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Original Research

Assessment of interleukin IL-17 levels in periodontitis patients: an observational study

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ABSTRACT:
Background: Periodontal disease is the most common localized dental inflammatory disease caused by bacterial infection of the periodontal cavity associated with subgingival plaque. Interleukin-17 (IL-17) is a proinflammatory cytokine secreted by Th-17 cells. It is a powerful activator of neutrophils as it regulates G-CSF and its receptor and chemokine expression. Hence; the present study was undertaken for assessing salivary IL-17 levels in periodontitis patients. **Materials & methods:** A total of 45 chronic periodontitis patients and 45 healthy controls were included in the present study. All the patients were recalled in the morning and salivary samples were taken. All the samples were sent to laboratory where an auto-analyser was used for evaluation of serum IL-17 levels. All the results were recorded in Microsoft excel sheet and were analysed by SPSS software. **Results:** Mean IL-17 levels among the patients of the chronic periodontitis group and the control group was found to be 8.13 pg/ml and 1.39 pg/ml respectively. Significant results were obtained while comparing the mean IL-17 levels among the patients of the periodontitis group and the control group. **Conclusion:** IL-17 levels are raised significantly in periodontitis patients.
Key words: Interleukin, Periodontitis

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INTRODUCTION

Periodontal disease is the most common localized dental inflammatory disease caused by bacterial infection of the periodontal cavity associated with subgingival plaque. This is often associated with diverse pathological conditions like inflammation of gums (gingivitis), degeneration of periodontal ligament and dental cementum and alveolar bone loss. Gingivitis is the initial stage of disease which may or may not progress into severe condition like periodontitis which shows evidences of cavity formation, loss of clinical attachment level and degeneration of alveolar bone.[1-3]

Cytokines play an important role in the pathology associated with chronic inflammatory diseases. In recent years, the etiology of periodontal tissue breakdown is primarily attributed to the interaction of bacterial antigens and inflammatory cells resulting in the production of cytokines. IL-1 alpha, IL-1 beta, IL-6 and tumor necrosis factor alpha are proinflammatory cytokines that have been identified in gingival crevicular fluid (GCF). Interleukin-17 (IL-17) is a proinflammatory cytokine secreted by Th-17 cells. It is a powerful activator of neutrophils as it regulates G-CSF and its receptor and chemokine expression. It contributes in the pathogenesis of various autoimmune and inflammatory diseases. It regulates antimicrobial activity of molecules like calgranulins, β-defensins, and mucin. Its increased level has been documented in CP. Although periodontal infection (P. gingivalis) induces IL-17, the protective role of IL-17 against bone destruction has also been suggested.[4-6] Hence; the present study was undertaken for assessing salivary IL-17 levels in periodontitis patients.

MATERIALS & METHODS

The present study was conducted with the aim of assessing the salivary IL-17 levels in periodontitis

patients. A total of 45 chronic periodontitis patients and 45 healthy controls were included in the present study. Criteria described by American Association of Periodontology were used for establishing the diagnosis of chronic periodontitis. Complete demographic details of all the subjects were obtained. Mouth mirror, probe and tweezers were used for doing clinical examination. Exclusion criteria for present study included:

- Patients with history of any other systemic illness,
- Patients with any known drug allergy,
- Patients with history of any other metabolic disorder,
- Patients with presence of any form of malignant neoplasm

After meeting the exclusion criteria, all the patients were recalled in the morning and salivary samples were taken. All the samples were sent to laboratory where an auto-analyser was used for evaluation of serum IL-17 levels. All the results were recorded in Microsoft excel sheet and were analysed by SPSS software.

RESULTS

Mean age of the patients of the chronic periodontitis group and the control group was found to be 41.3 years and 42.9 years respectively. There were 30 males and 15 females in chronic periodontitis group while there were 28 males and 17 females in the control group. Mean probing depth among the patients of the chronic periodontitis group and the control group was found to be 3.17 mm and 0.92 mm respectively. Mean clinical attachment levels among the patients of the chronic periodontitis group and the control group was found to be 3.29 mm and 0.97 mm respectively. Mean pocket depth and mean clinical attachment levels among the patients of the chronic periodontitis group were significantly higher in comparison to the patients of the control group.

