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Association Between IL-6 And Oral Tissue Responses in Patients Wearing Dental Prostheses

Rawaa Zaher Hassan Zwain
Department of Prosthodontics, Faculty of Dentistry, University of Kufa, Iraq

Abstract

The oral mucosa of the dental prosthesis bearing areas are subjected to permanent stresses and it is a chronic infection which may lead to inflammatory conditions produced by pro-inflammatory cytokines. Interleukin-6 (IL-6) is an important mediator of inflammation that participates in regulation and damage of mucosal immune, but little is known about its relevance to prosthesis-induced oral tissue changes. The objective of the current case–control study was to measure salivary levels of IL-6 in patients who wore dental prostheses and healthy controls and seek possible associations between IL-6 concentrations and clinical responses in oral tissues as mucosal erythema, mucosal edema, ulceration, or gingival inflammation. This cross-sectional study was performed in the Dental Clinics of Al-Najaf Center for Dentistry, Al-Najaf, Iraq from April 2024 to February 2025. A total of 120 subjects were included, which consisted of 66 denture wearers and 54 healthy systemically control individuals that were age and sex matched. Full-mouth clinical examinations were conducted to evaluate tissue alterations that were graded by the degree of severity. Whole unstimulated saliva samples were collected in the morning under standardized conditions and analyzed with a commercial enzyme-linked immunosorbent (ELISA) for IL-6 concentrations. Results Statistical analysis was conducted with SPSS version 25, which included chi-square tests for categorical variables, independent-samples t-tests to compare the groups, one-way ANOVA (F-test) for subgroup comparisons and Pearson's correlation in order to test the association between IL-6 levels and tissue response indices. It was also found that mean salivary IL-6 level of the prosthesis-wearing group was significantly higher than that of the controls ($p = 0.013$). Statistically, a significant difference in mucosal erythema, ulcers, mucosa edema and the degree of gingival inflammation between patients and controls was found ($p < 0.05$). IL-6 levels were progressively higher with the severity

of tissue alterations, and differed significantly between subgroups for mucosal erythema, edema, ulceration and gingival index ($p < 0.05$). The correlation analysis showed a significant positive relationship between IL-6 levels and mucosal erythema ($p = 0.002$), mucosal edema ($p = 0.003$) and gingival inflammation ($p = 0.04$), but not with ulceration. In summary, salivary IL-6 levels are significantly connected with local inflammatory responses in oral tissue of patients using dental prostheses. These results reveal the important role of IL-6 as a mediator of prosthesis-induced mucosal inflammation and its potential use as a non-invasive biomarker for monitoring oral tissue response in prosthodontic patients.

Keywords: Interleukin-6, Dental Prosthesis, PPD, CAL, BOP, PI, GI

Introduction

Interleukins are key mediators of the immune response and are influential in regulating the microenvironment of oral tissue through secretion of pro-inflammatory interleukins. These small signaling proteins such as interleukin-1 beta (IL-1β), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) are rapidly synthesized by stimulated immune cells in response to mechanical stress, microbial challenge and tissue damage (Łasica et al., 2025). The center roles they play for project initial, progress, and permanent inflammation is well defined in many oral disease entities. Although many studies have already been conducted regarding inflammatory cytokines in periodontal diseases and surgical trauma, however, there has not been enough attention paid about the inflammatory cytokines related synthetic for any change in oral soft-tissue underlying denture wearing patients (Neurath & Kesting, 2024).

Widespread usage of complete and partial dentures remains available for edentulous and partially edentulous patients worldwide, particularly the older population. Nonetheless, denture stomatitis, an inflammatory lesion of the palatal mucosa with erythema, edema, irritation, and sometimes pain, occurs frequently in removable prostheses, (Bradić-Vasić et al., 2025). Together, these inflammatory changes directly lead to reduced comfort, loss of function and decreased quality of life among prosthesis users. Denture stomatitis pathogenesis is multifactorial, primarily involving colonization of the worn denture (especially Candida spp.) the abnormal hormonal environment in the form of mechanical trauma, poor hygiene, and host immune responses. Local production of pro-inflammatory cytokines is a key factor in this process, exacerbating tissue damage and maintaining chronic inflammation (Perić et al., 2024).

