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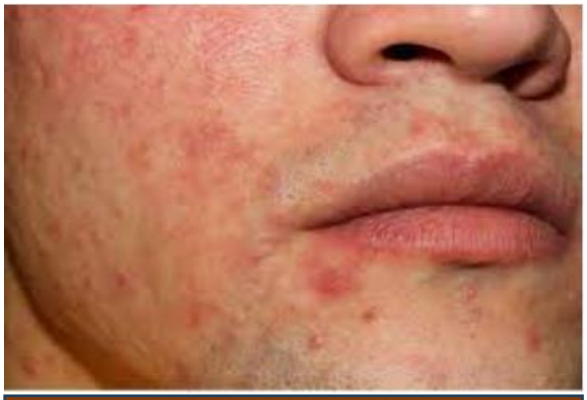
ASSOCIATION BETWEEN HELICOBACTER PYLORI INFECTION AND SEVERITY OF ACNE VULGARIS

Introduction:



ACNE VULGARIS

Acne vulgaris is a chronic inflammatory skin condition that affects approximately 9.4% of the global population, with over 85% of adolescents experiencing it. This condition can persist into adulthood, particularly among females, leading to significant psychological and social consequences. Clinically, acne presents as non-inflammatory lesions (blackheads and whiteheads) and inflammatory lesions (papules, pustules, nodules, and cysts), often resulting in scarring and hyperpigmentation.[1]

Helicobacter pylori (H. pylori) is a Gram-negative, spiral-shaped microaerophilic bacterium primarily implicated in various gastric and extra-gastric diseases. The global prevalence of H. pylori infection is approximately 50%, with significant regional variation influenced by socio-economic factors. Recent literature has begun to explore the association between H. pylori and acne vulgaris (AV), particularly in more severe cases, in addition to its established links to conditions such as chronic urticaria, psoriasis, and rosacea. However, studies on the impact of H. pylori infection across different severities of AV are limited.[2] So, this study aimed to explore the association of H. pylori infection among AV patients and correlate it with the severity of the disease.

Methods:

This case-control study included 45 patients with AV and 45 age- and sex-matched healthy individuals as controls. Comprehensive history-taking encompassed personal, drug, and family histories, along with a focused assessment of the present illness. Clinical examinations involved general and dermatological assessments to classify AV types and severity using the Global Acne Grading System (GAGS). Laboratory investigations included H. pylori antigen detection in stool and serum H. pylori IgG antibody measurement via ELISA kits. Stool samples (1–2 mL or 1-2 g) were collected and stored at -20°C or 2–8°C for immediate testing, while blood samples (1 mL) for IgG detection were refrigerated at 2–8°C for up to 7 days. The

assay procedures employed monoclonal antibody-based ELISA for stool antigen detection and dilutions for serum IgG measurement, with optical density measured at 450 nm for interpretation. Data analysis was performed using IBM SPSS (v22.0), utilizing appropriate statistical tests to assess significance, with a p-value of ≤0.05 considered statistically significant.

Results:

The study comprised 45 patients with AV and 45 matched controls, revealing a sex distribution of 58 females (64.4%) and 32 males (35.6%), with a mean age of 18.7 years. The duration of AV ranged from 7 to 144 months, with a median of 36 months, and 39 patients (86.6%) had a family history of AV. Types of AV included inflammatory (26.7%), comedonal (13.3%), and mixed (60.0%). Severity assessment using the GAGS classified AV as mild in 35.6%, moderate in 35.6%, and severe in 28.8% of patients.

Laboratory findings indicated that 38.9% of participants tested positive for H. pylori stool antigen and 45.6% for serum IgG antibodies. A significant difference was noted in positive stool antigen rates between AV patients and controls ($P < 0.001$), as well as in serum antibodies ($P = 0.006$) (Table 1). The stool antigen positivity was significantly associated with acne severity ($P < 0.001$), and patients with severe AV exhibited a significantly higher rate of serum IgG antibodies (92.3%) compared to healthy controls (31.1%, $P = 0.001$) and mild AV patients (31.3%, $P = 0.007$). No significant differences were found between mild and moderate AV or between other comparative groups.

