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WORM INFECTION IN THE COMPROMISED HOST

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Abstract

Worm infections, caused by parasitic worms or helminths, affect billions globally, particularly compromising individuals with weakened immune systems due to conditions like HIV, malnutrition, or medications. Transmission occurs through ingestion of contaminated food/water or skin penetration. Worms like Ascaris, hookworms, and Strongyloides cause various complications like hyper-infection, anemia, and blockages. Diagnosis involves fecal tests, blood tests, and imaging. Treatment primarily relies on antiparasitic medications. Prevention strategies include hygiene practices, safe food handling, and mass de-worming. Establishing robust healthcare systems is crucial to mitigate the impact of worm infections on compromised individuals. To conclude this review, parasitic worm infections, particularly in immunocompromised individuals, pose significant health risks and require careful management. Effective prevention strategies, including sanitation improvements and hygiene practices, are crucial. Accurate diagnosis through various laboratory techniques facilitates appropriate treatment selection. It is therefore recommended that continued research into diagnostic methods and treatment options be an essential for better management and control of these infections and also a well-developed medical healthcare system should be put in place to control the devastating effects of worm infection on compromised host.

Keywords: *worm infection, immunocompromised. Helminthes parasites*

1.0 INTRODUCTION

World Health Organization (WHO) estimates, *Ascaris lumbricoides* infects over 1 billion people, *Trichuris trichiura* 795 million, and hookworms 740 million. Infections caused by intestinal parasites are widespread [1]. Current assessments suggest that at least 25% of the world's population is chronically infected by enteric parasites [2]. These infections would be expected to be appreciably higher in developing countries due to higher prevalence of infections in the general population. However, Intestinal opportunistic parasitic infections in Human Immunodeficiency Virus (HIV)-infected subjects present commonly as diarrhea [3]. Although, HIV infection is not highly prevalent in the Middle East [4], it is a rapidly spreading infection in this region [5]. Globally it was estimated that 42 million people were HIV-infected with the majority of patients (29.4 million) from Africa. Since, the first described cases of acquired immunodeficiency syndrome (AIDS), a high prevalence of gastrointestinal infections has been reported in such patients, in the form of diarrhea associated with parasitosis. Studies investigated the existence of interaction between HIV and parasitic infections in co-infected individuals. Parasitic infections particularly helminthes cause chronic immune activation [6]. Though proving evidences are insufficient, such immune modulation 2 was shown to increase host susceptibility, thereby, promoting HIV infection and disease progression [7]. Parasites that can cause infection include; *Cappilaria hepatica, Cappilaria philippinensis, Trichuris trichiura, Ascaris lumbricoides, Strongyloides stercoralis, and Ancylostoma duodenale/Necator americanus* are also frequently encountered in developing countries. A person with intestinal worms may also experience dysentery which is the presence of blood and mucus in a watery stool. Intestinal worms can also cause a rash or itching around the rectum or vulva. In some cases, worm is passed out in the stool during a bowel movement. Some people may have intestinal worms for years without experiencing any symptoms. [8]

An immunocompromised host is a patient who does not have the ability to respond normally to an infection because of an impaired or weakened immune system. This inability to fight infection can be caused by a number of conditions, including diseases (e.g., diabetes, human immunodeficiency virus [HIV] infection), malnutrition, and drugs. Immunocompromised children are becoming more commonly encountered in daily clinical practice. This is likely due to the increased prevalence of HIV infection, improved survival rates of children with malignancy, and an increased number of children receiving immunosuppressive agents for differing reasons. Immunocompromised children are prone to various infections, including those affecting the gastrointestinal tract, which can manifest as severe protracted diarrhoea, chronic malabsorption, failure to thrive, and malnutrition. Among different pathogens causing gastrointestinal infection, parasites are likely to have significant roles as the primary cause or comorbidity of diarrhoea in immunocompromised children. The incidence of parasitic infection in HIV patients with diarrhoea was reported to be 53-83%. Similar prevalence rates (42%) were also found in children with malignancy [9].

There are two main routes of transmission:

1. Fecal-oral route: Ingestion of parasite by mouth is the commonest method of transmission of parasites. It can occur through contaminated food or water with faeces containing the infective stage of the parasite e.g. *Trichuris trichiura*. It can also occur through the ingestion of uncooked or not properly cooked meat, fish or vegetables e.g. *Taenia species*.
2. Penetration through skin:
   The infective stage of some certain parasite enter the body through the skin, and through the blood migrate to the site where they can mature and reproduce e.g. the infective (filariform) stage of hookworm and *Strongyloides stercoralis.*

2.0 TYPES OF WORMS FOUND IN IMMUNOOMPROMSED AND THEIR EFFECTS

The following are the types; Strongyloides stercoralis, Capillaria philippinensis, Hymenolepsis nana, Ascaris lumbricoides, Schistosoma mansoni, Hook worm, *Capillaria hepatica, Trichuris trichiura, Enterobius vermicularis* etc.

