Full Length Research Paper

Prevalence of *Helicobacter pylori* and other intestinal parasites amongst duodenal and gastric ulcer patients at Imo state University Teaching Hospital, Orlu, south eastern Nigeria

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Abstract

The prevalence of *Helicobacter pylori* and intestinal parasites amongst duodenal, gastric and peptic ulcer patients at Imo State University Teaching Hospital, Orlu was investigated between February and August, 2012. A total of 1,206 ulcer patients and 409 non ulcer patients were examined. Early morning stool samples and venous blood samples were collected from the patients from the general out - patient department (GOPD) and Community Medicine department (COMED) and sent to Microbiology and Parasitology Laboratory for analyses. The patients were examined for ulcer using faecal occult blood (FOB) test. Stool analysis for intestinal parasites was carried out using direct wet mount and stool concentration techniques (flotation and sedimentation methods). The blood samples were analysed serologically for *H. pylori* antibody using proprietary rapid test kits (Global test kits.) Out of 1,206 ulcer patients confirmed positive by faecal occult blood test, 703 (58%) were positive for *H. pylori* IgG antibody serological test, 407 (33.7%) were infected by *Entamoeba histolytica,* 113 (9.4%) were infected by *Cryptosporidium parvum*, 93 (7.7%) were infected by hookworm, 62 (5.1%) were infected by *Ascaris lumbricoides*, 31 (2.6%) were infected by *Strongyloides stercoralis*, 19 (1.6%) were infected by *Trichuris trichura* and 11 (0.9%) were infected by *Giardia intestinalis*. Out of 409 non ulcer patients who were negative for faecal occult blood test, 7 (1.7%) were positive for *H. pylori* antibody serological test, 187 (45.7%) were positive for *Entamoeba histolytica*, 23 (5.6%) had hookworm, 11 (2.7%) had *Ascaris lumbricoides*, 6 (1.5%) had *Trichuris trichiura*, 5 (1.2%) had *Cryptosporidium parvum* and 3 (0.7%) had *Strongyloides stercoralis*. Statistical analysis of the data using analysis of variance (ANOVA) showed positive correlation ($p < 0.0001$) between prevalence of intestinal parasites and ulcer disease. There was significant difference ($p < 0.05$) in the prevalence of *H. pylori* and intestinal parasites (*Cryptosporidium parvum,* hookworm and *Strogyloides stercoralis*) between ulcer and non - ulcer patients. This study has implicated *H. pylori* and other intestinal parasites as probable causes of ulcer amongst patients in Orlu and its environs in south eastern Nigeria.

Keywords: *Helicobacter pylori*, intestinal parasites, duodenal, gastric ulcer.

INTRODUCTION

Duodenal, gastric and peptic ulcers are amongst growing public health problems of developing countries and rural communities in Nigeria. An ulcer has been defined as a hole or sore in the lining of the stomach or other parts of the digestive system (Getchell et al., 1991). Ulcer may occur in different parts of the gastrointestinal tract, leading to different types of ulcers: duodenal, gastric and peptic ulcers. Duodenal ulcers are usually deep and

sharply demarcated. They tend to penetrate through the sub-mucosa, often into the muscularis propria (McGuigan, 1991). Peptic ulcers form in the lining of the duodenum (Forbes and Jackson, 1993). Gastric ulcers are deep, penetrating beyond the mucosa of the stomach and is similar histologically to duodenal ulcer but often with more extensive gastritis surrounding the ulcer. Almost all benign gastric ulcers are found immediately distal to the junction of the antral mucosa with the acid – secreting mucosa of the body of the stomach (McGuigan, 1991). Sometimes ulcers form in the lining of the stomach.

