CHAPTER ONE

INTRODUCTION

INTRODUCTION

1.1 Introduction

Helicobacter pylori (*H. pylori*) infection is a major cause of peptic ulcer and other upper gastrointestinal tract diseases. *H. pylori* infection occurs in childhood and persists life-long if not treated. Many of the infected patients develop chronic gastritis, and considerable percentage of them develops ulcer disease or gastric cancer (Chey *et al*., 2013). Almost 50% of world population is infected with *H. pylori*. Its prevalence is highly variable in relation to geography, ethnicity, age, and socioeconomic-factors, with 80% in developing countries and 20-50% in developed countries. Transmission of *H. pylori* is largely by the oral-oral or fecal-oral routes. There is a relation between *H. pylori* infection and peptic ulcer disease. *H. pylori* is one of the most important factors causing peptic ulcer development and ulcer complications such as upper gastrointestinal hemorrhage and stomach cancer (Ting-Chun & Chai-long , 2014). Thus, diagnostics tests are very important for diagnosis, treatment and eradication confirmation to prevent infection complications. The diagnosis of *H. pylori* infection can be made through many laboratory tests. The techniques are divided into two groups, invasive and non-invasive tests. All invasive tests are based on endoscopic examination during which biopsy specimens are obtained for direct (histological analysis, isolation) or indirect (urease test) diagnosis of *H. pylori* infection. The difficulties associated with this approach are risk and discomfort to the patient. Moreover, *H. pylori* tend to colonize in patches and may be missed by biopsy. Culturing of *H. pylori* from biopsy is difficult and time consuming. These difficulties may lead to false negative results. Non-invasive methods reveal the presence of *H. pylori* by measuring the activity of urease (urea breath test), then by confirming

the presence of antibodies in the serum. The urea breath test is highly sensitive and specific; however, it requires ingestion of isotopically labeled urea as well as specialized instrumentation for detection of C14 or C13. Other non-invasive tests were also evaluated in several studies including, detection of *H. pylori* antigens in stool and presence of *H. pylori* in saliva. Stool antigen tests have recently been welcomed with great expectations as they are convenient to the patients and can be easily performed even in small laboratories. The antigen test is a rapid, non-invasive method which provides direct detection of an active infection. However, serological tests are reported to be unreliable for the diagnosis of *H. pylori* since they may return false negative results for up to 60 days after infection and remain positive for considerable time after eradication (Abdulrahman *et al*., 2014). The aim of this study was therefore planned for comparative evaluation of stool antigen tests and blood antibody tests detection methods using commercially available kits for the diagnosis of *H. pylori* infection in symptomatic patients.

1.2 Rationale

Sudan is one of the developing countries with high rate of *H. pylori* infection and the serological diagnostic tests and stool antigen tests are widely used. Several studies reported that the percentage of positive *H. pylori* antibody (serum) in infected patients was higher than *H. pylori* antigen (fecal antigen) (Abdulrahman *et al.*, 2014). Recently in Sudan majority of the investigations for *H. pylori* using commercial kits for the detection of *H. pylori* antibody in the serum of infected patients yielded positive results. While, *H. pylori* stool antigens yielded less positive results. Many patients continue to complain from the same symptoms even after treatment. This has led researchers in the medical field to question the reliability of serum antibody tests knowing the chances of

false positive results. Serum diagnosis is favored only for the price of the kits compared to the stool antigens which are much sensitive and expensive than the serum kits. Hence, it is essential to evaluate the sensitivity and specifity of both stool (antigen) and serum (antibody) for the detection of *H. pylori* from symptomatic Sudanese patients. Urea breath test and culture obtained by invasive method (biopsy) could be useful tools in the diagnosis of *H. pylori* infection but they might not be applicable to all cases since they are expensive and time consuming. Despite the disadvantages of immunochromatography tests (ICT) they remain as important methods for the detection of *H. pylori* in Sudanese healthcare settings and it is necessary to confirm the results obtained by antigen detection tests as well as antibody detection tests. The detection, treatment and eradication of *H. pylori* are essential measure in minimizing the risks of complications such as peptic ulcer, duodenal ulcer and stomach cancer.

1.3 Objectives

1.3.1 General objectives

To compare between antigen antibody detection tests used for diagnosing *H. pylori* infection among symptomatic patients in Aboguta city.

