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The potential role of *Helicobacter pylori* infection in the pathogenesis and exacerbation of psoriasis, with emphasis on E-selectin levels and specific histochemical features; a cross-sectional study

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Abstract

Introduction: Several studies have suggested a potential link between *Helicobacter pylori* infection and psoriasis; however, the underlying mechanisms remain unclear. One proposed pathway is mediated through endothelial activation and inflammatory responses, potentially indicated by changes in E-selectin expression and related histochemical features.

Objectives: To assess the role of *H. pylori* infection in the expression of E-selectin in psoriasis patients and to evaluate related histological and immunohistochemical changes, aiming to provide insights into psoriasis pathogenesis and potential therapeutic targets.

Patients and Methods: A cross-sectional study was conducted on 93 patients with psoriasis, who were evaluated using the Psoriasis Area and Severity Index (PASI), *H. pylori* IgG antibodies, serum E-selectin levels, histopathological examination, special staining (Masson trichrome), and Ki-67 immunostaining. Patients were categorized into *H. pylori*-positive (n = 35) and *H. pylori*-negative (n = 58) groups.

Results: *H. pylori*-positive patients had significantly higher PASI scores (p = 0.007) and elevated E-selectin levels, particularly in severe cases ($P < 0.001$). Masson trichrome staining showed increased collagen in *H. pylori*-positive patients (64.3 ± 5%) compared to negative group (53.1 ± 2%), though not statistically significant ($P = 0.152$). Additionally, Ki-67 expression was significantly lower in *H. pylori*-positive individuals (0.51 ± 0.01%) than in *H. pylori*-negative ones (0.92 ± 0.01%, $P = 0.04$).

Conclusion: *H. pylori* infection may contribute to the development and exacerbation of psoriasis by promoting endothelial activation and inflammation, suggesting its potential role in disease pathogenesis.

Keywords: E-selectin, *H. pylori*, Histochemistry, Psoriasis

Introduction

Psoriasis is a skin autoimmune disorder characterized by persistent proliferation and inflammation. It is represented by characteristic erythematous plaques capped by silvery scales, especially on the extensor surfaces, scalp, and lumbosacral area (1,2). The eyes, joints, and other systems may also be impacted by the disease. Psoriasis waxes and wanes with flare-ups and has no complete cure. It is a global disease whose prevalence varies from 0.2% to 4.8% (3,4). Many patients with psoriasis acquire depression due to their low quality of life. The disease has a multifactorial, complex, and incompletely understood etiology, including an interaction

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Key point

- *H. pylori*-positive psoriasis patients had significantly higher PASI scores ($P = 0.007$), indicating more severe disease.
- Serum E-selectin levels were significantly elevated in patients with severe psoriasis ($P < 0.001$). suggesting endothelial activation.
- Ki-67 expression was significantly reduced in *H. pylori*-positive patients ($P = 0.04$), indicating lower cellular proliferation.
- Our study suggests *H. pylori* may play a role in psoriasis pathogenesis through inflammation and endothelial activation.

of genetic susceptibility and environmental triggers (3,5).

One of the characteristic features of psoriasis is the aberrant activation of T-cells, which results in the excessive production of inflammatory cytokines. Endothelial cells play a vital role in inflammation by producing adhesion molecules that enhance the attraction of leukocytes to regions of tissue damage or infection. E-selectin is a major type of adhesion molecule that is surface-expressed by endothelial cells in response to inflammatory cytokines (6,7).

Elevated E-selectin levels have been found in psoriasis skin lesions and patient serum, indicating that endothelial activation and leukocyte recruitment play essential roles in the disease's etiology. E-selectin's elevated expression in psoriasis suggests that it may serve as a biomarker for disease severity and a possible therapeutic target (8). Studies on other chronic skin diseases have shown that E-selectin is expressed on vascular endothelial cells in atopic dermatitis lesions, contributing to the recruitment of inflammatory cells. E-selectin also plays phase-dependent roles in chronic contact hypersensitivity responses, influencing the severity and progression of the condition (9).

Helicobacter pylori bacteria colonize stomach mucosa contributing to peptic ulcer disease, chronic gastritis, and various extra-gastric systemic complications. Emerging evidence suggests an association between *H. pylori* infection and multiple extra-gastrointestinal conditions, including cardiovascular pathologies, immune-mediated disorders, and dermatological conditions such as psoriasis (10).

