

Association Between Smoking and Vertical Post-extraction Alveolar Ridge Resorption: a Randomized Controlled Trial

Ralitsa Yotsova¹, Anzhela Bakhova², Tsvetalina Gerova-Vatsova³

1. Department of Oral Surgery, Faculty of Dental Medicine, Medical University of Varna, Bulgaria

2. Department of Social Medicine and Health Care Organization, Faculty of Public Health, Medical University of Varna, Bulgaria

3. Department of Periodontology and Dental Implantology, Faculty of Dental Medicine, Medical University of Varna, Bulgaria

Abstract

Post-extraction bone resorption causes a significant reduction in the vertical and horizontal dimensions of the alveolar ridge. Over the years, various socket preservation methods have been adopted to minimize bone loss. However, it has been demonstrated that multiple local and systemic factors influence the resorption process. Tobacco smoking is one of the systemic factors thought to influence the resorption process negatively. This randomized clinical trial aimed to assess the effect of smoking on vertical alveolar bone resorption during the first three months after single tooth extraction, with or without an SP procedure.

Materials and methods: The research involved 80 patients who required a single-tooth extraction and underwent either a socket preservation procedure or unassisted socket healing. The heights of the socket plates were measured twice, first after the intervention and then after 3 months, using cone-beam computed tomography.

Results: A statistically significant difference was found only in the second experimental group between smokers and non-smokers ($\chi^2 = 7.2$, $p = 0.007$). No statistically significant difference was observed in the vertical resorption of both plates between smokers and non-smokers after 3 months.

Discussion: Although the harmful effects of smoking on bone healing and resorption have been well-documented, the results of this study did not present any evidence that smoking affects the vertical postextraction loss of the alveolar crest for 3 months after the extraction.

Conclusion: More long-term preclinical and clinical studies are necessary to assess the enduring effects of smoking (and its intensity) on postextraction resorption, if any. Additionally, its influence on both bone quantity and quality should be examined using suitable radiological, histological, and histomorphometric methods.

Keywords: alveolar ridge resorption; post-extraction resorption; socket preservation; alveolar ridge preservation; smoking

Introduction

Tooth extraction triggers a cascade of processes, resulting in the healing of the extraction site and, simultaneously, the resorption of alveolar bone (1).

After tooth extraction, the alveolar ridge significantly decreases in size both vertically and horizontally (2-4). The vertical reduction of the buccal wall is about 10–20% within the first six months. Horizontal loss on the buccal side is even more notable, reaching 30–60% in the first 6–7 months. Afterwards, a yearly reduction of 0.5–1% in the bone contour can be expected (5). Without intervention, approximately 50% of the alveolar bone volume is lost during the first 2–3 years after extraction (6).

The resorption of postextraction sockets in both jaws is more prominent than in the buccal region. This occurs because the cortical bone in this area is often thin, knife-like, and lacks support. The presence of dehiscences and fenestrations further promotes remodelling, resulting in a concavity of the alveolar ridge on the buccal side (7).

Preserving alveolar ridge height is crucial for maintaining functional stability and aesthetics. It allows for the following implant placement and/or prosthetic rehabilitation, avoiding proximity to the maxillary sinus and mandibular canal, surgical and prosthetic complications, and reducing the need for more invasive treatment, such as guided bone regeneration, bone grafting, sinus lift, and alveolar ridge augmentation (8).

Prevention of postextraction resorption is limited by appropriate methods of socket preservation (SP). Most often, this is solved by methods of guided regeneration in the postextraction alveolus. Various procedures have been proposed for SP, such as minimally traumatic extraction, soft tissue and bone grafts, the use of barrier membranes, and immediate implantation.

Multiple surgical procedures are performed to preserve the hard and soft tissues. However, there is no clear clinical criterion (area of dentition, number of preserved alveolar walls, thickness, and height of buccal alveolar bone) for the selection of biomaterials or fillers, nor whether a barrier membrane is needed to isolate them (guided bone regeneration – GBR) or whether socket sealing is necessary (9).

