ORIGINAL RESEARCH

Impact of Vitamin D3 on Postorthodontic Treatment Stability: A Randomized Controlled Trial

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Abstract

Aim: This study aims to clinically evaluate whether the local administration of vitamin D3 enhances postorthodontic tooth stability over a period of 3 months immediately after debonding.

Materials and methods: Patients aged 15–30 years with dental malocclusion and moderate crowding in the lower arch (Little's irregularity score of 4–6) were selected and randomly split into the experimental and the control groups. After complete alignment and leveling, vitamin D3 injection was delivered to the experimental group and the control group was given a placebo injection with 0.9% normal saline mixed with 2% lignocaine. Fourteen days after the injection, the lower archwires were removed from the control and experimental groups.

Results: Relapse was significantly higher in control than in the experimental group at all-time intervals. Statistically significant values of relapse were observed at T2 and T3 intervals between the two groups, with greater relapse in the control group than in the experimental group. Inter-canine width, arch perimeter, and intermolar width showed mild changes over 3-month period, but there were no significant differences between the two groups.

Conclusion: Relapse was seen in both the control and the experimental in the first 4 weeks of the study. Still, the control group showed a greater relapse rate in the following 8th week and 12th week when compared to the experimental group.

Keywords: Arch perimeter, Crowding, Intercanine width, Inter-molar width, Postorthodontic stability, Relapse, Vitamin D3.

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Introduction

Orthodontic treatment aims to achieve optimal esthetics and function by correcting malocclusions. Orthodontic tooth movement (OTM) involves mechanical, chemical, and cellular events within the surrounding tissues of the teeth due to external forces, allowing structural changes for tooth movement within the alveolar housing.[1]

Orthodontics, particularly fixed appliance therapy, can correct malocclusions, including crowding, spacing, rotations, deep bite, open bite, and crossbite, which are prevalent among patients. However, corrected teeth are prone to relapse, or the tendency to return to their original positions, primarily due to the recoil forces of gingival fibers, pressures from lips, cheeks, and tongue, and ongoing growth and tooth encounters.[2]

Achieving long-term stability postorthodontic treatment remains a significant challenge for orthodontists. Long-term relapse is influenced not only by the outcome of orthodontic treatment, but also by physiological changes in the dentition and tissue, and the forces surrounding them. Fixed or removable retention appliances are used to reduce relapse risk, but evidence regarding treatment protocols and duration of wear remains limited.[3] Long-term use of retention appliances is often considered the gold standard for minimizing relapse.

In addition to retention appliances, other measures to enhance tooth position stability include clear thermoplastic retainers, medications, and gingival fiber-cutting procedures.[4] External agents modifying bone remodeling responses have been studied with varying success. These include osteoprotegerin gene transfer, pharmacological agents like bone morphogenetic proteins (BMPs), bisphosphonates, vitamin D3, iPRF, antibiotics, low-level laser therapy, cytokines, hormones, and mechanical vibrations.[5,6]

Studies have suggested that calcitriol (vitamin D3) may enhance bone formation and periodontal tissue remodeling by increasing osteoblastic activity, potentially improving postorthodontic stability.[7,8] Vitamin D3 has been associated with both acceleration and retardation of OTM. Locally injected vitamin D3 has increased

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OTM rate by boosting local inflammatory mediators and bone remodeling, particularly in the first 7 days post-injection.[9]

Despite advancements in orthodontic retention strategies, there is limited evidence on adjuvant approaches to improve long-term tooth stability post-treatment. Systemic administration of vitamin D has been shown to decrease OTM rate and relapse by increasing bone resistance and mineral density.[10] Recent animal studies have highlighted the potential of vitamin D3 in enhancing bone remodeling and reducing relapse, but its local effects in humans remain underexplored.[11] Given the association of vitamin D3 with increased bone mineral density and altered bone trabeculae structure, further clinical evaluation is necessary to determine its efficacy in postorthodontic retention.

