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NSC 106

Medical Mircobiology and Parasitology

Module 7

NSC 106 (Medical Microbiology and Parasitology) Module 7

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Module 7: Introduction

Nematode infections in humans include ascariasis, trichuriasis, hookworm, enterobiasis, strongyloidiasis, filariasis, and trichinosis, among others. The phylum Nematoda, also known as the roundworms, is the second largest phylum in the animal kingdom, encompassing up to 500,000 species. The module will describe the general features and life cycles of nematodes, soil transmitted helminthes, blood and tissue nematodes and air-borne nematodes.

At the end of this module, you should be able to:

- discuss the various types of nematodes in relation to epidemiology, pathology and control.

Unit 1 General Features and Life Cycles of Nematodes

1.0 Introduction

Members of nematoda are elongated, with bilaterally symmetric bodies that contain an intestinal system and a large body cavity. Many roundworm species are free living in nature. Recent data have demonstrated that approximately 60 species of roundworms parasitise humans. Intestinal roundworm infections constitute the largest group of helminthic diseases in humans. According to a 2005 report by the World Health Organisation (WHO), approximately 0.807-1.221 billion humans have ascariasis, 604-795 million have trichuriasis, and 576-740 million have hookworm infections worldwide.

2.0 Objectives

At the end of this unit, you should be able to:

- list the various examples of nematodes with thier common names
- describe the general morphological features of nematodes
- explain the life cycles of the major groups of nematodes.

3.0 Main Content

3.1 General Features

Nematodes are cylindrical rather than flattened; hence the common name roundworm. The body wall is composed of an outer cuticle that has a noncellular, chemically complex structure, a thin hypodermis, and musculature. The cuticle in some species has longitudinal ridges called alae. The bursa, a flap-like extension of the cuticle on the posterior end of some species of male nematodes, is used to grasp the female during copulation.

The cellular hypodermis bulges into the body cavity or pseudocoelom to form four longitudinal cords; a dorsal, a ventral, and two lateral cords which may be seen on the surface as lateral lines. Nuclei of the hypodermis are located in the region of the cords. The somatic musculature lying beneath the hypodermis is a single layer of smooth muscle cells. When viewed in cross-section, this layer can be seen to be separated into four zones by the hypodermal cords. The musculature is innervated by extensions of muscle cells to nerve trunks running anteriorly and posteriorly from ganglion cells that ring the midportion of the esophagus.

The space between the muscle layer and viscera is the pseudocoelom, which lacks a mesothelium lining. This cavity contains fluid and two to six fixed cells (celomocytes) which are usually associated with the longitudinal cords. The function of these cells is unknown.

The alimentary canal of roundworms is complete, with both mouth and anus. The mouth is surrounded by lips bearing sensory papillae (bristles). The oesophagus, a conspicuous feature of nematodes, is a muscular structure that pumps food into the intestine; it differs in shape in different species.

The intestine is a tubular structure composed of a single layer of columnar cells possessing prominent microvilli on their luminal surface.

The excretory system of some nematodes consists of an excretory gland and a pore located ventrally in the mid-esophageal region. In other nematodes this structure is drawn into extensions that give rise to the more complex tubular excretory system, which is usually H-shaped, with two anterior limbs and two posterior limbs located in the lateral cords. The gland cells and tubes are thought to serve as absorptive bodies, collecting wastes from the pseudocoelom, and to function in osmoregulation.

Nematodes are usually bisexual. Males are usually smaller than females, have a curved posterior end, and possess (in some species) copulatory structures, such as spicules (usually two), a bursa, or both. The males have one or (in a few cases) two testes, which lie at the free end of a convoluted or recurved tube leading into a seminal vesicle and eventually into the cloaca.

The female system is tubular also, and usually is made up of reflexed ovaries. Each ovary is continuous, with an oviduct and tubular uterus. The uteri join to form the vagina, which in turn opens to the exterior through the vulva.

Copulation between a female and a male nematode is necessary for fertilisation except in the genus Strongyloides, in which parthenogenetic development occurs (i.e., the development of an unfertilised egg into a new individual). Some evidence indicates that sex attractants (pheromones) play a role in heterosexual mating. During copulation, sperm is

transferred into the vulva of the female. The sperm enters the ovum and a fertilisation membrane is secreted by the zygote. This membrane gradually thickens to form the chitinous shell.

A second membrane, below the shell, makes the egg impervious to essentially all substances except carbon dioxide and oxygen. In some species, a third proteinaceous membrane is secreted as the egg passes down the uterus by the uterine wall and is deposited outside the shell. Most nematodes that are parasitic in humans lay eggs that, when voided, contain either an uncleaved zygote, a group of blastomeres, or a completely formed larva. Some nematodes, such as the filariae and Trichinella spiralis, produce larvae that are deposited in host.

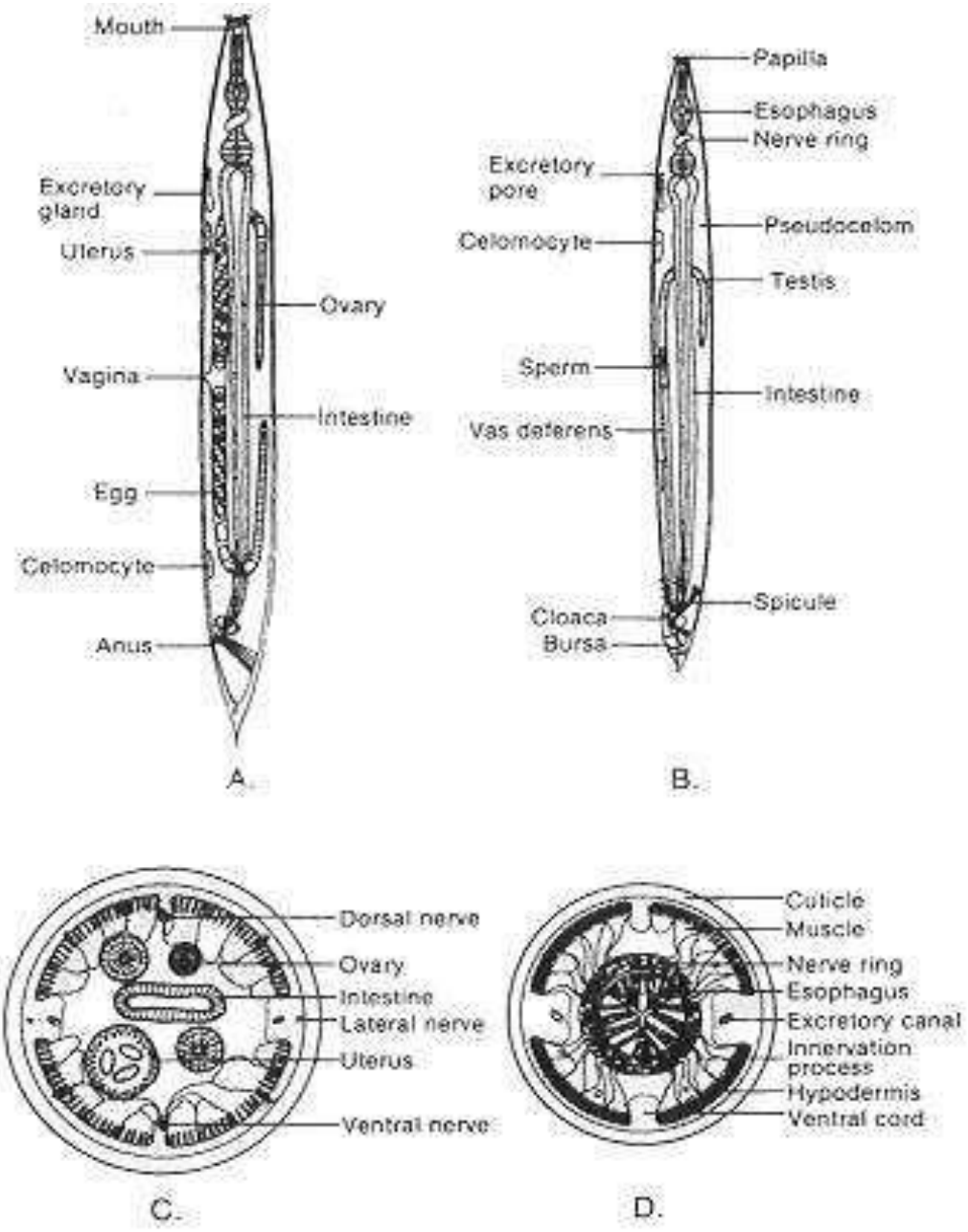

Fig. 1: Structure of Nematodes. (A) Female. (B) Male. Transverse Sections through

the Mid Region of the Female Worm (C) and through the Esophageal Region (D)

3.2 The Basic Life Cycle of the Major Groups of Nematodes

The life cycles of the parasitic species vary considerably, as would be expected from such a large and diverse group. There are however a number of common features. Firstly, the parasite undergoes a series of moults through larval stages (designated L1 to the adult L5 form). Secondly, in most (but not all) nematodes it is the L3 larvae that is the infective form, important exceptions to this being the Ascarids, such as Ascaris lumbricoides and the pinworms, where it is either the L1 larvae, or eggs containing L1 or L2 larvae that are infective.

Thirdly the L3 form onwards in all species undergoes a migration within the body of the definitive host as it matures into the adult parasite, usually via the bloodstream or lymphatic system to the heart, lungs, trachea, and then to the intestine. Finally, in most cases the parasite leaves the definitive host as thin walled eggs in the faeces, important exceptions being the viviparous filarial worms (where L1 larvae infect intermediate hosts, usually in the blood meals of biting arthropods), Strongyloides stercoralis, (where the L1 larvae are found in the faeces), and the viviparous Trichinela Spiralis, where the larvae do not leave the body as such, but develop to the L3 stage which then encysts in the muscles, infection being by ingestion of undercooked contaminated meat.

Infection of the definitive host may be by a variety of routes, such as the oral route, where eggs are accidentally ingested, also many filarial worms are infective via the bite of flies, as previously decribed, and the L3 larvae of many nematodes such as the hookworms and other related nematodes are directly invasive. In terms of complexity, the simplest life cycles are those of the pinworms, where adults living in the colon mate and lay eggs which pass out in the faeces, infection being either by the oral route with eggs, or perianally, where eggs hatch around the anus and L1 larvae migrate back through the anus.

