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Prevalence of intestinal parasites in patients with abdominal discomfort in Mayo area

By

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Dedication

To my parents who used to encourage me.

To my kids who suffered a lot during this study.

To the greatest man in my life who made my world a happier place and standout as the most worthy, royal and loving my dear husband, I send you my heartfelt thanks and respect for all you have done.
Special thanks are due to my supervisor Dr. Musa M. Kheir, Associated Professor of Internal Medicine, Faculty of Medicine, University of Khartoum for his help and kind advice.

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Thanks also given to Miss. Widad A/Magsood for her great help in typing this manuscript.

Lastly I would like to thank Mr. Hassan Ali for his help of data analysis. For all I record my sincere thanks.
### Abbreviations

<table>
<thead>
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<tr>
<td>A. Doudenale</td>
<td>Ancylostioma duodenale</td>
</tr>
<tr>
<td>A. Lumbricoides</td>
<td>Ascaris humbricoides</td>
</tr>
<tr>
<td>CAA</td>
<td>Circulating Anodic Antigen</td>
</tr>
<tr>
<td>CCA</td>
<td>Circulating Cathodic Antigen</td>
</tr>
<tr>
<td>E. coli</td>
<td>Entamoeba coli</td>
</tr>
<tr>
<td>E. Histolytica</td>
<td>Entamoeba histolytica</td>
</tr>
<tr>
<td>G. Iambia</td>
<td>Giardia Iambia</td>
</tr>
<tr>
<td>H. nana</td>
<td>Hymenolepis nana</td>
</tr>
<tr>
<td>N. Americans</td>
<td>Nector americanus</td>
</tr>
<tr>
<td>S. haematobium</td>
<td>Schistosoma haematobium</td>
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<tr>
<td>S. mansoni</td>
<td>Schistosoma mansoni</td>
</tr>
<tr>
<td>S. Japonicum</td>
<td>Schistosoma japonicum</td>
</tr>
<tr>
<td>S. Stercolaris</td>
<td>Strongyloides stercolaris</td>
</tr>
<tr>
<td>SPSS</td>
<td>Statistical Package for Social Sciences</td>
</tr>
<tr>
<td>T. saginata</td>
<td>Taenia saginata</td>
</tr>
<tr>
<td>T. solium</td>
<td>Taenia solium</td>
</tr>
<tr>
<td>TH1</td>
<td>T- cell helper 1</td>
</tr>
<tr>
<td>Th2</td>
<td>T- cell helper 1</td>
</tr>
<tr>
<td>INF-y</td>
<td>Interferon-Gamma</td>
</tr>
<tr>
<td>HLA</td>
<td>Histocompatibility antigen</td>
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<tr>
<td>CNS</td>
<td>Central nervous system</td>
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### Abstract

...
Faecal samples from 100 individuals attending Mayo Hospital were examined for intestinal parasitic infections. The finding revealed an overall prevalence rate of intestinal parasitic infection of 74%. The commonest intestinal parasite in Mayo Giardia lambia, its prevalence rate was (71.6%). The prevalence rate of other parasites were as follows:

- E. histlytica (25.6%),
- S. mansoni (22.9%),
- Taenia saginata (4.1%),
- H. Nana (4.1%),
- E. coli (2.7%),
- Entrobious vermicularis (1.3%).

Infection by a single parasite was found in 62.3%, while mixed parasites were found 6.5%. The most vulnerable age group is between 15-40 years, males predominated the females and parasitic infection is more common among poor, illiterate patients.
انتشار وعوامل م timeZone، جنوب.

أكتوبر بين الفترات في مادة مناطق اختيرت 2001 ينائر حتي 2002 تم خلالها جموعهم تم براز عينة فحص 10 حضر شخص من الصباحية الفترات أثناه خارجية للريع.

診断 84% 1 و سدى لحافرك! حقض_ad تهمة

اشتراع 81.6 وقفة الآن جزء من كل هذا التكامل تم ذاتيا (25.8) ناهيا وقفاء! لاحظ! أوف أي ذكر في 55% 41) توقف جزء منه (41) توقف فرد يسا (2.9) داي دئه داي 

(1.3) هي التي تدريجيا (2.7) 1 وسدى وقفاء! لاحظ!

إلى نقطة الدوام الذي يخرجونها وقودة! نوع الاستخدام لفترة سريعة البصق 84% البعدياً بآ

. تقديرات كفاءة وسعي الدفاعي! وغلق! لاحظ! داي ذكر! داي ذكر!
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**APPENDIX (Questionnaire)**
The term parasitic is used to determine infection with protozoa and helminths (metazoa). The former (protozoa) is unicellular microscopic in size and develops within the host, small number is needed to produce disease. But the metazoa (helminth) is macroscopic, multicellular organism, that doesn't multiply within the host, the disease needs repeated exposure to infective organisms, so it is common in endemic areas.

The parasitic diseases are endemic in developing countries especially the less developed countries forming a major health problem.

Intestinal parasites are among the most common human infections and have been associated with widespread morbidity and malnutrition. One of the most important recent changes in the epidemiology of prolosgoal disease is the association of selective infections with HIV, and more recently the molecular and cellular biology have revolutionized the molecular parasitology.
Prevalence of intestinal parasites:

Intestinal parasites are distributed worldwide with high rate in many regions. The global prevalence showed considerable variation in distribution. The seasonal variation was due to climatic and geographical factors.\(^{(1,2)}\)

Pathogenesis of intestinal parasitic infection:

Host related factors: environmental factors has important role in transmission of parasites, e.g. individuals who live in houses without proper water supply are more susceptible to water borne parasite as *E. histolytica* and *Giardiasis* and defecation habit in the same damp wet places was important for hookworm and strongyloidiasis.\(^{(3,4)}\) Also poor facilities of food handling is important in faecal oral transmission of diseases.\(^{(5)}\)

Low socioeconomic status may produce structural changes in the small intestine, this was found to affect absorptive capacity and immune status of the host and may increase susceptibility to infection, e.g. nutritional deficiency of iron if associated with hookworm infection will result in anemia and strongyloid infection result in hypoalbuminaemia. *Diphylobothrium latum* causes
megaloblastic anemia due to parasite host competition for food containing cyanocobalamin.\textsuperscript{(6,7)} The co-existence of other diseases also influences the outcome of intestinal parasitic infection, so bacillary dysentery will help transformation of \textit{Entamoeba dispar} to pathogenic. Megacolon facilitates strogyloloides larvae to repenetrate the colon mucosa. The presence of an immunosuppressive disease like malignancy facilitate the auto-infection of strongyloides.\textsuperscript{(8)}

Immunity has important role in defense against intestinal parasites; immundeficiency resulting from malignancy or chemotherapy e.g. steroid has influenced the pathogenicity of strogyloloides (severe disseminated strogylolidosis, so it is important to screen for presence of strongyloides before starting treatment. In schistosomiasis T. cell mediated immunity is important for producing granuloma, which is pathology of schistosomiasis.\textsuperscript{(9)} In cryptosporidium, microsporidum infection and isospora belli, diarrhoea is established in patients with human immuno-deficiency syndrome.\textsuperscript{(10)}
Parasitic factors:

Density of the parasite is the cornerstone in helminthic infection. Being unable to replicate within the host, so increased density will result in severe disease with complication, more in lighter infection, so rectal prolapse was related to heavy *Trichuris trichuria* and intestinal obstruction in heavy *ascariasis*.

**Virulence of organisms and adaptation to human host:**

Human is a definitive and intermediate host, but the disease may depend mainly on pathogenicity of parasite, sometimes more pathogenic than other and when the human is the intermediate host, the infections are mild and transient. (11)

**Concomitant infection:** It was observed that mixed infection with parasitic disease will help spread of the parasite as in *E. histolytica*. (11)

**Response to modified host:** If human becomes debilitated or immunosuppressed, autoinfection by parasites such as strongyloidis stercolaris and *E. histolytica* may result in severe disease.

**Climatic and seasonal factors:** It was observed that in Europe transmission of ascaris was interrupted by low
temperature in winter. In Saudi Arabia it was limited to rainy seasons, in tropical areas where high temperature and increased rainfall permit continuous transmission all the year round.

