iJRASET
International Journal For Research in Applied Science and Engineering Technology



INTERNATIONAL JOURNAL FOR RESEARCH

IN APPLIED SCIENCE & ENGINEERING TECHNOLOGY

Volume: 10 **Issue:** III **Month of publication:** March 2022

DOI: https://doi.org/10.22214/ijraset.2022.40649

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International Journal for Research in Applied Science & Engineering Technology (IJRASET)
ISSN: 2321-9653; IC Value: 45.98; SJ Impact Factor: 7.538
Volume 10 Issue III Mar 2022- Available at www.ijraset.com

A Review Paper on Helicobacter Pylori

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Abstract: Helicobacter pylori (H. pylori) infection is a primary cause of gastroduodenal ulcer disease, gastric cancer, and a variety of other gastric and extra gastric diseases. The medical management of H. pylori is a dynamic process that requires frequent reconsideration due to changing epidemiologic conditions (e.g., immigration), changing resistance patterns with therapeutic implications, and new understanding relating to the reasons for pathogen eradication.
Keywords: helicobacter pylori, pathogenic infection, gastric cancer.

I. INTRODUCTION

Helicobacter pylori (H. pylori) is a spiral-shaped gram-negative bacterium that affects up to half of the world's population, with a higher frequency in developing nations. Chronic or atrophic gastritis, peptic ulcer, gastric lymphoma, and gastric cancer are all caused by H. pylori. [1] Barry J. Marshall, a Nobel Laureate, and Robin Warren, an Australian researcher, discovered the bacteria Helicobacter Pylori in 1984 and decoded its function in gastritis and peptic ulcer illness. It was a long-held notion in medical teaching and practise at the time they announced their findings that stress and lifestyle factors were the primary causes of peptic ulcer disease. They changed their minds, and it was soon discovered that H. pylori is responsible for more than 90% of duodenal ulcers and up to 80% of stomach ulcers. [2] Infection with Helicobacter pylori (H. pylori) is very widespread all over the world. Infection is thought to be acquired during early childhood in the great majority of affected people. In transmission, the mother is crucial. The stomach is the primary reservoir, and bacteria are most likely passed from person to person by faecal-oral, oral-oral, or sexual pathways, as well as gastric-oral routes. Infection is frequently linked to poor sanitation, overcrowding, and inadequate water sources. Fortunately, some recent studies have shown that global prevalence rates are declining, especially among children. In underdeveloped nations, the prevalence is estimated to be about 70%, while in the United States and other industrialised countries, the prevalence is estimated to be 30%-40%. While the frequency of H. pylori infection is declining in northern and western Europe, the infection is still widespread in southern and eastern Europe and Asia. [3]

II. LITERATURE REVIEW

We wanted to focus on the pathogenicity of H. pylori in this research because it was previously thought that the gastrointestinal environment was sterile due to its high acidity. [4] Chronic active gastritis is the most common complication following H. pylori infection. The intragastric distribution and severity of this chronic inflammatory process are influenced by a number of parameters, including the invading strain's features, the host's genetics and immune response, food, and acid production levels. Recognizing the function of H. pylori in the aetiology of upper gastrointestinal pathology requires an understanding of these components. [5].

A. Pathogenicity

The bacterium uses a variety of methods to achieve successful colonisation in such hostile settings, including enhanced motility, strong adhesion to epithelial cells, and an enzymatic apparatus that allows the formation of a suitable milieu for infection perpetuation. Finally, virulence factors such cytotoxin associated antigen A (Cag A), vacuolating cytotoxin (Vac A), duodenal ulcer promoting gene A protein (Dup A), outer inflammatory protein (Oip A), and gamma-glutamyl transpeptidase have been identified (GGT). Furthermore, the host immune system plays an important role in the infection's progression, most likely through a Th1-polarized response to the pathogen. [6]