The most diverse is probably that of S. stercoralis, where there are a number of alternative lifecycles which it may undergo, either as a completely free living soil nematode, or as the standard infective L3 larvae with tissue migration to the intestine, or even occasionally full completion of the life cycle within the intestine, and finally in immunocompromised hosts a life-threatening disseminated infection can occur, with parasites found throughout the body.

4.0 Conclusion

In this unit, you learnt that nematodes are roundworms with pseudocoelom (lacking a mesothelium lining). The alimentary canal is complete having mouth and anus. The intestine is a tubular structure composed of a single layer of columnar cells possessing prominent microvilli on their luminal surface. The excretory system of some nematodes consists of an excretory gland and a pore with complex tubular excretory system, which is usually H-shaped. Nematodes are usually bisexual. Males are usually smaller than females. Copulation between a female and a male nematode is necessary for fertilisation except in the genus Strongyloides, in which parthenogenetic development occurs (i.e., the development of a unfertilised egg into a new individual). Parasite undergoes a series of moults through larval stages with L3 larva mostly being the infective stage. Infection of the definitive host may be

by accidental ingestion of eggs, bite of flies and skin penetration by the infective L3 larval form as in the case of hookworms.

5.0 Summary

Nematodes are roundworms with pseudocoelom (lacking a mesothelium lining). The alimentary canal is complete having mouth and anus. The intestine is a tubular structure composed of a single layer of columnar cells possessing prominent microvilli on their luminal surface. The excretory system of some nematodes consists of an excretory gland and a pore with complex tubular excretory system, which is usually H-shaped. Nematodes are usually bisexual. Males are usually smaller than females. Copulation between a female and a male nematode is necessary for fertilisation except in the genus Strongyloides, in which parthenogenetic development occurs (i.e., the development of a unfertilised egg into a new individual). Parasite undergoes a series of moults through larval stages with L3 larva mostly being the infective stage. Infection of the definitive host may be by accidental ingestion of eggs, bite of flies and skin penetration by the infective L3 larval form as in the case of hookworms.

6.0 Self-Assessment Exercise

Activity: Concduct a physical examination of a mature roundworm and report your findings in the log book.

Answer the following questions:

1. Mention examples of nematodes with their common names (LO1.)
2. With the aid of well labeled diagrams describe the general features of roundworms (LO2).
3. Highlight the basic life cycles of nematode (LO3).

7.0 References/Further Reading

Ukoli, F.M.A. (1990). *Introduction of Parasitology in Tropical Africa.* Chichester.

John Wiley and Sons Ltd.

Unit 2 Soil Transmitted Helminths

1.0 Introduction

Soil-transmitted helminth (STH) infection is highly endemic in tropical and subtropical areas of sub-Saharan Africa, Asia and Latin America, where up to 2 billion people have active infections. STH infection has remained largely neglected by the global health community because the people most affected areamong the most impoverished and because the infection causes chronic ill health with insidious clinical presentations, rather than severe acute illness or high mortality. However, it is now recognised that STH infection causes significant morbidity worldwide with 39 million disability adjusted life years (DALYs) lost each year - more than those lost to malaria (36 million yearly) and approaching those lost to tuberculosis (47 million yearly). Hookworm infection alone causes the loss of 22 million DALYs.

2.0 Objectives

At the end of this unit, you should be able to:

- give examples of soil transmitted helminthes (STHS).
- describe their life cycles with emphasis on the route of infection.
- describe the diagnostic features of the parasites.

3.0 Main Content

3.1 Ascaris Lumbricoides (Large Roundworm of Man)

Infection with this roundworm is extremely common, with estimates of the annual incidence of infection being greater than 1500 million cases, or around one quarter of the world's population. In addition to the species in man, Ascaris lumbricoides, a morphologically indistinguishable species Ascaris suum is found in the pig. Other related genera include Parascaris in equines, and Toxascaris in a variety of domesticated animals.

Morphology

The adult Ascaris lumbricoides are large white, or pinkish-white, cylindrical roundworms, slightly narrower at the head. The more slender males measure between 10 to 30cm long and have a curved tail with two spicules, but no copulatory bursa. The females are very similar, being slightly larger; it's between 20 to 35cm long, a vulva size approximately. A third of the length of the body down from the head, and have a blunt tail. They are both characterised by having a smooth,finely striated, cuticle, and a mouth, which is characteristic of all of the Ascarids (e.g. Toxocara), having three lips each equipped with small papillae. Internally they follow the generalised body plan of all nematodes, and have a cylindrical oesophagus opening into a flattened ribbon like intestine. The eggs consist of a thick transparent inner shell which is covered in a thick, warty, albuminous coat.

Fig. 1: Eggs unfertilised (left) fertilised (right) of A. lumbricoides

Life Cycle

These parasites have a direct life cycle, with no intermediate hosts. The adult parasite lives in the lumen of the small intestine of man, usually only feeding on the semi-digested contents of the gut, although there is some evidence that they can bite the intestinal mucous membrane and feed on blood and tissue fluids. The female parasite is highly prolific, laying an estimated 2 million eggs daily. In the intestine, these only contain a unembryonated mass of cells, differentiation occurring outside the host. This requires a temperature less than 30°C, moisture and oxygen, before the development of the young L1 larvae after approximately 14 days. Eggs containing the L2 larvae take another week to develop, before they are infective to man, and may remain viable in the soil for many years if conditions are optimal.

Infection occurs on ingestion of raw food, such as fruit or vegetables that is contaminated with these infective eggs. The eggs then hatch in the small intestine, to release the L2 rhabditiform larvae (measuring approximately 250 by 15µm in size. These do not simply grow into the adult forms in the intestine, but must then undergo a migration through the body of their host. These L2 larvae penetrate the intestinal wall, entering the portal blood stream, and then migrate to the liver, then heart, then after between 1 to 7 days, the lungs. Here they moult twice on the way to form the L4 larvae, (measuring approximately 1.5mm long), then burrow out of the blood vessels, entering the bronchioles. From here they migrate up through the air passages of the lungs, to the trachea. They then enter the throat and are swallowed, finally ending up in the small intestine where they mature and mate, to complete their life cycle.

Pathology of Infection

The majority of infections (~85%) appear to be asymptomatic, in that there is no gross pathology seen. However the presence of these parasites appears to be associated with the same general failure to thrive in their hosts seen with many of these intestinal nematodes. In terms of more easily identified pathology, this may be divided into three areas:

Pathology Associated with the Ingestion and Migration of Larvae. Severe symptoms of Ascaris infection may be associated with the migrating larvae, particularly in the lungs. If large numbers of these larvae are migrating through the lungs simultaneously this may give

rise to a severe haemorrhagic pneumonia. More commonly, as is the case with most infections, the haemorrhages are smaller in scale, but still may lead to breathing difficulties, pneumonia and/or fever. A complication here is that many of the parasites proteins are highly allergenic. Due to this the presence of the migrating larvae in the lungs is often associated with allergic hypersensitivity reactions such as asthmatic attacks, pulmonary infiltration and urticaria and oedema of the lips.

Pathology Associated with Adult Parasites in the Intestine

The most common symptoms of infection are due to the adult parasite, and consist of rather generalised digestive disorders, such as a vague abdominal discomfort, nausea, colic (etc.). These symptoms are dependent to some extent on the parasite's burden of the host, which in severe cases may consist of many hundreds or even thousands of parasites, although these are extreme cases. In the case of these heavy infections the presence of many of these large parasites may contribute to malnutrition in the host, especially if the hosts (often children) are undernourished. A more serious, and potentially fatal, condition may arise in these more heavy infections, where the mass of worms may block the intestine and need to be surgically removed. This may also occur sometimes on treatment for other intestinal nematodes such as hookworms, where the curative drug dose for these parasites irritates the ascarids.

Pathology due to "Wandering" Adults outside of the Intestine

Adult parasites often leave the small intestine to enter other organs, (sometimes in response to anti-helminthic drugs used to treat other intestinal nematode infections), where they may cause various types of pathology, sometimes with severe consequences. For example adult Ascaris worms may migrate to the bile duct, which may then become blocked causing jaundice and a general interference in fat metabolism. Adult parasites may also migrate to the appendix, or through the intestinal wall, both conditions which may cause a fatal peritonitis as they may well carry intestinal bacteria to these sites. They may, alarmingly, sometimes migrate forward through the intestinal tract, to be either vomited up or emerging through the nose. More seriously, if they enter the trachea they may cause suffocation.

Diagnosis

Definitive diagnosis is by demonstration of the characteristic eggs in faecal samples or by identifying adult worms passed out spontaneously by the host.

Epidemiology and Control

Infection occurs through ingestion of parasites' eggs in food. The eggs are highly resistant to adverse environmental conditions. This with other factors highlighted below are often associated with transmission of infection;

- Lack or inadequate waste disposal facilities
- Improper washing of hands before eating
- Improper washing of fruits and vegetables before consumptions
- Unkept rooms and dwelling places that harbour mechanical carriers of parasites, etc.

Provision of good waste disposal system and good personal hygiene will help to control infections.

3.2 The Human Hookworms

The hookworms belong to the Order Strongylida, a very large order, and of great interest as it contains many important pathogens of man and domesticated animals. This order is further subdivided into three Superfamilies, the Strongyloidea (the hookworms in man), and two related groups, the Superfamily 'Trichostrongyloidea', intestinal nematodes which are of veterinary importance in many domesticated animals

In man there are two species capable of causing intestinal infections, Ancylostoma duodenale native to parts of Southern Europe, North Africa and Northern Asia parts of Western South America, and Necator americanus in Central and Southern Africa, Southern Asia, Australia and the Pacific Islands. These are very important human pathogens. It has been estimated that there are 1200 million cases of hookworm infection in man annually, of which about 100 million of which are symptomatic infections with accompanying anaemia. In addition, the larvae of several species of hookworms infecting domesticated animals may penetrate human skin, causing pathology even though they do not develop to the adult parasites in man.

