspects

Most infected humans remain asymptomatic. Only in severe infections (> 1000 worms; >10,000 eggs per gram of faeces) do symptoms occur: these include diarrhoea (dysentery type), malnutrition or anaemia. In undernourished children with chronic diarrhoea and tenesmus there is sometimes prolapse of the rectum, in which the worms can be seen on the prolapsed mucosa.

Diagnosis

Diagnosis is based on faecal examination. No concentration technique is necessary for clinically relevant infections. The Kato-Katz technique can be used to quantify egg numbers. Sometimes the worms can be seen on the rectal mucosa (rectoscopy or during anal prolapse). Normally there is no eosinophilia (since there is no larval migration).

Treatment

- Mebendazole 100 mg BD x 3 days, or 500 mg single dose (but less active: 65-70%)
- Albendazole 400 mg BD x 3 days (for cure rate above 90%)
- Ivermectine is also less active
- The combination treatment albendazole plus oxantel pamoate showed higher cure rates and higher

egg reduction rates than mebendazole or albendazole alone.
- The new tribendimidine drug has limited activity

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Hookworms

Summary

- Blood-sucking worms 1 cm long (but never seen in the stool); in the jejunum
- Transmission by larvae: transcutaneous and oral
- Brief local itch after skin penetration, lung passage (but very rarely noticed)
- Generally asymptomatic
- In severe infection iron deficiency anaemia

Life cycle

There are two important hookworms: *Necator americanus* and *Ancylostoma duodenale*. [L. necator = murderer; Gr. ancylo = hook, stoma = mouth]. There are a few zoonotic hookworms which are of much less clinical importance and seldom cause infections in humans (e.g. *Ancylostoma ceylanicum*, *A. caninum*, *A. malayanum*, *Cyclodontostomum purvisi*). The adult worms are found in the small intestine. It is estimated that the life span of adult worms is 5 to 15 years. *Necator* lives longer than *Ancylostoma*. The adults measure approximately 1 cm. A few weeks or months after infection eggs can be found in the faeces. Once the eggs arrive in the outside world with the faeces, they take one week to mature to infectious larvae. At first they are rod-shaped = rhabditiform, later thread-shaped = filariform. They may survive for weeks or months (at an optimal temperature and humidity for as much as 2 years). A soil with neutral pH is optimal for their development, as is shade and a sufficiently high temperature (23°C to 30°C is ideal). If the faeces mix with urine the eggs die. Frost, direct sunlight and a soil saturated with salt or water are unfavourable conditions.

Infection occurs via the mouth (*A. duodenale*) or via the skin (*A. duodenale* and *N. americanus*). If they enter through the skin, the young parasites have to pass through the lungs. A new dimension in the epidemiology of hookworm disease emerged when it was found that insufficiently cooked meat from paratenic hosts (= an intermediate host in which no development of the parasite occurs) such as pigs, cattle, rabbits and sheep can be responsible for transmission. The adult hookworms bore a hole in the mucosa of the duodenum and the small intestine and suck blood. They adhere with hooked

teeth in their mouth (*Ancylostoma*) or with two buccal cutting plates (*Necator*). *A. duodenale* sucks 5 to 10 times more blood than *N. americanus* (approximately 30 µl per day for *Necator* and 260 µl for *Ancylostoma*). Blood loss is caused primarily by parasite release of anticlotting agents -anticoagulant peptides that inhibit activated factor X and factor VIIa/tissue factor complex and that inhibit platelet activation- which causes continuous blood loss in the stool and only secondly due to actual blood consumption by the worm.

