Effect of smoking on immediate loaded implants placed by flapless computer guided surgery to support full arch fixed restoration in edentulous maxilla. One-year clinical and radiographic trial

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ABSTRACT

**Purpose:** The aim of this prospective case-control clinical trial was to evaluate the effect of smoking on immediate loaded implants placed by flapless computer guided surgery to support full arch fixed restoration in edentulous maxilla.

**Materials and Methods:** This study was conducted on 10 male patients with completely edentulous maxillary ridge whom divided into 2 groups. Study group comprised of 5 smoker patients who smoked > 10 cigarettes per day. The control group comprised of 5 participants who had not smoke for at least 5 years and were case matched to study group. All patients received 6 implants in the maxillary ridge using computer guided surgery and flapless surgical protocol and the implants were immediately loaded with fixed acrylic bridge. 6 months later, final ceramo-metal screw-retained prosthesis was delivered. Clinical (plaque scores, gingival scores, pocket depth, implant stability, width of keratinized mucosa) and radiographic outcomes (crestal bone loss) were measured at implant loading, 6 and 12 months later.

**Results:** Implant survival rate was 93.3% and 80% for non-smoker and smoker groups with significant difference between groups. At 6 and 12 months, smoker group recorded significant higher plaque and gingival scores, pocket depth, and crestal bone loss than non-smoker groups. At 6 months only, smoker group recorded significant higher implant stability than non-smoker groups, however the difference disappeared after 12 months. No difference in width of keratinized mucosa was observed between groups.

**Conclusion:** Within the limits of this study, smoker patients were associated with worsen clinical and radiographic outcomes of immediate loaded implants placed by flapless computer guided surgery to support full arch fixed restoration in edentulous maxilla compared to non-smoker patients.

**Key Words:** Flapless, Guided, Implant, Maxilla, Smoking.

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cause periodontal bone loss\textsuperscript{14}. There is no consensus on the relation between number of cigarettes smoked and implant failure\textsuperscript{11}. According to a systematic review and meta-analysis, smoker patients were associated with significant higher implant failure and marginal bone loss than non-smoker patients\textsuperscript{13}. Conversely, Sgolastra et al\textsuperscript{16}, in another systematic review reported that there is low evidence of smoking as a risk factor for development of peri-implantitis.

The implant failures in smoker patients may be affected by other confounding factors as a history of periodontitis, characteristics of the implant (shape and surface treatment), the opposing occlusion, the used surgical protocol, and the loading protocol\textsuperscript{13}. Controversy exists in the literature regarding to the effect of smoking and these confounding factors on implant failure. Regarding surgical protocol, some studies\textsuperscript{17, 18} reported statistically higher implant failure after stage-two surgery in smoker patients than after initial placement. Another study showed that implants placed in extraction sockets using flapless protocol and immediately loaded with acrylic prostheses had no significant difference in marginal bone loss and implant survival rate between smokers and non-smokers\textsuperscript{19}. Regarding prosthetic protocol, Romanos et al. reported that clinical outcomes (implant survival and marginal bone loss) of platform switched implants inserted in heavy smokers and immediately loaded, without subsequent removal of the abutments appear to be comparable to those for nonsmokers\textsuperscript{14}. Similarly, Cassetta et al\textsuperscript{20} reported that marginal bone loss was not affect by smoking status when immediate loaded implants were inserted using the mucosa supported stereolithographic guides and flapless approach in maxillary and mandibular arches. In contrast, Sanna et al.\textsuperscript{9} reported that smokers were associated with significant higher implant failures and bone loss than non-smokers when implants were inserted in edentulous arches using computer guided flapless approach and immediately loaded with fixed prosthesis. There is no consensus in the literature about the surgical and prosthetic protocol that can reduce the risk of smoking on implant success. However, submerging of implants may reduce contact with the nicotine and its products and prevent formation of bacterial biofilms, which may enhance healing of implants\textsuperscript{13}.

