Relationship between *Helicobacter Pylori*, Chronic Idiopathic Urticaria and Atopic Dermatitis

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ABSTRACT

**Background:** *Helicobacter pylori* (*H. pylori*) is the microorganism responsible for the most frequent and persistent bacterial infection worldwide. *H. pylori* infection affects nearly half of the world’s population. In the developing countries, the prevalence of infection is as high as 90%, whereas in the developed countries, excluding Japan, the prevalence is below 40% (¹). An association between *H. pylori* infection and skin diseases such as chronic idiopathic urticaria and atopic dermatitis has been suggested (²).

**Aim of the work:** this review aimed to focus on the relationship between *Helicobacter pylori*, chronic idiopathic urticaria and atopic dermatitis. This association was reviewed in the following lines.

**Methodology:** we used scientific websites such as PubMed, Google Scholar and Research Gate to get related articles about this subject. **Results:** several studies have reported the presence of *H. pylori* DNA in environmental water sources. Diagnostic tests are usually divided into invasive (endoscopic-based) and noninvasive methods. Using proton-pump inhibitors (PPIs) in combination with several antibiotics such as amoxicillin plus clarithromycin or metronidazole have been considered as the first-line treatment.

**Conclusion:** *H. pylori* has a great prevalence among world’s population. *H. pylori* infection could play a role in the pathogenesis of a variety of skin diseases.

**Keywords:** *H. pylori*, urticaria, atopic dermatitis.

INTRODUCTION

*Helicobacter pylori* (*H. pylori*) is a spiral-shaped microaerophilic gram-negative bacterium that colonizes the gastric mucosa and induces a strong inflammatory response with release of various bacterial and host-dependent cytotoxic substances (¹-³). Epidemiological and experimental data had pointed to a strong relation of *H. pylori* infection with the development of many extragastric diseases, such as cardiovascular, immunologic and some skin diseases (⁴).

Most infections are probably acquired in childhood, but geographic area, age, race, socioeconomic status and hygiene seem to play roles in the prevalence of *Helicobacter pylori*.

Higher rates of infection tend to occur at a younger age in the developing countries compared to the developed countries and in regions characterized by lower socioeconomic status and higher density living (⁵). Since *Helicobacter pylori* (*H. pylori*) identification in 1983, an increasing amount of knowledge has collected, with this pathogen having been directly involved in the pathogenesis of several dermatological diseases (⁶). An association between *H. pylori* infection and skin diseases such as chronic idiopathic urticaria and atopic dermatitis has been suggested (²).

This review concerned with the association between *H. pylori* infection and occurrence of skin diseases especially urticarial and atopic dermatitis.

METHODOLOGY

We used scientific websites such as PubMed, Google Scholar and Research Gate to get related articles about this subject. The research process involved specific keywords "correlation between *H. pylori* infection and urticarial, correlation between *H. pylori* infection and atopic dermatitis, correlation between *H. pylori* infection and skin diseases and complication of *H. pylori* infection " to find more articles on the subject. We were more concerned about English published articles only which published from 1995 to 2017.

*H. pylori* epidemiology, sources, diagnosis, treatment and complications

*Helicobacter pylori* (*H. pylori*) is the microorganism responsible for the most frequent and persistent bacterial infection worldwide. *H. pylori* infection affects nearly half of the world’s population. In the developing countries, the prevalence of infection is as high as 90%, whereas in developed countries, excluding Japan, the prevalence is below 40% (²).

Several studies have reported the presence of *H. pylori* DNA in environmental water sources (⁷-⁹), but this probably reflects contamination with either naked DNA or dead *H. pylori* organisms. Spread via fecal contaminants is supported by the occurrence of *H. pylori* infections among institutionalized young people during outbreaks of gastroenteritis (¹⁰).
Other possible sources include contaminated food, as *H. pylori* may survive briefly in refrigerated food (14). Coupled with the extreme sensitivity of *H. pylori* to atmospheric oxygen pressure, lack of nutrients and temperatures outside the 34 to 40°C range (12), direct person-to-person transmission remains the most likely transmission route. Diagnostic tests are usually divided into invasive (endoscopic-based) and noninvasive methods. Invasive diagnostic tests include endoscopic image, histology, rapid urease test, culture and molecular methods. Non-invasive diagnostic tests included urea breath test, stool antigen test, serological, and molecular examinations (13).