Clinical aspects

At the site where the hookworms penetrate, the skin may rarely develop a rash and itch (called “ground itch”). This is short-lived and rarely noticed. Lung passage also rarely produces symptoms, but may be accompanied by Loeffler’s syndrome. There are few intestinal symptoms. When infection with *A. duodenale* occurs by the oral route, the early migrations of third-stage larvae cause a syndrome known as Wakana disease, which is characterized by nausea, vomiting, pharyngeal irritation, cough, dyspnoea and hoarseness. Significant infections (>1000 worms) may result in pronounced anaemia. The haemoglobin level may sometimes be very low. Children and pregnant women in whom the iron supplies are already low, are particularly affected. Hypoproteinaemia may also occur and results in oedema and anasarca. Protein deficiency also has consequences for the production of immunoglobulins. Some patients exhibit geophagia. In history, certain regions in the USA were famed for their “quality” clay and people would cover great distances to eat this iron-containing soil.

Differential diagnosis:

Differentiation from *Strongyloides* larvae is based chiefly on the difference in morphology of the “head” end. The mouth is elongated in ancylostomes and shorter in *Strongyloides*. Sometimes, if intestinal transit has been swift eggs of *Strongyloides stercoralis* may be found in the faeces. These too should be differentiated from hookworm eggs.

Diagnosis

The eggs are found in fresh faeces. In an old stool (>24 hours) the eggs will have hatched and rhabditiform larvae can be seen (Gr. rhabdos = rod). There is mild eosinophilia. Since an adult hookworm lays approximately 25,000 eggs per day, as a very rough estimate 100 eggs per gram of faeces corresponds to 1 adult worm. The Kato-Katz concentration technique can be used to estimate the number of eggs per gram of faeces. The eggs of N. americanus and A. duodenale are morphologically indistinguishable.

Eggs of *Oesophagostomum* are morphologically identical to those of hookworms. Identification of the latter parasite can only be made by coproculture (identification of the typical stage 3 larvae).

Treatment

- Mebendazole 2 x 100 mg/day for 3 days. Also give iron supplementation and folic acid in anemia.
- Albendazole may be used in treatment (400 mg single dose) and is generally effective.
- Pyrantel 10 mg/kg for 3 days or levamisole 2.5mg/kg once or twice (less used nowadays)
- *Necator* and *Ancylostoma duodenale* are less sensitive to ivermectin (cure rate around 30%).
- Tribendimidine has a promising activity on hookworms

Prevention

Mass chemotherapy together with health education and sanitary provisions are strategies which are often used for morbidity control. The most heavily infected individuals are the chief target group. There are however increasing concerns about long-term sustainability. Wearing footwear only partly prevents infection because oral infection is also important for *Ancylostoma duodenale*. Children are the main victims as they rarely wear shoes and their whole skin is a portal of entry.

Cutaneous larva migrans
Some larvae from animal hookworms may penetrate human skin, but do not migrate deeper to the underlying tissues and organs. Their cycle thus reaches a dead end in the skin. Examples are the hookworms of dogs and cats (Ancylostoma braziliense, Ancylostoma caninum) and animal Strongyloides species. The migration of these larvae causes very itchy red lines on the skin which slowly move about (i.e. creeping eruption). A single oral administration of 12 mg of ivermectin (or albendazole 400 mg x 5 days) is effective.

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Strongyloides stercoralis

Summary

- Infection with small worms 3 mm long (never seen in the stool) – small intestine
- Transmission by larvae is transcutaneous or oral
- Importance of endogenous re-infection and multiplication, which lead to very long-term infections

- Hypereosinophilia, larva currens with itch, chronic lung problems
- Hyperinfection in immunosuppression with steroids, HTLV-1