2.1 *Strongyloides stercoralis*

Etiology: *Strongyloides stercoralis,* the causative agent of strongyloidiasis, is an intestinal nematode found worldwide in moist soil contaminated by human faeces. This organism currently infects approximately 100 million people worldwide [10].

Mode of transmission: Unique among the human parasitic nematodes, *S. stercoralis* transforms into the rhabditiform larvae into invasive filariform larvae in the gut; this event is referred to as an auto-infectious cycle. It has an auto-infective cycle that allows infection to persist in the host indefinitely without the need for an external environment [10].

Predisposing factors: Warm moist temperature, lower socioeconomic status, and poor sanitation leading to faecal contamination of soil [10].

Effect/Impact: In immunocompromised hosts, *Strongyloides stercoralis* can cause potentially fatal hyper-infection syndrome, marked by increased infective larvae in stool and sputum, leading to gastrointestinal bleeding and respiratory distress. Disseminated infection affects various organs, particularly in individuals with immune deficiencies, malignancies, or immunosuppressive therapies. [10] In both immunocompetent and immunocompromised patients, high IgG antibody titres to Strongyloides filariform larval antigens are found, alongside specific IgA responses. Immediate hypersensitivity is prominent, but the role of IgA antibodies remains unclear. Low IgE levels may create a permissive environment for nematode proliferation. Hygiene and sanitation practices are crucial. Exercising good personal hygiene, using sanitary facilities, and not walking on bare foot. [9] The gold standard for Strongyloides diagnosis is serial stool examination, but it's insensitive, requiring up to seven tests for 100% sensitivity. Specialized methods include Baermann concentration, Horadi Mori culture, and duodenal aspirate, which is more sensitive than stool examination. Duodenal biopsy may also reveal parasites. Larvae may be visible in bronchoalveolar lavage fluid. (BAL). [11].

Many of the serologic tests that are available are quite sensitive, but cross-react with other filarial parasites, schistosomes, and *Ascaris lumbricoides*, decreasing the specificity of the tests. Furthermore, it can be difficult to distinguish between active cases and historical cases since antibodies can persist for some time. For acute and chronic strongyloidiasis, Ivermectin or Albendazole [11].

2.2 *Capillaria philippinensis*

Etiology: *Capillaria philippinensis* is endemic to the Philippines, with epidemics in Northern Luzon. It's also found in Thailand and sporadically in East and Southeast Asia. Recent cases have emerged in northern Egypt. This nematode causes human intestinal capillariasis, belonging to the genus *Capillaria* in the phylum Nematoda. Genus *Capillaria* falls under the phylum Nematoda, order Trichurida and family Trichinellidae [11].

Mode of transmission: Transmission primarily occurs through under-cooked fish consumption, posing a zoonotic risk to birds. Thick-shelled eggs pass in human stool, embryonating in 5-10 days. Larvae hatch in freshwater fish and penetrate human intestines upon ingestion. *Capillaria philippinensis* adults (males: 2.3-3.2mm; females: 2.5-4.3mm) reside in the small intestine, laying eggs with or without shells. These eggs lead to internal autoinfection, causing oviparous and larviparous cycles, potentially resulting in hyperinfection [11].

Predisposing factors: Eating undercooked or raw infected fish [11].

Effects/Impacts: Intestinal capillariasis initially manifests as abdominal/gastrointestinal disease, which can become serious if not treated because of autoinfection. A protein-losing enteropathy can develop which may result in complications such as cardiomyopathy, severe emaciation, cachexia (loss of weight and muscle loss), and death. Treatment consists of electrolyte replacement and administration of an antidiarrheal agent and mebendazole or albendazole [11].

2.3 *Hymenolepsis nana*

Etiology: *Hymenolepsis nana* also known as dwarf tapeworm is the most common human cestodes infection, with an estimated cestodes infection of 50-75 million carriers worldwide. [12]. Endemic in Asia, Africa, Southern, and Eastern Europe, helminthiasis thrives in areas with poor sanitation and hygiene, commonly

affecting children. Transmission happens through contaminated food, water, or arthropods [13].

