Chapter 22

COMMON WORM INFESTATIONS IN SA

NEMATODES (ROUNDWORMS)

ASCARIS LUMBRICOIDES (COMMON ROUNDWORM)

Ascaris lumbricoides is the largest intestinal nematode (roundworm) that infects humans and is one of the most common helminth infections globally. *Ascaris suum* is a roundworm parasite of pigs and can infect humans. It has become more prevalent in areas where exposure to pigs enables ingestion of infectious eggs. Use of pig faeces for fertilizer has increased its transmission. Clinical infection is similar to *A. lumbricoides*.

Ascaris eggs, passed in stool, are deposited in the soil where they embryonate and become infective after two to four weeks. Transmission occurs via ingestion of water or food contaminated with *Ascaris* eggs. The eggs hatch in the small intestine releasing larvae which penetrate the intestinal wall and migrate to the liver and lungs. The larvae ascend to the pharynx and are swallowed, and once back in the intestine, they mature to adult worms.

Most patients are asymptomatic, but all infected patients should be treated. When symptoms occur, it is usually during the late phase as adult worms (intestinal symptoms) but can occur early during larval migration (pulmonary manifestations).

Peripheral eosinophilia as seen on a full blood count is a diagnostic clue and stool can be examined for eggs during late stage infection.

If pulmonary manifestations are severe, systemic corticosteroids may be administered. Antihelminthic therapy is not to be used because the effect on larvae causing the pulmonary manifestations is unknown. Stool should be examined two months after the pulmonary symptoms developed and antihelminthic treatment given if needed.

ASCARIS LUMBRICOIDES: ANTIHELMINTHIC THERAPY

Mebendazole (Vermox®) 100 mg PO 12 hourly for 3 days or 500 mg PO as a single dose (contraindicated during pregnancy)

OR

Albendazole (Zentel®) 400 mg PO given as single dose (contraindicated during pregnancy and children under 1 year)

Piperazine (50–75 mg/kg PO once daily up to a maximum of 3.5 g for 2 days) is seldom used because other alternatives are more efficacious and less toxic, however, it may be useful for cases in which intestinal or biliary obstruction is suspected because the drug paralyses the worm aiding in expulsion.

Endoscopic or laparoscopic extraction of the worms may be necessary in the setting of hepatobiliary disease.

Neither mebendazole nor albendazole is recommended during pregnancy. For most worm infestations, therapy can be deferred until after delivery.

ENTEROBIUS VERMICULARIS (PINWORM)

Enterobiasis is the most common helminthic infection in the United States and Western Europe. Humans are the only natural host and infection occurs in all socioeconomic conditions. *E. vermicularis* is transmitted by ingestion of infective eggs. An adult worm deposits eggs on the perianal folds. Autoinfection occurs by scratching the area and transferring the eggs to the mouth with contaminated hands. Infection can also occur by contact with contaminated environmental surfaces.

Once ingested, they hatch and release larvae into the small intestine. Adult worms attach in the caecum and appendix. Female worms migrate through the rectum onto the perianal skin to deposit eggs.

Most infections are asymptomatic. The most common symptom is perianal itching (pruritus ani) and scratching can lead to secondary infection. Adult worms can migrate to extra-intestinal sites and vulvovaginitis has been described.

Diagnosis is made using sticky cellulose tape pressed against the skin of the perianal region and viewed on a slide. The eggs can be visualised best if an early morning or evening sample is taken.

ENTEROBIUS VERMICULARIS: ANTIHELMINTHIC THERAPY

Mebendazole (Vermox®) 100 mg PO as a single oral dose in adults and children. Repeat after 2 weeks to eliminate re-infection

OR

Albendazole (Zentel®) 400 mg PO as a single oral dose. Repeat after 2 weeks

Simultaneous treatment of all family members is necessary for eradication of other reservoirs of infection. All bedding and clothes should be washed. Hygienic measures include clipping of fingernails, frequent handwashing and regular baths.

