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Evaluation of the effect of orthognathic surgery on alveolar bone level: a cone-beam computed tomography study

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ABSTRACT

Aims: Three-dimensional examination of preoperative and postoperative changes in alveolar bone levels with cone-beam computed tomography (CBCT) in cases with skeletal class II and III anomalies treated with different surgical methods, supported by cephalometric images.

Methods: A total of 32 patients, 18 girls and 14 boys, who applied to Dicle University Faculty of Dentistry Department of Orthodontics for orthognathic surgery-supported orthodontic treatment and were treated with orthognathic surgery after initial orthodontic treatment was started, preoperatively in Dicle University Oral Diagnosis and Radiology Department. It was created by retrospectively examining CBCT images taken before and after. To examine changes in alveolar bone level, 28 measurements were made using alveolar bone levels and reference points determined on teeth.

Results: When the preoperative and postoperative groups were compared, a significant difference was found in the upper anterior bone level, upper palatinal bone thickness, lower anterior bone level, upper trifurcation buccal, upper distobuccal root middle buccal, lower bifurcation buccal, lower distal root middle buccal values at the $p<0.05$ level. A statistically significant difference was observed in the enamel cement joint width value in the comparison between the sexes. When the correlation between class II and class III anomalies was examined, it was observed that lower anterior bone thickness, lower anterior bone level/root and lower lingual bone level/root values were statistically associated with more alveolar bone loss in class III patients.

Conclusion: Orthognathic surgery causes alveolar bone loss in the patient. In order to minimize the side effects of the operation on the patient's periodontal tissues, oral hygiene, applied forces, fixation between the jaws and methods should be carefully evaluated.

Keywords: Alveolar bone loss, cephalometry, orthognathic surgery, cone-beam computed tomography

INTRODUCTON

During the birth, growth and development of a person, the facial structures formed anatomically depend primarily on genetics; secondarily, it depends on environmental factors. Dentofacial deformities occur as a result of abnormal or disproportionate growth of facial structures. This situation may occur prenatally or developmentally; it may also occur as a result of postnatal factors such as trauma, infection and other external factors.[1]

Depending on these factors; in order to correct the musculoskeletal system, dento-osseous and soft tissue deformities of the jaws and related facial structures, diagnosis, treatment planning and application should be carried out in a coordinated manner by combining orthodontics and maxillofacial surgery.[2]

Studies have found that approximately 20% of the world's population has some type of facial deformity.[3] This situation directly affects individuals' quality of life and social satisfaction.[4,5]

The aims of orthognathic surgery treatment are multifaceted. When planning treatment, improving facial aesthetics, providing a functional occlusion, protecting and widening the airway if possible, ensuring or maintaining periodontal health, healthy temporomandibular joint, and most importantly, eliminating the patient's main complaints with the least complications should be taken into consideration.[6]

Complications that mostly occur during routine orthodontic treatment; periodontal problems, alveolar bone loss and root resorption. Studies have mostly focused on periodontal tissues and osseous bone losses due to orthodontic treatment. However, studies on bone loss after orthognathic surgery, which is performed more frequently today, have been observed very rarely.

It should not be forgotten that good oral hygiene and periodontal condition before surgery are important factors affecting the success of surgery and the risk of complications.

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Various complications related to orthognathic surgery that occur after treatment and the changes that may occur in the alveolar bone have become a matter of concern. Since orthognathic surgery procedures depend on many different factors in different ways, different procedures and periods, the complications that may arise and the responses of the tissues need to be carefully evaluated in a broad context. When the literature was reviewed, no study was found in which the alveolar bone level of both anterior and posterior teeth was evaluated together with the changes in the anterior incisor tooth angles in class II and class III cases after orthognathic surgery.

Therefore, the aim of our study is to plan and start treatments at Dicle University Faculty of Dentistry, Department of Orthodontics; To examine pre-and postoperative alveolar bone levels, both anteriorly and posteriorly, in class II and class III cases that underwent orthognathic surgery at Dicle University Faculty of Medicine, Department of Plastic, Reconstructive and Aesthetic Surgery.

METHODS

Purpose and Type of Research

The material of this study was collected from a total of 32 patients, 18 girls and 14 boys, who applied to Dicle University Faculty of Dentistry Department of Orthodontics for orthodontic treatment supported by orthognathic surgery and were treated with orthognathic surgery by the same surgeon using the same operation method after the initial orthodontic treatment was started. It was created by retrospectively examining cone beam computed tomography (CBCT) images taken before and after the operation in the department of radiology.

Ethical Aspect of Research

The ethics committee report with protocol number 2021-06 was received from the Local Ethics Committee of Dicle University Faculty of Dentistry Deanery (Date: 27.01.2021, Decision No: 2021-06). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

In our study, the entire patient database was evaluated and an attempt was made to evaluate as many patients as possible who met the conditions.

Population and Sample of the Research

In our study, 32 patients between the ages of 18-46 applied to Dicle University Faculty of Dentistry Orthodontics Department for orthognathic surgery-supported orthodontic treatment; While only 5 of them have class II malocclusion, the remaining 27, the majority, have class III malocclusion. In addition, maxillary advancement-mandibular setback was applied to 21 class III patients, mandibular setback was applied to 3 class III patients, maxillary advancement was applied to 3 patients, and mandibular advancement was applied to 5 class II patients.

Data Collection and Analysis

CBCT images taken before and after surgery, with a minimum period of 6 months, a maximum of 21 months, and an average of 9.9 months, were used. Preoperative CBCTs were taken to be used for anatomical dental and osseous examination and orthognathic surgery planning.

A total of 56 measurements were made for each patient, both upper and lower and before and after surgery, from the alveolar sections around the incisors and molars using CBCT images. NemoStudio 2019 (NemoStudio, Software Nemotec, SL, Spain) software was used to examine the CBCT images and make measurements on the relevant sections.

Linear Measurements

Upper anterior bone level (UABL): Distance between the buccal enamel cementum border and the buccal alveolus crest, parallel to the upper incisor axis.

Upper palatal bone level (UPBL): Distance between the palatal enamel cementum border and the palatal alveolus crest, parallel to the upper incisor axis.

Upper anterior bone thickness (UABT): Distance between the root tip and the intersection of the buccal maxillary curvature perpendicular to the upper incisor axis.

Upper palatal bone thickness (UPBT): The distance between the root tip and the intersection of the palatal maxillary curvature, perpendicular to the upper incisor axis.

Upper root length: The distance between the intersection point between the upper incisor-enamel-cement junction width and the tooth axis and the root tip.

Upper enamel cementum junction width: Distance between the upper incisor buccal and palatal cementum enamel junctions.

Lower anterior bone level (LABL): Distance between the buccal enamel cementum border and the buccal alveolus crest, parallel to the lower incisor axis.

Lower lingual bone level (LLBL): The distance between the lingual enamel cementum border and the lingual alveolar crest parallel to the lower incisor axis.

Lower anterior bone thickness (LABT): The distance between the root tip and the intersection of the buccal mandibular symphysis, perpendicular to the lower incisor axis.

Lower lingual bone thickness (LLBT): The distance between the root tip and the intersection of the lingual mandibular symphysis, perpendicular to the lower incisor axis.

Lower root length: The distance between the intersection point between the lower incisor-enamel-cement junction width and the lower incisor axis and the root tip.

Upper trifurcation buccal: The shortest distance between the buccal alveolar border and the tooth in the horizontal section of the upper first molar at the trifurcation level.

Upper trifurcation palatal: The shortest distance between the palatal alveolar border and the tooth in the horizontal section of the upper first molar at the trifurcation level.

Upper distobuccal root middle buccal: The shortest distance between the buccal alveolar border and the tooth in the horizontal section of the upper first molar tooth at the level of the middle of the distobuccal root.

Upper distobuccal root mid-palatal: The shortest distance between the buccal alveolar border and the tooth in the horizontal section of the upper first molar at the level of the middle of the distobuccal root.

Upper mesiobuccal alveolar height: The distance between the crest of the alveolar crest and the crest of the mesial tubercle in the frontal section of the upper first molar at the level of the mesial tubercle.

Upper middle alveolar height: The distance between the alveolar crest and the buccal ridge crest in the frontal section of the upper first molar at the buccal ridge level.

Upper distobuccal alveolar height: The distance between the top of the alveolar crest and the top of the distobuccal tubercle in the frontal section of the upper first molar at the level of the distobuccal tubercle.

Lower bifurcation buccal: The shortest distance between the buccal alveolar border and the tooth in the horizontal section of the lower first molar at the bifurcation level.

Lower bifurcation lingual: The shortest distance between the lingual alveolar border and the tooth in the horizontal section of the lower first molar at the bifurcation level.

Lower distal root middle buccal: The shortest distance between the buccal alveolar border and the tooth in the horizontal section of the lower first molar at the level of the middle of the distal root.

Lower distal root mid-lingual: The shortest distance between the buccal alveolar border and the tooth in the horizontal section of the lower first molar at the level of the middle of the distal root.

Lower mesiobuccal alveolar height: The distance between the alveolar crest and the mesiobuccal tubercle in the frontal section of the lower first molar at the level of the mesiobuccal tubercle.

Lower middle alveolar height: The distance between the alveolar crest and the buccal middle tubercle in the frontal section of the lower first molar at the level of the buccal middle tubercle.

Lower distobuccal alveolar height: The distance between the top of the alveolar crest and the top of the distobuccal tubercle in the frontal section of the lower first molar at the level of the distobuccal tubercle (Figure 1-4).

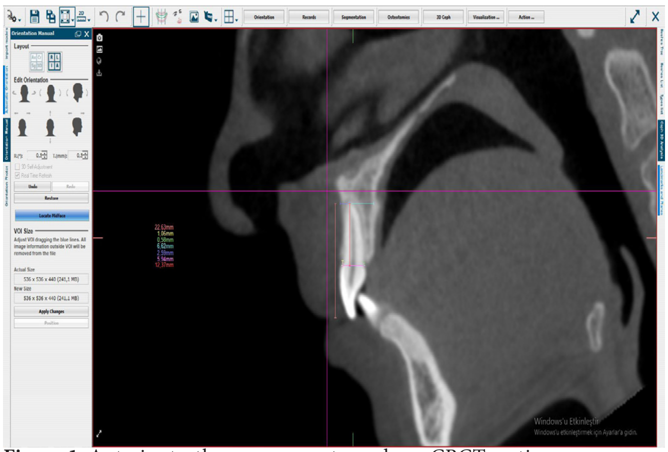

Figure 1. Anterior teeth measurements made on CBCT section

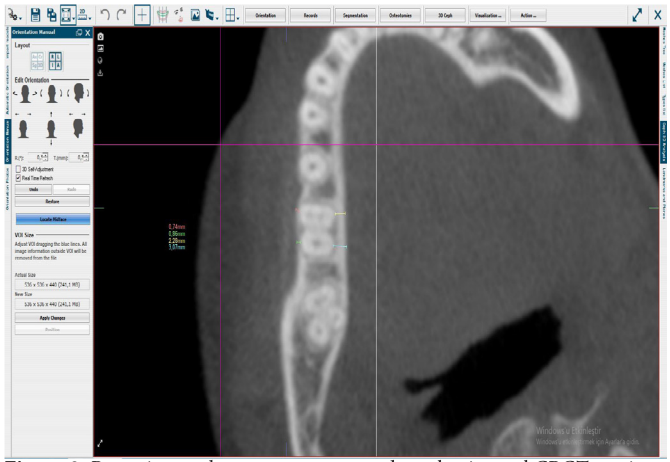

Figure 2. Posterior teeth measurements made on horizontal CBCT section

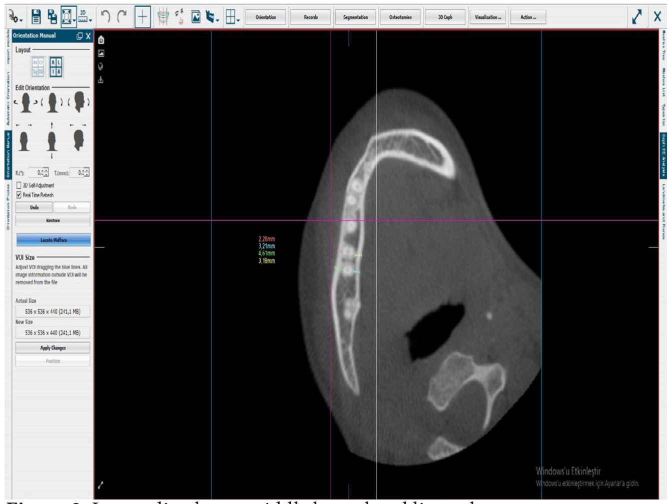

Figure 3. Lower distal root middle buccal and lingual measurements

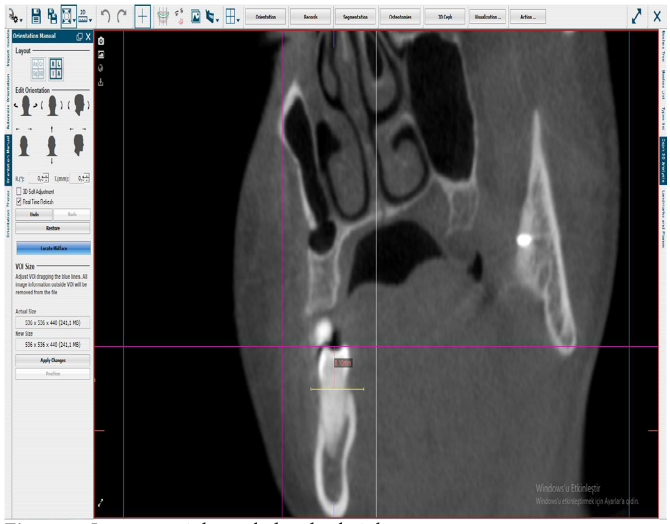

Figure 4. Lower mesiobuccal alveolar height

Statistical Analysis

Normal distribution of the data was tested with the Shapiro-Wilk test. Mann-Whitney u test was used to compare non-normally distributed measurements in 2 groups, and Kruskal-Wallis and Dunn multiple comparison tests were used to compare more than 2 groups. Wilcoxon signed rank test was used to evaluate the changes in measurements before and after surgery.

Relationships between numerical variables were evaluated with the Spearman correlation coefficient. Analyzes were made using the SPSS for Windows 24 program. A p-value of less than 0.05 was considered statistically significant.

RESULTS

For sample size estimation, we used the upper anterior bone level data from Kim et al.[16] according to the power analysis performed using G*Power Software version 3.1.9.2 (Universität Düsseldorf, Germany), it was determined that the alpha error probability was 0.05 and the sample size should consist of 15 patients for 80% power.

When the gender distribution of the patients is examined, it is seen that 56.3% are female and 43.8% are male. When the class distribution is examined, it is seen that 84.42% are class III and 15.6% are class II. When the group distribution of the patients is examined, it is seen that 65.6% are patients who have undergone double jaw surgery, 25% have only mandible surgery, and 9.4% have only maxilla surgery (Table 1).

There is a statistically significant difference between preoperative and postoperative measurements of upper anterior bone level, upper palatal bone thickness, upper root length, UABL/root UPBL/root, (UABT+UPBT)/CEJW, lower anterior bone level, lower root length LABL/root, LLBL/root, (LABT+LLBT)/CEJW, upper trifurcation buccal, upper DBR middle buccal, lower bifurcation buccal, lower DBR middle buccal, L1-mandibular plane values (p<0.05).

It is seen that the postoperative measurements of the upper anterior bone level, UABL/root, UPBL/root, lower anterior bone level, LABL/root, LLBL/root, (LABT+LLBT)/CEJW values are higher than the preoperative measurements.

Postoperative measurements of preoperative measurements of upper palatal bone thickness, upper root length, (UABT+UPBT)/JECW, lower root length, upper trifurcation buccal, upper DBR middle buccal, lower bifurcation buccal, lower dr middle buccal, L1-mandibular plane values appears to be higher.

L1-mandibular plane, (UABT+UPBT)/CEJW, lower anterior bone thickness, (LABT+LLBT)/CEJW values were statistically

Table 1. Comparison of pre-and post-operative measurements in millimeters p<0.05

	Pre-operative	Post-operative		
Variables	**Mean±SD**	**Mean±SD**	**Average difference [95% CI]**	**p**
UABL	2.3±1.01	2.75±1.23	-0.45 [-0.73--0.16]	0.004*
UPBL	3.05±2.1	3.41±2.02	-0.35 [-0.72-0.01]	0.052
UABT	3.46±1.35	3.48±1.24	0.02 [-0.45-0.49]	0.674
UPBT	5.18±2.4	4.32±1.83	-0.86 [-1.39--0.33]	0.004*
Upper root length	12.19±1.51	11.7±1.64	-0.49 [-0.74--0.23]	0.001*
UABL/root	0.19±0.09	0.24±0.11	0.05 [0.02-0.07]	0.001*
UPBL/root	0.26±0.2	0.31±0.22	-0.05 [-0.09--0.01]	0.004*
(UABT+UPBT)/SEJW	1.33±0.29	1.21±0.3	-0.12 [-0.17--0.06]	0.001*
LABL	5.41±2.43	6.86±2.47	-1.45 [-2.02--0.87]	0.001*
LLBL	7.58±3.73	8.27±3.2	-0.69 [-1.35--0.03]	0.070
LABT	4.61±1.96	4.29±1.83	-0.32 [-0.8-0.16]	0.065
LLBT	1.97±1.4	1.74±1.29	-0.24 [-0.5-0.03]	0.090
Lower root length	11.75±1.25	11.39±1.29	-0.36 [-0.63--0.09]	0.005*
LABL/root	0.47±0.23	0.61±0.23	0.14 [0.09-0.19]	0.001*
LLBL/root	0.65±0.31	0.73±0.27	0.08 [0.02-0.14]	0.002*
(LABT+LLBT)/CEJW	1.2±0.41	1.21±0.77	0.01 [-0.23-0.26]	0.003*
Upper trifurcation buccal	0.7±0.8	0.43±0.61	-0.27 [-0.47--0.07]	0.010*
Upper trifurcation palatinal	0.52±0.74	0.43±0.62	-0.09 [-0.28-0.1]	0.420
Upper DBR middle buccal	0.36±0.61	0.06±0.24	-0.31 [-0.48--0.14]	0.003*
Upper DBR middle palatinal	0.9±1.13	0.87±0.92	-0.03 [-0.29-0.24]	0.936
Upper mesiobuccal alveolar height	7.97±0.95	8.33±0.95	-0.36 [-0.78-0.06]	0.080
Upper middle alveolar height	8.42±1.3	8.76±1.15	-0.33 [-0.78-0.11]	0.150
Upper distobuccal alveolar height	8±1.05	8.1±1.08	-0.1 [-0.52-0.33]	0.531
Lower bifurcation buccal	0.39±0.57	0.13±0.36	-0.25 [-0.44--0.06]	0.016*
Lower bifurcation lingual	0.9±0.91	0.92±1.07	0.03 [-0.18-0.23]	0.871
Lower DR middle buccal	0.6±0.91	0.31±0.81	-0.29 [-0.56--0.02]	0.043*
Lower DR middle lingual	2.26±1.15	2.27±1.3	0.01 [-0.26-0.28]	0.964
Lower mesiobuccal alveolar height	8.21±1.4	8.33±1.18	-0.12 [-0.49-0.26]	0.504
Lower middle alveolar height	8.13±1.1	8.58±1.37	-0.45 [-0.85--0.04]	0.054
Lower distobuccal alveolar height	7.11±0.91	7.4±1.45	-0.3 [-0.7-0.11]	0.511
U1-palatal plane	111.38˚±10.13	113.41˚±8.44	2.03˚ [-0.55-4.61]	0.058
L1-mandibular plane	85.99˚±8.41	83.67˚±6.87	-2.32˚ [-4.21--0.42]	0.024*

SD: Standart deviation, UABL: Upper anterior bone level, UPBL: Upper palatinal bone level, UABT: Upper anterior bone thickness, UPBT: Upper palatinal bone thickness, LABL: Lower anterior bone level, LLBL: Lower lingual bone level, CEJW: Cement enamel junction width, LABT: Lower anterior bone thickness, LLBT: Lower lingual bone thickness significant. (UABT+UPBT)/CEJW value decreased more in women than in men.

When examined between class II and class III groups, the changes in lower anterior bone thickness, lower root length, LABL/root, LABL/root values are statistically significant.

Lower anterior bone thickness decreased more in class III patients than in class II patients. Lower root length decreased more in class II patients than in class III patients. Changes in LABL/root and LLBL/root values changed more in class II patients than in class III patients (Table 2, 3).

When patients who had only lower and only upper jaw surgery were compared, the change in lower anterior bone level changed more in patients who had only lower jaw surgery than in patients who had only upper jaw surgery. Lower anterior bone thickness measurement changed more in patients who had double jaw surgery than in patients who had single mandible surgery. Lower distal root mid-lingual measurement changed more in patients who underwent double jaw surgery than in patients who underwent single upper jaw surgery.

DISCUSSION

Today, the Le Fort I osteotomy procedure for the correction of severe dentofacial deformities has been modified and improved in recent years and has become one of the standard operations performed in oral and maxillofacial surgery.[7]

The surgery, that is, Le Fort 1, which is usually performed together with bilateral sagittal split ramus osteotomy (BSSRO), allows changes in all three directions of space, is frequently preferred to correct functional and cosmetic irregularities, and is used in the treatment of a wide variety of malocclusions.[8]

Oral rehabilitation combined with orthognathic surgery is a long and challenging process that relies on the cooperation of the patient throughout the treatment to achieve the goals of functional improvement, prevention and correction of deformities, and improvement of quality of life.[9]

No matter how accurate the diagnosis, how comprehensive the approach, and how meticulous the surgical technique, complications will occur in a small percentage of patients after orthognathic surgery. This situation is an expected possibility.