1.2 Specific objectives

To detect *H. pylori* antibodies in serum of symptomatic patients by immunochromatography test cards.

To detect *H. pylori* antigens in stool of symptomatic patient by immunochromatography test cards.

CHAPTER TWO

LITERATURE REVIEW

LITERATURE REVIEW

2.1 History and taxonomy of Helicobacter

Helicobacter pylori, previously *Campylobacter pylori* are a Gram-negative, microaerophilic bacteria found usually in the stomach. It was identified in 1982 by Australian scientists Barry Marshall and Robin Warren, who found that it was present in a person with chronic gastritis and gastric ulcers, conditions not previously believed to have a microbial cause. It is also linked to the development of duodenal ulcers stomach cancer. Important Human pathogens include; *H. pylori* (human, no animal reservoir), *H. cinaedi* (male homosexuals, rodents), *H. fenneliae* (male .homosexual, rodent) (David *et al*., 2012)

General Characteristics of Helicobacter pylori 2.2

Helicobacter pylori are a major human pathogen. It is a Gram-negative, spirally shaped bacterium. It is strictly micro-aerophilic, requires carbon dioxide and a rich growth media. It has a tuft of sheathed unipolar flagella (lophotrichous), unlike the unsheathed flagella of Campylobacters. It produces an exceptionally powerful urease, almost 100 times more active than *Proteus vulgaris,* which is vital to its survival in the stomach. It is more fragile (David *et al.,* 2012).

2.3 Epidemiology of *Helicobacter pylori*

The prevalence of *H. pylori* infection varies widely by geographic area, age, race, and socioeconomic status. Infection is common within family cluster and orally transmitted from person-to-person and more common within elders (uncommon within young children). In developed countries approximately 30% of the total population is infected. Of which 1% per year develop duodenal ulcer, one third eventually have peptic ulcer

disease (PUD). In developing countries the infection is hyper endemic (70-90%), and majority of the adults are infected but not diseased, due to protective immunity from multiple childhood infections (Hunt *et al*., 2010).

2.4 Pathogenesis and virulence factors

H. pylori are highly adapted to live only on gastric mucosa. The bacteria are non-invasive, being present in the mucosa. Although gastric acid is destructive to *H. pylori*, protection is provided by it is powerful urease, which acts on the urea passing through the gastric mucosa to generate ammonia which neutralizes acid around the bacteria. *H. pylori* virulence factors includes, multiple sheathed flagella, Adhesins, Mucinace (causes localized tissue damage), Urease (neutralizes the local acid environment) and Acid-inhibiting proteins (David *et al*., 2012).

Mechanism of Immune escape and Tolerance 2.5

Helicobacter pylori colonize the gastric mucosa of at least half of human population, causing worldwide infection that appears in early childhood and if not treated, it can persist for life. The presence of symptoms and their severity depend on bacterial components, host susceptibility, and environmental factors, which makes *H. pylori* to switch between commensalism and pathogenicity. *H. pylori* driven interaction with the host immune system underlie the persistence of the infection in humans, since the bacterium is able to interfere with the activity of innate and adaptive immune cells, reducing the inflammatory response in its favour. The immunomodulator VacA promotes immune tolerance and persistent *H.pylori* infection through its activities on T-Cells and Antigen-presenting cells. Once you are infected, unless treated, the infection usually stays for the rest of your life (Tiziana, *et al*., 2015).

2.6 Transmission of *Helicobacter pylori*

The exact mode of transmission of *H. pylori* is not known. It has the potential to spread by fecal contamination, and from person to person by saliva. Majority of the people who are infected were infected as children and this may explain why the rate of infection is so high in poorer countries and in socio-economic groups characterized by crowded living conditions, poor sanity condition and lack of clean water. Personal hygiene is also very important since food handlers and cookers could be a source of infection (Hunt *et al.*, 2010).

2.7 Symptoms & complications of *H. pylori* infection

The majority of people who are infected with *H. pylori* are disease free. The symptoms of infection are; upper abdominal pain, loss of appetite, nausea, vomiting, and abdominal discomfort. Most people develop stomach inflammation (gastritis) from the body response to the bacterium itself and to the bacterium products (Vac-A, cytotoxin) (Levine *et al.*, 2004).

