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Recent Advances in Research on the Association Between Chronic Urticaria and Helicobacter pylori

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Abstract. The association between Chronic Urticaria (CU) and Helicobacter pylori (H. pylori) infection has attracted progressively attention. This article systematically reviews research progress of these two conditions on regarding the epidemiology, pathogenesis, and treatment strategy aspects. Epidemiological data imply that the prevalence of H. pylori infection in patients with Chronic Urticaria (CU) is markedly higher than healthy individuals, and higher infection rates referred to females, young and middle-aged individuals, and those accompanied by gastrointestinal symptoms. In the way of efficacy prediction, patients infected with Cag A-positive H. pylori strains, serum Hp-IgE antibodies-positive and IL-17/IL-23 elevated levels achieve more significant relief symptom after H. pylori eradication, which suggesting that these indicators help screen potential beneficiaries. The pathogenesis refers to Th1/Th2 immune imbalance, IgE-mediated mast cell activation, intestinal barrier damage and neuroendocrine regulation disorder, the three through the "Gut-skin axis" exerting synergistic effects. As for treatments, Standard Triple Therapy and Bismuth Quadruple Therapy serve as core treatments, yet drug resistance remains a prominent challenge; Acupoint Catgut Embedding Therapy and Sequential Treatment Of Traditional Chinese and Western medicine (such as Ban Xia Xie Xin Decoction) can improve H. pylori eradication and clinical cure rates while reducing recurrence rates. Nevertheless, current researches are limited by small sample sizes, discrepant diagnostic and therapeutic criteria and insufficient mechanistic exploration. In the future, efforts should concentrate on conducting multicenter and forward-thinking studies. It's necessary to establish precise efficacy prediction models and explore comprehensively the scientific connotation behind integrated traditional Chinese and Western medicine therapy.

Keywords: Chronic Urticaria, Helicobacter Pylori, Prediction of Treatment Efficacy, Mechanism of Disease, Eradication Treatment

1. Introduction

Chronic Urticaria (CU) is a common allergic skin disease in daily life, which is accompanied by spontaneous urticaria or angioedema occurring at least twice a week and lasting six weeks or longer. The global incidence of CU is 4.4% [1], but different regions have notable discrepancies. The incidence in Asia (1.4%, 0.5–2.9) is obviously higher than in Europe (0.5%, 0.2–1.0) and North America (0.1%, 0.1–0.1). Among these, China holds the highest prevalence of urticaria, ranging

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from 2.6% to 4.2% [2], with over half of cases being chronic spontaneous urticaria, and females are considerably more influenced than males. Due to the reference of multiple internal and external factors as well as immune mechanisms, the mechanism of CU is complex and has not been completely clarified so far. Patients with CU appear to have severe itchiness, which can drastically impact their quality of life. Helicobacter pylori (H. pylori), a microaerophilic Gram-negative bacterium, widely exists in the anatomy and is closely associated with various gastrointestinal disorders, including duodenal ulcers and stomach cancer. H. pylori infection is a prevalent global infection, which affects around 43% of the world's population. In China, the prevalence of H. pylori infection is roughly 41.5%–44.2%, yet different regions and populations have distinct contrasts. The rural areas are superior to urban areas, and adults exceed children. In recent years, numerous studies have confirmed that there is a certain connection between CU and H. pylori, though thorough research to analyze the comorbidity of the two remains lacking [3-4]. Many experts and scholars regarding the role of H. pylori in the progress of disease in CU are undergoing a dynamic evolution. Early research revolving around H. pylori infection and CU associations has two major controversies. On the one hand, whether there is an actual relationship between the two, some studies demonstrate patients in CU with a higher H. pylori infection rate and symptoms improved after following eradication [5]. On the other hand, whether H. pylori acts as a direct pathogenetic factor or only through systemic inflammatory responses to aggravate the clinical condition indirectly [6]. These discrepancies are closely connected with limitations like inconsistent study designs, small sample sizes, and failure to distinguish eradication ups and downs.

Over the past, although meta-analyses have verified that H. pylori eradication improves the relief rate of CU, there is significant heterogeneity in treatment effects. Additionally, excessive treatment may give rise to increasing antibiotic resistance and disorder of the gut microbiota. Consequently, concentration of research has gradually shifted from "verifying associations" to "precision medical" and how to accurately identify the patients likely to benefit from it before treatment has become a critical problem requiring urgent resolution. This article systematically summarizes the association between H. pylori infection and CU, including epidemiological association, multidimensional pathogenesis, therapeutic heterogeneity of different eradication regimens and advances in integrated traditional Chinese and Western medicine therapies. It analyzes the insufficiency of current research and provides scientific evidence for the clinical diagnosis and treatment of H. pylori-related CU.

