Peri-implant parameters and C-reactive protein levels among patients with different obesity levels

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1 | INTRODUCTION

Obesity is defined as increased adipose tissue to a degree in which health is impaired and is mainly characterized by systemic low-grade inflammation. Individuals with a body mass index (BMI) of 27.5–34.9 kg/m², 35–39.9 kg/m², and ≥40 kg/m² are categorized as class I, II, and III obese, respectively. It is one of the main causes of morbidity and mortality in current society and has become a major public health problem. Recent data reveals that an estimate of 150 million individuals suffer from some form of obesity and it is estimated

Abstract

Background: It is hypothesized that peri-implant conditions are worse with increasing severity of obesity, because systemic low-grade inflammatory marker (C-reactive protein [CRP]) is higher in severe form of obese individuals.

Purpose: The aim of the cross-sectional retrospective study was to compare clinical and radiographic peri-implant inflammatory parameters in patients with different levels of obesity and correlate these parameters with CRP levels.

Materials and methods: Eighty-four patients who participated in this study were divided into 4 groups: class I obese (group 1), class II obese (group 2), class III obese (group 3), and nonobese individuals (group 4) were included. Clinical (plaque index [PI], bleeding on probing [BOP], probing depth [PD]) and radiographic (marginal bone loss [MBL]) peri-implant parameters were recorded. Serum CRP were quantified using enzyme-linked immunosorbent assay (ELISA). Clinical peri-implant parameters and serum CRP concentrations were analyzed using 1-way analysis of variance. The Pearson correlation coefficient was used to analyze correlations of CRP levels with any of the clinical and radiographic parameters assessed.

Results: Peri-implant PI, BOP, PD, and MBL were significantly higher in group-1, -2, and -3 patients as compared to nonobese individuals (P < .05). Peri-implant PI, BOP, PD, and MBL were significantly higher in obese patients of group-2 and group-3 as compared to obese patients in group-1 (P < .01). Mean PI, BOP, PD, and MBL were comparable between group-2 and group-3 patients (P > .05). A significant positive correlations were found between CRP levels and BOP (P = .0148) and PD (P = .0425); and significant negative correlation was found for MBL in group 3, respectively (P = .0212).

Conclusion: Clinical and radiographic peri-implant inflammatory parameters and serum CRP were significantly high in patients with severe form of obesity. Serum CRP levels correlated with peri-implant bleeding in obese patients. These findings are preliminary and long-term controlled trials are recommended to support these outcomes.

KEYWORDS
alveolar bone loss, bleeding on probing, C-reactive protein, dental implants, obesity
that the unexpected plateauing of adult obesity rates may increase up to 300 million by 2025.\textsuperscript{5} Concern is growing about the rising prevalence of overweight and obesity in adults in Saudi Arabia. An estimated prevalence of 36\% of adults suffer from obesity and it is projected that 41\% in men and escalating rise of 78\% in women by 2022, respectively.\textsuperscript{6}

Evidence suggests that obesity is associated with the build-up of constant systemic inflammation.\textsuperscript{7,8} A systemic proinflammatory state in obesity is suggested for increased alveolar bone loss with altered immune response because of compromised functional state of immune cells.\textsuperscript{9} C-reactive protein (CRP) in particular, is a marker of systemic inflammation and is associated with obesity and periodontitis.\textsuperscript{10} Elevated levels of CRP is characterized by dysregulated and increased production of several proinflammatory cytokines that are implicated in the pathogenesis of periodontal destruction.\textsuperscript{9,11,12}

In the past decade, obesity has been thoroughly investigated as one of the risk factors for periodontal tissue destruction and association between increased adiposity and poor periodontal outcomes is well elucidated.\textsuperscript{13–16} In a recent cross-sectional study by Abduljabbar and colleagues,\textsuperscript{17} it was concluded that obese individuals showed increased peri-implant probing depth (PD) and higher amount of marginal bone loss (MBL) as compared to nonobese individuals. However, the correlation of peri-implant tissue destruction with the severity of obesity remains uninvestigated. We hypothesize that peri-implant inflammatory parameters are compromised with increasing severity of obesity, because systemic low-grade inflammatory marker (CRP) is higher in severe form of obese individuals.\textsuperscript{7–9} Therefore, the aim of the present cross-sectional retrospective study was to compare clinical and radiographic peri-implant inflammatory parameters in patients with different levels of obesity and correlate serum CRP levels with clinical and radiographic peri-implant parameters.

