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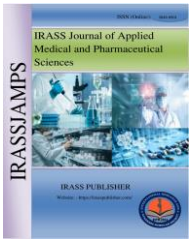

PREVALENCE AND ASSOCIATED RISK FACTORS OF HELICOBACTER PYLORI INFECTION IN ASYMPTOMATIC AND SYMPTOMATIC PATIENTS ATTENDING THE NKWEN DISTRICT HOSPITAL BAMENDA

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Abstract:

INTRODUCTION: *H. pylori* is an important global pathogen infecting many individuals worldwide. This bacterium causes gastritis and peptic ulcer disease and it is also an important risk factor for the development of stomach cancer. Despite progress in diagnosis and therapy in various regions of the world, it affects more than half of the population with a majority of the cases (about 80%) being asymptomatic and induces clinical symptoms in 15-20% of subjects

OBJECTIVE: The aim of this study was to determine the prevalence and associated risk factors of *H. pylori* infection among asymptomatic and symptomatic individuals attending the Nkwen District Hospital.

MATERIAL AND METHODS: This was a hospital based cross-sectional study conducted among 340 individuals who attended the Nkwen District Hospital Bamenda. The participants of this study were individuals presenting with symptoms of *H. pylori* infection who had the test request from the doctor, as well as those who were not having any symptoms randomly selected and accepted freely to participate in the study by signing the consent or assent forms assuring confidentiality. Individuals were allowed to participate in this research study if they had filled their questionnaires, were not undergoing treatment for the infection or had not been treated less than a month prior and also if they could give a stool sample.

Structured questionnaires were filled in other to determine the associated risk factors of *H. pylori* in patients with or without clinical symptoms. To determine the prevalence of this infection, their stools samples were collected and tested for the presence of stool antigen against anti-*Helicobacter pylori* antibody conjugated in a colloid gold nitrocellulose membrane strip.

An ethical clearance was obtained from the ethical review board of the University of Bamenda, administrative consent was gotten from the delegation of Public Health and from the Nkwen District Hospital administration.

Data was analyzed using the statistical software package for social sciences (SPSS) version 22.0 with 95% confidence interval and results displayed on frequency tables having respective percentages. The Chi-square test was used to compare the prevalence between groups and a factor was considered significant if $p \leq 0.05$

RESULTS: Three hundred and forty participants (340) of all ages who attended the Nkwen District Hospital Bamenda took part in this study. The overall prevalence of *Helicobacter pylori* infection in the participants was 32.9% (112/340). The prevalence in the asymptomatic population was 17.36% (33/190) and the prevalence in the symptomatic population was 52.66% (79/150). The associated risk factors of *Helicobacter pylori* infection in asymptomatic individuals were their age group, frequent eating spots, incomplete medications from previous

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infection, frequent intake of NSAIDs, previous operation in the stomach, fasting for long periods, smoking and alcohol consumption (P-values ≤ 0.05). The associated risk factors of *Helicobacter pylori* infection in symptomatic individuals were their age group, occupation, educational level, marital status, frequent eating spot, hand hygiene after using the toilet, source of drinking water, intake of NSAIDs, previous operation in the stomach, having a close relative in your household with stomach cancer or serious gastrointestinal disorders, fasting for long periods and alcohol consumption (p-values≤0.05).

CONCLUSION: In this study carried out to investigate the prevalence and associated risk factors of *Helicobacter pylori* infection in asymptomatic and symptomatic patients attending the Nkwen District Hospital Bamenda,

The overall prevalence of *Helicobacter pylori* infection in the participants was 32.9% (112/340). The prevalence in the asymptomatic population was 17.36% (33/190) and the prevalence in the symptomatic population was 52.66% (79/150).

The associated risk factors of *Helicobacter pylori* infection in asymptomatic individuals were their age group, frequent eating spots, incomplete medications from previous infection, frequent intake of NSAIDs, previous operation in the stomach, fasting for long periods, smoking and alcohol consumption. The associated risk factors of *Helicobacter pylori* infection in symptomatic individuals were their age group, occupation, educational level, marital status, frequent eating spot, hand hygiene after using the toilet, source of drinking water, intake of NSAIDs, previous operation in the stomach, having a close relative in your household with stomach cancer or serious gastrointestinal disorders, fasting for long periods and alcohol consumption.

Keywords: *Helicobacter pylori*, Prevalence, Associated Risk Factors, Symptomatic, Asymptomatic, Bamenda, All ages.

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1.0. Introduction

1.1. Background

Helicobacter pylori is a helix-shaped gram-negative bacterium that infects approximately 4.4 billion individuals worldwide having over 20 recognized strains [1]. When compared to other relatively common causes, such as bile reflux, autoimmune illnesses, long-term use of non-steroidal anti-inflammatory medicines, and ethanol intake, it is the most common cause of chronic gastritis [2]. The stomach remains a highly effective organ and microbial filter despite the innumerable daily injuries it sustains. This is primarily due to its ability to regenerate and repair itself through a cell migration process known as restitution, which begins in minutes (continuous regeneration via differentiation and proliferation of stem cells and progenitor cells leads to repair within days to months) [3]. It can be significantly affected by *H. pylori* infection which will intend cause an immune response in the host causing macrophages, lymphocytes and cytokines to intervene and further contribute to epithelial-mesenchymal transition as well as carcinogenesis [4].

Although the exact way of transmission is still unknown, research from the literature points to a variety of possible routes [5]. The bacterium has been isolated from the saliva, feces, and dental plaque of some affected individuals, which is consistent with these routes of transmission [6]. It has been demonstrated that houseflies can mechanically spread the bacterium, suggesting that inadequate sanitation is a risk factor [7]. It has a significant role in the development of gastric mucosal related lymphoid tissue lymphoma and gastric cancer [8]. It affects almost half of the population, with the majority of instances (approximately 80%) remaining asymptomatic and causing clinical symptoms in 15-20% of people, despite advancements in detection and treatment in different parts of the world [9]. In the light of the rising incidents of gastritis and duodenal ulcers in Cameroon, as well as the importance of *H. pylori* infection in the pathogenesis of gastroduodenal diseases, there is a need to also re-appraise its prevalence rate in a cross-section of asymptomatic individuals. Using this background information, the epidemiology of *H. pylori* in both asymptomatic and symptomatic individuals is essential for health planners and program managers to come up with several strategies to improve health care and will provide a great opportunity for screening and identifying this bacterium in this population which could have served as a natural reservoir for future generations.

1.2. Statement of problem

In Cameroon where there is a rise in the incidents of gastritis, duodenal ulcers and stomach cancers, there is an increase in the importance of *H. pylori* infection in the pathogenesis of such diseases. Over the years, several research studies have been carried out on the prevalence of *H. pylori* infection and its associated risk factors such as in patients with gastritis in West Cameroon by Nathan et al [7] and in Nairobi County, Kenya by Khamisi [6] with small attention paid to asymptomatic individuals and the complete eradication of the bacteria after diagnosis. This research will provide room for screening of *H. pylori* in individuals of all ages providing a tool for future diagnosis in asymptomatic patients as well as a call for eradication of the infection in symptomatic patients, preventing progression of the disease condition bringing out the associated risk factors relating to the infection. Despite the fact that several measures have been put in place to combat the

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spread of the *H. pylori* infection like talks from health personnel, the use of several diagnostic techniques [10] and treatment methods [11]it is still prevalent.

1.3. Research question(s)

- What is the prevalence of *Helicobacter pylori* infection in symptomatic and asymptomatic patients attending the Nkwen District Hospital Bamenda?
- What are the associated risk factors of *Helicobacter pylori* infection in asymptomatic and symptomatic patients attending the Nkwen District Hospital Bamenda?

1.4. Objectives

1.4.1. General objectives

- To evaluate *Helicobacter pylori* infection prevalence and associated risk factors in both symptomatic and asymptomatic patients at Nkwen District Hospital Bamenda.

1.4.2. Specific objectives

- To determine the prevalence of *Helicobacter pylori* infection in asymptomatic and symptomatic patients attending the Nkwen District Hospital Bamenda.
- To identify the associated risk factors of *Helicobacter pylori* infection in symptomatic and asymptomatic patients attending the Nkwen District Hospital Bamenda.

1.5. Hypothesis /impact of research

- There is prevalence of *Helicobacter pylori* infection in asymptomatic and symptomatic patients who attend the Nkwen District Hospital Bamenda.
- There are risk factors associated risk factors with *Helicobacter pylori* infection in asymptomatic and symptomatic individuals attending the Nkwen District Hospital Bamenda.

1.6. Significance of the Study

The results from this study will educate the general public about the prevalence and prognosis of *H. pylori* infection and also help to reduce the advancement of the disease through early diagnosis and reduce the rate of transmission of the infection through sensitization about the factors associated risk with the bacterium. This research will go on to strengthen other established research relating to this field as it will give more insights about the prevalence of *H. pylori* not only in patients posing with symptoms but also those without.

