IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 19, Issue 12 Ser.12 (December. 2020), PP 06-11
www.iosrjournals.org

To Evaluate And To Assess The Relationship Between Vitamin - D & Chronic Periodontitis A Clinico Biochemical Study.

Syed Owais Ahmed[1], Praveen B Kudva[2]
[1](*Department of periodontology & implantology, Sri Siddhartha Dental college & hospital, Sri Siddhartha Academy Of Higher Education(SSAHE),Tumkur, Karnataka, india).*
[2](*Department of periodontology & implantology, Sri Siddhartha Dental college & hospital, Sri Siddhartha Academy Of Higher Education(SSAHE),Tumkur, Karnataka, india).*

ABSTRACT.
INTRODUCTION
Chronic Periodontitis is an multifactorial, immuno inflammatory disorder which is initiated by the accumulation of periodontal pathogens in dental plaque. When plaque bacteria invades the gingival tissue it evokes an immune inflammatory response by stimulating the host response which results in the destruction of periodontal apparatus in the susceptible individuals. Vitamin D plays an vital role in bone metabolism and homeostasis. Additionally vitamin D also has immunomodulatory and anti inflammatory properties. Hence there is an rationale that vitamin D deficiency could progress the rate of periodontal destruction. This study was done to evaluate if any relationship exist between vitamin D deficiency and periodontal disease and to find out anti inflammatory properties of vitamin D.
MATERIALS & METHODS.
A total of 60 subjects were selected and categorized into 3 groups 1.clinically healthy 2. Gingivitis & 3.periodontitis. the periodontal status of patients in all the 3 groups was assessed by gingival index(loe and sillness 1964) , pocket probing depth using (UNC15 PROBE). Subjects in all the 3 groups were biochemically tested for serum vitamin D3 level and serum CRP level..
RESULT
The results obtained from this study revealed that vitamin D level is decreased in the periodontitis group and also revealed that there is inverse relationship between the mean vitamin D3 level and mean serum CRP level which showed the anti inflammatory effect of vitamin D.
CONCLUSION.
Deficiency of vitamin D might be an risk factor for progression of periodontal diseases. Hence further studies with larger sample size, larger duration of follow up and with more advance invasive procedures, may help in studying relationship between vitamin d and Periodontitis with more accuracy and precision.
KEYWORDS: *C-Reactive Protein, Vitamin D, Immuno Inflammatory*

Date of Submission: 10-12-2020 Date of Acceptance: 25-12-2020

I. Introduction.

Vitamin D is an fat soluble vitamin , whose genesis is from the skin, liver & kidneys. The vitamin D which is obtained from the diet and absorbed from intestine is also plays an vital role.1. Vitamin D crucial for wide variety of organ system, 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. The primary role of vitamin D is to maintain calcium equilibrium in the body it also plays an vital role in modulating immune response in chronic inflammatory disorder thus suppressing the excessive damage caused by the prolonged inflammation. [2]..Recent advancements have proved that biologically active form of vitamin D (1,25(oH)2 D) down regulates the pro inflammatory cytokines production and proliferation of the inflammatory cells, which adds to its anti inflammatory properties. Hence its deficiency can affect the periodontium both by lowering the bone mineral density and through the modulated inflammatory response.[3,4].Numerous recently executed studies has proved that vitamin D deficiency influences the progression of periodontal disease.[5-11]. Periodontitis is an chronic immune inflammatory disorder ,which is multi factorial, and initiated by the accumulation and maturation of the dental plaque. Endotoxins produced by the Plaque bacteria enters the gingival circulation and elicits an host immune response which causes destruction of periodontium in the susceptible host [12,13]Chronic infections, such as periodontitis, can evoke an acute-phase response,

increasing CRP levels[14,15]. Hence in this study in addition to evaluating relationship between vitamin d and periodontal disease we will also assess the anti inflammatory effect of vitamin D by correlating the serum Vitamin d3 level and serum CRP level.