Table 2. Comparison of differences in millimeters according to the operated jaw p<0.05

	Mand (n=8)	Mand+Max (n=21)	Max (n=3)	
Variables	**Mean±SD**	**Mean±SD**	**Mean±SD**	**p**
UABL	-0.31±0.59	-0.53±0.89	-0.22±0.6	0.853
UPBL	-0.07±0.65	-0.41±1.17	-0.75±0.59	0.316
UABT	-0.04±2.08	0.12±0.97	-0.52±1.16	0.392
UPBT	-0.68±2.26	-0.91±1.15	-0.98±1.5	0.592
Upper root length	-0.43±0.53	-0.5±0.8	-0.59±0.5	0.776
UABL/root	0.03±0.04	0.05±0.08	0.03±0.07	0.887
UPBL/root	-0.03±0.04	-0.05±0.13	-0.09±0.07	0.517
(UABT+UPBT)/SEJW	-0.11±0.06	-0.12±0.16	-0.11±0.31	0.807
LABL	-2.49±1.62	-1.23±1.49	-0.18±0.3	0.041*
LLBL	-2.22±2.74	-0.18±1.11	-0.17±0.92	0.203
LABT	0.43±1.85	-0.62±1.09	-0.25±0.42	0.037*
LLBT	-0.76±0.71	-0.09±0.7	0.12±0.63	0.077
Lower root length	-0.9±1.18	-0.17±0.47	-0.22±0.24	0.091
LABL/root	0.24±0.15	0.11±0.13	0.04±0.05	0.061
LLBL/root	0.23±0.24	0.03±0.09	0.04±0.08	0.055
(LABT+LLBT)/CEJW	-0.06±0.34	-0.13±0.12	1.19±2.08	0.113
Upper trifurcation buccal	-0.51±0.62	-0.24±0.5	0.16±0.61	0.442
Upper trifurcation palatinal	-0.18±0.51	-0.02±0.48	-0.37±1.01	0.893
Upper DBR middle buccal	-0.3±0.43	-0.23±0.47	-0.84±0.24	0.057
Upper DBR middle palatinal	0.24±1.04	-0.1±0.58	-0.2±1.06	0.256
Upper mesiobuccal alveolar height	-0.2±0.91	-0.4±1.33	-0.6±0.37	0.766
Upper middle alveolar height	-0.37±1.15	-0.39±1.28	0.19±1.44	0.640
Upper distobuccal alveolar height	-0.02±1.55	-0.1±0.94	-0.28±2.14	0.947
Lower bifurcation buccal	-0.14±0.82	-0.29±0.41	-0.28±0.48	0.595
lower bifurcation lingual	-0.08±0.45	0.07±0.66	-0.03±0.06	0.583
Lower DR middle buccal	-0.13±1.24	-0.35±0.52	-0.34±0.59	0.117
Lower DR middle lingual	0.39±0.66	-0.01±0.74	-0.86±0.23	0.024*
Lower mesiobuccal alveolar height	0.15±0.87	0.01±0.79	-1.69±1.91	0.086
Lower middle alveolar height	-0.46±1.27	-0.47±1.01	-0.24±1.94	0.899
Lower distobuccal alveolar height	-0.21±0.93	-0.28±1.14	-0.63±1.78	0.847
U1-palatal plane	4.5˚±8.6	2.19˚±4.91	-5.67˚±13.5	0.565
L1-mandibular plane	-2˚±6.68	-1.72˚±4.67	-7.33˚±3.51	0.202

UABL: Upper anterior bone level, UPBL: Upper palatinal bone level, UABT: Upper anterior bone thickness, UPBT: Upper palatinal bone thickness, LABL: Lower anterior bone level, LLBL: Lower lingual bone level, LABT: Lower anterior bone thickness, LLBT: Lower lingual bone thickness, CEJW: Cement enamel junction width

Table 3. Correlations between differences with anomaly p<0.05

	Class II (n=5)	Class II (n=27)	
Variables	**Mean±SD**	**Mean±SD**	**p**
UABL	-0.15±0.52	-0.5±0.83	0.604
UPBL	-0.15±0.82	-0.39±1.06	0.500
UABT	0.44±2.46	-0.06±1.04	0.775
UPBT	-1.19±2.5	-0.8±1.27	0.836
Upper root length	-0.54±0.56	-0.48±0.74	0.697
UABL/root	0.02±0.04	0.05±0.08	0.696
UPBL/root	-0.05±0.05	-0.05±0.12	0.550
(UABT+UPBT)/SEJW	-0.12±0.04	-0.12±0.17	1.000
LABL	-2.55±1.94	-1.24±1.46	0.154
LLBL	-2.07±2.56	-0.44±1.59	0.169
LABT	1.08±1.51	-0.58±1.14	0.010*
LLBT	-0.82±0.68	-0.13±0.72	0.058
Lower root length	-1.43±0.9	-0.16±0.53	0.009*
LABL/root	0.26±0.14	0.11±0.13	0.045*
LLBL/root	0.22±0.19	0.05±0.14	0.022*
(LABT+LLBT)/CEJW	0.06±0.33	0±0.73	0.287
Upper trifurcation buccal	-0.51±0.74	-0.23±0.52	0.772
Upper trifurcation palatinal	-0.36±0.57	-0.04±0.53	0.255
Upper DBR middle buccal	-0.48±0.46	-0.27±0.47	0.257
Upper DBR middle palatinal	0.08±1.22	-0.04±0.66	0.309
Upper mesiobuccal alveolar height	-0.27±0.97	-0.38±1.21	0.897
Upper middle alveolar height	-0.11±1.36	-0.37±1.23	0.897
Upper distobuccal alveolar height	-0.04±0.48	-0.1±1.28	0.716
Lower bifurcation buccal	-0.02±0.97	-0.3±0.42	0.161
Lower bifurcation lingual	-0.07±0.6	0.04±0.59	0.317
Lower DR middle buccal	-0.32±1.6	-0.29±0.52	0.343
Lower DR middle lingual	0.38±0.63	-0.06±0.76	0.194
Lower mesiobuccal alveolar height	-0.24±0.8	-0.09±1.09	0.795
Lower middle alveolar height	0.18±1.05	-0.56±1.12	0.169
Lower distobuccal alveolar height	0.34±0.25	-0.41±1.18	0.065
U1-palatal plane	6±10.84	1.3±6.29	0.604
L1-mandibular plane	-3.2±7.6	-2.15±4.89	0.716

*Significant at 0.05 level, Mann Whitney u test, p: Statistical significance value, UABL: Upper anterior bone level, UPBL: Upper palatinal bone level, UABT: Upper anterior bone thickness, UPBT: Upper palatinal bone thickness, LABL: Lower anterior bone level, LLBL: Lower lingual bone level, LABT: Lower anterior bone thickness, LLBT: Lower lingual bone thickness

The aim should be to minimize these complications, increase patient comfort and have less traumatic experiences.

Many studies on the incidence and types of complications have been reported in the literature. In their study, Sousa and Turrini[10] confirmed the low prevalence of postoperative complications with a literature review and showed that the data during osteotomy were approximately 12% sensory change, 3.4% infection, 2.5% fixation problems, and 1.8% unintended fracture rates.

It should not be forgotten that good oral hygiene and periodontal condition before surgery are important factors affecting the success of surgery and the risk of complications. Lupi et al.[11] they also stated in their study that the degree of bone loss during adult orthodontic treatment may be higher than that observed in adolescents, especially if poor periodontal condition is not treated before orthodontic treatment begins. This situation paves the way for periodontal and osseous defects in the adult patient profile where orthognathic surgery is especially applied.

Nelson et al.[12] showed in their study that orthodontic variables such as the type of tooth movement (especially intrusion, lingual tipping) and treatment duration are more important factors for attachment loss resulting from the use of appliances during orthodontic treatment.

Steiner et al.[13] in their study on monkeys, it was shown that orthodontic movement in the labial direction caused loss of marginal bone and connective tissue attachment and gingival recession.

Yoonji et al.[14] in their study, they emphasized the need to reconsider excessive orthodontic movement, especially in skeletal class III adult patients, according to the patient's anatomical boundaries and periodontal health.

As a result of the studies carried out by all these researchers, different complications related to orthognathic surgery and the changes that may occur in the alveolar bone have become a matter of concern. Since orthognathic surgery procedures are different and depend on many different factors, the complications that may arise need to be carefully evaluated in a broad context. Although we want to examine the isolated relationship of orthognathic surgery with alveolar bone loss, we must state that we cannot completely isolate the effects of orthodontic treatment and orthognathic surgery on bone from each other.

Considering this information, our study aimed to evaluate pre-and post-operative alveolar bone levels in patients who underwent orthognathic surgery following orthodontic treatment.

Many studies have been conducted in the literature involving different numbers of patients. For example, Nicodemo et al.[15] in their study based on 29 patients aged between 17 and 46, with angle class III malocclusion and indication for surgical intervention; The patients received orthodontic preparation between 1 year and 1 year and 6 months, and then underwent orthognathic surgery.

Kim and Kook[16] conducted a study on tomography images taken at least 1 month before the surgery in 20 patients with class III crossbite and open bite who were indicated for orthognathic surgery, and found that alveolar bone level losses in the mandibular incisors were greater, especially in the lingual area, compared to the maxilla, and that the maxillary incisors were affected in the palatal area. They stated that the bone thickness on their faces was significantly greater than the lingual of the mandibular incisors and emphasized that special attention should be paid to bone loss in the lower incisor region during orthodontic treatment, especially in class III orthognathic surgery patients.

Radiographs and advanced imaging techniques are of great importance in evaluating alveolar bone changes. Bholsith et al.[18] stated that cephalometric analysis is one of the basic tools of craniomaxillofacial surgery as well as orthodontic diagnosis, and they also defined cephalometry as a two-dimensional reflection of three-dimensional structures.

Cephalometric films may have disadvantages such as non-homogeneous growth and distortions of lateral structures and incorrect landmark positions due to overlapping structures. Incorrect head position may cause incorrect diagnosis.

Choi et al.[15] reported in their study that diagnoses regarding bone structure can be made with excellent accuracy by cone beam computed tomography (CBCT). In addition, they stated that CBCT overcomes the limitations of traditional two-dimensional radiographs and provides three-dimensional images that facilitate measurements from buccal and lingual bone plates and reflect much more reality.[18] Considering this information, CBCT was preferred in our study in order to minimize distortion and to avoid errors caused by incorrect head position during the measurement of all anatomical points used in our parameters.

Lee et al.,[19] in their study on 25 class III orthognathic surgery patients, stated that the IMPA (L1-Mandibular Plane) angle, which was 92.17 degrees before surgery, decreased to 87.42 degrees after the operation. In our study, the L1-Mandibular plane angle decreased from 85.99 to 83.67 degrees. The researcher stated that excessive buccal incisor movement in orthodontic treatment before surgery causes alveolar bone resorption, and their findings are also compatible with our study.

Kim and Park[20] looked at UABL (upper anterior bone level), UPBL (upper palatinal bone level), UABT (upper anterior bone thickness), UPBT (upper palatal bone thickness), LABL (lower anterior bone level), LLBL (lower lingual bone level), LABT (lower anterior bone thickness), LLBT (lower lingual bone thickness) values in their study and found that the alveolar bone thickness in the upper jaw was thicker than in the lower jaw symphysis, but inversely with the thickness, bone losses were greater in the lower jaw symphysis region than in the maxilla anterior. In our study, alveolar bone losses are concentrated in the mandible, and mandibular anterior losses and mandibular lingual losses are greater than maxillary losses.

Many researchers have suggested that excessive labial or lingual movement of maxillary and mandibular incisors should be avoided to prevent irreversible bone loss that leaves the tooth with less bone support.[21,22] In our study, alveolar resorption occurred not only in the anterior region but also in the molar region with tooth movement directed towards the cortical bone, with the values of upper trifurcation buccal, upper disto buccal root middle buccal, lower bifurcation buccal and lower distal root middle buccal.

It is recommended that the use of elastics used after orthognathic surgery should not exceed physiological limits in terms of force and duration, and that the treatment time and amount of surgical movement should be kept as minimal as possible and the treatment should be completed with minimum tension after the operation.[23]

Current studies have shown that anterior tooth inclination causes losses such as fenestration and dehiscence, as well as local alveolar bone loss, if long-term and severe force is applied, especially in the mandible anterior.[24]

Steiner et al.[13] in their study, they found that orthodontic movement in the labial direction caused loss of marginal bone and connective tissue attachment. In our study, as seen from the decrease in UABL (upper anterior bone level), LABL (lower anterior bone level) values and changes in incisor angles, it is thought that tooth movement in the direction of the cortical bone causes alveolar bone resorption and decrease in bone thickness.

Yoonji et al.[14] in their study, they emphasized the need to reconsider excessive orthodontic movement, especially in skeletal class III adult patients, according to the patient's anatomical boundaries and periodontal health. As seen in our study, lower anterior bone thickness decreased more in class III patients than in class II patients. Our findings are consistent with Yoonji et al.[16] it is similar to their study.

Wehrbein et al.[25] they suggested that significant sagittal incisor movement and rotation are critical risk factors for progressive lingual and labial bone loss in patients with class III anomalies with narrow symphysis and increased vertical direction growth. The study supports our study by finding that lower anterior bone thickness decreased more in class III patients than in class II patients.

Jäger et al.[26] showed in their study that a change in tooth position changes the thickness of the labial and lingual cortical plates at the level of the root apex. As a result of our measurements, a decrease in root length was detected in maxillary teeth. It was observed that the upper incisor buccal and palatal bone levels were more resorbed than the root. The ratio of upper buccal and palatal bone thickness to cementum-enamel junction has decreased. These data support the postoperative changes in terms of UPKK (upper palatal bone thickness), (UABT+UPBT) CEJW (upper anterior bone thickness+upper palatal bone thickness/cement enamel junction width) values. In our study, it was observed that bone loss occurred in the upper incisor buccal region, and bone thickness decreased in the upper incisor palatal region.

Sun et al.[27] in their study, it was reported that the labial inclination of the mandibular incisors showed a positive correlation with the labial and total alveolar bone thickness and a negative correlation with the lingual alveolar bone height. The finding of a moderate positive correlation between lower mesiobuccal alveolar height and L1-mandibular plane in our study supports this finding.

Another CBCT study reported alveolar bone loss around the incisors in skeletal class III patients treated with orthognathic surgery. In the study, it was observed that the vertical alveolar bone level decreased more in the mandibular incisors compared to the maxillary incisors, especially on the lingual side.[16] In our findings, the lower jaw lingual bone level parameter decreased more than the upper jaw palatal bone level parameter. The findings are parallel to the study.

In the treatment of class III patients with mandibular prognathism, particular attention should be paid to the alveolar bone around the mandibular anterior teeth. Especially in extraction cases, mandibular anterior teeth are more vulnerable to bone defects during retraction compared to maxillary anterior teeth.[28] Therefore, it is also very important to evaluate and identify clinical factors associated with changes in alveolar bone dimensions in orthodontic decompensation before surgery. However, in skeletal class III patients with thin mandibular symphysis and increased vertical height, even a small amount of periodontal inflammation may pose a risk of bone loss and destruction.[29]

Guo et al.[29] emphasized in their study that the alveolar bone level, especially in the mandibular incisors, should be considered specifically in skeletal class III patients.

It should not be forgotten that orthognathic surgery procedures are performed on adult patients. In our study, patients with bone loss from both maxillary (upper anterior bone level, upper palatal bone thickness, UABL/root, UPBL/root, (UABL+UPBL)/CEJW, upper trifurcation buccal, upper DBR middle buccal $p<0.05$) and mandibular (lower anterior bone level), lower root length, AAKS/root, ALKS/root, (AAKK+ALKK)/MSBG, lower bifurcation buccal, lower DK middle buccal $p<0.05$) are evaluated. Being an adult patient, slowing down of bone regeneration or even having a risk of residual bone degeneration, and the possibility of an underlying systemic disease such as osteoporosis, osteopenia, vitamin D/calcium deficiency, menopause or pregnancy, and bisphosphonate use should be taken into consideration during the treatment planning phase.

Santos et al.[30] found in their study that alveolar bone dehiscence increased significantly only on the lingual side of the lower first molar in class II group patients. Parallel to this, in our study, lower 1st molar distal root middle lingual bone measurement changed more in patients who underwent double jaw surgery than in patients who underwent single upper jaw surgery. All 5 class II patients in our study underwent mandibular advancement surgery. The observation of bone loss on the lingual side of the lower molar teeth in operations involving the lower jaw is supported by the study.

Bondemark[31] found that the average alveolar bone loss per patient in adolescents who received orthodontic treatment for 2 years during a 5-year observation period varied between 0.1 and 0.5 mm. In our study based on the data obtained, the average bone loss was found to be 0.45 mm at the upper buccal level and 0.35 mm at the upper palatal level, while the lower incisor region was found to be 1.45 mm at the buccal level and 0.69 at the lingual level.

Nelson et al.[12] also did not find a relationship between maxillary osteotomy and lower jaw bone loss as a result of their study. The findings support our findings in that the change in the lower anterior bone level in our study, when comparing the patients who had only lower and only upper jaw surgery, changed more in patients who had only lower jaw surgery than in patients who had only upper jaw surgery, and that the maxilla was not affected statistically significantly by the surgery performed on the mandible.

CONCLUSION

Every surgical procedure carries an element of risk. However, the risks and potential morbidity of orthognathic treatment are relatively low and generally short-term.

Considering the patient's age, systemic status, surgery-related factors, dental and anatomical differences; it is not possible to make a definitive diagnosis regarding the increase or decrease of complications in orthognathic surgery depending on gender. Specific studies in this field should be increased and patient profiles in different subcategories should be diversified.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was carried out with the permission of the Local Ethics Committee of Dicle University Faculty of Dentistry Deanery (Date: 27.01.2021, Decision No: 2021-06).

Informed Consent

All patients signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Lye KW. Effect of orthognathic surgery on the posterior airway space (PAS). *Ann Acad Med Singap*. 2008;37(8):677-682.
2. Te Malik NA. Presurgical Orthodontic Phase. In: textbook of oral and maxillofacial surgery. *Fourth.* 2016,421-430.
3. Westermark A, Shayeghi F, Thor A. Temporomandibular dysfunction in 1,516 patients before and after orthognathic surgery. *Int J Adult Orthodon Orthognath Surg.* 2001;16(2):145-151.
4. Helm S, Petersen PE. Causal relation between malocclusion and periodontal health. *Acta Odontol Scand.* 1989;47(4):223-228.
5. Posnick JC. Definition and prevalence of dentofacial deformities. Orthognathic surgery: principles and practice. *St. Louis, MO: Elsevier.* 2014,61-68.
6. Naini FB, Gill DS. Foreword. Orthognathic surgery: principles, planning and practice. *John Wiley & Sons.* 2017,19-20.
7. Garg S, Kaur S. Evaluation of post-operative complication rate of le fort ı osteotomy: a retrospective and prospective study. *J Maxillofac Oral Surg.* 2014;13(2):120-127.
8. Panula K, Finne K, Oikarinen K. Incidence of complications and problems related to orthognathic surgery: a review of 655 patients. *J Oral Maxillofac Surg.* 2001;59(10):1128-1136.
9. Nicodemo D, Ferreira LM. Questionnaire of the psychosocial profile of the patient with anophthalmia with indication of ocular prosthesis. *Arq Bras Oftalmol.* 2006;69(4):463-470.
10. Sousa CS, Turrini RN. Complications in orthognathic surgery: a comprehensive review. *J Oral Maxillofac Surg.* 2012;24(2):67-74.
11. Lupi JE, Handelman CS, Sadowsky C. Prevalence and severity of apical root resorption and alveolar bone loss in orthodontically treated adults. *Am J Orthod Dentofacial Orthop.* 1996;109(1):28-37.
12. Nelson PA, Artun J. Alveolar bone loss of maxillary anterior teeth in adult orthodontic patients. *Am J Orthod Dentofacial Orthop.* 1997;111(3):328-334.
13. Steiner GG, Pearson JK, Ainamo J. Changes of the marginal periodontium as a result of labial tooth movement in monkeys. *J Periodontol.* 1981;52(6):314-320.
14. Choi YJ, Chung CJ, Kim KH. Periodontal consequences of mandibular incisor proclination during presurgical orthodontic treatment in class III malocclusion patients. *Angle Orthod.* 2015; 85(3):427-433.
15. Nicodemo D, Pereira MD, Ferreira LM. Effect of orthognathic surgery for class III correction on quality of life as measured by SF-36. *Int J Oral Maxillofac Surg.* 2008;37(2):131-134.
16. Kim Y, Park JU, Kook YA. Alveolar bone loss around incisors in surgical skeletal class III patients. *Angle Orthod.* 2009;79(4):676-682.
17. Bholsithi W, Tharanon W, Chintakanon K, Komolpis R, Sinthanayothin C. 3D vs 2D cephalometric analysis comparisons with repeated measurements from 20 thai males and 20 thai females. *Biomed Imaging Interv J.* 2009;5(4):e21.
18. Garib DG, Yatabe MS, Ozawa TO, da Silva Filho OG. Alveolar bone morphology under the perspective of the computed tomography: defining the biological limits of tooth movement. *Dental Press J Orthod.* 2010;15:192-205.
19. Lee S, Hwang S, Jang W, Choi YJ, Chung CJ, Kim KH. Assessment of lower incisor alveolar bone width using cone-beam computed tomography images in skeletal class III adults of different vertical patterns. *Korean J Orthod.* 2018;48(6):349-356.
20. Kim Y, Park JU, Kook YA. Alveolar bone loss around incisors in surgical skeletal class III patients. *Angle Orthod.* 2009;79(4):676-682.
21. Vardimon AD, Oren E, Ben-Bassat Y. Cortical bone remodeling/tooth movement ratio during maxillary incisor retraction with tip versus torque movements. *Am J Orthod Dentofacial Orthop.* 1998;114(5):520-529.
22. Wainwright WM. Faciolingual tooth movement: its influence on the root and cortical plate. *Am J Orthod.* 1973;64(3):278-302.
23. Gaitan-Romero L, Shujaat S, Ma H, et al. Evaluation of long-term hard tissue relapse following surgical-orthodontic treatment in skeletal class II patients: a systematic review and meta-analysis. *Int J Oral Maxillofac Surg.* 2021;50(4):477-486.
24. Lannetti G, Fadda TM, Riccardi E, Mitro V, Filiaci F. Our experience in complications of orthognathic surgery: a retrospective study on 3236 patients. *Eur Rev Med Pharmacol Sci.* 2013;17(3):379-384.
25. Wehrbein H, Bauer W, Diedrich P. Mandibular incisors, alveolar bone, and symphysis after orthodontic treatment. A retrospective study. *Am J Orthod Dentofacial Orthop.* 1996;110(3):239-246.
26. Jäger F, Mah JK, Bumann A. Peridental bone changes after orthodontic tooth movement with fixed appliances: a cone-beam computed tomographic study. *Angle Orthod.* 2017;87(5):672-680.
27. Sun B, Tang J, Xiao P, Ding Y. Presurgical orthodontic decompensation alters alveolar bone condition around mandibular incisors in adults with skeletal class III malocclusion. *Int J Clin Exp Med.* 2015;8(8):12866-12873.
28. Zhang G. Influence of orthodontic tooth movement on alveolar bone morphology and bone mineral density. *J Tissue Eng.* 2015; 19(21):3440-3444.
29. Guo R, Zhang L, Hu M, Huang Y, Li W. Alveolar bone changes in maxillary and mandibular anterior teeth during orthodontic treatment: a systematic review and meta-analysis. *Orthod Craniofac Res.* 2020;24(2):165-179.
30. Dos Santos MC, Iwaki LCV, Valladares-Neto J, Inoue-Arai MS, Ramos AL. Impact of orthognathic surgery on the prevalence of dehiscence in class II and class III surgical-orthodontic patients. *Angle Orthod.* 2021;91(5):611-618.
31. Bondemark L. Interdental bone changes after orthodontic treatment: a 5-year longitudinal study. *Am J Orthod Dentofacial Orthop.* 1998;114(1):25-31.

Original Article *Dicle Dent J.* 2024;25(2):38-45

Investigation of the effect of denosumab and ozone application on bone healing in critical size bone defects

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ABSTRACT

Aims: In maxillofacial surgery, various drugs are used in order to accelerate the recovery of defects caused by any reason. Several studies have shown that denosumab from bisphosphonate group drugs used in osteoporosis patients has positive effects on new bone formation. In general medicine and dentistry, ozone is in widespread use as an alternative treatment and has positive effects on new bone formation. The aim of this study was to evaluate new bone formation by performing denosumab and ozone on critical-sized rat calvarium defects.