Different factors lead to development of ulcers in different people. Some of these are poor diet, stress, heredity and too much caffeine, alcohol, tobacco and aspirin. A peptic ulcer occurs when the stomach makes too much hydrochloric acid and there is a weakening in the protective lining of the stomach or duodenum (Getchell et al., 1991). Smokers have a 50% greater chance of developing peptic ulcers than non - smokers. Smoking makes ulcers difficult to treat, habits that often go with smoking such as drinking coffee, make ulcers worse and harder to heal (Getchell et al., 1991; McGuigan, 1991). Large amounts of alcohol causes the stomach to produce too much stomach acid which causes or worsens ulcer. Peptic ulcer develops when there is an unfavourable balance between acid – pepsin secretion and mucosal resistance: in the pathogenesis of duodenal ulcer, evidence favours the importance of absolute or in most instances, relative gastric hyper-secretion. In contrast, for gastric ulcer, defective mucosal resistance appears to be the major contributing factor (McGuigan, 1991). Duodenal ulceration is very common in the developed world, but the cause of duodenal ulcer diathesis remains unknown. Recent attention has focused on the potential role of *Helicobacter pylori*, which is often found in the mucus and on the surface of cells in gastric biopsies from patients with duodenal ulcers, many of whom have an associated antral gastritis (Forbes and Jackson, 1993) The acid in the digestive juices causes ulcers and stress may worsen this condition (Getchell et al., 1991). Gastric colonization with *Helicobacter pylori* has been reported in 80 to 100 percent of patients with duodenal ulcer (McGuigan, 1991). This has been proposed as a potential contributing factor in the pathogenesis of duodenal ulcer. At present, it is uncertain whether *H. pylori* play a role in producing duodenal ulcer or if its presence reflects a commensal association (McGuigan, 1991). Acid – pepsin appears to be important in the pathogenesis of gastric ulcer, however, in contrast to duodenal ulcer, gastric ulcer patients generally have acid secretory rates that are normal or reduced compared with non – ulcer subjects. Although many patients with gastric ulcer have reduced rates of acid secretion, true achlorhydria (in response to pentagastrin stimulation) almost never occurs in patients with benign gastric ulcer. Ten to twenty percent of patients with gastric ulcers also

have duodenal ulcers. Patients with both duodenal and gastric ulcers tend to have acid secretory patterns that parallel those of duodenal ulcers. Patients with pyloric channel ulcer have acid secretory rates and clinical patterns similar to those found with common duodenal ulcer (McGuigan, 1991). It is clear that acid secretion by the stomach is required for production of a duodenal ulcer, but the factors which render the acid – secreting subject susceptible to duodenal ulceration have not been defined completely.

Helicobacter pylori (formerly known as *Campylobacter pylori*) is a short (0.2 to 0.5μm in length), spiral shaped micro-aerophilic gram – negative bacillus which has been suggested as a potential cause of certain forms of acute and chronic gastritis. Gastric colonization has also been associated with duodenal and gastric ulcer. With gastric colonization, *H. pylori* are found in the deep portions of the mucus gel layer that coats the gastric mucosa and between the mucus gel layer and the apical surfaces of the gastric mucosal epithelial cells. They also may be located in the regions of the tight junctions between adjacent mucosal epithelial cells. They do not invade the gastric mucosa. *H. pylori* is associated with inflamed gastric mucosal epithelium in the stomach as well as with metaplastic gastric epithelium found elsewhere e.g. in the duodenal bulb of most patients with duodenal ulcer. This is evidence that *H. pylori* is the cause or at least a principal cause of a form of gastritis that has been designated active chronic gastritis. *H. pylori* has been identified in gastric samples by histologic examination, culture, urease activity and by endonuclease analysis. On stained tissue sections, *H. pylori* is Giemsa – positive and faintly haematoxylin – positive. It can be cultured successfully from biopsy material, but usually not from gastric secretions. *H. pylori* produce large amounts of urease. Antibodies (IgG and IgA) to *H. pylori* have been identified in sera of individuals with *H. pylori* colonization. Previous workers, (McGuigan, 1991) reported high degree of correlation between these serum antibodies and histologic gastritis.

More often, intestinal amoeba make their way to the sub mucosal layer where they multiply rapidly and form colonies, destroying the tissues around by lytic necrosis and forming an abscess which breaks down to form an ulcer (Paniker, 2007). Amoebic ulcer is the typical lesion seen in intestinal amoebiasis. The ulcers are multiple and confined to the colon, being most numerous in the caecum and next in the sigmoido – rectal region. Also the intestinal coccidian protozoan parasites (*Cryptosporidium parvum*), which causes gastrointestinal tract infection (cryptosporidiosis) was brought to the forefront as a clinically important human parasite because of its strong association with gastroenteritis amongst immune-deficient individuals (Current, 1983; Oyerinde, 1999; Obiajuru et al., 2008).

