

## GASTRIC HELICOBACTER PYLORI INFECTION / PEPTIC ULCERS AND PERIODONTAL DISEASE: A LITERARY OVERVIEW

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### ABSTRACT

H. Pylori is an important factor in pathogenesis of numerous diseases including metabolic, gastrointestinal and vascular disorders. The oral cavity has been proposed as a reservoir for *Helicobacter pylori* that could be responsible for refractoriness of gastric infection to triple therapy (antibiotics, antimicrobials, proton pump inhibitors). Analysis of data suggests that periodontal treatment in combination with systemic therapy could be a promising approach to increasing the therapy's efficacy and decreasing the risk of infection recurrence. Some authors have given conflicting evidences, to date there has been no clear cut evidence / study which directly correlates H. Pylori infection of periodontal pockets / plaque leading to peptic ulcer, duodenal ulcers and stomach cancers.

**Key words:** H. Pylori infection, peptic Ulcers, Periodontal disease.

### INTRODUCTION

This update will review the contribution on of selected published studies to advance our understanding towards the *Helicobacter Pylori* infection, its mode of transmission, the relationship of infection with periodontal disease and plaque.

H. Pylori is a common bacterium and approximately 50 percent of world's population has been estimated to be infected. Humans are the principle reservoir. The prevalence of H. Pylori infection varies widely by geographic area, age, race, ethnicity and socioeconomic status.[1]. H. Pylori, a micro organism perfectly at home in the highly acidic "soup" of your stomachs, whose PH is about 1.5 – 2.5 [2]. Most bacteria can't survive in stomach acid but H. Pylori can. It contains an enzyme, urease that makes it to make ammonia an alkaline compound that neutralize acid molecules. By secreting these protective substance bacteria avoid destruction and able burrow through the mucous lining to reach underlying cells. There they multiply and stay in your stomach indefinitely.<sup>1</sup>

Rates appear to be higher in developing than in developed countries with most of the infections occurring during childhood and they seen to be decreasing with improvements in hygiene practices. H. Pylori cause gastritis and has been associated with several serious diseases of the gastrointestinal tract including duodenal ulcer and gastric

cancers, since its discovery in 1982 by Warren and Marshall.<sup>1</sup>

The Australian discovery came as quite surprise to medical world, because previously ulcers had been attributed mainly to stress, spicy foods, excess stomach acid and un healthy life style choices, such as smoking and drinking coffee and alcohol. It turns out that these factors do not cause ulcers but they can surely aggravate them.<sup>2</sup>

The majority of recent studies have not found tobacco use or alcohol consumption to be risk factors for H. Pylori infection. Adequate nutritional status, especially frequent consumption of fruits and vegetables, vitamin c appears to protect against infections with H. Pylori. In contrast food prepared under less than ideal conditions or exposed to contaminated water or soil may increase risk. Overall inadequate sanitation practices, low social class and crowded or high – density living conditions seen to be related to a higher prevalence of H. Pylori infection. This finding suggests that poor hygiene and crowded conditions may facilitate transmission of infection among family members and is consistent with data on intrafamilial and institutional clustering of H. Pylori infection.<sup>1</sup>

Understanding the route of *H. Pylori* transmission is important, if public health measures to prevent its spread

are to be implemented, transmission of *H. Pylori* following endoscopy is the only proven mode. The most likely mode of transmission is from person to person, by either the oral – oral route (through vomits or possibly saliva) or perhaps the fecal oral route. Detection of *H. Pylori* DNA in vomits, saliva, dental plaque, gastric juice and fecus. Water borne transmission, probably due to fecal contamination may be an important source of infection especially in parts of world in which untreated water is common. Recent studies in United States have linked clinical *H. Pylori* infection with consumption of *H. Pylori* contaminated well water.