LITERATURE REVIEW

2.1. Introduction

2.1.1. *Helicobacter pylori* infection in asymptomatic individuals

Clinical data over an extended period of time suggest that this virus is not harmless. Approximately 80% of *H. pylori*-affected cases globally are asymptomatic [9]. Iron and vitamin malabsorption, pancreatic secretory dysfunction, and disturbances of intestinal myoelectric activity are among its potential side effects [9]. Before life-threatening complications arise, some PUD (peptic ulcer disease) patients have no symptoms [13]. Strong positive correlations between BMI and asymptomatic PUD were found in one investigation. Furthermore, research has shown that obese people with higher BMIs have higher "somatic" pain thresholds due to increased endorphin plasma levels [14]. In patients with a family history of gastric cancer, asymptomatic infectious eradication of *H. pylori* is a preventative measure [9]. However, it has been suggested that silent infections have altered oxygen metabolism, and the existence or lack of infection symptoms is dependent on a variety of factors, such as environmental factors, intricate bacterial-host interactions, and epigenetic factors [9]. Additionally, either reinfection or recrudescence may result in an *H. pylori* recurrence following treatment [15];

- Both small amounts of dormant *H. pylori* lurking in the human body (primarily in vitro factors like therapeutic scheme, treatment time window, and re-examination time) and false-negative results (primarily in vivo factors like oral colonization, biofilm formation, and formation of coccoid forms) can cause recrudescence. Usually, it happens in a year.
- Infection with a new strain or a strain that is similar to the original H. pylori stain is known as reinfection.

Also knowledge of *H. pylori* status in asymptomatic individuals is important so if symptoms start to arise, not only will they be treated for other symptoms like pain and nausea but also measures will be put in place for *H. pylori* eradication.

2.1.2. *Helicobacter pylori* infection in symptomatic individuals

About 15-20% of people globally experience symptoms of chronic gastritis due to *Helicobacter pylori* infection [9]. The negative effects of *Helicobacter pylori* cytotoxins and increased release of pro-inflammatory cytokines and prostaglandins (inflammatory mediators) lead to inflammatory and destructive alterations of the stomach mucosa [16]. Enterochromaffin cells proliferate as a result of these cytokines. These cells are the primary source of serotonin in the GIT, where tryptophan hydroxylase (TpH-1) converts L-tryptophan to serotonin. In the digestive system, these cells release serotonin, which makes up around 90% of the hormone's entire body pool. The type of receptor determines how serotonin works (5-HT1 produces fundus relaxation and stomach secretion; 5-HT2, 5-HT3, 5-HT4 cause gastric motility [17]. 5-HT3 receptors are found in the GIT, mostly on vagal sensory fibers that transmit signals from the GIT to the central nervous system (CNS), which includes the limbic system, the brain, and the sensory cortex, which is where pain is located and consciously felt [18]. Early satiety, nausea, vomiting, and visceral hypersensitivity are all caused by 5-HT3 activation [19]. After *H. pylori* is eradicated, TpH-1 is downregulated, which suggests that the bacteria is responsible for the disruption of serotonin homeostasis in the stomach [9]. Nevertheless, a variety of factors, such as the quantity of enterochromaffin cells, TpH-1 gene polymorphism, or the existence of antibodies against this enzyme, may influence the degree of TpH expression [20]. Chronic active gastritis is the main condition that follows *H. pylori* colonization and is present in all symptomatic positive people [6]. Numerous variables, including the traits of the invading strain, host genetics and immune response, food, and the degree of acid production, influence the intragastric distribution and intensity of this chronic inflammatory process [6].

2.2. Epidemiology of *Helicobacter pylori* infection

Prioritizing and tailoring public health initiatives to better manage the disease's burden will be made easier with an understanding of the worldwide epidemiologic patterns of *H. pylori* [11]. Poor socioeconomic conditions appear to be linked to increased *H. pylori* prevalence in both industrialized and

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developing nations [21]. The potential pathologic outcome of the infection appears to be controlled by the age at which this bacterium is acquired: people who contract *H. pylori* early in life are more likely to experience more severe inflammation, which may lead to atrophic gastritis and a higher risk of gastric ulcer, gastric cancer, or both [22]. Conversely, since children are more likely to be exposed to germs through family transmission, especially from infected parents or siblings, incidence in children is correlated with prevalence in adults [23]. Prior research in Cameroon found that the prevalence of *H. pylori* was 92.2% in gastric biopsies of patients with gastroduodenal diseases and 72% in biopsy samples showing signs of gastritis [24]. All around the world, peptic ulcers and chronic gastritis are very common [25]. *H. pylori* gastritis is the main cause of active persistent gastritis, and it can induce major side effects such adenocarcinoma and mucosa associated risk factors lymphoid tissue (MALT) lymphoma [26].

H. pylori infection is becoming less common worldwide, while this decline has only been seen in industrialized nations [27]. Eliminating *H. pylori* greatly reduced inflammation in the stomach, accelerated the healing of ulcers, and stopped gastric cancer [28]. Antibiotic use must be supplemented with reinfection prevention strategies because eradication therapy does not prevent future reinfection [29]. Vaccination is a clear substitute for antibiotics [29]. Unfortunately, the development of a therapeutic *H. pylori* vaccination is hampered by the fact that *H. pylori* strains can infect individuals again even when antibodies are present [29]. Nonetheless, a preventative *H. pylori* vaccine was found to be effective in a field test conducted on school-age children [30]. This indicates that *H. pylori* infection may be avoided in situations with a low bacterial burden [29]. In the interim, we recommend that eradication be coupled with sanitation and clean water supply provision, education to enhance hygiene, and perhaps coordinated eradication across families [29].

2.3. Pathogenesis of *Helicobacter pylori* infection

More than half of the world's population is infected with *Helicobacter pylori*, which is frequently the cause of persistent bacterial infections [31]. Numerous factors, including host immunological responses, environmental factors, and bacterial factors, interact to induce disease [32]. Proteases, vacuolating cytotoxic A (VacA), and specific phospholipases are among the biochemicals produced by *H. pylori* that harm epithelial cells, break down tight junctions, and ultimately trigger apoptosis. In addition to causing inflammation, cytotoxic associated gene A (CagA) has the potential to induce cancer. Adhesins (which adhere to epithelial cells and cause colonization), porins, iron transporters, flagellum-associated proteins (which facilitate mucous movement and chemotaxis to the epithelium), and proteins with lesser-known functions are the five most significant outer membrane proteins that *H. pylori* possesses [6]. Phospholipids and lipopolysaccharide (LPS) make up *H. pylori's* outer membrane, just like those of other common gram-negative bacteria. LPS's O antigen might be fucosylated and resemble the gastric epithelium's Lewis blood group antigens [6]. Additionally, it generates Urease, an enzyme that converts urea—which is typically released in the stomach—into carbon dioxide and ammonia, which are nutrients for bacteria and damage the gastric epithelium. After receiving a proton (H^+), the ammonia is converted to ammonium, which neutralizes stomach acid. Additionally, the organism can build biofilms and change from a spiral to a viable but coccoid form, both of which are likely to help the bacterium survive [6].

2.4. Associated risk factors of *Helicobacter pylori* infection

Understanding of some of the associated risk factors may thus be very important for the recognition of *H. pylori* in the etiology of gastrointestinal pathologies [6]. The associated risk factors of *H. pylori* infection have been investigated in several countries which includes [13];

- **Low level of education:** These persons usually have low knowledge about prevention of the infection.
- **Number of residents per household:** Living with a lot of other people can make you more susceptible to *H. pylori.*
- **Drinking municipal or tank water:** One way to lower the risk of *H. pylori* is to have a consistent supply of clean water.
- **Poor sanitation:** This covers food exposure and inappropriate food handling. It has been demonstrated that houseflies can manually spread the bacterium, suggesting that unsanitary practices increase the chance of infection [7].
- Living in a rural area.

2.5. Signs and symptoms of *Helicobacter pylori* infection

About 15-20% of people experience clinical symptoms from *H. pylori*, while the majority of cases (about 80%) are asymptomatic [9]. According to other research, symptoms either go away or become less frequent and severe with or without getting rid of the bacteria [33]. Iron deficiency anemia of unknown source, epigastric pain, persistent vomiting, and malnourishment could all be symptoms of infection-related consequences [34]. Additional related symptoms include

- Ulcers; more severe vomiting usually accompanied with blood, stool is usually dark, pain related to hunger which may be relieved by food or not, pain can radiate to the back, heart burn.
- Belching, bloating and nausea.

2.6. Transmission of *Helicobacter pylori*

The mode of transmission still remains unclear, but literature data suggests different modalities of transmission of the infection which included; [6]

- Person-to-person (through tubes or saliva)
- Faeco-oral (water contaminated with feces may predispose to infection and if it’s municipal, potentiate it’s spread.
- Oro-oral (the bacteria may colonize dental plaque and saliva and be transmitted by saliva to another).
- Gastro-oral (typical in children when *H. pylori* use mucous vomitus of children).
- Gastro-gastric (through endoscopes).