Mode of Transmission: Humans or rodents act as definitive hosts, with arthropods (beetles and fleas) as intermediate hosts. Eggs release oncospheres in the intestine, developing into cysticercoid larvae. These mature into adult worms, laying eggs in the intestine and stool. Autoinfection occurs when eggs release oncospheres, penetrating villi and continuing the cycle internally, extending the lifespan [13].

Predisposing factors: Absence of proper sanitation, contact with environments contaminated with human faeces, inadequate treatment of excreta and waste, consumption of untreated water, presence of infected person in the household, and bathing in contaminated irrigation canals [13].

Effects/Impacts: They absorb all the nutrients in the food through the intestinal lumen and can cause death in extreme cases and usually in young children and immunocompromised. Symptoms include: vomiting, nausea, anaemia, bloody diarrhea, dizziness, extremity pain, headache, increased appetite and behavioral disturbances [13].

2.4 *Ascaris Lumbricoides*

Etiology: Ascariasis, the most common human helminthic infection globally, is prevalent in tropical and subtropical regions with poor sanitation. WHO estimated 1,450 million cases of *Ascaris lumbricoides* infection, causing around 60,000 deaths annually. Sporadic cases occur in rural, impoverished regions of developed countries. *Ascaris* derived from pigs may also infect humans [5]. The incubation period for roundworm is around 4-6 weeks, meaning it can be over a month between ingesting the roundworm and them passing eggs in their stools which may infect other animals.

Mode of transmission: Infection occurs through ingestion of soil containing embryonated eggs. Upon hatching in the stomach or duodenum, larvae penetrate intestinal walls, migrate to lungs via bloodstream, and are swallowed after development. In intestines, they mature into adult worms, with females producing about 200,000 eggs daily excreted in stool. Eggs take 30-40 days to become infective. Heavy infections in children can cause malnutrition [14].

Predisposing factors: Consumption of contaminated food and water, bathing in unsanitised environment, consumption of unwashed vegetables and fruit in regions that lack sanitation infrastructure or that use human feces for fertilizer. Children who are 3 to 8 years old are most likely to be infected because of their contact with soil while playing [14].

Effects/Impacts: Heavy infections in children lead to stunted growth via malnutrition; adults often exhibit no acute symptoms. High worm burdens may cause abdominal pain, intestinal obstruction, perforation, or complications like biliary tract occlusion, appendicitis, or nasopharyngeal expulsion, especially in immunocompromised patients [14].

2.5 *Schistosoma mansoni*

Etiology: *S. mansoni*, prevalent in Africa, South America, and parts of Asia, causes schistosomiasis. It inhabits mesenteric venous plexuses. Larvae inhabit freshwater snails, and eggs are excreted in feces, occasionally urine. Cercariae in freshwater infect humans [14].

Mode of transmission: *Schistosoma mansoni* requires two hosts to complete its life cycle: humans as definitive hosts and Biomphalaria snails as intermediate hosts. Infection occurs when humans contact water with infective cercariae. Cercariae penetrate skin, transform into schistosomula, migrate through tissues to blood vessels, then reach the portal circulation, mate, and mature in the mesenteric veins. Eggs are excreted in stool, continuing the cycle [14].

Predisposing factors: Consumption of contaminated water, contact with contaminated feces, poor sanitation, poor hygiene.

Effects/Impact: Adult *Schistosoma mansoni* parasites live in the portal vasculature, where female worms lay eggs intended to exit the host. However, many eggs lodge in the liver, causing granulomata, ulceration, and thickening of the bowel wall. Antigens released from eggs induce a Th2 response, leading to granulomatous lesions in the liver and skin rash at the site of cercarial penetration [14].

3.0 RELATIONSHIP BETWEEN COMPROMISED HOSTS AND WORM INFECTION

In persistent hepatitis B virus (HBV), infection is characterized by a weak adaptive immune response, thought to be due to inefficient CD4+ T cell priming early in the infection and subsequent development of a quantitatively and qualitatively ineffective CD8+ T cell response [15]. In HIV patients, the virus targets and destroys CD4+ T cells, weakening the immune system and allowing parasitic worms to evade host defenses. HIV attaches to CD4+ proteins but also requires chemokine receptors like CCR5 and CXCR4 for entry into other cell types, facilitating infection and disease progression [16]. Alcohol impairs gut communication, damages epithelial cells, and weakens gut barrier function, facilitating microbe leakage. It disrupts lung cilia function, impairs immune cells, and weakens lung epithelial barriers. Alcohol-related lung damage often remains unnoticed until respiratory infections worsen lung diseases beyond those in nondrinkers [17]. Marijuana impairs the immune system, making the body vulnerable to various infections and reducing cancer cell destruction. Narcotics like heroin, morphine, and fentanyl also weaken immunity. Injecting drugs elevates the risk of viral (HIV, hepatitis B or C) and bacterial/fungal infections, especially hazardous for immunocompromised individuals. [18]