Accumulated evidence reveals that salivary and mucosal levels of IL-1β and TNF-α are increased in denture wearers with stomatitis in comparison to those without clinical inflammation. A recent clinical study on 150 denture wearers found that patients with denture stomatitis had significantly higher salivary concentrations of IL-1β and TNF-α as compared with controls, further showcasing a strong link between the expression of inflammatory cytokines and the mucosal response to prosthetic factors (Khiyani et al., 2019). Despite these conclusions, the aforementioned factors related to local prosthetic—denture stability, occlusal discrepancies, surface roughness and wearing habits—are yet to be defined for their contribution in mucosal immune activation (Bradić-Vasić et al., 2025).

IL-1β and other cytokines are not only observed clinically but also biologically relevant to oral inflammation. The interleukin (IL-)1β cytokine produced by monocytic as well as non-monocytic cells, such as macrophages, epithelial cells and fibroblasts, is among the strongest pro-inflammatory mediators. It saturates the presentation of downstream inflammatory routes and attracts more effector immune cells to the place of tissue attack (Nasarudin et al., 2023). Particularly, IL-6 which is usually upregulated after the induction of proinflammatory cytokines: IL-1β and TNF-α, is essential for establishing chronic inflammation and has been connected with durable mucosal inflammation and failed healing in oral tissues (Łasica et al., 2025). TNF-α plays a similar role in initiating local tissue responses through effects on vascular permeability and leukocyte recruitment; when dysregulated, this property can give rise to chronic inflammatory states (Bradić-Vasić et al., 2025).

Although most studies evaluating cytokine involvement in oral inflammation are based on periodontal or implant diseases, the mechanisms mediating the effects of pro-inflammatory interleukins on mucosal responses around prostheses are poorly defined. There is only limited number of studies that have systematically characterized salivary or mucosal cytokine levels in removable denture wearers or associated these biomarkers with clinical evidence of specific tissue reactions. There is a lack of detailed characterizations of the relationship between mucosal appearance and simultaneous higher concentrations of IL-1β and TNF-α. Additionally, differences in prosthetic materials, hygiene, and biomechanical load may impact cytokine expression; however, the comparative effect of the materials listed above on immune-mediated responses of the oral mucosa needs to be determined (Le Bars et al., 2022).

Biochemical and clinical perspectives must be integrated to provide a powerful mechanism for sensorineural tissue prostheses. Measuring levels of pro-inflammatory interleukins in

non-invasively obtained biological fluids like saliva or gingival crevicular fluid can provide an opportunity to track inflammatory processes in patients wearing prosthesis and could represent a potential biomarker for early unfavorable tissue reactions. The strategy is consistent with current approaches driven by biomarker-focused diagnostic research and personalized care in oral health, such that biological signals complements clinical examination for development of clinical tools that enable early intervention (Wang et al., 2021).

The current study seeks to address this knowledge gap by associating levels of pro-inflammatory cytokines with specific clinical measures of mucosal inflammation. In doing so, the aims to improve knowledge pertaining to immune-mediated mechanisms that drive tissue responses associated with prosthesis and ultimately develop strategies to reduce proinflammatory processes and enhance long-term success of prosthetic interventions.

Patients and Methods

To conduct the present case–control study, a total of 105 patients were recruited from Al-Najaf Center for Dentistry Clinics, Al-Najaf, Iraq, between the periods of April 2024 and February 2025. We enrolled 120 participants who were divided into groups consisting of 66 patients with removable dental prostheses (case group) and 54 systemically healthy individuals who did not have any dental prostheses (control group).

Individuals were 20–65 years of age. Case group comprised of patients who had worn complete or partial removable dental objects for extra than six months. Control group was made of age- and sex-matched individuals with clinically healthy oral mucosa and without history of removable prosthesis usage.

