Clinical Oral Investigations (2022) 26:5351–5358
https://doi.org/10.1007/s00784-022-04502-0

ORIGINAL ARTICLE

Is periodontal inflamed surface area associated with serum and salivary levels of IL-1β, visfatin, and omentin-1 in overweight/obese patients?

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Received: 19 January 2022 / Accepted: 13 April 2022 / Published online: 22 April 2022

Abstract

Objectives The aim of this study was to evaluate the levels of salivary and serum interleukin (IL)-1β, visfatin, and omentin-1 in the relationship between periodontal disease and overweight/obesity as well as to reveal the possible role of periodontal inflamed surface area (PISA) in this association.

Materials and methods Ninety-six individuals (69 females, 27 males) were divided into 4 groups as systemically healthy (H) and non-periodontitis (HnP, $n=23$), systemically healthy and periodontitis (HP, $n=24$), overweight/obese (O) and non-periodontitis (OnP, $n=25$), and overweight/obese and periodontitis (OP, $n=24$). Periodontal parameters were measured, and PISA was calculated. IL-1β, visfatin, and omentin-1 levels in saliva and serum samples were analysed.

Results Periodontal parameters deteriorated, salivary and serum IL-1β and visfatin levels were increased, and omentin-1 levels were decreased in OnP and OP groups, compared to HnP and HP groups. Salivary and serum IL-1β and visfatin levels were increased and omentin-1 levels were decreased in periodontitis groups, compared to HnP and OnP groups. PISA was negatively correlated with salivary omentin-1 and positively correlated with salivary and serum visfatin in H and O groups, whereas a positive relationship was found between PISA and salivary and serum IL-1β in H group.

Conclusions PISA may be negatively associated with salivary omentin-1, while positively correlated with salivary and serum visfatin in overweight/obese patients.

Clinical relevance.

Co-evaluation of PISA and adipokines seems to be an innovative approach to evaluate the association between periodontitis and overweight/obesity.

Keywords Adipokines · Obesity · Overweight · Periodontal diseases

Introduction

Periodontal diseases are mostly caused by the exposure of host periodontal tissues to microbiota in the microbial dental plaque, and the resulting inflammation may cause tissue destruction and tooth loss in some individuals. Researchers have shown that the presence of a periodontal pocket that can harbour pathogenic microorganisms can initiate a host response and produce a systemic effect [1–7]. It has been claimed that it is necessary to determine the amount of inflammatory periodontal tissue to measure the inflammatory load caused by periodontitis, which is a risk factor for other diseases [8]. For this purpose, periodontal inflamed surface area (PISA) has been developed, which reflects the surface area of the bleeding pocket epithelium in square millimetres [8].

The common risk factors and pathogenesis mechanisms of periodontal disease and obesity are still not fully explained, although many studies have shown that obesity may worsen the periodontal condition [1, 9, 10] and a positive relationship exists between PISA and body mass index (BMI) [10]. Adipose tissue induces inflammatory processes and oxidative stress disorders, which create a similar pathophysiology between both diseases, and secretes proinflammatory

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cytokines and hormones called adipocytokines. This consequence suggests a potential interaction between obesity, periodontitis, and chronic-disease incidence [11].

Visfatin is one of the last identified adipocytokines that visceral adipose tissue produces [11]. It has been shown to play an important role in immune response and inflammation and to be regulated by some cytokines and lipopolysaccharides, such as interleukin (IL)-1β, tumour necrosis factor alpha (TNF-α), and IL-6 [12]. Obese individuals have presented elevated visfatin levels [13]. Visfatin levels have been reported to increase [14–17] and decrease after periodontal treatment in patients with periodontal disease [17]. Similarly, it was documented that serum [18], salivary [19], and gingival crevicular fluid (GCF) [20] visfatin levels increased and decreased after periodontal treatment in obese and periodontitis patients.