II. Materials And Methods.

A total of 60 patient reporting to department of Periodontology and implantology were screened for their periodontal status by gingival index (loe and sillness1964) and pocket probing depth using UNC 15 probe.

Selection Criteria:-

Exclusion criteria:- Patient with systemic disorders, pregnant and lactating mothers, patients on long term medication (steroids and antibiotics),patients on vitamin D or calcium Supplements, smokers, patients who underwent periodontal therapy within previous 3 months were excluded from study.

Inclusion Criteria:- Patients without any systemic disorders.

60 subjects were selected based upon the above mentioned criteria and categorize into 3groups.
1 Clinically healthy
2 Gingivitis
3 Periodontitis.

Subject in all the 3groups were biochemically tested for serum vitamin D3 level and to assess its anti inflammatory properties ,serum CRP level was assessed. In this study serum vitamin D3 level <20ng/ml is considered as deficient & serum CRP < 3mg/L is considered normal The study protocol was approved by the institutional ethical community of Jaipur Dental College.

STATISTICAL ANALYSIS.

statistical analyses were done with Statistical Package for Social Sciences version 15.0 (SPSS Inc., Chicago, IL, USA) software package..One way ANOVA test was used for comparison between means of groups and to determine the significance of each parameter of the study. Multivariate Tukey honest significant difference test was used to evaluate the relationship between Mean vitamin D scores and Mean serum CRP level . Pearson's correlation coefficient was used to correlate between various parameters.

III. Results.

The results obtained in this study showed that mean PPD in group 1, group 2, group 3 were, 2.9500, 2.9500, 5.6500 respectively [table 1&2] and the difference among them was statistically significant at $p<0.05$. the mean GI in group 1,2 & 3 are 0.8100, 1.5650, 2.5500 [table 3 &4]and the difference was statistically significant at $p<0.05$. The mean serum vitamin D3 level in group 1.2&3 are 40.0500, 31.5500& 17.7000 respectively[table 5 &6] and the difference amongst them were statistically significant at $p <0.05$. The mean serum CRP levels in group 1,2 &3 are 1.0300, 1.9550 & 3.7800 respectively[table 7&8] and the difference among the 3 groups were statistically significant at $p <0.05$. The correlation between SERUM CRP and vitamin D3 between group 1,2 & 3 are -.268, -.085&-.199 [table9,10,11] respectively. Even though there was an inverse correlation between two parameters but it was not statistically significant which can be attributed to the less sample size

TABLE 1

- Mean PPD of different groups analyzed using oneway ANOVA

		N	Mean	Std. Deviation	p	95% Confidence Interval for Mean		Minimum	Maximum
						Lower Bound	Upper Bound		
ppd	Healthy	20	2.9500	0.39403	<0.001	2.7656	3.1344	2.00	4.00
	Gingivitis	20	2.9500	0.51042		2.7111	3.1889	2.00	4.00
	Periodontitis	20	5.6500	1.22582		5.0763	6.2237	4.00	8.00
	Total	60	3.8500	1.50508		3.4612	4.2388	2.00	8.00

The mean difference is significant at the 0.05 level
PPD(POCKET PROBING DEPTH)

TABLE 2

Comparing mean PPD among all the 3 groups using Posthoc Tukey HSD

Dependent Variable	(I) grp	(J) grp	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
ppd	healthy	gingivitis	0.00000	0.25288	1.000	-.6085	.6085
		periodontitis	-2.70000*	0.25288	0.000	-3.3085	-2.0915
	gingivitis	healthy	.00000	0.25288	1.000	-.6085	.6085
		periodontitis	-2.70000*	0.25288	0.000	-3.3085	-2.0915
	periodontitis	healthy	2.70000*	0.25288	0.000	2.0915	3.3085
		gingivitis	2.70000*	0.25288	0.000	2.0915	3.3085

*. The mean difference is significant at the 0.05 level.