B. Colonization

Surface adhesins, which preferentially interact with mucin 5 (MUC5AC) and Lewis (Le) determinants, are responsible for Helicobacter pylori colonisation of the stomach mucosa. MUC5AC synthesis is increased in response to H. pylori infection, which could represent a potential mechanism for bacterial adhesion. [7] The role of the transferrin receptor (TFRC) in H. pylori attachment to stomach epithelial cells, which allows iron acquisition, was validated in one investigation. Overexpression of ferritin light chain (FTL) in the gastric mucosa of H pylori-infected individuals was revealed to be critical for epithelial cell differentiation into intestinal metaplasia in the same study. [8]

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Special mechanisms are required for effective colonisation in the unfavourable stomach environment. H. Pylori's motility is given by its sheathed flagella. Flagella must be heavily glycosylated with the unique nine-carbon sugar pseud aminic acid in order to contribute to efficient motility (Pse). Pseud aminic acid biosynthesis is a five-step process that necessitates the sequential activity of five enzymes (PseB, Pse C, Pse H, Pse G, and Pse I). However, only two of the required enzymes, PseB and Pse C, have crystal structures, so the crystal structure of the Pse H-acetyl-CoA complex is of particular interest. [9] Chemotaxis allows a bacterium to alter its swimming motion in response to extracellular chemical signals, which is critical for successful colonisation and infection establishment. At least 10 H. pylori genes are involved in chemotactic stimuli receipt, signal transduction, and processing. [10] T1pA, B, C, and D, as well as Che A kinase and numerous coupling proteins, have all been found in H. pylori. As different investigations have shown in recent years, these proteins are all critical for bacterial colonisation. [11] In the contact between bacteria and host, adhesion molecules and surface receptors of stomach cells are also significant. Bab A, Sab A, Alp A and Alp B, Oip A, Hop Q, and Hop Z are some of the key adhesin molecules with various roles. [12]

C. Immunological Aspects

H. pylori infection triggers a wide range of innate and adaptive immunological responses in the host. Various H. pylori antigens, such as lipoteichoic acid, lipoproteins, lipopolysaccharide, HSP-60, Nap A, DNA, and RNA, bind to gastric cell receptors, including TLR1, TLR2, TLR4, TLR5, TLR6, and TLR10 on epithelial cell membranes, and TLR9 in intracellular vesicles, after initial contact with the pathogen. The activation of NF-B and c-Jun N terminal kinases, as well as other signalling pathways, leads to the production of proinflammatory cytokines. In addition to pathogen-associated molecular patterns activating receptors, Cag A injection through T4SS causes the generation of cytokines, which is another NF-B-dependent mechanism. CD4+ and CD8+ T cells, which are part of adaptive immunity, are also enlisted. In terms of cytokine profiles in general, Studies have revealed a Th1-polarized response in H. pylori-positive patients, with low levels of IL-4 (a Th2 cytokine) and increased levels of gamma interferon, tumour necrosis factor, IL-1, IL-6, IL-7, IL-8, IL-10, and IL-18. H. pylori-specific serum IgM antibodies can be identified in patient serum four weeks after infection in terms of immunoglobulin synthesis.

Serum IgA and IgG immunoglobulins are directed against various bacterial antigens during chronic infection. In most H. pylori-positive people, this inflammation is asymptomatic, but it raises the risk of duodenal and gastric ulcer disease, as well as the development of stomach cancer. [13]

D. Diagnosis

Infection progresses in a variety of ways, all of which are influenced by host variables. Some H. pylori infection endoscopic evaluation strategies, such as magnifying endoscopy techniques and chromoendoscopy, are used to diagnose H. pylori infection. In addition, the diagnostic value of histology and the urea breath test in specific clinical situations and patient groups has recently been investigated. Recent research suggests that the number of biopsy fragments used in the fast urease test be increased. The gold standard technique for antibiotic susceptibility testing is bacterial culture from a stomach biopsy, which is recommended. Serology is utilised for initial screening, and the stool antigen test is employed in particular when the urea breath test is unavailable, although molecular approaches have gained popularity for detecting antibiotic resistance. [14]

