Response to Periodontal Therapy in Patients Who Had Weight Loss After Bariatric Surgery and Obese Counterparts: A Pilot Study

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Background: Periodontitis and obesity are both chronic health problems, and the literature supports an association between the two. Weight loss after bariatric surgery (BS) has been shown to decrease overall mortality as well as the development of new health-related conditions in morbidly obese patients. The present study aims to assess whether significant weight loss would improve the response to non-surgical periodontal therapy in obese patients.

Methods: This study included 30 obese (body mass index >30 kg/m²) patients affected with chronic periodontitis. Of these, 15 patients had previously undergone BS and lost ≥40% of their excess weight for ≥6 months after surgery. The other 15 patients were also obese but did not have the surgery, nor did they lose weight to serve as a control group. All participants received non-surgical periodontal therapy (scaling and root planing and oral hygiene instructions). Probing depth (PD), clinical attachment level (CAL), bleeding on probing (BOP), gingival index (GI), and plaque index were measured at baseline and at 4 to 6 weeks after the periodontal treatment. Descriptive statistics, linear mixed-effects models, and linear regression models were used for data analysis.

Results: The mean age of the study participants was 47.1 ± 11.5 years, and 36.7% of the participants were males. There was a statistically significant improvement after periodontal therapy in the BS group compared with the obese group (P <0.05). The PD had a mean reduction of 0.45 mm in the BS group versus 0.28 mm in the control group. The reduction in CAL was 0.44 mm versus 0.30 mm, percentage of BOP sites was 16% versus 15%, and GI was 1.03 versus 0.52 in the BS and control groups, respectively.

Conclusion: An improved response to non-surgical periodontal therapy is observed in obese patients who had significant weight loss after BS compared with obese patients who did not have such a surgery. J Periodontol 2012;83:684-689.

KEY WORDS
Bariatric surgery; obesity; periodontal diseases; periodontitis; weight loss.

Obesity is a major public health problem, and it has been implicated as a risk factor for several chronic health conditions, including diabetes, hypertension, and stroke, and is thus associated with increased mortality. An association between obesity and periodontitis was first noted in a rat model by Perlstein and Bissada (1977). More recently, several epidemiologic studies supported this observation. A hyperinflammatory state observed in obesity is proposed as a mechanism to explain this association. Genco et al. suggested that the hyperinflammatory state of obesity would result in an increased insulin resistance and thus predispose to diabetes, which in turn would increase the risk for periodontal disease. In contrast, a recent study suggested that obesity does not modify the local and systemic response to periodontal therapy.

Several methods have been proposed for weight loss in obese patients, including dieting and physical exercise, pharmacologic treatment, and surgical intervention. A recent systematic review concluded that bariatric surgery (BS) is a clinically effective and cost-effective intervention for moderately to severely

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obese individuals compared with non-surgical interventions. Surgery provides significant, sustained weight loss for patients who are morbidly obese and has been shown to significantly decrease overall mortality.\(^{16-18}\) Weight loss resulting from surgery also wards off the development of additional morbidities.\(^{18}\) Schauer et al.\(^{19}\) suggested that BS is a successful treatment for type 2 diabetes because of changes in hormonal secretion from the gastrointestinal tract\(^{20}\) as well as reduction of inflammatory mediators such as tumor necrosis factor-alpha (TNF-\(\alpha\)), interleukin-6 (IL-6), and leptin after the surgery.\(^{21}\) The reduction in proinflammatory cytokines secondary to significant weight loss may have an indirect effect on periodontal health. Therefore, the purpose of this exploratory study was to assess whether significant weight loss by BS improves the response to non-surgical periodontal therapy.

