Research Article

Orthodontic Gingival Enlargement: About A Series Of Cases

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Abstract

Introduction

The etiology of gingival growth is very varied. Poor dental hygiene, irritation due to anatomical variations and orthodontic appliances are all factors favoring their appearance.

The clinical implications of orthodontic augmentation are important and can impact long-term stability.

Methodology

A cross-sectional descriptive study was carried out at the periodontology unit of Sidi Bel Abbes, covering 30 patients who consulted for treatment of the gingival enlargements they presented, from 01/09/2024 to 02/03/2025.

The objective of the study was to estimate the frequency of orthodontic overgrowths as well as the factors associated with their appearance.

Results

Our results showed that orthodontic growth represents 43% of gingival overgrowth.

Thus 2/3 of patients are female with almost equal frequency in children (≤15 years) and adults.

In addition, the level of hygiene of patients with these increases is 84% moderate with PI (Silness and Loe) =2.

Conclusion

Orthodontic growth requires special attention in order to improve the aesthetic and functional results of orthodontic treatment on the one hand and preserve periodontal health on the other hand.

Keywords— orthodontic treatment, gingival overgrowth, gingival enlargement, children.

I. INTRODUCTION

Gingival enlargement is a clinical manifestation with a multifactorial etiology. In children and adolescents, its development can be promoted by the accumulation of dental biofilm, inadequate oral hygiene, certain anatomical factors, and the presence of orthodontic appliances. Orthodontic devices, particularly fixed appliances, can promote biofilm retention and inflammatory changes in the gingiva [1,2].

Orthodontic treatment should therefore be considered a contributing or aggravating factor in a multifactorial context rather than a single cause. A recent systematic review concludes that the risk and prevalence of gingival enlargement associated with orthodontic treatment are particularly high in adolescents [1].

II. METHODOLOGY

A descriptive cross-sectional study was carried out at the periodontology unit of Sidi Bel Abbes, on 86 patients who consulted for the management of gingival growth between September 1, 2024 and March 2, 2026. The Excel data provided made it possible to distinguish 26 patients with growth associated with orthodontic treatment (ODF group) and 60 patients without orthodontic etiology.

The objective was to estimate the frequency of gingival enlargement associated with orthodontic treatment in the pediatric/adolescent population and to study its relationship with age, sex, plaque index (PI), gingival index (GI), and periodontal diagnosis. Subjects aged 15 years or younger were considered to belong to the pediatric/adolescent group.

Qualitative variables were compared using Pearson's chi-squared test. The significance level was set at $\alpha = 0.05$. When necessary, Fisher's exact test was also considered.

III. RESULTS

A. Frequency of orthodontic growth according to age

Of the 30 patients aged 15 years or younger, 14 belonged to the ODF group, i.e. 46.7%, while 16 were not associated with orthodontic treatment, representing 53.3%. Among patients over 15 years of age, 12 had orthodontic treatment and 44 did not (Fig. 1).

The association between age group and the presence of an increase associated with ODF is statistically significant: $x2 = 5.899$, $df = 1$, $p = 0.015$. Fisher's exact test confirms this association ($p = 0.026$).