This study aims to clinically evaluate whether the local administration of vitamin D3 enhances postorthodontic tooth stability over a period of 3 months immediately after debonding.

Materials and Methods

This prospective, randomized, double-blinded, parallel-arm study was conducted in the Department of Orthodontics and Dentofacial Orthopedics, India, from June 2022 to November 2022. The study was approved by the Institutional Review Board and Institutional Human Ethics Committee on June 2022 (IHEC-I/0672/22) and registered with the Clinical Trial Registry of India (CTRI/2022/10/046600).

The sample size was calculated using G*Power software version 3.1.9.4. A total sample size of 14 (7 in each group) was determined to provide 80% power with an alpha error of 5%. The selection criteria include both male and female patients aged 15–30 years, patients with any type of malocclusion and moderate crowding in the lower arch (Little's irregularity score 4–6), patients undergoing non-extraction fixed orthodontic treatment, patients with periodontal sound dentition and patients in good general health. Exclusion criteria are patients requiring extractions for orthodontic treatment, history of previous orthodontic treatment, poor oral hygiene, missing permanent teeth in the mandibular arch (except third molars), any systemic diseases, long-term use of medications, recent history of viral fever (e.g., dengue) and pregnancy.

Systematic random sampling was used, with even numbers assigned to group A (control) and odd numbers to group B (experimental). All patients were treated by the same operator. Figure 1 shows the consolidated standards of reporting Trials (CONSORT) flow diagram. All treatment procedures were explained to the patients and their guardians, and informed consent was obtained from those who met the selection criteria and were willing to participate.

Methodology

Patients meeting the selection criteria were randomly assigned to two groups:

Group A (control): Received a placebo injection containing 5 mL of 0.9% saline mixed with 2% lignocaine.

Group B (experimental): Received 5 mL of vitamin D3 (trade name: ARACHITOL 6 L) diluted with 2% lignocaine, administered intraligamentously in the lower anterior segment.

Intraoral photographs and alginate impressions were taken initially for both groups. Fixed appliance therapy was initiated, and bonding was completed. After alignment and leveling, injections were administered. Fourteen-day post-injection, lower archwires were removed in both groups. Both groups were followed for 3 months at 4-week intervals (4th, 8th, and 12th weeks).

Digital superimposition of follow-up cast models was used to assess alignment and stability. Parameters measured included: (Fig. 2) Little's irregularity index (LII): Measures horizontal linear displacement of contact points of mandibular anterior teeth; Inter-canine width: Distance between the cusp tips of right and left canines; Inter-molar width: Distance between the mesial marginal ridges of the first permanent molars; arch perimeter: Space available in the dental arch for aligning teeth.[12–14]

Study models were evaluated at T0—immediately after removal of lower arch wire; T1—4th week (1st review after removing lower