Cryptosporidium infections has been associated with short term diarrhoeal illness in immune – competent

individuals, chronic life threatening cholera – like illness in immune-compromised individuals and duodenal ulcer in some other individuals (Fayer and Ungar, 1986; Mbanugo and Agu, 2005; Obiajuru et al., 2008). Antibodies to herpes simplex have been reported to be higher in titre and more frequent in sera of patients with duodenal ulcer than in normal (McGuigan, 1991). The main sign of an ulcer is pain caused by digestive juice coming in contact with the ulcer. Peptic ulcer is characterized by intermittent epigastric pain that is relieved by food, with night waking and heartburn. However, not all ulcers are painful. Many patients do not have symptoms and become aware of their ulcers only when complications occur (Getchell et al., 1991; Forbes and Jackson, 1993). Some workers, (McGuigan, 1991) emphasized that many patients with active duodenal ulcer have no ulcer symptoms and this leads to a significant, although not precisely quantifiable, underestimate of duodenal ulcer frequency in the population. Ulcers cause severe bleeding or blockage of the alimentary canal. They eat through the wall of the stomach or duodenum. Complications of duodenal ulceration include haemorrhage, pyloric stenosis caused by scarring, and perforation (Forbes and Jackson, 1993). The absolute prevalence of duodenal ulcer in the population is not known, however, estimates have ranged from 6 to 15 percent (McGuigan, 1991). Variations in estimates may be explained by differences in the populations examined, study designs, diagnostic methods e.g. Endoscopy vs radiologic examination and by actual changes occurring in the frequency of duodenal ulcer. In the past 40 years, the frequency of duodenal ulcer and its complications has been decreasing in the United States and England especially in males. The reasons for this reduction are not known (McGuigan, 1991).

MATERIALS AND METHODS

Selection of respondents

Male and female duodenal, gastric and peptic ulcer patients aged 25 to 60 years were selected from the general out - patient and Community medicine departments at Imo State University Teaching Hospital, Orlu between February and August, 2012 for the study. Ethical permit was obtained from the Teaching hospital. Patients consent to participate in the study was obtained and study questionnaires were given to them to complete and return on the spot.

Collection of samples

Patients who indicated willingness to participate in the study were referred to the Microbiology and Parasitology

Laboratory of the hospital for sample collection.

The Laboratory scientist in charge of serology tests was instructed to collect 1ml of venous blood from each patient and centrifuge it at 3500rpm for 5minutes to separate the serum for serological test. Thereafter, each patient was given a sterile disposable bottle and swab stick to produce stool sample for faecal occult blood test and parasitological examinations. A total of 1,206 blood and stool samples were collected from ulcer patients and 409 blood and stool samples were collected from non - ulcer patients.

Laboratory analyses of samples

Screening for peptic, gastric and duodenal ulcer

The patients were examined for duodenal, gastric and peptic ulcer using faecal occult blood (FOB) test. Each Stool sample was examined for occult blood using haemascreen rapid occult blood test kit. According to the manufacturers' instructions, the applicator sticks provided by the manufacturers was used to apply a very thin smear of each stool sample on the indicated area of the test paper marked with Roman numeral I. Using the same applicator stick, sample was taken from a different portion of the same stool and smeared on the area of the same test paper marked with Roman numeral II. The stick was discarded and the test slide was sealed to enclose the smeared samples. The perforated section on the back of the test slide marked 1 and 2 was opened. Two drops of hema – screen[TM] developing solution was applied to exposed test paper. The results were read within 30 – 60 seconds using the embedded auto control as a standard. Any trace of blue colour on the test areas was interpreted as positive while no indication of blue colour was interpreted as negative result.

Stool analysis for intestinal parasites

Each stool sample was examined parasitologically using direct wet mount and stool concentration techniques (flotation and sedimentation methods) for intestinal parasites as described by previous workers (Chesbrough, 2002; Obiajuru and Ozumba, 2009). A drop of physiological saline was placed on one end of a grease – free slide and a drop of Lugol's iodine was placed on the other end of the slide. A small portion of each stool sample was emulsified in the saline and iodine respectively, using sterile swab stick. They were covered with coverslips and examined microscopically using x10 and x45 objectives. Each stool sample was further processed for concentration technique using flotation and sedimentation methods respectively as described in (Obiajuru and Ozumba, 2009). Both were examined microscopically using x10 and x45 objectives.

