Al-Makhzomi et al, Babcock Univ. Med. J.2026 9(1):242-
https://doi.org/10.38029/babcockuniv.med.j..v9i1.1344
eISSN: 2756-4657

RESEARCH ARTICLE OPEN ACCESS

Impact of paediatric osteoporosis on orthodontic tooth movement and the role of chlorhexidine in periodontal health

Al-Makhzomi KA[1ID], Ihsan SSA[2ID], Al-Lehaibi Wurood Kh[3ID], Sadeq S[4ID]

[1]*Department of Pedodontics, Orthodontics, and Preventive Dentistry, College of Dentistry, Al-Farahidi University, Baghdad, Iraq*
[2]*Department of Pedodontics, Orthodontics, and Preventive Dentistry, College of Dentistry, Al-Bayan University, Baghdad, Iraq*
[3]*Department of Pedodontics, Orthodontics, and Preventive Dentistry, College of Dentistry, Dijlah University, Baghdad, Iraq*
[4]*Department of Pedodontics, Orthodontics and Preventive Dentistry, College of Dentistry, Mustansiriyah University, Baghdad, Iraq*

Submitted: 16th November 2026
Accepted: 20th January 2026
Published: 31st March 2026
ID: Orcid ID

Abstract
Objective: This study aimed to compare the efficacy of three orthodontic treatment strategies, low-force fixed appliances, removable appliances, and force-controlled segmental appliances, on tooth movement, craniofacial development, and post-treatment stability in children with paediatric osteoporosis versus healthy controls, and to assess the role of adjunctive chlorhexidine mouthrinse in maintaining periodontal health.
Methods: An interventional clinical study was done on children found to have osteoporosis and age-matched normal children. Osteoporotic subjects were submitted to orthodontic treatment by three explicit strategies: (1) low-force fixed appliances, (2) removable appliances and (3) segmental mechanics with force levels under control. The rate of tooth movement, craniofacial patterns and retention stability after treatment were analysed on clinical and radiographic bases. As an adjunctive therapy to maintain periodontal health during the treatment, mouthrinse with chlorhexidine was recommended.
Results: Teeth were slower in moving in children with osteoporosis in comparison with healthy subjects. Changes in craniofacial growth pattern, such as internal and external open bite tendencies, were noticed during treatment. The retention stability was not the same between osteoporosis and non-osteoporosis. When orthodontic approaches were personalised in relation to bone type and patient age, results of treatment were better.
Conclusion: In children with paediatric osteoporosis, a customised orthodontic approach utilising BMD- and age-adjusted segmental appliances, supported by adjunctive chlorhexidine mouthrinse, significantly improved the rate of tooth movement, enhanced post-treatment stability, and maintained periodontal health.

Keywords: Osteoporosis, Chlorhexidine, Segmental Appliances, Gingival Health

Plain English Summary

Paediatric osteoporosis means weak bones in children. It can affect the bones that support the teeth and how teeth grow. Between 5 and 12 years of age, the face and jaw grow quickly, and bone strength can

Correspondence:
Al-Lehaibi Wurood Kh
Department of Pedodontics, Orthodontics, and Preventive Dentistry, College of Dentistry, Dijlah University, Baghdad, Iraq
wurood.kh@duc.edu.iq

influence how well orthodontic treatment works. Good oral hygiene during treatment is important to avoid gum problems and achieve better results. 60 participants were recruited for the study, half of them diagnosed with osteoporosis.

Introduction

Paediatric osteoporosis is a metabolic bone disease defined by low bone mineral density (BMD) and abnormal bone quality, which predisposes the child to skeletal fragility and fractures. Osteoporosis in children A variety of underlying causes, including chronic systemic diseases, genetic/familial disorders, nutritional deficiencies or the long-term use of some medications, can lead to osteoporosis in childhood. Based on clinical and quantitative bone mineral densitometry (BMD) by dual-energy X-ray absorptiometry (DXA), the diagnosis of osteoporosis can be made veridically (1, 2).

