

Psoriasis and Periodontitis: Exploring an association or lack thereof

Abstract

Objectives: Psoriasis is a common, chronic, non-communicable skin disease with no clear etiology or cure. Periodontitis is a chronic inflammatory condition which is now known to significantly influence various systemic diseases as an established risk factor. This study aimed at comparatively evaluating the periodontal status of Psoriatic patients vis. a vis. that of age and gender matched systemically healthy volunteers. An attempt was also made to explore a possible association, if any, amongst the two diseases. **Materials and Methods:** Forty two residents of Chandigarh, suffering from Psoriasis and attending the Psoriasis Clinic of Department of Dermatology & Venereology, Post Graduate Institute of Medical Education & Research, Chandigarh were recruited over a period of 10 months (Case group) and their periodontal status was compared with forty two age and gender matched systemically healthy volunteers (Control group) randomly selected from the Out Patient Department of Periodontics, Dr. Harvansh Singh Judge Institute of Dental Sciences & Hospital, Panjab University, India. Their serum IL-33 levels were evaluated and compared in an attempt to identify an underlying common pathological pathway. **Results:** The periodontal status was comparable in the two groups in terms of the debris index ($p = 0.932$), calculus index ($p = 0.088$), plaque index ($p = 0.097$), and mean clinical attachment loss ($p = 0.401$). A higher bleeding points index was recorded amongst healthy individuals as compared to the Psoriasis group, the difference being statistically significant ($p = 0.001$). The mean number of teeth were more in the Psoriasis group as compared to the healthy group ($p=0.034$). IL 33 levels were also not significantly different ($p = 0.491$). **Conclusion:** Contrary to currently available evidence in literature, the study did not find a statistically significant association between Psoriasis and Inflammatory Periodontal Disease.

Keywords: Association, inflammation, periodontal disease, periodontitis, psoriasis

Introduction

Recent times have seen a sudden and justifiable interest in relating Periodontitis with a number of systemic conditions with the aim of improving understanding and overall patient care. However, in the midst of potentially lucrative associations it does become something of a caveat that to prove an association may make one lose sight of the quality of evidence supporting these claims. Psoriasis has been reported to have an association with Periodontitis, however, a meta-analysis on the subject seemed to suggest otherwise in light of the evidence available on the matter.^[1,2] Psoriasis is a chronic skin disease of unknown etiology presenting on the skin of affected people as erythematous scaly plaques, particularly on the extensor surfaces of knees and elbows, scalp, buttocks, and lower back.^[3,4] In addition to the skin, it affects the joints

of the spine and other joints, known as psoriatic arthritis.

Periodontal diseases are caused by microbial plaque, are chronic in nature, and no longer thought to be a localized entity but one which significantly influence the systemic condition of an individual. They present clinically as destruction of tissues and bone surrounding the teeth, leading to mobility and finally tooth loss. The World Health Organization (WHO) Global Report on Psoriasis, Geneva 2016 acknowledges the association between Periodontal disease and increased risk for Psoriasis.^[1] Association between the two diseases may be due to common underlying pathological pathways.

In periodontal diseases, IL-33 has an osteoclastogenic role by inducing osteoclast differentiation and increased production of bone resorption factors (c- Src

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and cathepsin K).^[5] It has been reported that treating periodontal disease leads to an attenuation of the risk for subsequent psoriasis.^[6] Early recognition and control of such modifiable risk factors hence might pave the path for better patient management and reduce the morbidity/mortality associated with psoriasis. With the recent increasing attention on links between periodontal disease and the development of psoriasis, we felt a compelling need to explore the association of periodontal disease and psoriasis in the regional population of Chandigarh suffering from psoriasis.

Materials and Methods

The study was designed as a descriptive cross-sectional study. The sample size was calculated to be 42 with a confidence level of 95%. Eighty-four subjects in the age group of 18–65 years were enrolled over a period of ten months: Case group, 42 subjects with psoriasis and Control group, 42 age and sex matched systemically healthy subjects. The diagnosis of patients with psoriasis was based on case history and clinical examination. Patients with all types of psoriasis and under medication for the same (methotrexate 0.3–0.5 mg/kg once weekly dose), were included in the study randomly using coin toss method. Control group included age and sex matched systemically healthy subjects selected randomly using coin toss method. Presence of minimum 20 teeth in the oral cavity and willingness to participate in the study were other criteria mandatory for inclusion into the study. Subjects with any known systemic disease, tobacco users, pregnant/lactating mothers, patients with <20 teeth in the oral cavity, alcoholics, subjects who have undergone periodontal therapy in previous 6 months, subjects under any medication (except methotrexate for psoriasis).

