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VITAMIN-D_3 DOES NOT ACCELERATE ORTHODONTIC TOOTH MOVEMENT IN HUMANS. A COMPARATIVE STUDY

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ABSTRACT

Introduction: Prolonged treatment time in orthodontics has been the focus of attention of researchers since decades and many techniques have been suggested for accelerating tooth movement. The role of Vitamin D_3 is among one of such suggested means but results are variables. **Objectives:** The aim of this clinical study was to determine the effect of Vitamin D_3 on the rate and amount of orthodontic tooth movement in humans when injected locally at site. **Materials and Method:** A prospective split mouth clinical trial was carried out on 15 subjects who were indicated for bilateral therapeutic extraction of the first premolars followed by canine retraction in the maxillary arch. Vitamin D_3 in a vehicle of local anaesthetic solution was injected into the buccal vestibule immediately distal to the canine to be retracted on experimental side; whereas on the contralateral side, only local anaesthetic solution was injected as control in the same site on the 7th, 21st & 47th days of canine retraction. The amount of canine retraction was assessed keeping maxillary palatal rugae as stable landmark and the values obtained were compared by pre- and post- canine retraction occlusograms. **Results**: The present study showed that experimental teeth that had received injections containing Vitamin D_3 had moved considerably

lesser than matched control teeth ($p<0.001$). **Conclusions:** The application of vitamin D_3 did not accelerate the orthodontic tooth movement.

KEYWORDS: Vitamin D_3, orthodontic tooth movement, canine retraction, occlusograms, palatal rugae.

INTRODUCTION

One of the primary factors in the orthodontic treatment is treatment time. Several methods both biological and mechanical have been used for accelerating tooth movement. These clinical methods range from *Electro-Magneto-Mechanical devices* (e.g. lasers[1,2], electromagnetic fields[3], direct electrical current[4], mechanical vibrations[5,6]); *Surgical means* (e.g. distraction[7,8], interseptal alveolar surgry[9], corticotomy[10], piezocision technique[11]) to *Pharmacological means* [local or systemic drugs e.g. prostaglandins[12,13], corticosteroids[14], nitro- L-arginine[15], PTH[16], nicotine[17], Vitamin D_3 (cholecalciferol).[18,19] The clinical studies have shown that vitamin D_3 accelerates tooth movement in animals. Therefore, the present study was designed to determine whether the rate and amount of orthodontic tooth movement can be enhanced by the local injection of vitamin D_3 in humans also.

METHODOLOGY

The study was a prospective split mouth clinical trial involving 15 subjects in the age group of 13-25 years who reported to the Department of Orthodontics and Dentofacial Orthopedics requiring fixed orthodontic treatment. Informed consent was obtained from the patients prior to the treatment. The ethical clearance was sought from the local body and approval was obtained from an identified institutional review board. Healthy subjects with no radiographic evidence of periodontal disease (probing depth values less than 3 mm in the whole dentition) and indicated for therapeutic extraction of bilateral maxillary first premolars (Angle's class II division 1 cases, crowding, bimaxillary protrusion) and who have not used anti-inflammatory drugs, steroids or antibiotics over past 3 months; were selected for the study. The exclusion criteria were: Subjects with systemic/bone/metabolic/hormonal disease; hypersensitivity to vitamin D or its analogues/derivatives; subjects who had undergone major surgical procedure or with artificial valves or joints.

Procedure

Each subject's maxillary arch was divided into right-left and randomly allocated as experimental and control sides. The subjects were orthodontically treated using preadjusted

edgewise appliance (0.022" x 0.028" slot, MBT prescription, 3M Unitek, U.S.A). Initial leveling and aligning was carried out using 0.016" Nickel Titanium (Optima, Orthodontic supplies Ltd, Leicestershire, U.K.) and 0.018" round stainless steel (A.J. Wilcock, Whittle Sea, Victoria, Australia) wires. Individual canine retraction was carried out on 0.019" x 0.025" stainless steel wire (Optima, Orthodontic supplies Ltd, Leicestershire, U.K.), using NiTi closed coil springs (Orthoforce G&H wire company, Indiana, U.S.A) placed between the maxillary first molars and canines delivering 150 grams of force following complete healing of the alveolar sockets. The force was calibrated using a Dontrix gauge (Libral traders Pvt. Ltd, New Delhi) (Figure 1 A & B). Anchorage was reinforced using transpalatal arches on the maxillary first molars in all the cases from the alignment stage.

