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TITLE

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I. Introduction And Literature Review

1.1 Introduction and literature review:

Gastritis is group of conditions with one thing in common inflammation of the stomach is most often the result of infection , this may lead to ulcer and cancer. The most common bacteria cause gastritis is *Helicobacter pylori* .

1.2 Helicobacter pylori :

Helicobacter pylori, a gram-negative bacterium found on the luminal surface of the gastric epithelium, was first isolated by Warren and Marshall in 1983.[(1)] It induces chronic inflammation of the underlying mucosa. The infection is usually contracted in the first few years of life and tends to persist indefinitely unless treated.[(2)]Its prevalence increases with older age and with lower socioeconomic status during childhood and thus varies markedly around the world.[(3)] The higher prevalence in older age groups is thought to reflect a cohort effect related to poorer living conditions of children in previous decades. At least 50% of the world's human population has *H. pylori* infection. [(2)] The organism can survive in the acidic environment of the stomach partly owing to its remarkably high urease activity; urease converts the urea present in gastric juice to alkaline ammonia and carbon dioxide. [(4)]

1.2.1 General characteristic of Helicobacter pylori :

Since the introduction of *Helicobacter pylori* to the medical community by Marshall and Warren almost two decades ago, *Helicobacter pylori* has been the focus of basic biochemical and clinical research and debate. Its relevance to human disease, specifically to peptic ulcer disease, gastritis, and gastric malignancy, is indisputable. Many questions, however, still remain concerning the optimal diagnostic and therapeutic regimens with which to approach the organism *Helicobacter pylori* is a gram negative, microaerophilic bacterium that can inhabit various areas of the stomach, particularly the antrum. It causes a chronic low-level inflammation of the stomach lining and is strongly linked to the development of duodenal and gastric ulcers and stomach cancer. Over 80% of individuals infected with the bacteria are asymptomatic. The bacterium was initially named *Campylobacter pyloridis*, then renamed *C. pylori* (pylori = genitive of pylorus) to correct a Latin grammar error. When 16S rRNA gene sequencing and other research showed in 1989 that the bacterium did not belong in the genus *Campylobacter*, it was placed in its own genus, *Helicobacter*. The genus derived from the ancient Greek "spiral" or "coil". The specific epithet pylōri means "of the pylorus" or pyloric valve (the circular opening leading from the stomach into the duodenum), from the Ancient Greek word πυλωρός, which means gatekeeper. More than 50% of the world's population harbor Helicobacter pylori in their upper gastrointestinal tract. Infection is more prevalent in developing countries, and incidence is decreasing in Western countries. [(5)]

1.2.2 Microbiology:

Helicobacter pylori is a helix-shaped (classified as a curved rod, not spirochaete) Gram-negative bacterium, about 3 micrometers long with a diameter of about 0.5 micrometers. It is microaerophilic; that is, it requires oxygen, but at lower concentration than is found in the atmosphere. It contains a hydrogenase which can be used to obtain energy by oxidizing molecular hydrogen (H2) that is produced by intestinal bacteria. It produces oxidase, catalase, and urease. It is capable of forming biofilms and can convert from spiral to a possibly viable but non cultural coccoid form, both likely to favor its survival and be factors in the epidemiology of the bacterium. The coccoid form can adhere to gastric epithelial cells in vitro.

Helicobacter pylori possess five majors outer membrane protein (OMP) families. The largest family includes known and putative adhesions. The other four families include porins, iron transporters, flagellum-associated proteins and proteins of unknown function. Like other typical Gram-negative bacteria, the outer membrane of Helicobacter pylori consistsof phospholipids and lipopolysaccharide (LPS). The O antigen of LPS may be fucosylated and mimic Lewis blood group antigens found on the gastric epithelium. The outer membrane also contains cholesterol glucosides, which are found in few other bacteria. *Helicobacter pylori* has 4-6 lophotrichous flagella; all gastric and enterohepatic *Helicobacter* species are highly motile due to flagella.

The characteristic sheathed flagellar filaments *of Helicobacter* are composed of two copolymerized flagellins, *FlaA* and *FlaB*. [6]

1.2.3 Genome:

Helicobacter pylori consist of a large diversity of strains, and the genomes of three have been completely sequenced. The genome of the strain "26695" consists of about 1.7 million base pairs, with some 1,550 genes. The two sequenced strains show large genetic differences, with up to 6% of the nucleotides differing. Study of the *Helicobacter pylori* genome is centered on attempts to understand pathogenesis, the ability of this organism to cause disease. Approximately 29% of the loci are in the "pathogenesis" category of the genome database. Both sequenced strains have an approximately 40 kb-long Cag pathogenicity island (a common gene sequence believed responsible for pathogenesis) that contains over 40 genes. This pathogenicity island is usually absent from Helicobacter pylori strains isolated from humans who are carriers of *Helicobacter pylori* but remain asymptomatic .

