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Helicobacter pylori Infection: From Biological Characteristics and Gastrointestinal Pathogenesis to Diagnosis and Treatment— A Comprehensive Review

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ABSTRACT

Objective: *Understanding why H. pylori can occasionally be a deadly infection and figuring out how to get rid of it in those who are most at risk for serious illness are crucial in this second decade of rigorous study.* ***Method:*** *The biology of H. pylori colonization, stomach and intestinal microbiota, mucosal damage mechanisms, major gastrointestinal disorders, gastric carcinogenesis, diagnostic techniques, antibiotic resistance, and current eradication options are all covered in this narrative overiew.* ***Results:*** *Over the last decade, Helicobacter pylori has become one of the most common human infection, infecting practically everyone who has been exposed to filthy conditions from infancy. It is also found among high socioeconomic classes but less commonly. H. pylori is an important factor in the pathogenesis of duodenal and, to a lesser extent, gastric ulcers in addition to inducing chronic active gastritis. Furthermore, this bacteria is now considered as a risk factor for lymphoma and stomach cancer. However, the majority of infections don't have any clinical effects.* ***Novelty:*** *Improving long-term gastrointestinal health, therapy selection, and prevention all depend on an understanding of this integrated host-microbe connection.*

INTRODUCTION

Helicobacter. Since histological study of gastric biopsies revealed it as curved bacteria in the stomach and its isolation by [1], it has been a prominent subject of medicinal and biological research.

Pylori, a common gastrointestinal pathogen, infects roughly 50% of people. Several gastrointestinal illnesses are linked to this bacterium. H. pylori, a microaerophilic, Gram-negative spiral bacterium, is linked to chronic superficial gastritis, chronic active gastritis, peptic ulcer disease, and gastric cancer. It seems that H. pylori infection of the stomach occurs during early infancy. Acute and chronic active gastritis, which appears to last a lifetime, result from the bacteria colonising the gastric mucosa. Chronic active gastritis may go undiagnosed for years or decades or evolve to a peptic ulcer[2].

The gram-negative bacterium H. pylori has dimensions of 0.5 to 1.0 mm in diameter, 2.5 to 3.5 mm in length, and 1 to 2 mm in periodicity. It is characterised by an acurved, spiral, or gull-wing shape. One to six polar-sheathed flagellae protrude from one of its rounded ends, and its surfaces are smooth. It was initially referred to as "pyloric Campylobacter," followed by C. pyloridis, C. pylori, and ultimately H. pylori, as a result of its morphological resemblance to C. jejuni [3], Only the non-acid-secreting mucosa of the stomach is colonised by this organism, and it is not present in areas with a high concentration of parietal cells. Consequently, it is possible to witness it in the gastric antrum. The contamination of potable water may be a contributing factor in certain

developing countries, despite the fact that the route of transmission for this infection is unknown [4], In the United States and other locations, direct touch or intake of food or drink contaminated with saliva, gastric contents, or feces [5], may be significant contributors. The recent discovery that H. pylori can be isolated from cats indicates that transmission from pets to people (or humans to pets) is also possible[6].

To improve diagnosis and therapy, we must understand how Helicobacter pylori colonises the stomach and causes digestive diseases. Advances in medical reseach have led to the development of numerous diagnostic methods, including invasive procedures like endoscopy and biopsy as well as non – invasive tests like the stool antigen test and urea breath test. Early infection detection is crucial for stopping the progression of the illness and minimizing sequelae. Effective treatment of Helicobacter pylori infection is crucial for controllingdigestive disorders in addition to diagnosis. In order to eliminate the bacteria and encourage standard treatment combines proton pump inhibitors with antibiotics [7].

RESEARCH METHOD

A narrative literature review was conducted to collect and consolidate scientific date regarding Helicobacter pylori infection. The literature search was conducted using PubMed, Scopus, and Google Scholar. “Helicobacter pylori”, H.pylori infection “, gastric infection”, “pathogenesis”, “gastritis”, “peptic ulcer disease”, “cancer”, “diagnosis”, “treatment” and “antibiotic resistance “were the terms that comprised the search strategy. Relevant English – language items were taken into account. Based on their abstracts and titles, potentially relevant papers were selected during the initial screening stage. Studies on the biological characteristics, pathogenic mechanisms, virulence factors, associated disease diagnostic method, treatment, and antibiotic resistance of H. pylori were included. After that, the findings were analyzed and qualitatively summarized in line with the main topics of the review.