Complication associated with *H. pylori* infection includes inflammation of stomach lining, ulcer and stomach cancers. *H. pylori* can damage the protective lining of your stomach and small intestine. This can allow stomach acid to create an open sore (ulcer). About 10% of people with *H. pylori* will develop an ulcer. *H. pylori* infection is strong risk for certain types of stomach cancers. The risk of developing stomach cancer is thought to be increased with long-term infection with *H. pylori.* However majority of people with *H. pylori* do not get stomach cancer. The increased risk is smaller. Your risk may be greater if you have *H. pylori* in addition to having a first-degree relative who has been diagnosed with stomach cancer (Chey et *al*., 2013).

2.8 Treatment & prevention of *H. pylori* infection

The therapy for *H. pylori* infection involves multiple steps. In addition to antibiotics to eradicate the bacteria, another goal is to decrease the amount of acid secretion in the stomach and remove risk factors from further stomach irritation.

Antibiotic therapy: two week course of combination antibiotic therapy is recommended, Clarithromycin, Metronidazole, Amoxicillin, and Tetracycline. It is important to confirm eradication of *H. pylori* (Alesandro *et al.*, 2014).

Since 50% of world population has detectable *H. pylori* in their stomachs, it is very difficult to prevent infection with this bacterium; however, suggestions have been made to prevent ulcers. Certain measures such as non-smoking, no drinking for alcohol, substituting acetaminophen (Tylenol and others) for aspirin for pain control, avoidance of caffeine, check for GI symptoms and immediate treatment, identify and reduce or avoid stress, wash hands with clean water to avoid contracting the bacterium, and if infected with *H. pylori*, antimicrobial treatment may avoid ulcer formation and extension of disease (Charles, 2015).

2.9 Diagnosis of *H. pylori* infection

Diagnosis *H. pylori* infection divided into two types;

2.9.1 Endoscopic testing (**invasive testing**): the invasive tests include biopsy urease testing; rapid urease testing, histology, brush cytology, and bacterial culture and sensitivity testing.

2.9.2 Noninvasive testing: the non-invasive tests include urea breath test (UBT)**,** serology**,** stool antigen assay; rapid stool antigen tests**,** salivary assays**,** urinary assays, and polymerase chain reaction (PCR) (Malfertheiner *et al.*, 2015).

The choice of test depends upon issues such as cost, availability, clinical situation, population prevalence of infection, pretest probability of infection and factors such as the use of proton pump inhibitors and antibiotics that may influence certain test result (Greenberge and Anderson, 2015).

2.9.2.1 Urea Breath test (UBT)

Urea breath can detect current *H. pylori* infection. This test is based on the knowledge that *H. pylori* produce abundant active urease, an enzyme that converts urea to ammonium and carbon dioxide. When infected with *H. pylori* high urease activity is present in the patient's stomach. A dose of urea labeled with either C-13 or C14 is ingested by the subject. The urease-catalyzed reaction then takes place in the mucus layer where *H. pylori* is present; the labeled carbon dioxide diffuses to the epithelial cells, and then is carried in the bloodstream. Ultimately it is released in the exhaled air. The labeled carbon dioxide in the subject's breath can be measured and it is amount relates the urease activity, which indicates the presence or absence of *H. pylori* infection. The sensitivity and specificity

of breaths range from 95-97%. So it is highly sensitive and specific: however, it requires ingestion of isotopically-labeled urea as well as specialized instrumentation for the detection of C-14 or C13, which is not available in developing countries hospitals (Sihe *et al*., 2013).

2.9.2.2 *H. pylori* stool antigen test

This is a rapid test based on monoclonal antibody immuno-chromatography of stool samples. This test has been reported to be very specific (98%) and sensitive (94%).The results are positive in the initial stages of infection and can be used to detect eradication after treatment (follow up). Stool antigen tests are appropriate where prevalence of *H. pylori* infection is less than 30%. Other test methods are based on a sandwich enzyme immunoassay with antigen detection. *H.pylori* stool antigen test is accurate (as urea breath test), simple, fast and relatively inexpensive. Therefore, physicians can trust this test and start patient's treatment (Ali, Muhammad. 2015).