Helicobacter Pylori Ag in stool					
Negative/ Positive	**Control**		**Case**		**P value**
	Count	**Percent**	**Count**	**Percent**	
Negative	39	80%	19	42.2%	<0.001
Positive	9	20%	26	57.8%	
Helicobacter pylori Ab in serum					
Negative	31	68%	18	40%	0. 006
Positive	14	31.1%	27	60%	

Table 1: Positive Helicobacter pylori Antigen in Stool and Antibody in Serum Among Case and Control Groups

Discussion:

The current study found a significant association between H. pylori infection and the severity of AV. H. pylori antigens were detected in 57.8% of AV patients, and IgG antibodies were present in 60%, both significantly higher than in healthy controls. Notably, patients with severe AV showed markedly elevated rates of both stool antigens and serum antibodies, aligning with previous findings by Khodaeinai et al. and Saleh et al. which also reported a correlation between H. pylori infection and increased acne severity.[3] This study's findings were consistent with those of Saleh et al., which showed that patients with severe AV had significantly higher levels of fecal H. pylori antigen compared to those with mild or moderate AV and healthy controls.[4]

Conclusion:

This study demonstrated a high prevalence of H. pylori infection in patients with AV, suggesting its potential role in the pathogenesis of the condition. Patients with severe AV exhibited significantly higher levels of H. pylori antigens in stool and antibodies in serum compared to those with mild AV and healthy controls.

H. pylori infection is prevalent in acne vulgaris, particularly in severe cases, suggesting its potential role in exacerbating the condition.

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3. Khodeiani E, Halimi M, Hagigi A. Severe acne vulgaris is associated with helicobacter pylori infection: first report in the literature. J Med Sci. 2014; 14:92–96.
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ASSESSMENT OF RISK FACTORS AND CHARACTERISTICS OF SCARRING IN PATIENTS WITH MONKEYPOX INFECTION

Introduction:

The monkeypox virus (MPOX) is a zoonotic, double-stranded DNA virus belonging to the Orthopox virus genus, presenting skin manifestations like smallpox but typically less severe and self-limiting, with lower case-fatality rates. Prior to May 2022, MPOX was primarily a

zoonotic and traveler-associated infection, with limited cases outside endemic regions. However, since May 2022, over 79,000 cases have emerged across 103 non-endemic countries. Human-to-human transmission occurs primarily through direct contact with skin lesions and respiratory droplets, with fomite transmission being less common. The incubation period ranges from 5 to 24 days,[1] followed by a prodromal phase of 2 to 4 days, during which patients may experience systemic symptoms such as fever, malaise, and lymphadenopathy. The characteristic lesions progress from whitish papules on an erythematous base to umbilicated pustules. Despite the WHO's declaration of a public health emergency in response to the outbreak from 2022 to 2023, cicatricial sequelae—such as hypertrophic, keloid, atrophic scars, and pigmentary changes—have been reported anecdotally in both the current outbreak and endemic cases. The risk of scarring following lesion resolution remains poorly defined, with limited objective data available due to insufficient long-term follow-up.[2] So, this study aimed to investigate the risk factors associated with scarring in MPOX patients and to characterize the nature of scarring in individuals treated at the sexually transmitted infection (STI) unit of a tertiary hospital.

Methods:

This prospective cohort study included confirmed MPOX cases from a tertiary referral hospital, diagnosed via PCR testing of skin, oropharyngeal, urethral, and anal samples using the VIASURE Monkeypox Virus reverse transcription (RT)-PCR kit. Data on epidemiological, clinical, and microbiological factors were collected from medical records and follow-up consultations at 12–15 months post-diagnosis to assess scarring and its impact on quality of life, using a 0 to 10 scale. Statistical analysis using SPSS version 28 involved the Shapiro–Wilk test for normality, the student's t-test or Mann–Whitney U test for numerical variables, and Pearson's chi-squared or Fisher's exact tests for categorical variables, with significance set at $P < 0.05$.

Results:

Among 72 individuals with confirmed MPOX diagnoses, 40 patients were analyzed after excluding 32 due to lack of follow-up. Of these, 19 (47.5%) developed scarring, while 21 (52.5%) did not. The mean age was significantly higher in the scar-free group (42.5 years) compared to the scarred group (36.2 years), and all scarred patients were male, whereas most non-scarred patients were female (95%) (Table 1). The study found that 28.6% of scar-free patients had HIV, compared to 21.1% in the scarred group. There was no significant difference in vaccination status. Systemic symptoms were observed in 67% of the non-scarred and 74% of the scarred patients, with significant differences in initial presentation, 57% of non-scarred patients had systemic symptoms initially, while 47% of scarred patients presented with skin lesions first. Scar characteristics included a mean of 19.7 scars per patient, with the genital area being the most affected. Bacterial superinfection occurred in 15.7% of scarred patients, and severe pain was reported in 21%. The quality-of-life impairment for scarred patients averaged 5.2 out of 10, with 36.8% reporting substantial impairment. Overall, the study highlights significant differences in demographic, clinical, and quality of life outcomes related to scarring after MPOX infection.