Methods: In our study, 40 Sprague Dawley rats were used. Rats were divided into 4 groups. Only grafts were placed in the control group. After applying graft to ozone group (O) and ozone and denosumab (O-D) groups, topical ozone was applied for 15 seconds. Denosumab group (D) and O-D group were injected subcutaneously (s.c) 10 mg/kg Prolia (denosumab) every 4 weeks for 8 weeks. 5 animals from each group at the end of week 4, while the other five animals in the group at the end of 8 weeks after being sacrificed for histopathological examination was performed. The differences between the groups were evaluated by statistical analysis.

Results: After histopathological examination, better bone formation was observed in the ozone and denosumab treated groups compared to the control group. There was no statistically significant difference between the groups except for the control group, however, new bone formation was determined in the groups treated with denosumab compared to the ozone group.

Conclusion: As a result of our study, we believe that the application of ozone and denosumab has a positive effect on the formation of new bone, but more comprehensive studies on the subject are needed.

Keywords: Rat, ozone, denosumab, new bone formation

INTRODUCTION

In oral and maxillofacial surgery, healing and bone formation in the operating area have been one of the most emphasized issues. Today, autogenous grafting is considered and used as the gold standard in bone healing. Although autogenous grafts are the gold standard, different graft materials (allografts, xenografts, alloplastic graft) can be used due to their disadvantages such as creating a second wound area, the risk of bone graft resorption, the risk of nerve injuries and the risk of infection.[1]

Ozone (O_3) is a natural compound consisting of three oxygen atoms. Ozone is not a radical molecule due to its chemical structure, but it is the third most powerful oxidant known. Since ozone cannot be stored and has a half-life of 40 minutes, it is an unstable gas that must be used all at once. O_3 has been frequently used in dentistry and oral surgery since 1993 due to its antimicrobial properties, analgesic effect, and regulatory effects on microcirculation and peripheral blood circulation.[2]

Bone tissue is considered one of the hardest tissues in our body. If bones are exposed to trauma, their repair abilities are highly developed, and therefore, new bone tissue is obtained in the affected area and the functions of the relevant area are restored.[3,4]

The disruption of the anatomical integrity of the bone due to external or internal forces is called a fracture. A number of physiological reactions begin to restore the damaged bone integrity. In bone healing, scar tissue does not form and it heals through restructuring. Fracture healing begins from the moment the fracture occurs and continues until the regular

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bone tissue and the fracture ends unite.[5] If fibrous tissue forms after the bone tissue heals, this indicates that the fracture has not healed.[6]

Denosumab is a recently FDA-approved monoclonal antibody for the treatment of osteoporosis in postmenopausal women at high risk of fracture.[7] It is also being evaluated for the prevention of osteoporosis in postmenopausal women, as well as for the treatment and prevention of bone loss in patients undergoing hormone ablation therapy for breast and prostate cancer.[8,9] The aim of this study was to evaluate new bone formation by performing denosumab and ozone on critical-sized rat calvarium defects.

METHODS

This study was supported by Dicle University Scientific Research Projects with project number DİŞ17.022. Ethics committee approval for our thesis study was received from Dicle University Animal Experiments Local Ethics Committee (Date: 30.05.2017, Decision No: 2017/09). The experimental stages of our study were carried out at Dicle University Prof. Dr. Sabahattin Payzın Health Sciences Research Center, and histopathological examinations were carried out at Dicle University Faculty of Medicine, Department of Histology and Embryology. This study was conducted on critical-sized bone defects opened experimentally in the rat calvarium.

In our study, 40 Sprague-Dawley male rats with an average weight of 350 grams were used. Sprague-Dawley rats aged 6 months were used in the study. During the experiment, all subjects were housed in special plastic cages in ventilated rooms with 12 hours of light and 12 hours of darkness, at a temperature of 24 degrees, free access to drinking water and standard food. Before starting our study, the health status of all subjects was checked by a veterinarian.

Study Groups Experimental animals were divided into 4 groups, with 10 rats in each group

Group 1 [Control Group (C)]: Only graft was applied to the rats in this group.

Group 2 [Ozone Group (O)]: Topical ozone and graft application was applied to the rats in this group.

Group 3 [Denosumab Group (D)]: Subcutaneous denosumab was given to the rats in this group and graft was applied.

Group 4 [Ozone and Denosumab Group (O+D)]: Topical ozone, subcutaneous denosumab and graft were applied to the rats in this group.

Graft was applied to the defect area of all rats in the study. Control group; The defect was opened only on the calvarium with a trephine drill and the graft was applied. In the ozone group; Topical ozone was applied to the rats in this group after the graft was placed in the defect area. Denosumab Group; Rats in this group were injected subcutaneous denosumab after placing the graft in the defect area. In the ozone and denosumab group; After the graft was placed in the defect area, topical ozone was applied to the rats in this group and subcutaneous denosumab was injected. 5 animals from each group were sacrificed at the end of the 4th week, and the other 5 animals in each group were sacrificed at the end of the 8th week. The removed tissues were stored in fixative solution and examined histopathologically.

Surgery Prodecure

General anesthesia was achieved by intramuscular injection using Ketamine (Ketalar®, Pfizer, Turkiye) 50 mg/kg and Xylazine (Rompun®, Bayer, Turkiye) 10 mg/kg in the anesthesia applied for surgical procedures in all rats. Asepsis and antisepsis criteria were complied with during surgical procedures in all subjects. All instruments to be used were sterilized before the operation. After the calvarial area of all subjects was shaved, they were stained with povidone-iodine and covered with a sterile cover, leaving the operation area exposed. To reach the calvarium, approximately 24 mm long skin and subcutaneous incisions were made in the anterior-posterior direction. By blunt dissection, the outer surface of the calvarium was reached by removing the soft tissues and stripping the periosteum. The defect was prepared with a 5 mm diameter trephine bur attached to a contra-angle handpiece under physiological saline cooling. Bovine xenograft (Hypro-Oss, Bioimplon GmbH, Gießen, Germany) was placed in the defect area. Topical ozone (W&H Prozone Ozone generator, Bürmoos, Austria) was applied for 15 seconds after the graft was applied to the ozone group (O) and ozone+denosumab (O+D) groups. Denosumab group (D) and ozone and denosumab (O-D) groups were injected subcutaneously (s.c) with 10 mg/kg Prolia (ready-to-use injector containing Denosumab 60 mg SC solution for 23 injections, Amgen, Turkiye) every 4 weeks for 8 weeks. After the operation, the working area was closed primarily with 3/0 silk suture. For infection control, 30 mg/kg Cefazolin was administered intramuscularly (i.m.) post-operatively. Subjects were taken to the recovery room in metal cages. In the post-operative period, 30 mg/kg Cefazol (i.m) and Carprofen 4 mg/kg subcutaneously were administered to the subjects who were fed orally, once a day, for the first five days after the surgery.

Histopathological Analyzes

In each group, half were sacrificed in the 4th week and the other half in the 8th week. At the end of the 4th and 8th weeks, the rats were given high doses of anesthesia and euthanized using the cervical blockade method. For histopathological analyses, the calvarial bone was resected, the calvarium sample of each subject was fixed in Zinc-Formalin for at least 8 hours, and following the fixation process, it was decalcified with 15% nitric acid solution for 72 hours. These fixed and decalcified samples were embedded in paraffin and sections with an average thickness of 4-5 µm were taken from the paraffin blocks of each sample. H-E, Masson Trichrome and Mallory-Azan staining methods were used as histochemical staining methods. The samples were scored and statistical analysis was performed considering the criteria of osteoblastic activity, bone trabeculae, collagen fiber distribution, connective tissue cells and osteocyte cells.

Semi-Quantitative Histologic Scoring

Scoring was performed using a 0 to 3 scoring system (0, none; 1, minimal; 2, moderate; 3, abundant).

Statistical Analysis

The data obtained in this study were analyzed with the IBM SPSS Statistics Version 22 package program. While investigating whether the variables came from a normal distribution, Shapiro Wilk's was used due to the number of units. While examining the differences between the groups, the Kruskal Wallis-H test was used because the variables did not come from a normal distribution. If significant differences were seen in the Kruskal Wallis-H test, groups with differences were determined with the PostHoc multiple comparison test. When examining the difference between two dependent variables, the Wilcoxon test was used because the variables did not come from a normal distribution. The significance level was set as $p<0.05$.

RESULTS

In our research, a total of 40 Sprague-Dawley adult male rats with an average weight of 350 g were used, 10 in each group. Our study consisted of 4 groups: control group (C), ozone (O), denosumab (D), ozone and denosumab (OD), and 5 animals from each group were sacrificed at the end of the 4th week, and the other 5 animals in the group were sacrificed at the end of the 8th week. The removed tissues were subjected to histomorphological examination in fixative solution. During the experimental study, it was observed that the rats tolerated the surgical procedure well, there were no adverse effects in terms of their nutrition, no infection occurred due to the operation, and the general health of the subjects was good.

Histopathological Findings (Week 4)

1. Control group: In the 4th week, intense mononuclear cell infiltration was observed in the grafted area of the control group calvarial bone. In the graft area, small bone trabeculae with increased matrix, an increase in osteoblast cells, and a development in the direction of mature bone cells were observed. Thickening and irregular distribution of collagen fibers were observed (Figure 1).

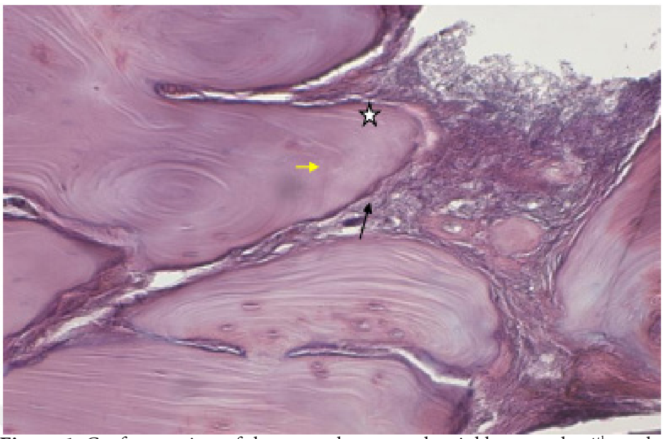

Figure 1. Graft area view of the control group calvarial bone at the 4th week. Dense mononuclear cell infiltration (⁎), small bone trabeculae (black →), Osteoblast cells (yellow →) (Staining: Hematoxylin-eosin (H-E), bar: 50µ

2. Ozone group: In the 4th week ozone group, in the area where the calvarial bone graft was applied, maturation of bone trabeculae and concentration in the matrix, an increase in osteoblastic activity around the bone trabeculae, and a prominence in mature osteocyte cells were observed. Within the graft area, an increase in normal connective tissue cells, hyperplasia in fibroblast cells, a decrease in inflammatory cells, thickening and irregular distribution in collagen fibers were observed (Figure 2).

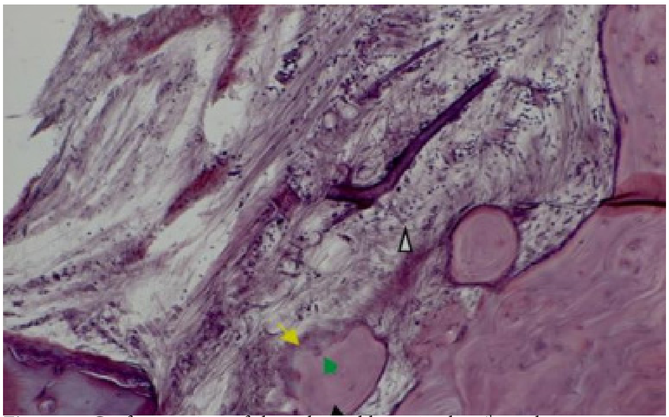

Figure 2. Graft area view of the calvarial bone in the 4th week ozone group. Increase in osteoblastic activity (yellow →) around the bone trabeculae (black →), prominence in osteocytecells (green →), increase in connective tissue cells (Δ) (Staining: Hematoxylin-eosin (H-E), bar: 50µ)

3. Denosumab group: In the 4th week denosumab group, in the area where the calvarial bone graft was applied, expansion of the bone trabeculae was observed due to the increase in matrix in the bone trabeculae, an increase in osteoblast cells in the periphery of the bone trabeculae, and a prominence in the bone lacunae. It was observed that there was a thickening of collagen fibers in the graft area and connective tissue cells with high mitotic activity due to a decrease in cell infiltration. It was observed that bone development accelerated and the number of bone cells increased due to the effect of the drug (Figure 3).

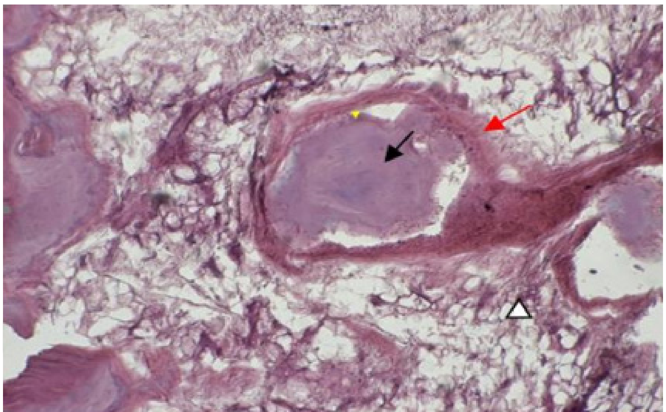

Figure 3. View of the graft area of the denosumab group calvarial bone at the 4th week. Expanded bone trabeculae (black →), dense collagen bands around the bone trabeculae (red →), increase in osteoblast cells (yellow →), significant increase in connective tissue cells (Δ) (Staining: Hematoxylin-eosin (H-E), bar: 50µ)

4. Ozone and Denosumab group: In the 4th week, in the ozone and denosumab group, it was observed that in the area where the calvarial bone graft was applied, collagen fiber synthesis increased, thickening of the fibers became evident, hypertrophy in fibroblast cells and an increase in connective tissue cells. Increased bone matrix development, hyperplasia of osteoblast cells in the periphery, and prominence of osteocyte cells within the lacuna were observed. It has been observed that new bone development accelerates further and there is a significant increase in the number of mature bone cells due to drug and ozone application (Figure 4).

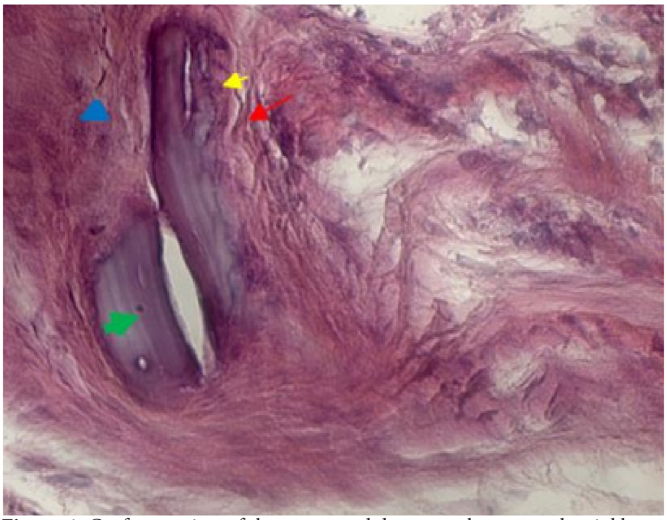

Figure 4. Graft area view of the ozone and denosumab group calvarial bone at the 4th week. Increase in collagen fiber synthesis (red →), prominence in osteocyte cells within the lacuna (green →), hypertrophy in fibroblast cells (blue →), increase in osteoblast cells (yellow →) (Staining: Hematoxylin -Eosin (H-E), bar: 50µ)

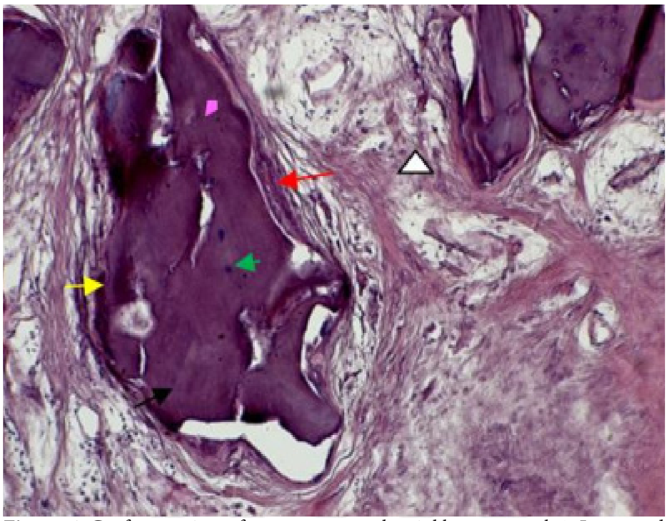

Figure 6. Graft area view of ozone group calvarial bone at week 8. Increased bone trabeculae (black →), dense collagen bands (red →), connective tissue cells (Δ), Osteocyte cells (green →), Osteoblast cells (yellow →), Osteon (pink →) (Staining: Hematoxylin-eosin (H-E), bar: 50µ)

Histopathological Findings (Week 8)

1.Control group: In the area where the calvarial bone graft was applied in the control group at the 8th week, the bone trabeculae in the graft area expanded due to the increase in osteoblastic activity, the amount of matrix increased, and the amount of osteocyte cells began to increase with the lacunae becoming evident in some places. It was observed that collagen bands were quite thick and connective tissue cells were diffusely increased (Figure 5).

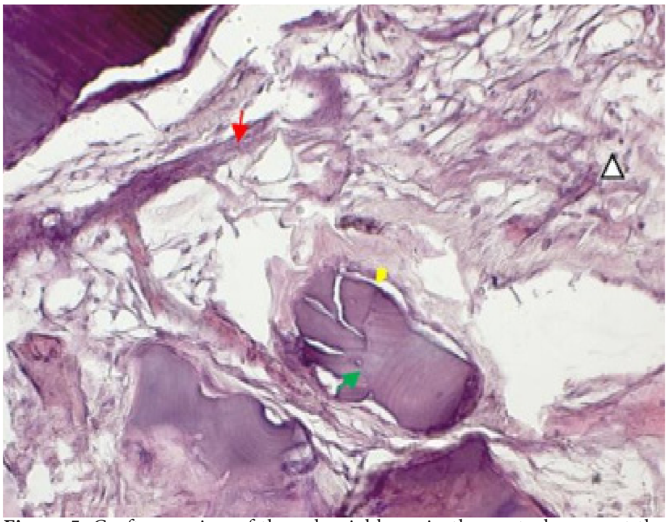

Figure 5. Graft area view of the calvarial bone in the control group at the 8th week. Increase in osteoblastic activity (yellow →) Increase in the number of osteocytes (green →), collagen fibers in the form of thick bands (red →), significant increase in connective tissue cells (Δ) (Staining: Hematoxylin-eosin (H-E), bar: 50µ)

2. Ozone group: In the 8th week ozone group, in the grafted area of the calvarial bone, bone trabeculae of varying sizes that had completed bone development were observed within the graft area. An increase in osteoblastic activity, prominence in osteocyte cells and an increase in the number of osteon structures were observed within these trabeculae. It was determined that collagen bands around the trabeculae in the graft area were extremely concentrated and the number of connective tissue cells increased (Figure 6).

3. Denosumab group: In the 8th week denosumab group, in the area where the calvarial bone graft was applied, a significant expansion of the bone trabeculae, a significant increase in osteoblastic activity in the periphery, an increased number of osteocyte cells and osteon structures located in the lacuna began to appear. In the graft area, it was observed that collagen fibers were formed in the form of tight and thick bands and there was an intense increase in connective tissue cells between them. New bone formation took shape (Figure 7).

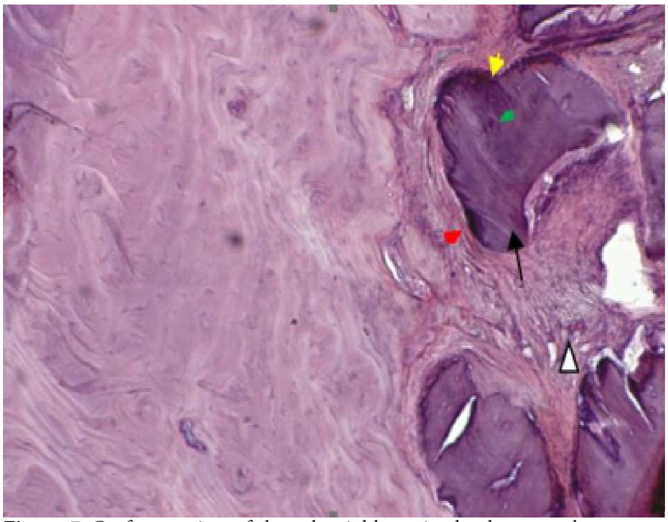

Figure 7. Graft area view of the calvarial bone in the denosumab group at the 8th week. Expanded bone trabeculae (black →), dense collagen bands (red →), connective tissue cells (Δ), Osteocyte cells (green →), Osteoblast cells (yellow →), (Staining: Hematoxylin-eosin(H-E), bar: 50µ)

4. Ozone and Denosumab group: In the 8th week, in the ozone+denosumab group, all elements suitable for new bone formation began to form in the expanded bone trabeculae in the area where the calvarial bone graft was applied. A significant hypertrophy of osteoblastic cells and an increased number of osteocyte cells settled in the lacuna were observed. Although osteon structures appear distinct, bone marrow has begun to form within the trabecular areas. Collagen fibers were arranged in thick and tight bands around the trabeculae, and connective tissue cells increased in clusters. All findings regarding new bone formation were seen as positive in this group (Figure 8).

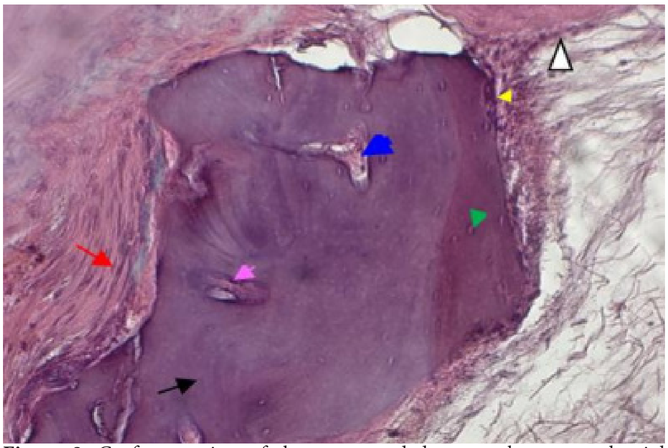

Figure 8. Graft area view of the ozone and denosumab group calvarial bone at the 8th week. Expanded bone trabeculae (black →), collagen fibers in the form of thick and tight bands (red →), increased connective tissue cells (Δ), a large number of increased osteocyte cells (green →) and Osteoblast cells (yellow →), prominent Osteon (pink →), formed bone marrow (blue →) (Staining: Hematoxylin-eosin (H-E), bar: 50µ)

There is a statistically significant difference between the groups in terms of bone formation 4th week scores ($p<0.05$). The bone formation score of the control group (K) at week 4 was significantly lower than the ozone and denosumab group (O+D) (Table1) (Figure 9).

