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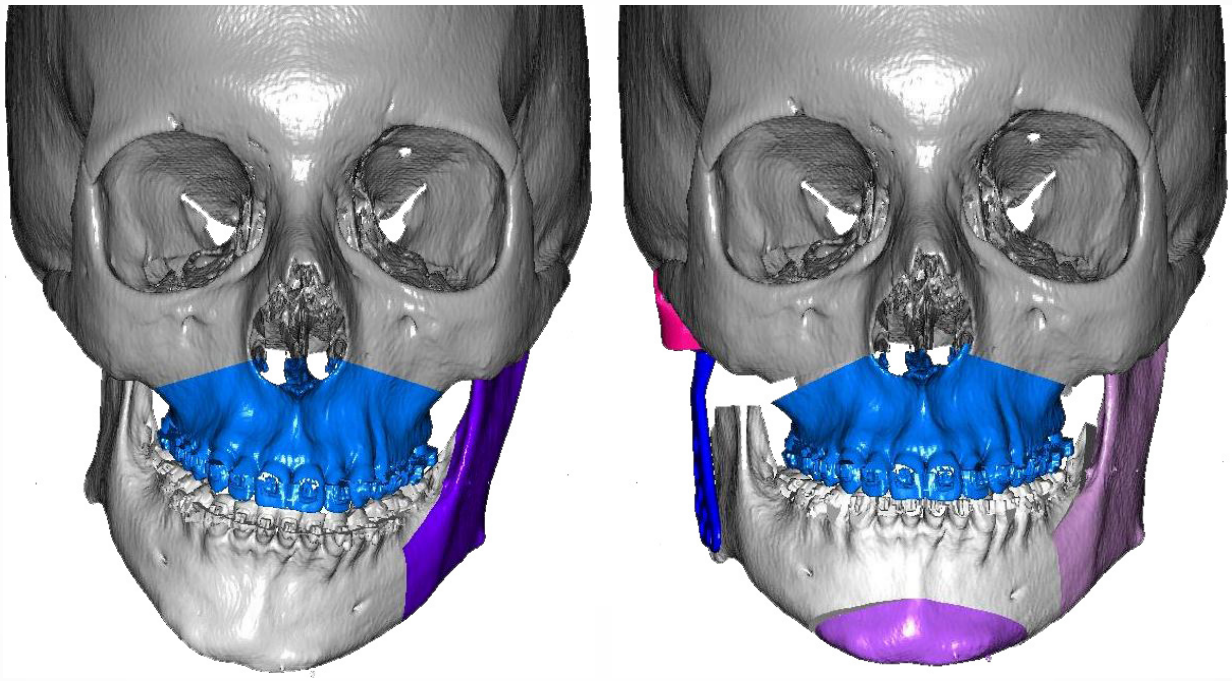

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Highly Predictable Augmentation of the Alveolar Ridge: Using a Ribbed Titanium Mesh in Two Stage Implant Surgery

Pediatric Surgery

Determination of the Coronoid Process Hyperplasia of the Mandible in Ankylosing Diseases of the Temporomandibular Joint in Children

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Journal of ***Diagnostics & Treatment of Oral & Maxillofacial Pathology*** goals to publish the cutting-edge and peer-reviewed articles on work in oral and maxillofacial surgery and neighboring specialties. The journal includes the following topics: implants surgery, head and neck imaging, microvascular and reconstructive surgery, oral and maxillofacial pathology, head and neck surgery/oncology, TMJ lesions/disorders, head and neck trauma, plastic surgery, pharmacology/drugs.

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Content

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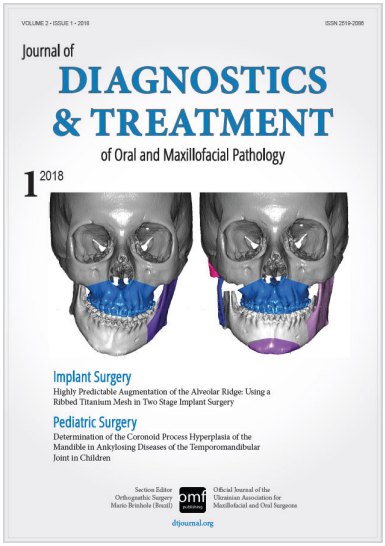

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On the lower image in hospital Dr. Mario Brinhole (on the left) with colleague Dr. Jose Galiano (on the right).

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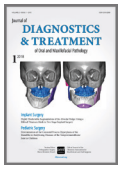

Determination of Coronoid Process Hyperplasia of the Mandible Upon Ankylosing Diseases of the Temporomandibular Joint in Children*

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ABOUT ARTICLE

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Electromyography of masticatory muscles

ABSTRACT

Purpose.
Ankylosing diseases of the temporomandibular joint (ADTMJ) in children – bone ankylosis and secondary deforming osteoarthrosis (SDOA) lead to an increase in the coronoid process (CP) on average by 1.5 times. The slice computed tomography (SCT) allows fully determining the changes occurring in the bone structures of the joint with its ankylosing diseases. The purpose of the work was to determine the parameters coronoid process, which affects the limitation of mouth opening, and indications for its resection in the ADTMJ, based on the treatment of SCT data.
Material and Methods.
The subject of the study were 33 SCT in children aged 6 to 14 years with ADTMJ and without lesions of TMJ. Anthropometric measurements of CP in children of the three groups were performed according to the proposed modified scheme of Levandoski panographic analysis.
Conclusion.
The proposed scheme of anthropometric measurements of SCT allows us to mathematically substantiate the stage of hyperplasia coronoid process in children and to determine the necessity of its surgical correction.

Introduction

Ankylosing diseases of the temporomandibular joint (ADTMJ) in children – bone ankylosis and secondary deforming osteoarthrosis (SDOA), make up from 53% to 86% among all joint diseases and 8-11% of surgical stomatological diseases in children [1, 3-5, 7].

The bone deformity, which constantly accompanies ADTMJ, is hyperplasia of the coronoid process (CP). According to various authors in ankylosing diseases, it increases by 1.5 times [6, 5, 10, 12, 13].

Timely and reliable diagnosis of bone ankylosis and SDOA TMJ is the basis for choosing the optimal individual therapeutic tactics [2, 3, 5, 6, 11, 15]. CT is used today in all diagnostic protocols to find out changes in the bone joint elements at ADTMJ [3, 6, 8, 9, 13-15]. The value of CT is that 3D image allows to evaluate the nature and extent of pathological changes not only in the joint, but also the processes of the lower jaw, the facial bones and their interrelations. To determine the degree of explosives used methodology by Levandoski [10-12, 14]. The latter involves calculating the height, the width of the base of the CP, the angle between the condylar process and coronoid process. The Levandoski method detects the presence of hyperplasia of CP, but does not answer the question of which exactly changed parameters require resection of the CP [10, 15].

The purpose of the work: to determine the parameters of hyperplasia of CP of mandible, which affect the limitation of mouth opening, and indications for its resection in the ADTMJ on the basis of data processing slice computed tomography (SCT).

Material and Methods

The subject of the study were 33 SCT children aged 6 to 14 years. Depending on the damage of TMJ is divided into three groups: I group – 8 children with SDOA TMJ, II group – 6 patients with bone ankyloses TMJ, III – control group of 19 children without TMJ lesions.

Anthropometric measurements of CP in children of the three groups were performed according to the

* This manuscript has not been presented

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proposed modified scheme of Levandoski panographic analysis. S is the width of the basal CP, measured as the anterior-posterior dimension from the lower point of the incisure of the mandibular branch, lowering the perpendicular to the leading edge of the basal coronoid process (Fig 1). The height (h) of the CP was determined as the perpendicular that was lowered from the top of the coronoid process to the line S. The angle α – is formed between the CP and the condylar process had sides passing between the highest points of the processes of the mandible and the lowest point of the incisure of mandible. Additionally, measure the distance L, which was determined between the top of the CP and the inner surface of the zygoma, as an indicator of the degree of mobility of the mandible. For the data processing of SCT, the Horos program was used.

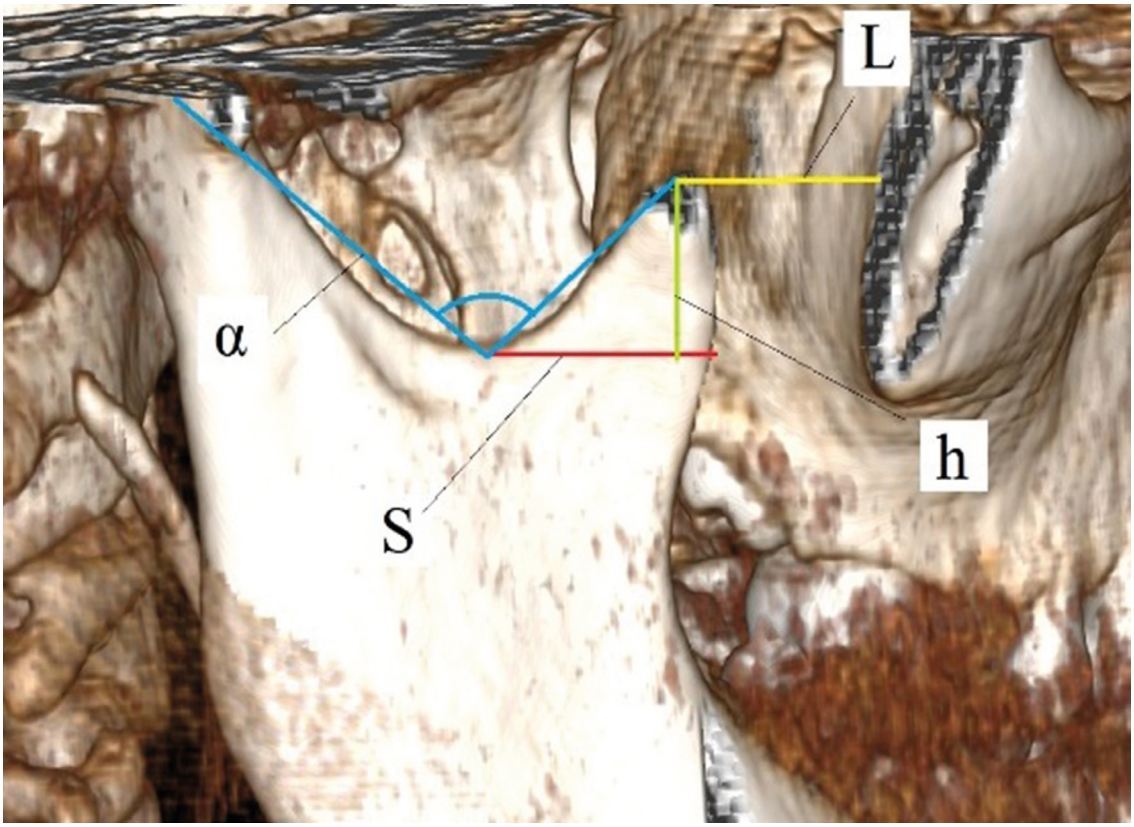

FIGURE 1. 3D reconstructed CT image. Scheme of anthropometric measurements of explosives according to the proposed modification. The height (h) of the CP was determined as the perpendicular that was lowered from the top of the coronoid process to the *red line S*. The angle α – formed between *blue lines* (CP and the condylar process) had sides passing between the highest points of the processes of the mandible and the lowest point of the incisure of mandible. Additionally, measure the distance L (*yellow line*), which was determined between the top of the CP and the inner surface of the zygoma, as an indicator of the degree of mobility of the mandible.

Electromyography (EMG) of temporal muscle was performed on an electromyograph of the type EEG-16 S Medicor®, Buclapest, Hungary. The difference in the muscle biopotentials was recorded by the biopolar method from the healthy and affected side, and the bioelectric activity (BEA) of the temporal muscle in the phase of relative rest (X1) and in the active phase (X2) was determined. The measurement range was the standard deviation.

Results

The technique of paired analysis of Levandoski by us was modified by introducing a new index of mobility of the jaw L. This indicator is a summary reflection of the changes that occur with CP. Increasing the height and width of the bases of the CP helps to change its position, namely the top and the back. This, in turn, leads to a decrease in the angle between the CP and the condylar proces, which affects the spatial displacement of the blood vessels in the movements of the lower jaw. In hyperplasia CP, this distance significantly decreases and when the jaw moves in the sagittal plane, it is blocked. Indicator L is fundamentally important for determining the mobility of the mandible (Fig 2). When opening the mouth there is a reduction of chewing muscles, the head of the condylar process of the mandible performs sliding movements, moves to the articular hump. Together with it, it moves in the sagittal direction and the CP, the apex of which approaches the inner surface of the chick bone and the mouth opens. With reduced distances between the top of the CP and the zygoma, there is a blockage of opening the mouth due to the contact of these bone anatomical formations, which is normally absent.

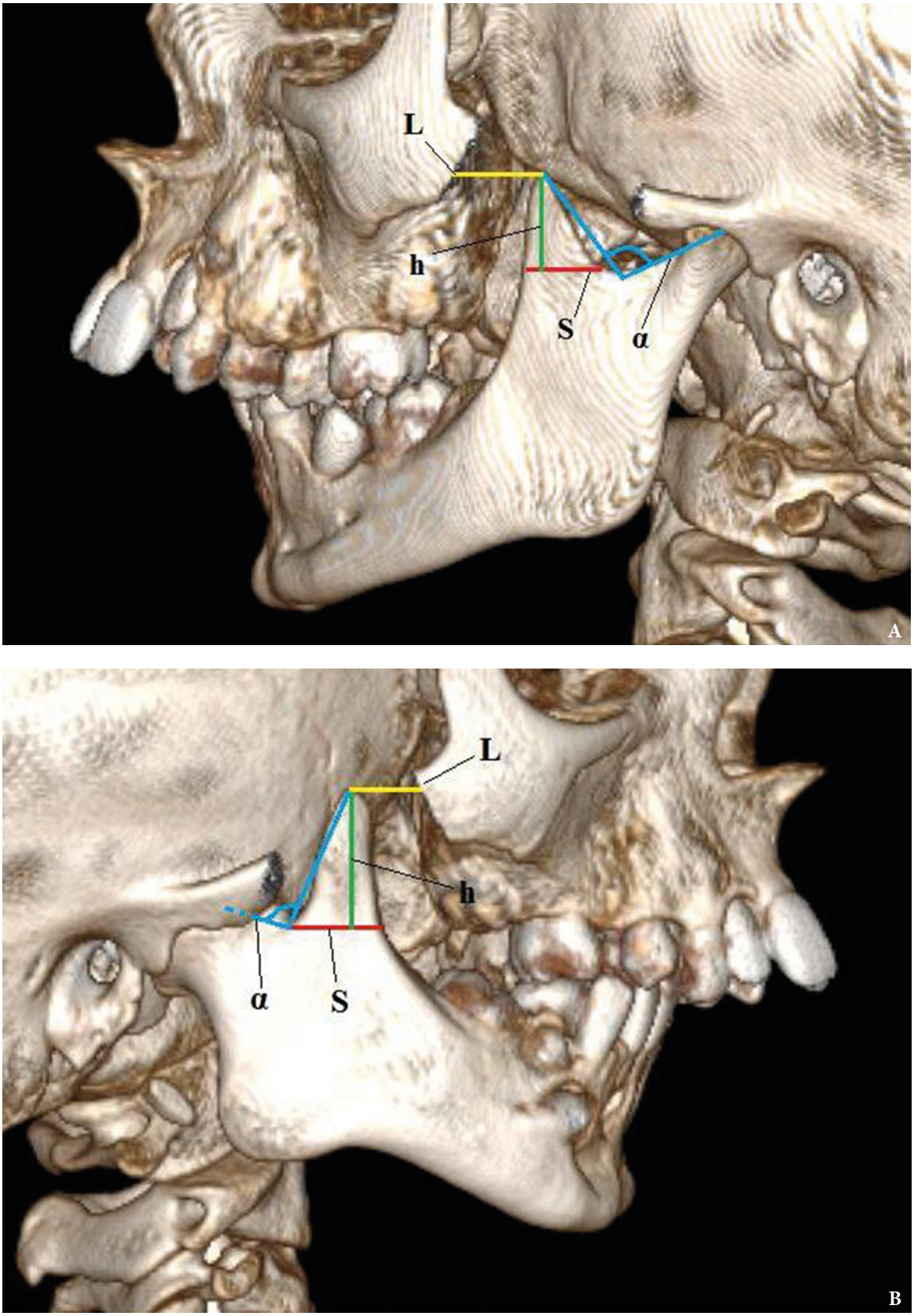

FIGURE 2. 3D reconstructed CT images (**A**, **B**). Scheme of calculations of anthropometric indices of the patient's CP and patient with unilateral SDOA: **A** – healthy; **B** – the affected side. The height (h) of the CP was determined as the perpendicular that was lowered from the top of the coronoid process to the *red line S*. The angle α – formed between *blue lines* (CP and the condylar process) had sides passing between the highest points of the processes of the mandible and the lowest point of the incisure of mandible. Additionally, measure the distance L (*yellow line*), which was determined between the top of the CP and the inner surface of the zygoma, as an indicator of the degree of mobility of the mandible.

The mean values of the CP jaw values for children in the norm of the age group from 7 to 12 years, which were determined according to the method proposed by us, were: S = 4.9 ± 0.87 mm; h = 9.6 ± 1.41 mm; α = 82.0° ± 2.11°; L = 7.25 ± 0.83 mm (Table 1). In this case, the mobility of the mandible was not disturbed and the opening of the mouth was free.

TABLE 1. Results of anthropometric measurements of CP in the control group children.

#	Age	Sex (m/f)	Affected side (R/L/R+L)*	Size of CP of Mandible (III Control Group – Norm)							
				L				R			
				S (mm)	h (mm)	α (°)	L (mm)	S (mm)	h (mm)	α (°)	L (mm)
1.	7	f	-	4.3	9.1	79	6.7	4	9	83	6.5
2.	7	f	-	4.9	8.5	81	6.6	4.8	8	81	6.7
3.	8	f	-	4.5	9.2	80	7.2	4.5	9.2	80	7.1
4.	8	m	-	4.5	9.6	85	7.3	4.6	9.4	80	7.6
5.	8	m	-	5	10.2	90	7.4	4.4	9.5	84	7.1
6.	9	f	-	5.2	9.8	84	7.6	5.2	10	79	7.6
7.	9	f	-	5.4	10.3	85	8.0	6	10.9	81	7.4
8.	9	m	-	4.9	8.5	79	7.1	4.9	9.5	85	7.2
9.	10	f	-	6	11.4	80	8.4	5.9	11.3	80	8.1
10.	10	m	-	6	10.9	78	7.6	5.9	10.9	80	8.3
11.	10	m	-	5.4	12.5	77	8.1	5.6	13.5	78	8.1
12.	11	f	-	6.2	12.4	79	8.1	6.3	12.3	80	8.5
13.	11	f	-	5.9	11.2	78	8.3	6	11	79	8.4
14.	11	m	-	6.6	11.4	83	9.3	6	12	77	9.0
15.	12	f	-	6.4	12.5	81	9.1	7	12.8	78	9.1
16.	12	f	-	7	13.8	83	8.9	7.2	13	77	8.9
17.	12	m	-	6.9	12.3	81	9.7	7.2	11.8	78	9.8
18.	13	m	-	7.6	13.6	80	9.9	8	13.1	77	10.0
19.	13	f	-	7.3	13.2	84	9.1	7.4	13.2	81	9.2
			M±m	5.8 ±0.84	11.1±1.43	81.4±2.53	8.1±0.81	5.8±0.92	11.1±1.40	79.9±1.70	8.1±0.84

** R — affected right side; L — affected left side; R + L — bilateral joint lesion*

We have not identified the gender differences in the anthropometric indicators of CP in healthy children. The largest increase in the values of the indicators was observed in children from 10 to 13 years: S = 1.6 ± 0.87 mm; h = 3.7 ± 1.41 mm; α = no significant changes; L = 1.3 ± 0.83 mm. Between the right and left sides there was a fluctuation in the values of anthropometric indicators, which can be explained by the habit of chewing more on one side, but they did not have any significant differences.

The results of measurements of the width of the S CP at the ADTMJ indicate an increase in its basis (Table 2).

For unilateral SDOA S of CP is 5.9 ± 1.03 mm, which is 1.0 mm more than normal (Fig 3). For bilateral SDOA, the width of the base of the CP reaches 6.8 ± 1.15 mm, increasing the difference by 1.9 mm. This indicator for ankylosis also had a steady tendency to increase: at unilateral 6.8 ± 0.89 mm / gain was + 2.5 mm, bilateral 6.4 ± 0.85 mm / gain + 2.0 mm in comparison with norm (Fig 3). The increase in the value of S for ankylosis and bilateral SDOA was the same and amounted to an average of 2.1 ± 0.85 mm. This can be explained by the fact that CP suffers the greatest burden precisely at these joint lesions due to the strain of chewing muscles and the imbalance of movements in the TMJ. Comparison of the healthy side index with one-sided ADTMJ with the norm – gave it an increase of only 0.3 mm with SDOA and 2.9 mm – with ankylosis.

The height h CP in children with one-sided SDOA was 14.8 ± 3.45 mm, which was 5.2 mm (N = 9.6 ± 1.41 mm) more than in control group children, and when compared with the unaffected side (10.7 ± 3.75 mm) more than 4.1 mm (Fig 4).

Indicator h CP for bilateral SDOA TMJ in children reached the values 17.8 ± 3.65 mm. The difference in the scores between one- (h^1) and two-way lesions (h^2) of this group was 3 mm (h^1 and h^2). In children with unilateral ankylosis, the value of the height of the explosives was 13.3 ± 1.88 mm, and bilateral – 15.1 ± 1.34 mm. The difference h^1 and h^2 was about 2.0 mm. This can be explained by the

TABLE 2. Results of anthropometric measurements of CP in children with SDOA and ancylosis TMJ.

#	Age	Sex (m/f)	Affected Side (R/L/R+L)*	Size of CP of Mandible (I Group – Patients With SDOA)							
				L				R			
				S (mm)	h (mm)	α (°)	L (mm)	S (mm)	h (mm)	α (°)	L (mm)
1.	7	f	L	5.5	14.5	67	4.2	4.0	9.6	69	6.1
2.	8	m	R+L	7.0	21.5	66	41	6.8	22.1	69	4.2
3.	9	m	L	6.6	17.5	58	4.0	5.7	10.3	65	6.9
4.	9	f	L	5.3	17.0	59	4.5	6.0	12.3	69	6.0
5.	10	m	R+L	8.2	15.3	69	4.5	8.2	15.8	65	4.0
6.	11	f	R+L	6.7	24.5	65	3.7	8.4	25.1	66	3.8
			M±m	6.4±1.03	17.6±3.60	64.0±3.67	4.2±0.23	6.5±1.28	16.7±3.75	67.2±1.83	5.2±1.17
#	Age	Sex (m/f)	Affected Side (R/L/R+L)*	Size of CP of Mandible (II Group – Patients With Ankylosis)							
				L				R			
				S (mm)	h (mm)	α (°)	L (mm)	S (mm)	h (mm)	α (°)	L (mm)
1.	7	f	R+L	4.9	15.6	65	3.2	5	14.3	65	3.7
2.	9	f	R	6.6	9.5	74	7.2	6.4	13.1	65	4.1
3.	9	m	R	5.7	11.2	69	6.7	5.8	11.3	70	4.6
4.	10	f	R+L	6.4	15.6	69	3.9	6.5	14.6	69	4.1
5.	10	f	R+L	7.5	17.1	61	4.6	7.7	16.3	66	4.2
6.	12	f	R	7.5	11.6	72	7.4	7.4	13.9	68	4.3
7.	13	m	R	7.9	12.2	62	8.3	7.5	13.0	69	4.1
8.	14	m	L	7.1	14.3	66	4.2	8.2	13.7	66	7.6
			M±m	6.7±0.80	12.9±2.12	67.3±3.75	5.7±1.71	6.8±0.89	13.5±1.88	67.3±1.75	4.6±0.76

** R — affected right side; L — affected left side; R + L — bilateral joint lesion*

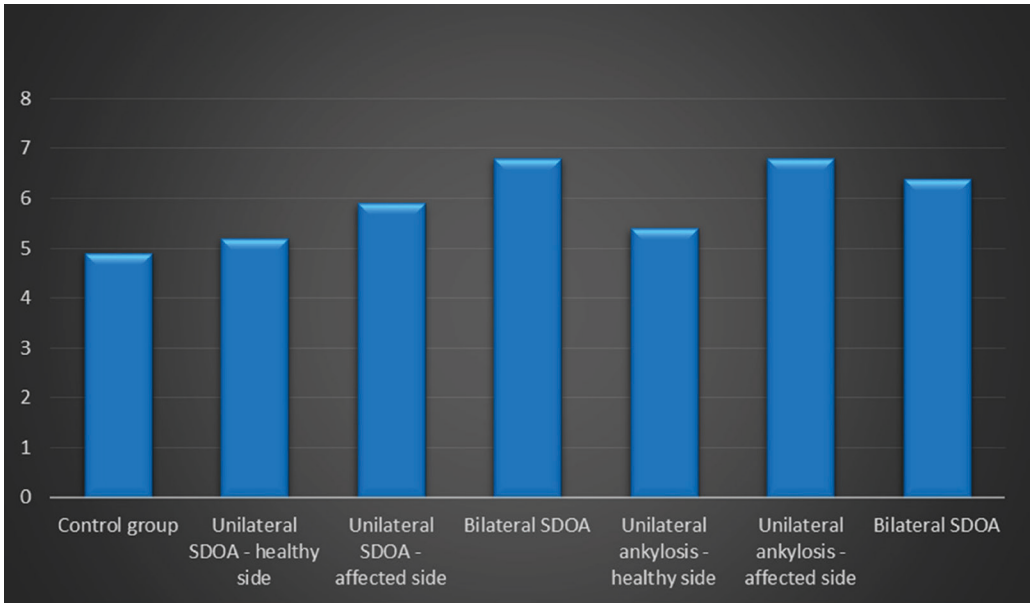

FIGURE 3. Results of measurements of S CP of mandible in patients I, II, III groups.

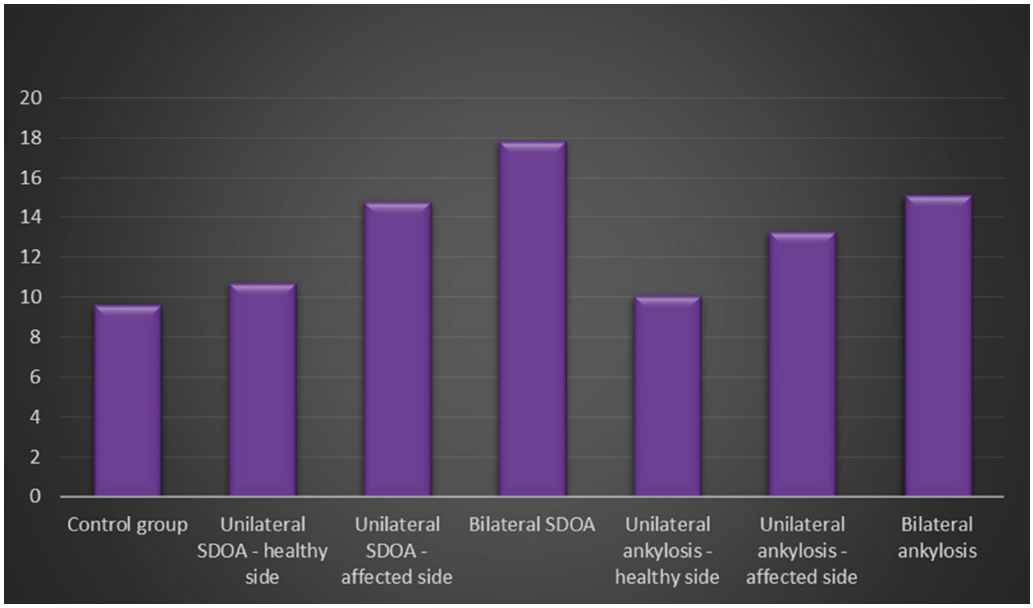

FIGURE 4. Results of measurements of S CP of mandible in patients I, II, III groups.

fact that with SDOA TMJ children retain small movements in the joint of the jaw and, accordingly, the load supports the trophism of the temporal muscle, which indirectly affects h CP. The difference was only when comparing the healthy side and affected by unilateral ankyloses and was 3.2 mm. Consequently, in all clinical cases, both groups had an increase in h, with the maximum being for bilateral SDOA and all types of ankylosis. The difference between the values h^1 and h^2 was greater when SDOA was 3 mm or less with 1.8 mm ankylosis. The comparative characteristic of the norm of height h with the healthy side with one-sided lesions gave it an average increase of 5.65 ± 2.34 mm, (SDOA – 6.55 mm / ankylosis – 4.62 mm). This indicator is a component of hyperplasia of the CP with ankylosis and SDOA TMJ and gives an idea of the mechanism of violation of opening the mouth in the patient. It should be noted that h is proportional to the maximum for bilateral joint damage. Such an increase in h CP is due to the vertical inclination of m.temporalis and the constant increased tonus of chewing muscles. This is evidenced by the data of EMG, namely the reduction of the temporal muscle, which, in the absence of movements in the joint, doubles the voltage of 2.041 μV (N = 0.942 μV).