Intestinal helminthes include *Cestode* infection (*Diphyllobothrium latum*, *H. Nana*, *Taeniasis*) and trematods infection the most common human parasites of which are (schistosomaisis and fasilopiasis and lastly the intestinal nematodes), which include *Ascaris lumbricoides*, *Hookworms*, *Entrobious vermicularis* and *Trichuris trichuria*. They are prevalent in temperate and tropical areas of the world especially those with overcrowding and poor sanitation and they cause little morbidity.
Types of intestinal parasites

Schistosomiasis:

Schistosomiasis is endemic in 74 countries and affects >200 million persons worldwide. The disease is ranked second to malaria in term of socioeconomic and public health importance in many tropical and subtropical areas. The infection has been demonstrated in the mummies of ancient Egypt and the etiology was known in 1851 by Bilharz who discovered the adult worm in mesenteric veins of an Egyptian.\(^{(12)}\) The circumstantial evidence indicates that *S. mansoni* might has been introduced to Sudan as early as 2600 BC by Egyptian soldiers and farmers when Northern Sudan especially Karma was under Egyptian occupation.\(^{(13)}\)

Schistosomiasis is one of the most important parasitic diseases (helminthes) of the human that caused by blood flukes (trematode) of the genus *schistosoma* of which three types are primary parasites of man:

1- Schistosoma haematobium, which is found in the Nile Delta, North to Cairo and South Africa, Subsahara from Mali to Ethiopia, belt South Mozambique. Also in
Madagascar and Angola, Brazil, Venezuela, Puerto-Rica, Antigua.\textsuperscript{(2)}

2- \textit{Schistosoma mansoni} is found in Middle-east and Africa.

3- \textit{Schistosoma japonicum} is found in Far-east.

In Sudan schistosomaisis both \textit{S. haematobium} and \textit{S. mansoni} are widely spread throughout the country with \textit{S. mansoni} showing the highest endemicity in Geriza Irrigated Areas.\textsuperscript{(14)}

In Sudan prevalence of infection with schistomiasis and of periportal fibrosis in areas not covered by controlled programm reached about 70\%, 18\% receptively and more than 50\% of those with periportal fibrosis have oesophageal varices and 3-4\% have hematemesis.\textsuperscript{(15)}

In endemic areas schistosma infection is acquired in childhood and the infection increases in prevalence and intensity with increasing age, but in adults obvious decline in intensity and not in prevalence has been observed.\textsuperscript{(8,12)}

\textbf{Transmission:}

There are many important factors in transmission of the disease, these include:

\begin{itemize}
  \item[a-] Suitable snails, \textit{S. haematobium} requires the
snail *Bulinus truncalius*, *S. mansoni* requires one form of the *Biomphalaria* genus.

b- Suitable environment for the snail.

c- Contamination of water by human excreta.

d- Man-water contact.

**Pathogenesis and pathology:**

The lesions in schistosomiasis are caused by schistosoma eggs, the adult worm causes little or no pathology, most of the eggs are laid in the lumen of the gut, the rest are laid in walls of organs or embolized into portal radicals or lung arterioles, collateral, which allow egg deposition in different organs in the body. Soluble egg antigen originated from secretory glands of miracidia endoresed within eggs diffuse out the eggshell and induces hypersensitivity response. The immunopathology of schistosomiasis is considered to be due to granuloma formation around eggs deposited in the tissue and is a manifestation of delayed hypersensitivity reaction through a T-cell mediated immune response. The granuloma is described to be composed of schistosoma eggs surrounded by cellular aggregates of oesinophils, mononuclear
phagocytes, lymphocytes, neutrophils, plasma cells and fibroblasts.

Fibroblasts observed to appear early and throughout the lengthy involution process, replace other cells. The pathology of schistosomiasis resulted from collection of granulomata and fibroblast obstructing vessels and fibro-inflammatory swelling which is containing millions of eggs. Von Lichtenberg compared the pathogenic variables and prognosis in hepatosplenomegaly of early infection. The cell proliferation, granulomas, reticuloendothelial hyperplasia and different inflammatory infiltrate all are reversible by specific chemotherapy when compared with the hepatosplenomegaly. These are induced by fibro-vascular pathology with periportal fibrosis, portal hypertension an associated effect on the spleen. The prognosis is far less dependent upon chemotherapy, but more upon surgical alleviation of mechanical obstructive condition. A range of chronic lesions of *S. mansoni* are found from scattered granulomas of intestinal tract to gross periportal hepatic fibrosis described as bilharzial clay pipe-stem fibrosis.
The disease passes in four stages:

**Stage I:** this is the stage of invasive itchy nodules (swimmer dermatitis).

**Stage II:** stage of migration of schistosomules resulting in Katayama like syndrome of acute febrile reaction with eosinophilia, fever, abdominal pain, cough, dyspnoea and hepatosplenomegaly, but no ova in stool.\(^{(15)}\)

**Stage III:** Established infection in which there is heavy egg production by parasite.

**Stage IV:** This is the stage of complications and fibrosis. Here the eggs are taken to the liver in extremely large amount, they block up portal veins, and the reaction to them causes dense fibrosis to take place known as Symmer’s fibrosis.\(^{(4)}\) There is hepatosplenomegaly and pulmonary hypertension due to granulomatous pulmonary arteritis and schistosomal cor-pulmonale might occur with *S. mansoni* infection. Nephropathy and glomerulonephritis consisting of deposition of immunocomplexes of host immunoglobulins with adults worm or egg antigen in the glomerular parenchyma and basement membrane also occurs in *S. mansoni* infection.\(^{(15)}\)
Proteinuria was common particularly in endemic areas, but renal failure occurs in small proportion. Amyloidosis which indicates impaired immunity was found in renal biopsy in patients with nephrotic syndrome and schistosomiasis in Egypt.\(^{(13)}\)

Neurological schistosomiasis may be due to \(S.\) \textit{mansoni}, but more common in \(S.\) \textit{japonicum}. Central nervous system (CNS) granuloma causes epilepsy and spinal cord granuloma may cause cord compression. Other ectopic lesions such as cutaneous lesions due to \(S.\) \textit{mansoni} in form of papular or nodular lesion are known but rare.

In the intestine egg deposition leads to fibrosis, polyps, ulceration and bleeding. If there is portal hypertension eggs may pass into systemic circulation to various organs and cause certain complications, in the lungs they cause lung fibrosis, pulmonary hypertension and cor-pulmonale. This is commonly caused by \(S.\) \textit{mansoni} and is found in Egypt and South America, but is not common in Sudan.\(^{(13)}\)

The pathological effects are proved to be related to the infecting dose of eggs which is related to the other clinical and biochemical changes.\(^{(13)}\) Other complications of
schistosomiasis include anaemia and infection with certain gram negative organisms such as *Salmonella* and *E. coli*. *Salmonella* infection is prolonged causing schistos-salmonellosis.

**Immunity:**

Antibodies against antigens released from eggs, cercaria and adult worms provide partial protection.

Immunoglobulins formed in response to the antigen are IgG, IgM, IgA and they are responsible for the splenomegaly.

**TH1, TH2 INF-γ** play an important role in resistance to reinfection in addition to the role of genetic factors (associated with HLA class II allele CDQBO201) and lastly concomitant infection with other intestinal parasites seems to reduce the risk of severity.

**Clinical presentation:**

Schistosomiasis can present with one of the following phases:

a- Dermatitis in previously exposed patients who present with purpuric, papular rash occurs in both *S. haematobium* and *S. mansoni*. 
b- Acute schistosomiasis Katayama like syndrome, characteristically in _S. japonicum_ but is seen in _S. mansoni_ and _S. haematobium_. It occurs in previously non-exposed patients e.g. Europeans. There are chills, sweat, abdominal pain, headache, cough, transient hepatosplenomegaly, lymphadenopathy, leucocytosis and eosinophilia.

c- Chronic schistosomiasis: incase of _S. haematobium_ patients present with fatigue, low grade fever, terminal haematuria, dull hypogastric pain, dysuria, urgency, frequency hydronephrosis, contracted calcified bladder, nephrotic syndrome, cor-pulmonale due to pulmonary hypertension as result of deposition of the ova in the pulmonary arterioles, this is not common in Sudan, but more in Egypt and South Africa. Hepatosplenomegaly, acute appendicitis, genital tract affection, pathological fractures, CNS granuloma causing epilepsy, paraplegia and transverse myelitis, may be encountered.

In chronic _S. mansoni_, the stage of invasion, toxaemia and hypersensitivity the same as in _S. haematobium_. In the
stage of acute intestinal disease there are dysenteric symptoms. In established disease (chronic intestinal form) widespread involvement of intestine by granulomata, fibrosis and ulceration occurs. The patient presents with abdominal pain, fatigue, polyps, bilharzioma and hepatosplenomegaly and portal hypertension.

Other complications are colonic perforation genitalia and amyloidosis which indicate immunity derangement.\textsuperscript{(3,15)}

**Diagnosis:**

History of contact with contaminated water and clinical manifestation are important steps in establishing the diagnosis. Eosinophilia is indicator for further investigations as well as hepatosplenomegaly.

The definitive diagnosis of schistosomiasis is by finding spined schistosome eggs in faeces or urine by concentration method is optimal in the detection of light infections. The cellophane thick faecal smear, the kato technique and its modification became standard diagnostic tool in both clinical and epidemiological studies. Biopsy usually from rectum for demonstration of ova. Hatching test is important to see the viability of ova. Sigmoidoscopy will
show rectal polyps, endoscopy for detection of any varices, liver biopsy for detection of periportal fibrosis.

