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Association Between Salivary Omentin-1 and C-reactive protein (CRP) levels with Periodontitis Severity in Male Patients with Type 2 Diabetes: A Cross-Sectional Study

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ABSTRACT

Background & Objective: Type 2 diabetes mellitus (T2DM) and periodontitis share a well-established bidirectional relationship, where systemic inflammation act as a critical mechanistic link. Omentin-1, an anti-inflammatory adipokine with potential metabolic regulatory functions, and C-reactive protein (CRP), a widely recognized marker of systemic inflammation, may play opposing but interconnected roles in this association. This cross-sectional study aimed to evaluate salivary levels of Omentin-1 and CRP in T2DM patients, at varying stages of periodontitis, in comparison with healthy controls.

Materials & Methods: In this cross-sectional study, 160 participants were enrolled, including 32 healthy controls and 128 T2DM patients stratified into four equal groups based on periodontitis stages I–IV. Unstimulated saliva samples were collected, and periodontal parameters—probing pocket depth, clinical attachment level (CAL), and bleeding on probing (BOP)—were recorded. Salivary levels of Omentin-1 and CRP were measured using ELISA.

Results: Omentin-1 levels were significantly higher in healthy controls (210.61 pg/mL) than in T2DM-periodontitis patients ($P < 0.001$), showing a progressive decline with advancing periodontitis stages (Stage I: 180.72; Stage II: 153.16; Stage III: 123.90; and Stage IV: 122.82 pg/mL). Conversely, CRP levels were significantly elevated in T2DM-periodontitis patients ($P < 0.001$), increasing with disease severity (Stage I: 120.4; Stage II: 178.0; Stage III: 289.4; and Stage IV: 501.0 pg/mL) compared to controls (45.3 pg/mL).

Conclusion: Salivary Omentin-1 levels decrease with the progression of periodontitis in T2DM patients while CRP levels increase, suggesting a potential protective role of Omentin-1 against inflammation. These biomarkers may serve as useful indicators for monitoring both periodontitis severity and metabolic dysfunction in T2DM, underscoring their clinical relevance.

Keywords: Omentin-1, C-reactive Protein, Periodontitis, Type 2 Diabetes Mellitus, Saliva, Biomarker, Inflammation

1. Introduction

Periodontitis (PD) is a chronic inflammatory disease that contributes to the global burden of chronic conditions. It is characterized by the progressive destruction of the tooth-supporting structures, including the gums, periodontal ligament, and alveolar bone (1). Type 2 Diabetes Mellitus (T2DM) arises from the body's inefficient utilization of insulin (2). According to WHO estimation, diabetes will rank as the sixth most common cause of death by 2030 (3).

Deterioration of the tissues that support teeth, such as the alveolar bone and periodontal ligament, is a hallmark of periodontitis. It is a chronic infection of the oral cavity caused by pathogenic biofilms that cause a persistent inflammatory response in gingival tissue (4, 5). Numerous reviews and epidemiological research have established the reciprocal link between T2DM and periodontitis (6, 7). Since periodontitis is the sixth consequence of diabetes mellitus, DM may accelerate the development of this condition (8). On the other hand, periodontitis may raise

the risk of complications from diabetes and is now recognized as a risk factor for declining glycemic control (9). Because periodontitis is a localized infection, it can raise systemic inflammatory levels of TNF-a, CRP, and IL-6, which in turn can lead to insulin resistance (10, 11).

Omentin-1 is secreted by visceral adipose tissue and epithelial cells. It is recognized for its dual role in metabolic regulation and inflammation (12). Additionally, it has anti-inflammatory capacity, hindering pro-inflammatory cytokines such as IL-6 and TNF-α, which are elevated in chronic inflammatory conditions (13). Recent studies suggest that Intelectin-1 levels are altered in chronic inflammatory states, positioning it as a proposed biomarker for disease severity and advancement (14).

C-reactive protein is a marker for systemic inflammation that is created by the liver as a reaction to IL-6 and other inflammatory cytokines. It is widely used to assess inflammation in clinical practice (15). The rationale for studying these biomarkers in saliva, is due to its non-invasive and reliable medium for assessing oral and systemic inflammation (16).