4.0 LABORATORY DIAGNOSIS OF PARASITIC WORM INFECTION

Human stool containing worms may appear as pieces or live worms, while pin-worms resemble thin, white threads or may be visible around the anus at night. Diagnosis involves laboratory analysis of stool samples for worm signs or eggs, confirming infection Diagnostic tests include:

a) Fecal test: A fecal test analyzes stool for parasites, larvae, or eggs, typically through macroscopic and microscopic examination. Macroscopic inspection checks for visible signs like blood, larva segments, or mucus, while microscopic examination detects helminth eggs using a microscope and wet mount technique. Iodine visualizes egg glycogen mass and nuclei, while normal saline observes larval motility. This method is rapid and cost-effective but may yield low egg counts, uneven distribution, and sample handling challenges. [19].

b) Concentration techniques: There are two concentration techniques: flotation and sedimentation. The sedimentation method employs the Formal-ether concentration method, which is rapid and effective for a variety of fecal parasites. It combines well with the Kato-Katz method and can be preserved using sodium acetate-acetic acid-formalin or diluted formalin. The Methiolate-iodine-formaldehyde-concentration technique is highly sensitive for protozoan parasites but less so for helminths. For floatation, Zinc sulfate technique is preferred for *Trichuris trichiura* and *Giardia lamblia*, while Saturated sodium chloride floatation is economical and useful for hookworm and *Ascaris lumbricoides*. [19]

c) Culture test: The Kato-Katz method is the WHO gold standard for assessing *Strongyloides stercoralis* prevalence due to its high sensitivity, egg quantification, cost-effectiveness, and minimal infrastructure requirements. The Baermann technique and Harada-Mori technique are alternatives for detecting scant larvae. Stoll's dilution egg-counting and McMaster methods quantify helminth numbers. [19]

d) Antigen detection: The method involves the use of coproantigens captured by ELISA assay. The principle involves the use of these assays which relies on the capture of parasites excretory/secretory (E/S) proteins using rabbit anti-E/S polyclonal antibodies. [19]

e) Polymerase chain reaction technique (PCR): Microscopy techniques for intestinal parasite diagnosis require expertise, multiple stool samples, and species-specific methods for concentration and staining. Their limitations in specificity, sensitivity, and variability have prompted increased use of PCR assays, offering higher sensitivity and specificity but requiring trained personnel and infrastructure, with risks of contamination and DNA damage. [19]

f) Imaging tests: Imaging tests such as CT scans, MRIs, or X-rays can be used to detect organ injury caused by parasites. [19]

g) Tape test: A tape test involves placing clear tape around the anus. The tape can be examined under a microscope for the presence of pinworms or their eggs. Sometimes, eggs of *Enterobius vermicularis* may be seen as

evidence of pinworms around a child's anus, even with the naked eye, within the first few hours of them falling asleep. [19]

h) Colonoscopy: It's rare that a colonoscopy is used to help diagnose a parasitic infection. However, it might be performed if symptoms persisted after medications. [19]

5.0 MANAGEMENT OF WORM INFECTION

5.1 TREATMENT OF WORM INFECTION

Prescription antiparasitic medications like albendazole, ivermectin, mebendazole, triclabendazole, and praziquantel are mainstays of treatment. Dosage and duration depend on the parasite type. Severe cases may require surgery or additional medications to manage complications. [3]

5.2 PREVENTION/CONTROL OF PARASITIC WORM INFECTION

The following tips can often help prevent parasitic worm infection:

Avoid or limit consumption of raw or under-cooked meat, fish, or poultry. Avoid cross contamination during food prep by keeping meat separate from other foods. Disinfect all cutting boards, utensils, and counter tops that touched raw meat. Don't eat watercress or other freshwater plants raw. Always carry hand sanitizer. [3]

CONCLUSION

Soil-transmitted helminth (STH) infections, affecting 1.5 billion people globally, stem from poor sanitation and transmit through contaminated soil. Roundworm, whipworm, and hookworms are prevalent, causing malnutrition, anaemia, diarrhoea, and reduced fitness. Infected children suffer nutritional and physical impairment, while girls and women face increased anaemia risk. Control relies on periodic de-worming, health education, and sanitation improvements. Immediate measures like preventive chemotherapy and enhanced living conditions are essential. Immunocompromised individuals in low-resource areas remain vulnerable, necessitating accessible diagnostics and better management strategies.

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