The angle α for SDOA and ankylosis was 64.0° ± 2.75° and 67.8° ± 2.75°, respectively, which was lower than in children without lung lesions (82° ± 2.11°) (Fig 5). It should be noted that in one-sided SDOA he < at 6° compared with the healthy side, and with ankylosis < only 2°. On average, the angle α decreased by 12 degrees relative to the norm.

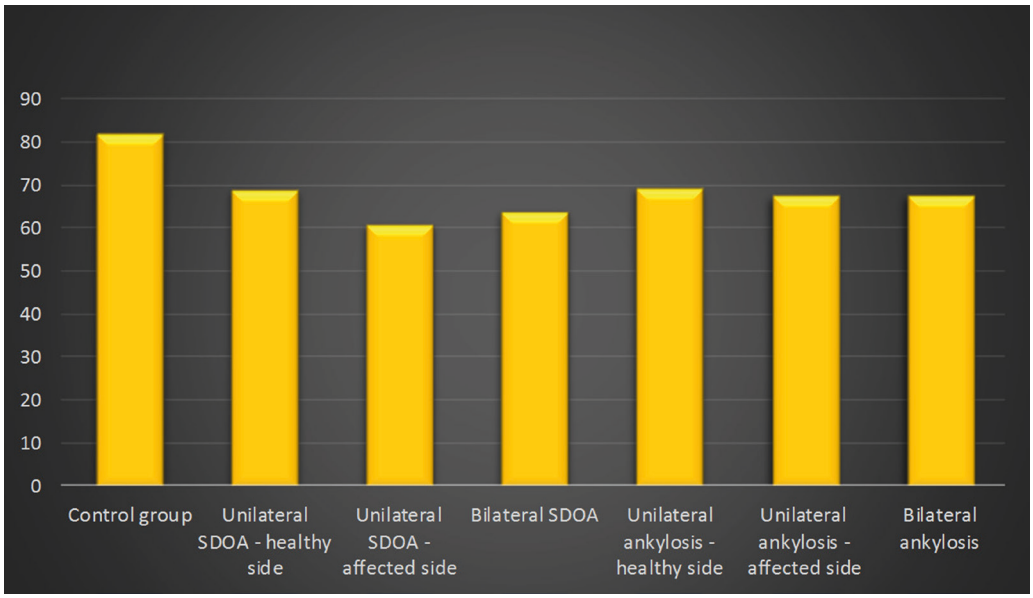

FIGURE 5. The results of measurements of the α CP of mandible score of patients in groups I, II, III.

Such changes of the angle at ADTMJ associated with the difficulty of the movements of the jaw and, consequently, the constant tension of the temporal muscle, whose myotatic vector, directed vertically upwards. The imbalance in the loading of chewing muscles in SDOA leads to an asymmetric hypertrophy of the temporal muscle. The obtained EMG data showed an increase in its voltage alone (X1) on the affected / healthy side – 2.041μV / 0.942μV, with compression (X2) – 81.61 / 107.52μV, respectively. With ankylosis, these rates increase. Voltage of temporal muscle at rest on the affected side was equal to 2.961μV, and at compression of 119.01 μV, indicating excessive bioelectric activity of it. This in turn leads to increased trophics in the affected area of the bone, which also contributes to hypertrophy of the CP of mandible. The expressed hypertrophy of the latter with ADTMJ is due also to the fact that the growth of the mandible changes the growth modulus from the condylar process of the jaw to its related vector – CP of mandible [3, 5].

The indicator L proposed by us indirectly indicates a violation of the mobility of the jaw. Normally, the values of L were within the range of 7.25 ± 0.83mm. In one-sided and bilateral SDOA TMJ, the average figures were 4.2 ± 0.23 mm and 4.1 ± 0.70 mm respectively; at ankylosis – 4.3 ± 0.76 mm and 3.95 ± 1.24 mm (Fig 6).

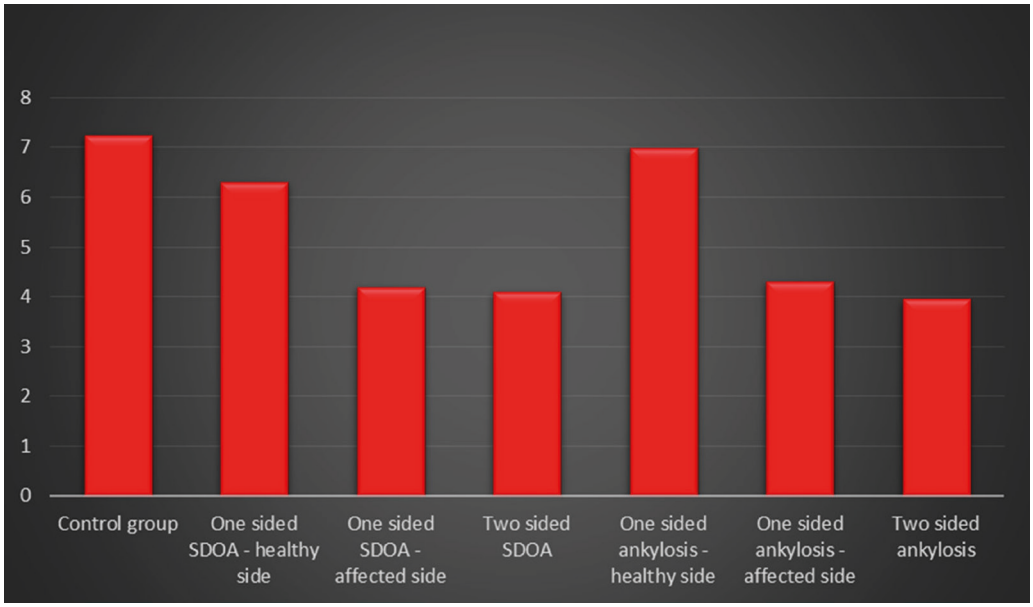

FIGURE 6. Results of measurements of the L index of CP of mandible in patients I, II, and III groups.

That is, in children of the 1st and 2nd groups he was reduced by an average of 3.1 mm. Comparing the indicator L with one-sided lesions of the TMJ with the healthy side, it was established that at: SDOA it was 4.2 ± 0.23 mm, which is 2.1 ± 1.01 mm less, compared to the healthy side, and 4.3 ± 0.76 mm – with ankylosis, which is 3.2 ± 1.17 mm smaller, respectively. This indicator is vividly illustrated by the fact that even at a reduced angle between the processes, if the indicator L is ≤7.0 ± 0.33 mm, then the free opening of the mouth in patients with ADTMJ is difficult. Normally, with bilateral contraction of the masticatory muscles, the lower jaw moves forward, with the distance between the vertex of the CP and the inner surface of the chick bone reduced to 3-4 mm, but the CP, while not touching the latter. At ADTMJ, this distance decreases by an average of 3 mm and when moving the CP to the front, he rests in the spine, which blocks the further opening of the mouth. Changes in height, width and angle of the CP at ADTMJ are a summary indicator based on our proposed indicator L, which reflects the biomechanics of the movements of the mandible at the opening of the mouth.

The determined changes in CP indexes on the unaffected side of the joint with SDOA and ankylosis can be explained by the fact that TMJ is a pair of joints and the occurrence of changes in one of them leads to "deviations" in the second one. It is important that, in the case of one-sided SDOA / ankylosis, the determination of changes in blood pressure on the unaffected side with known anthropometric indices makes it possible at the planning stage to determine the need for resection of the blood vessels not only on the affected side, but also on the healthy one.

The h / S ratio indicates an increase in CP in children with TMJ lesions, especially with bilateral SDOA and unilateral ankyloses up to 2.75 mm, which is 1.5 mm larger than N = 1.95 ± 0.62 mm. The h / L ratio, as the index of mobility of the jaw was higher in all children compared to control, namely: with bilateral lesions TMJ – 4.26 mm, with one-sided – 3.52 mm. This is almost 4 times more than normal in the case of bilateral

SDOA and ankylosis and, almost 3 times – at one-sided ADTMJ. Indicators of these relations, in our opinion, are extremely important, because they determine the degree of mobility of the jaw in its movements. And if they increase several times – h / L 4 times with ankylosis and 2.7 times with SDOA, then multiples of this changes the mobility index L, and thus the volume of movements of the mandible decreases towards the limitation of the opening of the mouth.

Thus, all three indicators of CP are altered with ADTMJ, with the largest changes developing in children of the II group with ankylosis TMJ and smaller in children of the 1st group with SDOA. With these diseases of the joint, the base of the joint increases with an average of 2.1 mm. The most significant changes occur at the height of the CP, where h varies from 13.3 ± 1.88 mm to 17.8 ± 3.65 mm, depending on the type of joint damage that was 5.9 mm higher than normal. Changes in h and S influence the angle α, which decreased with SDOA by 18°, And with bone ankylosis by 15° compared with N = 82° ± 2.11°. Probably the children are compensatory reorganization of the spatial orientation of the CP to maintain the mobility of the jaw. According to the results of SCT studies, it was determined that with an increase of h more than 14 mm and a S ≥6,0 mm and a ratio of h / S = 2.2 mm, h / L = 3.8 mm and L ≤ 4.5 mm, a blockage of the lower jaw occurs during sagittal and its vertical movements, which interferes with the free opening of the mouth in patients with ADTMJ. Thus, the combination of subjective indicators made it possible to determine the hyperplasia of CP of mandible and indications for its resection in children from ADTMJ at the stage of diagnosis according to SCT and accordingly plan the volume of surgical interventions.

Conclusions

1. The proposed scheme of anthropometric measurements of SCT allows us to mathematically substantiate the stage of hyperplasia of CP of mandible in children and determine the need for its surgical correction.
2. Our proposed indicator L is an indicator of the mobility of the mandible and reflects the changes that occur with the CP in the ADTMJ. Its values are taken into account in determining the indications for osteotomy CP.
3. It was found that the most significant changes were observed in bilateral SDOA and ankylosis: S = +2.1 mm; h = + 6.8 mm; α = -18°; L = -3.0 mm.
4. Indications for the resection of CP are an increase in h of more than 14 mm, S is 7.0 mm, α≤69°, and the h / L ratios = 4.4 mm and h / S = 2.1 mm and a decrease of L to 4.5 mm.

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Conflict of Interests

The authors declare that they have no conflict of interest.

Role of Author

The authors are equaly contributed to that article.

Ethical Approval

Approval was obtained from the Medical Ethics Committee of the Bogomolets National Medical University, Kyiv, Ukraine.

Patient Consent

Not required.

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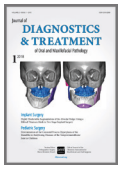

Prevalence of Aphthous Ulcer in Students of Ras Al Khaimah College of Dental Sciences*

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ABOUT ARTICLE

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Stress
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Behce't syndrome
Hormonal changes

ABSTRACT

Purpose.
Recurrent aphthous ulceration (RAU) is a common oral mucosal disease. RAU are painful ulcerations in the oral cavity that can cause bad breath and typically cause craters in the mouth. They are the most common type of lesions found in the oral cavity. The etiological involves in genetics, vitamin deficiencies, trauma, immune dysfunction and stress. This study was to explore the related risk factors of recurrent aphthous ulceration among dental college students.
Material and Methods.
We conducted a questionnaire survey among 80 students from the Ras Al Khaimah College of Dental Sciences (RAKCODS). The information collected includes report the prevalence, knowledge, experience and risk factors of aphthous ulcer in a sample of RAKCODS students.
Results.
The overall prevalence of RAU is 33 (41.25%) students reported of ever experiencing of RAU, however 47 (58.75%) students reported of never having had any experience.
Conclusion.
According to the results, there are many predisposing factors of RAU including sex, a positive family history and stress. Some measures should be taken to control the incidence of RAU which consist of prompting a correct way of living habits, paying attention to the health conscious diet, strengthen physical exercise, self-decompression and keeping good mentality.

Introduction

Aphthous ulcers are painful ulcerations in the oral cavity that can cause bad breath and determined effect on speech, nutrition and social interaction. The term *aphthous* has been derived from a Greek word *aphtha* which means ulceration. The multifactorial etiologic factors have already been implicated in the promotion and/or exacerbation of aphthous ulcer; these include positive family history, local trauma (Figs 1, 2), nutritional deficiency, food hypersensitivity, immune disturbance, smoking, and psychological stress [1]. The aim of this study to carry out the research among bachelor of dental surgery (BDS) students to add some knowledge related to the distribution, high frequency of aphthous ulcer by finding underlying etiology which is essential for better management of these cases.

Review of the Literature

Recurrent aphthous ulcer (RAU) is the most common inflammatory ulcerative condition of the oral mucosa. RAU occur in the non-keratinized areas such as lips, tongue, buccal mucosa and soft palate. They are usually painful, shallow round ulcers with an erythematous halo covered by a yellowish-gray fibro membranous layer. Many suggestions have been proposed but the etiology of recurrent aphthous ulcer is still controversial and its occurrence is related to a range of factors, precipitated factors include stress, physical or chemical trauma, food sensitivity, and genetic predisposition [2, 3].

The still unclear etiology has resulted in treatments that are largely empiric and aimed at symptom reduction. These ulcers may be associated with systemic conditions such as Behce't syndrome/HIV AIDS [4]. There are three major categories of aphthous ulcers – major, minor and herpetiform aphthous ulcers. Aphthous minor; commonly encountered painful, small, superficial ulcers of the oral glad bearing mucosa that occur episodically in clusters of one to five lesions. During an attack, new lesions may continually appear for a 3-4 week period with each lesion lasing 10-14 days. The floor-of-mouth and soft palate are common

* This manuscript has not been presented

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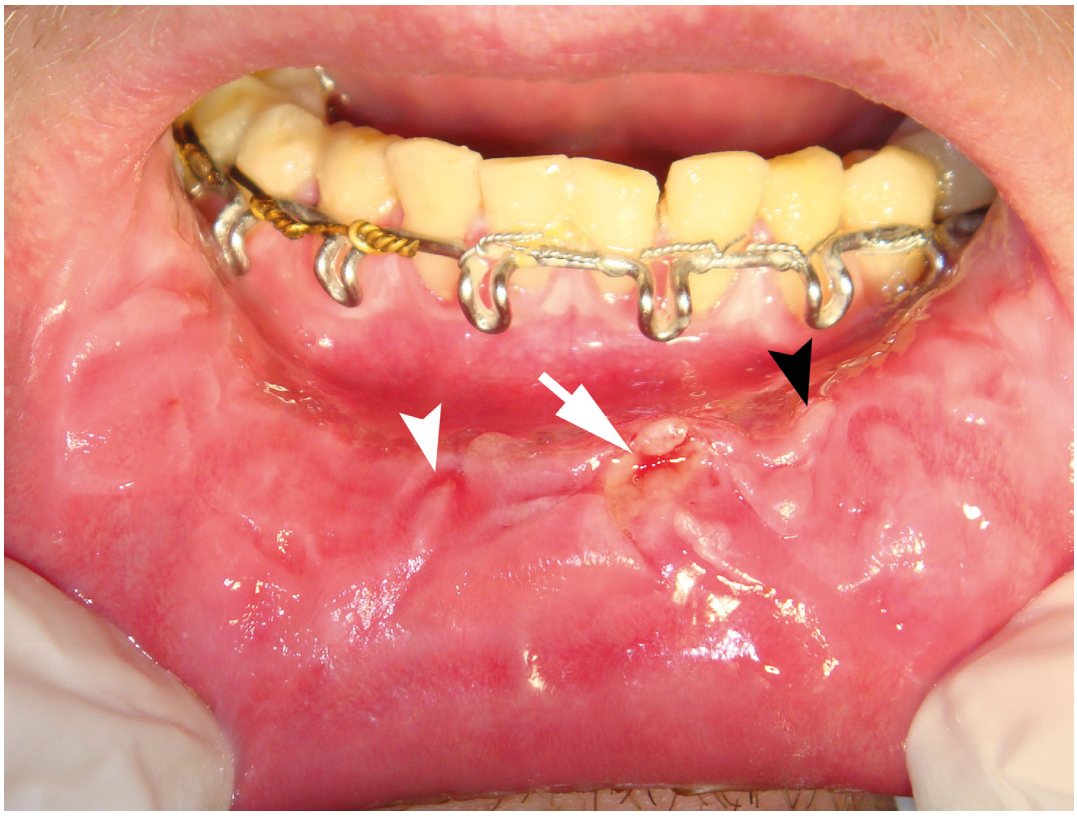

FIGURE 1. 35-year-old man with aphthous ulcer (*arrow)* on the lower lip mucosa caused by trauma with arch bar at 21st day of the treatment of mandibular fractures. Notes impressions (*white arrowhead*) and hyperplasia (*black arrowhead*) of the lip mucosa at the points of contact with arch bars. Image of **Figure 1** are courtesy of Ievgen I. Fesenko, *PhD*, *Assis Prof*; PHEI "Kyiv Medical University", Kyiv, Ukraine.

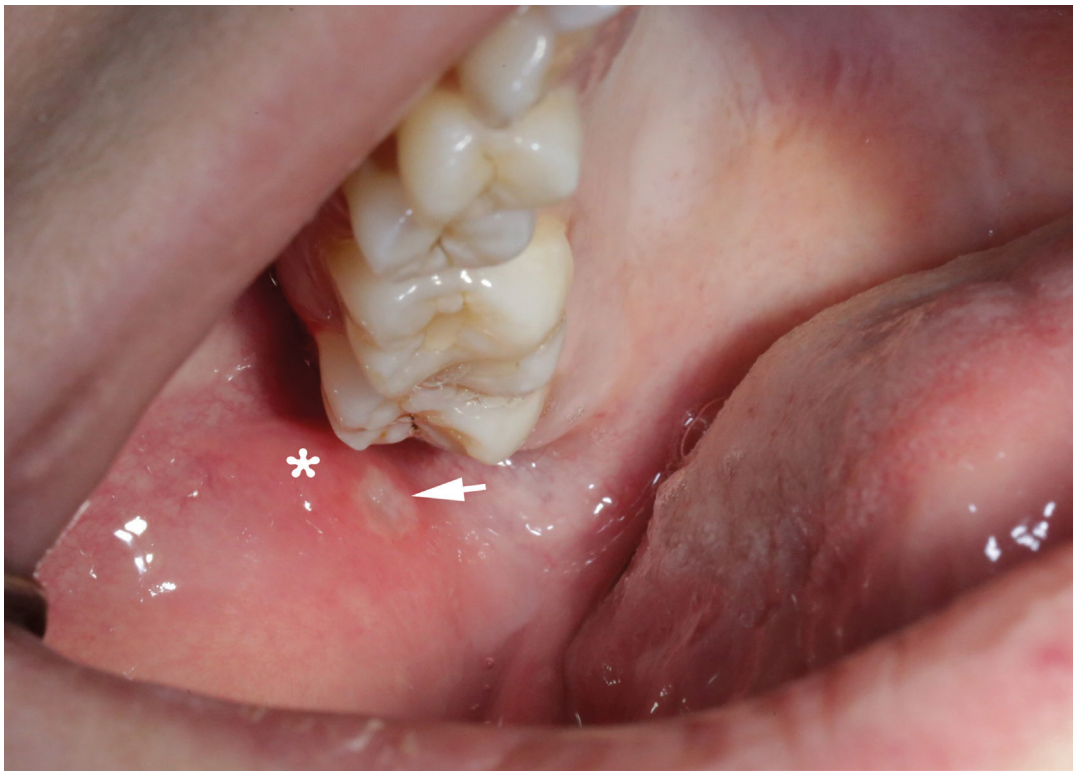

FIGURE 2. 28-year-old gentleman with aphthous ulcer (*arrow*) of the buccal mucosa caused by permanent trauma with upper wisdom tooth. Noted erythema and swelling (*asterisk*) of the mucosa around the ulcer. Image of **Figure 2** are courtesy of Ievgen I. Fesenko, *PhD*, *Assis Prof*; SCIEDECE – Scientific Center of Dentistry & Ultrasound Surgery, Kyiv, Ukraine.

locations for the minor sores which are typically small and shallow. They spare attached gingiva, hard palate and dorsum of tongue. Aphthous major; one or two uncommon large superficial painful ulcers, usually appear on labial mucosa and soft palate. They are larger than aphthous minor, they are around 5-20 mm in size, crater form and takes up to 6 weeks to heal. Scars are more likely to occur with the major ones which are larger and deeper. Herpetiform aphthous ulcers are the most numerous and intense [5, 6].

Aphthous ulcers (canker sores) are associated with local pain and discomfort. Symptoms usually last 2-10 days with minor and herpetiform ulcers and as long as 30 days with major ulcers. Most cases are self-limited and heal without squeal in 7-14 days; however, major ulcers heals slowly (10-30 days or longer) [6-9].

The primary morbidity with any type of aphthous ulcer (canker sore) in the pediatric population is dehydration due to poor oral intake. People are more likely to get them on a regular basis if they have a positive family history of cancer sores. These ulcers mostly occur from age of 10 years onwards but children as young as 2-years-old can get them. Most of them are first noted in adolescence or young adulthood and decrease in severity after menopause [10].

The frequency of occurrence is variable, ranging from several weeks to several years between episodes. The etiology of recurrent aphthous stomatitis (RAS) is not entirely clear, and aphthae are therefore termed idiopathic. RAS may be the manifestation of a group of disorders of quite different etiology, rather than a single entity. Despite many studies trying to identify a causal microorganism, RAS does not appear to be infectious, contagious, or sexually transmitted [11-15]. Immune mechanisms appear at play in persons with a genetic predisposition to oral ulceration [16].

There is no curative therapy to prevent the recurrence of ulcers and all available treatment modalities can only reduce the frequency or severity of the lesions. Vitamin supplements in people who are B12, folic acid deficient. Avoid spicy food, if caused by another illness, they will clear up when the illness is treated. Some herbs like *Aloe vera* and tea tree oil have been known to relieve pain and inflammation which is caused by the aphthous ulcers [17].

Therefore, the aim of the study is to explore the prevalence of aphthous ulcer in Dental College students (RAKCODS students).

Materials and Methods

INCLUSION AND EXCLUSION CRITERIA

Ras Al Khaimah College of Dental Sciences (RAKCODS) RAK Medical & Health Sciences University UAE students (80 persons) from 2nd, 3rd, 4th, and 5th year. 1st year students were excluded due to unavailability and limited knowledge.

STUDY DESIGN

Cross sectional by a questionnaire study.

DATA COLLECTION AND ANALYSIS

Data collection was done using a questionnaire. The questionnaires were distributed by the investigator and collected immediately after being filled with the help of class representatives. Among the variables in the questionnaire were; the students age, gender, ethnical backgroundand presence or absence of familial history of aphthous ulcers. The data was analyzed using MS Excel computer programs. The information gotten from the data collected was presented in the form of graphs and frequency tables.

A questionnaire containing a total of 12 questions in which 4 questions giving the personal details of the students which included name, age, sex and smoker/ nonsmoker were recorded. The names of these students were kept confidential. Whereas 8 questions related to aphthous ulcerations (which included whether patients has any history of AU or no, if they had history of RAU then what are the triggering factors, whether it is related to exam/stress or not, duration of the ulcer present, number of days took for healing, any medication patient taking for the same problem, during their visit whether they had any ulcer in the mouth, and any related comments) were recorded.

ETHICAL CONSIDERATIONS

This study is approved by local Ethical Committee. The permission was obtained from the responders and the respondents were assured of confidentiality. All the information obtained in this study will be used for academic purposes.

Results

This study was conducted in order to find out the knowledge, experience and risk factors of oral aphthous ulcers (RAU) among BDS students.The study sample was 80 students (Tables 1-10 and Figs 3-12).

RESULT 1 [EXPERIENCE OF APHTHOUS ULCER] (Table 1 and Fig 3)

TABLE 1. Experience of aphthous ulcer

Answers	Number of Students	Total
Yes	33	
No	47	
		80

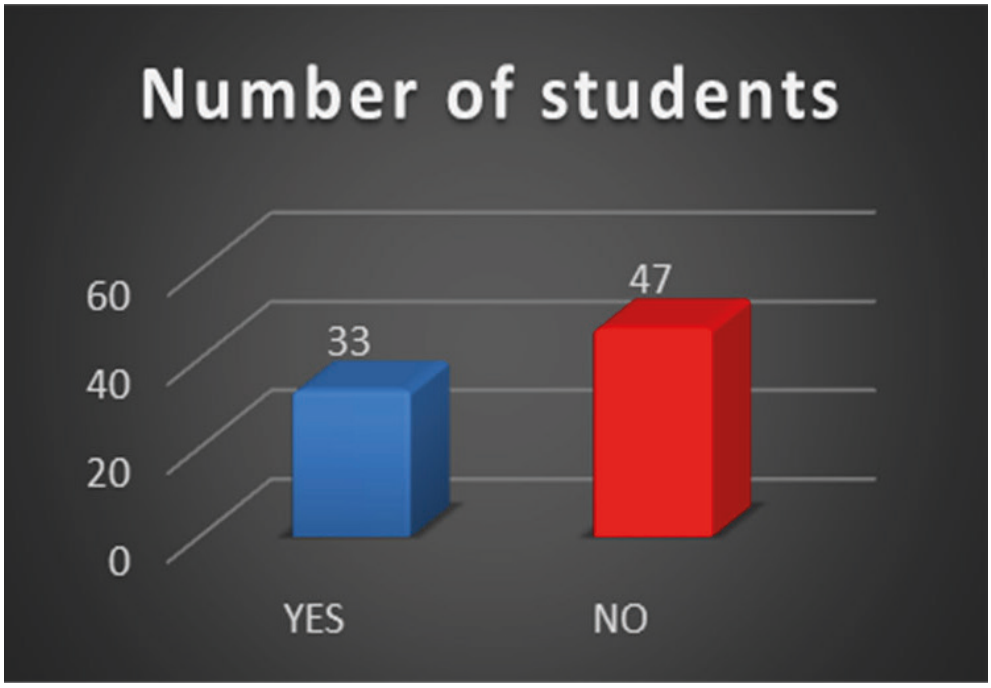

FIGURE 3. Graphic depicts experience of AU: (*left axis*) total number of students, (*column in blue*) "yes" answers, (*column in red*) "no" answers.

The study sample was 80 students. In total, 33 (41.25%) students reported of ever experiencing aphthous ulcer. 47 (58.75%) students reported of never having had any experience.