Omentin, also called intelectin, is one of the new adipocytokines highly expressed in human visceral adipose tissue and mouse small intestines [21]. Two forms of omentin have been identified: omentin-1 and omentin-2. Omentin-1 is the most researched and the main form in the human bloodstream [22]. Omentin-1 has an anti-inflammatory, antioxidant, and anti-apoptotic functions and may have a therapeutic role in bone metabolic diseases, atherosclerosis, and vascular diseases [23]. It has been indicated that omentin-1 level is negatively associated with an increase in BMI [24]. Omentin-1 decreases in periodontal disease and increases after periodontal treatment and also been reported to be lower in obese and periodontitis patients than in systemically healthy and periodontitis controls [25].

The rationale of this study was to exhibit whether the salivary and serum IL-1β, visfatin, and omentin-1 levels may have a potential role in the association between periodontal disease and overweight/obesity and to investigate the role of PISA in this relationship.

Materials and methods

Ethical approval was obtained from the Hatay Mustafa Kemal University Tayfur Ata Sökmen Medical Faculty Clinical Research Ethics Committee (No. 2018/149). Patients who came to Hatay Mustafa Kemal University, Department of Periodontology, for routine periodontal treatment were included in the study. We asked the individuals who agreed to participate in the study to sign an informed consent form, and we recorded their sociodemographics with the help of a questionnaire. We calculated the patients' BMI (kg/m^2) and divided a total of 96 subjects into 2 groups, systemically healthy (H) (BMI < 25, $n = 47$) and overweight/obese (O) (BMI ≥ 25, $n = 49$), according to their BMI values [26]. The patients were then divided into 4 subgroups according to their periodontal status: systemically healthy and non-periodontitis (HnP, $n = 23$), systemically healthy and periodontitis (HP, $n = 24$), overweight/obese and non-periodontitis (OnP, $n = 25$), and overweight/obese and periodontitis (OP, $n = 24$). Patients under 20 years of age and over 50 years of age, having less than 18 teeth, having any systemic disease other than obesity and/or using drugs, smoking, having received periodontal treatment within the last 6 months, having taken antibiotics within the last 3 months, having a history of chemotherapy/radiotherapy, and being pregnant/breastfeeding were excluded from the study. Patients having severe stage IV periodontitis, who need for complex rehabilitation due to significant tooth loss, and diagnosed with grade C periodontitis, whose destruction exceeds expectation given biofilm deposits, were also excluded due to affect the study results.

Periodontal examination

A specialist (E.D.) performed periodontal examination of all patients. Plaque index (PI) [27], gingival index (GI) [28], bleeding on probing (BOP), periodontal probing depth (PPD), and clinical attachment level (CAL) were measured using a UNC-15 probe. PISA was calculated with the help of an Excel file on https://www.parsprototo.info using the PPD, CAL, and BOP values at the 6 points of each tooth [8], in the following steps: (1) mean CAL and gingival recession (GR) for each particular tooth is measured; (2) periodontal epithelial surface area (PESA) is calculated for each specific tooth using linear mean CAL and GR; (3) PISA is calculated for each tooth via multiplying of PESA by the proportion of sites around the tooth with BOP; (4) the sum of the PISA for each tooth is calculated for each participant.

Periodontal healthy (BOP < 10%) and gingivitis (BOP ≥ 10%) patients who had PPD ≤ 3 mm were included in the non-periodontitis group [29]. The periodontitis group included stage II and III periodontitis patients who had interdental CAL at ≥ 2 non-adjacent teeth or buccal or oral CAL ≥ 3 mm with PPD > 3 at ≥ 2 teeth [30].

Analysis of serum and saliva samples

Unstimulated total saliva samples were collected at in the morning after an overnight fasting before the periodontal examination. The patients were asked to spit into a glass every 60 s from a sitting position for 10 min [31]. We then centrifuged the saliva samples at 4 °C, 4000 g for 10 min. Blood samples were taken from the antecubital vein after 8 h of fasting, and then serum samples were obtained by centrifuging the blood samples at 3000 rpm for 15 min. Saliva supernatants and serum samples were collected in Eppendorf tubes and stored at − 80 °C.