TRICHURIS TRICHIURA (WHIPWORM)

Trichuriasis is very common and it is estimated that up to a quarter of the world's population is infected with whipworm. It is found in individuals of all ages, associated with conditions of poor hygiene. The life cycle starts with the passage of unembryonated eggs in the stool. In the soil the eggs embryonate, become infective and once ingested hatch in the small intestine. Larvae mature in to adult worms which become established in the colon and caecum. The thin end of the worm is embedded in the bowel mucosa and the thick end is visible in the lumen. Most infections are asymptomatic, with symptoms only developing with moderate to heavy infections. Re-infection is common following treatment in endemic areas.

TRICHURIS TRICHIURA: ANTIHELMINTHIC THERAPY

Mebendazole (Vermox®) 100 mg PO 12 hourly for 3 days

OR

Albendazole (Zentel®) 400 mg PO once daily for 3 days

Treatment of trichuriasis with single oral doses of antihelminthics is not satisfactory.

HOOKWORM

There are two predominant species of hookworm that cause human infection: *Ancylostoma duodenale* and *Necator americanus*. The prevalence of hookworm infection is highest in Sub-Saharan Africa, Asia and other tropical regions. Three conditions are important for hookworm transmission: human faecal contamination of soil, favourable soil conditions for larvae survival and contact of human skin with the contaminated soil.

Hookworms (the infective larvae) enter the body, usually the feet, by penetrating the skin. The larvae migrate via blood vessels to the lungs during the following eight to 21 days. There they penetrate alveoli and ascend to the pharynx where they are swallowed. Larvae mature into adult worms in the small intestine and where they attach to the intestinal wall, they cause blood loss.

There are four potential clinical manifestations: symptoms due to dermal penetration of the larvae, transpulmonary passage, acute gastrointestinal symptoms and chronic nutritional impairment.

An unexplained eosinophilia seen on a full blood count is a major clue to hookworm infection. Stool examination for eggs is useful from eight weeks after dermal penetration with *N. americanus* and up to 38 weeks with *A. duodenale*.

HOOKWORMS: ANTIHELMINTHIC THERAPY

Mebendazole (Vermox®) 100 mg PO 12 hourly for 3 days

OR

Albendazole (Zentel®) 400 mg PO as a single dose

Iron replacement may be needed to restore haemoglobin levels.

STRONGYLOIDES STERCORALIS (THREADWORM)

Strongyloides stercoralis is endemic to sub-tropical and tropical regions and causes the disease strongyloidiasis. Infection caused by *S. stercoralis* can range from asymptomatic eosinophilia in the immunocompetent host, to disseminated disease with septic shock in the immunocompromised host. The life cycle begins when larvae in soil or other surfaces, come into contact with human skin. The larvae penetrate the skin and migrate to the lungs. They penetrate into alveoli and ascend to the pharynx where they are swallowed. In the duodenum and jejunum they burrow into the mucosa and mature into adult worms. Adult females produce eggs which hatch and release non-infectious larvae. The larvae can transform into infective (filariform) larvae within the gut and penetrate perianal skin or colonic mucosa, resulting in autoinfection of the human host.

This cycle of autoinfection can result in a high burden of adult worms in infected humans. In immunocompromised hosts, this massive worm burden can lead to dissemination of filariform larvae to multiple organ systems. The subsequent inflammatory response produces organ dysfunction, the manifestations of which are known as hyperinfection syndrome.

STRONGYLOIDES STERCORALIS: ANTIHELMINTHIC THERAPY

Albendazole (Zentel®) 400 mg PO 12 hourly for 3 days. Repeat after 3 weeks if necessary

OR

Ivermectin (not registered in South Africa) given as 200 μg/kg PO either on 2 consecutive days or 2 weeks apart. In studies, the efficacy of ivermectin is superior to albendazole.

Hyperinfection syndrome – prolonged or repeat treatment is advised; though there is no consensus on a fixed duration. Some experts advise 5–7 days but it would be reasonable to continue treatment until symptoms resolve and daily stool examination is negative for two weeks. Combination of ivermectin and albendazole has also been suggested.

Patients treated for *Strongyloides* should have follow-up stool specimens collected two to four weeks after treatment. Stool microscopy is insensitive, and if symptoms persist despite a negative stool examination, then it may still indicate possible treatment failure.

