

Mandibular Crowding - A risk factor in the Initiation and Progression of Gingival Inflammation

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ABSTRACT

This article deals with the impact of mandibular frontal crowding on the initiation and progression of gingival inflammation. For the purposes of this study, sixty patients were included, of which thirty with normal occlusion and another thirty with mandibular frontal crowding of their teeth were examined. In all patients, the accumulation, retention and elimination of the dental plaque was analyzed, whereas its impact on the integrity of the gingival tissue was followed by clinical observation of the gingival tissue and the recorded changes were shown through IGI and IGK. The respondents were observed several times, i.e. upon their first visit and then in 15, 30 and 45 days from their first check, i.e. after the elimination of the dental plaque and other overlays from their teeth as well as the elimination of the gingival inflammation.

The acquired results from the examinations showed that there is a significant difference in the accumulation and retention of the dental plaque between the two examined groups, namely between the group of patients with normal occlusion and those with mandibular frontal crowding (p-0000***). On the 15th day of therapeutic treatment, upon the examination, the presence of dental plaque was still noticeable, in both groups, as a result of the inadequate maintenance of oral hygiene. The difference from the acquired values of the amount of the dental plaque from both examined groups is significant (p-005). The same was noticed even after the 30th day of the treatment; however, after the 45th day, the significance of the difference in acquired results was (p-0000***). Significant differences were noticed between the values obtained from both examined groups in terms of the index of gingival inflammation and the index of gingival bleeding (p-0002***). The difference from the obtained values for IGI and IGK after the 15th day are insignificant; the difference from the obtained values for IGI and IGK after the 30th day was p-005 whereas after the 45th day p-0002***. These results point to the fact that mandibular crowding of frontal teeth represents a significant factor for the accumulation, retention and elimination of the dental biofilm and has indirect impact on the initiation and progression of gingival inflammation, namely the periodontal illness.

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INTRODUCTION

The growth and development of the craniofacial system happens under the influence of genetic, internal and external factors, as well as post-natal conditions of the environment seen from the physiological and pathological aspect. If, during growth, some parts of the face develop faster or slower than others, or if compensational mechanisms fail, then disharmony is established, leading later on to malocclusion. It actually represents a deviation from what is considered normal occlusion where upon the disruption of the homeostasis in all positions occurs, i.e. the disruption of the morpho-functional integrity of the entire masticatory organ [1].

Mandibular crowding of the lower incisors is a relevant topic for research, as an indirect risk factor in the etio-pathogenesis of gingival inflammation. Teeth crowding represents one of the most significant forms of malocclusion, and its representation according to Geiger [2] and Sutcliffe [3] varies between 40-58%. Research carried out by Serafimova and co. [4] in 2,092 children within the territory of the city of Skopje aged between 3 and 14, showed crowding in 146 children (or 32%). On the other hand, epidemiological studies carried out by Bajraktarova-Gorchuloska [5] in the territory of the Republic of Macedonia in 3,850 children aged between 6 and 15, in both frontal segments was 57.55%, of which only in the maxillary frontal segment it was 23.86%, whereas in the mandibular frontal segment - 18.59%.

The regular position and shape of the teeth represent an important factor in maintaining the functional integrity of the periodontal complex. A lot of research point at the significant relation between certain teeth malpositions and the situation of the periodontal-tissue complex. In this group of exceptions, which actually represent a risk factor of the initiation and progression of gingival inflammation, namely periodontal illness, the following are included: teeth crowding, rotated teeth, inclined teeth, deep overlay and open bite [6,7,8,9]. However, the presence of orthodontic anomalies only indirectly influences the initiation of gingival inflammation or periodontal illness, and that is through difficult elimination of the primary and dominant etiological factor - the dental plaque. However, data published in resources and references on the impact of orthodontic anomalies and gingival inflammation are contradicting and irreconcilable. Results obtained by Hellgren [7], Buckley [10] and El-Mangouro [11] point

at the impact of orthodontic anomalies on the periodontal health and well-being; On the other hand, Gould [12] and Katz [13] negate this kind of activity or influence.

The aim of the article is to observe the impact of lower frontal teeth crowding on the initiation and progression of gingival inflammation, namely the periodontal illness.

