

Intestinal helminthes: Nematodes

Treatment: Albendazole , Mebendazole for whole family

	Risk factor / Clinical presentation	Life cycle	Location	Pathogenicity	Complications/ characteristics
<i>Enterobius (Oxyuris) vermicularis</i>	Children are more often evolved than adults (families, army camp, nursery “appears during night”). presentation: Pruritus ani. Seen with naked eye	Both infective and diagnostic stages are the EGGS .	Adults worm located in the lumen of the cecum . female migrate to rectum to deposits her eggs on perianal skin	Autoinfection (contaminated fingers), Ectopic enterobiasis occurs in infected adult female when invade vulva and vagina result in valvo-vaginitis. adult worm can lodge in the lumen of appendix causing appendicitis.	Complication: inflammation 2ndry to bacterial infection of peri-anal region Diagnosis: CELLULOSE ADHESIVE TAPE , the examination should be done before defecation or bathing.
<i>Ascaris lumbricoides</i>	commonest human helminthes infection all over the world.	Infective stage: embryonated egg Diagnostic stage: unfertilized egg Cycle: fertilized -> larva “penetrate to the wall of duodenum” -> blood stream -> pulmonary circulation -> after 3 wks larva passes by coughing -> swallowed -> return to small intestine -> becomes mature fertilization takes place and eggs are in the stool.	large round worm which is normally located in the small intestine .	1-Migrating LARVA: Ascaris pneumonia. 2-Adult WORM: -malnutrition. -Can cause intussusception, intestinal ulcers Loeffler`s syndrome: Larvae in lung: pneumonia, cough, bloody sputum.	Feed on semi digested food.
<i>Trichuris trichiura</i>		Infective stage: embryonated egg Diagnostic stage: egg in stool	Adult live in large intestine		heavy infection: abdominal pain, bloody diarrhea. Rectal prolapsed in children is a common complication.

<p><i>Ancylostoma duodenale</i> & <i>Necator americanus</i></p>	<p>combination of intestinal inflammation and progressive iron-deficiency anemia and protein deficiency risk: walking barefoot through areas contaminated with fecal matter.</p>	<p>Infective stage: Filariform Larval Diagnostic stage: unfertilized egg</p> <p>Cycle: Filariform Larval finishing their journey in the small intestine where the larvae mature into adult worms. They mate inside the host, females laying up to 30,000 eggs per day The eggs need to be in soil for about one week to become FILARIFORM LARVA</p>	<p>small intestine</p>		<p>Anemia: due to withdrawal of blood by parasites and hemorrhage from punctured sites lead to severe anemia = microcytic hypochromic anemia.</p>
<p><i>Strongyloides stercoralis</i></p>	<p>Immune-compromised host.</p>	<p>The parasite shows 3 different modes of development:</p> <p>1-Direct development. 2-Indirect development: 3-AUTOINFECTION</p> <p>Internal: when the rhabditiform larva become a filariform larva in the intestine and penetrate the intestine External : fecal contamination of skin -Rh larva > filariform penetrates the skin</p> <p>Infective stage: Filariform larva diagnostic stage: rhabditiform larvae</p>		<p>Disseminated strongyloidiasis: in patient with immunodeficiency, uncontrolled diarrhea – granulomatous changes – necrosis--perforation--peritonitis--death.</p>	

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