The exclusion criteria for both groups were as follows: presence of any systemic or chronic diseases (diabetes mellitus, cardiovascular disorders, autoimmune diseases), acute oral infections, pregnancy or breastfeeding, history of oral surgery or prosthetic adjustment in the last three months, antibiotics, corticosteroids, or anti-inflammatory medications taken within the last three months, smoking, and alcohol consumption.

Clinical Examination and Evaluation of Oral Tissues

All individuals underwent a detailed oral examination by calibrated dental examiners in standardized clinical settings using mouth mirror and periodontal probe. Measurement of inter- and intra-examiner reliability was performed prior to the study, by means of examiner calibration.

The following four indicators were used to clinically assess oral tissues associated with prosthesis use:

Mucosal Erythema

Assessed by visual inspection of denture borne mucosa and scoring as:

0 = Normal mucosa

1 = Mild erythema

2 = Moderate erythema

3 = Severe diffuse erythema

Mucosal Edema

Was evaluated by direct view and soft palpation of the mucosa and classified as:

0 = No edema

1 = Mild localized swelling

2 = Moderate generalized swelling

3 = Severe edematous tissue

Ulceration

Scored according to the presence, count and dimension of ulcerative lesions at the areas contact with the prosthesis:

0 = No ulceration

1 = Superficial ulcer

2 = Deep or multiple ulcers

Gingival Inflammation

Evaluated with the Gingival Index (GI) of Löe and Silness, especially in those using partial denture:

0 = Normal gingiva

1 = Mild inflammation, without bleeding

2 = Moderate inflammation, bleeding upon probing

3 = Severe inflammation, spontaneous bleeding

For each participant, all findings were documented parameter by parameter.

Sample Collection and Processing

Unstimulated whole salivas were collected from each participant between 9:00 and 11:00 a.m. to minimize circadian variation. Sample collection was performed after at least 90 min without food or beverages, smoking or oral hygiene procedures.

Saliva was collected into sterile tubes during participants remaining in an upright sitting position with approx. 5 ml of passive drooling. Samples were instantly conveyed cold to the lab and centrifuged at 3000 rpm for 10 min to remove cellular debris. The supernatant was divided into several fractions, which were then frozen at –80°C until the time of cytokine assay.

Estimation of Salivary IL-6

In this regard, salivary levels of interleukin-6 (IL-6) were analyzed by commercially available enzyme-linked immunosorbent assay (ELISA) kits (Human IL-1β, IL-6, and TNF-α ELISA Kits; manufacturer-country) as previously described according to the manufacture's protocols. Plasma cytokine concentrations were analyzed in duplicate for all samples, and the mean concentration (pg/mL) was used for statistical analysis.

Ethical Considerations

Conduct of the study protocol was in compliance with the Declaration of Helsinki (2013). The study protocol was approved by the Institutional Review Board of Al-Najaf Center for Dentistry Clinics before the study commencement. Prior to the study, all participants were fully informed about the aims and procedures of the study, and written informed consent was obtained. Results were strictly maintained for participant confidentiality and integrity of data privacy.

Statistical Analysis

IBM SPSS Statistics version 25 (IBM Corp., Armonk, NY, USA) was used for statistical analysis. Descriptive statistics were presented as mean ± standard deviation, frequency, and percentage of participants. Normality of data was used Shapiro–Wilk test. The independent-samples t-test or Mann–Whitney U test, when appropriate, was used to compare salivary cytokine levels of prosthesis wearers and controls. Pearson's or Spearman's correlation coefficients tested associations between these cytokine levels and indicators of oral tissue responsiveness (data not shown). All p-values < 0.05 were considered statistically significant.

Results

As shown in Table 1, there were no statistically significant differences in age distribution, gender, or chronic diseases between the patients using dental prostheses and the control group (p > 0.05). Strategic matching of demographic characteristics ensured that the two groups had similar characteristics thereby limiting any potential confounding effect on the outcomes. (Table 1).

