Original Article

Helicobacter Pylori Infection: A Possible Predisposing Factor in Chronic Plaque-Type Psoriasis

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Background. Helicobacter pylori (H. pylori) is a common bacterial infection that is linked to disorders of the gastrointestinal tract. A possible relationship of H. pylori infection to a variety of dermatoses has been detected. Exposure to microbial infections like H. pylori is among a number of exogenous agents that might have a pathogenic role in the development of psoriasis, however, it is still a controversial subject. Objective. To evaluate the occurrence of H. pylori infection in chronic plaque-type psoriasis patients in order to determine a possible contribution in the pathogenesis of psoriasis. Patients and methods. H. pylori infection was assessed in psoriatic patients and controls by using H. pylori IgG quantitative enzyme immunoassay (ELISA test). Anti H. pylori IgG antibodies were estimated in the sera of 20 psoriatic patients without GIT symptoms and 20 healthy age and sex matched controls. Estimates were correlated with PASI score of sero-positive psoriatic patients. Results. The prevalence of H. pylori IgG seropositivity in psoriatic patients was statistically significantly higher compared with controls (67.7 ± 32.5 vs 33.9 ± 15.1, p < 0.05), and higher values were correlating with severe psoriasis. Conclusion. Based on these results, the link between H. pylori and psoriasis might be supported. We consider the pathogenic influence of H. pylori in psoriasis to be highly probable. (J Egypt Women Dermatol Soc 2010; 7: 39 - 43)

Keywords. H. pylori, gastrointestinal, psoriasis

Heliacobacter pylori (H. pylori), a microaerophilic, flagellated, curved or spiral, gram-negative bacterium, selectively colonizes the human stomach. Its infection is widespread throughout the world, and is present in about 50% of the global human population; with 80% in developing countries and 20 - 50% in industrialized countries. It is the major cause of gastritis, which plays a key role in the etiology of peptic ulcer and is a risk factor for gastric carcinoma.

In view of the excitement and interest generated by the link between H. pylori and gastric abnormalities, different investigators have thought to determine a role for the infection in a variety of non–gastrointestinal tract disorders. This is despite our current understanding that H. pylori infection is confined to gastric mucosa.

Several studies have pointed to a possible role of H. pylori infection in the pathogenesis of various extra gastric diseases involving dermatologic conditions. However, dermatologists seem to be unaware of the impact H. pylori may have on cutaneous pathology.

Among skin diseases that H. pylori has been related to is chronic urticaria. The best evidence came from studies investigating chronic urticaria in which chronic urticaria disappeared in many patients who proved to have H. pylori infection after careful eradication of the H. pylori. H. pylori was found to have a significant association with lichen planus and this supported a highly probable pathogenic role. Rosacea has often been linked with gastrointestinal disturbances. The abatement of H. pylori isolates and serology after metronidazole treatment was an evidence that suggested an etiologic relationship between rosacea and H. pylori infection. Moreover, some diseases which have been linked to H. pylori included: atopic dermatitis, Henoch-Schoenlein purpura, recurrent aphthous stomatitis, Sweet’s syndrome, Sjögren syndrome, systemic sclerosis and alopecia areata. In addition, a beneficial role of H. pylori eradication in Behçet’s disease has been reported.

Despite these observations, there have been some debates regarding the aetiopathogenic role of H. pylori in such proposed associations with various dermatologic conditions.

Psoriasis vulgaris is one of the skin diseases in which the role of H. pylori is still controversial. H. pylori infection in psoriasis has been implicated by many investigators being among a number of

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Patients and Methods

The study included 20 patients with clinically typical chronic plaque-type psoriasis without any gastrointestinal symptoms (16 males and 4 females), their age ranged from 26 - 55 years. Twenty age and sex-matched volunteers were included as a control group. A randomized blood sample was taken from the patients and controls attending the Department of Dermatology, at Ain Shams University Hospital, Egypt from March to August 2008. A written informed consent was taken from both the patients and controls.