The findings from multiple studies on omentin-1 levels have shown inconsistencies across different age groups in both male and female patients. Furthermore, the concentration of omentin-1 in the saliva of men with type 2 diabetes and periodontitis has not been thoroughly investigated. These gaps in the existing research serve as the primary motivation for conducting this study.

Our study aims to compare the salivary levels of omentin-1 and CRP in patients with type 2 diabetes mellitus across different stages of periodontitis and a healthy control group. Additionally, we assessed the potential of omentin-1 and CRP as biomarkers for evaluating periodontitis severity in T2DM patients.

2. Materials and Methods

2.1 Study Design

In this cross-sectional study, 160 male participants aged 40–60 years were included. Of these, 128 were patients diagnosed with type 2 diabetes mellitus (with a duration of over 8 years) and periodontitis, recruited from the Diabetes and Endocrinology Center at Marjan Hospital in Babylon. For all participants, a case sheet was completed, encompassing patient demographics (age, educational attainment), risk factors such as tobacco use, family history of periodontal disease, medical history, blood glucose levels, and periodontal parameters.

These participants were classified into four stages of periodontitis (Stage I to Stage IV) based on the staging system developed by Tonetti et al (17). The current staging system for periodontitis, initially introduced by Tonetti et al (17) is incorporated within a broader classification framework created by the World Workshop on the categorization of periodontal and peri-implant diseases and conditions. Staging is used to describe the severity (based on attachment loss and tooth loss due to periodontitis) and extent of periodontitis, as well as the complexity of its management. Additionally, 32 participants were considered as the control group. The control group comprised systemically healthy individuals (with no conditions such as diabetes or immunosuppression) attending routine dental check-ups without active oral complaints. These participants exhibited periodontal health, defined as probing depths ≤3 mm at all sites, the absence of bleeding on probing, no clinical attachment loss, no radiographic evidence of bone loss, and clinically healthy gingiva (no erythema, edema, or inflammation).

The reason for females' withdrawal from the study is females' experience cyclical hormonal fluctuations (menstrual cycle, pregnancy, and menopause) that directly influence inflammatory markers (CRP) and adipokines (Omentin-1) (18). Also, Hormonal changes (e.g., pregnancy gingivitis) can independently worsen periodontal health.

2.2 Criteria Inclusion and Exclusion Criteria

Participants included individuals aged 40–60 years (19), who voluntarily consented to the study, and had either type 2 diabetes mellitus (T2DM) with periodontitis (for the case group) or no systemic or periodontal diseases (for the control group). Exclusion criteria included individuals under 40 years of age, those who declined consent, and those who had undergone periodontal treatment or antibiotic therapy within the past six months. Additionally, participants with other systemic diseases (except diabetes), such as chronic kidney disease (which increases CRP due to uremic inflammation and reduces omentin-1 due to renal clearance impairment) (20), chronic respiratory diseases (which elevate systemic CRP due to lung inflammation) (21), hypoxia in COPD (which may worsen periodontitis severity) (21), female patients, current or recent smokers (within the past three years), e-cigarette users, individuals using inhalant drugs, and edentulous individuals were excluded.

2.3 Collection of Saliva Samples

Saliva collection was conducted between 8:00 a.m. and 11:00 a.m. Participants were allowed to rest for a few minutes before rinsing their mouths thoroughly with water. They were then instructed to lean forward slightly for five minutes without swallowing or speaking. A minimum of 3 mL of saliva, pooled in the anterior floor of the mouth, was collected by passive drooling into pre-weighed, airtight, serialized 50 mL polystyrene tubes (22). Absorbent paper towels were used to manage any accidental spillage. The samples were then centrifuged at 5,000 rpm for 10 minutes (23).

The supernatants were stored in pre-weighed, airtight, serialized tubes to ensure sample integrity and accurate measurement until further analysis (24). Only male participants were recruited to control for sex-specific hormonal influences on inflammatory biomarkers (25). This design eliminated confounding due to menstrual cycle variability and allowed for more homogeneous data

on T2DM–periodontitis interactions within a higher-risk male population (26).

2.4 Oral Examination: Clinical Periodontal Parameters

The clinical parameters were measured for all present teeth, including full-mouth probing pocket depth, clinical attachment loss, and bleeding on probing. Wisdom teeth were excluded from the assessment.