Morphology

The adult parasites are small cylindrical worms, 0.5-1.5mm long (Ancylostoma duodenale being slightly larger than Necator americanus). The posterior end of the male worm is equipped with a characteristic copulatory bursa, used to hold the female nematode in place during mating. The females themselves have a vulva situated near the center of the body, slightly anterior in Necator and slightly posterior in Ancylostoma. The anterior end of the parasites is formed into a Buccal capsule, absent in members of the other Strongylida superfamilies, by which the different genera and species within the group may be differentiated. For example members of the genus Necator have capsules equipped with cutting plates on the ventral margins, and within the capsule itself small dorsal teeth. In contrast, members of the genus Ancylostoma have pairs of teeth on the ventral margin of the capsule. The number of teeth will vary between different species of Ancylostoma, but is usually between one and four pairs.

Fig. 2: Scanning Electron Micrograph Of The Mouth Capsule of Ancylostoma Duodenale (Left), note the

Presence of four "Teeth," Two On each side and Necator Americanus (right)

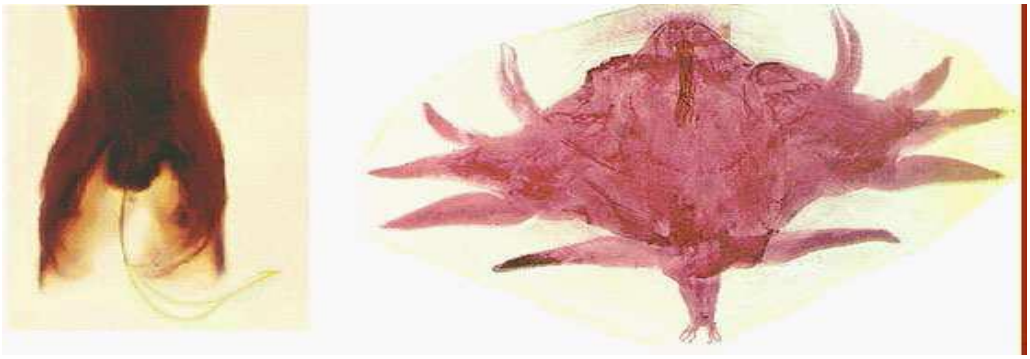

Fig.3: Left Picture: Copulatory Bursa and Spines of N. Americanus (A Side View); Right Picture: Copulatory Bursa of a Duodenale (A Top View). The eggs are bluntly rounded, thin shelled, and are almost indistinguishable between the different species, measuring approximately 60 by 40 µm, and the eggs of Ancylostoma being slightly larger than those of Necator

The Morphological Differences between Two species of Hookworms

Features	A. duodenale	N. americanus
Size	Larger	Smaller
Shape	Single curve, looks like C	Double curves, looks like S
Mouth	2 pairs of ventral teeth	1pair of ventral cutting plates
Copulatory bursa	Circle in shape	Oval in shape
Copulatory spicule	1pair with separate endings	1 p air of which unite to form a terminal hooklet
Caudal spine	Present	No
Vulva position	Post-equatorial	Pre-equatorial

Life Cycle

The life cycles of all the hookworms are very similar. The eggs are passed in the faeces, once exposed to air they mature rapidly if conditions are right, with both moisture and warmth essential for development. When matured, they hatch to liberate a rhabditiform i.e. having an oesophagus where a thick anterior region is connected via a neckline region with posterior bulb L1 larvae after a few days.

These larval nematodes feed on bacteria and organic material in the soil, where they live and grow for about two days before undergoing the first moult. After about five days more growth they moult again, to produce much more slender L3 larvae. The L3 larvae have a much shorter oesophagus, are a non-feeding form, and are the infective form of the parasite. Infection takes place by penetration of the skin, for example when walking with bare feet over contaminated damp soil, followed by entry into the circulatory system. Here they are carried to the heart, and then lungs.

Once in the lungs, they are too large to pass through the capillary bed there. Instead they become trapped, and the burrow through the capillary epithelium, entering the air spaces. They then migrate up through bronchi and trachae, and are then swallowed. Once swallowed, they pass into the intestine and bury themselves between the intestinal villi. Here they moult to form the L4 larvae, equipped with a buccal capsule allowing adherence to the gut wall. After about thirteen days post-infection they moult for the final time, producing immature adult worms. These mature over three to four weeks (i.e. five to six weeks after infection), then mate and commence egg lying to complete the life cycle. These parasites show a very high fecundity, female Necator americanus producing up to 10,000 eggs daily, while female Ancylostoma duodenale produces up to 20,000 eggs daily.

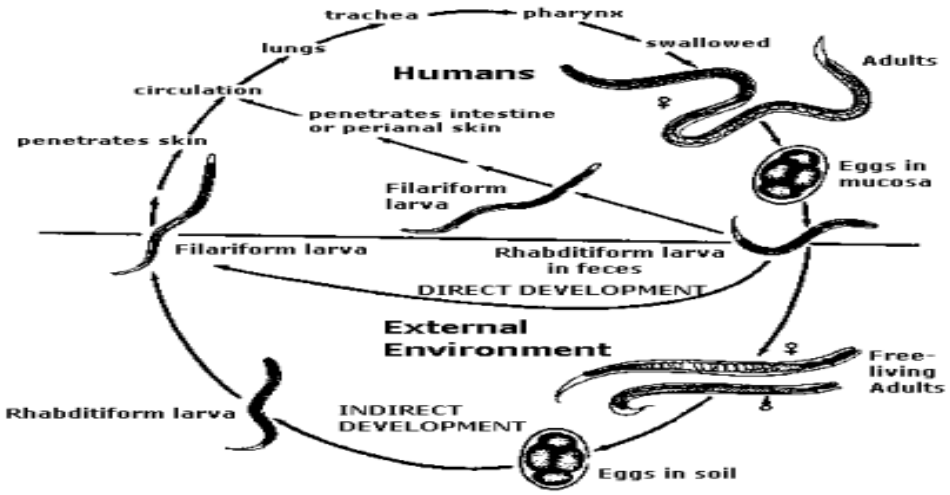

Fig. 4: Life Cycle of Human Hookworms (source: rrh.org.au)

Pathology of Infection

The Pathology associated with hookworm infections may be divided roughly into two areas. Firstly the pathology associated with the presence of the adult parasite in the intestine, and secondly the pathology associated with the penetration of, and migration of the larval worms within the skin. The adult hookworms attach themselves to the intestinal wall using their buccal capsules. Their preferred site of infestation is in the upper layer of the small intestine, but in very heavy infections (where many thousands of worms may be present) the parasites may spread down as far as the lower ileum. Once attached to the intestinal wall, the hookworm mouthparts penetrate blood vessels, and the parasites obtain nutrition by sucking blood.

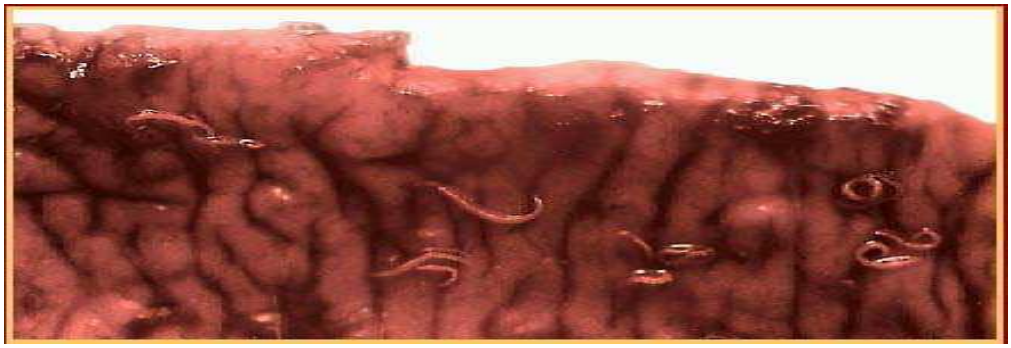

Fig. 5: Adults in Intestinal Mucosa

A single Necator americanus will take approximately 30 µl of blood daily, while the larger Ancylostoma duodenale will take up to 260 µl. The gross pathology of the disease is very dependent on the intensity of infection. Light infections appear asymptomatic, but in heavy infections, the continuous loss of blood leads to a chronic anaemia, with down to 2gm of haemoglobin per 100ml of blood in extreme cases. Experiments carried out in the 1930's showed that in dogs infected with 500 Ancylostoma caninum a similar species to the human parasite, nearly a pint of blood a day was lost. This leads to permenant loss of iron and many blood proteins as well as blood cells.

This in turn has consequences for further production of erythrocytes, which have been shown to contain less haemoglobin, as well as being reduced in size and smaller in numbers. This form of anaemia may be directly fatal, but more often, it induces more non-specific symptoms, the most noticeable being the severe retardation in growth and development, both physical and mental, in infected children, and a general weakness and lassitude, often wrongly interpreted as "laziness".

Diagnosis

Identify characteristic eggs in feacal samples. Note the eggs of N. americanus and A. duodenale are morphologically identical.

Fig. 6: Egg of Hookworm

Epidemiology and Control

The factors of epidemiological importance include;

- Poor sanitation through contamination of soil through direct defaecation on the ground.
- Skin exposure to infections e.g. by walking about bare-footed
- Favourable environmental conditions that enhance eggs and larval development.
- Loose, humus soil with reasonable drainage and aeration
- Even distribution of rainfall throughout the year.

Control is by improvement in the standard of sanitation, raising the nutritional status of the population especially in relation to iron content, and mass treatment with suitable worm expeller (vermifuge)

3.3 Trichuris Trichiura (Human Whipworm)

The first written record of Trichuris trichiura was made by Morgani, an Italian scientist, who identified the presence of the parasite in a case of worms residing in the colon in 1740. Exact Morphological description and figures were first recorded in 1761 by Roedere, a German physicist. Soon after morphology and visual representation of the worms, Trichuris trichiura was given taxonomy (during the 18th century). This is the third most common round worm of humans. It is distributed worldwide, with infections more frequent in areas with tropical weather and poor sanitation practices, and among children. It is estimated that 800 million people are infected worldwide. The southern United States is endemic for trichuriasis.