Exclusion criteria included patients that were under 18 years, pregnant or lactating females, and those who had taken antibiotics 40 days or H2 blockers 14 days prior to sample withdrawal. Patients having peptic ulcer diseases or any GIT complaints, autoimmune and skin disorders, or previous treatment of psoriasis were excluded as well. All candidates underwent history taking with special consideration to history of psoriasis (onset, precipitating factors, course and duration of the disease). Patients were diagnosed as having chronic plaque-type psoriasis on clinical basis. Psoriasis Area and Severity Index (PASI score) was used for evaluating the severity and the extent of psoriasis.

Psoriasis Area and Severity Index (PASI score)

Psoriasis Area and Severity Index is the golden standard method for evaluating the severity and the extent of psoriasis. PASI assesses four body regions, the head, trunk, upper extremities and lower extremities. For each region, the surface area involved is graded from 0 - 6 and each of three parameters (erythema, thickness and scaliness of the plaques) are graded from 0 - 4. The scores from the regions will be summed to give a PASI score ranging from 0 - 72 (PASI < 15 for mild psoriasis, PASI ≥ 15-25 for moderate psoriasis, PASI > 25 for severe psoriasis).

Detection of serum levels of H. pylori IgG

Helicobacter pylori infection was assessed in psoriatic patients and controls by using H. pylori IgG quantitative enzyme immunoassay kit (ELISA test), having a sensitivity and specificity of 93 and 95% respectively, (BioCheck, Ink. USA). In this assay procedure, purified H. pylori antigen was coated on the surface of microwells. Diluted patients’ sera were added to the wells, and H. pylori IgG-specific antibody, if present, binds to the antigen. All unbound materials were washed away. Enzyme conjugate was added, which binds to the antibody-antigen complex. Excess enzyme conjugate was washed off and a solution of TMB reagent was added. The enzyme conjugate catalytic reaction was stopped at a specific time. The intensity of the color generated was proportional to the amount of IgG-specific antibody in the sample. The results were read by a microwell reader compared in a parallel manner with calibrator and controls.

Statistical analysis

This was done using a PC with microstat™ software program. Parametric tests were used as the obtained data proved to be normally distributed by the goodness of fitness test. Descriptive statistics of the obtained data was presented as mean and standard deviation. Difference between the means of each two groups was tested using the student t-test. Correlation study was done using linear regression analysis. p < 0.05 were considered significant.

Results

Our study included two groups; 20 patients with chronic plaque type psoriasis that had no gastrointestinal symptoms and age and sex matched 20 healthy controls. According to PASI score, there were 3 mild psoriasis patients, 7 moderate psoriasis patients and 10 severe psoriasis patients.

Psoriatic patients and controls were tested for the presence of active H. pylori infection by means of ELISA based serological test. An overall anti H. pylori IgG antibody seropositivity of 70% (n = 14) was found in psoriatic patients compared with an overall value of 40% (n = 8) for the control group. The difference in seropositivity between patients and controls was statistically significant (67.7 ± 32.5 vs 33.9 ± 15.1, p < 0.05) (Figure 1). H. pylori seropositivity values were correlated with PASI score of seropositive patients. Out of 14 seropositive psoriatic patient;10 had severe psoriasis and 4 had moderate psoriasis. Higher anti H. pylori antibody values were found in patients with severe psoriasis (Figure 2).

Figure 1. Comparison between psoriatic patients and controls as regards mean value of anti - H. pylori antibody.
DISCUSSION

Psoriasis is a common chronic, and recurrent inflammatory skin disease affecting about 2% of the general population but its prevalence varies among geographical areas and ethnic groups. Clinical observations have suggested a relationship between the exposure of a number of exogenous agents and the development or exacerbation of psoriasis\textsuperscript{35}. Various micro-organisms were found to associate the provocation and/or exacerbation of psoriasis, such as; bacteria (Staphylococcus aureus, and Streptococcus pyogenes), viruses, and fungi\textsuperscript{36,37}. One of the newly suggested bacterial aetiologies is H. pylori which happens to be one of the most common bacterial infections in the world\textsuperscript{38}.