2.9.2.3 *H. pylori* serological tests

The serological tests has a high sensitivity and specificity (>90%). It is currently based on the detection of IgG antibodies against *H. pylori*. The antibody titer may remain elevated for a long time after *H. pylori* eradication and the number of false-positive results in age related increase with age. Serological tests are appropriate where the prevalence of *H. pylori* infection is greater than 30%. Serological tests are easy and cheap to perform and the potential for developing a rapid diagnostic test makes serology an interesting option for testing population in areas with little or no access to medical facilities (Ali, Mohammad. 2013).

CHAPTER THREE

MATERIALS &METHOD

MATERIALS & METHOD

3.1 Study design

This was a cross sectional study.

3.2 Study area

The study was carried out at Aboguta city in Aljazeera State Central Sudan between May and June 2015.

3.3 Study population

Randomly 100 symptomatic patients from both genders were enrolled for this study.

3.4 Data collection

A trained physician interviewed each patient and completed a detailed questionnaire & obtained data of age, sex, drug history and symptoms.

3.5 Samples collection

From each patient, 5ml of blood sample was taken into sterile plain tube to use for antibody detection and stool sample into sterile spoon-cover and outer labeled stool container for antigen detection. All of blood serum samples were examined for *H. pylori* antibodies and all of stool samples were examined for *H. pylori* antigens.

3.6 Sample processing

All samples were tested by a commercial kits made by Xiamen Boson Biotech and Healgen England, UK, 2015 used for the detection of *H. pylori* antigens and antibodies. Immunochromatography test cards (ICTs).

3.7 Data analysis: Data was analyzed by Microsoft Excel.

CHAPTER FOUR

RESULTS

RESULTS

Hundred patients participated in this study, 52% were female with mean age of 37 years and 48% were male with mean age of 39 years. *H. pylori* antibody was positive in 72 patients and negative in 28 patients, while *H. pylori* fecal antigen was positive in 36(36%) patients and negative in 64 patients. The total number of infected patients was 82(82%), while 18(18%) patients were non-infected. The antibody was positive in 72(88%), antigen positive in 36(44%), antigen positive and antibody positive in 23(28%), and 10(12%) patients with antigen positive and antibody negative. According to the gender 43 of the infected patients were male with serum antibody positive in 38(88%) and fecal antigen positive in 20(53%), while 39 of the infected patients were females with serum antibody positive in 34(87%) and fecal antigen positive in 16(47%). Moreover, the results revealed that the percentage of infected males was higher (90%) than infected females (75%) and fecal antigen was positive in (53%) of the infected males and (47%) of the infected females. The percentage of positive results in the symptomatic patients detected with antibody was higher (72%) compared to fecal antigen (36%).

Table 3.1 Frequency of antibody detected in patients blood

	Positive	**Negative**	**Total**
Male	38(79%)	10(21%)	48
Female	34(65%)	18(35%)	52
Total	72	28	100

Table 3.2 Frequency of antigen detected in patients stool

Sex	**Positive Ag**	**Negative Ag**	**Total**

Male	20(42%)	28(58%)	48
Female	16(31%)	36(69%)	52
Total	36	64	100

Table 3.3 Comparison between serum and stool results

	Positive N (%)	**Negative** N (%)	**Total** N (%)
Serum antibody	72 (72%)	28(28%)	100(%)
Stool antigen	36(36%)	64(64%)	100(%)

CHAPTER FIVE

DISCUSSION, CONCLUSIONS & RECOMMENDATIONS

DISCUSSION, CONCLUSION& RECOMMENDATIONS

5.1 DISCUSION

H. pylori infection is an important public health issue in our country as it is in all developing and developed countries. Many studies performed at

home and abroad indicate that *H. pylori* is the cause of several diseases that can progress to stomach cancer, especially chronic gastritis and peptic ulcer disease (gastric and duodenal ulcer).In *H. pylori* infected patients, first IgM class antibodies develop. Then both systematically and locally in gastric mucosa, IgG and IgA antibodies developed and the level of these two antibodies can be maintained for months-years. IgG positive antibody may continue for 3years, even if proper antibiotics were used (Giovanni *et al*., 2006). In this study, blood serum and stool samples of total 100 symptomatic patients was analyzed. *H. pylori* serum antibody was positive in 72 patients and negative in 28 patients. These results were similar to the results reported in Yemen by (Abdulrahman *et al.,* 2014) and close to that reported in Turkey by (Ahmet *et al.,* 2009) where 69% were positive. The results of stool antigen found in this study (36% positive) were varies from those reported in Yemen (49% positive) by (Abdulrahman *et al* 2014) and close to those reported in Turkey (29% positive) by (Ahmet *et al.,* 2009). The different between those studies might attribute to the sample size or the socioeconomic status in different countries.