Preparation of the solution

1 ml of commercially available Vitamin D_3 (Arachitol-6l, Solvay Pharma India Pvt. Ltd) containing 15 mg of vitamin D_3 per ml of solution was dissolved in 999 ml of 2% Xylocaine containing Adrenaline 1:200000 (AstraZeneca Pharma India Ltd) to prepare a stock solution having 15 µg (600 IU) of vitamin D_3 per ml of solution for local injection.

Administration of the solution (Figure 1 C)

Experimental side: 1ml of the prepared stock solution was injected into the buccal vestibule at level of distal margin of the root of the canine using a commercially available 2 ml syringe with a, 11/2 inch long 26-gauge needle (Unolok luer lock syringe, Hindustan Syringes &Medical Devices Ltd, New Delhi).

Control side: 1 ml of the control solution of the local anaesthetic was administered at similar site on the distal portion of the contralateral canine.

1ml of the stock solution was injected 3 times during treatment (on the 7th, 21st & 47th days of canine retraction) on both sides.

Data collection

The exact amount of canine retraction was assessed using occlusograms using maxillary palatal rugae as stable landmarks for taking readings for tooth movements before and after 60 days of canine retraction.

Fabrication of occlusograms (Figure 2 A)

The pre- and post-retraction casts were scanned using a flatbed scanning machine (Hewlett-Packard Scanjet G3010) along with two 15 cm metal rulers placed in the x- and y-axes. The magnification error was ascertained with the help of the metal rulers. The scanned images were printed. The printouts of the scans were traced on cellulose acetate paper using a 0.3 mm black lead pencil (Staedtler® Mars micro pencil). Points were marked on the traced images as shown in **Figure 2 B**. The canine distalization was then measured on each post-retraction cast using the digimatic caliper (Mitutoyo Corp, Japan), and the magnification error correction applied.

RESULTS

The collected data was analyzed statistically using the Mann-Whitney U test. The results clearly show that the mean rate of tooth movement on the control side (1.86mm) was greater than the experimental side (1.14mm) for the total observational time period of 60 days ($p<0.001$). Thus, tooth movement was slower on the experimental side as compared to the control side. (**Table 1 and Figure 3**).

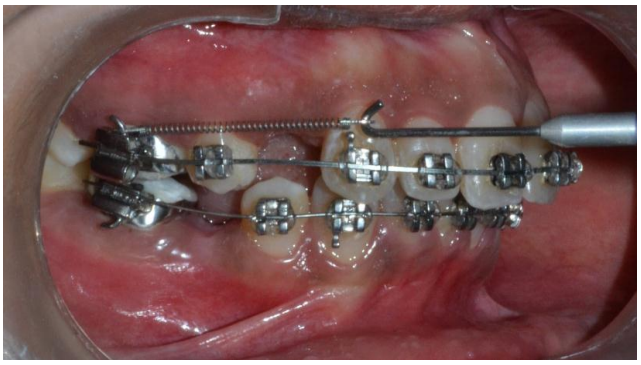

Figure 1(A): Showing calibration of force using Dontrix Gauge.

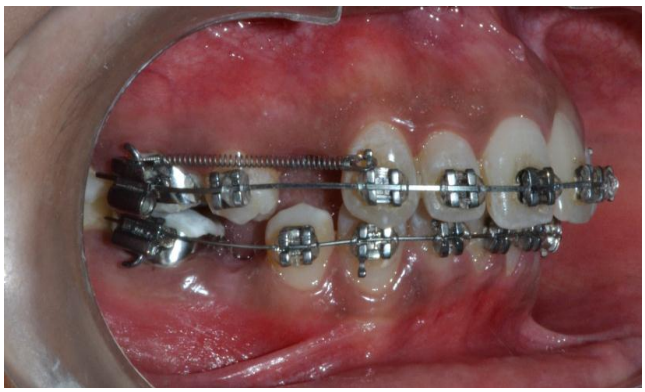

Figure 1(B): Placement of NiTi closed coil spring.

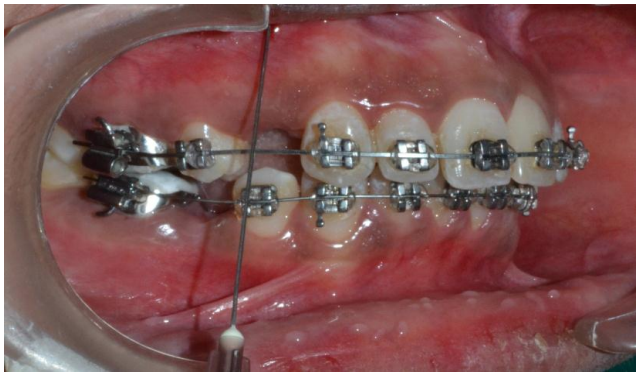

Figure 1(C): Local injection of Vitamin D_3 in the labial vestibule distal to the canine.