The *cagA g*ene codes for one of the major *Helicobacter pylori* virulence proteins. Bacterial strains that have the *cagA* gene are associated with an ability to cause ulcers. The *cagA* gene codes for a relatively long (1186 amino acid) protein. [7]

1.2.4 Epidemiology:

At least half the world's population is infected by the bacterium, making it the most widespread infection in the world. Actual infection rates vary from nation to nation, the people in under developed countries has much higher infection rates than the developed countries like North America, Australasia etc. where rates are estimated to be around 25% Infections are usually acquired in early childhood in all countries and is we found in a previous study that has been done in Brazil The [C^{13}] urea breath test (C^{13} -UBT) and *Helicobacter pylori* stool antigen test (HpSA) for the diagnosis of *H. pylori* infection in children were validated. The sensitivity, specificity, and positive and negative predictive values were 93.8, 99.1, 97.8, and 98.0%, respectively, for the C^{13}

-UBT and 96.9, 100, 100, and 98.0%, respectively, for HpSA. Both tests are appropriate for diagnosing *H. pylori* infection in children. The C^{13} urea breath test (C^{13}-UBT) and a new developed immunoassay for the detection of *Helicobacter pylori* antigens in stool, the H. pylori stool antigen test (HpSA) are noninvasive tests for *H. pylori* diagnosis. [8-9] However, the infection rate of children in developing nations are higher than in industrialized nations, probably due to poor sanitary conditions. In developed nations it is currently uncommon to find infected children, but the percentage of infected people increase with age, with about 50% infected for those over the age of 60 compared with around 10% between 18 and 30 years. The higher prevalence among the elderly reflects higher infection rates when they were children rather than infection at later ages. Prevalence appears to be higher in African-American and Hispanic populations, although this is likely related to socioeconomic rather than racial factors. The lower rate of infection in the developed countries is largely attributed to higher hygiene standards and widespread use of antibiotics. Despite high rates of infection in certain areas of the world, the overall frequency of *Helicobacter pylori* infection is declining. However, antibiotic resistance is appearing in *Helicobacter pylori*; there are already many metronidazole and clarithromycin resistant strains in most parts of the world [10] . *Helicobacter pylori* is contagious, although the exact route of transmission is not known. Person-to-person transmission by either the oral-oral or fecal-oral route is most likely. Consistent with these transmission routes, the bacteria have been isolated from feces, saliva and dental plaque of some infected people. Transmission occurs mainly within families in developed nations yet can also be acquired from the community in developing countries. *Helicobacter pylori* may also be transmitted orally by means of fecal matter through the ingestion of waste-tainted water, so a hygienic environment could help decrease the risk of *Helicobacter pylori* infection.

1.2.5 Pathologic findings:

Although extensive work has been performed to classify histopathologic changes seen with *Helicobacter pylori infection*, there is no consensus on classification; the Sydney system and the Houston Gastritis Workshop system have, however, been recognized as models. After colonization, there appears to be an intense neutrophilic infiltrate in the necks of the mucosal glands. Epithelial changes are common when there is irregularity of the surface architecture, and atrophy of the glands is typical of longstanding infection. Moreover, there is usually lymphocytic infiltration of the stroma and impaired mucus secretion. Finally, areas of patchy intestinal metaplasia may be seen, which are central to the development of neoplasia. [11]

1.2.6 Clinical manifestations:

1.2.6.1 Gastritis and gastric cancer:

Once infected with *Helicobacter pylori*, most persons remain asymptomatic. Some infected persons may even clear the infection, with seroreversion rates commonly reported to be in the range of 5% to 10%; it is not known if this seroreversion is spontaneous or results from elimination of the organism by antibiotic agents used to treat other conditions. However, the typical course of disease in infected patients begins with chronic superficial gastritis, eventually progressing to atrophic gastritis. This progression appears to be a key event in the cellular cascade that results in the development of gastric carcinoma. Existing data indicate a 90-fold increase in rates of gastric carcinoma in patients with severe, multifocal atrophic gastritis, compared with normal controls. The mechanism of tumorigenesis appears to involve DNA damage induced by different cytokines and free radicals released in the setting of chronic inflammation in susceptible persons. Although *Helicobacter pylori* is associated with the development of adenocarcinoma of the antrum and body of the stomach, it is also clearly linked with gastric mucosa–associated lymphoid tissue (MALT) lymphomas. *Helicobacter pylori* stimulates lymphocytic infiltration of the mucosal stroma; this infiltration may act as a focus for cellular alteration and proliferation, ultimately resulting in neoplastic transformation to lymphoma. It appears that *Helicobacter pylori* also produces proteins that stimulate growth of lymphocytes in the early stages of neoplasia. Most tellingly, it has been reported that regression of low-grade gastric MALT lymphoma can be achieved in 70% to 90% of patients with eradication of *Helicobacter pylori* infection. Recent work has shown endoscopic ultrasound examination to be invaluable in identifying the grade of MALT lymphoma and in predicting the efficacy of treating the Helicobacter pylori infection to obtain regression of the lymphoma Peptic ulcer disease, [(12)] The relationship between *Helicobacter pylori* infection and peptic ulcer disease has been studied exhaustively, and it is now accepted that the organism is the major cause, but not the only cause, of peptic ulcer disease worldwide. Eradicating the infection can alter the natural course of peptic ulcer disease by dramatically reducing its recurrence rate in treated patients, compared with untreated patients. This reduction occurs in patients with duodenal and gastric ulcers that have no history of nonsteroidal anti-inflammatory drug use.