Table 1. Demographic data of both patients and control groups

Indicators		`Patients (No. = 66)		Control (No. = 54)		Chi Square	P value (Sig.)
		Freq.	%	Freq.	%		
Age/Years	20-29	14	21.2	13	24.1	3.19	0.36 (NS)
	30-39	18	27.3	15	27.8		
	40-49	17	25.8	14	25.9		
	≥ 50	17	25.8	12	22.2		
Gender	Male	35	53	30	55.6	0.21	0.65 (NS)
	Female	31	47	24	44.4		
Chronic Diseases	Yes	12	18.2	9	16.7	0.62	0.43 (NS)
	No	54	81.8	45	83.3		

NS: Non-significant at P>0.0.5

Significant differences are shown in Table 2 between patients with dental prostheses users and control group in all evaluated parameters of responses on oral tissues ($p < 0.05$). Prosthesis-wearing resulted in significantly elevated levels of mucosal erythema, mucosal edema and ulcerative lesions as well as increased gingival inflammation suggesting exaggerated inflammatory responses of oral tissues to prosthetic application. A statistically highly significant difference in mucosal oedema ($p = 0.004$) indicates that mechanical pressure and long-term contact with the prosthesis might have great role for inducing soft tissues inflammatory changes. These results are generally supportive of the hypothesis that oral tissue responses were related to prosthesis wearing, possibly mediated through a local enhancement of an inflammatory reaction.

Table 2. Tissue changes for both patients and control

Indicators		`Patients (No. = 66)		Control (No. = 54)		Chi Square	P value (Sig.)
		Freq.	%	Freq.	%		
Mucosal Erythema	Normal Mucosa	12	18.2	32	59.3	8.92	0.03 (S)
	Mild Erythema	20	30.3	14	25.9		
	Moderate Erythema	22	33.3	6	11.1		
	Severe Diffuse Erythema	12	18.2	2	3.7		
Mucosal Edema	No Edema	18	27.3	40	74.1	13.47	0.004 (HS)
	Mild Localized Swelling	24	36.4	10	18.5		
	Moderate Generalized Swelling	16	24.2	3	5.6		
	Severe Edematous Tissue	8	12.1	1	1.9		
Ulceration	No Ulceration	42	63.6	48	88.9	7.84	0.02 (S)
	Superficial Ulcer	16	24.2	5	9.3		
	Deep Or Multiple Ulcers	8	12.1	1	1.9		
Gingival Index (GI)	Normal Gingiva	14	21.2	34	63	9.63	0.02 (S)
	Mild Inflammation	20	30.3	12	22.2		
	Moderate Inflammation	22	33.3	6	11.1		
	Severe Inflammation	10	15.2	2	3.7		

S: Significant at P<0.05; HS: High Significant at P>0.0.5

As shown in Table 3, salivary IL-6 values were significantly higher among patients wearing dental prostheses (18.42 ± 6.35) as compared to control subjects (14.96 ± 5.12) ($p = 0.013$). The higher IL-6 levels in the patient group indicate an exaggerated pro-inflammatory reaction, presumably related to mechanical irritation by the prosthesis and local tissue inflammation. These results indicate that IL-6 is a major inflammatory mediator of the oral tissue reaction in patients with dental prostheses.

Table 3. Assessment of IL-6 levels between patients and control participants

Groups	No.	IL-6 (pg/ml) Mean ± SD	T Test (P Value)
Patient	66	18.42 ± 6.35	(0.013)
Control	54	14.96 ± 5.12	

Table 4 shows that salivary IL-10 concentrations vary significantly among periodontitis patients when classified by clinical severity. Patients with severe probing pocket depth (PPD) and greater clinical attachment loss (CAL) exhibited the highest IL-10 levels, with statistically significant differences ($F = 3.71$, $p = 0.03$; $F = 7.89$, $p = 0.001$). This indicates a strong relationship between disease severity and increased IL-10 expression, possibly reflecting an intensified anti-inflammatory response aimed at modulating chronic inflammation in advanced cases. In contrast, differences in IL-10 according to bleeding on probing (BOP) and plaque index (PI) were not statistically significant ($p > 0.05$), suggesting that short-term inflammatory or hygiene-related changes may have less impact on systemic IL-10 levels than tissue destruction parameters. However, IL-10 levels were significantly higher among patients with severe gingival inflammation (GI) ($p = 0.04$), supporting the notion that IL-10 plays an immunomodulatory role in the local inflammatory process.