MATERIALS AND METHODS

The research was carried out in 30 respondents with Class 1 crowding of frontal teeth, based on the Angle classification [14], and 30 respondents with normal occlusion, according to Graber [15]. Examined patients were all males, aged between 18 and 24, who had not been orthodontically treated before. The group of respondents with lower frontal crowded teeth included all those who belonged to Class 1 malocclusion according to Angle, and were then divided in five groups according to the level of crowding of lower frontal teeth, expressed in millimeters.

- Group 1 - No crowding - Examination
- Group 2 - 7 mm crowding
- Group 3 - 6 mm crowding
- Group 4 - 5 mm crowding
- Group 5 - 4 mm crowding

An imprint from every respondent was taken and then a model was made which helped us determine the level of crowding of frontal teeth of the mandible. The presence of the dental plaque was verified through clinical examinations along with the clinical situation of the gingiva and they were expressed through their respective indexes (IGI and IGK).

The presence of dental plaque and the clinical situation of the gingiva were noticed at the first visit paid to the patients in question. A therapeutic treatment for eliminating the dental plaque, tartar and other local factors with the aim of healing the inflammatory process of the gingiva was carried out in every patient.

After recovery of the gingival inflammation, every patient was given guidelines for further maintenance of their oral hygiene.

Examinations were carried out after 15, 30 and 45 days from the initial therapeutic treatment.

The dental plaque in examined groups, as a dominant factor in the etio-pathogenesis of gingival inflammation was clinically visualized and expressed through the plaque index (PI)-Loe-Silness, whereas the gingival changes were presented through the gingival index (GI) and bleeding index (BI).

PLAQUE INDEX (PI)-LOE-SILNESS

The amount of the dental plaque was examined with a dentistry probe on all four sides of the tooth (buccal-distal, buccal, buccal-mesial and oral side) and was quantified as follows:-

- 0- There is no dental plaque .
- 1- There is a thin layer of dental plaque around the marginal edge of the gingiva..
- 2- There is moderate amount of dental plaque, which occupies more than 1/3 of the tooth surface, but it is also present in the gingival sulcus and periodontal pocket.
- 3 - Increased amount of dental plaque across the whole surface of the tooth.

The gingival inflammation was noticed and expressed through the

Loe&Silness index of gingival inflammation (IGI), which is interpreted as follows:

- 0 = Normal gingiva.
- 1 = Mild inflammation -- slight change in color, slight edema. No bleeding on probing.
- 2 = Moderate inflammation -- redness, edema and glazing. Bleeding on probing.
- 3 = Severe inflammation -- marked redness and edema. Ulceration. Tendency to spontaneous bleeding.

Each of the four gingival areas of the tooth is given a score from 0 to 3; this is the GI for the area. The scores from the four areas of the tooth may be added and divided by four to give the GI for the tooth; the scores for the individual teeth may be grouped to designate the GI for the group of the teeth; finally, by adding the indices for the teeth and dividing by the total number of the teeth examined, the GI for the individual is obtained. The index for the subject is thus an average score for the areas examined.

The interpretation of GI values is done by following the below-stated scale:

- 01- 1.1 -- Mild inflammation
- 1.1-2.0 -- Moderate inflammation
- 2.0-3.0 -- Severe inflammation

The assessment of the situation of gingival health was presented based on the index of gingival bleeding (IGB) in the examined groups.

- 0 - Healthy gingival tissues with no bleeding after probing.
- 1 - Bleeding on probing, plaque present, but no calculus or defective restoration margins, pockets.
- 2 - Bleeding on probing, calculus detected or defective restoration margins but pockets.
- 3 - Pocket within the color-coded area, i.e. pocket.
- 4 - Color-coded area disappears, indicating pocket.

The obtained results were statistically processed and expressed through the "t" test.

RESULTS

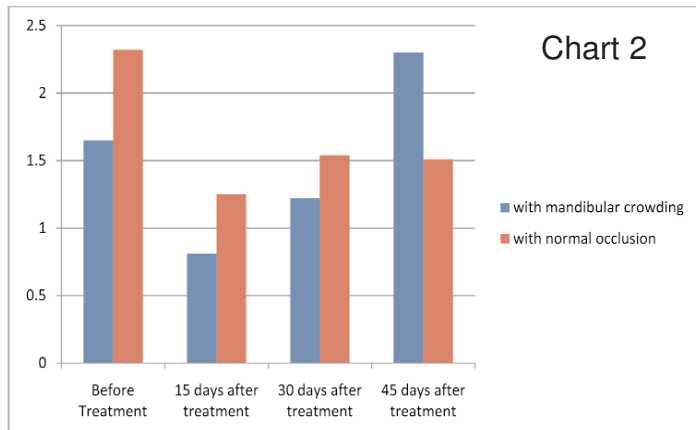
We can see from the Chart 1 that from the total number of examined patients, thirty had normal occlusion, without mandibular frontal crowding, representing the controlling group of respon-

Chart 1. Distribution of respondents based on the crowding level

Number of respondents	Crowding / mm
30	0
10	7
9	6
8	5
3	4
Total	60

dents. Ten respondents from this group had mandibular frontal crowding of 7mm, nine with 6mm, eight with 5mm and three with 4mm.