2. Epidemiologic association between CU and H. pylori infection

2.1. Prevalence of H. pylori infection in patients with CU

Multiple clinical researches have indicated that the occurrence of H. pylori infection is remarkably higher in patients with CU than in the healthy population [7-9]. Wang et al. detected H. pylori urease antibodies in 420 patients with CU and 450 healthy subjects undergoing physical examinations during the same period [10]. The results showed that the H. pylori positivity rate among CU patients was 38.6%, considerably higher than the 14.4% in the healthy control group ($P < 0.05$). Guo et al. collected 2,728 patients with CU and 3,500 healthy controls conducting a meta-analysis and subgroup analysis [3]. The results all showed that the H. pylori positivity rate in the CU group was higher than in the healthy control group, having statistically significant differences. In terms of population segmentation, H. pylori infection rates among female patients, young and middle-aged individuals and CU patients with concomitant gastrointestinal discomfort were higher. Additionally, patients experiencing positive test results usually exhibited symptoms including longer disease duration, more severe itching symptoms and a higher tendency for recurrent episodes. While

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infection rates may exist slight fluctuations across studies due to the differences between geographic location, testing methods, and case inclusion criteria, the overall conclusions on epidemiological differences are unified. This thoroughly verifies a closed connection between H. pylori infection and the incidence of CU in the population. It also provides epidemiological evidence to support routine H. pylori screening among high-risk populations in clinic.

2.2. Prediction of curative effect related to CU and H. pylori

Notwithstanding multiple randomized controlled trials have confirmed that H. pylori eradication therapy can cause some H. pylori-positive patients with CU complete relief or significantly improve symptoms, some patients still experience poor treatment responses [11-13]. To prevent unnecessary use of antibiotics and enhance treatment regimens, research has increasingly focused on accurately identifying patient subgroups most likely to benefit from H. pylori eradication therapy in recent years. At present, several clinically valuable predictors of treatment efficacy have been identified.

2.2.1. H. pylori virulence factors

H. pylori virulence factors are key determinants of its pathogenicity, and Cytotoxin-associated Protein A (Cag A) is one of the most extensively studied virulence factors. Cag A-positive H. pylori strains are significantly more virulent than Cag A-negative strains. Cag A is designed into gastric mucosal epithelial cells through the type IV secretion system, where it disrupts the cytoskeletal structure, compromises the integrity of the gastric mucosa, and leads to erosion and ulcer formation. Due to the disruption of the gastric mucosal barrier, mast cells in the lamina propria can directly contact H. pylori and their metabolic products (such as urease and vacuolating toxin Vac A), which activate them to release inflammatory mediators such as histamine, leukotrienes, and prostaglandins. These mediators can reach the skin throughout the body via the bloodstream, directly or indirectly activating skin mast cells and inducing urticaria and pruritus [14]. Clinical studies have shown that patients with CU infected with Cag A-positive H. pylori strains exhibit a significantly higher rate of symptom resolution following eradication therapy compared to those infected with Cag A-negative strains [15].

2.2.2. Serological markers

Positive serum Hp-IgE antibodies (specific immunoglobulin E antibodies produced by the body in response to H. pylori antigens, which are hallmark antibodies of Type I hypersensitivity). Traditionally, it was believed that H. pylori infection primarily induces the production of IgG and IgA antibodies. However, recent studies have found that some patients with H. pylori infection may produce specific Hp-IgE antibodies. Hp-IgE can combine with high approachability receptors (FcεRI receptors) on the surface of mast cells and basophils, sensitizing these cells. Upon re-exposure to H. pylori antigens, this can trigger cell degranulation, leading to the release of inflammatory mediators and inducing an urticaria attack. Therefore, for patients with positive serum Hp-IgE antibodies, the causal relationship between CU and H. pylori infection is more clearly established, and symptom improvement following eradication therapy is more pronounced [16, 17].

