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Clinical evaluation of the effect of vitamin D supplementation on the outcomes of non- surgical periodontal therapy in post- menopausal women with gingivitis: An interventional study

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ABSTRACT

Gingivitis is the inflammation of gingiva and a milder form of periodontal disease. Studies have shown that Vitamin D supplementation may reduce susceptibility to gingival inflammation through its anti-inflammatory effect by inhibiting antigen induced T cell proliferation and cytokine production. The aim of the study is to clinically evaluate the effect of vitamin D supplementation on the outcomes of non- surgical periodontal therapy in post-menopausal women with gingivitis. Total of 20 post-menopausal women visiting the department of Periodontology, D.A.P.M.R.V Dental College, were included in the study, with gingivitis (GI score >1). Out of which 10 were on vitamin D supplements (>400 IU/day) since one year and 10 subjects were not on any vitamin D supplements. Gingival, plaque and bleeding indices were recorded at baseline, 1, 2 and 3 months interval post scaling. On statistical analysis, there was significant improvement in all the three clinical parameters within the groups at different intervals (P<0.05) but however the difference in improvement (P>0.05) was not statistically significant between the groups.

Key words: Gingivitis, Vitamin D, Post- menopausal women, Non- Surgical therapy.

INTRODUCTION

Vitamin D plays a role in several physiological processes, including bone and calcium metabolism, cellular growth and differentiation, immunity and cardiovascular function.[1] In tradition, vitamin D has been associated with bone health and it is well-understood that vitamin D deficiency leads to rickets in children and osteomalacia/osteoporosis in adults.[2] However, it is now known that adequate vitamin D is important for optimal functioning of many organs and tissues throughout the body. [3] Vitamin D deficiency or insufficiency is prevalent in practically every segment of the population, including children and adults especially in post-menopausal women. [4]This world-wide pandemic remains generally unrecognized and untreated. Developing data indicate that vitamin D deficiency in addition to playing a significant role in the genesis of coronary risk factors also pre-disposes to hypertension, diabetes, the metabolic syndrome, left ventricular hypertrophy, congestive heart failure, and chronic vascular inflammation. Vitamin D is classified as a secosteroid in which one of the rings has been broken by ultraviolet B (UVB) sunlight and the main source of vitamin D is de novo synthesis in the skin.[5] Although vitamin D is consumed in food, dietary intake alone is often insufficient, supplying only 20% of the body's requirements.

The potential for extra skeletal effects of vitamin D arose with the discovery of thevitamin D receptor(VDR) on tissues with no involvement in calcium homeostasis (i.e. skin, placenta, pancreas, prostate and colon cancer cells, and activated T and B cells). [5]In recent years, the discovery of the VDR in the cells of the immune system and the fact that several of these cells produce the vitamin D hormone suggested that it could have immuno-regulatory properties. [6]

Antigen- presenting cells, including macrophages and dendritic cells, express the vitamin D activating enzyme 1a-hydroxylase (also known as CYP27B1). Thus, these cells can convert precursor 25(OH)D3, the major circulating form of vitamin D, to active 1,25-(OH)2D3. In this way these cells can induce responses in cells by binding their VDR and promoting transcriptional regulation. This autocrine mechanism is essential to two key features of immune function, namely innate antibacterial activity and presentation of antigen to cells of the adaptive immune response, such as activated T lymphocytes. [7]

Periodontal disease is an inflammatory condition, characterized by alveolar bone loss induced by the host immune response to bacterial attack. As vitamin D deficiency is associated with some inflammatory diseases and plays a significant role in bone homeostasis and immunity, vitamin D deficiency could negatively affect the periodontium and increase tooth loss. [8]

Menopause, which means 'without estrogen', is the time at which cyclic ovarian function or menstruation ceases. The majority of women experience menopause stage between the ages of 47 and 55 years when the production of estrogen decreases.[9] The mechanisms involved in this influence are not completely understood, but it is thought to be related to the action of estradiol on the connective tissue.[10]The menopause triggers a wide range of changes in women's body, including oral cavity Absence of ovarian sex steroids has been associated with worsening in gingival health. [11]Post –menopausal women reported to have increased gingivitis, periodontal disease, tooth loss and dry mouth.[12] The deficiency in hormones may alter immunologic factors and responses, including antigen expression and presentation, and cytokine production, as well as the expression of apoptotic factors, and cell death.[13]