PPD(Pocket Probing Depth)

TABLE 3

Mean GI of different groups analyzed using Oneway ANOVA-

		N	Mean	Std. Deviation	p	95% Confidence Interval for Mean		Minimum	Maximum
						Lower Bound	Upper Bound		
gi	healthy	20	0.8100	0.17442		.7284	.8916	.50	1.10
	gingivitis	20	1.5650	0.22070	<0.001	1.4617	1.6683	1.20	2.00
	Periodontitis	20	2.5500	0.22361		2.4453	2.6547	2.00	3.00
	Total	60	1.6417	0.74681		1.4487	1.8346	.50	3.00

GI-Gingival Index

*. The mean difference is significant at the 0.05 level.

TABLE 4

Comparing the mean GI among all the 3 groups with Posthoc using Tukey HSD

Variable	(I) grp	(J) grp	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
gi	healthy	gingivitis	-.75500*	.06561	.000	-.9129	-.5971
		periodontitis	-1.74000*	.06561	.000	-1.8979	-1.5821
	gingivitis	healthy	.75500*	.06561	.000	.5971	.9129
		periodontitis	-.98500*	.06561	.000	-1.1429	-.8271
	periodontitis	healthy	1.74000*	.06561	.000	1.5821	1.8979
		gingivitis	.98500*	.06561	.000	.8271	1.1429

TABLE 5

Mean Vitamin-D of different groups analyzed using oneway ANOVA.

		N	Mean	Std. Deviation	p	95% Confidence Interval for Mean		Minimum	Maximum
						Lower Bound	Upper Bound		
vid_d	healthy	20	40.0500	4.38268		37.9988	42.1012	30.00	47.00
	gingivitis	20	31.5500	3.85903	<0.001	29.7439	33.3561	24.00	38.00
	Periodontitis	20	17.7000	2.86724		16.3581	19.0419	12.00	24.00
	Total	60	29.7667	9.99554		27.1845	32.3488	12.00	47.00

The mean difference is significant at the 0.05 level.

TABLE 6

Comparing the mean vitamin D among different groups with Posthoc using Tukey HSD

Dependent Variable	(I) grp	(J) grp	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
vid_d	healthy	gingivitis	8.50000*	1.18773	.000	5.6418	11.3582
		periodontitis	22.35000*	1.18773	.000	19.4918	25.2082
	gingivitis	healthy	-8.50000*	1.18773	.000	-11.3582	-5.6418
		periodontitis	13.85000*	1.18773	.000	10.9918	16.7082
	periodontitis	healthy	-22.35000*	1.18773	.000	-25.2082	-19.4918
		gingivitis	-13.85000*	1.18773	.000	-16.7082	-10.9918

*. The mean difference is significant at the 0.05 level.

TABLE 7

- Mean CRP of different groups analyzed using oneway ANOVA.

		N	Mean	Std. Deviation	p	95% Confidence Interval for Mean		Minimum	Maximum
						Lower Bound	Upper Bound		
crp	healthy	20	1.0300	0.24301		.9163	1.1437	.60	1.40
	gingivitis	20	1.9550	0.48175	<0.001	1.7295	2.1805	1.00	2.60
	periodontitis	20	3.7800	0.61866		3.4905	4.0695	2.60	4.80
	Total	60	2.2550	1.24280		1.9340	2.5760	.60	4.80

CRP- C-Reactive Protien

The mean difference is significant at the 0.05 level

TABLE 8

Comparing mean serum CRP LEVEL among different groups with Posthoc using Tukey HSD

Dependent Variable	(I) grp	(J) grp	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
crp	healthy	gingivitis	-.92500*	.14987	.000	-1.2857	-.5643
		periodontitis	-2.75000*	.14987	.000	-3.1107	-2.3893
	gingivitis	healthy	.92500*	.14987	.000	.5643	1.2857
		periodontitis	-1.82500*	.14987	.000	-2.1857	-1.4643
	periodontitis	healthy	2.75000*	.14987	.000	2.3893	3.1107
		gingivitis	1.82500*	.14987	.000	1.4643	2.1857

*. The mean difference is significant at the 0.05 level.

