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A Clinical-Biochemical Study of Salivary Tumour Necrosis Factor-Alpha Levels in Obese Sudanese Patients with Chronic periodontitis

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ABSTRACT

Objectives: This study examined the impact of Body Mass Index (BMI) on salivary Tumour Necrosis Factor-Alpha (TNF-α) levels in individuals with chronic periodontitis (CP), both obese and non-obese, as well as in a healthy periodontal control group.

Materials and Methods: The clinical-biochemical investigation comprised 30 obese CP participants, 30 non-obese CP participants, and 30 periodontally healthy controls. The researcher looked at the periodontal clinical parameters (Gingival Index (GI), Simplified Oral Hygiene Index (sOHI), Probing Pocket Depth (PPD), and Clinical Attachment Level (CAL)) and anthropometric data (BMI). He also looked at salivary TNF-α levels using an enzyme-linked immunosorbent assay (ELISA). One-way ANOVA and partial correlation coefficient tests were employed to ascertain the extent of the association among TNF-α, CP, and obesity.

Results: Salivary TNF-α levels correlated with elevated BMI and CAL; however, no correlation was seen between CAL and BMI values. Individuals with obesity had elevated clinical indices (GI, sOHI, PPD, and CAL), BMI, and TNF-α in comparison to the other two groups.

Conclusions: Salivary TNF-α levels were significantly higher in obese people with CP, demonstrating a strong association between obesity and enhanced systemic inflammation. However, an insignificant link between BMI and CAL indicates that, while obesity may augment the inflammatory profile, it doesn't directly lead to accelerated periodontal tissue destruction in this group.

Keywords: Body mass index, Obesity, Chronic periodontitis, Tumour necrosis factor-alpha, saliva, Sudan.

1. Introduction

Chronic periodontitis is a persistent inflammatory condition of the periodontium resulting from the interplay between the host's immune response and potentially harmful gram-negative bacteria. The host's immune response is a way for the body to protect itself, but it may also cause tissue damage by making and releasing pro-inflammatory cytokines (1), mediators, and metalloproteinases (2). Periodontal disease poses a danger to systemic health. It contributes to the aetiology of several diseases, including diabetes mellitus (3), cardiovascular disease (4), oral and colorectal cancer, where a bidirectional relationship has been shown, with potential reciprocal influences (5).

This host-parasite relationship triggers a series of innate and adaptive immune responses. During inflammation, inflammatory cells infiltrate the periodontium, and activated macrophages produce various cytokines, such as prostaglandin E2 (PGE-2), interleukin-6 (IL-6), and tumour necrosis factor – alpha (TNF-α), all of which are

associated with the deterioration of periodontal tissue (1).

TNF-α is a pro-inflammatory cytokine generated mainly by macrophages, lymphocytes, and adipocytes in response to inflammation. It is necessary for starting an inflammatory response (6). Additionally, TNF-α serves as an endogenous mediator of immunity, apoptosis, cell proliferation, and differentiation (4). TNF-α is a distinct biomarker for inflammation, obesity, and periodontal disease, with elevated TNF-α levels potentially serving as a substantial risk factor for CP (6).

The TNF-α exerts several effects on periodontal tissues, including the promotion of osteoclastogenesis (bone resorption), regulation of MMP (connective tissue degradation), and augmentation of the immunological response to periodontal infections (7). In addition, TNF-α modulates the metabolic state of adipose tissues and precipitates insulin resistance in persons with diabetes and obesity (6). Lundin (2004) identified a link between TNF-α levels, obesity, and periodontal disease, indicating that each unit increase in BMI is associated with a 0.74 pg elevation in TNF levels in GCF (7).

The Body Mass Index (BMI), a conventional relative weight measure, is calculated by dividing body weight in kilogrammes by height in metres squared (kg /m²). It is frequently utilized in epidemiological studies to forecast obesity-related morbidity and mortality (7).