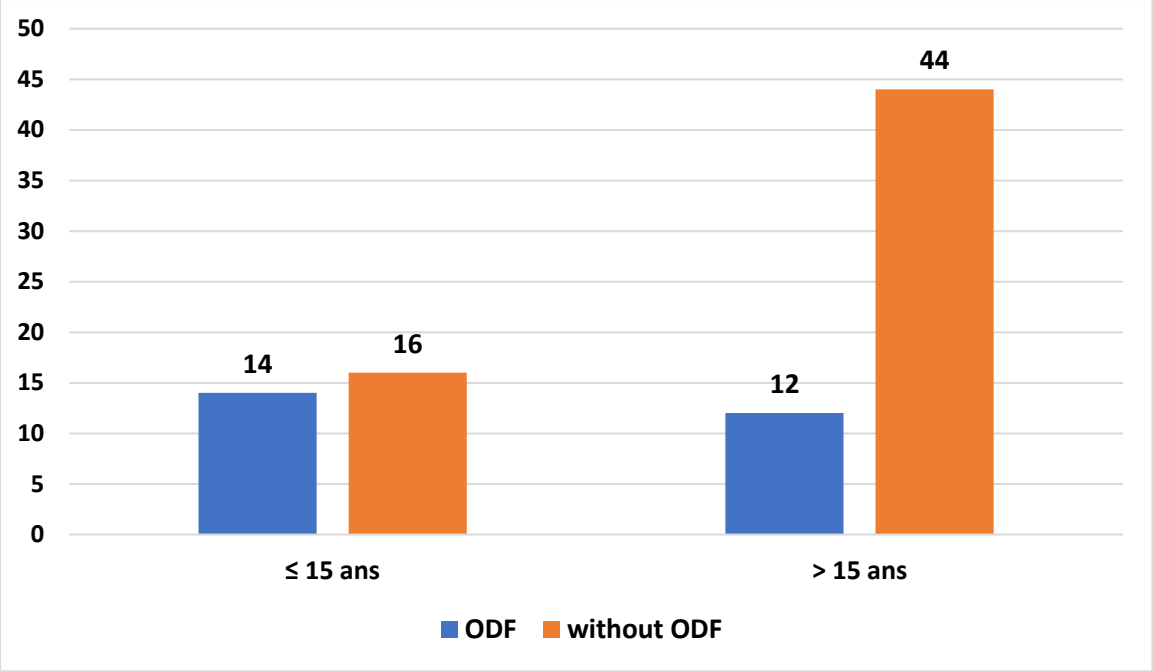

Figure 1: Description of the study population according to the frequency of orthodontic growth.

B. Distribution by sex

The ODF group comprised 19womenand 7 men, compared to 40 women and 20 men in the group without orthodontic treatment. The difference is not statistically significant (x2 = 0.346, df = 1, p = 0.556) (Fig. 2).

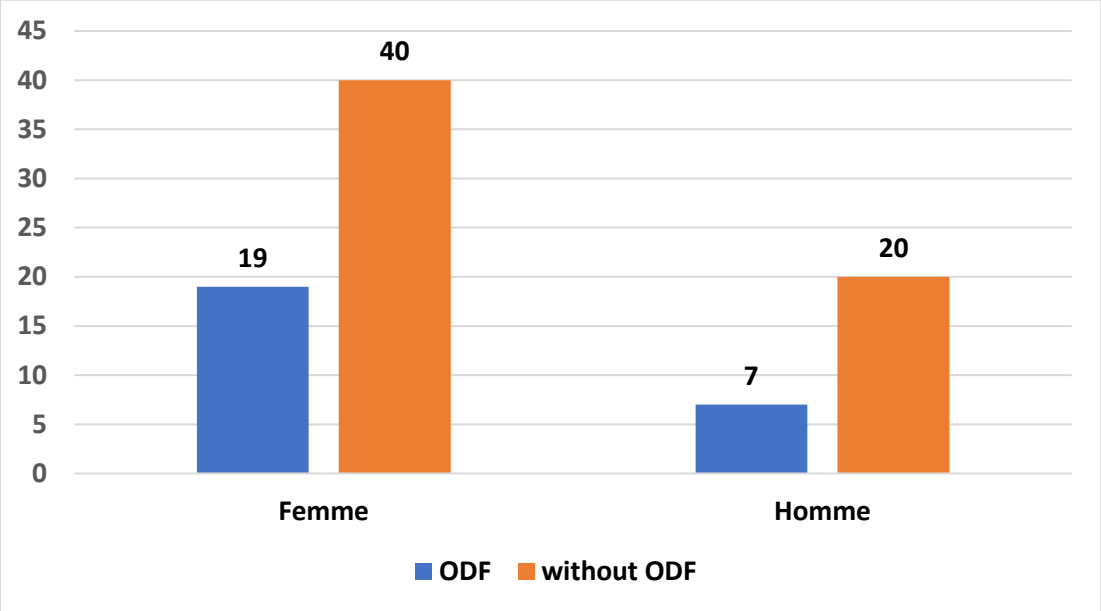

Figure 2: Distribution of orthodontic growth according to sex

C. Distribution according to the plate index (PI)

The PI values in the orthodontic treatment group were: PI=1 in 1 patient, PI=2 in 21 patients, and PI=3 in 4 patients. In the group without orthodontic treatment, they were 2, 45, and 13 patients, respectively. The association was not statistically significant (χ^2 = 0.454, df = 2, p = 0.797) (Fig. 3).

