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Comparison of Mmp-8 and Il-6 Levels in Resolved and Persistent Peri-Implant Mucositis

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ABSTRACT: Peri-implant mucositis is a plaque-associated inflammatory condition that may progress to peri-implantitis if left untreated. PICF biomarkers may complement clinical parameters in monitoring treatment response. Thus, the aim for my study was to compare PICF MMP-8 and IL-6 levels in resolved and persistent peri-implant mucositis following non-surgical therapy among 10 peri-implant mucositis sites, categorized after 3 months as resolved (Group A, n=5) or persistent (Group B, n=5). PI, BOP, PD and PICF MMP-8 and IL-6 levels were assessed at baseline and 3 months. We have found that, at 3 months, Group A showed significantly lower PI, BOP, PD, MMP-8, and IL-6 levels than Group B (all p=0.008). Group A demonstrated significant reductions in all clinical and biomarker parameters, whereas Group B showed no significant reduction in IL-6. Baseline MMP-8 and IL-6 were significantly higher in the persistent group. MMP-8 and IL-6 showed positive correlations with BOP and PD, and both demonstrated good-to-excellent discriminatory ability for distinguishing resolved from persistent mucositis. We have come to conclude that, PICF MMP-8 and IL-6 may serve as useful adjunctive biomarkers for monitoring treatment response and identifying persistent peri-implant mucositis after non-surgical therapy.

INTRODUCTION

Peri-implant mucositis is a plaque-associated inflammatory condition involving the soft tissues around dental implants (Renvert et al., 2019). It is clinically characterized by bleeding and/or suppuration on gentle probing, with or without increased probing depth (PD), but without bone loss beyond initial post-remodelling changes. Non-surgical mechanical

debridement and reinforcement of oral hygiene are considered the first-line treatment; however, clinical resolution is not uniform across all affected implants (Berglundh et al., 2018). Conventional clinical parameters, including plaque index (PI), bleeding on probing (BOP), and PD, remain essential for diagnosis and follow-up. However, these measures may not completely reflect subclinical inflammatory activity or identify sites likely to show a persistent inflammatory response after therapy (Alassy et al., 2019). Peri-implant crevicular fluid (PICF) is a readily obtainable, site-specific biological fluid that may provide additional information regarding peri-implant inflammatory status (Dursun and Tözüm., 2016). Moreover, matrix metalloproteinase-8 (MMP-8) is a neutrophil-derived collagenase associated with connective-tissue degradation, while interleukin-6 (IL-6) is a pro-inflammatory cytokine involved in local immune activation (La Monaca et al., 2025). Elevated PICF levels of MMP-8 and IL-6 have been reported in peri-implant inflammatory conditions, supporting their potential role as adjunctive diagnostic and monitoring biomarkers (Ghassib et al., 2019). Nevertheless, current evidence remains insufficient to establish whether these markers can predict disease course or response to non-surgical treatment. Despite the clinical usefulness of BOP, PI, and PD, these indicators may not reliably identify sites with persistent molecular inflammation or predict treatment response. Evaluation of MMP-8 and IL-6 in PICF may offer an objective, minimally invasive biomarker-based endpoint to complement clinical assessment. Establishing their predictive and discriminatory value could support earlier identification of non-resolving sites and help guide individualized maintenance or additional intervention.

Therefore, the present study, aims to compare PICF MMP-8 and IL-6 levels in resolved and persistent peri-implant mucositis among patients attending an Indian tertiary hospital. The study assessed the baseline biomarker levels, changes after 3 months of non-surgical therapy, associations with PI, BOP, and PD, and the diagnostic ability of individual and combined biomarkers to distinguish resolved from persistent mucositis.

Research Questions

1. Do peri-implant crevicular fluid (PICF) levels of MMP-8 and IL-6 differ between peri-implant mucositis that resolves and mucositis that persists after standard non-surgical therapy, in patients at an Indian tertiary hospital?
2. Do MMP-8 and IL-6 levels measured before treatment differ between the two groups, which would suggest they could predict how a site will respond?
3. How much do MMP-8 and IL-6 change from baseline to 3 months within each group, and does that change track changes in bleeding on probing (BOP), probing depth (PD) and plaque index?
4. Can MMP-8 and IL-6, alone or together, tell resolved and persistent mucositis apart, giving a biomarker-based endpoint to use alongside the clinical one?