Less than 1,000 organisms per milliliter are often seen in the sterile stomach and upper small intestine. The hydrochloric

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acid and resulting low pH of the stomach, along with gastric enzymes, typically destroy organisms that enter the stomach. Bile and pancreatic enzymes may kill other organisms once they reach the small intestine. Bacteria of oral, nasopharyngeal, or colon origin may colonize the stomach when the pH rises over 5.0.

2.7. Diseases caused by *Helicobacter pylori*

Numerous studies have shown that *H. pylori* infection is the bacterial cause of both malignant and non-malignant and non-malignant gastroduodenal diseases and are also involved in extra-duodenal disorders. Among these the most common disorders are [35];

- Peptic and duodenal ulceration
- Acute and chronic gastritis which may lead to atrophic gastritis
- Gastric adenocarcinoma (B-cell gastric lymphoma and MALT lymphoma).

H. pylori is a cause for concern in pregnancy as well. This may be due to the fact that there are immunologic adaptations in pregnancy to ensure maternal tolerance towards the semi-allogenic fetus. It leads to the following complications during pregnancy;

- Iron deficiency anemia: Due to low gastric Ph, reduction of stomach vitamin C levels, bacterium-host competition for dietary iron supply.
- The hallmark of hyperemesis gravidum (HG) is intense, prolonged vomiting, which frequently leads to electrolyte imbalance, weight loss, and dehydration.
- Others include miscarriage, pre-eclampsia and fetal growth restriction.

2.8. Laboratory diagnosis of *Helicobacter pylori*

Since the presence or absence of the bacterial infection dictates the type of treatment to be administered or serves as a helpful way to track the efficacy of antimicrobial treatment, testing for *H. pylori* infection has become a crucial component of the diagnostic process for gastric and duodenal inflammatory disease [6]. As a result, *H. pylori* testing is useless on its own; instead, it should be done to determine the etiology of an underlying problem, like gastric ulcer disease, or to avoid disease in people who have a history of infection or familial stomach cancer [6]. similar to when a patient has no symptoms.

2.8.1. Non-invasive methods

2.8.1.1. Stool antigen test using *Helicobacter pylori* strip

The detection of *H. pylori* antigen in feces is a quick, non-invasive test that is simple to conduct and collect specimens for. The test's benefits include the ability to identify active infections, track treatment efficacy, and verify recovery following antibiotic administration [6]. Additionally, unlike upper gastrointestinal endoscopy, the patient does not need to be prepared beforehand. This test has a greater sensitivity and specificity than the other *H. pylori* detection techniques. There is a 90% correlation between the *H. pylori* stool antigen test and reference techniques like endoscopy, histology, and the urea breath test [6].

Principle

This test is ready to use and is based on the use of latex microspheres and membrane technology. Antibodies against *H. pylori* are used to sensitize a nitrocellulose membrane [10]. Latex microspheres are coupled to monoclonal antibodies. On a membrane, this conjugate is dried. The faecal sample is diluted in the buffer that is supplied with the test. When the liquid phase of the faecal suspension comes into contact with the strip, the solubilized conjugate migrates with the sample by passive diffusion and both conjugate and sample material come into contact with a monoclonal antibody directed against a specific antigen of *H. pylori*. A red line will form and the conjugate-*H. pylori* complex will stay attached to the monoclonal antibody adsorbed onto the nitrocellulose membrane if the sample contains this particular *H. pylori* antigen. The solution keeps migrating until it comes into contact with a second reagent (the control reagent), which binds the migration control conjugate and creates a green control line, indicating that the test is functioning correctly. Result is visible within 10 minutes. It has a sensitivity of 92.3% and a specificity of 100% [10].

Requirements

- *H. pylori* strips.
- Dilution buffer.

Specimen

Stool

Test procedure

- Put on all appropriate personal protective equipment.
- Label a stool container with patient's name, sex, age, date, laboratory number and type of test is given to the patient.
- The patient is given instructions on how to produce and bring the stool sample to the laboratory.
    - First pass out urine into the toilet if you have to. The stool specimen should not come in contact with water or urine.
    - Carefully unscrew the cap from the plastic container. Do not touch the inside of the container or the lid with your fingers.
    - You can stool on tissue paper and carefully pick the sample with the applicator stick at different places and place in the cup and cap (if the outside of the container is contaminated with stool, wash the exterior with soap and water, dry and place in the bag provided).
    - Wash your hands with soap and water and bring the container to the lab.
- Add 14 drops of the dilution buffer solution into a labelled tube.
- Put the stool sample in a loop and dip it into the tube. Take two loops of 10µl for liquid samples and one loop for solid samples, each from a separate part of the sample.
- Discard the sampling loop.

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- To homogenize the preparation, vortex it. It is necessary to suspend the complete feces sample in the solution.
- The sensitive strip should be dipped in the direction that the red arrow points.
- Allow to react for 10 minutes [10].

Results and interpretation

The results are interpreted as follows;

- **Negative test result:** At the Control line (C) location, a green line emerges. There isn't another band.
- **Positive test result:** There is a red band at the Test line location (T) in addition to a green band at the control line position. Depending on how many antigens are present in the sample, the test line's intensity may change. Any red line, no matter how faint, should be interpreted as a favorable outcome.
- **Invalid test result:** A test method failure is indicated by the lack of a control line. Use a fresh strip to repeat tests that are invalid [10]. The most common causes of control line failure are inadequate specimen volume or improper procedural methods. Repeat the test using a fresh test cassette after going over the steps again. Stop using the test kit right away and get in touch with the distributor if the issue continues [35].
- **Note**: After the 10-minute reaction period has elapsed, do not consider the emergence of additional lines [10]. Figure 1 below provides an overview of the test process.

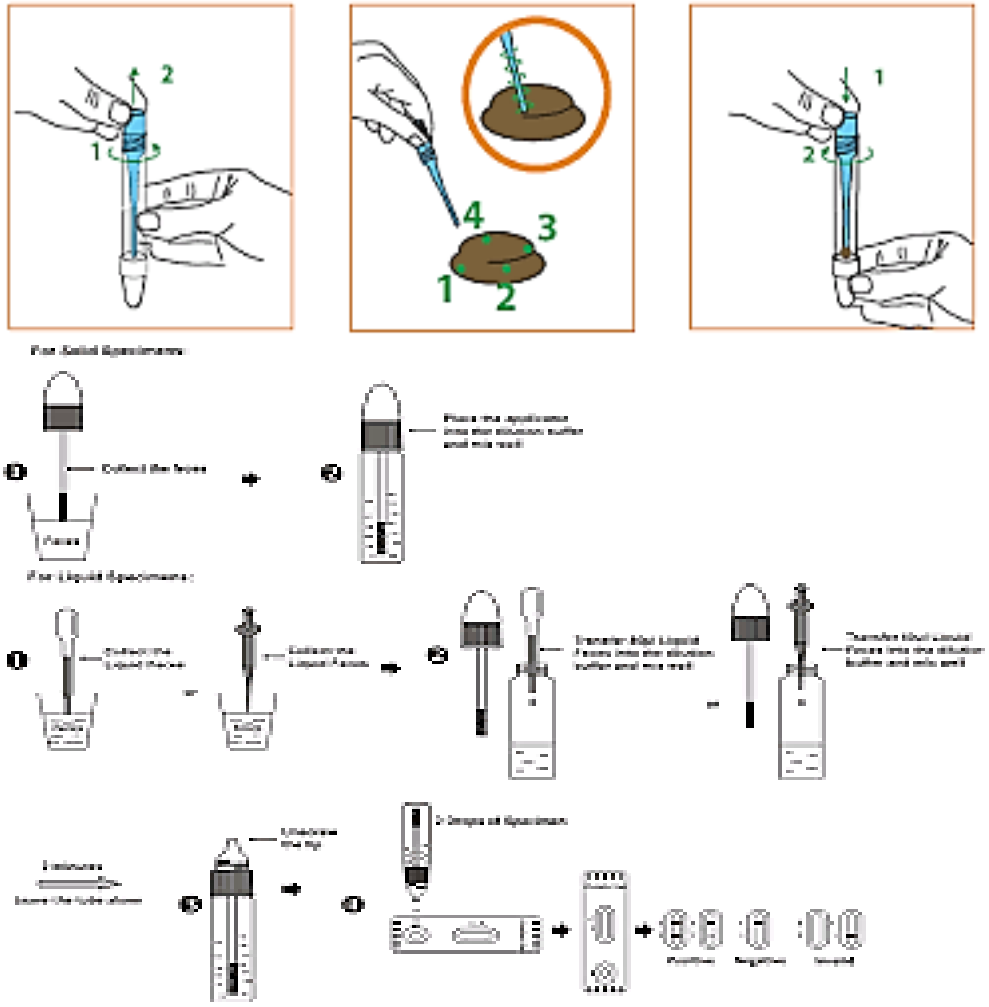

Figure 1: Summary of stool antigen test procedure.