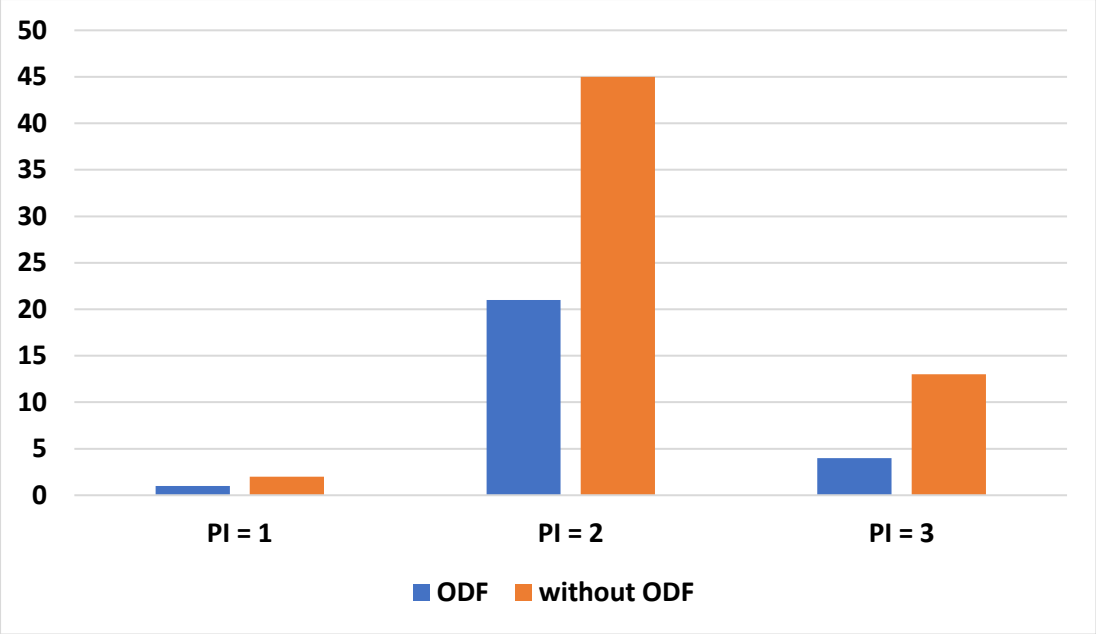

Figure 3: Distribution of orthodontic growth according to PI

D. Distribution according to the gingival index (GI)

In the orthodontic treatment group, GI=1 was observed in 1 patient, GI=2 in 23 patients, and GI=3 in 2 patients. In the group without orthodontic treatment, the numbers were 2, 44, and 14, respectively. The association was not statistically significant ($\chi^2 = 2.932$, df = 2, p = 0.231) (Fig. 4).

