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The Effect of Adiposity on the Outcome of Non-Surgical Periodontal Therapy

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ABSTRACT

Background: Non-surgical periodontal therapy constitutes the first step in controlling periodontal infections, and its outcome is affected by the presence of other systemic diseases and conditions. Obesity is a modifiable risk factor for periodontitis and its effect on the outcome of non-surgical periodontal therapy has not been clearly determined.

Aim: To determine the impact of central adiposity on the outcome of non-surgical periodontal treatment in patients with periodontitis.

Methodology: This prospective interventional study included 39 obese patients with periodontitis (Group A) and 39 normal-weight patients with periodontitis (Group B). The waist circumference (WC), waist/hip ratio (WHR), waist/height ratio (WHR), Simplified oral hygiene index (OHIS), bleeding on probing (BOP), probing pocket depth (PPD), number of sites with PPD ≥ 4 mm, and clinical attachment loss (CAL) were measured at baseline and 3 months after non-surgical periodontal treatment. Chi-square test was used to compare categorical variables between the groups. Independent samples t-tests and paired t-test were used to analyse the numerical intergroup and intragroup data, respectively. Pearson's correlation coefficients and multiple regression analyses were used to assess the strength and impact of central adiposity on periodontal treatment outcomes. Data were analysed using the IBM Statistical Package for Social Sciences version 20.0 and statistical significance was set at P < 0.05.

Results: At baseline, the mean PPD and CAL were comparable in both Groups A and B; while the mean percentage of sites with gingival BOP and percentage of sites with periodontitis were significantly higher in Group A. All periodontal parameters significantly improved after treatment in Groups A and B. However, participants in Group B had a better improvement in sites with gingival BOP and percentage of sites with periodontitis after treatment compared to Group A. Multiple regression analysis showed that central adiposity had a significant (P < 0.05) negative impact on the treatment outcome of gingival BOP, PPD and percentage of sites with periodontitis (PPD $\geq 4 \text{ mm}$) after adjusting for age, sex and socio-economic status.

Conclusions: The findings indicated that central adiposity has a negative impact on gingival bleeding on probing, probing pocket depth and percentage of sites with periodontitis.

Keywords

Central adiposity, non-Surgical periodontal therapy, Periodontitis, Treatment outcome.

Introduction

Recently, the medical literature has been inundated with various evidence associating the status of periodontal health to several systemic conditions [1,2]. One of such burgeoning areas of interest

seeks to establish the correlations between adiposity and chronic inflammatory periodontal diseases [1-3].

Periodontal diseases are pathological conditions affecting the gingival tissue and periodontal attachment apparatus [3]. Inflammatory periodontal disease has traditionally been divided into two categories; gingivitis and periodontitis [3,4]. Gingivitis is the milder form; it is reversible and can be defined as the inflammation of gingival tissues in the absence of attachment loss [3,4]. The more severe form is periodontitis which may results in irreversible tissue damage and tooth loss if left untreated. Periodontitis is a major dental disease having a global impact [3-5] The management of periodontal disease includes; control of risk factors, non-surgical (motivation and personal plaque control by the patient, scaling and root planing (SRP) with or without other adjunctive treatments) and surgical periodontal procedures [6]. Several studies have reported the connection between adiposity (overweight/obesity) and periodontal diseases [3-7] However, the effect of adiposity on the outcome of non-surgical periodontal therapy has not been proven due to the paucity of studies in this respect at the moment.

Adiposity manifests as obesity or overweight. It is an excessive or abnormal accumulation of fats that has the potential to impair health [8]. According to the World Health Organisation (WHO) an individual is overweight when the body mass index (BMI) ranges from 25kg/m2 to 29.9kg/m2 and obese if BMI is ≥30kg/ m2 [8]. Although BMI commonly defines overweight or obesity in adults, this index does not adequately describe the distribution of body fat. Abdominal/central obesity measures such as waist-tohip ratio (WHR), waist circumference (WC) and the waist-height ratio (WHtR) are more strongly associated with health risk and prevalence of periodontal disease than general obesity measured by BMI [9,10]. Obesity is a preventable non-communicable chronic ailment, yet a growing public health problem [8,11]. The WHO fact sheet of 2016 reveals that global prevalence of obesity has doubled since 1980 [8]. "In 2014, more than 1.9 billion adults 18 years and above (which represent 39% of adults) were overweight while over 600 million (which represent 13% of adults) were obese" [8]. This evidence is disturbing taking into consideration the health problems linked with obesity and overweight. Also, epidemiological studies globally have established the rising tendency of weight problems, especially in less developed countries [11]. A systematic review conducted by Chukwuonye et al. [12] revealed a high prevalence of overweight and obesity in Nigeria, ranging from 20.3%-35.1% and 8.1%-22.2% respectively. Also, the prevalence of overweight and obesity among patients accessing care at the oral and maxillofacial unit of Lagos University Teaching Hospital was 39.1% as reported in 2010 [13].

Adiposity has been identified as a risk factor for periodontal disease [3-7,11]. A systematic review and meta-analysis conducted by Chaffee and Weston [3] revealed that $BMI \ge 30 \text{ kg/m2}$ was significantly associated with a high risk for periodontal disease. Suvan et al. [4] also observed that individuals who are overweight

or obese are 2.3 times more likely to suffer from periodontal disease irrespective of traditional risk factors, compared with normal-weight individuals. Furthermore, the release of several mediators of inflammation by adipocytes impairs wound healing [4,5,14]. The altered host immune response, delayed wound healing and negative post-operative outcome generally associated with overweight/obese individuals bring to the fore, the possibility that adiposity may have a modifying influence on the clinical outcome following periodontal treatment [14].