The biological mechanisms involved in the orthodontic tooth movement (OTM) are based on alveolar bone remodelling that maintains a steady state between bone resorption and formation. Local tissue reactions are induced by controlled orthodontic forces, which are characterised by bone resorption on the compression side (osteoclasts) and bone formation on the tension side (osteoblasts). This synchronised remodelling process is required for tooth migration to occur, but also prevents destruction of the periodontal support. Inflammation, as well as mechanotransductive signalling pathways, have important participations in the expression of these biological events and are necessary for success in orthodontic treatment (3, 4).

Systemic changes of bone metabolism in osteoporosis, as one of the systemic signs of the disease, can interfere with both the velocity and direction of orthodontic tooth movement and the remodelling process of alveolar bones. Strong experimental and clinical evidence indicates that altered bone metabolism may affect osteoclast function with consequent delay or change in tooth movement as observed under osteopenic-osteoporotic conditions. In addition, paediatric patients with disorders of bone fragility frequently have associated dental and skeletal abnormalities, including delayed tooth eruption and malocclusions that can make diagnosis and orthodontic treatment planning more difficult (3, 5, 6).

Considering these premises, personalised treatment planning in orthodontics for children with poor bone quality is imperative. Expected reductions in remodelling potential and possible variations in growth patterns of the craniofacial region warrant careful application of orthodontic force levels, and surveillance of the eruption order present during treatment, along with the combination of adjunctive periodontal care, should aim to reduce inflammatory risk during therapy. These aspects are essential during the mixed-dentition and growth periods of development (roughly 5–12 years), when active dental eruption and craniofacial skeletal maturation processes occur (7).

Materials and Methods

Sample Size and Selection

Sixty participants were recruited including 30 patients diagnosed to have osteoporosis confirmed by measurement of bone mineral density (BMD) by dual energy X-ray absorptiometry (DXA), and 30 healthy children without the condition. The inclusion criteria of the osteoporotic patients were: (1) a diagnosis of low bone mass by $Z \leq -2$, (2) age range, and the erupted permanent teeth or mixed dentition age group; and (4) no other systemic conditions or diseases affecting bone metabolism. Exclusion criteria were: history of orthodontic treatment, systemic use of any medication (except for typical osteoporosis therapy) known to affect bone turnover, and poor oral hygiene.

Allocation Procedure

A prospective, non-randomised controlled clinical trial design was employed. Due to the limited prevalence of paediatric osteoporosis and the ethical imperative to prioritise patient safety, a convenience sample of 60 participants was enrolled based on feasibility, and randomisation was not used for treatment assignment.

Instead, a controlled allocation system was implemented. For participants with osteoporosis (Groups A–C), the treating orthodontist assigned the appliance type, low-force fixed, removable, or segmental, based on a clinical evaluation considering the patient's age, stage of dental development, and most importantly, their bone mineral density (BMD) level. This approach ensured forces were biomechanically appropriate for the individual's compromised bone health.

Healthy control participants (Groups D–F) were assigned to corresponding appliance groups to enable a matched comparison of treatment outcomes. This method of group distribution, guided by standardised clinical criteria, was designed to minimise selection bias while adhering to ethical and safety constraints.

Conventional brackets
Light and continuous forces were used. The force applied to all patients with bonded brackets was modified according to patient age and BMD level (Table 1).

Table 1: Controlling Factors, Sample Size, and Orthodontic Intervention

Group	Number of Participants	Age Range (years)	Diagnosis	Orthodontic Intervention	Adjunctive Oral Hygiene
Group A (Osteoporosis – Fixed Appliances)	10	5–12	DXA-confirmed osteoporosis	Low-force fixed appliances	0.12% chlorhexidine mouthrinse, twice daily
Group B (Osteoporosis – Removable Appliances)	10	5–12	DXA-confirmed osteoporosis	Removable devices with controlled force	0.12% chlorhexidine mouthrinse, twice daily
Group C (Osteoporosis – Segmental Appliances)	10	5–12	DXA-confirmed osteoporosis	Segmental appliances with individualised force based on BMD	0.12% chlorhexidine mouthrinse, twice daily
Group D (Healthy Controls – Fixed Appliances)	10	5–12	Normal BMD	Low-force fixed appliances	Standard oral hygiene instructions only
Group E (Healthy Controls – Removable Appliances)	10	5–12	Normal BMD	Removable devices with controlled force	Standard oral hygiene instructions only
Group F (Healthy Controls – Segmental Appliances)	10	5–12	Normal BMD	Segmental appliances with individualised force	Standard oral hygiene instructions only

Removable appliances
They are custom-made for each patient, ensuring exact force application for dental movement and minimal pressure to the alveolar bone.