Salivary and serum IL-1β (Catalogue no: E0143Hu), omentin-1 (Catalogue no: E3770Hu), and visfatin

(Catalogue no: E0025Hu) levels were measured in the laboratory of Hatay Mustafa Kemal University, Faculty of Medicine, Department of Medical Microbiology, using commercial kits (BT LAB Bioassay Technology Laboratory, Shanghai, China) according to the protocol the manufacturer provided with the ELISA technique. The kits' sensitivities were 10.07 pg/L, 2.53 ng/mL, and 0.23 ng/mL, and their detection ranges were 20–6000 pg/L, 5–1500 ng/mL, and 0.5–100 ng/mL, respectively.

Statistics

A package programme (SPSS version 21.0 for Windows; IBM Corp., Armonk, NY, USA) was performed for statistical analysis. Sample size was calculated using a previous study investigating the levels of salivary visfatin in obese patients with chronic periodontitis [19], since there has been no study comparing of the salivary and/or serum omentin-1 levels between periodontitis and overweight/obese patients. For visfatin levels, a sample of 22 patients were needed for each group to achieve a power of 90% and $\alpha = 0.05$. We determined the study's power using a software (G*power version 3.1.9.2 for Windows; University of Kiel, Kiel, Germany), and we calculated the power at $\alpha = 0.05$ as > 0.99 for all biomarkers. The parameters' normality and homogeneity were analysed, and non-parametric tests were conducted. Chi-square test for qualitative data [(n (%)] and Mann–Whitney *U* test for quantitative data [mean ± standard error (SE)] were performed to analyse the sociodemographics. Kruskal–Wallis test was used to analyse the periodontal and biochemical parameters and the Mann–Whitney *U* test with Bonferroni correction to determine the difference between groups. The relationships among obesity status and periodontal and biochemical parameters were evaluated by multivariate linear regression analysis adjusted for age and tooth brushing frequency. We considered $P < 0.05$ statistically significant.

Results

The study included ninety-six patients (69 female, 27 male). Table 1 shows the individuals' sociodemographic and anthropometric data. We observed that age, tooth brushing frequency, BMI, and waist-hip ratio were higher in the O group than in the H group ($P < 0.05$). Periodontal parameters were increased in overweight/obese groups (OnP and OP), compared to systemically healthy periodontal controls (HnP and HP) ($P < 0.05$) (Table 2).

Figure 1 demonstrates comparisons of the biochemical parameters among the groups. Salivary and serum IL-1β and visfatin levels were increased, and serum omentin-1 levels were decreased in the HP group, compared to the HnP group ($P < 0.05$). Serum IL-1β and salivary and serum visfatin levels were increased, and salivary and serum omentin-1 levels were decreased in the OP group, compared to the OnP group ($P < 0.05$). We determined that salivary and serum IL-1β and visfatin levels were increased, and omentin-1 levels were decreased in the OnP group, compared to the HnP group ($P < 0.05$), and in the OP group, compared to the HP group.

We noted that of the periodontal parameters, only GI and PISA were positively correlated with overweight/obesity ($P < 0.05$) (Table 3). Table 4 indicates the relationships between biochemical and periodontal parameters according to the overweight/obesity status. Periodontal