RESULT 2 [AGE DISTRIBUTION OF RESPONDENTS] (Table 2 and Fig 4)

TABLE 2. Age distribution of respondents

Age	Number of Respondents	Total
18-19	15	
20-21	20	
22-23	40	
Less than 30	5	
		80

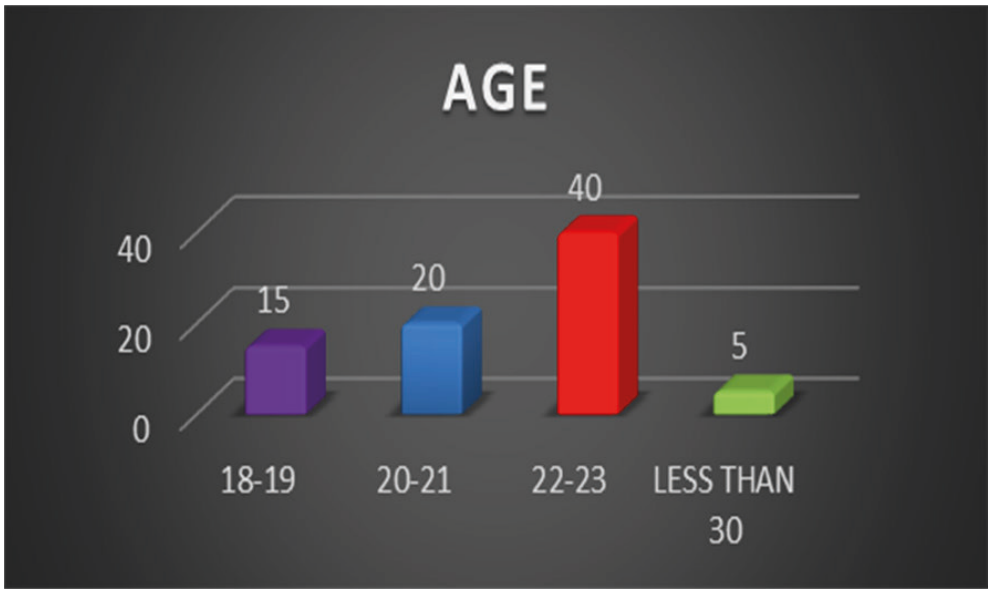

FIGURE 4. Graphic depicts the age distribution of respondents: (*left axis*) total number of students, (*column in purple*) 18-19-years-old age, (*column in blue*) 20-21-years-old age, (*column in red*) 22-23-years-old age, (*column in green*) less than 30-years-old age.

In total, 40 students age range between 22-23 years, 20 students age range between 20-21 years, 15 students age range between 18-19 years, and 5 students who are above 23 and below 30.

RESULT 3 [GENDER] (Table 3 and Fig 5)

TABLE 3. Gender

Gender	Number of Students	Total
Female	22	
Male	11	
		33

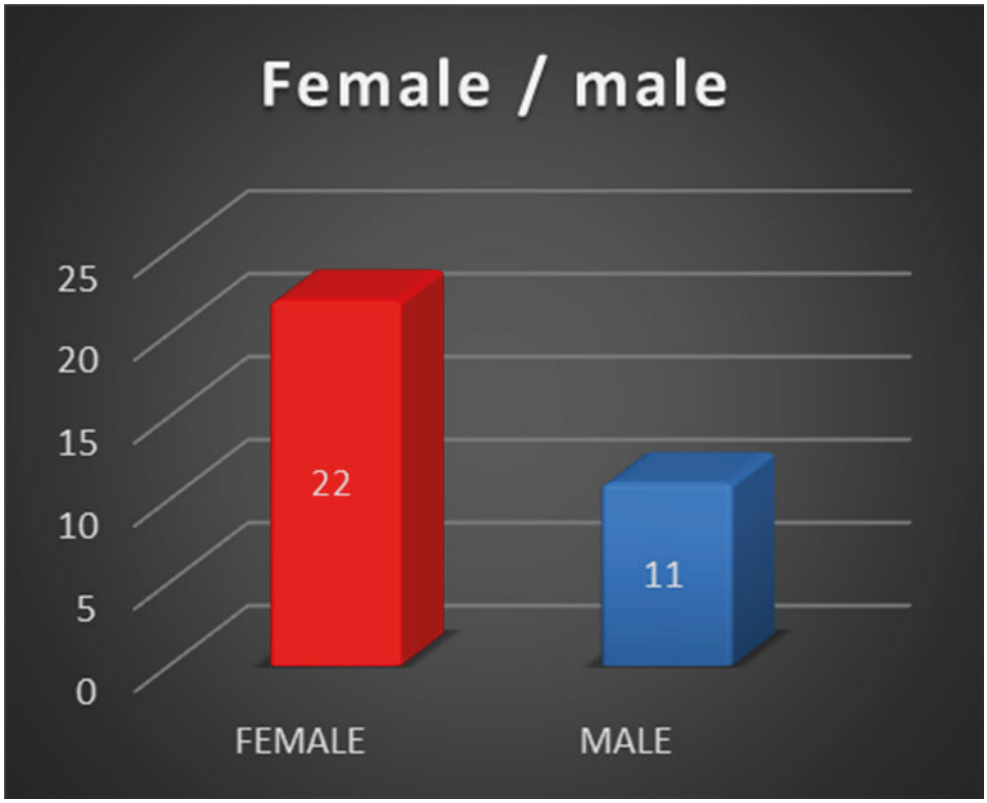

FIGURE 5. Graphic depicts the gender: (*left axis*) total number of students, (*column in red*) female, (*column in blue*) male.

In 33 (41.25%) students reported of ever experiencing aphthous ulcer. Of these, 22 (66.7%) students were female and 11 (36.4%) male.

RESULT 4 [CAUSES OF APHTOUS ULCER] (Table 4 and Fig 6)

TABLE 4. Causes of aphthous ulcer

Causes	Number	Total
Eating spicy food	6	
Trauma	11	
Vigorous brushing tooth	4	
Type of toothpaste	None	
Stress	12	
		33

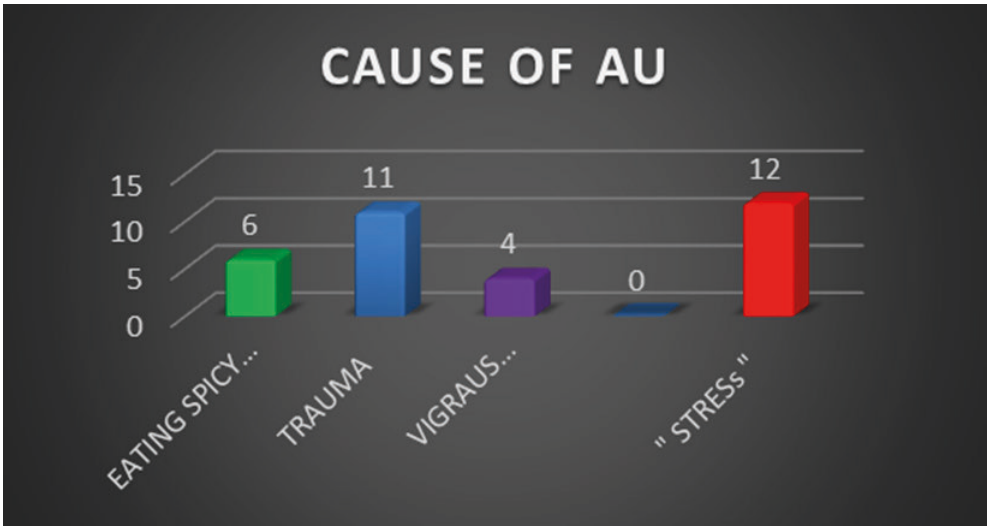

FIGURE 6. Graphic depicts the causes of AU: (*left axis*): total number of students, (*column in green*) eating spicy, (*column in blue*) trauma, (*column in purple*) vigorous brushing tooth, (*column in dark blue*) type of toothpaste, (*column in red*) stress.

In 33 (41.25%) students who reported of having experience on apthous ulcer. Of these, 12 (36.35%) students said that stress is the cause of RAU. 11 (33%) students said that trauma is the cause. 6 (18%) students said that spicy food is the cause, and 4 (12.1%) students said that hard brushing tooth is the cause.

RESULT 5 [LAST EXPERIENCE OF APHTHOUS ULCER] (Table 5 and Fig 7)

TABLE 5. Last experience of AU

Months	Number of Students	Total
< 6 months	12	
6-12 months	5	
1-1 years ago	6	
> 2 years	10	
		33

FIGURE 7. Graphic depicts the last experience of AU: (*left axis*): total number of students, (*column in purple*) < 6 months, (*column in blue*) 6-12 months ago, (*column in red*) 1-2 years ago, (*column in yellow*) > 2 years.

In total, 33 (41.25%) students reported of ever experiencing AU. Of these, 12 (36.4%) students inform that the last experience of AU was < 6 months. 10 (30.3%) students inform that the last experience of aphthous ulcer was > 2 years.

RESULT 6 [LOCATION OF APHTHOUS ULCER] (Table 6 and Fig 8)

TABLE 6. Location of aphthous ulcer

Locations	Number	Total
Buccal mucosa	21	
Dorsum of tongue	6	
Palate	3	
Floor of mouth	2	
Other	1	
		33

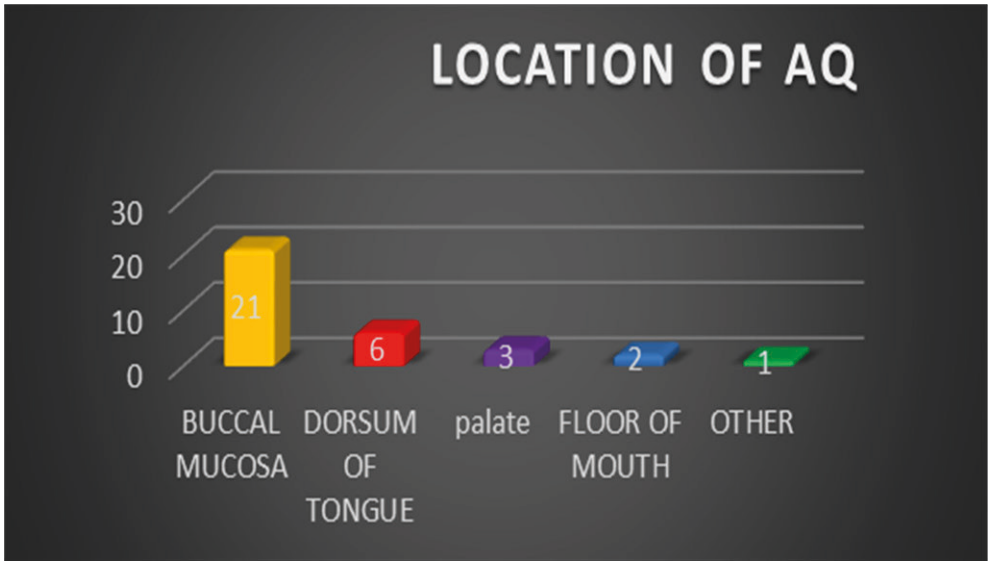

FIGURE 8. Graphic depicts the location of AU: (*left axis*) total number students, (*column in yellow*) buccal mucosa, (*column in red*) dorsum of tongue, (*column in purple*) palate, (*column in blue*) floor of mouth, (*column in green*) other.

In total, 33 (41.75%) students reported of ever experiencing AU. Of these, 21 (63%) students reported of having AU in buccal mucosa.

RESULT 7 [TYPE OF APHTHOUS ULCER] (Table 7 and Fig 9)

TABLE 7. Type of aphthous ulcer

Types	Number	Total
Major "large ulcer"	3	
Minor "small ulcer"	29	
Many small ulcers	1	
Other	None	
		33

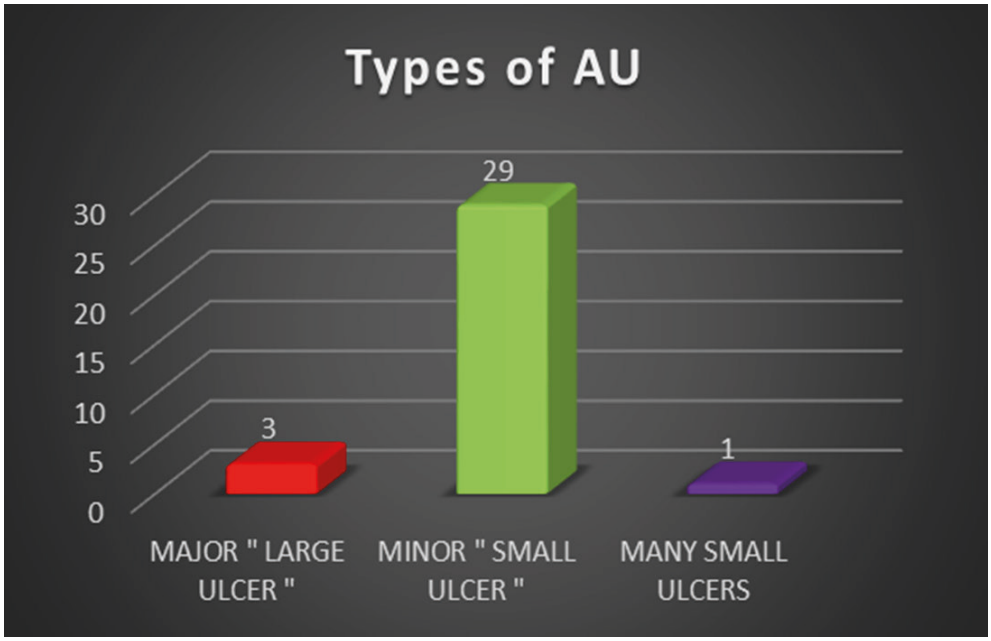

FIGURE 9. Graphic depicts the types of AU: (*left axis*) total number of ulcers, (*column in red*) major ulcer, (*column in green*) minor ulcer, (*column in purple*) many small ulcers.

In total, 33 (41.75%) students reported of ever experiencing AU. Of these, 29 (87.8%) students reported that they had "minor" type of AU.

RESULT 8 [SMOKING AS A RISK FACTOR] (Table 8 and Fig 10)

TABLE 8. Smoking as a risk factor

Answers	Number	Total
Yes	20	
No	60	
		80

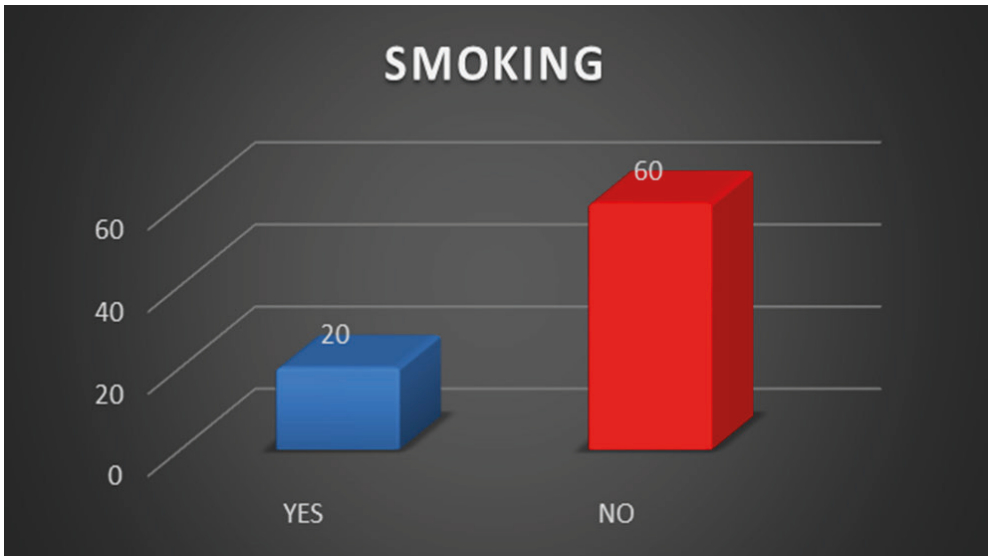

FIGURE 10. Graphic depicts the smoking as a risk factor: (*left axis*) total number of students, (*column in blue*) "yes" answers, (*column in red*) "no" answers.

The total student sample (80 students), 20 (25%) students were smokers whereas 60 (75%) students nonsmokers.

RESULT 9 [TAKING MEDICINES FOR THE TREATMENT OF AU] (Table 9 and Fig 11)

TABLE 9. Taking medicines for the treatment of AU

Answers	Number of Students	Total
Yes	5	
No	28	
		33

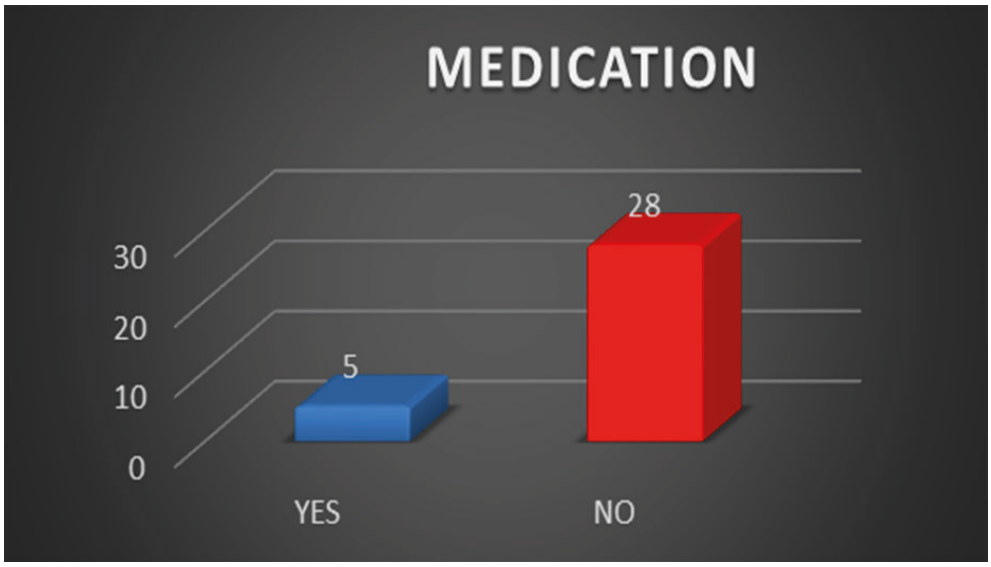

FIGURE 11. Graphic depicts the taking medicines for the treatment of AU: (*left axis*) total number of students, (*column in red*) "yes" answers, (*column in blue*) "no" answers.

In total, 33 (41.75%) students reported of ever experiencing AU. Of these, 28 (84.8%) students did not take any medication for the aphthous ulcer whereas 5 (15.2%) students took medication for the AU.

RESULT 10 [HISTORY OF AU IN OTHER FAMILY MEMBERS] (Table 10 and Fig 12)

TABLE 10. History of AU in other family members

Answers	Number of Family Members	Total
Yes	8	
No	25	
		33

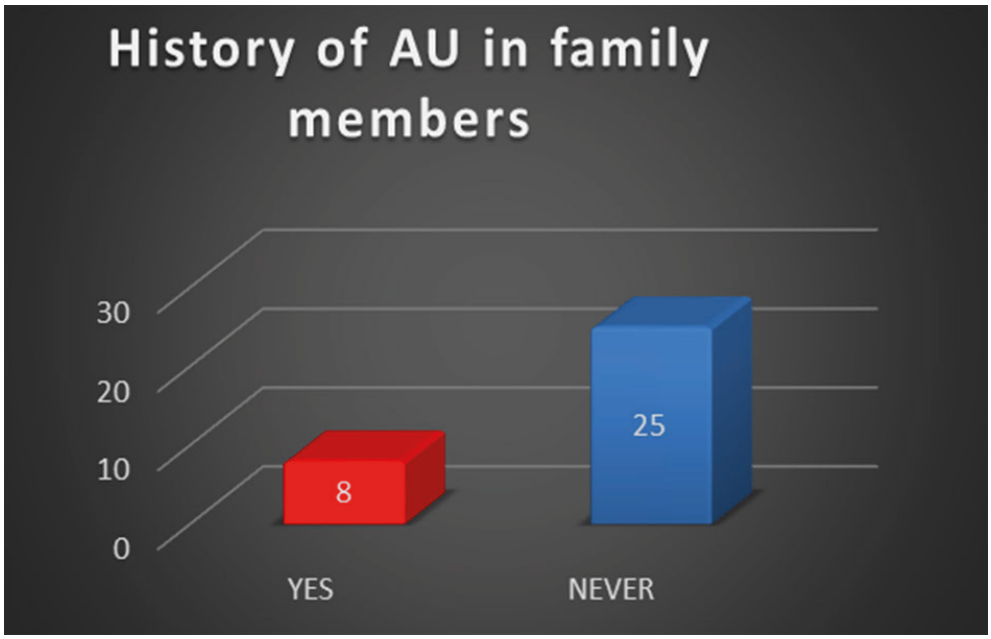

FIGURE 12. The history of AU in other family members: (*left axis*) total number of family members, (*column in red*) "yes" answers, (*column in blue*) "no" answers.

In total, 33 (41.75%) students reported of ever experiencing AU. Of these, 8 (24.2%) students said that other people in their family have had AU whereas 25 (75.8%) students said that no one else in the family has ever experienced AU.

Discussion

Recurrent aphthous ulcers are painful oral lesions with many predisposing factors and no proven effective treatment. A definitive cause of these ulcerations still remains elusive. This study was designed to evaluate the prevalence of RAU in BDS RAK Dental College Students. A total of 80 students participated in the research, and responded to the questionnaires provided. In total, 33 (41.25%) students reported of ever experiencing aphthous ulcer and 47 (58.75%) students reported of never having had any experience.

In our results as shown of the 33 students who reported of ever experiencing aphthous ulcer 29 (87.8%) students reported that they had "one small ulcer." This unlike Lelei Priscilla (2009) [1] who found that most of their students got the "many small ulcers."

Approximately 87% of patients with recurrent aphthous ulcerations were exhibiting minor type in our study. This finding in agreement with Field and Allan (2003) [18]. Also in the study of Naito *et al* (2014) [21]. Female BD patients with two or more RAU had almost double the risk for a subnormal score than did their male counterparts. This difference was agreed with our results which showed that higher occurrence in females during their second decade of life. This unlike Kaimenyi and Guthua [5] who found that occurrence in male is more than female.

Prabha *et al* in 2012 are agreed with our results as in results were shown. It is believed that psychological stress maybe a significant contributor of all students who had experienced apthous ulcer, 12 (36.35%) students said that stress is the cause of RAU. This finding also is in agreement with a research done by Lelei Priscilla in 2009 [1], and Gavic *et al* in 2014 [20].

The recurrence rates of RUA at the interval of 3-monthes are as high as 50% and these results obtained from Byahatti in 2013 [21, 22] and these are in agreement with our results. Safadi in 2009 [8] in his study of students of Jordanian dental students observed that 92% of subjects reported pain and two – thirds of subjects noticed that ulcers lasted for less than week whereas a minority of participants felt the duration extended beyond two weeks. The observations were evident of our results which indicated same results and suitability to recurrence in total of 33 (41.25%) students reported over experiencing.

Of the 33 students who reported of ever experiencing aphthous ulcer, 21 (63%) students reported of having AU in buccal mucosa as was shown in the our study. This finding is in agreement with a research done by Lelei Priscilla in 2009 [1] and Zhou [23]. The high percentage of AU on the buccal mucosa could be due to trauma during mastication.

In present study 20 (25%) students were smoking whereas 60 (75%) students were nonsmokers which have shown in results and this most probably agreed with Grady *et al* in 1992 and Chaopadhyay *et al* in 2007 which were reported that majority of the students reported not using tobacco. This is not completely reliable as the students may not have revealed the correct information for fear of scurvy by faculty. Also it has been suggested that cigarettes smoking prevents aphthous ulcers by causing increased keratinization of the oral mucosa [24, 25].

Regarding certain drugs have been associated with development of RAU; in our study in total of 28 (84.8%) as shown in our study that the students reported that didn't take any medications for AU whereas 15.2% took medication for relieve the pain. These associated with the results of Natah *et al* in 2004 which agreed in their study small percentages of patients used pain medications and antihypertensive drugs [22].

Present study revealed in the our study that students whose parents suffer from RAU more prone to RAU multivariate logistic regression analysis showed that genetic factors are risk factors of recurrent oral ulcers, which are similar to the findings of Koybasi *et al* in 2006 [26]. Family history was the most important factor for RAU among the investigated ones. Occur among sibling may be parents RAU status [27] with increased risk in children of two affected parents (67-90%), and correlation between the incidence of RAU and identitical twins was found [28].

However, the etiology of RAU still remains unclear and the currently available therapy remains inadequate. On the other hand, many factors have already been implicated in the promotion and/or exacerbation of RAU. The study may have its limitations in that the data collected was based on subject recall of ulcer experience.

Conclusions

Based on the findings of this study, the following was concluded: 1) The occurrence of AU in female is more than male; 2) There is a direct relationship between stress and occurrence of AU; 3) AU has an effect on speech, nutrition, and social interaction. Early detection and management of these patients by finding the cause is essential for better management of these cases.

Perceived benefits: 1) the information obtained will be used to design preventive programs against ulcers among dental students, 2) the information obtained will be sensitize the clinical BDS students level III and IV and V on need to screen for and educate their patients on risk factors and prevention of aphthous ulcer.

Recommendations: 1) educational programs on stress and their effects on RAU experiences should be carried out to inform dental students as this will be important especially for BDS year 3, 4 and 5 who deal with patients, 2) introduction of stress management programs, this will minimize occurrence of RAU especially during examination, 3) public health programs to be developed in order to educate the public about the ulcers for the well being of the public at large.

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Conflict of Interest

The authors declare that they have no conflict of interests.

Ethical Approval

Approval was obtained from the Ethical Committee of the RAK College of Dental Sciences. RAK Medical & Health Sciences University. Ras Al Khaimah, UAE.

Patient Consent

The permission was obtained from the responders and the respondents were assured of confidentiality.

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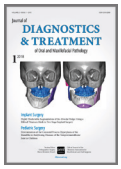

Detection of Titanium Particles in Soft Tissues Adjacent to the Fixators in Patients With Facial Fractures and Bone Defects*

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ABSTRACT

Background.
Open reposition and rigid internal fixation are the main methods of treatment for traumatic injuries of the facial skull and an important stage of bone-plastic, reconstructive, and orthognathic surgery. In contemporary maxillofacial surgery, fixators, implants, and endoprostheses made of titanium or its alloys are widely used due to the high corrosion resistance and biocompatibility. However, recent studies have shown that none of the metal implants used in maxillofacial surgery, orthopedics or traumatology is completely inert. Moreover, they always interact with the surrounding biological environment. Thus, a number of studies have revealed the release of titanium to the adjacent soft tissues.
Material and Methods.
Titanium fixators (plates and screws) removed in 12 patients in late terms after osteosynthesis, as well as biopsies of the periosteum and fibrous capsule adjacent to the fixation elements made of titanium were investigated. Microscopic fluorescence spectroscopic analysis (M4 TORNADO micro-ray fluorescence spectrometer; Bruker, Bremen, Germany) was used to determine the elemental composition of the removed soft tissue fragments. Scanning electron microscopy (microscope model JSM-6060; JEOL, Japan) was used to study structural changes on the surface of titanium plates and screws. The obtained results were analized with the use of Spirman correlation coefficient, calculated by the IBM SPSS Statistics v.23 software.
Results.
X-ray fluorescence analysis revealed the inclusion of titanium in all investigated samples with an average content of titanium 48.14% ± 31.1% in metal deposition areas. For samples removed in patients with traumatic facial fractures after metallosteosynthesis, the average content of titanium was 55.6%, and for reconstructive surgeries – 37.72%. The acquired maps of the element deposition showed no topographic inhomogenity of titanium particles distribution. The main distribution patterns were the following: 1) areas of clearly outlined intensive titanium inclusions (90.9-800 μm), and 2) diffuse titanium inclusions which were poorly demarcated. Electronic microscopy of the investigated fixators revealed deformation of the thread, bending of screws, deformation and surface defects of the plates caused by mechanical damage, including microcracks, sharp edges, scratches, dimples.

