International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
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ARTICLE TITLE	THE ROLE OF *HELICOBACTER PYLORI* IN CHRONIC SPONTANEOUS URTICARIA: FROM PATHOGENESIS TO CLINICAL IMPLICATIONS
DOI	https://doi.org/10.31435/ijitss.1(49).2026.5422
RECEIVED	07 February 2026
ACCEPTED	16 March 2026
PUBLISHED	27 March 2026
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THE ROLE OF *HELICOBACTER PYLORI* IN CHRONIC SPONTANEOUS URTICARIA: FROM PATHOGENESIS TO CLINICAL IMPLICATIONS

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ABSTRACT

Background: Chronic spontaneous urticaria (CSU) is characterized by the presence of pruritic wheals persisting for more than 6 weeks, which may or may not be accompanied by angioedema. In recent years, increasing attention has been given to the potential role of infectious factors in the pathogenesis of CSU, including Helicobacter pylori infection, which may represent a contributing factor to disease exacerbation.
Objective: This review aims to evaluate the association between H. pylori infection and chronic spontaneous urticaria. It addresses underlying pathophysiological mechanisms, the impact of bacterial eradication on clinical symptoms, and the clinical relevance of diagnostic and therapeutic approaches.
Methods: A literature review was conducted to assess the association between H. pylori infection and chronic spontaneous urticaria. Studies addressing disease pathomechanisms, the impact of bacterial eradication on the clinical course, and the role of diagnostic and therapeutic strategies were analyzed. Both clinical studies and meta-analyses published in peer-reviewed medical journals were included.
Results: H. pylori infection is more frequently observed in patients with CSU, although its prevalence does not consistently exceed that in the general population. Symptomatic improvement following bacterial eradication has been reported in some patients; however, the correlation remains inconclusive.
Conclusions: H. pylori infection may influence the course of chronic spontaneous urticaria by modulating immune responses and inflammatory processes, which may partly explain symptomatic improvement following eradication. However, study findings remain inconsistent, and limitations such as small patient cohorts and heterogeneity of treatment regimens hinder a definitive assessment of the bacterium's role in the disease.

KEYWORDS

Helicobacter Pylori, Chronic Spontaneous Urticaria, Eradication, CSU, Pathogenesis

CITATION

Anna Kamosińska, Julia Pająk, Antoni Majda, Natalia Sitko, Paulina Makowska, Joanna Gontarczyk, Alicja Laske, Martyna Sowa, Kacper Kucharski, Adam Kowal, Julia Sokołowska, Marcel Dawidowicz. (2026) The Role of Helicobacter Pylori in Chronic Spontaneous Urticaria: From Pathogenesis to Clinical Implications. *International Journal of Innovative Technologies in Social Science*. 1(49). doi: 10.31435/ijitss.1(49).2026.5422

Introduction

Urticaria is a common skin disorder, with an estimated 12-25% of the population experiencing at least one episode during their lifetime, whereas the chronic form affects approximately 0.5-1.5% of the population [1, 2]. Depending on the duration of symptoms, urticaria is classified as either acute or chronic [3]. Chronic urticaria is characterized by the presence of pruritic wheals persisting for more than 6 weeks, which may or may not be accompanied by angioedema. This condition can be classified into two main types: chronic spontaneous urticaria, which occurs without an identifiable triggering factor, and inducible urticaria, in which symptoms are elicited by specific physical or non-physical stimuli [4]. In recent years, increasing attention has been given to the potential role of infectious factors in the pathogenesis of chronic spontaneous urticaria, including *Helicobacter pylori* infection, which may represent one of the factors exacerbating disease severity [5]. The prevalence of *Helicobacter pylori* infection is significantly associated with socioeconomic status and increases with age. In developing countries, the proportion of infected individuals ranges from 80% to nearly 100%, whereas in developed countries, including those in Western Europe, the United States, and Australia, it ranges from 20% to 40%. Poland is considered a country with intermediate infection prevalence, estimated at approximately 40-60% of the population [1]. Urticaria can occur in both children and adults; however, it is more common in adults and also shows a higher prevalence among women [3]. Given the observed association between *H. pylori* infection and the occurrence of urticaria, several hypotheses have been proposed to explain this relationship. Some authors suggest that *H. pylori* infection increases gastric mucosal permeability, potentially leading to greater exposure of the body to allergens present in the gastrointestinal tract. Another hypothesis emphasizes the role of the immune response, in which antibodies produced during infection may

promote mast cell activation and histamine release in the skin [6]. Despite these considerations, the association between *H. pylori* infection and chronic idiopathic urticaria remains controversial. This is due to inconsistent study results, methodological limitations, and the lack of clear evidence for a causal relationship [7, 8].