TABLE 9

Correlation between vitamin D and CRP level in Healthy Group

Correlations

		vid_d	crp
vid_d	Pearson Correlation	1	-.268
	Sig. (2-tailed)		.253
	N	20	20

P<0.05 is considered as significant

TABLE 10

Correlation between vitamin D and CRP level in Gingivitis Group

Correlations

		vid_d	crp
vid_d	Pearson Correlation	1	-.085

	Sig. (2-tailed)		.721
	N	20	20

P<0.05 is considered as significant

TABLE 11

Correlation between vitamin D and CRP in periodontitis Group

Correlations

		vid_d	crp
vid_d	Pearson Correlation	1	-.199
	Sig. (2-tailed)		.399
	N	20	20

P<0.05 is considered as significant

IV. Discussion.

The result obtained in this study showed positive association between vitamin D deficiency and periodontal disease which were in accordance with studies done by rajashree dasari et al (2016)[22] which concluded that low level of serum vitamin D3 is associated with chronic periodontitis and serum vitamin D3 level can be used as a marker for disease progression.

Vitamin D has been found to affect bone metabolism by enhancing the calcium and phosphorus absorption and transportation and thus protects the strength of bone [16]. It has anti- inflammatory and immune modulatory activities as well, which can cause suppression of inflammatory processes associated with the periodontal disease [17] and its deficiency can aggravate chronic inflammatory disorders. It helps in the production of cathelicidin and other defensins to act against bacterial infection [18,19] and its deficiency can results in increased risk of infections. Furthermore, polymorphism in VDR gene is found to be associated with the development of disease and alveolar bone loss and clinical attachment loss [20,21].

This study also showed the positive correlation between periodontal disease and mean CRP levels which were in accordance with studies done with Stepan Podzimek et al(2015) who stated that CRP levels increase subsequently with the severity of the periodontal disease[23].

CRP is produced in the body whenever there is an inflammatory stimulus in the body such as chronic inflammation, infection, trauma and hypoxia. Plasma level of CRP raises markedly as 100 folds or more within or < than 72 hours following inflammatory injury or infections ,hence can be an effective marker to evaluate inflammatory conditions.[24,25,26].hence periodontitis being an chronic inflammatory disorder produces an pro inflammatory cytokines which contributes to increase in inflammatory burden thus increasing the levelsof CRP in plasma.

The results obtained from this study also revealed that there is inverse correlation between vitamin D and mean CRP levels which were also supported by

Marte C. Liefaard et al 2015 [27]. Who concluded that higher the vitamin D level the decrease is seen in serum CRP levels.

Even though we got inverse correlation between crp and vitamin D levels ,this correlation was not statistically significant which might be due to less number of sample size.

vitamin D receptor is present on leukocytes, T-helper cells and monocytes. 1.25-dihydroxyvitamin D has been shown to inhibit production of inflammatory markers such as IFN-γ, IL-2, and IL-5 by T-helper 1 lymphocytes [28,29]. Vitamin D also suppress the secretion of IL-6 by monocytes, which is the prime inducing factor of CRP synthesis from liver.[30,31].

Systematic review did by mohden mazidi et al 2016[32] revealed that vitamin D supplementation has no impact on serum CRP levels which were contradictory with the results obtained in our study.

Hence further studies with larger sample size, larger duration of follow up and with more advance invasive procedures, may help in studying relationship between vitamin d and Periodontitis with more accuracy and precision.