Obesity is a persistent and critical public health issue in both industrialized and developing countries, and it has been established as a risk factor for several systemic diseases, including cardiovascular conditions, such as heart disease and stroke, diabetes, osteoarthritis, and cancers (8,9). Obesity is becoming more common around the world, especially in African countries, where it affects around 24% of the population (10). Numerous studies in Sudan have shown a rise in obesity, with an estimated prevalence of 21.2% among the Sudanese population, especially in women (26.3%)(11).

Perlstein et al. were the first to show that there is a connection between obesity and periodontal disease (12). Subsequent research has investigated the correlation between periodontal disease and obesity, revealing that obese and overweight individuals are 1.8 times and 2.3 times more likely to be affected by CP, respectively (8).

The molecular mechanisms between adipose tissue and CP remain unidentified. Adipose tissue is thought to release adipokines that cause pro-inflammatory cytokines and hormones to be released. These cytokines and hormones then release a lot of inflammatory mediators that cause a pro-inflammatory state and change how the tissue reacts to bacterial antigens, which creates a pathophysiology that is similar to both diseases and increases the release of cytokines (IL-1, IL-6, and TNF-α), which makes infections more likely and is a significant factor (13).

Saliva is an exocrine secretion, producing around 1.0 to 1.5 litres every day, and consists predominantly of water (99%), alongside electrolytes, mucus, white blood cells, proteins, enzymes, epithelial cells, and anti-inflammatory substances (14). Whole saliva is a combination of fluids that come from the salivary glands, the mucosal transudate, and the nasal cavity and laryngeal mucosa. The flow rate of total unstimulated saliva is around 0.3–0.4 ml/min, but it can drop to 0.1 ml/min during sleep and rise to about 4-5 ml/min during gustatory stimulation (15).

Clinical parameters alone are inadequate for predicting future disease progression or molecular activity; hence, salivary biomarkers, like TNF-α offer a significant supplementary diagnostic value (16).

Saliva reflects both physiological and pathological alterations, making it a frequently utilized alternative diagnostic biofluid. Moreover, salivary cytokines have a more robust correlation with periodontal disease and the total inflammatory load in the oral cavity. Since systemic inflammation affects the salivary inflammatory load, it is essential to characterize this link for accurate salivary biomarker evaluation (16).

Salivary TNF-α is a sensitive biochemical indicator for evaluating periodontal disease activity and systemic health. Its application in the surveillance of periodontal disease is especially relevant in light of the increasing incidence of obesity in Sudan. Also, because it is easy to collect and non-invasive, it is a useful diagnostic tool for a wide range of oral and systemic disorders. Thus, this study sought to assess and compare salivary TNF-α levels, body mass index (BMI), and CP in obese and non-obese and periodontally healthy Sudanese adults.

2. Materials and Methods

An observational analytical cross-sectional study was performed at the Department of Periodontology, Khartoum Dental Teaching Hospital (KDTH), Sudan, to evaluate and compare salivary TNF-α levels, body mass index (BMI), and CP in obese and non-obese, and periodontally healthy Sudanese participants.

During data collection, participants who satisfied the study's inclusion and exclusion criteria were addressed in the Department of Periodontics at the KDTH, and the study's objective and importance were conveyed to them. As a result, those who might be interested in taking part were encouraged to do so and finally signed up for the study.

Using the Raosoft sample size calculator, the sample size was calculated to be 73, with a 95% confidence level, an absolute accuracy of 5%. To make up for any dropouts during the trial period, an extra 25% was added. There were 90 people in all, and they were split into three groups of 30 each: Group A had 30 obese people with CP, Group B had 30 non-obese people with CP, and Group C had 30 people who were periodontally healthy (the control group).

The American Academy of Periodontology (AAP) 1999 criteria were used to include participants with moderate to severe CP (characterized by a pocket depth (PPD) of 4 mm, clinical attachment loss (CAL) of 3 mm, at least one affected site per tooth, and a minimum of four distinct teeth), as well as individuals over 20 years of age of both sexes with at least 20 teeth (17).