2 | MATERIALS AND METHODS

2.1 | Ethical guidelines

The study was performed following guidelines recognized by the Declaration of Helsinki as revised in 2013 for experimentation involving human patients. An information sheet was provided to the patients that explained the objectives and methods of the present study and individuals had the right to retire from the study at any stage without penalty.

2.2 | Study groups

The study analyzed a total of 84 obese and nonobese individuals recruited from private dental referral clinic (Dr F Alkhudhairy, Saudi Arabia). These patients were further divided into 4 groups on the basis of BMI as follows; Group-1: class-I obese individuals with BMI 27.5–34.9 kg/m\textsuperscript{2}; Group-2: class-II obese individuals with BMI 35–39.9 kg/m\textsuperscript{2}; Group-3: class-III obese individuals with BMI $\geq$40 kg/m\textsuperscript{2}; Group-4: nonobese individuals with BMI 18.5–22.9 kg/m\textsuperscript{2}.\textsuperscript{4}

2.3 | Eligibility criteria

As inclusion criteria, all patients were (1) $>30$ years of age and; (2) obese individuals classified according to World Health Organization (WHO) classification of obesity for Asian cohort which defines BMI $\geq$27.5 kg/m\textsuperscript{2}; (3) nonobese individuals with a BMI ranging between 18.5 and 22.9 kg/m\textsuperscript{2}; and (4) dental implants which has been in service for $\geq$36 months.

Self-reported tobacco smokers, individuals using smokeless tobacco products,\textsuperscript{18} habitual alcohol users and patients with systemic diseases such as acquired immune deficiency syndrome/HIV, renal disorders, and cardiovascular disorders were excluded.

2.4 | Serum sampling for high-sensitivity CRP and lipid parameters

Blood samples were collected from the antecubic fossa of the left forearm of each patient in the morning and processed, divided into aliquots and stored locally at $-80^\circ$C until final analysis. High-sensitivity CRP was assessed using an automated immunoanalyzer (IMMULITE; Diagnostics Products Corporation, Los Angeles, California). Lipid parameters were measured in the serum samples including triglycerides, total cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and glucose parameters, including fasting blood glucose levels (FBGL), insulin, and homeostasis model assessment of insulin resistance (HOMA-IR). HOMA-IR was calculated as follows: [FBGL (mg/dL)] $\times$ [fasting serum insulin (mu/mL)]/405 formulate.\textsuperscript{19} The HOMA-IR score $>2.7$ estimates insulin resistance.

2.5 | Clinical peri-implant examination

A trained and calibrated examiner (FA), who was blinded to the study groups performed the clinical examinations. The overall kappa value for intra-examiner reliability was 0.91 which was considered a good agreement. Peri-implant status was based upon recording peri-implant plaque index (PI), bleeding on probing (BOP), and PD with references to the Consensus report of the Seventh European Workshop on Periodontology-2011.\textsuperscript{20} All recordings were measured at 6 sites per implant (mesiobuccal, mid-buccal, distobuccal, distolingual/palatal, mid-lingual/palatal, and mesiolingual/palatal) and presented as mean percentages per individual. PD was measured to the nearest millimeter using a manual graded probe (UNC-15 Hu-Friedy, Chicago, Illinois).

2.6 | Marginal bone loss

All radiographic examinations were performed by a trained and calibrated investigator (FK) who was blinded to the study groups (kappa = 0.83). Digital periapical radiographs were taken and viewed on a calibrated computer screen (Samsung SyncMaster digital TV monitor, Korea) using a software program (Image Tool 3.0 Program, Department of Dental Diagnostic Science, University of Texas Health Science Center, San Antonio, Texas). MBL was defined as the distance from the widest supracrestal part of the implant to the alveolar crest.\textsuperscript{21}
2.7 | Statistical analyses

Statistical analyses were carried out using a statistical software (SPSS v. 20, IBM, Chicago, Illinois). Data were expressed as means and standard deviations with percentages and ranges. Normality of distribution of the variables was tested with Shapiro-Wilk tests and confirmed with Q–Q plots. Between-group comparison of means was verified with Kruskal–Wallis test. The Pearson correlation coefficient was used to analyze for correlations of CRP levels with any of the clinical and radiographic peri-implant parameters assessed. Stepwise logistic regression analysis was employed to identify explanatory variables for peri-implant outcomes, controlling for the effect of possible covariate such as FBGL, TC, LDL, triglyceride levels, HOMA-IR, and oral hygiene care. The direction and strength of association between peri-implant outcomes and covariates were assessed by generating odds ratios, the precision of which could be measured by 95% confidence intervals. Differences were considered significant with \( P < .05 \).