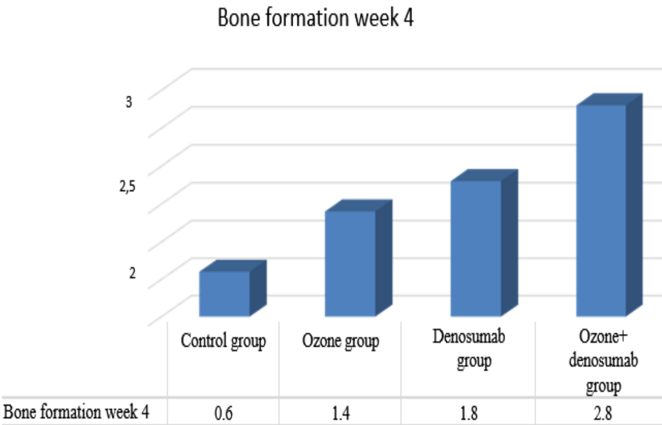

Figure 9. Scatter plot of bone formation week 4 scores by groups

There is a statistically significant difference between the groups in terms of bone formation 8th week scores ($p<0.05$). The bone formation 8th week score of the control group is significantly lower than the ozone and denosumab group (Figure 10) (Table 2).

There is statistically significant difference between the 4th week and 8th week findings in terms of bone formation scores in the control group ($p<0.05$). In the control group, the bone formation score at week 4 was significantly lower than the score at week 8. There is a statistically significant difference between the findings of the 4th week and the 8th week in terms of bone formation scores in the ozone group ($p<0.05$). In the ozone group, the bone formation score at week 4 was significantly lower than the score at week 8. There is a statistically significant difference between the 4th week and 8th week findings in terms of bone formation scores in the denosumab group ($p<0.05$). In the denosumab group, the bone formation score at week 4 was significantly lower than the score at week 8. There is a statistically significant difference between the 4th week and 8th week findings in terms of bone formation scores in the ozone and denosumab groups ($p<0.05$). In the ozone and denosumab group, the bone formation score at week 4 was significantly lower than the score at week 8 (Table 3) (Figure 11).

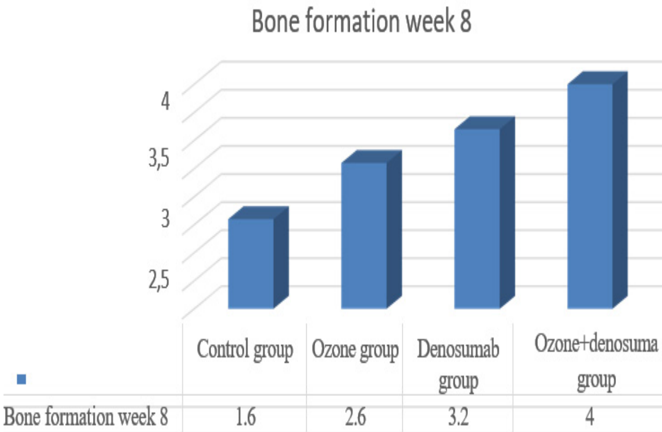

Figure 10. Scatter plot of bone formation week 8 scores by groups

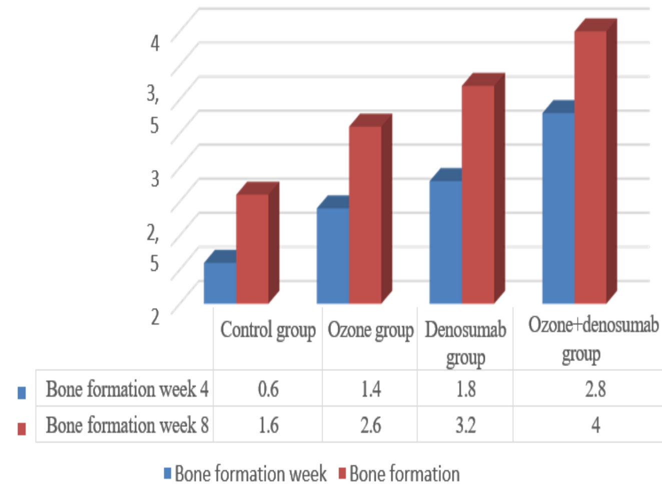

Figure 11. Scatter plot of bone formation week 4 and week 8 scores within the group

Table 1. Kruskal Wallis h test result regarding the difference between groups in terms of bone formation 4th week scores

		Group						Kruskal Wallis h Test		
		n	Mean	Median	Min	Max	SD	Mean Rank	h	p
Bone formation week 4	Control group	5	0.6	1	0	1	0.55	4.2	14.388	0.002
	Ozone group	5	1.4	1	1	2	0.55	8.8		
	Denosumab group	5	1.8	2	1	2	0.45	11.6		
	Ozone-denosumab group	5	2.8	3	2	3	0.45	17.4		
	Total	20	1.65	2	0	3	0.93		1-4	

Min: Minimum, Max: Maximum, SD: Standart deviation

Table 2. Kruskal Wallis h test result regarding the difference between groups in terms of bone formation 8th week scores

		Group						Kruskal Wallis h Test		
		n	Mean	Median	Min	Max	SD	Mean Rank	h	p
Bone formation week 8	Control group	5	1.6	2	1	2	0.55	3.6	16.139	0.001
	Ozone group	5	2.6	3	2	3	0.55	8.6		
	Denosumab gruop	5	3.2	3	3	4	0.45	12.3		
	Ozone+denosumab group	5	4	4	4	4	0	17.5		
	Total	20	2.85	3	1	4	0.99		1-4	

Min: Minimum, Max: Maximum, SD: Standart deviation

Table 3. Wilcoxon test result for the difference between bone formation 4th week and 8th week scores within the group

		n	Mean	Median	Min	Max	SD	Wilcoxon Test Mean Rank	z	p
Control group	Bone formation week 4	5	0.6	1	0	1	0.55	0	-2.236	0.025
	Bone formation week 8	5	1.6	2	1	2	0.55	3		
Ozone group	Bone formation week 4	5	1.4	1	1	2	0.55	0	-2.121	0.034
	Bone formation week 8	5	2.6	3	2	3	0.55	3		
Denosumab group	Bone formation week 4	5	1.8	2	1	2	0.45	0	-2.07	0.038
	Bone formation week 8	5	3.2	3	3	4	0.45	3		
Ozon+denosumab group	Bone formation week 4	5	2.8	3	2	3	0.45	0	-2.121	0.034
	Bone formation week 8	5	4	4	4	4	0	3		

Min: Minimum, Max: Maximum, SD: Standart deviation

DISCUSSION

One of the most important areas of maxillofacial surgery is the restoration of bone volume lost for any reason. Since 1950, the use of modern medicine as well as regenerative and complementary medicine approaches on some disease groups has increased significantly. Ozone therapy, one of the popular applications of regenerative and complementary medicine, has been applied in private treatment centers in our country for many years. Ozone is a naturally occurring compound containing three oxygen atoms. It is normally found in the atmosphere and filters out the sun's harmful ultraviolet rays. Ozone therapy is the application of an ozone/oxygen mixture to the circulation or body cavities. Ozone therapy has been applied in medicine and dentistry for many years. Ozone has various effects such as antimicrobial, antihypoxic, immunomodulatory, biosynthetic and analgesic.[10]

Most published articles considering the use of ozone in dentistry concern the antimicrobial effects of ozone.[11-14] Additionally, there is not enough evidence for ozone application in oral and maxillofacial surgery.[15] We chose to use ozone in our thesis due to its therapeutic effect, which facilitates wound healing and improves blood flow.[16,17]

Various animal species are used in thesis studies in the field of maxillofacial surgery. When we look at the literature, animal studies conducted with denosumab were mostly found in rat, mouse, rabbit and monkey species.[18-21] We chose to use rats in our study in accordance with the literature.[18-21]

Denosumab is a new agent with antiresorptive potential used in the treatment of osteoporosis, which has been proven to increase bone mass, microarchitecture and durability in human and animal studies. The drug developed against RANKL (receptor activator of nuclear factor-κB-ligand) is a human monoclonal antibody. Besides these; It also acts to inhibit the differentiation and function of osteoclasts. Studies have reported that denosumab application has an antiresorptive effect on bone remodeling.[22] Denosumab acts as a decoy receptor by binding to RANKL, preventing it from binding to RANK. When the RANKL/RANK connection is blocked, the differentiation of preosteoclasts into mature osteoclasts, their activation and survival are inhibited, and RANKL cannot produce bone resorption. Thus, it reduces resorption in cortical and trabecular bone. Denosumab drug shows its antiresorptive feature by blocking the life and production of osteoclasts that cause bone destruction. It also reduces bone turnover and increases bone mineral denalsity.[23]

Alpan et al.[24] they placed a xenograft into the calvarial defect they created in diabetic rats. And they examined the effects of ozone on bone regeneration. According to the results of the study, it was determined that ozone applied in gaseous form increased xenograft resorption and accelerated early bone healing in diabetic rats.[24] In our study, it was determined that when ozone and denosumab were used together, they showed a synergistic effect and provided better results in new bone formation.

Buyuk et al.[25] examined the effects of ozone at various concentrations on bone healing in their study with 48 rats. The rats were divided into 4 groups, 10, 25, 40 µg/ml ozone was injected into the expansion area made in the premaxillary suture, and 1 ml saline solution was injected into the control group. Bone regeneration in the suture area was examined histomorphometrically and new bone area, fibrotic area, osteoblast, osteoclast number and vascularization were examined. All parameters examined were found to be significantly higher in the experimental groups than in the control group. It was reported that the values in the 25 µg/ml ozone applied group were higher than both the other experimental groups and the control group.[25] In our study, it was observed that ozone application alone increased new bone formation in the reconstruction of bone defects.

Ominsky et al.[19] they investigated the effect of denosumab on ovariectomized monkeys. They reported that denosumab reduced biological markers of bone remodeling and increased cortical and trabecular bone mass. They reported that denosumab increases bone etstrength by preserving bone quality and increasing bone mass.[19] In our study, it was observed that denosumab, which has antiresorptive properties in the repair of bone defects, increased new bone formation both alone and in combination with ozone.

Kostenuik et al.[21] reported that, as a result of their study, they detected an increase in both trabecular and cortical bone mass in monkeys treated with denasumab. In our study, when the control and denosumab groups were compared, it was observed that the drug denosumab significantly increased the formation of new bone area.

Gerstenfeld et al.[26] in their study, they investigated the effects of alendronate and denosumab on fracture healing in a mouse model. A fracture line was created in the femur region and denosumab (10 mg/kg) and alendronate (0.1 mg/kg) were injected in groups. They found that the bone volume at the fracture line was higher and the bone structures were harder and more durable in the denosumab-treated group.[26] The results obtained from our study are in line with the literature.

Bernhardsson et al.[27] they investigated the effect of denosumab on screw fixation in their study on rats. They found that the drug denosumab increased bone density more than alendronate. Additionally, they reported that denosumab increased screw fixation in cancellous bone more than bisphosphonate.[27] In our study, it was observed that the application of denosumab, which has antiresorptive properties, in the repair of bone defects, increased new bone formation both alone and in combination with ozone.

CONCLUSION

When the findings obtained as a result of our study were evaluated, the following conclusions were reached:

- It has been observed that ozone application alone increases new bone formation in the reconstruction of bone defects.
- It has been observed that the application of denosumab, which has antiresorptive properties in the repair of bone defects, increases new bone formation when used alone.
- It was determined that when ozone and denosumab were used together, they showed a synergistic effect and gave better results in new bone formation.
- There are many different studies in the literature showing that ozone has a positive effect on new bone formation. The results in our study are similar to other studies.
- In line with the results, we think that denosumab application can be used in operations to increase bone volume. Studies investigating long-term results can be conducted on this subject. On the other hand, it was deemed necessary to evaluate this treatment in terms of dose and duration with animal and clinical studies involving larger experimental groups before clinical applications.
- When we look at the results of our thesis, we think that it will benefit clinical studies, but it should be supported by additional studies.

REFERENCES

1. Guler R, Dundar S, Bozoglan A, et al. The biomechanical investigation of osseointegration levels in titanium implants simultaniously placed with different bone grafts. *Niger J Clin Pract.* 2024;27:310-316.
2. Korkmaz H, Küçükkolbaşı H. Ozone applications in dentistry. *J Dent Fac Ataturk Uni.* 2013;7:125-134.
3. Gartner LP, Hiatt James I. Color Textbook of Histology 3rd edition. Saunders Elsevier, 2007,114-130.
4. Schenk Robert K. Biology of fracture. In: Browner B, Jupiter J, Levine A, Trafton P (Eds) Skeletal Trauma. *Saunders, Philadelphia.* 2003;1:29-74.
5. Kılıçoğlu SS. Fracture healing on microscobic level. *Ank Üni Tıp Fak Derg.* 2002;55(2):143-150.
6. ElHawary H, Baradaran A, Abi-Rafeh J, Vorstenbosch J, Xu L, Ionut Efanov J. Bone healing and inflammation: principles of fracture and repair. *Semin Plast Surg.* 2021; 35(03):198-203. doi: 10.1055/s-0041-1732334
7. Kendler DL, Cosman F, Stad RK, Ferrari S. Denosumab in the treatment of osteoporosis: 10 years later: a narrative review. *Adv Ther.* 2022;39:58-74. https://doi.org/10.1007/s12325-021-01936-y
8. Colella A, Yu E, Sambrook P, BAgSci TH, Goss A. What is the risk of developing osteonecrosis following dental extractions for patients on denosumab for osteoporosis? *J Oral Maxillofac Surg.* 2023;81(2):232-237.
9. McClung MR, Lewiecki EM, Cohen SB, et al. Denosumab in postmenopausal women with low bone mineral density. *N Engl J Med.* 2006;354:821-831.
10. Bocci V. Scientific and medical aspects of ozone therapy. state of the art. *Arch Med Res.* 2006;37(4):425-435.
11. Polydorou O, Halili A, Wittmer A, Pelz K, Hahn P. The antibacterial effect of gas ozone after 2 months of in vitro evaluation. *Clin Oral Investig.* 2012;16:545-550.
12. Huth KC, Jakob FM, Saugel B, et al. Effect of ozone on oral cells compared with established antimicrobials. *Eur J Oral Sci.* 2006;114(5):435-440.
13. Polydorou O, Pelz K, Hahn P. Antibacterial effect of an ozone device and its comparison with two dentin-bonding systems. *Eur J Oral Sci.* 2006;114(4):349-353.
14. Baysan A, Lynch E. Effect of ozone on the oral microbiota and clinical severity of primary root caries. *Am J Dent.* 2004;17(1):56-60.
15. Azarpazhooh A, Limeback H. The application of ozone in dentistry: a systematic review of literature. *J Dent.* 2008;36(2):104-116.
16. Das S. Application of ozone therapy in dentistry. *Indian J Dent Adv.* 2011; 3(2): 538-542.
17. Kazancioglu HO, Kurklu E, Ezirganli S. Effects of ozone therapy on pain, swelling, and trismus following third molar surgery. *Int J Oral Maxillofac Surg.* 2014;43(5):644.
18. Agın Demircan H, Gunes N, Guler R. A comparative investigation of the effects of resveratrol and dental pulp delivered mesenchimal stem cells on rat tibia bone defect healing. *Revista Científica.* 2024;34(2):rcfcv-e34372. doi: 10.52973/rcfcv-e34372

19. Ominsky L. Denosumab, a fully human RANKL antibody, reduced bone turnover markers and increased trabecular and cortical bone mass, density, and strength in ovariectomized cynomolgus monkeys. *Bone J.* 2011;49(2):162-173.

20. HelaS A. Inhibition of receptor activator of NF-B ligandby denosumab attenuates vascular calcium deposition in mice. *Am J Pathol.* 2009; 175(2):473-478.

21. Kostenuik PJ. Effects of denosumab, alendronate, or denosumab following alendronate on bone turnover, calciumhomeostasis, bone mass and bone strength in ovariectomized cynomolgus monkeys. *J Bone Miner Res.* 2015;30(4):657-669.

22. Kurban S, Mehmetoğlu İ. Osteoprotegerin, rank and rank ligand. *Turkish J Biochemist.* 2007;32(4):178-184.

23. Suresh, Abrahamsen B. Denosumab: a novel antiresorptive drug for osteoporosis. *Cleve Clin J Med.* 2015;82(2):105-114.

24. Alpan, AL, Toker H, Ozer H. Ozone therapy enhances osseous healing in rats with diabetes with calvarial defects: a morphometric and immunohistochemical study. *J periodontal.* 2016;87(8):982-989.

25. Buyuk SK, Ramoglu SI, Sonmez MF. The effect of different concentration of topical ozone administration on bone formation in orthopedically expanded suture in rats. *Eur J Orthod.* 2016; 38(3):281-285.

26. Gerstenfeld LC. Comparison of effects of the bisphosphonate alendronate versus the RANKL inhibitor denosumab on murine fracture healing. *J Bone Miner Res.* 2009;24:196-208.

27. Bernhardsson D. Anti-RANKL treatment improves screw fixation in cancellous bonein rats. *Inter J Care Injur.* 2015;46:990-995.

Comparison of two different types of molar distalization appliance

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ABSTRACT

Aims: The aim of this study was to examine the clinical efficacy of Modified Veltri (MV) and first class (FC) appliances, to investigate the effect on skeletal teeth and soft tissues in the patients and to compare the findings obtained.

Methods: The study included 40 individuals aged between 12 and 16 years with class II malocclusion (ANB <6°), dental crowding not requiring extraction, and no congenital tooth deficiency. MV and FC appliances were applied to 20 and 20 individuals, respectively. At the beginning of the study, at the end of distalization and three months after the reinforcement appliance was applied, cephalometric X-rays and plaster models were taken from the individuals and analyzed. The statistical significance of the changes that occurred during the distalization and reinforcement periods were evaluated by independent student's t test for each group, and the significance between the groups was checked by paired Student's t test.

Results: In the MV and FC groups, molar distalization was achieved in a similar time (4.29±0.97, 4.20±0.86). Skeletal changes were observed only in the MV group in SNB, ANB, SNGoGn, FMA ($p<0.05$) and B-PTV ($p<0.01$) values. In the first molar tooth, the MV group showed -2.16 mm distalization, 1.88 mm intrusion and 5.21° distal tipping, while the FC group showed -2.42 mm distalization and 1.19o distal tipping. During the consolidation period, 1.13 mm recurrence of distalization was observed in the MV group. In the MV group, overjet increased by 2.28 mm and overbite decreased by 1.89 mm. In the FC group, the overjet increased by 1.32 mm and the overbite decreased by 0.94 mm. After soft tissue distalization, Lu-E and Li-E values decreased by 1.45 mm and 1.01 mm in the MV group and by 1.38 mm and 1.30 mm in the FC group.

Conclusion: In this study, although MV and FC appliances provided a similar amount of distalization in a similar amount of time, recurrence was observed in the MV group during the reinforcement period. In addition, loss of anchorage was observed more in the MV group. Anchorage loss should be considered in the clinical application of MV and FC appliances.

Keywords: Distalization, veltri, first class

INTRODUCTION

In the treatment planning of dental class II cases, tooth extraction and molar distalization, which are methods of gaining space for the elimination of protrusion of the upper teeth or dental crowding, have been the subject of debate among researchers[1-3] for many years.

Gianelly and White[4] state that in borderline cases, the decision to extract becomes important because permanent tooth extraction will affect facial aesthetics.

Philip[5] argued that borderline cases can be successfully treated without extraction with the right mechanics at the right time in individuals with normal growth and development who do not have cooperation problems.

Distalization of the maxillary posterior teeth is the most commonly used non-extraction treatment approach. Headgear is the oldest and most common distalization method. However, patient cooperation is required. Intraoral fixed distalization devices have been developed in cases where patient cooperation cannot be achieved. Loss of anchorage during distalization of maxillary molars is the biggest disadvantage of these appliances. In addition, tipping and rotations occurring in distalized molars are also undesirable types of movement.[6-16]

Keleş,[12] reported that they achieved 4.5 mm distalization of the upper first molar tooth without distal tipping and extrusion movement with the Keleş Slider appliance. It was claimed that

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the molars moved distally due to the use of thick wire in the construction of the appliance and the force passing through the level of the center of resistance of the tooth. Fortini et al.[13,17] obtained bodily and rapid molar distalization with the first class (FC) appliance. Küçükkeleş et al.[18] suggested that loss of anchorage was high during molar distalization with the lip bumper supported Veltri appliance, so clinicians should pay attention to case selection when using this appliance.

In this study, it was aimed to investigate the clinical efficacy of the modified veltri (MV) and FC appliances introduced by Baccetti and Franchi,[19] to investigate the effect on skeletal, soft tissues and dentolaveolar structures in the patients and to compare the findings obtained.

METHODS

This study is a doctoral thesis completed before 2020. Institutional approval was obtained. All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

The following characteristics were taken into consideration in the selection of the patients:

- ANB <6°
- Tubercle-tubercle class II molar relationship of bilateral upper first molar (U6) and lower first molar (L6),
- Absence of severe space stenosis in the upper and lower jaw that would require extraction,
- Normal or retrusive upper and lower incisors in relation to the basal bone base,
- Normal or deep closure,
- Perpendicular face dimensions are low or within normal limits (SN-Go-Me <37°),
- Having a soft tissue profile that does not require extraction treatment,
- Absence of congenital tooth deficiency,
- Chronological age between 12-16 years.

The MV appliance was applied to 20 individuals (11 girls and 9 boys) and the FC appliance was applied to 20 individuals (12 girls and 8 boys). The mean age of the individuals before distalization (D1) was 13.64±1.46 in the MV group and 13.82±1.43 in the FC group.

Construction and Application of Appliances

The MV appliance was prepared and applied based on the form developed by Bacetti T. and Franchi L.[19] (Figure 1). A schedule was prepared for the parents to perform the screw twice a week, with 90° opening at each activation. Screw activations were continued until a class I molar relationship was achieved. The FC appliance was prepared as developed by Fortini et al.[13] (Figure 2). However, the parents were asked to perform the activation of the screws one half turn (180°) at two-day intervals. Screw activations were performed until a class I molar relationship was achieved (Figure 3, 4). After distalization (D2), the appliance was removed from the mouth and model and lateral cephalometric film records were taken from the patients.