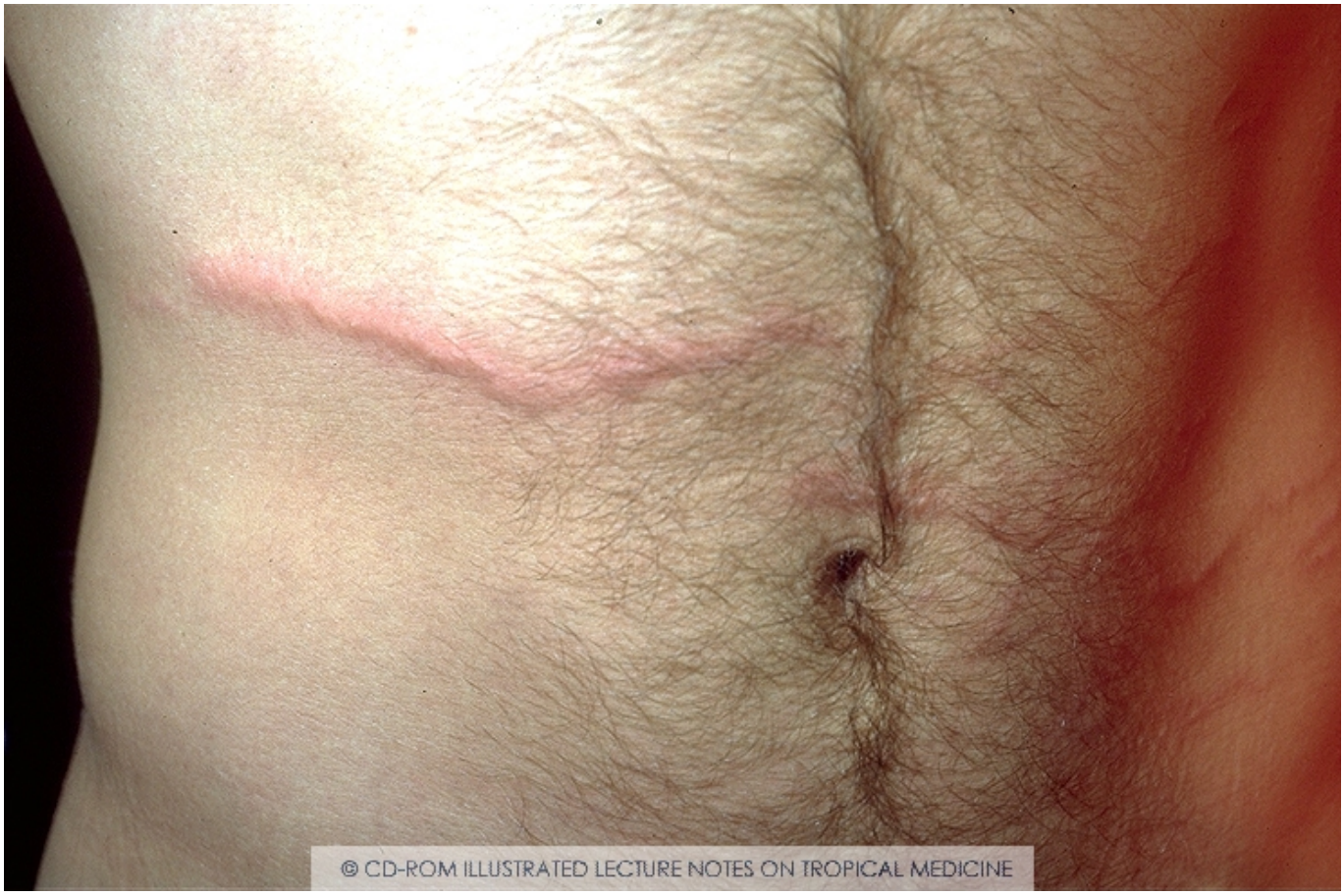
Life cycle

The adult female worm is found in the mucosa of the small intestine. Males cannot penetrate the intestinal mucosa and perish. Reproduction is asexual via parthenogenesis (=development of an embryo from an unfertilized egg cell). The females lay eggs after 2-3 weeks, from which larvae are quickly produced. Initially the larvae are described as rhabditiform. These quickly develop into filariform (infectious) larvae. These larvae may:

- either penetrate back into the intestinal mucosa (*Strongyloides* is one of the rare worms which can multiply in the human body).
- or pass to the perianal skin and from there again penetrate the body (auto re-infection). In auto re-infection there is always another lung passage. In this way an infection with *Strongyloides* may persist for a very long time (more than 30 years).
- or pass to the outside world with the faeces. From there after molting, they may go in either of two directions. The larvae either again penetrate the skin of a human (sometimes even via the mouth) or they develop to adult worms in the outside world. They may then via sexual reproduction in their turn lay eggs, from which new larvae develop. The worm can thus survive without a host.

