INTRODUCTION

According to the original Branemark protocol, implant placement should be posted several months after tooth extraction until complete healing of alveolar bone occurred[1]. However, alveolar bone resorption after extraction especially in maxilla usually reduces buccolingual residual bone volume, creates buccolingual discrepancies between the implant and the prosthesis[2]. Immediate implant placement in extraction sockets was first proposed by Schulte and Heimke[3] and it has several advantages such as preservation of alveolar bone from resorption, prevention of soft tissue shrinkage, provision for emergence profile, reduction of treatment time, reduction of surgical appointments (as extraction, implant placement and bone grafting procedures are performed at the same visit) and improvement of the patient’s psychology and comfort[4, 5]. Furthermore, immediate implant placement in extraction sockets maintain height and width of the bone and maintain the implants in the same position and inclination of pre-existed teeth[6].

The compromised tooth needed to be extracted is usually associated with infection which comes from periapical pathology, failure of endodontic treatment, and the periodontal disease. This infection conventionally contraindicates implant placement after extraction as it may compromise healing, affect osseointegration and cause implant failure[7, 8] as a result of bacterial contamination of the implant surface during the initial stages of healing[9]. Controversy exists in the literature regarding the success rate of immediate implant insertion into infected extraction sites. Some authors found satisfactory results[10 - 12], while others showed failures of implants when installed into infected extraction sites compared to noninfected sockets[13 - 15]. Alsaadi et al.[16] reported higher failure rate if implants are placed in sockets with periapical lesions. However, several studies[14, 17 - 21] showed similar
survival rate for implants placed immediately in infected or non-infected sockets after controlling the infection by prophylactic antibiotics and adequate debridement and curettage of the alveolar bone. It has been reported that the presence of periapical infection did not preclude immediate implant placement provided that sockets are properly cleaned and decontaminated[2, 14].

When placing the implants immediately after extraction, a gap will be formed between the fixture body and the socket wall which needs to be filled with biocompatible bone material to enhance three-dimensional osteointegration and bone formation[5]. Several augmentation materials can be utilized such as autogenous bone graft, allografts, hydroxyapatite, xenografts, and growth factors[5]. It has been reported that all these materials provided similar clinical outcomes in bone regeneration[22, 23].

According to systematic reviews[8, 24], there is scarce information about the effect of different confounding variables that may influence the survival of immediate implants inserted in infected socket such as the type of infection (periodontal or apical), loading type (immediate or delayed), the intactness of the ridge (intact or fenestrated) and the type of bone regeneration material. Zuffetti et al.[10] reported that placement of implants into infected sockets immediately after teeth extraction had similar success rate if the implants are delayed loaded or immediately loaded. However, the effect of the intactness of the ridge (intact or fenestrated) especially in the posterior maxillary region on survival rate of the implants immediately placed in extraction socket still scarce in the literature. Consequently, this study aimed to evaluate clinical and radiographic outcomes of immediate implant insertion in infected extraction sites of maxillary posterior teeth with buccal fenestrations combined with guided bone regeneration.

MATERIALS AND METHODS

Patient enrollment and study design:

Eight patients (4 males and 4 females, mean age 42 ± 3.5 years) were selected from the outpatient clinic attending the oral and maxillofacial department who need to replace their failed in dentition with dental implants. The inclusion criteria are:

1) Patients with non-restorable maxillary posterior teeth (premolars or molar teeth) that needed to be extracted due to failure of endodontic treatment or badly destructed teeth,

2) All teeth presented with periapical lesions due to infection which is diagnosed by peri-apical radiolucency preoperative cone beam computerized tomography (CBCT, (Figure 1)),

3) At least one tooth in each quadrant presented with buccal bone defect (apical fenestration),

4) Patient aged more than 20 years,

5) Presence of adequate quantity of native bone to achieve primary stability. The exclusion criteria include; active signs of periapical or periodontic infections, total absence of buccal or lingual bone walls, patients with history of periodontitis, smoker patients, systemic disease that may interfere with implant placement, chemotherapy or radiotherapy to head and neck region, bone metabolic disorders such as diabetes mellitus, history of clenching or bruxism, and pregnancy. The patients were informed about the protocol and objectives of the study before obtaining informed consents. The study protocol was approved by the local ethical committee of the faculty of Dentistry.

Figure 1: Evaluation of remaining non restorable teeth:
a) intra-oral view and b) radiographic evaluation using CBCT (cross-sectional images).