Serological tests for detection of IgA, IgG, IgM antibodies against schistosoma antigen during acute phase is used. A new simple computer assisted method is proposed based on analysis of distribution of sensory endings (sensillae) on body of cercariae revealed by impregnation with silver nitrate e.g. in *S. mansoni* species is multipolar in contrast to distribution of sensillae in *S. intercalcaum* species, which appears more uniform. Several methods had been used for detection of two schistosom adult worm circulating antigens, Circulating anodic antigen (CAA) and circulating cathodic antigen (CCA) detected by ELISA. These two glycoprotein circulating antigen are associated with the gut of adult worm, they are well characterized and genus specific and their presence indicate active infection, they have virtually 100% specificity and very high sensitivity, being expensive they are used at research level or individual patients or in small groups.

Other serological tests like radio immunoassay (RIA), indirect immunofluorescence test (IFAT), latex agglutination
test (LAT) and circumoval precipitation test (COR) are helpful in diagnosis.\textsuperscript{(20)}

Rectal biopsy is employed if faecal examination was negative. U/S is superior to physical examination in measuring liver and splenic size and is the best technique for grading schistosomal periportal fibrosis and portal hypertension.\textsuperscript{(21)}

**Treatment:**

Chemotherapy is important for cure and control of schistosomiasis. Three compounds are currently in use, metrofonate, oxamnique and praziquantel and all these drugs are recommended on WHO's list of essential drugs.

i- Praziquantel, Apyrazinoisoquinoline derivative, is a broad spectrum antischistosomes, with high efficacy and lack of serious side effects, single dose and cheap. It is taken orally and the standard dose is 40mg/kg body weight for *S. mansoni* and *S. haematobium*, and 60mg/kg for *S. japonicum*. The cure rate is 75-85\% for *S. haematobium* 63-85\% for *S. mansoni* and 80-90\% for *S. japonicum*.\textsuperscript{(22)} The most common side effects are mainly related to the GIT (abdominal pain, discomfort, nausea, vomiting
anorexia and diarrhoea). Dizziness, headache, drowsiness, pruritis and skin eruption, fever and fatigue. It is contraindicated during pregnancy.

ii- Oxamniquine: this is a tetrahydroquinoline derivative. It is effective against *S. mansoni* infection only. It is taken orally as 15-60mg/kg body weight in two or 3 divided doses. In Sudan, 60mg/kg is given while in South America only 15mg/kg is the dose.\(^6\) Side effects include change in the colour of the urine, dizziness, convulsions and transient increase in SGPT, SGOT fever. It is contraindicated during pregnancy.

iii- Metrofonate: this is an organophosphorus cholinesterase inhibitor, it acts only against *S. haematobium*. It is given orally in doses of 10mg/kg/body weight in three divided doses, two weeks apart. Cure rate is 80%. Side effects are nausea, abdominal pain, fainting, fatigue, sweating and bronchospasm.

Surgical treatment for portal hypertension in patients with hepatosplenic schistosomiasis includes spleenectomy, this results in reduction of venous portal blood flow to the liver, with increase in arterial hepatic blood flow.\(^{23}\)
Treatment of schisto-salmonellosis infection associated with other bacteria, although clinical response to antibiotic is good, bacteraemia would recur unless the underlying schistosomiasis is treated.

**Prevention and control:**

Active immunization against schistosomiasis is still under trials. Vaccine was observed to induce partial immunity, which probably became completely ineffective.\(^{(25,26)}\) Many programs to control schistosomiasis were done in endemic areas including Sudan. Important prevention measures are health education, sanitary disposal of faeces and urine, reduction of snail population (biologically),\(^{(26)}\) safe clean water for drinking and bathing, avoidance of swimming in fresh water in endemic countries and treatment of infected people help to reduce infectivity and morbidity.

**Giardiasis:**

This is an infection of upper small bowel by the Flagellate protozoa *Giardia lambia* and it is the most common human protozoan pathogen worldwide and causes both epidemic and endemic diarrhoeal disease. It is often
pathogenic in individuals who have no previous exposure to infection.\textsuperscript{(27)} Often is carried asymptomatic in countries where the infection is common and if symptomatic it is more in children.\textsuperscript{(28,29)}

In Sudan giardia intestinalis is commonly present in stools of adult patients who present with gastrointestinal symptoms at outpatient clinic.\textsuperscript{(30)}

\textit{Giardia lambia} has a simple life cycle alternating between trophozoite and cyst stage.\textsuperscript{(31)}

The trophozoite is a pear shaped flagellate (10-20\(\mu\)m \(\times\) 5-15 wide \(\times\) 2-4\(\mu\)m thick) and contains four nuclei resembles a face microscopy.\textsuperscript{(32)} It has a sucking disc by which it attaches itself to the mucosa of the intestine, has 4 pairs of flagellae and multiply by binary fission it is detected in liquid stool of symptomatic patients when drops off from the attaching side they usually encyst in the ileum. The cyst is oval in shape and has 4 nuclei, which is identified in the stool of asymptomatic carriers as well as liquid stool of symptomatic patients. The cyst survives in water for weeks or even months and resists to chlorine concentration (0.4mg/l) routinely used in community purification.
system.\textsuperscript{(32,34)} The cysts are infectious as soon as they hatch and encyst in upper gastrointestinal tract and liberate trophozoites. Man is the only source of infection and \textit{Giardia lambia} spreads by faecal oral route through contaminated food and water. The latter is thought to be responsible for high incidence of giardiasis in travelers returning from endemic areas. Direct person to person spread occurs as a result of sexual contact.\textsuperscript{(33,34)}

\textbf{Epidemiology:}

\textit{Giardia lambia} has a worldwide distribution. It is more common in the tropics and subtropic but countries of Eastern Europe Leningrad, USSR are endemic areas.\textsuperscript{(31)} All age groups are susceptible to infection but in developing Countries infection is usually universal by the age of 5 years and it is recurrent, so the primary immunity is not completely protective. In developed countries the disease is sporadic and more in children and homosexuals (oro-anal sexual behavior) campers, hiker and international travelers, and the overall prevalence in industrialized world lies between 2-5\%, nevertheless although within these low prevalence areas, there might be localized regions of high
prevalence. The susceptibility to infection is increased by achlorhydria, chronic pancreatitis, immune deficiency state, malnutrition, hypogammaglobulinaemia and it is more common in patients with AIDS.

**Pathogenesis and pathology:**

*Giardia lambia* is strictly small bowel noninvasive pathogen, when swallowed the cyst release the trophozoite in alkaline condition of the upper small intestine. The Histopathological effects of *Giardia lambia* on small intestine depend on shape of the clinical presentation.

In asymptomatic patients no abnormality may be identified by light microscopy, but electronmicroscopy will show microvillae changes. Symptomatic patients may reveal villous atrophy and crypt hyperplasia and plasma cell infiltration. The mechanism by which *Giardia lambia* results in diarrhoea is multi factorial.

1. Disaccharidase deficiency reduced B12 absorption.

2. Mechanical blockage of intestinal mucosa by large number of trophozoites which lead to malabsorption of fats and xylose.

3. Altered jejunal motility with or without bacterial growth.
4- Villous atrophy.

**Immunity:**

There is increased evidence to suggest that immunological factors are important in protection against reinfection, IgG in endemic areas should be detected in >80% of symptomatic cases. Studies in India and Gambia indicated that only 30% of patients with infection had detectable anti-giardia IgA which is likely to be the most important aspect of immune response for giardia clearance from the gut. Cellular immune response T. helper (CD4) were thought to be important and there had been no detailed studies on lymphocytes phenotype in human giardiasis.\(^{35,36,37}\)

**Clinical picture:**

*Giardia lambia* can presents in one of the following clinical forms:

1- Asymptotic carrier state up to 50% of infection.

2- Acute self-limiting diarrhoeal disease.

3- Chronic diarrhea associated with malabsorption.

4- Growth retardation in developing countries.

40-50% of infected patients have diarrhea.
Symptomatic patients develop, anorexia, nausea associated with explosive, watery, foul-smelling diarrhoea with passage of gases. Low-grade fever may be present.

Alternatively more indolent, non-dehydrating, intermittent diarrhoea persists. This may lead to malabsorption with weight loss. 20-40% had lactose intolerance. Rarely *Giardia lamblia* may be associated with urticaria cholecystitis, pancreatitis, arthritis, retinal arteritis and iridocyclitis.

