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Salivary omentin-1 following non-surgical periodontal therapy: Chronic periodontitis and type 2 diabetes mellitus

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Abstract:
Chronic periodontitis (CP) and type 2 diabetes mellitus (T2DM) exhibit a bidirectional inflammatory relationship, but the utility of salivary omentin-1 as a biomarker linking these conditions remains inadequately defined. Therefore, it is of interest to evaluate the salivary and serum omentin-1 levels in patients with CP with and without T2DM and assessed their changes following non-surgical periodontal therapy (NSPT). A prospective interventional study included 100 participants (35–50 years) allocated into four groups (n=25 each): CP+T2DM, CP only, healthy+T2DM and healthy controls, with omentin-1 quantified by ELISA at baseline and 3 months after NSPT. Thus, salivary omentin-1 is a promising non-invasive biomarker for monitoring periodontal inflammation and metabolic dysregulation, supporting its potential use for evaluating therapeutic response after NSPT.

Keywords: Chronic periodontitis (CP), type 2 diabetes mellitus, omentin-1, saliva, biomarker, non-surgical periodontal therapy

Background:
Chronic periodontitis (CP) is a multifactorial inflammatory disease leading to progressive destruction of tooth-supporting structures through dysbiotic biofilm and host immune dysregulation **[1].** Periodontitis has been increasingly linked to systemic metabolic conditions, with type 2 diabetes mellitus (T2DM) representing the most extensively studied bidirectional relationship **[2, 3].** Persistent hyperglycemia intensifies periodontal inflammation via advanced glycation end-products and oxidative stress, while periodontal inflammation worsens insulin resistance by elevating TNF-α and IL-6 **[4, 5].** Omentin-1 is an anti-inflammatory, insulin-sensitizing adipokine predominantly expressed in visceral adipose tissue whose circulating levels are reduced in obesity, insulin resistance and T2DM **[6, 7].** Emerging evidence suggests that periodontal inflammation may suppress omentin-1 expression, yet simultaneous salivary and serum assessment before and after NSPT in both diabetic and non-diabetic CP populations is lacking **[8, 9].** Because saliva is non-invasive and reflects systemic physiological changes, salivary biomarkers are increasingly explored for disease monitoring in both oral and systemic conditions **[10, 11].** Therefore, it is of interest to evaluate the salivary and serum omentin-1 levels in CP patients with and without T2DM, assessed the effect of NSPT and explored the correlation between omentin-1 and clinical periodontal parameters.

Materials and Methods:
This prospective interventional study was conducted in the Dental Outpatient Department, AIIMS Kalyani, West Bengal, with Institutional Ethics Committee approval (No.: MRU/RP/59) and in accordance with the Declaration of Helsinki. One hundred participants aged 35-50 years were enrolled and assigned equally to four groups: CP with T2DM, CP without T2DM, periodontally healthy with T2DM and periodontally healthy without T2DM (n=25 each). Sample size was calculated using OpenEpi (95% CI, 80% power) based on prior omentin-1 data (80±45 pg per mL healthy vs 60±20 pg per mL in CP), yielding a minimum of 96, rounded to 100. Inclusion criteria were age 35-50 years with or without T2DM and CP; exclusion criteria were systemic immunocompromising conditions, periodontal therapy within 6 months and pregnancy or lactation. Clinical periodontal parameters (PI, GI, BOP and PPD, CAL) were recorded at baseline and 3 months by a calibrated examiner (intra-examiner ICC > 0.85); CP was diagnosed based on PPD, CAL and BOP. Unstimulated whole saliva (5 mL, passive drool, 9:00-10:00 a.m.) and venous blood (5 mL) were collected, centrifuged at 5000 rpm for 10 minutes and stored at −80°C. Glycemic status was assessed by fasting blood glucose and HbA1c via high-performance liquid chromatography; participants with HbA1c ≥6.5% were classified as diabetic per ADA criteria. NSPT (full-mouth scaling and root planning plus oral hygiene instructions) was performed in both CP groups; healthy groups received no active periodontal treatment. Omentin-1 was quantified using ELISA (Elabscience Biotechnology Inc., USA; range 15.6-1000 pg per mL, sensitivity 9.38 pg per mL, intra or inter-assay CV <8 %/< 10%). Statistical analysis was performed with SPSS v25.0; one-way ANOVA with Tukey post-hoc and paired t-test were used for inter- and intra-group comparisons; Pearson correlation, multiple linear regression and ROC analysis were applied; P<0.05 was significant.