Test limitations

- The test is qualitative so cannot predict the quantity of antigens present in the sample.
- A positive test does not rule out the possibility that other pathogens may be present [10].

2.8.1.2. Blood antibody test using *Helicobacter pylori* Antibody Rapid Test Cassette (whole blood/serum/plasma).

This quick chromatographic immunoassay helps diagnose *H. pylori* infection by qualitatively detecting *H. pylori* antibodies in whole blood, serum, or plasma. Its specificity is 93.0% and its sensitivity is 96.8% [35].

Test principle

Anti-human IgG is immobilized in the test line region during this test method. Following its addition to the device's specimen well, the specimen reacts with the test's *H. pylori* antigen-coated particles. This mixture interacts with the immobilized anti-human IgG and migrates chromatographically over the course of the test. A positive result will be shown by the appearance of a colorful line in the test line region (T) if the specimen contains *H. pylori* antibodies. A negative result will be indicated by the absence of a colored line in this area if the material

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is devoid of *H. pylori* antibodies. A colored line will always show up in the control line region (C) to act as a procedural check, signifying that the correct volume of specimen was added and that the test protocol was closely adhered to [35].

Reagents

Test cassette containing *H. pylori* antigen coated particles and anti-human IgG coated on the membranes.

Specimen

- Whole blood, serum or plasma.

Test procedure

- Put on all appropriate personal protective equipment.
- Collect venous blood into a Dry or EDTA tube.
- Allow blood in the dry tube to clot. Centrifuge the blood to get either serum or plasma. If whole blood is needed, mix the blood in the EDTA tube or do a fingerpick.
- Remove the test cassette from the sealed foil pouch, label with patient's laboratory identification number and place on a clean flat surface.
- For serum or plasma specimen; Transfer 100µl of serum or plasma to the specimen well of the test cassette and start the timer. Read results at 10 minutes.
- For whole blood specimen; Transfer 75µl of whole blood to the specimen well of the test cassette, add 2 drops of buffer and start the timer. Read results at 10 minutes [35].

Results and interpretation

The results are interpreted as follows;

- **Positive; Two distinct colored lines appear.** Both are located in the test region (T) and the control region (C). Depending on the amount of *H. pylori* antibodies in the samples, the test region's color intensity will change. As a result, any color in the test area ought to be interpreted as positive [35].
- **Negative; One colored line appears in the control region (C).** In the test region (T), there isn't a visible colored line.
- **Invalid; Control line fails to appear.** The most common causes of control line failure are inadequate specimen volume or improper procedural methods. Repeat the test using a fresh test cassette after going over the steps again. Stop using the test kit right away and get in touch with the distributor if the issue continues [35].

Note: After the 10-minute reaction period has elapsed, do not consider the emergence of additional lines [35]. A summary of the test procedure is shown on figure 2.

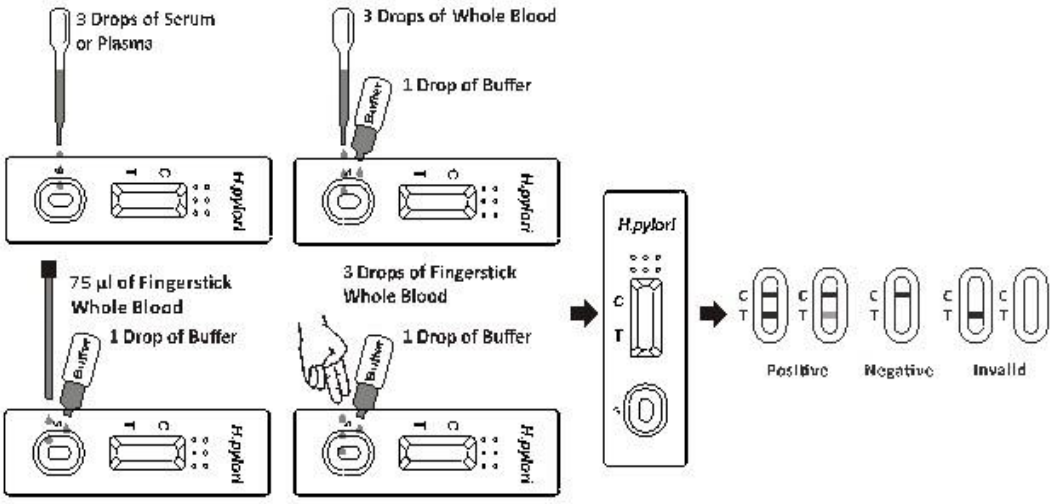

Figure 2: Summary of blood antibody test procedure.

Test limitations

- It is a qualitative test, hence neither the quantitative value nor the rate of increase in *H. pylori* antibody concentration can be determined.
- The test kit has been compared with culture/histology, demonstrating an accuracy of 94.6%. Therefore, if the test result is negative and clinical symptoms persist, additional testing using other clinical methods should be done [35].
- The test cannot differentiate between past and current infection. It cannot tell which antibody is present. It only tells that the test is either positive or negative [35].

2.8.2. Invasive methods

2.8.2.1. Rapid Urease Test

It is a widely used diagnostic procedure to identify *H. pylori*. It is a quick, inexpensive, and easy test that finds out if the stomach mucosa has the enzyme urease. This test gathers stomach lining cells using a process known as endoscopy and biopsy. Its sensitivity is 99% and its specificity is 100% [36] [37].

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Test principle

Urea is the product of decarboxylation of amino acids. Hydrolysis of urea produces ammonia and CO_2. The medium is alkalinized by the formation of ammonia, and the pH shift is indicated by the color change of phenol red from yellow at pH 6.7 to pink at pH 8.1. Urease-positive organisms that are rapidly active turn the entire medium pink in a day, while weakly positive organisms may take several days, and negative organisms produce no color change or yellow.

$(NH_2)_2CO + 2H_2O \longrightarrow CO_2 + H_2O + 2NH_3$.

Specimen

Biopsy of mucosa.

Reagents

Urea Agar medium (pH 6.7). It contains 1ml distilled water, one drop of 1% phenol red indicator and 100mg urea [38].

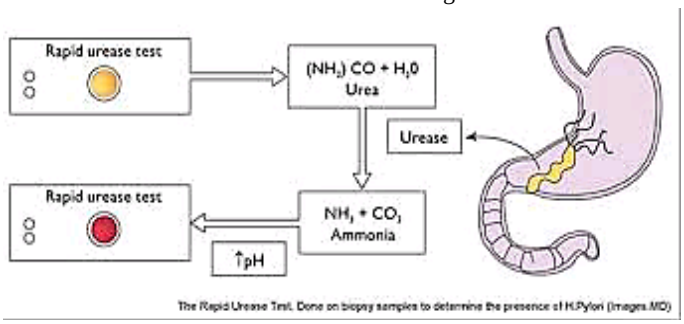
Procedure.

The test is performed at the time of gastroscopy (examination of the upper digestive tract-esophagus, stomach and duodenum). A biopsy of mucosa is taken from the antrum of the stomach and placed into Urea Agar Medium which is maintained at room temperature. If the bacterium is present, urease enzyme will be produced which will hydrolyze urea to ammonia producing a pink color [37] [39]. The result is read within 24 hours.

Result interpretation

- Positive; Pink color.
- Negative; Yellow color.

An illustration of the test result is shown on figure 3 below.



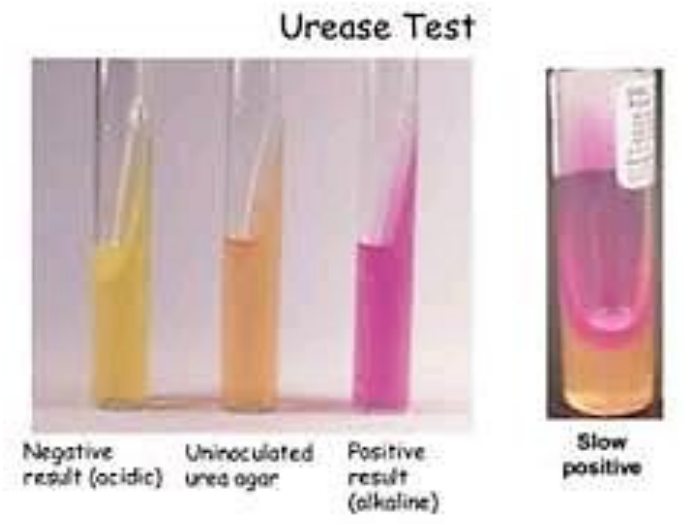


Figure 3: An illustration of rapid urease test result.

Test Limitations

- After prolonged incubation time, a false-positive alkaline reaction may be seen.
- Urea Agar cannot be used to determine the quantitative rate of urease activity as organism vary in their capability and rate of hydrolysis [37].

2.8.2.2. Histopathology

It is the study of disease in a tissue section. The tissue undergoes a series of steps before it reaches microscopy where diagnosis is made [39] [40].