Surgical and prosthetic protocol:

Preoperative cone beam CBCT was used to detect amount of bone available for initial stability of the implants, detect the relation to the vital structures (maxillary sinuses, nasal cavity) and roots of maxillary canine. Moreover, the CBCT was used to detect the proper implant dimensions (width and length) that will be placed after extraction in the intact bone. Preoperative prophylactic medications were given to all participant to control the infection. These medications include; chlorhexidine digluconate (0.2 %) mouthwash, and antibiotics (amoxicillin 625 mg + clavulanic acid 125 mg, Augmentin® 1 gm) given 24 hours prior to surgery. At surgical appointment, the patient was asked to rinse his mouth with chlorhexidine digluconate (0.2 %) immediately before surgery. Surgery was performed using local infiltration anesthesia (Articaine HCL 4 %, ArtPharmaDent, 1 : 200,000 epinephrine). A mucoperiosteal flap was
elevated due to existence of bone defects\cite{5}. A traumatic extraction was performed to the remaining roots using periotomes and elevators. Separation of the roots of molar teeth was performed using thin surgical bur. The extraction socket was thoroughly debrided using appropriate curette and then the sockets were irrigated with saline. The integrity and continuity of the socket walls was evaluated. Bone perforation to the periapical lesions for full debridement was performed if needed. Implant osteotomy was prepared using sequential drilling to 3 mm beyond the apex of the root (in premolar region). Osteotomy in the molar region was performed in the interradicular bone to gain sufficient primary implant stability. Osseointegrated sandblasted acid etched implants (Superline, Dentium, South Korea, 4 mm - 5.5 mm in diameter) was stabilized into the osteotomy site with a minimum insertion torque of 25 Ncm. If bone density is reduced, underpreparing of implant osteotomy was performed by omitting the last drill to gain adequate primary stability. All implants were leveled 2 to 3 mm apical to the gingival margin of the adjacent natural teeth. (Figures 2 and 3).

The gap between the implants and the bone walls of the sockets and bone defects (fenestrations) were filled with scaffold and granular bone grafting material which consisted of mixture of Xenograft (Intergraft, Neobiotech, particle size 0.2 - 1.0 mm, South Korea) and alloplastic material (Osteon II, 30 % hydrox apatite and 70 % β-tricalciumphosphate, Pore Size : 250 μm, Porosity : 70 %). Undermining of the flap was performed to avoid tension of the flap during suturing. The artificial bone was covered by Biodegradable collagen membrane barrier (Dentium, South Korea) and the flap was sutured tension free (using Vicryl 40- resorbable suture). Postoperative medications include chlorhexidine digluconate (0.2 %) mouthwash, and antibiotics (amoxicillin 625 mg + clavulanic acid 125 mg, Augmentin® 1 gm) given twice daily for 7 days. Moreover, analgesics (Ketolac® 10 mg), and non-steroidal anti-inflammatory (Alphintern) drugs were prescribed one day before surgery and continued after surgery for 5 days. The patients were instructed to apply ice bags after surgery to decrease postoperative edema. (Figures 4, 5 and 6).
All the patients were followed on a weekly basis for the initial 4 weeks. After 2 weeks, acrylic partial dentures with wrought wire claps were constructed to be used as professional restoration. The second surgery was made 6 months later. Cover screws were unthreaded and healing abutments were threaded to implants for 2 weeks to allow proper gingival healing. Impression posts were threaded to the implants and the impression was performed using open tray impression technique. Implant analogues were connected to the impression posts and the cast was poured. Prefabricated titanium abutments of adequate gingival height were used. Fixed metal ceramic prosthesis was constructed and tried in patient mouth for passivity. Interocclusal record was performed to register jaw relation and the bridge was cemented to the implants using glass-ionomer cement. Three-months based follow up visits were scheduled for all patients after cementation of the prosthesis. The second surgery was with wrought wire claps were constructed to be used as professional restoration. The second surgery was made 6 months later. Cover screws were unthreaded and healing abutments were threaded to implants for 2 weeks to allow proper gingival healing. Impression posts were threaded to the implants and the impression was performed using open tray impression technique. Implant analogues were connected to the impression posts and the cast was poured. Prefabricated titanium abutments of adequate gingival height were used. Fixed metal ceramic prosthesis was constructed and tried in patient mouth for passivity. Interocclusal record was performed to register jaw relation and the bridge was cemented to the implants using glass-ionomer cement. Three-months based follow up visits were scheduled for all patients after cementation of the final prosthesis to collect the data.