Most of the studies [3-5,7] revealing a relationship between obesity and periodontal diseases were cross-sectional studies; and only few [11,15-18] examined the effect of adiposity on the response to non-surgical periodontal therapy. Gerber et al. [19], in a recent systematic review, argued that the impact of obesity on the outcome of non-surgical periodontal treatment is debatable. He, however, concluded that not only is obesity associated with poorer periodontal health but may also result in inadequate response to non-surgical periodontal therapy [19].

The fact that the results of the few studies assessing adiposity as a possible modifying factor in response to periodontal therapy are contradictory [11,15,20] warrants more prospective studies. Additionally, there is limited literature on the topic in our environment [1,19] We hypothesized that adiposity does not have a significant impact on the outcome of non-surgical periodontal therapy. Therefore, this study aimed to determine the effect of central adiposity on the outcome of non-surgical periodontal treatment.

Materials and Method

This prospective interventional study was conducted among patients attending the Periodontology clinic at the Dental Centre of University of Port Harcourt Teaching Hospital (UPTH), Rivers State. The participants were recruited into the study between January and May, 2019. An equal number of cases and controls were selected and matched for age and gender. The inclusion criteria included; the presence of at least 20 natural teeth, patients between 18 and 60 years[11], non-smokers, patients with no established systemic diseases (such as Diabetes mellitus, hypertension, immunosuppression that can affect the outcome of non-surgical periodontal therapy) or suspicious with such diseases and confirmed by investigations, patients who have not had periodontal treatment in the last 6 months, patients with ability to give informed consent and willing to participate in the study.

Sample size was calculated using the formula (N = 2SD² ($Z_{\alpha/2}$ + Z_{β})²/(d)²) for sample size determination for testing a hypothesis, i.e., for comparison between two groups when the endpoint is quantitative data [21].

Sample Groups and Sampling Method

Group A- Obese participants with periodontitis

Individuals were categorised as obese if BMI is ≥ 30 kg/m², and concomitant WHR > 0.85 for females and WHR > 0.90 for males

according to the WHO criteria [8]. Also, with WHtR greater than 0.5 [22]. Obese participants with gingival BOP > 10%, PPD \geq 4mm and CAL \geq 2mm at \geq 2 non-adjacent sites [23] who met other inclusion criteria and gave consent were selected into this group using systematic random sampling. A daily register was created for all patients who satisfy the criteria for obesity thereby constituting a sampling frame. Obese participants were first enrolled and matched by sex and age with normal-weight participants.

Group B- Normal-weight participants with periodontitis

The normal-weight subjects are those with BMI ranging from 18.5 kg/m² to 24.9 kg/m² and WHR < 0.85 for females and <0.90 for males; and WHtR < 0.5 [22]. Participants with gingival BOP > 10%, PPD \geq 4mm and CAL \geq 2mm at \geq 2 non-adjacent sites [23] and the matching criteria (age and gender) were recruited into this group using systematic random sampling.

Standardisation and Calibration of the Investigator

Intra-examiner reliability was calculated by comparing 2 measurements (with an interval of one week) of PPD and CAL performed on ten patients with periodontitis not related to the study. Kappa statistics showed acceptable reliability with coefficients = 0.93 for PPD and 0.81 for CAL. The reliability testing also served as the pilot-test for the data collecting instrument.

Ethical Consideration

Approval for the study was obtained from the Research and Ethics Committee of the University of Port Harcourt Teaching Hospital, Port Harcourt, Rivers State (Protocol number: UPTH/ADM/90/S. II/VOL.XI/353). The minimal risk associated with the study was explained to the study participants, and the investigation and intervention were at no cost to the study participants.

Data were collected using self-developed structured questionnaires and clinical oral examination. Anthropometric measurements of the study participants were done in a screened cubicle in the presence of a dental nurse. The participant's weight was measured and recorded to the nearest 100g while participants were minimally clothed without shoes [22] using a weighing balance (Model ZT 180, Wincom company Ltd China, max capacity 180 kg). Participants were requested to remove their heavy outer garments (jacket, coat, etc.) as well as empty their pockets. The participants stood without any support, with their weight evenly distributed over the centre of the weighing scale [24]. Three (3) measurements were taken, and the mean was chosen as the weight of each participant.

Likewise, the height of each participant was measured in a standing position without shoes using a modified long ruler placed by the straight wall while the shoulders are in a normal position and eyes are in line with the external auditory meatus [22,24]. The participant with the feet close together stood backing the height rule; with their occiput, shoulder, buttocks, calves and heels touching the ruler [24] Participants were asked to look straight ahead. A small flat ruler was lowered on the head so that the hair

was pressed flat. The height was recorded to the nearest metre. Three (3) measurements were taken, and the average was chosen as the height of each participant.

The waist circumference was measured at the midpoint between the lower margin of the last palpable rib and the top of the iliac crest using a stretch-resistant tape according to the WHO data gathering protocol [9,22]. Hip circumference was measured around the widest portion of the buttocks, with the tape parallel to the floor. For both measurements, the subject stood with their feet close together, arms at the side and body weight evenly distributed. The measurements were taken at the end of normal respiration with the subject relaxed [9,24]. Each measurement was repeated twice; when the measurements are within 1cm of one another, the average was calculated. When the difference between the two measurements exceeds 1cm, the two measures were repeated [9].

BMI was calculated as the weight divided by the square of height (kilogrammes/square metres). The waist-to-hip ratio (WHR) and waist to height ratio (WHR) were calculated as the ratio of waist circumference to hip circumference and waist circumference to height respectively [8,9]. The anthropometric measurements were done at baseline and reassessed at follow-up visits to verify that the participants did not have a significant change in their adiposity status during the study.