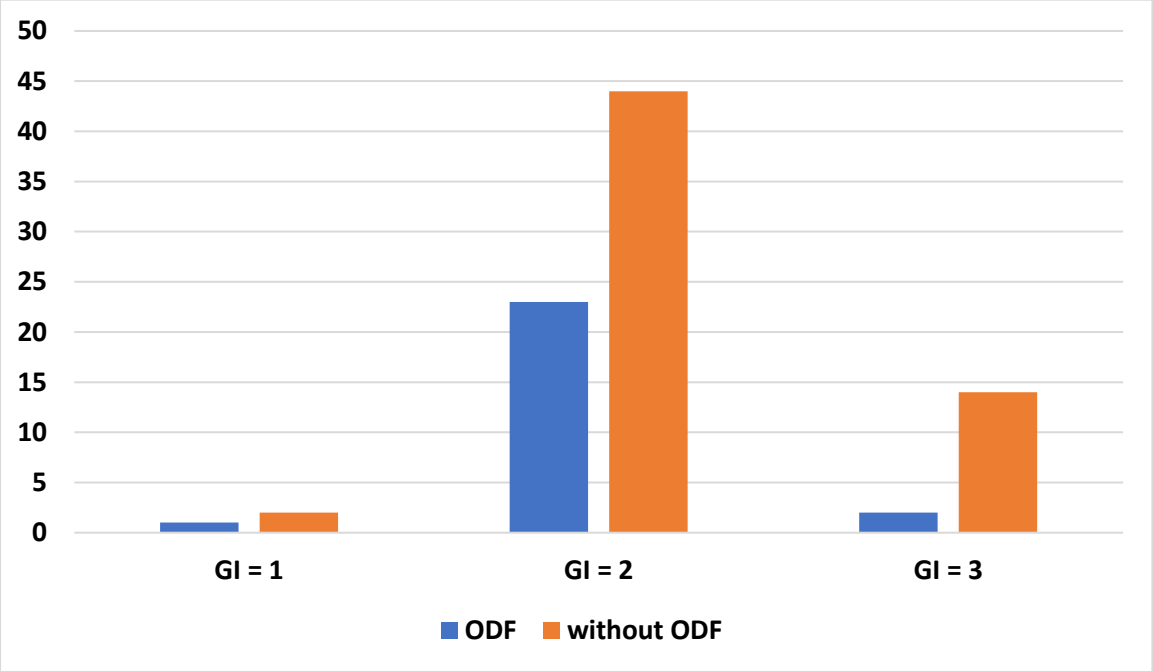

Figure 4: Distribution of orthodontic growth according to the GI.

E: Distribution according to periodontal diagnosis

In the orthodontic treatment group, 15 patients had gingivitis and 11 had periodontitis, representing 57.7% and 42.3% respectively. In the group without orthodontic treatment, 23 patients had gingivitis and 36 had periodontitis.

The comparison of gingivitis versus periodontitis does not show a statistically significant association ($\chi^2 = 2.556$, df = 1, p = 0.110) (Fig. 5).