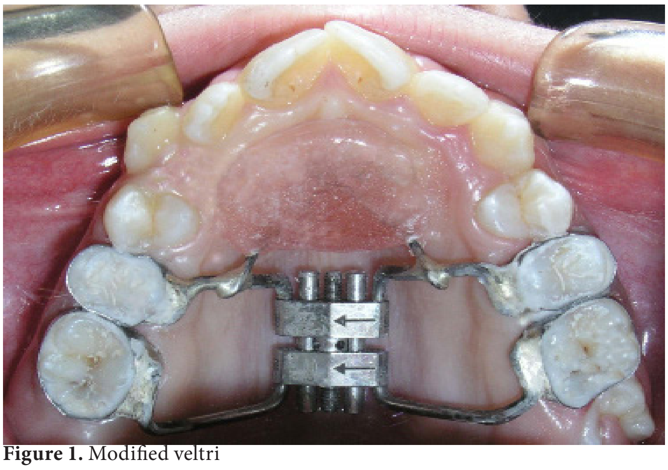

Figure 1. Modified veltri

Figure 2. First class

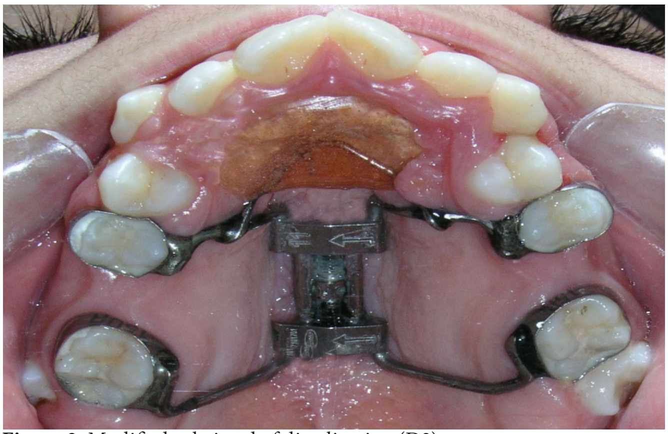

Figure 3. Modified veltri end of distalization (D2)

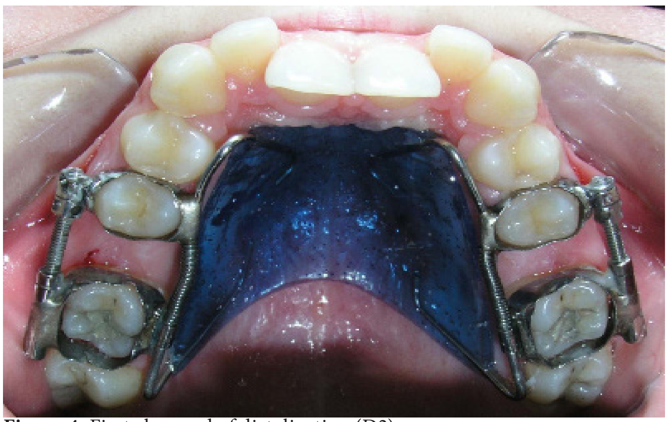

Figure 4. First class end of distalization (D2)

Stabilization Period

Reinforcement appliance was applied after the distalization of U6 was completed in the MV and FC patient groups. After the reinforcement appliance was applied, the individuals participating in the study were observed for three months without the application of any appliance (Figure 5). After three months, the appliance was removed from the mouth again and cephalometric films and plaster model records were obtained from both groups in order to monitor spontaneous changes (D3).

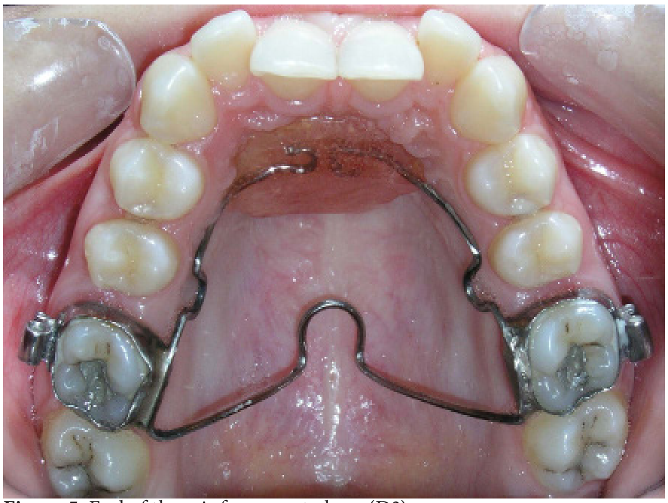

Figure 5. End of the reinforcement phase (D3)

Lateral Cephalometric Filming and Evaluation

All lateral cephalometric films were taken digitally (Vatech, PaX-400C, Korea) in the natural head position at the Oral Diagnosis and Radiology Clinic of Dicle University Faculty of Dentistry. To ensure standardization in the measurements and to monitor the effect of distalization appliances on the posterior teeth in the maxilla, marker wires prepared from 0.5 mm stainless steel wire were fixed in acrylic crowns on the right U6 and second premolar (U5) teeth before distalization (Figure 6).[20] These acrylic marker crowns were temporarily placed on the teeth and the first lateral cephalometric film was taken (Figure 7). The measurements of the maxillary posterior teeth were made on this radiograph (Figure 8). The second lateral cephalometric film was taken when the teeth were in centric occlusion. Skeletal and soft tissue and incisors and lower first molar (L6) values were measured on the second lateral cephalometric film (Figure 9-12).

A total of 28 parameters (13 angular and 15 linear) for cephalometric evaluation were created using measurements from Pancherz,[21] McNamara,[22] Ricketts[23] and Steiner[24] analyses (Table 1). The Frankfurt horizontal plane (FH), the plane passing through the orbital and anatomical porion points, was determined as the horizontal reference plane for the analyses. Pterygo vertical plane (PTV), the line drawn perpendicular to the FH plane from distal to the pterygopalatine fossa, was used as the vertical reference plane in the analyses.

Evaluation of Orthodontic Models

In both groups, plaster models were obtained by taking impressions from the upper jaws of the individuals before distalization (D1), after distalization (D2) and after reinforcement (D3). On these plaster models, the tubercle crests, anterior palatal raphe and posterior palatal raphe points of the U6 teeth were marked with a 0.5 mm pencil. Between these marked points, the midline line (MRL) was determined as the reference plane. Then, the occlusal surface of the models was placed on the glass of the photocopier and photocopies of the models were taken.[25] Angle measurements were made on these photocopies to determine whether there was rotation after distalization of the U6 teeth (Figure 13).

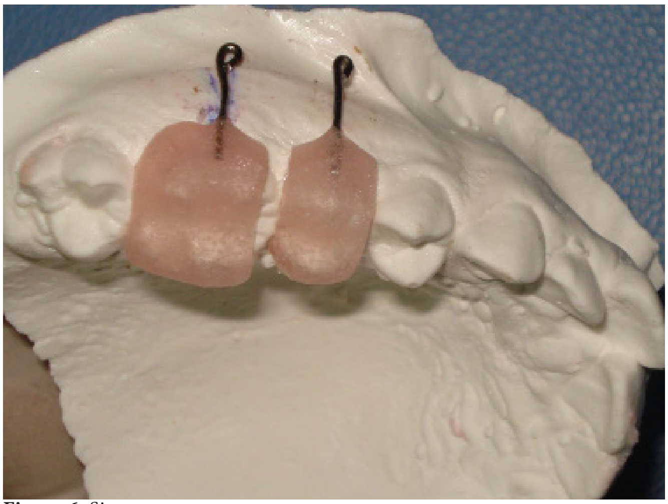

Figure 6. Sign crowns

Figure 7. Cephalometric radiograph with sign crown

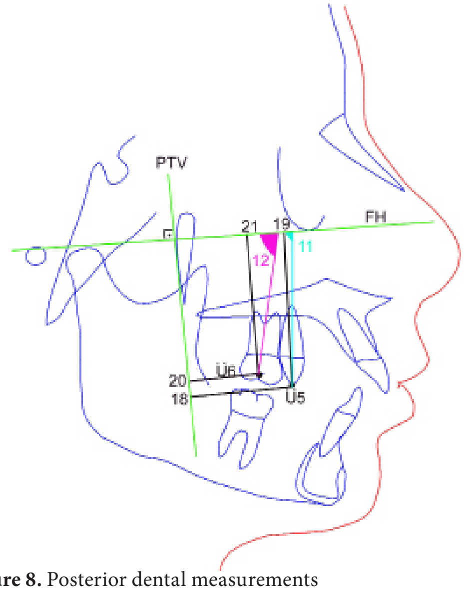

Figure 8. Posterior dental measurements

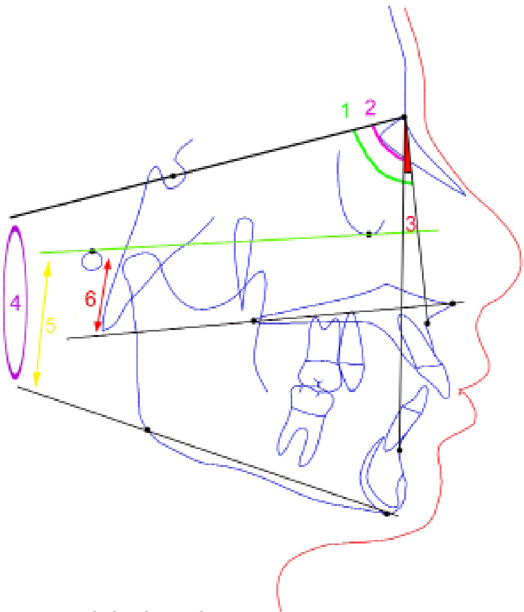

Figure 9. Skeletal angular measurements

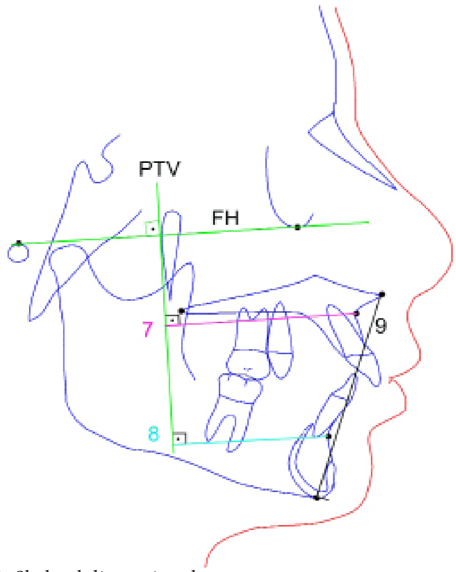

Figure 10. Skeletal dimensional measurements

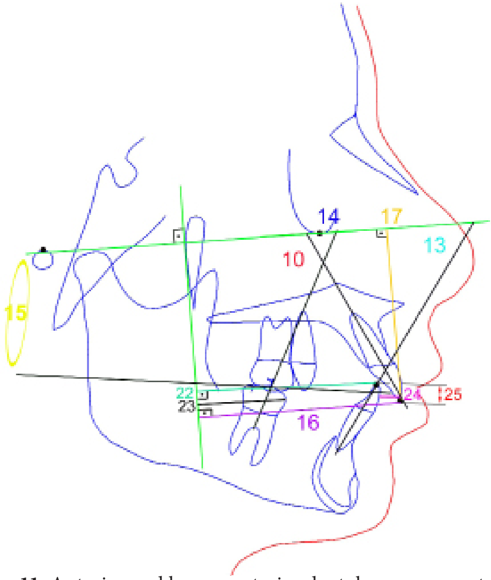

Figure 11. Anterior and lower posterior dental measurements

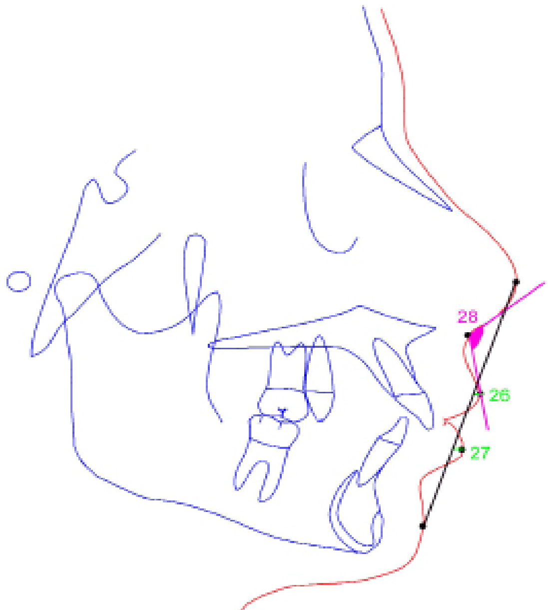

Figure 12. Soft tissue measurements

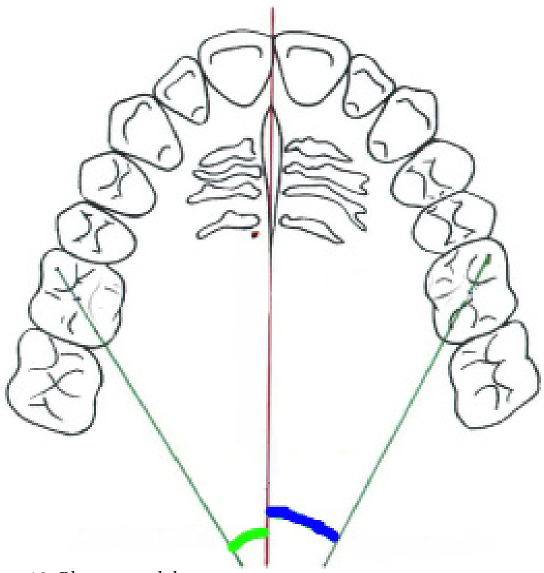

Figure 13. Plaster model measurements

Table 1. Measurements performed for cephalometric evaluation

SKELETEAL	SNA	SNA Angle formed between Sella-Nasion and Nasion-A.
	SNB	SNB Angle formed between Sella-Nasion and Nasion-B.
	ANB	ANB Angle formed between points A-N-B.
	SN-GoMe	SN-GoMe Angle formed between the Sella-Nasion and the mandibular plane.
	FMA	FMA Angle formed between the Frankfurt horizontal plane and the mandibular plane.
	PD-FH	PD-FH Angle between the Frankfurt horizontal plane and the palatal plane.
	OD-FH	OD-FH The angle formed between the FH and the occlusal plane.
	A-PTV	A-PTV Length of the perpendicular drawn from point A to the PTV plane.
	B-PTV	B-PTV The length of the perpendicular drawn from point B to the PTV plane.
	ANS-Me	ANS-Me The distance between the spina nasalis anterior and the Me points.
DENTAL	Overjet	The horizontal distance between the cutting edges of the upper and lower most advanced incisors.
	Overbite	The vertical bite distance between the incisal edges of the upper and lower most advanced incisors.
	U1-FH angle	The angle formed between the long axis of the upper most advanced incisor and the FH plane.
	U1-FH mm	The perpendicular distance of the incisal edge of the upper most advanced incisor to the FH plane.
	U1-PTV	The perpendicular distance from the incisor edge of the upper most incisor to the PTV plane.
	U5-FH angle	The angle formed between the line through the index wire in acrylic crowns placed on the upper second premolar and the FH plane.
	U5-FH mm	The perpendicular distance from the upper second premolar marker point to the FH plane.
	U5-PTV	The perpendicular distance from the upper second premolar marker point to the PTV plane.
	U6-FH angle	The angle formed between the line through the index wire in acrylic crowns placed on the upper first premolar and the FH plane.
	U6-FH mm	It is the perpendicular distance from the upper first molar marker point to the FH plane.
	U6-PTV	The perpendicular distance from the upper first molar landmark to the PTV plane.
	L6-FH angle	The angle formed between the FH and the line connecting the center of the crown and the furcation point of the lower first molar.
	L6-PTV	The perpendicular distance of the mesial contact point of the lower first molar from the plane of the PTV.
	L1-FH angle	The angle formed between the long axis of the lower most advanced incisor and the FH.
	L1-PTV	The perpendicular distance of the incisal edge of the lower most advanced incisor from the plane of the PTV.
SOFT TISSUEU	NLA	The angle formed by the line extending from the vermilion border of the upper lip to the subnasal and the tangent drawn from the subnasal to the lower border of the nose.
	Lu-E	Distance between line E and point Ls.
	Li-E	The distance between the E line and the Li point.

Statistical Analysis

Statistical evaluations were performed with the SPSS 10.0.0 program (Chicago, Illinois, USA). Comparisons were made both within and between groups to determine the statistical significance of the changes in cephalometric and plaster models in the MV and FC groups. For each group, the evaluation of the statistical significance of the changes in the distalization (D2-D1) and three-month reinforcement period (D3-D2) was performed by independent student's t-test, and the significance of the changes in the D2-D1 and D3-D2 periods of the groups was checked between the groups by paired student's t-test.

RESULTS

Distalization time (D2-D1) lasted an average of 4.29±0.97 months in the MV group and 4.20±0.86 months in the FC group. When the ages and distalization times of the individuals with MV and FC appliances were compared by independent student's t-test, no statistical difference was found ($p>0.05$).

To test the similarity of the groups before distalization (D1), skeletal, dental and soft tissue measurements made on lateral cephalometric radiographs were compared. In the comparison, only the mean OD-FH angle of 3.31° ($p<0.05$) and the mean U5-FH angle of 4.01° ($p<0.01$) were statistically significantly higher in the MV group (Table 2).

Findings of the MV Group

The cephalometric and model measurements of the MV group in the D1, D2 and D3 periods and the statistical comparison of the changes after distalization (D2-D1) and after reinforcement (D3-D2) are shown in Table 3 and Table 4.

Accordingly, in the evaluation of cephalometric measurements;

After distalization (D2-D1), there was a statistically significant increase ($p<0.05$, $p<0.01$) in the mean value of SNB, B-PTV (0.64°, 1.73 mm) and a statistically significant decrease ($p<0.05$) in the mean value of ANB, SN-GoMe and FMA (0.8).

After distalization, the mean overjet increased by 2.28 mm and the mean overbite decreased by 1.89 mm ($p<0.001$). After consolidation, overjet and overbite decreased by 0.45 mm and increased by 0.63 mm, respectively, at a statistically significant level ($p<0.01$).

After distalization, the upper incisors (U1) protruded a statistically significant average of 11.71° angular (U1-FH angle) and 4.39 mm dimensional (U1-PTV mm) ($p<0.001$, $p<0.01$). After reinforcement, U1 moved statistically significantly mean, angular 4.75° (U1-FH angle) and dimensional (U1-PTV) 1.49 mm distally ($p<0.001$, $p<0.05$).

After distalization, the upper second premolar (U5) moved statistically significantly mean, angular 9.68° (U5-FH angle) and dimensionally 4.94 mm (U5-PTV) mesially ($p<0.001$). After consolidation, U5 moved in a statistically significant mean, angular 12.12° (U5-FH angle) and dimensional 3.27 mm distally ($p<0.001$). After distalization, the U6-FH angle decreased by a statistically significant mean of 5.21° and U6

Table 2. Comparison of cephalometric measurements between MV and FC groups before distalization

Comparison of Baseline Measurements Between Groups	Veltri n=20	First Class n=20	Significance	
SKELETEAL	SNA	80.62±3.17	80.94±4.97	-
	SNB	76.84±2.65	76.63±3.32	-
	ANB	4.05±1.47	4.30±2.08	-
	SN-GoMe	30.47±4.16	30.90±4.08	-
	FMA	25.77±5.14	23.55±5.52	-
	PD-FH	2.93±4.25	0.63±3.85	-
	OD- FH	10.42±3.69	7.16±4.81	*
	A PTV	49.85±4.65	49.18±4.97	-
	B PTV	40.39±5.01	41.15±4.99	-
	ANS Me	65.58±5.27	62.16±6.22	-
DENTAL	Overjet	4.86±1.56	4.11±1.24	-
	Overbite	4.16±1.90	3.56±1.09	-
	U1-FH angle	100.96±7.66	103.11±7.57	-
	U1-FH mm	52.33±4.64	48.99±6.66	-
	U1-PTV	50.43±4.51	50.37±5.43	-
	U5-FH angle	80.99±5.24	85.79±4.64	**
	U5-FH mm	47.44±4.32	45.83±6.28	-
	U5-PTV	23.49±3.68	24.68±4.51	-
	U6-FH angle	91.88±5.42	88.64±7.30	-
	U6-FH mm	45.94±4.39	44.21±5.76	-
	U6-PTV	23.76±3.56	23.88±4.80	-
	L6-FH angle	67.86±5.34	69.53±3.44	-
	A6-PTV	21.14±3.46	23.03±4.77	-
	L1-FH angle	57.16±7.09	59.47±6.43	-
	L1-PTV	46.56±4.48	46.47±5.51	-
SOFT TISSUE	NLA	116.68±11.76	112.33±7.76	-
	Lu-E	-4.69±2.90	-5.16±2.28	-
	Li-E	-3.61±2.58	-3.95±2.61	-

- $p>0.05$, * $p<0.05$, ** $p<0.01$

tilted distally (p<0.01). U6-PTV decreased by a statistically significant mean of 2.16 mm, i.e. U6 moved in the distal direction (p<0.01). U6-FH mm decreased by a statistically significant mean of 1.88 mm, i.e. U6 was intruded (p<0.01). After consolidation, U6-PTV increased by a statistically significant mean of 1.13 mm, i.e. U6 moved mesially (p<0.05). After distalization, the lower incisor (L1) moved forward in the sagittal direction by a statistically significant mean of 1.93 mm (L1-PTV) (p<0.01). After distalization, the upper lip (Lu) and lower lip (Li) moved forward in the sagittal direction by a statistically significant mean of 1.45 mm (Lu-E) and 1.01 mm (Li-E), respectively (p<0.001). After reinforcement, Li-E increased by a statistically significant mean of 0.40 mm (p<0.05).

According to the evaluation of model measurements;

After distalization, only the left U6-OHD decreased by a statistically significant mean of 3.25° (p<0.05) (Table 4).

Findings for the FC Group

The statistical comparison of cephalometric measurements of D1, D2 and D3 periods, changes after distalization (D2-D1) and changes after reinforcement (D3-D2) in the FC group are shown in Table 5. Accordingly;

After distalization (D2-D1), there was no statistically significant difference (p>0.05) in skeletal values in the measurements.

After distalization, the mean overjet increased by 1.32 mm and the mean overbite decreased by 1.89 mm (p<0.001). After consolidation, overjet decreased by a statistically significant mean of 0.67 mm (p<0.01).

After distalization, the upper incisors (U1) protruded a statistically significant average of 3.49° angular (U1-FH angle) and 1.59 mm dimensional (U1-PTV mm) (p<0.05).

