Original Article

Association between Periodontitis, Smoking, and Psoriasis: A Cross-Sectional Study in Bareilly Population

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ABSTRACT

Background: This study examines the link between periodontitis and the occurrence of psoriasis, particularly exploring the influence of smoking on this association. **Materials and Methods:** A sample of 464 adults, divided equally between those with periodontitis (n = 232) and those without (n = 232), was analyzed. Baseline data included demographic details, smoking habits, and medical history for hypertension and diabetes. Psoriasis incidence rates were calculated/100 person-years, with subgroup analysis by periodontitis and smoking status. Cox proportional hazards models assessed psoriasis risk in individuals with periodontitis, accounting for smoking status and other covariates. Kaplan–Meier survival analysis illustrated the cumulative psoriasis incidence across groups. **Results:** Participants had an average age of 47.5 years (±9.8), and 52% were male. Smoking was more prevalent in the periodontitis group (34%) than in the non-periodontitis group (18%, $P < 0.01$). The psoriasis incidence was 4.0 cases/100 person-years among participants with periodontitis, compared to 2.6 in those without. Smokers with periodontitis exhibited the highest psoriasis incidence rate, at 4.8 cases/100 person-years. Cox analysis showed that individuals with periodontitis had a higher risk of psoriasis (hazard ratios [HR] 1.39, 95% confidence interval [CI] 1.03–1.87, $P = 0.03$), with an amplified risk in smokers (HR 1.61, 95% CI 1.17–2.15, $P = 0.01$). Kaplan–Meier curves indicated a significantly higher cumulative psoriasis incidence in smokers with periodontitis (log-rank test, $P < 0.01$). **Conclusion:** Periodontitis is associated with a greater likelihood of developing psoriasis, especially among smokers. Targeted interventions for periodontal health and smoking cessation may reduce psoriasis risk in this population.

Key words: Bareilly population, Cohort study, Inflammation, Periodontitis, Psoriasis, Smoking

INTRODUCTION

Psoriasis is a chronic inflammatory skin disease affecting approximately 1–3% of the population, with known systemic impacts on skin, nails, and joints and associations with conditions such as cardiovascular disease, obesity, and metabolic syndrome.[1,2] Similarly, periodontitis is a chronic inflammatory disease of the gums, which, due to the immune response triggered by bacterial infection, may contribute to systemic inflammation.[3] Studies increasingly suggest a link between periodontitis and psoriasis, theorizing that shared inflammatory pathways and immune dysregulation may drive this association.[4,5]

Notably, both periodontitis and psoriasis share common comorbidities and risk factors, including obesity, metabolic syndrome, and cardiovascular disease, which may contribute to mutual disease progression.[6] Smoking, known to worsen inflammatory processes in both conditions, is an independent risk factor for both psoriasis and periodontitis. Smoking may influence the oral microbiome and immune response, leading to exacerbation of gum inflammation and further promoting the inflammatory cascade involved in psoriasis.[7-9]

While these associations have been documented in various populations, there is limited data examining these links within specific regional groups, such as the population of Bareilly, where environmental and lifestyle factors may impact disease prevalence. This study aims to investigate the prevalence of psoriasis in individuals with periodontitis within the Bareilly population, emphasizing the role of smoking as a potential exacerbating factor. Understanding these connections may inform targeted preventive strategies to manage the burden of psoriasis and periodontitis in at-risk groups.

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MATERIALS AND METHODS

Study design and population

This observational cohort study was conducted in Bareilly, India, to examine the association between periodontitis and the incidence of psoriasis, with a particular focus on the influence of smoking. The study included 464 participants, with 232 individuals diagnosed with periodontitis and 232 without. Adults aged 20 years or older who presented to the Periodontology Department Institute of Dental Sciences and Rohilkhand Medical College, dermatological clinics in Bareilly for routine care or health screenings were invited to participate. Exclusion criteria included any prior diagnosis of psoriasis or periodontitis, incomplete medical records, and age under 20.

Data collection

Participants underwent comprehensive clinical evaluations to confirm the presence of periodontitis or psoriasis. Periodontitis was diagnosed based on clinical assessment criteria, including probing depth, clinical attachment level, and radiographic evidence of bone loss. Psoriasis diagnosis followed standardized criteria, with dermatologist-confirmed diagnosis required. Baseline data included demographics, lifestyle information (such as smoking status and alcohol consumption), body mass index (BMI), and medical history. Smoking status was categorized as a non-smoker, former smoker, or current smoker.