5.2 CONCLUSIONS

In conclusion this study showed that the distribution of *H. pylori* infection according to gender revealed that the rate was higher in males (90%) than in females (75%), *H. pylori* positive antibody in infected patients was higher 72/82 (88%) than antigen detected in stool 36/82 (44%). The variations between the results of antigen and antibody detection tests elaborate the importance of running different screening tests to avoid the possibility of getting false positive results. Moreover, if possible culture or urea breath test should be done to confirm the results and the eradication of the bacteria by medications upon follow-up.

5.3 RECOMMENDATIONS

1. If the diagnosis was based on serological tests, it is recommended to run more than one test i.e., antigen and antibody.

2. Serum antibody test and stool antigen screening tests can be used in areas with little or no access to medical facilities, but confirmation should be done with urea breath test or other advanced test.

3. If possible invasive techniques (endoscopy) should be used to confirm the laboratory screening tests.

4. Future studies should include symptomatic as well as asymptomatic individuals to confirm the results.

5. Molecular techniques might be used in future research to confirm the positive results obtained by screening techniques.

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APPENDIX

SUDAN UNIVERSITY OF SCIENCE AND TECHNOLOGY

COLLEGE OF GRADUATE STUDIES

Antigen and Antibody Detection Tests Used for the Diagnosis of *Helicobacter pylori* Infection in Symptomatic Patients in Abogota city

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Questionnaire

Patient name:……..

Age:

Gender:

Blood group:

Symptoms:

History of treatment:

Pt. Number	Sex	Age/y	*H. pylori* Ab	*H. pylori* Ag
1	F	33	-	-
2	F	65	-	-
3	F	30	+	-
4	M	75	+	+
5	F	60	+	+
6	M	25	-	+
7	M	58	-	+
8	F	45	+	+
9	F	45	-	-
10	M	20	+	+
11	M	65	+	-
12	M	22	-	-
13	F	35	+	+
14	M	30	+	-
15	M	25	+	-
16	F	21	-	-
17	F	55	+	-
18	F	40	-	-
19	F	26	-	+
20	F	22	+	-
21	F	32	-	+
22	M	73	+	-
23	F	22	+	-
24	M	26	+	+
25	M	36	+	-
47	F	18	+	-
48	M	28	+	-

Family history:

49	F	28	-	-
50	M	27	+	-

Pt. Number	Sex	Age/y	*H. pylori* Ab	*H. pylori* Ag
51	F	23	+	-
52	F	40	-	-
53	M	25	+	-
54	F	23	+	-
55	F	27	-	+
56	M	45	+	+
57	M	19	+	-
58	M	64	+	-
59	F	40	+	-
60	M	46	+	+
61	M	32	+	-
62	F	48	-	+
63	M	45	+	+
64	F	77	+	-
65	F	25	-	-
66	M	30	-	+
67	F	35	+	-
68	M	58	+	-
69	M	35	-	-
70	M	36	+	+
71	M	32	-	+
72	F	34	+	-
73	F	60	+	-
74	F	11	+	+
75	M	50	+	+

Pt. Number	Sex	Age/y	*H. pylori* Ab	*H. pylori* Ag
76	F	27	+	-
77	M	39	+	+
78	F	75	+	-
79	F	25	+	+
80	F	38	+	+
81	M	36	+	+
82	F	38	+	+
83	F	50	+	+

84	M	28	+	-
85	F	45	+	-
86	F	20	+	+
87	M	29	+	-
88	F	13	+	-
89	F	35	-	-
90	M	28	+	+
91	M	37	+	-
92	F	64	+	+
93	F	40	+	-
94	F	25	-	-
95	F	25	-	-
96	F	45	-	-
97	M	22	+	-
98	M	45	-	-
99	F	40	+	+
100	F	34	+	-