Clinical oral examinations of all the participants were done on a dental chair at the dental clinic with adequate light source for illumination. All clinical measurements (BOP, PPD and CAL) were taken using UNC-15 graduated periodontal probe from six sites per tooth on all the teeth present in the patient's mouth except the last molars at baseline and 3 months after periodontal treatment and recorded on a modified periodontal chart.

The oral hygiene status before and after treatment was assessed using the simplified oral hygiene index (OHI-S) [25]. OHI-S was graded as; Good (0 - 1.2), fair (1.3 - 3.0) and poor (3.1 - 6.0). BOP was recorded positive when bleeding occurs within 10 to 15 seconds of gentle probing of the orifice of the gingival crevice [26]. PPD was recorded as the distance from the free gingival margin to the bottom of the periodontal pocket and CAL as the distance from the cemento-enamel junction (CEJ) to the pocket base. Mean CAL of 1-2 mm is considered mild; moderate: 3-4 mm; while \geq 5 mm is severe periodontitis [23,27]. The participant's mean percentage of gingival BOP, mean PPD, percentage of sites with PPD \geq 4 mm and mean CAL were calculated from the periodontal chart. The periodontal parameters were taken at baseline and 3 months posttreatment. The outcome of treatment was defined as the difference between the pre- and post-periodontal parameters.

The study participants were given oral hygiene instructions to motivate them on oral hygiene measures (such as brushing techniques and interdental flossing). Also, the clinical procedures (scaling and subgingival root planing) for all the study participants were done by one of the researchers. Non-surgical periodontal therapy such as scaling and polishing with sub-gingival scaling and root planing (SRP) was done using ultrasonic scalers (UDS-J Woodpecker) and universal curettes. SRP was done after achieving local anaesthesia using 2% Xylocaine in 1:100,000 adrenaline. Copious irrigation was done using Normal saline and 0.2% chlorhexidine digluconate solution (0.2% w/v Corsodyl mouthwash by GlaxoSmithKline Consumer Healthcare, UK) intermittently. Participants were instructed to rinse with 10mls chlorhexidine mouthwash twice a day for two weeks. Participants were reviewed after one week to reinforce the post-operative instructions, and at three (3) months to record their periodontal parameters in the periodontal chart after treatment.

Data Analysis

Data were analysed using the IBM Statistical Package for Social Sciences version 20.0 (IBM SPSS Statistics, Armonk New York). Tables and charts were used for data presentation appropriately. Numerical variables (age, BMI, waist and hip circumference, BOP, PPD, CAL) were presented using means and standard deviation. Results were expressed in frequency and percentages for the categorical variables (age groups, sex, educational level, socio-economic status and severity of periodontitis). Categorical variables were compared using the Chi-square (χ^2) test with Fisher's exact correction. Intra- and intergroup mean comparison of independent and dependent variables was done using paired t-test and interdependent t-test respectively. The strength and direction of the relationship between adiposity measurements and treatment outcome of periodontal clinical parameters was tested using Pearson's correlation coefficients [28].

Multivariate regression analysis was used to determine if measures of adiposity have a unique impact on the outcome of non-surgical periodontal therapy after adjusting for age, sex and socio-economic status. Statistical significance was set at P < 0.05.

Results

A total of 84 participants were recruited for the study comprising 42 participants in each group. However, 78 (39 in each group) participants completed the study. The mean age of the study participants was 34.26 ± 9.47 years with an age range of 20-58 years, while the male to female ratio in each of the group was 1:1.4. Most of the study participants 56 (71.8%) were below 40years of age. The difference in mean age between the two groups was not statistically significant (P = 0.208). Most of the study participants were in the 30-39 age group and had a tertiary level of education with class 2 socio-economic status. There was no statistically significant difference in the participants' age groups, gender distribution, educational status and socioeconomic status (SES) between the groups (Table I). Over the study period, changes in the study participants' weight, waist, and hip circumference were not statistically significant. Also, there was no statistically significant difference (P = 0.350) in the severity of periodontitis at baseline between the two groups.

Table 1: Sociodemographic data of study participants.

Sociodemographic	Gre	oups			
variables	Group A	Group B	Total	χ², P-value	
Age groups					
20-29	10 (25.6)	12 (30.8)	22 (28.2)		
30-39	18 (46.2)	16 (41.0)	34 (43.6)	2 24 0 700#	
40-49	6 (15.4)	5 (12.8)	11 (14.1)	5.24, 0.799"	
50-59	5 (12.8)	6 (15.4)	11 (14.1)		
Gender					
Male	16 (41.0)	16 (41.0)	32 (41.0)	0.05 1.000#	
Female	23 (59.0)	23 (59.0)	46 (59.0)	0.05, 1.000	
Educational status					
Primary	6 (15.4)	6 (15.4)	12 (15.4)		
Secondary	11 (28.2)	6 (15.4)	17 (21.8)	182 0 227#	
Tertiary	22 (56.4)	27 (69.2)	49 (62.8)	4.02, 0.227	
Socio-economic status					
Class 1	5 (12.8)	5 (12.8)	10 (12.8)		
Class 2	19 (48.7)	17 (43.6)	36 (46.2)		
Class 3	7 (18.0)	5 (12.8)	12 (15.4)	12.58	
Class 4	5 (12.8)	2 (5.1)	7 (8.9)	0.083#	
Class 5	3 (7.7)	10 (25.6)	13 (16.7)		
Age in years, mean \pm SD	36.18 ± 8.27	34.50 ± 10.40	34.26 ± 9.47	0.208β	
Age range	20 - 52	20 - 58	20–58		

 χ^2 = Chi-square value, "Fisher's exact p-value, ^{β} t-test.