RESULTS AND DISCUSSION

Results

Research has repeatedly "Helicobacter pylori", which has a persistent capacity to colonize the gastric mucosa, is still a major bacterial pathogen in the human gastrointestinal system. According to research, this bacteriums motility, urease activity, and ability to tolerate immunological stresses make it highly to the gastrointestinal environment. These traits support the infection persistence and long – term stomach colonization [8].

The pathogenicity of Helicobacter pylori depends on a wide range of virulence characteristic that make it a powerful pathogen. For example, the enzyme urease secertes ammonia, which neutralizes stomach acid and causes direct damage to epithelial cells.Injecting tha CagA protein into stomach cells the bacteria modifies the cytoskeleton of the cells and causes acute inflammatory reaction by releasing mediators like IL-8. Simultaneously, the bacteria releases the toxin VacA, which enters cell, causes acidic

vacuolation, and finally pushes the cell into programd death. In order to prevent the germs from being washed away and to enable the very effective administration of these toxins, surface adhesins guaranty a strong adhesion to the stomach mucosa. The interaction of strain virulence, host immunological genetics, and enviromantal, variables ultimately determines the clinical course of the infection, which can range from mild inflammation to the formation of ulcers or tumors [9,10].

Overall, the data reviewed shows a high correlation between chronic gastritis and persistent "Helicobacter pylori" infection. Long-term infection has been found to be a major contributing factor to the development of peptic ulcer disease and is consistently associated with chronic gastritis. Moreover, prolonged antigenic stimulation has been linked to gastric mucosa-associated lymphoid tissue (MALT) lymphoma, whereas increasing damage to the stomach mucosa can result in atrophic gastritis[11].

According to the reviewed data, host vulnerability, environmental variables, and interactions with the gut microbiota all affect the likelihood of illness development in addition to bacterial virulence.

The association between "Helicobacter pylori" and the digestive system goes beyond the stomach, according to recent investigations covered in this paper. Through changes in stomach acidity, mucosal inflammation, immunological responses, and drug exposure, infection can affect the makeup of the gut microbiota. Similarly, gut microbial populations may alter as a result of eradication therapy, albeit the length of time and clinical importance of these alterations are still unknown[12].

Overall, the findings support the hypothesis that "H. pylori" infection involves a complicated host-microbe interaction in which the disease's progression is influenced by the gut microbiota, immune responses, stomach function, and bacterial characteristics. Therefore, the reviewed evidence highlights how important it is to understand "H. pylori" as a persistent gastrointestinal infection with consequences ranging from serious gastrointestinal issues to chronic gastritis[13].

Discussion

Biological and Structural Characteristics of Helicobacter pylori

As a microaerophilic bacterium, Helicobacter pylori needs less oxygen to develop as best it can. This feature enables the bacteria to endure under the particular conditions of the gastric mucosa. Lipopolysaccharides found in the bacterial cell wall are crucial for boosting the host's immune system. During an infection, these chemicals cause tissue damage and inflammation [14].

Additionally, Helicobacter pylori has outer membrane proteins that help it adhere to gastric epithelial cells. Helicobacter pylori also possesses outer membrane proteins that facilitate adhesion to gastric epithelial cells. Establishing infection and sustaining colonization in the stomach depend on these proteins, significant genetic variability in the bacteria adds to differences in the severity of the illness among those who are afflicted. Helicobacter pylori capacity to elude the human immune system is another amazing characteristic, the immunological reaction brought on by the illness. Chronic

inflammation brought on by this persistence may result in serious stomach disorders [15].

Factors Influencing H. pylori Infection Rates

The transmission of illness varies widely throughout populations and is influenced by environmental, social and demographic variables. Studies show that Helicobacter pylori infection is far higher common in underdeveloped countries than in industrialised ones. The primary reasons for this disparity are living conditions, hygiene, and access to medical treatment [16].

Helicobacter pylori usually spreads through fecal – oral or oral – oral pathways. Close contact with infected people and contaminated food and water are major factor in the bacteria's transmission. The risk of transmission to other family members is higher in homes where one person is afflicted [17].

Another significant aspect affecting infection rates is age. According to research, the majority of people get the infection as children, especially in areas where the bacteria is quite common. Once acquired, the infection may persist throughout life if not treated effectively [18].

Infection rates also depend on socioeconomic status. Poor housing, overcrowding, and lack of clean water increase Helicobacter pylori infection. However, greater cleanliness and healthcare systems have reduced infection rates in many developed countries[19].