Morphology

Adult worms are usually 3–5 cm long, with females being larger than males as is typical of nematodes. The thin, clear majority of the body (the anterior, whip-like end) is the oesophagus, and it is the end that the worm threads into the mucosa of the colon. The widened, pinkish gray region of the body is the posterior, and it is the end that contains the parasite's intestines and reproductive organs.Trichuris trichiura has characteristic football-shaped eggs, which are about 50-54µm long and contain polar plugs (also known as refractile prominences) at each end.

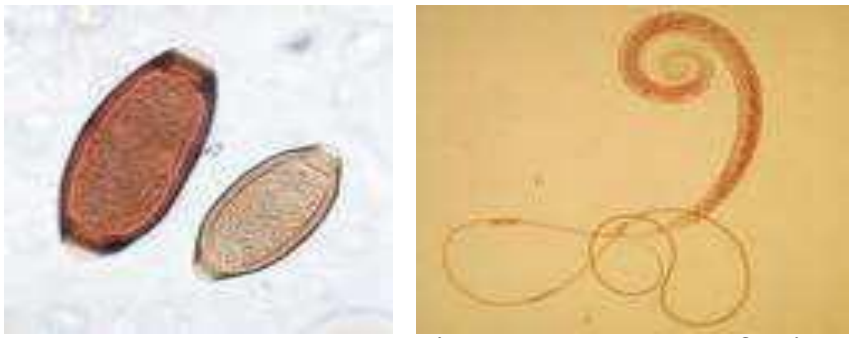

Fig.7: Egg and adult of Trichuris Trichiura

Life Cycle and Transmission

Humans can become infected with the parasite due to ingestion of infective eggs by mouth contact with hands or food contaminated with egg-carrying soil. However, there have also been rare reported cases of transmission of Trichuris trichiura by sexual contact. Some major outbreaks have been traced to contaminated vegetables (due to presumed soil contamination).

Unembryonated eggs (unsegmented) are passed in the faeces of a previous host to the soil. In the soil, these eggs develop into a 2-cell stage (segmented egg) and then into an advanced cleavage stage. Once at this stage, the eggs embryonate and then become infective, a process that occurs in about 15 to 30 days). Next, the infective eggs are ingested by way of soil-contaminated hands or food and hatch inside the small intestine, releasing larvae into the gastrointestinal tract. These larvae burrow into a villus and develop into adults (over 2–3 days). They then migrate into the cecum and ascending colon where they thread their anterior portion (whip-like end) into the tissue mucosa and reside permanently for their year-long life span. About 60 to 70 days after infection, female adults begin to release unembryonated eggs (oviposit) into the cecum at a rate of 3,000 to 20,000 eggs per day, linking the life cycle to the start.

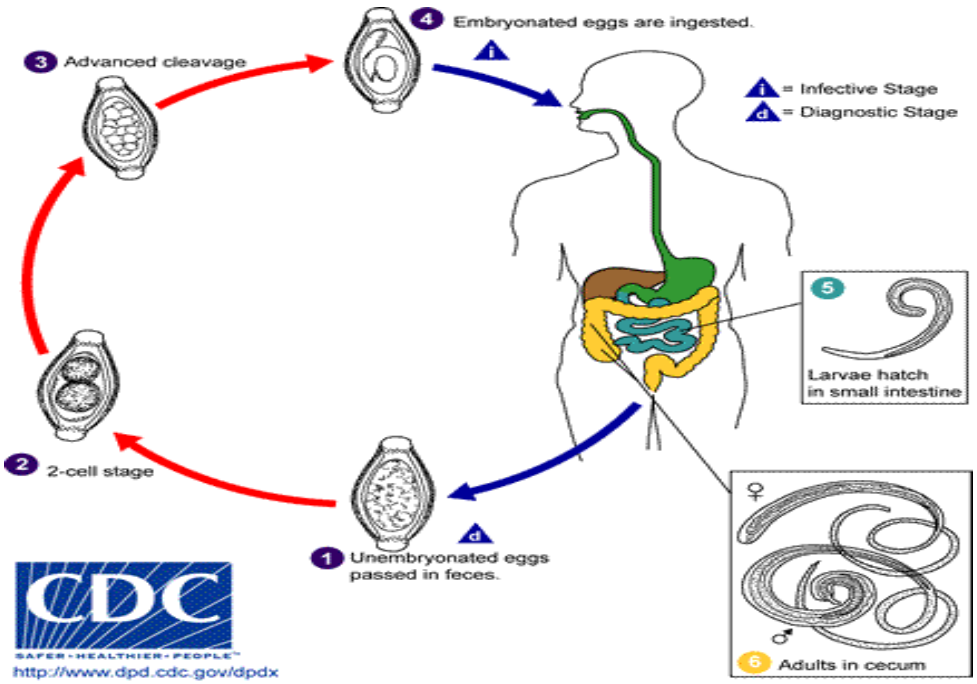

Fig. 8: Life Cycle of Trichuris Trichiura

Signs and Symptoms

Light infestations are frequently asymptomatic (have no symptoms). Heavier infestations, especially in small children, can present gastrointestinal problems including abdominal pain and distention, bloody or mucous-filled diarrhoea, and tenesmus (feeling of incomplete defecation, generally accompanied by involuntary straining).

While damage may be done to the GI tissue and appendicitis may be brought on (by damage and oedema of the adjacent lumen) if there are large numbers of worms or larvae present, it

has been suggested that the embedding of the worms into the ileo-cecal region may also make the host susceptible to bacterial infection. Severe infection may also present with rectal prolapse, although this is typically seen only in heavy infections of small children. High numbers of embedded worms in the rectum cause oedema, which causes the rectal prolapse. The prolapsed, inflamed and oedematous rectal tissue may even show visible worms.

Growth retardation, weight loss, nutritional deficiencies, and anaemia (due to long-standing blood loss) are also characteristic of infection, and these symptoms are more prevalent and severe in children.

Diagnosis

A stool ova and parasites examination reveals the presence of typical whipworm eggs. Typically, the Kato-Katz thick-smear technique is used for the identification of the Trichuris trichiura eggs in the stool sample. Although colonoscopy is not typically used for diagnosis, but there have been reported cases in which colonoscopy has revealed adult worms. Colonoscopy can directly diagnose trichuriasis by identification of the threadlike form of worms with an attenuated, whip-like end. Colonoscopy has been shown to be a useful diagnostic tool, especially in patients infected by only a few male worms and with no eggs presenting in the stool sample.

Epidemiology

Trichuris trichiura is the third most common nematode (roundworm) of humans. Infection of trichuris trichiura is most frequent in areas with tropical weather and poor sanitation practices. Trichuriasis occurs frequently in areas in which human feces are used as fertiliser or where defecation onto soil takes place. Trichuriasis infection prevalence is 50 to 80 percent in some regions of Asia (noted especially in China and Korea) and also occurs in rural areas of the southeastern United States. Infection is most prevalent among children, and in North America, infection occurs frequently in immigrants from tropical or sub-tropical regions. It is estimated that 600-800 million people are infected worldwide with 3.2 billion individuals at risk.

Control and Prevention

Improved facilities for faeces disposal have decreased the incidence of whipworm. Handwashing before food handling and avoiding ingestion of soil by thorough washing of food that may have been contaminated with egg-containing soil are other preventive measures. Mass Drug Administration (preventative chemotherapy) has had a positive effect on the disease burden of trichuriasis in East and West Africa, especially among children, who are at highest risk for infection. Improvement of Sewage and Sanitation systems, as well as improved facilities for faeces disposal have helped to limit defecation onto soil and contain potentially infectious faeces from bodily contact.

A study in a Brazil Urban Centre demonstrated a significant reduction in prevalence and incidence of geohelminth infection, including trichuriasis, following implementation of a city-wide sanitation programme. A 33% reduction in prevalence of trichuriasis and a 26% reduction in incidence of trichuriasis were found in the study performed on 890 children ages 7–14 years old within 24 different sentinel areas chosen to represent the varied environmental conditions throughout the city of Salvador, Bahia, Brazil. Control of Soil

Fertilisers has helped eliminate the potential for contact with human faecal matter in fertiliser in soil.

4.0 Conclusion

In this unit, we have discussed in details what soil transmitted helminthes are with various examples. Also discussed were their life cycles and their diagnostic features.

5.0 Summary

STH infection is caused by four major nematode species: Ancylostoma duodenale and Necator americanus (hookworms), Ascaris lumbricoides (roundworm) and Trichuris trichiura (whipworm). Infection is prevalent in areas with overpopulation and inadequate sanitation in tropical and sub-tropical countries, where the climate supports the survival of the parasite eggs or larvae in the warm and moist soil. After infective larvae enter the human body they develop into adult worms and parasitise the gastrointestinal tract, sometimes for years. Some species of worms can produce up to 200,000 eggs per day. Eggs are excreted in the faeces and remain viable in the soil for several weeks or years depending on the species. It is common for a single individual, especially a child, to be infected with all three types of worms.

Although STH infection rarely causes fatality, chronic infection with high worm burden can lead to serious health consequences. Infection is typically most intense and debilitating in school-age children, resulting in malnutrition, physical and intellectual growth retardation, and cognitive and educational deficits. A. lumbricoides may cause intestinal obstructions that require surgery, and T. trichuria may cause chronic colitis. Hookworm infection causes iron-deficiency anaemia because the worms feed on the intestinal wall causing tissue damage and blood loss. Hookworm infection is a leading cause of morbidity in children and pregnant women, and can have adverse results for the mother, the foetus and the neonate.

6.0 Self-Assessment Exercise

Activity: Conduct a physical examination of Ascaris lumbricoides, Ancylostoma duodenale, and Trichuris trichiura under the microscope and report your findings in the log book.

Answer the following questions:

1. State the morphological features of ascaris lumbricoides and trichuris trichiura (LO1)
2. What strategies you will implore for the control of soil transmitted nematodes? (LO2)
3. Describe the life cycles of hookworm with emphasis on the route of infection (LO2)

7.0 References/ Further Reading

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Unit 3 Blood and Tissue Nematodes

1.0 Introduction

Filariasis is caused by nematodes (roundworms) that inhabit the lymphatics and subcutaneous tissues. Eight main species infect humans. Three of these are responsible for most of the morbidity due to filariasis: Wuchereria bancrofti and Brugia malayi cause lymphatic filariasis, and Onchocerca volvulus causes onchocerciasis (river blindness). The other five species are Loa loa, Mansonella perstans, M. streptocerca, M. ozzardi, and Brugia timori. (The last species also cause lymphatic filariasis).