Independent samples t-test was used to compare the baseline periodontal characteristics of the two groups, as shown in Table II. The baseline OHIS, percentage of sites with gingival BOP, and percentage of sites with PPD \geq 4mm were significantly higher in Group A compared to Group B. The difference in the baseline mean probing pocket depth (PPD) and clinical attachment loss (CAL) between Groups A and B were not statistically significant with P-values of 0.053 and 0.276, respectively. Likewise, the percentage of sites with deep pockets (PPD \geq 6 mm) showed no statistically significant difference (P = 0.166) between Group A and B.

Paired samples t-test performed to determine the response of clinical periodontal parameters to non-surgical periodontal therapy after three months in Groups A and B showed that there was a significant decrease in all clinical periodontal parameters in the two groups at three months post-therapy compared to baseline values. However; the difference in OHIS and CAL between the two groups at 3 months post-therapy was not statistically significant (P = 0.051 and 0.057 respectively).

An independent t-test was performed to compare the mean of differences in pre- and post-treatment periodontal parameters in Group A to that of Group B (Table III). Participants in Group B had a better reduction in sites with gingival BOP, percentage of sites with shallow pockets (PPD ≤ 5 mm) and the percentage of sites with periodontitis (PPD ≥ 4 mm) after 3 months of NSPT compared to Group A. The clinical attachment gain in Group B (1.39 \pm 0.59 mm) was higher than in Group A (1.30 \pm 0.51 mm). However, this was not statistically significant, t (76) = -0.67, P = 0.504.

Table 2: Means (±SD) of clinical	periodontal	parameters f	for both Gro	ups A and B	at baseline and	l follow-up visits.
		1	1		1		1

Variables		Group A	Group B	Between groups P-values
OUR	Baseline	3.15 ± 0.74	2.81 ± 0.75	0.001*
OHIS	3 months IgP	1.0 ± 0.34 0.033^*	$\begin{array}{c} 0.83 \pm 0.38 \\ 0.041 * \end{array}$	0.051
	Baseline	45.80 ± 9.85	38.14 ± 9.72	0.002*
Bleeding on probing (%)	3 months	28.29 ± 8.27	9.66 ± 2.19	< 0.001*
	IgP	< 0.001*	< 0.001*	
	Baseline	4.59 ± 0.30	4.44 ± 0.34	0.053
Probing pocket depth (mm)	3 months	3.38 ± 0.38	3.03 ± 0.36	< 0.001*
	IgP	0.022*	< 0.001*	
Percentage of sites with PPD = 4 mm	Baseline	6.78 ± 5.23	3.54 ± 1.84	< 0.001*
	3 months	6.06 ± 4.49	2.40 ± 1.63	< 0.001*
	IgP	0.002*	0.007*	
	Baseline	3.59 ± 2.21	2.39 ± 1.40	0.010*
Percentage of sites with $PPD = 5 \text{ mm}$	3 months	2.39 ± 2.02	0.20 ± 0.28	< 0.001*
	IgP	< 0.001*	0.011*	
	Baseline	1.32 ± 0.97	0.98 ± 1.05	0.166
Percentage of sites with PPD $\geq 6 \text{ mm}$	3 months	0.26 ± 0.52	0.00 ± 0.00	0.005*
	IgP	0.021*	< 0.001*	
	Baseline	11.65 ± 7.37	6.91 ± 3.09	< 0.001*
Percentage of sites with PPD \geq 4mm	3 months	6.24 ± 4.35	3.29 ± 2.34	< 0.001*
	IgP	< 0.001*	< 0.001*	
	Baseline	3.35 ± 0.74	3.53 ± 0.58	0.276
Clinical attachment loss (mm)	3 months	2.05 ± 0.79	2.14 ± 0.52	0.577
	IgP	< 0.001*	0.024*	

*Statistically significant (P < 0.05), IgP = Intragroup P-value.

Table 3: Comparison of the mean differences in periodontal parameters between pre- and post-periodontal therapy in Groups A and B.

	Group A	Group B	P-value
OHIS	2.15 ± 0.69	1.98 ± 0.51	0.274
Bleeding on probing (%)	17.51 ± 5.27	28.47 ± 8.87	0.001*
Probing pocket depth (mm)	1.21 ± 0.45	1.40 ± 0.37	0.061
Percentage of sites with $PPD = 4 \text{ mm}$	1.25 ± 0.68	2.14 ± 1.42	0.002*
Percentage of sites with $PPD = 5 mm$	1.47 ± 0.68	2.19 ± 1.32	0.006*
Percentage of sites with $PPD \ge 6 \text{ mm}$	1.07 ± 0.98	0.98 ± 1.05	0.711
Total Percentage of sites with $PPD \ge 4 \text{ mm}$	3.78 ± 1.58	5.31 ± 2.97	0.010*
Clinical attachment loss (mm)	1.30 ± 0.51	1.39 ± 0.59	0.504

Data are presented as Mean \pm SD

*Statistically significant (P < 0.05)

A Pearson's correlation coefficient was computed to determine the strength and direction of the relationship between measures of central adiposity and the treatment outcome of non-surgical periodontal treatment (i.e., the difference in the pre- and posttreatment periodontal parameters) (Table IV). The results showed a moderate, negative correlation between measures of central adiposity and treatment outcome of gingival BOP, which was statistically significant (BMI: r = -0.538, n = 78, P = 0.001), (WHR: r = -0.439, n = 68, P = 0.001) and (WHtR: r = -0.551, n = 78, P = 0.000). Likewise, there was a significant, weak and negative correlation between measures of central adiposity (BMI, WHtR) and treatment outcome of PPD and the percentage of sites with shallow pockets (PPD ≤ 5 mm).