E. Treatments

According to studies, there is no commonly recommended treatment for H. pylori infection. Treatment for an infectious condition, on the other hand, should be based on culture and susceptibility tests performed on biological samples (e.g., urine, sputum) acquired from each patient. [15] A standard triple therapy consisting of a PPI (Protein-Pump Inhibitor) and two antibiotics (clarithromycin and amoxicillin/metronidazole) is commonly used as the first-line regimen for the treatment of infection. However, due to increased resistance of H. pylori to clarithromycin and metronidazole, bismuth-containing therapy or 10-day sequential therapy has been proposed to replace standard triple therapy. [16] For patients who have failed to eradicate H. pylori with first-line medications, the second-line therapy is unknown. A levofloxacin-based triple therapy, on the other hand, is a well-accepted rescue treatment. Patients who require third-line therapy should be sent to a medical centre and treated according to the results of an antibiotic susceptibility test, according to most guidelines. [17] Alternative remedies, such as phytomedicines and probiotics, have been utilised to help eradicate the disease. According to several research, probiotics aid in the restoration of the intestinal microbiota disrupted by antibiotics, as well as a reduction in side effects and, as a result, increased treatment adherence, supporting successful therapy. [18] However, probiotics alone will not heal the illness; they must be used in conjunction with antibiotics. And because the role of probiotics is still debatable, research is still being conducted on it.

F. Vaccines

Efforts to create an efficient H. pylori vaccination began in the early 1990s. In animal models, studies have shown that preventive and therapeutic protection against H pylori infection can be accomplished. Third Military Medical University and Chongqing Kang Wei Biotechnology in China produced an oral recombinant H pylori vaccination employing urease B subunit fused with heat-labile enterotoxin B subunit. In phase 1 and phase 2 clinical trials, the vaccine was evaluated. Up to a year after immunisation, Zeng and co-workers (2015) found that oral administration of the H. pylori vaccine gave good protection against infection in children aged 6–15 years. [19] Recent research has focused on using bioinformatics to generate reverse vaccines, and five antigenic epitopes have been prioritised as prospective vaccine candidates: Bab A, sab A, Fec A, vac A, and omp16. [20] Nano vaccines are also being studied, and they appear to be a promising alternative for eliciting an effective immune response against H. pylori infection. [21] Epi Vax/H. pylori vaccine, Helicovaxor®, Sichuan University/Urease epitope vaccine, Southern Medical University/Lp220 vaccine, China Pharmaceutical University/Probiotic vaccine delivery, MCRI/Gastric Cancer Vaccine are some of the vaccines in development. [22]

G. Controversy

There has been a debate for many years about whether the H. pylori bacterium, despite its detrimental consequences, has any positive properties. H. pylori is harmful to a tiny fraction of people, but there is enough data to suggest it is also beneficial, according to a 2007 NIH symposium on the topic. Infection with H. pylori causes asymptomatic infection in about 85% of people, symptomatic peptic ulcer disease in 15% of people, and gastric cancer in less than 1% of people. These findings show that H. pylori may possibly play a role in preventing the onset of autoimmune illness. Furthermore, H. pylori deficiency has been associated to an increased risk of disorders such as multiple sclerosis and celiac disease. Inflammatory bowel disease (IBD) is more common in locations with lower rates of H. pylori colonisation, according to epidemiological studies. Some evidence suggests that H. pylori infection may protect against the development of IBD. Other benefits of H. pylori include suppression of tuberculosis-causing bacteria (Mycobacterium tuberculosis), protection from asthma, Crohn's disease, oesophageal reflux, diarrheal disorders, and oesophageal cancer. This debate has sparked debate over whether eradication of H. pylori is necessary to help restore the host's health, or whether alternative strategies should be developed to control the bacteria's virulence, avoiding the development of ulcers and adenocarcinomas while retaining the bacterium's beneficial effects. [23][24]

III. CONCLUSIONS

This paper focuses on providing an overview of the detrimental consequences of helicobacter pylori that have been developed or proposed, as well as the bacteria's benefits. As a result, we will discuss and examine the above Literature survey in order to learn more about the bacteria and how to prevent its spread. As a result, we have come to the end of this paper, which demonstrates both the harmful and beneficial aspects of H. pylori.