Reviewing the literature, several studies evaluated the clinical efficacy and predictability of computer planned flapless guided surgery and immediate loading of the implants with full arch fixed restorations\textsuperscript{21 - 23}. However, the effect of the smoking on clinical and radiographic outcomes of immediately loaded implants inserted using computer guided flapless surgery was scarce in the literature and limited to trials which evaluate both maxillary and mandibular arches\textsuperscript{8, 20}. Moreover, these studies did not concern with evaluation of peri-implant soft tissue parameters and implant stability. Accordingly, the aim of the present clinical trial was to evaluate the effect of smoking on immediate loaded implants placed by flapless computer guided surgery to support full arch fixed restoration in edentulous maxilla. The authors hypothesized that there will be no significant difference in clinical and radiographic outcomes between smoker and non-smoker patients.

**MATERIALS AND METHODS**

**Patient cohort and study design:**

This case-control study was conducted on 10 male patients with completely edentulous maxillary ridge who were divided into two groups. The study group comprised of 5 smoker patients with completely edentulous maxillary ridge (mean age = 54 ± 4.6 years) who were selected from the outpatient clinic of the oral and maxillofacial department. The patients smoked > 10 cigarettes per day for at least 5 years. The control group comprised of 5 participants who had not smoke for at least five years and the participants were case matched to study group regarding age, gender, status maxillary and mandibular arch. For both groups, the inclusion criteria are:

1) sufficient bone quantity and quality to receive six implants of at least (10 mm in length and 3.5 mm in diameter). This was evaluated by preoperative diagnostic cone beam computerized tomography (CBCT).

2) natural teeth and/or fixed restoration presented in the mandibular arch and complete dentures in the maxillary arch with bilateral balanced occlusion.

3) Sufficient interarch and restorative space to allow construction of fixed restorations in the maxillary arch. The exclusion criteria include:

1) patient with history of periodontitis,

2) public disease that affect bone activity such as diabetes mellitus,

3) patient with chemotherapy or radiotherapy,

4) patients with normal maxillomandibular relations, and

5) patients with bad oral hygiene. Patients were required to sign an informed consents after explaining the objectives of the study. The study protocol was approved by the ethical committee of the faculty of Dentistry. All patients received 6 implants in the maxillary ridge using computer guided surgery and flapless surgical protocol and the implants were immediately loaded with fixed acrylic bridge. Six months later, the final ceramo-metal fixed full arch screw-retained prosthesis was delivered.

**Surgical and prosthetic procedures:**

For all participants, scaling and root planning was performed for mandibular natural teeth, adjustment of the occlusal plane of the natural teeth was performed using selective grinding, and fixed/ removable restoration that replace missing teeth (if present) in the mandibular arch was constructed. New maxillary complete denture was constructed with bilateral balanced occlusion. A mucosal
supported Sterolithographic surgical guide was constructed to be used for computer guided flapless implant placement. Radiopaque markers (Gutta purcha) were added to the buccal and palatal polished surfaces of the denture, then dual scan protocol was made for each participant using cone beam computerized tomography (i-CAT, Hatfield, PA, USA). The first scan was performed while the patients wearing the denture and close in centric occlusion. The second scan was performed for the denture alone (placed on the table of the device). The two scans were overlapped together and loaded into a computer software (On Demand). The scans were used to construct a three-dimensional image of the edentulous maxillary ridge. Using the computer software, planning for proper position and orientation of the implant was performed. Moreover, proximity to vital structures (maxillary sinus, naso-incisive foramina and the nasal cavity) were identified and the selection of implants length and width was completed. Implants were positioned in the central/lateral incisors area, canine area and second premolar area (Figure 1).

The plan was used to direct 3D printer to print tissue supported stereolithographic guide using prototyping technology (In2Guide) with metal sleeves positioned over implant sites. The metal sleeves are compatible with computer guided surgical kit provided by implant manufacture (Dentium, South Korea) which contain the drills of increasing diameter with wide metal shanks and stoppers that fit accurately in the sleeves to provide total guided implant placement (Figure 2).