In a study it was concluded that Vitamin D supplementation may reduce susceptibility to gingival inflammation through its anti-inflammatory effect by inhibiting antigen induced T cell proliferation and cytokine production, [14]at the same time, it is unknown whether vitamin D exerts anti-inflammatory effects relevant to human disease. Average vitamin D and calcium intakes in the general population are below current recommendations of 400 to 600 IU and 1,000 to 1,200 mg daily, respectively. [15]Although there is a growing consensus that such daily targets are inadequate and higher vitamin D intakes (800 to 1,000 IU daily) are now recommended by professional organizations. [16]The present study aims to clinically evaluate the effect of vitamin D supplementation on the outcomes of non- surgical periodontal therapy in post-menopausal women with gingivitis.

EXPERIMENTAL SECTION

Materials and methods

A clinical study was conducted with subjects who were recruited from D A P M R V Dental College and Hospital, Bangalore, India. Ethical clearance was obtained for the study and all subjects signed informed consent documents for participation in the study.

Inclusion criteria

- Total of 20 post-menopausal women between 45 to 60 years were included in the study, with gingivitis (GI score >1).
- Out of which 10 were on vitamin D supplements (>400 IU/day) since one year and 10 subjects were not on any vitamin D supplements.

Exclusion criteria

- Patient who has undergone any sort of periodontal therapy within last one year.
- History of any systemic diseases.
- History of diseases or conditions use of medications that might affect periodontal health.
- History of use of medications that might affect bone and mineral metabolism.
- Treatment with oestrogen within the last 6 months.
- Treatment with bisphosphonates in the past 12 months or lifetime exposure to bisphosphonates for >3 years.

- Patients who are under antibiotics past last 6 months.

Clinical assessment

The following clinical parameters were recorded, plaque index (PI) (Silness.P and Loe H 1964), gingival index (GI) (Loe H and Silness.P 1963), and bleeding on probing(SBI) (Muhlemann H.R and Son S 1971).All the three indices (GI, PI, and BOP) are measured and recorded at baseline. Thorough scaling is performed and same clinical parameters were reassessed in all the 20 subjects at 1 month, 2 months and 3 months interval. All the values at each time are recorded on appropriate forms during assessment.

Statistical analysis

Statistical test used is t- test/Mann-Whitney test Null Hypothesis: There is no significant difference in the mean value between two groups i.e. $\mu_1=\mu_2$. Alternate Hypothesis: There is a significant difference in the mean value between two groups i.e. $\mu_1\neq\mu_2$.Level of Significance: $\alpha=0.05$. Decision Criterion: We compare the P-Value with the level of significance. If $P<0.05$, we reject the null hypothesis and accept the alternate hypothesis. If $P\geq0.05$, we accept the null hypothesis.

RESULTS AND DISCUSSION

The results of the study are as follows:

Comparison of PI within Group 1 between two time intervals:

Comparison of PI within Group 1 between two time intervals has been shown in Table 1. The plaque index scores at baseline, 1 month, 2 months and 3 months were 1.70 ± 0.63, 1.43 ± 0.50, 1.25 ± 0.51,1.03 ± 0.41 respectively. The mean difference in the values between baseline & 1 month, baseline & 2 months and baseline & 3 months were 0.269, 0.442 and 0.662 respectively. The mean difference in the values between 1 month & 2 months, 1 month & 3 months and 2 months & 3 months were 0.173, 0.393 and 0.220 respectively. The difference in mean PI was found to be statistically significant between all the time intervals.

Comparison of GI within Group 1 between two time intervals: Paired t-test)

Comparison of GI within Group 1 between two time intervals has been shown in Table 2. The gingival index scores at baseline, 1 month, 2 months and 3 months were 1.72 ± 0.66, 1.58 ± 0.55, 1.49 ± 0.47,1.36 ± 0.49 respectively. The mean difference in the values between baseline & 1 month, baseline & 2 months and baseline & 3 months were 0.145, 0.233and 0.361respectively. The mean difference in the values between 1 month & 2 months, 1 month & 3 months and 2 months & 3 months were 0.088, 0.216and 0.128respectively. The difference in mean GI was found to be statistically significant between all the time intervals except between baseline and 1 month.