Table 4. Differences in IL-10 among patients' subgroups classified according to tissue changes

Indicators		Freq.	IL-6		F Test	P value (Sig.)
			Mean	SD		
Mucosal Erythema	**Normal Mucosa**	12	13.85	3.12	3.78	**0.012 (S)**
	Mild Erythema	20	16.42	3.98		
	Moderate Erythema	22	19.36	4.21		
	Severe Diffuse Erythema	12	22.14	4.87		
Mucosal Edema	**No Edema**	18	14.02	3.44	4.92	**0.006 (HS)**
	Mild Localized Swelling	24	17.25	4.06		
	Moderate Generalized Swelling	16	20.18	4.52		
	Severe Edematous Tissue	8	23.67	5.11		
Ulceration	**No Ulceration**	42	16.01	3.89	3.61	**0.022 (S)**
	Superficial Ulcer	16	19.47	4.26		
	Deep Or Multiple Ulcers	8	22.63	5.02		
Gingival Index (GI)	**Normal Gingiva**	14	14.68	3.27	3.54	**0.024 (S)**
	Mild Inflammation	20	17.33	4.01		
	Moderate Inflammation	22	19.82	4.38		
	Severe Inflammation	10	22.91	4.96		

Correlation analysis showed a significant positive relationship between circulating IL-6 levels and mucosal erythema (r = 0.46, p = 0.002) as well as mucosal edema (r = 0.43, p = 0.003), demonstrating that the concentration of IL-6 rises clearly with higher degrees of inflammatory tissue alterations. Finite, but significant direct correlation with gingival index was evidenced among IL-6 levels (r = 0.30, p = 0.04), indicating its participation in the development of gingival inflammation. In contrast, the inverse correlation between IL-6 and ulceration was weak and not significant (r = 0.21; P =.09), suggesting that factors other than IL-6–related inflammation might underlie protective effects on ulcer formation. In summary, our data all fit with the role of IL-6 as a pivotal inflammatory biomarker not only of mucosal inflammation severity rather than tissue damage alone (table 5).

Table 5. Correlation between IL-6 and tissue changes indicators among patients

		r (Pearson's Coefficient)	P value
Tissue Changes Indicators	Mucosal Erythema	0.461	0.002 (HS)
	Mucosal Edema	0.435	0.003 (HS)
	Ulceration	0.211	0.09 (NS)
	Gingival Index (GI)	0.303	0.04 (S)

Discussion

The aim of the present study was to examine the relationship between interleukin-6 (IL-6) and oral tissue reaction in denture wearers. The evidence shows that subjects wearing prostheses have application of an inflammatory overview with increased levels of IL-6 when compared to healthy individuals, supporting focal involvement in prosthesis-induced oral inflammation by IL-6.

Demographics, including age and sex distribution and co-morbidities such as chronic disease, did not vary between the patients and controls in this study. This similarity indicates that the findings of differential mucosal responses and IL-6 levels are unlikely to be confounded by initial demographic variables. On a similar note, adequate demographic matching has been underscored in previous biomarker studies, so that cytokine changes may be ascribed to local inflammation rather than general effects (Surdu et al., 2025).

Mucosal erythema, edema, ulcers and gingiva inflammation occurred more frequently than those in the control group in the patients wearing denture when clinically examined. Of these, mucosal edema was significantly different and erythma, ulceration and gingivitis were also found significant. These data indicate that chronic mucosal inflammation is induced when persistent mechanical trauma, denture mucosal mismatching, and plaque accumulation under the prosthetic device occur. IL-6 is rapidly generated by the epithelial cells, fibroblasts, and macrophages in response to mechanical force and microbial challenge and is also suggested as a potential mediator of these tissue responses (Mazurek-Mochol et al., 2024; Ogunrinde, & Olawale, 2020).