Several studies have also suggested that *H. pylori* infections play a role in pathogenesis of other skin diseases, such as immune thrombocytopenic purpura and chronic urticaria (11). However, a recent study found that patients with psoriasis had higher rates of *H. pylori* infection, but the infection did not affect psoriasis severity (12).

The cascade caused by *H. pylori* is posited to exacerbate psoriatic lesions (2,13); elevated levels of E-selectin may serve as another mechanism through which *H. pylori* can exacerbate psoriasis, and some studies have demonstrated that *H. pylori* can induce the expression of E-selectin on endothelial cells. This up-regulation facilitates the adhesion and migration of leukocytes to the site of infection, playing a role in inflammatory response (14).

The association between *H. pylori* affection and psoriasis severity remains controversial, with some studies suggesting a significant correlation, while others report no clear link, highlighting the need for further investigation (12,15). Similarly, E-selectin has been implicated in psoriasis pathogenesis, but its precise role and correlation with disease severity require further elucidation (16).

Objectives

This study aims to investigate the relationship between *H. pylori* infection, E-selectin expression, and disease severity in patients with psoriasis, hypothesizing that *H. pylori* infection may exacerbate psoriasis by increasing E-selectin expression and promoting an inflammatory cascade in psoriasis.

Patients and Methods

Study design

This cross-sectional study included 93 patients, aged 10–60 years, who were referred to dermatology clinics at the Faculty of Medicine, Al-Azhar University, Cairo, Egypt, between June 2023 and August 2024.

Inclusion and exclusion criteria

All patients were diagnosed with psoriasis based on both clinical and histopathological criteria during the study period were included. Exclusion criteria included patients who had received antibiotics, proton pump inhibitors, or H2 receptor antagonists within the preceding month; those who had undergone phototherapy or local and/or systemic anti-psoriatic treatment; those for whom the required blood tests could not be performed or paraffin blocks were unavailable; pregnant individuals; smokers; and patients with hypertension, diabetes mellitus, acute or chronic organ failure, local or systemic infections, other autoimmune disorders, or malignancy.

Data collection

Following enrollment based on the aforementioned criteria, data were collected prospectively using a standardized data collection form to ensure uniformity. Each participant underwent a detailed clinical assessment, relevant laboratory investigations, and histopathological examination. The collected data included demographic information (age and sex), clinical features (type and severity of psoriasis), laboratory parameters (serum *H. pylori* IgG and E-selectin levels), and histological features, including Ki-67 immunohistochemical staining. Clinical and laboratory data were extracted directly from patient records and laboratory reports. Histopathological evaluations were performed by researchers specializing in histology, pathology, and dermatopathology—both onsite in Cairo and remotely by collaborating researchers from Egypt and Saudi Arabia. All data were collected and entered by trained clinicians and pathology staff under the supervision of senior investigators to ensure accuracy and consistency.

Clinical and laboratory assessment of the candidates

- Comprehensive medical and medicinal history taking.
- Local and systemic medical examination.

Psoriasis diagnosis

The clinical morphology and lesion site are used to make the diagnosis. Histopathology aids in the diagnosing process and distinguishes psoriasis from other dermatoses.

Psoriasis severity assessment

The severity of psoriasis in patients was assessed using the Psoriasis Area and Severity Index (PASI), which evaluates the severity of lesions based on the area covered, erythema, scaling, and thickness. PASI scores range from 0 to 72, with higher scores indicating more severe disease (17,18). Plaque psoriasis has three severity levels; PASI mild (<5), moderate (5–10), and severe (>10) (5).

H. pylori antibody detection

The serum was extracted from the blood and kept at -20 °C until analyzed for all assays. Serum IgG antibodies against *H. pylori* were assessed using a microplate enzyme immunoassay (EIA) and a specialized antibody detection kit (E-Plate Eiken *H. pylori* antibody, Eiken Chemical Co., Ltd., Tokyo, Japan). All samples were analyzed following the manufacturer's guidelines, with a cutoff value set at 10 U/mL (19).

E-Selectin measurement

E-selectin levels were determined using a commercially available enzyme-linked immunosorbent assay (ELISA) kit [Aviva's human E-Selectin ELISA kit (Orgenium, Vantaa, Finland)], based on the standard sandwich ELISA technique. Human E-selectin-specific polyclonal antibodies were pre-coated onto 96-well plates. According to the manufacturer, the intra-assay and inter-assay coefficients of variation were both below 10% for all assays.