The mechanism by which *Helicobacter pylori* induces peptic ulcer disease is incompletely understood but most likely involves a combination of genetic predisposition of the host, virulence factors of the organism (eg, *VacA* and *CagA* proteins), mechanical damage to the mucosa, and alterations of gastric and duodenal secretions.

1.2.6.2 Non-ulcer dyspepsia:

Non-ulcer dyspepsia comprises a constellation of varied symptoms, including dysmotility-like, ulcer-like, and reflux-like symptoms. Many possible causes have been suggested for non-ulcer dyspepsia, including lifestyle factors, stress, altered visceral sensation, increased serotonin sensitivity, alterations in gastric acid secretion and gastric emptying, and *Helicobacter pylori* infection. A recent study also highlighted the role played by psychosocial impairment (eg, depression, somatization, anxiety) in patients with non-ulcer dyspepsia. In a study linking *Helicobacter pylori* infection to non-ulcer dyspepsia, patients with the latter condition were twice as likely to be positive for the organism. However, despite such epidemiologic evidence, treatment studies have failed to consistently show that eradication of Helicobacter pylori results in improvement of non-ulcer dyspepsia symptoms. Consequently, eradication of the organism can not be considered the standard of care in all patients with non-ulcer dyspepsia, because *Helicobacter pylori* infection is only a single part of the multi-factorial etiology of the disease [(13)].as we found in this study that has been done in Iran despite many changes, no large studies comparing the different diagnostic tests *for Helicobacter pylori* have been performed in the past 10 years. In this time, monoclonal stool antigen immunoassays and in office C ^{13} -urea breath tests (UBTs) have appeared. The aim of this study was to evaluate the accuracy of invasive and noninvasive tests in a large series of dyspeptic patients. And the Rates of positive test results were similar (54%) for the rapid urease test, histopathological examination, and the stool test. By contrast, 75% of UBT results were positive, and the UBT was associated with a very low specificity (60%). For this reason, the delta cutoff value for the UBT was recalculated as 8.5%. Sensitivities and specificities with this new cutoff value were 95% and 100%, respectively, for the rapid urease test; 94% and 99%, respectively, for histopathological examination; 90% and 93%, respectively, for the stool test; and 90% and 90%, respectively, for the UBT. [(14-15)]

1.2.6.3 Gastroesophageal reflux disease:

Much attention has been focused on the possible relationship between infection with *Helicobacter pylori* and gastroesophageal reflux disease (GERD) in its various manifestations (eg, esophagitis, Barrett's esophagus). Some investigators have suggested a link between the presence of *Helicobacter pylori* and a decreased risk for developing esophagitis and Barrett's esophagus; although this inverse association is supported by many prevalence studies, others fail to show it. Studies have also indicated that certain strains of *Helicobacter pylori*,

notably the CagA- positive strain, may be protective against the development of Barrett's esophagus. Moreover, Labenz and colleagues have shown that the incidence of esophagitis may in fact, increase after eradication of the organism. Treatment of *Helicobacter pylori* infection can lead to exacerbation of GERD in many patients, prompting many gastroenterologists to defer endoscopic antral biopsies in patients with significant GERD and absent ulcer. Conversely, other studies using endoscopic findings, pH probe measurements, and histology to determine the presence *of Helicobacter pylori* did not find any association between GERD (in any of its manifestations) and infection with *Helicobacter pylori*. Clearly, more definitive studies are necessary to define the relationship, if any, between these 2 entities [(16)].

1.2.6.4 Other disease associations:

Investigators have further postulated a relationship between *Helicobacter pylori* infection and cardiovascular disease and iron-deficiency anemia. These associations, however, require much more study before a causal relationship is established. [(17)]

1.2.7 Diagnostic testing:

Currently, there are several popular methods for detecting the presence of *Helicobacter pylori* infection, each having its own advantages, disadvantages, and limitations. Basically, the tests available for diagnosis can be separated according to whether or not endoscopic biopsy is necessary. Histological evaluation, culture, polymerase chain reaction (PCR), and rapid urease tests are typically performed on tissue obtained at endoscopy. Alternatively, simple breath tests, serology, and stool assays are sometimes used, and trials investigating PCR amplification of saliva, feces, and dental plaque to detect the presence of *Helicobacter pylori* are ongoing.