Measurements of clinical and radiographic outcomes:

The implant success criteria of Albrektsson et al.\[25\] were utilized which include; no detectable implant mobility, no radiographic peri-implant radiolucency, no infection, and bone loss less than 2 mm. The implant was considered survived if it still functioning and fulfill the success criteria. Plaque index was measured according to Mombelli et al.\[26\]: score 0 = no plaque, score 1 = plaque detected by a probe, score 2 = plaque seen by naked eye, score 3 = a lot of soft matter. Gingival index was measured using Loe and Silness\[27\] scores: score 0 = no bleeding, score 1 = pinpoint bleeding, score 2 = linear bleeding, score 3 = profuse bleeding. Pocket depth was measured by plastic periodontal probe which inserted in the peri-implant sulcus to measure the distance between gingival margin and the most apical probing depth. Plaque index, Gingival Index, pocket depth were measured at mesial, distal, buccal and lingual surface of each implant. The width of keratinized mucosa around each implant was calculated in mm using a periodontal probe from the free gingival margin to the muco-gingival junction\[28\].

Crestal bone loss was measured at mesial, distal, buccal, and lingual surface of each implant using CBCT (i-CAT Vision, Hatfield, PA, USA). In the panoramic window of the CBCT software (OnDemand3DApp), marginal bone height was measured at mesial and distal surface of each implant. In the cross-sectional window of the CBCT software, marginal bone height was measured at buccal and palatal surface of each implant. To estimate marginal bone level, the distance from implant abutment junction to the bone contact with implant was measured using the ruler measure tool of the software to give bone level\[29\]. Crestal bone loss was calculated by subtraction of alveolar bone heights at follow-up visits from bone level at base line (prosthesis delivery). The bone loss measurement for mesial, distal, buccal and lingual surfaces were summed for all implants and the average was used in the analysis. All clinical and radiographic outcomes were collected after insertion of the prosthesis (base-line), 6 months, 12 months and 24 months later.

Statistical analysis:

The normal distribution of data was verified by Shapiro Wilk test. Life table analysis was used to calculate cumulative implant survival. Friedman test was used to compare non-parametric data (plaque and gingival indices) between different time intervals, then Wilcoxon signed ranks test was used for multiple-comparisons between time intervals. Repeated measures analysis of variance was utilized to compare parametric data (probing depth, mucosal width, and crestal bone loss between time intervals followed by Bonferroni post hoc test. Data was analyzed with SPSS program version 25 (SPSS Inc., Chicago, IL, USA). P < .05 was considered significant.

RESULTS

A total of 21 implants were placed in 8 patients. Five patients had 15 implants (71.4 %) inserted in premolar and molar regions (3 for each patient) and 3 patients had 6 implants (28.6 %) inserted in premolar regions only (2 for each patient). Nine implants (42.8 %) were installed in molar regions and a total of 12 (57.1 %) implants were inserted in premolar region. At second stage surgery, 2 implants failed to integrate (one implant in the location of second molar area and another in the location first premolar area) resulting in 97 % survival rate. After 6 months of loading, additional implant failed in the area of first molar resulting in 95 % survival rate at the end of this interval. No additional implant failure occurred thereafter, therefore; the cumulative survival rate was 95 % after 24 months. The failed implants were associated with suppuration, bone loss and mobility. The failed implants were removed and the prosthesis was cemented to the remaining implants. The life table analysis showing the survival rate of the implants.
at each time interval and the cumulative survival rate is presented in (Table 1). There was a significant difference in plaque scores between different time intervals \((p = 0.034)\). Plaque scores significantly increased from prosthesis insertion to 6 months then significantly increased from six months to 12 months. However, no difference in plaque between 12 months and 24 months was observed. Gingival index did not differ between different observation times \((p = 0.027)\). Gingival scores increased significantly from baseline to 6 months, then decreased significantly at 12 months. However, no significant difference in gingival scores between 12 months and 24 months was detected. There was a significant difference in pocket depth between different time intervals \((p = 0.009)\). Pocket depth increased significantly from baseline to 6 months. No significant difference in pocket depth was observed between 6 months and 12 months. However, pocket depth significantly decreased from 12 months to 24 months. Mucosal width did not differ between time intervals \((p = 0.63)\). Crestal bone loss differed significantly between observation times. Crestal bone loss significantly increased with passage of time \((p = 0.001)\). Crestal bone loss significantly increased from 6 months to 12 months, then significantly increased from 12 months to 24 months.