Histopathology study

Biopsies were taken from the lesions, fixed in formalin 10% solution, and subsequently paraffin blocks were made. Hematoxylin and eosin (H&E) stained slides were examined by at least three pathologists (onsite and via remote consultation with pathologists abroad) to confirm the diagnosis of psoriasis according to the histological criteria (parakeratosis, psoriasiform hyperplasia), polymorphonuclear leukocytes and lymphocytes infiltrates within the dermis and epidermis and no granular layer. Masson trichrome stain, and periodic acid Schiff (PAS) reaction, were conducted to stain sections (5-6 µm thickness) (20).

Ki-67 immunostaining

For the extraction of the antigen, dewaxed and rehydrated sections (4 µm thick) were heated up in a 10 mmol/L citrate buffer. Adding 3% hydrogen peroxide reduced the body's endogenous peroxide action. Following that, slices were treated with antibody panel included Ki-67 (1:100 dilution; Thermo Fisher Scientific, Fremont, CA, USA). Ki-67 expression was evaluated with respect to intensity (mild, moderate, or strong. Then, slices were treated with an antibody panel, including Ki-67 (1:100 dilution; Thermo Fisher Scientific, Fremont, CA, USA). Accordingly, Ki-67 expression was assessed based on intensity (mild, moderate, or strong) (21).

Outcomes

The primary outcome was to assess the difference in serum E-selectin levels between *H. pylori*-positive and *H. pylori*-negative psoriasis patients. Secondary outcomes included;

- Comparison of PASI scores between the studied groups.
- Histopathological differences, particularly collagen deposition assessed by Masson trichrome staining.
- Evaluation of Ki-67 expression as a marker of epidermal proliferation.
- Correlation between *H. pylori* seropositivity and severity of psoriasis or histological markers.

Statistical analysis

For statistical analysis, the Image-Pro PLUS software (version 4.5; Media Cybernetics, Silver Spring, MD, USA) was utilized for image processing. Data was analyzed using the Statistical Package for the Social Sciences (SPSS) software (version 16; SPSS Inc., Chicago, IL, USA). Quantitative variables were presented as mean and standard deviation, while qualitative variables were expressed as frequencies. Statistical comparisons included Student's T-test for assessing differences between two quantitative groups, one-way analysis of variance (ANOVA) for comparisons among more than two groups, Tukey's HSD is a post hoc test conducted to compare the mean of each sample to the mean of each other sample, the Pearson's correlation test to examine relationships between variables, and the chi-square test for analyzing categorical data. Univariate regression analysis and multivariate regression analysis were used to evaluate the correlation between the PASI score and other parameters. P value < 0.05 was considered statistically significant.

Results

Of 93 participants in the study, 8 had mild psoriasis, 68 had moderate psoriasis, and 17 had severe forms of the illness. In total, 35/93 (37.6%) of our patients with psoriasis were positive for *H. pylori* IgG antibody titers. Table 1 compares demographic factors such as age and sex between *H. pylori*-positive and -negative patients with psoriasis.

Table 2 shows a comparison of key clinical characteristics between *H. pylori*-positive and negative patients with psoriasis. There was no significant statistical difference between the two groups in terms of disease duration,

Table 1. Comparison of demographic variables across patients with and without *H. pylori* and psoriasis

		H. pylori IgG		Significance test	*P* value
		Negative (n = 58)	Positive (n = 35)		
Age (y) (Mean ± SD)		33.44 ± 19.42	37.81 ± 17.19	t= 1.097	0.276
Sex	Male	30 (51.7%)	18 (51.5%)	χ^2 = 0.0008	0.978
	Female	28 (48.3%)	17 (48.5%)		
Occupation	Employed	32 (55.3%)	24 (68.5%)	χ^2 = 2.0137	0.570
	Unemployed	6 (10.3%)	3 (8.5%)		
	Student	10 (17.2%)	3 (8.5%)		
	Housewife	10 (17.2%)	5 (8.5%)		
BMI (kg/m^2)		24.48 ± 4.91	23.15 ± 7.82	t= -1.009	0.316

BMI, body mass index.