Preoperative medications include prophylactic antibiotic ([Augmentin® 1 gm (amoxicillin 875 mg + clavulanic acid 125 mg)] was given the day before surgery (every 12 hours), then continued seven days postoperatively twice-daily. Chlorhexidine digluconate 0.2 % mouth rinse started one day before surgery and continued for 7 days postoperatively. An interocclusal rubber base centric record was made over the surgical guide to be used for guide fixation. The flapless surgical protocol was followed. The surgical guide was fixed to the edentulous maxilla using the drills and anchor pins. A tissue punch was used to make a circular incision over each implant site, then the circle of the soft tissue was removed. Implant osteotomy preparation was performed by sequential drilling of the computer guided surgical kit (Figures 3). In case of reduced bone density, the final drill was omitted (under preparation for implant osteotomy) to obtain at least 35 Ncm insertion torque at implant placement to give adequate primary stability required for immediate loading[23].

Figure 1: Planning of implant position using the On Demand software.
Six implants (SuperlineII, Dentium, Korea) were inserted using the implant mount through the guide (fully guided implant placement) with at least 35 Ncm insertion torque. Post operative panoramic radiograph was performed to verify implant placement (Figure 4).

Straight abutments of adequate gingival height were screwed to the implant fixtures. Rubber base impressions were made to abutments and poured with extrahard stone. A centric rubber base interocclusal record was made. On the resultant cast, a full arch acrylic provisional fixed restoration was performed using the conventional method and cemented to the abutments with soft cement (Zinc oxide eugenol) on the same day of implant placement (about 3 hours after implant placement). The occlusion was adjusted to provide even and homogenous occlusal contacts. Anti-inflammatory medications (Alphintern) and analgesics (Ketolac 10 mg) were prescribed 3 times daily for 7 days post surgically in addition to antibiotics and mouth wash. Post-operative instruction included application of ice packs, avoid hard food and maintain soft diet. Patients were followed up at least once per week for the first 3 weeks, then follow up visits were scheduled every 3 months. Six months later, Open tray impression was made on the implant level, and fixed porcelain fused to metal screw retained restoration was constructed. Tibase abutments (Dentium, south Korea) were used for screw retained prosthesis. The plastic pattern of the bridge was designed using CAD/CAM over the Tibase abutments, then printed using prototyping. The resin bridge was tried in patient mouth for passive fit, then casted with cobalt chromium alloy framework. The framework was pained with opaquer and porcelain powder, then the powder was fired and glazed. The prosthesis included 12 artificial teeth (from first molar area on one side to first molar area on the other side) (Figure 5). The occlusion of the final prosthesis was canine guided occlusion.
Said Said bone height at baseline. Measurements were made on both the mesial and distal aspects of each implant and the mean was subjected to statistical analysis.

Clinical and radiographic parameters were measured at immediate loading of the implants with fixed acrylic prosthesis (baseline), 6 months later (before removal of the acrylic prosthesis), then after 12 months (6 months after insertion of the metal ceramic prosthesis).

Statistical analysis:
Data was analyzed with SPSS program version 25 Statistical Packages for Social Science (SPSS Inc., Chicago, IL, USA). The normal distribution of data was verified by Shapiro Wilk test. Kaplan Meier analysis was used to calculate implant survival and the difference in implant survival between groups was calculated with Log rank test. Friedman test was used to compare plaque and gingival scores between different observation times, and Wilcoxon signed ranks test was used to compare between each two times. For between-group comparisons, the non-parametric Mann-Whitney test was used. Repeated measures ANOVA was used to compare pocket depth, implant stability, width of keratinized mucosa, and crestal bone loss between observation times and groups followed by Bonferroni test for multiple comparisons. The threshold for statistical significance was set at \( P < 0.05 \).