*H. Pylori* probably uncommon in domestic cats. Several studies have suggested sheep as a possible source of *H. Pylori* transmission knowledge of the epidemiology and mode of transmission of *H. Pylori* is important to prevent its spread and may be useful in identifying high risk populations, especially in areas that have high rates of gastric lymphoma, gastric cancer and gastric ulcer.<sup>1</sup>

### What is a peptic ulcer?

Peptic ulcer is a sore on the lining of the stomach or duodenum. A peptic ulcer in the stomach called a gastric ulcers and one that occurs in duodenum is called a duodenal ulcer. Peptic ulcers are common. Each year in United States about half a million people develop a peptic ulcer.<sup>3</sup>

### What causes peptic ulcers?

Together the stomach acid and *H. Pylori* irritates the lining of stomach or duodenum and causes an ulcer. *H. Pylori* infection often begins in childhood. Symptoms usually do not occur until adult hood, although most people never have any symptoms.

*H. Pylori* cause more than half of peptic ulcer worldwide. Yet most people infected with *H. Pylori* never develop ulcers. Why bacterium causes ulcers in some people and not in others is not known. Most likely development of ulcers depends on characteristics of infected person, the type of strain, of *H. Pylori* present and factors researchers have yet to discover. [3]

### NON-GASTRO-INTESTINAL ILLNESS

Skin conditions, depression, low energy levels and headaches. Subsequent findings have shown a relationship with rosacea and urticaria. These conditions may be linked in part to the increased gut permeability, or leaky gut, associated with *H. pylori*.

In addition to influencing gut permeability, *H. pylori* infection can also impede the absorption of iron and vitamin B12 by affecting digestive activity in the stomach and duodenum. This may contribute to the slightly raised risk of cardiovascular problems in sufferers, although these may also be related to other effects of infection such as low levels of antioxidants or high levels of inflammation.

Other vascular conditions such as headaches, migraines and Reynaud's phenomenon have all been linked with *H. pylori*.

Higher rates of infection than expected have been found in those suffering with autoimmune diseases such as rheumatoid arthritis, Sjogren's syndrome and autoimmune thyroiditis, but the mechanism in this connection is not yet understood. *H. pylori* has also been found in bile and in the liver as well as in many sites in the mouth where it is associated with periodontal disease and halitosis. Recent work has found connections with hyperismus gravidum (a severe form of morning sickness), spontaneous recurrent abortion and in colon and pancreatic cancer

### Symptoms of peptic ulcer

Abdominal discomfort is the most common symptom of both duodenal and gastric ulcers

- Is a dull or burning pain in stomach
- Occurs when stomach is empty lasts for minutes to hours and comes and goes for several days or weeks
- Poor appetite
- Bloating
- Nausea
- Vomiting

Some people experience only mild symptoms or more at all.<sup>3</sup> *Helicobacter Pylori* infection may increase the risk of the following. Between 10 – 20% of those with *H. Pylori* will experience peptic (gastric and duodenal) ulceration 1% will experience stomach cancer as a result of the infection. These outcomes are due to the between genetic tendencies, the virulence of the strain of *H. Pylori* and environmental factors. Blood influence the development of gastric cancer, which is more prevalent in type A blood group and which are more prevalent in type O, *H. Pylori* itself has different strains of which *cag A* is most strongly associated with ulceration and cancerous outcomes. Environmental influence show over crowding, poor water supply and hygiene also impact on acquisition rates.<sup>4</sup>

### How an *H. Pylori* is induced ulcer diagnosed?

#### Non invasive techniques

Patients taking an NSAID are asked to stop, reduce the dose or switch to another medication.

The doctor tests to see if *H. Pylori* is present. Testing is important because *H. Pylori* induced ulcers are treated differently than ulcers caused by NSAIDS

Non invasive tests to detect *H. Pylori* in a patient's blood, breath or stool. Because the breath test and stool test more accurately detect *H. Pylori* than blood test.