Table 2. Comparison of clinical variables across *H. pylori*-positive and negative patients with psoriasis

		H. pylori IgG		Significance test	*P* value
		Negative (n = 58)	Positive (n = 35)		
Duration of illness (y) (Mean ± SD)		4.12 ± 3.15	3.98 ± 4.89		
Family history	Yes	15 (26%)	8 (22.8%)	χ^2 = 0.1059	0.745
	No	43 (74%)	27 (77.2%)		
Affected sites*	Upper limbs	39 (67.2%)	26 (74.3%)	χ^2 = 0.5777	0.965
	Lower limbs	42 (72.4%)	27 (77.2%)		
	Head	32 (55.2%)	18 (51.4%)		
	Trunk	29 (50%)	17 (48.6%)		
	Nail	21 (36.2%)	16 (45.7%)		
Arthritis	Yes	3 (5.2%)	1 (2.8%)	χ^2 = 0.2843	0.594
	No	55 (94.8%)	34 (97.2%)		
PASI	Mild	5 (8.6%)	3 (8.5%)	χ^2 = 9.8245	0.007
	Moderate	48 (82.8)	20 (57.2%)		
	Severe	5 (8.6%)	12 (34.3%)		

PASI, Psoriasis area severity index, *Multiple sites can be affected.

positive family history, anatomical distribution, or arthritis. On the other hand, patients with *H. pylori* had significantly higher PASI scores (P=0.007).

Table 3 compares E-selectin levels across varying degrees of psoriasis severity and shows that individuals with severe psoriasis had significantly greater levels of E-selectin. Moreover, a post hoc test employed to compare the mean of each severity category to the mean of each other severity category and find significant variation between each pair of them regarding E-selectin level.

As displayed in Table 4, The PASI score was significantly correlated with E-selectin level and *H. pylori* IgG titer with univariate regression analysis, and correlated with E-selectin level, *H. pylori* IgG titer, sex, and arthritis with multivariate regression analysis

The following clinical and laboratory outcome metrics demonstrated significant correlations: PASI scores and E-selectin levels (r=0.287; P = 0.005; Figure 1), *H. pylori* IgG titers, and E-selectin levels (r=0.275; P=0.008; Figure 2), as well as PASI scores and *H. pylori* IgG titers (Figure 3).

Hematoxylin and eosin-stained skin biopsies displayed that patients with *H. pylori* had: i) regular epidermal hyperplasia with parakeratosis; ii) epidermal hyperproliferation and elongation of the rete ridges; iii) vasodilation of the superficial vasculature; iv) dense dermal inflammatory and lymphomonocytic infiltrate; v) supra-papillary thinning, dilated blood vessels in the papillary dermis, supra-basilar mitoses, and intra-epidermal cellular

Table 3. Comparison of E-selectin level across variable degrees of severity of psoriasis

		E-selectin (ng/mL) (Mean ± SD)	Significance test	*P* value
PASI	A-Mild (n = 8)	40.57±30.31	F= 10.795	<0.001*
	B- Moderate (n = 68)	72.45±29.08		
	C-Severe (n = 17)	98.15±31.75		
PASI pair		**Tukey HSD Q statistic**		***P* value**
	A vs B	4.0666		0.014
	A vs C	6.4024		0.001
	B vs C	4.5177		0.005

Table 4. Univariate and multivariate regression analysis of PASI score

	Univariate		Multivariate	
	r	*P* value	β	*P* value
E-selectin (ng/mL)	0.397	<0.001	0.299	0.004
H pylori IgG titer (U/mL)	0.328	<0.001	0.411	<0.001
BMI (kg/m²)	-0.032	0.377	0.062	0.471
Age of onset (years)	0.171	0.963	-0.086	0.411
Duration of disease (years)	0.128	0.871	0.042	0.801
Sex	0.082	0.071	0.291	0.044
Family history	0.062	0.064	0.141	0.089
Arthritis	0.068	0.082	0.253	0.049

Abbreviations: r, correlation coefficient; β, standardized partial regression coefficient;

infiltration (Figure 4). Biopsies from patients without *H. pylori* showed similar histological alterations; however, inflammatory processes were less pronounced, and overall skin architecture appeared better preserved.