2.2.3. Serum interleukin-17 (IL-17) and interleukin-23 (IL-23)

IL-17 and IL-23 are key cytokines involved in chronic inflammation and autoimmune responses. H. pylori infection can activate dendritic cells and macrophages to secrete interleukin-23 (IL-23),

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which induces the naive T cells to differentiate into the T helper 17 (Th17) cells; Th17 cells secrete large amounts of IL-17. IL-17 promotes neutrophil infiltration, induces the release of various inflammatory mediators, and exacerbates inflammatory responses in the skin and throughout the body. Clinical studies have shown that H. pylori-positive patients with CU who have significantly elevated serum levels of IL-17 and IL-23 exhibit a more pronounced reduction in symptom scores and a lower recurrence rate following eradication therapy [6, 18].

3. Pathogenesis of CU caused by H. pylori infection

3.1. Mechanisms of immune dysregulation

Imbalance of T helper 1 (Th1)/T helper 2 (Th2) Immune Responses: H. pylori infection modulates the production of interferon-γ (IFN-γ) and interleukin-4 (IL-4) by peripheral blood mononuclear cells, leading to an imbalance in Th1 and Th2 subsets and exacerbating immune dysfunction [19].

IgE Antibody Production: H. pylori, acting as an antigen, can induce the body to produce specific Hp-IgE antibodies. Since IgE antibodies bind to FcεRI receptors on the surface of mast cells and basophils, re-exposure to H. pylori antigens can trigger cell degranulation, releasing active substances such as histamine and leukotrienes, which increase vascular permeability and cause capillary dilation, resulting in a series of skin allergy symptoms [6, 16, 18].

3.2. Mechanisms of intestinal dysbiosis

H. pylori has settled in the gastric mucosa over the long term and produced urease to break down urea. The urea is converted into alkaline ammonia by urease, which neutralizes gastric acid, stimulates the secretion of gastric acid and gastrin, and causes vasodilation in the gastric mucosa. Due to the gastrin in the blood dilating the skin blood vessels and increasing vascular permeability, the risk of gastrointestinal exposure to allergens increases, leading to the emergence of urticaria [20]. Simultaneously, H. pylori infection can disrupt the balance of the gut microbiome through a top-down effect by means of the "gut-brain axis," which decreases gut microbiota diversity, leading to a slump in the adequacy of friendly bacteria like Bifidobacterium and Lactobacillus and an excessive proliferation in opportunistic pathogens like Enterobacteriaceae and Klebsiella species. The imbalance of the gut microbiota further damages the intestinal mucosal barrier and increases intestinal permeability (leaky gut). This allows food antigens, incompletely degraded allergens and inflammatory mediators to enter the circulation, activating a systemic Th2-type immune response. It induces mast cell degranulation and the release of histamine and continuously drives skin allergies and inflammation, constituting a key mechanism underlying the persistent and recurrent distinction of CU [21, 22].

3.3. Neuroendocrine dysregulation

Neurotransmitter Imbalance: H. pylori infection causes intestinal chromaffin cells and the synthesis of serotonin (5-HT) to increase. Simultaneously, there is increased release of neuropeptide P (substance P) and decreased levels of gamma-aminobutyric acid (GABA). These neurotransmitters directly stimulate cutaneous sensory nerves to cause itching and trigger mast cell degranulation in a non-IgE-dependent manner [21]. Impaired intestinal barrier, gut microbiota imbalance, and neuroendocrine dysregulation are not independent of one another; rather, form a synergy through the "gut-skin axis" collectively generating systemic immune dysregulation and cutaneous inflammation.

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H. pylori infection firstly disrupts gastrointestinal internal environment and microecological order. On the one hand, it weakens the function of impaired intestinal barrier, allowing allergens to enter the blood circulation, on the other hand, it abnormally adjusts the secretion of intestinal neurotransmitters through the neural, humoral, and immune pathways of the "gut-skin axis" transmitting these disordered signals upward. Subsequently, owing to the disruption of the local immune microenvironment of the skin, the activation of inflammatory cells and the massive release of inflammatory factors, thus amplifying skin allergic reactions. Multiple pathological mechanisms intertwine and mutually exacerbate one another, constantly interfering the body's immune homeostasis so that leading to the recurrent and persistent distinction of CU ultimately [23, 24].