Individuals who smoked, dipped snuff, took antibiotics or nonsteroidal anti-inflammatory medicines within the preceding three months, or had periodontal treatment within the same timeframe were excluded. Pregnant, lactating women, or women who used birth control or hormone supplements were also excluded.

The World Health Organization (WHO) criteria for BMI states that a BMI of greater than 30 kg/m² is considered obese, whereas a BMI of less than 30 kg/m² is considered not obese (7).

The recruited participants had a structured on-site interview, and their responses were documented on a prepared data collection sheet along with socio-demographic factors. Each participant got a clinical periodontal evaluation in a dental chair with a graduated Williams periodontal probe. The gingival index (Löe & Silness, 1963) was assessed at the four surfaces (mesial, facial, distal, and lingual/palatal) of all teeth. (18). Probing pocket depth (Glavind & Löe, 1967) (19) was measured at six sites per tooth: the mesiobuccal, distobuccal, mid-buccal, mesiolingual/palatal, distolingual/palatal, and mid-lingual/palatal.

For the Simplified Oral Hygiene Index (s-OHI) (Greene & Vermillion, 1964) (20), six chosen teeth were checked for calculus, debris, and stains on their facial surfaces (teeth 16, 11, 26, and 31) and lingual surfaces (teeth 36 and 46).

A ruler that was attached vertically to a solid base was used to measure the participants' height in centimetres. Then, an Omron digital weighing scale was used to find out how much they weighed (in kg). Before the weight test, participants were told to take everything out of their pockets and take off their shoes and purses. Then, an online BMI calculator was used to figure out each person's BMI.

Participants were asked not to eat or drink, brush their teeth, or chew gum for at least an hour before the sample was taken. Before the sample collection, participants were told to rinse their mouths with water and wait 10 minutes in the clinic. After that, the participants were told to sit up straight and let saliva build up on the floor of their mouths before spitting unstimulated saliva into a sterile, graded test tube. The spitting method was used to get four millilitres from each person. To prevent alterations in antioxidants, all saliva samples were collected between 9:00 am and 12:00 pm. The tube samples were packed in an ice bag right away and sent to the Institute of Endemic Diseases (IED) at Khartoum University, Faculty of Medicine. First, the saliva was centrifuged at 3000 rpm for 15 minutes following the standard centrifugation protocols. Next, the clear supernatant was pipetted into a clean, pre-coded, and properly labelled microvial tube. It was then stored in the refrigerator at -80 °C until the sample was ready to be analysed.

The quantitative sandwich ELISA approach was used to look at the salivary TNF-α. The BioLegend TNF-α - ELISA MAXTM kit (which can detect seven pg/mL of saliva) was used. The quantitative sandwich ELISA approach was performed like this: On the first day, an amount of 100 µL of diluted capture antibody solution was put into each well of the plate. The plate was then sealed and placed in an incubator overnight at 4°C. The next day, each well of the incubated plate was washed four times with 300 µL of wash buffer. Then, an amount of 200 µL of 1X Assay Diluent was added to each well to block the plate. Finally, the plate was sealed and put in an incubator for an hour at room temperature while being shaken on a plate shaker that moved in a circle at 500 rpm with a 0.3 cm radius. The plate was cleaned four times in an hour. We filled the right wells with 100 µL of diluted standard and 100 µL of the saliva sample.

The plate was sealed and shaken at room temperature for two more hours. After washing the plate four times, a 100 µL diluted detection antibody solution was put in each well. After that, the plates were sealed and shaken for an hour at room temperature. After washing four times, each well was filled with 100 µL of diluted Avidin-HRP solution. Finally, the plate was closed and shaken for 30 minutes at room temperature. After cleaning the plate five times and letting it soak for 30 seconds each time, an amount of 100 µL of freshly mixed Tetramethylbenzidine (TMB) substrate solution was added to each well. The plate was then left in the dark for 15 minutes. Finally, an amount of 100 µL of stop solution was added to each well, and the absorbance was measured within 15 minutes.