Masson's trichrome staining revealed increased collagen fiber formation in the epidermis of patients with *H. pylori*. These collagen fibers were more disordered and unevenly distributed compared to those in patients without *H. pylori* (Figure 5). Although collagen levels increased from 53.1 ± 2% in patients without *H. pylori* to 64.3 ± 5% in *H. pylori*-positive individuals, the difference was not statistically significant ($P = 0.152$).

Periodic acid–Schiff (PAS) staining of patient specimens showed an increase in PAS-positive components, including glycoproteins, proteoglycans, and mucopolysaccharides, which contribute to the thickening of the skin's basement membrane and cell membranes. These changes were less pronounced in specimens from Patients without *H. pylori* (Figure 5). The PAS-positive reaction rate decreased from 51 ± 3% in specimens from

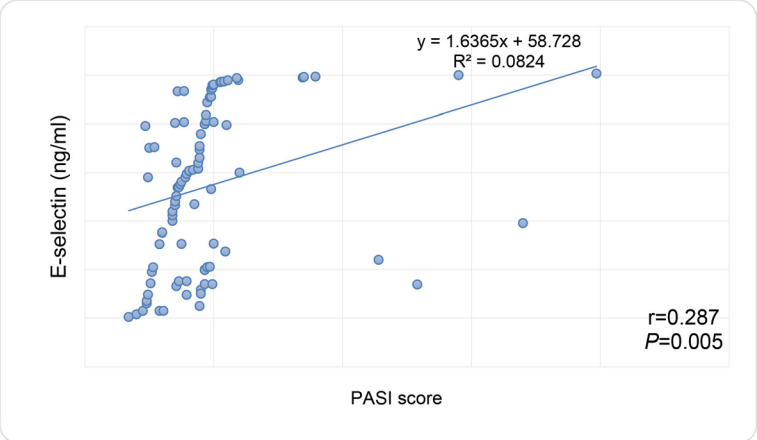

Figure 1. Correlation between PASI scores and E-selectin levels.

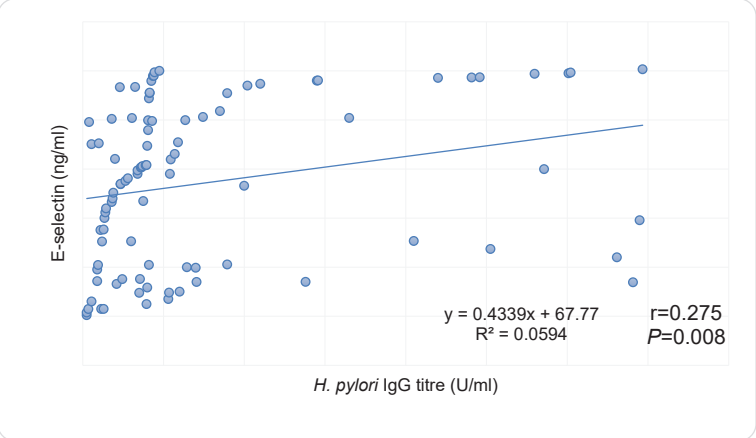

Figure 2. Correlation between *H. pylori* IgG titers and E-selectin levels.

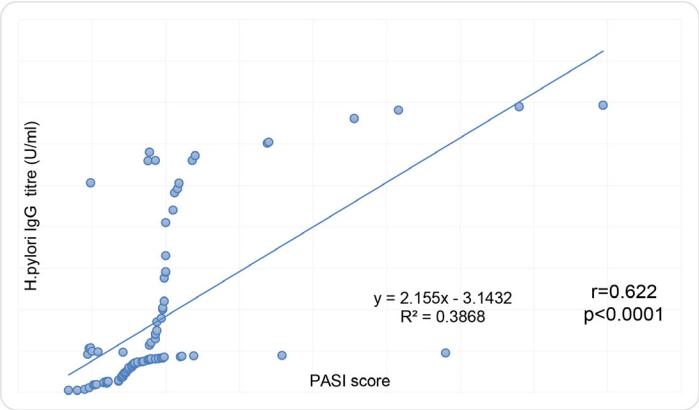

Figure 3. Correlation between PASI scores and H. pylori IgG titers.