**MATERIALS AND METHODS**

The sample for the present study was recruited from obese patients who presented for treatment at the University Hospitals Case Medical Center in Cleveland, Ohio. The study was conducted from January to October 2010 after obtaining the Institutional Review Board approval from the University Hospitals Case Medical Center, Cleveland, Ohio (#11-09-37, December 18, 2009) and a signed written informed consent from each participant. The diagnosis of obesity was made by two physicians involved in this study (AS and LK) using the established clinical medical practice and National Institutes of Health guidelines.\(^{22}\)

Participants were recruited based on inclusion criteria: 1) \(\geq 18\) years of age, 2) diagnosis of chronic periodontitis (classified as a mean clinical attachment loss of \(\geq 2\) mm), 3) \(\geq 20\) teeth present, 4) a body mass index (BMI) >30 kg/m\(^2\), and 5) a loss of \(\geq 40\%\) excess weight after BS. Excluded from the study were: 1) pregnant females, 2) corticosteroid users, and 3) those who had undergone periodontal treatment \(\leq 1\) year before the study. A total of 30 obese patients (11 males and 19 females; mean age: 47.1 \(\pm\) 11.5 years) affected with chronic periodontitis were included. Of these, 15 patients had previously undergone BS and maintained a \(\geq 20\%\) loss of their excess weight at \(\geq 6\) months after surgery. The other 15 patients were also obese but did not have the surgery, nor did they lose weight to serve as a control group.

The following data were collected for all participants: age, sex, medical history, BMI, and medications used. In the weight loss group (BS), the percentage of weight loss after BS was also recorded. To collect baseline data, a calibrated examiner (DL) assessed all participants using a complete periodontal evaluation that included the following: probing depth (PD) and clinical attachment level (CAL) at six sites per tooth, gingival index (GI) of Löe and Silness,\(^{23}\) plaque index (PI) of Silness and Löe,\(^{24}\) and percentage of sites with bleeding on probing (BOP). Intraclinical and interclinical examiner reliability analyses were performed on five randomly selected patients who were unrelated to the study until no significant difference in the correlation between measurements was noted (\(P = 0.22\)).

Non-surgical periodontal therapy, consisting of scaling, root planing, and oral hygiene instructions, was performed after the periodontal examination. Re-examination 4 to 6 weeks after therapy was performed by the same calibrated examiner (DL).

Statistical analyses included both descriptive and analytical tests. An independent samples \(t\) test was used to assess the differences in age, number of teeth present, BMI, and periodontal parameters between the control and test groups at baseline. An \(\chi^2\) test was used to examine the differences in male/female ratio between the two groups. Changes in periodontal measurements before and after treatment within each group were analyzed using a two-sided matched-pair \(t\) test. Linear mixed-effects models compared the differences between BS and control groups after therapy for all periodontal measurements except for BOP. Linear mixed-effects models adjusted for age, sex, number of teeth, and baseline values and a random patient effect were used to account for correlation of measurements from the same patient. For BOP, the percentage of sites with bleeding per patient was calculated. The analysis was performed using the linear regression model adjusting for age, sex, and baseline values. The level of significance, \(\alpha\), was set at \(\leq 0.05\). To check the appropriateness of the sample size, a post hoc power calculation was conducted using a stand-alone power analysis program\(^{1}\) and showed that the study has a 94\% power to detect an effect size (\(\hat{h}\)) of 1.2 in CAL at an \(\alpha \leq 0.05\) level.

**RESULTS**

The mean age of the study participants was 47.1 \(\pm\) 11.5 years, and 36.7\% of the participants were males. No significant difference between the surgical group (BS) and control group was found regarding age, sex, or number of teeth present (Table 1). The only significant difference was in BMI, which was 35.1 kg/m\(^2\) in the control group and 39.4 kg/m\(^2\) in the BS group (\(P < 0.001\)).

At baseline, PD, CAL, BOP, and GI were significantly lower in the BS group compared with the control group, whereas PI was not significantly different between the two groups (Table 2). Descriptive statistics of the periodontal measurements before and after periodontal therapy are presented in Table 2. Both groups showed improvement in all periodontal measurements after the periodontal treatment (Table 2).
As shown in Figure 1, the BS group had a statistically significantly greater improvement in mean PD (0.45 versus 0.28 mm), CAL (0.44 versus 0.30 mm), and GI (1.03 versus 0.52) compared with the control group (P = 0.007, 0.03, and 0.001, respectively). The reduction in the percentage of BOP sites was also significantly higher in the BS compared with the control group (15.79% versus 14.79%, P = 0.001).