Results:
All 100 participants (25 per group) completed the study; no dropouts were recorded; age (P=0.84) and sex distribution (P=0.92) were comparable across groups **(Tables 1, 2).** HbA1c and fasting blood glucose were significantly higher in diabetic groups (P<0.001), confirming appropriate metabolic grouping **(Table 3).** At baseline, CP groups showed significantly higher PI,

GI, BOP, PPD and CAL than healthy groups (P<0.001); among CP groups, the T2DM subgroup exhibited greater periodontal destruction **(Table 4).** Baseline salivary and serum omentin-1 differed significantly across groups (P<0.001); lowest levels were in CP+T2DM (42.6±11.3 pg per mL salivary; 58.3±14.9 pg per mL serum) and highest in healthy controls (89.6±15.2; 118.4±18.9 pg per mL) **(Table 5).** Following NSPT, PI, GI, PPD and CAL were significantly reduced in both CP groups at 3 months (P<0.001), with greater improvement in the non-diabetic group **(Table 6).** Both CP groups showed significant post-NSPT increases in salivary and serum omentin-1 (P<0.001); in CP+T2DM, salivary omentin-1 rose from 42.6±11.3 to 61.4±13.6 pg per mL and serum from 58.3±14.9 to 81.9±16.8 pg per mL **(Table 7).** Pearson correlation showed significant negative associations between omentin-1 and PPD (r=−0.67 salivary; r=−0.63 serum) and CAL (r=−0.69 salivary; r=−0.65 serum) (P<0.001) **(Table 8).** Multiple linear regression identified CAL, PPD and diabetic status as independent predictors of salivary omentin-1 (adjusted R^2=0.48) **(Table 9).** ROC analysis demonstrated good diagnostic performance for salivary omentin-1 (AUC=0.86, sensitivity 82%, specificity 79%) and serum omentin-1 (AUC=0.89, sensitivity 85%, specificity 83%) in distinguishing periodontitis from healthy controls **(Table 10).**

Table 1: Age distribution of study participants

Group	Mean Age (years) ± SD	p-value
CP + T2DM	42.1 ± 4.3	
CP + No T2DM	41.6 ± 4.1	
No CP + T2DM	42.4 ± 3.9	
No CP + No T2DM	41.8 ± 4.0	0.84

Table 2: Gender distribution of study participants

Gender	CP + T2DM	CP + No T2DM	No CP + T2DM	No CP + No T2DM	p-value
Male	14	13	15	14	
Female	11	12	10	11	0.92

Table 3: Glycemic characteristics of study participants

Group	HbA1c (%) Mean ± SD	FBG (mg/dL) Mean ± SD	p-value
CP + T2DM	8.2 ± 1.1	168.4 ± 24.6	
CP + No T2DM	5.3 ± 0.4	96.2 ± 10.3	
No CP + T2DM	7.8 ± 0.9	158.6 ± 22.8	
No CP + No T2DM	5.1 ± 0.3	92.4 ± 8.7	<0.001

Table 4: Baseline periodontal parameters among study groups

Parameter	CP + T2DM	CP + No T2DM	No CP + T2DM	No CP + No T2DM
Plaque Index	2.34 ± 0.42	2.18 ± 0.39	1.21 ± 0.28	1.08 ± 0.24
Gingival Index	2.41 ± 0.36	2.22 ± 0.34	1.18 ± 0.26	1.04 ± 0.23
BOP (%)	68.4 ± 9.1	61.3 ± 8.4	21.6 ± 5.2	18.4 ± 4.9
PPD (mm)	5.82 ± 0.64	5.34 ± 0.58	2.84 ± 0.31	2.61 ± 0.29
CAL (mm)	6.21 ± 0.71	5.78 ± 0.65	2.96 ± 0.34	2.73 ± 0.32

P < 0.001 for all parameters. BOP=Bleeding on Probing; PPD=Probing Pocket Depth; CAL=Clinical Attachment Level.

Table 5: Baseline salivary and serum omentin-1 levels among study groups

Group	Salivary Omentin-1 (pg/mL)	Serum Omentin-1 (pg/mL)	p-value
CP + T2DM	42.6 ± 11.3	58.3 ± 14.9	
CP + No T2DM	56.2 ± 12.8	71.6 ± 15.4	
No CP + T2DM	69.2 ± 13.7	92.8 ± 16.3	
No CP + No T2DM	89.6 ± 15.2	118.4 ± 18.9	<0.001