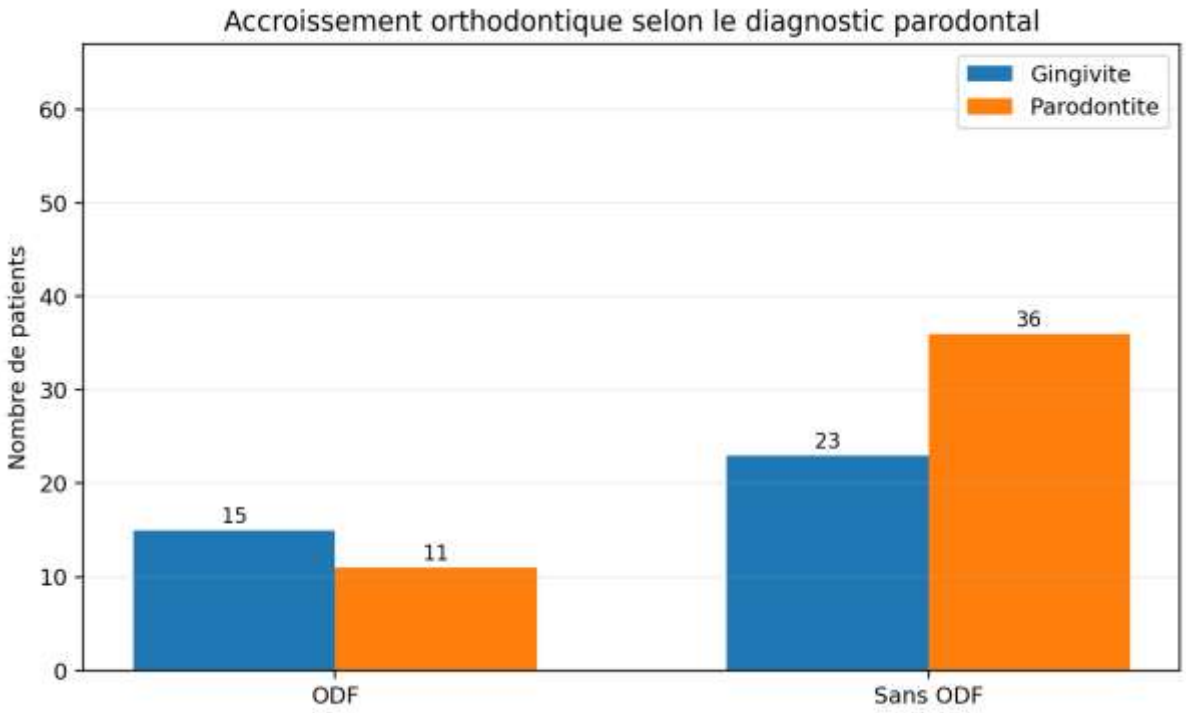

Figure 5: Distribution of orthodontic growth according to periodontal diagnosis

IV. DISCUSSION

the present findings support a multifactorial model of orthodontic-associated gingival enlargement. In our series, age was the only factor demonstrating a statistically significant association, with a higher frequency among patients aged 15 years or younger.

- Analysis of the raw data confirms that the association with age is the main statistically significant finding in this series. In patients aged 15 years or younger, 14 out of 30 (46.7%) belonged to the group with orthodontic-associated gingival growth, compared to 12 out of 56 (21.4%) in subjects over 15 years of age. This difference is consistent with data from recent literature, which report a particularly high risk and prevalence of orthodontic-associated gingival overgrowth in adolescents [1].

The systematic review by Rocha-Eiroa et al. reported heterogeneous sex-related findings and, in the pooled literature, a higher rate in males in several pediatric and adolescent samples [1]. Differences in sample composition, age distribution, orthodontic exposure and diagnostic criteria may explain the discrepancy between our series and published data.

Treatment duration is another potentially important variable that could not be evaluated in our dataset. Pinto et al. demonstrated a progressive increase in gingival enlargement with longer periods of fixed orthodontic treatment [3]. This is relevant to our findings because younger patients may remain under orthodontic care for a considerable period, potentially increasing cumulative exposure to plaque-retentive factors. The lack of treatment-duration data in the present study prevents determination of whether the higher frequency observed in younger patients is partly related to longer or more intensive orthodontic exposure.

Evidence comparing bracket ligation systems is less consistent: a systematic review and meta-analysis found no convincing difference in plaque and gingival indices between conventional and self-ligating brackets, particularly because of limitations and heterogeneity among studies [4]. Thus, prevention should focus less on a single appliance component and more on overall plaque control, appliance design, patient compliance and regular professional monitoring [4,15].

- The etiology appears to be multifactorial. Biofilm retention around brackets and other orthodontic components, local mechanical factors, and changes in the microbial environment can contribute to the onset or worsening of gingival enlargement [1,2,3]. Hormonal variations during puberty can also modulate the gingival response. [5,6].

The factor most consistently identified in the systematic review by Nibali et al. [7]. Inadequate plaque control is the risk factor most consistently observed for gingival enlargement.

In the data from Hussain et al. [8], patients with plaque-induced enlargement exhibited significantly higher plaque and hygiene indices than the controls.

The microbiological dimension is also increasingly recognized. Orthodontic appliances can modify the oral environment by changing available surfaces, plaque maturation and local ecological conditions. A review by Mulimani and Popowics described significant effects of orthodontic appliances on the oral environment and microbiome [9]. Ristic et al. also demonstrated increases in clinical and microbiological parameters after placement of fixed appliances in adolescents [10].

The biological response is not limited to plaque accumulation. Gong et al. reported higher levels of periodontal pathogens and inflammatory mediators, including interleukin-1β, at sites presenting orthodontic treatment-induced gingival enlargement, with clinical and microbiological improvement following periodontal therapy [6]. These findings support the concept that orthodontic-associated enlargement represents an interaction between a local microbial challenge and the host inflammatory response.

Hosadurga et al. investigated the relationship between sex hormones and gingival enlargement in adolescents receiving fixed orthodontic treatment and suggested that hormonal status may influence gingival susceptibility [5]. Consequently, the higher frequency observed in the younger patients in our series may reflect the combined effects of orthodontic appliances, biofilm retention and developmental or hormonal changes rather than orthodontic treatment as an isolated causal factor.

