INTESTINAL PARASITIC INFESTATIONS IN SEVERELY MALNOURISHED CHILDREN ADMITTED AT THE MOI TEACHING AND REFERRAL HOSPITAL, ELDORET, KENYA.

BY

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SM/PGCHP/04/2008

A THESIS SUBMITTED TO MOI UNIVERSITY IN PART FULFILLMENT OF THE REQUIREMENTS FOR THE AWARD OF THE DEGREE OF MASTER OF MEDICINE IN CHILD HEALTH AND PAEDIATRICS

SCHOOL OF MEDICINE, DEPARTMENT OF CHILD HEALTH AND PAEDIATRICS

NOV 2013

DECLARATION:

This thesis is my original work and has not been presented to any other university/institution.

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ACKNOWLEDGEMENTS

I acknowledge my thesis supervisors Prof Ayaya S.O and Dr Marete Irene for their guidance in the development of this thesis. I also thank the laboratory technicians in the department of Child Health and Paediatrics for their untiring efforts in the examination of specimens for parasites. I am grateful to the statistician for valuable input in analysis of data. I also thank the staff of the department of Paediatrics and my fellow registrars for their encouragement during the writing of my thesis. Last but not least, I thank my friends and family for their unwavering support during this time

DEDICATION

To my Children Kimutai Obed and Cherotich Beryl, who make every workday worthwhile.

ABSTRACT

Title: Intestinal Parasitic infestations in severely malnourished children admitted at the Moi Teaching and Referral Hospital, Eldoret, Kenya.

Background: Malnutrition in children predisposes to diarrhea, infections and long term health problems. Intestinal parasite infestation is commoner than in eutropic children and is often symptomatic and severe. Though intestinal parasites and severe malnutrition frequently co-exist, the exact extent is unknown. The purpose of the study was to establish prevalence.

Objective: To determine the prevalence of intestinal parasites and associated clinical and demographic factors.

Methods: An 8-month cross-sectional study was carried out between Nov 2011 and June 2012 on severely malnourished children aged 6-59 months whose parents gave consent, and excluded those with known underlying malignant or metabolic disease admitted during the study period. Data was collected using a structured questionnaire. It included demographic, clinical, sanitation and nutrition histories of the subjects. Stool specimens were analysed using formol-ether concentration technique for presence of parasite ova and cysts. Data was processed by SPSS, STATA and variables compared by Chi Square and Fisher's analytical tests.

Results: A total of 130 severely malnourished children were studied, median age was 25 months with a male to female ratio of 1:1.24. The subjects' guardians had a median age of 26 years, 61% were unemployed, 53% were single parents, and 56% attained primary school education. Malnutrition types were marasmus 50%, marasmic-kwashiorkor, 32 and kwashiorkor 18%. Diarrhoea was significantly present in children with kwashiorkor and marasmic-kwashiorkor 78% (P-Value 0.0491). Chronic diarrhea was present in 49% of subjects with diarrhoea, stools were profuse and watery in 52%. There was a previous diarrhoeal episode in 79% and 22% had been dewormed prior commonly with Mebendazole. Tap water was a common source but subjects who used river water for daily use were 11 times more likely to be infested with intestinal parasite. Parasites were found in 24% of subjects, 77% of whom had diarrhoea. The parasites species that were isolated were protozoa 15% (*Giardia lamblia* 9(6.9%), *Entamoeba hystolytica* 4(3%), *Entamoeba coli* 4(3%), *Chilomastix mesnilii* 3(2.3%)) and helminths (9%) (*Ascaris lumbricoides* 4(3%), *Strongyloides stercoralis* 2(1.5%), *Ancylostoma duodenale* 2(1.5%), *Taenia solium* 2(1.5%), *Taenia saginatum* 1(0.8%).

Conclusions and Recommendations: Intestinal parasites, commonly protozoa, among malnourished children are associated with severe, chronic and recurrent diarrhea. Diarrhoea is a common presentation of parasite infestation. Malnourished children should be dewormed and given antiprotozoal agents as well. Use of River water in this region is associated with parasite infestation and should be treated before use.

LIST OF ABBREVIATIONS:

GIT - Gastrointestinal Tract

INS - International Nutrition Society

IPIs -Intestinal Parasitic Infections

IREC -Institutional Research Ethics Committee

KDHS -Kenya Demographic Health Survey

MTRH - Moi Teaching and Referral Hospital

O & C - Ova and Cysts

O & P - Ova and Parasites

ORS - Oral Rehydration Solution

PEM - Protein Energy Malnutrition

SPSS - Statistical Package for Social Scientists

UNICEF -United Nations Children's Fund

WASH - Water, Sanitation and Hygiene Program of UNICEF

WHO - World Health Organisation

OPERATIONAL TERMS/ DEFINITIONS:

Protein-Energy Malnutrition: Is the lack of sufficient Energy or Protein to meet the body's metabolic demands. It could be due to inadequate dietary intake, increased demands due to disease or increased nutrient losses.

Severe malnutrition: This is Weight-for-Age less than 60 percent or presence of edema of both feet in a child with 60-80 percent of expected weight for age. The clinical entities included are: kwashiorkor, marasmus and marasmic-kwashiorkor (Wellcome Classification)

Marasmus: Is an undernutrition entity that is due to diets chronically deficient in calories as well as protein and other nutrients. Affected children are generally wasted and are less than 60 percent of expected weight for age.

Kwashiokor: Weights are 60-80 percent of expected for age and children have edema.

The symptoms in this entity are attributable to diets relatively deficient in protein.

Marasmic-kwashiorkor is a malnutrition entity in which weight-for-age is below 60% of expected for age and presence of edema.

Diarrhoea: Defined as 3 or more liquid stools passed in a 24-hour period.

An episode of diarrhea is considered terminated when an individual has at least 2 days free of diarrhea.

Persistent/ Chronic diarrhea: Persistent diarrhea is an episode of diarrhoea that lasts for at least 14 days.

Underweight: Underweight children are those whose weights are between 60-80 percent of expected for age and have no edema

Parasites: a parasite is an organism that depends on another for its existence. There are two main types of intestinal parasites: **helminthes and protozoa**.

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CHAPTER ONE

INTRODUCTION

1.1 Background:

Severe malnutrition is primarily a problem of developing countries. Every year, 10 million children under five years die worldwide.[2] About half of these are associated with under nutrition and about two million with diarrhoea. Diarrhoea is a common direct consequence of parasite infestation in the malnourished. Intestinal parasitic infections (IPIs) are globally endemic and have been described as constituting the greatest single worldwide cause of illness and disease[1–3]. IPIs are linked to lack of sanitation, lack of access to safe water and improper hygiene; therefore they occur wherever there is poverty. IPIs deprive the poorest of the poor of health, contributing to economic instability and social marginalization. The poor people of under developed nations experience a cycle where under nutrition and repeated infections lead to excess morbidity that can continue from generation to generation. People of all ages are affected by this cycle of prevalent parasitic infections; however, children are the worst affected [2, 4.] In East Africa, severe malnutrition is estimated to constitute 16 percent of all admissions to children's wards.[3] Attacks of diarrhea in undernourished children tend to be more severe and of longer duration, suggesting a vicious cycle of diarrhoea and under nutrition.

Severe malnutrition is defined as weight for age less than 60 percent or clinical signs of severe malnutrition (Wellcome Classification). The clinical entities included are: kwashiorkor, marasmus and marasmic-kwashiorkor.[4] According to KDHS statistics 2003, the proportion of severely malnourished children was 12 percent nationally; majority of whom were stunted.[5]

Malnourished children suffer many complications, the commonest of which is diarrhea. In one study, malnourished children have a 24-fold higher prevalence of diarrhea.[10] The causes of diarrhea in these children are many (bacterial, viral, parasitic) but this study aims at determining the frequency of parasitic causes. There is a high prevalence of intestinal worms among children in marginal agricultural areas,[6] such areas being found in parts of the Rift Valley.

Almost all children living in endemic zones are infected by GI parasites.[6,7] However only 3-5% percent develop diarrhoea directly related to parasite infection.[7] The clinical expression of the parasitoses, however, is largely determined by host defenses; and when they are weakened, as in protein energy malnutrition, parasitic diarrhea is frequent and severe.[8] Diarrhea caused by Strongyloides or Giardia is common and severe in malnourished children, while well-nourished children remain healthy carriers. These parasites therefore require specific treatment in the malnourished.[8]

Children are most at risk of both malnutrition and parasite infestation.[10,11] It affects their physical, emotional and cognitive wellbeing with adverse lifelong health consequences making them less productive as adults. The country's general development is thus retarded.[13,14]

Co-infections are associated with majority of deaths among severely malnourished children. Diarrhoea was the leading co-morbidity in one study at Kilifi District hospital, affecting 45 percent of severely malnourished children.[9]

CHAPTER TWO

LITERATURE REVIEW

2.1 Background:

Intestinal parasitic infections are among the major public health and socio-economic concerns that adversely affect the well-being of the poor in developing countries. It has been estimated that *Ascaris lumbricoides*, hookworm and *Trichuris trichiura* infect 1,450 million, 1,300 million and 1,050 million people worldwide, respectively, while schistosomiasis affects over 200 million people (1). *Entamoeba histolytica* and *Giardia lamblia* are also estimated to infect about 60 million and 200 million people worldwide, respectively (2).Complications associated with malnutrition are probably the commonest cause of death in young children worldwide. For example, in an urban community in the Gambia over 35% of deaths in children aged 0-3 years were found to be caused by diarrhea coupled with malnutrition.[11] The importance of the distinction between acute and persistent diarrhea (episodes of more than 14 day's duration) has been recognized.[1] Studies from different countries have shown that up to one half of deaths related to diarrhea were linked to persistent diarrhea, the type commonly due to parasites. Such figures may vary by area, season and environment.

Diarrhea (especially persistent diarrhea) often causes deterioration of nutritional status, and poor nutritional status has been shown to increase the duration and severity of diarrhea. Effective management of diarrhea helps prevent future illness, including diarrhea, since maintenance of nutritional status helps to maintain immunocompetence. Thus there are important implications for both prevention and management of diarrhea in children.

Diarrheal illnesses account for 10-80 percent of growth retardation in the first few years of life word-wide,[2] with the magnitude of effect possibly modified by other factors such as etiology and clinical type of diarrhoea, the source and adequacy of dietary intake, treatment and feeding practices. Understanding the mechanisms of diarrhea-induced undernutrition and appropriate treatment of diarrhea is important for managing the immediate illness and also for maximizing benefits for children's wellbeing in the long term.[2]

Most parasites cause persistent diarrhea, the type of diarrhea associated with greatest mortality among affected children.[11] While it is clear that parasites may lead to malnutrition, the extent to which malnutrition itself causes increased parasite infestation is unknown. Parasites in malnourished children cause severe and frequent diarrhea.[8] The two conditions do frequently co-exist. Human intestinal parasites occur in their greatest numbers in the wet tropics and sub-tropics.[10] The mechanisms by which intestinal parasites interfere with nutritional status include causing reduction in food intake, malabsorption, maldigestion, gastrointestinal nutrient losses, inflammatory responses (immunological host response) and anemia.[12]

Giardia lamblia is often associated with severe malnutrition[6, 8, 10] in certain areas, and may merit particular attention.