Patient information	Patients without scars	Patients with scars
Age (range)	42.5 (22–69)	36.2 (23–49)
Male	20 (95)	19 (100)
Female	1 (5)	0 (0)
HIV positive	6 (28.6)	4 (21.1)
HIV negative	15 (71.4)	15 (78.9)
Vaccination	8 (38.1)	2 (10.5)
Non-vaccination	13 (61.9)	17 (89.5)
Arthromyalgia	8 (38)	8 (42)
Systemic symptoms	14 (66.7)	14 (74)
Fever	8 (38)	12 (63)
Asthenia	9 (43)	11 (58)
Headache	6 (29)	6 (32)
Adenopathies	12 (57)	13 (68)
Pharyngitis	6 (29)	4 (21)
Urethritis	4 (19)	2 (11)
Proctitis	5 (24)	3 (16)

Table 1. Epidemiological and clinical characteristics of patients with and without scars

Discussion:

The current study highlighted an intriguing finding regarding smallpox vaccination and monkeypox outcomes there was a lower proportion of vaccinated patients who developed scarring, although it was not statistically significant. This contrasts with previous research suggesting that vaccination correlates with milder disease and reduced scarring risk.[3,4] Additionally, the study found that HIV infection did not significantly increase the risk of scarring in monkeypox patients, diverging from earlier literature that associated HIV with a higher likelihood of extensive necrotic lesions and subsequent scarring.[5]

Conclusion:

This prospective study, the longest follow-up on MPOX patients to date, reveals that scarring is a more frequent complication than previously reported, significantly affecting quality of life and psychological well-being. Young patients with risk factors are particularly susceptible to severe sequelae, underscoring the need for vaccination as primary prevention. Close monitoring and early intervention for central facial and genital lesions, especially when they present initially, are essential to reduce long-term complications.

Reference:

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MPOX scarring is more common than previously recognized, especially in young patients, highlighting the critical need for vaccination and early intervention.

ASSOCIATIONS BETWEEN CLINICAL CHARACTERISTICS AND BIOLOGIC THERAPY SWITCHING IN PSORIASIS TREATMENT

Introduction:

Psoriasis is a chronic, non-communicable, immune-mediated disorder characterized by painful, disfiguring, and disabling symptoms, primarily affecting the skin and nails with a papulosquamous morphology. The complex genetic architecture of psoriasis contributes to its variability in presentation and severity.[1] While global prevalence was estimated at 2-3%, epidemiological data vary, with rates from 0.91% to 8.5% in adults and 0.0% to 2.1% in children.[2] Although there were several medications available, biologics have greatly improved treatment outcomes for many psoriasis patients, allowing for remission or low disease activity. However, some may need to switch therapies due to ineffectiveness or side effects, and the risk factors for these changes are not well understood. Comorbidities like arthritis and metabolic syndrome can complicate treatment, and biomarkers such as C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and neutrophil-to-lymphocyte ratio (NLR) are important for assessing disease activity. Understanding the factors behind therapy changes is essential for optimizing patient care.[3] So, this study aimed to investigate the association between clinical characteristics and laboratory data that may predict the necessity for switching biologic therapies in psoriasis management.

Methods:

This retrospective study reviewed patients with psoriasis vulgaris (PsV), psoriatic arthritis (PsA), or generalized pustular psoriasis (GPP) treated with biologics. Eligible patients initiated first-line biologic therapy and were observed for over 24 weeks, categorized by the number of biologic switches: none, one, two, or more. Baseline data included age, sex, body mass index (BMI), and disease duration. Laboratory parameters, including CRP, ESR, NLR, MLR, and PLR, were collected within 3 months before assessment. Comorbidities were evaluated, and disease severity was assessed using Psoriasis Area Severity Index (PASI) scores. Descriptive statistics summarized continuous variables as median (IQR) whereas categorical variables as counts (%). The Kruskal-Wallis and Fisher exact tests were used for analysis, with drug-

specific survival rates calculated via the Kaplan–Meier method, considering p <0.05 as significant.