Clinical aspects

Mild infection is generally asymptomatic. In severe infections there may be intestinal discomfort or diarrhoea. During lung passage symptoms may occur depending on the number of larvae. Auto re-infection via the skin may give sometimes rise to significant itching, chiefly peri-anal. Migration of the larvae in the skin leads to itching red swollen lines (on the rump, arms, face, etc.). These lines may occur anywhere and progress swiftly (up to 10 cm per hour). The swelling is the result of an urticarial reaction to the migrating larva (the larva itself is only 0.2 mm long). These lesion disappear spontaneously a few hours later, to reappear once more at a different site and this rather typical symptom is called “larva currens” (observed at some moment in about 20% of infected individuals).



Strongyloides stercoralis, larva currens. Such recurrent migrating linear urticarial stripes are pathognomonic for infection with this parasite. Copyright ITM

Immune suppression (especially HTLV-1 infection), achlorhydria (low gastric acid secretion), haematological malignancies including lymphoma, nephropathy, transplant patient taking immunosuppression (cyclosporine, tacrolimus), cytotoxic medication but especially the long-term use of systemic corticoids, all increase the risk of hyperinfection. In such cases there is extensive multiplication with spread of the larvae to all organs (including the brain) due to a dysfunction of the Th-2 helper cells. Symptoms include purpura-like skin lesions (initially often peri-umbilical), severe diarrhoea, pulmonary symptoms (dyspnoea, bronchospasms, bloody sputum) and meningo-encephalitis. Hyperinfection with *Strongyloides stercoralis* may be accompanied by bacterial septicaemia (with usually Gram-negative bacteria). Mixed infection may occur. This probably depends on mechanical damage to the colon wall; adhesion of intestinal bacteria to the outside of migrating larvae and excretion of bacteria from the intestinal system of the parasite. Hyperinfection has a high mortality (75%). In chronic and persistent infection an underlying infection with HTLV-1 or use of

glucocorticoids should be considered. There have been fewer hyperinfections in AIDS patients than one would expect at first sight.

Diagnosis

The eggs hatch very rapidly in the intestine and are often not found in a faecal specimen. Larvae are found in the faeces. Often the numbers are not so high and specific concentration techniques, e.g. the Baermann method or modified agar plate method need to be used. In general, the diagnosis of *S. stercoralis* infection is difficult in the tropics as well as in travellers. Larvae can also be detected via duodenal intubation. Differentiation from hookworm larvae is necessary. Eosinophilia is almost always present, except when immune suppression exists. A history of larva currens is suggestive of strongyloidiasis and is enough to start treatment even if no larvae are found in the faeces. In hyperinfection larvae may be found in the sputum or in broncho-alveolar lavage fluid. The sputum must be regarded as infectious. If this sputum is cultured on blood agar, bacterial colonies can be seen which form a curvilinear pattern, reminiscent of a pearl necklace. This follows the migration of a larva on the agar plate, with translocation of the bacteria.

PCR on a stool sample is the most sensitive test, but is not widely available.

An ELISA test detecting IgG to filariform larvae in serum, can be used in immunocompetent hosts. However ELISA results can be falsely negative in immunocompromised hosts and in acute infection (as seen in travellers) during the window period. Cross-reactivity may occur in the presence of other helminth infections. As a whole serology is widely used in travel medicine to diagnose (past) exposure to *Strongyloides* but is almost nowhere available in the tropics.

Treatment

- Thiabendazole was used in the past, but had many side effects. Albendazole (400 mg twice daily for 3 to 7 days) is moderately effective. Mebendazole is not active.
- Ivermectin PO (200 µg/kg single dose) is easy to use and effective and at present is the **first line treatment**. Some experts recommend a second course after 1-2 weeks (an RCT is ongoing to answer this question). If immunosuppression is present, the cure rate with ivermectin is lower, certainly if cortisone has been taken. In such cases, successive courses of treatment should be administered. It should be mentioned that there are parenteral ivermectin formulations for veterinary use. They are not (as yet) registered for use in humans, but anecdotal case reports mention success with them.
- In hyperinfection it is important not to forget to use antibiotics, in view of the risk of severe

septicaemia.