In Chart 2 are given the mean values and significance of differences between the two examined groups, i.e. the group of respondents with normal occlusion and those with frontal mandibular crowding, before the treatment and 15, 30 and 45 days



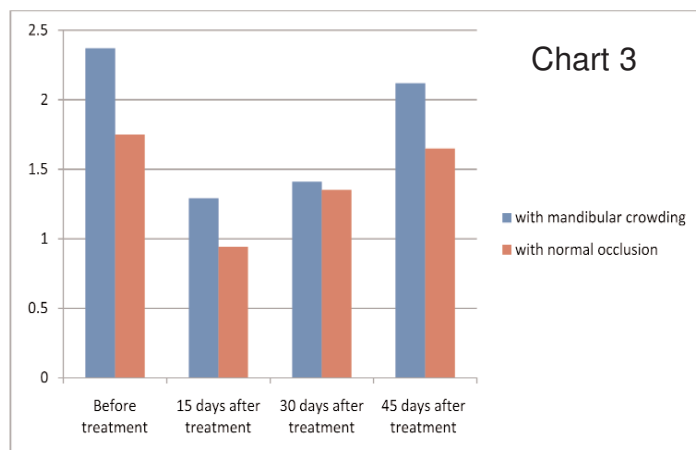
after the therapeutic treatment, meaning after the elimination of the dental plaque and the recovery of the gingival inflammation.

We can see from this chart that there is significant difference between the values obtained in relation to the presence of the dental plaque in patients with normal occlusion and those with frontal mandibular crowding upon the first visit, i.e. before the beginning of the therapeutic treatment (p=0000***).

The mean value of the dental plaque in respondents with normal occlusion before the therapeutic treatment was 1.65, whereas in those with frontal mandibular crowding - 2.32. 15 days after the first therapeutic treatment, the mean value of the dental plaque in respondents with normal occlusion before the therapeutic treatment was 0.81, whereas in those with frontal mandibular crowding - 1.25.

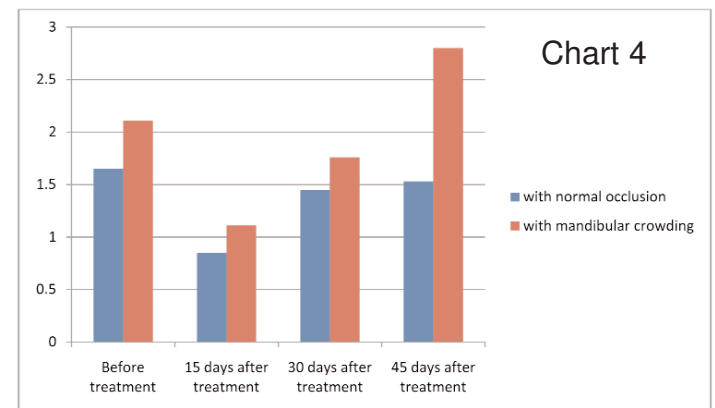
This difference between the two examined groups is not statistically significant (p<005). 30 days after the therapeutic treatment, the mean value of the dental plaque in respondents with normal occlusion before the therapeutic treatment was 1.22, whereas in those with frontal mandibular crowding - 1.54 (p=005). The difference between the two examined groups is even more statistically significant after the 45th day of the therapeutic treatment (p=0002***).