Atwood and Coy suggested four categories of local factors that influence the rate of resorption. The first group (anatomical factors) includes the periodontal biotype, the characteristics of the soft tissues covering the alveolar ridge, and the ridge itself. The second group of factors (metabolic) influences the activity of osteoblasts and osteoclasts. The third group (functional) includes the forces exerted on the crest. All these factors influence cellular activity, bone deposition, and resorption. The last group is the prosthetic factors, which are determined by the type and timing of prosthetic rehabilitation. All the categories described are interconnected in the resorption process (10). In addition to local factors, systemic factors are also considered to have an influence on the rate of bone resorption, including metabolic disorders and smoking.

Although the negative impact of smoking on systemic and oral health is proven and indisputable, its effect on post-extraction resorption of the socket bone walls is not unambiguously documented in the scientific literature. There are a few contradictory statements regarding whether it accelerates and/or increases bone loss in human studies and animal models (11-13).

This randomized clinical trial aimed to assess the effect of smoking on vertical alveolar bone resorption during the first three months following single tooth extraction with or without an SP

procedure. The three-month period aligns with the early implantation phase, as SP is usually performed in preparation for implant placement.

Materials and Methods

Study Design and Setting

A randomized clinical trial was conducted at the Medical and Dental Centre of the Medical University of Varna following the CONSORT guidelines (13). The study received approval from the University Ethics Committee (approval number 118, dated June 23, 2022) and was registered on ClinicalTrials.gov (NCT06621498).

Study Subjects

The study involved 80 patients, smokers and non-smokers, who underwent a single-tooth extraction. The participants were divided into four groups through a random (1:1:1:1) allocation by an independent party (a statistician). The groups were defined as follows: group 1 – SP using a dense non-resorbable polytetrafluoroethylene (PTFE) membrane; group 2 – SP with PTFE and autologous platelet-rich plasma (PRP); group 3 – SP with an autologous palatal or tuberosity soft-tissue graft; group 4 – natural healing. The patients' enrolment is presented in Fig. 1.