2.2 Prevalence of intestinal parasites:

Prevalence varies with regions, and intestinal parasites are endemic in the tropics.[7,8] Almost all children living in endemic zones are infected by gastrointestinal parasites.[7] However only 3 to 5% develop diarrhea directly related to parasite infection,[7] this figure rising several fold in malnourished children in whom diarrhoea is severe and prolonged.[7,8]

An estimated 10% of the world's population is infected with *E histolytica*, the highest prevalence is in developing countries with the lowest levels of sanitation.[13] This results in up to 100,000 deaths annually. *G lamblia* is the most commonly isolated intestinal parasite throughout the world. Rates of 20-40% are reported in developing countries, especially in children.[13] In study in Gambian children, it was shown that malnourished children harbour parasite more often than eutrophic children and they do not respond immediately to usual medications like metronidazole.[11]

Children with diarrhoea marginally harbour parasites more frequently than those without diarrhoea, particularly the diarrhoeagenic parasites.[14] In a prospective survey, 1130 children were studied for parasitic infections associated with acute diarrhea and/or protein-energy malnutrition at the Jos University Teaching Hospital, Jos, Nigeria. Intestinal parasites were isolated in 29.2% of the children with E. *histolytica*, S. *mansoni*, Hookworm and A. *lumbricoides* predominating.[14] In a similar study in Nigeria in which under 5 year-olds with diarrhea were investigated for parasites, 23.3 percent had prasites.[15] It was concluded that parasitic infestations are commonly associated with childhood diarrhea.

Diarrhea due to helminths is rare and only Strongyloides stercoralis induces severe diarrhea in malnourished child[16,17] and must be treated in emergency with Ivermectin to avoid dissemination[6,7,8].

2.3 Pathogenesis and Pathophysiology

While it is clear that parasites may lead to malnutrition, the extent to which malnutrition itself causes increased parasite infestation is not clearly known. Intestinal parasites may be associated with a reduction in food intake, maldigestion,

malabsorption, endogenous nutrient loss, and anaemia.[12,13] Behavioral effects of parasitic infestation may be important: the blindness resulting from warm destruction of sight may lead to malnutrition. These interactions are cyclic and closely linked, causing a malnutrition-infection complex described as follows: Inadequate dietary intake leads to low nutritional reserves. This is associated with lowering of immunity, probably with almost all nutrient deficiencies; particularly with protein-energy and Vitamin A deficiencies there may be progressive damage to mucosae, lowering resistance to colonization and invasion by pathogens. Under these circumstances, disease will be of potentially increased incidence, severity and duration. The disease processes itself exacerbates loss of nutrients, both by the host's metabolic response, and by physical loss from the intestine. These factors themselves exacerbate the malnutrition leading to further possible damage to defense mechanisms.

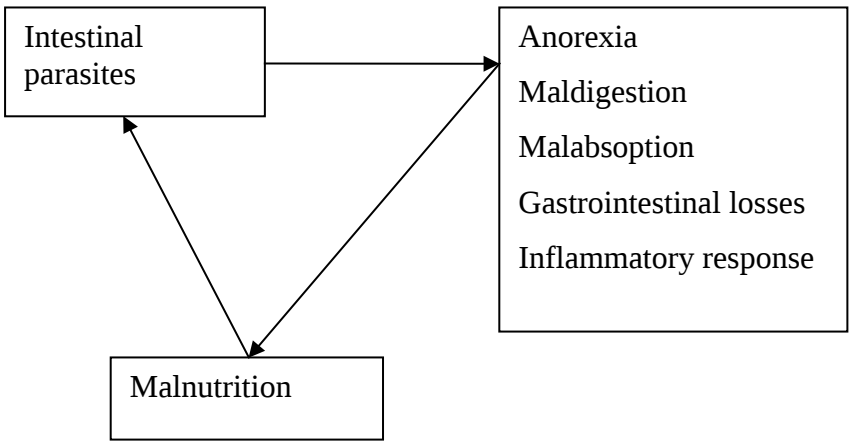


Figure 1: mechanisms of parasite-host nutrition interactions.[12]

Anorexia

Shown in rat studies that food consumption is markedly depressed in rats infected with *N. brasiliensis.* this is now recognized as a feature of cytokine activity and could explain occurrence of anorexia in humans too.

Maldigestion and malabsorption

With the possible exception of *T. trichiura,* malabsorption and or maldigestin of fat protein and carbohydrate have been described in these parasite infections. Panamanian *children with A. Lumbricoides* have been found to have lactose maldigestion which recovers following successful treatment. Many of these symptoms are associated with abnormalities and frank injury to the mucosa of the small bowel. In giardiasis, the severity of the malabsorption correlates with the degree of mucosal damage. In Gambian children small intestinal injury of this type has been shown to be associated with marked faltering of both length and height growth. (Lunn et al, 1991)

Gastrointestinal losses

With the exception of *A. Lumbricoides*, these parasites have been implicated in precipitating a protein-losing enteropathy. In protein and energy-malnourished animals the effect is exaggerated resulting in extremely low plasma albumin concentrations and in some cases in hypoproteinaemic edema.

In human infections, loss of iron (as hemoglobin) tends to be a more critical problem than protein loss and severe iron deficiency anaemia is the classical symptom of heavy hookworm and *T. trichiura* infections. Such losses can be important especially if the protein content of a child’s diet is low. In one study of Jamaican children with severe trichuriasis, the mean protein loss was estimated at 7g/d which represented

about 25% of the daily intake(cooper *et al*, 1992). Hypoproteinaemic edema has been reported in association with hookworm, *T. trichiura* and *S. stercoralis* infections (Stephenson, 1987), so it seems that these parasitic infections, when combined with a poor quality diet can lead to the development of kwarshiorkor.

Inflammatory responses

Gastrointestinal parasite infections elicit both local inflammatory and systemic acute phase responses in their hosts. Lamontagne *et al*. (1984) have demonstrated raised levels of alpha-1-protease inhibitor and other acute phase proteins during both the larval lung phase and the adult intestinal phase of infection. Activation of the systemic acute phase is initiated by cytokine release from activated macrophages but results in a wide range of metabolic alterations throughout the body (Grimble, 1989). Most important in terms of nutritional status are anorexia at time of raised nutrient requirements and a mobilization of skeletal muscle. These processes combine to cause either weight loss or marked growth faltering.

One feature of intestinal parasitic infections is that there are two potential sources of mucosal injury. First, damage resulting from the movement and feeding of the parasites, and second, that caused by the host's immune response to the presence of parasite and causing damage to villi and enterocytes.[19] This latter effect at times is the more severe, leading to chronic disease with the inflammatory damage persisting as long as the parasites remain.

2.4 Aetiology and specific presenting features

Protozoa: A wide range of intestinal protozoa infect the human intestinal tract [6,8,18] Many of these are worldwide in distribution, but the range of species and their

prevalence is higher in developing areas with low levels of sanitation and hygiene, be they temperate or tropical. *Entamoeba histolytica* tends however, to be endemic in many tropical regions of the world.[18]

The two best recognised pathogens are *Giardia duodenalis* (= *G. lamblia*; *G. intestinalis*) and *Entmoeba histolytica.* The latter species causes amoebiasis which can vary from the asymptomatic cyst passer (lumenal amoebiasis) to the patient presenting with non-dysenteric colitis, frank amoebic dysentery, amoeboma or extraintestinal invasive amoebiasis (eg liver/lung abscess). Giardiasis (often termed "Bushwalker's Diarrhoea) may vary from asymptomatic to a malabsorbtive and often chronic diarrhoea lasting weeks or months if untreated.[10,16] Its interpretation as a significant pathogen is more variable and in regions of low sanitation and hygiene and with a host of other more serious intestinal pathogens, it may be so common as to be left untreated.

The role of *Blastocystis hominis* as a pathogen is controversial but it can be associated with a watery and often chronic diarrhea.[10] A sensible approach to interpretation when finding this species in a stool specimen is to consider it significant if found in a patient with watery diarrhoea and in whom no other pathogens have been isolated after adequate laboratory investigations.[7]

Both *Cryptosporidium* and *Isospora* can cause a watery diarrhoea, mostly in children or in patients with AIDS while *Cyclospora* is also recognised as a cause of a watery diarrhoea – mostly in travellers to developing regions.[6,7] The Microsporidia, which include the genera *Microsporidium* and *Enterocytozoon* amongst others, also tend to be pathogens in AIDS patients and again can be associated with a watery diarrhoea. AIDS itself is a common cause cause of malnutrition.[6,7,8,10]

Dientamoeba fragilis is a flagellate which is probably transmitted in the egg of the threadworm (*Enterobius vermicularis*) and, with *Trichomonas hominis* it is believed, arguably, by some, to be one of the " lesser lights of the protozoal diarrhoea syndrome"[18]

Diagnosis of these infections is mostly based upon repeat stool examinations, species being identified by trophozoite/cyst morphology and size after staining wet drop preparations with iodine or permanent faecal smears with stains
such as Gomori or iron haematoxylin. [17,18]

Intestinal helminthes: Comprise a wide range of species including Trematodes (like *schistosoma spp*), Cestodes (Like *Taenia spp*.) ,Nematodes (like *Ascaris spp*, A. *duodenale*, S. *stercoralis*, T. *trichiura*) and Acanthocephalans.[18] Like the intestinal protozoan species, some are world-wide in distribution but tending to be more common in areas of poor sanitation and hygiene. These may occur in both temperate and tropical regions but some species are confined to the tropics, requiring warm soil for development or needing intermediate hosts such as water snails which can only survive in warm water.

Whether or not helminth infections cause symptoms, usually depends on the worm load and, unlike protozoan infections, worm load is usually dependant on the infective dose. [6,16,18] Another important feature of helminth infections is that of eosinophilia which usually occurs where there is a tissue invasive stage in the life cycle (as opposed to those species in which all stages are confined to the lumen of the gut).[13,16] Eosinophilia is thus often a useful clue to the presence of an invasive helminth infection and is not a feature of protozoan infections.

Trematodiases

All trematodes are transmitted from snail intermediate hosts[17,18] and are not common in the Rift Valley where there are no marshy areas or large water bodies. They are therefore not discussed further.

Cestodiases

Most adult tapeworms in the intestine cause little or no overt discomfort to the host but they may contribute to malnutrition by competition for food in the gut.[8,15]

Occasionally, however, the larger species (*Taenia* or *Diphyllobothrium*) may cause intestinal obstruction and *T. saginata* can cause a simulated appendicitis through wandering gravid proglottids blocking the appendix. Taeniasis due to *T. solium* (the Pork Tapeworm) is however particularly dangerous as the eggs passed by the human definitive (or final) host are infective not only to the pig, but also to humans by autoinfection (re-swallowing eggs passed by self) or heteroinfection (swallowing eggs passed by someone else and contaminating food or water). This can result in cysticercosis, or infection with *Cysticercus cellulosae*, the cysticercus larva of *T. solium. Diphyllobothrium latum* can cause a megaloblastic (macrocytic) anaemia in the host if attached high up in the small intestine due to competition for Vitamin B12.[10,13]

Infection of humans usually results from eating raw or undercooked fish or meat of the intermediate host animal depending on the species of tapeworm but in some species, infection may follow accidental ingestion of an insect or mite intermediate host. This can occur in conditions of poverty and/ or poor waste disposal.