Chart 3 shows the mean values of the IGI in examined groups, i.e. the group of respondents with normal occlusion and those with frontal mandibular crowding, before the treatment and 15, 30 and 45 days after the therapeutic treatment, meaning after the elimination of the dental plaque and the recovery of the gingival inflammation. The IGI mean value in respondents with normal occlusion before the therapeutic treatment was 1.75, whereas in



those with frontal mandibular crowding - 2.37. The difference in values between the two examined groups is significant (p=0002***). After the provided therapeutic treatment and the recovery of the inflammatory condition of the gingiva, on the 15th day the inflammation occurred again, and the IGI mean value in respondents with normal occlusion was 0.94, whereas in those with frontal mandibular crowding - 1.29. The difference in values between these two groups in this case is not statistically significant. However, this difference is significant and increases after the 30th day of the treatment, whereupon p=0.005, whereas after day 45, the difference is significant (p=0002***).

Chart 4 shows the mean values of the IGB in examined groups, i.e. the group of respondents with normal occlusion and those with frontal mandibular crowding, before the treatment and 15, 30 and 45 days after the therapeutic treatment. The IGB mean value in respondents with normal occlusion was 1.65, whereas in those with frontal mandibular crowding - 2.11. The difference in



values between the two examined groups is significant (p=0002***). After the provided therapeutic treatment and the recovery of the inflammatory condition of the gingiva, the IGI mean value in respondents with normal occlusion was 0.85, whereas in those with frontal mandibular crowding - 1.11. The difference in values between these two groups in this case is not statistically significant. However, this difference is significant and increases after the 30th day of the treatment, whereupon the IGI mean value in respondents with normal occlusion was 1.45, whereas in those with frontal mandibular crowding - 1.76. The difference in values between these two groups in this case is statistically significant. After day 45, the IGI mean value in respondents with normal occlusion was 1.53, whereas in those with frontal mandibular crowding - 2.10. The difference in this case is significant (p=0002***).

DISCUSSION

The results obtained from this study confirm that orthodontic anomalies have an indirect influence on the periodontal health and represent a significant local risk factor in the initiation and progression of the gingival inflammation, which is in line with a lot of other data provided by renowned authors in the field such as Fildens [16], Gabris [17] and Glans [18]. Even though the prevalence in the initiation and progression of the gingival inflammation, namely periodontal illness, falls over the microorganisms of the dental biofilm, we should never neglect other gen-

eral local factors, which definitely play a crucial role as additional factors in the accumulation, retention and difficult mechanical removal of the primary etiologic factor, i.e. the dental biofilm, as well as the disordered immune-biological response to periodontal-tissue complex. Our results relating to the accumulation and retention of the dental plaque are in compliance with the above-mentioned references, i.e. there are statistically significant differences in the mean values of the amount of the dental plaque between the two examined groups, with and without teeth crowding. The results obtained after the therapeutic treatment, after the 15th, 30th and 45th day, of the accumulation and retention of the dental plaque, showed repetitive emergence of the plaque, as a result of the inadequate maintenance of the oral hygiene in both examined groups, i.e. in respondents with and without teeth crowding. After the 15th day of the realization of the therapeutic treatment, there was a difference in the mean values of the amount of the dental plaque between the two examined groups, though it was not significant. The difference became significant after the 30th day of the therapeutic treatment and even more evident after the 45th day.

The increased accumulation of the dental plaque in the examined groups is followed by the progression of inflammatory changes of the gingival tissue, as a result of the activity of microorganisms of the dental biofilm. In fact, during the pre-therapeutic treatment of both groups, namely after the very first visit of the respondents, the gingival inflammation presented through the Loe-Silness inflammation index and the bleeding index, showed significant differences in values obtained from the two examined groups. After the therapeutic treatment, inflammatory changes of the gingival tissue were recovered and the respondents were given instructions on how to maintain their oral hygiene in home conditions. After the 15th day of the therapy, a relapse of gingival inflammation happened, which was more emphasized in patients with teeth crowding, compared to those with normal occlusion, even though that difference was not statistically significant after the first 15 days of the treatment. The significance of the difference in values between the two examined groups, determined by respective indexes, was evident after the 30th day, namely the 45th day of the treatment. These examinations are in compliance with those carried out by Bengt [19], Barzam [20], and Pagaca [21], who point out the relation and the role of tooth malposition as an indirect etiologic factor in the initiation and progression of the gingival inflammation or periodontal illness, through the creation of conditions for the accumulation, retention and difficult elimination of the dental plaque from regions occupied by crowded teeth.

