Enterobiasis:-

Background

Enterobius vermicularis, commonly referred to as pinworm, has the largest geographical distribution of any helminth. Discovered by Linnaeus in 1758, it was originally named *Oxyuris vermicularis* and the disease was referred to as *oxyuriasis* for many years. It is believed to be the oldest parasite described and was recently discovered in ancient Egyptian mummified human remains as well as in DNA samples from ancient human coprolite remains from North and South America.

Enterobius is one of the most prevalent nematodes in the United States and in Western Europe. At one time, in the United States there are an estimated 42 million infected individuals. It is found worldwide in both temperate and tropical areas. Prevalence is highest among the 5-10 year-old age group and infection is uncommon in children less than two years old. Enterobiasis has been reported in every socioeconomic level; however spread is much more likely within families of infected

individuals, or in institutions such as child care centers, orphanages, hospitals and mental institutions. Humans are the only natural host for the parasite. Infection is facilitated by factors including overcrowding, wearing soiled clothing, lack of adequate bathing and poor hand hygiene, especially among young school-aged children. Infestation follows ingestion of eggs which usually reach the mouth on soiled hands or contaminated food. Transmission occurs via direct anus to mouth spread from an infected person or via airborne eggs that are in the environment such as contaminated clothing or bed linen. The migration of worms out of the gastrointestinal tract to the anus can cause local perianal irritation and pruritus. Scratching leads to contamination of fingers, especially under fingernails and contributes to autoinfection. Finger sucking and nail biting may be sources of recurrent infection in children. Spread within families is common. *E. vermicularis* may be transmitted through sexual activity, especially via oral and anal sex. When swallowed via contaminated hands, food or water, the eggs hatch releasing larvae . The larvae develop in the upper small intestine and mature in 5 to 6 weeks without undergoing any further migration into other body cavities (i.e., lungs). Both male and

female forms exist. The smaller male is 2-5 mm in length and 0.3 mm in diameter whereas the female is 8-13 mm long and up to 0.6 mm in diameter . Copulation occurs in the distal small bowel and the adult females settle in the large intestine where they can survive for up to 13 weeks (males live for approximately 7 weeks). The adult female can produce approximately 11,000 eggs.

A gravid female can migrate out through the anus to lay her eggs. This phenomenon usually occurs at night and is thought to be secondary to the drop in host body temperature at this time. The eggs embyonate and become infective within 6 hours of deposition. In cool, humid climates the larvae can remain infective for nearly 2 weeks, but under warm, dry conditions, they begin to lose their infectivity within 2 days. Most infected persons harbor a few to several hundred adult worms.