V. Conclusion.

Deficiency of vitamin D might be an risk factor for progression of periodontal diseases. Hence further studies with larger sample size, larger duration of follow up and with more advance invasive procedures, may help in studying relationship between vitamin d and Periodontitis with more accuracy and precision

Reference

[1]. Ko JA, Lee BH, Lee JS, Park HJ. Effect of UV-B exposure on the concentration of vitamin D2 in sliced shiitake mushroom (*Lentinus edodes*) and white button mushroom (*Agaricus bisporus*). *J Agric Food Chem.* 2008;56(10):3671-3674.
[2]. Adams JS, Hewison M. Update in Vitamin D. J Clin Endocrinol Metab 2010;95:471-8.
[3]. Walters MR. Newly identified actions of the Vitamin D endocrine system. Endocr Rev 1992;13:719-64.
[4]. D'Ambrosio D, Cippitelli M, Cocciolo MG, Mazzeo D, Di Lucia P, Lang R, *et al.* Inhibition of IL-12 production by 1,25-dihydroxyvitamin D3. Involvement of NF-kappaB downregulation in transcriptional repression of the p40 gene. J Clin Invest 1998;101:252-62.
[5]. Anand N, Chandrasekaran SC, Rajput NS. Vitamin D and periodontalhealth: Current concepts. J Indian Soc Periodontol 2013;17:302-8.
[6]. Holick MF. Vitamin D deficiency. N Engl J Med 2007;357:266-81.
[7]. Garcia N, Miley D, Dixon DA. Vitamin D and periodontal disease. In: Watson RR, editor. Handbook of Vitamin D in Human Health: Prevention, Treatment and Toxicity. Wageningen Academic Publishers; 2013. p. 243-53.
[8]. Boggess KA, Espinola JA, Moss K, Beck J, Offenbacher S, Camargo CA Jr. Vitamin D status and periodontal disease among pregnant women. J Periodontol 2011;82:195-200.
[9]. Dietrich T, Joshipura KJ, Dawson-Hughes B, Bischoff-Ferrari HA. Association between serum concentrations of 25-hydroxyvitamin D 3 and periodontal disease in the US population1-3. Am J Clin Nutr 2004;80:108-13.
[10]. Hildebolt CF. Effect of Vitamin D and calcium on periodontitis. J Periodontol 2005;76:1576-87.
[11]. JosephR, NagraleAV, JoserajMG, Pradeep KumarKM, KaziyarakathJA, Chandini R. Low levels of serum Vitamin D in chronic periodontitis patients with type 2 diabetes mellitus: A hospital-based cross-sectional clinical study. J Indian Soc Periodontol 2015;19:501-6.
[12]. Tomar N, Gupta C, Kaushik M, Wadhawan A. Nutrigenomics: A perio-nutrition interrelationship. J Oral Rev Res 2017;9:32-6.
[13]. Shiva G, Deepa D. Applications of ozone therapy in dentistry. J Oral Rev Res 2016;8:86-91.
[14]. Buhlin K, Gustafsson A, Pockley AG, Frostegard J and Klinge B. Risk factors for cardiovascular disease in patients with periodontitis. *Eur Heart J* 2003; 24:2099-107.
[15]. Craig RG, Yip JK, So MK, Boylan RJ, Socransky SS and Haffajee AD. Relationship of destructive periodontal disease to the acute phase response. *J Periodontol* 2003; 74:1007-16..
[16]. DeLuca HF. The Vitamin D story: A collaborative effort of basic science and clinical medicine. FASEB J 1988;2:224-36.
[17]. Kamen DL, Tangpricha V. Vitamin D and molecular actions on the immune system: Modulation of innate and autoimmunity. J Mol Med (Berl) 2010;88:441-50.
[18]. Dimeloe S, Nanzer A, Ryanna K, Hawrylowicz C. Regulatory T cells, inflammation and the allergic response-The role of glucocorticoids and Vitamin D. J Steroid Biochem Mol Biol 2010;120:86-95