Results:

In this study of 285 patients, including 135 with psoriasis vulgaris (PsV), 137 with psoriatic arthritis (PsA), and 13 with generalized pustular psoriasis (GPP), 125 patients (43.9%) maintained their initial biologic therapy, while 56.1% switched at least once, primarily from Tumor Necrosis Factor inhibitors (TNFi) to other biologics. Those who switched more frequently had significantly higher PASI scores at therapy initiation (12.3 vs. 6.6, p=0.016) and a higher prevalence of concurrent arthritis (64.1% vs. 43.2%, p=0.012). Dyslipidaemia was less common in frequent switchers (12.8% vs. 25.6%, p=0.036). Patients with two or more switches also had a higher NLR of 2.65 compared to 2.30 in those with no switches (p=0.011). CRP and ESR levels tended to be higher in patients with two or more switches (Table 1). Regarding drug survival, median durations were significantly longer for IL-17 inhibitors (54 months) and IL-23 inhibitors (55 months) compared to TNFi (25 months, p=0.007), highlighting the greater long-term efficacy of IL-17i and IL-23i in these patients.

Number of switches	None	Once	Two or more	p-value
Patients, No.	125	82	78	
Concurrent arthritis	54 (43.2%)	39 (47.6%)	50 (64.1%)	0.012
Comorbidities, Dyslipidaemia	32 (25.6%)	23 (28.0%)	10 (12.8%)	0.036
NLR, median	2.3 (1.60–3.27)	2.55 (1.91–3.45)	2.65 (2.03–3.97)	0.011
CRP, median	0.12 (0.05–0.42)	0.09 (0.05–0.31)	0.18 (0.05–0.57)	0.517
ESR, median	13.5 (7–25)	16 (7–29)	24 (8.25–37)	0.169
MLR, median	0.23 (0.18–0.30)	0.25 (0.19–0.36)	0.25 (0.09–0.31)	0.109
PLR, median	14.18 (10.98–19)	16.1 (12.14–20.94)	15.77 (12.42–21.79)	0.137

Table 1: Clinical Characteristics and Baseline Biomarkers by Number of Switches

Discussion:

The current study found that a higher NLR was significantly associated with increased rates of biologic switching, underscoring the impact of systemic inflammation on treatment outcomes. In contrast, previous studies identified demographic factors like age and sex as contributors to switching rates, suggesting a shift in focus toward inflammatory markers in treatment adherence.[4] Patients on IL-17 and IL-23 inhibitors had longer drug survival than those on TNF inhibitors, underscoring the greater efficacy and safety of newer biologics. This supports existing literature and emphasizes the need for personalized treatment strategies based on the patient's inflammatory profile.[5]

Conclusion:

This study demonstrated a correlation between elevated PASI scores, NLR, and comorbid arthritis with the frequency of biological treatment switching. A treatment strategy that integrates clinical presentation and inflammatory biomarkers may be crucial for optimizing patient management in psoriasis.

Elevated PASI scores, NLR, and arthritis correlate with increased biologic treatment switching, emphasizing the need for integrated psoriasis management.

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CASE REPORT ON CHRONIC SPONTANEOUS URTICARIA WITH WHEALS LASTING FOR MORE THAN A WEEK

Introduction:

Chronic spontaneous urticaria (CSU) is characterized by localized, temporary edema in the dermis, leading to wheals of varying sizes and shapes, often accompanied by pruritus and burning sensations. These wheals typically resolve within 30 minutes to 24 hours without leaving residual marks.[1] Urticarial vasculitis (UV) presents as edematous erythema with post inflammatory hyperpigmentation lasting more than 24 hours. A survey of 883 physicians across 92 countries identified key diagnostic features for UV, including prolonged wheals (72%) and skin biopsy results (63%). Complete blood count and serum C-reactive protein tests may aid in diagnosis.[2] This report presented a case of wheals that gradually expanded over several days and ultimately resolved within 10 days, with no significant inflammatory response or histological evidence of vasculitis.

Case presentation

A 58-year-old man presented with a 2-month history of edematous, ring-shaped erythematous lesions accompanied by severe itching. Lesions, initially the size of a red bean, enlarged over

days and resolved within 10 days without scarring, except for mild desquamation on the palms. He had no significant family history or atopic background but was allergic to shellfish.