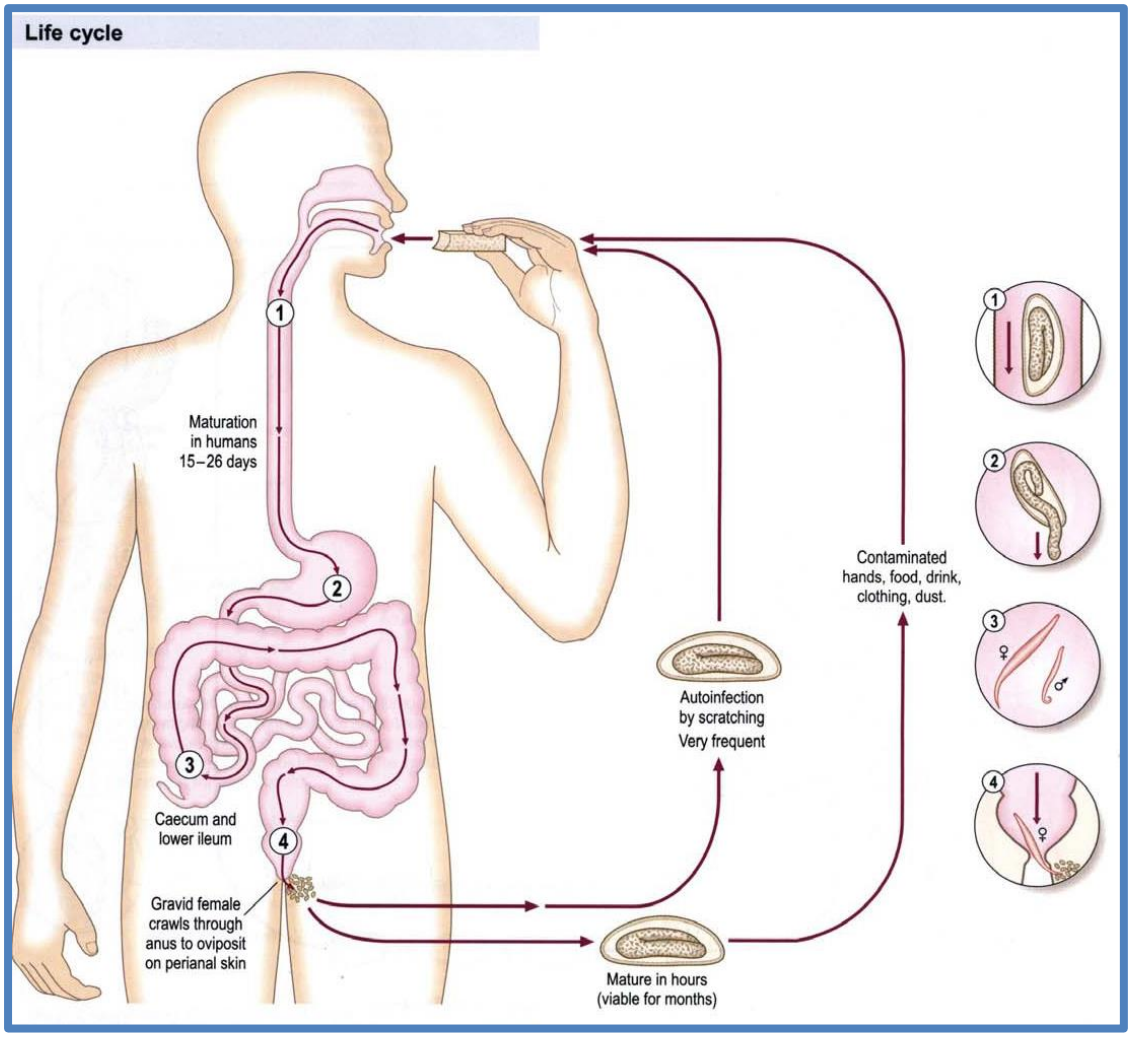
Disease Signs and Symptoms

The majority of enterobiasis cases are asymptomatic; however the most common symptom is perianal or perineal pruritus. Th is varies from mild itching to acute pain. Symptoms tend to be most troublesome at night and, as a result, infected individuals oft en report sleep disturbances, restlessness and insomnia. Th e most common complication of infection is secondary bacterial infection of excoriated skin. Folliculitis has been seen in adults with enterobiasis.

Gravid female worms can migrate from the anus into the female genital tract.

Vaginal infections can lead to vulvitis, serous discharge and pelvic pain. There are numerous reports of granulomas in the vaginal wall, uterus, ovary and pelvic peritoneum caused by E. vermicularis dead worms or eggs. Pre-pubertal and adolescent girls with E. vermicularis infection can develop vulvovaginitis. Scratching may lead to introital colonization with colonic bacteria and thus may increase susceptibility to urinary tract infections. Although ectopic lesions due to E. vermicularis are rare, pinworms can also migrate to other internal organs, such as the appendix, the prostate gland, lungs or liver, the latter being a result of egg embolization from the colon via the portal venous system. Within the colonic mucosa or submucosa granulomas can be uncomfortable and may mimic other diseases such as carcinoma of the colon or Crohn’s disease. E. vermicularis has been found in the lumen of uninfl amed appendices in patients who have been operated on for

acute appendicitis. Although eosinophilic colitis has been described with enterobiasis, eosinophilia is uncommon in infected individuals.



Diagnosis

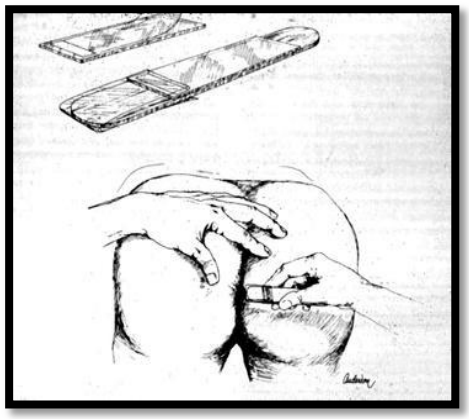
The diagnosis of E. vermicularis infestation rests on the recognition of dead adult worms or the characteristic ova. In the perianal region, the adult female worm may be visualized as a small white “piece of thread”. The most successful diagnostic method is the “Scotch tape” or “cellophane tape” method .