Table 3. Measurements of D1, D2 and D3 periods of the MV group; statistical evaluation of changes after distalization (D2-D1) and reinforcement (D3-D2)

Modified Veltri n=20		D1 Mean±SD	D2 Mean±SD	D3 Mean±SD	D2-D1 Mean±SD	p	D3-D2 Mean±SD	p
SKELETEAL	SNA	80.62±3.17	80.53±3.15	80.86±3.23	-0.09±3.34		0.27±1.38	
	SNB	76.84±2.65	77.49±3.19	76.69±2.70	0.64±1.27	*	-0.80±0.90	**
	ANB	4.05±1.47	3.35±1.96	3.81±1.88	-0.70±1.30	*	0.46±0.96	*
	SN-GoMe	30.47±4.16	29.71±4.47	29.66±4.62	-0.73±1.39	*	-0.05±1.06	
	FMA	25.77±5.14	24.42±5.00	24.27±5.05	-1.35±2.30	*	-0.15±1.45	
	PD-FH	2.93±4.25	2.64±3.62	2.53±3.64	-0.25±1.93		-0.11±1.61	
	OD-FH	10.42±3.69	9.29±3.87	9.88±3.71	-1.13±3.37		0.59±1.99	
	A PTV	49.85±4.65	50.04±4.85	51.03±4.43	0.18±2.20		0.99±3.18	
	B PTV	40.39±5.01	42.12±6.09	41.95±5.18	1.73±2.57	**	-0.17±2.45	
	ANS Me	65.58±5.27	64.70±5.92	65.34±6.27	-0.87±3.07		0.64±1.23	*
DENTAL	Overjet	4.86±1.56	7.14±1.99	6.69±1.93	2.28±1.49	***	-0.45±0.67	**
	Overbite	4.16±1.90	2.26±2.16	2.89±1.78	-1.89±1.14	***	0.63±0.91	**
	U1-FH angle	100.96±7.66	112.67±11.20	107.91±10.16	11.71±6.83	***	-4.75±3.81	***
	U1-FH mm	52.33±4.64	51.07±5.40	51.12±4.89	-1.25±3.60		0.05±1.79	
	U1-PTV	50.43±4.51	54.83±5.74	53.34±5.12	4.39±2.91	**	-1.49±2.65	*
	U5-FH angle	80.99±5.24	90.67±7.13	78.55±6.43	9.68±6.95	***	-12.12±7.02	***
	U5-FH mm	47.44±4.32	46.47±5.83	46.31±4.65	-0.97±4.60		-0.15±2.69	
	U5-PTV	23.49±3.68	28.44±4.78	25.16±3.10	4.94±2.88	***	-3.27±2.98	***
	U6-FH angle	91.88±5.42	86.67±6.55	87.21±5.45	-5.21±7.85	**	0.54±6.64	
	U6-FH mm	45.94±4.39	44.06±4.04	44.56±4.54	-1.88±2.49	**	0.40±1.14	
	U6-PTV	23.76±3.56	21.69±3.96	22.73±3.13	-2.16±3.33	**	1.13±2.43	*
	L6-FH angle	67.86±5.34	67.27±5.22	67.08±4.11	-0.58±5.88		-0.19±6.01	
	A6-PTV	21.14±3.46	22.55±4.05	23.20±2.92	1.41±3.33		0.65±2.80	
	L1-FH angle	57.16±7.09	55.84±5.73	56.12±5.57	-1.32±3.09		0.28±3.01	
	L1-PTV	46.56±4.48	48.50±5.11	48.64±4.51	1.93±2.30	**	0.14±1.88	
SOFT TISSUE	NLA	116.68±11.76	112.58±7.59	113.95±9.39	-4.10±9.70		1.37±5.72	
	Lu-E	-4.69±2.90	-3.24±2.1493	-3.59±2.16	1.45±1.46	***	-0.34±0.83	
	Li-E	-3.61±2.58	-2.60±2.07	-2.20±2.10	1.01±0.98	***	0.40±0.65	*

* p<0.05, ** p<0.01, *** p<0.001, SD: Standart deviation

Table 4. Model measurements of the MV and FC group for D1, D2 and D3 periods, statistical evaluation of changes after distalization (D2-D1) and reinforcement (D3-D2)

Model Measurement of MV and FC Groups Mean±SD		D1	D2	D3	D2-D1		D3-D2	
		Mean±SD	Mean±SD	Mean±SD	p	Mean±SD	p	
MV	Right U6-OHD	25.02±5.15	22.15±7.13	25.12±8.18	-2.87±6.19	-	2.97±6.63	-
	Left U6-OHD	29.75±4.06	26.50±6.22	26.87±6.61	-3.25±4.36	*	0.37±5.07	-
FC	Right U6-OHD	28.47±5.28	33.99±6.25	34.17±6.72	4.52±4.15	***	1.17±5.25	-
	Left U6-OHD	30.72±5.39	33.95±5.39	34.12±3.75	3.22±6.32	*	0.17±2.57	-

-p>0.05, * p<0.05, *** p<0.001, MV: Modified veltri, FC: First class, SD: Stantard deviation

Table 5. Measurements of D1, D2 and D3 periods, changes after distalization (D2-D1) and after reinforcement (D3-D2) and statistical evaluation of the FC group

First Class n=20		D1 Mean±SD	D2 Mean±SD	D3 Mean±SD	D2-D1 Mean±SD	p	D3-D2 Mean±SD	p
SKELETEAL	SNA	80.94±4.97	81.08±4.81	81.00±4.04	0.14±1.61		-0.08±2.41	
	SNB	76.63±3.32	76.48±3.30	76.70±3.07	-0.15±1.18		0.22±1.72	
	ANB	4.30±2.08	4.59±1.81	4.28±1.67	0.28±1.00		-0.31±1.23	
	SN-GoMe	30.90±4.08	31.22±4.12	30.91±5.31	0.31±2.39		-0.31±2.07	
	FMA	23.55±5.52	23.59±5.23	25.16±6.19	-0.04±2.55		1.57±5.19	
	PD-FH	0.63±3.85	0.07±3.88	1.39±2.93	-0.55±1.94		1.32±4.78	
	OD- FH	7.16±4.81	7.64±4.78	8.95±5.32	0.47±3.44		1.31±4.30	
	A PTV	49.18±4.97	49.34±3.61	48.94±3.74	0.16±2.68		-0.60±2.68	
	B PTV	41.15±4.99	40.76±4.38	39.26±5.48	-0.39±3.22		-1.50±5.09	
	ANS Me	62.16±6.22	61.12±5.15	61.17±4.96	-1.03±2.48		0.04±1.92	
DENTAL	Overjet	4.11±1.24	5.43±2.25	4.76±1.46	1.32±1.21	***	-0.67±0.97	**
	Overbite	3.56±1.09	2.62±1.27	2.62±1.27	-0.94±1.05	***	0.15±1.47	
	U1-FH angle	103.11±7.57	106.61±10.64	103.23±9.52	3.49±5.83	*	-3.38±8.30	
	U1-FH mm	48.99±6.66	48.63±5.68	49.70±3.63	-0.35±2.21		1.06±5.90	
	U1-PTV	50.37±5.43	51.97±5.29	49.07±5.65	1.59±2.97	*	-2.90±5.23	*
	U5-FH angle	85.79±4.64	90.99±5.20	81.83±8.40	5.19±6.84	**	-9.16±9.66	***
	U5-FH mm	45.83±6.28	46.06±6.19	45.58±3.30	0.23±2.25		-0.48±4.49	
	U5-PTV	24.68±4.51	28.03±3.53	24.41±4.06	3.35±4.08	**	-3.62±2.62	***
	U6-FH angle	88.64±7.30	87.45±9.90	87.01±8.74	-1.19±8.21		-0.43±7.98	
	U6-FH mm	44.21±5.76	44.48±5.47	43.73±3.29	0.72±2.03		-0.24±3.56	
	U6-PTV	23.88±4.80	21.46±4.13	21.76±4.23	-2.42±2.43	***	0.30±4.12	
	L6-FH angle	69.53±3.44	67.46±5.82	68.89±4.60	2.07±5.17		1.43±6.92	
	A6-PTV	23.03±4.77	23.46±4.04	22.58±4.14	0.43±2.81		-0.88±4.13	
	L1-FH angle	59.47±6.43	58.81±6.34	57.58±6.46	-0.65±2.63		-1.23±6.8	
	L1-PTV	46.47±5.51	46.06±4.42	45.77±4.73	-0.40±2.89		-0.29±4.27	
SOFT TISSUEU	NLA	112.33±7.76	108.05±7.97	108.42±9.43	-4.28±10.28		0.37±6.97	
	Lu-E	-5.16±2.28	-3.77±2.41	-3.22±2.29	1.38±1.25	***	0.55±1.95	
	Li-E	-3.95±2.61	-2.65±2.63	-1.92±2.90	1.30±1.84	**	0.72±2.51	

* p<0.05, ** p<0.01, *** p<0.001, SD: Standart deviation

After reinforcement, U1 moved statistically significantly mean, dimensional (U1-PTV) 2.90 mm distally ($p<0.05$).

After distalization, U5 moved statistically significantly mean, angular 5.19° (U5-FH angle) and dimensional 3.35 mm (U5-PTV) mesially ($p<0.01$). After consolidation, U5 moved in a statistically significant mean, angular 9.16° (U5-FH angle) and dimensional 3.62 mm (U5-PTV) distally ($p<0.001$).

After distalization, U6-PTV decreased by a statistically significant mean of 2.42 mm, IE U6 moved distally ($p<0.001$).

After distalization, the upper lip (Lu) and lower lip (Li) moved forward in the sagittal direction by a statistically significant mean of 1.38 mm (Lu-E) and 1.30 mm (Li-E), respectively ($p<0.001$, $p<0.01$).

The evaluation of the model measurements is shown in Table 4, accordingly;

After distalization, right U6-OHD increased by an average of 4.52° ($p<0.001$) and left U6-OHD increased by an average of 3.22° ($p<0.05$) at a statistically significant level.

Comparison of Differences in the D2-D1 and D3-D2 Periods of MV AND FC Groups

The comparison of cephalometric changes in the MV and FC groups in the D2-D1 and D3-D2 periods is shown in Table 6. Accordingly; After distalization (D2-D1), there was a statistically significant difference between the groups in SNB, ANB, SN-GoMe angles and B-PTV distances ($p<0.05$). After consolidation (D3-D2), a statistically significant difference was found between the groups in SNA and SNB angles ($p<0.05$). Statistically significant differences were found between the groups in overjet distance and U5-FH angle after distalization ($p<0.05$). Statistically significant differences were found between the groups in overbite, U1-PTV and A1-PTV distances ($p<0.01$). There was a statistically significant difference between the groups in the U1-FH angle ($p<0.001$). The comparison of the model measurements after distalization and reinforcement in MV and FC groups is shown in Table 7, according to this; a statistically significant ($p<0.001$) difference was observed in right U6-OHD° and left U6-OHD° after distalization.

Table 6. Comparison of the findings of distalization (D2-D1) and reinforcement periods (D3-D2) between MV and FC groups

Comparison Between Groups		D2-D1			D3-D2		
		MV n=20 Mean±SD	FC n=20 Mean±SD	p	MV n=20 Mean±SD	FC n=20 Mean±SD	p
SKELETEAL	SNA	-0.09±3.34	0.14±1.61		0.27±1.38	0.08±2.41	*
	SNB	0.64±1.27	-0.15±1.18	*	-0.80±0.90	0.22±1.72	*
	ANB	-0.70±1.30	0.28±1.00	*	0.46±0.96	-0.31±1.23	
	SN-GoMe	-0.73±1.39	0.31±2.09	*	-0.05±1.06	-0.31±2.07	
	FMA	-1.35±2.30	-0.04±2.55		-0.15±1.45	1.57±5.19	
	PD-FH	-0.25±1.93	-0.55±1.94		-0.11±1.61	1.32±4.78	
	OD-FH	-1.13±3.37	0.47±3.44		0.59±1.99	1.31±4.30	
	A PTV	0.18±2.20	0.16±2.68		0.99±3.18	0.60±2.68	
	B PTV	1.73±2.57	-0.39±3.22	*	-0.17±2.45	-1.50±5.09	
	ANS Me	-0.87±3.07	-1.03±2.48		-0.64±1.23	0.04±1.92	
DENTAL	Overjet	2.28±1.49	1.32±1.21	*	-0.45±0.67	-0.67±0.97	
	Overbite	-1.89±1.14	-0.94±1.05	**	0.63±0.91	0.15±1.47	
	U1-FH angle	11.71±6.83	3.49±5.83	***	-4.75±3.81	-3.38±8.30	
	U1-FH mm	-1.25±3.60	-0.35±2.21		0.05±1.79	1.06±5.90	
	U1-PTV	4.39±2.91	1.59±2.97	**	-1.49±2.65	-2.90±5.23	
	U5-FH angle	9.68±6.95	5.19±6.84	*	-12.12±7.02	-9.16±9.66	
	U5-FH mm	-0.97±4.60	0.23±2.25		-0.15±2.69	-0.48±4.49	
	U5-PTV	4.94±2.88	3.35±4.08		-3.27±2.98	-3.62±2.62	
	U6-FH angle	-5.21±7.85	-1.19±8.21		0.54±6.64	-0.43±7.98	
	U6-FH mm	-1.88±2.49	0.72±2.03		0.40±1.14	0.24±3.56	
	U6-PTV	-2.16±3.33	-2.42±2.43		1.13±2.43	0.30±4.12	
	L6-FH angle	-0.58±5.88	-2.07±5.17		-0.19±6.01	1.43±6.92	
	L6-PTV	1.41±3.33	0.43±2.81		0.65±2.80	-0.88±4.13	
	L1-FH angle	-1.32±3.09	-0.65±2.63		0.28±3.01	-1.23±6.18	
	L1-PTV	1.93±2.30	-0.40±2.89	**	0.14±1.88	-0.29±4.27	
SOFT TISSUEU	NLA	-4.10±9.70	-4.28±10.28		1.37±5.72	0.37±6.97	
	Lu-E	1.45±1.46	1.38±1.25		-0.34±.083	0.55±1.95	
	Li-E	1.01±0.98	1.30±1.84		0.40±0.65	0.72±2.51	

*p<0.05, ** p<0.01, *** p<0.001, MV: Modified veltri, FC: First class, SD: Standart deviation

Table 7. Statistical comparison of model measurements, changes after distalization (D2-D1) and after reinforcement (D3-D2) in MV and FC groups

Model Comparison Between MV and FC Groups	D2-D1			D3-D2		
	MV n=20 Mean±SD	FC n=20 Mean±SD	p	MV n=20 Mean±SD	FC n=20 Mean±SD	p
Right U6-OHD	-2.87±6.19	4.52±4.15	***	2.97±6.63	1.17±5.25	-
Left U6-OHD	-3.25±4.36	3.22±6.32	***	0.37±5.07	0.17±2.57	-

-p>0.05, ***:p<0.001, MV: Modified veltri, FC: First class, SD: Standart deviation

DISCUSSION

Molar distalization is a method applied to obtain a Cl I molar and canine relationship in the treatment of class II malocclusions. For this purpose, many distalization appliances have been developed from past to present.[6,12,17,20] Clinicians evaluate the advantages and disadvantages of these appliances compared to each other and prefer the mechanics that are suitable for each case. There are no studies in the literature comparing the effects of Modified Veltri and first class appliances. For this purpose, in this study, the effects of molar distalization with Modified Veltri and first class appliances on skeletal teeth and soft tissues were compared. In the cephalometric comparison of the groups at the beginning of the study, it was determined that the groups were mostly similar, with a difference only in OD-FH and U5-FH angle measurements.

Evaluation of Changes in Skeletal Structure

In the study, the decrease in FMA, SNGoGn angles after distalization in the Modified Veltri group indicates that the plane of the mandible rotated counterclockwise. The counterclockwise displacement of point B due to this rotation suggests that it causes differences in SNB, ANB and B-PTV values. The fact that the distalized molar tooth also intrudes in the vertical direction indicates the possibility of a reduction and rotation in the mandibular plane angle. In the literature, some researchers.[26,27] reported that the upper first molar was intruded while distalizing. However, they did not make a skeletal evaluation. Haydar et al.[28] reported in their study that extrusion occurred in the molars with intraoral molar distalization, but this did not have an effect on the mandibular plane angle. There was no difference in statistical values after distalization in the first class group. Fortini et al.[17] reported that 1.22 mm molar extrusion occurred with molar distalization but did not cause any sagittal or vertical skeletal change. Moschos et al.[29] also reported no skeletal change in their study.

The results of our study are compatible with this study. However, changes in some skeletal values were observed in the studies of some researchers.[30,31] It has been interpreted that these changes may be due to the upper first molars being pushed backwards in the arch. In this study, the fact that dental differences were observed only in the sagittal direction in the first class group and the upper first molars moved vertically only in the Modified Veltri group caused statistical differences in skeletal values between the groups.

In the Modified Veltri appliance group, it was determined that the skeletal values that changed due to the movement of the upper first molars after distalization recurred to some extent after reinforcement (D3-D2). Dental recurrence also caused recurrence in skeletal changes. It reinforces the idea that the skeletal changes occurred due to dental movements.

Evaluation of Changes in Dental Structures

In both groups with modified Veltri and first class appliances, an increase in overjet distance and a decrease in overbite distance were observed due to the protrusion of the incisors after distalization. This situation shows the loss of anchorage reflected to the incisors with distalization. In many studies[12,17,18,32] protrusion of the incisors and increases in overjet distance are inevitably seen in all intraoral distalization mechanics consisting of conventional anchorage units. In this study, less protrusion was observed in the first class group compared to the modified Veltri group. The large acrylic area of the first class appliance suggests that it strengthens the anchorage against the distalization force with tissue support in the palatinal region. Thus, the reciprocal force transferred to the incisors may have decreased compared to the modified Veltri appliance.

When the position of the upper second premolar after distalization was evaluated in the modified Veltri group, 4.94 mm of anchorage was lost compared to 2.16 mm of distalization. In the literature, Küçükkeleş et al.[18] reported that 25%-80% anchorage loss can be seen in their study. In our study, anchorage was tried to be increased with the nance button added to the Hyrax screw that creates the distalization force. However, since the distalization force was close to the deepest point of the palate, it created a clockwise moment. This moment on the screw may have further increased the loss of anchorage. This moment caused the appliance to move clockwise in the mouth. The rotation of the appliance caused distalization and intrusion of the posterior teeth. This suggests that the anchorage should be increased in future studies on this appliance. In the study of Küçükkeleş et al.,[18] it was reported that anchorage loss occurred at 4.17 mm. against 4.17 mm. distalization. Therefore, they suggested that anchorage units should be increased. In the light of this information; tooth and palate tissue supported intraoral distalization mechanics show that it is difficult to provide distalization without anchorage loss.

In our study, 5.19° mesial tipping and 3.35 mm mesial movement of the upper second molar was found in the first class group after distalization. Fortini et al.[17] found 2.2° mesial tipping and 1.7 mm mesial movement of the upper second premolars after distalization. Moschos et al.[29] reported that premolars or deciduous second molars moved mesially with a tipping of 1.86 mm and 1.85 degrees. In this study, the fact that the activation of the appliance was performed at different speeds and the age of the patients was different suggests that the presence of second molars may have increased the loss of anchorage.

Since the designs of the anchorage units of the first class and Veltri appliances and the point of origin of the distalization force are different in both appliances, it may have caused different anchorage losses in the supported teeth. In our study, in the modified Veltri group, 1.88 mm intrusion and 2.16 mm molar distalization were achieved with 5.21° distal tipping in approximately five months. The fact that the crown distalization rate is higher than the root distalization rate with this appliance and the moment motion generated in the appliance affect the formation of body molar distalization. In the study of Küçükkeleş et al.,[18] 4.61° distal tipping and 1.11 mm. intrusion movement were reported along with 4.17 mm molar distalization amount. The results of our study are similar to this study, but less molar distalization was obtained.

In the model analysis of the modified Veltri group, distobuccal rotation was observed in the right and left upper first molars after distalization. This rotation is thought to be caused by the relationship of the distalization force with the resistance point of the upper molar and the moment motion of the appliance. More body movement was obtained in the first class group compared to the Modified Veltri group. In the model analysis of the first class group, it was observed that mesiobuccal rotation occurred in the right and left upper first molars after distalization. In our study, doubling the activation amount of the vestibular screw in the appliance may have created a more severe buccal force and therefore may have caused mesiobuccal rotation. Itoh et al.[33] reported that mesiobuccal rotation ranging between 0°-29° occurred in their study with magnets.

The MV and FC appliances utilized in the present study employed conventional anchorage systems. Nevertheless, the anchorage losses observed in the present study could be mitigated through the utilization of the widely utilized mini screw-assisted molar distalization appliances. However, rotations were observed in maxillary molars.[34] No statistically significant differences were observed in the magnitude of molar distalization, molar distal tipping, or molar intrusion among appliances used in distalization, with the anchorage being placed in the palate, zygoma, or buccal area.[35]

In the modified Veltri group, there was an increase in the distance of the lower incisors to the reference PTV plane after distalization. We think that this increase, which occurred without protrusion of the lower incisors, is due to the counterclockwise rotation of the mandibular plane angle (SN-GoMe) during the distalization period. In our study, in both first class and Modified Veltri groups, almost all of the anchorage lost during the distalization period was recovered spontaneously with the distal movement of the second molars and even the second molar was dragged further distal than its initial position with the effect of interdental fibrils. It was observed that the distalization achieved during the stabilization period in the Modified Veltri group was lost by 1.13 mm and 0.3 mm in the first class group as a result of mesial movement. We think that more recurrences occurred in the Modified Veltri group due to excessive tipping of the upper first molar caused by molar distalization.

Changes in Soft Tissue

In the group with Modified Veltri and first class, it is seen that the lips approach the E plane in the sagittal direction due to the protrusion of the upper incisors after distalization. It suggests that the lower lip position at the end of reinforcement approaches to the E plane to some extent with the effect of growth. In the upper lip profile change, lip thickness is also important as well as the position of the incisors.[36] Although the amount of overjet was higher in the modified veltri group compared to the first class group, no statistically significant difference was found when the amount of soft tissue protrusion was compared between the groups.

CONCLUSION

Molar distalization was achieved in a similar time and at a similar rate with the modified veltri and first class appliances. Anchorage loss was less in the first class group. At the end of reinforcement, recurrence was observed similarly in the anchored tooth group and molars. The modified Veltri appliance was rotated in the mouth. Due to this rotation, intrusion movement occurred in the molars. In the clinical applications of these appliances, more effective clinical results can be achieved if anchorage losses are prevented with skeletal support applications.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study is a doctoral thesis completed before 2020. Institutional approval has been obtained.

Informed Consent

All patients signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Luppanapornlarp S, Johnston LE. The effects of premolar extraction: a long term comparison of outcomes in clear-cut extraction and nonextraction class II patients. *Angle Orthod.* 1993;63:257-272.
2. Dewel BF. Serial extraction: Precautions, Limitations and Alternatives. *Am J Orthod.* 1976;69:95-97.
3. Hazar S, Akyalçin S, Boyacioğlu H. Soft tissue profile changes in Anatolian Turkish girls and boys following orthodontic treatment with and without extractions. *Turkish J Med Sci.* 2004;34(3):171-178.
4. Gianelly AA, White WL. Anthony Gianelly on current issues in orthodontics. *J Clin Orthod.* 1996;30:239-446.
5. Philip C. Tandem concept in the nonextraction treatment of class II malocclusion. *Am J Orthod.* 1975;63:119-164.
6. Blechman AM. Magnetic force systems in orthodontics. Clinical results of a pilot study. *Am J Orthod Dentofac Orthop*. 1985;87: 201-210.
7. Gianelly AA, Bednar J, Dietz V. Japanese Ni-Ti coils used to move molars distally. *Am J Orthod Dentofac Orthop.* 1991;99:564-566.
8. Locatelli R, Bednar J, Dietz VS, Gianelly A. Molar distalization with superelastic NiTi wire. *J Clin Orthod.* 1992;26:277-279.
9. Hilgers JJ. The pendulum appliance for class II non-compliance therapy. *J Clin Orthod.* 1992;26:706-717.
10. Jones RD, White JM. Rapid class II molar correction with an open coil jig. *J Clin Orthod.* 1992;26:661-664.
11. Carano A. The distal jet for upper molar distalization. *J Clin Orthod.* 1996;26:374-380.
12. Keleş A. Maxillary unilateral molar distalization with sliding mechanics: a preliminary investigation. *Eur J Orthod.* 2001; 23:507-515.
13. Fortini A, Lupoli M, Parri M. The first class appliance for rapid molar distalization. *J Clin Orthod Dentofac Orthop*. 1999;33:322-328.
14. Veltri N. Espansione mascellare a 360 gradi. Sistematica dell'utilizzo di apparecchi fissi con vite per la correzione delle anomalie del mascellare superiore. *Boll di Inf Ortod Leone*. 1999; (63):25-28.
15. Giancotti A Cozza P. Nickel titanium double-loop system for simultaneous distalization of first and second molars. *J Clin Orthod.* 1998;255-260.
16. Gulati S, Kharbanda DP Parkash H. Dental and skeletal changes after intraoral molar distalization with sectional jig assembly. *Am J Orthod Dentofac Orthop.* 1998;(114):319-327.
17. Fortini A, Lupoli M, Giuntoli F, Franchi L. Dentoskeletal effects induced by rapid molar distalization with the first class appliance. *Am J Orthod Dentofac Orthop.* 2004;125:697-705.