**Table 1:** Life table analysis showing survival rate of the implants at each time interval and the cumulative survival rate:

<table>
<thead>
<tr>
<th>Beginning of interval</th>
<th>No. of implants get in interval</th>
<th>No. of implants withdrawn</th>
<th>No. of risky implants</th>
<th>% Terminated</th>
<th>% Survived</th>
<th>Cumulative % Survived at End of Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>84</td>
<td>19</td>
<td>74.500</td>
<td>2</td>
<td>0.03</td>
<td>0.97</td>
</tr>
<tr>
<td>6</td>
<td>63</td>
<td>20</td>
<td>53.000</td>
<td>1</td>
<td>0.02</td>
<td>0.98</td>
</tr>
<tr>
<td>12</td>
<td>42</td>
<td>21</td>
<td>31.500</td>
<td>0</td>
<td>0.00</td>
<td>1.00</td>
</tr>
<tr>
<td>18</td>
<td>21</td>
<td>0</td>
<td>21.000</td>
<td>0</td>
<td>0.00</td>
<td>1.00</td>
</tr>
<tr>
<td>24</td>
<td>21</td>
<td>21</td>
<td>10.500</td>
<td>0</td>
<td>0.00</td>
<td>1.00</td>
</tr>
</tbody>
</table>

Number and distribution of insertion torque of the inserted implants at time of implants placement is presented in (Table 2). Six implants (28.5 %) had insertion torque from 20 - 30 Ncm, eleven implants (52.3 %) had insertion torque from 30 - 40 Ncm, and four implants (19.1 %) had insertion torque more than 40 Ncm.

**Table 2:** Number and distribution of insertion torque of the inserted implants at time of implants placement:

<table>
<thead>
<tr>
<th>Implant number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insertion torque from 20 Ncm to 30 Ncm</td>
<td>6</td>
</tr>
<tr>
<td>Insertion torque from 30 Ncm to 40 Ncm</td>
<td>11</td>
</tr>
<tr>
<td>Insertion torque &gt; 40 Ncm</td>
<td>4</td>
</tr>
</tbody>
</table>

Clinical (plaque scores, gingival scores, probing depth, and width of keratinized mucosa) and radiographic (crestal bone loss) outcomes at different time intervals is presentation to 6 months then significantly increased from six months to 12 months. However, no difference in plaque between 12 months and 24 months was observed. Gingival index did not differ between different observation times \((p = 0.027)\). Gingival scores increased significantly from baseline to 6 months, then decreased significantly at 12 months. However, no significant difference in gingival scores between 12 months and 24 months was detected. There was a significant difference in pocket depth between different time intervals \((p = 0.009)\). Pocket depth increased significantly from baseline to 6 months. No significant difference in pocket depth was observed between 6 months and 12 months. However, pocket depth significantly decreased from 12 months to 24 months. Mucosal width did not differ between time intervals \((p = 0.63)\). Crestal bone loss differed significantly between observation times. Crestal bone loss significantly increased with passage of time \((p = 0.001)\). Crestal bone loss significantly increased from 6 months to 12 months, then significantly increased from 12 months to 24 months.

**Table 3:** Assessment of different clinical and radiographic parameters at different time intervals:

<table>
<thead>
<tr>
<th></th>
<th>Base line</th>
<th>6 months</th>
<th>12 months</th>
<th>24 months</th>
<th>Friedman ( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plaque scores Med (Mini-Maxi)</td>
<td>0(0-1)a</td>
<td>1(1-2)b</td>
<td>1.5(1-2)c</td>
<td>1.5(1-2)c</td>
<td>0.034*</td>
</tr>
<tr>
<td>Gingival scores Med (Mini-Maxi)</td>
<td>0(0-1)a</td>
<td>1.5(1-2)b</td>
<td>1(1-2)c</td>
<td>1(1-2)c</td>
<td>0.027*</td>
</tr>
<tr>
<td>Pocket depth X±SD</td>
<td>1.5 ± 3.4a</td>
<td>2 ± 0.54b</td>
<td>2.1 ± 0.52b</td>
<td>1.5 ± 0.44c</td>
<td>0.009*</td>
</tr>
<tr>
<td>Keratinized mucosa X±SD</td>
<td>2.8 ± 0.75a</td>
<td>3.0 ± 0.64a</td>
<td>2.9 ± 0.67a</td>
<td>2.9 ± 0.68a</td>
<td>0.63</td>
</tr>
<tr>
<td>Crestal bone loss X±SD</td>
<td>-</td>
<td>0.87 ± 0.34a</td>
<td>1.3 ± 0.36b</td>
<td>1.5 ± 0.41c</td>
<td>0.001*</td>
</tr>
</tbody>
</table>