Introduction

Open reduction and rigid internal fixation are the main methods of treatment for traumatic injuries of the facial bones and an important stage in bone-plastic, reconstructive and orthognathic surgery. A large number of available fixators differ in shape, size and design features. The most common are metal plates and screws, which have the adequate stiffness and strength to provide stabile fixation of the bone fragments under cyclic functional load [1].

Stainless steel [1], titanium, and its alloys, zirconium alloys, tantalum are used for fixators manufacturing. According to modern concepts, these materials, should be biocompatible (from the chemical, physico-mechanical and biological points of view) to avoid toxic and carcinogenic effect, as well as any kind of immune response [1]. However, studies of recent years have shown that none of the metal implants used in maxillofacial surgery, orthopedics or traumatology is completely inert. Moreover, they always interact

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with the surrounding biological environment [2, 3]. The release of metal from plates and screws into the living tissues after the implantation and the resulting pathological changes of varying severity have been reported for most alloys used to date [4-7]. The release of metal from the fixator results from the corrosion, friction and micro-destruction during the interaction of the 'fixator-bone' system elements under the functional load or mechanical damage to plates and screws at installation or removal [2, 7].

It has been proved that stainless steel, which was widely used for the manufacture of fixators in the second half of the past century, undergo significant biodegradation and cause local tissue reactions [8-11]. The constituent metals, including chromium, nickel, molybdenum and iron showed a certain degree of toxicity [8, 12-14]. Meachim and Winter reported that the high content of corrosion products around stainless steel implants was associated with chronic inflammatory reaction. Therefore, in contemporary maxillofacial surgery, the most widely used are fixators, implants, endoprostheses of titanium and its alloys [7, 12, 15-20] due to the fact that along with good mechanical properties they have high corrosion resistance and biocompatibility exceeding the similar characteristics of medical steel [7, 15-17, 19, 21-24].

High corrosion resistance and biocompatibility of titanium implants is determined by the formation of a passivating surface layer of titanium oxides [25, 26].

Nevertheless, the biocorrosion of titanium fixators in long terms following the implantation was detected both by light and electron microscopy in a series of studies [2, 11, 19, 20, 27-31]. Ferguson *et al* [33] reported the ionization and release of the metal from the surface of titanium implants into surrounding soft tissues. This process is often accompanied by the changing of the peri-implant color of soft tissue into stable greyish [7], although the impregnation of small metal particles may be present and visible at the microscopic level even if there is no macroscopic change of the tissue color [33]. Larger metal grit can get into the tissues through existing surface defects that arise during the manufacture of the fixator as well as due to corrosion, surface contamination or mechanical damage while installing, removing or operating [34].

The main mechanisms behind the release of metal into the tissue are mechanical wear and corrosion. The processes of the release of metallic micro and nano particles as well as metal ions are accelerated when the protective oxide layer becomes thinner due to the plate bending, microcracking, damage to the surface of the plate or screw with a drill, screwdriver or other surgical instruments [25, 26]. According to A. Rosenberg (1993) [35], pigmentation of tissues due to metallosis was more pronounced around the curved sections of the plates. Friction in the plate-screw and plate-bone systems is another important factor involved in the degradationof the fixator surface and the occurence of small metallic inclusions in the tissues. However, the analysis of literature points to the lack of consensus as to the mechanisms of the metal release into the tissue, as well as the degree of titanium miniplates surface degradation in the long-term presence inside the human body [12]. Biological effects caused by the release of titanium into the tissues also remain poorly studied, and the results of the related research are often controversial.

It has been established that metal implants and products of their degradation can cause both local and general reactions of varying severity in the human body [10, 33, 36]. A number of publications suggest that titanium, which is believed to be a bio-inert material, has the potential to cause chronic inflammation and some immunological responses [3, 7, 9-11, 14, 19, 26, 27, 33]. Although clinical trials have not provided convincing evidence of significant damage caused by the continued preservation of titanium plates in the human body, titanium particles in the tissues are associated with the activation of monocytes and macrophages, the release of mediators of bone resorption, fibroblast stimulation, affected bone healing, hypersensitivity reactions, and impaired immune response [37]. Titanium can be "attacked" by several different types of immune cells, namely macrophages, histiocytes, giant cells of foreign bodies, lymphocytes and granulocytes [7, 11, 38] releasing active forms of oxygen and contributing to further degradation of the implant surface, which is usually very slow. A significant increase in titanium content in such internal organs as lungs, spleen, liver and kidneys following the experimental installation of titanium implants to the long bones and mandible was also reported [39, 40].

Intracellular location of titanium particles may be caused by phagocytosis [41], but in most cases they are extracellularly located and surrounded by fibrous connective tissue [2, 7] with no or moderate manifestations of a chronic inflammatory reaction [41].

It should be noted that titanium alloys used in maxillofacial surgery include vanadium and aluminum, which are significantly more toxic than titanium. Ions of vanadium affect lipid metabolism, have a cytotoxic effect on tissues and cause the destruction of some enzymes. The ions of aluminum suppress synthesis of ATP, therefore the high content can significantly reduce the metabolic activity of bone tissue and slow down mineralization. Aluminium also suppresses erythropoiesis and affects the central nervous system. The cellular toxicity caused by aluminum is associated with Alzheimer's disease, parkinsonism and osteomalacia [44]. Some studies reported the presence of aluminum both on the surface of titanium plates [28] and in soft tissues adjacent to them [2, 43]. However, the cumulative effects of small quantity of titanium alloy corrosion products still need the further investigations [45].

The tissue response to corrosion and release of metal particles into the surrounding tissue are the main arguments in favor of removing the metal miniplates after fracture healing [1, 26, 43]. According to the literature, the frequency of plates removal in patients after osteosynthesis is from 3 to 18% and more. In 22% plates are removed in absence of any complications, at patients' requests [17, 45, 46]. At the same time, the removal of fixators can present significant technical difficulties. It creates discomfort for the patient associated with the need for an additional surgery [15-18] the risks of which may exceed the positive effect, since scientific studies did not reveal a reliable relationship between the intensity of metallosis and manifestations of inflammation [35, 41, 44].

In addition, the severity of the metallosis is variable in different patients, and the factors affecting it remain underinvestigated. Obviously, the optimization of strategy for the removal of fixators in the remote postoperative period and prevention of negative effects associated with their installation requires an in-depth study of the mechanisms of the fixator interaction with biological tissues and understanding the processes which determine the release of metal particles from their surface into the surrounding biological environment.

The aim of the study was to investigate the microstructural changes on the surface of fixation elements (titanium plates and screws), and to determine the content and distribution of titanium and other chemical elements in adjacent soft tissues, as well as factors influencing these parameters in the long-term period following osteosynthesis of the facial bones.

Materials and Methods

Materials of the study included titanium fixators (plates and screws), removed in 12 patients in the long terms following osteosynthesis, as well as biopsy samples of the periosteum and fibrous capsule adjacent to the fixing titanium elements. All patients were treated in the Center of Maxillofacial Surgery and Stomatology in Kyiv Regional Hospital and gave their consents to participate in the study. The expertise of the research materials was conducted according to the approval (#106, November 07, 2017) of Bioethics Commission of Bogomolets National Medical University.

The average age of patients was 30 years, the ratio of men and women in the group was 2:1. All patients underwent the osteosynthesis of the facial bones (8 patients) or reconstructive surgeries on the jaws (4 patients) with the use of titanium fixators. The following types of fixators were used: I-Plant (Ukraine), Stryker (Kalamazoo, Michigan, USA), and Conmet (Moscow, Russia). All the fixators were made of medical titanium (Grade 4). The lenght of the period from installation to the removal of the fixator was from 5 months to 3 years (an average of 11.6 ± 11 months). The reasons for removing the fixators were: exposure of fixation elements (33.3%), removal of the fixator during the regular stages of reconstructive interventions in multi-stage surgical treatment (33.3%), patients' complaints of pain and discomfort in the fixator area (25%) and patient's requests (8.3%). Surgeries were performed according to standard protocols by use of intra-oral access in 91.6% of cases (in one patient an external access was used to remove the reconstructive plate). Information on the local status and patients' general health, the use of medicines, bad habits, working and everyday life conditions, peculiarities of primary surgical intervention, the course of the postoperative period, the clinical and radiological findings of treatment were transferred to the patient's database to analyse the factors related to the intensity of surface degradation and ion exchange between fixators and surrounding tissues.

When removing the fixator, surrounding soft tissues and the bone surface were carefully examined to detect macroscopic signs of metalosis and inflammatory reactions. The attention was paid to the stability of the fixator and the degree of the fixation elements integration with the surrounding bone. The periosteum or fibrous capsule adjacent to the fixation elements were removed and fixed in 10% formaldehyde solution. To determine the elemental composition of the removed soft tissue fragments in accordance with standard analytical techniques, a micro-X-ray fluorescence spectral analysis was carried out by micro-X-ray fluorescence spectrometer (model M4 TORNADO) manufactured by Bruker (Bremen, Germany). The objects of the study were placed in the working chamber of the spectrometer where pressure of 20 mbar was created by means of vacuum pump. The sample was translated into the focal plane using autofocus. The objects of the study (soft tissue biopsy) were exposed to the X-ray beam. Atoms passed into an excited status then emitted fluorescent radiation, which is unique for each element, its intensity was recorded by the detector. The source of X-ray radiation in the spectrometer was a microfocus X-ray tube with operating parameters as follows: voltage of 50 kV and current of 500 μA.

Scanning electron microscopy (SEM) by raster electron microscope JSM-6060 (JEOL, Japan), micron marker 100 micrometer (μm)-500μm, was used for detailed study of structural changes on the surface of titanium plates and screws. The removed fixators were carefully washed with 10% formaldehyde solution to remove the residual biological tissues, then they were degreased, washed in 96% alcohol, and dried in vacuo. Electron microscopy was carried out in different fields of view at magnification of 1:30 and acceleration voltage of 30 kilovolts (kV).

The obtained results were analized with the use of Spirman correlation coefficient, calculated by the IBM SPSS Statistics v.23 software.

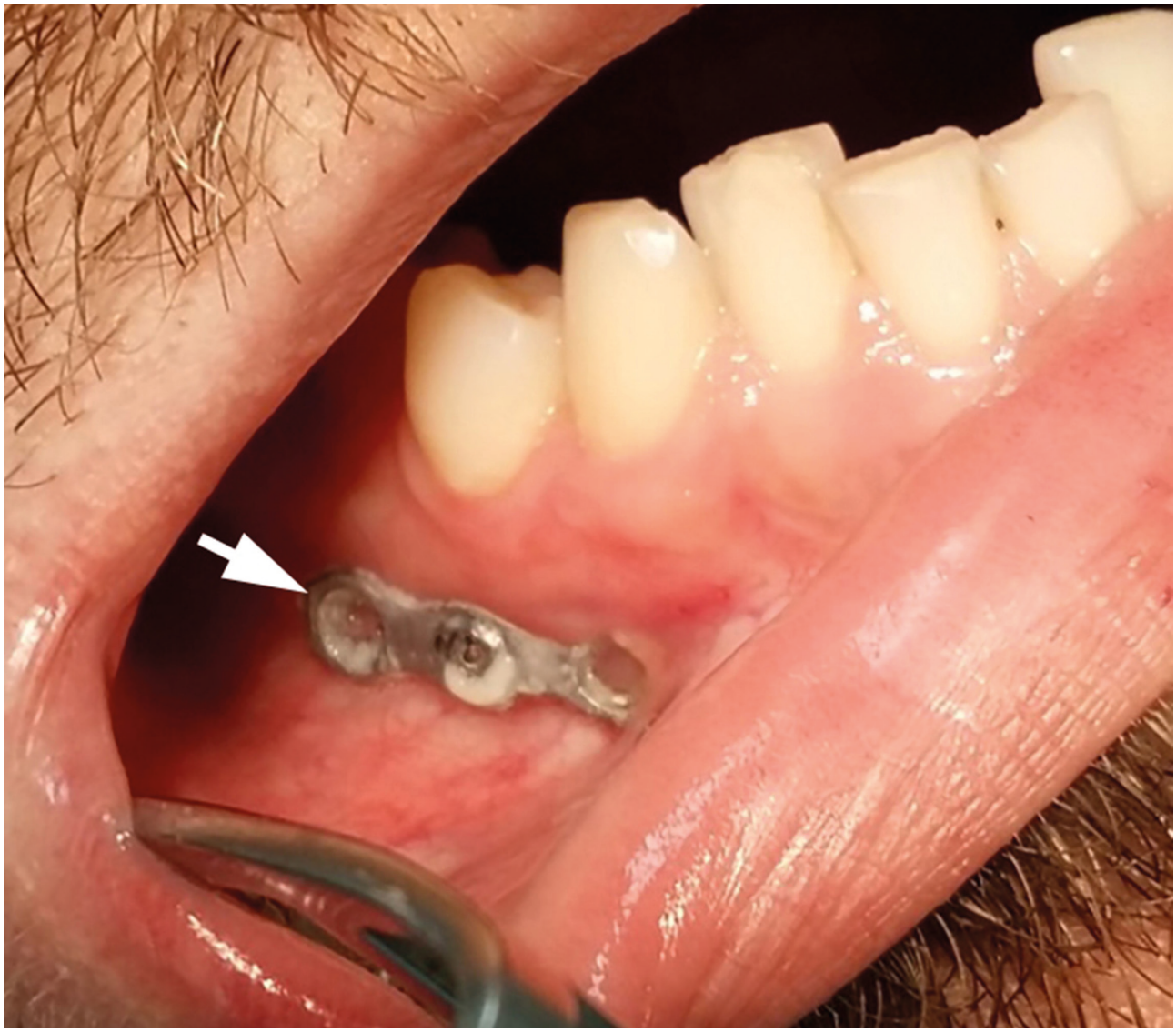


FIGURE 1. A 11.5-month follow-up photograph after mandibular fracture shows exposure of titanium miniplate (*arrow*) in the oral cavity without significant signs of chronic inflammation.

Results

At the removal of the fixation elements, macroscopic signs of chronic inflammation in adjacent soft tissues were noted in 1 (8.3%) patient. Exposure of fixators was noted in 4 (33.3%) patients (Fig 1). Local grey coloring was seen in 8 (66.6%) patients, predominantly in the area of the installed screws. In most observations, the loosening of at least one of the fixing screws was noted.

In all the cases, electronmicroscopy of fixation elements (plates and screws), which were removed in late terms after the installation, revealed signs of surface damage, including macrostructural ones such as deformation of the thread or bending of the screws (Fig 2), various deformations and surface defects of the plates (Fig 3), microcracking, sharp edges, and metal scratches, tongues and splinters. According to our data, the degradation of the surface of titanium fixators resulting from corrosion can hardly ever be determined. In some cases, on the surface of the fixators, there were small dimples which resembled the shells of corrosion, but their true nature was difficult to establish.

The study of soft tissues by X-ray fluorescence analysis of the scanning plane revealed the spectra of the following elements of the periodic table: phosphorus (P), sulfur (S), calcium (Ca), titanium (Ti), chromium (Cr), iron (ferrum) (Fe), nickel (Ni), copper (cuprum) (Cu), zinc (Zn), strontium (Sr), rhodium (Rh) (Table 1; Figs 4, 5). The applied method allowed not only detecting the presence of metals in the tissues, but also studying the features of the distribution. Thus, the presence of sites with an increased content of certain chemical elements in some cases was conditioned by the relief of the plate, contours of its holes, the turns of thread of the fixing screws (Figs 6, 7).

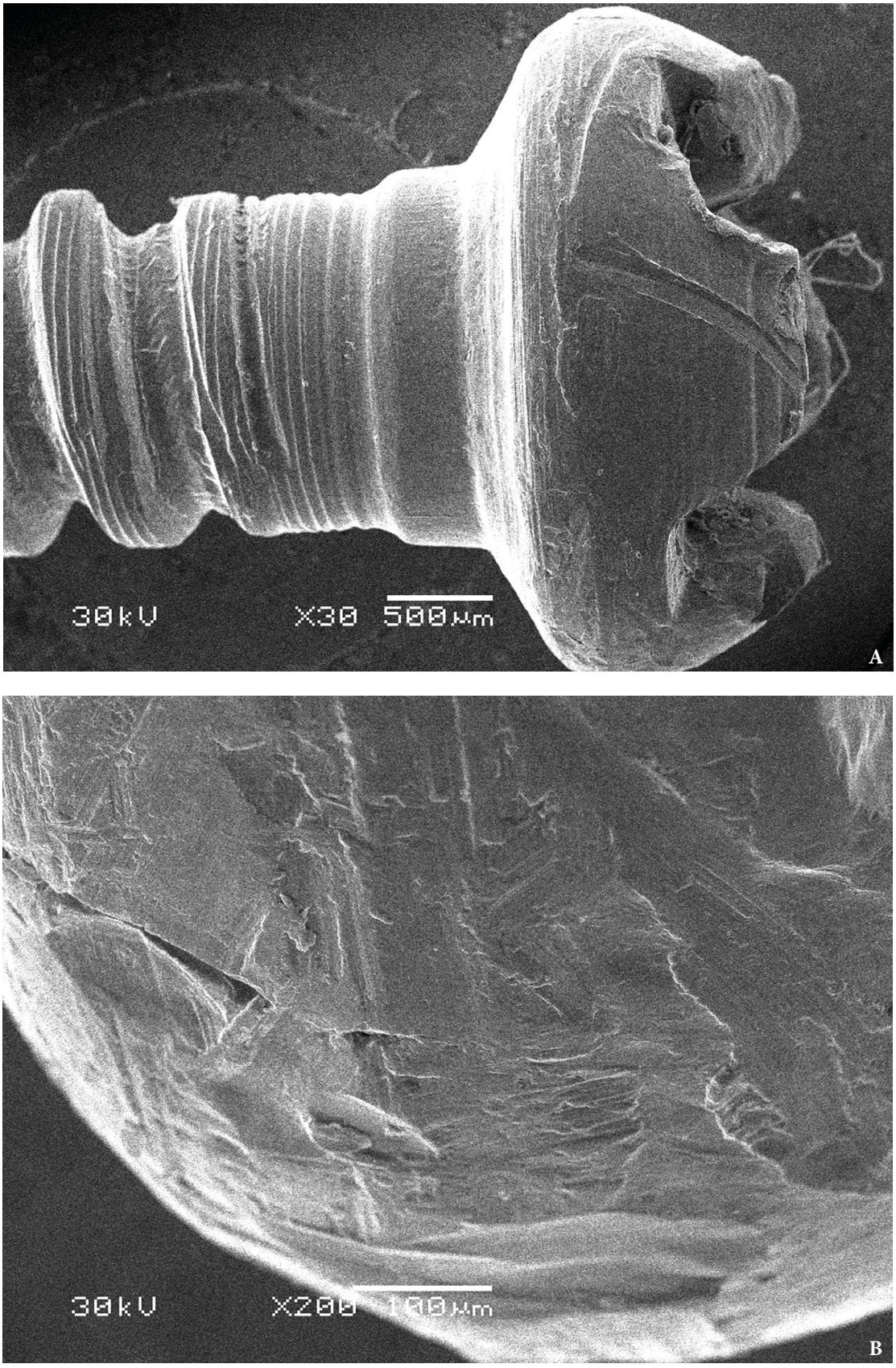


FIGURE 2. (**A**, **B**) SEM surface of the removed titanium screw in different maginifications (**A**: magnification, x 30; scale bar, 500 µm; voltage, 30 kV) (**B**: magnification, × 200; scale bar, 100 µm; voltage, 30 kV). There is seen deformation of screw threads and screw hinge, its bend, numerous defects of the surface, including microcracks, sharp edges, metal scratches, tonges and splinters.

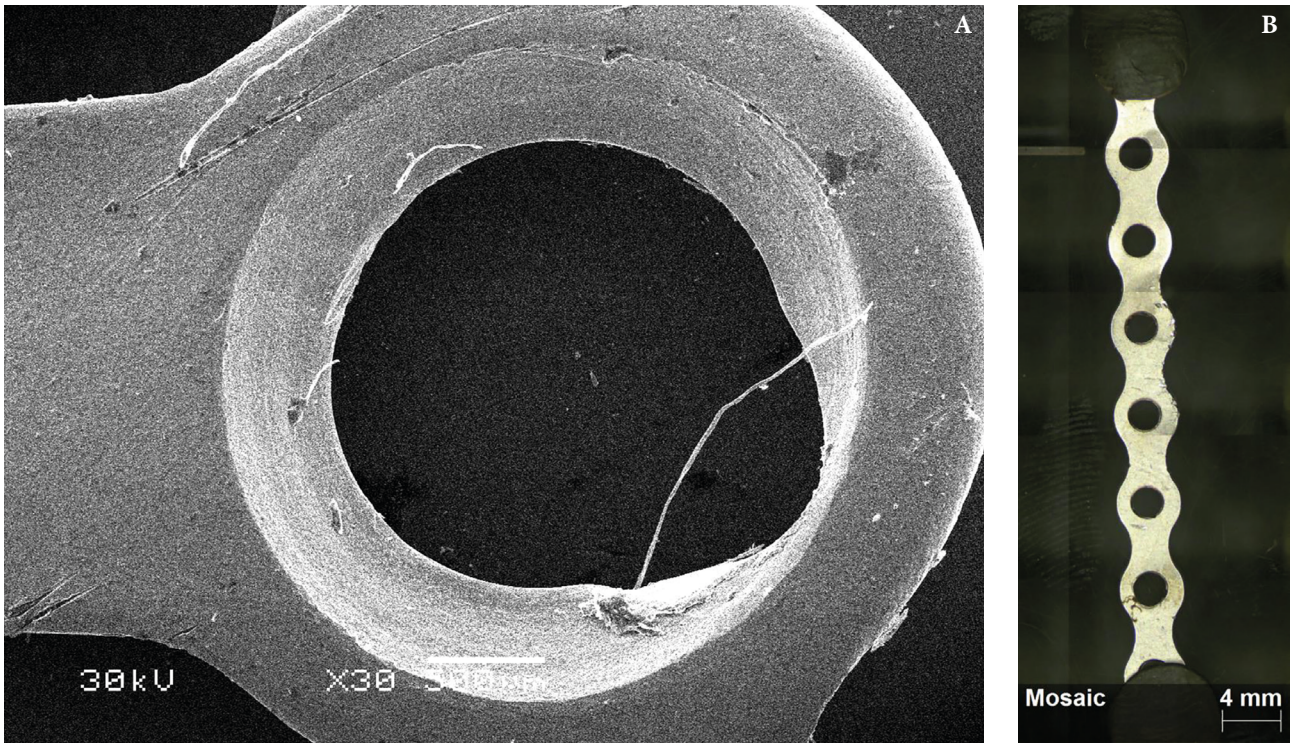


FIGURE 3. Appearance of the surface of the removed titanium miniplate at SEM (**A**: magnification, × 30; scale bar, 500 μm) and at optical magnification, × 10 (**B**; scale bar, 4 mm). The deformation of the screw hole, scratches, surface defects, microcracks, sharp edges, metal tonges, and splinters are seen.

TABLE 1. The obtained spectrum and concentration of chemical elements in the investigated areas. Mass percent (%)

Spectrum	P	S	Ca	Ti	Fe	Ni	Zn	Sr	Rh
Point 2	1.35	3.34	0.46	88.22	5.39	0.06	0.12	1.06	0.00
Point 1	0.40	0.68	0.17	97.29	1.29	0.04	0.01	0.10	0.00
Mean value	0.88	2.01	0.32	92.76	3.34	0.05	0.07	0.58	0.00
Sigma	0.67	1.88	0.20	6.42	2.90	0.01	0.08	0.68	0.00
Sigma mean	0.47	1.33	0.14	4.54	2.05	0.01	0.05	0.48	0.00

P – phosphorus; *S* – sulfur; *Ca* – calcium; *Ti* – titanium; *Cr* – chromium; *Fe* – ferrum (iron); *Ni* – nickel; *Cu* – cuprum (copper); *Zn* – zinc; *Sr* – strontium; *Rh* – rhodium

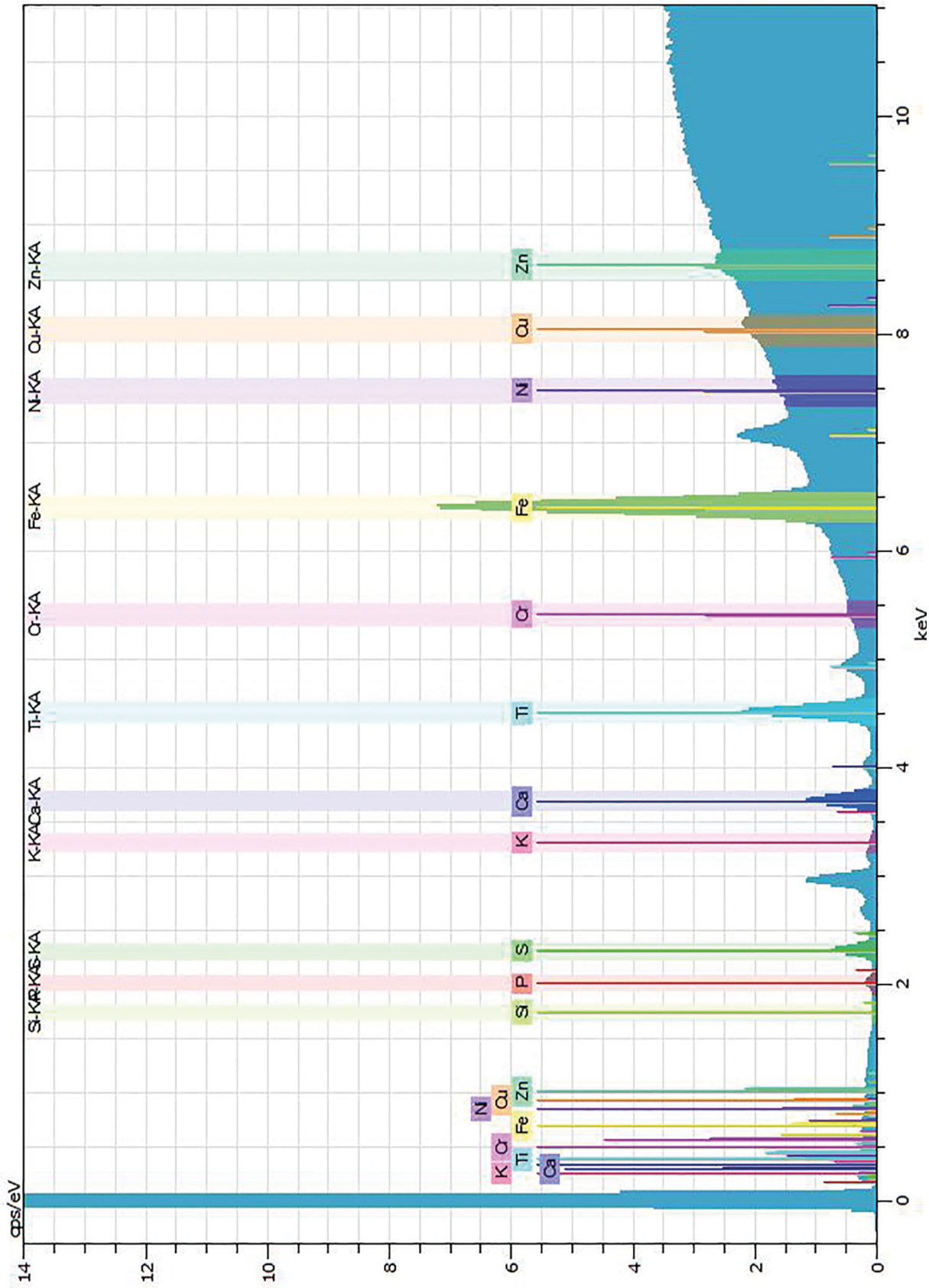


FIGURE 4. The obtained spectrum and concentration of chemical elements in the investigated areas (percentages by mass).