Infection of the stomach with H. pylori induces a local immune response with infiltration of the mucosa by macrophages, neutrophils and lymphocytes. Although the innate and adaptive immune responses are activated, the bacterium is rarely eliminated and infections can last for decades if left untreated. Most infections are asymptomatic, but overt diseases can occur in 10 - 20% of infected individuals\textsuperscript{1}. Although the infection is noninvasive, it triggers a marked local inflammatory response and a systemic immune response\textsuperscript{39,40}. H. pylori infection of the stomach could conceivably produce effects elsewhere by altering levels of systemic inflammatory mediators\textsuperscript{41}.

There is evidence that H. pylori has a critical role in different extragastric diseases. The discovery of a number of other new Helicobacter species has stimulated research into different extragastric diseases, in which an infectious hypothesis is plausible\textsuperscript{42}. In the present study, sero-prevalence of anti H. pylori antibody in sera of psoriatic patients were detected. H. pylori infection was found in 70% of 20 psoriasis patients and in 40% of 20 controls, with a statistically significant difference denoting a probable significant association. In addition, patients with severe psoriasis had significantly higher sero-positive values which also implied an influential potential on the clinical severity.

These findings are consistent with several studies that were carried out regarding the association of H. pylori with psoriasis. Several authors have reported significant anti H. pylori antibody values in sera of patients with psoriasis\textsuperscript{37,32,42,43}. Moreover, Qayoom and Ahmad\textsuperscript{44} reported sero-positivity in 20 psoriatic patients (40%) and 5 controls (10%) and supported a causal role of H. pylori in the pathogenesis of psoriasis. Furthermore, an interesting finding was noted by Hübner and Tenbaum\textsuperscript{45} who reported complete remission of palmo-plantar psoriasis through H. pylori eradication therapy. Other studies\textsuperscript{46,47,48} have also confirmed the beneficial effect of H. pylori testing and eradication in many dermatologic diseases. While different infections have been implicated in the causation of psoriasis, the concept of H. pylori infection as a possible causative factor is relatively a new one. What makes this association worthy of investigation is the chronic and asymptomatic nature of H. pylori infection and its highly endemic nature. How H. pylori can cause psoriasis is not known with certainty as the cutaneous pathology of H. pylori is far from being clear.

The postulate role of H. pylori in the pathogenesis of extraintestinal manifestations is based on the fact that local inflammation has systemic effects. The bacterium colonizes gastric mucosa and induces a strong inflammatory response with release of various bacterial and host-dependent cytotoxic substances. Gastric H. pylori infection is a chronic process lasting for decades, and persistent infection induces a chronic inflammatory and immune response able to induce lesions both locally and remote to the primary site of infection. The systemic effects may involve increased mucosal permeability to alimentary antigens, immunomodulation, an autoimmune mechanism or the impairment of vascular integrity\textsuperscript{43}. The various immunopathogenesis during the H. pylori-caused diseases might be due to an unbalanced Th1 or Th2 mediated response post infection\textsuperscript{11,43}. Superantigen activation of T-cells may be a supplementary piece of the puzzle, as H. pylori secretes enterotoxins (T-cell activation toxins) that bind to a T-cell receptor and induce the expression of a skin homing receptor and cutaneous lymphocytic antigen (CLA) on T-cells\textsuperscript{47}.

Our hypothesis is that H. pylori could be at least the provoking factor behind psoriasis, particularly as a chronic infecting agent maintaining psoriasis. However, the etiology of psoriasis is probably multifactorial and the possibility still remains that H. pylori may trigger psoriasis in certain susceptible individuals. An autoimmune mechanism has been suggested in psoriasis and H. pylori has been associated with autoimmune processes in susceptible patients. The present study showed the evidently higher prevalence of H. pylori in psoriatic patients than those found in controls.

Conclusion

Based on these results, we consider the pathogenic influence of H. pylori in psoriatic patients to be
highly probable. A large scale studies and further investigation for the eradication of *H. pylori* in psoriatic patients with *H. pylori* sero-positivity is required for a definite confirmation.

**REFERENCES**