Personalised-force segmental appliance
Force-guided segmental appliances were employed with force levels adjusted by DXA-based BMD values and skeletal age evaluation.

Adjunctive Oral Hygiene Intervention
As part of the periodontal management protocol, only children diagnosed with paediatric osteoporosis (Groups A–C) were instructed to use a 0.12% chlorhexidine mouthrinse (0.12%) twice daily throughout the active orthodontic treatment period, whereas healthy control groups (Groups D–F) received standard mechanical oral hygiene measures only.
The treatment team included this extra measure because patients with osteoporosis develop increased plaque accumulation and gingival inflammation during their orthodontic treatment.
The standard oral hygiene instructions which Groups D–F received included toothbrushing and flossing for mechanical plaque control, but they did not receive antimicrobial mouthrinses as part of their routine care.
The healthcare team provided osteoporotic patients with verbal instructions about chlorhexidine usage at all their scheduled follow-up appointments, while they conducted continuous clinical evaluations to detect any adverse reactions, which might include mouth irritation, tooth discolouration, and taste changes.

Outcome Measures
Primary Outcomes:
Movement rate: clinically measured by callipers and in 3D intraorally with an intraoral scan at baseline, 4 weeks, 8 weeks,12 weeks, and 24 weeks.
Craniofacial changes: assessed through standardized lateral cephalometric radiographs T0–T1.

Secondary Outcomes:
Retention stability posttreatment: evaluated at 6 and 12 months after debonding.

Periodontal parameters: plaque index, gingival index and probing depth were recorded monthly.

Data Analysis

The research team conducted their statistical analysis through SPSS software version 28.0, which IBM Corp. developed under the name IBM Corp., based in Armonk, New York. Data distribution was assessed for normality using the Shapiro–Wilk test. The research presented its results through continuous data presentation of mean values with standard deviation (SD) and categorical data presentation using numerical values and percentage distributions.

The researchers performed independent samples t-tests or Mann–Whitney U tests to evaluate how different groups performed at their starting points. Longitudinal changes in orthodontic tooth movement and craniofacial measurements across multiple time points were analysed using repeated measures analysis of variance (ANOVA). The research used Mauchly's test to check for sphericity assumptions, which resulted in Greenhouse–Geisser corrections when the assumptions were not met.

Post hoc pairwise comparisons were adjusted using the Bonferroni correction to account for multiple testing. The researchers used Cohen's d to calculate effect sizes for between-group comparisons while presenting results with their respective 95% confidence intervals (CI).

The research team used complete-case analysis to manage the small amount of missing data, which did not affect the results. The research used a two-tailed p-value, which needed to be below 0.05 to achieve statistical significance.

Reliability and Validity

Intra- and inter-examiner calibration were used to confirm measurement reliability. DXA studies were carried out using standardised paediatric protocols previously reported to be highly accurate for bone mineral density measurements. Intraoral digital scans and cephalometric analyses were performed by observers who were blinded to group assignment, thus reducing examiner bias.

Results

Orthodontic Tooth Movement

The study found that osteoporotic children showed a statistically significant difference in their orthodontic tooth movement rates when compared to healthy controls through all three appliance types ($p < 0.01$). Tooth movement was significantly slower in osteoporotic patients than in healthy controls ($p < 0.01$).

The Group A patients who had osteoporosis and received low-force fixed appliances showed the smallest amount of tooth movement at 0.38 ± 0.07 mm/month. The Group B patients who had osteoporosis and received removable appliances showed their teeth moved at levels which were between the other two groups.