1.2.7.1 Histology:

Histologic evaluation has traditionally been the gold standard method for diagnosing *Helicobacter pylori* infection. The disadvantage of this technique is the need for endoscopy to obtain tissue.

Limitations also arise at times because of an inadequate number of biopsy specimens obtained or failure to obtain specimens from different areas of the stomach. In some cases, different staining techniques may be necessary, which can involve longer processing times and higher costs.

However, histologic sampling does allow for definitive diagnosis of infection, as well as of the degree of inflammation or metaplasia and the presence/absence of MALT lymphoma or other gastric cancers in high-risk patients.

1.2.7.2 Culture:

Because *Helicobacter pylori* is difficult to grow on culture media, the role of culture in diagnosis of the infection is limited mostly to research and epidemiologic considerations. Although costly, time-consuming, and labor intensive, culture does have a role in antibiotic susceptibility studies and studies of growth factors and metabolism.

1.2.7.3 Polymerase chain reaction:

With the advent of PCR, many exciting possibilities emerged for diagnosing and classifying *Helicobacter pylori* infection. PCR allows identification of the organism in small samples with few bacteria present and entails no special requirements in processing and transport. Moreover, PCR can be performed rapidly and cost- effectively, and it can be used to identify different strains of bacteria for pathogenic and epidemiologic studies. As suggested earlier, PCR also is being evaluated for its utility in identifying *Helicobacter pylori* in samples of dental plaque, saliva, and other easily sampled tissues. The major limitation of PCR is that relatively few laboratories currently have the capability to run the assay. In addition, because PCR can detect segments of *Helicobacter pylori* DNA in the gastric mucosa of previously treated patients, false-positive results can occur, and errors in human interpretation of bands on electrophoretic gels can likewise lead to false-negative results.

1.2.7.4 Rapid urease testing:

Rapid urease testing takes advantage of the fact that *Helicobacter pylori* is a urease producing organism. Samples obtained on endoscopy are placed in urea-containing medium; if urease is present, the urea will be broken down to carbon dioxide and ammonia, with a resultant increase in the pH of the medium and a subsequent color change in the pH dependent indicator. This test has the advantages of being inexpensive, fast, and widely available. It is limited, however, by the possibility of false positive results; decreased urease activity, caused either by recent ingestion of antibiotic agents, bismuth compounds, proton pump inhibitors, or sucralfate or by bile reflux, can contribute to these false-positive results

1.2.7.5 Urea breath test:

A urea breath test similarly relies on the urease activity of *Helicobacter pylori* to detect the presence of active infection. In this test, a patient with suspected infection ingests either C^{14} - labeled or C^{13} - labeled urea; C^{13} - labeled urea has the advantage of being non-radioactive and thus safer (theoretically) for children and women of childbearing age. Urease, if present, splits the urea into ammonia and isotope-labeled carbon dioxide; the carbon dioxide is absorbed and eventually expired in the breath, where it is detected. Besides being excellent for documenting active infection, this test is also valuable for establishing absence of infection after treatment, an important consideration in patients with a history of complicated ulcer disease with bleeding or perforation. In addition, a urea breath test is relatively inexpensive (whichever isotope is used), is easy to perform, and does not require endoscopy. However, if the patient has recently ingested proton pump inhibitors, antibiotic agents, or bismuth compounds, a urea breath test can be of limited value. Therefore, at least 1 week should separate the discontinuing of antisecretory medications and testing for active infection, and 4 weeks should separate treatment of *Helicobacter pylori* infection and testing for eradication of the organism. Moreover, except for major medical centers or tertiary referral centers where results are usually available in fewer than 24 hours, a urea breath test may be further limited by a turnaround time of several days (or longer) required for transport of samples and analysis by specialized laboratories not present in many community settings. (18)

1.2.7.6 Serologic tests:

In response to *Helicobacter pylori* infection, the immune system typically mounts a response through production of immunoglobulins to organism-specific antigens. These antibodies can be detected in serum or whole-blood samples easily obtained in a physician's office. The presence of IgG antibodies to *Helicobacter pylori* can be detected by use of a biochemical assay, and many different ones are available. Serologic tests offer a fast, easy, and relatively inexpensive means of identifying patients who have been infected with the organism. However, this method is not a useful means of confirming eradication of *Helicobacter pylori*; several different samples and changes in titers of specified amounts over time would be needed. In addition, few patients become truly seronegative, even after eradication of the organism. In low-prevalence populations, serologic tests should be a second-line methodology because of low positive predictive value and a tendency toward false-positive results. Serologic tests may be useful in identifying certain strains of more virulent *Helicobacter pylori* by detecting antibodies to virulence factors associated with more severe disease and complicated ulcers, gastric cancer, and lymphoma. (19)