Table 1. Prevalence of H. pylori and intestinal parasites amongst ulcer and non - ulcer patients

Patients	Number Exam	Number Infected (%)						
		H. pylori	Hookworm	*Ascaris lumbricoides*	*Strongyloides steroralis*	*Trichuris trichura*	*Cryptosp*	*Giardia intestinalis*
Ulcer patients	1,206	703	93	62	31	19	113	11
Non-ulcer patients	407	7	23	11	3	6	5	0
Total	1,613	710	116	73	34	25	118	11

Helicobacter pylori IgG antibody test

The blood samples were examined serologically for *H. pylori* IgG antibodies using immune-chromatographic rapid test kits (Global). The test device and patients' serum were allowed to attain room temperature before commencement of test. According to the manufacturers' instructions, each blood sample was centrifuged at 3500rpm for 5 minutes and a sterile Pasteur pipette was used to separate the clear serum into a clean test tube. The test device was removed from the foil pouch and placed on a clean level surface. A sterile Pasture pipette was used to transfer 100ul of patient's serum to the well of the test device marked "S", avoiding air bubbles. A timer was set for 10minutes within which the red line(s) appear. Two distinct red lines (one on the control C and the other on the test T) indicate positive result. One red line only on the control C indicates negative result. One red line only on the test T or no line at all on both control C and test T indicate invalid result and the test repeated with new test kit / device.

RESULTS

Out of 1,206 ulcer patients confirmed positive by faecal occult blood test, 703 (58.3%) were positive for *H. pylori* IgG antibody serological test, 407 (33.7%) were infected by *Entamoeba histolytica*, 113 (9.4%) were infected by *Cryptosporidium* species, 93 (7.7%) were infected by hookworm, 62 (5.1%) were infected by *Ascaris lumbricoides*, 31 (2.6%) were infected by *Strongyloides stercoralis*, 19 (1.6%) were infected by *Trichuris trichura* and 11 (0.9%) were infected by *Giardia intestinalis*. Out of 409 non ulcer patients who were negative for faecal occult blood test, 7 (1.7%) were positive for *H. pylori* IgG antibody serological test, 187 (45.7%) were positive for *Entamoeba histolytica*, 23 (5.6%) had hookworm, 11 (2.7%) had *Ascaris lumricoides*, 6 (1.5%) had *Trichuris trichura,* 5 (1.2%) had *Cryptosporidium parvum* and 3 (0.7%) had *Strongyloides stercoralis*. Table 1 summarizes the prevalence of *Helicobacter pylori* and other intestinal parasites amongst ulcer and non - ulcer

patients at Orlu. There was significant difference (p < 0.05) in the prevalence of intestinal parasites (*H. pylori* and *Cryptosporidium* sp.) between duodenal, gastric and peptic ulcer patients and non - ulcer patients.

Table 2 summarizes the age related prevalence of *Helicobacter pylori* and intestinal parasites amongst ulcer and non - ulcer patients in Orlu. As shown, the most prevalent isolates amongst ulcer patients were *H. pylori* (58.3%)*, Cryptosporidum parvum* (9.4%) and hookworms (7.7%).The prevalence of intestinal parasites were significantly lower amongst non – ulcer than ulcer patients. Statistical analysis of the data on age related prevalence of intestinal parasites amongst ulcer and non - ulcer patients showed no significant difference (p > 0.05) between the different age groups.

Figure 1 shows the comparative analysis of the prevalence of *Helicobacter pylori* and intestinal parasites amongst ulcer and non - ulcer patients in Orlu. Statistical analysis of the data using analysis of variance (ANOVA) showed positive correlation (p < 0.0001) between prevalence of intestinal parasites (*H. pylori, Cryptosporidium,* Hookworms, *Strongyloides stercoralis* and *Ascaris lumbricoides*) and duodenal, gastric and peptic ulcer disease.   Figure 2 shows comparative analysis of the prevalence of *H. pylori* and intestinal parasites amongst different age groups of ulcer patients. As shown, the highest prevalence of *H. pylori* (63.4%) was observed amongst ulcer patients aged 41 – 50 years followed by 61.1% observed amongst ulcer patients aged 51 – 60 years. Amongst ulcer patients aged 31 – 40 years, prevalence of *H. pylori* was 53.7% and 50.3% amongst those aged 25 – 30 years. Similarly, the prevalence of *Cryptosporidum parvum* amongst ulcer patients was 14.3% amongst those aged 25 – 30 years, 11.2% amongst those aged 31 – 40 years, 9.3% amongst those aged 41 – 50 years and 4.2% amongst those aged 51 – 60 years. Comparative analysis of hookworm infections amongst ulcer and non – ulcer patients revealed 6.3% and 4.9% prevalence respectively amongst those aged 25 – 30 years. Amongst those aged 31 – 40 years, the prevalence of hookworm infection was 8.6% for ulcer patients and 6.9% for non – ulcer patients, amongst those aged 41 – 50 years, prevalence of

