



Incidence of gingival hyperplasia among children of 14-17 years of age undergoing orthodontic treatment

¹Aayush Ranjan Deb, ²Lalit Patil, ³Avinash Jaiswal, ⁴Shreya pandey, ⁵Aslam Hisam Qasmi, ⁶Jaspinder kaur,

¹Intern, Sri Ramachandra Faculty of Dental Sciences, Porur, Chennai, Tamil Nadu

²Associate Professor, Department of Pediatric and Preventive Dentistry, Dr. D. Y. Patil Dental College and Hospital, Dr. D. Y. Patil Vidyapeeth, Pune

³Department of orthodontic and dentofacial orthopedic, Senior lecturer, Dental college Azamgarh. avinashortho1586@gmail.com

⁴Department of orthodontic and dentofacial orthopedic, Senior lecturer, Dental college Azamgarh Pshreya256@gmail.com

⁵Department of orthodontic and dentofacial orthopedic, Post graduate student, Dental college Azamgarh, draslambaig@gmail.com

⁶Post graduate student Department of periodontology, B.j.s dental college and hospital Ludhiana

Corresponding author:-

Aayush Ranjan Deb, Intern, Sri Ramachandra Faculty of Dental Sciences Porur, Chennai, Tamil Nadu

Abstract

Background: To study the incidence of gingival hyperplasia in children of 14 to 17 years undergoing orthodontic treatment.

Materials & methods: A total of 120 patients were enrolled. Subjects of the age group 14 to 17 years were selected. All subjects were undergoing fixed orthodontic treatment. They were divided into the categories according to the duration of orthodontic treatment. G0 (control), G1, G2 and G3. Data was collected and result was evaluated using SPSS software. P-value with less than 0.001 was considered significant.

Results: A total of 120 subjects were enrolled. They were divided into groups G0, G1, G2 and G3 as 30 in each groups. In G0, the gingival hyperplasia was absent in almost all the cases. In G1, 24 cases did not showed gingival hyperplasia and in 6 cases it was present. In G2, total of 13 had gingival hyperplasia and in 17 was absent

Conclusion: The duration of orthodontic treatment significantly influenced the occurrence of gingival hyperplasia.

Keywords: orthodontic treatment, gingival hyperplasia, children.

DOI Number: 10.14704/nq.2022.20.11.NQ66199

NeuroQuantology 2022; 20(11): 2043-2046

Introduction

Gingival overgrowth is defined as the excessive overgrowth of the gingival tissue. Gingival overgrowth has several causes, including poor oral hygiene, medications, serious systemic illnesses, hematological disorders, genetic conditions and it even can be idiopathic. ⁽¹⁾ Drug-induced gingival hyperplasia (DIGH) is a periodontal side effects of certain drugs, causing swelling, bleeding, and problems with chewing, aesthetics, and pronunciation. In more severe

cases, it can cause high mobility and detachment of the teeth due to alveolar bone absorption. All of these effects lead to the deterioration of the patient's quality of life (QOL). More than 20 drugs are associated with DIGH, principal among them are immunosuppressants, calcium channel blockers, and anticonvulsants. ⁽²⁾

Periodontal health is an important factor that may be used to evaluate the success of orthodontic therapy. Periodontal complications are reported to be one of the most common side effects linked to



orthodontics. ⁽³⁾ Also, properly aligned teeth are easier to clean, and perhaps correct occlusion may promote healthier periodontium. The periodontal complications associated with orthodontic therapy mainly include gingivitis, periodontitis, gingival recession or hypertrophy, alveolar bone loss, dehiscences, fenestrations, interdental fold, and dark triangles. ^(4,5) Presence of microbial plaque is reported to be the most important factor in the initiation, progression, and recurrence of periodontal disease in reduced periodontium. ⁽⁶⁾

The reasons behind these periodontal complications involve patient factors and the technique used in the treatment. ⁽⁷⁾ Patient factors include past periodontal condition, increased susceptibility, and poor oral hygiene. Hence, this study is conducted to evaluate the incidence of gingival hyperplasia in children of 14 to 17 years undergoing orthodontic treatment.