Comparison of SBI within Group 1 between two time intervals: (Wilcoxon Signed Ranks test)

Comparison of SBI within Group 1 between two time intervals has been shown in Table 3. The gingival index scores at baseline, 1 month, 2 months and 3 months were 1.50 ± 1.08, 1.30 ± 0.95, 1.30 ± 0.48, 0.50 ± 0.53 respectively. The mean difference in the values between baseline & 1 month, baseline & 2 months and baseline & 3 months were 0.200, 0.200 and 1.000 respectively. The mean difference in the values between 1 month & 2 months, 1 month & 3 months and 2 months & 3 months were 0.000, 0.800 and 0.800 respectively. The difference in mean SBI was found to be statistically significant between baseline & 3 months ($P<0.05$), 1 month & 3 months ($P<0.05$) as well as between 2 months & 3 months ($P<0.01$).

Comparison of PI within Group 2 between two time intervals: (Paired t-test)

Comparison of PI within Group 2 between two time intervals has been shown in Table 4. The plaque index scores at baseline, 1 month, 2 months and 3 months were 1.66 ± 0.66, 1.58 ± 0.63, 1.46 ± 0.62,1.28 ± 0.61 respectively. The mean difference in the values between baseline & 1month, baseline & 2 months and baseline & 3 months were 0.078, 0.197 and 0.379 respectively. The mean difference in the values between 1 month & 2 months, 1 month & 3 months and 2 months & 3 months were 0.119, 0.301and 0.182 respectively. The difference in mean PI was found to be statistically significant between all the time intervals.

TABLE: 1Comparison of PI within Group 1 between two time intervals

Time Interval	Mean	Std Dev	SE of Mean	Mean Difference	t	P-Value
Baseline	1.70	0.63	0.20	0.269	5.763	<0.001*
1 Month	1.43	0.50	0.16			
Baseline	1.70	0.63	0.20	0.442	8.131	<0.001*
2 Months	1.25	0.51	0.16			
Baseline	1.70	0.63	0.20	0.662	6.237	<0.001*
3 Months	1.03	0.41	0.13			
1 Month	1.43	0.50	0.16	0.173	4.691	0.001*
2 Months	1.25	0.51	0.16			
1 Month	1.43	0.50	0.16	0.393	5.121	0.001*
3 Months	1.03	0.41	0.13			
2 Months	1.25	0.51	0.16	0.220	2.588	0.029*
3 Months	1.03	0.41	0.13			

**Denotes significant difference*

TABLE: 2Comparison of GI within Group 1 between two time intervals:

Time Interval	Mean	Std Dev	SE of Mean	Mean Difference	t	P-Value
Baseline	1.72	0.66	0.21	0.145	2.121	0.063
1 Month	1.58	0.55	0.17			
Baseline	1.72	0.66	0.21	0.233	2.938	0.017*
2 Months	1.49	0.47	0.15			
Baseline	1.72	0.66	0.21	0.361	4.346	0.002*
3 Months	1.36	0.49	0.16			
1 Month	1.58	0.55	0.17	0.088	2.927	0.017*
2 Months	1.49	0.47	0.15			
1 Month	1.58	0.55	0.17	0.216	7.541	<0.001*
3 Months	1.36	0.49	0.16			
2 Months	1.49	0.47	0.15	0.128	8.085	<0.001*
3 Months	1.36	0.49	0.16			

**Denotes significant difference*

TABLE: 3 Comparison of SBI within Group 1 between two time intervals

Time Interval	Mean	Std Dev	SE of Mean	Mean Difference	Z	P-Value
Baseline	1.50	1.08	0.34	0.200	-1.414	0.157
1 Month	1.30	0.95	0.30			
Baseline	1.50	1.08	0.34	0.200	-0.816	0.414
2 Months	1.30	0.48	0.15			
Baseline	1.50	1.08	0.34	1.000	-2.428	0.015*
3 Months	0.50	0.53	0.17			
1 Month	1.30	0.95	0.30	0.000	0.000	1.000
2 Months	1.30	0.48	0.15			
1 Month	1.30	0.95	0.30	0.800	-2.530	0.011*
3 Months	0.50	0.53	0.17			
2 Months	1.30	0.48	0.15	0.800	-2.828	0.005*
3 Months	0.50	0.53	0.17			