This is best done immediately after arising in the morning before the individual defecates or bathes. The buttocks are spread and a small piece of transparent or cellulose acetate tape is pressed against the anal or perianal skin several times. The strip is then transferred to a microscope slide with the

adhesive side down. The worms are white and transparent and the skin is transversely striated. The egg is also colorless, measures 50-54 × 20-27 mm and has a characteristic shape, flattened on one side. Examination of a single specimen detects approximately 50% of infections; when this is done on three consecutive mornings sensitivity

rises to 90%. Parija et al. found a higher sensitivity if lacto-phenol cotton blue stain was used in detecting eggs aft er the tape test was performed. Six consecutive negative swabs on separate days are necessary to exclude the diagnosis. Stool examination for eggs is usually not helpful, as only 5-15% of infected persons will have positive results. Rarely, E. vermicularis eggs have been found in cervical

specimens (done for routine Papanicolaou smears), in the urine sediment, or the worms have been seen during colonoscopy. Serologic tests specific for E. vermicularis are not available.



Treatment:

Several effective drugs such as pyrantel, pyrvinium and mebendazole can be used for single dose therapy while piperazine given daily for one week. It is necessary to repeat the treatment after two weeks to ensure elimination of all worms.

Hookworm

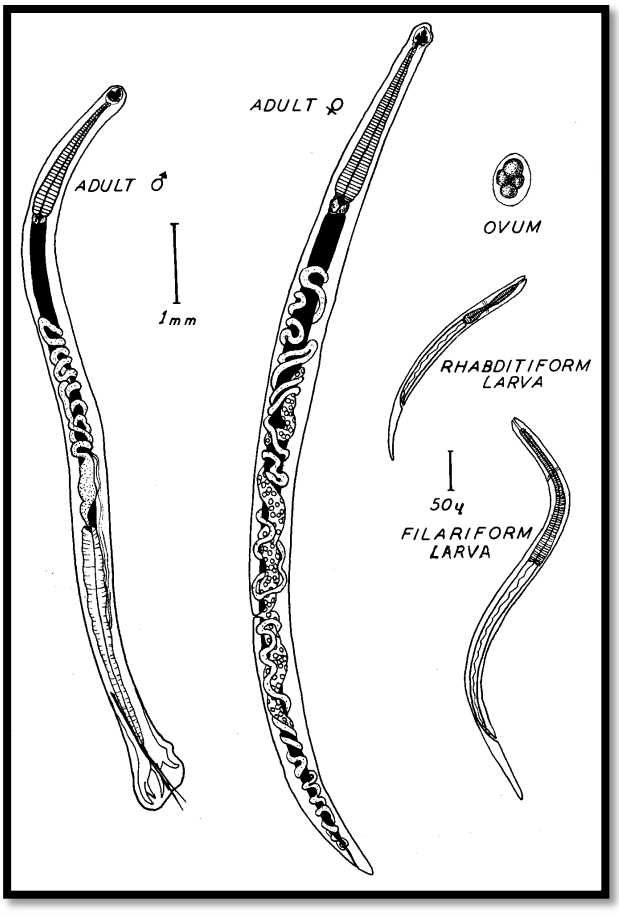
Human hookworm infection is a soil-transmitted helminth infection caused primarily by the nematode parasites Necator americanus and Ancylostoma duodenale. It is one of the most important parasitic infections worldwide, ranking second only to malaria in terms of its impact on child and maternal health. An estimated 576 million people are chronically infected with hookworm and another 3.2 billion are at risk, with the largest number of afflicted individuals living in impoverished rural areas of sub-Saharan Africa, southeast Asia and tropical regions of the Americas. N. americanus is the most widespread hookworm globally, whereas A. duodenale is more geographically restricted in distribution. Although hookworm infection does not directly account for substantial mortality, its greater health impact is in the form of chronic anemia and protein malnutrition as well as impaired physical and intellectual development in children.

Humans may also be incidentally infected by the zoonotic hookworms Ancylostoma caninum, Ancylostoma braziliensis, which can cause self-limited dermatological lesions in the form of cutaneous larva migrans. Additionally, Ancylostoma ceylanicum, normally a hookworm infecting cats, has been reported to cause hookworm disease in humans especially in Asia, whereas A. caninum has been implicated as a cause of eosinophilic enteritis in Australia.