Exposure and outcome assessment

The primary exposure variable was periodontitis status, while the primary outcome was the incidence of newly diagnosed psoriasis over the follow-up period. Smoking was assessed as a potential modifying factor. Participants were classified into four groups: (1) non-smokers without periodontitis, (2) non-smokers with periodontitis, (3) smokers without periodontitis, and (4) smokers with periodontitis.

Statistical analysis

Descriptive statistics were calculated for the baseline characteristics of participants. Cox proportional hazards regression was used to evaluate the risk of developing psoriasis among individuals with periodontitis compared to those without, adjusting for potential confounders, including age, gender, BMI, socioeconomic status, and lifestyle factors. Interaction terms were included to assess the modifying effect of smoking on the relationship between periodontitis and psoriasis. Results were expressed as hazard ratios (HRs) with 95% confidence intervals (CIs). Kaplan–Meier curves were plotted to visualize the cumulative incidence of psoriasis across groups, with significance tested by the log-rank test.

RESULTS

Baseline characteristics

The mean age of participants was 47.5 years (±9.8), and 52% were male. Smoking was more common in the periodontitis group, with 34% identified as current smokers compared to 18% in the non-periodontitis group ($P < 0.01$). Common comorbidities such as hypertension and diabetes were also more prevalent among those with periodontitis ($P < 0.05$) [Table 1].

Incidence of psoriasis

During the study period, 45 new cases of psoriasis were documented. The incidence rate was higher in participants with periodontitis compared to those without, at 4.0 cases/100 person-years versus 2.6 cases/100 person-years, respectively. Among smokers with periodontitis, the psoriasis incidence rate was particularly elevated at 4.8 cases/100 person-years, whereas non-smokers with periodontitis had an incidence rate of 3.4/100 person-years [Table 2].

Psoriasis risk analysis

Cox proportional hazards regression analysis indicated that individuals with periodontitis had a higher risk of developing psoriasis compared to those without (HR 1.39, 95% CI 1.03–1.87, $P = 0.03$). Smokers with periodontitis demonstrated an even greater risk of psoriasis (HR 1.61, 95% CI 1.17–2.15, $P = 0.01$), whereas non-smokers with periodontitis showed an increased but lower risk (HR 1.28, 95% CI 1.01–1.65). The interaction between periodontitis and smoking was statistically significant ($P < 0.05$), suggesting an amplifying effect of smoking on psoriasis risk among those with periodontitis [Table 3].

Kaplan–Meier analysis

Kaplan–Meier survival curves showed a consistently higher cumulative incidence of psoriasis among individuals with periodontitis, with smokers in this group displaying the highest incidence over time (log-rank test, $P < 0.01$) [Table 4].

Conclusion of findings

This study confirms periodontitis as an independent risk factor for psoriasis and highlights smoking as a significant modifier of this

Table 1: Baseline characteristics

Characteristic	Periodontitis group (n=232)	Non-periodontitis Group (n=232)	P-value
Age (years)	47.5±9.8	47.5±9.8	0.96
Gender (M/F)	120/112	121/111	0.85
Smokers (%)	34 (79)	18 (42)	<0.01
Hypertension (%)	30 (70)	22 (51)	0.04
Diabetes (%)	28 (65)	20 (46)	0.03

Table 2: Incidence of psoriasis

Group	Incidence rate (cases/100 person-years)
Periodontitis (overall)	4.0
Non-periodontitis (overall)	2.6
Smokers with periodontitis	4.8
Non-smokers with periodontitis	3.4

Table 3: Psoriasis risk analysis (cox proportional hazards regression)

Group	Hazard ratio	95% Confidence interval	*P*-value
Periodontitis versus non-periodontitis	1.39	1.03–1.87	0.03
Smokers with periodontitis versus non-smokers with periodontitis	1.61	1.17–2.15	0.01
Non-smokers with periodontitis versus non-periodontitis	1.28	1.01–1.65	0.04

Table 4: Kaplan-Meier survival analysis

Group	Cumulative incidence of psoriasis (%)	Log-rank test *P*-value
Periodontitis (overall)	18	<0.01
Non-periodontitis (overall)	10	
Smokers with periodontitis	22	
Non-smokers with periodontitis	15	

risk. These findings point to the importance of early interventions in oral health and smoking cessation, particularly in populations vulnerable to these combined risks.