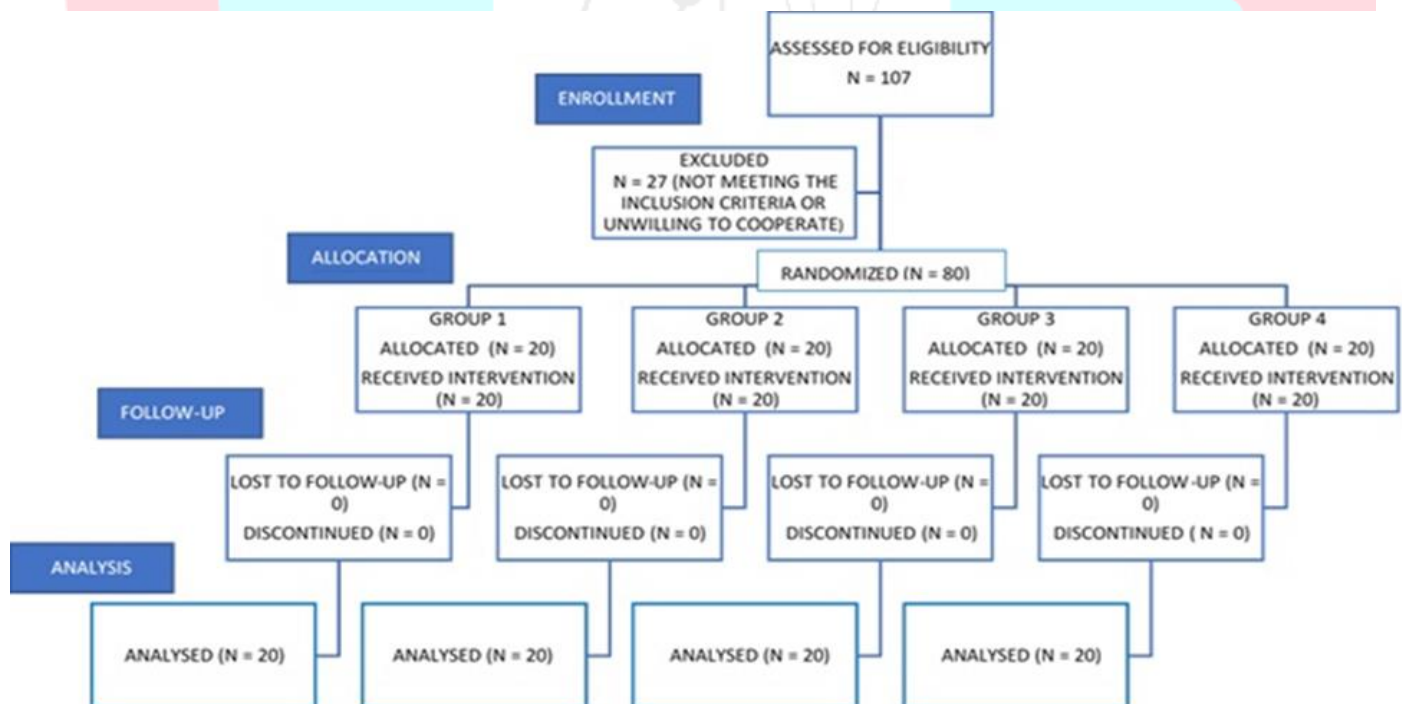


Figure 1. Flow Diagram of the participants.

Eligibility Criteria

The inclusion criteria for this trial were: patients between 18 and 65 years of age, requiring a single-tooth extraction, and those with good systemic health. The exclusion criteria included any local (acute inflammation or neoplasm in the oral cavity) or systemic contraindications (uncontrolled blood pressure, diabetes mellitus, or other metabolic diseases, hemodialysis, pregnancy, etc.) to oral surgery, as well as non-cooperative patients.

The sample size (SS) was determined using a formula for testing a hypothesis in clinical trials or interventional studies (14).

$$SS = 2SD^2(Z_{\alpha/2} + Z_{\beta})^2/d^2$$

SD—standard deviation; $Z_{\alpha/2}$ —standard variate for a significance level; Z_{β} —standard variate for power; d —effect size.

Z values were obtained from the Z table at a type I error of 5% and 80% power ($Z_{\alpha/2} = 1.96$; $Z_{\beta} = 0.84$). The SD value was sourced from previously reported vertical alveolar bone loss (15). The value of 0.8 was chosen as a large effect size ($d = 0.8$).

$$SS = 2(0.77)^2(1.96 + 0.84)^2/0.8^2 = 14.5$$

Data Collection

The heights of the socket plates were measured twice, immediately after the intervention and after 3 months, using cone-beam computed tomography (CBCT) images (Planmeca Pro Max 3D Max). For this purpose, the buccal and oral vertical dimensions were taken on paraxial slices in the middle of the dental socket, from the highest point of the plate to the bottom of the maxillary sinus/ ceiling of the mandibular canal. When interradicular septa were preserved, two measurements were taken for the socket – one medial and one distal, providing more investigation sites than the number of participants. The collected data included patients' demographics (age, gender) and smoking habits (smokers or non-smokers). For most of them, it was difficult to strictly define themselves as light or heavy smokers (taking 10 cigarettes/day as a reference point). Therefore, this division was not further analysed.

Statistical Analysis

IBM SPSS (v. 25.0) and Jamovi software (v. 2.6.44) were used for the statistical analysis of the obtained data (presented by descriptive statistics, the Student's t-test, and the Mann-Whitney U test). According to the distribution, the results were presented through measures of central tendency (mean and median) and variation (standard deviation (SD) and interquartile range (IQR)). Graphical presentation was performed using MS Office Excel 2016. A value of $p=0.05$ was considered statistically significant.

Results

The distribution of participants by gender and age (based on the WHO age group classification) is shown in Figs. 2 and 3, respectively.

The number of smokers (53) was higher in the study compared to non-smokers (27). When dividing the patients into study groups, it is evident that the number of smokers was greater in each group

(Fig. 4). A statistically significant difference was found only between smokers and non-smokers in the second experimental group ($\chi^2 = 7.2$, $P = 0.007$).

Tables 1 and 2 present the reported vertical resorption of both socket bone walls (buccal and oral) across the four study groups, categorised by smoking status (Tables 1 and 2). No statistically significant difference was reported in any of the groups.