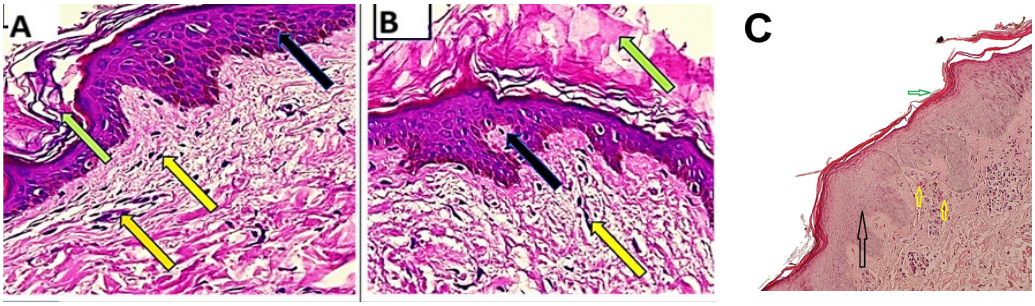

Figure 4. A case of psoriasis (A-C) shows epidermal hyperplasia (black arrows), regular epidermal hyperplasia with parakeratosis (green arrow, superficial), and vasodilation of the superficial vasculature accompanied by a lympho-monocytic dermal infiltrate (yellow arrow, dermal) (H&E, ×400 original magnification of A&B and 200x of C).

patients with *H. pylori* to 39 ± 3% in those from patients without *H. pylori*. Immunohistochemistry revealed an increase in Ki-67 expression. This expression was more prominent in *H. pylori*-positive specimens than in *H. pylori*-negative specimens, indicating increased keratinocyte cell proliferation and differentiation. Ki-67 immunohistochemistry expression dropped considerably from 0.92 ± 0.01% in *H. pylori*-negative to 0.51 ± 0.01% in *H. pylori*-positive individuals ($P = 0.04$; Figure 6).

Discussion

This study aimed to investigate the relationship between *H. pylori* infection and E-selectin expression in patients with psoriasis from both clinical and histopathological perspectives.

Our results showed that 37.6% (35/93) of patients with psoriasis tested positive for *H. pylori* IgG antibodies. Additionally, patients with *H. pylori* infection had significantly higher PASI scores. Serum E-selectin levels were also markedly elevated in patients with higher PASI scores.

Correlation analysis revealed significant positive associations between E-selectin levels, PASI scores, and *H. pylori* infection status. Furthermore, PASI scores were significantly correlated with *H. pylori* IgG titers. Histopathologically, biopsies from *H. pylori*-positive patients exhibited more pronounced inflammatory changes, including increased collagen deposition, basement membrane thickening, and enhanced cellular proliferation.

These findings suggest that *H. pylori* infection may contribute to psoriasis exacerbation by promoting endothelial activation and inflammation, as evidenced by the observed correlations. The histopathological alterations further support this hypothesis.

Several studies have reported that mean PASI scores are significantly higher in patients with psoriasis who are *H. pylori*-positive compared to those who are *H. pylori*-negative (22-25). For example, Mesquita et al. found that psoriasis patients with *H. pylori* infection had significantly higher PASI scores than those without the infection (25). Similarly, Fathy et al, used ELISA to screen for *H. pylori* in 20 patients with psoriasis and 20 age- and gender-matched healthy controls. Their results showed that seropositivity was significantly more prevalent among patients with psoriasis, and—consistent with our study—higher antibody titers correlated with greater disease severity (23).

Another important study by Su et al evaluated 300 patients with psoriasis and 150 healthy individuals to assess the prevalence of *H. pylori* seropositivity, its relationship with PASI scores, and its impact on treatment response.