**Diagnosis:**

Stool examination by fresh stool analysis (3 sample within 10 days or by concentration technique using formaline-ethyl acetate or zinc-sulphate,\(^{(37)}\) looking for cyst in formed stool or trophozoite form unformed stools,\(^{(34)}\) if stool analysis was negative other methods can be used:

a) Direct Immunoflourescence Assay, positive in 90%.

b) ELISA with sensitivity and specificity >95%.

c) Enterotest (string test), jejunal aspirate by string test, biopsy of the bowel by Crosby capsule.

d) Gel diffusion (serological tests are not helpful in endemic areas).
e) Endoscopy.

**Treatment:**

Treatment of asymptotic people is controversial because of rapid reinfection. Administration of antigiardial drugs decreases the severity of symptoms and duration of illness.\(^{(38)}\)

Giardiasis can be treated satisfactorily with the following drugs with more than 90% efficacy by nitroimidazoles which include:

1- Metronidazole, dose 15 mg/kg b.d for 10 days.

2- Tinidazole, dose 50 -75 mg/kg in a single dose.

3- Quinacrine (mepacrine), 2 mg/kg, 3 times per days for 5-7 days.

4- Furazolidone (80% effectiveness), 2mg/kg, 3 times daily, 7-10 days.

**Prevention:**

i- Public health measures are important to ensure availability of clean water supply.

ii- Health education to promote excellent personal
hygiene to interrupt the infection cycle.

iii- Boiling of water for one minute or 0.5ml of 2% tincture of iodine in 1 litre for at least 60 minutes before drinking renders parasite non-infective.

The antigenic variability of *G. lambia* combined with the presently ill-defined correlates of protective immunity greatly hinder vaccine development for this infection, where active immunization in the form of vaccine was feasible or even approachable continue to be evaluated.⁽³⁹⁾
Amoebiasis:

**Definition:**

According to the WHO definition, it is a condition in which the organism *Entamoeba histolytica* (E.H) is harboured by some one regardless of the symptoms.\(^{30}\) It is an infection of the large intestine produced by *E. histolytica*. It is an asymptomatic carrier states in most individuals but can produce spectrum of diseases ranging from chronic, mild to fulminate dysentery. *E. histolytica* is a worldwide disease affecting 480 million, the majority are asymptomatic but 10% develop amoebiasis of the colon and less frequently in the liver. It is of worldwide distribution\(^ {40}\) and highest in developing countries, but the prevalence in certain areas e.g. Egypt may reach 80%.\(^ {30}\)

**Biology and epidemiology:**

Infection results from acid resistant cyst in faecally contaminated water or food. Release of cysts occurs in small bowel leading to colonization of the colon with trophozoites.
Transmission also occurs from direct faecal oral contact because of poor hygiene and oral sexual practice is another way transmission.

Epidemiological and biological studies indicate that there are two species, the pathogenic *E. histolytica* and *E. dispar* infection with the latter is more common but does not result in invasive disease or humoral response and 10% only present with invasive amoebiasis. In favourable condition virulence, dose of organism, host resistance, nutritional status and genetic make up of the host are all important in determining the degree of infection. High incidence of invasive amoebiasis occurs include parts of South-East Asia and Mexico.\(^{(40)}\) Damage to intestinal mucosa by any process e.g. Bacillary dysentery, malaria, schistosomiasis all activate the organisms.\(^{(41)}\)

The high risk groups in developed countries are homosexual, the institutionalized mentally abnormal population, travelers and immigrant, young age pregnant women, malnutrition and patients using steroids.
**Pathogenesis and host immunity:**

*E. histolytica* trophozoites (adult amoeba) causes disease by adhering to colonic mucins disrupting mucosal barriers with proteolytic enzymes and contact dependent lysis of host. The trophozoites adherence to colonic mucin is mediated by a galactose-binding surface lectin, attachment by this lectin is the first step in the amoeba lysis of human cells.

Infection with *E. dispers* usually clears within 8-12 months, but cure with invasive amoebiasis is associated with resistance to recurrent disease, and immunity for a year or more to asymptomatic intestinal infection.

A protective immunity is mediated by development of a serum antibody and amoebicidal cell mediated immune response with lymphokine activated macrophages and CD8. Acute amoebiasis is associated with occurrence of antigen specific suppression of cell mediated response to *E. histolytica* the facilitating parasite survival in tissue.
mucosal secretory IgA antiamoebic antibody response that develop after *E. histolytica* infection has a protective role against recurrent infection.\(^{(31)}\)

**Clinical presentation:**

The patient presents with clinical disease syndrome which may be intestinal or extraintestinal. In intestinal disease the majority of cases *E. histolytica* are symptomatic cyst passer or patients may present with acute recto colitis (dysentery), characterized by the sub-acute onset of bloody diarrhoea over days, abdominal tenderness, weight loss, fever occurs in only one third, fulminate colitis with perforation is uncommon, patients are in toxic state, acutely ill and have rigid tender abdomen. Toxic megacolon is an unusual complication associated with use of steroids when amoebic colitis is mistaken for inflammatory bowel disease. Chronic nondysenteric amoebic colitis usually manifests with years of intermittent bloody diarrhoea, the syndrome is symptomatically indistinguishable from ulcerative colitis.

Amoeboma is a rare segmental form of chronic non dysenteric colitis, commonly found in the caecum and ascending colon may present with abdominal pain and is
commonly confused with colonic carcinoma.

Extra-intestinal disease consists mainly of amoebic liver abscess which occurs up to 5 month after onset of intestinal infection presentation and may be acute with high grade fever, marked upper abdominal pain and weight loss of less than one third has diarrhoea, the abscess may extend to pericardium, peritonium, empyema disseminated to lung and brain

Amoebic dysentery may be complicated by death from exhusion, haemorrhge, perforation, liver abscess, peritonitis or intussusception especially in children. Ulcerative colitis is rare, colonic stricture and skin ulceration may be extensive around the anal region. In Papua, New Guinea, Ano-genital amoebiasis is the commonest form of infection seen in the laboratory and it may resemble squamous cell carcinoma. Rarely complication may include balanitis with granuloma or involvement of prostate abscess formation in spleen, psoas muscle, buttocks or thigh. Brain abscesses may occur in presence or absense of liver abscess and emypema due to solely *E. histolytica* has been reported.\(^{(34)}\)

**Diagnosis:**
Stool examination should be in minimum of 3 specimens and fresh stool analysis should be done within 30 minutes.\(^{(37)}\)

The diagnosis is confirmed by microscopic findings of active trophozoites containing RBCs and this should be differentiated carefully from macrophages and polymorph.

Sigmoidoscopy is used when stool is negative, for presence of ulcer and trophozoite from a scraping.

ELISA used for diagnosis can replace microscopy for survey purposes.\(^{(42)}\)

**Treatment:** metronidazole 400mg three times a day for 10 days. This will sterilize abscess but can not be guaranteed to eradicate the bowel infection. Furamide 0.5 g three times a day for 10 days irraddicate the bowel infection. Chloroquine is effective in invasive amoebiasis, but less effective in the treatment of organism in the lumen.\(^{(43)}\)

**Prevention:** The high standard of personal hygiene, and environmental sanitation are the main means of prevention. In endemic areas avoid hazardous foods and drinking only boiled water or bottled drinks thorough wash of salad vegetables or sterilized in sodium hypochloride solution.
**Others protozoan:** They are protozoan diseases that become more important after emergence of AIDS.

### i. Cryptosporidiosis:

*Epidemiology and pathogenesis:* It is an important cause of infectious diarrhoea in human, caused by oocidian protozoan, cryptosporidium spp. The organisms are widely prevalent in animals as well as poultry, birds and reptiles and are present as oocysts in their faeces. Human infection occurs worldwide and community studies in African countries had shown prevalence rate between 6% and 12% in patients with diarrhoea.\(^{(44)}\) In developed world cryptosporidium was found in an outbreak in Denmark in 1990. The proportion of asymptomatic carriers was higher in areas of low socioeconomic condition in developing countries than developed region.\(^{(45)}\)

Transmission is either direct faecal-oral person to person or animal to person) or water borne faecally contaminated natural water supply, this is an important source of outbreaks or travelers diarrhoea. The organisms can survive routine chlorination.
**Immunity:** IFN-gamma has a protective immunity against Cryptosporidium parvum but the mechanism by which IFN-gamma leads to protective immunity is poorly understood. It was observed that resolution of infection is dependent on the expression of TH1 cytokines in the mucosa of mice and that INF-gamma may participate in the control of parasite.\(^{(46)}\)

**Clinical features:** Infection can be asymptomatic. Acute onset of watery diarrhoea and abdominal discomfort, fever and vomiting may occur, it is self limited in immunocompetent persons and usually subsides within two weeks. In immuno-compromised especially patient with AIDS the disease is serious with persistent profuse watery diarrhoea lasting for weeks and sometimes for months. Dehydration and malnutrition occur commonly leading to death. Here the infection involves the whole length of the intestine from stomach to rectum.