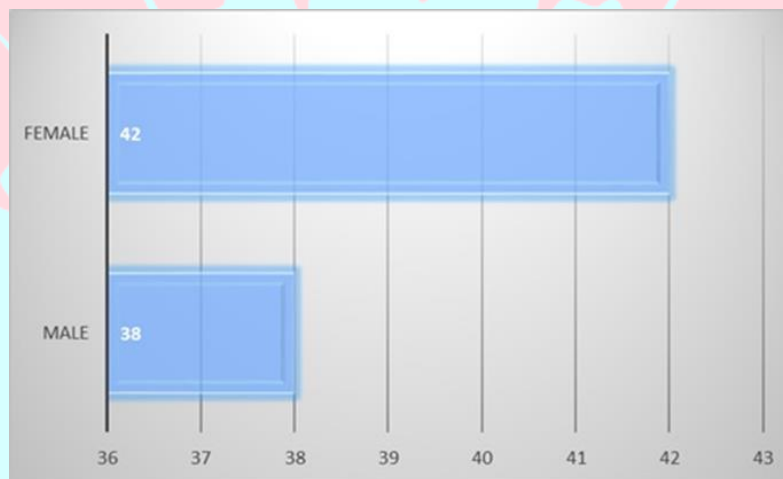


Figure 2. Gender distribution of the participants.



Figure 3. Age distribution of the participants.

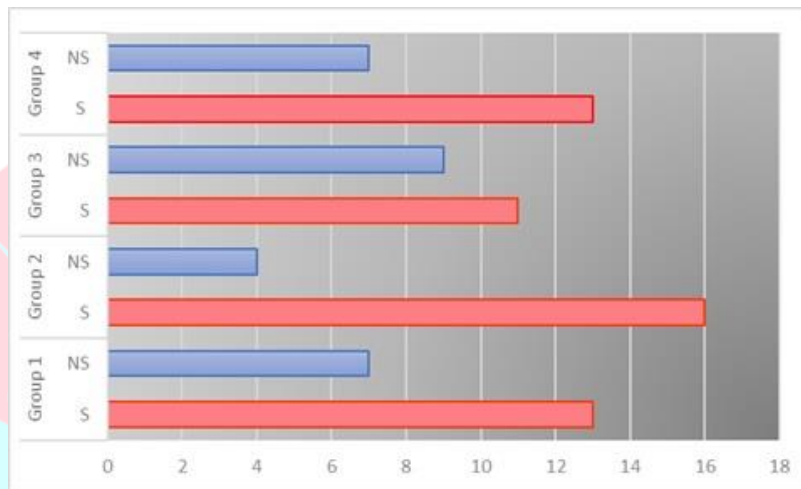


Figure 4. Smoking distribution in the study groups.

Table 1. Vertical resorption of the buccal wall (mm) according to smoking

Group	Smoking	N	Mean (SD)	Meadin (IQR)	T	Mann-Whitney	p
Gr. 1	Yes	14	1.0 (0.5)	-	- 0.6	-	0.5
	No	10	1.2 (0.8)	-			
Gr. 2	Yes	20	-	0.8 (0.7)	-	76.000	0.8
	No	7	-	0.8 (1)			
Gr. 3	Yes	11	1.8 (1.2)	-	0.7	-	0.5
	No	10	1.5 (0.6)	-			
Gr. 4	Yes	15	3.7 (2.2)	-	0.9	-	0.4
	No	9	3.0 (1.5)	-			

Table 2. Vertical resorption of the oral (palatal/lingual) wall (mm) according to smoking

Group	Smoking	N	Mean (SD)	Meadin (IQR)	T	Mann-Whitney	p
Gr. 1	Yes	14	1.2 (0.5)	1.3 (0.8)	-	54.500	0.4
	No	10	1.0 (0.7)	1.0 (1.2)			
Gr. 2	Yes	20	0.8 (0.5)	-	-1.0	-	0.3
	No	7	1.1 (0.7)	-			
Gr. 3	Yes	11	1.3 (0.5)	-	0.02	-	0.1
	No	10	1.3 (0.7)	-			
Gr. 4	Yes	15	2.3 (1.1)	-	0.9	-	0.4
	No	9	1.8 (1.1)	-			