Table V showed the univariate regression coefficients and adjusted R^2 values of the measures of adiposity and the dependent outcome

variables (i.e., BOP, PPD and percentage of sites with PPD). WHtR had the highest impact on BOP, PPD and percentage of sites with periodontitis (PPD \geq 4 mm) compared to BMI and WHR. Multiple linear regression was done to assess the influence of central adiposity on the treatment outcome of gingival BOP, PPD and the percentage of sites with PPD \geq 4 mm after NSPT while adjusting for age, gender and SES. The regression equations of the relationship between the treatment outcome of BOP, PPD, percentage of sites having periodontitis (PPD \geq 4 mm) respectively as dependent variables and the individual measures of adiposity (BMI, WHR and WHtR) respectively as independent variables were statistically significant.

Table VI showed the model summary of the influence of WHtR on the outcome of gingival BOP after 3 months of NSPT. In Model 1, a significant regression equation was found (F (1, 76) = 28.81, P < 0.001), with an adjusted R² of 0.293. This means that WHtR

Table 4: Pearson's correlation of measures of adiposity and periodontal treatment outcome (N = 78).

Mean Differences in pre- and post-treatme	ent periodontal parameters	BMI	WHR	WHtR
OTHE	r	0.154	0.035	0.103
OHIS	P-value	0.209	0.775	0.403
\mathbf{D}_{1}	r	-0.538	-0.439	-0.551
Bleeding on probing (%)	P-value	0.001*	0.001*	0.001*
Durling an electric double (man)	r	-0.264	-0.173	-0.279
Probing pocket depth (mm)	P-value	0.029*	0.160	0.021*
	r	-0.373	-0.256	-0.342
Percentage of sites with $PPD = 4 \text{ mm}$	P-value	0.002*	0.029*	0.004*
Demonstrate of citize critic DDD - 5 mm	r	-0.312	-0.317	-0.293
Percentage of sites with $PPD = 5 \text{ mm}$	P-value	0.010*	0.008*	0.015*
	r	-0.069	0.052	-0.038
Percentage of sites with $PPD \ge 6 \text{ mm}$	P-value	0.574	0.674	0.758
Total Demonstrate of sites with DDD > 4 mm	r	-0.346	-0.248	-0.310
Total Percentage of sites with $PPD \ge 4$ min	P-value	0.004*	0.042*	0.010*
	r	-0.026	-0.003	-0.104
Chinical attachment loss (mm)	P-value	0.834	0.981	0.398

r = Pearson's correlational coefficient

*Statistically significant (P < 0.05)

Table 5: Summary of univariate regression coefficients and adjusted R^2 of measures of adiposity and outcome of BOP, PPD, percentage of sites with PPD ≥ 4 mm.

Outcome variables		Regression coefficient (B)	P-value	Adjusted R ²
Bleeding on Probing	BMI	-0.74	0.001*	0.279
	WHR	-43.10	0.001*	0.181
	WHtR	-45.72	0.001*	0.293
	BMI	-0.02	0.029*	0.056
Periodontal Pocket depth	WHR	-0.96	0.160	0.015
	WHtR	-1.06	0.021*	0.064
Percentage sites with PPD \geq 4 mm	BMI	-0.13	0.004*	0.076
	WHR	-6.17	0.042*	0.047
	WHtR	-7.02	0.010*	0.082

Table 6: Multiple linear regression analysis of WHtR, outcome of gingival BOP and sociodemographic variables.

Model	Variables Unstandardized	Davalara	95.0% Confider			
	variables	Coefficients B	P-value	Lower Bound	Upper Bound	
1	(Constant)	49.94	< 0.001*	39.74	60.13	R = 0.551
	WHtR	-45.74	< 0.001*	-62.74	-28.73	$R^{2} = 0.304$ Adjusted R ² = 0.293 F (1,76) = 28.81, P < 0.001
2	(Constant)	37.37	< 0.001*	23.67	51.06	R = 0.628
	WHtR	-48.81	< 0.001*	-65.33	-32.29	$R^2 = 0.395$
	Gender	2.28	0.260	-1.73	6.28	Adjusted $R^2 = 0.357$
	Age	0.28	0.008*	0.07	0.49	F (4,73) = 10.27,
	SES	0.16	0.820	-1.27	1.59	P < 0.001

*Statistically significant

explains 29.3% of the variance in the outcome of gingival BOP after therapy. For every unit increase in WHtR, the outcome of gingival BOP reduced by 45.7%. However, the reduction increased to 48.8% after adjusting for gender, age and socioeconomic status in Model 2. Model 2 also had a significant regression equation (F (4, 73) = 10.27, P < 0.001), with an adjusted R² of 0.357. WHtR had a statistically significant impact on the treatment outcome of gingival BOP in the two Models with P < 0.05 after adjusting for the other independent variables.