Principle

Hematoxylin and Eosin staining technique functions to recognize different types of tissues and their morphological changes. Hematoxylin has a deep blue-purple color which stains nuclei of cells blue. Eosin has a pink color which stains the cytoplasm and extracellular matrix pink [39].

Specimen

Esophagus, stomach or duodenal biopsies.

Reagents

- Hematoxylin and Eosin stains.
- Paraffin.
- Alcohol
- Xylene.

Test procedure

- Obtain biopsies from the esophagus, stomach or duodenum and fixed in 10% formalin then sent to the laboratory.
- Make 4mm thick sections from the biopsies.
- Process in alcohol and xylene then embed in paraffin.
- Section the tissues into many 5-micron sections, then select the best and place on a slide.
- Place in the incubator for 15 minutes and stain using hematoxylin and eosin stain.
- Observe microscopically [40].

Results interpretation

An observation of a discontinuous mucosa is an indication of gastric and esophageal ulcers.

Limitation

- Hematoxylin and Eosin stains are inefficient in that not all features of a tissue are stained [39].
- It requires expert pathologist to provide histological data on inflammatory and atrophy [6].

NOTE; Other invasive methods include: microbiological culture and Polymerase Chain Reaction [41].

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2.9. Treatment of *Helicobacter pylori* infection

The identification of *Helicobacter pylori* led to a significant advancement in the management of stomach and duodenal disorders. Eliminating the *H. pylori* bacterium, which is thought to be a contributing cause, can either treat or prevent these illnesses [42]. Concurrent use of three or four medications can result in an acceptable *H. pylori* eradication effectiveness of above 80%. Proton Pump Inhibitors (PPIs) such as omeprazole, lansoprazole, and esoprazole are among these medications; they are frequently used in conjunction with antibiotics such as amoxicillin (Amx), clarithromycin (Cl), and tetracycline (Tetr) and chemotherapeutics such as metronidazole (Met) or tinidazole, levoflaxacine (Lev), moxifloxacine, furazolidone (Fur), and bismuth salts [42].

- **First-line therapies**

Treatment options. Twice daily for 7, 10 or 14 days.

PPI + Amx + Met

PPI + Cl + Met

PPI + Amx + Cl

- **Second-line therapies**

Treatment options. For 7, 10 or 14 days, quadruple therapy.

PPI + Amx + Met + Tet

PPI + Bismuth + Amx + Met

PPI + Bismuth + Tetr + Met

For 10-14 days

PPI + Bismuth + Tet + Met

For 10 days

PPI + Amx +Lev

PPI + Fur + Tet + Bismuth

PPI + Fur + Lev

- **Third-line therapies**

H. pylori sensitivity to Amx, Cl, Met, Lev, and Tetr.

treatment based on H. pylori's antibiotic sensitivity.

2.10. Prevention of Helicobacter pylori infection

Based on the results of recent studies, the following measures may help to reduce transmission of *H. pylori* [43] [44].

- Practice of good hygiene and hand washing especially after visiting the toilet and with food preparation.
- All patients with gastrointestinal symptoms that may be associated risk factors with *H. pylori* infection should be tested and treated to prevent exposure to family members.
- Patients should complete the full course of therapy (antibiotics and acid blockers) to maximize the potential for cure.
- Support policies to improve living conditions in developing countries.
- Maintain proper nutrition to prevent iron deficiency anemia.
- Increase cruciferous vegetable intake (like cabbage).

Stress and spicy foods do not cause ulcers but prevent them from healing quickly or make the pain worse [44].

3.0) MATERIALS AND METHODS

3.1. Study Site

This study was carried out in Nkwen District Hospital PMI Bamenda (NDH). Bamenda is the chief town of the North West Region –Cameroon with over 594,000 inhabitants as of 2023 demographic data. NDH is strategically located in the heart of Nkwen. It was upgraded to a Sub Divisional Medicalized Center around the year 2000 and it's the oldest and biggest district hospital in the region. The center has Mother to Child Services (antenatal consultation, maternity, vaccination and family planning), Medico-Consultation services (General consultation, pediatric, medical checkup, theatre) and other units like physiotherapy, non-communicable diseases, HIV treatment unit, Dental cabinet.

3.2. Study design and population

This was a hospital based cross sectional study carried out on individuals of all ages who were attending NDH at the time of study including both the outpatient and inpatient departments. The participants were recruited after signing the consent or assent forms and answering all the questions on the questionnaire that were provided. The sample size of 384 was calculated using Cochran formula of 95% confidence interval and the subjects were chosen randomly.

This study was carried out over a period of two (2) months, beginning from 1st April 2023 to 31st May 2023.

3.3. Sampling techniques

Exhaustive sampling was used where every person who met the inclusion criteria was selected.

- Individuals who were presenting with symptoms associated risk factors with *H. pylori* infection who had the test request from the doctor and accepted to participate in the research.
- Randomly selected individuals who were not presenting with any symptoms of *H. pylori* infection.

This study excluded;

- Individuals with incomplete questionnaires.
- Individuals who were undergoing treatment for *H. pylori* infection or had been treated for the infection less than one month prior.
- Patients who were critically ill and could not give stool sample.

3.4. Data collection

Interviewer guided questionnaires were administered to obtain information on socio-demographic characteristics, dietary habits, as well associated risk factors linked to *H. pylori* infection in both asymptomatic and symptomatic individuals. These questionnaires provided original data to be used for statistics. *H.*

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pylori stool antigen strips were used to carry out the tests for the bacterium.

3.6. Ethical considerations

Authorizations to carry out this research study were gotten from the institutional review board of the Faculty of Health Sciences University of Bamenda, Regional delegation of Public Health and Director of the NDH. Participants included only volunteers who signed the consent or assent forms that were attached to each coded questionnaire. They could freely withdraw from the study at any time without any consequences.

3.7. Data analysis

The data collected was analyzed using the statistical software package for social sciences (SPSS) version 22.0 and results displayed on frequency tables along with their percentages. The chi square test was used to determine significant associations between variables at 95% confidence interval, with a p-value set at ≤ 0.05.

3.8. Sample collection and laboratory procedures

The participants were informed about the entire procedure and stool cups were handed to them. All laboratory procedures were carried out while wearing the appropriate personal protective equipment. The test carried out was the stool antigen test using *H. pylori* strip. This test is preferable to other diagnostic methods because it is very sensitive, non-invasive and also best in conforming post-treatment eradication as compared to serology for *H. pylori*, which can remain positive for months to years even after eradication. The test was carried out following the manufacturer's instructions.

- After the individual met the inclusion criteria, a stool container with patient's name, sex, age, date, code and type of test is given to the patient.
- The patient was given instructions on how to produce and bring the stool sample to the laboratory.
  - First pass out urine into the toilet if you have to. The stool specimen should not come in contact with water or urine.
  - Carefully unscrew the cap from the plastic container. Do not touch the inside of the container or the lid with your fingers.
  - You can stool on tissue paper and carefully pick the sample with the applicator stick at different places and place in the cup and cap (if the outside of the container is contaminated with stool, wash the exterior with soap and water, dry and place in the bag provided).
  - Wash your hands with soap and water and bring the container to the lab.
- The sample was received and time noted.
- The test supplies were tested with a known control to ensure that the strips were in good shape and the buffer functioning well.
- 14 drops of the dilution buffer solution were added into a labelled dry tube.
- A loop containing the stool sample was dipped into the tube (for liquid samples, take 2 loops of 10µl, for solid samples, take 1 loop and it should be taken from different areas of the sample).
- The sampling loop was discarded.
- The sample-buffer solution was gradually swirled to obtain homogeneity. The entire stool sample must be suspended into the solution.
- The sensitized strip was then dipped into the solution in the direction indicated by the red arrow and the alarm set.
- It was allowed to react for 10 minutes and observed [10].

Results and interpretation

The results were interpreted as follows;

- **Negative test result:** At the Control line (C) location, a green line emerges. There isn't another band.
- **Positive test result:** There is a red band at the Test line location (T) in addition to a green band at the control line position. Depending on how many antigens are present in the sample, the test line's intensity may change. Any red line, no matter how faint, should be interpreted as a favorable outcome.
- **Invalid test result:** There is a red band at the Test line location (T) in addition to a green band at the control line position. Depending on how many antigens are present in the sample, the test line's intensity may change. Any red line, no matter how faint, should be interpreted as a favorable outcome.

3.9. Study variables

The following variables were of interest to us in this study.

3.9.1. Independent variables

i. Socio-demographic characteristics

This included gender, age, marital status, employment status, level of education and living conditions.

ii. Clinical variables

Which included; *H. pylori* infection and treatment history, previous gastroduodenal disorders, heart burn, acid regurgitation, excessive belching, nausea, vomiting, early satiety, family member with *H. pylori* infection or previous gastroduodenal disorder, smoking and alcohol consumption, treatment options used prior to consultation.

iii. Dependent variable

The dependent variable was a positive *H. pylori* positive test.