RESULTS
Of 60 implants placed (30 implant in each group), 2 implants (in 2 patients) failed in non-smoker group, and 6 implants (4 in one patient and 2 in another patient) in a smoker group. All implant failures occurred in the first six months after loading was professional acrylic prosthesis resulting in implant survival rate of 93.3 % and 80 % for non-smoker and smoker groups respectively. No implant failures happened after one year. The failed implants in non-smoker group occurred as a result of implant overloading with the absence of inflammation or pus formation and presence of implant mobility. The failed implants in smoker group were associated with peri-implantitis, inflammation, pus formation, mobility.
and crestal bone loss. In non-smoker group, the failed implant was removed and the final prosthesis was supported by 5 implants in the 2 patients. In the smoker group, the patient with 4 failed implants was excluded from the study and the final prosthesis was supported by 4 implants in the other patient. Kaplan Meier analysis of the survival rates of both groups is presented in (Figure 6). There was a significant difference in implant survival rate between groups (Log rank test, \( p = 0.048 \)).

Comparison of plaque and gingival scores between smokers and non-smokers at different time intervals is presented in (Table 1). For both groups there was a significant difference in plaque and gingival scores between observation times. Multiple comparison of plaque and gingival scores between observation times is presented in the same table. Plaque and gingival scores increased significantly from baseline to six months in both groups, then decreased significantly from six months to 12 months. At baseline, no significant difference in plaque and gingival scores was noted between groups. At 6 and 12 months, smoker group recorded significant higher plaque and gingival scores than non-smoker groups.

![Figure 6: Kaplan Meier analysis of the implant survival rate of both smokers and non-smoker groups.](image)

**Table 1:** Comparison of plaque and gingival scores between smokers and non-smokers at different time intervals:

<table>
<thead>
<tr>
<th></th>
<th>Baseline (at loading) M (min-max)</th>
<th>6 months M (min-max)</th>
<th>12 months M (min-max)</th>
<th>Freidman Test (p value)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Plaque scores</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-smokers</td>
<td>0.00a (0.00 - 0.00)</td>
<td>1.50b (1.00 - 2.00)</td>
<td>1.0 c (0.50 - 1.50)</td>
<td>0.004*</td>
</tr>
<tr>
<td>Smokers</td>
<td>0.00a (0.00 - 0.00)</td>
<td>2.00b (1.00 - 3.00)</td>
<td>1.50c (1.00 - 3.00)</td>
<td>0.002*</td>
</tr>
<tr>
<td>Mann-Whitney test (p value)</td>
<td>1.00</td>
<td>0.007*</td>
<td>0.003*</td>
<td></td>
</tr>
</tbody>
</table>

|                  |                                  |                      |                       |                        |
| **Gingival scores** |                                  |                      |                       |                        |
| Non-smokers      | 0.00a (0.00 - 0.00)a             | 1.00b (0.50 - 1.50)  | 0.50 c (0.50 - 1.00)  | 0.021*                 |
| Smokers          | 0.00a (0.00 - 0.00)a             | 3.00b (2.00 - 3.00)  | 2.00c (1.00 - 3.00)   | < 0.001*               |
| Mann-Whitney test (p value) | 1.00 | < 0.001* | < 0.001* |

M; median, min; minimum, max, maximum, \(*p\) is significant at 0.05. Different letters show significant difference between each 2 time intervals (Wilcon signed ranks test, \(p < 0.05\)), while similar letters show no difference.
Comparison of pocket depth, stability of implants, width of keratinized mucosa, and crestal bone loss between smokers and non-smokers at different time intervals is shown in (Table 2). Multiple comparisons between each two observation times are presented in the same table. Pocket depth significantly increased with advance of time in both groups. At baseline there was no significant difference in pocket depth between groups. At 6 and 12 months, smoker group recorded significant higher pocket depth than non-smoker group. There was a significant difference in implant stability between observation times in both groups. Implant stability significantly decreased from baseline to 6 months then significantly increased again at 12 months for both groups. At baseline, and 12 months, there was no significant difference in implant stability between groups. At 6 months, non-smoker group presented significant higher implant stability non-smoker group. The width of keratinized mucosa significantly decreased with time in both groups. However, no significant difference in the width of keratinized mucosa between groups at different time intervals. Crestal bone loss significantly increased from 6 months to 12 months in both groups. Smoker group presented significant higher crestal bone loss than non-smoker group at 6 and 12 months.