CUTANEOUS LARVA MIGRANS

This is a parasitic infection caused by accidental penetration of the skin by larvae of certain nematodes. Humans are considered end hosts and the parasites are generally unable to penetrate through the dermis. They therefore remain limited to the skin.

The rash manifests as an erythematous, serpiginous eruption which may be vesicular and which advances one to two centimetres per day from the site of penetration. It is usually intensely pruritic. The condition is normally benign and self-limited, with the condition clearing spontaneously within approximately four weeks. Prolonged infestations of up to a year have however been described. Common exposures include walking or sun-tanning on a beach or children playing in a sandpit in which animals may have defecated. While classically associated with tropical or sub-tropical climates, travel has resulted in this condition being encountered in most parts of the world.

A diagnosis is based on the classical clinical presentation. A minority of patients may demonstrate eosinophilia on the full blood count.

Complications include secondary bacterial infections and possible allergic reactions.

CUTANEOUS LAVA MIGRANS: ANTIHELMINTHIC THERAPY

Adults: Albendazole (Zentel®) 400 mg PO once daily for 3 days OR 200 mg PO 12 hourly for 5 days

Children > 2 years of age: Albendazole as for adults

Children 1–2 years of age: Albendazole 200 mg PO once daily for 3 days (repeat after 3 weeks if necessary)

OR

Ivermectin (not registered in South Africa) 200 μg/kg PO daily for 1–2 days

Antihistamines are helpful for the management of pruritis.

CESTODES (TAPEWORMS)

INTESTINAL TAPEWORM INFECTIONS

Cestodes (tapeworms) are flat worms that can live as parasites in the human gastrointestinal tract. Certain tapeworms are primarily human pathogens, while others have animals as their natural hosts but can also cause human infection. Intestinal tapeworm infections in humans are caused by:

- *Taenia* species. *T. solium* (pork tapeworm), *T. saginata* (beef tapeworm) acquired by eating undercooked pork or beef containing cysticerci.
- *Diphyllobothrium* species. Acquired by eating raw or undercooked fish.
- *Hymenolepis* species (dwarf tapeworm).
- *Dipylidium caninum.* These usually infect dogs and cats. Humans may accidentally be infected after ingestion of infected fleas.

The diagnosis is generally established by identifying eggs or proglottids in the stool.

INTESTINAL TAPEWORMS: ANTIHELMINTHIC THERAPY

Praziquantel (Biltricide®) 5–10 mg/kg PO given as a single dose. For *Hymenolepis* species use 25 mg/kg PO as a single dose

OR

Niclosamide (Yomesan®) 2 g (4 x 500 mg tablets) PO as a single dose (chewed). In children 2–6 years, the dose is 1 g (2 x 500 mg tablets) as a single dose, and in children less than 2 years the dose is 500 mg (1 x 500 mg tablet) as a single dose. For *Hymenolepis* species use 2 g PO on first day, followed by 1 g daily for 6 days.

CYSTICERCOSIS

Cysticercosis is due to tissue infection with larval cysts of the pork tapeworm, *Taenia solium*. Human infection occurs following ingestion of *T. solium* eggs. Following ingestion, embryos hatch in the small intestine, invade the bowel wall and spread hematogenously to the brain, striated muscles, liver, or other tissues. Over a period of three to eight weeks, tissue cysticerci develop.

Clinical syndromes related to cysticercosis are divided into:

- Neurocysticercosis, which is divided into:
  - Parenchymal forms – usually presenting with seizures.
  - Extra-parenchymal forms, which include:
    - Intraventricular cysts
    - Subarachnoid cysts
    - Ocular disease
    - Spinal disease

  These extra-parenchymal forms may be associated with symptoms of elevated intracranial pressure, e.g. headaches, nausea and vomiting and may be accompanied by altered mental status and other neurological deficits depending on the site of infection.
- Extra-neural cysticercosis, which involves:
  - Muscle tissue
  - Subcutaneous tissue
  - Cardiac tissue (rare)

  A reasonable clinical approach to diagnose cysticercosis is to begin with CT imaging of the brain, and serology testing. If CT findings are inconclusive, a subsequent MRI may be appropriate.