Among the agents of lymphatic filariasis, Wuchereria bancrofti is encountered in tropical areas worldwide; Brugia malayi is limited to Asia; and Brugia timori is restricted to some islands of Indonesia. The agent of river blindness, Onchocerca volvulus, occurs mainly in Africa, with additional foci in Latin America and the Middle East. Among the other species, Loa loa and Mansonella streptocerca are found in Africa; Mansonella perstans occurs in both Africa and South America; and Mansonella ozzardi occurs only in the American continent.

Another tissue invading parasite is Trichinella spiralis whose larval form is found in the muscular tissue of the host animal. Trichinella spiralis is in fact a complex of three closely related worm species. They are morphologically identical, but differ in their host specificity and their biochemical characteristics. T. spiralis spiralis occurs in moderate regions and infects mainly pigs. T. spiralis native occurs in the polar regions (polar bear, walrus). These parasites are resistant to freezing which is important for meat storage. T. spiralis nelsoni occurs in Africa and southern Europe with a reservoir in wild carnivores and wild pigs. T. britovi and T. pseudospiralis rarely cause infections. T. pseudospiralis can also infect some birds as well as mammals, unlike the other Trichinella species.

2.0 Objectives

At the end of this unit, you should be able to:

- explain examples of blood and tissue invading parasites
- describe their life cycles and clinical features associated with their infections.
- describe the methods of diagnosis of their infections.

3.0 Main Content

3.1 Filarial Worms

Wuchereria Bancrofti
Different species of the following genera of mosquitoes are vectors of W.bancrofti filariasis depending on geographical distribution. Among them are:

Culex (C. annulirostris, C. bitaeniorhynchus, C. quinquefasciatus, and C. pipiens); Anopheles (A. arabinensis, A. bancroftii, A. farauti, A. funestus, A. gambiae, A. koliensis, A. melas, A. merus, A. punctulatus and A. wellcomei); Aedes (A. aegypti, A. aquasalis, A. bellator, A. cooki, A. darlingi, A. kochi, A. polynesiensis, A. pseudoscutellaris, A. rotumae, A. scapularis, and A. vigilax); Mansonia (M. pseudotitillans, M. uniformis); Coquillettidia (C. juxtamansonia). During a blood meal, an infected mosquito introduces third-stage filarial larvae onto the skin of the human host, where they penetrate into the bite wound. They develop in adults that commonly reside in the lymphatics. The female worms measure 80 to 100 mm in length and 0.24 to 0.30 mm in diameter, while the males measure about 40 mm by .1 mm. Adults produce microfilariae measuring 244 to 296 µm by 7.5 to 10µm, which are sheathed and have nocturnal periodicity, except the South Pacific microfilariae which have the absence of marked periodicity. The microfilariae migrate into the lymph and blood channels moving actively through lymph and blood. A mosquito ingests the microfilariae during a blood meal. After ingestion, the microfilariae loose their sheaths and some of them work their way through the wall of the proventriculus and cardiac portion of the mosquito's midgut and reach the thoracic muscles. There the microfilariae develop into first-stage larvae and subsequently into third-stage infective larvae. The third-stage infective larvae migrate through the haemocoel to the mosquito's prosbocis and can infect another human when the mosquito takes a blood meal.

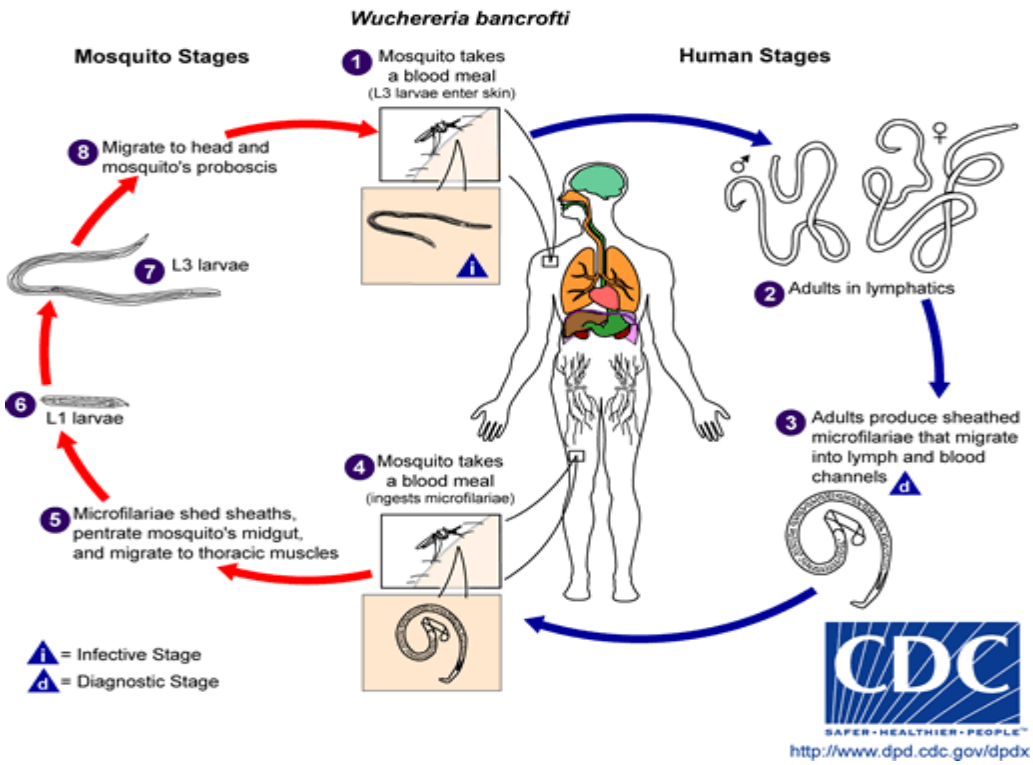

Fig. 1: Life Cycle of W. Bancrofti

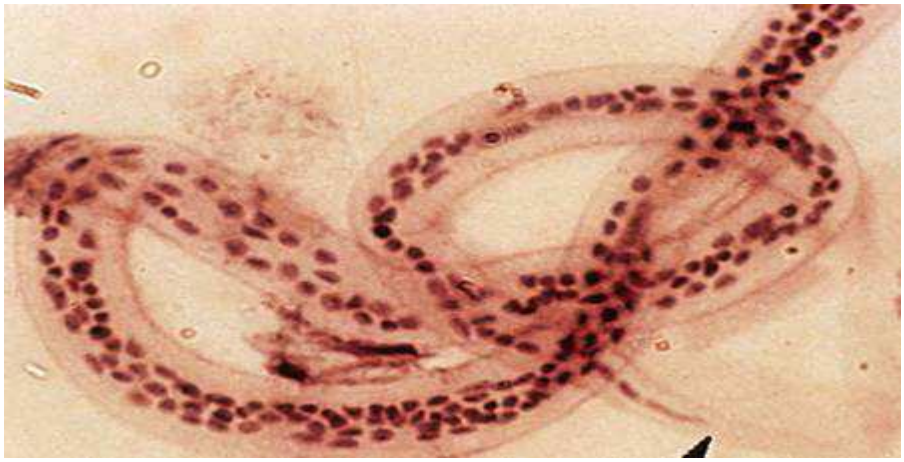

Fig. 2: Microfilaria of W. Bancrofti

Onchocerca Volvulus

During a blood meal, an infected blackfly (genus Simulium) introduces third-stage filarial larvae onto the skin of the human host, where they penetrate into the bite wound. In subcutaneous tissues the larvae develop into adult filariae, which commonly reside in nodules in subcutaneous connective tissues Adults can live in the nodules for approximately 15 years. Some nodules may contain numerous male and female worms. Females measure 33 to 50 cm in length and 270 to 400 µm in diameter, while males measure 19 to 42 mm by 130 to 210 µm. In the subcutaneous nodules, the female worms are capable of producing microfilariae for approximately 9 years.

The microfilariae, measuring 220 to 360 µm by 5 to 9 µm and unsheathed, have a life span that may reach 2 years. They are occasionally found in peripheral blood, urine, and sputum but are typically found in the skin and in the lymphatics of connective tissues. A blackfly ingests the microfilariae during a blood meal. After ingestion, the microfilariae migrate from the blackfly's midgut through the haemocoel to the thoracic muscles. There the microfilariae develop into first-stage larvae and subsequently into third-stage infective larvae. The third-stage infective larvae migrate to the blackfly's proboscis and can infect another human when the fly takes a blood meal.