4. Study on efficacy heterogeneity of CU based on different H. pylori eradication regimens

4.1. Standard triple therapy

In patients with CU, the standard triple therapy regimen—combining the proton pump inhibitor lansoprazole with two antibiotics (amoxicillin and clarithromycin)—demonstrated significantly higher efficacy compared to oral cetirizine alone in patients with CU complicated by H. pylori infection ($P < 0.05$). Clinical studies have shown that the standard triple therapy generally achieves an efficacy rate of over 80% and is relatively safe [10, 25]; it was once the top-ranking therapy for H. pylori infection. In recent years, however, due to the increasing antibiotic counteraction of H. pylori, its efficacy has gradually declined. Currently, in mainland China and parts of Europe, it has been gradually replaced by Bismuth-containing quadruple therapy.

4.2. Bismuth-containing quadruple therapy

Bismuth-containing quadruple therapy involves adding a bismuth-containing agent to the Standard Triple Therapy regimen, which can improve the eradication rate of H. pylori infection. A 1-year revisiting study of a Bismuth-containing quadruple regimen with Amoxicillin and Clarithromycin as first-class therapy for H. pylori eradication demonstrated that this regimen achieves a high H. pylori eradication rate in clinical practice, with a 1-year recurrence rate of 2.4% [26]. It is highly safe, with no reported cases of worsening clinical symptoms. However, a small number of adverse reactions still occur, affecting patient compliance. Nevertheless, with the widespread use of antibiotics, the resistance rate of H. pylori to drugs such as Amoxicillin and Clarithromycin has gradually increased; in regions with high resistance rates, the efficacy of the bismuth-containing quadruple therapy may decline significantly [27].

4.3. Acupoint catgut embedding therapy

Acupoint catgut embedding therapy namely embed absorbable catgut strings into acupoints to reach therapeutic effects through continuous stimulation of the meridians. It helps regulate organ function, promote metabolism and enhance the body's immune system. Yang et al. treated patients with H. pylori induced CU using a combination of acupuncture thread embedding and medication. This regimen boosted the cure rate of CU induced by H. pylori infection [28]. Its H. pylori eradication rate was superior to acupuncture thread embedding or medication alone, which reduced the recurrence rate and avoided the adverse effects associated with long-term medication. Xiao et al. treated patients with H. pylori induced CU using acupoint thread embedding supplemented with the *Ma Xing Shi Gan Decoction* [29]. This regimen significantly diminished the abnormally high

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expression of immune cytokines in patients, elevated basophil counts, improved immune dysfunction and inhibited the body's inflammatory response. Although thread embedding is generally safe, adverse reactions may occur. Adverse reactions such as allergic reactions to catgut strings and persistent subcutaneous indurations on patients do not resolve over time.

4.4. Sequential therapy combining traditional Chinese and Western medicine

Sequential therapy combining traditional Chinese and Western medicine refers to a treatment approach in which traditional Chinese medicine and Western medicine are systematically combined during the course of disease treatment, based on changes in the patient's condition and therapeutic needs at different stages. Cao et al. administered a four-drug regimen for H. pylori eradication for two weeks in conjunction with Western medication, followed by two weeks of treatment with *Ban Xia Xie Xin Decoction* [30]. The results showed that the efficacy of the sequential therapy group was significantly superior to that of the Western medicine group and the combined therapy group. *Ban Xia Xie Xin Decoction* originates from Zhang Zhongjing's Treatise on *Cold Damage and Miscellaneous Diseases* from the Han Dynasty. Composed of pinellia ternata, dried ginger, scutellariae radix, coptidis rhizoma, ginseng, jujube, and licorice, it functions to harmonize the liver and spleen, regulate cold and heat, and eliminate stuffiness and dissipate masses. It is primarily indicated for epigastric fullness with mixed cold and heat patterns, characterized by epigastric fullness without pain, or vomiting, borborygmi, and loose stools, with a greasy and slightly yellow tongue coating. Zhao et al. suggested that *Ban Xia Xie Xin Decoction* may directly inhibit H. pylori while simultaneously weakening the virulence of its virulence factors to exert its anti-Hp effect [31]. Results from the RT-PCR molecular biology technique showed that the mRNA expression levels of H. pylori toxic factors in vitro decreased to varying degrees as the viscosity of *Ban Xia Xie Xin Decoction* increased, indicating that the decoction downregulates the mRNA expression of H. pylori virulence factors. This sequential treatment model—firstly using medicine to rapidly control symptoms and eradicate the pathogen, followed by decoction to consolidate therapeutic effects and regulate the constitution—effectively leverages the respective therapeutic advantages of both Western and Chinese medicine, significantly improving clinical outcomes for patients with H. pylori-positive CU while reducing the recurrence rate of the disease.