Table 1 Sociodemographics of the groups

Variables	H ($n = 47$)	O ($n = 49$)	P^a
Age (years)	32.11 ± 1.11	38.61 ± 1.24	**0.000**
Gender			
Female	35 (50.7%)	34 (49.3%)	0.580
Male	12 (44.4%)	15 (55.6%)	
Education level			
Primary school	22 (42.3%)	30 (57.7%)	0.093
High school	7 (46.7%)	8 (53.3%)	
University	13 (54.2%)	11 (45.8%)	
Master/doctorate	5 (100%)	0 (0%)	
Personal Income (TRY)			
≤ 1600	16 (44.4%)	20 (55.6%)	0.880
1600–3200	16 (53.3%)	14 (46.7%)	
3200–4800	8 (53.3%)	7 (46.7%)	
≥ 4800	7 (46.7%)	8 (53.3%)	
Dental visit frequency			
Every 6 months	5 (62.5%)	3 (37.5%)	0.556
Every year	6 (46.2%)	7 (53.8%)	
Upon a complaint	30 (45.5)	36 (54.5%)	
Never before	6 (66.7%)	3 (33.3%)	
Brushing frequency			
2–3 times in a day	17 (56.7%)	13 (43.3%)	**0.036**
1 time in a day	26 (56.5%)	20 (43.5%)	
2–3 times in a week	1 (16.7%)	5 (83.3%)	
Rarely	3 (21.4%)	11 (78.6%)	
Interdental agent			
Yes	17 (63%)	10 (37%)	0.086
No	30 (43. 5%)	39 (56.5%)	
BMI (kg/m^2)	22.09 ± 0.36	29.87 ± 0.5	**0.000**
WHR			
Female	0.76 ± 0.01	0.79 ± 0.01	**0.018**
Male	0.91 ± 0.04	0.94 ± 0.02	0.157

H systemically healthy, *O* overweight/obese, *TRY* Turkish Lira, *BMI* body mass index, *WHR* waist-hip ratio

[a]*P* values were computed with chi-square test for categorical variables [n (%)] or Mann–Whitney *U* tests for continuous variables (mean ± standard error)

Bold denotes statistical significance at $P < 0.05$

Table 2 Comparisons of periodontal parameters (mean ± standard error)

Variables	HnP ($n=23$)	HP ($n=24$)	OnP ($n=25$)	OP ($n=24$)	P^a
PI	0.85 ± 0.1	1.71 ± 0.1[b]	1.07 ± 0.07[c]	1.97 ± 0.11[bd]	**0.000**
GI	0.79 ± 0.08	1.72 ± 0.1[b]	1.09 ± 0.07[bc]	2.20 ± 0.08[bcd]	**0.000**
BOP	16.68 ± 4.1	54.66 ± 5.26[b]	25.27 ± 4.19[c]	74.45 ± 2.81[bcd]	**0.000**
PPD	1.71 ± 0.11	3.15 ± 0.16[b]	1.93 ± 0.1[c]	3.81 ± 0.16[bcd]	**0.000**
CAL	1.73 ± 0.11	3.26 ± 0.17[b]	1.96 ± 0.1[c]	4.04 ± 0.23[bcd]	**0.000**
PISA	544.07 ± 61.01	1026.33 ± 75.66[b]	642.78 ± 47.22[c]	1950.37 ± 236.44[bcd]	**0.000**

HnP systemically healthy and non-periodontitis, *HP* systemically healthy and periodontitis, *OnP* overweight/obese and non-periodontitis, *OP* overweight/obese and periodontitis, *PI* plaque index, *GI* gingival index, *BOP* bleeding on probing, *PPD* periodontal probing depth, *CAL* clinical attachment level, *PISA* periodontal inflamed surface area

[a]*P* values were computed with Kruskal–Wallis *H* test

[b]Significant difference from HnP group ($P<0.05$, Mann–Whitney *U* test with Bonferroni correction)

[c]Significant difference from HP group ($P<0.05$, Mann–Whitney *U* test with Bonferroni correction)

[d]Significant difference from OnP group ($P<0.05$, Mann–Whitney *U* test with Bonferroni correction)