**Diagnosis** is by faecal smear will show acid fast oocyst using either ziehl-nielson or safranin-methylene blue stain.

**Treatment** in immuno-competent the disease is self limiting, no effective therapy for protracted, often fatal
diarrhoea in immuno-compromized, but azithromycin or parvomyocin may reduce the parasite load and lessen the magnitude of diarrhoea.\(^{(33,45,47)}\)

**ii- Microsporidiosis:**

*Epidemiology and pathogenesis:* it is a worldwide disease and intracellular protozon. They have emerged as important cause of chronic disabling diarrhoea in severely immunocompromised specially in AIDS patients among the genera microspordia, enterocytozoon bieneusi, Encephalitozoon hellem, nosema corneum and septata intestinalis account for most human infection and little is known about the mode of transmission.

*Clinical picture:* infection in non HIV patients is quite rare and mostly involves the eye and CNS. In AIDS patients persistent chronic watery diarrhoea is often associated with crampy abdominal pains, nausea, vomiting, malabsorption and wasting invariably follows. It is diagnosed by electronmicroscopic demonstration of parasites in small intestinal mucosal biopsy specimens. Stool analysis for detection of spore by atrichrome staining method.

*Treatment:* is uncertain but albendazole 400 mg b.d
suppressive therapy may be necessary in AIDS patients.

**iii- Isospora belli:**

It is worldwide distributed, most prevalent in Latin America and Africa. It produces self-limited diarrhoea in immuno-competent, but causes persistent severe diarrhoea in AIDS.

It is diagnosed usually by stool examination and treated with trimethoprim 160mg plus sulfamethoxazole 800 mg q.i.d for 10 days, then b.d. for 3 weeks, suppressive therapy may be necessary in person with AIDS.

**iv- Cyclospora:**

It is worldwide distribution. Diarrhoea has been reported in residence and travelers to many areas of the world including Europe, America, Nepal, South-East Asia. It produces severe watery diarrhoea with flatulence, bloating, dyspepsia and fatigue, lasting for weeks and prolonged disease in AIDS patients.

Diagnosis by detection of cyclospora oocytes in faceses by microscopy (acid-fast-round bodies) and jejunal aspirate and electronmicroscopy also may reveal them.

**Ascariasis:**
Ascaris lumbricoides, the most common human intestinal nematode, is the causative agent of ascariasis with an estimated worldwide prevalence of over one billion people, especially in moist tropical and subtropical regions, but also in cooler climates. Infection was estimated to be 47% or 531 millions persons were infected. Although characterized with low morbidity and mortality rates, the global prevalence of ascariasis still results in approximately 20,000 deaths annually primarily as a consequence of intestinal obstruction.\(^{(48)}\) In humans, transmission is usually by hand to mouth route by way of contaminated agricultural products and food, or from dirty hands, by eggs which remain in adverse environmental conditions which allows persistent infection.\(^{(49)}\) Three phases of ascariasis may be present, namely, the pulmonary, intestinal, and the complication stages. Although generally asymptomatic heavy infestation may cause serious pulmonary disease caused by the habitual migration of larvae through the lung, liver causing their damage.\(^{(50)}\) In transient pulmonary infiltrates, fever, cough, dyspnoea and eosinophilia (Loffler's syndrome) lasting one to several weeks after clinical manifestation.
Partial or complete obstruction of biliary and intestinal tracts, serious complications as convulsions, meningism, epilepsy small tumour (granuloma in the eyes retina or brain).\(^{(50)}\) Malnutrition, malabsorption syndrome characterized by steatorehoea and low vitamin A level has been reported in Latin American children with ascariasis. Peritonitis either pyogenic or granulomatous was recognized.

It is diagnosed by direct smear, rarely by concentration technique because it lays about 200,000 egg per day. Serological test was of little value due to cross-reaction with other helminthic antigen.\(^{(50)}\) Ultrasonographic examination will show specific signs (strip signs, four lines inner tube or double tubes or target sign of worm masses or spaghetti like or zigzag sign can be present in any form of abdominal ascariasis.\(^{(51,52)}\)

Treatment with mebendazole, albendazole and pyrantel pamoate are the most widely used agents.

**Anclystoma duodenale:**

It is prevalent in tropical and temperate areas of the world especially those with overcrowding and poor sanitation. It is mainly found in South Europe, North Africa,
North India, North China, South America, South-East Asia, Pacific and West Africa and Nectar Americans which present in sub-Saharan Africa, South-East Asia, Pacific and America. The adult parasites attached to the villi of small intestine will suck blood causing abdominal discomfort, diarrhoea cramps, anorexia, weight loss, and in advanced disease, hypochromic microcytic anemia. There is koilonychia, high cardiac output failure and humic murmur which could be seen in radiographic examination.\(^{(50,53)}\) Another clinical presentation often associated hookworm infection is cutaneous larva migrans it is self-limiting condition of skin eruption.

Hookworm is diagnosed by finding the characteristic round eggs containing convoluted larvae and serological diagnosis is not practical since there were so many cross reactions with other helminthes.\(^{(54)}\)

Mebendazole is the treatment of choice in dose of 100mg orally b.i.d for 3 days for both A. duodenal and Nectar Americans. Currently there is no available antihelminthic drugs showed convincingly an effect on arrested larvae in tissues. The ideal method for preventing
hookworm infection is by improvement of hygienic condition and using foot wear (only practical means of prevention).\textsuperscript{(55)}

**Strongyloides stercoralis:**

*Strongyloides stercoralis* is present in endemic warm climates worldwide, tropical and subtropical countries and in South-East Asia and institutes of mentally retards.\textsuperscript{(56,57,58)} In immunologically normal individuals, infection is usually asymptomatic or causes mild GIT dysfunction, manifest as pain bloating or bleeding, urticarial skin eruption and migratory serpiginous rash (Larvae curran).\textsuperscript{(59)} In immunocompromised hosts, intestinal auto-infection may lead to heavy worm load causing chronic diarrhoea and malabsorption of fat and B\textsubscript{12}.\textsuperscript{(48)} Also disseminated disease with wasting and pulmonary involvement with hyperinfection syndrome which is similar to adult respiratory distress syndrome can occur. CNS, pancreas, any eye invasion by the larvae results in symptom and signs attributable to these organs.\textsuperscript{(60)}

*Strongyloides stercoralis* is diagnosed by finding larvae
in host tissue, GIT and pulmonary secretion, and filariform larvae in faeces.\textsuperscript{61} They are demonstrated by direct faecal smear, formal-ether concentration method and filter paper culture. Duodenal aspiration is used if stool examination was negative and jejunal biopsy will show oedema, cellular and eosinophilic infiltration of mucosa with partial villous atrophy.\textsuperscript{62} Serological test such as ELISA and serum IgG reactivity to larval protein of strongloides stercolaries were useful in the diagnosis, but has a considerable cross-reaction with other helminth.\textsuperscript{63}

Treatment with albendazole in a dose of 400 mg b.d for 3 days. Thiabendazole 25mg/kg b.d for 3 days tabs and suspension. The side effects include nausea and vomiting and dizziness. Both drugs are effective for treatment of massive strongyloidosis and response is very well.\textsuperscript{66} Prevention can be achieved by avoiding skin contact with contaminated soil, and immuncompromised patients in endemic area should avoid walking barefoot. In case of latrogenic immuncompromised patients give prophylaxes with thiabendazole, 25mg/kg body weight daily for 2 days.

\textit{Trichuris trichuria} (Whip worm):
It affects about 755 millions in the world and more common on overcrowded areas with poor sanitary facilities, it is usually asymptotic, rarely a heavy infestation will cause abdominal pain, blood streaked diarrhoea, anaemia, rectal prolapse in children.\(^{(65)}\)

Whip-worm (\textit{Trichuis trichuria}) is diagnosed by identifying the football shaped eggs in smear or more recently by detecting specific IgG in saliva.\(^{(66)}\) It is treated by mebendazole as the same dose as for ascariasis.

\textbf{Enterobius vermicularis:}

Infection with \textit{Entrobius Vermicularis} (Pin-worm), is a worldwide diseases, usually affecting children, often several members of the family and institutions are affected, most of cases are asymptomatic or associated with perianal pruritis if persisted after treatment it is due to reinfection in which larvae migrate up the bowel.\(^{(50)}\) Itching at night will lead to sleep deprivation, and scratch of perianal areas lead to secondary bacterial infection and cellulites and even abscess.\(^{(67)}\) Rarely it can cause appendicitis, vulvovaginitis, urethritis and peritonitis.

It is diagnosed by identifying the ova on a piece of
c cellulphane tape applied to perianal region early morning and swab from under finger nails or by finding the adult worm around the anus usually at night.

Enterobiasis is treated successfully by Mebendazole given to the patient and close associated members of the family.