Discussion

It is well known that smoking can significantly harm systemic health, affecting various body systems. Concerning bones, their negative impact is linked to impaired healing and increased resorption, which is nearly three times higher than in non-smokers. Similarly, smoking hampers reparative processes in the oral cavity, slowing bone deposition and accelerating bone loss. It is also associated with a higher frequency and severity of issues in smokers compared to non-smokers (17). In dental implant treatment, smoking raises the risk of implant failure and peri-implant bone loss (18-21). Poor periodontal health and the presence of bone defects are more prevalent among smokers (21-23). Additionally, it is believed that smoking may influence the inflammatory and cellular response in the bone following extraction, although the precise mechanism remains unclear. Smoking has been reported to increase horizontal resorption and reduce bone density in post-extraction areas during the first six months after tooth loss (17). Similar vertical bone resorption and density in the centre of the postextraction socket were observed after 6 months in both smokers and nonsmokers. Still, a significant difference was observed in horizontal resorption and bone density in the apical part of the socket, which was more pronounced in smokers (17).

Part of the harmful effect of smoking on healing processes is attributed to nicotine, which is a cytotoxic, vasoactive substance. In vivo and in vitro studies have demonstrated its negative impact on revascularisation, bone healing, and healing processes in guided bone regeneration. Nicotine also suppresses the expression of a wide range of cytokines, including those associated with neovascularisation and osteoclast differentiation. It has also been found to affect fibroblast proliferation, increase collagenase activity, and suppress the synthesis of fibronectin and type 1 collagen (11).

Although smoking is considered a factor influencing post-extraction resorption and the outcomes of SP procedures, in this study, no statistically significant difference was observed in the vertical resorption of both plates after 3 months in smokers and non-smokers. It must be noted that the distribution of smokers and non-smokers was not equal. In addition, some other factors were not evaluated, such as hormone-age relation factors, which are crucial in females, primarily due to a significant portion of early elderly and middle-aged individuals. Further research with a larger sample size and more homogeneous participants is needed to support these findings. Another possible limitation is the short follow-up period, which cannot be illustrative for the long-term effect on smoking after the extraction. Moreover, the presented findings are based only on CBCT analysis, and conclusions on how smoking affects bone density and quality in the postextraction sockets cannot be drawn. Bone histology and histomorphometry would be helpful for these purposes. Therefore, the results of this study should be interpreted with caution.

Several other studies have reported similar results for SP in smokers and non-smokers (24-26). It is worth noting that no difference was observed in the control group in this study either. These findings are partly consistent with those reported by Hoffman et al. (12), who conducted a retrospective, randomised trial on the use of non-porous PTFE membranes alone for RP in 276 post-extraction sockets. The authors examined the influence of various factors, including smoking, on bone level and demonstrated that smoking did not affect healing. Nearly half of the participants were smokers, but no difference in treatment outcomes was found between the two groups.

Limitations

This clinical trial has several limitations, including a short follow-up period of 3 months, which is suitable for early implantation but may not be informative enough for the timing of late and delayed implantation, as well as the relatively small sample size. A larger sample size would enable a more precise classification of smokers into light and heavy smokers.

Future directions

Further long-term preclinical and clinical trials should be conducted to validate or refute the presented findings and address the limitations of this research. Histological and histomorphometric analysis would be beneficial for the quantitative and qualitative evaluation of the healing bone in postextraction sites.

Discussion

Although the harmful effects of smoking on bone healing and resorption have been well-documented, the results of this study did not present any evidence that smoking affects the vertical postextraction loss of the alveolar crest for 3 months after the extraction. Further longitudinal preclinical and clinical trials are needed to observe the long-term effects of smoking (and its heaviness) on postextraction resorption, if any. Furthermore, its impact not only on bone quantity but also on quality should be evaluated using appropriate radiological, histological, and histomorphometric investigations.

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Corresponding author:

Ralitsa Yotsova

Department of Oral Surgery, Faculty of Dental Medicine, Medical University of Varna, Bulgaria

E-mail: r.yotsova@abv.bg

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