5. Discussion

Although current studies have preliminarily confirmed a significant association between H. pylori infection and CU and have made some progress in the exploration of pathogenesis and the optimization of treatment, the whole research still has many limitations. Epidemiological researches on the association between H. pylori and CU are usually single center and small sample retrospective analyses, which lacking large scale and multicenter prospective cohort studies covering different countries, regions, age groups and gender globally. This result in an inaccurate reflection of the true prevalence, clinical characteristics and regional distribution of H. pylori associated CU, making it difficult to exclude the interference of confounding factors on study results.

For the treatment, different eradication treatments for the effect heterogeneity of CU is widely recognized. Notwithstanding Standard Triple Therapy and Standard Quadruple Therapy are currently the first-class treatments, H. pylori's resistance to antibiotics is yearly increasing with the widespread use of antibiotics, the seeking of new treatment methods is necessary. Although Integrated Traditional Chinese and Western medicine treatment for H. pylori associated CU has shown promising clinical prospects, it lacks unified diagnostic and therapeutic standards and

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efficacy evaluation system for Integrated Traditional Chinese and Western Medicine. Marked discrepancy exist in different studies among diagnostic classification, treatment regimens, treatment duration and efficacy assessment, making it difficult to compare and validate research findings. Furthermore, most studies only focus on clinical efficacy observations, while basic research on the effective ingredients, targets of action and pharmacological mechanisms of Chinese herbal formulas seriously behind, it is impossible to clarify the scientific content of integrated Chinese and Western medicine treatment. Additionally, there remains a lack of high-quality randomized controlled clinical trial evidence to support issues regarding the best time, mixing ratio and safety evaluations for combining acupuncture point embedding and herbal decoction of traditional Chinese therapies with Western medicine.

6. Conclusion

H. pylori infection is closely associated with CU for the occurrence, progression, and chronicity, which have become unignorable risk factors for some CU patients. Epidemiological data suggest that the prevalence of H. pylori infection in patients with CU is greatly higher than in the healthy population. Furthermore, patients infected with Cag A-positive strains, serum H. pylori IgE antibodies-positive, and IL-17/IL-23 elevated levels achieve more significant relief from symptoms after H. pylori eradication. The mechanism of H. pylori infection-induced CU is complex, referring to multiple dimensions like Th1/Th2 immune imbalance, activation of IgE-mediated mast cells, disruption of intestinal barrier function, and neuroendocrine dysregulation, which form a multisystem and multipathway pathogenic network. When it comes to treatment, the Standard Triple Therapy and Bismuth-containing Quadruple Therapy remain the core regimens for H. pylori eradication, but the matter of antibiotic resistance is becoming increasingly prominent. Integrated traditional Chinese and Western medicine treatment approaches, like acupoint thread implantation and sequential therapy combining traditional Chinese and Western medicine, can exert synergistic effects. These methods significantly improve H. pylori eradication rates and CU clinical cure rates as well as decreasing disease recurrence rates and demonstrating distinct therapeutic advantages and extensive application prospects.

Aiming at the deficiency of the current studies, in the first place, the scope of the literature search focusing primarily on Chinese and English publications from the past decade was limited, which may have introduced selection bias. Additionally, most epidemiological studies were single-center, small-sample, and retrospective designs; that's why there were significant differences in diagnostic criteria and H. pylori testing methods across studies and led to substantial heterogeneity in results. Posteriorly, indicators like Cag A, Hp-IgE, and IL-17/IL-23 have been identified for predicting treatment efficacy, yet the limited sample size, lack of external validation, and the absence of predictive models integrating multiple biomarkers restricted their clinical translational value. Besides, integrated traditional Chinese and Western medicine treatments lack rigorously designed randomized double-blind controlled trials for research on the active components and mechanisms of action for acupoint thread implantation and *Ban Xia Xie Xin decoction*, remaining largely unexplored. Revisiting periods are often less than 6 months, and data on long-term recurrence rates and the distribution of drug-resistant strains are missing. In the future, efforts should concentrate on conducting multicenter, large-sample, and forward-thinking studies with standardized diagnostic and efficacy criteria. It is necessary to clarify the molecular mechanisms of the "gut-skin axis", develop and validate personalized efficacy prediction models, utilize high-quality technologies to evaluate traditional Chinese medical therapies, and strengthen drug resistance monitoring and long-term

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follow-up. Thus, these efforts will facilitate the transition from "correlation validation" to "precision intervention".

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