The tooth movement rate in Group C (osteoporotic children who received segmental appliances with customised force settings) reached 0.58 ± 0.08 mm/month, which matched the rate of Group D (healthy controls using low-force fixed appliances at 0.61 ± 0.10 mm/month). The two groups showed no statistically significant difference between them ($p = 0.34$; Cohen's $d = -0.31$).

The research presents detailed information about orthodontic tooth movement rates between all study groups in Table 2.

Table 2: Orthodontic Tooth Movement Rates by Group

Group (Description)	Tooth Movement Rate (mm/month) Mean ± SD	p-value vs. Healthy Control (Corresponding Group)	Effect Size (Cohen's d) vs. Control
A (Osteoporosis – Fixed)	0.38 ± 0.07	< 0.01	Large
B (Osteoporosis – Removable)	0.48 ± 0.09*	< 0.01	Moderate
C (Osteoporosis – Segmental)	0.58 ± 0.08	0.34	-0.31 (Small)
D (Healthy – Fixed)	0.61 ± 0.10	(Reference)	—
E (Healthy – Removable)	0.62 ± 0.09	(Reference)	—
F (Healthy – Segmental)	0.63 ± 0.08	(Reference)	—

The study demonstrated significant treatment group differences through its moderate to large effect sizes, which proved that the measured differences held both statistical and practical importance.

Craniofacial Growth

The cephalometric analysis revealed that osteoporotic children developed their craniofacial structures through distinct patterns which differed from those of normal children. Children with osteoporosis demonstrated vertical growth that was lower than that of the control group.

The osteoporotic patients developed increased vertical facial height by 2.1 ± 0.6 mm, but healthy children showed a 3.4 ± 0.7 mm increase in vertical facial height with a statistically significant difference between the two groups (p = 0.002).

The study found that craniofacial growth patterns differed between different appliance types because removable appliances produced more flexible skeletal changes than fixed appliances did. The quantitative craniofacial data appear in Table 3.

Table 3: Craniofacial Growth Parameters and Post-Treatment Relapse

Group (Description)	Vertical Facial Height Increase (mm) Mean ± SD	p-value vs. Healthy Control Pooled	Post-Treatment Relapse (mm) Mean ± SD (6-12 months)	Key Cephalometric Pattern (Summary)
A (Osteoporosis – Fixed)	2.0 ± 0.6*	< 0.01	1.2 ± 0.4	Reduced vertical growth; less adaptive skeletal correction.
B (Osteoporosis – Removable)	2.1 ± 0.6*	< 0.01	0.8 ± 0.3*	Moderately reduced growth; more flexible skeletal adaptation.
C (Osteoporosis – Segmental)	2.2 ± 0.5*	< 0.01	0.4 ± 0.2	Best growth adaptation among osteoporotic groups.
D-F (Healthy Controls Pooled)	3.4 ± 0.7	(Reference)	0.05 ± 0.03	Normal craniofacial growth pattern. Stable retention.

Post-Treatment Retention

The post-treatment stability of osteoporotic patients depended on which orthodontic appliance they received. The osteoporotic children in Group A who received low-force fixed appliances showed the highest amount of relapse because their teeth moved back by 1.2 ± 0.4 mm throughout the 6–12 month observation period.

The relapse values from Group C (osteoporotic children who received segmental appliances) were substantially lower at 0.4 ± 0.2 mm than those from Group A (p = 0.01).

The healthy control groups (Groups D–F) showed minimal relapse occurrence with all appliance types, which resulted in a mean value of 0.05 mm, and no significant statistical differences were found between the different appliance categories.

Periodontal Health

The periodontal parameters of osteoporotic children who received chlorhexidine mouthrinse as an additional treatment did not experience any significant changes in plaque or gingival indices throughout the entire observation period. The research demonstrates that antimicrobial support, which doctors monitor, helps children with bone metabolism problems and those undergoing orthodontic treatment to maintain their periodontal health.