**Denotes significant difference*

TABLE: 4Comparison of PI within Group 2 between two time intervals

Time Interval	Mean	Std Dev	SE of Mean	Mean Difference	t	P-Value
Baseline	1.66	0.66	0.21	0.078	4.275	0.002*
1 Month	1.58	0.63	0.20			
Baseline	1.66	0.66	0.21	0.197	5.687	<0.001*
2 Months	1.46	0.62	0.20			
Baseline	1.66	0.66	0.21	0.379	7.387	<0.001*
3 Months	1.28	0.61	0.19			
1 Month	1.58	0.63	0.20	0.119	5.305	<0.001*
2 Months	1.46	0.62	0.20			
1 Month	1.58	0.63	0.20	0.301	8.132	<0.001*
3 Months	1.28	0.61	0.19			
2 Months	1.46	0.62	0.20	0.182	7.634	<0.001*
3 Months	1.28	0.61	0.19			

**Denotes significant difference*

TABLE: 5Comparison of GI within Group 2 between two time intervals

Time Interval	Mean	Std Dev	SE of Mean	Mean Difference	t	P-Value
Baseline	1.65	0.70	0.22	0.063	3.427	0.008*
1 Month	1.59	0.68	0.21			
Baseline	1.65	0.70	0.22	0.119	3.476	0.007*
2 Months	1.53	0.65	0.20			
Baseline	1.65	0.70	0.22	0.185	4.519	0.001*
3 Months	1.47	0.66	0.21			
1 Month	1.59	0.68	0.21	0.056	2.783	0.021*
2 Months	1.53	0.65	0.20			
1 Month	1.59	0.68	0.21	0.122	4.833	0.001*
3 Months	1.47	0.66	0.21			
2 Months	1.53	0.65	0.20	0.066	4.468	0.002*
3 Months	1.47	0.66	0.21			

**Denotes significant difference*

TABLE: 6Comparison of SBI within Group 2 between two time intervals

Time Interval	Mean	Std Dev	SE of Mean	Mean Difference	Z	P-Value
Baseline	1.50	1.08	0.34	0.300	-1.342	0.180
1 Month	1.20	0.79	0.25			
Baseline	1.50	1.08	0.34	0.600	-1.730	0.084
2 Months	0.90	0.32	0.10			
Baseline	1.50	1.08	0.34	1.000	-2.332	0.020*
3 Months	0.50	0.53	0.17			
1 Month	1.20	0.79	0.25	0.300	-1.342	0.180
2 Months	0.90	0.32	0.10			
1 Month	1.20	0.79	0.25	0.700	-2.333	0.020*
3 Months	0.50	0.53	0.17			
2 Months	0.90	0.32	0.10	0.400	-2.000	0.046*
3 Months	0.50	0.53	0.17			

**Denotes significant difference*

TABLE: 7Comparison of PI between the two groups

Time Interval	Group	Mean	Std Dev	SE of Mean	Mean Difference	t	P-Value
Baseline	Group I	1.70	0.63	0.20	0.040	0.138	0.892
	Group 2	1.66	0.66	0.21			
1 Month	Group I	1.43	0.50	0.16	-0.151	-0.596	0.558
	Group 2	1.58	0.63	0.20			
2 Months	Group I	1.25	0.51	0.16	-0.205	-0.808	0.430
	Group 2	1.46	0.62	0.20			
3 Months	Group I	1.03	0.41	0.13	-0.243	-1.040	0.312
	Group 2	1.28	0.61	0.19			