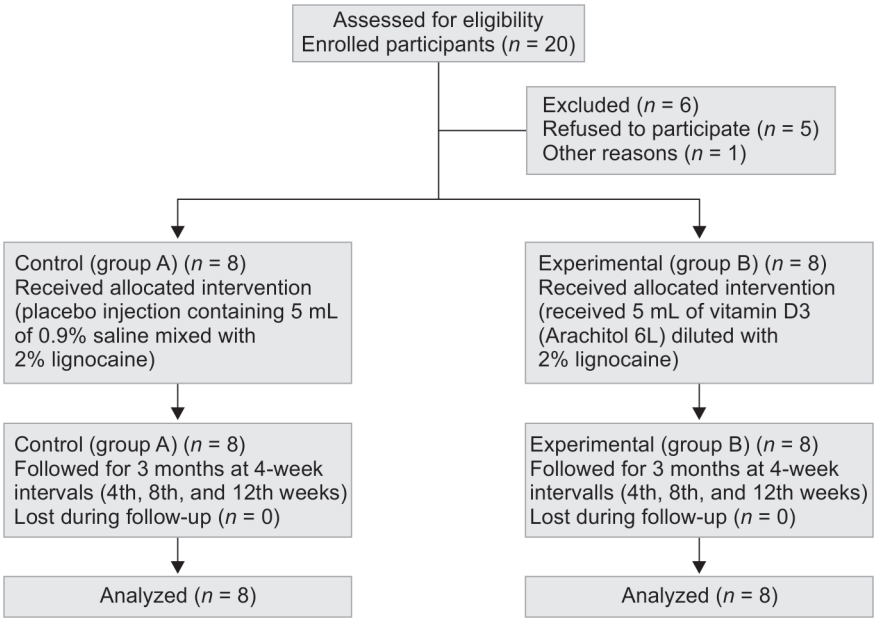
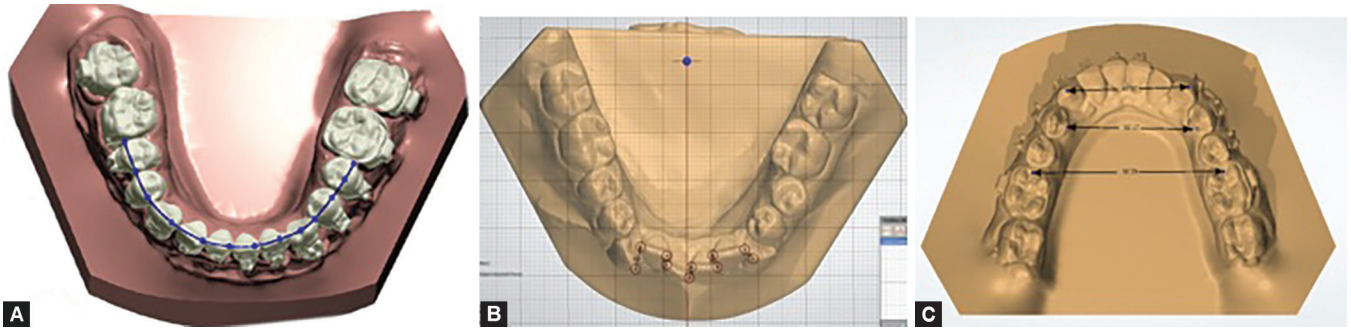


Fig. 1: Consolidated standards for reporting trials (CONSORT) flow diagram



Figs 2A to C: Scanned digital cast model for analysis: (A) Measurement of Little's irregularity Index; (B) Arch perimeter analysis in digital model; (C) Measurement of intercanine and intermolar width in digital model