Table 6: Intra-group comparison of periodontal parameters before and after NSPT

Parameter	CP+T2DM Baseline	CP+T2DM 3mo	CP No-T2DM Baseline	CP No-T2DM 3mo	p
Plaque Index	2.34±0.42	1.21±0.31	2.18±0.39	1.02±0.29	<0.001
Gingival Index	2.41±0.36	1.18±0.28	2.22±0.34	1.04±0.26	<0.001
PPD (mm)	5.82±0.64	3.96±0.51	5.34±0.58	3.52±0.47	<0.001
CAL (mm)	6.21±0.71	4.31±0.62	5.78±0.65	3.94±0.56	<0.001

Table 7: Intra-group comparison of salivary and serum omentin-1 before and after NSPT

Group	Salivary Baseline	Salivary 3mo	Serum Baseline	Serum 3mo	p
CP + T2DM	42.6±11.3	61.4±13.6	58.3±14.9	81.9±16.8	<0.001
CP + No T2DM	56.2±12.8	78.5±14.2	71.6±15.4	102.4±18.1	<0.001

Table 8: Correlation between omentin-1 levels and periodontal parameters

Parameter	Salivary Omentin-1 (r)	Serum Omentin-1 (r)	p-value
Probing Pocket Depth	−0.67	−0.63	<0.001
Clinical Attachment Level	−0.69	−0.65	<0.001

Table 9: Multiple linear regression analysis for salivary omentin-1 levels

Predictor	β	p-value
Clinical Attachment Level	−0.42	0.002
Probing Pocket Depth	−0.36	0.006
Diabetic Status	−0.31	0.01

Adjusted R^2 = 0.48

Table 10: ROC analysis for salivary and serum omentin-1

Biomarker	AUC (95% CI)	Cut-off (pg/mL)	Sens. (%)	Spec. (%)	p-value
Salivary Omentin-1	0.86 (0.78–0.93)	65	82	79	<0.001
Serum Omentin-1	0.89 (0.82–0.95)	80	85	83	<0.001

Discussion:

Both salivary and serum omentin-1 levels were reduced in CP and further decreased in the co-presence of T2DM, a pattern consistent with the systemic anti-inflammatory suppression described in metabolically dysregulated states **[12].** The chronic inflammatory burden of CP likely suppresses omentin-1 via elevated TNF-α and IL-6, while T2DM-associated metabolic dysregulation compounds this reduction through insulin resistance pathways **[13].** These findings extend prior work by Patil *et al.*, who reported salivary omentin-1 improvement after NSPT in periodontitis alone, by demonstrating the same response in diabetic CP patients simultaneously assessed for serum levels **[14].** Zhang *et al.* similarly demonstrated altered omentin-1 concentrations alongside TNF-α and IL-6 in periodontitis patients receiving adjunctive treatment, reinforcing omentin-1 as a responsive inflammatory mediator**.** NSPT-induced reduction in bacterial load and periodontal inflammation likely decreases systemic inflammatory mediators, thereby restoring adipokine regulation and partially improving insulin sensitivity **[15].** The significant negative correlation

between omentin-1 and clinical periodontal parameters (PPD, CAL) supports its potential as a measure of periodontal disease severity and inflammatory burden **[16].** Salivary omentin-1's association with serum levels validates the use of saliva as a surrogate diagnostic medium; several reviews confirm that salivary biomarkers reliably mirror systemic inflammatory status in metabolic and oral diseases **[17].** The good ROC performance of salivary omentin-1 (AUC=0.86) supports its utility as a chair-side, non-invasive biomarker, particularly given the advantages of salivary collection over venepuncture. Age-adjusted salivary IL-1 data in a pilot study by Aeran *et al.* **[18]** further supports the principle that salivary cytokines and adipokines carry periodontal diagnostic value without the need for serum sampling.

Conclusion:

Salivary and serum omentin-1 are significantly reduced in CP, with further decrease in T2DM co-morbidity and rise significantly following NSPT, validating their responsiveness to periodontal inflammation control. Omentin-1 levels correlated with periodontal destruction and showed good ROC diagnostic accuracy in both salivary and serum matrices, reinforcing the periodontal-systemic inflammatory interface. This study establishes salivary omentin-1 as a promising novel non-invasive biomarker for monitoring the inflammatory interplay between periodontitis and T2DM, advancing the concept of integrated periodontal-metabolic care.

Limitations:

It was single-centre with 3-month follow-up and other inflammatory biomarkers and microbiological parameters were not evaluated. Glycemic control variability during follow-up may also have influenced biomarker levels; therefore multicenter longitudinal studies with larger samples and longer follow-up are warranted. Salivary omentin-1 may aid in early identification of patients at heightened risk of combined periodontal and metabolic inflammatory complications, supporting its incorporation into routine chair-side diagnostics.

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