LITERATURE REVIEW

Peri-implant mucositis is a plaque-associated inflammatory condition confined to the peri-implant soft tissues and is characterized clinically by BOP, with or without suppuration and increased PD, in the absence of progressive bone loss (Berglundh et al., 2018). It is regarded as a reversible precursor of peri-implantitis; hence, early diagnosis and management are essential to prevent disease progression (Renvert et al., 2019). Non-surgical mechanical debridement with reinforcement of oral hygiene is the standard first-line treatment for peri-implant mucositis (Suárez-López Del Amo et al., 2016). Published evidence indicates that this approach generally reduces plaque accumulation, BOP, and PD; however, complete resolution is not consistently achieved in all sites. This variable response highlights the limitation of relying exclusively on conventional clinical outcomes and supports the need for objective molecular indicators of persistent inflammation (Herrera et al., 2023). PICF is a useful site-specific biological medium for evaluating inflammatory and tissue-destructive processes around implants (Alassy et al., 2019). Matrix metalloproteinase-8 (MMP-8) is a neutrophil-derived collagenase involved in degradation of extracellular collagen, whereas interleukin-6 (IL-6) is a pro-inflammatory cytokine associated with immune activation and inflammatory tissue response (Keskin et al., 2020). Increased levels of these biomarkers have been reported in peri-implant inflammatory conditions compared with healthy implants (Alassy et al., 2019).

In addition to this, a systematic review and meta-analysis reported significantly higher PICF IL-6 concentrations in peri-implant mucositis than in healthy peri-implant tissues. IL-6 was further elevated in peri-implantitis. MMP-8 has also demonstrated potential as a biomarker of peri-implant inflammation and tissue breakdown, although its diagnostic threshold and longitudinal behaviour after treatment require further clarification (Ghassib et al., 2019). Few longitudinal studies have

specifically compared biomarker profiles between sites with resolved and persistent peri-implant mucositis after non-surgical therapy (Moaven et al., 2022). Thus, evaluating baseline and 3-month PICF MMP-8 and IL-6 levels, their association with PI, BOP, and PD, and their diagnostic accuracy may identify clinically useful biomarker-based endpoints. Hence, the present study investigates whether individual or combined MMP-8 and IL-6 measurements can distinguish resolved from persistent peri-implant mucositis in an Indian tertiary-care setting.