Life Cycle

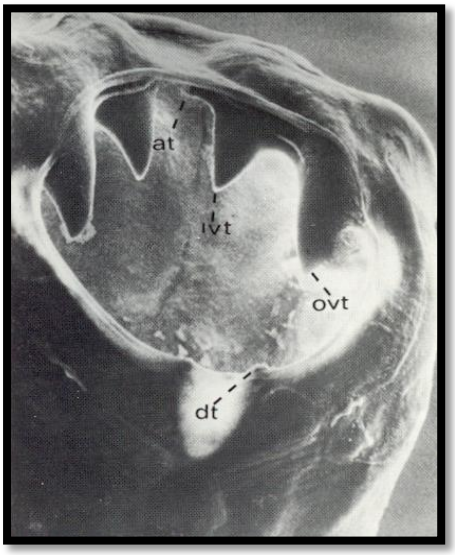
Hookworm transmission occurs when third-stage infective filariform larvae come into contact with skin . Hookworm larvae have the ability to actively penetrate the cutaneous tissues, most often those of the hands, feet, arms and legs due to exposure and usually through hair follicles or abraded skin. Following skin penetration, the larvae enter subcutaneous venules and lymphatics to gain access to the host's afferent circulation. Ultimately, they enter the pulmonary capillaries where they penetrate into the alveolar spaces, ascend the trachial tree to the trachea, traverse the epiglottis into the pharynx and are swallowed into the gastrointestinal tract. Larvae undergo two molts in the lumen of the intestine before developing into egg-

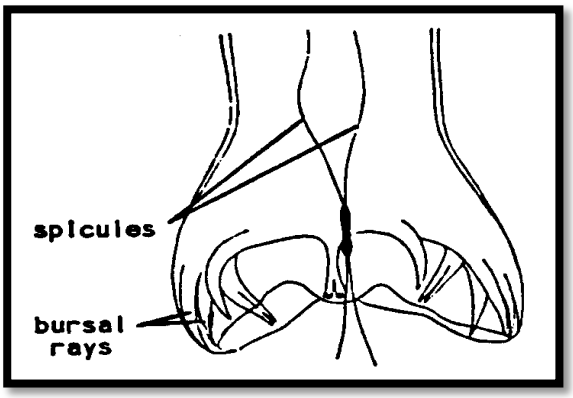
laying adults approximately five to nine weeks after skin penetration. Although generally one centimeter in length, adult worms exhibit considerable variation in size and female worms are usually larger than males. Adult Necator and Ancylostoma hookworms parasitize the proximal portion of the human small intestine where they can live for several years, although differences exist between the life spans of the two species: A. duodenale survive for on average one year in the human intestine whereas N. americanus generally live for three to five years . Adult hookworms attach onto the mucosa of the small intestine by means of cutting teeth in the case of A. duodenale or a rounded cutting plate in the case of N. americanus. After attachment, digestive enzymes are secreted that enable the parasite to burrow into the tissues of the submucosa where they derive nourishment from eating villous tissue and sucking blood into their digestive tracts. Hemoglobinases within the hookworm digestive canal enable digestion of human hemoglobin, which is a primary nutrient source of the parasite.



The body is curved with the dorsal aspect concave and the ventral aspect convex, the anterior end of the worm is bent likely dorsally hence the name hook worms. The buccal capsule provided with a hard chitin-like substance carries two pairs of hook-like teeth ventrally and dental plate with median cleft dorsally, in the depth of the capsule there is a pair of small teeth.

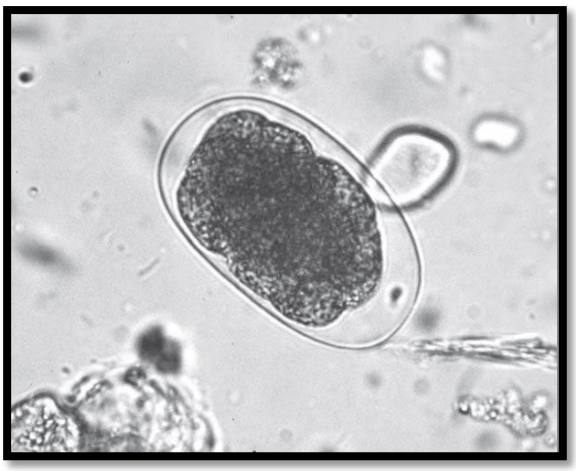
The male measures about 8-11 mm length, the posterior end is expanded as umbrella like structure supported by fleshy rays (copulatory bursa). In this worm the number of rays 13 distributed on three lobes, one dorsal and two lateral (tripatit) three of these rays supported the dorsal lobe and ten supported the lateral lobes. The pattern of distribution of rays helps in distinguishing between the different species. The cloaca is situated in the bursa, there are two long bristle-like spicules project from the bursa.





The female is larger 10-13 mm length, the posterior end is conoid. The vulva opens ventrally, so during the copulation the male attaches its copulatory bursa to the vulva therefore appears as Y-shape.