18. Kucukkeles N, Cakirer B, Mowafi M. Cephalometric evaluation of molar distalization by hyrax screw used in conjunction with a lip bumper. *World J Orthod*. 7(3):261-268.

19. Bacetti T, Franchi L. A new appliance for molar distalization. *Repr from Ortho News*. 2001;1:2-6.

20. Keles A, Sayınsu K. A new approach in maxillary molar distalization: intraoral bodily molar distalizer. *Am J Orthod Dentofac Orthop*. 2000;117(1):39-48.

21. Pancherz H. The mechanism of class II correction in Herbst appliance treatment. *Am J Orthod.* 1982;82:104-113.

22. Chiu PP, McNamara JA, Franchi L. A comparison of two intraoral molar distalization appliances: Distal jet versus pendulum. *Am J Orthod Dentofac Orthop.* 2005;128(3):353-365.

23. Ricketts RM. The influence of orthodontic treatment on facial growth and development. *Angle Orthod.* 1960;30:103-133.

24. Steiner CC. Cephalometrics for you and me. *Am J Orthod.* 1953; 39:729-755.

25. Champagne M. Reliability of measurements from photocopies of study models. *J Clin Orthod.* 1992;26:648-650.

26. Bondemark L. A comparative analysis of distal maxillary molar movement produced by a new lingual intra-arch Ni-Ti coil appliance and a magnetic appliance. *Eur J Orthod.* 2000;22:683-695.

27. Doğan K, Başaran G, Hamamcı N, Hamamcı O. Noncompliance therapy: Veltri appliance. *World J Orthod.* 2009;10:1-6.

28. Haydar S, Uner O. Comparison of Jones jig molar distalization appliance with extraoral traction. *Am J Orthod Dentofacial Orthop.* 2000;117(1):49-53.

29. Papadopoulos MA, Melkos AB, Athanasiou AE. Noncompliance maxillary molar distalization with the First Class Appliance: a randomized controlled trial. *Am J Orthod Dentofac Orthop [Internet]*. 2010;137:586.e1-586.e13.

30. Bussick TJ, McNamara J. Dentoalveolar and skeletal changes associated with the pendulum appliance. *Am J Orthod Dentofac Orthop*. 2000;117:333-343.

31. Ghosh J, Nanda R. Evaluation of an intraoral maxillary molar distalization technique. *Am J Orthod Dentofac Orthop.* 1996;110: 639-646.

32. Ngantung V, Nanda RS, Bowman S. Posttreatment evaluation of the distal jet appliance. *Am J Orthod Dentofac Orthop.* 2001;120: 178-185.

33. Itoh T, Tokuda T, Kiyosue S, Hirose T, Matsumoto M. Molar distalization with repelling magnets. *J Clin Orthod.* 1991;25:611-617.

34. Vilanovaa L, Castillob A, Bellini-Pereiraa S, at al. Three-dimensional changes after maxillary molar distalization with a miniscrew-anchored cantilever. *Angle Orthod.* 2023;93(5):513-523.

35. Cerattia C, Serafinb M, Del Fabbroc M, Caprioglio A. Effectiveness of miniscrew-supported maxillary molar distalization according to temporary anchorage device features and appliance design: systematic review and meta-analysis. *Angle Orthod.* 2024;94(1): 107-121.

36. Maddalone M, Losi F, Rota E, Baldoni MG. Relationship between the position of the ıncisors and the thickness of the soft tissues in the upper jaw: cephalometric evaluation. *Int J Clin Pediatr Dent*. 2019;12(5):391.

Review
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Benign neoplasms of gingiva and alveolar mucosa

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ABSTRACT

Clinicians encounter a variety of oral lesions that can originate from various etiologies, such as from infective, idiopathic, inflammatory, reactive, and neoplastic changes. Neoplastic changes are rare compared with other affecting conditions, however, the oral cavity is one of the areas where tumors and tumor-like lesions most commonly develop and include both non-odontogenic and odontogenic lesions. Diseases affecting the oral mucosa are diverse and cover a broad spectrum of benign or malignant lesions. To make an accurate diagnosis, a clinician must take a comprehensive clinical history and have adequate information about the signs and symptoms, such as location, size, color, and morphology of the oral mucosal lesion. This review aims to describe the clinical, radiographic, microscopic, and treatment aspects of benign neoplasms that might affect the gingiva and alveolar mucosa.

Keywords: Benign lesions, gingiva, alveolar mucosa

INTRODUCTION

The term neoplasia means new growth[1] and tend to grow independently of adjacent tissues. Neoplasms are often called tumors although not all neoplasm are malignant. The clinical characteristics of a tumor allow it to be categorized as benign or malignant.[2]

Tumors have two main components:[3]

1. Parenchyma consisting of neoplastic cells
2. Supportive, host-derived, non-neoplastic stroma consisting of connective tissue and blood vessels, and host-derived inflammatory cells

The biological behavior of the tumor is determined by the parenchyma and the tumor is named after this component. The stroma serves as support for the growth of parenchymal cells.

COMPARISON OF BENIGN AND MALIGNANT TUMOR CHARACTERISTICS[4]

Benign and malignant tumors vary from each other according to their degree of differentiation, growth rate, local invasiveness, and metastatic potential. While benign tumors are similar to the tissue from which they originate and show good differentiation, malignant tumors are poorly differentiated or are completely undifferentiated (anaplastic). Benign tumors grow slowly, while malignant tumors usually grow faster. Benign tumors are well circumscribed and have a capsule structure, whereas malignant tumors are poorly circumscribed and invade adjacent tissues. While benign tumors remain localized at the site of origin, malignant tumors are locally invasive and can metastasize to distant sites.

BENIGN NEOPLASMS OF THE GINGIVA AND ALVEOLAR MUCOSA[5]

- Squamous papilloma
- Fibroma
- Giant cell granuloma
- Traumatic neuroma
- Neurofibroma
- Schwannoma
- Leiomyoma
- Hemangioma
- Lymphangioma
- Congenital epulis
- Peripheral odontogenic tumor
- Fibromatosis

Squamous Papilloma

This is a benign, asymptomatic, non-plaque-related gingival lesion with exophytic finger-like protrusions (Figure 1), thought to be caused by the human papilloma virus, formed by the proliferation of stratified squamous epithelium.[6] HPV type 6 and type 11 are associated with squamous papilloma[7] and

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it has also been suggested that this neoplasm can occur due to trauma.[8] In the intraoral region, it most commonly affects the palate, tongue, and lip mucosa.[9] Squamous papillomas are typically seen in individuals aged 30-50 years, but also reported in children under 10 years[8,10] and constituting approximately 8% of oral tumors in children.[10] Koilocytes are seen in the spinous layer of squamous papillomas and connective tissue shows varying degrees of keratinization.[11] They can exhibit as a pedicled lesion with a cauliflower-like surface.[8] They present as solitary masses rarely exceeding 5 mm and range from white to pink and red in appearance.[11]

Figure 1. Squamous papilloma and finger-like protrusions[11]

Differential diagnosis includes verruca vulgaris, verruciform xanthoma, condyloma acuminatum, giant cell fibroma, and squamous cell carcinoma.[12] Verruciform xanthoma is distinguishable as they are not parakeratinized. Condyloma acuminatum is mostly larger than squamous papilloma.[11] Treatment of squamous papilloma usually involves complete excision, but there remains the potential for recurrence.[6]

Fibroma

Fibromas are benign, reactive lesions resulting from prolonged irritation in the oral cavity that are often found on the buccal mucosa, lips, or along the lateral edges of the tongue.[13] Clinically, oral fibroma appear as a hard, smooth swelling that is similar in color to the surrounding soft tissue[14] (Figure 2). Fibromas have a dome-shaped structure, but can be pedunculated.[13] Although usually seen in older people, fibromas can occur at any age, but rarely affect adults.[15]

Figure 2. Irritation fibroma[16]

A biopsy should be performed after excision to eliminate other possible pathologies. Studies have shown the incidence of fibroma to range between 5% and 8.4% compared with similar oral pathologies. Conservative excision is preferred as a treatment and recurrence is not expected.[17]

Giant cell fibroma

First described by Weathers and Calliham in 1974, oral giant cell fibroma is a benign tumor characterized by the presence of star-shaped and giant multinucleated cells in the subepithelial fibrotic connective tissue.[18] It constitutes 2-5% of fibrous lesions in the oral cavity and idiopathic stimulation might have a role in its etiology[19], however, there are debates about its etiology.[20] Giant cell fibroma is generally more common in patients under age 30 years and predominant in women.[21]

Although it is most observed in the gums, the tongue and buccal mucosa are the most common sites.[22] It is seen as an asymptomatic exophytic lesion in the oral cavity, less than 1 cm in size, and can have a pedicle[21] (Figure 3). Surgical excision is the preferred treatment and recurrence is not expected.[22]

Figure 3. Giant cell fibroma[21]

Neurofibroma

Neurofibroma is a benign tumor originating from Schwann cells and perineural fibroblasts.[23] It is most commonly considered a skin lesion, but can also occur as an oral lesion. Neurofibroma is most commonly seen intraorally on the tongue and buccal mucosa, but studies report neurofibroma formation on the lips and gums.[24] These neoplasms present clinically as solitary or as a component of neurofibromatosis. The solitary type, which is more commonly found in younger people, appears as slow-growing, soft, painless lesions ranging from small nodules to larger masses.[25] Although neurofibromas can be seen centrally, they are rare. In rare cases, it can present in various forms on radiographic findings ranging from well-circumscribed to poorly circumscribed and with a unilocular or multilocular appearance (Figure 4).[26]

Histologically, neurofibromas are circumscribed and unencapsulated tumors consisting of spindle-shaped cells with elongated, thin nuclei and scant cytoplasm, surrounded by a collagen matrix located in a myxoid stroma.[28] Surgical excision is the preferred treatment and recurrence is not expected.[25]

Schwannoma

Schwannoma is a slow-growing, single, encapsulated tumor originating from the Schwann cells of the peripheral nerve sheath.[29] Of all nerve sheath tumors, schwannoma is the most common, accounting for approximately 89% of cases[30], however, its incidence in the oral cavity is rare.[31] The regions with the highest incidence of schwannoma lesions are the tongue, palate, floor of the mouth, buccal mucosa, lips, and gums[32] (Figure 5). Except for any peripheral, olfactory, and ocular cranial nerves containing Schwann cells that form the myelin sheath, other cranial nerves or autonomic neurons can be responsible for its etiology.[29]

Figure 4. Neurofibroma on the floor of the mouth[27]


Figure 5. Schwannoma lesion on the floor of the mouth[33]

Approximately 90% of schwannomas are sporadic and may develop together with neurofibromatosis type 1 (NF1), NF2, and schwannomatosis.[34] It is more common between the ages of 20-50 years and there is no gender predominance. Histopathological examination commonly shows an encapsulated tumor consisting of two different histopathological areas. Antoni a tissue has hypercellular spindle cells that palliate eosinophilic areas (verocay bodies) and is S100 positive. Antoni B tissue is hypocellular with loose connective tissue.[30] Schwannoma are generally solitary, smooth, mobile, slow-growing, and minimally invasive tumors. To eliminate the risk of recurrence, total surgical excision is the preferred treatment.[30]

Leiomyoma

Leiomyoma is a tumor of smooth muscle origin that is mostly associated with the gastrointestinal tract, uterus, and skin.[35] The oral cavity lacks smooth muscles other than the blood vessel wall, so its incidence in the mouth and maxillofacial region is low.[36] Intraoral lesions are most commonly found in the lips, tongue, buccal mucosa and palate, gingiva, and mandible.[36] Leiomyoma is characterized by an asymptomatic, slow-growing hard mass with average dimensions ranging from 1-2 cm and a history of less than one year[37] (Figure 6).


Figure 6. Leiomyoma[38]

Leiomyoma is usually seen in the oral cavity between the ages of 30-50 years.[36] Histologically, it contains small cells with eosinophilic cytoplasm and basophilic nuclei.[38] Diagnosis is difficult due to its nonspecific clinical appearance, therefore, histopathological examination and electron microscopy are used for definitive diagnosis. Complete excision is the preferred treatment.[39]

Hemangioma

Hemangiomas are a spectrum of congenital, benign vascular tumors recognized in neonates, infants, and children.[40] Occasionally, hemangiomas might not be noticed at birth, but presents the first 8 weeks of life.[41] There can be a period of remission in adulthood.[42] These lesions are characterized by hyperlocalized proliferation of endothelial cells with a central lumen.[40] It is especially common in the head and neck regions of women. Oral hemangiomas are most commonly seen on the lips, buccal mucosa, tongue, and palate.[43]

Clinically, oral hemangiomas usually appear as asymptomatic, reddish-blue or dark blue, soft, well-circumscribed, lobulated, sessile, or pedunculated (Figure 7). The sizes of these lesions can range from a few millimeters to a few centimeters.[44]

Differential diagnosis is supported by advanced imaging methods, such as Doppler ultrasonography or magnetic resonance imaging.[45] In cases where imaging methods are not sufficient, histopathological evaluation is considered the most reliable diagnostic method of oral hemangiomas.[44] When intraoral hemangioma lesions were examined by ultrasound, all lesions were submucosal, well-circumscribed, lobulated, unencapsulated, hypoisoechoic, had hyperechoic foci (echogenic septa), and heterogeneous lesion areas were detected.[46]

Figure 7. Submucosal hemangioma in the right molar region of the buccal mucosa[45]

Lymphangioma

Lymphangiomas are benign, hamartomatous malformations resulting from lymphatic tissue sequestration that is twice as common in men than women.[47] They are rarely seen in the oral cavity and when they do occur, are more common on the dorsum of the tongue, followed by the palate, buccal mucosa, gums, and lips.[48] Superficial lesions consist of raised nodules that are pink or yellowish in color (Figure 8). Deeper lesions appear as soft, diffuse masses with normal color.[49]

Figure 8. Blister-like lesions on the tongue[47]

Lymphangioma is a common cause of macroglossia in children, which is associated with swallowing and chewing difficulties, speech disorders, airway obstruction, mandibular prognathism, and open bite.[48] Tasca, Myatt and Beckenham[50] stated in their study that Ludwin's angina might develop depending on the infected base of the tongue lymphangioma. Histopathologically, they are divided into three groups: capillary, cavernous, and cystic. Marked dilatation of lymphatic vessels is evaluated by histopathology. With microscopic evaluation; small capillary-sized vessels are seen in the capillary type, large dilated lymph channels are present in the cavernous type, and large macroscopic cystic spaces are seen in the cystic type.[51] The cavernous type has the highest intraoral incidence.[52]

It is reported that lymphangiomas are associated with Turner syndrome, Noonan syndrome, trisomies, cardiac anomalies, fetal hydrops, and fetal alcohol syndrome.[53] Surgical excision is the preferred method in treatment. Alternatively, radiation therapy, cryotherapy, electrocautery, sclerotherapy, steroid application, embolization and ligation, Nd-YAG and CO2 laser surgery, or radiofrequency tissue ablation techniques can also be used.[48] Lymphangioma is difficult to completely remove due to its unencapsulated structure and infiltrating character, which increases the incidence of recurrence. Although large lesions in the neck and tongue can result in airway obstruction and death, the prognosis is mostly positive for patients.[54]

Epulis

Epulis is also called congenital granular cell epulis.[55] The color of these lesions usually resemble the oral mucosa and can be pedicled or sessile. Their size can vary between a few mm and 1 cm and are more common in female babies (~8-10:1).[56] Although more common in the maxilla than in the mandible, epulis occurs in the gingival mucosa of the alveolar crest in the anterior region (maxilla:mandibula ratio 3:1).[57] While most are single lesions, they can occur as multiple lesions in approximately 10% of cases.[58] Large or multiple lesions can cause airway obstruction and difficulty when feeding[59] (Figure 9).

Figure 9. Epulis[60]

Histologically, these lesions have large and round polygonal cells, eosinophilic granular cytoplasm, and round or oval slightly basophilic nuclei are seen.[57] Histopathologically, this lesion is similar to granular cell tumors seen in adults and is difficult to differentiate using a light microscope.[61] Immunohistochemical staining of S-100 often helps distinguish between the two, with granular cell tumor having positive staining.[61] Babies with airway and digestive tract obstruction should undergo surgery as soon as possible after birth.[62] From histopathological examination, numerous islands of proliferative squamous epithelium are observed and are clearly separated from the surrounding stroma by a flattened cell layer at the periphery.[63] Complete surgical excision is generally the preferred treatment and is successfully treated with local excision.[64] Epulis is usually asymptomatic and is detected by routine radiographic examinations or when tooth eruption is delayed.[65]

Fibromatosis

Fibromatosis is a benign lesion characterized by slowly progressing localized and generalized fibrous growth of the gingiva that does not exceed the mucogingival border. Fibromatosis could be hereditary, syndrome-related, drug-related, or due to inflammation.[66] It can be attributed to various etiological factors, such as poor oral hygiene, plaque accumulation, malabsorption, hormonal stimulation, various blood dyscrasias, or long-term use of certain drugs, such as phenytoin, nifedipine, or cyclosporine.[67] The hereditary type is an autosomal dominant condition with high genetic heterogeneity.[68] Hereditary gingival fibromatosis can be idiopathic or associated with other syndromes, such as Zimmermann–Laband syndrome or hypertrichosis; juvenile hyaline fibromatosis; Rutherfurd, Jones, and Ramon syndrome; and tuberous sclerosis.[69,70] Fibromatosis affects 1:175,000 people and there is no gender predominance.[70] Connective tissue defects due to gene mutations play a role in the pathogenesis of the disease.[71] Sex hormones and epidermal growth factor also play a role in the abnormal proliferation of gingival fibers.[72]

Fibromatosis appears asymptomatic and non-hemorrhagic, with a firm consistency and normal gingival color. Gingival enlargement can be either generalized or localized. It is usually observed as an idiopathic gingival fibromatosis type generalized lesion. Idiopathic gingival fibromatosis involves overgrowth of all parts of the gingiva on both the maxilla and the mandible (Figure 10).

Figure 10. Gingival fibromatosis[66]

Fibromatosis can cause functional and aesthetic problems.[66] Gum overgrowth is a large hard, flexible and dense fibrous tissue that expands over the teeth. It can cause negative aesthetic and psychological effects on patients by causing malocclusion; delayed eruption of permanent teeth; and speech, articulation, and chewing disorders.[73] Periodontal disease develops in fibromatosis patients.[74] The clinical and histological features of non-syndromic and syndromic gingival fibromatosis are similar.[75]

Treatment methods such as electrocautery-laser are used, but the most effective treatment is conventional gingivectomy.[74] Good oral hygiene should be maintained to reduce recurrence. However, genetic predisposition might also be a reasons for recurrence, therefore, even with good oral hygiene, the long-term treatment effects are unpredictable.[69]

CONCLUSION

The oral cavity is an area that is often inadequately examined in general practice. Oral lesions are relatively common conditions, but clinicians can find it difficult to distinguish benign from malignant lesions. Increased knowledge of common symptoms of oral lesions could increase the practitioner's confidence in performing oral examinations and managing any identified pathology. More importantly, physicians should keep in mind the red flags in oral pathology that could indicate malignancy.

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The authors have no conflicts of interest to declare.

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All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Schmidt CW. NTP nonneoplastic lesion atlas: a new tool for toxicologic pathology. *Environ Health Perspect*. 2014;122(3):A76-A79.
2. Kumar V. Robbins Basic Pathology. In: Kumar V, Abbas AK, Aster JC, eds. Robbins Basic Pathology. 10th ed. Elsevier:2023:189-196.
3. Schoen FJ. Tumors Associated with Biomaterials and Implants. In: Ratner BD, Hoffman AS, Schoen FJ, eds. Biomaterials Science. 3rd ed. Elsevier:2012:558-565.
4. Jang SJ, Gardner JM, Ro JY. Diagnostic approach and prognostic factors of cancers. *Adv Anat Pathol*. 2011;18(2):165-172.
5. Fowler CB. Benign and malignant neoplasms of the periodontium. *Periodontol 2000*. 1999;21(1):33-83.
6. Datta P, Panda A, Lenka S, Satpathy A. Squamous cell papilloma of the gingiva with a "garlanding a tooth" appearance: report of an unusual case. *J Indian Soc Periodontol*. 2020;24(6):572.