Before the study started, the Sudan Medical Specialisation Board, the Khartoum State Ministry of Health (KMOH), and the KDTH all gave their permissions. An informed consent form in Arabic was obtained before the commencement of the study. A literate co-patient signed the informed permissions for patients who could not read or write. Before signing or marking the informed consent form, the participant read it out loud if a co-patient was present. To keep patient data safe and private, the data collected was coded and stored on a computer that only the researcher could access using a password.

The data was analysed using the IBM Statistical Package for Social Sciences (SPSS), version 23. Descriptive statistics were determined, such as mean and standard deviation (SD) for continuous variables and frequency and percentage for categorical variables. Inferential statistics were followed using parametric and non-parametric tests for univariate analysis. The level of significance was set at $p < 0.05$.

3. Results

An observational, analytical, cross-sectional hospital-based study was performed at the Department of Periodontics at Khartoum Dental Teaching Hospital (KDTH) in Sudan to examine the correlation between salivary TNF-α levels and periodontal status, as well as BMI.

Ninety participants were divided into three study groups. Each group consisted of 30 participants. Group A were obese with CP and a BMI > 30. kg/m², Group B were non-obese with CP. Group C (Control) were non-obese and periodontally healthy. Most of the people in the study were females (68.9% of the total). The mean age for group A was 38.1 ±9.77 years, for group B was 37.26 ±9.40 years, and for group C was 37.26 ±9.40 years. Comparison between the mean age in the 3 groups demonstrated no statistical significance ($p = 0.93$). The mean BMI for group A was 32.60 ±4.02 kg /m², for group B was 22.31 ±4.90 kg /m², and for group C was 25.47 ±4.33 kg /m². Comparison between the mean BMI values in the 3 groups demonstrated strong statistical significance ($p < 0.001$) (Table 1). The mean age of the participants was 37.54 ±4.51 years, with ages ranging from 22 to 50 years. The mean BMI was 26.79 ±6.16 kg /m², with a range of 14.50 to 47.60 kg /m².

Table 1: Distribution of participants according to gender among the different study groups

Gender	Group A (Obese participants with CP)	Group B (Non-obese participants with CP)	Group C (Periodontally healthy control)	Total
	N (%)	N (%)	N (%)	N (%)
Female	21 (23.30)	20 (22.20)	21(23.30)	62 (68.9)
Male	9 (10.00)	10 (11.10)	9(10.00)	28 (31.1)8
Total	30 (33.30)	30 (33.30)	30(33.30)	90 (100)
Criteria	**Mean ±SD**	**Mean ±SD**	**Mean ±SD**	**P-value (0.05)**
Age	38.1 ±9.77	37.26 ±9.40	37.26 ±9.40	0.93
BMI	32.60 ±4.02	22.31 ±4.90	25.47 ±4.33	0.001

The participants were divided into three age groups: 20–30, 31–40, and 41–50 years. The mean age for the

20-30 age group was 26.2 ±2.82 years in group A, 26.0 ±2.62 years in group B and 26.0 ±2.83 years in group C. The one-way ANOVA test was used to compare the mean age in the 3 different groups, which demonstrated no statistical significance (p = 0.98). The mean age for the 31-40 age group was 36.1 ±3.32 years in group A, 36.4 ±3.24 years in group B and 36.1 ±3.31 years in group C. The one-way ANOVA test was used to compare the mean age in the 3 different groups, which demonstrated no statistical significance (p = 0.97). The mean age for the 41-50 age group was 47.6 ±3.05 years in group A, 47.5 ±3.05 years in group B and 47.6 ±3.05 years in group C. The one-way ANOVA test was used to compare the mean age in the 3 different groups, which demonstrated no statistical significance (p = 1.00) (Table 2).