TABLE: 8 Comparison of GI between the two groups

Time Interval	Group	Mean	Std Dev	SE of Mean	Mean Difference	t	P-Value
Baseline	Group I	1.72	0.66	0.21	0.070	0.231	0.820
	Group 2	1.65	0.70	0.22			
1 Month	Group I	1.58	0.55	0.17	-0.012	-0.043	0.966
	Group 2	1.59	0.68	0.21			
2 Months	Group I	1.49	0.47	0.15	-0.044	-0.174	0.864
	Group 2	1.53	0.65	0.20			
3 Months	Group I	1.36	0.49	0.16	-0.106	-0.409	0.688
	Group 2	1.47	0.66	0.21			

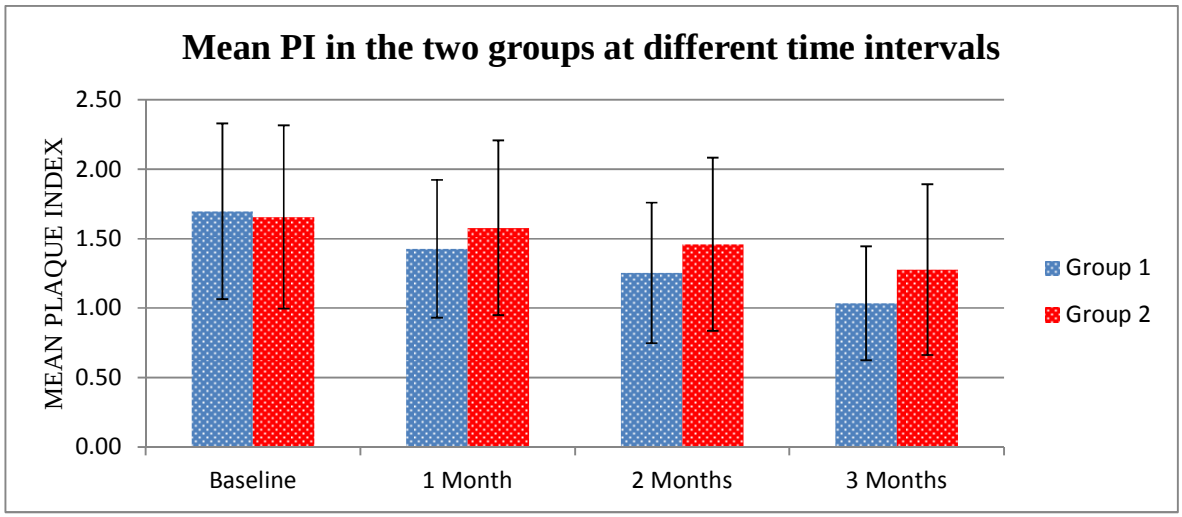
Comparison of GI within Group 2 between two time intervals:

Comparison of GI within Group 2 between two time intervals has been shown in Table 5. The gingival index scores at baseline, 1 month, 2 months and 3 months were 1.65 ± 0.70, 1.59 ±0.68, 1.53 ± 0.65, 1.47 ± 0.66 respectively. The mean difference in between the values between baseline & 1 month, baseline & 2 months and baseline & 3 months were 0.063, 0.119and 0.185 respectively. The mean difference in the values between 1 month & 2 months, 1 month & 3 months and 2 months & 3 months were 0.056, 0.122 and 0.066 respectively. The difference in mean GI was found to be statistically significant between all the time intervals.