4.0. RESULTS AND DISCUSSIONS

4.1. Socio-demographic characteristics of the participants

The table below shows the demographic representation of participants. A total of 340 participants were recruited for this study. 234(68.8%) participants were female and 106(31.2%) were male with 190(55.9%) of the participants being asymptomatic and 150(44.1%) symptomatic. The majority of participants were within the age range of 0-20 years.

Table 3: Socio-demographic representation of the participants

Variable	Category	Frequency	Percentage

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Sex	Female	234	68.8
	Male	106	31.2
	Total	**340**	**100.0**
Age group	0-20	194	57.1
(years)	21-30	58	17.1
	31 and above	88	25.9
	Total	**340**	**100.0**
Marital status	Cohabiting	6	1.8
	Divorced	10	2.9
	Married	91	26.8
	Single	233	68.5
	Total	**299**	**100.0**
Religion	Christians	227	66.8
	Muslims	29	8.5
	Others	84	24.7
	Total	**340**	**100.0**
Occupation	Employed	88	25.9
	Unemployed	5	1.5
	Student	202	59.4
	Retired	20	5.9
	Children < 2 years	25	7.4
	Total	**340**	**100.0**
Level of education	None	40	11.8
	Primary	133	39.1
	Secondary	59	17.4
	Higher	108	31.8
	Total	**340**	**100.0**

4.2. Prevalence of *Helicobacter pylori* infection among patients attending the Nkwen District Hospital Bamenda

A univariate analysis of *H. pylori* stool antigen test was done to establish the overall prevalence of the infection. *Helicobacter pylori* stool antigen test was used as the diagnostic test of choice in accordance with the results from the comparison of stool antigen and blood antibody test methods of detection of *H. pylori* infection by Abdulrahman et al [39] and comparison of five diagnostic methods for *H. pylori* by Mohammad et al [41]. Out of the 340 participants, 112(32.9%) were reactive(positive), with 33(29.5%) being asymptomatic and 79(70.5%) being symptomatic. The remaining 228(67.1%) participants were non-reactive(negative), with 157(68.9%) being asymptomatic and 71(31.1%) being symptomatic. The prevalence for symptomatic individuals only was 52.66% (79/150) and that for asymptomatic individuals was 17.36% (33/190). The prevalence of 52.66% in the symptomatic population was similar to the study carried out in Cameroon on patients with gastro-duodenal pathologies in 2008 by Ndip et al with a prevalence of 52.27% [5] and lower than that carried out on patients with peptic ulcers in Nairobi County, Kenya in 2019 by Khamisi with a prevalence of 46.2% [6]. The prevalence of 17.36% in the asymptomatic population was lower than those in similar studies carried out among women in a rural population in 2004 by Escobar with a prevalence of 44.8% [25] and in the asymptomatic population of middle aged to elderly people in 1998 by Rothenbacher et al with a prevalence of 34.8% [21]. The findings in this study were similar to those done by Nathan et al [7] and Escobar et al [23], that the prevalence of the infection may be due to differences in age group of participants, educational levels, alcohol consumption and sources of drinking water. The results showed that, the prevalence of *H. pylori* infection in patients attending the NDH Bamenda was 32.9% ($p<0.001$). The overall prevalence, 32.9% was lower than those in similar studies carried out in the North West Region of Cameroon in 2017 by Edith et al with a prevalence of 47.4% [45], and in West Cameroon in 2018 by Nathan et al with a prevalence of 43.4[7]. The low prevalence can be explained by the fact that this study was not focused only on patients with gastritis. This overall prevalence was higher than that carried out among children living in a rural setting of Sub-Saharan Africa in 2017 by Asante et al with a prevalence of 14.2% [46]. The high prevalence in Bamenda can be due to increased immigration of individuals from rural areas who never had access to proper health care due to recent conflicts in the region.

Table 4: Overall prevalence of Helicobacter pylori amongst patients.

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Test status	Asymptomatic	Prevalence	Symptomatic	Prevalence	Overall frequency	Overall prevalence
Positive	33	17.36	79	52.66	112	32.9
Negative	157	82.4	71	47.34	228	67.1
Total	**190**	**100.0**	**150**	**100.0**	**340**	**100.0**

4.3. Associated risk factors of *Helicobacter pylori* infection

The associated risk factors of *H. pylori* infection were assessed by asking a total of thirteen (13) questions which included the biological, community and behavioral characteristics of the participants which increases the chances of them acquiring the bacteria and developing the infection.

Table 5: Associated risk factors of Helicobacter pylori infection.

Variable	Option	Frequency	Percentage
Where do you frequently eat?	At home	268	78.8
	In restaurants	39	11.5
	By the roadside	29	8.5
	Others	4	1.2
	Total	**340**	**100.0**
Family size	1-5	158	46.5
	6-10	83	24.4
	11 and above	99	29.1
	Total	**340**	**100.0**
Do you have good toilets in or around your home?	Yes	293	86.2
	No	47	13.8
	Total	**340**	**100.0**
Do you wash your hands properly with soap after using the toilet?	Always	136	40.0
	Never	5	1.5
	Sometimes	199	58.5
	Total	**340**	**100.0**
What is your main source of drinking water?	Tap	126	37.1
	Bottled	113	33.2
	Bore hole	68	20.0
	Stream	21	6.2
	Wells	12	3.5
	Total	**340**	**100.0**
Did you complete your prescribed medication for previous *Helicobacter pylori* infection?	Yes	37	51.4
	No	35	48.6
	Total	**72**	**100.0**
Where you re-tested after completion of treatment?	Yes	4	5.6
	No	63	87.5
	I have forgotten	5	6.9
	Total	**72**	**100.0**
Do you always take anti-inflammatory drugs?	Yes, always	28	8.2
	Yes, sometimes	127	37.4
	No	185	54.4
	Total	**340**	**100.0**
Have you ever had an operation in your stomach, bowel or appendix?	Yes	56	16.5

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	No	284	83.5
	Total	**340**	**100.0**
Do you have any close relative in your household with stomach cancer or serious stomach disorders?	Yes	70	20.6
	No	270	79.4
	Total	**340**	**100.0**
Do you fast or go long periods without eating?	Yes	77	22.6
	No	263	77.4
	Total	**340**	**100.0**
Do you smoke?	Yes, always	22	6.5
	Yes, sometimes	23	6.8
	No	295	86.8
	Total	**340**	**100.0**
Do you consume alcohol?	Yes, always	37	10.9
	Yes, sometimes	34	10.0
	No	269	79.1
	Total	**340**	**100.0**

4.4. Relationship between prevalence of *Helicobacter pylori* and socio-demographic factors

There is a probability that a socio-demographic factor has a relationship with the prevalence of the infection if the p-value was less than or equal to 0.05. Based on the relationship between the socio-demographic factors and the prevalence of *Helicobacter pylori* infection in asymptomatic individuals, only the age group was significant (p=0.023) with the highest prevalence between the age group of 0-20 years similar to a study carried out by Rothenbacher et al [25]. This may be due to the fact that members of this age group are very active as compared to those of the other age groups exposing them to the bacteria. Assessing the relationship between the socio-demographic factors and prevalence of the infection in symptomatic individuals, age group was significant (p<0.001) with the highest prevalence between the age group of 0-20 years. The Marital status was also significant (p=0.003) with majority being single. Educational level was significant (p<0.001) with most participants having only primary education. This association is an indication that infection with this bacterium occurs as a result of ignorance and poor knowledge. Occupation was also significant (p<0.001) with most cases being students similar to the study carried out by Nathan et al. This may be due to the fact that some students might not have the resources to cater for their health needs.

Table 6: Association between socio-demographic characteristics and prevalence of H. pylori infection

Variable	**Option**	**Asymptomatic**		**Symptomatic**		**Chi-square**		**p-value**	
		Positive	**Negative**	**Positive**	**Negative**	**Asymptomatic**	**Symptomatic**	**Asymptomatic**	**Symptomatic**
Sex	Male	10	56	18	22	**0.346**	**1.286**	p=**0.354**	**p=0.171**
	Female	23	101	61	49				
	Total	**33**	**157**	**79**	**71**				
Age group (years)	0-20	18	116	18	42	**7.578**	**21.223**	p=**0.023**	p<**0.001**
		6	25	20	7				
	21-30	9	16	41	22				
	31 and above								
	Total	**33**	**157**	**79**	**71**				
Marital status	Cohabiting	0	3	1	2	**3.820**	**13.626**	p=**0.282**	p=**0.003**
	Divorced	0	4	6	0				
	Married	10	28	35	18				
	Single	23	122	37	51				
	Total	**33**	**157**	**79**	**71**				
Religion	Christians	24	119	48	36	**0.330**	**5.047**	p=**0.848**	p=**0.080**
	Muslims	4	14	8	3				