Materials & methods

A total of 120 patients were enrolled. Subjects of the age group 14 to 17 years were selected. All subjects were undergoing fixed orthodontic treatment. They were divided into the categories according to the duration

of orthodontic treatment. G0 (control), including individuals for corrective orthodontic treatment previously for fixed appliances, G1 composed of patients undergoing fixed orthodontic treatment for 1 year (10-14 months). G2, composed of patients undergoing fixed orthodontic treatment for 2 years (22- 26 months) and G3, composed of patients undergoing fixed orthodontic treatment for 3 years. Clinical examination was done using a dental mirror and periodontal probe. Chi- square test was done. Data was collected and result was evaluated using SPSS software. P-value with less than 0.001 was considered significant.

Results

A total of 120 subjects were enrolled. They were divided into groups G0, G1, G2 and G3 as 30 in each groups. In G0, the gingival hyperplasia was absent in almost all the cases. In G1, 24 cases didnot showed gingival hyperplasia and in 6 cases it was present. In G2, total of 13 had gingival hyperplasia and in 17 was absent. Whereas in G3, 14 cases showed presence of gingival hyperplasia and in 16 was absent. P-value was significant with < 0.001.

Table: Incidence of gingival hyperplasia and duration of orthodontic treatment.

| Duration of orthodontic treatment | Gingival hyperplasia | | P- value |
|-----------------------------------|----------------------|---------|----------|
| | Absent | Present | |
| G0 | 30 | 0 | <0.001* |
| G1 | 24 | 6 | |
| G2 | 17 | 13 | |
| G3 | 16 | 14 | |

* : significant.

Discussion

The most commonly reported adverse effects of orthodontic treatment can be both local and systemic. This includes, tooth discolorations, decalcification, root resorption, periodontal complications, psychological disturbances, gastrointestinal complications, allergic reactions, infective endocarditis, and chronic fatigue syndrome. ⁽⁸⁾

It has been shown that orthodontic forces represent a physical agent capable of inducing an inflammatory reaction in the periodontium. ⁽⁹⁾ In our study, a total of 120 subjects were enrolled. They were divided into groups G0, G1, G2 and G3 as 30 in each groups. In G0, the gingival hyperplasia was absent in almost all the cases. In G1, 24 cases



did not show gingival hyperplasia and in 6 cases it was present.

In one of the studies, the effect of the duration of fixed orthodontic treatment on gingival enlargement (GE) in adolescents and young adults. The sample consisted of 260 subjects (ages, 10-30 years) divided into 4 groups: patients with no fixed orthodontic appliances (G0) and patients undergoing orthodontic treatment for 1 year (G1), 2 years (G2), or 3 years (G3). Clinical examinations were conducted by a calibrated examiner and included the plaque index, the gingival index, and the Seymour index. Poisson regression models were used to assess the association between group and GE. They observed increasing means of plaque, gingivitis, and GH in G0, G1, and G2. No significant differences were observed between G2 and G3. ⁽¹⁰⁾

Another study showed adolescents tend to have higher chances of gingivitis and gingival enlargement (GE) compared to adults. Patients undergoing orthodontic treatment were selected. Participants were divided into three age groups and GH was graded as 0, 1 and 2 as per the classification of the American Academy of Periodontology. Group 1 had 21 patients (39.7%); Group 2 had 24 patients (45.3%) and Group 3 had 8 patients (15.1%). The highest frequency (48%) of GE was observed among the Group 1 age group (10-19 years). Differences in frequency of GH according to age groups were found to be statistically significant ($p=0.046$). Differences in GH according to the frequency of practicing oral hygiene measures were statistically significant ($p<0.001$). Highest frequency of GH was observed among the adolescents. ⁽¹¹⁾ In our study, in G2, total of 13 had gingival hyperplasia and in 17 was absent. Whereas in G3, 14 cases showed presence of gingival hyperplasia and in 16 was absent. P-value was significant with <0.001 .