Bold denotes statistical significance at $P<0.05$

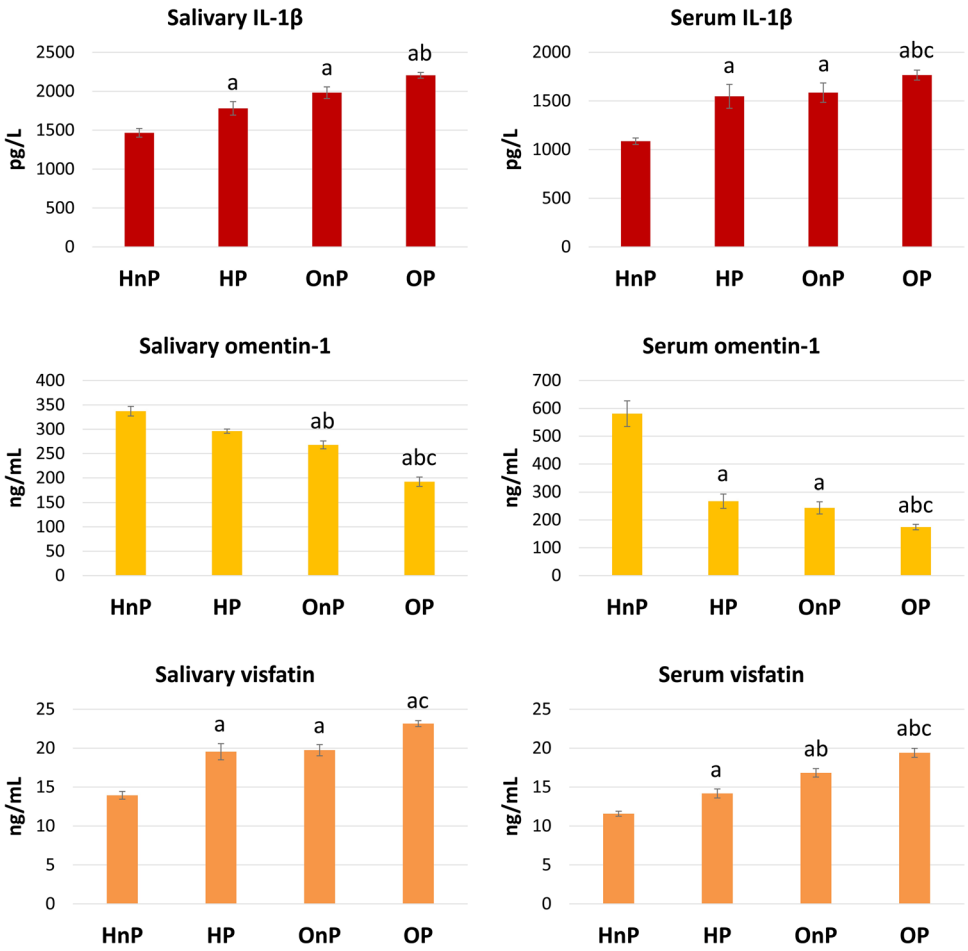

Fig. 1 Comparison of the biochemical parameters among the groups. HnP, systemically healthy and non-periodontitis; HP, systemically healthy and periodontitis; OnP, overweight/obese and non-periodontitis; OP, overweight/obese and periodontitis; IL, interleukin. Significant difference from HnP (a), HP (b), and OnP group (c)

parameters were generally negatively correlated with salivary and serum omentin-1 levels and positively correlated with IL-1β and visfatin levels. PISA was negatively correlated with salivary omentin-1 and positively correlated with salivary and serum visfatin levels in the H and O groups, and we observed a positive relationship between PISA and salivary and serum IL-1β in the H group ($P<0.05$).

Table 3 Multivariate linear regression analysis adjusted for age and tooth brushing frequency between overweight/obesity and periodontal parameters

Dependent variables	Standardized β[a] (95% CI)	*P*
PI	0.181 (−0.05 to 0.52)	0.109
GI	0.285 (0.09 to 0.69)	**0.011**
BOP	0.199 (−1.40 to 25.68)	0.078
PD	0.190 (−0.07 to 0.90)	0.094
CAL	0.176 (−0.12 to 0.98)	0.121
PISA	0.399 (319.84 to 1042.89)	**0.000**

PI plaque index, *GI* gingival index, *BOP* bleeding on probing, *PPD* periodontal probing depth, *CAL* clinical attachment level, *PISA* periodontal inflamed surface area