Table VII showed the model summary of the influence of WHtR on the outcome of mean PPD after 3 months of non-surgical periodontal therapy. Model 1 showed a significant regression equation (F (1, 66) = 5.58, P = 0.021), with an adjusted R² of 0.064. Hence WHtR can significantly explain 6.4% of the variance in the outcome of mean PPD after treatment. For every unit increase in WHtR, the outcome of mean PPD will reduce by 1.06 mm at follow-up. This however reduced to 0.91 mm after adjusting for gender, age and socioeconomic status in Model 2. WHtR made

Model	Variables	Unstandardized	Davalara	95.0% Confiden		
	Coefficients B	Coefficients B	r-value	Lower Bound	Upper Bound	
1	(Constant)	1.94	< 0.001*	1.39	2.47	R = 0.279
	WHtR	-1.06	0.021*	-1.96	-0.16	$R^{2} = 0.078$ Adjusted R ² = 0.064 F (1,76) = 5.58, P = 0.021*
2	(Constant)	1.82	< 0.001*	1.08	2.57	R = 0.386
	WHtR	-0.91	0.048*	-1.81	-0.00	$R^{2} = 0.149$ Adjusted R ² = 0.095 F (4,73) = 2.76, P = 0.035
	Gender	-0.17	0.130	-0.38	0.05	
	Age	0.00	0.688	-0.01	0.01	
	SES	0.08	0.040*	0.00	0.16	

Table 7: Multiple linear regression analysis of WHtR, outcome of mean PPD and sociodemographic variables.

*Statistically significant

Table 8: Multiple linear regression analysis of WHR, outcome of the percentage of sites with $PPD \ge 4$ mm and sociodemographic variables.

Model Varial	Variables	Unstandardized Coefficients B	D voluo	95.0% Confiden		
	variables		r-value	Lower Bound	Upper Bound	
1	Constant)	8.68	< 0.001*	5.51	11.84	R = 0.310
	WHtR	-7.02	0.010*	-12.31	-1.72	$R^{2} = 0.096$ Adjusted R ² = 0.082 F (1,76) = 7.01, P = 0.010*
2	Constant)	12.53	< 0.001*	8.22	16.83	R = 0.442
	WHtR	-6.90	0.010*	-12.09	-1.71	$R^2 = 0.195$
	Gender	-0.82	0.201	-2.07	0.45	Adjusted R ² = 0.144
	Age	-0.04	0.221	-0.11	0.03	F $(4,73) = 3.819$, P = 0.008*
	SES	-0.41	0.072	-0.86	0.04	

*Statistically significant P < 0.05

a statistically significant unique impact on the outcome of mean PPD after non-surgical periodontal treatment in the two Models with P < 0.05 after adjusting for the other independent variables.

Table VIII showed the model summary of the influence of WHtR on the outcome of the percentage of sites with PPD ≥ 4 mm after 3 months of non-surgical periodontal therapy. Model 1 showed a significant regression equation (F (1, 76) = 7.01, P = 0.010), with an adjusted R² of 0.082. Hence WHtR can significantly explain 8.2% of the variance in the outcome of the percentage of sites with PPD ≥ 4 mm after treatment. For every unit increase in WHtR, the outcome of the percentage of sites with PPD ≥ 4 mm will reduce by 7% at follow-up. This reduced to 6.90% after adjusting for gender, age and socioeconomic status in Model 2. WHtR made a statistically significant unique impact on the outcome of the percentage of sites with PPD ≥ 4 mm after non-surgical periodontal treatment in the two models with P < 0.05 after adjusting for the other independent variables.

Overall, central adiposity had a significant impact on the treatment outcome of gingival BOP, PPD and percentage of sites with periodontitis (PPD ≥ 4 mm).

Discussion

The association between obesity and periodontitis is one of the most recent fields of research in periodontal medicine, and the possible underlying biological mechanisms remain unclear.[19].

Obesity and periodontitis are both chronic low-grade inflammatory diseases that can potentially worsen the systemic inflammatory response [15,20] The association of adiposity and periodontitis has been attributed to increased oxidative stress by adipocytes which increases the level of cytokines in the periodontium leading to periodontal tissue destruction [29,30]. Also, adipose tissue releases pro-inflammatory cytokines which impair wound healing [5,14]. In this study, the impact of adiposity on the outcome of non-surgical periodontal therapy within a period of 3 months in obese patients with periodontitis when compared to age- and gender-matched normal-weight patients with periodontitis was determined.

The young age group (71.8% were below 40years of age) in this study is in contrast to those reported in similar studies [11,15,17,31] where the mean age of obese patients with periodontitis ranged from 44.0 to 48.8 years and those of normal-weight with periodontitis ranged from 42.5 to 48.4 years. This can be attributed to increased prevalence of chronic diseases and occurrence of complications due to obesity in the older age groups [32,33]. Moreso, many of the individuals excluded from this study were older subjects with chronic systemic diseases, in order to enable the potential effects of adiposity to be determined. This may have influenced the higher percentage of the younger age group reported in the current study. This was however, similar to the findings of Al-Zahrani and AlGhamdi [33] who reported a significant association between obesity and periodontitis among individuals between 18

and 34 years of age, while no significant association was found for individuals \geq 35 years of age.

Some of the baseline clinical periodontal parameters, such as the mean percentage of gingival BOP, mean percentage of sites with PPD of 4mm and 5mm, and the total percentage of sites with PPD \geq 4 mm were significantly higher in the obese group as observed in other similar studies [17,18] but contrasted to other studies [15,31,33] where the difference in baseline periodontal parameters between obese and normal-weight with periodontitis were not statistically significant.

Although the baseline oral hygiene status was comparable between the obese and normal-weight participants with periodontitis, the percentage number of sites with gingival BOP was significantly higher in the obese groups. This probably reflects the role of adiposity in exacerbating gingival inflammation. The higher baseline percentage of sites with gingival BOP among obese subjects with periodontitis compared to normal-weight with periodontitis in this study was similar to findings reported by Al-Zahrani and AlGhamdi [33]. However, while the difference was statistically significant in the current study, the previous study [33] did not observe a statistically significant difference. The previous study [33] recruited only female participants but this study had both male and female. It is possible that hormonal fluctuations in their study participants was responsible for the insignificant difference in the mean percentage of sites with BOP. In contrast, other studies [15,17,31] reported higher percentage of sites with gingival BOP in normal-weight with periodontitis compared to the obese subjects with periodontitis at baseline.