A written signed informed consent was obtained from the patients willing for participation. The research protocol was approved by both the Panjab University Institutional Ethical Committee and the PGIMER Institutional Ethical Committee. The trial was registered in CTRI with the id CTRI/2018/04/013475.

Data collection

Self-report information pertaining to socio-demographic, oral health, and general health factors was gathered at baseline. All patients underwent a detailed general physical examination. The severity of psoriasis was assessed and recorded by Psoriasis Area and Severity Index (PASI) as described by Cohen *et al.* (2005).^[7]

Oral and periodontal examination

Measurements of periodontal parameters were made by a single trained examiner for both the groups. Probing pocket depth (PD) was measured at six sites (mesio-buccal, mid-buccal, disto-buccal, mesio-lingual, mid-lingual, and disto-lingual) per tooth using William's Periodontal

probe. Gingival recession was determined by measuring the distance from the cemento-enamel junction to the gingival margin in millimeters and rounded down to the next millimeter. The clinical attachment loss (CAL) was calculated from recession and probing depth measures and represented as the distance in millimeters from cemento-enamel junction to periodontal ligament attachment. Sites that bled upon gentle probing were recorded and percentage of bleeding sites to total sites was calculated in each subject. Oral hygiene was evaluated by using the criteria of the Plaque Index of Silness and Løe (1964).^[8] The simplified Oral Hygiene Index given by Greene and Vermillion (1964) was used to record the Debris Index and Calculus Index.^[9] Number of teeth present in the oral cavity and number of missing teeth were also recorded.

Plasma/serum collection

10 ml of venous blood was collected from the antecubital vein using a standard veni-puncture method and transferred to the laboratory. The blood sample was allowed to clot at room temperature and, after 1 h, serum was separated by centrifugation. The serum was transferred to storage vials and stored at -70°C till required for biochemical assays (IL 33 level estimation). All the samples sent for biochemical assessment were thawed only once.

Statistical analysis

Data analysis was conducted using IBM SPSS STATISTICS (version 23.0). Frequencies were compared by using Chi square test. Data found to be normal were analyzed using student's t-test, otherwise, Mann-Whitney test was applied. Spearman's Correlation Coefficient was used to find correlations.

Results

The mean age of the population was 38.07 years. The number of males ($n = 26$) and females ($n = 16$) were similar in both groups. The effect of residence was, however, found to be statistically significant, with 59.5% of the psoriatic subjects residing in rural areas compared to controls (9.5%). Mean duration of Psoriasis (in years) in the case group was 11.82 with mean PASI score recorded as 10.54. Koebner phenomenon was absent in the majority of patients (81%) along with the Auspitz sign being absent in all but one individual. All individuals included in the study were diagnosed with Psoriasis vulgaris.

Intergroup correlations have been presented in Table 1. A higher bleeding points index was recorded amongst healthy individuals as compared to the psoriasis group, the difference being statistically significant ($p = 0.001$). The mean number of teeth present in the oral cavity were more in the psoriasis group as compared to the healthy group, the difference being statistically significant ($p = 0.034$). The mean number of missing teeth was found to be more

in the healthy group with the difference being statistically significant ($p = 0.034$). There was no statistically significant correlation found amongst the psoriasis and healthy groups in terms of debris index (DI) ($p = 0.932$), calculus index (CI) ($p = 0.088$), plaque index (PI) ($p = 0.097$), mean clinical attachment loss (CAL) ($p = 0.401$), and IL 33 levels ($p = 0.491$). Intragroup correlations have been presented in Tables 2 and 3.