Diagnosis is by finding proglottids or eggs in a faecal specimen.[10]

Nematodiases

Again, most intestinal nematode infections are asymptomatic, particularly when worm loads are low. However, the size of the adult *Ascaris* makes it liable to cause intestinal obstruction where loads are heavy, although even single migratory adult worms may cause bile duct or tracheal obstruction or intestinal perforation.[10] Heavy loads of hookworm (*Ancylostoma duodenale*, *A. ceylanicum* and *Necator americanus)* can also cause an iron deficiency (microcytic hypochromic) anaemia due to their blood sucking activities in the host small intestine, and *A.caninum* has been reported to be associated with eosinophilic enteritis.[13]

In enterobiasis, anal pruritis is common and appendiceal blockage with an appendicitis-like presentation or presentation with a vaginal discharge may follow ectopic wanderings by the adult threadworms. In trichuriasis, bloody diarrhoea can result when very heavy worm loads are encountered. Strongyloidiasis is uncommon in children.[16,18]

The trichinellids are nematodes which infect through the ingestion of meat containing the infective larvae. The adult male and female worms mature in the small intestine of the host and produce larvae which migrate through the gut wall to encyst in the muscles. During the initial stage of adult maturation in the gut, the host may suffer a transient diarrhoea, but most of the symptomatology results from the migrations and settling of the larvae in the muscles.[10,13,16] This phase results, depending on worm load, in the host developing a severe allergic/toxaemic response with myositis, oedema (especially around the eyes), eosinophilia, fever and even death. Five species of the genus are recognised – *T. spiralis* (widespread); *T. nelsoni* (Africa); *T. nativa* (Arctic);

T. britovi (Palaearctic) and *T. pseudospiralis* (widespread, but diagnosed from a human in Tasmania).

Diagnosis of intestinal nematode infections relies largely on the recovery of eggs in stool specimens, although the eggs of *E. vermicularis* need to be collected by the use of an anal tape technique.[10] Nematode eggs can usually be identified on shape and size. In the case of *S.stercoralis*, larvae are passed in the faeces and repeat stool examination or examination of duodenal fluid may be required.

The commonest diarrhoeagenic parasites in malnutrition are discussed briefly below.

E. vermicularis

E. vermicularis, commonly referred to as the pinworm or seatworm, is a nematode, or roundworm, with the largest geographic range of any helminth.[18] It is the most prevalent nematode in the United States. Humans are the only known host, and about 209 million persons worldwide are infected. More than 30 percent of children worldwide are infected.[18,20]

Adult worms are quite small; the males measure 2 to 5 mm, and the females measure 8 to 13 mm. The worms live primarily in the cecum of the large intestine, from which the gravid female migrates at night to lay up to 15,000 eggs on the perineum. The eggs can be spread by the fecal-oral route to the original host and new hosts. Eggs on the host's perineum can spread to other persons in the house, possibly resulting in an entire family becoming infected. Ingested eggs hatch in the duodenum, and larvae mature during their migration to the large intestine. Fortunately, most eggs desiccate within 72 hours. In the absence of host autoinfection, infestation usually lasts only four to six weeks.[10,13].

Disease secondary to *E. vermicularis* is relatively innocuous, with egg deposition causing perineal, perianal, and vaginal irritation.[21] The patient's constant itching in an attempt to relieve irritation can lead to potentially debilitating sleep disturbance. Rarely, more serious disease can result, including weight loss, urinary tract infection, and appendicitis.[20,21]

Pinworm infection should be suspected in children who exhibit perianal pruritus and nocturnal restlessness. Direct visualization of the adult worm or microscopic detection of eggs confirms the diagnosis, but only 5 percent of infected persons have eggs in their stool. The “cellophane tape test” can serve as a quick way to clinch the diagnosis.[10]

A. duodenale and N. americanus

Two species of hookworm, *A. duodenale* and *N. americanus*, are found exclusively in humans. *A. duodenale*, or “Old World” hookworm, is found in Europe, Africa, China, Japan, India, and the Pacific islands.[10,21]. *N. americanus*, the “New World” hookworm, is found in the Americas and the Caribbean, and has recently been reported in Africa, Asia, and the Pacific.

N. americanus ranges from 10 to 12 mm in length for females and 6 to 8 mm for males. It is distinguished from its slightly larger European cousin by its semilunar dorsal and ventral cutting plates at the buccal cavity compared with *A. duodenale's* two pairs of ventral cutting teeth. The eggs of both worms are 60 to 70 μm in length and bounded by an ovoid transparent hyaline membrane; they contain two to eight cell divisions.

Both species share a common life cycle. Eggs hatch into rhabditiform larvae, feed on bacteria in soil, and molt into the infective filariform larvae. Enabled by moist climates and poor hygiene, filariform larvae enter their hosts through pores, hair follicles, and even intact skin. Maturing larvae travel through the circulation system until they reach alveolar capillaries. Breaking into lung parenchyma, the larvae climb the bronchial tree and are swallowed with secretions. Six weeks after the initial infection, mature worms have attached to the wall of the small intestine to feed, and egg production begins.[10,13,21]

While larvae occasionally cause pruritic erythema or pulmonary symptoms during their migration to the gut, [20,21] hookworm infection rarely is symptomatic until a significant intestinal worm burden is established. A transient gastroenteritis-like syndrome can occur because mature worms attach to the intestinal mucosa.

The greatest concern from infection is blood loss. Aided by an organic anticoagulant, a hookworm consumes about 0.25 mL of host blood per day. The blood loss caused by hookworms can produce a microcytic hypochromic anemia.[21] Compensatory volume expansion contributes to hypoproteinemia, edema, pica, and wasting. The infection may result in physical and mental retardation in children. Eosinophilia has been noted in 30 to 60 percent of infected patients.

While clinical history, hygiene status, and recent travel to endemic areas can give important clues, definitive diagnosis rests on microscopic visualization of eggs in the stool.

G. lamblia

G. lamblia is a pear-shaped, flagellated protozoan that causes a wide variety of gastrointestinal complaints. Giardia is arguably the most common parasite infection of humans worldwide.[13,19] Because giardiasis is spread by fecal-oral contamination, the

prevalence is higher in populations with poor sanitation, close contact, and oral-anal sexual practices.[20] The disease is commonly water-borne because Giardia is resistant to the chlorine levels in normal tap water and survives well in cold mountain streams. Because giardiasis frequently infects persons who spend a lot of time camping, backpacking, or hunting, it has gained the nicknames of “backpacker's diarrhea” and “beaver fever.”[21]

Food-borne transmission is rare but can occur with ingestion of raw or undercooked foods. Giardiasis is a zoonosis, and cross-infectivity among beaver, cattle, dogs, rodents, and bighorn sheep ensures a constant reservoir.[21]

The life cycle of Giardia consists of two stages: the fecal-orally transmitted cyst and the disease-causing trophozoite. Cysts are passed in a host's feces, remaining viable in a moist environment for months. Ingestion of at least 10 to 25 cysts can cause infection in humans.[13,17,18]. When a new host consumes a cyst, the host's acidic stomach environment stimulates excystation. Each cyst produces two trophozoites. These trophozoites migrate to the duodenum and proximal jejunum, where they attach to the mucosal wall by means of a ventral adhesive disk and replicate by binary fission.

Giardia growth in the small intestine is stimulated by bile, carbohydrates, and low oxygen tension.[13,21] It can cause dyspepsia, mal-absorption, and diarrhea. A recent theory suggests that the symptoms are the result of a brush border enzyme deficiency rather than invasion of the intestinal wall.[18,20] Some trophozoites transform to cysts and pass in the feces.

Clinical presentations of giardiasis vary greatly. After an incubation period of one to two weeks, symptoms of gastrointestinal distress may develop, including nausea,

vomiting, malaise, flatulence, cramping, diarrhea, steatorrhea, and weight loss. A history of gradual onset of a mild diarrhea helps differentiate giardiasis or other parasite infections from bacterial etiologies. Symptoms lasting two to four weeks and significant weight loss are key findings that indicate giardiasis.

Chronic giardiasis may follow an acute syndrome or present without severe antecedent symptoms. Chronic signs and symptoms such as loose stool, steatorrhea, a 10 to 20 percent loss in weight, malabsorption, malaise, fatigue, and depression may wax and wane over many months if the condition is not treated.

Rarely, patients with giardiasis also present with reactive arthritis or asymmetric synovitis, usually of the lower extremities.[17,18] Rashes and urticaria may be present as part of a hypersensitivity reaction.

Cyst excretion occurs intermittently in both formed and loose stools, while trophozoites are almost only found in diarrhea. Stool studies for ova and parasites (O&P) continue to be a mainstay of diagnosis despite only low to moderate sensitivity. Examination of a single stool specimen has a sensitivity of 50 to 70 percent; the sensitivity increases to 85 to 90 percent with three serial specimens.[20,21] Because Giardia is not invasive, eosinophilia, and peripheral or fecal leukocytosis do not occur.

E. histolytica

Amebiasis is caused by *E. histolytica*, a protozoan that is 10 to 60 μm in length and moves through the extension of finger-like pseudo-pods.[20] Spreading occurs via the fecal-oral route, usually by poor hygiene during food preparation or by the use of “night soil” (crop fertilization with human waste), as well as by oral-anal sexual

practices. Spreading is frequent in persons who have a deficient immune system. Crowding and poor sanitation contribute to its prevalence in Asia, Africa, and Latin America. Approximately 10 percent of the world's population is infected, yet 90 percent of infected persons are asymptomatic.[17,20,21] Of the roughly 50 million symptomatic cases occurring each year, up to 100,000 are fatal.[21] The stable reservoir of infective cases complicates eradication. After malaria, it is likely that *E. histolytica* is the world's second leading protozoan cause of death.[20,21]

Much like Giardia, the two stages in the *E. histolytica* life cycle are cysts and trophozoites. Infective cysts are spheres of about 12 μm in diameter that have one to four nuclei and can be spread via the fecal-oral route by contaminated food and water. The pseudopodal trophozoite is about 25 μm across, has a single nucleus, and may contain red blood cells of the host in various stages of digestion. Ingested cysts hatch into trophozoites in the small intestine and continue moving down the digestive tract to the colon. Also like Giardia, some ameba trophozoites become cysts that are passed in the stool and can survive for weeks in a moist environment. However, trophozoites can invade the intestinal mucosa and spread in the bloodstream to the liver, lung, and brain.[20]

Amebiasis can cause both intraluminal and disseminated disease. In the intestinal lumen, *E. histolytica* can disrupt the protective mucus layer overlying the colonic mucosa. The resulting epithelial ulcerations can bleed and cause colitis,[17,20] usually two to six weeks after initial infection. Acute progression to malaise, weight loss, severe abdominal pain, profuse bloody diarrhea, and fever can occur, often leading to a misdiagnosis of appendicitis, especially in children. In chronic smoldering cases, inflammatory bowel disease can be misdiagnosed, and treatment with steroids only exacerbates the infection.