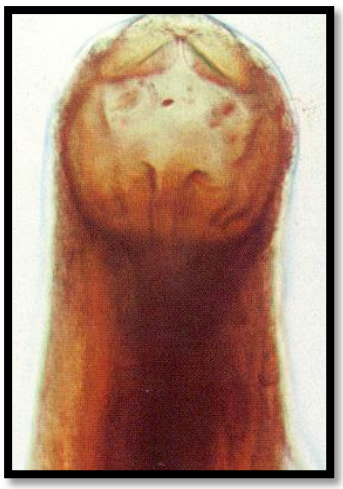
The egg is oval or elliptical, measuring 40 X 60 µm, colorless, not bile stained, with a thin transparent hyaline shell membrane, when released by the worm in the intestine, the egg contain unsegmented ovum. During its passage down the intestine, the ovum develops. When passed with feces its contain a segmented ovum usually 4 blastomeres. There is a clear space between the ovum and shell.

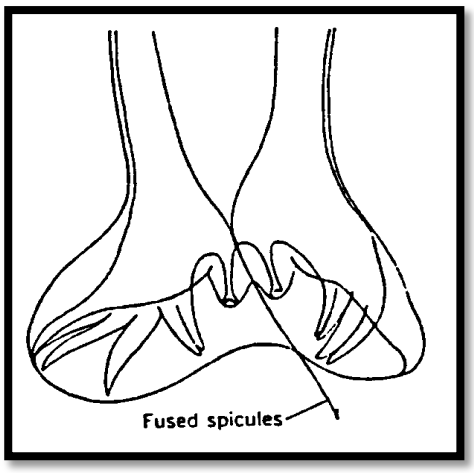


The filariform larvae are non-feeding and can life in the soil for about five weeks waiting their host and also ascend on blades and grass or other vegetation. Direct sunlight, drying or salt water can kill the larvae. So the infection rarely may take place by the oral route, the filariform larvae being carried on vegetables or fruits. These larvae may penetrate the buccal mucosa to reach venous circulation and complete the life cycle. Alternatively the larvae may be swallowed and develop directly into the adult in the small intestine without tissue phase. Transmammary and transplacental transmission has been reported for *Ancylostoma* but not for *Necator.*

Necator americanus

The adult worm are slightly smaller than *Ancylostoma duodenale*, the male measures about 7-9 mm and female 9-11 mm. The anterior end is bent in a direction opposite to the general curvature of the body. While *Ancylostoma duodenale* the bent is in the same direction. They have the smaller buccal cavity with two pairs of cutting plates instead of teeth. The copulatory bursa supported by 14 rays distributed on two lobes so this named (bipartite). The copulatory are fused at the end to form barbed tip.





Clinical Manifestations

The clinical features of hookworm infection can be separated into the acute manifestations associated with larval migration through the skin and other tissues and the acute and chronic manifestations resulting from parasitism of the gastrointestinal tract by adult worms. Migrating hookworm larvae provoke reactions in many of the tissues through which they pass, including several cutaneous syndromes that result from skin-penetrating larvae. Repeated exposure to N. americanus and A. duodenale filariform larvae can result in a hypersensitivity reaction known as "ground itch", a pruritic local erythematous and papular rash that appears most commonly on the hands and feet. In contrast, when zoonotic hookworm larvae (typically A. braziliensis,A. caninum) penetrate the skin, usually after direct contact between skin and soil or sandy beaches contaminated with animal feces, they produce cutaneous larva migrans, most often on the feet, buttocks and abdomen. Since these zoonotic larvae are unable to complete their life cycle in the human host, they eventually die aft er causing a typical clinical syndrome of erythematous linear tracts with a serpiginous appearance and intense pruritus. These tracts can elongate by several centimeters a day; larvae can migrate for up to one year, but the lesions usually heal spontaneously within weeks to months although secondary pyogenic infection may occur at these sites as well as those of ground itch. One to two weeks following skin invasion, hookworm larvae travel through the vasculature and enter the lungs, where they can uncommonly result in