**Diphyllobothrium latum:**

These are the largest parasites that infect humans, ranging up to 10 meters in length. Infection acquired by ingestion of parasite cysts in tissues of smoked or undercooked fresh water fish.

The prevalence rate worldwide is 2%, but endemic in lakes or delta region of Scandinavia, the Former Soviet Union, Japan, Europe, Chile and North America. Patients with *D. Latium* infection can be asymptomatic or have few symptoms of non-specific weakness, dizziness, craving for salt, diarrhoea and intermittent abdominal discomfort occasionally vomiting, severe abdominal pain, weight loss, multiple infection may occurs and cause biliary obstruction. 1-2% of patients develop vitamine B₁₂ deficiency been consumed by the parasite.
Diagnosis is by stool examination and finding of characteristic operculated eggs. Treatment is by niclosamide and praziquantel.

Infestation is prevented by avoiding consumption of raw, smoked or salted fish from endemic area cooking above 56°C for 5 minutes or freezing -20°C for 24 hours is sufficient for killing the parasitic cyst.

**Hymenolepis nana (Dwarf tape worm):**

Present in warm, dry climate and is prevalent in Southern and Eastern Europe, Asia, Africa, Central and South America and Australia. It is the only human tapeworm that doesn't require an intermediate host. Intensive infection probably has a significant clinical consequences and occurs more common in institutionalized, malnourished or immunodeficient individuals.

Clinical manifestations vary with intensity and may include diarrhoea, anorexia, abdominal pain and pallor. Phlyctenular kerato-conjunctivitis statistically associated with *H. nana.* It is treated with prolonged and high dose of niclosamide or praziquantel because the parasite is relatively resistant to drug therapy.
**Prevention:** sanitation and hand washing are essential for control. Mass chemotherapy is used in closed communities to decrease endemic transmission.

**Taeniasis:**

**Definition:**

Is a disease caused by *Taenia saginata* (Beef tapeworm) and *taenia solium* (pork worm).

*Taenia saginata* is a cosmopolitan infection in which the man carries the adult and cattle the larvae. Ethiopia has the highest rate in the world. It is importance lies in the economic loss of beef meat. Human get infected by eating undercooked meat containing cysticerci, the larval stages encysted in the muscle of infected herbivores by swallowing the eggs infective eggs eating the raw abd. Visira marara or umfif-fit was important in high incidence of infection in Sudanese people.

Clinical picture: commonly the patient presented with worm observed motile white proglottides) in stool.(34) suffers embarrassment when they force their way out of the anus umbid. Worm doesn't suck blood and rarely cause serious pathology in the gut. Rarely patient presented with hunger
pains of intestinal obstruction.

**Diagnosis:** *T. saginata* is diagnosed by egg found in stool. Occasionally worms observed in vomitus. Esionophilia was reported not to be a feature of established infection which should be distinguished from that of *T. solium* in former the proglottide contain 15-20 worm branches on each side and the latter has 13 or more.\(^{35,36}\)

*T. saginata* may measure about 10 meters in length more typically 2.5 meters. Man represent the continuous source of infection to animals as long as tapewormlives in his intestine.\(^{35}\)

**Treatment:**

1- Niclosamide is the drug of choice. Dose age above 6 years 2gm before breakfast, and 1 gm separated in 1 hour. The drug is not absorbed and it kills the worm, which is expelled in digested state, give purge 2 hour after the drug. The side effects are nausea, vomiting, abdominal pain, diarrhoea, dizziness and headache and pruritis, it is safe in pregnancy.

2- Praziquantel, 10 -12mg for all age groups. Side effects mild but frequent, dizziness, myalgias, nausea, vomiting,
diarrhoea and abdominal pain. Albendazole is also used.

**Epidemiology and control:** The disease occurs when people eat raw or very undercooked meat from cattle. Control measures are environmental sanitation and meat inspection. Immediately after niclosamide treatment, the stool contains countless thousands of eggs and special care should be taken to ensure that it is safely disposed off. There is evidence that seagulls feeding on sewage outflows containing proglottides may help to disseminate the infection into pastures in the U.K.

*Taenia solium* is much less common infection than *T. saginata* but far more important because of its ability to cause severe disease in human. The human is the definitive host, the pig is the intermediate host. It is distributed worldwide when people eat raw or undercooked pork for this reason it is rare in Muslims and Orthodox Jews.

Taeniasis and cysticercosis caused by tapeworm *T. solium* are prevalent in humans in many developing countries of Latin America, Africa and Asia that lack proper sanitary infrastructure and adequate hygienic condition. As a result of migration from endemic areas an increase in
neurocysticercosis cases has also occurred in developed countries.\(^3\) Human neurocysticercosis is a disabling and occasionally fatal disease.\(^1,2\)

**Clinical presentation:** symptoms are usually trivial (Unless cysticercosus developed). They resemble those of *T. saginata*.

Human cysticercosis may be symptomless, worms may encyst in muscles or subcutaneous tissue and discovered by finding skin swelling (0.5-1mm) or accidentally discovered x-rays some year latter when calcified in skeletal muscles. In cerebral cysticercosis patient usually present with secondary epilepsy.

The diagnosis is by stool analysis and finding eggs, which should be differentiated from that of *T. sagenata* eggs. Biopsy of skin nodules for cyst. X-ray will show typical spindle shape calcification in muscles more in thighs, serological studies to defect cystocercal antibodies, immunologic assay such as ELISA and western blotting. C.T has been used as a reference or gold standard.

Immunological diagnosis of taeniasis has been recently evaluated in field studies by ELISA for coproantigen
detection. Values of 11% and 1.5% for cysticercosis and taeniasis respectively has been reported.$^{(4,10)}$

Ultrasonographic examination, the double reflective, ribbon-like structure in the lumen of the intestine seems to be characteristic to the U/S appearance of intestinal taeniasis.

Treatment of *T.solium* and cysticercosis is either with niclosamide or praziquantel as in *T. saginata*. Once diagnosed it should be treated as soon as possible because of the risk of acute infection with cysticercosis, which needs longer period of treatment.
OBJECTIVES

The epidemiological studies provide essential basis for the planning of an appropriate and cost effective control measures and study of this aspect is necessary for:

1- Knowing the prevalence of the increasing parasitic infection.

2- Age, sex distribution among adult affected by the disease.
MATERIALS & METHODS

Study design and population:

A cross-sectional study was performed on 100 patients, >15 years of age, 52 females and 48 males, subjects were selected by random sampling of any patient attending hospital at Mayo area with abdominal discomfort irrespective of other complaints were included in this study.

Study Period:

The study was conducted in the period between October 2001 and January 2002.

Study area:

The study site, Mayo area, is situated in Southern of Khartoum State, surrounded by Mahlia El Kalakla from West and Mahlia Elazahary from East, Soba irrigated scheme from the South. The estimated population is about 785453. There are 44 primary schools and only 4 secondary schools. Health services are not satisfactory, which consist of one hospital and 4 primary health care centers with limited facilities. The houses are made of low price materials (mud) and the houses backyards, which are used by children as
playgrounds, serve as a convenient waste disposal site in some families. The inhabitants drink water brought by containers which are made of cistern from the available 13 wells. The women when employed work as household servants and the majority of the men work as construction workers (manual workers).

**Data collections:**

Before starting collection of the data for the study, all individuals received detailed information about the procedures.

Examination of cases was within the morning hours of the working days of the week. According to well-defined questionnaire in which every patient was given a code number, patients were asked about, name, age, sex, occupation, level of education, residence now and previously, water supply, type of latrine and hygiene. History was obtained by clinician using standardized questionnaire which contained the following data, presence or absence of diarrhoea, anaemia, abdominal discomfort, weight loss, pruritis ani, worm observation and physical examination was done in comfortable condition, searching for skin
lesions, pallor, koilonychia, keratitis, angular stomatitis, abdominal distension, hepatosplenomegaly. The stool samples were collected in properly labelled containers. All patients gave fresh samples. Stool analysis examination looking for consistency presence or absence of mucus, blood, presence or absence of intestinal protozoa and helminthes (ova, cyst, larvae, traphozoites) and Kato-katz thick smear technique for detection of helminthes and hatching test.

**Data analysis:**

Statistical analysis was done using the SPSS (statistical package for social sciences), The students t-test, Chi-square test and Fishei exact tests, odd ratio were calculated at 95% confidence intervals to assess the association between stool analysis, epidemiological, clinical and hygienic data.
RESULTS

Faecal specimens from a total of 100 individuals were examined. The total sex distribution showing 52% females and 48% males (Fig. 1) and the overall prevalence rate was 74% (Fig. 2). The age group ranging from 15-60 years and age group above 15-30 years showed a high frequency (Fig. 3).