- In our series, no statistically significant association was observed between ODF and sex, PI, or GI. However, these results do not allow us to conclude that biofilm or inflammation do not play a biological role; they only indicate that a statistical association was not demonstrated in this sample.

The 2026 consensus emphasizes that periodontal tissues in children and adolescents require age-appropriate examination and that orthodontic treatment should ideally be initiated in the presence of healthy gingival tissues [11]. The finding of no statistically significant association in our study should therefore be viewed as a characteristic of this dataset rather than as evidence that periodontal inflammation is irrelevant.

The absence of a significant association is particularly interesting when compared with previous research. Pinto et al., in a study of 260 subjects aged 10–30 years, found that longer exposure to fixed orthodontic appliances was associated with increasing plaque, gingivitis and gingival enlargement, and reported substantially higher rates of enlargement among orthodontic patients than among subjects without fixed appliances [3]. Similarly, studies of adolescents have documented increases in plaque and gingival inflammation after appliance placement [9,12]. More recent cross-omics research has also linked orthodontic-associated gingival enlargement with differences in plaque, bleeding on probing, probing depth and the oral microbiome [13]. The difference between these findings and our non-significant PI result may therefore reflect study design, sample size, the timing of clinical examination and the use of a categorical plaque index rather than longitudinal measurements.

- Periodontal assessment before orthodontic treatment, oral hygiene education, and regular monitoring during treatment are therefore essential. The 2026 consensus on gingival and periodontal diseases in children and adolescents emphasizes the importance of periodontal management tailored to age and risk factors [11].

During treatment, oral-hygiene education should be adapted to the appliance and to the patient's age and manual dexterity. The 2026 consensus recommends detailed instruction in brushing around brackets and wires, use of interdental aids and professional plaque removal, with periodontal reviews commonly performed every 3–4 months according to individual risk [11].

Persistent biofilm accumulation may therefore increase the inflammatory challenge to the gingival tissues. Earlier longitudinal studies demonstrated increases in plaque accumulation, gingival inflammation and microbiological changes following placement of fixed appliances [9,10,12]. More recent evidence also indicates transient periodontal deterioration and shifts in the oral microbiome during active orthodontic treatment [9].

A 2025 randomized clinical trial comparing nonsurgical periodontal treatment, conventional gingivectomy and diode-laser gingivectomy in orthodontic patients with anterior gingival enlargement reported improvements in clinical crown length, periodontal pocket depth and gingival-overgrowth measures following treatment [14]. Such interventions should nevertheless be individualized according to the clinical situation.

- Limits
- the orthodontic group comprised only 26 patients, which limits statistical power and increases the possibility of type II error for associations involving sex, PI, GI and periodontal diagnosis.

- the use of categorical PI and GI scores may reduce sensitivity compared with continuous measurements or more comprehensive periodontal parameters.

V. CONCLUSIONS

In this series of 86 patients with gingival enlargement, 26 were receiving associated orthodontic treatment. The frequency associated orthodontic treatment was significantly higher in patients aged 15 years and younger than in older subjects (46.7% versus 21.4%, p=0.015 on the Chi-square test).

No statistically significant association was found with sex (p=0.556), plaque index (p=0.797), gingival index (p=0.231) or gingivitis/periodontitis diagnosis (p=0.110).

These results support the importance of enhanced periodontal monitoring in children and adolescents wearing orthodontic appliances. Prevention should focus on biofilm control, oral hygiene education, and regular assessment of gingival health.

Future research should use larger, preferably multicenter longitudinal cohorts beginning before orthodontic appliance placement and continuing throughout treatment.

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REFERENCES

[1] 1. Rocha-Eiroa MD, Ramírez-Rámiz A, Brunet-Llobet L, Cahuana-Bartra P, Rabassa-Blanco J, Miranda-Rius J. Gingival enlargement induced by orthodontic therapy: a systematic review. Int J Dent. 2026;2026:2172939. doi:10.1155/ijod/2172939.