METHODOLOGY

This prospective, comparative pilot study was conducted in the tertiary care hospital in India among systemically healthy adults aged 25–65 years with at least one screw- or cement-retained implant-supported restoration in function for ≥12 months were screened, and peri-implant mucositis using 2017 World Workshop classification. Bleeding was registered by its presence at more than 1 spot, line or profuse & suppuration on gental probing without boneloss was diagnosed by periapical radiograph. Patients with peri-implantitis, uncontrolled diabetes (HbA1c >7%), systemic conditions or medications affecting inflammation or bone metabolism (immunosuppressants, bisphosphonates, corticosteroids, NSAIDs, statins), antibiotic use or periodontal/peri-implant therapy within the previous 3–6 months, smoking or smokeless tobacco use, alcohol abuse, pregnancy or lactation, untreated periodontitis, mobile or fractured implants, loose abutments or screws, or inability to attend follow-up were excluded; when multiple implants were affected, only the most inflamed implant was included, so that the patient was the unit of analysis. At baseline, a single calibrated examiner recorded the modified plaque index, modified sulcus bleeding index, BOP, suppuration and PD at six sites per implant using a manual periodontal probe at a force of approximately 0.2 N. PICF was then collected from the deepest bleeding site: the site was isolated with cotton rolls and gently air-dried, and a PerioPaper strip was inserted 1 mm into the peri-implant sulcus for 30 s. Its volume was measured with a calibrated Periotron, and the strip was eluted in phosphate-buffered saline and stored at −80 °C until analysis. All patients received a standardized non-surgical protocol consisting of oral hygiene instruction, professional mechanical plaque removal and submucosal instrumentation with titanium or PEEK-tipped instruments, and glycine/erythritol air-polishing, without adjunctive antimicrobials. Clinical parameters and PICF sampling were repeated at 3 months, and patients were allocated by the EFP treatment endpoint into Group A (resolved; ≤1 BOP site and no suppuration, n = 5) or Group B (persistent; ≥2 BOP sites, ≥1 site with profuse BOP, or suppuration, n = 5). Enrolment was consecutive until each group reached five cases. MMP-8 (ng/mL) and IL-6 (pg/mL) were measured in duplicate using commercial human sandwich ELISA kits according to the manufacturer's instructions by a laboratory technician blinded to group allocation, and results were expressed as both concentration and total amount per site. Data were analysed using IBM SPSS Statistics (version 26) with significance set at $p < 0.05$. Normality was assessed with the Shapiro–Wilk test, and data were presented as mean ± SD and median (IQR). Intergroup comparisons were made with the Mann–Whitney U test (exact p-values), intragroup changes from baseline to 3 months with the Wilcoxon signed-rank test, and categorical variables with Fisher's exact test. Effect sizes were reported as $r = Z/\sqrt{N}$ with 95% confidence intervals. Associations between biomarkers and clinical parameters were assessed with Spearman's rank correlation, and the ability of MMP-8 and IL-6 to discriminate resolved from persistent mucositis was explored with receiver operating characteristic (ROC) curve analysis (AUC, cut-off, sensitivity and specificity).

RESULTS

TABLE 1(A) : BASELINE CHARACTERISTICS & NORMALITY

VARIABLE	GROUP A RESOLVED	GROUP B PERSISTENT	TEST	P- VALUE
Age	46.60±6.80/47.00 (42.00-51.00)	52.80±6.83/52.00 (49.00-58.00)	Mann- Whitney U=6.0	0.222
Male/ Female	3/2	2/3	Fisher's Exact	1.000
Maxilla/Mandible	2/3	3/2	Fisher's Exact	1.000
Retention, Screw/ Cement	3/2	1/4	Fisher's Exact	0.524
Suppuration at 3 Months, Present/Absent	0/5	2/3	Fisher's Exact	0.444

Table 1 (A) showed that, The resolved and persistent groups were comparable for age, sex, jaw location, restoration retention, and 3-month suppuration status, with no statistically significant differences as th p value was 0.222, 1.000, 1.000, 0.524 and

0.444 respectively. Although persistent cases were older and more often cement-retained, these trends were not statistically significant.

TABLE 1(B) : SHAPIRO-WILK (Group A/Group B)

PARAMETER	P- VALUE
MMP-8 (Baseline)	0.991/0.960
IL-6 (Baseline)	0.972/0.823
PD (Baseline)	1.000/0.884
BOP (Baseline)	0.314/0.314
BOP (at 3 Month)	0.006/0.815

Table 1 (B) showed that baseline MMP-8, IL-6, and probing depth values were normally distributed in both groups (p>0.05). Baseline bleeding on probing was also normally distributed. At 3 months, BOP was non-normally distributed in Group A (p=0.006) but normally distributed in Group B (p=0.815); therefore, non-parametric analysis may be appropriate for between-group comparison of 3-month BOP.