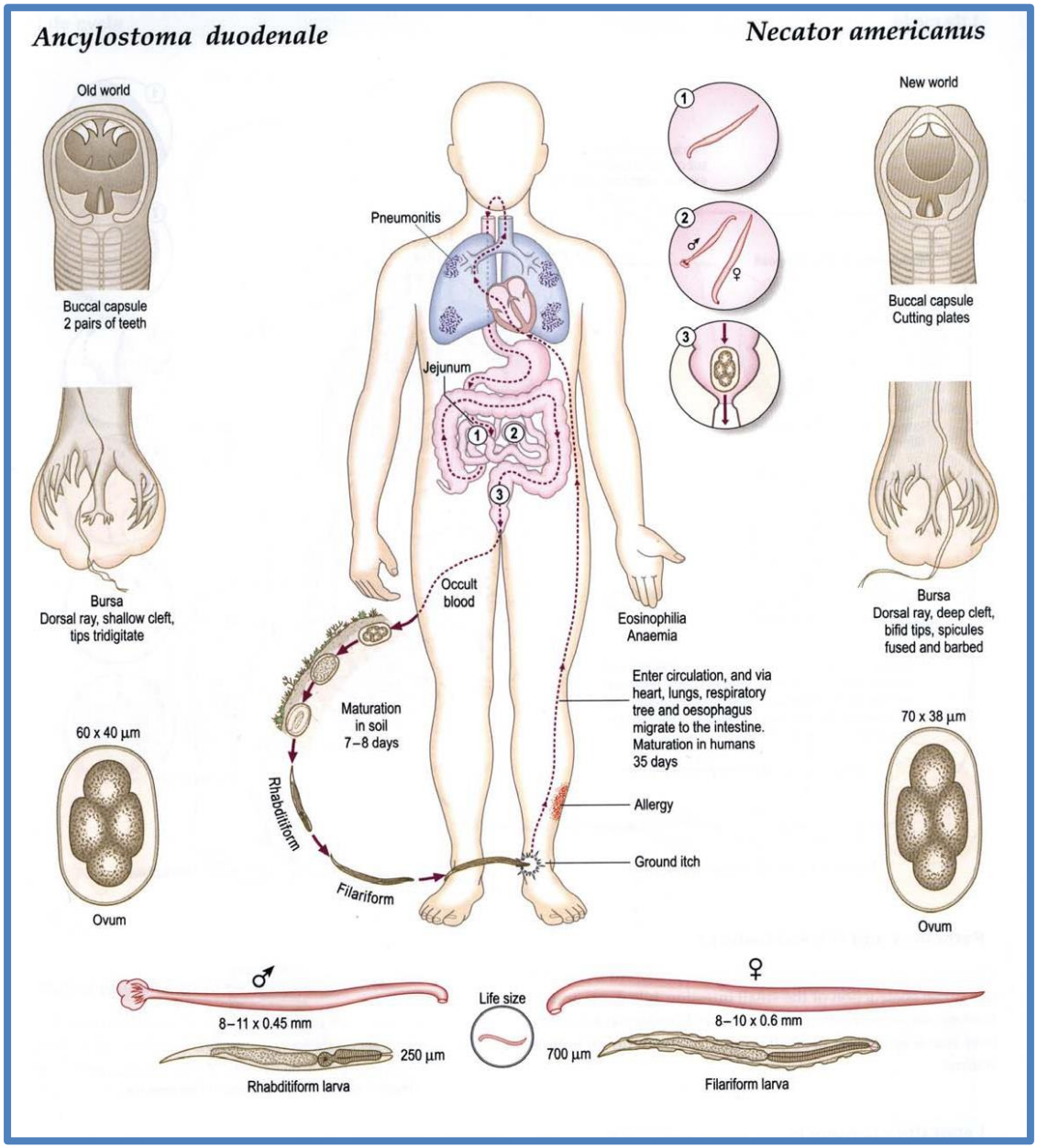
pneumonitis. The pulmonary symptoms that may develop are usuall mild and transient, consisting of a dry cough, sore throat, wheezing and slight fever. The pulmonary symptoms are more pronounced and of longer duration with A. duodenale than with N. americanus infection. Acute symptomatic disease may also result from oral ingestion of A. duodenale larvae, referred to as the Wakana syndrome, which is characterized by nausea, vomiting, pharyngeal irritation, cough, dyspnea and hoarseness. In hookworm infection, the appearance of eosinophilia coincides with the development of adult hookworms in the intestine. The major pathology of hookworm infection, however, results from the intestinal blood loss that results from adult parasite invasion and attachment to the mucosa and submucosa of the small intestine. Usually only moderate and high intensity hookworm infections in the gastrointestinal tract produce clinical manifestations, with the highest intensity infections occurring most often in children, although even in low intensity infections, initial symptoms may include dyspepsia, nausea and epigastric distress. A. duodenale may also result in acute enteritis with uncontrollable diarrhea and foul stools that may last indefinitely.