Rarely, a reactive collection of edematous granulation and fibrous tissue called an ameboma can grow into the lumen, causing pain, obstruction and, possibly, intussusception. Toxic megacolon, pneumatosis coli (intramural air), and peritonitis also may occur.[17,19]

Tissue penetration and dissemination are possible.[10] Trophozoites that penetrate the intestinal wall spread through the body via the portal circulation. Amebas are chemotactic, attracting neutrophils in the circulation. Amebic liver abscesses form because of toxin release and hepatocyte damage, and usually develop within five months after infection. Symptoms of a developing abscess include fever, dull pleuritic right upper quadrant pain radiating to the right shoulder, and pleural effusions. Diarrhea is present in only one of three patients with abscess. Fever is the presenting symptom in 10 to 15 percent of patients, and therefore amebic abscess should be considered in patients with a fever of unknown origin. Abscesses may rupture into the pleural space, peritoneum, or pericardium, requiring emergency drainage.

Traditional O&P stool testing for amebiasis should use at least three fresh samples to increase sensitivity. Positive stool samples are likely to be heme positive and to have low neutrophils but may contain Charcot-Leyden crystals, indicating the presence of eosinophils. Biopsy of colonic ulcer edges may yield intramural trophozoites but carries with it the risk of perforation.[10,13,21]

2.5 Treatment of Malnutrition and Intestinal Parasites

Apart from managing diarrhea and its attendant complications, other complications of malnutrition should be attended to optimize patient care. Treatment involves stabilization for the first 7 days (Hypoglycaemia, hypothermia, dehydration, infection

treatment and initial feeding) and rehabilitation (Electrolytes, micronutrients, sensory stimulation, feeding to achieve catch-up growth) over the next 5 weeks.

Emergency management is outlined in the table below.

Table 1: General Management of the severely Malnourished Child:

Treat shock Shock is if the child is lethargic **or** unconscious and cold hands **Plus either**: Slow capillary refill (longer than 3 seconds) **or** Weak fast pulse Monitor closely: use the Critical Care Pathway Initial Management Chart	**If child is in shock:** 1. Give oxygen 2. Give sterile 10% glucose (5ml/kg) by IV 3. Give IV fluid at 15ml/kg over 1 hour, using: • Ringers' lactate with 5% dextrose or • half-normal saline with 5% dextrose or • half-strength Darrow's solution with 5% dextrose • if all of the above are unavailable, Ringer's lactate 4. Measure and record pulse and respirations every 10 minutes **If there are signs of improvement** (pulse and respiration rates fall) repeat IV 15ml/kg for one more hour **If there are no signs of improvement** assume child has septic shock. In this case: 1. Give maintenance fluids (4ml/kg/h) while waiting for blood 2. Order 10ml/kg fresh whole blood 3. Refer child to ward quickly for slow transfusion
Treat severe dehydration Assume severe dehydration if there is profuse watery diarrhoea and signs such as sunken eyes, slow skin pinch, absent tears, dry mouth, very thirsty, reduced urine output, rapid pulse and respirations.	**DO NOT GIVE IV FLUIDS EXCEPT IN SHOCK** 1. Give ReSoMal 5ml/kg every 30min for 2 hours. Do not give standard ORS to severely malnourished children 2. Measure and record pulse and respirations every 30 minutes. 3. Refer child to ward quickly for continued rehydration: give ReSoMal 5-10 ml/kg/hour for next 4-10 hours. **STOP** rehydration if 3 or more signs of rehydration or any signs of overhydration (increased respiratory rate and pulse rate, increase oedema and puffy eyelids). Only give ReSoMal for **up to 10 hours.**
Treat very severe anaemia Very severe anaemia is Hb	**If very severe anaemia** (or Hb 4-6g/dl AND respiratory distress)**:** 1. Give whole blood 10ml/kg body weight slowly over 3

less than 4g/dl	hours. If signs of heart failure, give 5-7ml/kg packed cells rather than whole blood. 2. Give furosemide 1ml/kg IV at the start of the transfusion
Treat hypoglycaemia Hypoglycaemia is a blood glucose <3mmol/L Assume hypoglycaemia if no dextrostix available	**If hypoglycaemia and conscious:** Give 10% glucose (50ml) or sugar solution (1rounded teaspoon sugar in 3 tablespoons of water). 10% glucose is best, but give sugar solution rather than wait for glucose. **If unconscious:** Give glucose IV (5ml/kg of sterile 10% glucose), followed by 50ml of 10% glucose or sucrose by NG tube. Then refer child to ward quickly to begin feeding straightaway.
Treat hypothermia Hypothermia is a rectal temperature <35.5^{0}C (95.9^{0}F) or an underarm temperature <35^{0}C (95^{0}F).	**If hypothermia:** 1. Give 10% glucose as above and refer child to ward quickly to begin feeding straightaway. 2. Keep warm. Put the child on the mother's bare chest (skin to skin contact) and cover them. Ensure child is covered when being transported to ward.
Emergency Eye Care Corneal Ulceration	**If corneal ulceration:** 1. Give Vitamin A immediately (<6 months 50,000IU, 6-12 months 100,000 IU, >12 months 200,000IU) 2. Instil one drop atropine (1%) into affected eye to relax the eye and prevent the lens from pushing out.

Treatment of intestinal parasites may often be desirable alongside food supplementation. WHO recommends that in areas where the prevalence of mild to moderate underweight in children is greater than 25%, and where parasites are known to be widespread, high priority should be given to deworming programmes for treatment of parasites.[4,19] It is also of particular priority in vitamin-A deficient areas. Cases of severe malnutrition are also frequently suffering from intestinal parasite infestation, which should therefore be treated as part of nutritional rehabilitation. In one study, more than 20 percent of diarrhoeas in infants with severe malnutrition were

dramatically improved by specific treatment of S. stercoralis or Giardia with thiabendazole or metronidazole.[16, 18] The summary of drugs used and dosages is as below:

Protozoans: where indicated, common drugs are Metronidazole 15mg/kg tds x 5-10 days and Tinidazole 2g/d x 3-5d or 600-800 mg/d x 5 days. Albendazole (400 mg bd x 21d) can be used for *Giardia* and the microsporidia while co-trimoxazole (160 mg TMP; 800 mg SMX OD x 10d, then BD x 21d) is used for *Cyclospora* and azythromycin (1200 mg/d x 27d) for *Cryptosporidim*

Trematodes: A good broadspectrum anthelmintic for most trematode infections, including schistosomiasis, is praziquantel (10-20mg STAT) or Niclosamide 2g stat.

Cestodiases: Praziquantel (25mg/kg single dose) is effective for treatment of most species. Niclosamide can be used as an alternative.

Nematodiases: Good broadspectum anthelmintics for intestinal nematodes include Mebendazole (<10kg 50mg bd x 3d; > 10kg 100mg bd x 3d), Albendazole (<10 kg 200mg stat; > 10kg 400mg stat) and Pyrantel emboate (11-20mg/kg STAT). *Trichuris* and *Strongyloides* tend to be more difficult to eradicate and are best treated with albendazole.

Albendazole/mebendazole+corticosteroids is recommended for the treatment of trichinosis, which is diagnosed by muscle biopsy or by antibody serology.

CHAPTER THREE

3.1 Justification:

Diarrhea and malnutrition are a leading cause of childhood morbidity and mortality as elucidated above. In 1990s the prevalence and incidence of severe malnutrition declined but despite this, severe malnutrition remains an important problem.[5]

Human intestinal parasites occur throughout the world but it is in the wet tropics and subtropics where they are found in their greatest numbers.[6, 7, 8] A basic requirement for the continued survival of these organisms is an inadequate an unhygienic method of

waste disposal. Consequently, intestinal parasites reach their highest prevalence and intensity in impoverished parts of the world, the same areas where malnutrition in children remains a major problem. Since we live in the subtropics where poverty and poor hygiene are common, and parasitoses are endemic and therefore infection with the same is the rule.[7,10]

Parasite infestation and malnutrition do frequently co-exist, the extent to which is unknown.[10] This study has attempted to establish prevalence and associated clinical and socio-demographic factors. Intestinal parasites can directly cause malnutrition and other life-threatening complications like anaemia and multiorgan damage from fibrosis of intestinal walls, liver, spleen and other organs.[10] They can also cause intestinal obstruction by *Ascaris* worms, migratory worms may cause bile duct or tracheal obstruction or intestinal perforation or appendiceal blockage.[6] These are serious complications which could be avoided by concerted efforts to diagnose specific parasites and give appropriate therapy especially in the malnourished in whom these infections are especially serious.[8]

About 10 percent of diarrheal episodes in less developed countries lead to persistent diarrhea and require specialized treatment in addition to rehydration therapy.[2] this type of diarrhea is commonly due to parasites and it is associated with greatest mortality among affected children; and stool examination for giardiasis is indicated if there is poor weight gain despite good food intake.[8]

This study therefore aims to demonstrate the disease burden of intestinal parasites in order to call for greater efforts in management of affected children by both health care providers and the community at large. We live in a zone where the prevailing

environmental and social factors promote malnutrition and infestation with parasites, the two conditions which when co-existent lead to deterioration of health, poor cognition and long term complications including death. While these patients remain in poor health and economically non-productive, the country's resources are spent to cover their hospital costs thus retarding general development. Effective management of diarrhea helps prevent future illness, including diarrhea, since maintenance of good nutritional status helps to maintain immune-competence. A population that is healthy is a productive population. Thus there are important implications for both prevention and management of parasitic diarrhea in malnourished children.

3.2 Research Question:

What is the Prevalence of intestinal parasite infestations among severely malnourished children?

What are the risk factors associated with intestinal parasites in severely malnourished children?

3.3 Objectives

Broad:

To determine prevalence of intestinal parasites and factors associated with infestation in severely malnourished children admitted at MTRH.

Specific:

1. To determine the prevalence of intestinal parasites in severely malnourished children admitted to the children's wards of Moi Teaching and Referral Hospital.

2. To describe clinical and socio-demographic characteristics associated with intestinal parasite infestation in these children

3. To determine the clinical and socio-demographic factors associated with intestinal parasitic infestations among these children.

CHAPTER FOUR

METHODOLOGY

4.1 Study design

This is a cross-sectional descriptive study.

4.2 Study site

The study was carried out in the pediatrics wards of MTRH in Eldoret, Kenya. Eldoret is a town in western Kenya and the administrative centre of Uasin Gishu county of

Rift Valley Province. Lying south of the Cherangani Hills, it has an average elevation of 2095 metres. It is currently the fastest growing town, and the 5th largest town in Kenya. Eldoret is 300 kilometers northwest of the capital Nairobi. The county of Uasin Gishu has 3 constituencies: Eldoret North, Eldoret South and Eldoret East. The county had a 2009 population (Kenya National Bureau of Statistics) of 894,179, with 548,620 people living in rural areas and 345,559 living in urban areas. The main economic activity in the region is agriculture, with large scale farming of wheat and maize. MTRH serves as a teaching hospital for Moi University's schools of medicine, dentistry and nursing and other medical institutions in the Region. It is also the referral hospital serving the western region of Kenya, with a catchment population of approximately thirteen million. The general wards admit about 400 children per month with general medical conditions with an average number of 10-20 severely malnourished children per month. Other conditions include pneumonia, malaria, diarrhoeal diseases and an oncology unit. The prevalence of malnutrition and IPIs are not known.