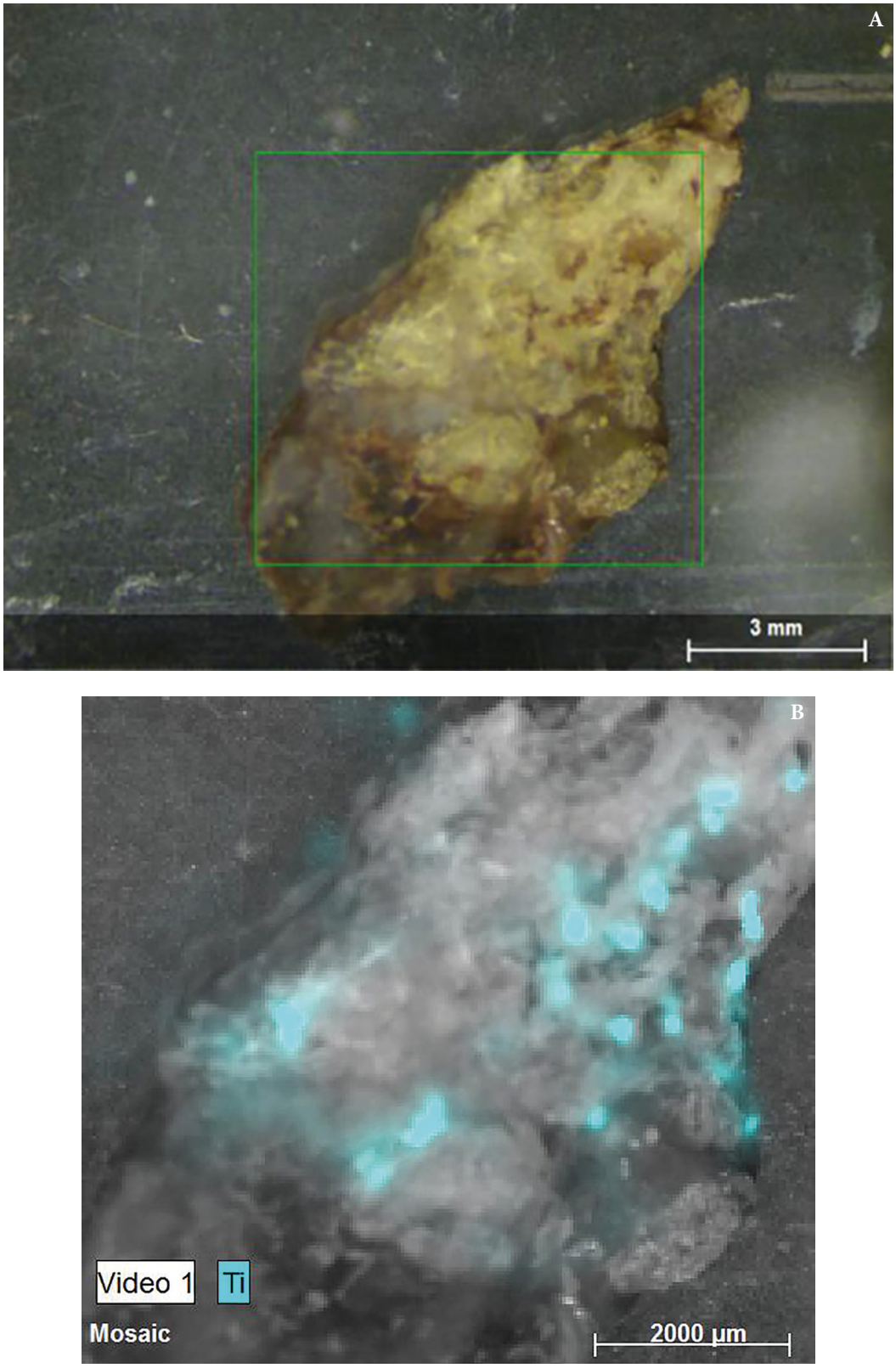


FIGURE 5. Analysis of the study of scanning area of biopsysample of soft tissue. (**A**) Area of scanning (scale bar, 3 mm.) (**B**) Distribution map P, S, Ca, Ti, Fe, Zn (scale bar, 2000 µm.)
(Fig 5 continued on next page.)

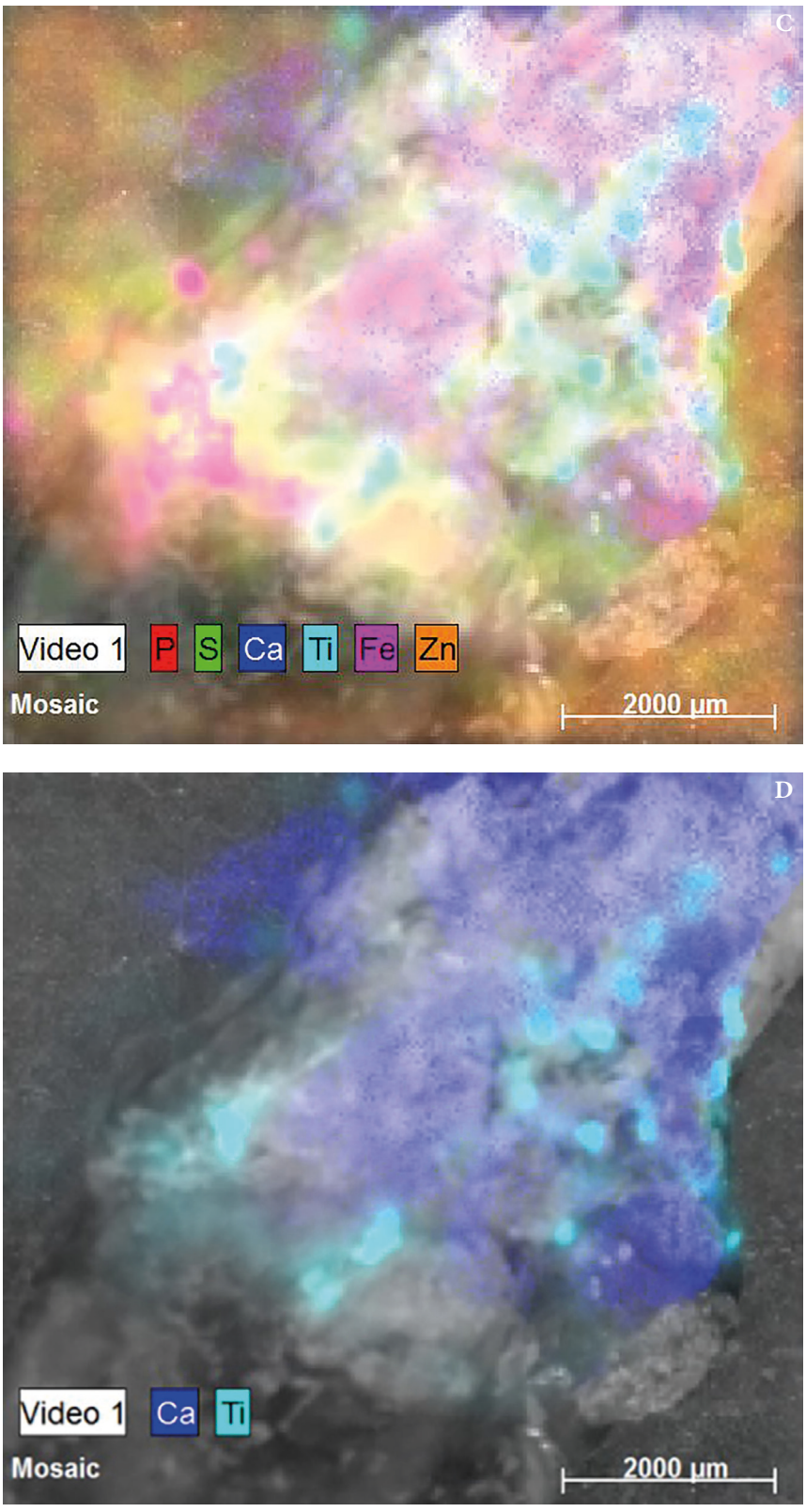


FIGURE 5. (cont'd). (**C**) Distribution map Ti on the scan area (other elements are hidden). (**D**) Map of Ca, Ti distribution on the scan area (scale bar, 2000 µm.)

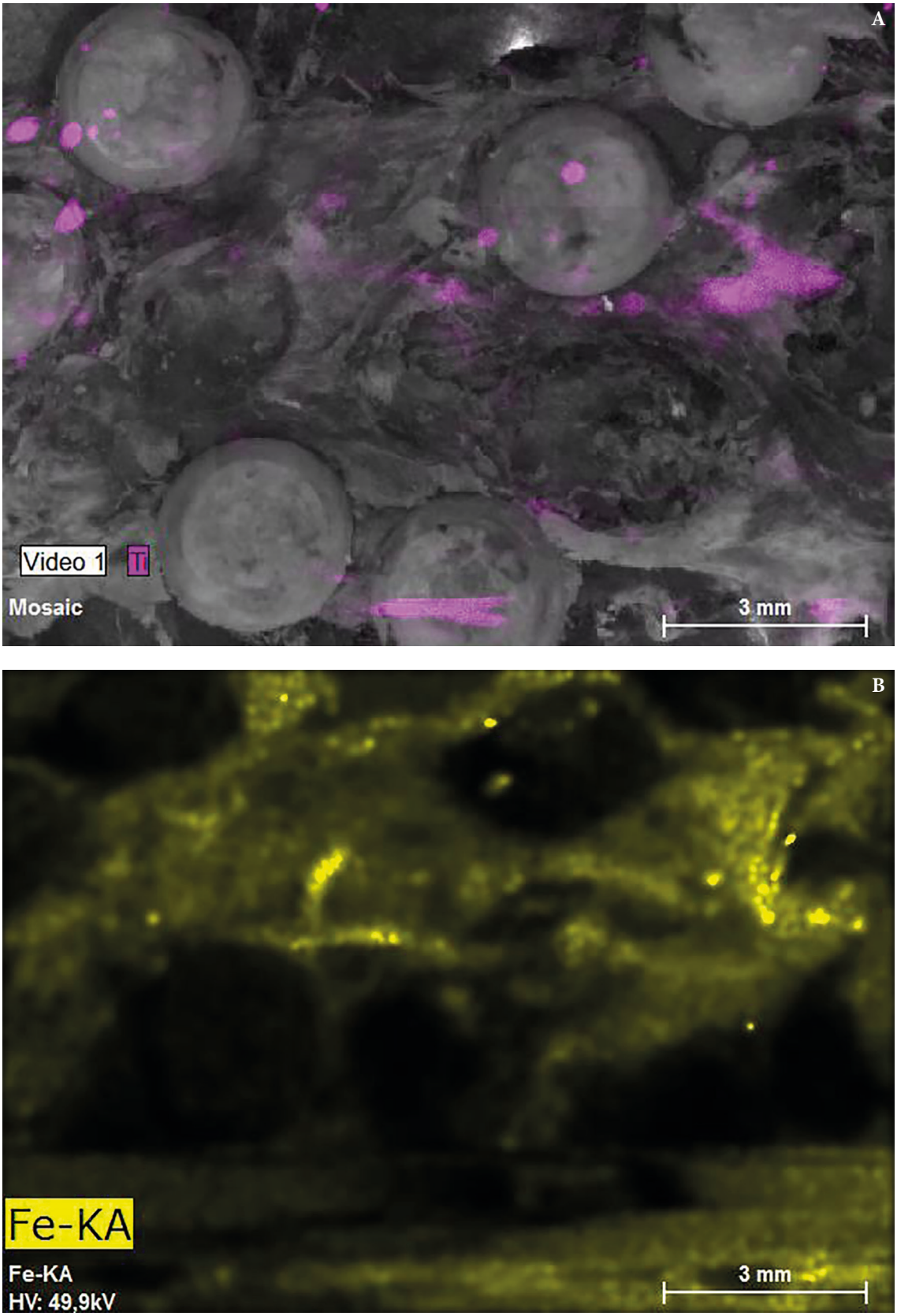
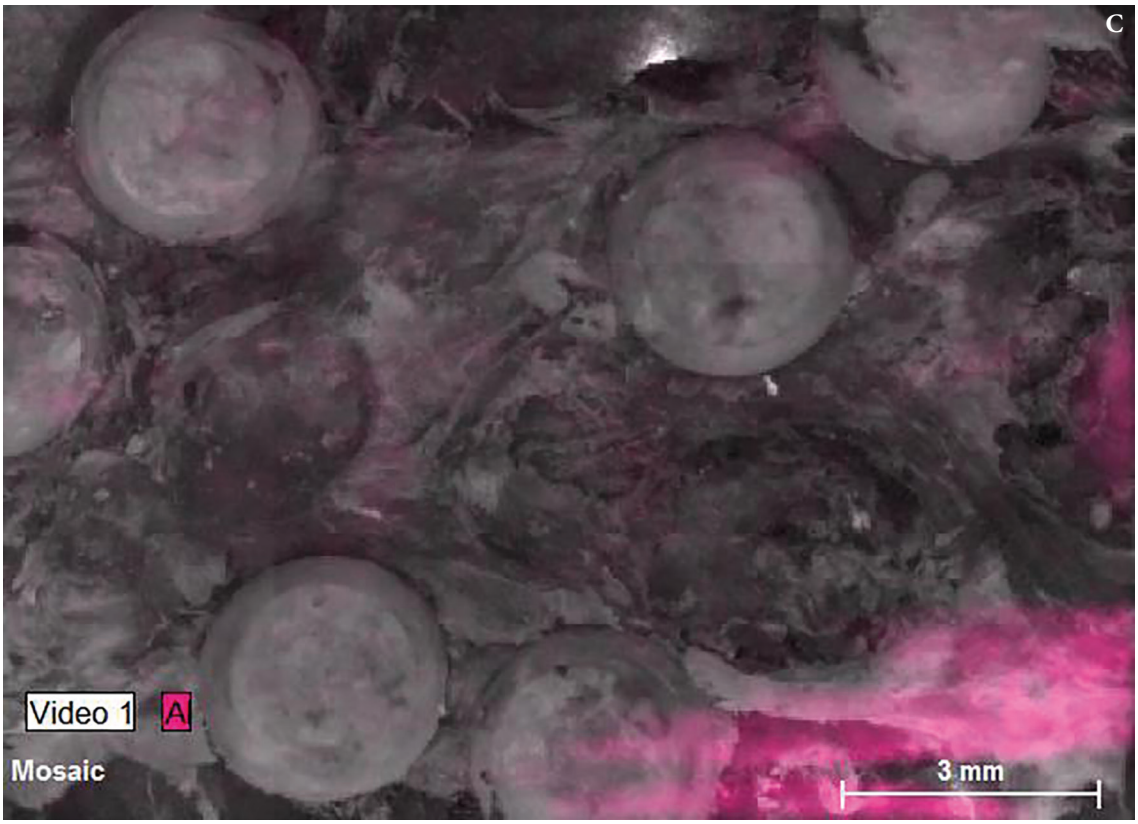


FIGURE 6. Ti (**A**), Fe (**B**), Al (**C**), and Ca (**D**) distribution maps on the scanning area of soft tissue adjacent to the removed titanium grid (two types of titanium distribution are noted: the first one is represented by clearly outlined intense inclusions sized 100 -800 μm and more and high titanium content (up to 90%); around these particles and in areas close to the fixator, there are poorly outlined diffuse inclusions of titanium (second type) with lower percentage content (**A**). The detected iron could be either of biological origin or it could get into the tissues from the surface of the surgical instruments used to install the fixators (**B**) (scale bar, 3 mm.) **(Fig 6 continued on next page.)**



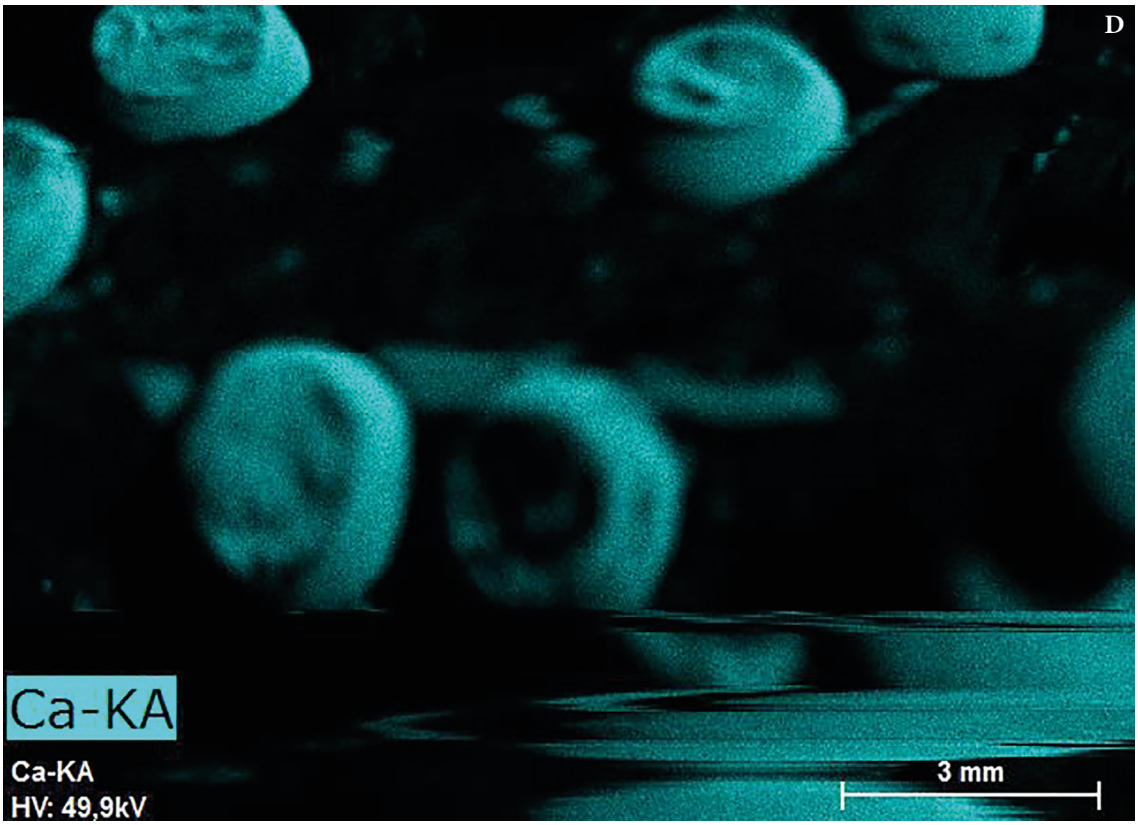


FIGURE 6. (cont'd). Al was detected in very small quantities and it was topographically linked with the sites of titanium deposition (**C**). The revealed Ca was unevenly distributed, its increased content was seen in areas of periosteal osteogenesis, including in the areas of free holes (**D**) (scale bar, 3 mm).

The received maps showed a quite uniform distribution of P, S, Cu and Zn which are normally present in large quantities in soft tissues. Ca was present in all the specimens studied, but the distribution parttens were nonuniform. An increase in its content was seen in sections of periosteal osteogenesis around the plate, including the area of its free openings (Fig 6D). Moderate amount of Fe was noted in all samples. The detected iron could be either of biological origin, due to its presence in hemoglobin, or it could get into the tissues from the surface of the surgical instruments used to install the fixators. Thus, in 41.6% of cases in some local sites along with high Fe deposits there were found Cr and Ni, which are constituents of medical steel (Fig 7). Small amount of Sr was seen in all observations, which is generally characteristic of this geographic area. In 3 (25%) cases, insignificant amount of Al (4.57 ± 5.13%) was detected which was topographically linked with areas of titanium deposition.

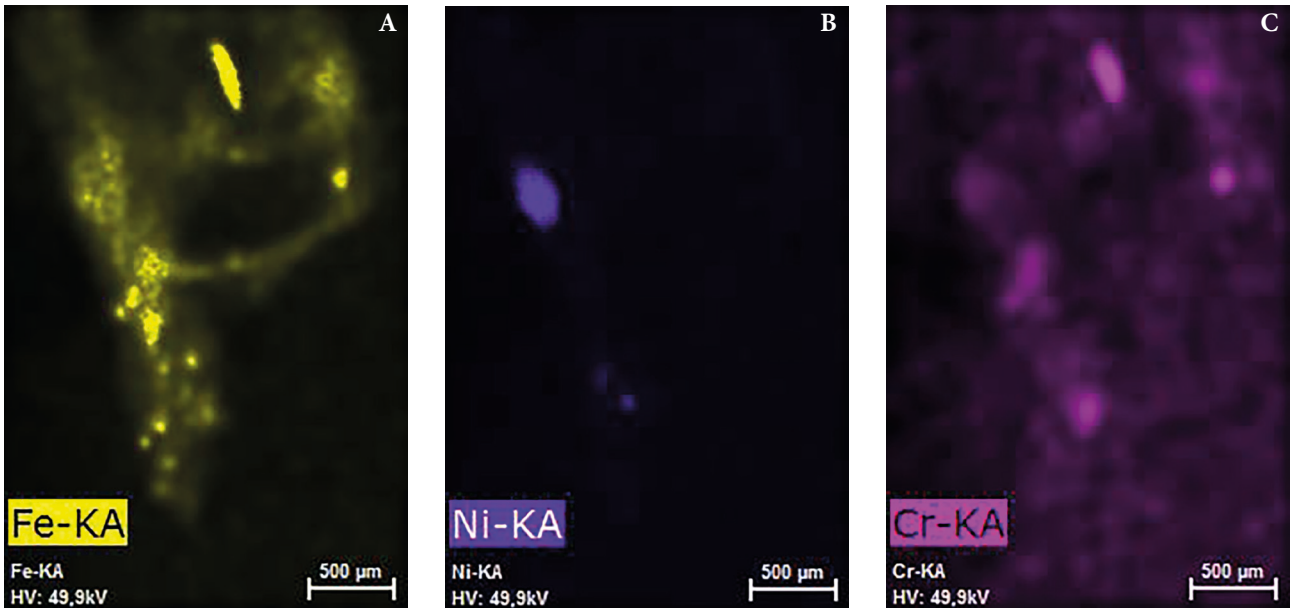


FIGURE 7. Fe, Ni, Cr distribution maps (**A-C**) in the area of scanning (scale bar, 500 µm) of soft tissues adjacent to the removed titanium screw. In some local sites along with high Fe deposits, there is seen Cr and Ni, which are constituents of medical steel.

The presence of Rh in the spectrum can be explained by the material of M4 TORNADO tube emitting continuous radiation and bremsstrahlung which affects the spectral background of the excited spectrum inclusion.

The average content of titanium of 48.14 ± 31.1% at sites of local deposition was detected in all the studied samples (Fig 8). Samples removed following osteosynthesis in patients with traumatic fractures of the facial skull bones, showed the average content of titanium at the sites of metal deposition being 55.6 ± 29.4%, whereas in the samples removed in patients with reconstructive and restorative interventions, where fixators had been less loaded, it was less (37.72 ± 30.2%). As can be seen from the above, although titanium alloys are considered bioinert and the ones which do not actually interact with the internal environment, the data suggest that titanium plates and screws on the surgical site eventually undergo active transformations resulting from physical and chemical processes. The latter can proceed more intensively if the fixation elements are exposed to a significant stress and deformation.

The acquired maps of the element distribution show uneven distribution of titanium with two types of titanium inclusions and different character of their distribution revealed in all cases. The first type is represented by clearly outlined intensive inclusion with dimensions of 100-800 µm and more and high titanium content (up to 90%). The presence of such inclusions can be explained by the separation of large debrises of fixing elements during the procedures through the contact between the moving drill and the hole of the plate or between the screw thread, plate and bone. Around these particles and in tissues adjacent to the fixators, there were also detected poorly demarcated, diffuse inclusions of titanium (second type) with a lower percentage of this chemical element, which probably resulted from surface corrosive degradation of the fixator.

The analysis of the obtained data revealed no significant impact of the titanium content in tissues on the development of inflammatory complications and exposure of the plate ($r = 0.465$, $p > 0.05$). The correlation between the content of titanium and the duration of the period while the plate remained in the human body ($r = 0.38$, $p > 0.05$), between the content of titanium and the type of plate ($r = 0.237$, $p > 0.05$) also turned out to be unsignificant.

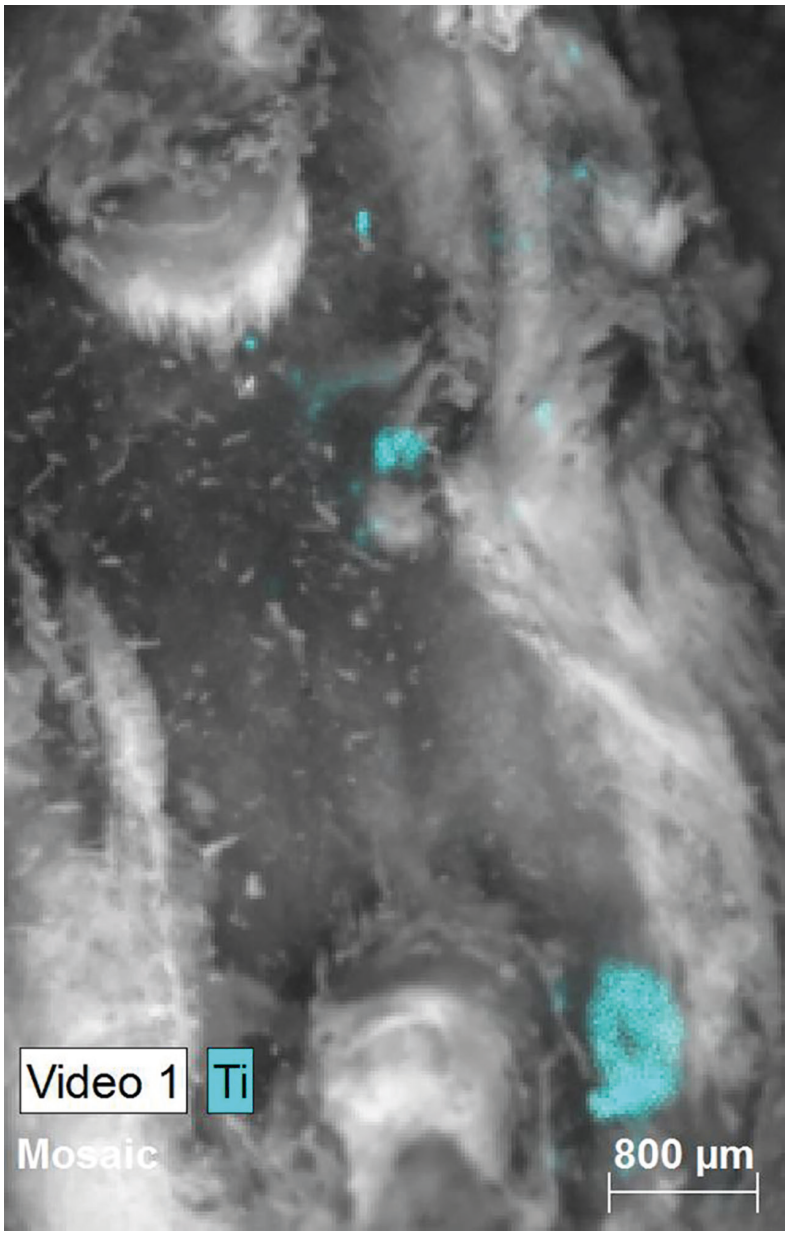


FIGURE 8. Ti distribution map. In the scan area of the removed fragment of the periosteum adjacent to the plate (magnification, × 10; scale bar, 800 µm), there are clearly limited intensive inclusions sized 100-800 µm with titanium content (*blue color*) of 92.76%.