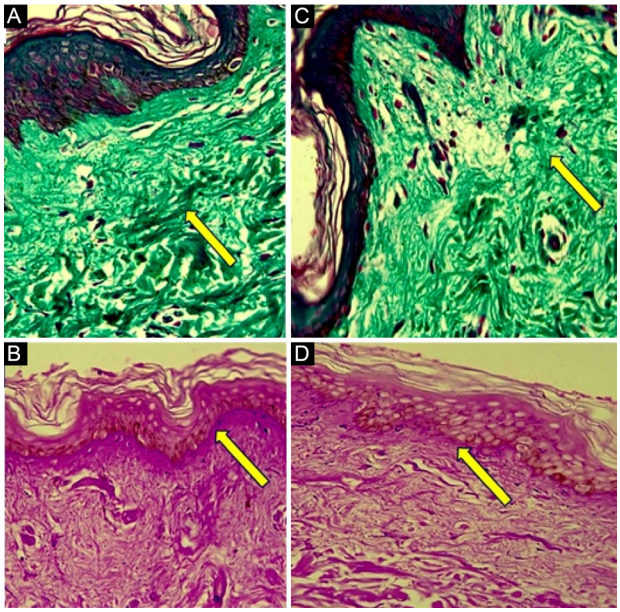

Figure 5. Specimens from patients with *H. pylori* show increased collagen production with fragmented and disorganized collagen fibers (A: yellow arrow, Masson's trichrome stain), as well as strong PAS-positive material and thickened basement membranes (B: yellow arrow). Specimens from patients without *H. pylori* display a lower amount and more organized distribution of collagen fibers (C: yellow arrow, Masson's trichrome stain) and weak PAS-positive material with minimal basement membrane thickening (D: yellow arrow).

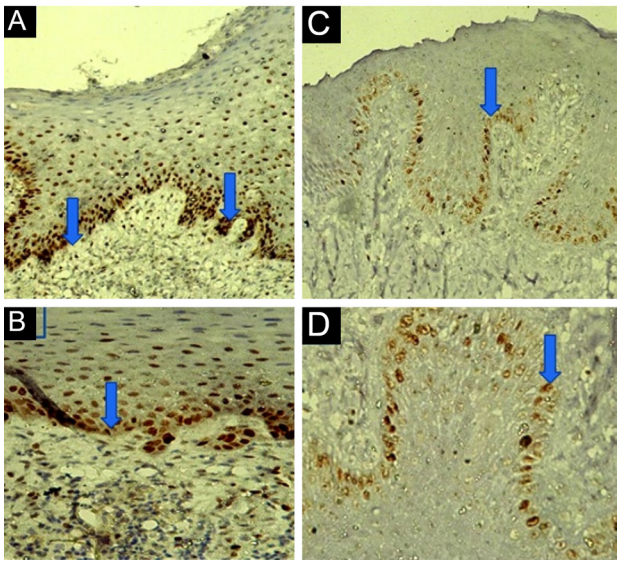

Figure 6. Microscopic images of Ki-67–stained tissue samples from psoriatic lesions reveal varying levels of Ki-67 expression, indicated by brownish-colored cells (blue arrows). Samples from patients with a positive *H. pylori* titer (A & B) show increased Ki-67 expression in rapidly dividing cells, whereas those with a negative *H. pylori* titer (C & D) exhibit lower levels of Ki-67 expression (brownish-colored cells, blue arrows).

Their findings also demonstrated significantly higher PASI scores in *H. pylori*-positive patients compared to *H. pylori*-negative ones (25). However, some studies have reported no statistically significant difference in PASI scores between *H. pylori*-positive and -negative psoriasis patients (12,26).

E-selectin, an endothelial adhesion molecule, plays a crucial role in leukocyte recruitment during inflammation. Elevated E-selectin levels have been reported in various inflammatory conditions, including psoriasis. Our findings are in line with these reports, demonstrating higher serum E-selectin levels in patients with more severe psoriasis, as reflected by their PASI scores. This supports previous studies reporting a positive correlation between E-selectin levels and disease severity, suggesting that E-selectin may serve as a biomarker for disease activity (27–29). However, some investigators have found no significant correlation between PASI scores and serum E-selectin levels (30).

Our observation of increased E-selectin levels in psoriasis patients with positive *H. pylori* IgG titers further supports the hypothesis that *H. pylori*-induced systemic inflammation may exacerbate endothelial activation (31).

To our knowledge, no previously published studies have correlated histopathological changes in psoriatic skin biopsies with *H. pylori* IgG titers, as our study has conducted.

Although the exact mechanisms remain unclear, *H. pylori* infection may contribute to psoriasis by triggering an aggressive immune response and increasing the production of pro-inflammatory cytokines such as TNF-α (tumor necrosis factor alpha), interleukin (IL)-1 and IL-6. These cytokines circulate systemically and may exacerbate psoriatic inflammation. Furthermore, *H. pylori* infection can directly influence endothelial cells by increasing the expression of adhesion molecules such as E-selectin, thereby facilitating leukocyte migration to inflamed regions (32,33).