Relationship Between H. pylori and the Gastrointestinal Tract

The stomach, small intestine, colon, immune system, and resident microbiota may all be seen as interconnected axs in the connection between H. pylori and the GI tract. Despite the fact that H. pylori mostly colonizes the stomach, alterations in gastric physiology, inflammatory mediators, immunological modulation, microbial metabolites, and antimicrobial exposure can all have an impact outside of the colonization site [20].

Persistent infection alters the local microbial habitat and causes chronic inflammation at the stomach level. The organisms that enter the duodenum and gut may be impacted by changes in acid secretion. Furthermore, downstream microbial populations may be impacted by inflammatory products and changes in the function of the epithelial barrier. On the other hand, the makeup of intestinal microbes may affect mucosal and systemic immune responses, changing the host's reaction to H. pylori [21].

This connection is crucial when considering eradication therapy, antibiotic combinations used to treat h.pylori can reduce microbial diversity and alter the abundance of specific gut taxa. Digestion, the production of short – chain fatty acids, vitamin metabolism, colonization resistance, and immunologyical development all depand on the gut microbiota. Current reviews show that many alterations are transient, while some research describe longer – lasting changes, emphasizing the need to strike a balance between effective eradication, antibiotic stewardship,and maintaining microbial equilibrium. Even though H. pylori dose not frequently predominate in the colon, gastric infections can have an indirect impact on the microbial makeup of the intestines.

Evidence of dysbiosis along the GI tract during eradication therapy and in connection with H. pylori infection has been reported in recent critical reviews [20,21].

This relationship might be explained by a number of processes. First, the quantity and kinds of bacteria that enter the small intestine are changed by H. pylori-related variations in stomach acid output. Second, immunological and epithelial activities are altered by persistent inflammation. Third, vulnerable gut bacteria are directly suppressed by antibiotic exposure during eradication. Lastly, by lowering stomach acidity, proton pump inhibitors, which are a common component of eradication regimens, might alter the GI microbial ecology [22].

Diseases Associated with Helicobacter pylori

Helicobacter pylori infection is associated with a variety of substantial gastrointestinal disorders.

Chronic gastritis is one of the most prevalent illnesses associated with this bacteria. The stomach lining is persistently inflamed with this illness, which may not cause any symptoms for a long time.Peptic ulcer disease is another serious illness linked to Helicobacter pylori. This syndrome arises when bacterial infection and increased gastric acid output cause ulcers to form in the stomach or duodenum [23].

In 1994, Helicobacter pylori was recognised as a human class I carcinogen by the International Agency for Research on Cancer (IARC) of the World Health Organization (WHO). It was discovered during this IARC meeting that human stomach cancer risk was significantly increased by H. pylori infection. Gastric adenocarcinoma (GAC) affects 1%–3% of those who have an H. pylori infection, which affects around half of the world's population. About 80% of GAC cases are linked to H. pylori infection, making it the main risk factor for GAC. Furthermore, mucosa-associated lymphoid tissue lymphoma, a rare form of stomach cancer, is linked to Helicobacter pylori infection [24].

Types, Classification, and Characteristics of Helicobacter Species

The majority of the microaerophilic organisms in the Helicobacter genus are positive for oxidase and catalase, and many are also urease positive, although not all. Helicobacter species have two main lineages: enterohepatic (nongastric) and gastric. Both families have strong organ specialization, therefore stomach helicobacters cannot colonise the intestine or liver, and vice versa [24].

Detection of Helicobacter spp. in the stomach.Gastric Helicobacter species are currently believed to be adapted to the adverse environment of the gastric mucosal surface and may be present in the stomach of all mammals. All known stomach Helicobacter species are urease positive and are extremely motile with flagella. Urease provides short-term survival in the extremely acid gastric lumen, while mobility helps gastric mucosa reach a neutral pH. This may explain why stomach mucosa colonisation requires both elements. On entry, gastric Helicobacter species travel chemotactically toward mucus. The spiral structure and flagellar motility allow the stomach Helicobacter species to live in the more pH-neutral thick mucus layer [25,26].

Felis Helicobacter: Initially identified from a cat's stomach, the spiral-shaped Helicobacter felis was subsequently discovered in dogs. The Helicobacter species Bizzozero described in 1893 and designated H. felis was likely it. A microscope can identify H. felis, a zoonotic Helicobacter species, by its helical structure and periplasmic fibers. High humidity is necessary for H. felis to grow well, if at all, on the typical growth media used to cultivate H. pylori. H. felis is extremely motile and grows like a grass on agar plates rather than forming colonies[27].