**DISCUSSION**

The results show a better response to non-surgical periodontal therapy in participants who had BS than in obese controls. This contrasts with the conclusions of Zuza et al.\(^\text{12}\) that obesity does not seem to play a negative role in the response to non-surgical periodontal therapy. One reason for the difference is that the present study focuses on severely obese participants who were candidates for BS, whereas the previous study included only obesity Class I and II. After weight loss in the BS group, a reduction in total adipocytes might have resulted in a decrease in adipokines and, subsequently, proinflammatory mediators released by those adipose cells. As such, a systemic inflammatory reduction associated with fewer adipocytes might have played a role in reducing the insulin resistance, resulting in lower blood glucose levels and thus a better outcome after periodontal therapy. Rubino et al.\(^\text{20}\) proposed a significant decrease in blood glucose, insulin, and leptin with a decrease in adrenocorticotropic hormone only 3 weeks after the BS, when there was still no significant change in weight loss. The authors attributed their findings to the hormonal changes resulting from the surgical procedure itself. After the BS, food and salivary secretions go directly into the distal jejunum, bypassing the duodenum and the proximal jejunum. This bypass might have stimulated the release of gut hormones that inhibit the brain appetite center and suggest an endocrinal mechanism for controlling diabetes. In the present study, presurgical and post-surgical glucose levels tended to be lower after surgery (Table 3). Future studies are needed to closely examine the relationship between change in glucose tolerance after BS and periodontal therapy outcomes. This hormonal mechanism could possibly explain the better response in the BS group in the present study despite a lower BMI in the control group.

Recently, Shimada et al.\(^\text{25}\) proposed that non-surgical periodontal treatment decreased the serum leptin, IL-6, and C-reactive protein (CRP) levels of the average population. Therefore, the significant reduction in leptin after the periodontal therapy may be another mechanism for the reduction in glucose levels in patients with diabetes after periodontal treatment, which would re-emphasize the influential role of leptin, which is considered to have a dual function as a hormone and a cytokine.\(^\text{25}\) As a hormone, leptin has a negative feedback signal on the hypothalamus to

### Table 1.

**Demographic, Dental, and Systemic Health Characteristics of the Study Sample**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control (n = 15)</th>
<th>Surgery (n = 15)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)*</td>
<td></td>
<td></td>
<td>0.778</td>
</tr>
<tr>
<td>No. of teeth present*</td>
<td></td>
<td></td>
<td>0.583</td>
</tr>
<tr>
<td>Males (%)</td>
<td></td>
<td></td>
<td>0.103</td>
</tr>
<tr>
<td>BMI (kg/m(^2))*</td>
<td></td>
<td></td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

* Data are presented as mean ± SD.