1.2.7.7 Stool antigen testing:

Stool antigen testing is a relatively new methodology that uses an enzyme immunoassay to detect the presence of *Helicobacter pylori* antigen in stool specimens. A cost effective and reliable means of diagnosing active infection and confirming cure, such testing has a sensitivity and specificity comparable to those of other noninvasive tests. Questions remain regarding possible cross reactivity with other *Helicobacter* species presents in the intestines, but definitive studies are lacking. (20)

1.2.8 Management:

1.2.8.1 General treatment principles:

Determining the optimum treatment of *Helicobacter pylori* infection is difficult, because the organism lives in an environment not easily accessible to many medications and because emerging bacterial resistance presents an added challenge. Moreover, many of the recommended regimens are difficult for patients to take, leading to problems with compliance; specifically, having to take a large number of pills at least twice daily and coping with unpleasant adverse effects do little to encourage patient cooperation. Despite these obstacles, current regimens can obtain cure rates in excess of 85% in most patient populations. (21)

1.2.8.2 Patient management in primary care:

The majority of patients infected with *Helicobacter pylori* present initially in primary care, suffering from dyspeptic symptoms with or without alarm symptoms. This is where many of them can and should be treated for the infection, even though, in the absence of endoscopy, the primary care physician may not have an accurate diagnosis of the underlying disease pathology. A further consideration is the increasing media, and hence patient, awareness of *Helicobacter pylori*, and its relationship to diseases such as gastric cancer. In this environment, primary care physicians need to have a clear understanding of the major role that they play in the management of the infection. The recommendations given here are particularly relevant to management in primary care, but many of them apply across clinical practice. Two strongly recommended indications which should be noted here as particularly relevant in primary care are patients who are first-degree relatives of gastric cancer patients and eradication therapy in response to patients' wishes after full consultation. As recommended in the original Maastricht Consensus Report, a `test and treat' approach should be offered to adult patients under

the age of 45 years (the age cut-off may vary locally according to the mean age of gastric cancer onset) presenting in primary care with persistent dyspepsia. Several studies have since been published which support this recommendation. [22]

1.2.8.2.1 Antibiotic agents:

Currently, antibiotic agents used to treat *Helicobacter pylori* infection are administered in combination, with no single agent ever used as monotherapy because of a lack of efficacy and the potential development of resistance. Metronidazole has activity independent of pH, but resistance to the drug is common in many parts of the world This problem with resistance is ameliorated somewhat, however, when the drug is used with clarithromycin. Metronidazole can have unpleasant adverse effects (e.g. nausea) and a disulfiram- like reaction to alcohol ingestion is possible, although exceedingly rare. Clarithromycin has lower rates of resistance (approximately 7%–11%) but is not acid stable, may cause dysgeusia and is more expensive than other antibiotic agents. Resistance to amoxicillin is rare, but this drug usually requires the co-administration of a proton pump inhibitor because its activity is pH- dependent. Finally, tetracycline has the advantage of low cost and low occurrence of resistance but can cause discoloration of the teeth in children and photosensitivity reactions. [23]

1.2.8.2.2 Adjunctive agents:

The most popular agents currently used in combination with antibiotic agents to eradicate *Helicobacter pylori* infection are the proton pump inhibitors i-e omeprazole being the most widely studied drug. Omeprazole acts not only by directly inhibiting bacterial microsomal enzymes but also by raising intra-gastric pH, thus facilitating the action of antibiotic agents, reducing gastric secretions, and increasing antibiotic concentrations in the stomach. Other adjunctive agents include histamine receptor antagonists and ranitidine bismuth citrate, which has anti-secretory properties in addition to the antibacterial action of bismuth (i.e., interruption of the bacterial cell wall). Ranitidine bismuth citrate is no longer available. [24]

1.2.8.2.3 Current regimens:

Presently, the most efficacious regimens include 2 antibiotic agents and at least 1 adjunctive agent for 14 days. In literature citation study carried out has claimed adequate cure rates with a 7-day course of quadruple therapy (2 antibiotics, 2 adjunctive agents), but other studies have not confirmed this finding. Most clinicians treat *Helicobacter pylori* infection with a triple drug or even quadruple-drug approach. The 1998 guidelines suggested the following 3 regimens to be optimal. [25]

1. Administration of a proton pump inhibitor, clarithromycin and either metronidazole or amoxicillin for 2 weeks.
2. Administration of ranitidine bismuth citrate (this guideline preceded the drug's withdrawal in the United States), clarithromycin and either metronidazole, amoxicillin, or tetracycline for 2 weeks.