4.3 Study population

The study population was severely malnourished children admitted to the paediatrics wards of MTRH during the study period.

4.4 Eligibility criteria

The sample population was constituted by children admitted to the paediatrics wards during the study period with severe malnutrition (by Wellcome Classification) aged between 6 and 59months whose guardians gave consent

4.5 Exclusion Criteria

The children who had known underlying chronic illnesses like diabetes, thyroid disorders and malignancies were excluded from the study.

4.6 Sample Size Determination:

Sample size was mainly computed using fisher's formula.

Where;

n = Is the required study population, which is all children admitted during the study period who had severe malnutrition.

Where;

$n =$ Is the anticipated sample size to be considered for the study

$Z_{\alpha/2} = 1.96$, standard normal variate

$p =$ Estimated prevalence of intestinal parasites in severely malnourished admitted children (Ecuador , in which the study subjects characteristics were most similar to the current study) is 90% .

$d =$ Margin of error at 5% (standard value of 0.05)

Calculating sample size yields the following figure;

$$n = \frac{1.96^2 \times 0.90 \times 0.10}{0.05^2}$$

$$n = \frac{0.3457}{0.0025}$$

$$n = 139, subjects$$

Adjusting for 5% non-response yields

$$n = \frac{139}{0.95} = 146, subjects$$

4.7 Adjusting for finite population correction.

The sample size (n) was adjusted using Equation shown below;

$$n_o = \frac{n}{1 + \frac{n - 1}{N}}$$

Where n_o the required sample size and N is the population size.

$n_0 = \frac{146}{1 + \frac{146 - 1}{1000}} = 128$. We therefore approximated study sample to 130 subjects after adjusting for finite population.

4.8 Sampling procedure:

Consecutive patients admitted who met the eligibility criteria were recruited into the study until the study sample size was achieved over 8 months. This involved taking anthropometric measurements of all children to determine their nutritional status and the severely malnourished children who were eligible admitted into the study. A questionnaire was administered to the guardians of the children and the stool specimens collected and analyzed for presence of parasites.

4.9 Data Collection Instruments and procedures:

Research assistants were trained by the investigator before commencement of the study. The two-day training included understanding the nature of the study, the study population, inclusion and exclusion criteria and ethics to be observed during interview and recruitment of subjects for the study. All patients admitted were screened for severe malnutrition by weighing them all and weight for age tabulated (Wellcome Classification). Those who met the inclusion criteria (severe Malnutrition, aged 6-59months and guardians consented) were recruited into the study. The nature and aim of the study was explained to parents/guardians of prospective participants and consent obtained, before questionnaires were administered.

Data was collected using data collection sheet(see annex) filled by research assistants and/or the principal investigator by questioning parents/guardians, to collect dietary, sanitation, demographic data and anthropometric measurements of patients (See Wellcome Classification of Malnutrition annex). The data collection sheet also collected clinical history of presenting illness to help determine factors that could be associated with patient's condition. Stool samples were then collected before the children were dewormed in the wards, as this would have tampered with study results. For this purpose, the caretaker was given a pair of clean gloves a water-tight labeled container and an applicator stick with which to pick patient's stool after it was passed in a wider container or potty, to take 2 scoops (approximately 15 grams) into the container provided, for transportation to the laboratory. The stool specimens were collected within one hour of collection and taken to the laboratory for analysis (diarrhoeic stools were examined immediately when it was possible for presence of trophozoites of protozoa. Normal stools preserved in 10% formal saline awaiting analysis using formal-ether concentration technique by laboratory technicians all within one week to look for Ova and Cysts which stayed viable after fixation). Mostly the diagnosis was done by presence of Ova and Cysts.

The patients' ages, weights, expected weights and clinical data were recorded on the space provided on the questionnaire. Regular calibration of the weighing machine was done by technicians to certify correctness. Study numbers and not patients' names appeared on the questionnaires. .Interviewers submitted completed instruments to the principal investigator at the end of each working day. Filled questionnaires were sorted out and edited on a daily basis. They were also checked for completeness and appropriateness by the researcher.

The microscopes for use in stool examination were regularly calibrated, cleaned daily to remove contaminants and checked to ensure good working order before use each time. The stool samples were analysed using the Formol-Ether concentration method and analysed by two identified laboratory technicians examining the samples separately to minimize individual errors, another independent technician, acting as tie-breaker, looking at the slides only when the results of the two primary technicians differed. Results for microscopy for Ova and Cysts were entered in another sheet with study numbers matching the one in questionnaire for each patient. (See laboratory request form and laboratory procedure for stool analysis, Annex).

4.10 Data Analysis:

Data was entered in SPSS then exported to STATA version 11.0 which was used to perform all analyses. Categorical variables were presented in form of frequency tables and bar graphs while continuous variables were mainly summarized using means. Chi-square test was used to compare categorical variables. Fisher's exact test was also used to compare categorical variables where some cells had expected value of less than 5. Bivariate analysis was performed by comparing study subjects with diarrhoea and those without in terms of type of malnutrition and presence of parasites. Logistic regression model was additionally fitted to assess factors associated with parasite infestation. Level of significance was set at $p < 0.05$.

4.11 Ethical Considerations

Approval to carry out the study was sought from IREC before commencement.

Permission to undertake the study in MTRH was obtained from the hospital's administration. Also informed consent was obtained from the parents/guardians of patients participating in the study.

The participants as well as those patients whose parents did not give consent received the same level of care accorded to other patients. There were no immediate benefits to the participants, and the guardians were informed in advance. The parents/ guardians were provided with gloves and spatula for specimen collection and air-tight container for storage to ensure safety. Information obtained was handled with confidentiality. No names were used in any of the data collection and/or entry forms, only serial numbers. The information obtained was stored safely and only accessed by the investigator for the intended purpose. The results of this study shall be availed to the Moi Teaching and Referral Hospital for use.

CHAPTER FIVE

RESULTS

5.1 Socio-demographic Characteristics of Study Subjects

A total of 130 children were studied. The median age among study subjects in this population was 2 years (Range of 1-3 years). The male to female ratio was 1:1.24. The guardians' level of education ranged from none to secondary level, majority attaining primary level of education. Majority of the guardians' occupation was in unskilled labor, and the mean number of dependants was 4 (IQR=3-11) (***Table 1***).

Table 2: Demographic Data of study subjects

Characteristic	**n, (%)**
Child characteristic	
Median age (Range)	2 (1-3)
Gender	
Male	58 (44.6)
Female	72 (55.4)
County of residence	
Uasin Gishu	106 (81.5)
Nandi	9 (6.9)
Busia	1 (0.8)
Other	14 (10.8)
Parent/Guardian	
Mean Age (sd)	26.64 (4.65)
Occupation	
Farming	7 (5.4)
Business	18 (13.9)
Unemployed/ Casual day labour	79 (60.8)
Employed skilled labor	26 (20.0)
Marital status	
Single	35 (26.9)
Married	61 (46.9)

Separated	34 (26.2)
Education level	
Uneducated	30 (23.1)
Primary	73 (56.2)
Secondary	27 (20.8)
Median number of dependants, (range)	**4 (3-11)**

The prevalence of Diarrhoea in the study population was 68.5% as seen in the chart below.

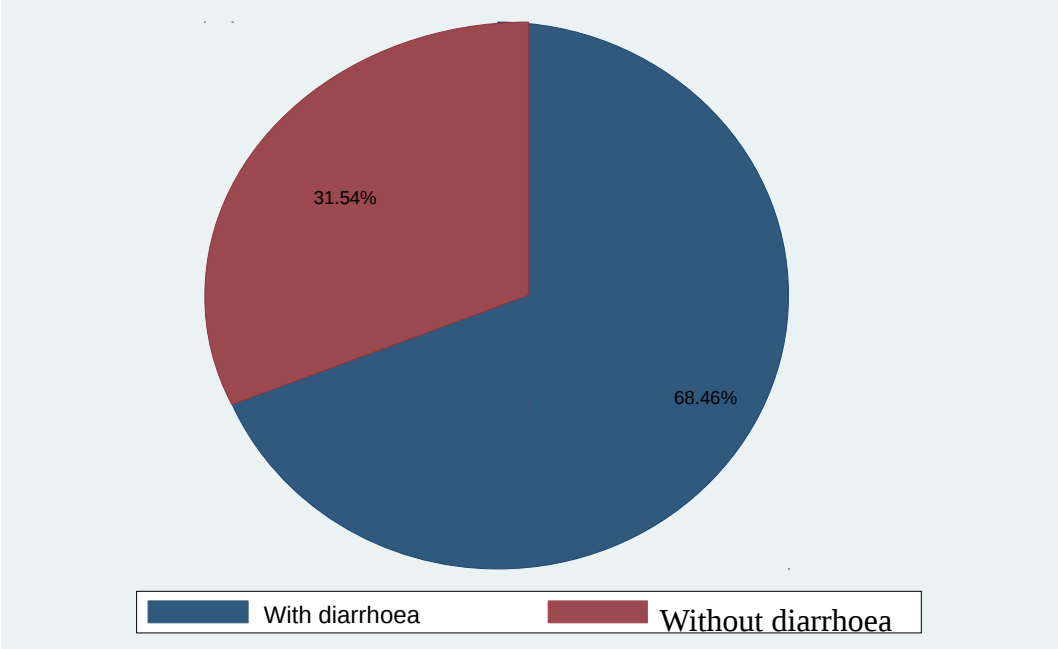


Figure 2: Proportions of Subjects with and without diarrhoea

5.2 Malnutrition Types and presence of Diarrhoea

A higher proportion of those with diarrhoea had edematous types of malnutrition. The association between Kwashiorkor and diarrhea was statistically significant (p=0.0491). Most subjects without diarrhoea were marasmic. (***Table 2***).

Table 3: Malnutrition Types in subjects with and without diarrhoea.

Clinical condition	With diarrhoea,	Without diarrhea	P-value

	89, n (%)	n=41 (%)	
Type of severe malnutrition	n=89	n=41	
Kwashiorkor	19 (21.4)	5 (10.0)	
Marasmus	38 (42.7)	27 (65.8)	
Marasmic-Kwashiorkor	32 (36.0)	9 (22.0)	0.0491[2]

[2]**Chi-square test**

5.3 Diarrhoeal Characteristics of the study Subjects

There were almost similar proportions of subjects with acute and chronic diarrhea. Majority subjects were reported to pass profuse and watery stools. Majority had previous episode of diarrhoea in the three months prior, and had not been dewormed. A large proportion was given over-the-counter drugs as first-Aid for the diarrhea and only 6% gave ORS before admission. (***Table 3 and 4 below***).

Table 4: Diarrhoeal History in Subjects with Diarrhoea.