A. Measurement of Bleeding on Probing

Periodontal probes were carefully inserted into the sulcus or pocket until slight resistance was encountered, in order to record the BOP score. The probing force was estimated to be between 20 and 25 grams (0.20–0.25 N) to minimize tissue trauma. If bleeding occurred within 30 seconds of probing, a score of 1 was assigned to the site; sites that did not bleed received a score of 0 (27).

B. Measurement of Probing Pocket Depth

Probing pocket depth refers to the distance from the gingival margin to the tip of the periodontal probe. A PPD measurement of 3 mm or less is considered indicative of good oral health while a PPD greater than 3 mm suggests the presence of periodontitis (27).

C. Measurement of Clinical Attachment Loss

Clinical attachment loss refers to the measurement from the cement enamel junction (CEJ) to the deepest point reached by the probe tip during periodontal probing. The distance between the CEJ and the gingival margin also indicates gingival recession. The primary difference between PPD and CAL lies in their use of different anatomical reference points (27).

2.5 Biochemical Analysis

The measurement of C-reactive protein (CRP) and Omentin-1 levels in saliva samples was performed using a microplate reader (Mindray MR-96A) and sandwich ELISA kits specific for each biomarker. These kits utilized microplates pre-coated with CRP- and Omentin-1-specific antibodies. Standards or samples were added to the designated wells, followed by the addition of biotin-conjugated antibodies specific to CRP and Omentin-1.

After incubation, horseradish peroxidase (HRP)-conjugated avidin was added and incubated further. Subsequently, the TMB substrate was added. In wells where CRP or Omentin-1, biotin-conjugated antibodies, and HRP-avidin complexes were present, a color change was observed due to the enzyme-substrate reaction. This reaction was terminated by adding sulfuric acid solution, and absorbance was measured spectrophotometrically at a wavelength of 450 nm. The concentrations of CRP and Omentin-1 in the samples were calculated by plotting the absorbance values against the standard curves.

2.6 Statistical Analysis

All data were expressed as mean ± standard deviation (SD). Statistical analysis was conducted using the Statistical Package for the Social Sciences (SPSS), version 24. For group comparisons, the Kruskal-Wallis test, a non-parametric method, was applied. A p-value of less than 0.05 was considered statistically significant.

3. Result

3.1 Demographic Characteristics of Study Groups

A total of 160 male participants aged between 40 and 60 years were included in the study, with an overall mean age of 51.33 years (SD ± 6.45). The study groups were categorized as follows:

Group One: 32 participants, mean age 48.43 years (SD ± 6.15), Group Two: 32 participants, mean age 49.62 years (SD ± 5.89), Group Three: 32 participants, mean age 51.87 years (SD ± 7.11), Group Four: 32 participants, mean age 55.78 years (SD ± 5.21) and Control Group: 32 participants, mean age 50.93 years (SD ± 5.55).

Table 1 presents the statistical analysis of salivary C-reactive protein levels among T2DM patients with various stages of periodontitis and healthy control participants. The results show that CRP levels were significantly higher in all periodontitis groups compared to the control group. Furthermore, a progressive increase in CRP concentration was observed from Stage I to Stage IV periodontitis among T2DM patients. The Kruskal-Wallis test revealed a statistically significant difference among the groups ($p < 0.001$), indicating that salivary CRP levels are positively associated with the severity of periodontal disease in diabetic individuals.

Table 2 presents the statistical analysis of salivary Omentin-1 levels among type 2 diabetes mellitus (T2DM) patients with varying stages of periodontitis and healthy control subjects. The results demonstrate that salivary Omentin-1 levels were significantly higher in the control group compared to all T2DM periodontitis groups. Moreover, a progressive decline in Omentin-1 concentration was observed from stage I to stage IV periodontitis. The Kruskal-Wallis test yielded a p-value < 0.001, indicating statistically significant differences in Omentin-1 levels among the study groups.

Table 3 presents the correlation coefficients between periodontal clinical parameters and salivary biomarkers (CRP and Omentin-1) within the control group. A weak negative, non-significant correlation was observed between probing pocket depth and Omentin-1, as well as between PPD and CRP. Clinical attachment level showed a weak positive, non-significant correlation with Omentin-1 and also a weak positive, non-significant correlation with CRP. Bleeding on probing demonstrated a weak negative, non-significant correlation with Omentin-1. Overall, no statistically significant associations were found between periodontal parameters and salivary biomarker levels in the control group.