CONCLUSION

Based on the realized clinical examinations in the examined groups, we can say that frontal mandibular teeth crowding has an indirect influence on the initiation and progression of the periodontal illness. This kind of indirect impact happens through the emergence of the accumulation, retention and difficult elimination of the dental plaque, as the main etiologic factor in the etio-pathogenesis of this illness. This can also be confirmed by the statistically significant differences in terms of the presence of the dental plaque in respondents with normal occlusion and those

with frontal mandibular crowding. The results obtained from the realized therapeutic treatment after day 15, speak in favor of the difficulty of the elimination of the dental plaque, when it began to accumulate again. The difference in values between the two examined groups began to be evident after the 30th and 45th day of the therapeutic treatment. In addition, we also had the progression of inflammatory changes of the gingival tissue expressed through the index of gingival inflammation and gingival bleeding. Based on all of the above, we come to our final conclusion of this study, which suggests an interdisciplinary approach by orthodontists and periodontists in dealing with orthodontic anomalies and the treatment of the periodontal tissue complex. Special attention should also be paid to the motivation of patients for maintaining good oral hygiene as well as using supplementary items for mechanical removal of the dental biofilm.

REFERENCES

1. Jaksic H, Scepan I, Glisic B. *Ortodonskadijagostika*. Belgrade 2000.
2. Geiger AM. Occlusal studies in 188 consecutively treated cases of periodontal disease. *Am Dent J* 1962;48:330-60.
3. Sutcliffe P. Chronic anterior gingivitis in epidemiological study in schoolchildren. *Brit. Dent. J* 1968;125:47-55.
4. Serafimova S, Gorculoska N, Gorgova J. *Frekvencijata na okluzalno anomalii na teritorijata na Skopje. V Kongresna stomatolozita na SFRJ (zbornik na trudovi)* 1975:663-675.
5. Bajraktarova-Gorculoska N. *Promenina dental nitelakovi i struktura dental nitelakovi kaj slucajso primarnateskoba (habilitacione trud)* 1977. Skopje
6. Boyd R. Longitudinal evaluation of a system for self-monitoring plaque control effectiveness in orthodontic patients. *J Clin Periodontol* 1983;10:380-8.
7. Hellgren B, Serling J. Oral hygiene and gingival inflammation in orthodontic patients. *J Clin Periodontol* 1997;24:2:81-5.
8. Maeda S, Maeda Y, Ono Y, Nakamura K, Matsui T. Interdisciplinary approach and orthodontic orthons for treatment of advanced periodontal disease and malocclusion: a case report. *Quintessence Int* 2007;38;8:653-62.
9. Balazi I. *Ortodonskite anomalii I nivniottretman-mozenrizik faktor na gingivalna ta inflamacija. magisterski trud*, 2012, Skopje.
10. Buckley LA. The relationship between malocclusion and periodontal disease. *J Periodontol* 1972 43:415-7.
11. El-Mangouro NH, Gafaar SG, Mostat YA. Mandibular anterior crowding and periodontal disease. *Angle Orthod* 1987;85:35-8.
12. Gould MSE, Picton OCA. The relationship between irregularities of the teeth and periodontal disease: a pilot study. *Brit Dental J* 1996;121:20-3.
13. Katz RV. An epidemiological study of the relationship between various states of occlusion and the pathological condition conditions. *Dent Res* 1977;3:433-9.
14. Angle EH. Classification of malocclusion. *Den Cosmos* 1998;412-48.
15. Graber TM. *Orthodontics: principles and practice*, second edi-

tion. Philadelphia:WB.Saundersen Company 1972.pp.180-203.

16. Feldens EG,Kramer PF,Feldens CA,Fereirra CH.Distribution of plaque and gingivitis and associated factors in 3 to 5 years old Brazilian children.J Dent Children 2006,73 (1):4-10.
17. Gabris K,Marton S,Madiene M.Prevalence of malocclusion in Hungarian adolescent.Eor J Dent 2006;28 (5):467-70.
18. Glans R,Larsen E,Ogaard B.Longitudinal changes in gingival condition in crowded and non-crowded dentitions subject to fixed orthodontic treatment.Am J OrthodDentofacijalOrthop 2003;124:6:679-82.
19. Bengt I,Javobson U,Sture N.A clinical study od the relationship between crowding of teeth,plaque and gingival condition of ClinPeriodontolog 1974:214-222.
20. BarzamAdulwahah.Low Arch Crowding in Relation To Periodontal Disease;MDJ 2008;5:2:213-225.
21. Estela Santos Gusmao at all.Association between malpositioned teeth and periodontal disease.Dent Press J Orthod 2011;16:4:87-94.

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