Since H. felis infection does not cause canine or feline gastritis, it may be part of cats' and dogs' stomach flora. Thus, H. felis' significance in cat and dog stomach disorders is uncertain.Like other gastric Helicobacter species, it has two flagellin genes (flaA and flaB) and a urease gene cluster [27].

Mustelae Helicobacter: Campylobacter pylori subsp mustelae, the ferret pathogen, was found shortly after H. pylori. Later, however, it was recognized as H. mustelae, since it was found to differ from H. pylori in several characteristics. H. mustelae is a relatively tiny rod with many polar and lateral sheathed flagella. It's interesting to note that, according to its 16S rRNA gene sequence, urease sequences, and fatty acid profile, H. mustelae is phylogenetically closer to the enterohepatic Helicobacter species[28].

The pathogen Helicobacter acinonychis: They have an impact on cheetahs and other massive animals. Previously known as Helicobacter acinonyx[29], H. acinonychis is susceptible to antibiotic therapy, as is the case with H. pylori infection, and employs comparable mechanisms for antimicrobial resistance. Twenty-four[30].

Heilmannii Helicobacter: It has been isolated from a range of domestic and wild animals, including cats, dogs, and nonhuman primates. It has also been found in a tiny portion of gastritis-affected people. Gastrospirillum hominis was the species' original name. Despite the lack of periplasmic fibres, the morphology of H. heilmannii is similar to that of H. felis [27], Human H. heilmannii infections may cause dyspeptic symptoms, gastritis, and, in rare instances, ulcer disease. Nonetheless, the inflammation may be naturally temporary and is often less severe than that seen in H. pylori-positive people [31].

Methods for Detecting H. pylori

Noninvasive approaches use bacterial enzymes, antigens, antibodies, or DNA sequences, while invasive procedures use gastrointestinal endoscope tissues. Both conventional and sophisticated detection methods have been used to diagnose H. pylori. Method selection depends on numerous aspects. In addition to patient age, these criteria include diagnostic test availability, endoscopic requirement, cost, accessibility, and technique pros and cons [32]. Young individuals with undiagnosed dyspepsia are treated with "test-and-treat" noninvasive testing instead of Oesophago-Gastro-Duodenoscopy. This method saves money, time, and stress. For adult patients 40 years or older with dyspepsia but no concern signs, such as persistent vomiting, weight loss, dysphagia, a palpable abdominal mass, jaundice, gastrointestinal bleeding, and no first-degree relative with gastric cancer, the noninvasive "test-and-treat" strategy is recommended. Test

accuracy is determined by sensitivity and specificity, which help healthcare providers make decisions and improve patient care [33].

Endoscopy

Traditional white-light endoscopy and narrowband imaging (NBI) are the two main methods for diagnosing Helicobacter pylori (H. pylori) infection [34], NBI clearly detects slight pathological changes in color. Dyspepsia symptoms, especially in locations with low H. pylori incidence, or weight loss, dysphagia, gastrointestinal haemorrhage, abdominal masses, or iron deficient anemia warrant gastrointestinal endoscopy. Advanced techniques such as blue laser imaging help provide accurate assessment of gastritis and early detection of gastric cancer.The optimal age for endoscopy is 35 years. Endoscopy coupled with stomach mucosal biopsy for fast urease tests and histological investigation is still the gold standard for diagnosis [35].

Histopathology

Histopathology is the gold standard for diagnosing stomach bacteria (H. pylori), although its accuracy depends on the amount of germs and biopsy location. However, it is not the perfect reference standard, as clinicians read pathological slides differently, and occasionally the results need to be reviewed together, despite its great efficiency [36], Histological assessment yields a diagnostic sensitivity between 70% and 95%, which can be greatly increased with the use of specific stains such regular yozin or immunohistochemistry[37].

Culture

In specialized labs, biopsy samples are cultured for Helicobacter pylori (H. pylori). It is mostly employed for scientific research or when other therapies have failed; it is not a common diagnostic technique.This process takes a long time and necessitates certain circumstances, such low temperatures and certain air conditions [38], in addition to the utilisation of certain culture medium that might not be easily accessible in diagnostic facilities. Culturing can be performed at a lower cost using biopsy samples compared to taking a gastric juice sample or a biopsy test, but its sensitivity decreases when the bacterial count is low.Bacterial colonies form in smaller numbers when using biopsy samples, and this number varies depending on the severity of the infection. It's important to note that the bacteria remain viable when secreted in gastric juice[39].