TREATMENT OF CYSTICERCOSIS

Treatment of cysticercosis depends on the site of involvement and the symptoms experienced.

ANTIPARASITIC AND CORTICOSTEROID THERAPY

Antiparasitic and corticosteroid therapy is indicated in those patients with a single enhancing cyst, those with multiple cysts, for patients with subarachnoid cysts, for patients with symptomatic subcutaneous or intramuscular lesions and for patients with involvement of extraocular muscles or an optic nerve. Antiparasitic therapy is not recommended for patients with calcified cysts or for patients with diffuse cerebral oedema.

CYSTICERCOSIS: ANTIHELMINTHIC AND CORTICOSTEROID THERAPY

Treat with albendazole (Zentel®) 15 mg/kg per day PO (usually 800 mg/day in 2 divided doses) for 7 days in those with a single enhancing lesion, and for 10–14 days in those with multiple cystic lesions. Treat for at least 28 days for those with subarachnoid disease

OR

Praziquantel (Biltricide®) 50–100 mg/kg/day PO given 8 hourly is an effective alternative antiparasitic agent

Corticosteroids should be administered to patients with parenchymal neurocysticercosis receiving treatment with antiparasitic therapy to reduce the inflammation associated with the dying organisms. Give prednisone 1 mg/kg/day or dexamethasone 0.1 mg/kg/day for 5–10 days followed by a rapid taper.

ANTI-EPILEPTIC THERAPY

Anti-epileptic drug therapy should be administered to patients who present with seizures, or prophylactically to those with multiple lesions, particularly if the lesions are degenerating and are surrounded by inflammation. Anti-epileptic drug therapy is not indicated when calcified, inactive lesions are detected in asymptomatic patients.

OTHER TREATMENT OPTIONS

Those with hydrocephalus require CSF diversion. For patients with intraventricular cysts, intraocular disease or solitary symptomatic subcutaneous or intramuscular lesions, surgical excision is recommended.

ECHINOCOCCOSIS (HYDATID CYST DISEASE)

Humans serve as inadvertent intermediate hosts of *Echinococcus* species, which are carried as tapeworms by dogs and other canids. In South Africa, *E. granulosus* is prevalent, and sheep in which the larval cysts are found, are the intermediate hosts. In humans, echinococcosis presents as cystic disease and the presentation depends upon the site of the cysts and their size. Hydatid cysts may be found in almost any site of the body. The liver is affected in approximately two-thirds of patients, the lungs in approximately 25%, and other organs including the brain, muscle, kidneys, bone, heart, and pancreas in a small proportion of patients. Single organ involvement is commonly seen. The diagnosis is often made using both imaging and serological tests.

TREATMENT OF ECHINOCOCCOSIS

In most cases of hydatid liver disease, treatment consists of both antihelminthic therapy and percutaneous or surgical therapy. In previous years treatment of symptomatic cysts was by open surgical resection to remove the complete intact cyst. Because there is risk of secondary seeding and/or anaphylaxis if the cyst ruptures, the recommended approach is to visualise the cyst, remove a fraction of the fluid, and instill a protoscolicidal agent such as hypertonic (20%) saline, to kill the germinal layer and daughter cysts prior to resection. Thirty minutes after instillation, the cyst should be removed intact. However, other treatment modalities are becoming more popular.

These include:

- Laparoscopic surgery – usually for anteriorly located hepatic cysts.
- PAIR (Percutaneous Aspiration, Introduction of a protoscolicidal agent and Reaspiration) – performed under ultrasound or CT guidance. This is used particularly for uncomplicated cysts in the liver, abdominal cavity, lung and bone.

- Chemotherapy alone can be used in:
  - Patients with inoperable disease
  - Patient who are unfit for surgery
  - Peritoneal cysts
  - Multiple small liver cysts or cysts deep in the liver parenchyma
  - Recurrent cysts following surgery

Open surgery is still the preferred option for large liver cysts (> 10 cm in diameter, especially if associated with multiple daughter cysts), superficially located cysts which have a risk of rupture and cysts in the lungs and brain.