Table 2: Comparison of pocket depth, stability of implants, width of keratinized mucosa, and crestal bone loss between smokers and non-smokers at different time intervals:

<table>
<thead>
<tr>
<th></th>
<th>Baseline (at loading) mean ± SD</th>
<th>6 months mean ± SD</th>
<th>12 months mean ± SD</th>
<th>Repeated ANOVA (p value)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pocket depth</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-smokers</td>
<td>1.45 ± 0.28a</td>
<td>1.70 ± 0.25b</td>
<td>2.0 ± 0.51c</td>
<td>0.003*</td>
</tr>
<tr>
<td>Smokers</td>
<td>1.57 ± 0.25a</td>
<td>2.1 ± 0.48b</td>
<td>2.46 ± 0.68c</td>
<td>0.005*</td>
</tr>
<tr>
<td>t-test (p value)</td>
<td>1.00</td>
<td>0.031*</td>
<td>0.011*</td>
<td></td>
</tr>
<tr>
<td><strong>Stability of implants</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-smokers</td>
<td>62 ± 2.2a</td>
<td>59 ± 3.3b</td>
<td>61 ± 2.7a</td>
<td>0.011*</td>
</tr>
<tr>
<td>Smokers</td>
<td>61 ± 2.5a</td>
<td>56 ± 3.5b</td>
<td>60 ± 2.9a</td>
<td>0.017*</td>
</tr>
<tr>
<td>t-test (p value)</td>
<td>0.54</td>
<td>0.008*</td>
<td>0.89</td>
<td></td>
</tr>
<tr>
<td><strong>Keratinized mucosa</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-smokers</td>
<td>2.1 ± 0.50a</td>
<td>1.8 ± 0.41b</td>
<td>1.50 ± 0.32c</td>
<td>0.002*</td>
</tr>
<tr>
<td>Smokers</td>
<td>2.0 ± 0.48a</td>
<td>1.7 ± 0.39b</td>
<td>1.4 ± 0.31c</td>
<td>0.004*</td>
</tr>
<tr>
<td>t-test (p value)</td>
<td>0.35</td>
<td>0.54</td>
<td>0.65</td>
<td></td>
</tr>
<tr>
<td><strong>Crestal bone loss</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-smokers</td>
<td>-</td>
<td>0.6 ± 0.20a</td>
<td>1.0 ± 0.33b</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td>Smokers</td>
<td>-</td>
<td>0.9 ± 0.31a</td>
<td>1.4 ± 0.38b</td>
<td>0.014</td>
</tr>
<tr>
<td>t-test (p value)</td>
<td>-</td>
<td>0.003*</td>
<td>0.001*</td>
<td></td>
</tr>
</tbody>
</table>

*p is significant at 0.05. Different letters show significant difference between each 2 time intervals (Bonferroni, p < 0.05), while similar letters show no difference.