3 | RESULTS

3.1 | Characteristics of the study groups

In total, 84 individuals were included. Among all the 4 groups, there were 25 class I obese patients (group 1), 22 class II obese patients (group 2), 12 class III obese patients (group 3), and 25 nonobese patients (group 4) as controls (Figure 1). A total of 39 implants in group 1, 35 in group 2, 26 in group 3, and 43 in group 4 were assessed. The mean duration of implants in the study groups ranged from 42.1 to 78.3 months. The mean age of obese (class I, II, III) and nonobese individuals was 49.3 years (42–56 years), 51.8 years (40–54 years), 50.4 years (45–59 years), and 52.1 years (39–58 years), respectively. Mean CRP levels among obese class I, II, and III patients was 3.18 mg/L, 3.34 mg/L, and 3.97 mg/L, respectively. FBGL in obese class I, II, and III individuals were 104.8 mg/dL, 126.3 mg/dL, and 132.7 mg/dL, respectively, whereas, FGBL in nonobese individuals was 86.1 mg/dL. The mean BMI of obese and nonobese individuals was 32.2 kg/m², 38.8 kg/m², 44.3 kg/m², and 21.1 kg/m², respectively. Among patients in groups 1, 2, and 3, the duration of obesity was 6.1 years, 8.7 years, and 9.3 years, respectively. Metabolic parameters including TC, LDL, TG, and HOMA-IR showed statistical difference between group 3 and group 4 subjects (Table 1).

3.2 | Clinical and radiographic peri-implant parameters

The mean scores of peri-implant PI \( (P < .05) \), BOP \( (P < .05) \), and PD \( (P < .05) \) were significantly higher in group-1, -2, and -3 patients as compared to nonobese individuals. Peri-implant PI \( (P < .01) \), BOP \( (P < .01) \), and PD \( (P < .01) \) were significantly higher in obese patients of group-2 and group-3 as compared to obese patients in group-1. Peri-implant PI was significantly higher in group-3 and group-4 patients as compared to group-1 patients \( (P < .05) \). Peri-implant BOP and PD were comparable in obese individuals of group-2 and group-3 (Table 2).

MBL was significantly higher in group-1, group-2, and group-3 patients as compared to nonobese individuals \( (P < .05) \). MBL was significantly higher in group-2 and group-3 patients as compared to group-1 patients \( (P < .01) \). Similarly, there was no significant difference in MBL between group-2 and group-3 patients \( (P > .05) \) (Table 2).

3.3 | Correlation of CRP with clinical and radiographic peri-implant parameters

Pearson correlation coefficient was calculated to analyze for any correlations among CRP levels and the clinical and radiographic parameters assessed in all 4 groups (Table 3). When the individual groups were analyzed, a significant positive correlations were found between CRP levels and BOP \( (P = .0148) \), and PD \( (P = .0425) \); and significant negative correlation was found for MBL in group 3, respectively \( (P = .0212) \).

3.4 | Regression analysis to control metabolic parameters

The logistic regression analysis revealed that peri-implant outcomes showed statistically significant difference even after adjusting for FBGL, TC, LDL, HDL, and TRG \( (P < .05) \) (Table 4).

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**FIGURE 1** Flow diagram showing recruitment of study participant
To our knowledge from indexed literature, this is the first study to assess the clinical and radiographic peri-implant parameters among different levels of obesity and correlate these parameters with the levels of CRP. The present study was based on the hypotheses that peri-implant inflammatory parameters (PI, BOP, PD) and radiographic bone loss (MBL) is compromised in severe obesity forms as compared to individuals with mild or no obesity. The present result showed that peri-implant parameters were compromised in obese patients than nonobese patients, however, class II and class III obese patients showed similar peri-implant tissue damage. Moreover, there was a significant positive correlation between CRP levels and BOP and PD in class III obese patients. Obesity has been associated with metabolic disturbances and its effects on other chronic diseases such as osteoarthritis, cardiovascular complications, type 2 diabetes mellitus, and cancers are well-documented. It is speculated that obesity increases the risk of periodontal attachment loss. The pathological mechanisms involved in peri-implant tissue damage in obesity may be well-explained by similar mechanisms involved in periodontitis. It is proposed that expansion of white adipose tissue leads to generalized chronic inflammation.