Some studies suggest that *H. pylori* infection may be associated with metabolic disorders and obesity by influencing ghrelin and leptin—hormones that regulate appetite and metabolism—which could, in turn, affect body mass index (BMI). However, *H. pylori* may also contribute to weight loss in some individuals due to its effects on gastric inflammation and reduced appetite (34). Increased E-selectin levels have been linked to higher BMI, as obesity induces chronic inflammation and endothelial dysfunction (35).

The relationship between BMI and *H. pylori* infection remains controversial, with some studies suggesting a potential association, while others report conflicting results (34,36). In our study, we did not find a significant correlation between BMI and *H. pylori* infection, which aligns with certain reports indicating that factors such as dietary habits and genetic predisposition may play a more critical role in this relationship. Further research is needed to clarify the potential mechanisms linking BMI and *H. pylori* infection, particularly in different populations

Several studies have explored the potential impact of *H. pylori* eradication on psoriasis severity, yielding mixed results. Campanati et al (37) conducted a study

involving 210 patients with psoriasis and 150 healthy controls, finding that while the prevalence of *H. pylori* infection was similar between groups; infected patients exhibited more severe psoriasis. Interestingly, patients who successfully underwent *H. pylori* eradication therapy showed a significant improvement in their PASI scores compared to those who did not receive eradication treatment. Conversely, another study reported that *H. pylori* eradication had no impact on psoriasis and found no definitive evidence linking *H. pylori* infection to psoriasis (38).

Given these conflicting findings, the relationship between *H. pylori* eradication and psoriasis severity remains inconclusive. Further large-scale, randomized controlled trials are necessary to elucidate the potential therapeutic benefits of *H. pylori* eradication in psoriasis management.

Our findings have important clinical implications for the management of psoriasis. First, screening for and treating *H. pylori* infection in patients with psoriasis could potentially alleviate disease severity.

Second, targeting E-selectin and other endothelial adhesion molecules may offer a novel therapeutic approach for psoriasis. Given the significant role of E-selectin in leukocyte recruitment and inflammation, therapies aimed at reducing E-selectin expression or blocking its interaction with leukocytes could help mitigate the inflammatory response in psoriasis.

Psoriasis pathogenesis is influenced by genetic, environmental, and immunological factors, which may vary across different ethnic and geographic populations. Although research indicates that the clinical features of psoriasis in Egyptian patients are generally similar to those observed in other ethnic groups, some minor differences exist (39, 40). Regional factors such as diet, microbiome composition, and genetic predispositions may play a role in disease expression (40,41).

Occupational factors may play a role in psoriasis severity, particularly in individuals exposed to environmental irritants, chemical agents, or high levels of psychological stress. Studies have suggested that physically demanding jobs, frequent hand washing, and prolonged contact with irritants may exacerbate psoriatic lesions (42,43). In our study, we collected occupation data but one of the limitations of this study, we did not perform a subgroup analysis to determine its specific impact on disease severity. Future studies with a larger sample size and targeted occupational assessments are needed to better understand this relationship.

Conclusion

In summary, this study highlights a potential association between *H. pylori* infection and elevated E-selectin expression in patients with psoriasis. The findings suggest that *H. pylori* infection may contribute to the pathogenesis and exacerbation of psoriasis by promoting endothelial activation and systemic inflammation. Future research should aim to validate these findings through large-scale studies and clinical trials, with particular attention to their potential therapeutic implications.

Limitations of the study

This study has several limitations. First, as cross-sectional study, it cannot establish causality, and longitudinal studies are needed to evaluate the effects of *H. pylori* eradication on psoriasis progression. Second, the study relied on serological detection of *H. pylori*, which does not distinguish between active and past infections. More sensitive diagnostic methods, such as urea breath tests or stool antigen tests, could offer a more accurate assessment of *H. pylori* infection status.

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Conflicts of interest

The authors declare that they have no competing interests

Ethical issues

The research conducted in this study adhered to the principles

outlined in the Declaration of Helsinki and was approved by the Ethics Committee of bioethics committee of Al-Azhar Faculty of Medicine, Cairo, Egypt (Ethical code#His._395Med. Research_00000147. Date: 2 Feb. 2024). All patients provided the consent form before admission to the hospital or any intervention or biopsy taking. The authors have fully complied with ethical issues, such as plagiarism, data fabrication, and double publication.

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