Condition	n=89, (%)
Length of current episode	
< 2 weeks	45 (50.6)
>2 weeks	43 (48.3)
Nature of stools passed	
Mucoid	24 (27.0)
Bloody	15 (16.9)
Profuse and watery	47 (52.8)
Had worms	3 (3.4)

Table 5: Diarrhoeal recurrence and Management in all subjects

Diarrhoea history	n=130 (%)
Previous episode in the past 3/12	
Yes	103 (79.2)
No	27 (20.8)
Dewormed in past 3 months	
Yes	29 (22.3)
No	101 (77.7)
First-Aid measure for diarrhea before admission	
None	44 (33.8)
Plain Water	31 (23.8)
Over the counter medications	47 (36.2)
Oral Rehydration Solution	8 (6.2)

5.4 Sanitation and Nutritional Characteristics

Majority of the study subjects used tap water, and more than half the guardians reported treating it before use and also majority washed their raw foods before eating. (***Table 5***).

Table 6: Water and Sanitation Practices Among all Subjects

Social and hygiene practices	n,%
Source of water for daily use	
Tap	80 (61.5)
Borehole	41 (31.5)
River	9 (6.9)
Treatment of drinking water	
Yes	76 (58.5)
No	54 (41.5)
Distance of toilet from water source(metres)	
>=50	53 (40.8)
< 50	77 (59.2)
Wash raw foods before eating	
Yes	116 (89.2)
No	14 (10.8)

Most subjects were exclusively breastfed for less than 4 months. Diet was adequate in slightly less than half of all study subjects (***Table 6)***.

Table 7: Nutritional History Among all subjects

Nutrition	n,%
Length of exclusive breastfeeding	
< 4months	75 (57.7)
>=4months	55 (42.3)
Frequency of meals	
Once	6 (4.6)
2-4 times	71 (54.6)
>5 times	53 (40.8)
Amount	
Adequate	56 (43.1)
Inadequate	74 (56.9)

Table 8: Association Between Guardian Education level and Malnutrition Type

Kwashiorkor was a common malnutrition type among children of uneducated guardians as seen in this table.

	Malnutrition type, n (%)			
Education level	**Kwashiorkor**	**Marasmus**	**Marasmic-Kwashiorkor**	**P-value**
Uneducated	18 (75.0)	3 (4.6)	7 (17.1)	
Primary	6 (25.0)	43 (66.2)	23 (56.1)	
Secondary	0 (0.0)	19 (29.2)	11 (26.8)	<0.0001[1]

[1]**Fishers exact test**

Table 9: Distribution of Specific Parasites in the study population.

Type of parasite/absence	n (%)
Parasites species present	**31(24)**
Giardia lamblia	9(6.9)
Entamoeba histolytica	4 (3.0)
Entamoba coli	4 (3.0)
Chilomastix mesnilii	3 (2.3)
Ascaris lumbricoides	4 (3.0)
Strongyloides stercoralis	2 (1.5)
Ancylostoma duodenale	2 (1.5)
Taenia solium	2 (1.5)
Taenia saginatum	1 (0.8)

The prevalence of intestinal parasites was 24% among the study subjects. The other 99(76%) did not have parasites. *Giardia lamblia* was the commonest protozoan while *Ascaris lumbricoides* was the commonest helminth.

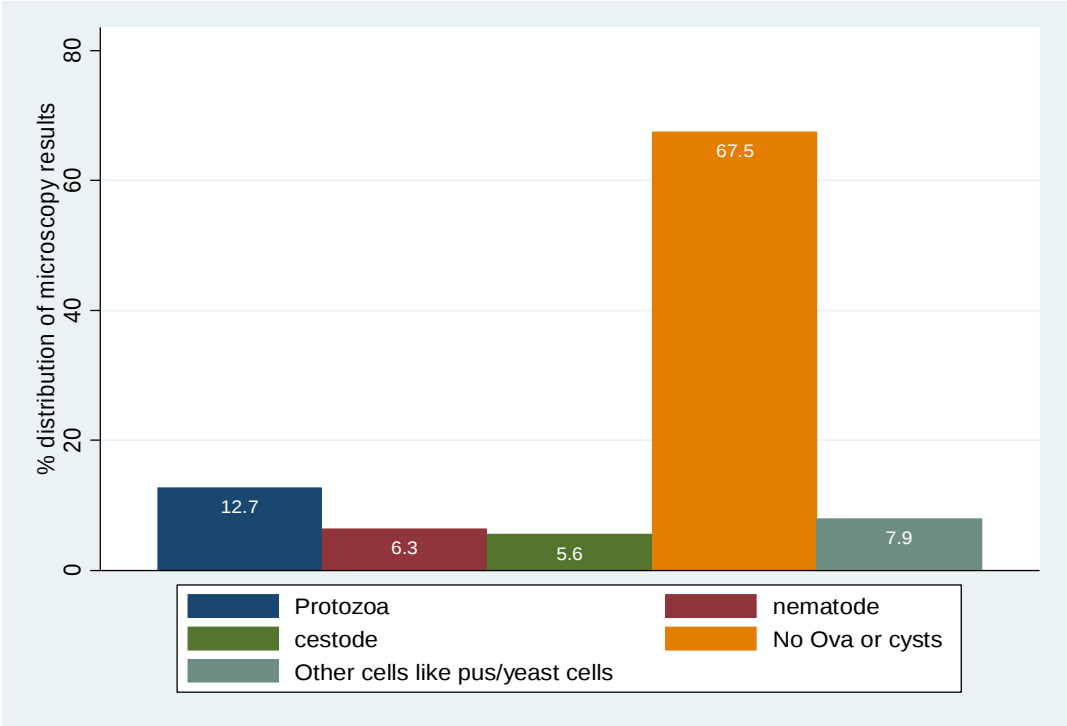


Figure 3: Distribution of Parasite Types among all Study Subjects

Most subjects had no parasites in their stools. The commonest parasites isolated were protozoa.

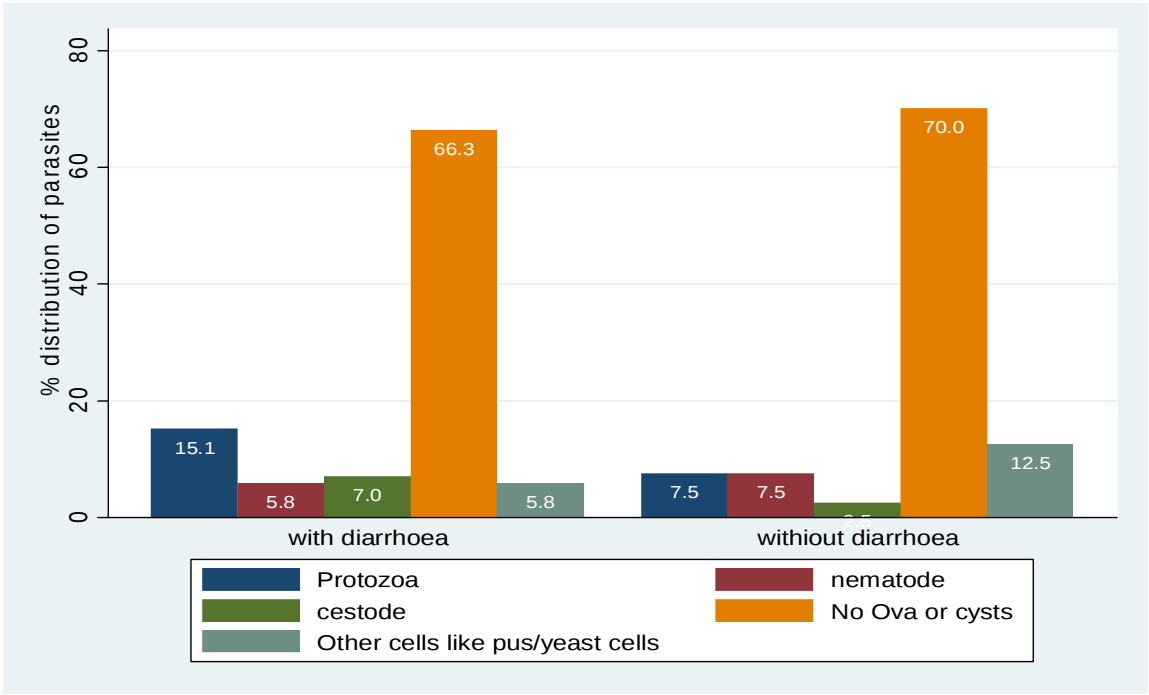


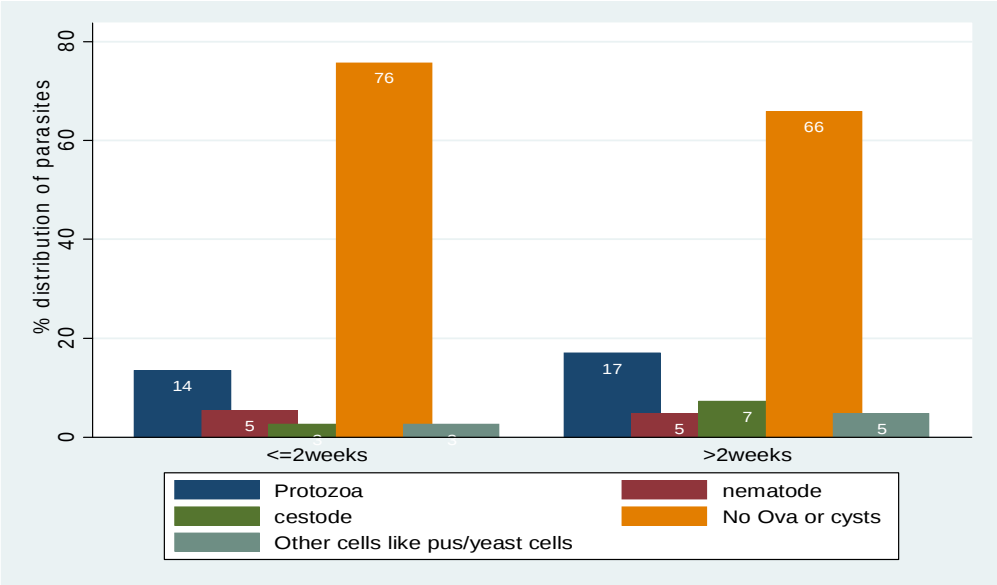
Figure 4: Parasite types among Subjects with/without diarrhoea

The subjects with diarrhoea had parasites more often than those without diarrhoea. Protozoa were commonest among those with diarrhea while nematodes were increased in those without diarrhoea.

Table 10: Association between presence of diarrhoea and parasite infestation among study subjects

A total of 31 subjects had intestinal parasites, forming a prevalence of 24% of all the 130 children studied. Majority of these subjects with parasites also had diarrhoea but the association was not statistically significant (p=0.219; OR=1.76, 95%CI=0.22-1.43).

	Presence of parasite infestation			
Presence of diarrhoea	**Yes, n(%)**	**No, n(%)**	**P-value**	**OR (95%CI)**
Yes	24 (77.4)	65 (66.7)		
No	7 (22.6)	34 (34.3)	0.219	1.76 (0.22-1.43)



Fisher's exact test, $p = 0.897$

Figure 5: Association of Duration of diarrhea with parasite Type

In figure above, Intestinal parasites were more prevalent in subjects with chronic diarrhoea. Protozoans were the commoner parasites in both groups but mostly found in subjects with chronic diarrhea.