Table 2: Age grouping among the study groups

Age (years)	Group A (Obese participants with CP)		Group B (Non-obese participants with CP)		Group C (Periodontally healthy control)		P-value
	N (%)	Mean ± SD	N (%)	Mean ± SD	N (%)	Mean ± SD	
20-30	9 (30)	26.2 ±2.82	9 (30)	26 0 ±2.62	9 (30)	26 0 ±2.83	0.98
31-40	10 (33.3)	36.1 ±3.32	10 (33.3)	36.4 ±3.24	10 (33.3)	36.1 ±3.31	0.97
41-50	11 (36.7)	47.6 ±3.045	11 (36.7)	47.5 ±3.045	11 (36.7)	47.6 ±3.045	1.0

The mean GI score for group A was 1.63 ±0.30, while in group B it was 1.58 ±0.26, and in group C it was 0.0 ±0.0. The mean PPD for group A was 2.84 ±0.74, while in group B it was 2.58 ±0.41 and in group C it was 1.59 ±0.35. The mean CAL for group A was 1.35 ±0.99, while in group B it was 1.04 ±0.54, and in group C it was 0.0 ±0.0. The mean s-OHI for group A was 2.86 ±0.60, while in group B it was 2.68 ±0.75 and in group C it was 0.66 ±0.22. Groups A and B, compared to group C, demonstrated the highest mean values for all the periodontal clinical parameters (GI, PPD, CAL, and sOHI). The mean values were compared among different study groups by using one-way ANOVA and Post Hoc analysis by Tukey's HSD test. Comparison between the three different groups demonstrated strong statistical significance (p < 0.001) (Table 3).

Table 3: Mean values of clinical parameters among study groups

Clinical parameters	Group A (Obese participants with CP)	Group B (Non-obese participants with CP)	Group C (Periodontally healthy control)	P-value
	Mean ±SD	Mean ±SD	Mean ±SD	
GI	1.63 ±0.30	1.58 ±0.26	0.0 ±0.0	0.001
PPD	2.84 ±0.74	2.58 ±0.41	1.59 ±0.35	0.001
CAL	1.35 ±0.99	1.04 ±0.54	0.0 ±0.0	0.001
s-OHI	2.86 ±0.60	2.68 ±0.75	0.66 ±0 .22	0.001

The mean salivary TNF-α level in group A was 277.6 ±197.45 pg/mL, whereas it was 208.1 ±166.61 pg/mL in group B, and in group C it was 100.9 ±83.5 pg/mL. The mean values were compared among different study groups by using one-way ANOVA and Post Hoc analysis by Tukey's HSD test. Comparison between the three different groups using the one-way ANOVA test showed a strong statistically significant difference in the salivary levels of TNF-α between the periodontally healthy study group and group A, having the highest value (p < 0.001) and group B (p value 0.026) (Table 4).

Table 4: Mean values of salivary TNF-α among study groups

Groupings	Salivary biomarker Mean Salivary TNF-α levels for groups in homogeneous subsets (Mean ±SD)	P-value for Comparisons with Group C (Periodontally healthy control)
Group A (Obese participants with CP)	277.60 ±197.45	0.001
Group B (Non-obese participants with CP)	208.10 ±166.61	0.026
Group C (Periodontally healthy control)	100.89 ±83.5	

The partial correlation coefficient was employed to evaluate the association among the continuous variables (BMI, CAL, and TNF-α level) while adjusting for OHI. The statistical analysis showed that there were strong positive links between BMI and salivary TNF-α in the whole study group. The periodontally healthy control group (Group C) displayed the most robust linear relationship, evidenced by a Pearson correlation coefficient of r=0.880 (p<0.001). Next came the obese CP group (Group A), which also showed a strong positive correlation (r=0.794, p<0.001). Conversely, the non-obese CP group (Group B) exhibited no statistically significant correlation between BMI and salivary TNF-α levels, indicating a disconnection of these variables in the context of local infection, absent systemic adiposity. Additionally, a significant and positive correlation was identified between TNF-α and CAL salivary levels in obese and non-obese participants with CP groups. However, no correlation was seen between BMI and CAL in either the obese or non-obese with CP cohort (r = 0.110 (0.56) and r = 0.104 (0.584), respectively (Table 5).