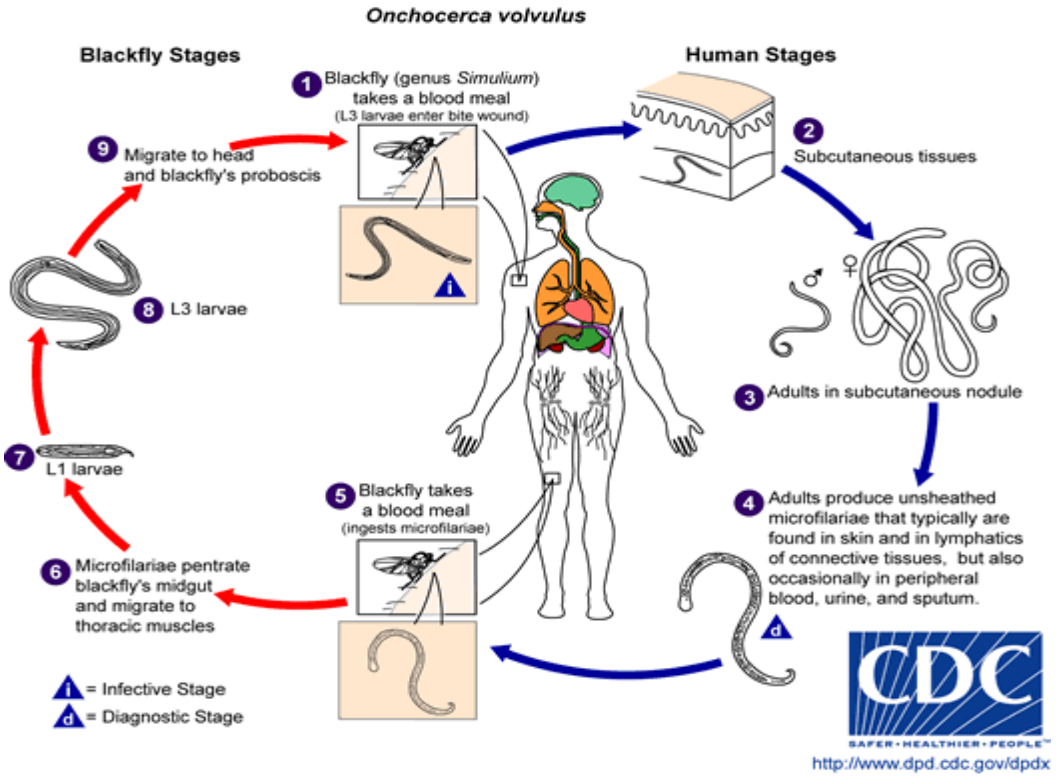

Fig. 3: Life Cycle of O. Volvulus

Loa Loa

The vectors for Loa loa filariasis are flies from two species of the genus Chrysops, C. silacea and C. dimidiata. During a blood meal, an infected fly (genus Chrysops, day-biting flies) introduces third-stage filarial larvae onto the skin of the human host, where they penetrate into the bite wound. The larvae develop into adults that commonly reside in subcutaneous tissue. The female worms measure 40 to 70 mm in length and 0.5 mm in diameter, while the males measure 30 to 34 mm in length and 0.35 to 0.43 mm in diameter. Adults produce microfilariae measuring 250 to 300 µm by 6 to 8 µm, which are sheathed and have diurnal periodicity. Microfilariae have been recovered from the spinal fluids, urine, and sputum.

During the day they are found in peripheral blood, but during the noncirculation phase, they are found in the lungs. The fly ingests microfilariae during a blood meal. After ingestion, the microfilariae lose their sheaths and migrate from the fly's midgut through the haemocoel to the thoracic muscles of the arthropod. There the microfilariae develop into first-stage larvae and subsequently into third-stage infective larvae. The third-stage infective larvae migrate to the fly's proboscis and can infect another human when the fly takes a blood meal.

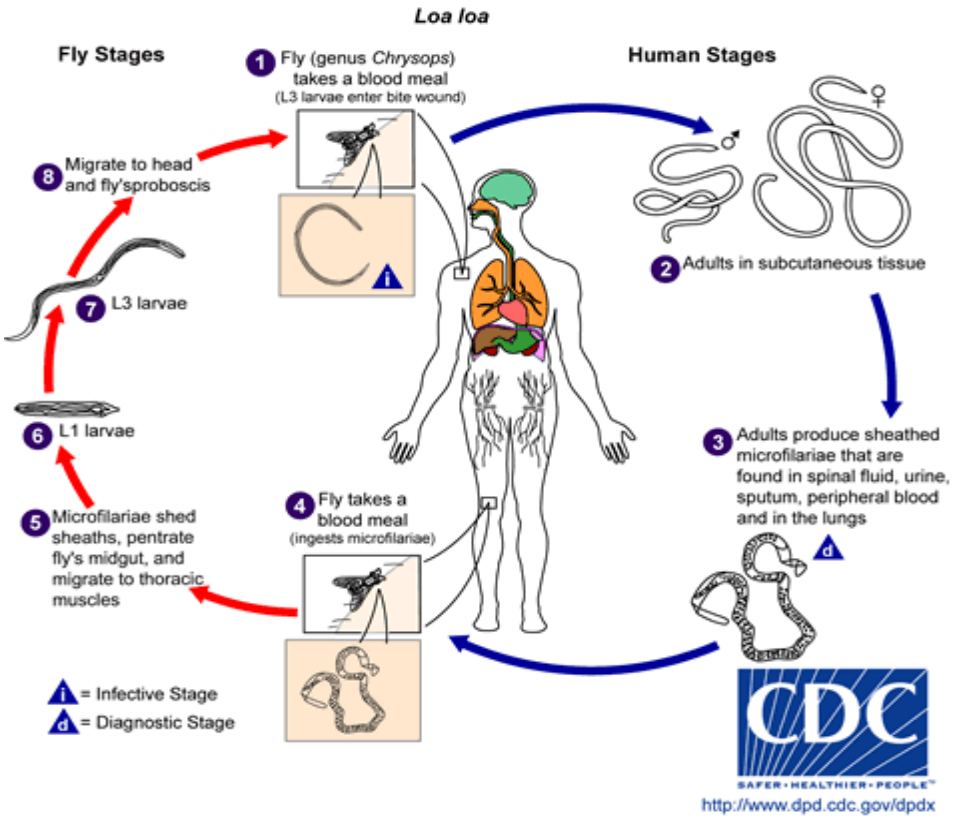

Fig. 4: Life Cycle Loa Loa

Brugia Malayi

The typical vector for Brugia malayi filariasis is mosquito species from the genera Mansonia and Aedes. During a blood meal, an infected mosquito introduces third-stage filarial larvae onto the skin of the human host, where they penetrate into the bite wound. They develop into adults that commonly reside in the lymphatics. The adult worms resemble those of Wuchereria bancrofti but are smaller. Female worms measure 43 to 55 mm in length by 130 to 170 µm in width, and males measure 13 to 23 mm in length by 70 to 80 µm in width. Adults produce microfilariae, measuring 177 to 230 µm in length and 5 to 7 µm in width, which are sheathed and have nocturnal periodicity. The microfilariae migrate into the lymph and enter the blood stream reaching the peripheral blood. A mosquito ingests the microfilariae during a blood meal.

After ingestion, the microfilariae lose their sheaths and work their way through the wall of the proventriculus and cardiac portion of the midgut to reach the thoracic muscles. There the microfilariae develop into first-stage larvae and subsequently into third-stage larvae. The third-stage larvae migrate through the haemocoel to the mosquito's prosbocis and can infect another human when the mosquito takes a blood meal.

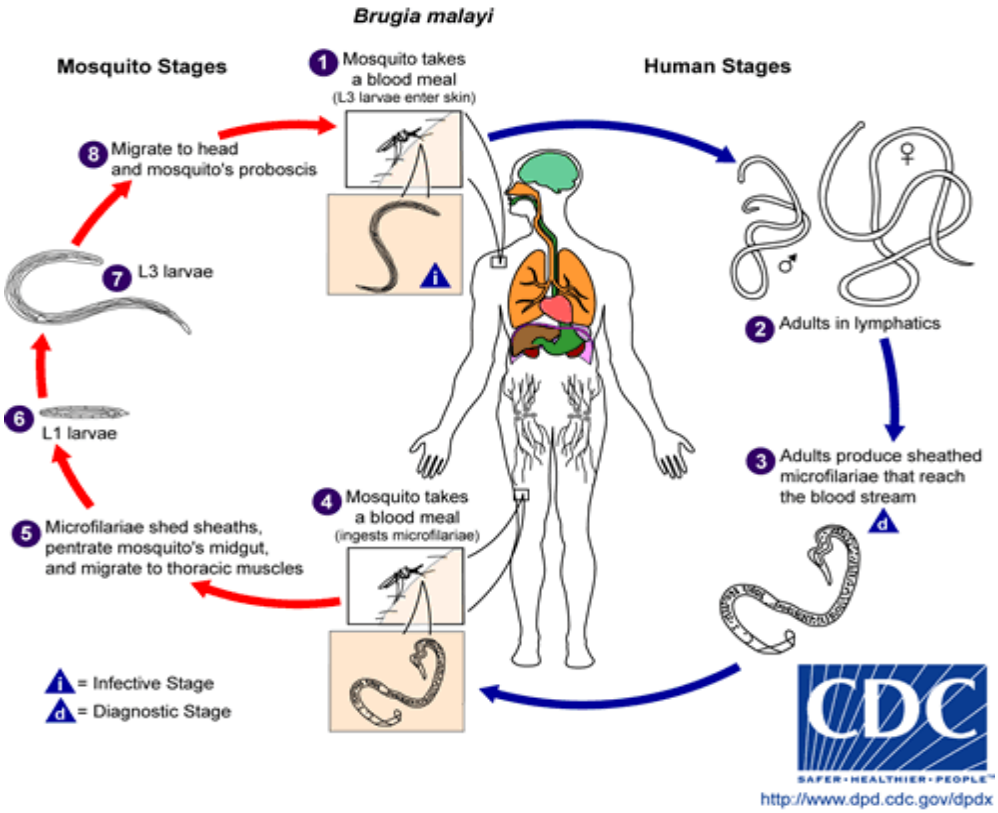

Fig. 5: Life Cycle of Brugia Malayi

Clinical Features and Pathology

Lymphatic filariasis most often consists of asymptomatic microfilaremia. Some patients develop lymphatic dysfunction causing lymphedema and elephantiasis (frequently in the lower extremities) and, with Wuchereria bancrofti, hydrocele and scrotal elephantiasis. Episodes of febrile lymphangitis and lymphadenitis may occur. Persons who have newly arrived in disease-endemic areas can develop afebrile episodes of lymphangitis and lymphadenitis. An additional manifestation of filarial infection, mostly in Asia, is pulmonary tropical eosinophilia syndrome, with nocturnal cough and wheezing, fever, and eosinophilia.

Onchocerciasis can cause pruritus, dermatitis, onchocercomata (subcutaneous nodules), and lymphadenopathies. The most serious manifestation consists of ocular lesions that can progress to blindness. Loiasis (Loa loa) is often asymptomatic. Episodic angioedema (Calabar swellings) and sub-conjunctival migration of an adult worm can occur. Infections by Mansonella perstans, which is often asymptomatic, can be associated with angioedema, pruritus, fever, headaches, arthralgias, and neurologic manifestations.

Mansonella streptocerca can cause skin manifestations including pruritus, papular eruptions and pigmentation changes. Eosinophilia is often prominent in filarial infections. Mansonella ozzardi can cause symptoms that include arthralgias, headaches, fever, pulmonary symptoms, adenopathy, hepatomegaly, and pruritus.