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Enterobius vermicularis

Summary

- Cosmopolitan distribution
- Humans are the reservoir of this 1 cm long worm
- Ileocaecal region ? Anal region: anal itch

Life cycle

This parasite is cosmopolitan. There is no intermediate host. Infection is via ingestion of eggs e.g. by eating food touched by contaminated hands or by handling contaminated clothes or bed linens. Eggs accumulate in the ileo-caecal region. After copulation the males die. The females migrate via the colon to the anus and lay their eggs chiefly at night as they creep over the peri-anal skin. This explains the nightly itching. Self-infection occurs by transferring infective eggs to the mouth with hands that have scratched the perianal area. Retro-infection or the migration of newly hatched larvae from the anal skin back into the rectum, may also occur. In rare cases there is vaginal itch because the females can also hide there. Sometimes the parasites are found in the appendix. The eggs must be sought not only in the faeces, but also on the peri-anal skin (using Scotch tape or other transparent sticky tape). In women the eggs may be found in the urine due to contamination. Apart from the itch there are few problems. There is a possible association between infection with *Enterobius* and infection with the possibly pathogenic amoeboflagellate, *Dientamoeba fragilis*. A hypothesis is that Enterobius vermicularis serves as a vector for *D. fragilis*, as *D. fragilis* DNA has been detected within surface-sterilized eggs of *E. vermicularis*.

Treatment

- Mebendazole 100 mg (Vermox®), to be repeated after 1 and 2 weeks. Albendazole is also effective.
- Ivermectin 12 mg single dose, to repeat after 2 weeks
- Pyrantel pamoate 10 mg/kg base once (max. 1 g); to repeat in 2 weeks
- Vanquin® (pyrvinium) may also be used as an alternative to mebendazole. The faeces may

discolour red.

Since the eggs can adhere to all objects e.g. underclothing, sheets and so on, these should be changed. In a family it is best to treat all the family members, even those without symptoms.

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Capillaria philippinensis

Summary

- Infections with *Capillaria philippinensis* are rare, but potentially fatal
- Transmission by eating infected fish
- Endogenous multiplication resulting in chronic malabsorption and diarrhoea

Life cycle

Capillaria philippinensis is a nematode which causes severe infections. The parasite was discovered in 1960 in Luzon, an island in the Philippines. Subsequently it was also found in Thailand, Indonesia, Egypt, Japan, Taiwan, Korea and Iran. It is a parasite of fish-eating waterbirds. The infection occurs due to eating infected fish which live in fresh or brackish water. The larvae are found in the muscles of the fish. It is an intestinal nematode which has an intermediate host (most nematodes don't). After developing to adult forms the parasites, which are 2 to 4 mm long, live in the mucosa of the small intestine. The worm is capable of multiplication in the human intestine (cf. *Strongyloides*). This phenomenon may lead to severe infection (high worm load). The incubation period can be very long (many months). Chronic watery diarrhoea, malabsorption and cachexia follow. The diarrhoea can be high volume (several litres per day). Ascites, pleural fluid and severe electrolyte imbalance including hypokalaemia may occur. The infection is sometimes fatal if not treated in time.

Diagnosis and treatment

Diagnosis is made by means of faecal examination. Often it is necessary to analyse multiple stool samples before eggs are found. Intestinal biopsy can show worm fragments. Every infection must be treated promptly with mebendazole, 200 mg x 2 per day for 20 days or albendazole x 10 days. Cooking fish prevents the infection. Eating raw fish is a culinary habit in many Asiatic countries and this is difficult to change.