TABLE 2: INTERGROUP COMPARISON

PARAMETER	TIME	GROUP A	GROUP B	U	EXACT P	R (95% Cl)
PI	Baseline	1.26±0.24/1.20 (1.10-1.40)	1.62±0.29/1.69 (1.40-1.80)	4.0	0.095	0.53 (0.00-0.87)
PI	3 Month	0.44±0.11/0.40 (0.40-0.50)	1.06±0.24/1.00 (0.90-1.20)	0.0	0.008	0.80 (0.33-0.95)
BOP (%)	Baseline	63.34±13.94/66.70 (50.00-66.70)	80.00±13.92/83.30 (66.70-83.30)	5.0	0.151	0.48 (0.00-0.85)
BOP (%)	3 Month	6.68±9.15/0.00 (0.00-16.70)	56.66±19.01/50.00 (50.00-66.70)	0.0	0.008	0.81 (0.36-0.95)
PD (mm)	Baseline	4.00±0.38/4.00 (3.80-4.20)	4.56±0.36/4.60 (4.30-4.80)	3.0	0.056	0.59 (0.00-0.89)
PD (mm)	3 Month	3.12±0.19/3.10 (3.00-3.20)	4.24±0.36/4.20 (4.00-4.50)	0.0	0.008	0.79 (0.33-0.95)
MMP-8 (ng/mL)	Baseline	155.48±12.23/155.60 (148.20-162.50)	210.54±22.14/210.30 (196.40-224.80)	0.0	0.008	0.79 (0.33-0.95)
MMP-8 (ng/mL)	3 Month	67.76±9.23/68.58 (62.40-71.80)	187.84±20.64/189.40 (171.60-198.20)	0.0	0.008	0.79 (0.33-0.95)
IL-6 (pg/mL)	Baseline	20.08±2.65/19.70 (18.60-21.40)	28.22±5.52/28.90 (26.30-31.80)	2.0	0.032	0.66 (0.05-0.91)
IL-6 (pg/mL)	3 Month	8.00±1.44/8.00 (7.20-8.90)	25.24±3.93/25.30 (22.10-27.60)	0.0	0.008	0.79 (0.33-0.95)

Table 2 showed that, at 3 months, Group A showed significantly lower PI, BOP, PD, MMP-8, and IL-6 than Group B, with large effect sizes. Baseline MMP-8 and IL-6 were also significantly higher in Group B, whereas baseline PI, BOP, and PD were comparable.

TABLE 3 : INTRAGROUP CHANGE

GROUP	PARAMETER	CHANGE	REDUCTION	Z	P-VALUE	R (95% Cl)
A	PI	0.82±0.16	65.2	-2.03	0.042	0.64 (0.02-0.91)
A	BOP	56.66±9.12	91.0	-2.06	0.039	0.65 (0.04-0.91)
A	PD	0.88±0.47	21.3	-2.02	0.043	0.64 (0.02-0.90)
A	MMP-8	87.72±3.12	56.6	-2.02	0.043	0.64 (0.02-0.90)
A	IL-6	12.08±2.18	60.0	-2.02	0.043	0.64 (0.02-0.90)
B	PI	0.56±0.05	35.0	-2.06	0.039	0.65 (0.04-0.91)
B	BOP	23.34±9.14	30.3	-2.03	0.042	0.64 (0.02-0.91)
B	PD	0.32±0.04	7.0	-2.12	0.034	0.67 (0.07-0.91)
B	MMP-8	22.70±3.22	10.8	-2.02	0.043	0.64 (0.02-0.90)
B	IL-6	2.98±7.53	6.7	-0.94	0.345	0.30 (0.00-0.78)

Group A demonstrated significant reductions in PI, BOP, PD, MMP-8, and IL-6 from baseline to 3 months, with large effect sizes. Group B also showed significant improvements in PI, BOP, PD, and MMP-8; however, the reductions were smaller than in Group A. IL-6 reduction in Group B was not statistically significant (p=0.345p = 0.345p=0.345). Overall, Group A exhibited greater clinical and biochemical improvement.

TABLE 4 (A) : Spearman Correlation

PAIR	BASELINE	3 MONTHS
MMP-8 vs BOP	0.864 (0.001)	0.982 (<0.001)
MMP-8 vs PD	0.467 (0.174)	0.564 (0.090)
IL-6 vs BOP	0.553 (0.097)	0.704 (0.023)
IL-6 vs PD	0.358 (0.310)	0.794 (0.006)
MMP-8 vs IL-6	0.721 (0.019)	0.685(0.029)

Table 4 (A) showed that, at baseline, MMP-8 showed a significant positive correlation with BOP and IL-6, while other correlations were non-significant. At 3 months, MMP-8 remained strongly correlated with BOP and IL-6. IL-6 showed significant positive correlations with BOP and PD, indicating that higher inflammatory biomarker levels were associated with greater clinical inflammation.