The protozoan parasites were the more common in Mayo area with prevalence rate (67.8%) of faecal specimens (Table 1). Giardia lambia and E. histolytica were the commonest organism with prevalence rate of 44.9%, 15.8% respectively, the overall prevalence rates in Mayo, infection with G. lambia was highest among people aged 15-30 years (35.7%) and E. histolytica was not found in individuals above 60 years as shown in Table 2. There was no significant difference in infection rate between males and females (Table 3). There were only 2 cases where stool specimen showed both E. histolytica and E. coli infection. No
helminthes larvae were found in stool specimens examined (Table 3).

The commonest helminthes parasite was *S. mansoni* (14.1%) and the overall prevalence was highest in subject (15-30 years) and the infection decreases with increasing age (Table 2). The prevalence of other intestinal helminthes as follow. *Taenia saginata* (1.7%) *H. nana* (2.5%) and *Enterobious vermicularis* and it is obvious that infection with *H. nana* and *Enterobious vermicularis* (0.8%) decreased with increase in age.

The presenting symptoms of patients are shown in Table 4, abdominal discomfort (93%), the most frequent complaint, other symptoms are diarrhoea (83%), weight loss (38%) and anaemia (29%), Pruritis ani (17%), skin manifestations (1%), 2% were pale, koilonychia (1%), abdominal distension (3%), hepatomegaly (1%), splenomegaly (1%), (Figures 4 to15 respectively). Microscopic stool analysis findings were blood was found in stool of 22 patients and mucus in 44 patients (Figures 16 & 17) and the consistency of the stool was found to be soft in 74.5% (Fig. 18). Interestingly the relation between blood and protozoa
infection were calculated and was found to be statistically significant $X^2 = 9.49$, $P = 0.008$. $X^2 = 12.59$, $P < 0.04$ in overall prevalence of parasites. Prevalence according to education we studied the relation between education and prevalence rate of intestinal parasites revealed (44%) for illiterate (31%) for primary school and 22% for secondary school and 3% for university (Fig. 19). The lower education, the highest rate of infection as shown in Table 5. The presence of intestinal parasites by residence showed that most of the infected individuals were from rural areas and western and central area of Sudan showed the highest prevalence rate (36%, 28% respectively) compared to northern Sudan (10%). The difference is statistically calculated and was found to be significant $X^2 123.92$, $P < 0.05$ (Fig. 20).

Distribution of patients by the source of water supply showed 9% were drinking from wells and 9% from tap water, 1% from sea (Fig. 21).

Distribution of patients according to their hygiene revealed that 89.9% had a good hygiene and 10.1% had bad hygiene (Fig. 22).
Distribution of patients by sewage disposal and parasitic infestation showed that (94%) had pit halls and 6% dispose their sewage outdoors, and no siphons (Fig. 23). The differences were calculated and was found to be statistically insignificant. Presence of intestinal parasite and occupation were studied (Table 6). It revealed that the highest occupational hazard are housewives (43%), followed by labourer (38%) (Fig. 24).

Frequency of intestinal parasites according to stool analysis was studied by direct and Kato-Katz methods, direct stool examination revealed 36 parasites, while Kato-Katz showed 47 parasites (Figures 25 & 26).
## Table 1

Distribution of intestinal parasite among 83 adult persons in Mayo area.

<table>
<thead>
<tr>
<th>Type of parasite</th>
<th>No. of parasite</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Giardia lambia</em></td>
<td>53</td>
<td>71.6</td>
</tr>
<tr>
<td><em>Entamoeba histolytica</em></td>
<td>19</td>
<td>25.6</td>
</tr>
<tr>
<td><em>Entamoeba coli</em></td>
<td>2</td>
<td>2.7</td>
</tr>
<tr>
<td><em>Schistosoma mansoni</em></td>
<td>17</td>
<td>22.9</td>
</tr>
<tr>
<td><em>Tina saginata</em></td>
<td>2</td>
<td>2.7</td>
</tr>
<tr>
<td><em>Hymenolybis nana</em></td>
<td>3</td>
<td>4.1</td>
</tr>
<tr>
<td><em>Enterobious vermicularis</em></td>
<td>1</td>
<td>1.3</td>
</tr>
</tbody>
</table>
Table 2

Age specific rate of intestinal parasite among
74 adult in Mayo area.

<table>
<thead>
<tr>
<th>Age</th>
<th>Parasite</th>
<th>15-30</th>
<th>31-40</th>
<th>41-50</th>
<th>&gt; 60</th>
<th>Overall prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>No. examined</td>
<td>62</td>
<td>(62%)</td>
<td>24</td>
<td>(24%)</td>
<td>10</td>
<td>(10%)</td>
</tr>
<tr>
<td>G. lambia</td>
<td>43</td>
<td>(69.3%)</td>
<td>8</td>
<td>(36.4%)</td>
<td>1</td>
<td>(10%)</td>
</tr>
<tr>
<td>E. histolytica</td>
<td>12</td>
<td>(19.3%)</td>
<td>6</td>
<td>(27.5%)</td>
<td>1</td>
<td>(10%)</td>
</tr>
<tr>
<td>E. coli</td>
<td>2</td>
<td>(3.2%)</td>
<td>0</td>
<td>(0.0%)</td>
<td>0</td>
<td>(0.0%)</td>
</tr>
<tr>
<td>S. mansoni</td>
<td>12</td>
<td>(19.3%)</td>
<td>3</td>
<td>(13.6%)</td>
<td>1</td>
<td>(10%)</td>
</tr>
<tr>
<td>T. saginata</td>
<td>0</td>
<td>(0.0%)</td>
<td>0</td>
<td>(0.0%)</td>
<td>1</td>
<td>(10%)</td>
</tr>
<tr>
<td>H. nana</td>
<td>1</td>
<td>(16.6%)</td>
<td>1</td>
<td>(4.5%)</td>
<td>0</td>
<td>(0.0%)</td>
</tr>
<tr>
<td>E. vermicularis</td>
<td>1</td>
<td>(16.6%)</td>
<td>0</td>
<td>(0.0%)</td>
<td>0</td>
<td>(0.0%)</td>
</tr>
</tbody>
</table>

70
Table 3

Prevalence of intestinal parasite by sex among 100 adult in Mayo area.

<table>
<thead>
<tr>
<th>Parasite</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. examined</td>
<td>48</td>
<td>52</td>
</tr>
<tr>
<td><em>E. lambia</em></td>
<td>20</td>
<td>19</td>
</tr>
<tr>
<td><em>G. histolytica</em></td>
<td>10</td>
<td>9</td>
</tr>
<tr>
<td><em>E. coli</em></td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td><em>S. mansoni</em></td>
<td>11</td>
<td>6</td>
</tr>
<tr>
<td><em>T. saginata</em></td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td><em>H. nana</em></td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td><em>E. vermicularis</em></td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>
### Table 4

**Frequency of symptoms in 100 patients in Mayo area.**

<table>
<thead>
<tr>
<th>Parasite</th>
<th>Symptoms</th>
<th>Diarrhoea</th>
<th>Anaemia</th>
<th>Abdominal discomfort</th>
<th>Wt. loss</th>
<th>Pruritis ani</th>
<th>Worm observation</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>G. lambia</em></td>
<td></td>
<td>22</td>
<td>11</td>
<td>38</td>
<td>19</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td><em>E. histolytica</em></td>
<td></td>
<td>22</td>
<td>8</td>
<td>20</td>
<td>10</td>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td><em>S. mansoni</em></td>
<td></td>
<td>12</td>
<td>6</td>
<td>11</td>
<td>6</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td><em>T. saginata</em></td>
<td></td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td><em>H. nana</em></td>
<td></td>
<td>3</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><em>E. vermicularis</em></td>
<td></td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>
Table 5

Distribution of patients by education and diarrhoea in Mayo area.

<table>
<thead>
<tr>
<th>Level of education</th>
<th>Total No. examined</th>
<th>No. of patients with diarrhoea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Illiterate</td>
<td>44</td>
<td>24</td>
</tr>
<tr>
<td>Primary school</td>
<td>31</td>
<td>14</td>
</tr>
<tr>
<td>Secondary school</td>
<td>22</td>
<td>18</td>
</tr>
<tr>
<td>University</td>
<td>3</td>
<td>3</td>
</tr>
</tbody>
</table>

$X^2 = 9.39, \quad P = 0.02$ (significant)
Table 6

Distribution of patients by occupation and parasitic infection in Mayo area.

<table>
<thead>
<tr>
<th>Occupation</th>
<th>Total No. examined</th>
<th>Parasitized</th>
<th>Non parasitized</th>
</tr>
</thead>
<tbody>
<tr>
<td>Labourer</td>
<td>39</td>
<td>39</td>
<td>0</td>
</tr>
<tr>
<td>Employee</td>
<td>3</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Housewives</td>
<td>41</td>
<td>26</td>
<td>15</td>
</tr>
<tr>
<td>Students</td>
<td>15</td>
<td>15</td>
<td>0</td>
</tr>
<tr>
<td>Unemployee</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>
DISCUSSION

This was a hospital-based study, which has its limitation, it doesn't reflect what happen in the community, being dealing with selected and symptomatic patients only. Yet useful information can be obtained.