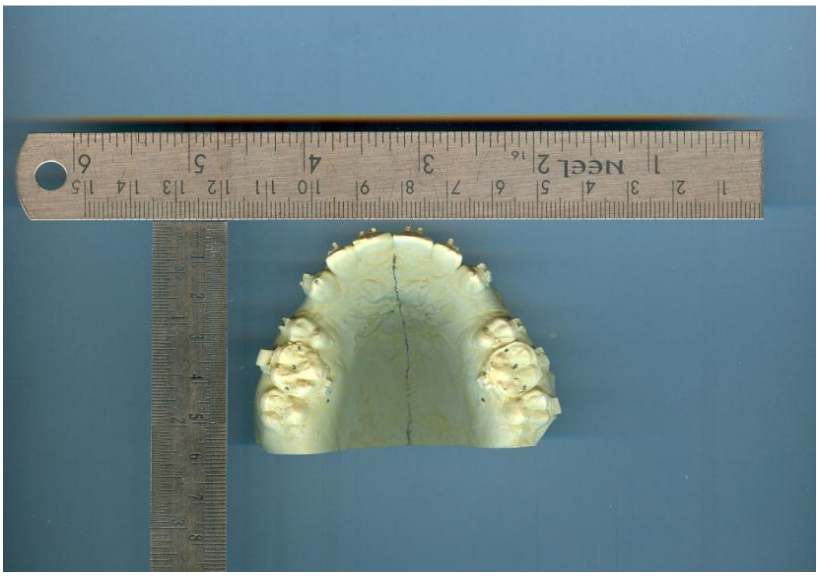

Figure 2(A): Showing Fabrication of occlusograms.

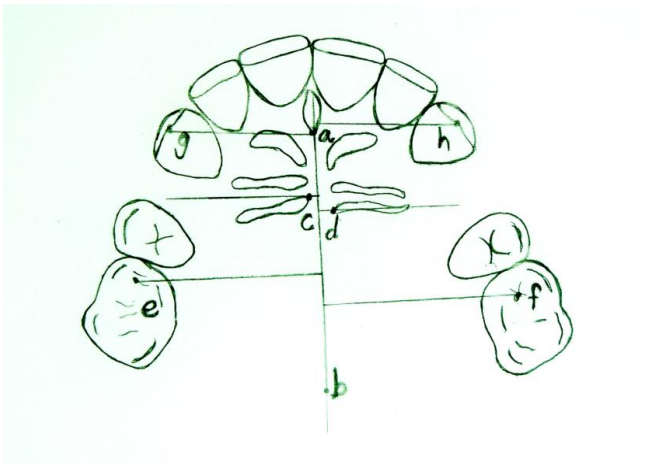

Figure 2 (B) Points were marked on the traced images.

a-b: Mid palatal suture.

c: Medial end of right third palatal ruga.

d: Medial end of left third palatal ruga.

e: Central fossa of maxillary right first permanent molar.

f: Central fossa of maxillary left first permanent molar.

g: Cusp tip of right maxillary canine.

h: Cusp tip of left maxillary canine.

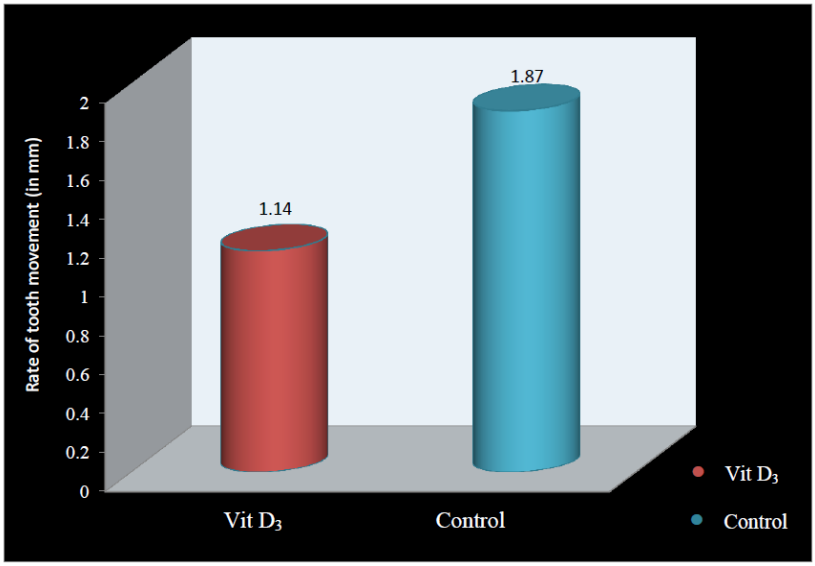

Figure 3: Comparison of canine distalization in vitamin D3 and control groups.