TABLE 4 (B) : ROC ANALYSIS

BIOMARKER	AUC (95%Cl)	P-VALUE	CUT-OFF	SENSITIVITY	SPECIFICITY
Baseline MMP-8	1.00 (1.00-1.00)	0.008	177.0 ng/mL	100%	100%
Baseline IL-6	0.92 (0.73-1.00)	0.032	21.85 pg/mL	80%	80%
3 Month MMP-8	1.00 (1.00-1.00)	0.008	122.25 ng/mL	100%	100%
3 Month IL-6	1.00 (1.00-1.00)	0.008	15.3 pg/mL	100%	100%

Table 4 (B) showed that, at baseline and 3-month MMP-8 demonstrated excellent discriminatory ability between groups, with an AUC of 1.00, 100% sensitivity, and 100% specificity. IL-6 also showed good baseline accuracy (AUC = 0.92) and perfect discrimination at 3 months (AUC = 1.00). These biomarkers may effectively predict treatment outcome.

DISCUUSION

The present study demonstrated significant reductions in PI, BOP, PD, MMP-8, and IL-6 after non-surgical therapy in the resolved group. Although the persistent group showed improvement in PI, BOP, PD, and MMP-8, the magnitude of reduction was smaller, and IL-6 did not decrease significantly. These findings indicate that clinical resolution is accompanied by a substantial reduction in local inflammatory and collagenolytic activity in PICF. The significantly lower 3-month MMP-8 and IL-6 levels in the resolved group support the potential value of these biomarkers for monitoring treatment response. Similar findings were observed by another study and showed significant reductions in BOP, IL-6, and MMP-8 after mechanical debridement of peri-implant mucositis at 3 months (Isola et al., 2025). However, their study did not identify an additional benefit from adjunctive photodynamic therapy (Isola et al., 2025), whereas the present study specifically differentiated sites according to resolution or persistence of mucositis. Furthermore, baseline MMP-8 and IL-6 values were higher in the persistent group, suggesting that greater pre-treatment inflammatory burden may be associated with an unfavourable therapeutic response. This observation is consistent with another study where authors reported that there was elevated PICF IL-6 concentrations in peri-implant mucositis compared with healthy implants, supporting its role as a marker of peri-implant inflammation. In contrast, previous evidence has reported inconsistent ability of MMP-8 to discriminate between peri-implant mucositis and peri-implantitis, indicating that biomarker values should be interpreted together with clinical findings (Ghassib et al., 2019). Moreover, the strong positive correlations between MMP-8, IL-6, BOP, and PD at 3 months further suggest that these PICF biomarkers reflect ongoing peri-implant inflammation. The excellent ROC performance observed for 3-month MMP-8 and IL-6 indicates their possible role as adjunctive markers for distinguishing resolved from persistent mucositis. However, the small sample size and single-centre design necessitate cautious interpretation and validation in larger longitudinal multicentre studies. Thus, PICF MMP-8 and IL-6 appear to be promising adjunctive biomarkers for monitoring therapeutic response and identifying persistent peri-implant mucositis after non-surgical therapy.

LIMITATIONS

The study was of small sample size, single-centre design, and short 3-month follow-up, which may restrict generalizability and statistical power. Potential confounders, including implant surface characteristics, prosthetic design, oral hygiene compliance, and patient-related risk factors, may have influenced outcomes. The diagnostic accuracy of MMP-8 and IL-6 requires

validation in larger, multicentre longitudinal studies.

CONCLUSION

The non-surgical therapy resulted in significant clinical and biochemical improvement in resolved peri-implant mucositis. The resolved group demonstrated greater reductions in PI, BOP, PD, MMP-8, and IL-6 than the persistent group. Higher baseline and 3-month MMP-8 and IL-6 levels were associated with persistent inflammation, and both biomarkers showed good discriminatory potential, particularly at 3 months. PICF MMP-8 and IL-6 may serve as useful adjunctive biomarkers alongside clinical parameters for monitoring treatment response and identifying persistent peri-implant mucositis. Larger multicentre studies with longer follow-up are required to validate these findings.

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