3. A proton pump inhibitor, bismuth, metronidazole and tetracycline for 2 weeks. More recent recommendations outlined in a postgraduate course offered by the American Gastroenterology Association propose the use of newer proton pump inhibitors. For patients who fail initial triple-drug therapy, according to follow-up testing, subsequent therapy should involve using a different combination of available antibiotic agents, increasing the duration of treatment, or incorporating a course of quadruple therapy. Culture with sensitivity testing should be performed after 2 treatment failures . And as we found in a previous study has been done in Italy To evaluate the agreement between a mAb-based stool test (HP StAR) and the urea breath test (UBT) in monitoring (H pylori) infection after eradication therapy [26]. We found in the results by cross sectional study that Among 250 patients. 240 (96.0%) had concordant UBT and Hp StAR tests. The remaining 10 (4.0%) patients had discordant tests (positive Hp StAR and negative UBT) with the Hp StAR inaccurate in five cases (false positive) and UBT inaccurate in the other five cases (false negative). And the Overall accuracy for both tests was 98%. [27-28]

1.2.8.3 Herbal treatment of *Helicobacter pylori* infection:

Many hundreds of plants worldwide are used in traditional medicine as treatment for bacterial infections. Some of these have also been subjected to in vitro screening but the efficacy of such herbal medicines have seldom been rigorously tested in controlled clinical trials. Conventional drugs usually provide effective antibiotic therapy for bacterial infections but there is an increasing problem of antibiotic resistance and a continuing need for new solutions. Although natural products are not necessarily safer than synthetic antibiotics, some patients prefer to use herbal medicines. Thus, healthcare professionals should be aware of the available evidence for herbal antibiotics. This review was undertaken to assess critically those antibacterial herbal medicines that bean have subjected to controlled clinical trials. In a recent study, anti-*Helicobacter pylori* activity of 50 commonly used Unani (traditional) medicine plants from Pakistan that are extensively utilized for

the cure of gastrointestinal disorders to explore the natural source for pilot compounds against Helicobacter pylori. [29]

Curcumin is the substance that gives the spice turmeric its yellow color. Curry powder, which is used extensively in Indian cuisine, is largely made of turmeric and other spices. Curcumin contains many powerful antioxidants and anti-inflammatory compounds, which have been shown to support colon health, a healthy cardiovascular system, and most recently brain health. Dozens of studies have shown that it is a chemo-preventative, and more recently it has been shown to exert a strong antibacterial effect against *Helicobacter pylori*. Studies carried furnished results showing a significant in vitro effect of its extracts against *Helicobacter pylori*, leading researchers to conclude that curcumin could be considered a valuable support in the treatment of the infection.[30] In a recent study, researchers found that licorice extract produced a potent effect against strains of Helicobacter pylori that are resistant against clarithromycin, one of the antibiotics typically used in the three antibiotic treatment regimens. The authors concluded that this study provides hope that licorice extract can form the basis for an alternative therapeutic agent against *Helicobacter pylori.* Research study-based communication found that licorice extracts are also effective against *Helicobacter pylori* strains that are resistant to both amoxicillin and clarithromycin, making them viable as chemo preventive agents for peptic ulcer or gastric cancer in *Helicobacter pylori* infected individuals. [31]

1.3 Rational:

Helicobacter pylori has generated public health interest, it induces chronic inflammation of the underlying mucosa. The infection is usually contracted in the first few years of life and tends to persist indefinitely unless treated. At least 50% of the world's human population has H. pylori infection. About 40% of people in the UK have *H.pylori* in their stomach so it's very common to diagnosis it. However about 15% of people with the condition get ulcer either in the stomach (gastric ulcer) or in the duodenum (duodenal ulcer). [10] Although ulcer tend to cause indigestion, occasionally they become much more serious as they can bleed or even burst(perforate), which happen if the ulcer burrows deep enough into the stomach lining to make a hole. Because there are millions of people who have both *H.pylori* and sever indigestion, it can be tempting to draw the conclusion that one leads to another. The *H.pylori* can survive in the acidic environment of the stomach partly owing to its remarkably high urease activity; urease converts the urea present in gastric juice to alkaline ammonia and carbon dioxide. Several test and procedure are used to determine the *H.pylori* infection, divided into direct (invasive) and indirect (non-invasive). In my research I compromised between ICT test for antigen detection and urea breath test in diagnosis of *H.pylori* The urea breath test is more accuracy but expensive. The ICT has low cost so I want to approved that ICT test for antigen detection has the same accuracy in compare with urea breath test.

1.4 Objectives:

1.4.1 General objectives:

To compare between ICT test and urea breath test in diagnosis of *H. pylori* in Khartoum state.

1.4.2 Specific objectives:

1. To detect the presence H.*pylori antigen* by using ICT test.
2. To detect the presence *H.pylori* by using urea breath test .
3. To compare the accuracy and cost between ICT and urea breath test in the diagnosis of *H.pylori*.