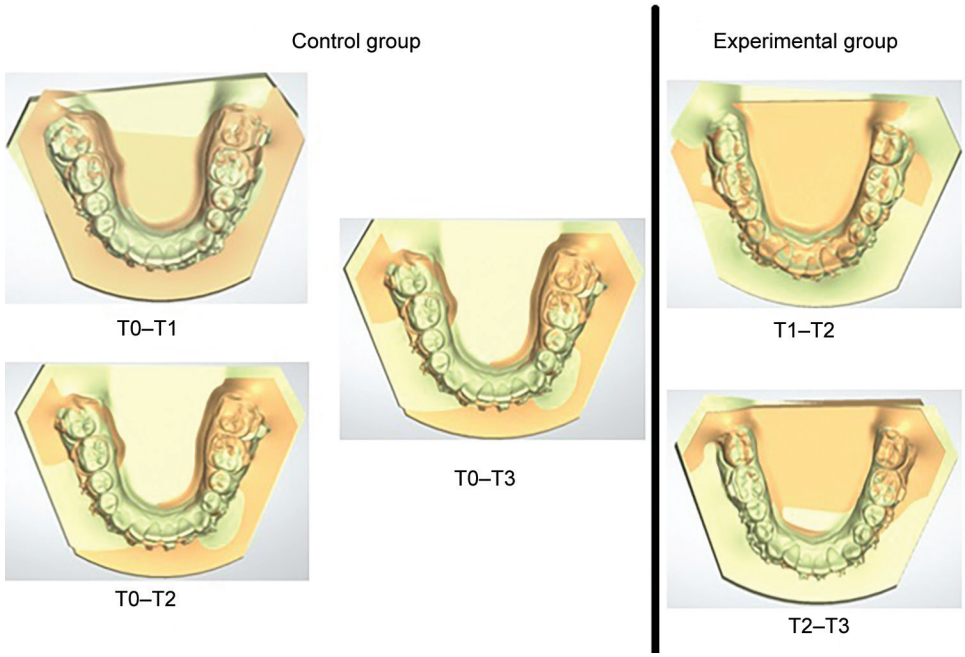


Fig. 3: Superimposition of digital models (control and experimental group) at different time intervals

arch wire); T2—8th week (2nd review after removing lower arch wire); T3—12th week (3rd review after removing lower arch wire) for relapse using LII, inter-canine width, inter-molar width, and arch perimeter. Any detected relapse was addressed with further finishing and detailing of the occlusion (Fig. 3).

Statistical Analysis

All statistical analyses were performed using Statistical Package for Social Science (SPSS, version 17) for Microsoft Windows. The data obtained were of independent variables which are normally distributed, therefore analysis of variance tests were performed. Results from statistical analysis were represented as numbers and percentages, the data were expressed as mean and SD. An unpaired *t*-test was used to compare the correlation of variables between the two groups. A two-sided *p*-value < 0.05 was considered statistically significant.

Results

The study included 14 participants (7 males, 7 females) aged 15–30 years. Equal distribution of participants across groups ensured homogeneity. The Little`s irregularity index of the lower anterior in both the control and the experimental group was kept at 0 mm as the baseline value (indicating perfect alignment) at the start of the study, the mean values of the control group were 1.20 ± 0.3 mm, 1.92 ± 0.77 mm and 2.41 ± 0.49 mm at T1, T2 and T3 respectively and for the experimental group were 0.8 ± 0.34 mm., 1 ± 0.4 mm and 1.17 ± 0.24 mm at T1, T2 and T3 respectively. Analysis of variance indicates that significant relapse was observed in both groups, but the control group showed a higher relapse rate from T1 to T3 than the experimental group (Table 1).

The control group had a mean inter-canine width value of 28.76 ± 0.84 mm, 29.5 ± 1.08 mm, 28.98 ± 1.12 mm, 28.48 ± 0.97 mm and 28.41 ± 1.02 mm at pre-Rx , T0 , T1, T2 and T3 respectively, whereas in experimental group were 28.26 ± 1.42 mm, 28.80 ± 1.45 mm, 28.64 ± 1.52 mm, 28.49 ± 1.41 mm and 28.42 ± 1.41 mm at pre-Rx , T0, T1, T2 and T3 intervals respectively. ANOVA indicated no significant difference in inter-canine width within the groups at all-time intervals. Inter-canine width decreased from T0 to T3 in the control group (Table 2).

The arch perimeter in the control group had a mean value of 88.28 ± 2.46 mm (baseline value), 90.48 ± 2.52 mm, 89.44 ± 2.20 mm,

Table 1: Comparison of Little`s irregularity index between groups A and B at different time intervals

Parameter	*Time*	*Groups*	*Mean*	*SD*	*p-value*
Little's irregularity index	Pre-Rx	Group A	5.386	0.9459	0.469
		Group B	5.857	1.3758	
	T0	Group A	00	0.000a	**
		Group B	00	0.000a	
	T1	Group A	1.2043	0.67784	0.216
		Group B	0.8300	0.33961	
	T2	Group A	1.9271	0.76702	0.016*
		Group B	1.0086	0.40818	
	T3	Group A	2.4129	0.48904	0.001*
		Group B	1.1757	0.23957	

p-value <0.05* (statistically significant); *p*-value<0.001** (statistically significant)

Table 2: Comparison of inter-canine width between groups A and B at different time intervals