### TABLE 1  General characteristics of the study groups

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Group-1 Obese I</th>
<th>Group-2 Obese II</th>
<th>Group-3 Obese III</th>
<th>Group-4 Nonobese</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of study participants (n)</td>
<td>25</td>
<td>22</td>
<td>12</td>
<td>25</td>
</tr>
<tr>
<td>Mean age in years (range)</td>
<td>49.3 (42–56)</td>
<td>51.8 (40–54)</td>
<td>50.4 (45–59)</td>
<td>52.1 (39–58)</td>
</tr>
<tr>
<td>Total number of implants</td>
<td>39</td>
<td>35</td>
<td>26</td>
<td>43</td>
</tr>
<tr>
<td>Implant position (maxilla/mandible)</td>
<td>22/17</td>
<td>21/14</td>
<td>16/10</td>
<td>21/22</td>
</tr>
<tr>
<td>Duration of implants in months (mean ± SD)</td>
<td>73.4 ± 12.6</td>
<td>51.2 ± 14.9</td>
<td>42.1 ± 21.8</td>
<td>78.3 ± 16.5</td>
</tr>
<tr>
<td>Brushing frequency (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Once daily</td>
<td>85</td>
<td>81</td>
<td>93</td>
<td>77</td>
</tr>
<tr>
<td>Twice daily</td>
<td>15</td>
<td>19</td>
<td>7</td>
<td>23</td>
</tr>
<tr>
<td>Mean CRP in mg/L (range)</td>
<td>3.18 (2.65–3.34)</td>
<td>3.34 (2.97–3.89)</td>
<td>3.97abc (3.85–4.16)</td>
<td>2.56 (1.77–2.83)</td>
</tr>
<tr>
<td>Fasting blood glucose in mg/dL (mean ± SD)</td>
<td>104.8 ± 7.6c</td>
<td>126.3 ± 8.8k</td>
<td>132.7 ± 8.4k</td>
<td>86.1 ± 7.2</td>
</tr>
<tr>
<td>Mean BMI in Kg/m² (range)</td>
<td>32.2 (30.9–33.8)</td>
<td>38.8 (36.6–38.9)</td>
<td>44.3 (40.4–47.5)</td>
<td>21.1 (20.9–22.7)</td>
</tr>
<tr>
<td>Duration of obesity in years (range)</td>
<td>5.1 (5–7)</td>
<td>8.7a (6–10.7)</td>
<td>9.3a (7–11.4)</td>
<td>–</td>
</tr>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>195 ± 37</td>
<td>199 ± 32</td>
<td>209 ± 44c</td>
<td>178 ± 28</td>
</tr>
<tr>
<td>HDL (mg/dL)</td>
<td>105 ± 21</td>
<td>108 ± 24</td>
<td>116 ± 24c</td>
<td>101 ± 22</td>
</tr>
<tr>
<td>Triglyceride (mg/dL)</td>
<td>41 ± 7</td>
<td>43 ± 6</td>
<td>45 ± 6</td>
<td>41 ± 6</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>3.8 (0.8–7.3)c</td>
<td>5.3 (1.1–9.7)c</td>
<td>7.0 (2.2–12.1)c</td>
<td>1.1 (0.62–4.9)</td>
</tr>
</tbody>
</table>

**Abbreviations:** BMI, Body mass index; CRP, C-reactive protein; LDL, low-density lipoprotein; HDL, high-density lipoprotein; HOMA-IR, homeostasis model assessment of the insulin resistance.

aSignificantly different from group-1 (P < .05).
bSignificantly different from group-2 (P < .05).
cSignificantly different from group-4 (P < .05).