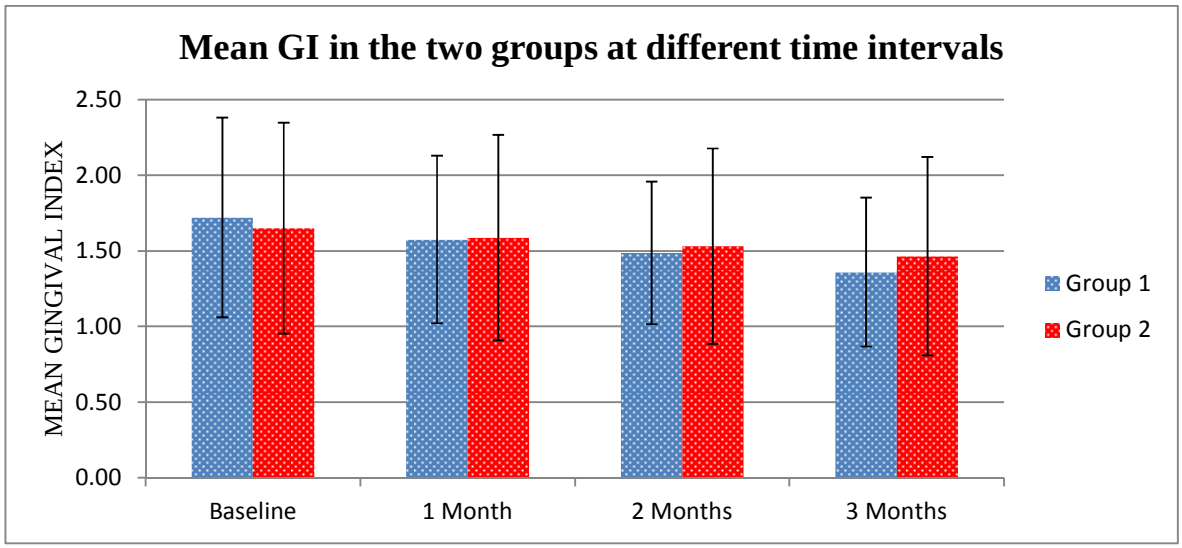
TABLE: 9 Comparison of SBI between the two groups

Time Interval	Group	Mean	Std Dev	SE of Mean	Mean Difference	Z	P-Value
Baseline	Group I	1.50	1.08	0.34	0.000	1.000	1.000
	Group 2	1.50	1.08	0.34			
1 Month	Group I	1.30	0.95	0.30	0.100	0.872	0.912
	Group 2	1.20	0.79	0.25			
2 Months	Group I	1.30	0.48	0.15	0.400	0.045	0.165
	Group 2	0.90	0.32	0.10			
3 Months	Group I	0.50	0.53	0.17	0.000	1.000	1.000
	Group 2	0.50	0.53	0.17			

GRAPH: 1



GRAPH: 2

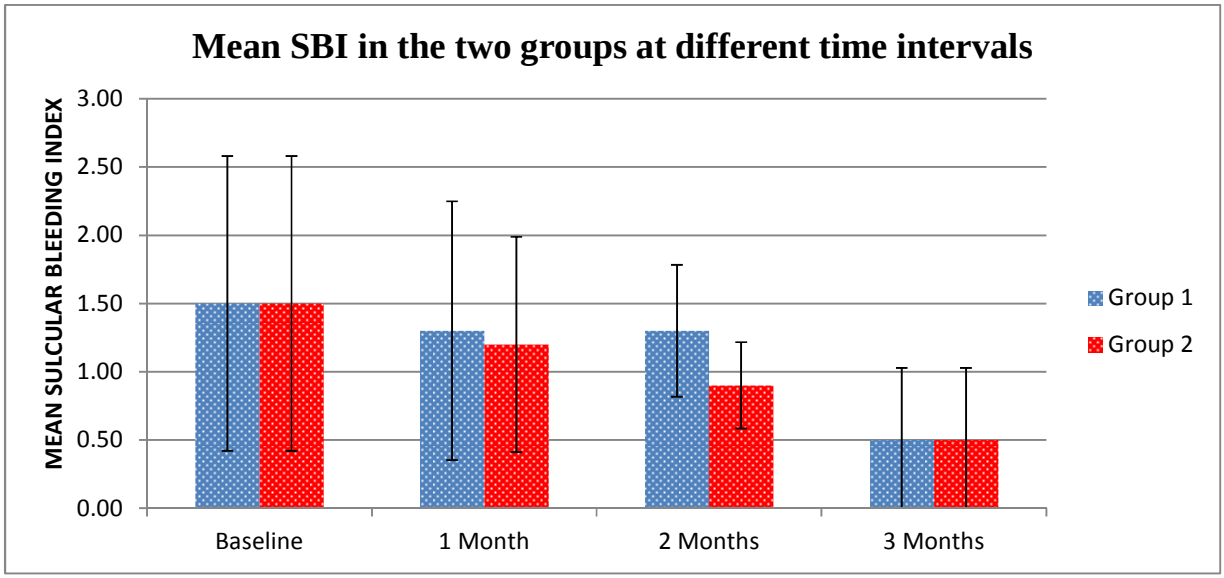


Comparison of SBI within Group 2 between two time intervals:

Comparison of SBI within Group 2 between two time intervals has been shown in Table 6. The gingival index scores at baseline, 1 month, 2 months and 3 months were 1.50 ± 1.08, 1.20 ± 0.79, 0.90 ± 0.32, 0.50 ± 0.53 respectively. The mean difference in the values between baseline & 1 month, baseline & 2 months and baseline & 3 months were 0.300, 0.600 and 1.000 respectively. The mean difference in the values between 1 month & 2 months, 1 month & 3 months and 2 months & 3 months were 0.300, 0.700 and 0.400 respectively. The difference in mean

SBI was found to be statistically significant between all the time intervals except baseline and 1 month &1 monthand months & 3 months (P<0.05).

GRAPH: 3



Comparison of PI between the two groups: (t-test)
Comparison of PI scores between two groups between different time intervals has been shown in Table 7, Graph 1. In Group I the scores were 1.70± 0.63 at baseline, 1.43± 0.50 at 1 month, 1.25 ±0.51 at 2 months, 1.03± 0.41 at 3 months and in Group II the scores were 1.66± 0.66 at baseline, 1.58± 0.63 at 1 month, 1.46±0.62 at 2 months, 1.28± 0.61 at 3 months. The mean difference in the values between two groups at baseline, 1 month, 2 months and three months were 0.040, -0.151, -0.205, -0.243 respectively. No significant difference was observed between the two groups at any of the time intervals with respect to mean PI (P>0.05).

Comparison of GI between the two groups: (t-test)
Comparison of GI scores between two groups between different time intervals has been shown in Table 8, Graph 2. In Group I the scores were 1.72± 0.66 at baseline, 1.58 ± 0.55 at 1 month, 1.49 ± 0.47 at 2 months, 1.36± 0.49 at 3 months and in Group II the scores were 1.65

± 0.70 at baseline, 1.59± 0.68 at 1 month, 1.47±0.66 at 2 months, 1.53± 0.65 at 3 months. The mean difference in the values between two groups at baseline, 1 month, 2 months and three months were 0.070, -0.012, -0.044, -0.106respectively. No significant difference is observed between the two groups at any of the time intervals with respect to mean GI (P>0.05).

Comparison of SBI between the two groups: (Mann-Whitney test)
Comparison of SBI scores between two groups between different time intervals has been shown in Table 9, Graph 3. In Group I the scores were 1.50± 1.08 at baseline, 1.30± 0.95 at 1 month, 1.30 ± 0.48 at 2 months, 0.50± 0.53 at 3 months and in Group II the scores were 1.50± 1.08 at baseline, 1.20 ± 0.79 at 1 month, 0.90±0.32 at 2 months, 0.50± 0.53 at 3 months. The mean difference in the values between two groups at baseline, 1 month, 2 months and three months were 0.000, 0.100, 0.400, and 0.000respectively. No significant difference is observed between the two groups at any of the time intervals with respect to mean SBI (P>0.05).

Gingivitis is the inflammation of gingiva and a milder form of periodontal disease. Studies have shown that Vitamin D supplementation may reduce susceptibility to gingival inflammation through its anti-inflammatory effect by inhibiting antigen induced T cell proliferation and cytokine production.[14] Vitamin D plays an important role in calcium homeostasis, promoting calcium absorption in the intestine and stimulating osteoblasts to enable normal bone growth and preservation. The immunomodulatory effect of vitamin D has been linked to modulation of

bacterial-mediated infections, with low levels of vitamin D being associated with an increased risk of infectious diseases. [17]Very few studies have been reported to evaluate the anti-inflammatory effects of vitamin D on gingival status improvement.