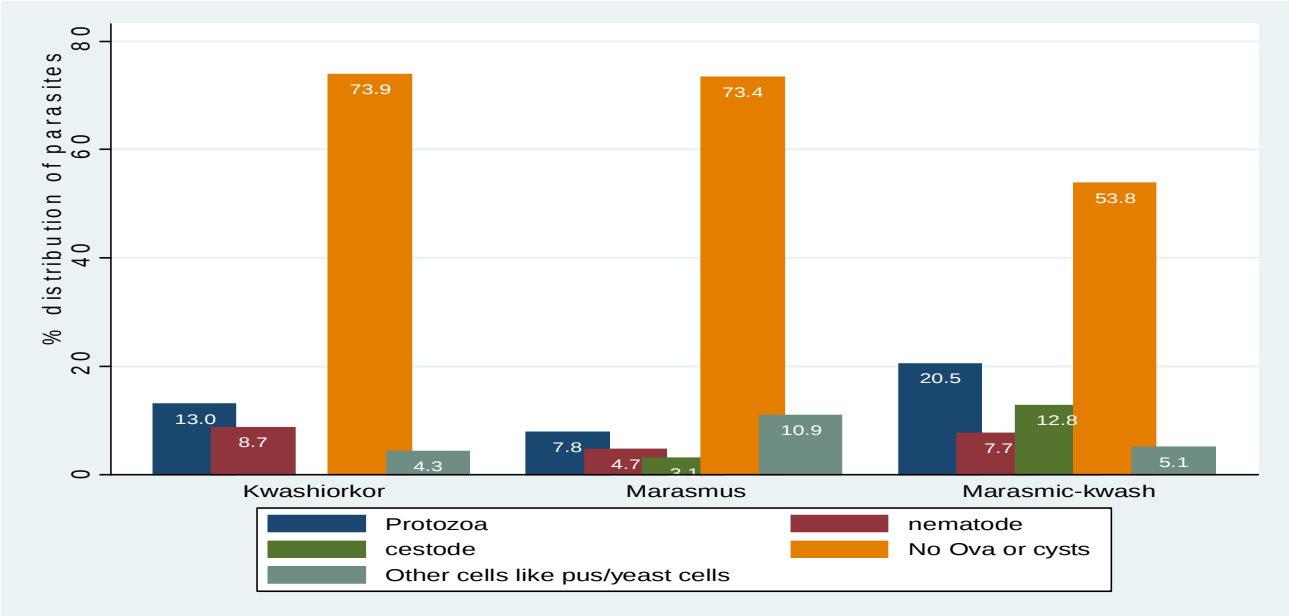


Figure 6: Malnutrition types versus Types of Parasites

In the figure above, we can see that parasite prevalence was highest among subjects with Marasmic-Kwashiorkor, commonly protozoa.

Table 11: Unadjusted and adjusted logistic regression model of factors associated with parasite infestation.

	Unadjusted			Adjusted		
Covariate	**P-value**	**O.R**	**95% CI**	**P-value**	**O.R**	**95% CI**
Guardian age in years	0.684	1.02	0.937-1.050	0.196	1.08	(0.962 - 1.211)
Gender (male vs. female***)**	0.944	0.97	0.432-2.186	0.557	0.53	(0.062-4.478)
Marital status						
(married vs. single*)	0.292	1.76	0.616-5.017	0.855	0.86	(0.166 - 4.435)
(separated vs. single*)	0.385	1.67	0.524-5.341	0.84	0.82	(0.112 - 5.939)
Educational level						
(Primary vs. uneducated*)	0.931	1.05	0.363-3.023	0.322	0.46	(0.101 - 2.122)
(Secondary vs. uneducated*)	0.458	1.57	0.476-5.184	0.577	2.17	(0.143 - 33.009)
Water source for daily use						
(Borehole vs. tap*)	0.929	1.04	0.418-2.597	0.611	1.52	(0.305 - 7.516)
(River vs. tap*)	**0.034****	4.63	1.120-19.159	**0.032****	**10.89**	(1.227 - 96.599)
Treatment of water before drinking						
(No vs. Yes*)	0.232	0.6	0.254-1.394	0.669	1.51	(0.227 - 10.029)
Distance of toilet from water source (metres)						
(<50 vs. >50*)	0.88	0.94	0.414-2.128	0.556	1.45	(0.419 - 5.041)
Washing raw foods before eating (No vs. yes***)**	0.382	0.5	0.106-2.367	0.418	0.32	(0.021 - 4.973)

* Baseline category
**significant level ($p<0.05$)

Multivariate logistic regression model

When adjusted for other factors, water source remained significantly associated with parasite infestation. Obtaining water for daily use from the river was associated with 11-fold higher chance for presence of parasite infestation (AOR=10.89, 95% CI=1.227-96.599). Distance from toilet to water source of less than 50m was also

associated with a higher risk for parasite infestation though the association was not statistically significant (AOR=1.45, 95% CI=0.419-5.041) (**table 9**).

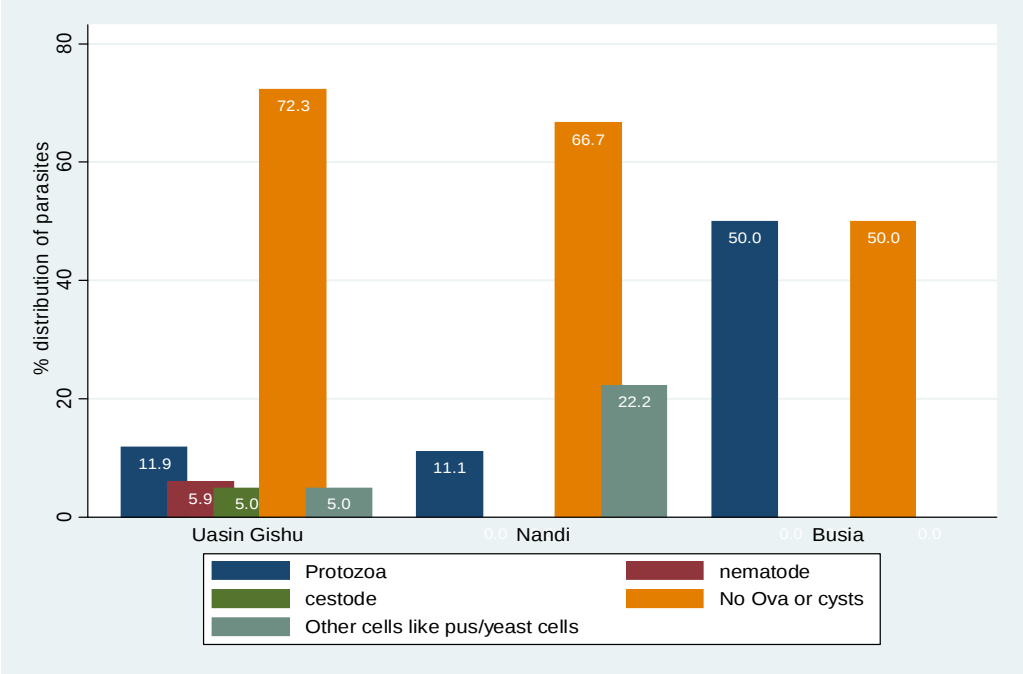


Figure 7: Distribution of parasite type by County of Residence

Prevalence of protozoa was highest and the only parasite found in subjects from Western region.

CHAPTER SIX

DISCUSSION

Parasitic diarrhea in the malnourished is common and severe. The relationship between parasitic infestation and malnutrition is bidirectional, parasites cause

malnutrition while malnutrition aggravates the course of diarrhea due to parasites.[16,20,22]

6.1 Demographics:

A total of 130 children aged between 6-59 months were recruited into the study. Most of the patients studied were aged between one and three years, with a mean age of 26 months; majority of whom were females, 72 (55%). Most children were above one year likely due to increased environmental explorative tendencies among this age group, unlike those under one year, thus predisposing them to parasites. This is also the time when most children have been completelely weaned off breastfeeding and have newborn siblings, so the feeding is erratic and are prone to acute malnutrition.[20,22] In a community-based study in under 5-year-old Sudanese children, those over 3 years of age were most affected.[24] Female sex was associated with severe PEM in a prior study in this hospital in 2001, [25]

Most patient caretakers were young with a median age of 26.7 years, were mostly unemployed or did casual labor 79(60.8%), were single or separated 69 (53%), had attained only primary school education (56.2%), and had a high number of dependants (mean of 4). These are all indicators of low economic power associated with severe PEM. All these indicators have been shown in several studies to be positively correlated to presence of severe malnutrition.[25 ,26] In the Sudan study, malnutrition rates were highest among the illiterate, overcrowded and large-sized families.[24]

6.2 Malnutrition type:

Majority of the patients studied had maramus, 65(50%), followed by marasmic-kwashiorkor 41 (32 %) and kwashiorkor 24(18%). This pattern of distribution is

similar to that seen in malnourished children admitted in Basrah hospital in Iraq where 84% had marasmus, 8.2% had marasmic-kwash and 6.4% had kwashiorkor.[27]

Diarrhea was common among children with kwashiorkor(22% vs 10%) and marasmic-kwash (42% vs 26%) compared to those patients with marasmus (64% vs 36%); the association of diarrhea with kwashiorkor and with marasmic-kwash was statistically significant (P-Value 0.0491). This is because kwashiorkor and marasmic-kwash are the decompensated forms of severe malnutrition and they get multiple infections and malabsorptive syndromes making them more prone to diarrhea. The marasmic patients are patients suffering general starvation and therefore have less diarrhea.[11, 14]

6.3 History of Diarrhea and other clinical symptoms:

Majority study subjects had diarrhea (68%) while 32% did not. Diarrhea is a common manifestation of parasite infestation in the malnourished due to higher prevalence of gut barrier dysfunction[17,20].

Among those with diarrhoea, 51% had acute diarrhoea while 48% had chronic diarhoea. This is because all the subjects were severely malnourished with diminished immunity and therefore were all prone to diarrhoeal diseases of similar magnitude. The percentage with chronic diarrhoea however, is far higher in this population than in the eutrophic population where only 3-20% is said to develop chronic diarrhea.[14,28,29]

Majority of the study subjects (79.2%) had had previous episodes of diarrhea and only a minority had been dewormed three months prior (22%). This further shows how low immunity in the malnourished children predisposes them to persistent and recurrent

intestinal and other infections[10, 15, 16]. The low coverage of deworming is probably due to health-seeking behavior among this population. First Aid measures for diarrhea provided by the caretakers were also poor as most of them gave nothing at all (34%), Plain water (23%) or over-the-counter medications (36%) while only 6% gave ORS as recommended by WHO. Use of ORS is way below the WHO survey that ORS use was at 20% in 1999.[1]

6.4 Social Conditions and Dietary Practices:

Dietary practices were similar in both groups with and without IPIs but for unexplained reasons, more subjects with diarrhoea (44%) than those without (38%) were breastfed for longer than 4 months and also treated their water more often (62% versus 58%). Breastmilk is the best and safest food source for children and strengthens the child's immune system and confers protection against infections.[34,36] Less than half of the children in this study were exclusively breast-fed for more than 4 months. No significant association was found between breast feeding and parasitic infection which is comparable to studies done in central Africa and Bangladesh.[37,40,41] Though a study done in Egypt showed less incidence of giardiasis in children exclusively breast-fed for six months.[44]

There was an association, though not significant, between intestinal parasitic infections and the occupational status of the guardians. The higher prevalence was noticed among those with employed guardians. This may be explained by the amount of time spent in child caring and supervision in relation to personal hygienic habits which is limited in case of working guardians[20]. In the current study, higher guardian education was associated with lower prevalence of intestinal parasitic infection from 42.8 % (for illiterate) to 23.3% children of secondary educated guardians. These

observations were in agreement with findings reported from other countries like India[31], Ethiopia[34] and Nepal.[35].