Table 4 displays the correlation coefficients between periodontal clinical parameters and salivary biomarkers in T2DM patients with stage I periodontitis. A statistically significant positive correlation was found between probing pocket depth and Omentin-1 ($r = 0.36$, $p = 0.04$).

Additionally, clinical attachment level showed a significant positive correlation with both MDA ($r = 0.51$, $p = 0.002$) and CRP ($r = 0.59$, $p < 0.001$) in stage 1 group.

Table 5 presents the correlation coefficients between periodontal clinical parameters and salivary biomarkers in T2DM patients with stage II periodontitis. A statistically significant positive correlation was observed between PPD and C-reactive protein ($r = 0.38$, $p = 0.02$), while the correlation between PPD and Omentin-1 was weak and not statistically significant. Additionally, clinical attachment level showed a significant positive correlation with CRP ($r = 0.67$, $p < 0.001$). In contrast, the correlation between CAL and Omentin-1 was weak and non-significant. These findings indicate that CRP may be more closely associated with periodontal tissue destruction in stage II periodontitis among T2DM patients.

Table 6 presents the correlation coefficients between periodontal clinical parameters and salivary biomarkers in T2DM patients with stage III periodontitis. Probing pocket depth showed a weak, non-significant negative correlation with Omentin-1 and a weak, non-significant positive correlation with C-reactive protein. In contrast, clinical attachment level exhibited a strong and statistically significant positive correlation with CRP ($r = 0.68$, $p < 0.001$), while the correlation between CAL and Omentin-1 was weak positive and non-significant.

Table 7 presents the correlation coefficients between periodontal clinical parameters and salivary biomarkers in T2DM patients with stage IV periodontitis. Probing pocket depth (PPD) showed a weak, non-significant weak positive correlation with Omentin-1 and a weak, non-significant negative correlation with CRP. Clinical attachment level exhibited a significant positive correlation with CRP ($r = 0.54$, $p = 0.001$), whereas the correlation between CAL and Omentin-1 was weak and non-significant. Bleeding on probing showed weak, non-significant positive correlations with both Omentin-1 and CRP.

Table 1. Salivary C-reactive protein levels across study groups, presented using median values, interquartile ranges (IQR), and full ranges (minimum–maximum).

Groups	Median (Q2)	Interquartile Range (IQR)	Full Range (Min–Max)	Clinical Interpretation	Kruskal Wallis test	P value
Healthy Controls	45.3	30.1 – 66.7	0.05 – 83.7	Normal levels	85.67	0.000
Stage I	120.4	79.6 – 283.4	30.1 – 326.7	Mild elevation		
Stage II	178.0	100.4 – 310.4	36.1 – 529.3	Significant inflammation		
Stage III	289.4	120.4 – 514.2	42.8 – 904.5	Disease		
Stage IV	501.0	268.3 – 652.5	29.6 – 1031.0	Severe inflammation		

Table 2. Omentin-1 Levels by using median values, interquartile ranges, and full ranges for study groups.

Groups	Median (Q2)	Interquartile Range (IQR) (Q1–Q3)	Full Range (Min–Max)	Kruskal-Wallis test	P value
Stage I	180.72	108.49 – 245.82	67.59 – 494.66	25.67	0.000
Stage II	153.16	106.43 – 191.70	47.33 – 376.91		
Stage III	123.90	78.48 – 172.74	27.86 – 311.43		
Stage IV	122.82	80.12 – 170.54	45.50 – 361.38		
Control	210.61	150.78-290.34	99.86-664.11		

Table 3. Association between periodontal clinical measures and salivary biomarkers (CRP and Omentin-1) among control participants. This table presents the correlation analysis assessing the relationship between periodontal parameters (e.g., probing depth, clinical attachment level, bleeding on probing) and salivary biomarker levels within the healthy control group.

Groups	Periodontal Parameters	Salivary Biomarkers			
		omentin-1		CRP	
Controls	PPD	r	-0.27	r	-0.003
		p	0.12	p	0.98
	CAL	r	0.06	r	0.12
		p	0.7	p	0.49
	BOP	r	-0.07	r	-0.22
		p	0.7	p	0.2

Table 4. Association between periodontal clinical measures with salivary biomarkers among control participants in Stage I Group.