Table 5: Correlation between BMI and TNF-α, TNF-α and CAL, BMI and CAL, while adjusting for OHI

Compared parameters	Partial correlation coefficient (p-value) Group A (Obese participants with CP)	Partial correlation coefficient (p-value) Group B (Non-obese participants with CP)	Partial correlation coefficient (p-value) Group C (Periodontally Healthy control)
BMI and TNF-α	0.79 (0.05)	0.72 (0.07)	0.88 (0.03)
TNF-α and CAL	0.40 (0.03)	0.57 (0.001)	-
BMI and CAL	0.11 (0.56)	-0.10 (0.58)	-

4. Discussion

Obesity is a global chronic condition marked by excessive fat accumulation and a persistent low-grade inflammatory response. However, it is one of the most ignored public health problems that might lead to a higher risk of CP. The WHO claimed that obesity afflicted 650 million individuals in 2022 (21). Chronic periodontitis is one of the most common dental diseases in the world and the main reason for people losing (22). Periodontal disease and obesity are interconnected through an exact aetiology shown by analogous inflammatory mediators (23).

Animal studies showed that obesity induces oxidative stress in periodontal tissues. It also makes the periodontal tissue react more strongly to suspected bacterial infections, which leads to a stronger immune response and, finally, loss of alveolar bone (24). Human studies indicated that being overweight increases susceptibility to illness and enhances the reactivity of the immune system. In periodontal disease, adipose tissue releases several inflammatory mediators (25).

The present study examined the relationship between periodontal parameters and salivary TNF-α levels in obese and non-obese individuals with CP, as well as in periodontally healthy subjects. Additionally, the present study correlated the salivary concentration of TNF-α with the BMI in each cohort. Genco et al. (26) developed a model linking inflammation to obesity, diabetes, and periodontal infection, demonstrating that pro-inflammatory cytokines generated by adipocytes disrupt insulin signalling, leading to insulin resistance, a characteristic of diabetes. Hyperinflammation is also linked to diabetes, and the negative effects of hyperinflammation on periodontal tissues make the

body more likely to have oral infections (27).

The BMI was employed to measure obesity, because height and weight are the most common and easy-to-use measurements. Nonetheless, the present study's correlation between BMI and CAL was statistically insignificant, potentially due to BMI's overall relationship with adiposity, despite its occasional misclassification of body fat content, particularly misidentifying the shortest and tallest individuals as obese. In addition, periodontitis is a disease of cumulative destruction. While obesity-driven inflammation, as seen by the high TNFα can be measured in the saliva almost immediately, the transition from inflammation to permanent bone loss (CAL) takes years, if not decades. Furthermore, the pro-inflammatory state might be active (as evidenced by the current TNFα results, but the physical destruction of the periodontal ligament, as demonstrated by the CAL, may not yet have reached a statistically separable threshold between the obese and non-obese groups (28). This conclusion aligns with the results of Ylöstalo et al., who identified a moderate connection between BMI and exacerbated PPD in dentate individuals aged 30 to 49, despite controlling for confounding factors by limiting the sample to never-smokers and non-diabetic participants. Ylöstalo, conversely, contended that weight may be inaccurately categorised owing to variations in the distribution of adipose tissue and muscle mass among individuals, underscoring that BMI is not the most effective measure for determining obesity (29).

The present investigation identified a strong positive correlation between the BMI and salivary TNF-α in both the obese and periodontally healthy cohorts. The periodontally healthy control group (group C) had a higher correlation coefficient (r=0.880) than the obese periodontitis group (r=0.794) (group A), which could be due to the "biological noise" that comes with being sick. In the periodontally healthy control group (group C), the salivary TNF-α levels probably show a baseline metabolic state in which BMI is the only factor causing a change in cytokine levels, creating an almost linear signal. In Group A (Obese Periodontitis), the total salivary TNF-α pool originates from two separate sources: systemic adipose tissue, which consistently induces systemic inflammation, and the localised periodontal tissue destruction. The imperfect alignment between the intensity of local infection and the level of systemic adiposity probably causes some variation that makes the correlation coefficient a little lower, which strengthens the function of salivary TNF-α as a highly sensitive biomarker that continues to respond to metabolic alterations, even in the absence of active oral pathology. In individuals with obesity, the relationship between adipocytes and periodontal infection results in a linear rise in cytokine expression. A similar conclusion was drawn by Iwashita et al., who found that loss of adipose tissue homeostasis induces an increase and activation of immune cells in adipose tissue, leading to impaired immune function in obesity (30).