### Invasive techniques

1. Endoscopy or upper gastrointestinal series.
2. Biopsy
3. If ulcer is bleeding

By using endoscope inject medicines that help blood clot or to guide a heat probe that burns tissue to stop bleeding – a process called cauterization.

For an upper GI series, the patient drinks white, chalky liquid called barium. The barium makes oesophagus, stomach and duodenum and any ulcers show up on X ray.<sup>3</sup>

### How is an H. Pylori induced ulcer treated?

Peptic ulcers caused by H. Pylori are treated with drugs that kill bacteria, reduce stomach acid and protect the stomach and duodenal lining.

Medicines that reduce stomach acid include proton pump [PPIS] inhibitors and histamine receptor blockers (H2 blockers). Both acid reducing help to relieve peptic ulcer pain after a few weeks and promote ulcer healing.

While PPIS cannot kill H. Pylori, research shows they do help fight the H. Pylori infection. Bismuth subsalicylate coats ulcers, protecting them from stomach acid. Although bismuth subsalicylate may kill H. Pylori, it is used with not in place of antibiotics in some treatment regimens.

In United States, clarithromycin based triple therapy, for short is the standard treatment for an ulcer caused by H. Pylori. The doctor prescribes antibiotic clarithromycin, PPI and antibiotics amoxicillin or metronidazole for 10 days. Because research shows higher rates with 14 days of treatment, some doctors now prescribe triple therapy for this longer period, bismuth quadruple therapy is another patent strategy used in United States. The patent a PPI, bismuth subsalicylate, and antibiotics tetracycline and metronidazole for 10 to 14 days.

Bismuth quadruple therapy is used to treat patients in one of several situations, including if the patient.

- Cannot take amoxicillin because of a penicillin allergy
- Has been treated before with a macrolide antibiotics, such as clarithromycin
- Is still infected with H. Pylori because triple therapy failed to kill the bacteria

At least 4 weeks after treatment, doctors test patients using a breath and stools test, to be sure the H. pylori infection has been cured.[3] The new quinolones and rifamycin derivative have recently demonstrated their efficacy in treatment of H. Pylori infection.[4]

### Dietary Recommendations

A diet based on optimum nutrition shows that genetic susceptibility to H. Pylori can be modified mounting

scientific evidence to show that such a diet not only helps to prevent acquisition of bacteria and protects from disease consequences in those who are infected. In one study, research on vitamins and humans has shown that in amounts of 5g a day it can rid the body of H. Pylori in up to 30%. Epidemiological studies have also shown, the diets high in fruit, therefore rich in vitamin c, as well as other antioxidant nutrients are associated with lower rate less disease out come when infection is present.

Fibre is essential for the proper functioning of the digestive system and there is an inverse association between fibre levels, particularly soluble sources from fruits, vegetables, oats, legumes and seeds, and duodenal ulceration and gastric cancer. The introduction of steel rolling mills to produce white flour in the latter part of the nineteenth century coincided with a rise in cases of duodenal ulcer.

The benefits of the probiotic family of lactobacilli have also been proven through research. Studies show possible benefits from fructo-oligosaccharides (FOS), the food for these beneficial gut bacteria as well. Various strains of Lactobacillus acidophilus, and Lactobacillus salivarius inhibit the growth of helicobacter in mice, probably through the production of lactic acid, although they also interfere with H. pylori's ability to stick to cells. The same study showed that as well as eliminating existing colonies of H. pylori, the presence of lactobacilli prevented colonisation by the bacteria. Consumption of tea, both black and green, and red wine is associated with lower incidence of the infection, while coffee and spirits have negative associations.

Foods to be avoided in any anti - H. pylori diet are those high in sugar which can be used by the bacteria, chocolate, coffee, dairy products, red and processed meat, pickled products, refined grains, salt and spirits. These foodstuffs have all been variously associated in many research papers with higher incidence of, or disease resulting from, infection with H. pylori.