The regimens that utilize proton-pump inhibitors (PPIs) in combination with several antibiotics such as amoxicillin plus clarithromycin or metronidazole have been considered as the first-line treatment for *H. pylori* infection (14).

In some countries, new first-line treatments are not accepted because of a lack of national validation studies and a lack of studies of clarithromycin resistance (15).

The Maastricht IV/Florence Consensus Report recommended the bismuth-containing quadruple therapy as an alternative for first-line empirical treatment in areas with the clarithromycin resistance over 15%-20%. If this regimen is not available sequential therapy or a non-bismuth quadruple therapy (the so-called “concomitant” treatment) is recommended (16).

Currently, *Helicobacter pylori* (*H. pylori*) infection is confirmed to correlate with chronic gastritis, peptic ulcer disease, Mucosa Associated Lymphoid Tissue (MALT)-lymphoma, precancerous changes in the stomach (atrophy, intestinal metaplasia) and gastric cancer. At the same time, *H. pylori* eludes the immunological response evoked by the host. This chronic infection has the local production and systemic diffusion of pro-inflammatory cytokines, which may influence the remote organic systems and result in extragastric manifestations (17).

Some studies suggested that *H. pylori* infection could play a role in the pathogenesis of a variety of skin diseases (18). The best evidence for such link is found for chronic urticarial (19).

Worthy of note, many case reports have described interesting associations between *H. pylori* infection and atopic dermatitis (AD) (20).

**Correlation between chronic urticarial infection and *H. pylori* infection:**

Chronic urticaria (CU) is defined as the occurrence of daily, or almost daily, wheals and itching for at least 6 weeks. It is a common and potentially debilitating skin condition that affects up to 1% of the general population with variable duration, typically several months but occasionally decades (21).

Chronic urticaria is associated with various autoimmune disorders (22). There is a possible association with malignancies, although data are conflicting (23). None of the theories of pathogenesis of CU has been fully established (24). The best-developed hypotheses included the autoimmune theory, theories involving histamine-releasing factors and the cellular defects theory.

Attempts have been made to associate some common chronic infections such as *Helicobacter pylori* with CU (25).

There is increasing evidence for systemic effects of gastric *H. pylori* infection, which may be involved in extra gastrointestinal disorders such as vascular, autoimmune and skin diseases.

A possible relationship between chronic idiopathic urticaria and *H. pylori* infection has been suggested in preliminary studies, in which antibiotic eradication of *H. pylori* lead to regression of urticaria in up 100% of cases (25-27). Regarding the possible mechanisms involved in the relationship between *H. pylori* infection and chronic urticaria, a number of speculations and theories have been put forward. One possible explanation might be that the immunologic stimulation induced by infection might, through mediator release, causes a non-specific increase of the skin vessel sensitivity to agents increasing vascular permeability (28).

A number of agents might act through this mechanism. As a matter of fact, increased production of interleukin 8 (IL-8), platelet-activating factor (PAF) and leukotrienes (LT) B4 and C4 has been observed in the gastric mucosa of *H. pylori* infected patients and these mediators exert evident actions on the skin (29, 30).

Another possibility would be that urticaria patients might develop specific IgE antibodies to *H. pylori*, an attractive explanation that still requires confirmation (31). In this context, Liutu *et al.* (32) have reported gre ater rates of total IgE increase in patients with chronic urticarial and *H. pylori* infection than in those with chronic urticaria but without such infection.
There have also been observations reported of increased serum *H. pylori* IgE and basophil-bound IgE in subjects with infection (33) and increased basophil counts in peripheral blood in patients with dyspepsia and *H. pylori* positivity have also been reported (34).

CONCLUSION

*H. pylori* infection affects nearly half of the world’s population. Recent evidence suggested that *H. pylori* infection could play a role in the pathogenesis of a variety of skin diseases. There is increasing evidence for systemic effects of gastric *H. pylori* infection, which may be involved in extra gastrointestinal disorders such as vascular, autoimmune and skin diseases (27). It has been hypothesized an important role of *Helicobacter pylori* (*H. pylori*) infection in the host immune network arrangement and its influence on the development of allergic diseases such as atopic dermatitis (30).

REFERENCES

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