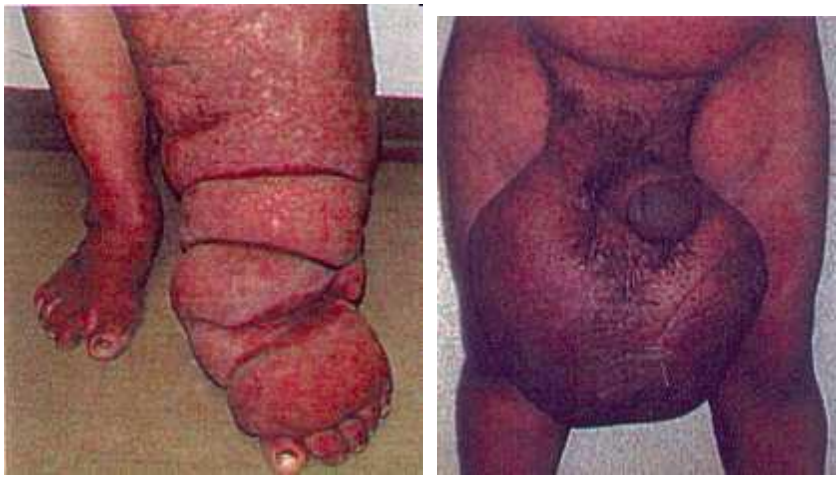

Fig. 6: Elephantiasis Caused by Infection by W. bancrofti

Treatment and Control

Ivermectin is effective in killing the larvae, but does not affect the adult worm. Preventive measures include vector control, treatment of infected individuals and avoidance of black fly.

Laboratory Diagnosis of Filarial Worms

Identification of microfilariae by microscopic examination is the most practical diagnostic procedure.

Microscopy

Examination of blood samples will allow identification of microfilariae of Wuchereria bancrofti, Brugia malayi, Brugia timori, Loa loa, Mansonella perstans, and M. ozzardi. It is important to time the blood collection with the known periodicity of the microfilariae. The blood sample can be a thick smear, stained with Giemsa or haematoxylin and eosin. For increased sensitivity, concentration techniques can be used.

These include centrifugation of the blood sample lysed in 2% formalin (Knott's technique), or filtration through a Nucleopore® membrane. Examination of skin snips will identify microfilariae of Onchocerca volvulus and Mansonella streptocerca. Skin snips can be obtained using a corneal-scleral punch, or more simply a scalpel and needle. The sample must be allowed to incubate for 30 minutes to 2 hours in saline or culture medium, and then examined for microfilariae that would have migrated from the tissue to the liquid phase of the specimen.

Preparing Blood Smears for Microscopy Examination
If one uses venous blood, blood smears should be prepared as soon as possibleafter collection (delay can result in changes in parasite morphology and staining characteristics).

Thick Smears

Thick smears consist of a thick layer of dehemoglobinised (lysed) red blood cells (RBCs). The blood elements (including parasites, if any) are more concentrated (app. 30×) than in an equal area of a thin smear. Thus, thick smears allow a more efficient detection of parasites (increased sensitivity). However, they do not permit an optimal review of parasite morphology. For example, they are often not adequate for species identification of filaria

parasites: if the thick smear is positive for filaria parasites, the thin smear should be used for species identification.

How to Prepare a Thick Smears

1. i. Place a small drop of blood in the centre of the pre-cleaned, labeled slide.
2. ii. Using the corner of another slide or an applicator stick spread the drop in a circular pattern until it is the size of a dime (1.5 cm2).
3. iii. A thick smear of proper density is one which, if placed (wet) over newsprint, allows you to barely read the words.
4. Lay the slides flat and allow the smears to dry thoroughly (protect from dust and insects!). Insufficiently dried smears (and/or smears that are too thick) can detach from the slides during staining. The risk is increased in smears made with anticoagulated blood. At room temperature, drying can take several hours; 30 minutes is the minimum; in the latter case, handle the smear very delicately during staining. You can accelerate the drying by using a fan or hair dryer (uses cool setting).

Protect thick smears from hot environments to prevent heat- fixing the smear.

5. Do not fix thick smears with methanol or heat. If there will be a delay in staining smears, dip the thick smear briefly in water to haemolyse the RBCs.

Thin Smears

Thin smears consist of blood spread in a layer such that the thickness decreases progressively toward the feathered edge. In the feathered edge, the cells should be in a monolayer, not touching one another.

How to Prepare Thin Smears

- Place a small drop of blood on the pre-cleaned, labeled slide, near its frosted end.
- Bring another slide at a 30-45° angle up to the drop, allowing the drop to spread along the contact line of the 2 slides.
- Quickly push the upper (spreader) slide toward the unfrosted end of the lower slide.
- Make sure that the smears have a good feathered edge. This is achieved by using the correct amount of blood and spreading technique.
- Allow the thin smears to dry. (They dry much faster than the thick smears, and are less subject to detachment because they will be fixed.)
- Fix the smears by dipping them in absolute methanol.

Special Procedures for Detecting Microfilariae

Blood Microfilariae:

A. Capillary (Fingerstick) Blood

Since microfilariae concentrate in the peripheral capillaries, thick and thin smears prepared from fingerstick blood are recommended.

B. Anticoagulated (EDTA) venous blood (1 ml) should be concentrated by one of the following methods:

1. Centrifugation (Knott's Technique)

a. Prepare 2% formaldehyde (2 ml of 37% formaldehyde + 98 ml H2O).

b. Mix 9 ml of this 2% formaldehyde with 1 ml of patient's venous blood. Centrifuge at 500 × *g* for 10 minutes; discard supernatant. Sediment is composed of WBCs and microfilariae (if present).

c. Examine as temporary wet mounts.

d. Prepare thick and thin smears; allow to dry and dip in absolute methanol before Giemsa staining to enhance staining of microfilariae.

2. Filtration

- Place millipore or Nucleopore membrane filter (5 µm pore) in filter holder with syringe attachment.
- Mix 1 ml of venous blood (in EDTA) with 10 ml of 10% Teepol® 610 (Shell Co.); allow to stand for several minutes to allow lysis; transfer to a 10 ml Luer-Loc® syringe; attach the filter apparatus.
- Force the solution through the 5 µm pore filter, followed by several syringes of water to wash out the remaining blood, then 1 or 2 syringes full of air to clear excess fluid.
- Prepare a temporary wet mount by removing the filter and placing it on a glass slide, adding a drop of stain or dye and a coverslip.
- For permanent preparations, pass 2 to 3 ml of methanol through the filter while it is still in the holder; remove filter and dry it on a glass slide; then stain it with Giemsa stain, horizontally (so that the filter does not wash off the slide); coverslip filter before examining.

Diagnostic Findings

- Antigen detection using an immunoassay for circulating filarial antigens constitutes a useful diagnostic approach, because microfilaremia can be low and variable. A rapid-format immunochromatographic test, applicable to Wuchereria bancrofti antigens, has been evaluated in the field. However, antibody detection is of limited value. Substantial antigenic cross reactivity exists between filaria and other helminths, and a positive serologic test does not distinguish between past and current infection.
- Molecular diagnosis using polymerase chain reaction is available for W. Bancrofti and B. malayi.
- Identification of adult worms is possible from tissue samples collected during nodulectomies (onchocerciasis) or during subcutaneous biopsies or worm removal from the eye (loiasis).

3.2 Trichinella Spiralis

Historical aspect
In 1835, a man died of tuberculosis in St Bartholomew's Hospital, London. Dr Paget, a first-year student, carried out the autopsy and observed fine hard white inclusions in the muscles. Similar inclusions had been observed by doctors from time to time in the past, but were attributed to commonplace muscle calcification, which quickly blunted the dissecting scalpel. Dr Paget inspected the lesions with a hand lens and quickly recognised their worm-like structure. The name "Trichina spiralis" was suggested. This name Trichina had already been given to a certain fly, however, and the name was later changed to "Trichinella". The discovery of the parasite was published by the famous biologist and palaeontologist Richard Owen, at that time assistant conservator of the museum of the Royal College of Surgeons.

In 1859 Rudolph Virchow carried out transmission experiments in which infected human muscle was fed to a healthy dog. After only 3 to 4 days adult Trichinella worms were found in the dog's duodenum and jejunum.

Life Cycle
More than 100 species of mammals are susceptible to the infection. Infections with Trichinella spiralis affect chiefly carnivores and omnivores, although infection of horses has also been described. People become infected with this nematode by eating raw or insufficiently cooked infected meat, often pork or wild boar. The larvae of Trichinella spiralis which are in the meat develop in a few days into adult worms (2-4 mm) in the wall of the small intestine. There they lay larvae (100 mm).

These spread via the bloodstream to various muscles, including the heart, where they undergo encapsulation (Trichinella pseudospiralis does not form a capsule). The larvae cannot continue to survive in the heart. The larvae are localised within the cells of the muscles, which is unique for a worm. After penetrating the muscle cell, a larva excretes a number of signal molecules and proteins, which convert the cell to what is called a nurse cell.

In the cell the behaviour of the worm is rather similar to that of a virus. Many of its proteins are glycosylated and often carry an unusual sugar (tyvelose). These proteins are excreted from a special organ in the larva (the stichosome). Various muscle proteins such as actin and myosin change or disappear, nuclear division is stimulated and mitochondria are damaged. Local angiogenesis is stimulated by excretion of a blood vessel growth factor and new blood vessels, originating from nearby venules, develop and form a network around the infected cell. The metabolism of the nurse cell and the parasite is essentially anaerobic. After 1 to 4 months the adult worms die. The larvae in the muscles sometimes survive for years and can remain viable for a long time even in rotting flesh. Trichinella is unique among worms in that all development stages take place in the same host. There is never a free stage outside the mammalian body.

Symptoms
Infection may be asymptomatic. In typical cases there is diarrhoea, abdominal pain, vomiting and fever a few days after eating infected meat. After 10 days the fever increases, the patient is very ill and debilitated; there are muscle pains and a typical peri-orbital oedema (differential diagnosis acute trypanosomiasis and nephrosis). This oedema is caused by invasion of the small muscles around the eye. There may be signs of myocarditis, encephalitis, urticaria and asthma. There is often very significant eosinophilia. The myositis

causes an increase in the muscle enzymes (creatine phosphokinase, CK). After a few months the symptoms are reduced or disappear. Mild infections are self-limiting.