Discussion

The study of mechanisms behind the interaction of titanium fixators with biological tissues during their prolonged presence in a human body is very important for defining strategy and indications for removing fixation elements, and for long-term prognosis of surgical interventions.

Numerous studies indicate that metal particles and ions can be released from the surface of the plates into the surrounding tissues, under the influence of mechanical, chemical and biological factors. According to our data, titanium inclusions in soft tissues adjacent to the fixation elements were found in all 100% of samples in terms of more than 5 months. Jonas *et al* [12], and Theologie-Lygidakis *et al* [47] reported somewhat lower figures: according to their research, titanium inclusions were detected in 20-68% of investigated biopsy samples by means of light, transient electron microscopy, X-ray microanalysis, or electron diffraction. Sections of biopsy specimens used by the authors were of different thicknesses following preliminary preparation. We associate discrepancies in the results obtained with the technical limitations of the research methods used by the authors [7, 11, 31, 38], in comparison with which X-ray fluorescence analysis has greater accuracy and informativity.

When analyzing the distribution of metals in samples of biological tissues, we also found two types of titanium inclusions that had different characteristics. More often, titanium was represented as intense, clearly otlined

inclusions (particles) sized from 100 to 800 μm and more. The content of titanium in these areas averaged 48%. In addition, around these particles and in areas adjacent to the plate, the diffusion of titanium inclusion was poorly outlined.

Based on light and electron microscopy findings, several authors also reported the presence of 2 types of titanium particles in soft tissues following a long-term implantation of miniplates: 1) colloidal particles located in histiocytes, fibroblasts or intercellular space, and 2) larger metal fragments [2, 7, 10, 11, 20, 27, 31, 38].

According to the researchers, larger particles of titanium [7, 28], resulted from the mechanical damage during the installation of fixation elements, including damage to the surface by surgical instruments, by a drill to form holes for fixing screws, titanium chipping while tightening these screws, and friction that occurs between the fixation elements under functional load conditions, especially due to insufficient stability of the 'fixator-bone' system [7, 28, 36]. Such a mechanism for the formation of large titanium particles is indirectly confirmed by the deposits of iron, chromium and nickel (which are the constituents of the medical steel used for manufacturing of surgical instruments) close to the large titanium inclusions in 41.6% of observations.

According to the authors, small colloidal particles of titanium are of different origin. It is believed that they are likely to arise as a result of the titanium biocorrosion [7, 19, 26, 38].

The mechanism of osteosynthesis devices corrosion is complex and probably includes four main components: depassivating, fretting, galvanic component and exposure to local factors of surrounding biological environment [48, 49]. Titanium plates and screws exhibit high corrosion resistance in the presence of a surface oxide layer that is chemically inert. The loss of this layer under the exposure to mechanical, chemical and biological factors (depassivation) results in a partial dissolution and degradation of the titanium surface that occurs intensively in the presence of reactive oxygen forms and electrolytic (electro-chemical) processes [12, 25]. The protective oxide layer on the surface of the plates quickly restores, except for the conditions when the 'fixator-bone' system is not sufficiently stabile and its elements are exposed to constant friction during repeated masticatory and non-masticatory movements.

To understand which of the mechanisms for the release of titanium into tissues is more important, it is significant to study the surface of the fixators removed at different terms following osteosynthesis. When conducting this study on the surface of all removed fixators we found such signs of mechanical damage as scratches, microcracks, surface defects, dimples, sharp edges, metal tongues and splinters that may have occurred during the manufacturing, installation, operation, and removal of the fixator. The above defects are a likely source of titanium fragments in the tissue adjacent to the fixator. In the case of infection or exposure of the plate, they act as retention points for the fixation of microorganisms and the formation of biofilms responsible for the development of chronic inflammatory processes and they are the main reason for the removal of fixators [21, 34]. No defects that could be uniquely qualified as signs of corrosive degradation were seen on the surface of the fixators. In few observations, we noted minor single surface defects of rounded shape similar to the corrosion shells occurring on the surface of steel structures. Such defects could have occurred during manufacturing of plates as evidenced by the studies conducted by Acero *et al* (1990) [50], Torgersen and Gjerdet (1994) [51].

Langford [34] reported similar findings resulted from the analysis of surface changes of removed plates and screws during an observation period of up to 13 years following osteosynthesis of the facial bones. He notes that surgical procedures and defects in the production of titanium miniplates were likely to be the main source of metal particle release into the tissue. In his study, no evidence was found to confirm that titanium miniplates installed for osteosynthesis of the facial bones should be routinely removed due to corrosion [34].

Interestingly, the signs of diffusion of small colloidal titanium inclusions associated with the corrosion process were observed mainly around large particles (fragments) of the metal. Probably the particles arising from mechanical damage and defects in the surface of the plate deprived of protective oxide layer are the main source of corrosive release of metal ions into the tissue due to increased surface area, depassivation, and capability of triggering cellular and tissue responses. The degradation in this regard may occur more intensively than the destruction of the fixator surface contacting with biological tissues. In favor of this hypothesis, French (1984) [8] shows that the formation of metal particles significantly increases the surface area available for the oxidation and release of ions into biological tissues. Jonas observed the initial signs of surface degradation of titanium alloys in the areas of fixator damage and believed that damage caused by the procedure for plate installing was the starting point for biocorrosion. Similar results were reported in other studies [15, 35, 41]. According to French [8], even in stainless steel fixators, reliable signs of corrosion were noted only on a small area of the screw-plate contact. The researcher did not find the link between the severity of corrosion and the duration of period when the fixator remained in the human body. In his opinion, it is indicative of the fact that the most intensive processes of corrosion proceed immediately following the installation the fixator, then they slow down and almost stop.

Our study found no significant correlations between the content of titanium in the tissues and the time the plate remained in the human body, either. In addition, the release of metal particles did not seem to depend on the manufacturer or the type of plate used.

The biological significance of the biodegradation

of metal fixators with the release of metal particles into tissues and the related potential risks are the subjects of discussion. According to Rae (1986) [52], metal particles of 1 to 10 μm are capable of activating monocytes and macrophages *in vitro*, and they also stimulate the release of mediators of bone resorption, prostaglandin E2 and interleukin-1, directly stimulate fibroblasts, and increase the synthesis of collagen. This determines their potential capability of causing inflammatory reactions. In addition, according to a number of researchers, interleukin-1, a potent bone resorbing agent, may be responsible for the loosening and loss of screws in the absence of infectious suppurative and inflammatory complications.

However, numerous studies of biopsy samples of soft tissue adjacent to the fixator, in the overwhelming majority of cases, revealed only minimal or poorly marked signs of chronic inflammation with minor lymphocyte-macrophage infiltration, less often with granular formation and small focal areas of necrosis. Such a tissue response was seen only in the presence of metallic inclusions in the tissues and was topographically related to them. At the same time, French [8] reported about a time-related decrease in the severity of the inflammatory tissue response in cases the fixator remained in the body for a prolonged period. The response did not depend on the degree of metallosis.

We did not find a significant correlation between the content of titanium in the tissues and the clinical manifestations of inflammation in the area adjacent to the fixator, either. Such manifestations were mainly conditioned by the occurrence of an infection, exposure of the plate and biomechanical characteristics of the system (instability, loosening, and loss of screws).

Literature review shows that to date, there is no convincing clinical evidence of titanium fixators contribution in the occurrence of inflammatory reactions in the surrounding soft tissues due to corrosion and the release of metal particles, the capability of aggravating the remote prognosis of surgical interventions and causing harm to the patient's health. Hypothetically, corrosion and mechanical damage to titanium fixators made of alloys containing vanadium and aluminum (metals whose toxicity is proved), can lead to their release into the tissue. The effect of these toxic components was studied only in isolated studies, which did not confirm the crucial role of aluminum and vanadium in the occurrence of inflammatory reactions in tissues adjacent to the titanium miniplates. According to our data, a very little amount of aluminum (4.57 ± 5.13%) was detected in the tissues adjacent to the fixator only in 25% of cases. There was no evidence of the presence of vanadium in biopsy samples.

Another potential risk is associated with the capability of titanium depositing not only in the tissues adjacent to the fixator, but also in tissues and organs distant from the site of osteosynthesis. Onodera *et al* (1993) [53] identified titanium particles in the submandibular lymph nodes of the patient 2 years after the reconstructive plate was installed on the mandible. Bessho *et al* (1993) [19] showed that titanium released from the miniplates can enter the vascular system and spread from the implantation site to distant organs. Biological effects of titanium in this case are practically uninvestigated.

The insight into the uncertainty about the long-term side effects of metal plates was provided in the recommendation of the Strasbourg Osteosynthesis Research Group (S.O.R.G.) in 1991, which concluded that the removal of non-functional titanium miniplates is desirable, provided that the procedure does not cause a significant risk to the patient. However, based on a survey of a significant number of maxillofacial surgeons, Matthew and Frame (1999) [41] found that miniplates and screws are not routinely removed. The decision to remove miniplastes in the maxillofacial area is taken in the presence of complications, certain clinical symptoms or at the patient's insistence [34]. In these conditions, the role of measures aimed at reducing the penetration of metal particles in the tissue and the associated negative effects significantly increases.

So, our research suggests that titanium fixators interact with the surrounding biological environment. This is accompanied by the release of metal particles into adjacent tissues, which was observed in all investigated samples. The main mechanisms involved into release of titanium into the adjacent biological tissues are corrosion and mechanical damage to the surface of the fixator by surgical instruments, drills, etc. during its installation, the contact of the plate and the thread of the fixing screws when they are screwed in, the friction of the elements in fixator-bone system, especially with insufficient stability of osteosynthesis, loosening of screws, plastic deformation of plates. According to our data, the degradation of the surface of titanium fixators due to corrosion can hardly ever be determined. Biocorrosion occurs mainly around small particles (debrises) of titanium and in areas of mechanical damage to the surface deprived of a protective oxide layer therefore more exposed to the chemical and biological factors of the environment. The main way to reduce titanium penetration into surrounding tissues is to minimize mechanical damage to the plate during its installation and operation. This implies, in particular, to follow the manufacturer's protocol for fixator installation, to avoid plate bending in the wound and the contact of the plate with the drill while making holes in the bone, to use titanium or ceramic drills, to install screws perpendicular to the surface of the plate, not at an angle to it, to employ surgical techniques and fixators that ensure the functional stability of the 'fixator-bone' system and minimize friction between its elements, to avoid conditions under which the plate is exposed to plastic deformation and destruction at the micro and macro levels in the process of functioning. Given the potential risk of the release of toxic impurities from titanium alloys into tissues, it is reasonable to search and develop new materials and alloys with improved biological and mechanical properties.

Conclusions

1 Following osteosynthesis and reconstructive interventions on the facial bones, titanium miniplates and screws interact with the surrounding biological environment which results in the release of metal particles into the adjacent tissues observed in all 100% of the studied biopsy samples wthin the time periods from 5 months to 3 years.

2. The main mechanisms involved in titanium release into surrounding tissues are corrosion and mechanical damage to the surface of the fixator by surgical instruments during its installation, the contact of the plate and the thread of the fixing screws when they are screwed in, and the friction of the elements in 'fixator-bone' system under functional load. In this case, the biocorrosion is of lesser importance and it occurs predominantly around tiny particles (debrises) of titanium and on sites of mechanical damage to the surface of the fixator, which loose the protective oxide layer and became exposed to the chemical and biological influences of the environment.
3. The distribution of metals in samples of biological tissues was characterized by the presence of two types of titanium inclusions that had different characteristics. More often, titanium was detected as intense, clearly outlined inclusions sized from 100 to 800 μm, with a high content of titanium (an average of 48.1 ± 31%) resulted from mechanical damage to the fixation elements during their installation. In addition, around these particles and in areas adjacent to the plate, there were also detected poorly outlined diffuse inclusions of titanium, where its content was lower.
4. There was no significant correlation between the content of titanium in tissues and the time the plate remained in the human body ($r = 0.38$, $p > 0.05$), between the content of titanium and the development of inflammatory complications or exposure of the plate ($r = 0.465$, $p > 0.05$), between the titanium content and the type of plate used ($r = 0.237$, $p > 0.05$).
5. The main approach to reduce titanium penetration into surrounding tissues is to minimize mechanical damage to the plate during its installation and functioning. This implies, in particular, to follow the protocol for fixator installation, to avoid plate bending in the wound and the contact of the plate with the drill while making holes in the bone, to install screws perpendicular to the surface of the plate, not at an angle to it, toemploy surgical techniques and fixators that ensure the functional stability of the 'fixator-bone' system and minimize friction between its elements, to avoid conditions under which the plate is exposed to plastic deformation and destruction at the micro and macro levels, and to use alloys with improved biological and mechanical properties.

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Conflict of Interests

The authors declare that they have no conflict of interest.

Role of Author

The authors are equaly contributed to that article.

Ethical Approval

The study protocol (#106, November 07, 2017) was approved by the Bioethics Commission of the Bogomolets National Medical University, Kyiv, Ukraine.

Patient Consent

Not required.

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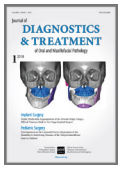

Highly Predictable Augmentation of the Alveolar Ridge: Using a Ribbed Titanium Mesh in Two-Stage Implant Surgery at the Mandible. Report of Clinical Cases and Surgical Technique*

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ABSTRACT

Purpose.
The aim of this prospective surgical note was to evaluate the highly predictable horizontal bone gain of the alveolar ridge augmentation in two-stage implant surgery at the mandible with titanium mesh.

Material and Methods.
Five patients treated with 10 implants and simultaneous guided bone regeneration with ribbed titanium meshes (i–Gen®, MegaGen, Seoul, Republic of Korea) were selected for inclusion in the present surgical note. Primary outcomes were highly predictable horizontal bone gain of the alveolar ridge augmentation, secondary outcomes were biological and prosthetic complications.

Results.
After the removal of titanium meshes, the cone beam computed tomography (CBCT) showed a mean horizontal bone gain of 2 mm. The most frequent complications were mild postoperative edema (40% of patients) and discomfort after surgery (60% of patients); these complications were resolved within one week. Titanium mesh exposure occurred in 0 patients. And implant survival rate of 100% (implant-based).

Conclusions.
The horizontal ridge reconstruction with titanium meshes placed simultaneously with dental implants achieved predictable satisfactory results.

Introduction

In our opinion the best way to restore partially dentition defect for nowadays is dental implantation.

Dental implants are a predictable treatment procedure for the prosthetic rehabilitation of partially and fully edentulous patients [1–3]. But there is a lot of cases in our everyday practice (35%) that seems with CBCT not an adequate bone volume to place implants.

An adequate bone volume is required for insertion of dental implants [4, 5]; the absence of a sufficient amount of horizontal and vertical bone is a problem that can affect the survival and success rates of dental implants in the short, medium, and long term [4, 5]. Since frequently patients present with bone defects of variable entity [4, 5], different surgical techniques have been proposed to restore the ideal anatomical conditions required for implant insertion or to allow simultaneously positioned implants to succeed [6–14]. These techniques include onlay/inlay bone grafting [6, 7], distraction osteogenesis [8], maxillary sinus augmentation [9], inferior alveolar nerve transposition [10], alveolar ridge split [11], and guided bone regeneration (GBR) with resorbable [12] and nonresorbable membranes, such as those in polytetrafluoroethylene (PTFE) [13] or titanium [14], partial extraction therapies [28]. GBR is considered one of the most predictable of these techniques in terms of clinical outcomes, as reported by several systematic reviews of the literature [12–15], particularly where it is employed for the regeneration of defects of small and

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medium entities [16], or around dental implants [17]. The operating principle of GBR involves the placement of a mechanical barrier for the protection of the clot and the isolation of the bone defect from the surrounding connective tissues, in order to facilitate the selective recruitment of the mesenchymal cells responsible for new bone formation [12-15, 17]: this can allow the regeneration of the bone defect.

Bone regeneration with GBR has been demonstrated to be predictable, whether or not bioma-terials are positioned below the membrane and are contained by it [12, 14, 16].

An ideal membrane should possess the following characteristics: biocompatibility, space maintenance capabilities, and ease of use [13, 14, 17, 18]. In the last few years, several types of membranes with different designs have been introduced, to facilitate the containment of the rege-nerative material that is often positioned below it and to prevent its dispersion, but also to simplify the work of the surgeon and the application of the membrane itself [13-18].

In particular, the titanium meshes represent a valid solution, because they meet most of the ideal requirements that a membrane should possess [14, 15]. Several clinical studies have demonstrated that titanium meshes can promote the formation of new bone, when positioned before [19-24] or simultaneously with dental implants [25-27].

The proper placement and stabilization of the titanium mesh into the defect site is of fundamental importance for the success of the regenerative therapy [13, 16-18]; one of the difficulties with these membranes can be related to this, particularly in case of simultaneous placement of the implant, for regeneration of small and medium size defects [17, 18, 25-27].

Recently, titanium meshes that can be fixed directly on the implant have been introduced, but there is still a lack of clinical studies evaluating the efficiency and predictability of these membranes [18, 26].

Therefore, the purposes of the report are 1) to evaluate the horizontal bone gain and the de-gree of complications in patients treated with titanium meshes positioned simultaneously with den-tal implants and fixed over them 2) to give for colleagues a new approach for the bone augmenta-tion technique.

In our clinical cases (target group) there were five missed tooth 3.6 (Fig 1) for some years with vestibular horizontal bone atrophy, that we exam on CBCT (Fig 2).

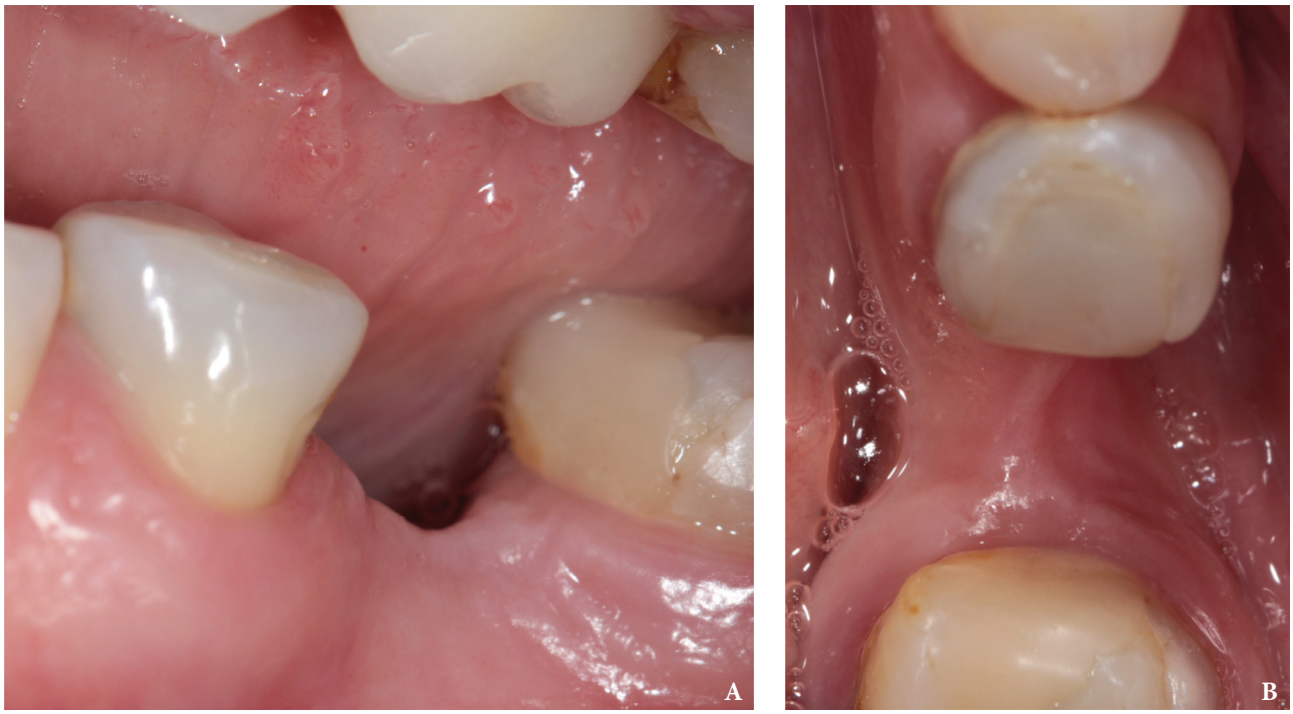

FIGURE 1. Preoperative clinical view in an area of a loosed tooth 3.6.

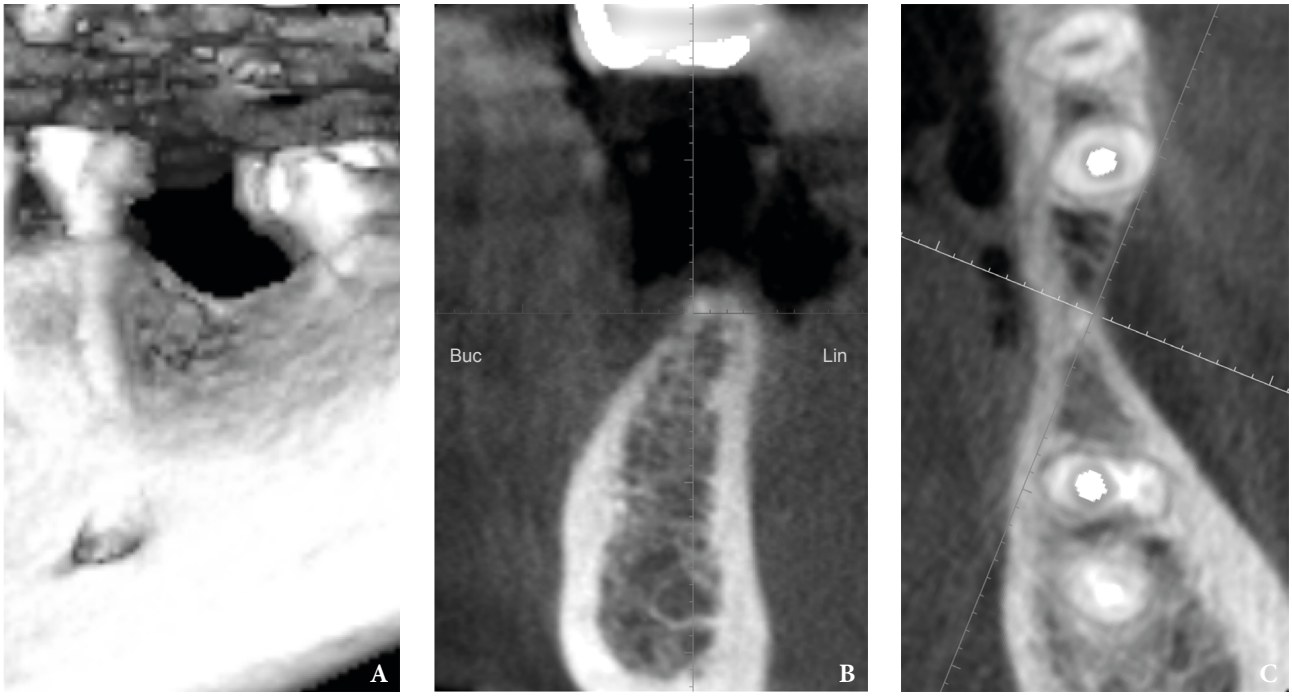

FIGURE 2. Preoperative cone beam computed tomography (CBCT). 3D reconstructed (**A**), coronal (**B**: Buc = buccal side, Lin = lingual side.), and axial (**C**) scans of the mandibular bone in are of missed tooth 3.6.

We prefer to restore this partial edentulous using implant placement (AnyOne; MegaGen, Seoul, Republic of Korea) with GBR (Laddec; OST Développement, Clermont-Ferrand, France) and titanium mesh i-Gen (MegaGen, Seoul, Republic of Korea) (Fig 3) to achieve predictable vestibular bone gain before the implants and do one step surgery.

FIGURE 3. Ribbed titanium mesh before bone augmentation surgery at mandible.

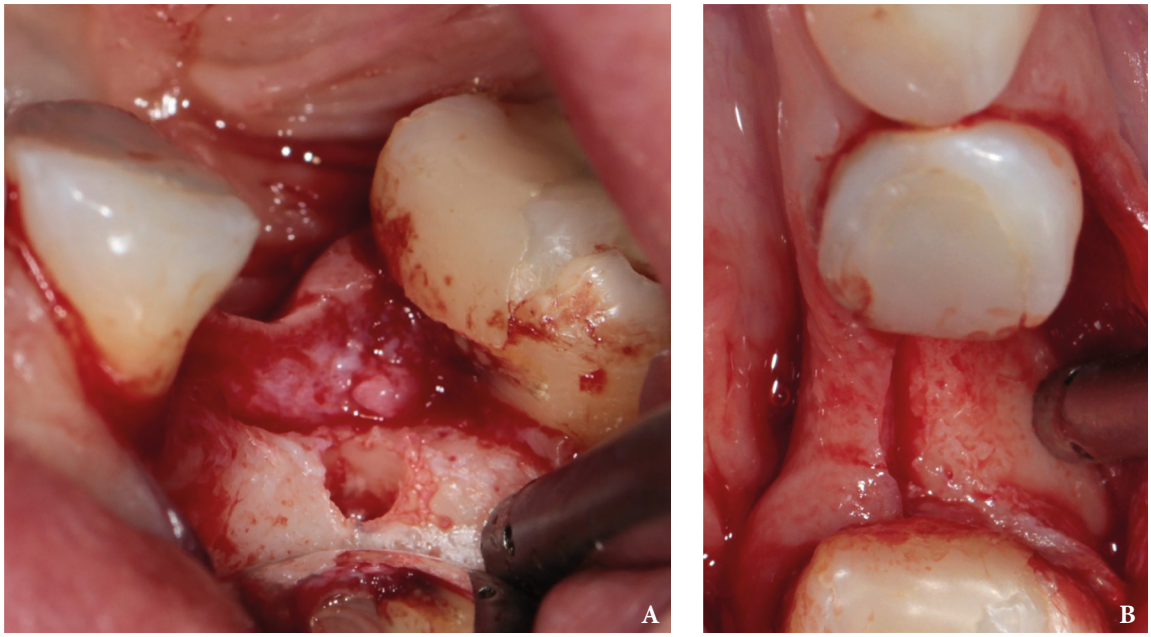

FIGURE 4. Elevation of full-thickness flap exposing the deficient alveolar ridge (**A, B**). Preparation of the surgical site.

Surgical procedures begins with local anesthesia and incision (one crestal and two horizontal). Full-thickness flap to expose the residual bone (Fig 4). Osteotomy starting with a 2.0 mm diameter pilot drill, then protocol preparation for implant site we choose (4.0-10, 4.0-11.5, 4.5-10 AnyOne) (Fig 5A). Implant placement. Osteotomy of the cortical bone. Regenerative material (Laddec; OST Développement, Clermont-Ferrand, France) filled the vestibular bone defect and covered with advanced platelet rich fibrin (APRF) [22] and a ribbed titanium mesh (Fig 5B) is fixing on implant with screw (i-Gen; MegaGen, Seoul, Republic of Korea). APRF was achieved using Choukroun A-PRF Centrifuge System (A-PRF™; Nice, France). The soft tissues were adapted over the membranes with mobilizing the flap, sutured with horizontal mattress and single loop sutures (Nylon 5.0, RE-SORBA Medical GmbH, Germany) (Fig 5C). Postoperative and 1-week recommendations were given.