[a]Standardized *β* coefficient represents the change in periodontal parameters (dependent variables) for a change of status from non-overweight/obese to overweight/obese (predictor variable)

Bold denotes statistical significance at $P < 0.05$

Discussion

Nesse et al. [8] proposed the necessity of a score indicating the total inflammatory load of periodontitis and defined PESA and PISA based on periodontal inflammation's possible effects on systemic health. PESA indicates the entire surface area of the periodontal pocket epithelium, whereas PISA reflects the inflammatory load caused by periodontitis and the surface area of the bleeding pocket epithelium. The relationship between PISA and serum and salivary adipokines had not been evaluated even though the relationship between periodontitis and obesity has been extensively investigated in the literature [1, 9–11, 32–34]. A recent meta-analysis showed that vaspin, omentin-1, chemerin, IL-10, progranulin, monocyte chemoattractant protein 4, IL-1β, and interferon gamma may play a key role in the relationship between obesity and periodontal disease [32]. To reveal the relationship between PISA and adipokines, we evaluated IL-1β and omentin-1 levels similarly to Brum

Table 4 Multivariate linear regression analysis adjusted for age and tooth brushing frequency between biochemical and periodontal parameters according to overweight/obesity status [standardized β[a] (95% CI)]

Variables	Group	Salivary IL-1β	Serum IL-1β	Salivary omentin-1	Serum omentin-1	Salivary visfatin	Serum visfatin
PI	**H**	0.171 (0.00;0.00)	0.263 (0.00;0.00)	**−0.402 (−0.01;0.00)**	**−0.553 (0.00;0.00)**	**0.538 (0.04;0.104)**	**0.444 (0.04;0.18)**
	O	**0.392 (0.00;0.00)**	0.207 (0.00;0.00)	**−0.459 (−0.01;0.00)**	−0.202 (0.00;0.00)	**0.380 (0.02;0.13)**	0.286 (0.00;0.12)
GI	**H**	0.237 (0.00;0.00)	**0.306 (0.00;0.00)**	**−0.395 (−0.01;0.00)**	**−0.577 (0.00;0.00)**	**0.589 (0.05;0.11)**	**0.549 (0.07;0.20)**
	O	**0.478 (0.00;0.00)**	**0.312 (0.00;0.00)**	**−0.558 (−0.01;0.00)**	**−0.328 (−0.01;0.00)**	**0.469 (0.04;0.15)**	**0.356 (0.01;0.15)**
BOP	**H**	0.294 (0.00;0.05)	0.188 (−0.01;0.03)	−0.250 (−0.40;0.04)	**−0.547 (−0.10;-0.04)**	**0.524 (1.58;4.82)**	**0.458 (2.09;8.31)**
	O	**0.452 (0.02;0.07)**	**0.357 (0.01;0.05)**	**−0.520 (−0.41; −0.14)**	**−0.482 (−0.26; −0.06)**	**0.418 (1.34;6.43)**	**0.420 (1.41;7.03)**
PD	**H**	**0.318 (0.00;0.00)**	0.276 (0.00;0.00)	**−0.426 (−0.02;0.00)**	**−0.579 (0.00;0.00)**	**0.563 (0.06;0.17)**	**0.484 (0.08;0.29)**
	O	**0.422 (0.00;0.00)**	0.196 (0.00;0.00)	**−0.541 (−0.02; −0.01)**	**−0.502 (−0.01;0.00)**	**0.516 (0.09;0.28)**	**0.323 (0.01;0.24)**
CAL	**H**	**0.326 (0.00;0.00)**	0.284 (0.00;0.00)	**−0.439 (−0.02;0.00)**	**−0.595 (0.00;0.00)**	**0.578 (0.07;0.18)**	**0.475 (0.08;0.30)**
	O	**0.413 (0.00;0.00)**	0.195 (0.00;0.00)	**−0.523 (−0.02; −0.01)**	**−0.475 (−0.01;0.00)**	**0.533 (0.11;0.33)**	**0.343 (0.02;0.29)**
PISA	**H**	**0.480 (0.22;0.80)**	**0.497 (0.19;0.66)**	**−0.391 (−6.75; −0.94)**	−0.281 (−0.1;0.03)	**0.331 (3.00;52.90)**	**0.302 (0.88;93.89)**
	O	0.268 (−0.07;2.01)	0.130 (−0.43;1.14)	**−0320 (−11.09; −0.76)**	−0.240 (−6.68;0.87)	**0.350 (24.87;206.6)**	**0.318 (12.4;214.9)**