7. Syrjänen S. Oral manifestations of human papillomavirus infections. *Eur J Oral Sci*. 2018;126(S1):49-66.
8. Orenuga O, Oluwo A, Oluwakuyide R, Olawuyi A. Recurrent oral squamous papilloma in a pediatric patient: case report and review of the literature. *Niger J Clin Pract*. 2018;21(12):1674-1677.
9. Darwish G. Squamous papilloma of the soft palate: a case report. *Cureus*. 2023;15(4):e37423. doi: 10.7759/cureus.37423
10. Jaya A, Nagarathna C, Aishwarya N. A case report of squamous papilloma of the hard palate in a pediatric patient. *J Indian Soci Pedodont Prevent Dentistry*. 2020;38(1):91.
11. Betz SJ. HPV-related papillary lesions of the oral mucosa: a review. *Head Neck Pathol*. 2019;13(1):80-90.
12. Jaju PP, Suvarna PV, Desai RS. Squamous papilloma: case report and review of literature. *Int J Oral Sci*. 2010;2(4):222-225.
13. Javid K, Kurtzman GM, Boros AL. Oral fibroma removal with a Er, Cr: YSSG Laser. *Compend Contin Educ Dent*. 2020;41(5):e17-e22.
14. Jiang M, Bu W, Chen X, Gu H. A case of irritation fibroma. *Advanc Dermatol Allergol*. 2019;36(1):125-126.
15. Esmeili T, Lozada-Nur F, Epstein J. Common benign oral soft tissue masses. *Dent Clin North Am*. 2005;49(1):223-240.
16. Alsuwailem R, Elrahawy K, Nagy A. Fibroma of buccal mucosa: case report and literature review. *Eur Scientif J Febr*. 2017;13(6): 267-272.
17. Arslan S, Çobanoğlu B, Ural A, Sayğin İ, Işik AÜ. A 15-year retrospective study of 160 cases of benign lip lesions. *J Laryngol Otol*. 2015;129(12):1224-1227.
18. Magnusson BC, Rasmusson LG. The giant cell fibroma a review of 103 cases with immunohistochemical findings. *Acta Odontol Scand*. 1995;53(5):293-296.
19. Jimson S. Giant cell fibroma: a case report with immunohistochemical markers. *J Clin Diagnost Res*. 2013;7(12): 3079. doi: 10.7860/JCDR/2013/6476.3859
20. Sabarinath B, Sivaramakrishnan M, Sivapathasundharam B. Giant cell fibroma: a clinicopathological study. *J Oral Maxillofac Pathol*. 2012;16(3):359.
21. Bagheri F, Rahmani S, Azimi S, Bigom Taheri J. Giant cell fibroma of the buccal mucosa with laser excision: report of unusual case. *Iran J Pathol*. 2015;10(4):314-317.
22. Kuo RC, Wang YP, Chen HM, Sun A, Liu BY, Kuo YS. Clinicopathological study of oral giant cell fibromas. *J Formosan Med Assoc*. 2009;108(9):725-729.
23. Neville BW, Damm DD. Oral and maxillofacial pathology. 4th ed. Elsevier Health Sciences: 2015.
24. Powell CA, Stanley CM, Bannister SR, McDonnell HT, Moritz AJ, Deas DE. Palatal neurofibroma associated with localized periodontitis. *J Periodontol*. 2006;77(2):310-315.
25. Sekhar P, Nandhini G, Kumar Kr, Kumar Ar. Solitary neurofibroma of the palate mimicking mucocele: a rare case report. *J Oral Maxillofac Pathol*. 2019;23(4):23.
26. Allen CM, Miloro M. Gingival lesion of recent onset in a patient with neurofibromatosis. *Oral Surg Oral Med Oral Pathol Oral Radiol Endodontol*. 1997;84(6):595-597.
27. Broly E, Lefevre B, Zachar D, Hafian H. Solitary neurofibroma of the floor of the mouth: rare localization at lingual nerve with intraoral excision. *BMC Oral Health*. 2019;19(1):197.
28. Vivek N, Manikandhan R, James P, Rajeev R. Solitary intraosseous neurofibroma of mandible. *Indian J Dental Res*. 2006;17(3):135.
29. Swain SK, Samal S, Das S, Padhy R. A large intraoral sublingual schwannoma in a pediatric patient: a case report. *Iran J Otorhinolaryngol*. 2021;33(118):335-337.
30. Sheikh MM, De Jesus O. Vestibular Schwannoma. [Updated 2023 Aug 23]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK562312/
31. Cohen M, Wang MB. Schwannoma of the tongue: two case reports and review of the literature. *Eur Arch Oto-Rhino-Laryngol*. 2009;266(11):1823-1829.
32. da Silva LFM, Duarte BG, Boiça BA, Rocha-Junior HV, Pereira-Stabile CL. Intraoral schwannoma: a case report. *Oral Maxillofac Surg*. 2013;17(4):319-321.
33. Farooqui A, Khalekar Y, Farooqui J, Kasat V. Schwannoma of floor of mouth: a case report. *J Indian Acad Oral Med Radiol*. 2017;29(2):135.
34. Bakir M, Enabi J, Almeshal H. Unusual lower lip swelling: a rare case of lip schwannoma. *Cureus*. 2021;13(11):e19242. doi: 10.7759/cureus.19242
35. Campelo VES, Neves MC, Nakanishi M, Voegels RL. Nasal cavity vascular leiomyoma - case report and literature review. *Braz J Otorhinolaryngol*. 2008;74(1):147-150.
36. Veeresh M, Sudhakara M, Girish G, Naik C. Leiomyoma: a rare tumor in the head and neck and oral cavity: report of 3 cases with review. *J Oral Maxillofac Pathol*. 2013;17(2):281.
37. Kaur G, Gondal R. Oral leiomyoma. *J Oral Maxillofac Pathol*. 2011;15(3):361.
38. Gianluca S, Marini R, Tonoli F, Cristalli MP. Leiomyoma of oral cavity: case report and literature review. *Ann Stomatol*. 2011;2(1-2):9-12.
39. Dutt KC, Bindra S, Awana M, Talwar M, Lehl G. Intraosseous leiomyoma of the mandible: a case report of the rare entity and review of literature. *J Maxillofac Oral Surg*. 2017;16(2):145-151.
40. Balaji S, Balaji P. Large hemangioma of the tongue. *Indian J Dental Res*. 2020;31(6):979.
41. Azma E, Razaghi M. Laser treatment of oral and maxillofacial hemangioma. *J Lasers Med Sci*. 2018;9(4):228-232.
42. Neville BW, Damm DD, Allen CM, Bouqout JE. Oral and Maxillofacial Pathology. 3rd ed. Elsevier: 2009.
43. Narwal A, Sen R, Singh V, Gupta A. Masson′ s hemangioma: a rare intraoral presentation. *Contemp Clin Dent*. 2013;4(3):397.
44. Reddy KVK, Roohi S, Maloth K, Sunitha K, Thummala VR. Lipoma or hemangioma: a diagnostic dilemma? *Contemp Clin Dent*. 2015;6(2):266.
45. Ademi Abdyli R, Abdyli Y, Perjuci F, Gashi A, Agani Z, Ahmedi J. Sclerotherapy of intraoral superficial hemangioma. *Case Rep Dent*. 2016;2016:4320102.
46. Derindağ G, Sarıca İ, Çağlayan F. Examination of oral hemangiomas by intraoral ultrasonography. *Oral Radiol*. 2021; 37(4):687-692.
47. Sunil S, Gopakumar D, Sreenivasan BS. Oral lymphangioma-case reports and review of literature. *Contemp Clin Dent*. 2012;3(1): 116-118.
48. Asokan GS. Lymphangioma of the tongue-a case report and review of literature. *J Clin Diagnost Res*. 2014;8(9):ZD12. doi: 10.7860/JCDR/2014/9890.4792
49. Goswami M, Singh S, Gokkulakrishnan S, Singh A. Lymphangioma of the tongue. *Natl J Maxillofac Surg*. 2011;2(1):86.
50. Tasca RA, Myatt HM, Beckenham EJ. Lymphangioma of the tongue presenting as Ludwig's angina. *Int J Pediatr Otorhinolaryngol*. 1999;51(3):201-205.
51. Guelmann M, Katz J. Macroglossia combined with lymphangioma: a case report. *J Clin Pediatr Dentistry*. 2004;27(2):167-169.
52. Mosca RC, Pereira GA, Mantesso A. Cystic hygroma: characterization by computerized tomography. *Oral Surg Oral Med Oral Pathol Oral Radiol Endodontol*. 2008;105(5):e65-e69.
53. Tewari A, Munjal M, Kamakshi, Garg S, Sood D, Katyal S. Anaesthetic consideration in macroglossia due to lymphangioma of tongue: a case report. *Indian J Anaesth*. 2009;53(1):79-83.
54. Rajendran R, Sivapathasundaram B. Shafer's Textbook of Oral Pathology. 6th ed. Elsevier: 2009.

55. Ye Y, Tang R, Liu B, Li Y, Mo Y, Zhang Y. Prenatal diagnosis and multidisciplinary management: a case report of congenital granular cell epulis and literature review. *J Int Med Res*. 2021; 49(10):030006052110537.

56. Conrad R, Perez MCN. Congenital granular cell epulis. *Arch Pathol Lab Med*. 2014;138(1):128-131.

57. Godra A, D'Cruz CA, Labat MF, Isaacson G. Pathologic quiz case: a newborn with a midline buccal mucosa mass. *Arch Pathol Lab Med*. 2004;128(5):585-586.

58. Yuwanati M, Mhaske S, Mhaske A. Congenital granular cell tumor-a rare entity. *J Neonatal Surg*. 2015;4(2):17.

59. Bosanquet D, Roblin G. Congenital epulis: a case report and estimation of incidence. *Int J Otolaryngol*. 2009;2009:508780.

60. Pekçetin ZS, Senemtaşı A, Koçak GE, Kumral S, Yaltirik M, Koray M. Congenital epulis of the newborn: a case report. *Open J Stomatol*. 2018;08(04):120-126.

61. Cheung JM, Putra J. Congenital granular cell epulis: classic presentation and its differential diagnosis. *Head Neck Pathol*. 2020;14(1):208-211.

62. Kokubun K, Matsuzaka K, Akashi Y, et al. Congenital epulis: a case and review of the literature. *Bull Tokyo Dent Coll*. 2018;59(2):127-132.

63. do Rosário Mardones N, de Oliveira Gamba T, Flores IL, De Almeida SM, de Castro Lopes SLP. Squamous odontogenic tumor: literature review focusing on the radiographic features and differential diagnosis. *Open Dent J*. 2015;9(1):154-158.

64. Anjana R, Murali N, Malathi N, Suresh R. Management of a rare case of peripheral squamous odontogenic tumor of the gingiva. *J Indian Soc Periodontol*. 2016;20(3):336-339. doi: 10.4103/0972-124X.179897

65. de Oliveira MA, Reis B, Pallos D, Kim YJ, Braz-Silva PH, Martins F. The importance of histopathological examination to the final diagnosis of peripheral odontogenic tumors: a case report of a peripheral odontoma. *Case Rep Dent*. 2019;2019:9712816.

66. Papadopoulos KS, Pantazidou G, Karagkouni E, Papadopoulos G, Papaioannou I. Local mucosal flap for the treatment of gingival defect after gingival fibromatosis excision. *Cureus*. 2020;12(12): e12016 doi: 10.7759/cureus.12016

67. Lai B, Muenzer J, Roberts MW. Idiopathic gingival hyperplasia: a case report with a 17-year followup. *Case Rep Dent*. 2011;2011: 986237.

68. Resende EP, Xavier MT, Matos S, Antunes AC, Silva HC. Nonsyndromic hereditary gingival fibromatosis: characterization of a family and review of genetic etiology. *Special Care Dentistry*. 2020;40(3):320-328.

69. Coletta RD, Graner E. Hereditary gingival fibromatosis: a systematic review. *J Periodontol*. 2006;77(5):753-764.

70. Yadav A, Kumari A, Bansal MB, Asrani KH. Nonsyndromic with recurrent idiopathic gingival fibromatosis: a rare case report. *Int J Clin Pediatr Dent*. 2021;14(1):158-160.

71. Gao Q, Yang K, Chen D, et al. Antifibrotic potential of MiR-335-3p in hereditary gingival fibromatosis. *J Dent Res*. 2019;98(10): 1140-1149.

72. Shi J, Lin W, Li X, Zhang F, Hong X. Hereditary gingival fibromatosis: a three-generation case and pathogenic mechanism research on progress of the disease. *J Periodontol*. 2011;82(7): 1089-1095.

73. Li N, Wang W, Sun Y, Wang H, Wang T. Seven-year follow-up of a patient with hereditary gingival fibromatosis treated with a multidisciplinary approach: case report. *BMC Oral Health*. 2021; 21(1):473. doi: 10.1186/s12903-021-01830-7

74. Boutiou E, Ziogas IA, Giannis D, Doufexi AE. Hereditary gingival fibromatosis in children: a systematic review of the literature. *Clin Oral Investig*. 2021;25(6):3599-3607.

75. Costa CRR, Braz SV, de Toledo IP, et al. Syndromes with gingival fibromatosis: a systematic review. *Oral Dis*. 2021;27(4):881-893.

Case Report
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Surgical treatment of chronic sinus pain developing after dental implant placement: a case report with 3-month follow-up

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ABSTRACT

Dental implant surgery in the maxillary posterior region with insufficient alveolar bone height can be performed in one or two stages, depending on the amount of residual bone. Anatomical structures in the area where the procedure will be performed increase surgical sensitivity, and failure to make the necessary preparations may lead to various complications. During dental implant surgical procedures, various complications may be encountered in the relevant area. A 56-year-old male patient applied to our clinic with the complaint of severe and persistent pain. With panoramic and cone beam computed tomography evaluation, it was determined that the apical parts of the last two implants were in the sinus. After obtaining consent, two adjacent implants were explanted within the same session on the planned day, and the procedure area was closed with buccal fat tissue obtained from the patient and autologous platelet-rich fibrin.

Keywords: Buccal fat pad, dental implant, explantation, maxillary sinus, sinus lifting

INTRODUCTION

Dental implants are increasingly used in the oral rehabilitation of partially and completely edentulous areas.[1] This method is preferred by many physicians over traditional fixed or removable partial dentures because its functionality and aesthetics are similar to natural teeth.[2-4] However, in these procedures, it is necessary to pay attention to the anatomical structures during the application and if there is a pathological condition, it is necessary to postpone the procedure or turn to a different option. Preliminary evaluation of the remaining alveolar bone height and sinus during implant placement in the upper jaw posterior edentulous area is very important for the success and prognosis of the surgical procedure. The presence of a pathological kondüitin in the area may result in acute infection, pain-fever-swelling-discharge, and failure of implant surgery. In the presence of chronic sinusitis, surgical procedures may cause the chronic process to turn into an acute painful form. If any pathology is detected in the patient's history or imaging during the evaluation of the sinus, ear, nose and throat consultation is required. Our aim in this case report is to present the management of the surgical field by removing the factors that cause maxillary sinus-related pain from the area. The only solution in treatment management is to carefully remove the source of pain from the area and use safe antibiotics.

CASE

A 56-year-old male patient applied to Kırıkkale University Faculty of Dentistry Periodontics Clinic with the complaint of persistent pain in the left maxillary sinus area (Figure 1).

Figure 1. Panoramic image of the patient

According to the anamnesis, it was learned that the patient had no systemic disease, had a history of maxillary sinusitis, and had a dental implant procedure 4 months ago. When intraoral examination was performed, there was no redness around the implants, no bleeding on probing, no pus formation, and no deep probing depth was observed. With panoramic (Figure 1)

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and CBCT evaluation, it was determined that the apical part of the last two implants was located in the maxillary sinus and there was a thickening of the Scheneiderian membrane. Based on the patient's complaints and radiographic evaluation, the ear, nose and throat clinic was consulted. It was decided to remove the implants located in the area of teeth 26 and 27. The patient was informed about the procedure. 1 day before the procedure, once a day Ceftinex 600 mg (PharmaVision Industry and Tic. Inc., İstanbul, Turkiye) twice a day, Kloroben 1.5 mg/ml+1.2 mg/ml mouthwash (Drogsan Pharmaceutical Industry. and Tic. Inc., Ankara, Turkiye) and Bi-profenid 100 mg (Sanofi Pharmaceutical Industry. and Tic. Inc., Kırklareli, Turkiye) was started 1 hour before the procedure.

Following routine surgical preparations, an envelope flap was created to provide a comfortable field of view. Bone tissue was removed around both implants under serum cooling with rond drills and trephane drills, respectively. The released implants were removed without any problems by moving them counterclockwise towards the oral cavity (Figure 2). The cavity was evaluated. Granulation tissues were cleaned and irrigation was performed with physiological saline and rifampicin (Figure 3).

Figure 2. Removing implants

Figure 3. Checking the defect

After the irrigation process, a 1 cm horizontal incision was made on the mucosa at the level of the 2nd molar tooth to reach the cheek fat tissue. The buccinator muscle was passed and the buccal fat tissue was reached. The liberated fat tissue was excised and placed in the sockets of the resulting implants (Figure 4).

Figure 4. Transfer and adaptation of buccal fat tissue to the cavity

In addition, the membrane obtained from 2 tubes of autologous PRF obtained from the patient was covered over the fat tissue. Both incision lines were closed primarily using 4.0 silk suture (Figure 5). Precautions and recommendations after the procedure were explained. Ceftinex 600 mg once a day (PharmaVision Industry and Tic. Inc., İstanbul, Turkiye), Kloroben[1] 1.5 mg/ml+1.2 mg/ml mouthwash twice a day (Drogsan Pharmaceuticals Industry. and Tic. Inc., Ankara, Turkiye), Bi-profenid 100 mg twice a day (Sanofi Pharmaceutical Industry. and Tic. Inc., Kırklareli, Turkiye), Aerius 5 mg once a day (Sanofi Pharmaceutical Industry. and Tic. Inc., Kırklareli, Turkiye), Otrivine care 1 mg/1 ml (Zentiva Health Products Industry and Trade Inc., Kırklareli, Turkiye) was prescribed twice a day. Cold application was recommended on the day of the procedure and the day after. A follow-up appointment was made for 14 days later.

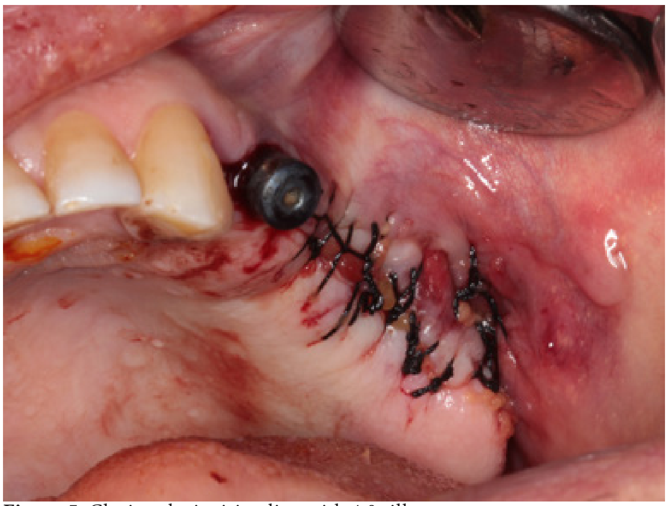

Figure 5. Closing the incision line with 4.0 silk suture

After the procedure, a periapical image was taken from the relevant area (Figure 6).

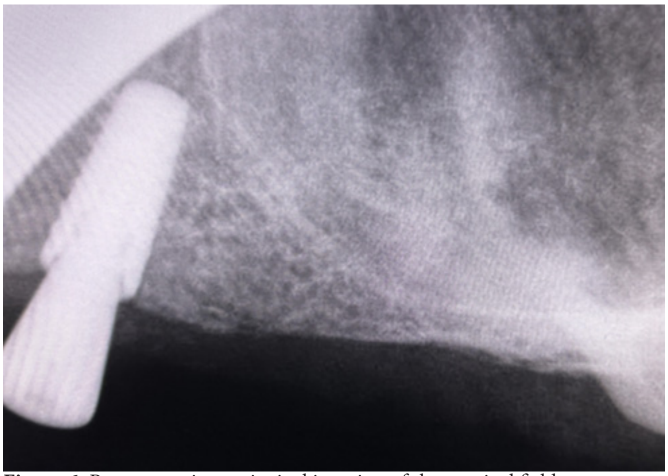

Figure 6. Post-operative periapical imaging of the surgical field

During the postoperative follow-up, recovery was uneventful. The patient reported pain in the upper left cheek area for the first day and swelling that started the next day. The stitches were removed on the 14th day after surgery. At a one-month follow-up, it was observed that the pain was gone and the closure of the wound areas was in proper form. After the surgery, the patient was successfully followed for 3 months and no problems were observed.

DISCUSSION

Maxillary sinusitis is a common and important complication after dental implant surgery. A review of the literature revealed numerous case reports. In this case report, the procedure and recovery process applied to a patient who applied to the clinic with the complaint of sinus pain, which partially decreased during use and then increased again, although he used different types of antibiotics and painkillers many times after the procedure, was reported.

Factors affecting maxillary sinus pneumatization and alveolar bone resorption include prosthetic rehabilitation, causes of tooth loss, and muscle activities.[5] During the edentulous period, the amount of remaining bone decreases due to these factors. Dental implants planned for the area can be placed in one or two stages by elevating the sinus floor. For an appropriate treatment, it is important to carefully evaluate the sinus characteristics. Sinus physiology is affected by the variable anatomy of the sinus floor and conditions such as extension and perforation of the elevated membrane.

Additionally, postoperative hematoma and swelling may play a key role in the development of sinusitis by leading to decreased osteomeatal patency.[6] In patients with chronic maxillary sinusitis, changes in the physiology of the chronically infected sinus due to damage to the Scheneiderian membrane during the procedure may cause acute sinusitis in the post-procedure period.[6] 10% of sinusitis cases are odontogenic in etiology, this rate increases to 40% in different reports. Common causes of odontogenic sinusitis vary; These include inflammatory cysts, odontogenic cysts, peri-implantitis and foreign bodies. Studies show that although the majority of odontogenic sinusitis sources are dental-related, an increasing number of cases tend to be caused by dental implants and augmentation procedures. In a retrospective study (including 480 patients), implant-related etiology was reported to be 30%.[7] In our study, implant-related sinusitis was present, and when we evaluated different studies, removal was indicated.[8] It is recommended to use a Bichat fat pad as a large defect will appear after the removal of the implants, the vitality of the hard tissue will be impaired and the possibility of oro-antral fistula development is high.[9] In this case, the existing gaps were closed with Bichat fat pad and autologous PRF. In cases where chronic symptoms are present in the sinus, removal of the implant and use of Bichat fat pad or palatal flap shifting techniques to prevent oroantral fistula development may increase success in long-term follow-up after the procedure. Even though the features of dental implants improve, complications are inevitable. Today, the relationship between dental implants and sinusitis needs to be understood and taken into consideration. The SCDDT treatment protocol recommended for sinusitis-related implants is compatible with our study.[10] Effective treatment must be applied in a way that does not cause or cause inflammation in patients.

CONCLUSION

In this article, we present the 6-month follow-up of the patient who was treated by removing the implants and closing the gap with buccal fat tissue and autologous PRF as a result of persistent pain after surgery. In the presence of an inflammatory condition in the maxillary sinus, it may be necessary to pre-evaluate the area in detail before surgery, suppress the inflammation, or plan different dental procedures by protecting the area.

REFERENCES

1. Kang DW, Kim SH, Choi YH, Kim YK. Repeated failure of implants at the same site: a retrospective clinical study. *Maxillofac Plast Reconstr Surg.* 2019;41:27.
2. Wolff R, Hsu A, Heo Y, et al. Two-year follow-up comparison of three surgical techniques for implant placement in posterior maxilla with limited alveolar bone height. *Int J Oral Maxillofac Implant.* 2022;37:171-180.
3. Maruyama N, Maruyama F, Takeuchi Y, et al. Intraindividual variation in core microbiota in peri-implantitis and periodontitis. *Sci Rep.* 2014;4:6602.
4. Hainarosie R, Pietrosanu C, Cherecheanu A, et al. Risk factors for dental implant failure and medicolegal implications. *Rom J Leg Med.* 2019;27:52-56.
5. Lovasova K, Kachlik D, Rozpravkova M, Matusevska M, Ferkova J, Kluchova D. Three-dimensional CAD/CAM imaging of the maxillary sinus in ageing process. *Ann Anat-Anatomisc Anzeiger.* 2018;218:69-82.
6. Kozuma A, Sasaki M, Seki K, et.al. Preoperative chronic sinusitis as significant cause of postoperative infection and implant loss after sinus augmentation from a lateral approach. *Oral Maxillofac Surg.* 2017;21(2):193-200.
7. Molteni M, Bulfamante AM, Pipolo C, et al. Odontogenic sinusitis and sinonasal complications of dental treatments: a retrospective case series of 480 patients with critical assessment of the current classification. *Acta Otorhinolaryngol Ital.* 2020;40(4):282-289.
8. Saibene AM, Pipolo C, Borloni R, Felisati G. ENT and dentist cooperation in the management of odontogenic sinusitis: a review. *Acta Otorhinolaryngol Ital.* 2021;41(1):116-123.

9. Bravo Cordero G, Minzer Ferrer S, Fernández L. Odontogenic sinusitis, oro-antral fistula and surgical repair by Bichat's fat pad: literature review. *Acta Otorrinolaringol Esp.* 2016;67(2):107-113.

10. Saibene AM, Collurà F, Pipolo C, et al. Odontogenic rhinosinusitis and sinonasal complications of dental disease or treatment: prospective validation of a classification and treatment protocol. *Eur Arch Otorhinolaryngol.* 2019;276(2):401-406.