In the present study, mean IL-17 levels among the patients of the chronic periodontitis group and the control group was found to be 8.13 pg/ml and 1.39 pg/ml respectively. Significant results were obtained while comparing the mean IL-17 levels among the patients of the periodontitis group and the control group.

Table 1: Demographic and clinical data

Parameter	Chronic periodontitis group	Control group	p-value
Mean age (years)	41.3	42.9	0.12
Males (n)	30	28	0.39
Females (n)	15	17	
Pocket depth (mm)	3.17	0.92	0.00*
Clinical attachment level (mm)	3.29	0.97	0.02*

*: Significant

Table 2: Comparison of salivary IL-17 levels

Parameter	Chronic periodontitis group	Control group	p-value
Mean IL-17 (pg/ml)	8.13	1.39	0.00*
SD	4.33	0.58	

*: Significant

DISCUSSION

Periodontitis is a bacteria-induced chronic inflammatory disease of tooth-supporting tissues, i.e., the gingiva, periodontal ligament, and alveolar bone. Long-term bacterial accumulation at the gingival margin results in matured biofilm and enrichment of periodontal pathogens in subgingival areas. Persistent bacterial burden and sustained inflammation of the gingival tissues induce the deepening of the shallow gingival crevice around the affected tooth to form a deep periodontal pocket.[6-8]

The gingival epithelium forms the first line of defense against periodontal bacteria by producing antimicrobial peptides and chemokines, while dendritic cells, which are potent antigen-presenting cells, activate the naive T cell response. Part of the host immune response is due to the expression of interleukin (IL)-23 by dendritic cells, differentiation of Th17 cells, and the activation of the IL-23/IL-17 cascade. Th17 cells, which are distinct from the other CD4+ T cell subsets, are the main source of IL-17. This cytokine plays an essential role in the pathogenesis of inflammatory diseases, including periodontitis.[7-9] Hence; the present study was undertaken for assessing salivary IL-17 levels in periodontitis patients.

In the present study, mean probing depth among the patients of the chronic periodontitis group and the control group was found to be 3.17 mm and 0.92 mm respectively. Mean clinical attachment levels among the patients of the chronic periodontitis group and the control group was found to be 3.29 mm and 0.97 mm respectively. Mean pocket depth and mean clinical attachment levels among the patients of the chronic periodontitis group were significantly higher in comparison to the patients of the control group. Gürsoy UK et al assessed salivary interleukin (IL)-17 and tumor necrosis factor (TNF)-α levels in subjects with T2DM. Unstimulated whole saliva samples, together with full-mouth periodontal recordings (plaque index [PI], bleeding on probing [BOP %], gingival index [GI], probing pocket depth [PPD], and clinical attachment level [CAL]), were collected from 123 subjects with T2DM. When the subjects with detectable salivary IL-17 were categorized in tertiles, the scores of PPD and BOP%, and salivary TNF-α concentrations were significantly elevated in the highest ($P = 0.007$, $P = 0.002$ and $P < 0.001$, respectively) and middle ($P = 0.052$, $P = 0.022$, and $P = 0.003$, respectively) tertiles compared to subjects with non-detectable salivary IL-17. The adjusted association between PPD measurements and salivary

IL-17 concentrations was significant (P = 0.008). Poorly-controlled glycemic status relates to the severity of periodontal disease in T2DM.[10]

In the present study, mean IL-17 levels among the patients of the chronic periodontitis group and the control group was found to be 8.13 pg/ml and 1.39 pg/ml respectively. Significant results were obtained while comparing the mean IL-17 levels among the patients of the periodontitis group and the control group. Batool H et al determined levels of salivary IL-6 and IL-17 in patients with calculus associated chronic periodontitis. Between healthy controls and CP patients (moderate and severe disease), a statistically significant difference was observed in the concentrations of IL-6 and IL-17. In CP patients, the highest mean ± SD of salivary IL-6 and IL-17 was observed in severe CP, followed by moderate and mild CP. Regarding level of IL-6, a statistically significant difference was observed between mild and severe disease and between moderate and severe subcategories of CP patients. Similarly, statistically significant difference was observed in the level of IL-17 between mild and moderate, mild and severe disease, and moderate and severe disease. The levels of salivary IL-6 and IL-17 were increased significantly in calculus associated CP patients as compared to healthy controls and these levels increased with the progression of CP.[11] DM Isaza-Guzmán et al investigated if the salivary levels of IL-17, IL-21, IL-22, and its ratio regarding salivary IFN-γ may be linked with the periodontal clinical status. One hundred and five chronic periodontitis (CP) subjects and 44 healthy controls (HC) were recruited. A statistically significant increase in salivary levels of IFN-γ and IFN-γ/IL-22 ratio in CP group could be detected, but there was no significant domination of any Th17 cytokine that could be of predictive value for health/disease status. Univariate and binary logistic regression analyses revealed a strong and independent association of IFN-γ salivary levels and IFN-γ/IL-22 ratio with disease status. An interaction effect of ageing on IFN-γ levels also could be noted. While salivary levels of IFN-γ and IFN-γ/IL-22 ratio may act as strong/independent indicators of the amount and extent of periodontal breakdown, the low detection frequency of Th17 cytokines in saliva samples make these determinations useless for the detection of disease presence and/or its severity.[12]