Discussion

Psoriasis is an immune-mediated, chronic inflammatory proliferative disorder of the skin which involves the skin, joints, and nails. Although psoriasis has been associated with stress, bacterial infection, and inflammatory disorders, the association between psoriasis and periodontitis cannot be explained merely on the basis of chronic bacterial inflammation. Patients with psoriasis have been reported to have lesser number of teeth and reduced bone levels as compared to healthy controls, indicating that there exists an association between psoriasis and chronic form of periodontal disease.^[10] Our study reports results contrary to these findings with the mean number of teeth present in the oral cavity more in the psoriasis group as compared to the healthy group, the difference being statistically significant ($p = 0.034$). The mean number of missing teeth was found to be more in the healthy group with the difference being statistically significant ($p = 0.034$). IL-33 plays a role in affecting periodontal bone loss by virtue of its induction of RANKL. There is an ambiguity in data pertaining to the local and systemic concentration of IL-33 with reports ranging from hardly detectable to highly increased levels.^[11-14] A number of studies have shown that IL-33 levels are increased in skin lesions of psoriasis

with this being implicated as arising from systemic circulation.^[15-18] Our study found no significant correlation between IL 33 levels, Duration of psoriasis, PASI score, Debris index (DI), Calculus index (CI), Plaque index (PI), Bleeding points index (BPI) & Mean Clinical Attachment Loss (CAL) amongst the psoriasis group subjects. Similarly, no significant correlation was found between IL 33 levels, Debris index (DI), Calculus index (CI), Plaque index (PI), Bleeding points index (BPI), and Mean Clinical Attachment Loss (CAL) amongst the systemically healthy group subjects.

Conclusion

Contrary to currently available evidence in literature, the study did not find a statistically significant relationship between psoriasis and inflammatory periodontal disease. Alternatively, individuals with psoriasis were found to have an improved periodontal status as compared to the healthy group studied.

Ethical approval

The study was an observational trial. The research protocol was approved by Institutional Ethical Committee. The trial was registered in CTRI with the id CTRI/2018/04/013475.

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Written informed consent

Was obtained from all individuals participating in the study.

Table 1: Intergroup correlation of assessed parameters

	DI	CI	PI	BPI	CAL	Number of teeth (Mean)	Number of missing teeth (Mean)	IL 33 levels pg/ml
Psoriasis group	1.689	1.447	1.8373	0.6704	1.1223	29.69	2.31	26.65176
Healthy group	1.855	1.662	1.5609	1.7585	1.3487	28.38	3.62	34.72016
<i>P</i>	0.932	0.088	0.097	0.001**	0.401	0.034*	0.034*	0.491

*Significant; **highly significant; Debris index (DI); Calculus index (CI); Plaque index (PI); Bleeding points index (BPI); Mean Clinical Attachment Loss (CAL); Interleukin 33 (IL 33)

Table 2: Intragroup correlation of assessed parameters amongst the Psoriasis cohort

Parameters	IL33	Duration of psoriasis	PASI score	DI	CI	PI	BPI	Mean CAL
IL33	1.000	0.171	-0.063	0.002	-0.023	0.040	-0.011	0.109
Duration of Psoriasis	0.171	1.000	-0.072	0.314	0.319	0.404	-0.158	0.315
PASI score	-0.063	-0.072	1.000	0.234	0.205	0.156	0.268	0.334
DI	0.002	0.314	0.234	1.000	0.788	0.735	0.549	0.574
CI	-0.023	0.319	0.205	0.788	1.000	0.805	0.416	0.605
PI	0.040	0.404	0.156	0.735	0.805	1.000	0.354	0.695
BPI	-0.011	-0.158	0.268	0.549	0.416	0.354	1.000	0.280
Mean CAL	0.109	0.315	0.334	0.574	0.605	0.695	0.280	1.000

Interleukin 33 (IL 33); Psoriasis Area and Severity Index (PASI); Debris index (DI); Calculus index (CI); Plaque index (PI); Bleeding points index (BPI); Mean Clinical Attachment Loss (CAL)

Table 3: Intragroup correlation of assessed parameters amongst the systemically healthy cohort

Parameters	IL33	DI	CI	PI	BPI	Mean CAL
IL33	1.000	0.184	0.165	-0.276	-0.069	-0.063
DI	0.184	1.000	0.792	0.250	0.212	0.374
CI	0.165	0.792	1.000	0.265	0.078	0.434
PI	-0.276	0.250	0.265	1.000	0.377	0.129
BPI	-0.069	0.212	0.078	0.377	1.000	0.197
Mean CAL	-0.063	0.374	0.434	0.129	0.197	1.000

Interleukin 33 (IL 33); Debris index (DI); Calculus index (CI); Plaque index (PI); Bleeding points index (BPI); Mean Clinical Attachment Loss (CAL)

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Conflicts of interest

There are no conflicts of interest.