Rapid Urease Test

Numerous bacterial species produce the enzyme urease, which hydrolyses urea (carbamide) into carbon dioxide and ammonia [40]. When determining whether H. pylori is present in the stomach mucosa, the rapid urease test (RUT) is recommended as a first-line diagnostic procedure. After the biopsy is obtained, it is placed in a solution containing urea and a pH indicator. The indicator changes colour as a result of the urease's catalytic conversion of urea into ammonia and CO2, which raises pH. Biopsy samples should be taken from the body and gastric antrum for better outcomes. several organisms, including Staphylococcus aureus and Klebsiella pneumonia. The stomach sample used for RUT may be recovered for other H. pylori testing, including PCR, which is another important factor [40].

Urea Breath Test

The gold standard noninvasive method for diagnosing H. pylori is the UBT [15, 22]. The UBT analyses urea conversion using a radioactive carbon isotope. This test measures the activity of urease produced by H. pylori in the stomach following oral administration of isotopically labelled urea (13C or 14C), which is then hydrolysed into ammonia and carbon dioxide. 13C-labeled urea outperforms 14C because it is stable and non-radioactive[41].

Stool Antigen Test

Without stopping proton pump inhibitors, the fecal antigen test detects Helicobacter pylori bacteria in stool using enzyme immunoassay (EIA) and immunochromatography (ICA). The test is easy to perform and store in many hospitals and small laboratories without requiring specialized equipment or personnel, making it particularly useful in remote areas and developing countries.The fecal antigen test (SAT) using ELISA is an excellent alternative when the urea breath test (13C-UBT) is unavailable[41].

Molecular Methods

DNA/RNA-based molecular techniques are used to improve the diagnosis of both invasive and non-invasive Helicobacter pylori (H. pylori) infections.Polymerase chain reaction (PCR) tests are highly sensitive, enabling them to detect even very small numbers of live bacteria in clinical samples.Their importance lies in their ability to amplify target DNA to identify spherical H. pylori forms, which are difficult to culture and identify histologically[42].

Eradication Test

A Helicobacter pylori eradication test is essential to detect any relapse or treatment failure, especially in certain cases such as peptic ulcer disease, early-stage gastric cancer, MALT lymphoma, or those who continue to experience symptoms. Urea breath or stool tests are done four weeks following treatment to confirm eradication. Patients should avoid antibiotics, bismuth medications, and proton pump inhibitors for one month and two weeks before the test to avoid false-negative findings[43].

Treatment of H. pylori Infection

Helicobacter pylori (H. pylori) infection is treated with a combination of three medications: a medicine that decreases stomach acid (such as a proton pump inhibitor) and two separate antibiotics. The purpose of this combination is to attack the germs in two different methods, increasing the possibility of total eradication while preventing the development of antibiotic resistance[44].

The treatment plan typically lasts between one and two weeks (7 to 14 days). Results show that the longer the patient adheres to the treatment, up to 14 days, the higher the cure rate, increasing it by an additional 7% to 9%. Antibiotic resistance is the primary reason for treatment failure. Resistance to clarithromycin reduces the cure rate by half (50%), while resistance to metronidazole reduces it by 37%.Eliminating Helicobacter pylori depends on choosing the right combination of antibiotics, adhering to the full

course of treatment (14 days to ensure the best result), and the patient's discipline in taking the medications at the specified times[45].

CONCLUSION

Fundamental Finding : Overall, the reviewed research shows that Helicobacter pylori plays a significant role in gastrointestinal disorders, especially given its capacity to last in the stomachs acidic environment. Chronic inflammation of the stomach mucosa and even severe gastrointestinal issues may result from persistent colonization. **Implication :** The research that is now available also suggests that interaction with the stomach microbiota, therapeutic efficacy, and bacterial traits all affect how an infection turns out. Therefore, minimizing infection-related consequences still depends on early and accurate H. pylori testing followed by suitable eradication therapy. **Limitation :** The reviewed research indicates that treatment outcomes can be affected by therapeutic efficacy, bacterial traits, interaction with the stomach microbiota, and antibiotic resistance, which may contribute to treatment failure. **Future Research :** Furthermore, enhancing treatment results and lowering treatment failure need ongoing monitoring of antibiotic resistance.

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