### TABLE 2  Peri-implant clinical and radiographic parameters among patients in group 1, 2, 3, and 4

<table>
<thead>
<tr>
<th>Peri-implant characteristics</th>
<th>Group-1 Obese I (n = 25)</th>
<th>Group-2 Obese II (n = 22)</th>
<th>Group-3 Obese III (n = 12)</th>
<th>Group-4 Nonobese (n = 25)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean plaque index in % (range)</td>
<td>20.5a (13.1–21.7)</td>
<td>27.8b (18.5–30.9)</td>
<td>28.1b (26.6–34.6)</td>
<td>13.6 (5.2–16.4)</td>
</tr>
<tr>
<td>Mean bleeding on probing in % (range)</td>
<td>19.1c (13.4–22.4)</td>
<td>25.4d (16.7–32.5)</td>
<td>27.8d (24.3–32.8)</td>
<td>11.7 (5.1–14.6)</td>
</tr>
<tr>
<td>Mean probing depth in mm (range)</td>
<td>2.4c (1.9–2.8)</td>
<td>3.1b (2.7–3.8)</td>
<td>3.6b (2.3–4.1)</td>
<td>1.2 (0.6–2.0)</td>
</tr>
<tr>
<td>Mean marginal bone loss in mm (range)</td>
<td>1.8c (1.9–3.3)</td>
<td>2.6d (2.4–4.5)</td>
<td>2.7d (3.3–5.1)</td>
<td>0.9 (0–2.5)</td>
</tr>
</tbody>
</table>

aCompared to groups 3 and 4.
bCompared to groups 1 and 4.
cCompared to groups 2, 3, and 4.

dCompared to groups 1 and 2.
different mechanisms that is over expression of proinflammatory cytokines (interleukin (IL)-1β, IL-6, tumor necrosis factor-alpha) into the bloodstream and stimulating macrophages. Hence, this chronic inflammation increases the susceptibility of obese patients to infectious challenges by down regulating the immune response. In addition, the increased prevalence of periodontal tissue damage in obese individuals becomes strong with unhealthy diet. It is proposed that diet rich in high cholesterol and fatty acids may modulate alveolar bone loss regardless of increased BMI. These immunological and metabolic findings may explain the increased susceptibility of patients with obesity to peri-implant breakdown.

The finding of the present study states that the PD levels were higher in group 2 and group 3 patients. In a recent study by Maciel and colleagues, it was concluded that obese patients with deep PDs harbored higher levels of periodontopathogenic bacteria as compared to nonobese individuals. Although the cause of this association is still unknown, it is speculated that obesity-associated immune changes in periodontal tissues alter the pocket environment and/or host defenses, affecting the subgingival microbial colonization. Further studies assessing the microbiological and immune-inflammatory aspect of peri-implant sites in obese patients are recommended to confirm this hypothesis.

Numerous studies have reported a strong association between systemic CRP levels and poor periodontal and peri-implant health. Furthermore, higher levels of CRP are documented in obese patients as compared to nonobese individuals. It needs to be stressed that sites with peri-implantitis are histologically characterized by elevated levels of proinflammatory cells and cytokines which may further increase the degree of BOP. Our study demonstrated a significant positive association of CRP levels with BOP and PD. This possible association between CRP levels and BOP may reflect the potential significance of the local peri-implant inflammatory burden in systemic inflammation (obesity).

It has been reported that chronic hyperglycemia is associated with excessive formation of advanced glycation end products (AGEs) in the tissues. AGEs are coupled with impaired fibroblastic growth and increased production of proinflammatory cytokines (including IL-1β and IL-6). Moreover, it is indicated that chronic hyperglycemia weakens the chemotactic and phagocytic function of neutrophils (that prevent destruction of bacteria in deep pockets), which may be a possible justification for enhanced peri-implant tissue damage. It is noteworthy that in the present study that some obese patients in group 2 (class II) and group 3 (class III) were hyperglycemic, therefore it is speculated that the oxidative stress (induced as a result of hyperglycemia) worsened peri-implant inflammation in these patients thereby further compromising clinical (PI, BOP, PD), and radiographic (MBL) parameters of peri-implant inflammation. However, the effect of FBGL levels on peri-implant inflammation in obese patients were controlled (through stepwise logistic regression analysis); nevertheless, the effects of hyperglycemia cannot be overruled.