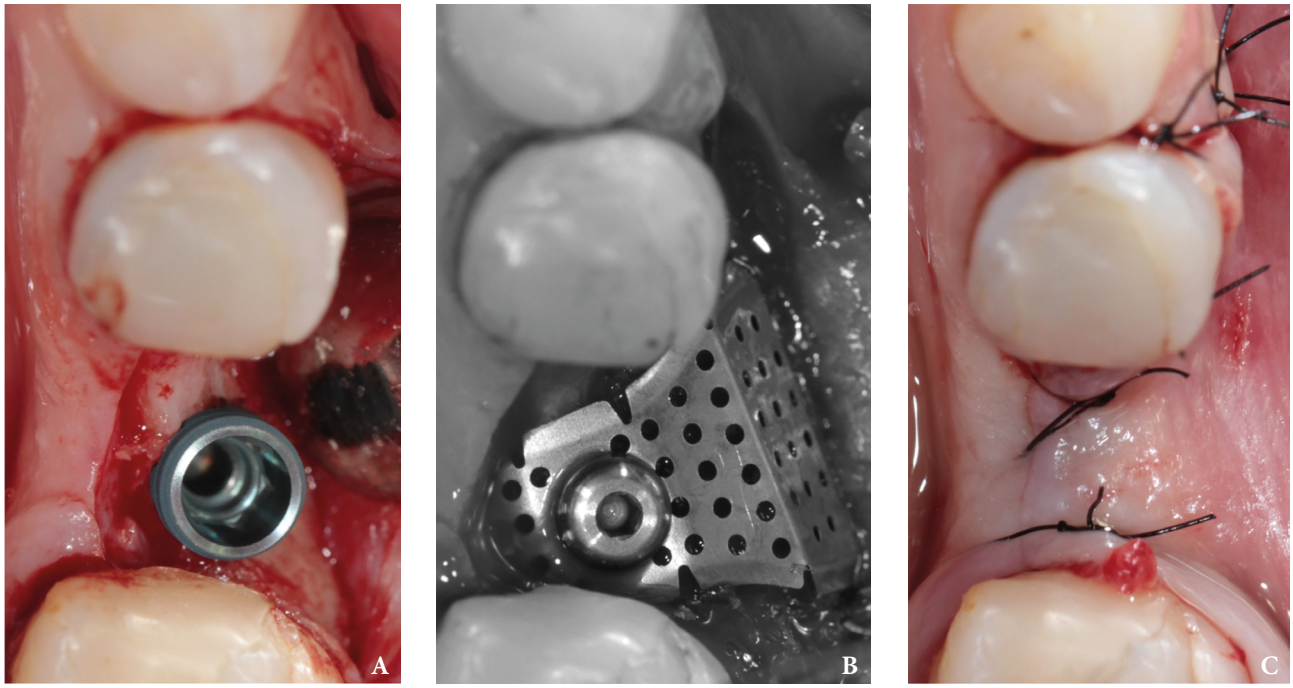

FIGURE 5. Placement the implant (**A**), titanium mesh (**B**), and sutures (**C**). The titanium meshes are connected to the implants and screwed on with the aid of a connecting screw; particulate bone grafts are placed below the titanium mesh screwed on implant.

After 3 months, a second stage surgery was performed at the recipient sites. The fixtures were uncovered, and the titanium screws and meshes were removed; transmucosal healing abutments were positioned and sutures were performed around them. Two weeks later, impressions were taken, and temporary resin restorations (single crowns, screw-retained) were provided (Fig 6). 1-month later we fixed ceram-zirconia screw retained crowns on titanium-bases (Ti-bases) (Fig 7).

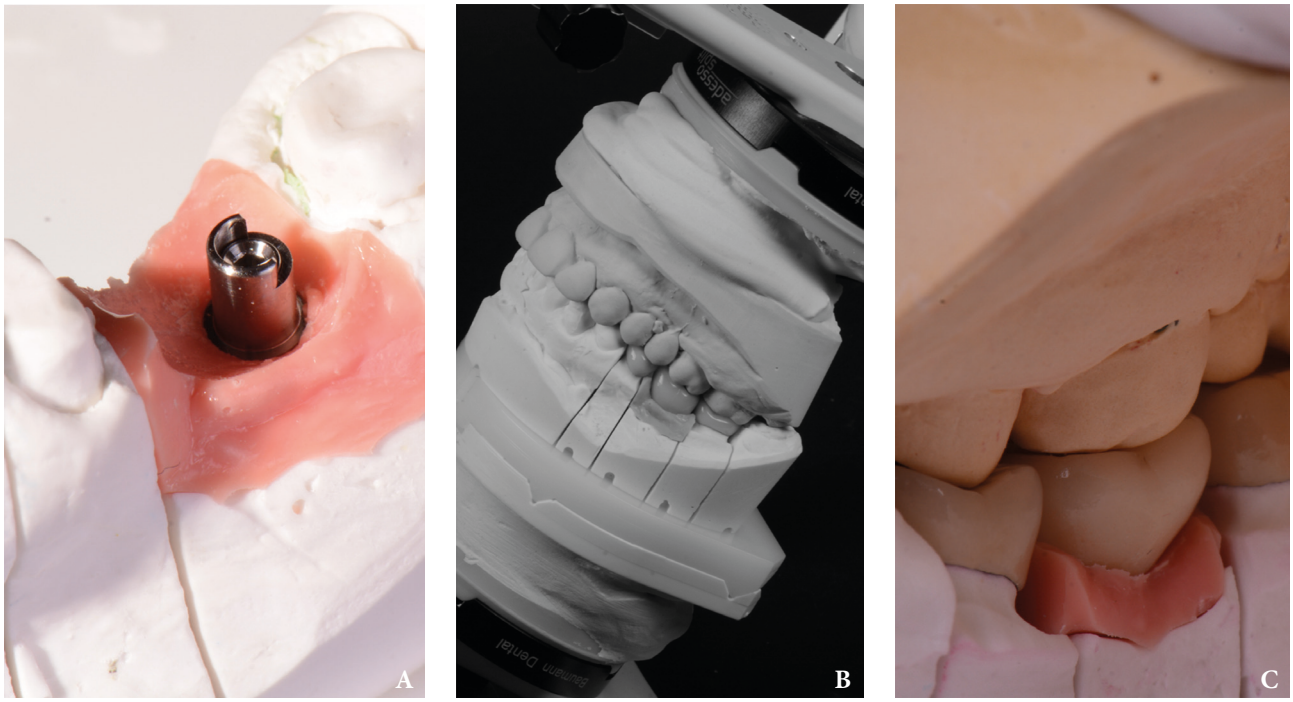

FIGURE 6. Consecutive stages (**A-C**) of the laboratory workflow.

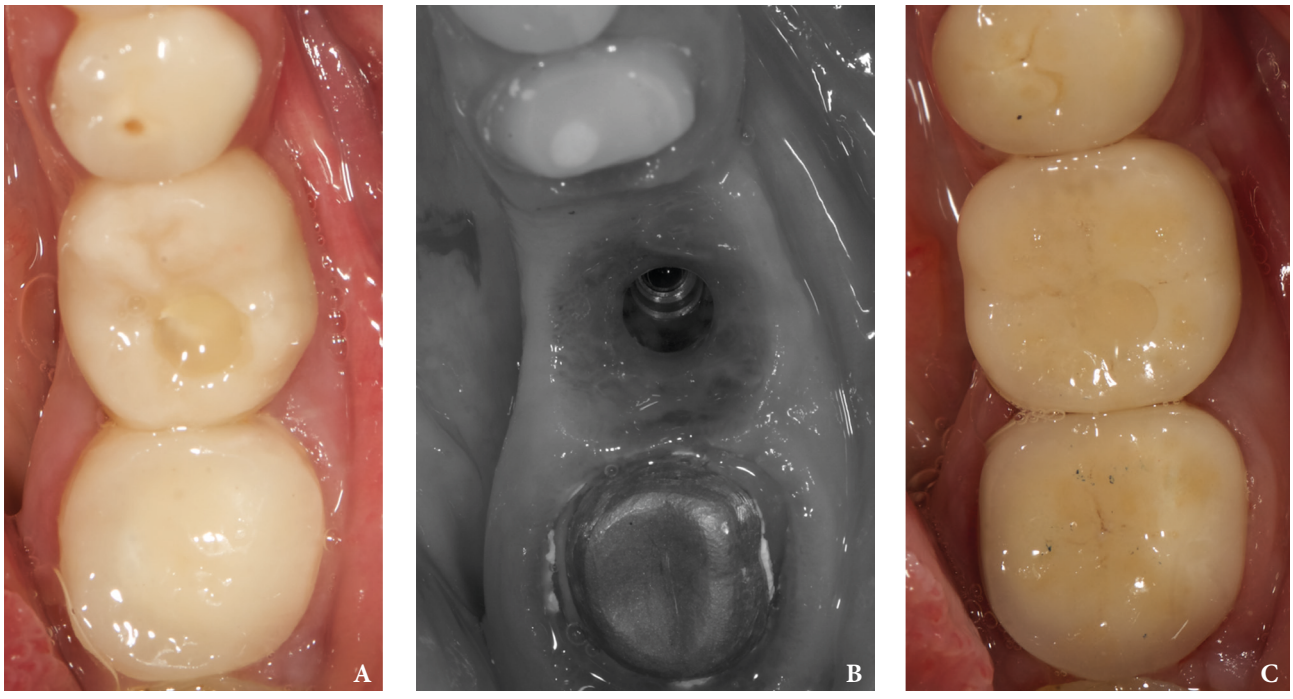

FIGURE 7. View of temporaries (**A**), emergence profile in keratinized gingiva (**B**), and fixed cream-zirconia screw retained crown (**C**).

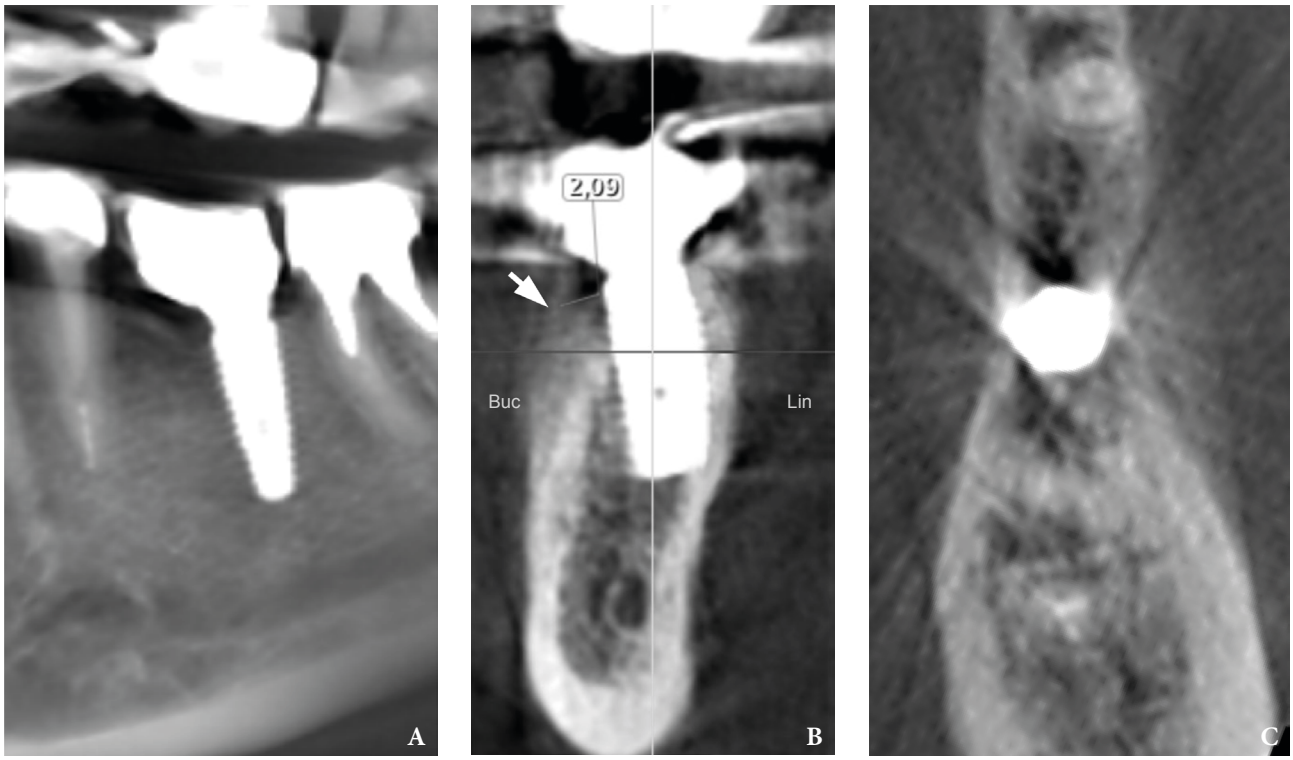

FIGURE 8. CBCT scans (**A**: panoramic view; **B**: coronal scan; **C**: coronal scan) with the highly predictable horizontal modeling of alveolar ridge, 4 months after surgery. (**B**) Buc = buccal side, Lin = lingual side. Noted additional 2.09 mm new bone (*arrow*) at the vestibular side of the implant (at the level of its cervical portion).

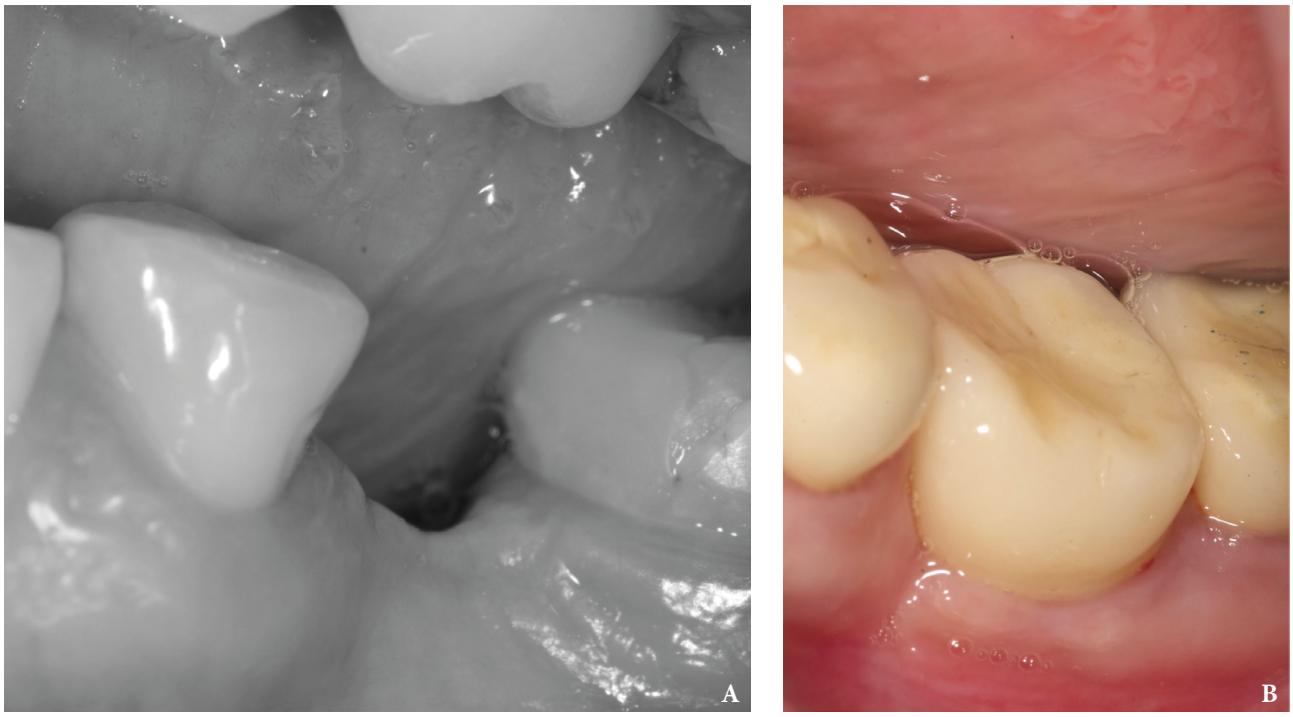

FIGURE 9. The view before (**A**) and after rehabilitation with permanent crown 4.5-month postoperatively (**B**).

PRIMARY OUTCOMES

Early biological complications: early complications were those that occurred immediately after surgery, or in the immediate aftermath (1-2 weeks), such as pain/discomfort, swelling/edema, and extraoral contusion. No one mesh exposure [22] on regenerative stage we fixed using special design form meshes (i-Gen, MegaGen, Korea).

SECONDARY OUTCOMES

The highly predictible horizontal augmentation of alveolar ridge were measured in the CBCT sections (in mm), 4 months after surgery we done (Fig 8). Pre- and postop clinical photographs (Fig 9) clearly demonstrate very precise result.

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Conflict of Interests

The authors declare no conflict of interest.

Role of Author and Co-authors

Oleg I. Mastakov (material collection, concept of the paper and writing)
Bohdan R. Kondratiuk (material collection)
Anna I. An (material collection)
Ievgen I. Fesenko (editing)

Patient consent

Written patient consent was obtained to publish the clinical photographs.

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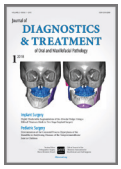

Osteoradionecrosis of the Jaws: A Report of Nineteen Consecutive Cases*

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Radiation-induced oral mucositis (RIOM)
Xerostomia after radiation therapy
Retabolil

ABSTRACT

Purpose.
To study the clinical-radiological symptoms in post-radiological osteonecrosis of the jaws.
Methods.
The survey is based on the clinical study of 19 patients with osteoradionecrosis of the jaws that appeared after the radiation impact on the soft tissues what surrounds jaws, which was performed after the removal of malignant tumors of the soft tissues of the maxillofacial area.
Results.
Based on the examination of patients, clinical and radiological symptoms were studied in cases of osteoradionecrosis of the jaws, described methods of treatment and prevention of this disease.
Conclusions.
In osteoradionecrosis of the jaws there is a significant destruction of bone tissue, which is accompanied by the rejection of sequesters. After X-ray influence on the soft tissues that surrounds jaws, changes in tissues and organs of the oral cavity are observed.

Introduction

Radiation damage to organs and tissues occurs as a result of exposure to ionizing radiation. The use of ionizing radiation in the treatment of malignant tumors of various organs and systems is associated with the risk of post-radiation complications. Osteoradionecrosis (ORN) (synonyms: post-radiation necrosis of the jaws, post-radiation osteomyelitis) of the jaw bones occurs after exposure to ionizing radiation, which can be associated with the treatment of malignant tumors, blood diseases and other organs and systems. ORN develops in 5-10% of patients who received large doses of radiation in the jaw region [1-4].

Material and Methods

This examination is based on the clinical examination and treatment of 19 patients with ORN of the jaws, which appeared after radiation exposure of the soft tissue that surrounds jaws, carried out after the removal of malignant tumors of the soft tissues of the maxillofacial region. We have observed patients and surgical interventions both in the Maxillofacial Surgery Department of Shupyk National Medical Academy of Postgraduate Education and in other surgical departments of Kyiv and other cities of Ukraine.

The purpose of the survey is to study clinical and radiological symptoms in post-radiation osteonecrosis of the jaws.

Results

There are early (acute) and late (chronic) manifestations of radiation exposure. The development of necrotic foci in tissues (skin, soft tissues, and jaws) after radiation depends on the size of the dose, the volume of the irradiated zone, etc. They manifest themselves in skin burns resembling thermal burns and are characterized by the fact that radiation burns of the skin do not develop immediately after exposure, and after a while, i.e. through a latent period. The duration of the latter is shortened with an increase in the dose of ionizing radiation. Late radiation damage most often occurs as a consequence of radiation therapy of malignant tumors.

Many authors believe that the following factors most often contribute to the appearance of ORN of the jaw: the failure of the irradiation technique (exceeding the permissible dose of irradiation), underestimation of concomitant local (presence of carious teeth, chronic periodontitis or complicated forms of periodontitis) or general (diabetes mellitus or another chronic pathology)

* This manuscript has not been presented
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diseases. It should be noted that osteoradionecrosis appears more often in people who abuse alcohol or smokers.

In the radiation zone in the postoperative radiation exposure, in addition to the affected tissues, healthy tissues also enter, including the mucous membrane of the oral cavity and alveolar processes, teeth and jaw bones.

It should be noted that the clinical signs of ORN, teeth and tissues of the oral cavity are quite typical. In all patients in the beginning there is a radiation-induced oral mucositis (RIOM) (synonym: radiomucositis) of mucous membranes of lips, cheeks, and tongue [5]. The clinical picture of radiation damage to the mucous membrane develops gradually. First there is hyperemia and swelling of the mucous membranes, in the future – erosion. The post-radiation reaction has its own peculiarities of manifestation in various parts of the mucous membrane. The first clinical signs on the mucosa, which do not have a keratinized layer in the epithelium, i.e. cheeks, the bottom of the oral cavity and the soft palate, are manifested by slight hyperemia and swelling, which gradually increase. Owing to an intense keratinization, the mucous membrane becomes turbid, loses its luster, thickens, folding appears, and the surface layer is not removed during scraping. Modified areas of the mucosa may resemble leukoplakia or oral lichen planus. As the dose of irradiation increases, the keratinized epithelium is rejected in some areas and erosions appear, covered with a sticky necrotic coating – focal radiation-induced oral mucositis, then the epithelium is rejected in large areas, the erosions merge and the focal radiomucositis is transformed into a large (diffused) radiation-induced oral mucositis. With post-radiation effects in the oral cavity the tropism of the mucous membrane changes, burning, dryness, blanching of the mucous membrane is observed. Often post-radiation stomatitis is developed, as well as the phenomenon of hemorrhagic syndrome, the presence of infection provoke the formation of ulcers and necrosis. Necrosis developing in the oral cavity, is always more intense in the area of adherence to the mucous membrane of metal prostheses and seals, in such places where there are usually accumulations of microbes. There are signs of damage to the edges of the gums and tonsils, followed by the damage of the lateral surfaces of the tongue and palate, increased swelling of the membranes of the mouth, lips and face.

The pathological process that occurs in the mucosa of the oral cavity is complicated by the damage of the salivary glands. Initially, there is increased salivation (within a few days), which quickly gives way to dry mouth before complete xerostomia [6-10] is developed. As a result of the death of taste buds of the tongue there is a taste disorder. Initially, sensations in the tongue can manifest as a glossalgia, then there is a perversion of taste, and later its loss. It is known that radiation changes in the oral cavity are largely reversible. After cessation of irradiation or during a break in treatment, the mucous membrane returns fairly quickly (within 2-3 weeks) to normal. Long-term radiation exposure can lead to irreversible changes in the salivary glands and mucous membrane (edema, hyperemia, telangiectasia, atrophy, radiation ulcers).

Post-radiation pathologic process of the jaws (osteoradionecrosis) is develop in the long term after irradiation and is most often manifested in the form of aseptic necrosis of the bone. Post-radiation damage to jaw bones and teeth can be both isolated and combined with radiation damage to the skin and soft tissues. The causes of osteoradionecrosis are vascular, morphological, degenerative changes in tissues and in the organs of the oral cavity (salivary glands), as well as the immunosuppressive effect of ionizing radiation on tissues. It is believed that the post-radiation osteonecrosis of the lower and upper jaws arises when the oral cavity dryness (xerostomia) affects the teeth. Predisposing factors for infection of the jaw injured by ionizing radiation are untreated dental diseases (periodontitis, etc.) [11-14]. As a result of this infection, post-radiation osteomyelitis develops, characterized by the presence of purulent inflammation in addition to the typical changes in bone structure for radiation injuries.

Most often ORN develops within the first or second year after the end of radiation therapy. In rare cases, they can appear at an earlier time – in a few weeks or at a later date – after 3 years or more. One of the first clinical signs of the development of post-radiation osteonecrosis is the emergence of osteoporosis foci, which can be detected by radiography. Clinical symptoms are often pains localized in the lower and less frequently in the upper jaw. When infected, areas of ulceration or even necrosis of the mucous membranes of the alveolar process may appear. Radiological features (X-ray, CT, MRI): foci of rarefaction (destruction) of bone tissue (osteolytic foci), in some cases it is possible to detect sequesters (Figs 1, 2). Sequesters can be easily detected upon clinical examination of patients (Fig 3).

Discussion

The essence of pathological changes in the bone is a violation (deterioration) of its blood supply and mineral composition. In some cases, post-radiation lesions of the upper and lower jaw can occur in the absence of clinical symptoms, i.e. aseptic necrosis of the bone develops. Therefore, the term "dead-jaw syndrome" is often found in the literature. In some cases, as a result of the presence of chronic odontogenic foci in the patients, infection of pathological foci develops (hyperemia and edema of the alveolar processes, soreness, fistulas with other clinical signs of inflammation) [15-18]. In very sharp cases ORN of the mandible is complicated by abscesses and phlegmons of the soft tissues of the maxillofacial region and neck, as well as sepsis, thrombophlebitis of facial veins and other severe purulent-inflammatory processes.

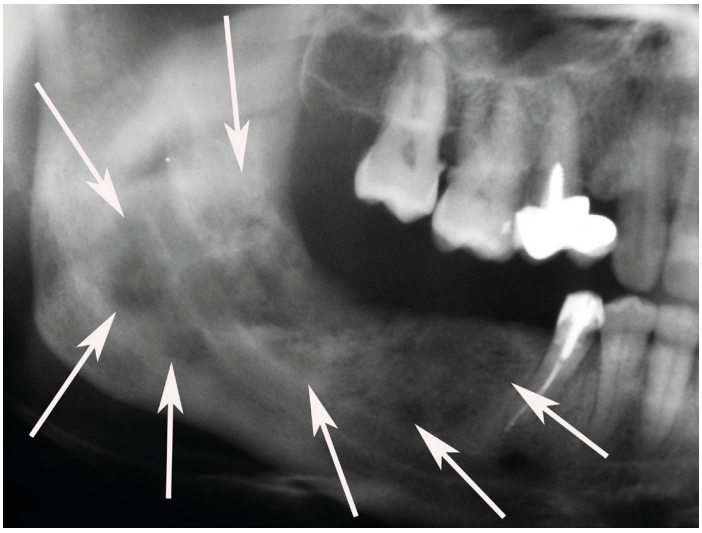
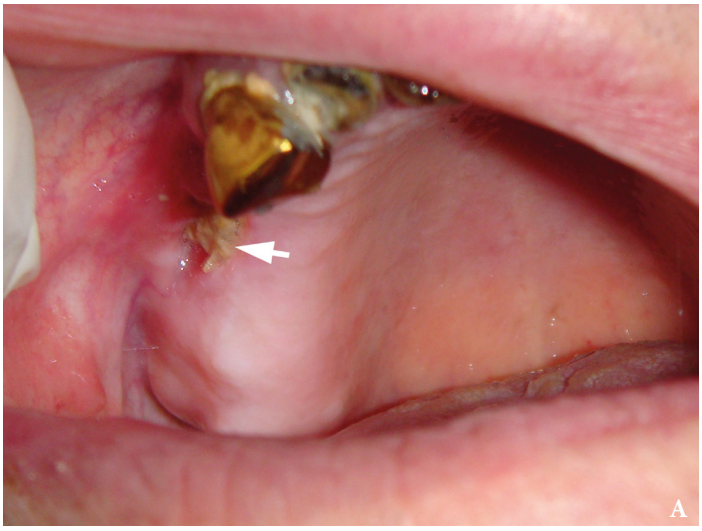


FIGURE 1. Radiograph shows destruction foci at the right mandible (*arrows*) in a 52-year-old gentleman with osteoradionecrosis.