Groups	Periodontal Parameters	Salivary Biomarkers			
		omentin-1		CRP	
Stage I group	PPD	r	0.36	r	-0.20
		p	0.04	p	0.25
	CAL	r	-0.016	r	0.59
		p	0.92	p	0.000
	BOP	r	0.16	r	0.056
		p	0.35	p	0.76

Table 5. Association between periodontal clinical measures with salivary biomarkers among control individuals in Stage II Group.

Groups	Periodontal Parameters	Salivary Biomarkers			
		omentin-1		CRP	
Stage II group	PPD	r	0.02	r	0.38
		p	0.90	p	0.02
	CAL	r	0.038	r	0.67
		p	0.83	p	0.000
	BOP	r	0.09	r	-0.143
		p	0.5	p	0.43

Table 6. Association between periodontal clinical measures and salivary biomarkers (CRP and Omentin-1) among control participants in the Stage III Group. This table details the correlation coefficients between key periodontal parameters—probing pocket depth (PPD), clinical attachment level (CAL), and bleeding on probing (BOP)—and salivary biomarker levels within this group.

Groups	Periodontal Parameters	Salivary Biomarkers			
		omentin-1		CRP	
Stage III group	PPD	r	-0.002	r	0.21
		p	0.9	p	0.24
	CAL	r	0.18	r	0.68
		p	0.32	p	0.000
	BOP	r	0.21	r	-0.04
		p	0.24	p	0.82

Table 7. Association between periodontal clinical measures and salivary biomarkers (CRP and Omentin-1) among control participants in the Stage IV Group. This table summarizes the correlation analysis between key periodontal parameters (e.g., probing pocket depth, clinical attachment level, bleeding on probing) and salivary biomarker levels within this group.

Groups	Periodontal Parameters	Salivary Biomarkers			
		omentin-1		CRP	
Stage IV group	PPD	r	0.12	r	-0.146
		p	0.48	p	0.42
	CAL	r	0.20	r	0.54
		p	0.26	p	0.001
	BOP	r	0.07	r	0.08
		p	0.6	p	0.63

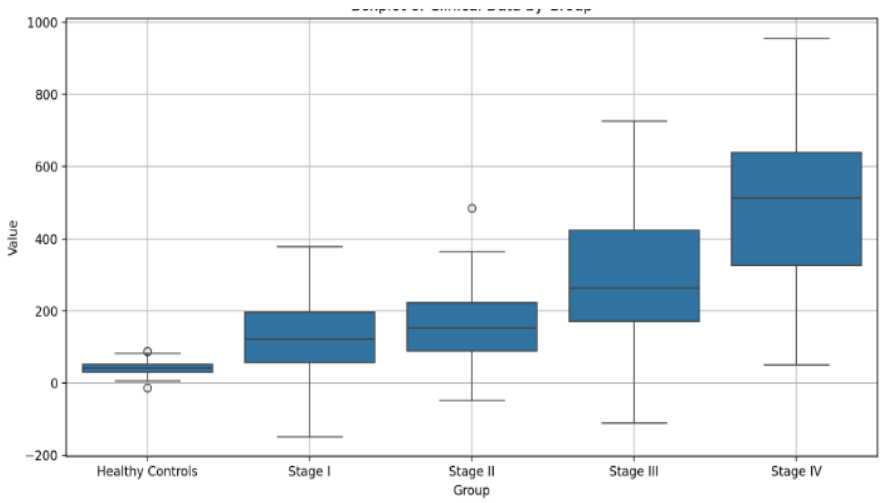

Figure 1. Boxplot illustrating the distribution of salivary CRP levels across different clinical groups, ranging from healthy controls to T2DM patients with stage IV periodontitis. Each box represents the interquartile range (IQR), with the horizontal line inside the box indicating the median value. The plot provides a clear comparison of CRP value distributions and highlights the progressive increase in inflammation severity across the groups. (**Designed by Authors, 2025**).