[19]. Wang TT, Nestel FP, Bourdeau V, Nagai Y, Wang Q, Liao J, *et al.*Cuttingedge:1,25-dihydroxyvitamin D3 is a direct inducer of antimicrobial peptide gene expression. J Immunol 2004;173:2909-12.
[20]. Amano Y, Komiyama K, Makishima M. Vitamin D and periodontal disease. J Oral Sci 2009;51:11-20.
[21]. Deng H, Liu F, Pan Y, Jin X, Wang H, Cao J. BsmI, TaqI, ApaI, and FokI polymorphisms in the Vitamin D receptor gene and periodontitis: A meta analysis of 15 studies including 1338 cases and 1302 controls. J Clin Nutr 2011;80:108-13.
[22]. Rajashree Dasari, Veerendranath Reddy Panthula, Sandeep Nandakumar, Rekha Rani Koduganti, Himabindu Gireddy, Sangeeta Sehrawat , Low serum 1,25(OH)2 D levels: A risk factor for periodontitis. Journal of Dr. NTR University of Health Sciences 2016;5(4).
[23]. **Stepan Podzimek et al,** C-Reactive Protein in Peripheral Blood of Patients with Chronic and Aggressive Periodontitis, Gingivitis, and Gingival Recessions. Hindawi Publishing Corporation Mediators of Inflammation Volume 2015, 7 pages.
[24].] I. S. Gomes-Filho, J. M. F. Coelho, S. S. da Cruz et al., "Chronic periodontitis and C-reactive protein levels," Journal of Periodontology, vol. 82, no. 7, pp. 969–978, 2011.
[25]. S. Black, I. Kushner, and D. Samols, "C-reactive protein," The Journal of Biological Chemistry, vol. 279, no. 47, pp. 48487–48490, 2004.
[26]. T. Bansal, A. Pandey, D. Deepa, and A. K. Asthana, "C-reactive protein (CRP) and its association with periodontal disease: a brief review," Journal of Clinical and Diagnostic Research, vol. 8, no. 7, pp. E21–E24, 2014.
[27]. : Liefaard MC, Ligthart S, Vitezova A, Hofman A, Uitterlinden AG, Kiefte-de Jong JC, et al. (2015) Vitamin D and C-Reactive Protein: A Mendelian Randomization Study. PLoS ONE 10(7): e0131740. doi:10.1371/journal.pone.0131740.
[28]. Cantorna MT, Zhu Y, Froicu M, Wittke A. Vitamin D status, 1,25-dihydroxyvitamin D3, and the immune system. Am J Clin Nutr. 2004; 80(6 Suppl):1717S–20S. PMID: 15585793.
[29]. Mahon BD, Wittke A, Weaver V, Cantorna MT. The targets of vitamin D depend on the differentiation and activation status of CD4 positive T cells. J Cell Biochem. 2003; 89(5):922–32. PMID: 12874827.
[30]. Zhang Y, Leung DY, Richers BN, Liu Y, Remigio LK, Riches DW, et al. Vitamin D inhibits monocyte/ macrophage proinflammatory cytokine production by targeting MAPK phosphatase-1. J Immunol. 2012; 188(5):2127–35. doi: 10.4049/jimmunol.1102412 PMID: 22301548.
[31]. Dickie LJ, Church LD, Coulthard LR, Mathews RJ, Emery P, McDermott MF. Vitamin D3 down-regulates intracellular Toll-like receptor 9 expression and Toll-like receptor 9-induced IL-6 production in human monocytes. Rheumatology (Oxford). 2010; 49(8):1466–71.
[32]. mohden mazidi et al 2016 Impact of vitamin D supplementation onC-reactive protein; a systematic review andmeta-analysis of randomized controlledtrials. BMC Nutrition (2018) 4:1

Syed Owais Ahmed, et. al. "To Evaluate And To Assess The Relationship Between Vitamin - D & Chronic Periodontitis A Clinico Biochemical Study." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 19(12), 2020, pp. 06-11.