Severity of peri-implant inflammation in patients with obesity may be associated with the attitude toward oral hygiene and duration of the obesity. The higher percentage of PI among obese patients may be attributed to the neglected attitude toward oral hygiene care. Furthermore, in the present study, duration of obesity among patients in class II (8.7 years) and class III (9.3 years) obese individuals was almost similar and higher than class I (5.1 years) obese individuals. It is speculated that duration of obesity among patients could have influenced the previously existing peri-implant inflammatory state in these patients. Moreover, the extrapolation of the changes in the anthropometric values among the test groups was not possible, and therefore, this may have disfigured the peri-implant outcomes between various studied groups. In addition it is suggested that patients with peri-implant inflammation with a longer history of obesity may be more susceptible to tissue destruction. Further studies are warranted to test these hypotheses.

There are certain limitations in the present study that should not be disregarded. The present study only assessed clinical and radiographic peri-implant parameters. Perhaps the sampling of peri-implant

### Table 3

<table>
<thead>
<tr>
<th>Peri-implant inflammatory parameters</th>
<th>Group-1</th>
<th>Group-2</th>
<th>Group-3</th>
<th>Group-4</th>
</tr>
</thead>
<tbody>
<tr>
<td>PI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation coefficient</td>
<td>.8001</td>
<td>.6713</td>
<td>-.1976</td>
<td>-.5167</td>
</tr>
<tr>
<td>P value</td>
<td>.9126</td>
<td>.7128</td>
<td>.2462</td>
<td>.0381*</td>
</tr>
<tr>
<td>BOP</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation coefficient</td>
<td>.5127</td>
<td>-.3572</td>
<td>.1634</td>
<td>-.3145</td>
</tr>
<tr>
<td>P value</td>
<td>.1728</td>
<td>.5793</td>
<td>.0148*</td>
<td>.4810</td>
</tr>
<tr>
<td>PD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation coefficient</td>
<td>.1341</td>
<td>.4559</td>
<td>.1916</td>
<td>-.7669</td>
</tr>
<tr>
<td>P value</td>
<td>.8725</td>
<td>.3958</td>
<td>.0425*</td>
<td>.0412*</td>
</tr>
<tr>
<td>MBL</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation coefficient</td>
<td>.4201</td>
<td>-.1570</td>
<td>-.5883</td>
<td>-.4492</td>
</tr>
<tr>
<td>P value</td>
<td>.0177</td>
<td>.3650</td>
<td>.0212*</td>
<td>.8264</td>
</tr>
</tbody>
</table>

*Significant at $P < .05$.  

**Table 4** Logistic regression model for covariates influencing peri-implant outcomes with odds ratios, 95% confidence intervals (CI), and P values

<table>
<thead>
<tr>
<th>Covariate</th>
<th>Odds ratio</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FBG</td>
<td>2.14</td>
<td>0.78, 5.13</td>
<td>.039</td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>2.59</td>
<td>1.18, 4.72</td>
<td>.032</td>
</tr>
<tr>
<td>LDL</td>
<td>3.72</td>
<td>1.01, 6.11</td>
<td>.075</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>2.29</td>
<td>1.52, 6.88</td>
<td>.017</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>1.74</td>
<td>1.21, 4.1</td>
<td>.002</td>
</tr>
</tbody>
</table>

Abbreviations: FBG, fasting blood glucose; LDL, low-density lipoprotein; HDL, high-density lipoprotein.  
Bold denotes statistically significant result at $P < .05$ in direction of increased risk.
crevicular fluid for the detection of proinflammatory cytokines may have resulted in the analysis of site specific destruction around implants among different levels of obesity. In addition, the cross-sectional nature of the present study assessed clinical and radiographic findings at only 1 time point and that longitudinal study may have helped to analyze the evolution of destruction of soft and hard peri-implant tissue. Moreover, the unmatched number of study individuals in group-3 (class III obese) with other study groups may have skewed the results. With these limitations, patients with severe form of obesity should be considered as having increased susceptibility toward peri-implant inflammation. It is recommended that clinicians should educate and warn obese patients about the biological response around dental implants and provide frequent oral hygiene screening and maintenance procedures in patients with increasing severity of obesity (high risk).

5 | CONCLUSION

Within the limits of this cross-sectional retrospective study, it is concluded that clinical and radiographic peri-implant inflammatory parameters were significantly high in patients with severe form of obesity. Serum CRP levels correlated with peri-implant bleeding in obese patients. These findings are preliminary and long-term controlled trials are recommended to support these outcomes.

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CONFLICT OF INTEREST

The authors declare that they have no conflict of interests and all authors have read and approved the final draft.

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