The mean baseline PPD was higher in the obese participants compared to the normal-weight with periodontitis in this study, but the difference was not statistically significant. This finding corroborates other similar studies [11,17,18,33]. Likewise, as observed in this study, Altay et al. [31] reported a non-significant higher mean CAL at baseline among normal-weight with periodontitis compared to the obese with periodontitis. This is in contrast to other studies [15,17,33] that reported a higher mean CAL at baseline in obese patients with periodontitis compared to normal-weight with periodontitis, although their mean difference was also not statistically significant. The lower mean CAL in the obese with periodontitis in this study may be attributed to less gingival recession in them.

While some cross-sectional studies [5,7,10] found a significant association between obesity and baseline PPD and CAL, longitudinal studies [17,31,33,34] did not, as observed in this study. It can be inferred that obesity does not appear to have an impact on mean PPD and CAL after adjusting for age and gender in longitudinal studies. Although adiposity may be related to the early stages of periodontal disease it may not relate to the subsequent stages of periodontal tissue destruction [30,34]. The variation in the results from different studies may be due to the various definitions of periodontitis and adiposity used in their

studies [19,30,35]. However, the recent definition by the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Condition clarified the diagnostic criteria of periodontitis from periodontal health and is expected to lay to rest the confusion in defining periodontitis for research purposes. In addition, all of the previous studies [3,19] defined obesity by calculating BMI, and only a few of them included other measures of abdominal obesity such as WC, WHR and percentage of body fat [3,5,30]. Measures of central obesity such as WHR, WC and WHtR as used in this study are more strongly associated with periodontal pockets [36] and poor response to periodontal therapy than BMI [9,10,17].

Similar to findings in this study, Saxlin et al. [37] reported an association between the percentage of sites with PPD \geq 4 mm and obesity in a non-smoking population. Likewise, Kangas et al. [36] reported 40-60% higher likelihood of having teeth with PPD ≥ 4 mm in subjects with central obesity compared to normal-weight individuals. While the baseline percentage of sites with shallow pockets (PPD \leq 5 mm) were significantly higher in the obese with periodontitis in this study, Zuza et al. [15] observed more shallow pockets in the normal-weight with periodontitis group. Also, the higher percentage of deep pockets found among obese participants in this study contradicts the findings by Zuza et al. [15], where normal-weight with periodontitis participants had a higher percentage of deep pockets. Besides, Kangas et al. [36] reported a weak correlation of central obesity and deep pockets (PPD ≥ 6 mm) in non-diabetic and never-smoking subjects aged 30-49 years old.

This study showed that non-surgical periodontal therapy resulted in a significant improvement in all periodontal parameters irrespective of the adiposity status, as observed in other similar studies [15,18,31,33,35]. Positive participants' cooperation with oral hygiene instructions may have been partially responsible for this [18] In the present study, obese participants with periodontitis had a significantly higher mean percentage of gingival BOP than normal-weight with periodontitis at 3 months post-therapy. This is similar to the findings of Bouaziz et al. [18] but in contrast to Altay et al. [31] and Goncalves et al. [17] who observed a higher mean percentage of gingival BOP in normal-weight with periodontitis at 3 months follow up. The findings of Altay et al. [31] and Goncalves et al. [17] may be attributed to the comparable baseline mean gingival BOP between obese and normal-weight participants with periodontitis in their studies. The baseline mean percentage of gingival BOP was significantly higher in obese with periodontitis compared to normal-weight with periodontitis in this study.

Furthermore, in this study, the mean PPD was significantly more in obese with periodontitis participants compared to normal-weight with periodontitis at follow up. This is in contrast to the findings of Altay et al. [31] who reported a statistically non-significant higher mean PPD in normal-weight with periodontitis compared to obese with periodontitis at three months post-treatment. Likewise, the difference in mean CAL observed in normal-weight with periodontitis compared to their obese counterparts in this study at follow-up was not statistically significant. This is in contrast to Altay et al. [31] who reported a significant difference. Goncalves et al. [17] on the other hand, observed no apparent difference in the mean values of PPD and CAL between obese and normal-weight patients at three months follow up.

This study showed a trend toward a better treatment outcome of clinical periodontal parameters in normal-weight with periodontitis compared to obese participants with periodontitis. This corroborates the findings of Suvan et al. [11] and Goncalves et al. [17] where clinical periodontal condition among normal-weight participants was better than among obese with periodontitis at follow-up. However, only the outcome of gingival BOP and the percentage of sites with PPD \leq 5 mm after treatment were significantly better in normal-weight participants with periodontitis compared to obese participants in this study. The better outcome in PPD and CAL observed among normal-weight participants was not statistically significant. However, the outcome of PPD showed a weak negative correlation with central adiposity. This will indicate that obesity might have a negative influence on the clinical response to non-surgical periodontal therapy concerning gingivitis and the number of sites affected by periodontitis but not on the outcome of PPD and CAL. CAL being a measure of cumulative periodontal disease may require a long-term prospective study for its treatment outcome with adiposity to be ascertained.