Table 1: Comparison of canine distalization in vitamin D3 and control groups.

Group	Mean	SD	U-value	Z-value	P-value
Vit D_3	1.1427	0.3138	26.0000	-3.5879	**0.0003**
Control	1.8687	0.5228			

In this table, the p value denotes the level of significance as follows

$p > 0.05$ – Not significant, designated as NS.

$p < 0.05$ – Significant (significant at 95% CI), designated as S.

$p < 0.01$ – Highly significant (significant at 99% CI), designated as HS.

$p < 0.001$ –Very highly significant (significant at 99.9% CI), designated as VHS.

DISCUSSION

Orthodontic tooth movement is brought about by alveolar bone and periodontal ligament remodelling (bone resorption on the pressure site and bone formation on the tension site). The two principal cells – osteoblasts and osteoclasts maintain this balance between bone resorption and deposition during remodelling of the alveolar bone. Hence, one of the potent adjuncts used along with biomechanical forces is the one which selectively activate osteoclasts in pressure zone and fasten OTM. Vitamin D (Calcitriol) in its active form 1,25-dihydroxycholecalciferol (1,25D), is one of the known potent stimulators of osteoclastic activity.[20] Therefore, it was tested for its ability to enhance the rate of orthodontically specific resorption and tooth movement. Collins and Sinclair demonstrated that intraligamentary injections of vitamin D metabolite (1,25-dihydroxy-cholecalciferol) resulted in increase in the number of osteoclasts and 60% increase in tooth movement during canine retraction with light forces in cats.[18] Kale and colleagues studied the local applications of PGE_2 and 1,25-dihydroxy-cholecalciferol on the rate of tooth movement in rats and observed 1,25-dihydroxy-cholecalciferol(1,25-DHCC) is more effective in modulating bone turnover as its effects on bone formation and resorption are well-balanced.[19] But the receptors for vitamin D have been demonstrated not only in osteoclast but are present on osteoblasts too. The results of some clinical trials also indicate that vitamin D_3 increases bone mass and is in use for the treatment of osteoporosis.[21,22] Because of this beneficial effect on bone tissue, we can assume this pharmacological agent can inhibit orthodontic movement. Kawakami observed an increase in the mineral appositional rate on alveolar bone after orthodontic force application and they suggested that local application of vitamin D could intensify the re-establishment of supporting alveolar bone, after orthodontic treatment.[23] This creates confusion if Vitamin D_3 has role in bone resorption or in deposition/mineralization around tooth under orthodontic movement so this was kept main objective in present clinical study. Secondly till now all previous trials were carried out in animals so present study was bound to check the effects of vitamin D_3 in human beings. The result of this clinical study shows that local application of vitamin D_3 produces a significantly decreased amount of tooth movement after 60-day experimental period, contrary to results obtained by animal studies done previously and no obvious local or systemic clinical side effects were noted. It is hypothesized that the injection of vitamin D_3 might have resulted in the stimulation of osteoblasts leading to bone deposition on the distal surface of the canine, thus slowing down the movement of the same. Similar effect on bone like mineralization of osteoid, increases in bone mineral density measurements and reduced fracture rates is seen in patients with vitamin D deficiency when replenished with vitamin D. The action of vitamin D3 is not

isolated but also affected by other calcium-regulatory hormones, such as thyroxine, parathormone and calcitonin.

Our study had few limitations: although NiTi coil springs were used in the present study for optimum force delivery on 0.019" x 0.025" stainless steel wire for translation of the canines, the canines may have tipped and this tipping movement might have been measured on the occlusograms. This was a short term study carried out only for 60 days. Genetic and environmental factors may have influenced the response of the canines which was not counted. The intra-ligamentous route of delivery was avoided because of the pain factor involved. However, that would have been a more accurate method of drug delivery (closer and more local to the area of interest). Molecular level study can also help in explaining these contrary results of this study. Nonetheless, for future scope larger sample size with long term evaluation is required with varied doses of vitamin D_3. Furthermore, GCF can be studied to determine the changes at the cellular level. Also, studies can be performed to determine the role of the same solution in periodontally compromised orthodontic cases which might open up new avenues in the treatment of periodontally compromised cases. Its potential application for anchorage augmentation may be another exciting avenue in orthodontics.

CONCLUSION

Localized injections of vitamin D3 produced a significantly decreased amount of tooth movement after a 60-day experimental period, contrary to results cited in the past literature.

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