To our knowledge this is the first study to be done in adults. In this study the overall prevalence rate of intestinal parasites was 74%, this prevalence rate is comparable with a previous study done in Elengaz area school children,\(^{69}\) where the prevalence rate was found to be 64.4%, and this study shows prevalence rate more than that reported from other areas, among children under 5 years of age in the police camps in Khartoum State,\(^{70}\) where the prevalence rate was 44%. This is because of decreased crowding and the good hygienic behavior in this area and the prevalence rate is comparable to studies done abroad in Iraq (77.9%), similar to a study done by Lee KJ, Ahn YK, Yong TS in Korea where the prevalence rate was 78.1% and less prevalence in Saudi
Arabia, Bahrain and Zinwie (51.6%), which was stated due to high socioeconomic standard.\(^{(71,72,73)}\)

Males Predominance (57.4%) which is in agreement with a study done in Lagos and in Chandigarh.\(^{(74,75)}\) The age distribution of the patients is between 15 and 60 years and the median age was 20 years.

In this study the commonest parasites encountered were \textit{G. lambia}, \textit{E. histolytica}, \textit{S. mansoni} (Table 1). This result is comparable to a study done in Sudan among school children in Elengaz area where the commonest parasites were \textit{G. lambia} and \textit{H. nana}, 33.4\% and 26\% respectively. The prevalence rate of \textit{G. lambia} in Iraq was (35.4\%) and similar results were obtained in a study done by Araj GF, Abdull-Baki NY, \textit{et al.}, in Lebanon, where the prevalence rate was 20.7\% and \textit{G. lambia} was the commonest protozoal infection.\(^{(76)}\)

The results of this study showed that Giardia intestinalis (71.6\%) infection can be symptomatic in adults in an endemic area and this is in agreement with the results of a study done in Sudan (1967),\(^{(77)}\) which is opposite to
what had been said before (Moore, 1969, Babb, 1971).\(^{(78,79)}\)

The prevalence rate of \textit{Giardia intestinalis} in males versus females was almost the same, being 51.3%, 48.7% respectively. This is comparable to a study done in Lebanon.\(^{(76)}\)

Abdominal discomfort, diarrhoea and malabsorption as shown in Table 4 are comparable to study done in Sudan.\(^{(77)}\) Although we didn't carry out test for malabsorption, but the presence of weight loss in patient with giardiasis which out numbered that of other parasitic infestation most probably indicates malabsorption. The stools in \textit{Giardia intestinalis} is often mucoid. The symptoms observed in our patients, don't differ significantly from those already described in adult patient being infected for the first time.\(^{(80,81,82,83)}\)

The results showed that the second common intestinal parasite was \textit{E. histolytica} (25.6%) which was comparable to study done in Lebanon,\(^{(76)}\) and there was no significant difference between sexes. Adults in the age group 15-30 years were the most affected followed by the 31-40 years age group and infection decreased gradually with
increasing age to become zero by the age of 60 years as seen in Table 2. Infection rate was slightly higher in males than females, this is comparable to studies done in Sudan and Indonesia.\(^{84,85}\) Diarrhoea and abdominal discomfort were the only positive symptoms associated with \textit{E. histolytica}, so diarrhoea and abdominal discomfort could be symptoms worth exploring further for rapid identification of adult infection with \textit{E. histolytica}, this is agreement with a study done by Utizinfer J, (1999).\(^{86}\)

This study indicates that the prevalence rate of \textit{S. mansoni} was (22.9\%), and age prevalence curve peaks at 22 years age group comparable to result of study done in Zimbabwe,\(^{87}\) and less prevalence rates were found in Saudi Arabia, Egypt and Iraq and this was stated due to better education of the people in these countries, the water supply is better and in most areas women no longer use stagnant river tributeries for washing clothes. Diarrhoea, abdominal pain and mucus in the stools are the most frequent symptoms associated with \textit{S. mansoni} infection, this is in agreement with the results of studies done in Sudan (1992).\(^{88}\) and of other workers elsewhere (1974)\(^{89}\) there was
significant difference between male (64.7%) and females (35.2%) and this is due to the role of gender who work as farmers rather than due to sex preference.

This data indicates that the prevalence rate of *T. saginata* was 2.7% and diarrhoea was the commonest symptom associated with *T. saginata* which didn’t differ from that written in the literature, it was present in age group above 40 years because of traditional eating of raw meat (Umfit fit).

In our data the prevalence rate of *H. nana* was 4.1% and this was comparable to a study done in Sudan and age group (15-30 years) are more affected and diarrhoea and abdominal distension are the main symptoms.\(^{(90)}\)

This study showed that the prevalence rate of *E. vermicularis* is 1.3%. The low prevalence rate of this parasites is due to the fact that we examined the stool only for eggs and adult worm, and we did not carry the celphone tape examination, because people in this country are ashamed from this method.

The complete absence of soil transmitted helminthic diseases might not indicate better hygienic practice, since
this population represents a low socioeconomic class. This data was collected between Oct. 2001 and January which was considered off peak period of parasites and only 6% of the population accustomed to defecate in the open which may also explain the complete absence of soil transmitted diseases such as hookworm and strongyloidosis.

The results of direct stool examination was compared with Kato-Katz method, the latter is known to be more sensitive for the diagnosis of *S. mansoni, ascariasis, Trichuris trichuria, Taenia* infection.

Our results confirmed this and showed that combination of both methods would further increase the sensitivity for infection with *S. mansoni, T. saginata* and this with agreement with study done by Dirk Engels, Samuel N, *et al.*, (1996). (91) Our results showed 90% of population were coming from rural area. Compared to 10% from Khartoum State, and the difference is statistically significant. The poor education and high crowding index, poverty of food handlers associated with higher rates of infection by intestinal parasites (Fig. 4). So the trend towards urbanization seems to have caused deterioration of living condition and
sanitation standard in some areas of Khartoum State with the most vulnerable people experience an increase in intestinal parasite infection.

**CONCLUSION**

- Protozoal disease was found to be the most common intestinal parasite in Mayo area and *Giardia lambia*, *E. histolytica* were the commonest organisms.
- No helminthic larvae were present, but the commonest helminthic parasite was schistosomiasis.
- Poor hygiene, poverty, indiscriminate defecation, lack of health education, unawareness of the magnitude of the problem, minimally equipped and few staffed laboratories were found to be directly related to the high prevalence of intestinal parasite at this region.
RECOMMENDATIONS

Intestinal parasitic diseases are disabling and affecting the most active group in the community and the problem could be completely controlled if the laboratories are well equipped and the technicians are qualified and community awareness of the problems so:

1- Collection of reliable data is extremely important in order to improve planning and monitoring.

2- National research programmes are encouraged to know the life style of the population.

3- Clinical history of intestinal parasites in a family member should be taken into account by health personnel to prevent other members of the family from becoming infected.

4- Public health authorities should control street-food to reduce new cases of intestinal
parasites by means of hygienic and sanitary measures.

5- Health promotion, environmental sanitation and clean water supply.

6- Improvement of the economic status.

7- The treatment of positive cases.

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Prevalence of parasitic infection
in patient with abdominal discomfort

1- Index No. ............................
2- Date: ..............................................
3- Name: ..................................................
4- Age .........................................................
5- Sex .........................................................
6- Occupation ..............................................
7- Education: ..............................................
   - Illiterate  □  - Primary school  □
   - Secondary school  □  - University  □
8- Residence: ........ Now ...........Previously.........
9- Source of water: .............................................
   - Tap water  □  - Well water  □  - River water  □
10- Type of latrine: .................................................
11- Hygiene: ......................................................
History:  Yes  No
- Diarrhoea
- Anaemia
- Abd. discomfort
- Weight loss
- Pruritis ani
- Worms observation

**Examination:**

<table>
<thead>
<tr>
<th>1- General:</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Skin manifestation</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>- Pallor</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>- Koilonyxia</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>- Keratitis</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>- Angular stomatitis</td>
<td>☐</td>
<td>☐</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>2- Local:</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Abdominal distension</td>
</tr>
<tr>
<td>- Hepatomegaly</td>
</tr>
<tr>
<td>- Splenomegaly</td>
</tr>
</tbody>
</table>

**Diagnosis:**

**Stool analysis (Fresh):**

- Consistency: .................................................................
- Mucus: .................................................................
- Blood: .................................................................

**Direct smear:**

- Cyst ☐ - Ova ☐ - Trophozoid ☐ - Larvae ☐
Kato-Katz methods:

- Cyst
- Ova
- Larvae