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	Others	5	24	23	32				
	Total	**33**	**157**	**79**	**71**				
Occupation	Employed	14	32	26	16	**8.236**	**20.278**	p=**0.083**	p<**0.001**
	Unemployed	0	2	1	2				
	Student	15	104	34	49				
	Retired	0	2	17	1				
	Children < 2 years	4	17	1	3				
	Total	**33**	**157**	**79**	**71**				
	None	4	18	3	15	**6.242**	**32.109**	p=**0.100**	p<**0.001**
	Primary	9	74	18	32				
	Secondary	5	25	26	3				
Level of education	Higher	15	40	32	21				
	Total	**33**	**157**	**79**	**71**				

4.5. Relationship between prevalence of *Helicobacter pylori* infection and its associated risk factors

The relationship between the prevalence of *H. pylori* infection and its associated risk factors in asymptomatic patients was assessed similar to the studies carried out by Sun et al [28], Escobar et al [23] and Rothenbacher et al [25]. The frequent eating spots of participants was statistically significant (p=0.031) with majority of participants eating at home. This may be due to improper handling and storage of food exposing them to the bacteria. Completion of medication prescribed for previous infection with the bacteria was significant (p=0.001) with majority of them not completing their treatments. This may be due to the fact that some patients seize to complete their medication when their symptoms like gastrointestinal pain subsides causing infection to resurface over time. The intake of NSAIDs was significant (p=0.004) and prevalent in individuals who take them. Recent studies have suggested that elimination of *H. pylori* before treatment with NSAIDs decreases ulcer occurrence but no direct relationship for NSAIDs causing the bacteria to be prevalent [21]. This NSAIDs can further damage the epithelium making the environment favorable for the bacteria and due to the fact that the NDAIDs help to relieve pain and inflammation, some patients have no symptoms. Previous operation in the stomach, bowel or appendix was significant (p=0.008). This is due to the fact that the bacteria can be transmitted through medical equipment such as endoscopes and blades during such procedures [43]. Fasting for long periods was statistically significant (p=0.001) as well the smoking and alcohol consumption habits of participants (p<0.001). Smoking and alcohol consumption have been associated risk factorsin multiple studies with the onset of *H. pylori* infection, its increased persistence, and the efficacy of its eradication [32]. They also increase acidity in the stomach which may cause a dose-dependently negative association between *H. pylori* and smoking and alcohol consumption. The relationship between the prevalence of *H. pylori* infection and its associated factors in symptomatic patients was assessed similar to the studies carried out by Iwanczak et al [42] and Kouitcheu et al [40]. The frequent eating spots of participants was significant (p=0.021), Poor hand hygiene after using the toilet was significant (p=0.040) as well as source of drinking water (p<0.001). This means that strict adherence to preventive measures greatly reduce the prevalence of this infection. The participants who drank tap water were the most prevalent. This could be due to the fact that the water source may not be purified properly and exposed to this bacterium. Previous operation in the stomach, bowel or appendix was significant (p=0.014), fasting for long periods was significant (p=0.019) and alcohol consumption was also significant (p<0.001). Having a close relative in your household with stomach cancer or serious stomach disorders was significant (p<0.001). This can be due to person-to-person transmission of the bacteria [47].

Table 7: Relationship between prevalence of Helicobacter pylori infection and its associated risk factors

Variable	Option	Asymptomatic		Symptomatic		Chi-square		p-value	
		Positive	**Negative**	**Positive**	**Negative**	**Asymptomatic**	**Symptomatic**	**Asymptomatic**	**Symptomatic**
What is your frequent eating spot?	At home	28	127	56	57	**6.920**	**9.721**	p=**0.031**	p=**0.021**
	In restaurants	5	10	18	6				
	By the roadside	0	20	5	4				
	Others	0	0	0	4				
	Total	**33**	**157**	**79**	**71**				
What is your Household size?	1-5	15	72	39	32	**0.002**	**0.283**	p=**0.999**	p=**0.868**
	6-10	10	47	13	13				
	11 and above	8	38	27	26				
	Total	**33**	**157**	**79**	**71**				
Do you have good toilets in	Yes	28	144	67	54	**1.501**	**1.837**	p=**0.220**	p=**0.175**

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or around your home?	No	5	13	12	17				
		33	**157**	**79**	**71**				
	Total								
Do you wash your hands properly with soap after using the toilet?	Always	9	70	33	24	**3.365**	**6.422**	p=**0.067**	p=**0.040**
	Never	0	0	5	0				
	Sometimes	24	87	41	47				
		33	**157**	**79**	**71**				
	Total								
What is your main source of drinking water?	Bottled	13	62	24	27	**4.690**	**24.389**	p=**0.321**	p<**0.001**
	Tap water	10	53	31	19				
	Bore hole	10	29	11	18				
	Stream	0	8	13	0				
	Wells	0	5	0	7				
	Total	**33**	**157**	**79**	**71**				
Did you complete your treatment prescribed for previous Helicobacter pylori infection?	Yes	18	11	4	4	**11.036**	**0.000**	p=**0.001**	p=**1.000**
	No	2	15	9	9				
	Total	**20**	**26**	**13**	**13**				
Where you re-tested after completion of treatment?	Yes	2	1	1	0	**4.737**	**1.040**	p=**0.094**	p=**0.308**
	No	18	20	12	13				
	I have forgotten	0	5	0	0				
	Total	**20**	**26**	**13**	**13**				
Do you always take anti-inflammatory drugs?	Yes, always	0	10	10	8	**11.065**	**0.126**	p=**0.004**	p=**0.939**
	Yes, sometimes	20	49	31	27				
	No	13	98	38	36				
	Total	**33**	**157**	**79**	**71**				
Have you ever had an	Yes	10	19	20	7	**6.985**	**6.053**	p=**0.008**	p=**0.014**
	No	23	138	59	64				

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operation in your stomach, bowel or appendix?	**Total**	**33**	**157**	**79**	**71**				
Do you have any close relative in your household with stomach cancer or serious stomach disorders?	Yes	5	19	35	11	**0.230**	**14.598**	p=**0.632**	p<**0.001**
	No	28	138	44	60				
	Total	**33**	**157**	**79**	**71**				
Do you fast or go long periods without eating?	Yes	16	4	37	20	**61.096**	**5.530**	p=**0.001**	p=**0.019**
	No	17	153	42	51				
	Total	**33**	**157**	**79**	**71**				
Do you smoke?	Yes, always	0	5	9	8	**25.251**	**5.281**	p<**0.001**	p=**0.071**
	Yes, sometimes	5	0	14	4				
	No	28	152	56	59				
	Total	**33**	**157**	**79**	**71**				
Do you consume alcohol?	Yes, always	5	15	9	8	**29.790**	**19.952**	p<**0.001**	p**<0.001**
	Yes, sometimes	10	5	19	0				
	No	18	137	51	63				
	Total	**33**	**157**	**79**	**71**				

Conclusion

The results of this study revealed that the overall prevalence of *Helicobacter pylori* infection in the participants was 32.9% (112/340). The prevalence in the asymptomatic population was 17.36% (33/190) and the prevalence in the symptomatic population was 52.66% (79/150). The associated risk factors of *Helicobacter pylori* infection in asymptomatic individuals were their age group, frequent eating spots, incomplete medications from previous infection, frequent intake of NSAIDs, previous operation in the stomach, fasting for long periods, smoking and alcohol consumption. The associated risk factors of *Helicobacter pylori* infection in symptomatic individuals were their age group, occupation, educational level, marital status, frequent eating spot, hand hygiene after using the toilet, source of drinking water, intake of NSAIDs, previous operation in the stomach, having a close relative with stomach cancer or serious gastrointestinal disorders, fasting for long periods and alcohol consumption.

5.1. Limitations

The study was carried out for a short period which made it difficult to attain the desired sample size. Maybe the results could have been different if the required sample size of 384 was attained. Some participants refused to sign the concerned or assent forms hence could not participate in the study. Some participants failed to provide stool samples for analysis.

5.2. Recommendations

To the ministry of Public Health

Sensitization campaigns on *Helicobacter pylori* should be put in place to educate the general population about the risks of having this bacterium and ways to prevent infection so as to lower its spread and mortality rate.

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To the Hospital

Patients should be educated on the various associated risk factors of this infection and proper follow up made to ensure that the patients complete their medications like the use of rendezvous forms.

To the population

Parents and guardians should ensure that good drinking water is available at home for consumption, practice good hand hygiene by using soap and water and ensure that food is properly handled.

Always come for medical checkups when symptoms arise to prevent infection of *Helicobacter pylori* bacterium progressing to complications like cancers or tumors.