Table 2. Age – related - Prevalence of *H. pylori* and other intestinal parasites amongst ulcer and non ulcer patients

Patients	Age (years)	Number Exam	H. pylori	Hookworm	Ascaris lumbricoides	Strongyloides stercoralis	Trichuris trichura	Cryptosp parvum	Giardia intestinalis
					Number Infected		**(%)**		
Ulcer patients	25– 30	189	95	12	7	8	6	27	2
	31-40	313	168	27	16	11	3	35	4
	41-50	421	267	33	19	5	7	39	3
	51-60	283	173	21	20	7	3	12	2
Total	1206		703 (58.3)	93 (7.7)	62 (5.1)	31 (2.6)	19 (1.6)	113 (9.4)	11 (0.9)
Non-ulcer patients	25– 30	81	3	4	4	1	3	3	0
	31-40	102	3	7	2	0	2	1	0
	41-50	118	0	6	3	0	1	1	0
	51-60	106	1	6	2	2	0	0	0
Total		407	7 (1.7)	23 (5.6)	11 (2.7)	3 (0.7)	6 (1.5)	5 (1.2)	0
Total (Both)		1,613	710 (44.0)	116 (7.2)	73 (4.5)	34 (2.1)	25 (1.5)	118 (7.3)	11 (0.7)
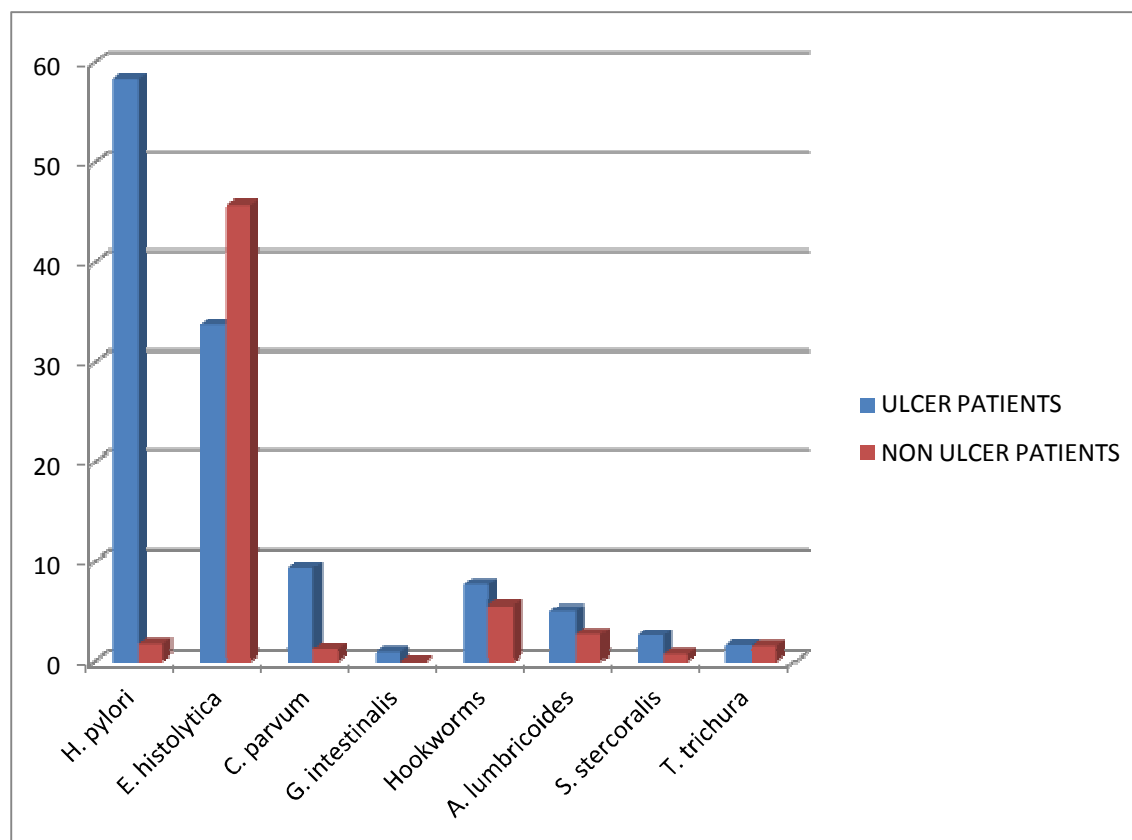
Figure 1. Comparative analysis of the prevalence of intestinal parasites amongst Ulcer and Non Ulcer patients in Orlu, South Eastern Nigeria
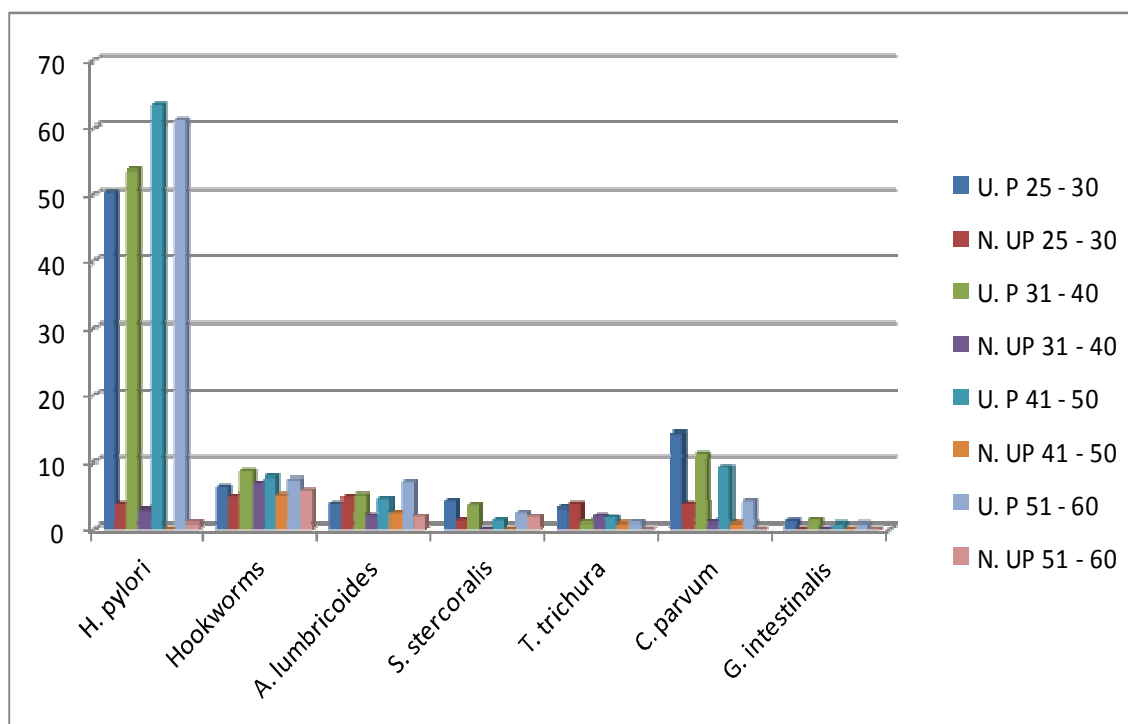
NOTE: U. P = Ulcer patients N. UP = Non ulcer patients

Figure 2. Comparative Analysis of *H. pylori* & intestinal parasites infections amongst different   age groups of ulcer and non – ulcer patients

hookworm infection was 7.8% for ulcer patients and 5.1% for non – ulcer patients. Amongst those aged 51 – 60 years prevalence of hookworm infection was 7.4% for ulcer patients and 5.7% for non – ulcer patients.