Initial examination revealed bean-sized edematous patches on the trunk (Figure 1a) and annular erythema on the extremities (Figures 1b and 1c). Laboratory results were normal, and a biopsy showed moderate lymphocytic infiltration and edema in the dermis, with no signs of vasculitis.

Diagnosed with atypical chronic spontaneous urticaria (CSU), the patient was treated according to Japanese guidelines. Despite high doses of antihistamines and other supportive medications, improvement was limited. After 1.5 months, he developed dysgeusia, which persisted even after discontinuation of some medications.

Over time, lesions became smaller and less itchy. After 10 weeks of treatment with betamethasone and adding cyclosporine, symptoms significantly improved. By 5 months post-diagnosis, the patient was considered cured.

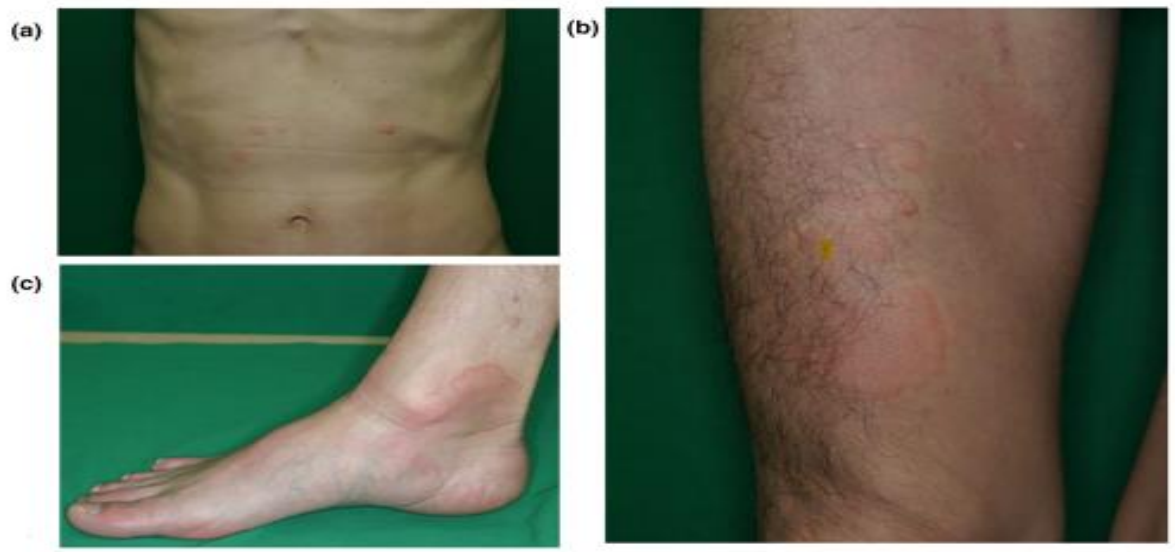

Figure 1. Clinical presentation at the initial visit:

(a) Red bean-sized edematous erythematous spots on the trunk.
(b) Edematous erythematous patches on the right thigh, ranging from thumb to walnut size.
(c) Annular erythema observed on the right foot. Each lesion gradually enlarged to palm size over several days, disappearing after 7–10 days.

Discussion:

This case presents a significant deviation from established definitions of chronic spontaneous urticaria (CSU), with wheals persisting for up to 10 days. In contrast, existing literature indicates that up to 20.8% of patients may experience wheals lasting longer than 24 hours, while the conventional understanding was that wheals typically resolve within a day.[1] Histopathological analysis revealed moderate lymphocytic infiltration and edema in the dermis, with no signs of vasculitis or features characteristic of conditions such as erythema multiforme. The absence of pressure-induced whealing suggested alternative inflammatory mechanisms beyond mast cell activation and histamine release. This aligns with Guitart's perspective that prolonged eruptions without vasculitis should still be classified as urticaria.[3]

Conclusion:

This case highlighted that urticaria wheals can last up to 10 days without evidence of urticaria vasculitis and can be managed using chronic spontaneous urticaria (CSU) guidelines. It suggests a broader disease spectrum for urticaria than previously recognized. Further research on the use of corticosteroids and cyclosporine for long-lasting wheals is needed. Reporting this case and gathering similar cases for analysis is essential.

Atypical chronic spontaneous urticaria can improve significantly with treatment using corticosteroids and cyclosporine after initial therapies fail.

Reference:

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