Med; median; Mini; minimum; Maxi, maximum; x; mean; SD; standard deviation, * p value is significant at 5 %. Dis-similar letters showed significant difference between each time intervals \((p < 0.05)\) for Wilcoxon and Bonferroni tests, while the same letters showed no difference.
DISCUSSION

Cumulative survival rate was 95 % after 24 months. Similarly, Zuffetti et al reported 97.9 % survival rate of the implants installed in infected sockets. They also reported no difference in survival rate of the implants installed in infected and normal extraction sites. Also, Bell et al. in a retrospective study found 97.5 % success rate of implants placed into sockets with chronic periapical pathology. The increased the survival rate of the implants (95 %) could be attributed to the preoperative treatment protocol which involves decontamination of socket by the use of antibiotic cover, adequate cleaning and decapitation, alveolar debridement, the use of bone grafting procedures which create adequate condition for bone regeneration and osteointegration. Moreover, the granulation tissue at the apex of the infected tooth is considered an inflammatory response to bacteria that protect the one from further infection. Consequently, adequate removal of the granulation tissue with proper curettage and debridement, helped in eradication of microorganisms, reduces inflammation and bone resorption. Furthermore, the use of antibiotic cover before surgery concluded the infection and improve implant survival as Dent et al. reported reduced implant failure when antibiotics were prescribed before surgery.

In addition, the two-stage surgical protocol and the delayed loading utilized in this study contribute significantly to the increased implant survival rate as it allowed undistributed healing of the implants, preventing contamination of the implants with oral microbiota, and prevent implant overload and increased implant micromotions caused by immediate loading which may interfere with the healing process. Two of the 3 failed implants are located in molar regions. The implant failure in this area could be attributed to the inadequate amount of bone required for increased initial implant stability. The inadequate bone may result from large in size of the roots or refused roots of molar teeth which may result in reduced interradicular bone. Two of the failed implants occurred after 6 months of implant placement and were associated with reduced insertion torque (< 30 Ncm) after implant placement. This reduced insertion torque may compromise osseointegration. A similar observation was reported in another study for implants placed in extraction sockets presented with periapical infection. Additional fixture failed 6 months after loading with fixed prosthesis. The implant failure may be due to increased implant overloading and reduced bone to implant contact.

In this study, implant stability was not measured during follow-up visits as it was not possible to remove the cement retained fixed prosthesis to attach the smart peg of the Ostell device (Resonance frequency analysis) to the threads of the implant. Moreover, if the periotest device was used for measuring the implant stability without removal of the fixed bridge, it would give inaccurate results as the bridge splint the implants together. Therefore, it was decided to use the insertion torque of the implant to represent implant stability. Most of the inserted implants had adequate insertion torque (> 30 Ncm). This may be due to the increased diameter of the implants used in this investigation (4 mm to 5.5 mm). It has been reported that wide implant diameter are associated with increased removal torque and decreased load on peri-implant bone. Moreover, the implant diameter was selected to be large than the diameter of peri-implant bone. This would increase implant stability. To further enhance primary implant stability, the depth of implant osteotomy was extended 3 mm in the bone apical to the periapical lesion. Furthermore, the omitting of the last drill during implant site preparation (under-preparation of implant osteotomy) could contribute to increased primary stability and insertion torque of the implants. Most of the inserted implants (52.3 %) had insertion torque from 30 - 40 Ncm. Similarly, Zuffetti et al. found that the majority of the inserted implants in infected sockets (49.7 %) had insertion torque ranging from 30 - 40 Ncm.