II.Materials and Methods

2.1 study design:

This is Cross sectional study that was conducted to fifty patient.

2.2 study area :

The study was carried out in (Khartoum hospital -Modren medical center) Khartoum _ Sudan.

2.3 study population :

2.3.1 Inclusion criteria:

This study will include all patient with gastric pain.

2.3.2 Exclusion criteria:

This study will not include any patient without gastric pain and any patient with gastric pain but refuse to participate in my research.

2.4 Sample Size:
50 Patients.

2.5 Sample Type:
1. Stool sample.
2. A breath sample after take one C^{14} urea capsule with appropriate amount of drinking water.

2.6 Ethical consideration:
Permission to carry out this research will be obtained from research board of the faculty of medical laboratory science, national university. The patient will be informed for the purpose of the study before collection of the specimen, and verbal consent well be taken.

2.7 Data analysis method:
T test.

2.8 Time schedule:
July to December.

2.9 Facilities required:
2.9.1 ICT for antigen detection from stool sample:
2.9.1.1 Specimen collection:

Stool sample was collected in clean container. The samples can be store in the refrigerator (2-8°C) for 1-2 days prior to testing. And the sample was totally frozen and through to room temperature before testing. Homogenous stool sample as thoroughly as possible prior to preparation.
2.9.1.2 Specimen preparation:
1. we take out the cap of the collection.

(a) and used the stick to pick up sufficient sample quantity. Then introduced the stick once into 4 different parts of the stool sample.
(b) making sure that at each insertion only the stick screw is covered with the sample to take the appropriate amount of fecal sample (50 mg) and then added it to the collection tube. For liquid sample added approx. 125µl in the collection tube using micropipette.
2. we closed the tube with the diluent and stool sample. Shaked the tube in order to assure good sample dispersion.

2.9.1.3 Materials:
2.9.1.3.1 Materials provided:

_ CerTest *H.pylori*.

_Instruction for use.

_Stool collection tubes with diluent.

2.9.1.3.2 Materials required but not provided:

_Specimen collection container.

_Disposable gloves.

-Timer.

2.9.1.4 Test procedure:
Allowed tests, stool samples and controls to reach room temperature (15-30°C) prior to testing. Don't open pouches until the performance of the assay.
1. Proceed to shake the stool collection tube in order to assure good sample dispersion.
2. Removed the CerTest H. pylori cared test from its sealed bag just before using it.
3. Taked the stool collection tube, cut the end of the cap and dispensed 3 drops in the circular window marked

with the letter S. avoided adding solid particles with the liquid.
4. Read the result at 10 minutes. Do not read the test result later than 10 minutes.

If the test dose not run due to solid particles, stir the sample added in the sample window(S) with the stick. If it doesn't work, dispense a drop of diluent until seeing the liquid running through the reaction zone.

2.9.2 Urea breath test:
2.9.2.1 Preparation:

Check that the patient has:
1. Not received any antibiotics for 4 weeks prior to the test.
2. Not received sucralfate for 2 weeks prior to the test.
3. Not received loswc, zoton or drink (including no water) and no brushing teeth, no chewing gum, no smoking for 6 hours (recommended) prior to the test

2.9.2.2 Performing the test:
1. Swallowed one urea [C^{14}] capsule with water on an empty stomach or two hours after eating.
2. Sit calmly for 15 to 25 minutes.

3. Unpacked and take out the mouthpiece and collection card body.

4. Connected the mouthpiece to the front end of the collection card body.

5. Blow reposefully through the mouthpiece, as long as possible.

6. You can exchange breath during blowing. Do not inhale from the mouthpiece.

7. Blow continuously for 1 to 3 minutes until the indicator of the collection card turns from orange to yellow.
8. Discard the mouthpiece into the dustbin. Hand over the collection card body to the operator for analysis and wait for the test result.

III.Result

- Cross sectional study done in modern medical center, sample size was 50 patient tested for detection of *H.pylori* by using ICT and UBT.
- Out of 50 sample the ICT test revealed positive samples 48 (96%) and 2 (4%) negative samples. (Table No 3-1)
- Out of 50 sample the UBT test revealed positive samples 46 (92%) and 4 (8%) negative samples. (Table No 3-2)
- One sample statistics done for both tests revealed mean 1.04, 0.198 Std deviation and 0.028 Std.Error mean for ICT.
- One sample statistic done for both tests revealed mean 1.08, 0.274 Std deviation and 0.039 Std.Error mean for UBT.
- There is no significant difference between the two means. (Table No 3-3)
- One sample T test done for ICT test, P value was 0.000, t =37.151. UBT, P value 0.000, t=27.867. No much differences between t score value. (Table No 3-4)
- Compare means between ICT and UBT show no big differences in the result and the P.value is significant at the level of 0.05 or less. (Table No 3-5)