References

- World Health Organization (WHO). Global Report on Psoriasis. Geneva: World Health Organization; 2016.
- Monson CA, Porfirio GJ, Riera R, Tweed JA, Petri V, Nagi A, *et al.* Periodontal aspects for psoriasis: A systematic review. *J Clin Res* 2016;3:1-8.
- Kaur I, Handa S, Kumar B. Natural history of psoriasis: A study from the Indian subcontinent. *J Dermatol* 1997;24:230-4.
- Nakib S, Han J, Li T, Joshipura K, Qureshi AA. Periodontal disease and risk of psoriasis among nurses in the United States. *Acta Odontol Scand* 2013;71:1423-9.
- Da Luz FA, Oliveira AP, Borges D, Brígido PC, Silva MJ. Physiopathological Role of IL-33: New Highlights in Bone Biology and a Proposed Role in Periodontal Disease. *Mediators of Inflammation*. 2014; 2014: 342410.
- Keller JJ, Lin HC. The effects of chronic periodontitis and its treatment on the subsequent risk of psoriasis. *Br J Dermatol* 2012;167:1338-44.
- Cohen AD, Van Dijk D, Naggan L, Vardy DA. Effectiveness of climatotherapy at the Dead Sea for psoriasis vulgaris: A community-oriented study introducing the 'Beer Sheva Psoriasis Severity Score'. *J Dermatolog Treat* 2005;16:308-13.
- Silness J, Loe H. Periodontal disease in pregnancy. II. Correlation between oral hygiene and periodontal condition. *Acta Odontol Scand* 1964;22:121-35.
- Greene JG, Vermillion JR. The simplified oral hygiene index. *JADA* 1964;68:7-13.
- Mahanonda R, Pichyangkul S. Toll-like receptors and their role in periodontal health and disease. *Periodontol* 2000 2007;43:41-55.
- Buduneli N, Özçaka Ö, Nalbantsoy A. Interleukin-33 levels in gingival crevicular fluid, saliva, or plasma do not differentiate chronic periodontitis. *J Periodontol* 2012;83:362-8.
- Papathanasiou E, Teles F, Griffin T, Arguello E, Finkelman M, Hanley J, *et al.* Gingival crevicular fluid levels of interferon- gamma, but not interleukin-4 or -33 or thymic stromal lymphopoietin, are increased in inflamed sites in patients with periodontal disease. *J Periodontal Res* 2014;49:55-61.
- Kursunlu SF, Öztürk VÖ, Han B, Atmaca H, Emingil G. Gingival crevicular fluid interleukin-36beta (-1F8), interleukin-36gamma (-1F9) and interleukin-33 (-1F11) levels in different periodontal disease. *Arch Oral Biol* 2015;60:77-83.
- Han X, Lin X, Yu X, Lin J, Kawai T, Larosa KB, *et al.* *Porphyromonas gingivalis* infection-associated periodontal bone resorption is dependent on receptor activator of NF-kappaB ligand. *Infect Immun* 2013;81:1502-9.
- Theoharides TC, Zhang B, Kempuraj D, Tagen M, Vasiadi M, Angelidou A, *et al.* IL-33 augments substance P-induced VEGF secretion from human mast cells and is increased in psoriatic skin. *Proc Natl Acad Sci USA* 2010;107:4448-53.
- Meehphansan J, Tsuda H, Komine M, Tominaga S, Ohtsuki M. Regulation of IL-33 expression by IFN-gamma and tumor necrosis factor-alpha in normal human epidermal keratinocytes. *J Invest Dermatol* 2012;132:2593-600.
- Hueber AJ, Alves-Filho JC, Asquith DL, Michels C, Millar NL, Reilly JH, *et al.* IL-33 induces skin inflammation with mast cell and neutrophil activation. *Eur J Immunol* 2011;41:2229-37.
- Meehphansan J, Komine M, Tsuda H, Karakawa M, Tominaga S, Ohtsuki M. Expression of IL-33 in the epidermis: The mechanism of induction by IL-17. *J Dermatol Sci* 2013;71:107-14.