CONCLUSION

From the above results, the authors conclude that IL-17 levels are raised significantly in periodontitis patients.

REFERENCES

1. Hajishengallis G. Immunomicrobial pathogenesis of periodontitis: Keystones, pathobionts, and host response. Trends Immunol. 2014; 35: 3–11.
2. Darveau RP. Periodontitis: A polymicrobial disruption of host homeostasis. Nat Rev Microbiol. 2010; 8: 481–90.
3. Lalla E, Papapanou PN. Diabetes mellitus and periodontitis: A tale of two common interrelated diseases. Nat Rev Endocrinol. 2011; 7: 738–48.
4. Pihlstrom BL, Michalowicz BS, Johnson NW. Periodontal diseases. Lancet. 2005; 366: 1809–20.
5. Oda T., Yoshie H., Yamazaki K. Porphyromonas gingivalis antigen preferentially stimulates T cells to express IL-17 but not receptor activator of NF-κB ligand in vitro. Oral microbiology and immunology. 2003;18(1):30–36.
6. Goswami J., Hernández-Santos N., Zuniga L. A., Gaffen S. L. A bone-protective role for IL-17 receptor signaling in ovariectomy-induced bone loss. European Journal of Immunology. 2009;39(10):2831–2839.
7. Tymkiw K. D., Thunell D. H., Johnson G. K., et al. Influence of smoking on gingival crevicular fluid cytokines in severe chronic periodontitis. Journal of Clinical Periodontology. 2011;38(3):219–228. doi: 10.1111/j.1600-051X.2010.01684.x.
8. Turner-Brannen E, Choi KY, Arsenault R, El-Gabalawy H, Napper S, Mookherjee N. Inflammatory cytokines IL-32 and IL-17 have common signaling intermediates despite differential dependence on TNF-receptor 1. J Immunol. 2011; 186: 7127–35.
9. Liu Y, Mei J, Gonzales L et al. IL-17A and TNF-alpha exert synergistic effects on expression of CXCL5 by alveolar type II cells in vivo and in vitro. J Immunol. 2011; 186: 3197–205.
10. Gürsoy UK, Sinem Yildiz Çiftlikli 2, Eija Könönen 1, Mervi Gürsoy 1, Başak Doğan 2. Salivary interleukin-17 and Tumor Necrosis Factor-α in Relation to Periodontitis and Glycemic Status in Type 2 Diabetes Mellitus. J Diabetes. 2015 Sep;7(5):681-8.
11. Batool H, Nadeem A, Kashif M, Shahzad F, Tahir R, Afzal N. Salivary Levels of IL-6 and IL-17 Could Be an Indicator of Disease Severity in Patients with Calculus Associated Chronic Periodontitis. Biomed Res Int. 2018;2018:8531961.
12. D M Isaza-Guzmán 1, N Cardona-Vélez 1, D E Gaviria-Correa 1, M C Martínez-Pabón 1, M C Castaño-Granada 1, S I Tobón-Arroyave 2 Association Study Between Salivary Levels of Interferon (IFN)-gamma, Interleukin (IL)-17, IL-21, and IL-22 With Chronic Periodontitis. Arch Oral Biol. 2015 Jan;60(1):91-9. doi: 10.1016/j.archoralbio.2014.09.002. Epub 2014 Sep 28.