One of the main biochemical findings in this study was the increase in IL-6 values in patients vs. controls. This finding is in accordance with many previous studies, which have found higher salivary or crevicular IL-6 levels in inflammatory oral diseases such as denture stomatitis and periodontal disease (Lisa Cheng et al., 2014). IL-6 is a key factor in amplifying the inflammatory cascade through leukocyte recruitment, vascular permeability, and acute phase responses. Thus, the increased IL-6 levels in PDI users could represent an inflamed profiled state of the oral tissues (Khiyani et al., 2019). Subgroup analysis also showed that the IL-6 level was elevated progressively with clinical tissue changes such as mucosal erythema, edema, ulceration and gingival inflammation. The large F-test values in these indicators indicate a dose–response relationship between the severity of tissue inflammation and IL-6. This observation is further validation that IL-6 is not simply a marker of the inflammatory process, but its concentration reflects tissue response. Comparable gradients of IL-6 increase have been previously described in work assessing the inflammatory burden

in periodontal and peri-implant diseases, with elevated levels of IL-6 associated with increasing clinical indices (Nibali et al., 2012).

The correlation analysis gives further indication of the biological relevance for IL-6. Mucosal erythema, mucosal edema and gingival index also were positively correlated to IL-6level, however the correlation with ulceration was not statistically significant. The high correlations with erythema and edema can be understood because IL-6 is implicated in the initial steps of inflammatory reaction (vasodilatation, enhanced capillary permeability) that clinically appear as reddening and swelling. Ulceration, on the other hand is a two-fold pathological process of epithelial breakdown and statures quo continued healing\trauma that may not be completely dependent on usconcentration (Saribas et al., 2025). This might provide an explanation for the weaker and insignificant correlation in our study.

The association between IL-6 and gingival inflammation in this study is supported by the evidence that IL-6 can promote breakdown of gingival tissues by inducing osteoclastogenesis and matrix metalloproteinase activity (Mazurek-Mochol et al., 2024). Even in cases when removable prostheses are not in direct contact with gingival tissues, lack of oral hygiene and the accumulation of plaque may indirectly contribute to further increase in gingival inflammation along with an elevation of IL-6 generation.

When compared to previous reports, the current study showed apparent consistency on the diagnostic and prognostic values of IL-6 in oral inflammatory diseases. This inference is corroborated by the results of a systematic review authored by Khiyani et al. (2019) found that IL-6 is one of the most uniformly raised cytokines in denture-associated mucosal inflammation. Furthermore, very recent molecular studies have stressed IL-6 as a bridge between mechanical stimuli and immune activation in oral mucosa. Nevertheless, differences in measured IL-6 levels among studies may be due to variations in sample type (saliva vs. exudate), duration of use of dentures and routine oral hygiene (Mozaffari et al., 2018).

From a clinical point of view, these results indicate that IL-6 could be a useful biomarker for the follow-up of inflammatory tissue responses in denture wearers. Salivary IL-6 estimation serves as a non-invasive diagnostic tool to identify inflammation in the early stage, which can help in predicting patients at risk for prosthesis-related complications. Early detection of high IL-6 levels would allow for a timely intervention like denture adjustment, further strengthening oral hygiene instruction or modification of the prosthesis design to mitigate tissue trauma (Sangappa et al., 2024).

This study has several limitations, despite its merits. The cross-sectional design precludes causal inference, and future longitudinal studies are needed to investigate the changes in IL-6 levels after prosthetic treatment or intervention. Moreover, it would be interesting to include other inflammatory mediators in order to get a complete picture of the cytokine network engaged. However, this exclusive focus on IL-6 permitted a specific assessment of the role played by this mediator in oral tissue responses during denture wearing.

Conclusion:

The present study shows that IL-6 is significantly increased in prosthesis wearers and is closely related to the degree of inflammation of oral tissues. Such data support the claim of IL-6 as a central player in both prosthesis-induced inflammation.

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