The mechanism mediating the pathogenesis of medication-triggered connective tissue

responses in the gingiva is still poorly understood. Some hypotheses have suggested the role of factors such as 1) fibroblasts, 2) inflammatory cytokines, and 3) matrix metalloproteinase (MMP) synthesis. ^(12,13) CsA, nifedipine, and phenytoin promote the modeling of periodontal fibroblasts through the synthesis of gingival fibroblasts or inhibition of the decomposition of gingival fibroblasts. ^(14,15) Phenytoin may increase the level of translatable collagen mRNA in human gingival fibroblast, while CsA, nifedipine, and phenytoin enhance the synthesis of collagenous proteins in vitro. ⁽¹⁶⁾

Conclusion

The duration of orthodontic treatment significantly influenced the occurrence of gingival hyperplasia.

References

1. Anil S, Smaranayake LP, Nair RG, Beena VT. Gingival enlargement as a diagnostic indicator in leukaemia. Case report. Aust Dent J. 1996;41:235–237
2. Dongari-Bagtzoglou A. Informational paper: drug-associated gingival enlargement. J Periodontol. 2004;75:1424–1431. doi: 10.1902/jop.2004.75.10.1424
3. Dannan A. An update on periodontic-orthodontic interrelationships. Journal of Indian Society of Periodontology. 2010;14(1):66–71.
4. Bragger U, Lang NP. The significance of bone in periodontal disease. Seminars in Orthodontics. 1996;2(1):31–38.
5. Romero M. Surgical solutions to periodontal complications of orthodontic therapy. Journal of Clinical Pediatric Dentistry. 2000;24(3):159–163.
6. Genco RJ, Borgnakke WS. Risk factors for periodontal disease. Periodontology 2000. 2013;62(1):59–94.
7. Meeran NA. Iatrogenic possibilities of orthodontic treatment and modalities of prevention. Journal of Orthodontic Science. 2013;2(3):73–86



8. Sonwane S, Ganesh P, Kumar BS. Is orthodontic treatment causes bacterial endocarditis? A review based random study. *International Journal of Molecular Medical Science*. 2013;3(2)
9. Tripuwabhrut P, Brudvik P, Fristad I, Rethnam S. Experimental orthodontic tooth movement and extensive root resorption: periodontal and pulpal changes. *European Journal of Oral Sciences*. 2010;118(6):596–603
10. Pinto AS, Alves LS, Zenkner JEDA, Zanatta FB, Maltz M. Gingival enlargement in orthodontic patients: Effect of treatment duration. *Am J Orthod Dentofacial Orthop*. 2017 Oct;152(4):477-482. doi: 10.1016/j.ajodo.2016.10.042. PMID: 28962731.
11. Eid HA, Assiri HA, Kandyala R, Togoo RA, Turakhia VS. Gingival enlargement in different age groups during fixed Orthodontic treatment. *J Int Oral Health*. 2014 Feb;6(1):1-4. Epub 2014 Feb 26. PMID: 24653595; PMCID: PMC3959129.
12. Morton RS, Dongari-Bagtzoglou AI. Regulation of gingival fibroblast interleukin-6 secretion by cyclosporine a. *J Periodontol*. 1999;70:1464–1471. doi: 10.1902/jop.1999.70.12.1464.
13. Bolzani G, Coletta RD, Junior HM, Almeida OP, Graner E. Cyclosporin a inhibits production and activity of matrix metalloproteinases by gingival fibroblasts. *J Periodontal Res*. 2000;35:51–58. doi: 10.1034/j.1600-0765.2000.035001051.x.
14. Deshmukh SN, Dive AM, Moharil R, Munde P. Enigmatic insight into collagen. *J Oral Maxillofac Pathol*. 2016;20:276–283. doi: 10.4103/0973-029X.185932.
15. Williamson MS, Miller EK, Plemons J, Rees T, Iacopino AM. Cyclosporine a upregulates interleukin-6 gene expression in human gingiva: possible mechanism for gingival overgrowth. *J Periodontol*. 1994;65:895–903. doi: 10.1902/jop.1994.65.10.895.
16. Duncan MR, Berman B. Stimulation of collagen and Glycosaminoglycan production in cultured human adult dermal fibroblasts by recombinant human interleukin 6. *J Invest Dermatol*. 1991;97:686–692. doi: 10.1111/1523-1747.ep12483971