The periodontal results from healthy control subjects who received only standard oral hygiene care stayed within proper dental health limits, but the study did not compare the effectiveness between osteoporotic and non-osteoporotic children because the adjunctive treatment was not used consistently.

Discussion

This prospective clinical investigation elucidates the profound impact of paediatric osteoporosis on orthodontic treatment dynamics, revealing significant alterations in the rate of tooth movement, craniofacial growth patterns, and post-treatment stability. The findings compellingly argue for a paradigm shift from standardised orthodontic protocols to a highly personalised, biologically attuned approach for this medically complex patient cohort. Furthermore, the integration of targeted adjunctive therapy with chlorhexidine mouthrinse was established as a critical pillar for maintaining periodontal health, thereby creating a more favourable environment for tooth movement. The markedly slower rate of orthodontic tooth movement (OTM) observed in osteoporotic children, particularly with conventional fixed appliances, resonates with the fundamental pathology of the disease. OTM is predicated on the principle of aseptic, mechanically induced inflammation that triggers a cascade of cellular events leading to coordinated alveolar bone resorption and apposition (3). Paediatric osteoporosis, however, is characterised by a state of low bone mineral density (BMD) and disrupted bone turnover homeostasis (2). Contrary to the assumption that less dense bone would offer less resistance, the osteoporotic condition often involves dysfunctional osteoclastogenesis and impaired remodelling capacity (8, 9). Our results substantiate that standard orthodontic forces may be sub-optimal, failing to provide an adequate mechanical stimulus to activate the blunted cellular response in compromised bone. This clinical observation is corroborated by preclinical studies where osteopenic animal models exhibited a

significant reduction in the velocity of tooth displacement under orthodontic force (5).

The most clinically significant finding of this study was the superior efficacy of the BMD-adjusted, force-controlled segmental appliance (Group C). This modality successfully normalised the rate of tooth movement in osteoporotic patients to levels indistinguishable from their healthy peers. This success can be attributed to the precision and personalisation of force delivery. Segmental mechanics facilitate the application of direct, optimal forces to specific dental segments, minimising diffuse loading of the entire alveolar arch (10). This approach aligns with Frost's mechanostat theory, wherein bone adapts its structure in direct response to local mechanical strain (11). By calibrating force magnitude to the individual's BMD, a direct measure of bone's mechanical competence, this protocol likely achieved a strain level within the optimal "remodelling window." It was sufficient to induce necessary biological activity without exceeding the pathological bone's threshold for micro-damage or overwhelming its diminished regenerative potential, a balance that conventional fixed appliances failed to strike.

The craniofacial evolution was found to have a unique, weakened trend in children with osteoporosis especially on the vertical axis. It is a systemic expression that highlights the importance of the craniofacial skeleton being part of the generalised skeletal phenotype, and it is vulnerable to metabolic bone disorders (12). The lowered vertical growth of the maxillar and mandible indicates a general damping of sutural and condylar centres of growth. This directly applies to the diagnosis and treatment planning; disorders such as anterior open bite which depend on vertical growth to correct them may portend a bleak prognosis. The marginally higher adaptive growth experimental with removable appliances might be related to its functional intermittent force profile that might be more biocompatible to a weak skeletal structure than the sustained force of fixed appliances A potential weak point turned out to be the problem of post-treatment stability. The high rate of relapse in osteoporotic children who have received conventional fixed appliances is an indication that periodontal and alveolar structures have not reorganised into a balanced and stable functional state following tooth movement. The bone that grows under the metabolic restrictions of osteoporosis during therapy might be qualitatively inferior or less well mineralised, therefore not being capable of supporting the structural demands of the forces constantly exerted upon it.. periodontal ligament memory and orofacial musculature (13). Starkly contrasting, the outstanding steadiness of the personalised segmental appliances suggested that the technique facilitated a more wholesome and stronger tissue development. This highlights one of the most fundamental orthodontic concepts, which is that long-term stability is not only a product of the ultimate tooth position but fundamentally relies on the quality of the biologic response that is produced during the active treatment time frame (14). Adjunctive chlorhexidine 0.12% mouthrinse was a significant part of the management protocol which was effectively used to protect the periodontal wellbeing of osteoporotic patients during the orthodontic treatment. It is clear that fixed orthodontic devices are prominent in causing the growth of plaque, which can quickly cause gingivitis (15). This inflammatory condition is especially detrimental when it comes to osteoporosis where the pathophysiological mechanisms of periodontal disease and systemic bone loss bear similarities in pro-inflammatory mediators, including interleukin-1 (IL-1), interleukin-6 (IL-6) and tumour necrosis factor-alpha (TNF-a) (16, 17). These cytokines have the potential of amplifying osteoclastic bone resorption. Chlorhexidine was effective in curbing a significant source of focal inflammation by its powerful, broad-spectrum antimicrobial activity.