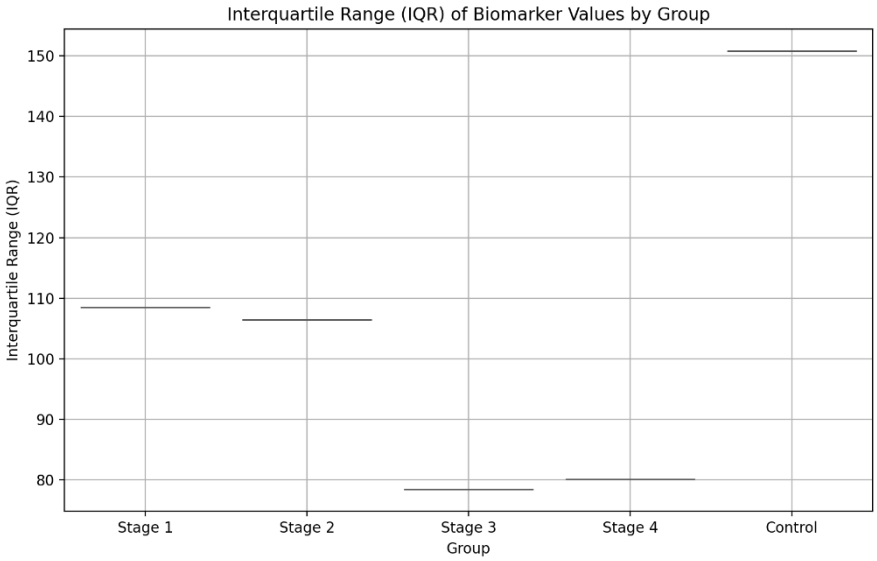

Figure 2. Boxplot for a clear comparison of how biomarker values change across different clinical stages, highlighting the variability and presence of outliers that may be important for clinical interpretation. (**Designed by Authors, 2025**).

4. Discussions

Diabetes is a well-established risk factor for periodontitis, increasing susceptibility to the disease by approximately threefold. This heightened risk is linked to elevated systemic inflammatory markers observed in diabetic individuals (28). Hyperglycemia activates multiple pathways that promote inflammation, oxidative stress, and apoptosis (29). This amplified inflammatory response contributes not only to microvascular and macrovascular complications but also exacerbates periodontal tissue inflammation. If periodontal tissue damage goes undiagnosed or is inadequately managed, it can progress to the destruction of both hard and soft tissues, ultimately leading to tooth loss (30).

The present study demonstrated a significant progressive decrease in salivary omentin-1 levels with advancing stages of periodontitis in type 2 diabetic patients. The lowest omentin-1 concentrations were observed in patients with Stage III and Stage IV periodontitis. In contrast, healthy controls exhibited the highest omentin-1 levels, followed by patients with Stage I and Stage II periodontitis. These differences were statistically significant ($p = 0.000$). These findings align with emerging evidence that omentin-1, an anti-inflammatory adipokine, is downregulated in chronic inflammatory states.

Our results are consistent with previous studies investigating omentin-1 in metabolic and inflammatory diseases. Moreno-Navarrete et al (31) reported reduced omentin-1 levels in patients with obesity and metabolic syndrome, suggesting a shared mechanism of adipokine dysregulation in chronic inflammation (32). Additionally, the progressive decline in omentin-1 observed here agrees with (28), Mubarok et al (32), who found an inverse correlation between periodontal inflammation severity and omentin-1 levels in diabetic patients.

This inverse relationship between periodontitis severity and omentin-1 levels carries important clinical implications, especially for diabetic patients. Omentin-1 plays a critical role in enhancing insulin sensitivity and exerting protective metabolic effects. Both chronic periodontitis patients—with and without T2DM—show significantly reduced serum and salivary omentin-1 compared to healthy controls. Notably, periodontal interventions such as scaling and root planning (SRP) not only increase omentin-1 levels but also improve periodontal health and glycemic control, as reflected by reductions in HbA1c (33, 34).

The decrease in omentin-1 observed in advanced periodontitis may worsen diabetic control by impairing insulin sensitivity and promoting systemic inflammation. This underscores the role of periodontitis as a modifier of metabolic function in diabetes. Importantly, non-surgical periodontal therapy has been shown to elevate omentin-1 levels while reducing inflammatory markers, suggesting periodontal treatment may improve metabolic regulation in diabetic patients (35).