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Oesophagostomiasis

Nematodes of the genus *Oesophagostomum* (*O. bifurcum, O. aculeatum, O. stephanostomum*) are widely distributed intestinal worms of monkeys. In some regions humans are accidental final hosts. Foci of *Oesophagostomum bifurcum* infections occur commonly in parts of West Africa (Northern Ghana and Togo) with very high prevalence in some villages. The eggs are morphologically identical to those of hookworms. The larvae develop when the eggs land on the ground, progressing through stages 1-3 in 5 to 7 days. Probably a number of stage 3 larvae can resist long periods of dehydration. Stage 3 larvae are swallowed with food or water and penetrate the human intestinal wall. They then develop further inducing **abscesses with a necrotic content (helminthoma)**. The worms may cause severe intestinal lesions, including eosinophilic granulomas in the intestinal wall (mostly caecum) and mesentery, deep abscesses and peritonitis. Epigastric or periumbilical masses may result.

As soon as the worms become adult they return to the intestinal lumen where they attach to the mucosa and mate. Adult worms in the intestinal lumen do not cause illness. In veterinary medicine the illness is known as “pimply gut” which refers to countless abscesses under the serosa.


CD-ROM ILLUSTRATED LECTURE NOTES ON TROPICAL MEDIC

Oesophagostomiasis with intestinal abscess spreading to the abdominal wall. Photo prof Gigase.

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Anisakiasis

Adult *Anisakis simplex* have been found in the stomachs of whales, seals, sea lions, walruses and dolphins. Humans are incidental hosts and the human “equivalent” of anisakiasis for sea animals is ascariasis. The eggs are eliminated with the faeces. In sea water the eggs hatch after embryonation after which the released larvae penetrate small crustaceans e.g. copepods or krill, which then in turn are eaten by fish or cephalopods. *Anisakis* larvae are usually restricted to the fish viscera in vivo only infesting the muscles after the fish has been killed, particularly if the fish is not promptly gutted and cleaned after its death. Humans become infected by eating undercooked or raw infected marine fish. The parasites which measure about 2-3 cm in length attach themselves to the gastric or intestinal mucosa by their anterior parts as far as the muscularis mucosa. This makes them visible during endoscopy.

In humans the parasites do not reach the adult stage and usually die off spontaneously after 3 weeks. The dying organism induces an inflammatory reaction and a tissue abscess develops with a predominance of eosinophils. **Gastric pain and nausea/vomiting** may occur within a few hours after eating infected fish or cephalopod but symptoms may have a late onset with abdominal pain appearing up to three weeks later. Late manifestations have rarely been described (several weeks to months) and are due to more distal intestinal infections. The infection is sometimes confused initially with appendicitis, stomach ulcer, duodenal ulcer, stomach cancer or Crohn’s disease. Rarely the worms perforate the intestinal wall and are found in the peritoneum. Eosinophilia is present. Approximately 95% of all cases in the world, which amounts to some 2000 cases annually, occur in Japan. Many different species of *Anisakis* larvae are being recognized as the cause of **urticaria and hypersensitivity reactions** after eating fish. The worm can in fact trigger quite dramatic hypersensitivity reactions even after it is dead. The first signs of an allergic reaction usually occur 60-120 minutes after ingestion, but may be delayed for up to 6 hours later probably due to passage of the food bolus through the gastro-intestinal tract. This means that urticaria and angio-oedema may occur at night. The diagnosis of allergy to *Anisakis simplex* is based on (1) a compatible anamnesis such as urticaria or angio-oedema after consumption of saltwater fish, (2) a positive skin prick test, (3) specific IgE against *Anisakis* simplex via radio-immunoassay, (4) negative reactions to the proteins of fish. There are some people who have antibodies to *Anisakis* without ever having exhibited

symptoms.

Therapy of anisakiasis consists of mechanical removal by means of surgery (in case of intestinal obstruction) or endoscopic extraction. Ivermectin and albendazole therapy has been suggested.

Thorough cooking to 70ºC or adequate freezing to -20ºC for a minimum of 72 hours are the best preventive measures.

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