IV. ACKNOWLEDGMENT

First and foremost, I'd like to convey my heartfelt gratitude to all of my college instructors who have literally led and supervised me throughout my academic career. I owe a huge debt of gratitude to you for your ongoing inspiration and invaluable advice throughout the project.

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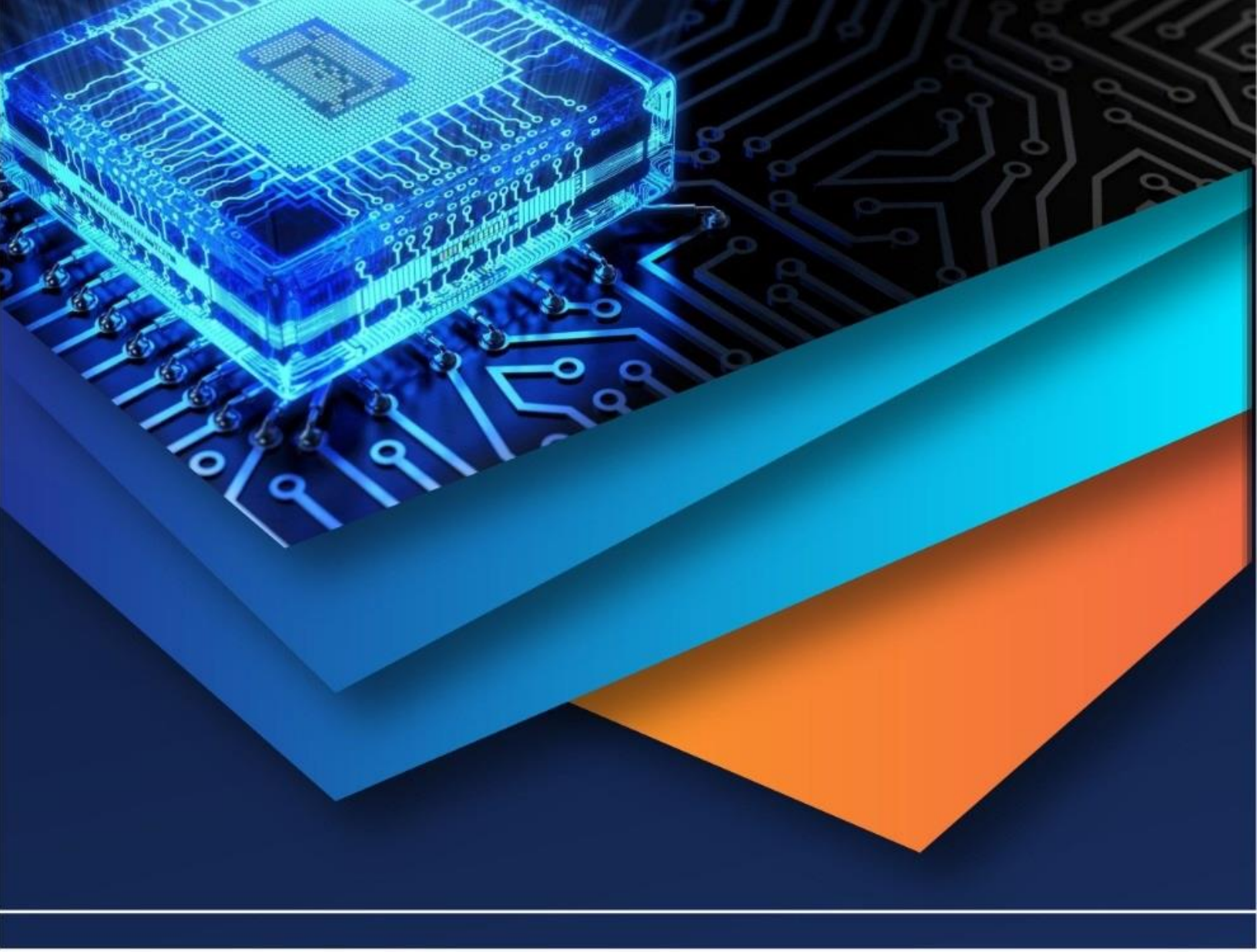
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