The mean percentage change in the gingival BOP in this study was comparable to earlier similar studies [15,17,18,31] where improvement in gingival BOP was better in normal-weight with periodontitis at follow-up. This study recorded a 17.5% change in percentage of gingival BOP among obese participants with periodontitis in contrast to other studies that recorded higher percentages; Goncalves et al. [17], Altay et al. [31] and Zuza et al. [15] reported 23.5%, 29.7% and 32.9% respectively. The 28.5% change in gingival BOP observed among normal-weight with periodontitis in this study was also lower to 32.9% [31] and 39.8% [15] reported in the previous studies but higher than 21.2% reported by Goncalves et al. [17] The difference in the results reported in the previous studies [15,17,18,31] may be attributed to their inclusion/exclusion criteria, the severity of periodontitis, treatment protocol and different treatment time used for the nonsurgical periodontal treatment.[19] Altay et al. [31] included smokers in their study while others [11,15,17,18] similar to the current study did not. Smoking has been found to affect the outcome of both non-surgical and surgical periodontal therapy [38,39]. In addition, treatment protocol in previous studies varies from 2 to 6 appointments for SRP completed between 7 to 14 days [17-19] In this current study, SRP was done in one appointment.

Although, the low response in gingival BOP to NSPT by the obese group may have resulted from poor compliance to oral hygiene instructions. There was, however, no significant difference in the oral hygiene status of obese and normal-weight subjects after therapy in this study as also reported by Goncalves et al. [17] Thus, it can be inferred that the difference in gingival BOP outcome is due to an intrinsic factor that is peculiar to the obese group. Moreover, it has been reported that the release of several mediators of inflammation by adipocytes impairs wound healing [14].

Increases in BMI, WC, WHR and WHtR in the current study, showed a significant moderate linear relationship with poor improvement in gingival BOP after therapy. In multivariable analysis model, a significant negative influence of obesity was observed for gingival BOP changes after adjusting for age, gender and socioeconomic factors. For every unit increase in WHtR, the outcome of gingival BOP reduced by 48.8% after adjusting for the independent variables.

This study shows significantly better improvement in the percentage of sites with periodontitis (PPD \ge 4mm) in the normalweight after treatment similar to the study done in Malaysia by Akram et al. [29] but contrasted with Duzagac et al. [20] that found no significant difference. In this study, obese patients with periodontitis had about 2.95% more sites with PPD \ge 4 mm at 3 months post-treatment than patients with normal-weight and periodontitis. This was comparable to the 3.2% reported by Suvan et al [11].

Bouaziz et al. [18] observed that the number of improving sites decreased with obesity after 3 months of NSPT. However, the linear relationship of changes in the percentage of sites with periodontitis and central adiposity was weak but significant in the current study. A unit increase in WHtR reduces the outcome of sites with periodontitis by 6.9% in this study. Suvan et al. [11] reported that for every 10kg/m2 increase in BMI, the mean percentage of sites with PPD > 4 mm increased by 2.5% (95% CI 1.10%, 3.80%). The different definitions of sites with periodontitis used in the previous studies [11,15,17,31] make it difficult to compare with the results of the present study. The significant negative correlation of all the measures of adiposity with the outcome of sites with PPD \geq 4mm in this study, suggests that abdominal obesity have a negative influence on the healing of sites with periodontal pockets although the small regression coefficient indicates a modest magnitude of the effect.

Similar to the findings by Akram et al. [29], this study showed no significant difference in the mean percentage of sites with deep pockets (PPD \geq 6 mm) in obese and normal-weight with periodontitis at follow-up. This was not surprising since both groups with periodontitis had a relatively lower percentage of sites with deep pockets at baseline. Moreover, the management of deep pockets may require more than non-surgical periodontal therapy. Persistent residual pockets greater than 6 mm were observed in obese subjects even after NSPT [17,18,29].

This study showed that the outcome of mean PPD was comparable between the obese and normal-weight with periodontitis. However, the change in mean PPD showed a significantly weak negative relationship with measures of adiposity; a unit increase in WHtR reduces the outcome of mean PPD by 0.91mm after adjusting for age, gender and socioeconomic variables. Similarly, Suvan et al. [11], in a study with a larger sample size of 260 participants reported a significant association between obesity and outcome of mean PPD after 2 months of NSPT independent of age, smoking status and dental plaque levels.

The improvement of mean CAL was also comparable between obese and normal-weight with periodontitis after 3 months of NSPT. The relationship between the change in mean CAL over 3 months and measures of central adiposity was very weak and non-significant. These findings are similar to the previous studies [17,18,31] that reported no significant difference in the improvement of mean CAL between obese and normal-weight with periodontitis after 3 months of NSPT. The short interval between baseline and post-treatment evaluation of periodontal parameters may have contributed to the findings in this study [19,30].

Similar to earlier studies [11,15-20,31,33] evaluating the effect of adiposity on the response to NSPT. This study also shows that the impact of adiposity on the outcome of non-surgical periodontal therapy is uncertain.

Limitations of the Study

- 1. The exclusion criteria (i.e., non-diabetic, non-smoker) for this study may limit the extrapolation of the results to the whole obese population.
- 2. The patients with obesity in this study were mostly moderately obese. Thus, these data cannot be extrapolated to more severe cases of obesity.
- 3. The small sample size employed limits the possibility of generalizing the findings.

Conclusion

After three months of non-surgical periodontal therapy, central adiposity had a significant negative impact on the non-surgical periodontal treatment outcome of gingival bleeding on probing, probing pocket depth and percentage of sites with periodontitis.

Recommendations

- 1. Long term prospective studies with larger sample size are needed to fully appreciate the impact of obesity on the treatment outcome of probing pocket depth and clinical attachment loss.
- 2. There is a need for dental practitioners to educate their obese patients on weight reduction as a preventive measure for the maintenance of periodontal health.

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