### Table 2.

**Descriptive Statistics of Periodontal Parameters in the Two Groups at Baseline and After Periodontal Treatment**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (mean ± SD)</th>
<th>Surgery (mean ± SD)</th>
<th>P Value Differences Between Groups at Baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline After Treatment</td>
<td>Baseline After Treatment</td>
<td></td>
</tr>
<tr>
<td>PD (mm)</td>
<td>3.1 ± 0.8 2.8 ± 0.7 &lt;0.001</td>
<td>2.6 ± 0.6 2.2 ± 0.5 &lt;0.001</td>
<td>0.004</td>
</tr>
<tr>
<td>CAL (mm)</td>
<td>3.1 ± 0.8 2.8 ± 0.7 &lt;0.001</td>
<td>2.6 ± 0.6 2.2 ± 0.5 &lt;0.001</td>
<td>0.002</td>
</tr>
<tr>
<td>BOP (%)</td>
<td>28.6 ± 27.8 13.6 ± 23.6 &lt;0.001</td>
<td>17 ± 23.6 1.3 ± 5.5 &lt;0.001</td>
<td>0.011</td>
</tr>
<tr>
<td>GI</td>
<td>1.42 ± 0.4 0.9 ± 0.6 &lt;0.001</td>
<td>1.2 ± 0.3 0.2 ± 0.4 &lt;0.001</td>
<td>0.003</td>
</tr>
<tr>
<td>PI</td>
<td>1.27 ± 0.5 0.4 ± 0.5 &lt;0.001</td>
<td>1.2 ± 0.4 0.1 ± 0.3 &lt;0.001</td>
<td>0.28</td>
</tr>
</tbody>
</table>
reduce the appetite and increase the metabolic rate.\textsuperscript{26,27} As a cytokine, leptin plays a significant role in the progression of the inflammatory process and thus has been implicated in the pathogenesis of autoimmune inflammatory conditions, such as diabetes\textsuperscript{28} and rheumatoid arthritis.\textsuperscript{29,30} Moreover, leptin can directly increase the production of cytokines such as IL-6, which could increase the release of CRP from hepatocytes.\textsuperscript{31} CRP, however, can directly inhibit the binding of leptin to its receptors, suggesting a mechanism of leptin resistance, with attenuation in its physiologic function.\textsuperscript{32} This suggested mechanism may explain the resistance to leptin observed in obesity. The leptin reduction after periodontal therapy shown by Shimada et al.\textsuperscript{25} may explain the better response to periodontal treatment after BS.

Another explanation for the better response to periodontal therapy in the BS group could be the change in lifestyle of patients. Patients who underwent BS were most likely motivated to adopt health-enhancing behaviors, such as diet improvement and physical exercise. A high-quality diet and an increased level of physical activity are associated with improved periodontal health.\textsuperscript{33-35} Therefore, the positive changes in lifestyle in the test group might also have contributed to the better periodontal response after periodontal therapy. Positive changes in the lifestyle, improved diet, and physical activity have been shown to reduce overall systemic inflammation, with positive effects on other conditions, such as cardiovascular diseases and chronic periodontitis.\textsuperscript{36,37}

A recent study by Goodson et al.\textsuperscript{38} proposed that oral bacteria might play a role in the pathology that leads to obesity. The authors suggested several mechanisms by which oral bacteria could affect body weight and contribute to obesity by increasing metabolic efficiency, increasing appetite, and redirecting energy metabolism by facilitating insulin resistance through increasing levels of TNF-\(\alpha\) or reducing levels of adiponectin. Additional investigation into the mechanism underlying the association between obesity and periodontitis is warranted.

In the present study, there was a significant difference in the periodontal parameters of the control and study groups at baseline. All periodontal measurements were greater in the control compared with the BS group except for the PI. The control group had more periodontal inflammation compared with the test group, despite their similar oral hygiene levels, suggesting a positive effect of weight loss on periodontal health. It has to be noted, however, that in the present study adjustments were made for the baseline periodontal parameters in the linear mixed-effect models that compared the changes in periodontal parameters after periodontal therapy between the two groups. Thus, the differences in the baseline values between the two groups are less likely to explain the better response to periodontal therapy in the BS group. Future investigations comparing periodontal health and response to periodontal treatment in obese patients before and after BS are needed to
definitively conclude that surgical weight loss plays a significant role.

CONCLUSIONS
This exploratory clinical study shows an improved response to non-surgical periodontal therapy in obese patients who lost significant weight after BS compared with obese patients who did not have such a surgery. Additional longitudinal studies with larger sample sizes are needed to confirm or refute the findings from the present study.

ACKNOWLEDGMENTS
This study was supported by the Department of Periodontics at Case Western Reserve University School of Dental Medicine. The authors report no conflicts of interest related to this study.

REFERENCES


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Submitted April 15, 2011; accepted for publication September 6, 2011.