[2] 2. Syrioti A, Charisi C, Zisis V, Spantidakis N, Fytros F, Poulopoulos K, et al. Gingival enlargement during orthodontic treatment: a narrative review. Cureus. 2026;18(6):e111127. doi:10.7759/cureus.111127.

[3] 3. 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;152(4):477-82. doi:10.1016/j.ajodo.2016.10.042.

[4] 4. Arnold S, et al. The effect of bracket ligation on the periodontal status of adolescents undergoing orthodontic treatment: a systematic review and meta-analysis. J Dent. 2016;54:13-24. doi:10.1016/j.jdent.2016.08.006.

[5] 5. Hosadurga R, Althaf MSN, Hegde S, Rajesh KS, Arun Kumar MS. Influence of sex hormone levels on gingival enlargement in adolescent patients undergoing fixed orthodontic therapy: a pilot study. Contemp Clin Dent. 2016;7(4):506-11. doi:10.4103/0976-237X.194099.

[6] 6. Gong Y, Lu J, Ding X. Clinical, microbiologic, and immunologic factors of orthodontic treatment-induced gingival enlargement. Am J Orthod Dentofacial Orthop. 2011;140(1):58-64. doi:10.1016/j.ajodo.2010.02.033.

[7] 7. Nibali L, Lu EM-C, Paz HES, Kang J, Bostanci N, Belibasakis GN, et al. Risk factors and management of gingival enlargement: a systematic review and meta-analysis. J Clin Periodontol. 2026. doi:10.1111/jcpe.70125.

[8] 8. Hussain ST, Mahendra J, Muralidharan J. Red-complex bacteria in patients with plaque-induced gingival enlargement undergoing fixed orthodontic therapy: a cross-sectional study. World J Dent. 2022;13(4):325-30. doi:10.5005/jp-journals-10015-2062.

[9] 9. Mulimani P, Popowics T. Effect of orthodontic appliances on the oral environment and microbiome. Front Dent Med. 2022;3:924835. doi:10.3389/fdmed.2022.924835.

[10] 10. Ristic M, Vlahovic Svabic M, Sasic M, Zelic O. Clinical and microbiological effects of fixed orthodontic appliances on periodontal tissues in adolescents. Orthod Craniofac Res. 2007;10(4):187-95. doi:10.1111/j.1601-6343.2007.00396.x.

[11] 11. Chapple I, Gizani S, Sanz M, Declerck D, Madianos P, Clerehugh V, et al. Gingival and periodontal diseases and conditions in children and adolescents: consensus report. J Clin Periodontol. 2026;53(7):1068-99. doi:10.1111/jcpe.70033.

[12] 12. van Gastel J, Quirynen M, Teughels W, Coucke W, Carels C. Longitudinal changes in microbiology and clinical periodontal variables after placement of fixed orthodontic appliances. J Periodontol. 2008;79(11):2078-86. doi:10.1902/jop.2008.070622.

[13] 13. Lv Y, et al. Cross-omics analysis reveals microbe-metabolism interactions characteristic of gingival enlargement associated with fixed orthodontic in adolescents. J Oral Microbiol. 2025;17(1):2513739. doi:10.1080/20002297.2025.2513739.
[14] Note. Deux références listées dans la version initiale n'étaient jamais citées dans le texte et ont été retirées : Eid HA, et al. (J Int Oral Health. 2014;6(1):1-4) et Jiang Q, et al. (J Am Dent Assoc. 2018;149(8):712-20.e12). Un doublon du consensus Chapple (ex-réf. 4 et 17) a également été supprimé ; la liste ci-dessus respecte l'ordre de première apparition des appels dans le texte.
[15] 14. Rihawi H, Al Habashneh R, Abdel-Hafez R, Alzoubi M. Comparison of two treatment modalities in the management of gingival enlargement during orthodontic treatment: a randomized clinical trial. Quintessence Int. 2025;56(3):226-35. doi:10.3290/j.qi.b5809024.
[16] 15. Bollen AM, Cunha-Cruz J, Bakko DW, Huang GJ, Hujoel PP. The effects of orthodontic therapy on periodontal health: a systematic review of controlled evidence. J Am Dent Assoc. 2008;139(4):413-22. doi:10.14219/jada.archive.2008.0178.