Plaque scores significantly increased from baseline to 6 months then significantly increased from six months to 12 months. Two reasons may explain this result. Firstly, it is difficult for the patient to perform adequate oral hygiene due to limited accessibility in the posterior maxillary region. Secondly, the cement-retained fixed prosthesis is not perfectly adapted to the implant’s mucosa, and the excess cemented 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. However, plaque indices did not differ between 12 months and 24 months. A similar observation was reported by Medikeri et al. for immediate implant placement in extraction sockets with periapical infection after placement of PRF and Allograft. Gingival scores increased significantly from baseline to 6 months. The increased in gingival scores after 6 months could be attributed to the increased plaque scores with may result in increased mucosal inflammation. However, gingival scores decreased significantly at 12 months and there was no significant difference in gingival scores between 12 months and 24 months. The decreased gingival indices may be due to plaque scores reached a plateau. In addition, the patients were instructed to use chlorohexidine mouth wash in the follow-up visits which may contribute to the reduced gingival index. Similarly, Montoya-Salazar et al. showed that gingival scores were comparable between prosthesis insertion 12, 24, and 36 months later for implants placed in infected sockets and they added that gingival scores did not differ between implants placed in infected and non-infected sockets.

Pocket depth increased significantly from baseline to 6 months. This could be explained by height crestal bone loss after 5 months together with increased peri-implant mucosal inflammation and 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. No significant difference in pocket depth was observed between 6 months...
and 12 months. However, pocket depth significantly decreased from 12 months to 24 months. The decreased pocket depth from 12 to 24 months could be attributed to the decreased plaque and gingival scores, consequently mucosal inflammation decreased and gingival recession may occur. Consequently, pocket depth decreased. In contrast, another author reported no difference in pocket depth between time intervals (over 24 months) for fixtures placed in infected sockets[17]. Also, Montoya-Salazar et al.[11] showed that pocket depth was comparable between prosthesis insertion 12, 24 and 36 months later for implants placed in infected and normal extraction sites.

Mucosal width did not differ between time intervals. Similarly, Crespi et al.[17] reported stable width of keratinized mucosa and marginal gingiva level in patients received immediate implants in chronic infected sockets. Also, Montoya-Salazar et al.[11] showed that both implants inserted in infected socket and non-infected sockets had no difference in keratinized mucosal width between time intervals. Conversely, in another study[24], the authors reported that the width of the keratinized mucosa increased significantly over the observation period for immediate implant placement with peri-apical pathology. However, in all studies the authors reported no difference mucosal width for implants inserted in infected and normal extraction sites.

In this study, Crestal bone loss was measured using cone beam CT instead of conventional periapical radiography as it provides Three-Dimensional information regarding bone resorption at labial and palatal implant surfaces in addition to mesial and distal surfaces. Conversely, the periapical films are two dimensional which provides information on mesial and distal bone resorption only. The use of cone beam CT in evaluation of marginal bone resorption was previously described in other studies[26, 30].

Bone losses in this study were 1.3 ± .36 after 12 months and 1.5 ± 0.41 after 24 months. These values of crestal bone loss were similar to the normal level of bone loss reported in the literature for implants inserted in non-infected sockets which is about 1.2 mm in the first year and 0.2 mm in each subsequent year[29]. These values are similar to crestal bone loss values obtained in another study[21] (1.7 mm) for implants place in infected sockets after 12 months. However, these values are higher than marginal bone loss values reported in other studies[17, 16] (0.79 to 0.86 mm). Crestal bone loss significantly increased from 6 months to 12 months, then significantly increased from 12 months to 24 months. The time dependent bone loss was not surprising and could be attributed to bone reaction to healing and implant loading[33]. A similar observation was noted in another study[20] in which the author reported that crestal bone loss increased significantly for implants placed in infected and normal sockets from one to 3 years. In contrast, Medikeri et al.[16] found no statistically significant difference in crestal bone levels around the implant between at baseline and at 12 months when these implants immediately installed in infected sockets. Also, other studies[17, 36] reported no significant difference in crestal bone loss between observation periods were reported for implants placed in sockets with periapical pathology. In addition, in all forementioned studies the authors reported no statistically significant differences in bone loss between implants inserted in infected and non-infected sockets.

Finally, the limitations of this study include the small sample size and the lack of control group (implants inserted in non-infected sockets). Future randomized clinical trials with increased patient sample and control group still needed to shed more light onto the other confounding factors that may affect the outcomes of implants placed in infected extraction socket.

CONCLUSION
Within the limits of this investigation, post extraction immediate implant insertion in infected sockets of maxillary posterior teeth presented with bone defects or buccal fenestrations and bone augmentation is a safe and predictable method as implants demonstrated good clinical and radiographic outcomes after 2 years.

CONFLICT OF INTEREST
The authors declare no conflict of interest.

REFERENCES
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