Conversely, the absence of such a correlation in the non-obese CP cohort is a noteworthy observation, which indicates that, for this cohort, the inflammatory response is not associated with body mass. In this cohort, the salivary TNF-α levels are probably affected more by the local microbial challenge and the strength of their immune response to dental plaque than by the adipose tissue distribution throughout their bodies. Furthermore, the elevated s-OHI Scores in this cohort support this claim and align with the study of Hasan et al., which demonstrated that localised infection may obscure the influence of BMI on the inflammatory profile in non-obese individuals (31). While there remains a possibility that BMI is inaccurate, this result is likely due to the gradual progression of periodontal damage.

From another perspective, in Sudanese populations, metabolic risk can sometimes occur at lower BMI thresholds than those used by the WHO. Some individuals in the "non-obese" cohort may have high visceral adiposity (the primary producer of TNF-α); these "metabolically obese" but "BMI-normal", individuals would have high salivary TNF-α. This "hidden" fat creates outliers that weaken the correlation when using BMI alone as the metric (10).

Pro-inflammatory mediators, such as TNF-α, respond rapidly to both systemic and localised inflammation, but the physical degradation of the periodontium (CAL) occurs gradually. Given that the mean age of the study's participants was around 37.5 years, it is likely that the pro-inflammatory condition associated with obesity has not persisted sufficiently to result in a greater attachment loss compared to non-obese individuals (32).

These findings further indicate that BMI may not be the most effective metric for assessing the likelihood of obesity-related diseases across all demographic groups.

Prior research indicated that waist circumference (WC) or waist-to-hip ratio (WHR) may serve as superior indicators of the inflammatory burden associated with excessive abdominal adiposity (32). The finding of the current study also aligns with the findings of Kim et al., who identified a positive correlation between periodontal attachment loss and waist circumference (WC), but no association with body mass index (BMI) (33). Kim's research demonstrated a correlation between elevated waist circumference and CP. It has been proven that waist circumference (WC) or waist-to-hip ratio (WHR) may serve as more robust indicators of disease risk compared to body mass index (BMI). Ongoing research aims to ascertain if BMI, WC, or both should be utilized to evaluate disease risk (33).

Kongstad et al. (2007) identified a negative correlation between BMI and CAL in a Danish study (23). They asserted that due to the varied definitions of CP in epidemiological research, the identified relationships in epidemiological studies should be interpreted within the framework of significant variability in the outcome variable. Kongstad et al. utilized the exact definition of CP, whereas the present study defined CP as a mean CAL above 3 mm. A Brazilian study that defined CP as CAL of more than 5 mm on more than 30% of teeth found that obesity was a significant risk factor for the disease (23).

Although the prevalence of obesity in African cultures is lower than in European ones (24), the health issues related to obesity may also affect African individuals with lower body mass. This conclusion aligns with Kim's research, which said that the WHO criteria for identifying obesity based on BMI may be insufficient for some populations, as the amount and distribution of body fat influences the risk associated with obesity. Furthermore, abdominal or visceral fat is associated with metabolic syndrome and cardiovascular risk factors (34).

A Dutch study involving men and women with waist circumferences over 102 cm in males and 88 cm in women identified a markedly elevated risk of metabolic disorders (35). Additionally, findings from research conducted in Asia (36) and the Middle East (37) indicated a favourable correlation. In Northern Europe (23), however, the data showed no statistically significant link, which means that the link between obesity and CP is different in different regions and races (33).