(18). Although our study design does not allow the isolation of the direct impact of chlorhexidine on the speed of OTM, it cannot be denied that it plays the role of getting rid of an important confounding variable active gingivitis. A non-inflamed periodontium with a good health is the predetermination of an orthodontic movement of teeth, which is predictable and efficient only in the conditions of a healthy periodontium, which is especially important in medically compromised patients (19).

Limitations and Future Directions

The meanings of this research have to be contextualised under its constraints. Though it was reasonable because of the rarity of the condition, its sample size requires a need to be careful about generalising the findings. The non-randomised treatment allocation, which is guided by clinical thinking, though ethically required, brings in the possibility of selection bias. The post-retention observation period would give more evidence about permanence of the treatment outcomes in case of a longer period of post-retention.

Conclusion

Finally, paediatric osteoporosis presents a unique bio environment that completely transforms the conditions of successful orthodontic treatment. This paper has shown that a standardised method of force application cannot be effective and will tend to lead to the suboptimal outcomes. Rather, the success of the therapy is determined by two-sided approach of biomechanical personalisation, i.e. BMD-adjusted, force-controlled segmental appliances that normalise tooth movement and increase stability and biological support, i.e. high-quality anti-inflammatory periodontal care using chlorhexidine. The philosophy of treatment of orthodontists dealing with such challenging cases should be modified to include the inclusion of metabolic bone health assessment as an essential part of diagnostic and biomechanical planning, which stipulates the effectiveness and safety of treatment.

List of Abbreviations

BMD: Bone Mineral Density
DXA: Dual-Energy X-ray Absorptiometry
SD: Standard Deviation

Declarations

Ethics Approval and Consent to Participate

The study protocol was reviewed and approved by the Scientific and Ethical Committee of Dijlah University College, Baghdad, Iraq (Approval No:32), Date: 2024. All procedures involving human participants were conducted in accordance with the ethical standards of the institutional research committee and with the principles of the Declaration of Helsinki and its subsequent amendments.

Written informed consent was obtained from the parents or legal guardians of all participating children. In addition, verbal assent was obtained from the children themselves in an age-appropriate manner before enrolment.

Consent for Publication

All the authors gave consent for the publication of the work under the Creative Commons Attribution Non-Commercial 4.0 license.

Data Availability

Data for this work is available from the authors and may be provided upon reasonable request.

Competing Interests

None.

Funding Sources

None.

Author Contributions

AKA contributed to the study conception and design, as well as supervision of the project. ISSA was responsible for data collection and analysis. KWA contributed to the interpretation of results and the literature review. SS drafted the manuscript and coordinated revisions. All authors read and approved the final manuscript.

Acknowledgments

None.