Consequently, this issue remains the subject of considerable discussion. Despite existing evidence, sources emphasize that the association continues to be debated. It should also be noted that chronic spontaneous urticaria significantly affects patients 'quality of life, leading to impairments in both professional and personal functioning. For these reasons, it remains an important and current research problem [1].

The aim of this review is to evaluate the association between *H. pylori* infection and chronic spontaneous urticaria, with a particular focus on the underlying pathophysiological mechanisms, the impact of bacterial eradication on symptoms, and the clinical relevance of diagnostic and therapeutic approaches in light of available studies and ongoing controversies.

Helicobacter pylori - characteristics and mechanisms influencing chronic spontaneous urticaria

Biological characteristics of *Helicobacter pylori*

Helicobacter pylori is a Gram-negative, spiral-shaped bacterium measuring 2–4 μm in length. It is highly adapted to colonize the stomach and is present in over half of the human population. As a microaerophilic organism, it requires reduced oxygen levels (2–5%), the presence of carbon dioxide (5–10%), and high environmental humidity for optimal growth, conditions that reflect its natural ecological niche within the mucus layer covering the gastric mucosa. It is equipped with 2 to 6 polar, sheathed flagella, allowing high motility and movement through the viscous gastric mucus. [9, 10] Additionally, through the use of specific adhesin molecules, such as BabA and SabA, the bacterium is able to adhere to gastric epithelial cells. This process occurs via the binding of adhesins to carbohydrates and lipids present on the host cell membrane, enabling persistent colonization of the gastric environment [10, 11]. A key factor in the bacterium's adaptation to gastric colonization is its ability to survive in acidic pH. Although *H. pylori* is a facultative neutrophile, with optimal growth at pH ranging from 5.5 to 8.0, it produces urease, which catalyzes the hydrolysis of urea into ammonia and carbon dioxide, leading to local neutralization of gastric acid in its immediate environment. This ability to survive in the stomach is a crucial aspect of its pathogenicity and enables the induction of a chronic host inflammatory response [9]. In addition to mechanisms enabling colonization and survival, *H. pylori* possesses virulence factors, such as the type IV secretion system (T4SS), the CagA protein, and the VacA cytotoxin, which allow the bacterium to interfere with host cell functions, leading to cellular damage, dysregulation, and promoting the development of diseases, including cancer [10].

Epidemiology and routes of transmission

The primary reservoir of *H. pylori* is humans, and transmission most commonly occurs through direct person-to-person contact [9, 12]. The main route of infection is fecal–oral, although oral–oral and gastric–oral transmission also play significant roles [9]. Although the bacterium is sensitive to atmospheric oxygen, it can temporarily survive in the external environment, including contaminated water or food, suggesting the possibility of indirect transmission [9, 12]. Infection is usually acquired in early childhood from close family members, and the risk increases with a higher number of household members and lower socioeconomic status [9]. Intra-familial transmission, primarily from mother to child, is considered a key mechanism for the spread of infection. Cases of *H. pylori* isolation from household animals have also been reported; however, evidence for widespread zoonotic transmission is limited and of marginal epidemiological significance [9, 12]. It should be emphasized that the precise mechanism of *Helicobacter pylori* transmission has not been fully clarified, and available data suggest the existence of multiple coexisting routes of infection.

Immune Response and Pathogenesis of Infection

Persistent colonization by *H. pylori*, together with the accompanying activation of the host immune response, leads to the development of chronic gastritis. This condition occurs in nearly all infected individuals, although most patients remain asymptomatic [9]. The primary mechanism initiating inflammation is the direct interaction of the bacterium with epithelial cells. This leads to the activation of signaling pathways, including the transcription factor NF-κB (nuclear factor kappa B) and MAP kinases (mitogen-activated protein kinases) [13, 14]. As a result, there is massive production and release of proinflammatory cytokines, primarily interleukins IL-1β, IL-6, IL-8, and tumor necrosis factor α (TNF-α) [13, 14, 15]. The released inflammatory mediators lead to the recruitment and infiltration of immune cells, primarily neutrophils, macrophages, monocytes, and T lymphocytes, thereby intensifying the local inflammatory response [9, 14, 15]. Since *H.*