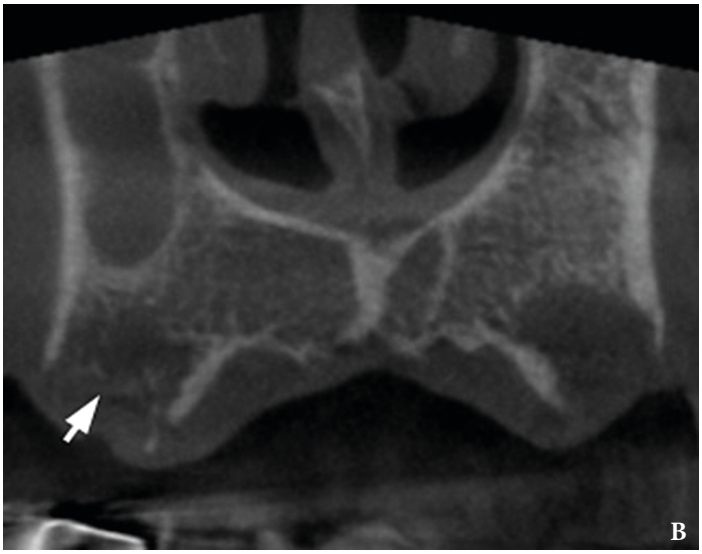


FIGURE 2. Osteoradionecrosis on the right maxilla in a 64-year-old man after radiotherapy. Sequester is indicated by *arrows* (**A**: clinical view; **B**: coronal cone beam CT scan). Images of **Figure 2** are courtesy of Ievgen I. Fesenko, *PhD*, *Assis Prof*; Kyiv, Ukraine.

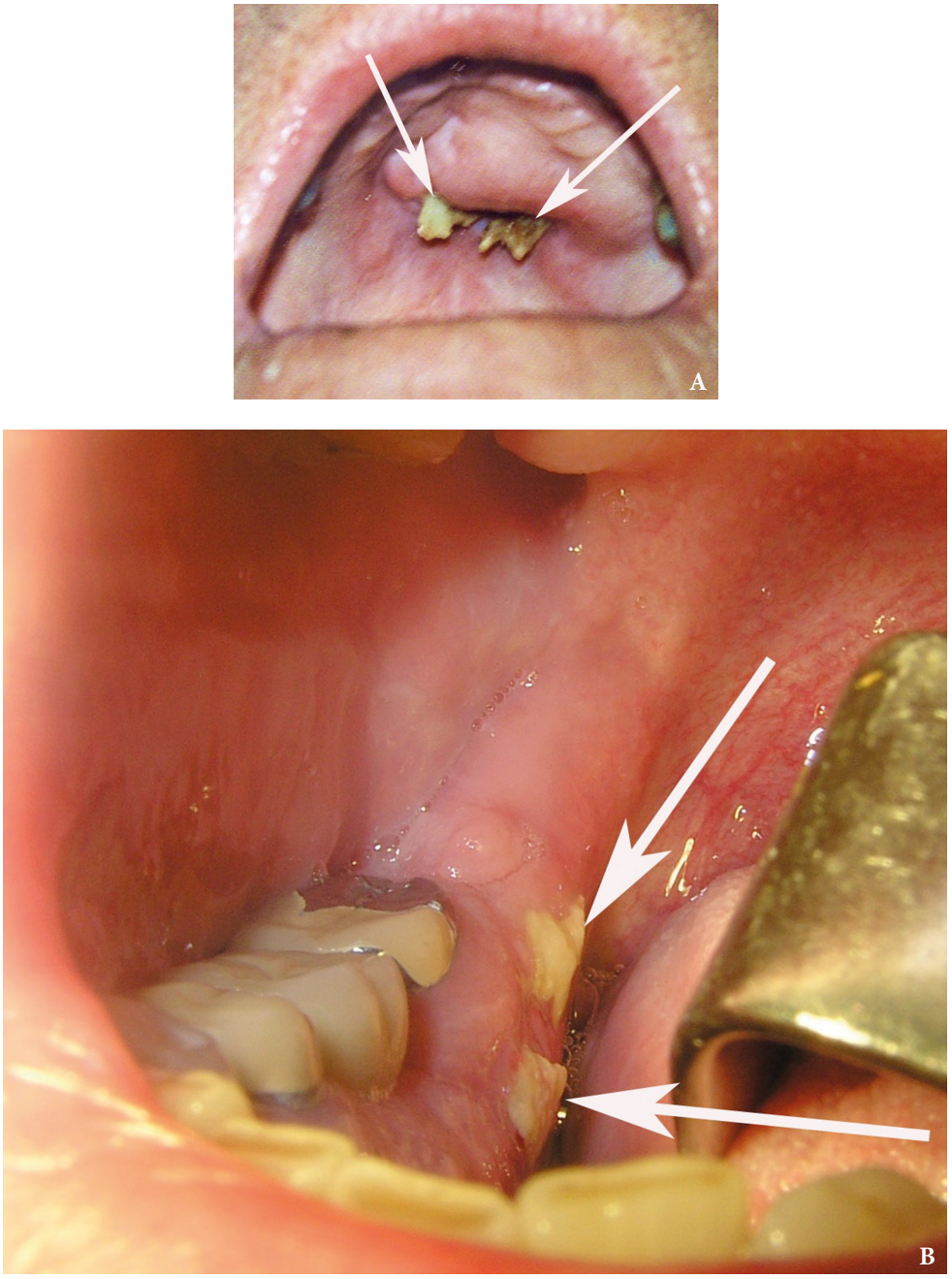


FIGURE 3. Sequestration (*arrows*) on the maxilla (**A**) and mandible (**B**) with osteoradionecrosis in patients of different age groups.

TREATMENT OF OSTEORADIONECROSIS

Treatment of osteoradionecrosis of the jaws in the early stages of its detection can be conservative and consists in prescribing, according to indications, anabolic steroids (Retabolil; Gedeon Rihter, Budepest, Hungary) in combination with calcium preparations. Retabolil enhances protein synthesis in patients with asthenia, cachexia, during radiation therapy, osteoporosis and other pathologies. Under the influence of the active substance, which is part of retabolil, the growth of damaged bone tissue is accelerated [19-22].

Treatment of post-radiation osteomyelitis is always operative and consists of carrying out sequestrectomy (with the removal of existing sequesters) or resection of the involved bone tissue also using microvascular free flaps [23-25]. But the flap surgeries have their own rate of complications [26]. General (antibiotic therapy) and local (antiseptic rinsing of the oral cavity) anti-inflammatory treatment is performed. General treatment of post-radiation lesions should always be combined with therapeutic measures that are aimed at increasing immunological resistance and normalizing the disturbed functions of the body.

There may be difficulties in the treatment of such patients. One of such difficulties in the treatment

of osteonecrosis may be that it is often difficult to determine radiography the difference between the healthy and affected bone tissue which determines the size of the sequestrectomy (necrectomy). Another difficulty is that it can be difficult to differentiate between post-ray bone necrosis and the recurrence of a malignant bone tumor. To solve this problem, the experience of the operating surgeon and carrying out pathomorphological studies of the material to be removed is necessary.

PREVENTION OF OSTEORADIONECROSIS

Prevention of osteoradionecrosis of the jaws consists in the sanitation of the oral cavity before radiotherapy, hygiene of the oral cavity. The teeth should not be extracted during radiotherapy and 2-3 months after [27, 28]. It is necessary to reduce the indirect effect of penetrating radiation by preliminary (preferably before irradiation) a month course of general and local remineralizing therapy in combination with a complex of antioxidants. In such cases, the use of antioxidants in tablets becomes necessary.

For remineralizing therapy, preparations containing calcium, phosphate and other trace elements are used: 10% calcium gluconate solution, 5-10% solution of acidified calcium phosphate, 3% remodent solution (fluoride is not included in its composition), calcium phosphate-containing gels, 5-10% calcium lactate solution, 2.5-10% calcium glycerophosphate solution.

The role of antioxidants is performed by vitamins and minerals contained in various products. The latter activate the human enzyme system. The best antioxidant products are natural products: blueberries, blackberries, grapes, eggplants, beets, grapefruits, persimmons, tomatoes, pumpkin, carrots, etc. Antioxidants must also be taken in the form of medicinal multivitamin preparations: vitrum antioxidant, vitrum-fort Q10, quercetin, selenium-forte, lipin, trofosan, coenzyme Q10, and others.

In the event that preventive measures before irradiation were not carried out, then after radiotherapy it is necessary to conduct the entire course of complex treatment for 5-6 months, combining it with dental interventions (sanitation of the oral cavity).

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Conflict of Interests

The authors declare no conflict of interests.

Role of Authors

The authors are equally contribute to that article.

Ethical Approval

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Patient Consent

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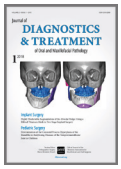

Tooth Root Injury and Orthodontic Microimplant Fracture Caused by Its Incorrect Placement: A Case Report*

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ABOUT ARTICLE

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Mesiobuccal (MB) canal of the tooth
Microimplant fracture
Tooth injury
Supernumerary tooth
Upper central incisor (U1)
Frankfurt horizontal (FH) plane

ABSTRACT

The purpose of this case report was to elucidate the condition after tooth injury while drilling and orthodontic microimplant fracture caused by its incorrect positioning. Among the investigation methods were CT, pulp vitality test, and endo-ice. That case clearly demonstrates and supports opinion of other authors [6-8] that injury of periodontium and tissues of the root while drilling and placement of the microimplants can cause no significant disturbances in the future. Even in case of drilling in close proximity to root canals.

Introduction

Microimplants (*synonyms:* mini-implants, miniscrews, temporary anchorage devices) as skeletal anchorage were implemented into clinical practice by Creekmore and Eklund in 1980s [1]. They used titanium screw below the nasal spine for intermaxillary fixation after orthognathic surgery, and intruded the maxillary incisors. Roberts *et al* [2] used implant fixture in the retromolar area. A canine was connected to the fixture with a bypass wire and used for mesialization of the mandibular molar to the edentulous area [1]. Then the specialists from the East Asia countries started to use widely microimplants and titanium plates as temporary anchorage devices [3]. The era of wide usage of microimplants lead, as any other surgical procedure, to some percentage of complications. According to Alves *et al* (2013) [4] among them: microimplant fracture, ulceration of the mucosa, periimplant mucositis, and damage of the tooth roots adjacent to the microimplant.

Case Presentation

A 26-year-old lady turned to SCIEDECE center seeking for orthodontic treatment, with main complains on crooked teeth, not satisfying smile. After proper investigations (plaster models, orthopantomogram, cephalogram/cephalometric analisys, intra- and extraoral phototography) she was diagnosed skeletal class I, low angle, light crowding on both arches (2.5 mm on upper arch, 4 mm on lower arch), upper incisors protrusion (U1/FH = 117), presence of the impacted supernumerary tooth 2.9. Treatment plan included all third molars extraction, tooth 2.9 removal within 3-6 month follow-up, full unremovable appliance placement, stripping 2 mm on upper arch, 2.5 mm on lower arch, 2 interradicular microimplants placement between upper second premolars and first molars for strong anchorage, while leveling and stripping space utilization. Upon these conditions it was essential to place microimplants maximally distal in the interradicular space as possible, to allow proper leveling. While second microimplant placement, a complication occurred – lower third of the microimplant was fractured. The cone beam computed tomography (CT) was performed. It showed impacted supernumerary tooth 2.9, that moved coronally in comparison to previous location, tip of the fractured microimplant near the mesiobuccal root of tooth 2.7 (Fig 1), and areas of drilling (Fig 2). The tooth 2.7 responded

* This manuscript has not been presented

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to pulp vitality test (Vitality Scanner 2006, SybronEndo, Glendora, California, USA), and wasn`t sensitive to endoice [5, 6]. We removed the fractured tip of the microimplant simultaneously with impacted tooth 2.9 (Fig 1D) under local anaesthesia (1.7 ml Ultracaine D-S forte, Sanofi-Aventis, Frankfurt am Main, Germany). No discoloration, pulpitis, ankylose symptomts were noted during following steps of orthodontic treatment. After 14 months of orthodontic treatment, the treatment was completed and 18-month follow-up showed a successful outcome.

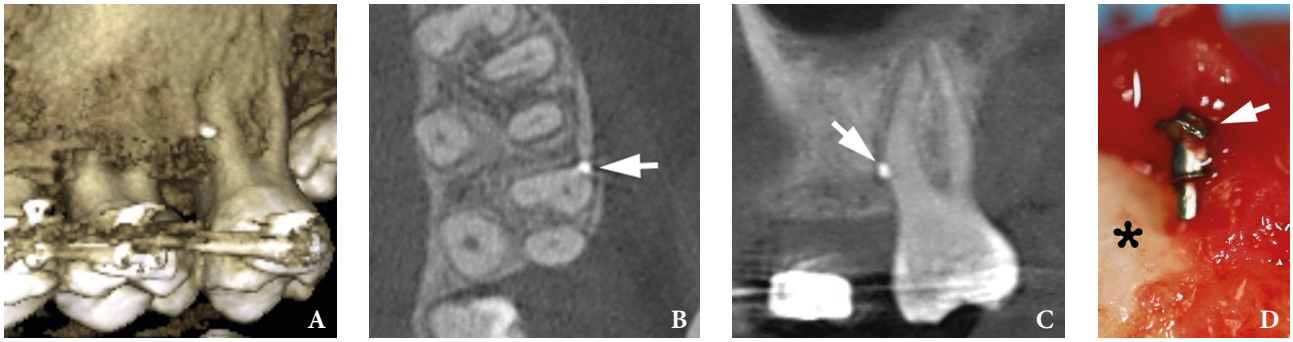

FIGURE 1. Cone beam CT scans (**A**: 3D reconstructed; **B**: axial; **C**: panoramic) shows fractured tip of microimplant (*arrows*) and its incorrect positioning into the tooth ligament. (**D**) Tip (*arrow*) of the fractured microimplant after removal (magnification, × 10) simultaneously with supernumerary tooth 2.9 (*asterisk*: crown of the removed tooth 2.9).

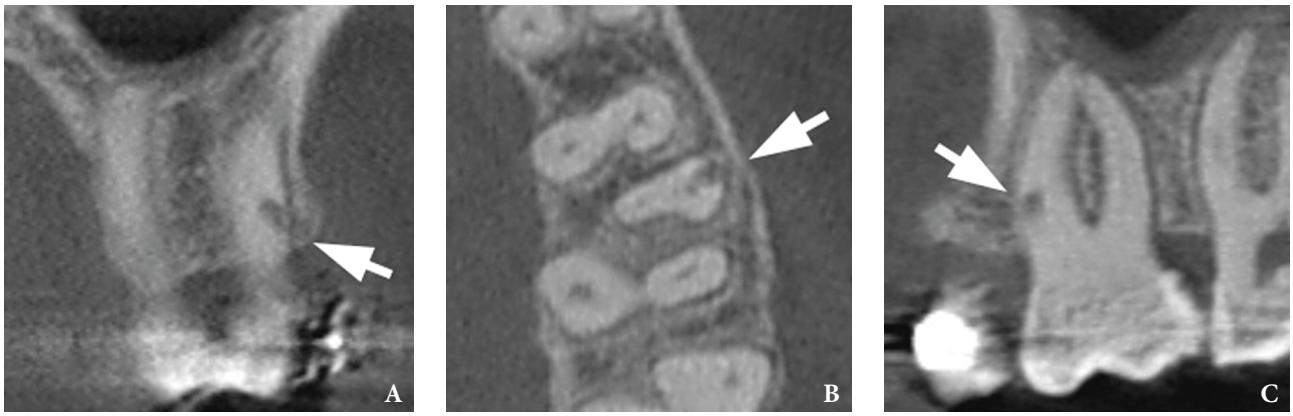

FIGURE 2. CT scans (**A**: coronal; **B**: axial; **C**: panoramic) shows area of drilling (*arrow*) causing the damage to the hard and soft tissues of the tooth root. Note proximity of the insertion hole to the first mesiobuccal (MB1) canal of the tooth 2.7.

Discussion

Orthodontic microimplants are frequently placed interradiucularly, so there is a risk of injury to the roots of the teeth. That can be a possible cause of pulpitis/periodontitis in some cases. However, iatrogenic root trauma is a rather rare complication. Animal studies have proved complete healing of insignificant damage to root tissue following implant removal, resulting in a normal periodontal structure [6-8]. In contrast, heavily injured tissue did not heal completely [7], but left a bony ankylosed area on the root surface, which can have a negative impact on orthodontic tooth movement. The defect is usually delayed by secondary cement [9, 10]. And histological examination of the roots in study of Asscherickx *et al* (2005) [10] demonstrated an almost complete repair of the periodontal structure (e.g. cementum, periodontal ligament and bone) in a period of 12 weeks, following removal of the microimplants. Few authors point out the significant difference in primary failure rate on the left side (9.29%) vs the right (5.12%) that reflects the technical sensitivity of the procedure for operator [11].

Conclusion

Our case clearly demonstrates and supports opinion of authors [6-8] that periodontium and root injury upon drilling and placement of the microimplants can cause no significant disturbances in the future. Even in case of drilling in close proximity to root canals.

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None.

Conflict of Interests

The authors declare no conflict of interests.

Role of Authors

The authors are equally contributed to that article.

Patient Consent

Written patient consent was obtained to publish the CT images.

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Future Events

for 2018-2020

2018

American Cleft Palate-Craniofacial Association 75th Annual Meeting
April 10 – 14, 2018
Pittsburgh, Pennsylvania, USA
www.meeting.acpa-cpf.org

24 Congress of the European Association for Cranio-Maxillo-Facial Surgery
September 18 – 21, 2018
Munich, Germany
www.2018.eacmfscongress.org

Plastic Surgery The Meeting
September 28 – October 2, 2018
Chicago, Illinois, USA
www.plasticsurgery.org

122 American Academy of Otolaryngology Annual Meeting (AAO-HNSF & OTO Expo 2017)
October 7 – 10, 2018
Atlanta, Georgia, USA
www.entannualmeeting.org

100th Annual Meeting of American Association of Oral and Maxillofacial Surgeons Scientific Sessions & Exhibition
October 8 – 13, 2018
Chicago, Illinois, USA
www.aaoms.org

Association of Oral & Maxillofacial Surgeons of India 43rd Annual Conference 2018 (AOMSI 2018)
October 11 – 13, 2018
Chennai, India
www.43chennai.aomsi.com

13th Asian Congress on Oral and Maxillofacial Surgery
November 8 – 11, 2018
Taipei, Taiwan
www.2018acoms.com

2019

24rd International Conference on Oral and Maxillofacial Surgery
May 21 – 24, 2019
Rio de Janeiro, Brazil
www.icoms2019.com.br

18th Meeting of the International Society of Craniofacial Surgery
September 16 – 19, 2019
Paris, France
www.iscfs.org

2020

14th International Facial Nerve Symposium
2020
South Korea
www.internationalfacialnerve.org

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- clinical cases (case reports)
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- reports of new equipment, instruments or technical innovations
- journal or book reviews
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Article and Abstracts

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One co-author is denominated as the corresponding author with all contact details:

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The abstract should include full title of the article, full names and surnames of the co-authors, affiliation, scien¬tific degree, specialty. Also the abstract should include short information about article content: purpose, material and methods, results, conclusions. Example how the Abstract should be looked like the authors can get from the published articles in current issue.

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Photographs, CT and MRI images, sonograms should be submitted in original with resolution of at least 300 dpi and saved in JPEG or TIFF file format.

Fundings

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Conflicts of Interest

At the end of the article the authors should specify about conflicts of interest (e.g., no conflict of interest).

Role of Co-authors in Writing

After specifying conflicts of interest the role of co-authors in writing of the article (concept and design of the study; material collection, mate¬rial processing, statistical data processing, writing text, editing, etc.) should be designated.

Patient Consent

Written patient consent should be obtained to publish the clinical images of the patients.

Acknowledgments

The authors can acknowledge the persons or institutions which they helped or useful in writing an article.

The Journal is recommended to use that internet source for the articles preparing according to *Vancouver References Style:* http://libguides.murdoch.edu.au/Vancouver/journal

Examples How to Form a Refference List

List all references in numerical order in the text.
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Example for the articles:

Fernandes RP, Quimby A, Salman S. Comprehensive reconstruction of mandibular defects with free fibula flaps and endosseous implants. *J Diagn Treat Oral Maxillofac Pathol* **2017**;1(1):6–10.

Example for the articles with more than three authors:

Neto AMR, Monteiro JL, Borba PM, et al. TMJ´s posterolateral dislocation with tympanic plate fracture – case report. *J Diagn Treat Oral Maxillofac Pathol* **2017**;1:59–64.

Example for the articles from the Journal Supplement:
Hammerle CH, Chen ST, Wilson Jr TG. Consensus statements and recommended clinical procedures regarding the placement of implants in extraction sockets. *Int J Oral Maxillofac Implants* **2004**;19(Suppl):26–8.

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Hammerle CH, Chen ST, Wilson Jr TG. Consensus statements and recommended clinical procedures regarding the placement of implants in extraction sockets. *Int J Oral Maxillofac Implants* **2004**;19:S26–8.

Examples for the book chapters:

Yuen HY, Ahuja AT. Benign clinical conditions in the adjacent neck. In: Sofferman RA, Ahuja AT, editors. Ultrasound of the thyroid and parathyroid glands. Springer, **2012**:229–33.

Example for the books:

Baskin J, Duick D, Levine R. Thyroid ultrasound and ultrasound guided FNA. 2nd ed. New York: Springer;
2008.

Example for the PhD/ScD work (dissertation for candidate/doctor of science):
Borkowski MM. Infant sleep and feeding: a telephone survey of Hispanic Americans. PhD [dissertation]. Mount Pleasant (MI): Central Micihigan University; 2002.

Kopchak AV. Clinico-biological and biomechanical study of methods for surgical treatment of mandibular fractures. ScD [dissertation]. Kyiv: Bogomolets National Medical University; 2014.

Example for references in Cyrillic:

Please indicate the language of writing in square brackets [Ukrainian] or [Russian].
Tymofieiev OO. Manual of maxillofacial and oral surgery [Russian]. 5th ed. Kyiv: Chervona Ruta-Turs; **2012.**
Tymofieiev OO. Diseases of the salivary glands [Ukrainian]. 1st ed. Lviv: VNTL-Klasyka; **2007.**

Examples for the internet links:

Seave A. Elsevier CEO using unique data sets and analytic processes to maintain competitive edge. The Forbes. February 25, 2016. Available at: https://www.forbes.com/sites/avaseave/2016/02/25/elsevier-ceo-using-unique-data-sets-and-analytic-processes-to-maintain-competitive-edge/#1d9e4b3979c2/. Accessed February 25, 2016.

Adult improving access to psychological therapies programme. NHS England. Available from URL:

https://www.england.nhs.uk/mental-health/adults/iapt/ (last accessed 3 March **2017**).

McManus S, Meltzer H, Brugha T, et al., editors. Adult psychiatric morbidity in England, 2007: results of a household survey. The NHS Information Centre for health and social care; 2017. Available from URL: http://www.hscic.gov.uk/catalogue/PUB02931/adul-psyc-morb-reshou-sur-eng-2007-rep.pdf (last accessed 3 March **2017**).

Example for conference paper in print proceedings:

Christensen S, Oppacher F. An analysis of Koza's computational effort statistic for genetic programming. In: Foster JA, Lutton E, Miller J, Ryan C, Tettamanzi AG, editors. Genetic programming: EuroGP 2002: Proceedings of the 5th European Conference on Genetic Programming; 2002 Apr 3-5; Kinsdale, Ireland. Berlin: Springer; **2002**. p. 182-91.

Example for conference paper from the internet:

Cloherty SL, Dokos S, Lovell NH. Qualitative support for the gradient model of cardiac pacemaker heterogeneity. In: Proceedings of the 2005 IEEE Engineering in Medicine and Biology 27 Annual Conference; 2005 Sep 1-4; Shanghai, China. New York: IEEE; **2005** [cited 2010 Sep 2]. p. 133-6. Available from: IEEE Xplore.

Example for A-V materials (DVD):

Acland RD, presenter. Acland's DVD atlas of human anatomy [DVD]. Baltimore (MD): Lippincott Williams & Wilkins; **2004**.

Example for A-V materials (YouTube/Vimeo video):

NRK. Medieval helpdesk with English subtitles [video file]. **2007** Feb 26 [cited 2014 Jan 28]. Available from:http://www.youtube.com/watch?v=pQHX-SjgQvQ

Example for A-V materials (Video recording):

Hillel J, writer. Out of sight out of mind: indigenous people's health in Australia [videorecording]. Bendigo: Video Education Australasia; **2003**.

Example for Readers/Study Guides:

Lynch M. God's signature: DNA profiling, the new gold standard in forensic science. Endeavour. 2003;27(2):93-7. Reprinted In: Forensic Investigation (BIO373) unit reader for forensic DNA component. Murdoch (WA): Murdoch University; **2005**.

Example for newspaper articles in print:

Hatch, B. Smoke lingers for those who keep hospitality flowing. Australian Financial Review. **2006** Jul 13: 14.

Example for newspaper article from the internet:

Devlin, H. Neuron breakthrough offers hope on Alzheimer's and Parkinson's. The Times [newspaper on the Internet]. **2010** Jan 28 [cited 2010 Jan 31]. Available from: http://www.timesonline.co.uk/tol/news/science/medicine/article7005401.ece.

Example for conversation citation:

In a conversation with a colleague from the School of Population Health (Jameson LI **2002**, oral communication, 7th August)...

Example for e-mail citation:

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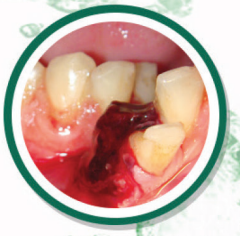
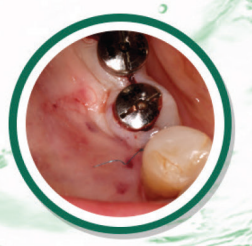
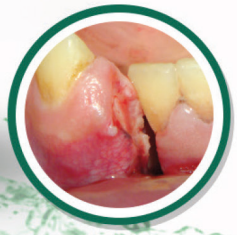
- **WOUNDS OF ORAL CAVITY[5]**







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LOCAL ANESTHETIC AND ANTI-INFLAMMATORY EFFECT[1]

SUMMARY OF PRODUCT CHARACTERISTICS

NAME OF THE MEDICINAL PRODUCT. Tantum Verde 0.15% mouthwash. QUALITATIVE AND QUANTITATIVE COMPOSITION. Each 100 ml contains: active ingredient: benzydamine hydrochloride 0.15 g (equivalent to 0.134 g of benzydamine). **Therapeutic indications.** Treatment of symptoms such as irritation/inflammation including those associated with pain in the oropharyngeal cavity (e.g. gingivitis, stomatitis and pharyngitis), including those resulting from conservative or extractive dental therapy. **Posology and method of administration.** Pour 15 ml of Tantum Verde mouthwash into the measuring cup, 2-3 times per day, using it either at full concentration or diluted. If diluted, add 15 ml of water to the graduated cup. Do not exceed the recommended dosage. **Contraindications.** Hypersensitivity to benzydamine or to any of the excipient. **PHARMACOLOGICAL PROPERTIES. Pharmacodynamic properties.** Pharmacotherapeutic group: Stomatologic drugs: other agents for local oral treatment, ATC code: A01AD02. Clinical studies demonstrate that benzydamine is effective in relieving suffering from localised irritation of the mouth and pharynx. In addition, benzydamine possesses a moderate local anaesthetic effect. **Pharmacokinetic properties.** Absorption. Absorption through the oropharyngeal mucosa is demonstrated by the presence of measurable quantities of benzydamine in human plasma. These levels are insufficient to produce systemic effects. Distribution. When applied locally, benzydamine has been shown to accumulate in inflamed tissues where it reaches effective concentrations because of its capacity to penetrate the epithelial lining.

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2. http://www.angelinipharma.com/wps/wcm/connect/com/home/Angelini+Pharma+in+the+world/
3. Тимофеев А.А. и др. "Особенности гигиены полости рта для профилактики воспалительных осложнений при переломах нижней челюсти". Современная стоматология 2015;1(75):52–8.
4. 4,5. Tymofieiev O.O. et al"Prevention of inflammatory complications upon surgeries in maxillofacial region". J Diagn Treat Oral Maxillofac Pathol. 2017;1:105–12.

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