### Supplements

Many plants have shown an ability to kill H. pylori in the test tube but animal or human research has yet to prove these connections. Among the substances tested in the laboratory and found to have activity against the microbe is garlic, glycyrrhizic acid, Iceland moss, Manuka honey, cinnamon, garlic, capsaicin and Rheum palmatum (rhubarb root). Barry Marshall notes that H. pylori is sensitive to berberine, and that citrus seed extract is moderately effective in eradicating H. pylori. Mastic gum from the Pistacia lentiscus plant, an ancient remedy for digestive problems, is currently being used with some success.

Research on vitamin C in both mice and humans has shown that in amounts of 5g a day, it can rid the body of H. pylori in up to 30% of individuals.

H. pylori has the potential to cause lifelong problems in those unfortunate enough to be infected with it, but following an optimum diet high in whole grains (excluding wheat), legumes, fruit, vegetables, fish and yoghurt, and avoiding aggregative foods, can reduce health risks and in some cases may help to eradicate infection.

#### supplements for the treatment of H.pylori infection

- Antioxidants: Vitamins A, C, E and selenium, N-acetyl cysteine, alpha lipoic acid.
- Probiotics: Lactobacillus salivarius and Lactobacillus acidophilus
- Prebiotics: F.O.S. 8
- Multivitamin and mineral
- Gastro-intestinal: (where appropriate) Hydrochloric acid (HCl), digestive enzymes, L-Glutamine
- Antibacterials: Garlic, citrus seed extract, mastic gum, berberine.<sup>5</sup>

Subjects drinking 3–6 units of wine / week had an 11% of lower risk of infection compared with those who did not drink wine and intake of 3–6 units of beer was also associated with a similar reduction in the risk of infection when compare to no beer intake.<sup>5</sup>

H. Pylori seropositivity was significantly higher among subjects with border line or low normal B12 levels.<sup>6</sup> If one member of a family is infected with H. Pylori other members are more likely to become infected.<sup>2</sup>

Researchers in Turkey demonstrated that chewing sugarless gum or natural mastic gums reduce the amount of bacterial accumulation in the mouth.<sup>[2]</sup>

#### Hope through Research

Some strains of the bacteria have become resistant to antibiotics that are used to destroy it. Researchers have identified and continue to study new antibiotic combinations that can kill these types of H. Pylori.

Other Promising Research may help identify treatments that

- Kill the H. Pylori bacteria with fewer medicines in less time
- Use different antibiotic combinations in back to back treatment
- Better protect the stomach lining when eliminating H. Pylori.

Researchers also are studying

- Characteristics of H. Pylori bacteria
- Traits of people who develop H. Pylori Ulcers
- Transmission of H. Pylori infection
- Vaccines to prevent and cure H. Pylori infection.[3]

#### Can H. Pylori infection be prevented?

No one knows for sure how H. Pylori spreads, so prevention is difficult, researchers are trying to develop a vaccine to prevent and even cure H. Pylori infection. To help prevent an H. Pylori infection, people should.

- Wash their hands with soap and water after using the bathroom and before eating
- Eat food that has been washed well and cooked properly
- Drink water from a clean, safe source<sup>3</sup>

#### Relationship between H. Pylori infection and periodontal disease

More than 300 bacterial species may be associated with periodontal pockets but only half of these have been cultivable.<sup>7</sup>

In view of potential recolonization subsequent to an initial treatment and difficulties in achieving complete eradication of H. Pylori from gastric mucosa, it is paramount that potential reservoir of this microorganism, other than stomach be identified.<sup>8</sup>