pylori is an extracellular pathogen, the activated type I immune response does not eliminate the bacterium but instead contributes to progressive tissue damage [9]. An important component of the inflammatory response mechanism is the production of reactive oxygen species (ROS), which further amplify and prolong the inflammatory cascade and cause damage to the mitochondrial DNA of epithelial cells [15]. Concurrently with these oxidative processes, mast cell activation, induced by specific bacterial virulence factors such as HP-NAP, plays a key role in pathogenesis. Their degranulation, resulting in the release of histamine and cytokines, constitutes an important mechanism for amplifying chronic inflammation and may also explain systemic hypersensitivity reactions in infected individuals [16]. In the context of the chronic nature of the infection, the mechanism of molecular mimicry is also highly significant. This involves the structural similarity between bacterial antigens and host antigens, which may lead to cross-reactive immune responses [17]. In *H. pylori*-infected patients, CD4+ Th1 lymphocytes can become activated and exhibit the ability to cross-recognize both bacterial proteins and the human proton pump (H^+/K^+-ATPase) located in gastric parietal cells [17, 18]. Data from conducted studies indicate that cross-reactive epitopes derived from nine different bacterial proteins have been identified, which stimulate T lymphocyte proliferation and enhance the Th1-type immune response [17, 18]. Furthermore, it has been shown that *H. pylori* lipopolysaccharides contain epitopes structurally resembling Lewis blood group antigens (Le^x and Le^y), which are physiologically expressed on human gastric epithelial cells. This phenomenon may enable the bacterium to evade the host immune system, promoting persistent infection, and may also induce the production of anti-Lewis antibodies capable of cross-reacting with the gastric mucosa, leading to tissue damage [19]. The described pathophysiological mechanisms of *H. pylori* infection—including chronic inflammation, immune cell activation, and autoimmune phenomena—may play a role in the pathogenesis of immune-mediated diseases, including chronic spontaneous urticaria [9, 17, 20].

Pathogenesis of Chronic Spontaneous Urticaria and the Potential Role of *Helicobacter pylori*

The pathogenesis of chronic spontaneous urticaria (CSU) is complex. Despite the absence of a clearly identifiable trigger, CSU is considered a disease of multifactorial etiology. Increasing evidence points to the involvement of immune dysregulation and chronic inflammatory processes, including infections, in its development. A key role is played by the activation and degranulation of mast cells, leading to the release of preformed mediators such as histamine and tryptase, which are responsible for the manifestation of disease symptoms. In addition, de novo-synthesized mediators, as well as numerous cytokines and chemokines, are released and contribute to the maintenance of the inflammatory state [2].

An important aspect of CSU pathophysiology involves autoimmune mechanisms, associated with the presence of autoantibodies directed against host antigens, including the FcεRI receptor or immunoglobulin E, which can directly activate mast cells [2]. Other pathways modulating the inflammatory response are also involved, such as activation of the coagulation cascade and the complement system. Generated thrombin can directly activate mast cells via PAR receptors, while the anaphylatoxin C5a enhances their degranulation [21]. Furthermore, IgE-independent activation of the MRGPRX2 receptor by certain drugs, neuropeptides, or eosinophil-derived mediators can also trigger mast cell degranulation and exacerbate disease symptoms [2, 22].

In the context of the complex pathogenesis of chronic spontaneous urticaria, increasing attention is being paid to the role of external factors, including chronic infections. *H. pylori* infection may modulate the host immune response through mast cell activation and the induction of a persistent inflammatory state. Additionally, autoimmune mechanisms, including molecular mimicry and the induction of autoantibodies, may further contribute to the maintenance of cutaneous inflammation. Clinical data indicate that in some patients with CSU, *H. pylori* eradication leads to symptom improvement, suggesting a potential role of this bacterium in exacerbating or sustaining the disease. [9, 16, 19]

Clinical significance

In some studies assessing the effect of *H. pylori* eradication on symptoms of chronic urticaria, it has been shown that successful elimination of the bacterium may lead to a reduction in symptom severity, and in some patients even to complete remission of skin lesions [23, 24]. However, it should be emphasized that this improvement does not occur in all patients, suggesting a multifactorial etiology of the disease and resulting in inconsistent findings regarding a direct causal relationship between the infection and the occurrence of skin symptoms [23, 25].

A meta-analysis conducted by Watanabe et al. compiled data from multiple earlier studies investigating the effectiveness of antibiotic therapy and the eradication of *H. pylori* in the treatment of chronic urticaria. Nine randomized clinical trials were analyzed, including a total of 361 patients, comparing the effects of

antibiotic therapy and dapsone. In both cases, clinical symptoms improved; however, bacterial eradication was associated with fewer reported adverse events. The data suggest that antibiotic therapy aimed at the eradication of *H. pylori* was associated with an approximately fourfold increase in the likelihood of complete disease remission [26].