DISCUSSION

Our findings showed that *Helicobacter pylori* a Gram – negative micro – aerophilic bacterium, *Cryptosporidium parvum* a coccidian parasite and hookworm are implicated with higher prevalence amongst ulcer patients than non – ulcer patients in Orlu, South Eastern Nigeria. This finding corroborates previous reports (McGuigan, 1991; Forbes and Jackson, 1993) which associated cryptosporidiosis and *Helicobacter pylori* infections with gastrointestinal tract ulcer disease in different parts of the world. Doudenal, gastric and peptic ulcer ranks amongst top ten common diseases in public and private hospitals in Imo State. Although much is now known concerning factors that contribute to the development of duodenal gastric and peptic ulcer, we do not completely understand its pathogenesis. On the other hand, there is paucity of information on the role of intestinal parasites in the development and spread of ulcer. Notwithstanding, it is known that a number of parasites and bacteria including *H. Pylori, Entamoeba* species, *Giardia intestinalis*,

hookworms, *Ascaris lumbricoides*, etc. cause injury on the gastrointestinal tract and these injuries are capable of causing ulcer.

The present study has observed higher prevalence of *Helicobacter pylori* and other intestinal parasites (*Cryptosporidum parvum, Ascaris lumbricoides* and hookworms) amongst ulcer patients than non – ulcer patients. This suggests that they play significant role in development of ulcer and/or contribute immensely to the worsening effects of duodenal, gastric and peptic ulcer amongst patients in Orlu South eastern Nigeria. Our investigations in some hospitals in this area showed that most clinicians are merely interested to establish that their patients have gastrointestinal tract ulcer and they proceed with treatment with little or no attention to parasites and micro-organisms as possible causes or secondary complications associated with such ulcers. The findings of the present study thus underscores the need for clinicians and healthcare providers in Orlu and its environs to pay adequate attention to n *Helicobacter pylori* and other intestinal parasites (*Cryptosporidum parvum, Ascaris lumbricoides* and hookworms) as emerging infectious diseases amongst ulcer patients in the area. The human gastrointestinal tract is a habitat for many organisms regarded as commensals under normal conditions. Under abnormal conditions these organisms become parasitic.

Our findings on age – related prevalence of *Helicobacter pylori* and other intestinal parasites amongst ulcer and non – ulcer patients showed that there is no correlation between age and the spread of intestinal parasites amongst ulcer patients. Similarly there is no correlation between ulcer and age. Both ulcer disease and gastrointestinal tract parasites affect all age groups randomly. We therefore suggests that clinicians and health care providers in this area should include diagnosis of gastrointestinal tract parasites and bacteria in management and treatment of ulcer patients irrespective of their ages or gender. This will help achieve better management as well as reduce waste of drugs and/or resources which is usually the case when aetiological agents of infectious diseases are unknown.

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Legend to Figure 1

PARASITE	ULCER PATIENTS = 1206		NON – ULCER PATIENTS = 409		
H. pylori IgG antibody	703	(58.3)	7	(1.7)	
H. pylori Culture	698	(57.9)	2	(0.5)	
Entamoeba histolytica	407	(33.7)	187	(45.7)	
Cryptosporidum parvum	113	(9.4)	5	(1.2)	
Giardia intestinalis	11	(0.9)	0	(0.0)	
Hookworms	93	(7,7)	23	(5.6)	
Ascaris lumbricoides	62	(5.1)	11	(2.7)	
Strongyloides stercoralis	31	(2.6)	3	(0.7)	
Trichuris trichura	19	(1.6)	6	(1.5)	

Legend to Figure 2

Age (yrs)	*Helicobac pylori*		*Hookworm*		*Ascaris lumbricoi*		*Strongyloi stercoralis*		*Trichuris trichura*		*Cryptosp. parvum*		*Giardia intestinali*	
	U. P	N.UP	U. P	N.UP	U. P	N.UP	U. P	N.UP	U. P	N.UP	U. P	N.UP	U. P	N.UP
25 - 30	50.3	3.7	6.3	4.9	3.7	4.9	4.2	1.2	3.2	3.7	14.3	3.7	1.1	0
31 – 40	53.7	2.9	8.6	6.9	5.1	2.0	3.5	0	1.0	2.0	11.2	1.0	1.3	0
41 – 50	63.4	0	7.8	5.1	4.5	2.5	1.2	0	1.7	0.8	9.3	0.8	.7	0
51 - 60	61.1	0.9	7.4	5.7	7.1	1.9	2.5	1.9	1.0	0	4.2	0	0.7	0

NOTE: U. P. = ULCER PATIENTS   N. UP = Non – ULCER PATIENTS
Figures = %.