As shown by many investigations, it seems that dental plaque has a more important role for harboring H. Pylori<sup>9-12</sup>. Therefore different gingival clinical states may have an effect on presence of this bacterium in the dental plaque.<sup>13</sup> Periodontal disease is one of the most common oral disease and highest amounts of plaques are found in affected patients.<sup>14</sup> Gebra et.al provided evidence that patients with periodontal disease show high prevalence 43% of H. Pylori in their dental plaque.<sup>13</sup> Umeda et.al showed that 41.2% of patients harbored H. Pylori in stomach or duodenum had periodontal pockets more than 4mm.<sup>15</sup> Bruce et.al in a survey of 4504 participants concluded that periodontal pockets with a depth of 5mm or more are associated with increased rods of H. Pylori sero-positivity<sup>16</sup>. Annam et.al chitsazi et.al and kamat et.al reported that there was no correlation between periodontal disease and H. Pylori infection<sup>17,18 19</sup>. The likely hood of infection with H. Pylori increases with age and infection is more common in people who live at or near poverty level<sup>20</sup>.

The oral cavity is a known niche for H. Pylori and H. Pylori infected individuals have been proven to have a higher rate of dental disorders. The presence of H. Pylori in the oral cavity is frequent and its habitat includes sub gingival and supra gingival biofilm and saliva as well as dorsum of tongue. It is expected that deeper pockets will favour the harboring of H. Pylori thus periodontal disease seen as a potential risk factor.<sup>21-24</sup>

People with severe gum problems are more likely than with healthy gums or with less extensive problems to test positive for *Helicobacter Pylori* bacteria associated with stomach ulcers.<sup>20</sup> *H. Pylori* induces can also cause gastric reflux. Gastric reflux can have severe dental consequences. Tooth enamel is designed to protect the sensitive tooth structure underneath. Acid reflux can attack the surface minerals in tooth enamel and erode them away. The result is teeth that are thinner, weakened and frequently sensitive to temperature extremes.<sup>20</sup>

Following triple therapy, the bacteria can persist in the oral cavity. The presence of dental disorders that could allow *H. Pylori* to be harbored in the oral cavity with possible ecological and immunopathological interactions of *H. Pylori* with other bacteria might be a causative and determining factor in recurrence of *H. Pylori* infection.<sup>23-25</sup>

Systemic antibiotic poly therapy alone is not as effective in preventing the reappearance of gastric *H. Pylori* in persons who are positive for the oral form of these bacteria. Given that periodontal treatment, significantly improved the outcome of systemic antibiotic therapy and periodontal pockets are indeed an important reservoir for *H. Pylori* that the presence of oral *H. Pylori* in persons with periodontitis and gingivitis may cause refractoriness of gastric infection to triple therapy alone.<sup>8</sup>

The mechanism of ability of *H. Pylori* to escape systemic treatment are not well understood. There is evidence indicating that antimicrobials cannot be efficient if the biofilm is not disrupted<sup>26, 27</sup>. The sub gingival dental biofilm known to provide protection from hosts immunological response, the bacteria growing in biofilm exhibit resistance to antimicrobial agents<sup>27, 28, 29</sup>. Adjunctive systemic antimicrobial therapy will depend on the debridement of sub gingival biofilm. Routine dental checkups during *H. Pylori* antibiotic and periodontal treatments should be applied to improve the clinical outcome and decrease the risk of *H. Pylori* recurrence.<sup>8</sup>

The periodontal pocket may provide an ideal micro environment for *H. Pylori* because of its unique micro aerophilic architecture and urea produced by sub gingival biofilm [*H. Pylori* is a urease producing bacteria]. Additionally gastric reflux provide constant source of *H. Pylori* impregnation of the biofilm. *H. Pylori* and periodontal status is important because the oral cavity is primary extra gastric reservoir for *H. Pylori* and infection by this pathogen appears to involve an oral route thus oral health status may influence *H. pylori* infection or re infection. The success rate for gastric eradication of *H. Pylori* has been shown to be dependent on prevalence of *H. Pylori* in oral cavity.<sup>30</sup>