Chronic hookworm disease occurs when the blood loss due to infection exceeds the nutritional reserves of the host, thus resulting in iron-deficiency anemia. It has been estimated that the presence of more than 40 adult worms in the small intestine is sufficient to reduce host hemoglobin levels below 11 g per deciliter, although the exact number depends on several factors including the species of hookworm and host iron reserves. Worm-for-worm, A. duodenale causes more blood loss than N. americanus: whereas each N. americanus worm produces a daily blood loss of 0.03 to 0.1 ml, the corresponding figure for A. duodenale is between 0.15 and 0.26 ml. The clinical manifestations of chronic hookworm disease resemble those of iron deficiency anemia due to other etiologies, while the protein loss from heavy hookworm infection can result in hypoproteinemia and anasarca. Th e anemia and protein malnutrition that results from chronic intestinal parasitism cause long-term impairments in childhood physical, intellectual and cognitive development. As iron deficiency anemia develops and worsens, an

infected individual may have weakness, palpitations, fainting, dizziness, dyspnea, mental apathy and headache.

Diagnosis:

Demonstration of the eggs in the feces by direct microscopy or concentration methods is the diagnostic test. In stool samples examined 24 hr. or more after collection the eggs may hatched and rhabditiform larvae may be present.



Treatment:

- Mebenadazole and pyrantel pamoate are the drugs of choice.
- Thiabendazole is less effective.
- The treatment includes relief of anaemia, oral iron is effective, but in severe cases a preliminary packed cell transfusion may be needed.

Blood and tissues dwelling nematodes

— A divers group of nematodes parasitised the blood vascular system and other organs and tissues of human. Human are only known definitive host for a few of these, some are shared with other vertebrates and for large number the human are relatively accidental host.

— These are includes :-

1- guinea worm.

2- filarial worms.

Dracunculiasis:-

Dracunculiasis (also know as dracontiasis) is caused by the "guinea worm" Dracunculus medinensis. It has been described in humans since antiquity with references to this infection being noted in the Bible and ancient Greek and Roman texts. A calcified adult worm has also been noted by X-ray in an Egyptian mummy and there are descriptions of the disease in ancient papyrus texts.

Epidemiology

Two important conditions need to be satisfied for transmission of Dracunculus to occur: emergent guinea worm lesions discharging larvae need to be in contact with drinking water and the intermediate

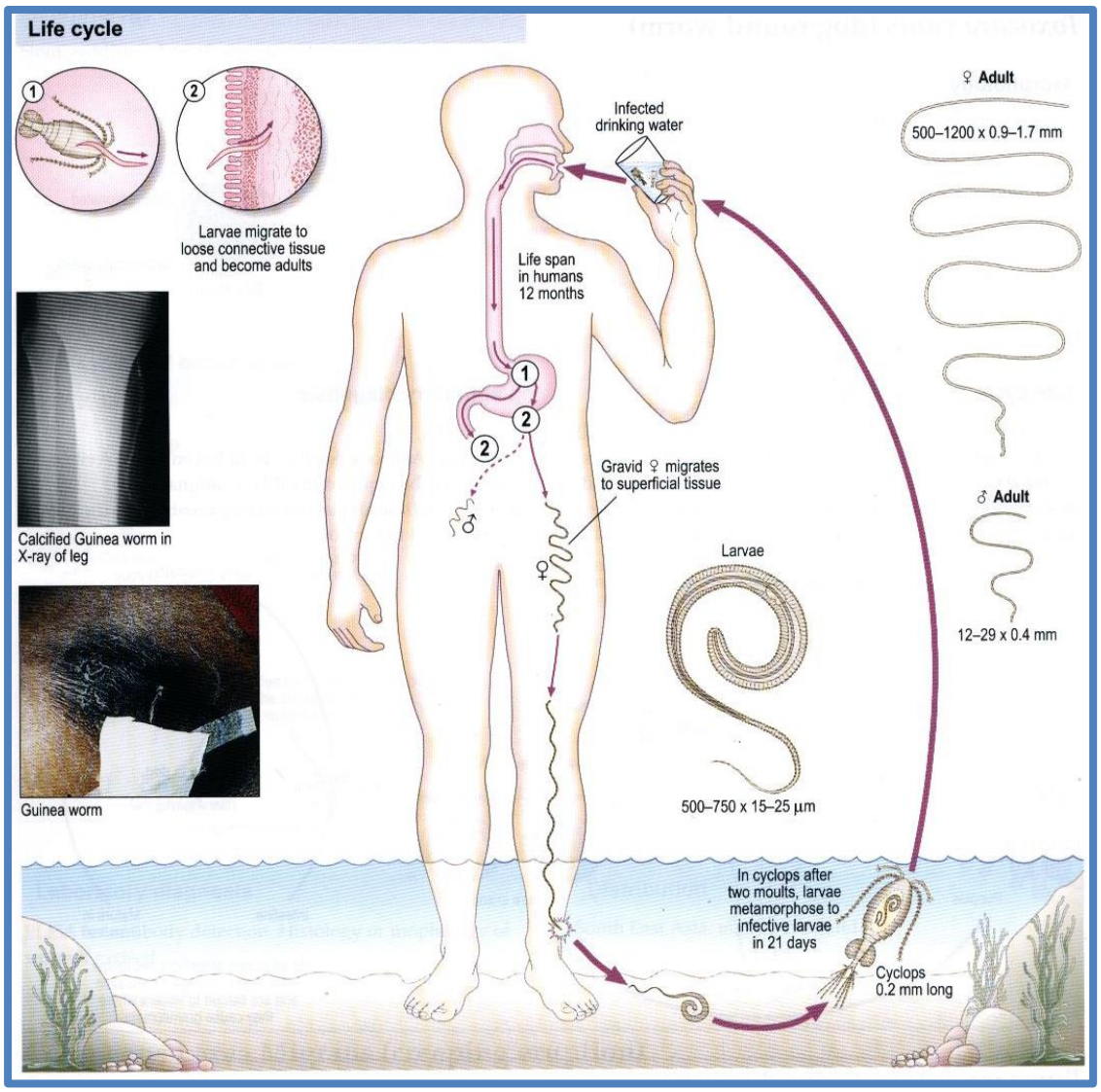
copepod host needs to be present in the water supply. Contamination of step wells, cisterns and ponds is common in endemic areas. Transmission in ponds is highest just before the rainy season, when the density of copepods may be the highest.