**DISCUSSION**

The implant survival rate was 93.3 % and 80 % for non-smoker and smoker groups with significant difference between groups. A similar reduced survival rate in a smoker group (84.2 %) was observed in another retrospective study[21] for implants placed using a non-submerged surgical protocol where 63.6 % of implants placed in the maxilla . Similarly, Sanna et al.[8] reported 81.2 % survival rate for smokers who received a flapless procedure and immediately loaded implants with prefabricated fixed complete dentures. The reduced implant survival rate in the smoker group concurred with the results of another systematic review[19] which is also reported a significant difference in implant failure rate between smokers and non-smokers in favor of non-smokers. The increased failure rate of the implants in smoker group may be due to the use of nicotine which cause a decrease in local blood flow resulting from vasoconstriction, which causes an increase in the inflammatory cells, and may delay or inhibit bone healing after surgery[32, 33]. The implant survival rate in non-smoker group (93.3) was similar to the survival rate reported in other studies[8, 21] for implants inserted with flapless approach and immediate loaded. In both groups, the immediate loading protocol could contribute to implant failures as it may induce implant micromotion and interfere with bone to implant contact in the critical healing period especially in bone with reduced density such as maxillary bone[34].
Plaque and gingival scores increased significantly from baseline to six months in both groups, then decreased significantly from six months to 12 months. The increased plaque and gingival scores after six months could be attributed to cement-retained fixed acrylic prosthesis which is not perfectly adapted to the implant’s mucosa, and the excess cement may skip to peri-implant sulcus unnoticed and the patients had a difficulty in performing adequate cleaning. This may cause peri-implant plaque accumulation and mucosal inflammation. When the cemented retained acrylic prosthesis was replaced with the screw retained prosthesis, oral hygiene becomes easy to performed, and plaque accumulation and gingival inflammation decreased again after 12 months. In line with this explanation, Dini et al. found that gingival scores and bleeding on probing was higher in cemented retained prosthesis than in screw-retained ones after one year\cite{14}. Similarly, Weber et al.\cite{14} found that cement-retained crowns seemed to worsen plaque and gingival scores compared to screw-retained crowns. The increased plaque and gingival scores in smoker group compared to than non-smoker group may be due to smoking enhance greater bacterial biofilm adhesion which may contain several types of microbata such as Fusobacterium, Tannerella and Mogibacterium leading to more plaque accumulation and gingival inflammation\cite{14, 29}. In line with our finding, Alghamidi et al.\cite{39} reported a significant increased in plaque scores in smoker patients receiving immediately loaded narrow diameter implants compared to nonsmokers. However, the authors reported no significant difference in gingival index between smokers and non-smokers. Conversely, Alamiri et al.\cite{39} reported that gingival index was significantly higher in non-smokers compared with smokers among patients with immediate loading.

Pocket depth significantly increased with advance of time in both groups. This may be due to the increased crestal bone loss with time in both groups together with increased peri-implant mucosal enlargement. The enlargement of thick peri-implant maxillary mucosa may have occurred due to gingival inflammation as a result of plaque accumulation under the prosthesis. The increased probing depth in smoker group was in line with the results of another study\cite{40} which reported that pocket depth increased significantly for immediate loaded implants than non-smoker group. Similarly, Sun et al.\cite{40} reported that probing depth was significantly higher after 6 and 12 months of implant loading in the smoker group compared to non-smokers. Conversely, Romanos et al.\cite{14} reported no difference in probing depth between smokers and non-smokers for immediately loaded platform-switched implants.

Primary stability, is considered an important factor for the success of immediate loading of implants\cite{29}. Implant stability significantly decreased from baseline to 6 months then significantly increased again at 12 months for both groups. The decrease in implant stability after 6 months could be attributed to the decrease in the bone to implant contact that occur during the initial healing period as a result of bone remodeling\cite{41}. The increase in implant stability again after 12 months may be due to the increased bone to implant contact occurred thereafter with increased bone density around implants and increased anchorage of the implants in the bone. The decrease in implant stability after 6 months and the increased implant stability again after 12 months was concurred with the results of another studies for immediate loading\cite{42} and delayed loading\cite{40} of the implants. There is controversy about the effect of immediate loading used in smoker patients on primary stability of the implants\cite{43}. After 6 months, smoker group recorded significant higher implant stability than non-smoker group. This may be due to smoking was found to reduce the percentage of bone to implant contact in the early phase of healing compared to non-smokers\cite{44}. A similar observation was reported in another study for implants placed in the posterior mandible after 3 months of implant placement\cite{40}.