IL interleukin, *PI* plaque index, *GI* gingival index, *BOP* bleeding on probing, *PPD* periodontal probing depth, *CAL* clinical attachment level, *PISA* periodontal inflamed surface area, *H* systemically healthy, *O* overweight/obese

[a]Standardized *β* coefficient represents the change in periodontal parameters (dependent variables) for each unit increase in the predictor variable as biochemical parameters

Bold denotes statistical significance at $P < 0.05$

et al. [32]. Additionally, we analysed visfatin levels in serum and saliva samples.

Wood et al. [35] presented a relationship between periodontal attachment loss and obesity, and fat metabolism plays an important role in this relationship. Al-Zahrani et al. [1] indicated the relationship between periodontal disease and obesity in individuals between the ages of 18 and 34 and did not find any relationship in individuals over the age of 35. We observed that periodontal parameters increased in overweight/obese groups, compared to systemically healthy periodontal controls.

Aoyama et al. [10] revealed that PISA and BMI were significantly associated, whereas Takeda et al. [36] reported that PISA was not associated with obesity parameters. Differences in results may be associated with the confounder factors that may play a role in the relationship. In our study, we used adjusted multivariate regression analysis to eliminate confounders and identified a positive relationship between overweight/obesity and GI and PISA. Therefore, we believe PISA is an important parameter that can be used in the relationship between overweight/obesity and periodontal disease.

IL-1β is a proinflammatory cytokine that induces bone resorption by triggering inflammatory reactions; therefore, it is considered a potential therapeutic target for periodontitis [37]. In our study, the increase in salivary and serum IL-1β levels in individuals with periodontitis, compared to non-periodontitis controls, as well as positive relationships between salivary IL-1β levels and PPD, CAL, and PISA and between serum IL-1β and GI and PISA are consistent with the literature.

Proinflammatory cytokines released from adipose tissue resulting in a hyper-inflamed condition that leads to periodontal attachment loss [9]. In our study, the increase in serum and salivary IL-1β levels in the O group, compared to the H group, is consistent with Abulnaja [38] findings. In the O group, positive associations between salivary and serum IL-1β and GI and BOP and between salivary IL-1β and PI, PPD, and CAL confirm that the exacerbated inflammatory response due to overweight/obesity may also contribute to the pathogenesis of periodontal disease.

Omentin-1 is an adipokine secreted from visceral adipose tissue. Circulating omentin levels and omentin gene expression in adipose tissue have been reported to decrease in obese, insulin-resistant, or type 2 diabetes mellitus patients, and they are inversely associated with BMI and waist circumference [24]. Similarly, salivary and serum omentin-1 levels were lower in the OnP and OP groups than in the systemically healthy periodontal controls in our study.

Researchers have not evaluated salivary and serum omentin levels in the relationship between obesity and periodontitis, but in limited studies, the association between omentin and periodontal disease was investigated. Studies have presented that gingival crevicular fluid (GCF) omentin-1 levels are lower in individuals with periodontitis and increase after periodontal treatment and researchers have argued that omentin may have an anti-inflammatory role in periodontitis [25]. Salivary and serum omentin-1 levels decreased in groups with periodontitis, compared to non-periodontitis controls, in our study. In addition, we identified negative correlations between salivary and serum omentin-1 levels and GI, PPD, and CAL.