Life Cycle

The copepod is the intermediate host for Dracunculus larvae and measures 1-3 mm in size. Following ingestion of an infected copepod the third stage infective larva is released.. The larva then penetrates through the intestinal mucosa and migrates to connective tissues, especially in the abdominal wall and thoracic wall and retroperitoneum, where it develops into an adult male or female worm. The adult female worms are cylindroidal, 1-2 mm in width and up to 800 mm in length; the males much smaller at about 40 mm in length. The female is likely fertilized in this site before migration begins to the subcutaneous tissues. By 8-9 months after ingestion the uterus of the female worm becomes filled with eggs which then develop into first stage larvae. Full maturation takes up to 12 months, contributing to its seasonal endemicity. The gravid worm, containing 1-3 million first stage larvae, then embarks on a period of subcutaneous migration eventually taking it to the surface. After migration the anterior end of the worm approaches the dermis, where first a papule and then a blister is formed. The lesions occur predominately on the distal lower extremities. On contact with fresh water the blister ruptures and first stage larvae (rhabditoid) are released as the uterus prolapses either through the mouth or body wall. The motile larvae are 15-25 µm × 500-750 µm in size and are subsequently ingested by the copepods. They penetrate through the intestinal wall. The larvae molt twice in the copepod over a period of 2-4 weeks and remain dormant as third stage (infective) larvae. Human infection again takes place after ingestion of infected copepods.

Clinical Manifestations

Initial ingestion of the infected copepod does not usually provoke any symptoms although urticarial reactions sometimes occur. Clinical disease is a direct result of the adult female worm which migrates in the subcutaneous tissue until stopping to discharge the larvae through the skin. The individual may notice a palpable or migrating worm in up to one-third of cases. Others may complain of allergic symptomatology prior to worm emergence: urticaria, infra-orbital edema, fever, dyspnea. The majority present with local signs of emergent worms. Emergent lesions are primarily located in the lower extremities (> 90%), which are also the body areas most likely to come in contact with fresh water and thus allow completion of the life cycle. Worms may also emerge from the upper extremity, trunk, or head. Errant worms have appeared rarely in unusual location such as the epidural space, testicle, orbit and eyelid.



Diagnosis

After the appearance of the skin ulcer and worm, the diagnosis can be certain. Discharged material from the gravid uterus will reveal larvae characteristic of *Dracunculus*. Characteristic calcifications can also be identified in areas where the worms have died and calcified and represent a "sarcophagus around a long departed parasite". These are most common in the lower extremities where they lie along tissue planes in a linear fashion; but have also been noted in the upper extremities and in the trunk where they may have a coiled appearance.

Treatment

Treatment of dracunculiasis is generally focused on mechanical removal of the worm and treatment of secondary infection. The traditional method has been to wind the protruding worm on a stick and with gentle traction to remove several inches at a time. Extraction of the worm may occasionally be difficult. Incomplete removal or breakage of the worm with spillage of contents can lead to a more significant inflammatory response and appears to increase the risk of secondary infection.