It should be noted that the observed therapeutic effect may result not only from bacterial eradication itself, but also from the nonspecific effects of antibiotic therapy. This possibility is supported by studies conducted by Kim et al., which found no clear association between the effectiveness of *H. pylori* eradication and the resolution of urticaria symptoms. Accordingly, the effectiveness of eradication therapy in improving symptoms of chronic spontaneous urticaria has not been conclusively established. Symptom resolution was not directly associated with successful bacterial elimination, as patients receiving antibiotics reported improvement in skin condition regardless of whether the infection was completely eradicated. Additionally, the authors suggest that antibiotics may influence the course of the disease indirectly, for example through anti-inflammatory effects or by eliminating other, undiagnosed infections. Consequently, it remains difficult to confirm that *H. pylori* is a direct cause of the disease, highlighting the need for further research [27].

Routine testing for Helicobacter pylori in all patients with chronic idiopathic urticaria is not currently recommended [25]. However, this does not mean that such diagnostic evaluation is entirely unjustified. It may be worth considering, particularly in patients who also present with gastrointestinal symptoms such as dyspepsia, epigastric pain, postprandial fullness, or heartburn, or when standard urticaria treatment fails to produce the expected results [23, 28]. It should also be taken into account in patients with a recurrent course of urticaria [7]. An additional argument in favor of testing may be the presence of comorbid conditions associated with this bacterium, such as gastric or duodenal ulcer disease, as well as a high prevalence of infection within a given population [29].

Eradication of *Helicobacter pylori* is not currently considered a standard treatment for all patients with chronic idiopathic urticaria [28, 30]; it is recommended primarily in those with confirmed infection and clear gastroenterological indications [28]. In such cases, a possible improvement in skin symptoms may be an additional benefit, although it is not guaranteed [5]. It should also be noted that the therapy requires the use of antibiotics, which may cause adverse effects and contribute to the development of bacterial resistance [31]. Therefore, the decision to initiate eradication therapy should be made on an individual basis, and further research is needed to better determine which patients are most likely to benefit from this approach.

Limitations of the studies

The role of *H. pylori* in the pathogenesis of chronic spontaneous urticaria has been the subject of numerous studies. These suggest a possible association between infection with this bacterium and the occurrence of skin lesions, indicating that the infection may contribute to both the initiation and exacerbation of disease symptoms [7]. Despite these promising findings, many authors emphasize significant limitations that substantially hinder the formulation of definitive conclusions.

In the meta-analysis by Cui et al., several important limitations of the analyzed studies were identified, particularly small sample sizes, which may negatively affect the accuracy and reliability of the results. Additionally, a lack of uniformity in the methods used to detect *H. pylori* was noted, making the interpretation of findings more challenging. The authors also pointed out the absence of clear pathological or etiological evidence supporting this association. Consequently, it remains unclear whether the relationship between infection and urticaria is direct or indirect [7]. Another important limitation is the variability in treatment regimens and the clinical outcomes assessed. The use of concomitant medications, such as antihistamines, as well as differing criteria for evaluating treatment efficacy, further complicate direct comparisons between studies [32]. In this context, it is also difficult to distinguish between the effects of *H. pylori* eradication and the nonspecific effects of antibiotic therapy on clinical improvement, which represents a significant interpretative limitation [27].

Moreover, studies by Fukuda et al. indicate that although eradication of *H. pylori* infection may lead to clinical improvement in some patients, the prevalence of the infection in individuals with CSU is not always higher than in healthy populations. This represents an additional factor complicating the formulation of clear conclusions regarding the role of *H. pylori* in chronic idiopathic urticaria [33].

An important limitation of the analyzed studies is the unclear pathogenesis of chronic spontaneous urticaria. The presence of multiple underlying mechanisms, including autoimmune processes, suggests that CSU represents a heterogeneous clinical syndrome rather than a single disease entity [34]. Although evidence indicates a potential association between *H. pylori* infection and the course of CSU, current data are insufficient

to establish a direct causal relationship. Methodological limitations and inconsistent findings across clinical studies indicate that the role of *H. pylori* in the pathogenesis of CSU remains uncertain and warrants further investigation [23, 32]

Conclusions

Data from the analyzed literature suggest that *H. pylori* infection may be involved in the pathogenesis of chronic spontaneous urticaria through modulation of the immune response and inflammatory processes, which may explain the clinical improvement observed in some patients following eradication. However, the available evidence remains inconclusive. Symptom improvement does not always correlate with successful bacterial eradication, and the complex and heterogeneous pathogenesis of the disease further complicates data interpretation. Therefore, current evidence does not support a definitive conclusion that *H. pylori* is a causative factor in urticaria. Existing study limitations, such as small sample sizes, variability in treatment regimens, and differences in the assessment of clinical outcomes, highlight the need for further clinical research to better define the role of this bacterium in the course of the disease.

Declaration on the use of AI: In preparing this work, the authors used Gemini for the purpose of improving language and readability, text formatting, and verification of bibliographic styles. After using this tool/service, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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