These findings support the American Diabetes Association's recommendations, which emphasize periodontal health as an integral component of diabetes management, given periodontitis's role in exacerbating systemic inflammation and insulin resistance (36). The consistent association between omentin-1 levels and disease progression suggests its potential as a dual biomarker for periodontal inflammation and metabolic dysregulation. Regular monitoring of omentin-1 could enhance treatment assessment and contribute to improved metabolic outcomes in diabetic patients with

periodontitis, highlighting the importance of interdisciplinary care (34).

Similarly, this study observed a clear progressive increase in salivary C-reactive protein (CRP) levels with advancing periodontitis stages in T2DM patients, with the highest levels in Stage IV. Healthy controls had significantly lower CRP levels compared to all periodontitis groups ($p = 0.000$), supporting the established link between periodontal inflammation and systemic inflammatory markers.

CRP, an acute-phase reactant produced primarily by the liver in response to proinflammatory cytokines such as IL-6, plays a central role in immune responses and microbial defense. Elevated CRP levels have been documented in individuals with chronic periodontitis and T2DM, indicating persistent systemic inflammation. Kim et al (37) showed increased CRP expression in the gingival tissues of periodontitis patients with T2DM compared to healthy controls, implicating CRP in periodontal tissue breakdown in diabetes (38).

Multiple studies corroborate these findings. Martínez-Aguilar et al (39) reported significantly higher serum CRP levels in periodontitis patients, especially those with T2DM, reinforcing CRP as a valuable inflammatory marker (39). Abdulhaq et al (40) demonstrated that nonsurgical periodontal therapy significantly reduces serum CRP levels in diabetic patients with chronic periodontitis, highlighting the systemic impact of periodontal inflammation and the therapeutic potential of periodontal treatment (40).

The elevated CRP levels in patients with periodontitis and T2DM reflect a persistent systemic inflammatory state that may contribute to disease progression. Thus, CRP monitoring could serve as a useful clinical marker for inflammation burden and guide treatment strategies. Importantly, periodontal therapy may reduce systemic inflammation and potentially improve glycemic control in diabetic patients (41).

The elevated CRP levels observed in patients with both periodontitis and type 2 diabetes mellitus (T2DM) indicate a persistent systemic inflammatory state that may contribute to disease progression in both conditions. CRP monitoring could therefore serve as a valuable clinical marker for assessing inflammatory burden and guiding treatment strategies. Importantly, periodontal therapy may help reduce systemic inflammation and could potentially enhance glycemic control in diabetic patients (41, 42).

Miller et al (43) also demonstrated that salivary biomarkers including omentin-1 and CRP, remain stable in periodontally healthy individuals, with no significant associations between these markers and clinical periodontal parameters.

5. Conclusion

This study found that salivary CRP levels were significantly elevated, while salivary omentin-1 levels were significantly reduced in T2DM patients with periodontitis compared to healthy controls. Moreover, these biomarker levels were significantly associated with the increasing severity of periodontitis, underscoring their potential role as biomarkers reflecting periodontal inflammation and metabolic dysregulation in diabetic patients.

6. Declarations

6.1 Acknowledgments

S.A.O. and D.K.A would want to express gratitude to Mustansiriyah University in Baghdad, Iraq (www.uomustansiriyah.edu.iq) for their assistance with this study.

6.2 Ethical Considerations

This study was conducted at the Diabetes and Endocrinology Center, Marjan Hospital, Babylon, and Al-Amin Center for Advanced Biotechnology and Research. Ethical approval was obtained from the Institutional Review Board (IRB) of Mustansiriyah University College of Dentistry (Approval No. REC145, dated December 1, 2023). Written informed consent was obtained from all participants prior to their inclusion in the study.

6.3 Authors' Contributions

Sajjad A. Jreo: Conceived and designed the study; collected samples from the Diabetes and Endocrinology Center at Marjan Hospital, Babylon; performed laboratory tests at the Al-Amin Center for Advanced Biotechnology and Research; analyzed the data; and wrote the manuscript. Dalia K. Abbas: Assisted in experimental design; contributed to data interpretation; critically reviewed the manuscript; and revised it for intellectual content. All authors read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

6.4 Conflict of Interest

The authors have no conflict of interest.

6.5 Fund or Financial Support

This research received no specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

6.6 Using Artificial Intelligence Tools (AI Tools)

The authors did not use any artificial intelligence (AI) tools in the preparation of this manuscript.

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