Table (3- 1): Frequency table for ICT test

ICT	Frequency	Percent
positive	48	96.0
negative	2	4.0
Total	50	100.0

Table (3- 2): Frequency table for UBT test

UBT	Frequency	Percent

positive	46	92.0
negative	4	8.0
Total	50	100.0

Table (3- 3): **One-Sample Statistic**

	N	Mean	Std. Deviation	Std. Error Mean
UBT	50	1.08	.274	.039
ICT	50	1.04	.198	.028

Table (3- 4): One-Sample Test

	t	df	Sig. (2-tailed)	Mean Difference
UBT	27.867	49	.000	1.080
ICT	37.151	49	.000	1.040

Table (3- 5): **Compare means UBT & ICT**

	N (Mean±SD)	p. value
ICT	50 (1.04±0.198)	.000
UBT	50 (1.08±0.274)	.000

P. value is significant at the level of 0.05 or less Independent T test was used.

IV. Discussion, conclusion and recommendation

4.1 discussion :

Helicobacter pylori has been considered to be the etiologic cause of gastritis, peptic ulcer disease and associated with development of gastric cancer.. In the developed countries, its prevalence is under 40% but in developing countries it is more than 80% .

H.pylori tests are established in clinical routine, but at present,.availability and costs should be considered in choosing the most suitable test. The UBT and the ICT (stool antigen) are currently considered to be the only reliable non-invasive tests for monitoring *H pylori* infection.

In this study that done by using cross sectional study for fifteen patient requested for tested by both ICT and UBT for the diagnosis of *H.pylori* , we found that when we use ICT show 96% was positive and 4% negative, and when we use UBT show 92% was positive and 8% negative according to the P.value show that there is no significant differences between both tests where the P.value at the level of 0.05 or less.

The results of our study is agreed with study done in Brazil in 2003 by Luciana de *et al*. Evaluation of C ^{13} Urea Breath Test and *Helicobacter pylori* Stool Antigen Test for Diagnosis of H. pylori Infection in Children from a Developing Country. Where their results show that The positive were 97.8% for the C ^{13} UBT and 100%, for HpSA. Both tests are appropriate for diagnosing H. pylori infection in children. [(8-9)]

Also, it agrees with another study done in Italy in 2005 by Francesco Perri *et al* for Comparison of antigen stool test (Hp StAR) with the 13C-urea breath test (UBT) in monitoring Helicobacter pylori eradication therapy and found that Among 250 patients (50±14 years), their result show that the positive were 96% for ICT test and 96% for UBT overall accuracy for both tests was 98%.at the end found that Both the UBT and the Hp StAR are equally accurate in monitoring H pylori infection. Nowadays, the choice of the “best” non-invasive H pylori test in the post-treatment setting should be done not only in terms of diagnostic accuracy but also in view of cost and local facilities. [(27-28)]

In another study that done by Xavier Calvet *et al in* 2009 for evaluate the accuracy of invasive and noninvasive tests in a large series of dyspeptic patients. The result show that Rates of positive test results were similar (54%) for the rapid urease test, histopathological examination, and the stool test. By contrast, 75% of UBT results were positive, and the UBT was associated with a very low specificity . The conclusion was found that the Histological examination and rapid urease testing showed excellent diagnostic reliability. The stool test seems to be a good, noninvasive alternative to endoscopy-based tests. By contrast, the infrared-based UBT evaluated in our study showed a lower than expected performance and this is disagree with our study. But show that the the Histological examination and rapid urease testing showed excellent diagnostic test for dyspeptic patients . [(14-15)]

4.2 Conclusion:

In this study the conclusion is that the immunochromatography test (ICT) for stool antigen and the urea breath test (UBT) show similar result and that mean they have the same accuracy in diagnosis, monitoring and management of *Helicobacter pylori*. But the ICT is low cost, simpler and more rapid to perform than the UBT.

4.3 Recommendations:

Based on the results and conclusions drawn out in this study the following recommendations are suggested for better understanding and accurate use of tests (ICT and UBT) in diagnosis and management of *H.pylpri* infection :

1. Both tests ICT and UBT has the same accuracy in diagnosis of *H.pylori* . But we recommended to use ICT test as alternative form UBT because the ICT test is simpler and more rapid to perform
2. ICT no need for specific staff training so we can use it in health center and peripheral center.
3. ICT test has no contraindication in use so we can use it in all type of patient. While the UBT has a contraindication to be use in pregnant woman and the patient should not eat or drink anything for at least 6 hours before performing the test.
4. ICT test has very low cost than UBT and nowadays the select of any test should be done not only in terms of diagnostic accuracy but also in view of cost, local facilities, and economic state of our country because our medical service is not very good.

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