GCF omentin levels have been indicated to be lower in obese and periodontitis patients than in healthy control and periodontitis groups [25]. In our study, salivary and serum omentin-1 levels were lowest in the OP group. Our results support the studies showing that omentin may have an anti-inflammatory effect in overweight/obese and/or periodontitis patients. Furthermore, although we saw no relationship between PISA and serum omentin-1, the negative correlation between PISA and salivary omentin-1 indicates that saliva and PISA may be used in the relationship between periodontal disease and overweight/obesity.

Circulating visfatin levels have been shown to be associated with white adipose tissue accumulation, and visfatin mRNA levels increased in the course of adipocyte differentiation [39]. Researchers have demonstrated that visfatin levels are higher in children with high BMI and that visfatin may have a significant effect on inflammatory diseases [13]. We detected higher salivary and serum visfatin levels in the OnP and OP groups than in the systemically healthy periodontal controls in our study. These results indicate that overweight/obesity may be associated with increased visfatin levels.

We observed that visfatin levels increased in periodontitis groups, compared to the non-periodontitis groups. In addition, we found positive relationships between salivary/serum visfatin levels and periodontal parameters and PISA. Pradeep et al. [15] examined the relationship between GCF and serum visfatin levels and periodontal disease and concluded that as the disease's severity increased, serum, and GCF visfatin concentrations elevated. Turer et al. [17] showed that GCF and serum visfatin levels were higher in chronic periodontitis than in gingivitis and healthy controls and decreased after periodontal treatment. Salivary visfatin levels have been exhibited to increase in individuals with periodontitis and to correlate with periodontal infection [16]. A recent study indicated that patients with gingivitis and periodontitis have higher visfatin expression than healthy controls [14]. All these results reveal that visfatin may have a role in the pathogenesis of periodontitis.

Serum [18] and salivary [19] visfatin levels have been shown to increase in obese and periodontitis patients, compared to healthy controls. Researchers have reported

that the total visfatin level of GCF is higher in obese and chronic periodontitis patients than in periodontitis patients and decreases after periodontal treatment [20]. We found visfatin levels were higher in overweight/obese and periodontitis patients than in other groups. Our results indicate that overweight/obesity and periodontitis may increase salivary and serum visfatin levels.

Our study has some limitations. Cross-sectional study design prevented us from revealing the cause-effect relationship. Because researchers have evaluated adipokine levels in saliva in a limited number of studies, it has been difficult for us to interpret the results. The inability to create subgroups of periodontal disease due to the small number of samples limited our ability to identify the role of disease severity in this relationship.

Conclusion

We observed that patients with periodontitis and overweight/obesity had increased periodontal inflammatory load, salivary/serum IL-1β, and serum visfatin levels while decreased salivary/serum omentin-1 levels, compared to systemically healthy and non-periodontitis/periodontitis controls. The negative relationships between PISA and salivary omentin-1 and the positive correlations between PISA and salivary/serum visfatin in overweight/obese patients reveal that PISA can be used as an important parameter in the association between periodontal disease and overweight/obesity.

Author contribution Conceptualization: Esra Sinem Kemer Doğan.
Formal Analysis: Esra Sinem Kemer Doğan; Nizami Duran.
Investigation: Esra Sinem Kemer Doğan.
Methodology: Esra Sinem Kemer Doğan; Nizami Duran.
Writing — original draft: Esra Sinem Kemer Doğan.
Writing — review and editing: Esra Sinem Kemer Doğan; Nizami Duran.

Funding Hatay Mustafa Kemal University Scientific Research Projects Commission (Project number: 18.M.094) financially supported the current study.

Declarations

Ethics approval This study was performed in line with the principles of the Declaration of Helsinki. The study protocol was approved by the Hatay Mustafa Kemal University, Tayfur Ata Sökmen Medical Faculty, Clinical Research Ethics Committee (No. 2018/149).

Consent to participate Written informed consent form was obtained from the patients.

Conflict of interest The authors declare no competing interests.

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