Parameter	*Intervals*	*Group*	*Mean*	*SD*	*p-value*
Inter-canine width	Pre-Rx	Group A	28.7571	0.84405	0.443
		Group B	28.2629	1.41633	
	T0	Group A	29.5157	1.07865	0.317
		Group B	28.8014	1.45473	
	T1	Group A	28.9829	1.12997	0.643
		Group B	28.6429	1.51796	
	T2	Group A	28.4814	0.96867	0.991
		Group B	28.4886	1.41035	
	T3	Group A	28.4114	1.01845	0.985
		Group B	28.4243	1.41243	

Table 3: Comparison of arch perimeter between groups A and B at different time intervals

Parameter	*Intervals*	*Group*	*Mean*	*SD*	*p-value*
Arch perimeter	Pre-Rx	Group A	88.2800	2.45794	0.278
		Group B	89.6314	1.96388	
	T0	Group A	90.4743	2.52831	0.683
		Group B	89.9114	2.51207	
	T1	Group A	89.4386	2.20013	0.826
		Group B	89.7057	2.25295	
	T2	Group A	88.9686	2.46553	0.595
		Group B	89.6557	2.23418	
	T3	Group A	88.5900	2.35982	0.461
		Group B	89.4814	2.08854	

88.97 ± 2.47 mm and 88.59 ± 2.08 mm at pre-Rx, T0, T1, T2 and T3 intervals respectively. In experimental group the values were 89.63 ± 1.96 mm (baseline value), 89.91 ± 2.51 mm, 89.71 ± 2.25 mm, 89.66 ± 2.23 mm and 89.48 ± 2.08 mm at pre-Rx, T0, T1, T2 and T3 respectively (Table 3). ANOVA indicated no significant difference in arch perimeter within the groups at all-time intervals. Arch perimeter tended to reduce from T0 to T3 in the control group.

Table 4: Comparison of inter-molar width between groups A and B at different time intervals

Parameter	*Intervals*	*Group*	*Mean*	*SD*	*p-value*
Inter-molar width	Pre-Rx	Group A	40.2100	1.41296	0.277
		Group B	41.1557	1.68555	
	T0	Group A	41.1214	1.44106	0.271
		Group B	42.0200	1.47340	
	T1	Group A	41.0557	1.45318	0.308
		Group B	41.8786	1.43688	
	T2	Group A	40.7143	1.38075	0.161
		Group B	41.8043	1.40792	
	T3	Group A	40.5057	1.36932	0.126
		Group B	41.7043	1.35562	

The intermolar width in the control group had a baseline value of 40.21 ± 1.41 mm pre-Rx, with 41.12 ± 1.44 mm, 41.06 ± 1.45 mm, 40.71 ± 1.38 mm and 40.50 ± 1.36 mm at T0, T1, T2, and T3 intervals respectively. Whereas in experimental group baseline value of 41.15 ± 1.68 mm at pre-RX, 42.02 ± 1.47 mm, 41.87 ± 1.43 mm, 41.80 ± 1.40 mm and 41.70 ± 1.35 mm at T0, T1, T2 and T3 respectively. ANOVA indicated no significant difference in inter-molar width within the groups at all-time intervals. No significant change in inter-molar width was observed in either group (Table 4).

The unpaired *t*-test was used to compare the little's irregularity index between groups at time intervals Pre Rx, T0, T1, T2, and T3. A statistically significant relapse was seen at T2 and T3 intervals between the groups, with the control group showing a greater magnitude of relapse than the experimental group. The unpaired *t*-test indicated no significant difference in inter-canine width, arch perimeter, and inter-molar width between the groups at all intervals.

The local administration of vitamin D3 significantly reduced relapse in alignment of the lower anterior teeth compared to the control group. However, no significant differences were observed in inter-canine width, inter-molar width, and arch perimeter between groups.