*H. Pylori* is an opportunistic bacterium that will use any method available for gaining access to human stomach. Working with patients was reported to increase the risk of transmission. The sero prevalence significantly increased during the graduation course ranging from 23.4% in students up to 38.6% in residents<sup>31</sup> that contact with patients during clinical practice may constitute a risk factor for acquiring *H. Pylori* infection. Significantly higher risk of infection in nursing staff compared to administrative staff, medical staff or paramedical staff are seen. The level of education was strongly related to prevalence, but not to the incidence of *H. Pylori* infection.<sup>32</sup>

Dentists are among health professionals that may be particularly prone to *H. Pylori* infections. This is related to fact that *H. Pylori* is commonly present in oral cavity in patients and can be disseminated during dental procedures<sup>33, 34, 35</sup>. The latter is associated with generation of so called aerosol cloud which reaches up to 1.5mm from its centre in patients oral cavity, this aerosol cloud contains majority of micro organisms present in the oral cavity and may be source of infection in dentist and dental assistants.<sup>36</sup>

The general prevalence of *H. Pylori* infection is not different between dentists and general population and observed a significant dependence of presence of *H. Pylori* in gingival sulcues in dentists upon the time of their professional activity. Dentists who showed positive presence of *H. Pylori* in gingival sulcus were working on average 6years longer than subjects who did not show the presence of *H. Pylori* in gingival sulcus. There was significant increase of frequency of *H. pylori* presence in gingival sulcus with length of time being active in dental profession.<sup>36</sup>

Interestingly, in male dentists the prevalence of gastric *H. Pylori* infection reached about 80% which is much higher than in female and than that in general population.<sup>36</sup>

There are two main methods currently accepted for the *H. Pylori* detection in oral cavity that is culture of germ and PCR but each of them has disadvantages.<sup>36</sup>

The consequences of *H. Pylori* infections can be severe, therefore the identification of professional hazard, as possibility of *H. Pylori* infection is very important, for taking an action to prevent risk among dentists.<sup>36</sup>

*H. Pylori* was associated with pathogenesis of serious disorders such as chronic antral gastritis of type B, peptic ulcers, mucosa associated lymphoid tissue lymphoma. In 1994 an international agency for research on cancer working group decided on basis of long retrospective evidence to classify *H. Pylori* as a human carcinogen for gastric cancers.<sup>36</sup>

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Recent studies now indicate that there may be significant relationships between H. Pylori and recurrent aphthous stomatitis, as well as herpes simplex virus type 1 [HSV – 1]. It was demonstrated that patients with H. Pylori infection suffered significantly more aphthous outbreaks and that these recurrences had significantly more severe symptoms than for non infected individuals. This relationship is based on a H. Pylori mediated autoimmune reaction involving T. Cell hypersensitivity. HSV I sero positivity was also associated with H. Pylori infection. HSV I and H. Pylori share the same route of transmission, both have been isolated from ulcerations of oral and gastric mucosa, and appears to be a linear association between sero positivity of antibodies against the 2 organisms. More studies will be needed to determine how H. Pylori infection predispose individuals to HSV I infection and if similar to recurrent aphthous stomatitis, H. Pylori eradication therapy leads to a reduction of HSV I infection.<sup>30</sup>

Long term professional dental plaque control was associated with less gastric re infection by H. Pylori, suggesting that dental plaque control may help to prevent H. Pylori induced gastric disease or re infection.<sup>38</sup>

Good oral hygiene not only prevents periodontal disease and gingivitis, it may also help, secondarily to reduce the risk of stomach infection or re infection with H. Pylori.<sup>2</sup>

## CONCLUSION

H. Pylori in dental plaque is seldom eliminated by H. pylori – eradication therapy, and this may act as a source for future re infection. Hence, eradication of H. Pylori from dental plaque should be made an important part of comprehensive management of H. Pylori associated gastric diseases. A combination of medical and dental treatment modalities may be required to prevent re infection of H. Pylori in future and all the health professionals should take necessary precautions while treating H. Pylori patients. Further detailed and comprehensive research should be continued regarding different aspects of H. Pylori bacteria.

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