DISCUSSION

This study underscores the significant link between periodontitis and an increased risk of developing psoriasis, with an amplified risk observed in smokers. The findings support the idea that these two chronic inflammatory conditions may share underlying immunological and inflammatory pathways that contribute to their association, and the observed interaction with smoking highlights the compounding effect of lifestyle factors on inflammatory diseases.

Periodontitis has been identified as a persistent inflammatory condition impacting the gums, with systemic consequences that extend beyond the oral cavity. Its role in systemic inflammation is thought to stem from the continuous exposure of the immune system to periodontal pathogens, which triggers an inflammatory response that can spread through systemic circulation. This pro-inflammatory state, characterized by elevated levels of cytokines such as interleukin (IL)-6, IL-17, and tumor necrosis factor-alpha, is also commonly observed in psoriasis.[7] Psoriasis itself is increasingly recognized as a systemic condition, affecting not only the skin but also linking to comorbidities such as cardiovascular disease, obesity, and metabolic syndrome. The presence of shared inflammatory mediators between periodontitis and psoriasis suggests that each condition may predispose individuals to the other, with chronic inflammation as a unifying factor.

Our study observed that smokers with periodontitis had the highest risk of developing psoriasis, highlighting the complex interaction between environmental factors and inflammatory diseases. Smoking is known to impair immune function, modify cytokine profiles, and influence the composition of the oral microbiome. These effects make the periodontal environment more susceptible to persistent inflammation and bacterial colonization, which can exacerbate the immune response.[8] In psoriasis, smoking has been shown to increase disease severity and incidence, potentially by influencing T-cell responses and other immune pathways involved in both diseases. Thus, smoking may act as a catalyst that intensifies the inflammatory cascade in individuals with periodontitis, thereby increasing the likelihood of psoriasis development.[9]

The presence of common comorbidities such as obesity, diabetes, and metabolic syndrome in both conditions further supports their relationship. These comorbidities are characterized by chronic low-grade inflammation and may act as intermediate factors, compounding the risk of inflammatory disorders in individuals with periodontitis. The inflammation associated with these metabolic conditions is thought to contribute to a cycle of immune dysregulation, where systemic inflammation from periodontitis may worsen insulin resistance or lipid profiles, creating an environment conducive to psoriasis onset or exacerbation.[6]

Bacterial pathogens in periodontitis, notably *Porphyromonas gingivalis*, are believed to play a pivotal role in this inflammatory network. This bacterium is capable of evading the immune system and promoting a pro-inflammatory environment by stimulating the release of cytokines such as IL-1β and IL-17, which are implicated in both periodontitis and psoriasis pathogenesis. The immune response to such pathogens can lead to the systemic spread of inflammatory mediators, increasing the risk of systemic inflammatory diseases such as psoriasis.[10] The potential for periodontal pathogens to trigger systemic inflammation suggests a plausible pathway through which periodontitis may influence psoriasis development, especially in individuals with other risk factors such as smoking.

These findings have practical implications for clinical management. The identification of periodontitis as a potential risk factor for psoriasis highlights the importance of integrating oral health monitoring into general healthcare, especially for populations at risk. Proactive periodontal care, including regular dental check-ups and treatment of gum disease, may help reduce systemic inflammation and potentially lower psoriasis risk. In addition, smoking cessation should be strongly encouraged in patients with periodontitis to minimize the compounded risk, as smoking appears to exacerbate the effects of periodontal disease on systemic health.[11]

There are limitations to consider in interpreting these results. The observational design of the study limits the ability to draw causal inferences, and while associations are evident, it is unclear whether periodontitis directly causes psoriasis or if shared inflammatory pathways lead to their co-occurrence. In addition, our sample is specific to a regional population, and thus, findings may vary in different demographic or environmental contexts. Despite these limitations, the study adds to a growing body of evidence that periodontitis is associated with psoriasis, particularly in the presence of smoking.

CONCLUSION

This study emphasizes the significant association between periodontitis and psoriasis, with smoking identified as an important

modifier of this relationship. These findings suggest that managing periodontal health and promoting smoking cessation may help mitigate psoriasis risk, particularly among individuals with multiple risk factors. Future research should aim to explore the biological mechanisms underlying this association further and assess whether targeted periodontal treatment or anti-inflammatory therapies could reduce psoriasis incidence.

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