Discussion

Postorthodontic retention remains a controversial yet critical aspect of orthodontics, aiming to maintain teeth in their corrected positions after active treatment while counteracting forces from muscles, lips, and tongue. Fixed bonded retainers are generally preferred over removable ones in cases where patient compliance may be an issue. The rationale for retention includes allowing gingival and periodontal tissue reorganization, minimizing growth-related changes, enabling neuromuscular adaptation to new positions, and maintaining stable teeth alignment.[15] Studies suggest that the periodontal tissue requires at least 232 days to reorganize.[16] Retention practices have evolved over time, with a shift toward invisible retainers and lifetime retention protocols becoming more common.

Orthodontic forces induce bone remodeling, involving stages of alveolar resorption and bone formation. Vitamin D plays a pivotal role in regulating calcium and phosphorus levels, essential for

bone metabolism. Studies have demonstrated a positive correlation between vitamin D receptor polymorphisms and enhanced bone remodeling.[17,18] In orthodontics, vitamin D3 has shown potential in promoting bone turnover, improving post-treatment stability, and reducing relapse rates by enhancing osteoblast activity.

Research supporting vitamin D's role in orthodontics includes intra-ligamental injections that increase osteoclast activity, accelerating tooth movement and promoting bone remodeling.[19] Boyce and Weisbrode reported that vitamin D3 injections combined with a calcium-rich diet in rats led to an initial increase in osteoclast activity, followed by a rise in osteoblastic activity, enhancing overall bone turnover.[20] Similarly, studies by Collins and Sinclair demonstrated that vitamin D injections in dogs increased osteoclast numbers, improving tooth movement and stability.[18] Kale et al. reported that 1,25-dihydroxycholecalciferol (1,25 DHCC) enhanced bone resorption and osteoblast activity, promoting stability.[8] Kawakami and Takano-Yamamoto's findings align, showing that topical vitamin D application accelerated tooth movement and improved positional stability by increasing osteoblast activity.[17]

In this study, parameters like Little's irregularity index, inter-canine width, inter-molar width, and arch perimeter were assessed to evaluate post-treatment relapse of mandibular anterior teeth. Little's irregularity index increased in both control and experimental groups, with the control group showing significantly higher relapse rates at T2 and T3 intervals. The relapse observed in the first 4 weeks was attributed to the severity of malocclusion and the time required for gingival and periodontal tissue reorganization. Elastic recoil of trans-septal fibers, which tends to rotate teeth back to their original positions, was also a contributing factor. Jasser et al.[2] highlighted that supracrestal fiberotomy significantly reduces rotational relapse.

Intercanine width changes were clinically insignificant in this study. A small increase during treatment was observed, followed by a return to baseline during the post-retention phase. These findings align with Freitas et al.,[21] who reported similar inter-canine width changes during and after treatment.

Arch perimeter exhibited the greatest difference at T2 in the control group, with values reducing from baseline.[22] The reduction was primarily attributed to mesial migration of mandibular molars and crowding of anterior teeth. These results are consistent with Glenn et al.,[23] who reported arch length decreases in non-extraction cases post-retention due to similar factors.

Inter-molar width showed minimal relapse toward baseline. Previous studies by Glenn et al.[23] and Azizi et al.[24] confirmed that inter-molar width changes were not significantly associated with anterior crowding or proclinations.

Limitations of the Study

It includes the need for a larger sample size, reliance on plaster models for relapse assessment, lack of CBCT analysis for bone density changes, unavailability of trial drug injection sites, and unaccounted bone densities of the study population.

Future Scope of Vitamin D3: in orthodontics includes studies on bone turnover in animals, bone morphometric changes in humans, complete elimination of post-treatment relapse, various applications in orthodontics, histological studies on bone morphometry, and targeted cell culture for bone augmentation. Vitamin D3 is not just a hormone for bones and teeth but a potential boon to orthodontists.

Conclusion

Relapse was seen in both the control and the experimental in the first 4 weeks of the study. Still, the control group showed a greater relapse rate in the following 8th week and 12th week when compared to the experimental group. The severity of malocclusion also influenced the amount of relapse. Inter-canine width, inter-molar width, and the arch perimeter showed changes during the study period but were not significant enough to present clinically.

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