References

1. Bittencourt BB, Antonio FFS, Silva AS, ***ET AL***. Pathogenesis and clinical management of *Helicobacter pylori* gastric infection. World Journal of Gastroenterology. 2019; 25(37): 5578-5580.
2. Yang H, Yang WJ and Hu B. Gastric epithelial histology and precancerous conditions. World Journal of Gastrointestinal Oncology. 2022; 14(2): 396-401.
3. Hoffmann W. Regeneration of the gastric mucosa and its glands from stem cells. Curr Med Chem. 2008; 15: 3133-3144.
4. Suarez-Carmona M, Lesage J, Cataldo D, ***ET AL***. EMT and inflammation: inseparable actors of cancer progression. Mol Oncol. 2017; 11: 805-823.
5. Ifeanyichukwu KO, Herrienta OO, Nnana II, ***ET AL***. Seroprevalence of *Helicobacter pylori* infection and Risk Factors among Asymptomatic Subjects in Delta State, Nigeria. Advances in Microbiology. 2017; 7: 641-645.
6. Khamisi MS. Prevalence of *Helicobacter pylori* infection among patients with peptic ulcers and the associated Risk Factors in Mbagathi Level V Hospital, Nairobi ounty, Kenya. 2019: 1-57.
7. Nathan EA, Seraphine NE, Lucy MN, ***ET AL***. *Helicobacter pylori* in patients with gastritis in West Cameroon: prevalence and risk factors for infection. BMC Res Notes. 2018; 11: 1-6.
8. Tursi AI, Cammarota G, Papa A, ***ET AL***. The Modes of Transmission of *Helicobacter pylori* infection. Recenti Progressi in Medicina. 1997; 88: 232-336.
9. Chonjnacki C, Poplawski T, Blonska A, ***ET AL***. Expression of tryptophan hydroxylase in gastric mucosa in symptomatic and asymptomatic *Helicobacter pylori* infection. Arch Med Sci. 2019; 15(2): 416-423.
10. Ricci A, Holton J and Vaira D. Diagnosis of *H. pylori*, invasive and non-invasive tests. Best pract Res Clin Gastroenterol. 2007; 21:299-313.
11. Iwanczak F and Iwanczak B. Treatment of *Helicobacter pylori* Infection in the Aspect of Increasing Antibiotic Resistance. Adv Clin Exp Med. 2012; 5: 671-680.
12. Dunn BE, Cohen H, Blaser MJ. *Helicobacter pylori*. Clin Microbiol. 1997; 10(4): 720-741.
13. Rothenbacher D, Bode G, Peschke F, ***ET AL***. Active infection with *Helicobacter pylori* in asymptomatic population of middle aged to elderly people. Epidemiol Infect. 1998; 120: 297-303.
14. Ramzan I, Wong BK and Corcoran GB. Pain sensitivity in dietary-induced obese rats. Physiol Behav. 1993; 54: 433-435.
15. Sjomina OPJ, Niv Y and Leja M. Epidemilogy of *Helicobacter pylori* infection. Helicobacter. 2018; 23: 1-30.
16. Michalkiewicz J, Helmin-Basa A, Grzywa R, ***ET AL***. Innate immunity components and cytokines in gastric mucosa in children with *Helicobacter pylori* infection. Mediators inflamm. 2015; 2015: 2-20.
17. Hansen MB. Neorohumoral control of gastrointestinal motility. Physiol Res. 2003; 52: 1-30.
18. Camilleri M. Serotogenic modulation of visceral sensation: lower. Gut. 2002; 51: 81-6.
19. Gershon MD, Tack J. The serotonin signaling system: from basic understanding to drug development for functional GI disorders. Gastroenterology. 2007; 132: 397-414.
20. Gut P, Czarnywojtek A, Fischbach J, ***ET AL***. Chromagranin A-unspecific neuroendocrine marker: Clinical utility and potential diagnostic pitfulls. Arch Med Sci. 2019; 12: 1-9.
21. Shi RXS, Zhang H, Ding Y, ***ET AL***. Prevalence and risk factors for *H. pylori* infection in Chinese populations. Helicobacter; 13(2): 157-165.
22. Mitchell A, Silva TM, Barrett LJ, ***ET AL***. Age-specific *H. pylori* seropositivity rates of children in an impoverished urban area of northeast Brazil. Journal of clinical microbiology. 2003; 41(3): 1326-1328.
23. Escobar ML and Kawakami E. Evidence of mother-child transmission of *H. pylori* infection. Arquivos de gastroenterologia. 2004; 41(4): 239-244.
24. Ndip RN, Malange TAE, Ojongokpoko JEA, ***ET AL***. *Helicobacter pylori* isolates recovered from gastric biopsies of patients with gastro-duodenal pathologies in Cameroon: current status of antibiogram. Trop Med Int Heath. 2008; 13: 848-54.
25. Ghazzawi IM and Obidat NA. The role of *Helicobacter pylori* infection in the pathogenesis of chronic urticaria. Pakistan Journal of Medical Sciences. 2004; 20: 101-104.26. Iwanczak F and Iwanczak B. Treatment of *Helicobacter pylori* Infection in the Aspect of Increasing Antibiotic Resistance. Adv Clin Exp Med. 2012; 5: 671-680.
26. Ozbek A, Ozbek E, Dursun H, ***ET AL***. Can *H. pylori* invade human gastric mucosa? An in vivo study using electron microscopy, immunohistochemical methods, and real-time polymerase chain reaction. Journal of clinical gastroenterology. 2010; 44(6): 416-422.
27. Veijola L, Myllyluoma E, Korpela R, ***ET AL***. Stool antigen test in the diagnosis of *H. pylori* infection. World journal of gastroenterology. 2017; 11:47-53.
28. Sun Y and Zhang J. *Helicobacter pylori* recrudescence and its influencing factors. J Cell Mol Med. 2019; 23: 7919-7925.
29. Stenstrom B, Helen MW, Fulurija A, ***ET AL***. *Helicobacter pylori* overcomes natural immunity in repeated infections. Clinical Case Reports. 2016; 4(11): 1026-1033.

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30. Zeng MXH, Mao JX, Li WD, ***ET AL***. Efficacy, safety, and immunogenicity of an oral recombinant *Helicobacter pylori* vaccine in children in China: a randomized, double-blind, placebo-controlled, phase 3 trial. Lancet. 2015; 386: 1424-1425.
31. Kao CY, Sheu BS and Wu JJ. *Helicobacter pylori* infection: an overview of bacterial virulence factors and pathogenesis. Biomed J. 2016; 39(1): 14-23.
32. Mclean LP and Wong U. Diagnosis and management of *H. pylori*. Journal of clinical gastroenterology and treatment. 2016; 2: 113-117.
33. Silva CRGS, Machado NC, Carvalho MA, ***ET AL***. *Helicobacter pylori* infection is high in paediatric nonulcer dyspepsia but not associated with specific gastrointestinal symptoms. Acta Paediatr. 2016; 105: 228-31.
34. Aguilera IM, Esther SDO, Angel AE, ***ET AL***. *Helicobacter pylori* infection in children. BMJ Paediatrics Open. 2020; 4: 1-6.
35. *Helicobacter pylori* antibody test cassette. Package Insert.
36. Osaki T, Mabe K, Hanawa T, ***ET AL***. Urease positive bacteria in the stomach induce a false positive reaction in a urea breath test for diagnosis of *H. pylori* infection. Journal Med Microbiol. 2008; 57(7): 814-819.
37. Bermejo F, Takahiro U, Graham DY, ***ET AL***. Diagnosis of *H. pylori* using the rapid urease test. Clinical microbiology procedure handbook. 2018; 2:120-145.
38. Michalkiewicz J, Helmin-Basa A, Grzywa R, ***ET AL***. Innate immunity components and cytokines in gastric mucosa in children with *Helicobacter pylori* infection. Mediators inflamm. 2015; 2015: 2-20.
39. Abdulrahman SN, Gamal AA, Mawhoob NA, ***ET AL***. Comparison of stool antigen and blood antibody test methods for detection of *H. pylori* infection. International journal of current microbiology and applied sciences. 2014; 3(12): 118-127.
40. Kouitcheu LB, Ngamga ML and Hubert L. BMC infectious diseases. 2018; 18:278-289.
41. Mohammad K, Fereshteh S, Ajhdarkosh H, ***ET AL***. Comparison of five diagnostic methods for *H. pylori*. Iranian Journal of microbiology. 2013; 5(4):396-401.
42. Iwanczak F and Iwanczak B. Treatment of *Helicobacter pylori* Infection in the Aspect of Increasing Antibiotic Resistance. Adv Clin Exp Med. 2012; 5: 671-680.
43. Cheung TK and Wong BC. Treatment of *H. pylori* and prevention of gastric cancer. Journal Dig Dis. 2008; 9(1): 8-13.
44. Johannes G, Arnoud H.M and Kuipers E.J. Treatment of *H. pylori* and prevention of H. pylori. Clinical microbiology journal.2006; 19(3): 449-490.
45. Edith LA, Samje M, Sanda KA, ***ET AL***, Knowledge, Practice and Prevalence of Helicobacter pylori infection in the North West Region of Cameroon. Clinical Biotechnology and Microbiology. 2017; 4(1): 135-143.
46. Asante YA, Larbi DS, Kunateh IA, ***ET AL***. Prevalence of *helicobacter pylori* infection among children living in a rural setting in Sub-Saharan Africa. BMC Public Health. 2017; 17: 360.
47. Sjomina OPJ, Niv Y and Leja M. Epidemilogy of *Helicobacter pylori* infection. Helicobacter. 2018; 23: 1-30.

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