The width of keratinized mucosa significantly decreased with time in both groups. This may be due attributed to the increased crestal bone loss, and probing depth with advance of time. Moreover gingival recession may occur after implant loading which contribute to decreased width of keratinized mucosa\cite{45}. It has been reported that increased plaque accumulation and tissue inflammation may result in marginal gingival recession and the decreased width of keratinized mucosa\cite{29}. However, no significant difference in width of keratinized mucosa was observed between smokers and non-smokers.

The mean crestal bone resorption after one year was $1.0 \pm 0.33$ mm and $1.4 \pm 0.38$ for non-smokers and smokers respectively. The value for nonsmokers is located within the normal limit of crestal bone loss reported in the literature that occurred during the first year ($1.2$ mm)\cite{29, 44}. However, for smoker, the crestal bone loss exceeds this value after one year. These values are similar to values obtained by Sanna et al.\cite{39} for immediately loaded fixed complete dentures using flapless implant placement for both maxillary and mandibular edentulous arches after one year. Crestal bone loss significantly increased from 6 months to 12 months for both groups. This unavoidable time dependent bone loss could be attributed to bone response to healing process and loading. Similarly, Sun et al.\cite{40} reported that marginal bone loss increased significantly at 12 months compared to 6 months after loading when implants are inserted in smoker male patients in posterior mandible.

Smoker patients showed significantly higher crestal bone loss than non-smoker patients. A similar observation was also reported in several studies in the literature\cite{8, 15, 44, 46, 47}. Sanna et al.\cite{39} reported significant higher crestal bone loss in smokers than non-smokers when implants were inserted in edentulous arches using computer guided flapless approach and immediately loaded with fixed
prosthesis. Similarly, Velasco-Ortega et al. reported more crestal bone loss in smoker patients with immediate loaded implants placed by guided surgery in edentulous mandible than non-smokers. In another systematic review, marginal bone loss in smoker patients was found to be significantly higher than non-smoker patients and higher in maxilla than mandible. In another study, the authors reported that smoking affect crestal bone loss more than the type of surgery (lap versus flapless). In contrast other studies reported no significant difference in crestal bone loss between smokers and non-smokers. Cassetta et al. reported that marginal bone loss was not affect by smoking status when immediate loaded implants were inserted using the mucosa supported stereolithographic guides and flapless approach in maxillary and mandibular arches. Similarly, Daher et al. found that marginal bone loss was not significantly different between smokers and non-smokers for immediate loaded implants in posterior maxilla.

The increased crestal bone loss in smoker group could be attributed to the smoking ingredients, such as the nicotine, which may delay or inhibit bone healing after surgery. Smoking may alter fibroblast function, reduce collagen synthesis, impair immune function by interfering with the functions of neutrophils, and lymphocytes. Moreover, smoking decrease blood flow due to vasoconstriction, which result in increasing inflammatory cells. The maxilla is more is more affected by nicotine than the mandible due large medullary bone and more vasculature. Consequently, maxilla is more permeable to the harmful ingredient of smoking. Moreover, the bacterial biofilm adhere faster to the mucosa of the smokers as stated previously which may increase mucositis and peri-implantitis and consequently lead to crestal bone loss. It has been reported that tobacco smoking can reduce bone quality, delay healing and increase bone loss specially in maxilla.

CONCLUSION

Within the limits of this study, smoker patients were associated with worsen clinical and radiographic outcomes of immediate loaded implants placed by flapless computer guided surgery to support full arch fixed restoration in edentulous maxilla compared to non-smoker patients.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

REFERENCES


30. Canullo L, Fedele GR, Iannello G, Jepsen S. Platform switching and marginal bone-level


49. Scolaro JA, Schenker ML, Yannascoli S, Baldwin K, Mehta S, Ahn J. Cigarette smoking increases