In a study conducted by Bashutski et al was shown that Vitamin D deficiency has negative effects on periodontal therapy outcomes for up to 1 year. [18]

In a study by Krallbat al it was shown that post –menopausal women have increased gingival inflammation, periodontal disease, tooth loss and dry mouth. [12]

In an another study by Huber et al he concluded that the deficiency in hormones in post-menopausal women may alter immunologic factors and responses, including antigen expression and presentation, and cytokine production, as well as the expression of apoptotic factors, and cell death.[13] Considering all the above mentioned concepts proposed by various authors , our study aimed to evaluate the effects of vitamin D supplementation on outcomes of non- surgical periodontal therapy in post-menopausal women with gingivitis.

The periodontal parameters assessed in this study were plaque index (PI), gingival index (GI) and sulcular bleeding index (SBI). Total of 20 subjects between 45 to 60 years were included in the study, with gingivitis (GI score >1) as the GI is based on two of the characteristic signs of inflammation-swelling (edema) and redness. According to the GI, the appearance of induced bleeding constitutes a worsening of the early symptoms. [19]The plaque index and Gingival Index have been used in several studies for evaluation of oral hygiene.[20, 21] Bleeding is the first sign to appear and it is the determining factor in the SBI. [19]

Antimicrobial defense of the gingival epithelium involves the recognition of microbes by cell surface receptors such as TLRs, which leads to the induction of host defense genes, such as those encoding defensins, cathelicidins, and cytokines. [22]It has been reported that the production of cathelicidins and defensins against infection in our body is dependent on sufficient circulating levels of vitamin D and 1,25(OH)2D3.The ability of vitamin D to induce these anti- microbial agents has shown to strengthen the physical barrier in the oral cavity may contribute significantly to the improvement of oral health. [22]

In the present study the intra- group comparison showed statistically significant improvement in plaque index, gingival index and sulcular bleeding index at all the time intervals (P value= <0.05).Non- surgical periodontal therapy refers to the conventional and conservative way of removing supra and sub- gingival bacterial plaque and calculus. The goal of this therapy is to establish and maintain healthy oral tissues by eliminating irritants from the surface and root of the tooth that promote plaque retention. In the present study the effect of vitamin D supplementation on the outcomes of non- surgical periodontal therapy in post-menopausal women with gingivitis has been evaluated.

In inter group comparison, Group I had a modest positive effect on gingival health when compared to Group II but the results were not statistically significant (P value= >0.05).The results of the present study fairly concurrent with the study done by Thomas et al. Thomas et al concluded that increased serum concentrations of vitamin D may be beneficial in regard to gingivitis susceptibility. This inverse association may be due to the anti-inflammatory effect of vitamin D. [14]

In a cross-sectional study of 116 subjects, serum concentrations of 25(OH) D were negatively correlated with serum concentrations of C-reactive protein. In a subsample of 24 patients from that study, vitamin D supplementation significantly reduced serum concentrations of C-reactive protein by 23%.This indicates the inverse association of inflammation and serum 25(OH)D levels. [23]

A study by Catherine A Peterson et al showed that Serum tumor necrosis factor alpha concentrations are negatively correlated with serum vitamin D concentrations in healthy women.[24]

In summary, as periodontal disease is associated with the adherence and colonization of pathogenic bacteria at the gingival epithelium, followed by the inflammation that occurs in response to microbial invasion. Prevention of colonization with direct antimicrobial activity, as well as an enhancement of the natural innate immune response, may have a profound effect. Studies have shown that Vitamin D possess anti- inflammatory, anti- microbial and

immuno-regulatory properties by inhibiting antigen induced T- cell proliferation and cytokine production by production of cathelicidins and defensins against infection from gingival epithelium respectively. [14, 19] Thus, in the present study the modest positive correlation between Vitamin D and inflammation reduction could be attributed to the above reasons.

Further studies are required to unravel the exact role of vitamin D supplementation on outcomes of non- surgical periodontal therapy in post-menopausal women.

CONCLUSION

Vitamin D deficiency is associated with some inflammatory diseases and plays a significant role in bone homeostasis and immunity. Post- menopausal women are known to have increased gingivitis and other oral problems due to lack of oestrogen. Hence the present study was conducted to clinically evaluate the effect of vitamin D supplementation on the outcomes of non- surgical periodontal therapy in post-menopausal women with gingivitis. However the difference between two groups was not statistically significant. Further long term interventional studies are required with larger sample size to arrive at a definitive conclusion.

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