Males and females had almost equal infection rates infection rates of 32% and 30% respectively but without significant difference ($p>0.05$), which suggested that parasitic diseases were independent of sex in this region. Similar findings were reported in general population of Nepal,[42] but in other studies in Ethiopia[45] and Cameroon[46] , boys were found to have significantly more IPIs associated with poorer personal hygiene in the boys.

6.5 Prevalence of intestinal Parasites and associated Risk Factors:

A significant proportion of study subjects had parasites in their stools (24%) while in 76% no parasites were isolated. This was similar to prevalence found in a study at Jos University teaching Hospital in Nigeria where 29% had intestinal parasites. Other studies have shown higher prevalences. In Nepal intestinal parasites were isolated in 41% of malnourished children.[32] In Pakistan, the prevalence was found to be at 35% and in Basrah Maternity and Children's Hospital in Iraq (2001), the prevalence was much higher at 59%.[27] The stool analysis techniques used in these centers were however much more sensitive and expensive (involving staining of specimens by the modified Ziehl-Neelsen method for recovery of acid-fast oocysts of *cryptosporidia*) and could not be used in this study due to time and financial limitation.

Intestinal parasites were isolated more in subjects with diarrhea (34%) than those without (27%); a trend that was demonstrated in all the above studies concluding that malnourished children with diarrhea harbor parasites more frequently than those without diarrhea.[14,15,32,45] The commonly isolated parasites were protozoa, 15% while

9% were helminthes. The commonest protozoa isolated was *Giardia lamblia* while *Ascaris lumbricoides* was the commonest helminth, the two parasites commonly found in studies in malnourished children.[21,24,28,46,47] The protozoa were more prevalent in subjects with diarrhea than those without, while the helminthes were equally distributed in both groups.[33] A similar trend was demonstrated in a study in Lagos Nigeria where protozoa were isolated in 17% subjects while helminthes were found in 6%.[14,15] In the Nepal study, the helminthes were more commonly found (27% of subjects) than protozoans (15%)[32] probably due to environmental and climatic differences. This region being in the Highlands with no waterlogging and farming being the main occupation, the waterborne helminthes are less prevalent. Protozoa have been found to be more diarrhoeagenic in malnourished children and worsen the course of malnutrition.[6,20,31,33]

6.6 Water source and Sanitation Practices:

There was no significant difference between those with and without parasite infestation in terms of sanitation practices, reflecting similar environmental influence. Most subjects were from peri-urban areas of Eldoret town so had tap water as their main source of drinking water supplied by the Municipal Council.

In this study, sourcing water from rivers and was significantly associated with IPIs. This could be explained by the fact that most subjects were from crowded peri-urban slums where waste disposal areas and toilets drained to the rivers and boreholes and only a few families reported treating water before drinking, so the water is faecally contaminated with parasites. WASH studies have demonstrated a clear association between poor hygienic and sanitation practices and infestation with intestinal parasites.[42]

Improving water supply and sanitation has a very significant effect on the mortality and morbidity of different infections. For diarrhoea, the reduction in morbidity (incidence and prevalence) is of 25% when access to water is improved; 22% when disposal of human waste is improved; and 16% when water quality is improved. But these effects are not entirely cumulative. Among their conclusions, these WASH studies stressed the necessity of improving the quality (protection, treatment) and quantity of water available to homes in order to remove the need to store water in receptacles that are a source of contamination. These improvements reduce the incidence of parasitic diarrhoea and malnutrition in children.[40,42,48]

6.7 Limitations

Due to the nature of this study being cross-sectional and cost limitation, only one stool sample was taken and not the recommended three over several days. Formol-ether concentration method was used to identify parasites in this study and may not identify parasites that require special staining techniques; therefore giving a possible lower prevalence of parasite infestation.

CHAPTER SEVEN

CONCLUSIONS AND RECOMMANDATIONS

7.1 Conclusions:

1. The prevalence of IPIs in Severely Malnourished children admitted at MTRH children is 24%.
2. Diarrhoea is a common clinical presentation of intestinal parasite infestation among severely malnourished children

3. Intestinal parasite infestation is associated with severe, chronic and recurrent diarrhea in the severely malnourished
4. Protozoa, commonly *Giardia lamblia* are common parasites among severely malnourished children
5. Drinking untreated river water is a risk factor for intestinal parasite infestation

7.2 Recommendations:

1. Antiprotozoal agents should be administered to malnourished children who present with diarrhoea. This should be in addition to routine antihelmintics
2. River water should be treated before drinking to avoid infestation with intestinal parasites.

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APPENDIX 1: IREC APPROVAL LETTER

INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE (IREC)

MOI TEACHING AND REFERRAL HOSPITAL
P.O. BOX 3
ELDORET
Tel: 33471/2/3

MOI UNIVERSITY
SCHOOL OF MEDICINE
P.O. BOX 4606
ELDORET
Tel: 33471/2/3

Reference: IREC/2010/27
Approval Number: 000506

26th July, 2011

Dr. Koech Hellen,
Moi University,
School of Medicine,
P.O. Box 4606-30100,
ELDORET, KENYA.

Dear Dr. Koech,

RE: CONTINUING APPROVAL

The Institutional Research and Ethics Committee has reviewed your request for continuing approval for your study titled:-

"The Association of Intestinal Parasites with Diarrhoea among Severely Malnourished Children Admitted to the Children's Wards at Moi Teaching and Referral Hospital, Eldoret, Kenya."

Your request has been granted Approval with effect from 26th July, 2011. You are therefore permitted to continue with your study.

Note that this approval is for 1 year; it will thus expire on 25th July, 2012. If it is necessary to continue with this research beyond the expiry date, a request for continuation should be made in writing to IREC Secretariat two months prior to the expiry date.

You are required to submit progress report(s) regularly as dictated by your proposal. Furthermore, you must notify the Committee of any proposal change (s) or amendment (s), serious or unexpected outcomes related to the conduct of the study, or study termination for any reason. The Committee expects to receive a final report at the end of the study.

Yours Sincerely,

27/07/2011
DR. W. ARUASA
AG.CHAIRMAN
INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE

INSTITUTIONAL RESEARCH & ETHICS COMMITTEE
APPROVED
26 JUN 2011
P.O. Box 4606 ELDORET

cc: Director - MTRH
Dean - SOM
Dean - SPH
Dean - SOD

APPENDIX 2: The Wellcome Classification of Malnutrition

All the children who had Kwashiorkor, Marasmus and Marasmic-Kwashiorkor are severely malnourished and were included in the study.

%expected weight for age	Edema	
	Present	Absent
60-80%	Kwashiorkor	Underweight
<60%	Marasmic-kwashiorkor	Marasmus
80-120%	-	Normal
>120%	-	Obese

APPENDIX 3: Consent Form

Hello. My name is ……., a research assistant for Dr Hellen Koech, a postgraduate student at Moi University.

She is conducting a study on parasitic causes of diarrhea in children with malnutrition, and would appreciate your participation in this study. I will ask you questions about the child's illness, the child's feeding habits, where you live and the people you live with. If you agree to participate in these studies, we will take a small amount of the child's stool for tests to see if the child has any parasites in the intestines. Should any findings be made, the child's management will changed accordingly. The child will continue to be managed in the ward like the other patients. If you choose not to participate in this study, your child will still get the best possible management for her condition. The questions will take 10-15 minutes. Whatever information you provide will be strictly confidential and no names will be used on these forms.

This information will help her in her studies and may also help the hospital and other policy makers to plan on how to improve the care we give to our children with similar illness. May I begin now?

Consent by respondent:

The nature and procedures involved in this study have been explained to me and I do/ do not (circle the response chosen) agree for my child to participate in the study

Sign…… Date……

Signature of interviewer ……. Date ……

Respondent agrees to be interviewed…-1…….Sign…….

Respondent does not agree to be interviewed…. -2…… Sign……

APPENDIX 4: Questionnaire:

Child's Particulars:

Demographics:

Child: Serial Number…… **IP**……

Age	
Sex	
Residence	

Parent/ Guardian

Age		
Occupation	Farming	
	Business	
	Unemployed/ Casual day labour	
	Employed skilled labor	
	Employed unskilled labor	
Marital status	Single	
	Married	
	Separated	
	Divorced	
Education Level	Uneducated	
	Primary	
	Secondary	
	Tertiary	
Total Dependants		

Clinical Data:

Malnutrition Type based on Wellcome Classification of Malnutrition

Child's weight		
Expected Weight for age		
Percentage of expected weight		
Edema present	Yes	
	No	
Type of severe malnutrition	Kwashiorkor	
	Marasmus	
	Marasmic-Kwash	

History of Diarrhoea

Length of current episode	< 2 weeks

	>2 weeks
Nature of stools passed	Mucoid
	Bloody
	Watery; profuse or frothy
	Had worms
Previous episode in the past 3/12	Yes
	No
Dewormed in past 3 months	Yes
	No
First-Aid measure for diarrhea before admission	None
	Plain Water
	Over the counter medications
	Oral Rehydration Solution

Social conditions and Hygiene practices:

Source of water for daily use	Tap
	Borehole
	River
Treatment of drinking water	Yes
	No
Distance of toilet from water source	>50 metres
	< 50 metres
Wash raw foods before eating	Yes
	No

Nutritional History:

Length of exclusive breastfeeding	< 4mo	
	>or =4mo	
Ordinary day diet (24-hr Recall)	Food type	Carbohydrate
		Protein
		Fat
		vitamins
	Frequency	Once
		2-4 times
		>5 times
	Amount	Adequate
		Inadequate

Stool Microscopy Results: …….

APPENDIX 5: Laboratory request Form and Laboratory Procedure

Child's serial number……..

Age ……..

Sex……

Type of severe Malnutrition……..

Specimen : stool

Microscopy Results…….

……

……

Signed(techn.1): Name…… Techn 2: Name……

Signature…….

Signature……..

Laboratory Technique:

Concentration Technique (formal ether):

1. Add 7ml of 10% formal saline to approximately 1g of stool and stir using an applicator stick or grind till an homogenous suspension is obtained

2. Fill the funnel with a wet four layer gauze and sieve or pour to centrifuge tube
3. Pass the stool suspension through the filter into the centrifuge tube till the 7-mark is reached
4. Remove the filter and discard
5. Add 3ml of ether or Diethyl Ether and mix well for 1 minute.
6. Centrifuge at 2500RPM for 1 minute.
7. Loosen the fatty plug using an applicator stick and pour away the supernatant by quickly inverting the tube
8. Replace the tube in the rack and allow the fluids to drain down and sediment
9. Mix well and transfer a drop to a slide and cover with a cover slip.
10. Use a ×10 objective then ×40 objective to look for various characteristic ova and cysts of parasites.