Diagnosis
Not many nematodes are found in muscle tissue. Occasionally a migrating third stage larva of Ancylostoma, Toxocara or Gnathostoma may be found (visceral larva migrans). Dracuncula medinensis may also be found in muscle tissue. Another, less common nematode which may be found here is Haycocknema perplexum (Tasmania).

Prevention

- Meat should be well boiled or roasted thoroughly.
- Importance of meat inspection. The diaphragm of a slaughtered animal is inspected (the piece of muscle is flattened between two glass slides and examined using transillumination). This technique (trichinoscopy) is not so good for Trichinella pseudospiralis because it is not surrounded by a capsule and is easily missed.
- Pig food (which may include infected rats) should be boiled for 30 minutes.
- To store pork for 10 days at -25° C is generally impractical in developing countries. In the West meat is sometimes irradiated with high doses of gamma rays, which will kill any larvae. Trichinella spiralis nativa is cold-hardy.

4.0 Conclusion

Blood and tissues are important ingredients for human survival, however despite their importance, several factors exist that can hinder or reduce their continous existence. Therefore, this unit discussed various Parasites affecting human blood and tissue. Also discussed were their life cycles and clinical features associated with their infections.

5.0 Summary

Filariasis is caused by nematodes that inhabit the lymphatics and subcutaneous tissues. Eight main species infect humans of whom three of these are responsible for most of the morbidity due to filariasis: Wuchereria bancrofti and Brugia malayi cause lymphatic filariasis, and Onchocerca volvulus causes onchocerciasis (river blindness) Infective larvae are transmitted by infected biting arthropods during a blood meal. The larvae migrate to the appropriate site of the host's body, where they develop into microfilariae-producing adults. The adults dwell in various human tissues where they can live for several years.

The agents of lymphatic filariasis reside in lymphatic vessels and lymph nodes; Onchocerca volvulus in nodules in subcutaneous tissues; Loa loa in subcutaneous tissues, where it migrates actively; Brugia malayi in lymphatics, as with Wuchereria Bancrofti; Mansonella streptocerca in the dermis and subcutaneous tissue; Mansonella ozzardi apparently in the subcutaneous tissues; and M. perstans in body cavities and the surrounding tissues. The female worms produce microfilariae which circulate in the blood, except for those of

Onchocerca volvulus and Mansonella streptocerca, which are found in the skin and O. volvulus which invade the eye.

The microfilariae infect biting arthropods mosquitoes for the agents of lymphatic filariasis; blackflies (Simulium) for Onchocerca volvulus; midges for Mansonella perstans and M. streptocerca; and both midges and blackflies for Mansonella ozzardi; and deerflies (Chrysops) for Loa loa). Inside the arthropod, the microfilariae develop in 1 to 2 weeks into infective filariform (third-stage) larvae. During a subsequent blood meal by the insect, the larvae infect the vertebrate host. They migrate to the appropriate site of the host's body, where they develop into adults, a slow process than can require up to 18 months in the case of Onchocerca.

Infections with Trichinella spiralis affect chiefly carnivores and omnivores. People become infected by eating raw or insufficiently cooked infected meat, often pork or wild boar. The larvae of Trichinella spiralis which are in the meat develop in a few days into adult worms (2-4 mm) in the wall of the small intestine. There they lay larvae (100 mm). These spread via the bloodstream to various muscles, including the heart, where they undergo encapsulation. Vector control in case of filariasis and proper cooking of pork (Trichinella spiralis) are the control measures.

6.0 Self-Assessment Exercise

Activity: Prepare thick and thin smears for two patients each.

Answer the following questions:

1. Write short note on Wuchereria bancrofti, Onchocerca volvulus, Loa loa (LO1).
2. Describe the life cycles and clinical features associated with Wuchereria bancrofti, Onchocerca volvulus, Loa loa infections (LO2).
3. Describe the laboratory diagnosis and treatment of filarial worms (LO3).

7.0 References /Further Reading

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Unit 4 Air-Borne Nematode

1.0 Introduction

The human pinworm Enterobius vermicularis is an ubiquitous parasite of man. It is estimated that over 200 million people are infected annually. It is more common in the temperate regions of Western Europe and North America, (it is being relatively rare in the tropics) and is found particularly in children. Samples of Caucasian children in the U.S.A. and Canada have shown incidences of infection of between30% to 80%, with similar levels in Europe, and although these regions are the parasites strongholds, it may be found throughout the world, again often with high degrees of incidence. For example in parts of South America the incidence in school children may be as high as 60%.

Interestingly non-Caucasians appear to be relatively resistant to infection with this nematode. As a species, and contrary to popular belief, E. vermicularis is entirely restricted to man, other animals harbouring related but distinct species that are non-infective to humans, but their fur may be contaminated by eggs from the human species if stroked by someone with eggs on their hands. In man anywhere where there are large numbers of children gathered together, (such as nurseries, play groups, orphanages etc.), especially if conditions are insanitary, are ready sources of infection, as one child may rapidly transmit the parasite to his or her fellows.

2.0 Objectives

At the end of this unit, you should be able to:

- describe the morphology of enterobius vermicularis
- describe the life cycle of enterobius vermicularis
- examine the pathology, diagnosis and control of enterobius vermicularis.

3.0 Main Content

3.1 Enterobius Vermicularis (The Human Pin-Worm)

Morphology
These creamy white coloured nematodes are relatively small, with the female measuring only approximately 10mm by 0.4mm wide. The females have a cuticular expansion at their anterior ends, with a long pointed tail. The male parasites, which are much less numerous than the females, are much smaller, measuring only up to 5mm long, and have a curved tail, with a small bursa like expansion, and a single spicule. The head has a mouth with three small lips.

Fig. 1: Adult Pinworm

Life Cycle

The adult parasites live predominantly in the caecum. The male and females mate, and the uteri of the females become filled with eggs. The gravid females (each containing up to 15000 eggs) then migrate down the digestive tract to the anus. From here they make regular nocturnal migrations out of the anus, to the perianal region, where air contact stimulates them to lay their eggs, before retreating back into the rectum. Eventually the female die, their bodies disintegrating to release many remaining eggs. These eggs, which are clear and measure about 55 by 30µm, then, mature to the infectious stage (containing an L1 larvae) over 4 to 6 weeks. To infect the host, typically these eggs must then be ingested. The ingested eggs hatch in the duodenum. The eggs themselves are sticky, and have a characteristic shape, shared with all members of the group Oxyuridea, with an asymmetrical form, flattened on one side, (see below);

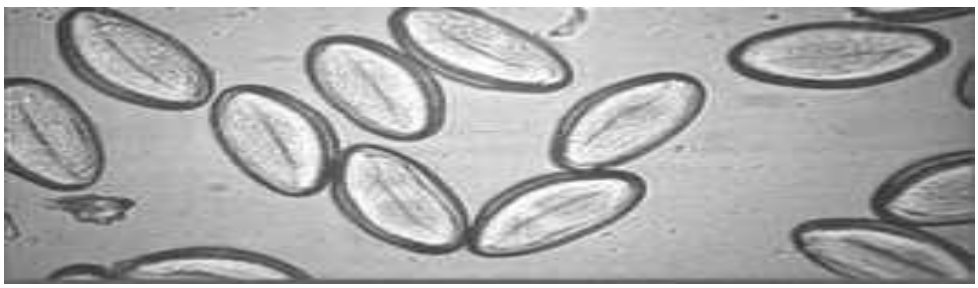

Fig. 2: The Ova of Enterobius Vermicularis

The larvae then undergo a series of moults, as they migrate down the digestive tract. The adult worms then mature in the caecum, before copulating to complete the cycle (typically 6 weeks). Occasionally the eggs hatch in the perianal region itself, the resulting L1 larvae being fully infective, crawling back through the anus, then migrating up the intestine to the caecum (retroinfection).

Pathology of Infection

The majority of infections with this nematode are asymptomatic, although in some cases the emerging females and the sticky masses of eggs that they lay may causes irritation of the perianal region, which in some cases may be severe. As the females emerge at night this may give rise to sleep disturbances, and scratching of the affected perianal area transfers eggs to the fingers and under the finger nails. This in turn aids the transmission of the eggs, both back to the original host (autoinfection), and to other hosts.

Diagnosis
Because eggs are rarely passed out with faeces, examination of faecal samples may not reveal them. This may account for negative results of enterobiasis in many of the surveys for helminth infections involving faecal samples in tropical Africa. The most reliable diagnosis is by the cellophane tape swab. This involves the attachment of a piece of cellophane to the perianal region overnight. This is then examined for eggs under the microscope. Alternatively, the anus and perianal area can be examined under bright light at night, at which time adult worms can be seen glittening in the light.

Epidemiology and Control
The eggs of the parasite are air-borne, caught in clothing, household linen, curtain, carpets, etc. As such, infection is common in dry season than rainy season in the tropics. Maitenance of high standards of personal and domestic hygiene is therefore imperative for control and prevention.

4.0 Conclusion

In this unit, the morphology of Enterobius vermicularis and their life cycle have been discussed. Also discussed were the pathology, diagnosis and control of Enterobius vermicularis.

5.0 Summary

Enterobius vermicularis is an air-borne parasitic infection common mostly in the temperate regions of the world. Adult female worms lay eggs in the perianal regions and infection occurs through direct ingestion of eggs containing the L1 larvae. Infection is usually asymptomatic but sometimes the sticky eggs could cause irritation of the perianal giving rise to scratching and sleep disturbance. Maitenance of high standards of personal and domestic hygiene is imperative for control and prevention.

6.0 Self-Assessment Exercise

Activity: Conduct a physical examination of an adult pinworm under the microscope and report your findings in the log book.

Answer the following questions:

1. With a well label diagram describe the morphology of enterobius vermicularis (LO1).
2. Describe the life cycle of enterobius vermicularis (LO2).
3. Explain the pathology, diagnosis and control of enterobius vermicularis (LO3).

7.0 Reference/Further Reading

Brown, H.W. & Neva, F.A. (1983). *Basic Clinical Parasitology*. (5th ed.). pp. 128-132.