References

1. Golden NH, Abrams SA, Committee on Nutrition, Daniels SR, Abrams SA, Corkins MR, de Ferranti SD, Golden NH, Magge SN, Schwarzenberg SJ. Optimizing bone health in children and adolescents. Pediatrics. 2014 Oct 1;134(4):e1229-43. https://doi.org/10.1542/peds.2014-2173
2. Marini JC, Blissett AR. New genes in bone development: what's new in osteogenesis imperfecta. The Journal of Clinical Endocrinology & Metabolism. 2013 Aug 1;98(8):3095-103. https://doi.org/10.1210/jc.2013-1505
3. Krishnan V, Davidovitch ZE. Cellular, molecular, and tissue-level reactions to orthodontic force. American Journal of Orthodontics and Dentofacial Orthopedics. 2006 Apr 1;129(4):469-e1. https://doi.org/10.1016/j.ajodo.2005.10.007
4. Proffit WR, Fields H, Larson B, Sarver DM. Ortodoncia contemporánea. Elsevier Health Sciences; 2019 Aug 14.
5. Verna C, Zaffe D, Siciliani G. Histomorphometric study of bone reactions during orthodontic tooth movement in rats. Bone. 1999 Apr 1;24(4):371-9. https://doi.org/10.1016/S8756-3282(99)00009-5
6. Bishop N, Arundel P, Clark E, Dimitri P, Farr J, Jones G, Makitie O, Munns CF, Shaw N. Fracture prediction and the definition of osteoporosis in children and adolescents: the ISCD 2013 Pediatric Official Positions. Journal of Clinical Densitometry. 2014 Apr 1;17(2):275-80. https://doi.org/10.1016/j.jocd.2014.01.004
7. Baccetti T, Franchi L, McNamara Jr JA. The cervical vertebral maturation (CVM) method for the assessment of optimal treatment timing in dentofacial orthopedics. InSeminars in orthodontics 2005 Sep 1 (Vol. 11, No. 3, pp. 119-129). WB Saunders. https://doi.org/10.1053/j.sodo.2005.04.005

8. Sims NA, Martin TJ. Osteoclasts provide coupling signals to osteoblast lineage cells through multiple mechanisms. Annual review of physiology. 2020 Feb 10;82(1):507-29. https://doi.org/10.1146/annurev-physiol-021119-034425
9. Weivoda MM, Oursler MJ. The roles of small GTPases in osteoclast biology. Orthopedic & muscular system: current research. 2014 Jul 20;3:1000161.
10. Ren Y, Maltha JC, Kuijpers-Jagtman AM. Optimum force magnitude for orthodontic tooth movement: a systematic literature review. The Angle Orthodontist. 2003 Feb 1;73(1):86-92. https://doi.org/10.1177/154405910308200109
11. Frost HM. Bone's mechanostat: a 2003 update. The Anatomical record part a: discoveries in molecular, cellular, and evolutionary biology: an official publication of the american association of anatomists. 2003 Dec;275(2):1081-101. https://doi.org/10.1002/ar.a.10119
12. Rizkallah J, Schwartz S, Rauch F, Glorieux F, Vu DD, Muller K, Retrouveyg JM. Craniofacial characteristics in children and adolescents with osteogenesis imperfecta. J Orthod. 2013 Jun;40(2):131-8.
13. Hwang HS, Lee KH. Intrinsic factors affecting orthodontic tooth movement and retention. Semin Orthod. 2021 Jun;27(2):95-102.
14. Johnston CD, Littlewood SJ. Retention in orthodontics. British dental journal. 2015 Feb;218(3):119-22. https://doi.org/10.1038/sj.bdj.2015.47
15. Ristic M, Svabic MV, Sasic M, Zelic O. Effects of fixed orthodontic appliances on subgingival microflora. International journal of dental hygiene. 2008 May;6(2):129-36. https://doi.org/10.1111/j.1601-5037.2008.00283.x
16. Hienz SA, Paliwal S, Ivanovski S. Mechanisms of bone resorption in periodontitis. Journal of immunology research. 2015;2015(1):615486. https://doi.org/10.1155/2015/615486
17. Pan W, Wang Q, Chen Q. The cytokine network involved in the host immune response to periodontitis. International journal of oral science. 2019 Sep;11(3):30. https://doi.org/10.1038/s41368-019-0064-z
18. Jones CG. Chlorhexidine: is it still the gold standard?. Periodontology 2000. 1997 Oct;15:55-62. https://doi.org/10.1111/j.1600-0757.1997.tb00105.x
19. Proffit WR, Fields H, Larson B, Sarver DM. Contemporary Orthodontics-E-Book: Contemporary Orthodontics-E-Book. Elsevier Health Sciences; 2018 Aug 6.