Antihelminthic therapy should begin at least seven days before surgery or percutaneous aspiration and be continued for a variable duration afterwards. Albendazole is the preferred agent and is given continuously at a dose of 10–15 mg/kg/day orally in two divided doses (usually 400 mg 12 hourly for adults). Albendazole is continued post-surgery or percutaneous aspiration for usually three months, however longer courses may be required. Albendazole is poorly absorbed and should be given with a fatty meal.

If given without surgical intervention, albendazole should be given continuously, without interruption for a period of up to 24 months.

Mebendazole is an alternative agent which is less well absorbed and not as effective as albendazole. The dose of mebendazole is 40–50 mg/kg PO daily.

TREMATODES (FLUKES)

SCHISTOSOMIASIS

Schistosomiasis (bilharzia) is the most common human trematode infection in Southern Africa and is caused by *S. haematobium* and *S. mansoni*.

Infection occurs after exposure of the skin to water contaminated with cercarial larvae of the trematode. Each schistosome species has a specific snail host as an intermediary, thus the distribution of the snail determines the distribution of the worm.

Schistosoma eggs are seeded into fresh water from contaminated faeces or urine. The eggs hatch and release miracidia and these penetrate snail intermediate hosts. They develop into cercariae which are released from the snail into the water. The cercariae penetrate human skin and migrate to the liver where they mature into adults. Adult worms will migrate to the venous plexus of the bladder (*S. haematobium*) or the mesenteric venules of the colon (*S. mansoni*). Eggs are deposited by the female worm into either the perivesical or mesenteric venule systems.

Infected individuals can have an acute schistosomiasis syndrome (Katayama fever) caused by a systemic hypersensitivity reaction to schistosome antigens. ‘Swimmers itch’ is a localised dermatitis at site of cercariae entry, typically on the lower legs or feet.

Chronic infection has an insidious onset of symptoms. Severity is related to the number of eggs that get trapped in tissues and their anatomical distribution. Hepatosplenic schistosomiasis results in periportal liver fibrosis with resulting portal hypertension and splenomegaly. Genitourinary schistosomiasis results in bladder wall fibrosis with hydronephrosis and renal failure.

A laboratory diagnosis can be made by detection of schistosoma eggs on microscopy of urine, stools or tissue specimens. The sensitivity of urine microscopy is highest for specimens collected between 10 am and 2 pm. Antigens can also be detected in urine or blood by means of serological tests. Molecular detection of *Schistosoma* DNA by means of PCR on urine, stool, tissue or CSF is a novel method with excellent sensitivity and is available at Ampath laboratories.

Eosinophilia is observed in 30–60% of patients. Hematuria and/or leukocyturia are common in the setting of *S. haematobium* infection.

TREATMENT OF SCHISTOSOMIASIS

Patients with schistosomiasis should be treated with the antihelminthic drug, praziquantel. Since praziquantel is not effective against the larval stages of schistosomes, treatment is most effective from four to six weeks after exposure. Follow-up after treatment includes monitoring of clinical manifestations, eosinophil count (in patients with eosinophilia), and microscopy for eggs in stool or urine.

SCHISTOSOMIASIS: ANTIHELMINTHIC THERAPY

Praziquantel 40 mg/kg PO as a single dose or in 2 divided doses is the standard treatment regimen and is sufficient for *S. haematobium* infection.

It has been suggested that higher doses (30 mg/kg daily for 2 days) may be needed, particularly in heavy *S. mansoni* infections.

Praziquantel can be given to pregnant or lactating women.

NOTE

- Treatment of schistosomiasis involving the central nervous system should include prednisone at a dose of one to two mg/kg orally since the inflammatory response provoked by praziquantel can lead to neurological worsening. Start the prednisone a few days before giving praziquantel and continue prednisone in patients with neuroschistosomiasis for at least two weeks.
- Supportive therapy should be offered to patients with Katayama fever – use prednisone in doses of 40 mg daily for five days.