The TNF-α, an adipokine that promotes inflammation, is implicated in several inflammatory conditions, such as CP and obesity (9). The TNF levels were elevated in the obese group compared to the non-obese and periodontally healthy groups, perhaps since TNF generated by adipose tissue may enhance the inflammatory response. Furthermore, the present study identified a statistically significant positive correlation with elevated BMI levels. The elevation of TNF-α levels may be attributed to adipose tissue secreting cytokines, e.g. TNF-α and IL-6, which are hypothesised to affect the relationship between body weight and periodontium (36).

The current study's results align with those of Gonçalves et al., who revealed that obese participants with CP exhibited elevated TNF-α levels compared to their non-obese counterparts, suggesting that obesity acts as a moderator of TNF-α levels, promoting pro-inflammatory responses in periodontal participants (38). Lundin et al. (2004) also looked at TNF-α and IL-8 levels in gingival crevicular fluid from 33 people. They found that when the participants were split into two groups based on their BMI (BMI > 40 and BMI < 40 kg /m²), the TNF-α levels in the gingival crevicular fluid were positively linked to people with the higher BMI (39). Finally, the present investigation utilized the CAL to evaluate periodontal disease damage, revealing a significant and positive correlation with increased TNF-α levels. This conclusion aligns with the research of Nishimura et al., who identified elevated TNF-α as a primary factor contributing to significant periodontal disease deterioration in obese individuals (40).

Saliva is a new way to find and keep track of different diseases, as well as a source of biomarkers for numerous systemic and local ailments, such as periodontal disease. Researchers may employ saliva as a diagnostic fluid to assess and monitor systemic health (16). Saliva is shown to contain TNF-α and other inflammatory mediators. Consequently, TNF-α may serve as a sensitive salivary biomarker that corresponds with the severity of the illness. Nonetheless, early detection in oral fluid renders it a cytokine-based diagnosis of periodontal disease (2).

The cross-sectional design of this study, however, impedes any definitive conclusion regarding the relationship between adiposity and the progression of CAL, which prevents causality findings since temporal snapshots are not typical. Due to the small sample size,

the study was unable to find a link between obesity and periodontal disease. Residual confounding may exist in these results, since variables, such as nutrition, exercise, and undiagnosed metabolic disorders, which were not comprehensively addressed in this investigation, might independently influence both systemic cytokine levels and periodontal health.

A larger sample size is necessary to confirm the correlation between obesity and periodontal disease. Researchers must longitudinally monitor individuals with periodontal disease to identify the inflammatory mediators present at various phases of the condition. Longitudinal research is essential before any definitive findings can be reached. Furthermore, experimental investigations are essential to assess the causal mechanisms underlying cross-sectional and longitudinal statistical correlations. To assist obese individuals and dental professionals in comprehending the oral health issues that may arise in obese participants, it is essential to enhance their knowledge of dental health.

5. Conclusions

This study demonstrates that obese Sudanese participants with CP exhibit significantly elevated levels of salivary TNF-α, which are closely associated with an elevated BMI and increased damage to the periodontium. It is essential to consider these findings as associative rather than causal. The data suggest that obesity-related systemic inflammation may increase the pro-inflammatory environment of the periodontium. The absence of a clear correlation between BMI and CAL in the periodontitis groups highlights the complexity of these illnesses, which arise from several aetiological factors. However, salivary TNF-α remains a viable non-invasive biomarker for monitoring the inflammatory load in obese individuals. Further study using bigger cohorts and improved metrics of body fat, such as waist-to-hip ratio, is necessary to determine the precise mechanisms by which obesity may accelerate the destruction of periodontal tissue over time.

Research indicated that monitoring periodontal health in obese people is crucial due to higher TNF-α levels, which may worsen inflammation. To reduce systemic inflammation, dentists should examine BMI and provide counselling on obesity as part of normal periodontal treatment.

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Conflict of Interests

The author declares that there is no conflict of interest.

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